



CONTENT

ORIGINAL ARTICLES

The prevalence of erectile dysfunction in male patients with diabetes mellitus in Rwanda: A cross-sectional study

Prevalence and risk factors of malaria and human immunodeficiency virus co-infection among pregnant women at Sokoto, Nigeria

Clinical presentation of splenomegaly at Kigali University Teaching Hospital, Rwanda -A retrospective descriptive study

Prevalence of primary infertility caused by chromosomal abnormalities and assessment of clinical manifestations in Rwandan patients

EDITORIAL

Non-Communicable Diseases-Challenging Agenda for The Rwandan Health Sector

CASE REPORTS

Disseminated Cysticercosis in Rwanda: A Case Report of a Patient Presenting with Difficulty with Walking and Skin Nodules

Severe Anemia by a Leech Infestation in a Pediatric Patient: A Case Report

Bedside Ultrasound Scan in the intensive care unit of a referral hospital in Kigali, Rwanda: case reports and a review of the literature-Case Series

Rwanda Medical Journal

The **Rwanda Medical Journal (RMJ)**, is a not-for-profit scientific medical journal created in 1967. It is published entirely online in open-access electronic format and is indexed by Bioline International.

Aim and Scope

The Rwanda Medical Journal aims at offering in-depth analysis of health-related issues from a Professional and research centered perspective. In this light, the journal will address fundamental concerns and dilemmas encountered in the health sector in Rwanda, acknowledging the multi-faceted nature of problems and solutions. It should reflect agreements and differences in goals and point of view among health professionals in order to foster communication and cooperation, new thinking and action, and new form of consensus.

The RMJ is an interdisciplinary research journal for publication of original work in all the health disciplines. Through a rigorous process of evaluation and peer review, The RMJ strives to publish original works of high quality for a diverse audience of healthcare professionals. The Journal seeks to deepen knowledge and advance scientific discovery to improve the quality of care of patients in Rwanda and internationally.

A new issue is published quarterly with supplements and publication materials are submitted online on [RMJ submission portal](#) and should fulfill the RMJ's instructions. Before submitting please ensure that you have reviewed [the author instructions](#). All articles that do not comply with the author instructions will be returned to the author for amendment.

PUBLISHER

Rwanda Biomedical Centre-Rwanda Health Communication Center (RBC-RHCC)
Kigali, Rwanda

CONTACTS

Email: rwandamedicaljournal@gmail.com

Website: <http://www.rwandapublichealthbulletin.org/>

ADDRESS

Rwanda Medical Journal Secretariat
KG 203St.
Kigali, Rwanda

EDITORS

Editor-In-Chief

Leon Mutesa, MD, PhD
*Center for Human Genetics
University of Rwanda*

Medical Editor & Editorial Assistant

Christian Nsanzabaganwa, MD
Rwanda Military Hospital, Kigali, Rwanda

Fidele Byiringiro, MD, MMed
Rwanda Military Hospital, Kigali, Rwanda

Deputy Editor-In-Chief

Peter Cartledge, MD, BSC, MBChB (Leeds), PCME, MSc
*Rwanda Human Resources for Health (HRH) Program
University Teaching Hospital of Kigali, Rwanda
Yale University*

Deputy Editor-In-Chief

Joseph Mucumbitsi, MD, MPH
*Honorary Associate Professor
Department of Pediatrics King Faisal Hospital, Kigali*

Rwanda Medical Journal

EDITORIAL BOARD MEMBERS

Jesse Raiten, MD

Anesthesiology, Critical Care, Perioperative Medicine
University of Pennsylvania, Anesthesiology and Critical Care
Department, Philadelphia, USA

Paulin Banguti, MD

Anesthesiology, Critical Care, Cardiac Anesthesiologist
College of Medicine and Health Sciences
University of Rwanda, Kigali

Vedaste Ndahindwa, MD, MSc

Biostatistics & Public health
School Public Health
University of Rwanda, Kigali

Michael Sinclair, MD

Cardiothoracic surgery
CHUB, Butare, Rwanda

Linda Baxter, CNM, MS

Clinical Educator & nursing
Great Barrington MA, USA

Maria Kidner, APRN, DNP, FNP-BC, FAANP

Clinical Educator & nursing
Essentia Health, Fargo, North Dakota, USA

Brian Swan, DDS, MPH

Dentistry
Cambridge Health Alliance, Cambridge, MA. USA

Eleana Stoufi, DDS, MSc, PhD

Dentistry, Oral Medicine & Oral Pathology
Harvard School of Dental Medicine

Ladan Basiri, MA, DMD

Dentistry
Washington DC, USA

Amelia Pousson, MD, MPH

Emergency medicine
CHUK, Kigali, Rwanda

Giles Cattermole, BM, BCH, FRCM, DTM&H

Emergency medicine & Medical ethics
King's College Hospital NHS Trust, London UK

Katie Cartledge, BSc, MBChB, DRCOG, DFSRH, RCGP

Family medicine & Medical education
International dispensary & UR, Kigali Rwanda

Tim Walker, MBBS(Hons), FRACP, MPHTM

Gastroenterology, Internal medicine & Tropical medicine.
Department of Medicine, Calvary Mater Hospital, Newcastle,
Australia.

Geldine Chironda, BSc, MSc, PhD

General Nursing, nephrology & Public Health
Human Resources for Health, Rwanda

Georges Ntakiyiruta, MD, MMed, FCS(ECSA)

General Surgery, Ejo Heza Surgical Center, Kigali
University of Rwanda

Lilian Omondi, BSc, MSc, PhD

General & Surgical Nursing
New York University School of Nursing, NY, USA
University of Rwanda, School of Nursing and Midwifery, Kigali

Florence Masaisa, MD, PhD

Hematology & Internal Medicine
College of Medicine and Health Sciences
University of Rwanda, Kigali

Claude Mambo Muvunyi, MD, PhD

Microbiology & Infectious Diseases
College of Medicine and Health Sciences
University of Rwanda, Kigali

Renee Pyburn, MSN, RN

Nursing
Rock Springs Behavioral Health,
Georgetown, TX.

Sheila Shabu, Bed, MSc, PhD

General Nursing
New York University School of Nursing and Human Resources for
Health Program, University of Rwanda School of Nursing and
Midwifery, Kigali. Rwanda

Cameron Page, MD

Internal Medicine
Brooklyn, New York, USA

Dirk Van Leeuwen, MD, PhD, FAASLD

Internal Medicine
Geisel School of Medicine at Dartmouth, Hanover NH, USA

Krs Bujarski, MD

Internal medicine & Neurology
Dartmouth-Hitchcock Medical Center, Lebanon, New Hampshire,
USA

Rwanda Medical Journal

Norbert Lameire, MD, PhD

Nephrology, Internal Medicine

Retired Professor of Medicine, University of Gent, Belgium

Joseph Friedman, MD

Neurology

New York, USA

Douglas Blackwell, MD, MPH

Pathology

St Louis University, Missouri, USA

Justin Wane, MD, MMed

Pathology

King Faisal Hospital, Rwanda

Craig Conard, MD, MPH, FAAP

Pediatrics, Community health & Medical Education

Yale University, Human Resources for Health, Rwanda

Natalie McCall, MD, MPH, FAAP

Pediatric emergency medicine

Yale University, Human Resources for Health, Rwanda

Tanya Rogo, MD, MPH&TM

Pediatrics & Infectious diseases

Brown University, Human Resources for Health, Rwanda

Stefan Jansen, MSc, PhD

Psychology & Mental Health

College of Medicine and Health Sciences

University of Rwanda, Kigali

Steven Rulisa, MD, PhD

Reproductive Health

College of Medicine and Health Sciences

University of Rwanda, Kigali

Chantal Ingabire, MSc

Social & Community Health

College of Medicine and Health Sciences

University of Rwanda, Kigali

David Skavadhl, MD

Surgery (general)

Maine Medical Center, Portland Maine USA

Jean Claude Byiringiro, MD, MMed, FCS (ECSA)

Surgery (Orthopedics)

Surgery (Education)

College of Medicine and Health Sciences

University of Rwanda, Kigali

Aline Uwimana, MD, MPH

Tropical & Infectious Diseases

Rwanda Biomedical Center

Dennis Hopkinson, MD

Rising Scholar and Internal Medicine Attending Physician, Virginia Commonwealth University

Lecturer, University of Rwanda College of Medicine and Health Sciences

US Fulbright Scholar 2018-2019, University of Rwanda

The Prevalence of Erectile Dysfunction in Male Patients with Diabetes Mellitus (DM) in Rwanda: A Cross-Sectional Study

Authors: A. Habumuremyi¹; J. Mukiza^{2,3*}; S. Habimana²; N. Koto-te-Nyiwa⁴; E. Niyigaba⁵; L. Bitunguhari^{6,7}

Affiliations: ¹Department of Internal Medicine, Kigeme District Hospital, Rwanda; ²School of Education, College of Education-University of Rwanda; ³Faculty of Nursing, University of Gitwe, Rwanda; ⁴Faculty of Medicine, University of Gbadolite, Gbadolite, Democratic Republic of the Congo; ⁵Integrated Polytechnique Regional College, Musanze (IPRC-Musanze), Rwanda; ⁶School of Medicine and Pharmacy, University of Rwanda, Rwanda; ⁷Department of Internal Medicine, Kigali University Teaching Hospital (CHUK), Rwanda

ABSTRACT

BACKGROUND: Diabetic men may experience erectile dysfunction (ED). Although commonly acknowledged to be a significant burden on diabetic men worldwide, nothing is known about the prevalence of diabetic ED in Rwanda.

The aim of this study was to determine the prevalence of ED in diabetic men in Rwanda with the focus on one private clinic (Fraternity Clinic) and 3 different public hospitals; University Teaching Hospital of Kigali (CHUK), University Teaching Hospital of Butare (CHUB), and Masaka District Hospital (MDH).

METHODS: A cross-sectional study was conducted on 125 diabetic men attending different health facilities; KUTH, BUTH, MDH and Fraternity Clinic and meeting the inclusion criteria. Data were collected from November 2017 to January 2018. During this period, 125 diabetic men between 20 and 70 years old were screened for ED by using international index of erectile function (IIEF-15) standards. Data were analyzed using the Statistical Package for Social Sciences (SPSS) version 20, and a Confidence Interval (CI) of 95% was used.

RESULTS: The median age of the study participants was 47.58 years (SD: 11.638). We found that 62.40% of patients had ED, in whom 21.60% had mild ED, 17.60% had mild to moderate ED, 15.20% had severe ED, 8% had moderate ED and 37.60% had no dysfunction.

CONCLUSION: The prevalence of ED in our study was found to increase with age. In our study, the determinants of ED were duration of diabetes mellitus, health insurance, site or health facility, level of education, and alcohol use.

Keywords (MeSH): Erectile Dysfunction, Diabetes Mellitus, Cross-Sectional Study, Impotence, Sexual Dysfunction

INTRODUCTION

DM is potentially identified as an important cause of disruption of normal sexual function in both men and women [1,2]. Erectile dysfunction is the inability to develop or maintain an erection of the penis during sexual activity [3], and it is the third most common complication of diabetes mellitus for men [1]. Poor glycemic control induces macrovascular changes, microvascular changes, neuropathy, and endothelial dysfunctions resulting

in ED for diabetic men [4]. The prevalence of ED due to diabetes mellitus worldwide occurs at 30% to 90% for diabetic men and also affects teens [2,5].

ED prevalence due to DM has been reported as 52% in the USA, 34% in Australia, 26% in Japan, 19.2% in Germany, 63% in Egypt and 54% in Morocco [5-8s] and is significantly higher compared to those without diabetes mellitus [9]. Although ED is commonly acknowledged to be a common problem in diabetic men worldwide, nothing

***Corresponding author:** Dr. Janvier Mukiza, PhD, Email: janvier.mukiza@gmail.com, Department of Internal Medicine, Kigeme District Hospital, Rwanda, School of Education, College of Education-University of Rwanda; **Potential Conflicts of Interest (Col):** All authors: no potential conflicts of interest disclosed; **Funding:** All authors: no funding was disclosed; **Academic Integrity:** All authors confirm that they have made substantial academic contributions to this manuscript as defined by the ICMJE; **Ethics of human subject participation:** The study was approved by the local Institutional Review Board. Informed consent was sought and gained where applicable; **Originality:** All authors: this manuscript is original has not been published elsewhere;

Review: This manuscript was peer-reviewed by three reviewers in a double-blind review process; **Type-editor:** Dennis Hopkinson (USA)

Received: 31st March 2019; **Initial decision given:** 11th April 2019; **Revised manuscript received:** 7th December 2019; **Accepted:** 24th February 2020

Copyright: © The Author(s). This is an Open Access article distributed under the terms of the Creative Commons Attribution License (CC BY-NC-ND) (click here), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. **Publisher:** Rwanda Biomedical Centre (RBC)/Rwanda Health Communication Center, P.O.Box 4586, Kigali.

ISSN: 2079-097X (print); 2410-8626 (online)

Citation for this article: Alain Habumuremyi, Janvier Mukiza, Sylvain Habimana et al. The prevalence of erectile dysfunction in male patients with diabetes mellitus (DM) in Rwanda: A cross-sectional study. Rwanda Medical Journal, Vol 77, no 2, pp 5-11, 2020

is known about the prevalence of diabetic ED for men in Rwanda.

There is no data to suggest that ED can be prevented by strict glycemic control [10], however patients with HbA1C above 7% have a seven times higher risk of ED compared to DM patients with good glycemic control [11]. Increased physical activity has been identified as protective against developing ED, but obesity is an independent predictor of ED [12,13] and obese men have a 1.5 to 3-fold increased risk of ED [14]. The previous studies reveal that physical activity in diabetic male patients will not only improve the quality of life but it may also help them to reduce morbidity, mortality and complications resulting from diabetes mellitus including ED [9].

Patients are often embarrassed when talking about their sexual history to the medical officer. This cross-sectional study was conducted to assess the prevalence of ED by focusing on diabetic men in Rwanda. This study was the first in Rwanda, and it provides the preliminary data for future consideration for diabetic men in Rwanda.

METHODS

Data were collected over a period of 3 months; from November 2017 to January 2018 during which 125 patients meeting inclusion criteria (See Figure 1) were screened and included in the final analysis. The study was a multicenter, cross-sectional study conducted at the three public hospitals; University Teaching Hospital Kigali (CHUK), University Teaching Hospital of Butare (CHUB), and Masaka District Hospital (MDH) and one private clinic known as Fraternity Clinic. Male inpatients and outpatients between 20 and 70 years old, with a new or previous diagnosed type I or type II DM were only included in this study, and each of the patients voluntarily agreed to sign the consent form. All patients included in this study presented the most common clinical signs of DM such as high fasting blood sugar level, polyuria, unexplained weight loss, or polydipsia. Patients severely sick (in DKA: diabetic ketoacidosis, HHS: hyperosmolar hyperglycemia state or unstable) were not identified as patients to be considered in this

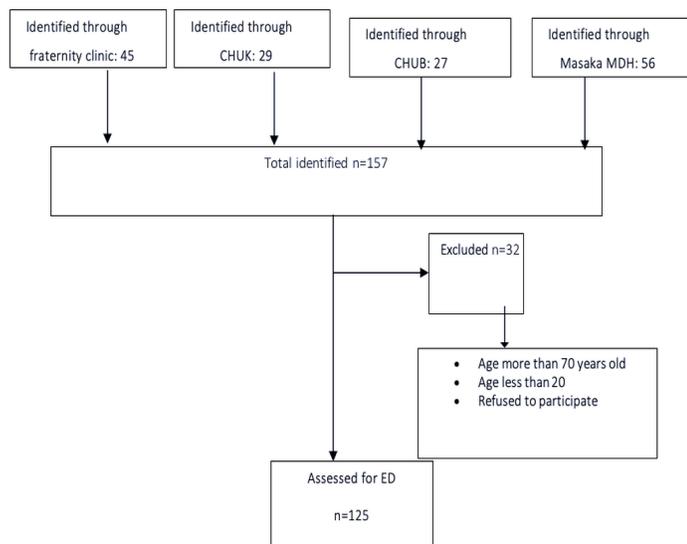


Figure 1: Diagram showing the participants selection criteria

study. For every day of consultation for non-communicable diseases, patients who agreed were seen in a private room and screened for inclusion and exclusion criteria and informed fully about the purpose of this study. We took note of demographic data (age, marital status, etc.), and social history (smoking, alcohol intake). Other information regarding medical and surgical history (hypertension and medications, heart disease and previous surgery especially pelvic surgery) were asked and documented.

A pretested questionnaire was used to obtain information and data were stored in a secure cupboard. Patient data were collected using detailed medical, sexual history, self-reported glycosylated hemoglobin. Medical officers were trained as research assistants before the commencement of the study. The questionnaires were pre-tested for the suitability of questions and necessary adjustments made. Questionnaires were checked every day for completeness by the principle investigator. The participants' sexual history was assessed using the international index of erectile function (IIEF-15) questionnaire. The IIEF-15 was translated into Kinyarwanda by a professional translator. The questionnaire was administered by the principal investigator with the help of the research assistant (nurses in charge of NCD: non-communicable diseases). Data as were analyzed by using the Statistical Package for Social Sciences (SPSS) version 20. In calculation of the sample size, we used a small percentage of patients with ED and we increased the P value for reducing the cost and P value < 0.07 was considered a significant risk factor and confidence Interval (CI) of 95% was used.

RESULTS

In this study, 125 patients meeting the inclusion criteria were screened and included in the final analysis. The median age was 47.58 years with a relatively normal distribution between 20 to 70 years and a slight skew towards older age (Figure 2).

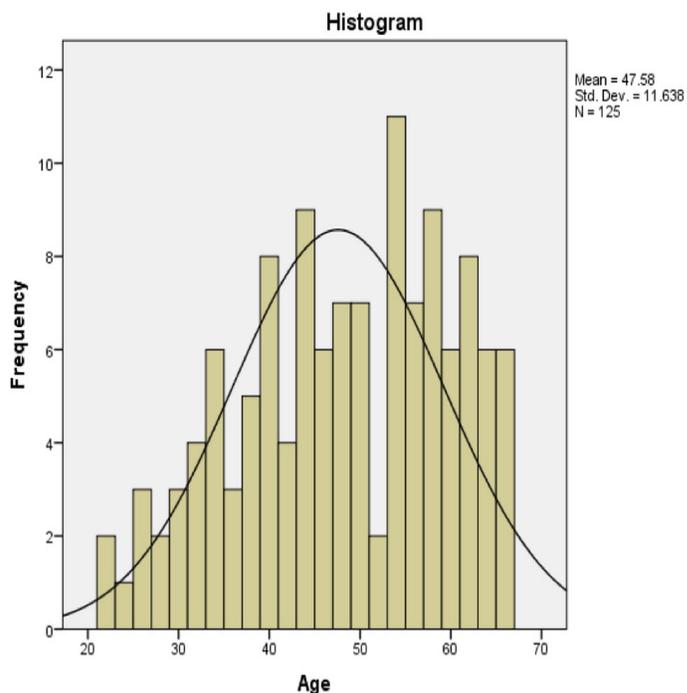


Figure 2: Distribution of age of studied sample

In Fraternity clinic, 45 patients were identified and 1 patient was older than 70 years old and another one was less than 20 years old while 8 patients refused to participate, and 10 patients from Fraternity clinic were consequently excluded in this study. In CHUK, 29 patients were identified in which, 5 patients were older than 70 years old and 5 patients refused to participate, and consequently 10 patients from CHUK were excluded in this study. In CHUB 27 patients were identified and 8 of them were excluded (refuse to participate). In Masaka DH 56 were identified 1 patient refused to participate while another one was older than 70 years old and 2 patients from Masaka DH were consequently excluded in this study. Another concern we considered only male patient, and this can explain the small number of participants.

Overall, 62.4% of patients reported some degree of erectile dysfunction, with 15.2% reporting severe dysfunction, 8% moderate dysfunction, 17.60% mild to moderate, 21.60% mild dysfunction and 37.60% had no dysfunction (Table 1). Despite nearly 2/3 of patients reporting erectile dysfunction, only 10% reported decreased satisfaction with overall sexual activity and, specifically, 33% reported orgasmic dysfunction (See Tables 1-2).

Table 1: Prevalence of erectile dysfunction

Prevalence of ED	N	%
Any	78	62.4
Severe	19	15.2
Moderate	10	8
Mild to moderate	22	17.6
Mild	27	21.6
No dysfunction	47	37.6

Severe, moderate definitions were based on the international index of erectile function

Table 2: Others domains of IIEF (International index of erectile function)

Characteristic	N	%
Orgasmic function		
Less	42	34
Normal	83	66
Intercourse satisfaction		
Moderate Impairment	30	24
Mild Impairment	55	44
Normal	40	32
Sexual desire		
Less	54	43
Normal	71	57
Overall satisfaction		
Less	13	10.4
Normal	112	89.6

Patients were mainly urban dwellers (80% vs 20%) and nearly 90% were married. 48.8% of patients had a primary school level of education and 68.8% had community-based health insurance

(See Table 3). Only 28.8% were diagnosed with DM more than 10 years ago and 3.2% were smokers, 1.6% were heavy alcohol users, 40.8% were hypertensive, 3.2% had history of a stroke, 24% had peripheral neuropathy, 57.6% had self-reported HbA1C, 36.8% were taking hypertensive drug and 7.2% had history of pelvic surgery (See Table 4).

There was no association with self-reported glycosylated hemoglobin with smoking, hypertension medication, pelvic surgery and arterial hypertension or age. There was less ED in patients using premium health insurance, patients from fraternity clinic (private) and patients with post-secondary school as level of education (Appendix 2). There was less ED in patients diagnosed with diabetes mellitus 10 years ago.

No correlation is found between ED and antihypertensive medication in our study and most of patients with ED were not on any

Table 3: Demographics data

Variables	N	%
Residency		
Countryside	25	20
Urban	100	80
Health facilities		
Fraternity clinic	35	28
CHUK	19	15.2
CHUB	19	15.2
Masaka district hospital	52	41.6
Marital status		
Married	111	88.8
Single	8	6.4
Divorced	1	0.8
Cohabiting	4	3.2
Widowed	1	0.8
Level of education		
Informal	8	6.4
Primary	61	48.8
Secondary	36	28.8
Post-secondary	20	16
Employment		
Peasant	18	14.4
Government	5	4
Private sector	31	24.8
Self-employee	53	42.4
Others	18	14.4
Health insurance		
Community based insurance	86	68.8
Premium	16	12.8
None	23	18.4

Table 4: Clinical data

Variables	N	%
Duration of diabetes		
<10years	89	71.2
>10years	36	28.8
Tobacco use: Yes	4	3.2
Alcohol use		
Never	55	44
Monthly or less	16	12.8
2 to 4times a month	40	32
2 to 3times a week	12	9.6
4 or more times a week	2	1.6
History of artery hypertension: Yes	51	40.8
History of stroke: Yes	4	3.2
Peripheral neuropathy: Yes	30	24
Glycosylated hemoglobin level		
<7%	32	25.6
>7%	72	57.6
Unknown	21	16.8
Medication of hypertension: Yes	46	36.8
History of pelvic surgery: Yes	9	7.2

hypertensive medications, and we found that severe ED was common in patients with self-reported HbA1C of more than 7% (Appendix 1).

DISCUSSION

The median age of all participants is 47.58 years and it is in accordance with that found in other similar studies [8,9,15,16-18]. The ED correlated with increasing age with 74.2% of ED are between 51 and 60 years old (Table 5), which is consistent with other similar studies [8, 19,20]. The prevalence of ED in our study (62.4%) (Table 1) was lower than that found in Saudi Arabia (84%) but notably higher than that reported in Nigeria (42%) [16,21]. First, the median age of participants in Saudi Arabia was 80 versus our population where patients were, on average, much younger and patients older than 70 years were excluded. The difference of prevalence can also be explained by difference in population characteristics and sample size. A study done in Tanzania [15] found the same association of ED with increasing age. This can be related to testosterone deficiency, atherosclerosis, psychogenic problem, decline in several organ functions or medication that induced ED [15,19].

We found that severe ED was common in patients with self-reported HbA1C more than 7% (Table 6) as shown in other studies [20,21] but the association was not significant for ED and self-reported HbA1C. Longstanding DM di was associated with ED as mentioned in several studies [15,19-21]. With HbA1C more than 7% and longer duration of diabetes mellitus, patients tend to have more microvascular, macrovascular, and neurologic complication of DM which contribute to ED pathophysiology.

Most of the patients with ED were married and it was found that there was no significant association between marital status and ED (Table 5) as found in similar previous studies [8,16,22].

In contrast to another study [6,] we found that ED is common amongst urban patients (Table 5) and it is evident that the ED can be underdiagnosed in rural areas (Table 5). This study found that 44% of patients had mild intercourse satisfaction which is similar to other findings [6]. However, the study reports that 57% (Table 2) of patients had normal sexual desire which is different from Braun and co-workers' study where sexual desire was more affected by ED [6]. It seems that other components of IIEF-15 were not affected by DM. This can be related to embarrassment of the patients when talking about sexual history.

The level of education was statistically associated with ED (Table 5). We found less ED in patients with a post-secondary primary school level of education as previously reported in the literature [8]. It is possible that patients with post-secondary level of education tend consult earlier, understand more the complications of DM and are more compliant on treatment. The prevalence of ED is higher among patients with lower income level. This finding is similar to that reported by Abdullah [20]. Without money, it is difficult to have a regular follow up and to pay medications and a glucometer. No correlation is found between ED and antihypertensive medication in our study and most of patients with ED were not on any hypertensive medications (See Table 6). This is consistent with the findings of Berrada and co-workers [8]. We found less ED in patients with premium as health insurance, and from Fraternity clinic (private clinic) (Tables 5). The difference was statistically significant for ED and health insurance, site or hospital. The reason is that most of the patients are financially stable and had high levels of education.

This study found that more patients with ED had no history of smoking (Table 6), which is different from findings of Kovac and co-workers [23]. It showed a high risk of ED in smokers and ex-smokers and this difference is due to the characteristics of the population under this study. The ED in a smoking patient is thought to be related to acceleration of atherosclerosis [23-25]. The difference is statistically significant between ED and alcohol consumption (patient taking beer 2-4 times per month) and stroke. This corresponds with the findings reported in literature for similar study [15].

CONCLUSION

This study was limited by Rwandans cultural barriers. The cultural barrier was due to reluctance of patients to talk about their sexual history. The prevalence of ED in our study is very high. ED is found to increase with age. In our study, the determinants of ED were duration of DM, health insurance, site or health facility, level of education, and alcohol use. The Rwanda Ministry of Health is recommended to do more education regarding DM and its related complications including ED. More effort needs to be put into screening and treatment as part of follow-up for diabetic patients, using the first 5 questions of international index of erectile function-5 (IIEF-5) and management of ED as well as diagnosing DM among all patients with ED are highly recommended.

Up to now, there is no standard treatment of ED in Rwanda except the treatment of the cause and risk factors. It is possible that ED in old people becomes irreversible case but young people can be recovered depending on the cause but no data that are available. ED can be stabilized using adjuvant treatment based on age, marital status and individual social life. Some drugs are available but no local study to assess the benefit. The local collaboration between the physician, urologists and gynecologists for future study is recommended for solving ED problem.

REFERENCES

1. Latini DM, Penson DF, Wallace KL, Lubeck DP, Lue TF. Longitudinal differences in psychological outcomes for Men with erectile dysfunction: Results from ExCEEDTM. *J. Sex Med.* 2006; 3(6):1068-1076.
2. Bivalacqua TJ, Hellstrom WJ, Kadowitz PJ, Champion HC. Increased expression of arginase II in human diabetic corpus cavernosum: in diabetic-associated erectile dysfunction. *Biochem. Biophys. Res. Commun.* 2001; 283(4):923-927.
3. Boris Schouten WV, Bohnen AM, Groeneveld FPMJ, Dohle GR, Thomas S, Ruud Bosch JH. Erectile Dysfunction in the Community: Trends over Time in Incidence, Prevalence, GP Consultation and Medication Use - the Krimpen Study: Trends in ED. *J. Sex Med.* 2010; 7(7):2547-2553
4. Binmoammar TA, Hassounah S, Alsaad S, Rawaf S, Majeed A. The impact of poor glycaemic control on the prevalence of erectile dysfunction in men with type 2 diabetes mellitus: a systematic review. *J. R. Soc. Med.* 2016; 0(0):1-10.
5. McCulloch DK, Campbell IW, Wu FC, Prescott RJ, Clarke BF. The prevalence of diabetic impotence. *Diabetologia.* 1980; 18(4):279-283.
6. Braun M, Wassmer G, Klotz T, Reifenrath B, Mathers M, Engelmann U. Epidemiology of erectile dysfunction: results of the "Cologne Male Survey". *Int. J. Impot. Res.* 2000; 12(16):305-311.
7. Shaeer KZM, Osegbe DN, Siddiqui SH, Razzaque A, Glasser DB, Jaguste V. Prevalence of erectile dysfunction and its correlates among men attending primary care clinics in three countries: Pakistan, Egypt, and Nigeria. *Int. J. Impot. Res.* 2003; 1:58-14.
8. Berrada S, Kadri N, Mechakra-Tahiri S, Nejari C. Prevalence of erectile dysfunction and its correlates: a population-based study in Morocco. *Int. J. Impot. Res.* 2003; 1:53-7.
9. Malavige LS, Wijesekara P, Ranasinghe P, Levy JC. The association between physical activity and sexual dysfunction in patients with diabetes mellitus of European and South Asian origin: The Oxford Sexual Dysfunction Study. *Eur. J. Med. Res.* 2015; 20(90):1-7.
10. Wessells H, Penson DF, Cleary P, Rutledge BN, Lachin JM, McVary KT, Schade DS, Sarma AV. Effect of intensive glycemic therapy on erectile function in men with type 1 diabetes in the diabetes control and complications trial/epidemiology of diabetes interventions and complications study. *J. Urol.* 2011; 185(5):1828-1834.
11. Ugwu T, Ezeani I, Onung S, Kolawole B, Ikem R. Predictors of

Data Availability

The demographic, clinical and statistical data used to support the findings of this study are included within the article.

Conflicts of interest:

The authors have no conflict of interest to declare.

Erectile Dysfunction in Men with Type 2 Diabetes Mellitus Referred to a Tertiary Healthcare Centre. *Adv. Endocrinol.* 2006; 2006:1-8.

12. Dorey G. Is smoking a cause of erectile dysfunction? A literature reviews. *Br. J. Nurs.* 2001; 10(7):455-465.
13. Cheng JY, Ng EM, Ko JS, Chen RY. Physical activity and erectile dysfunction: meta-analysis of population-based studies. *Int. J. Impot. Res.* 2007; 19(3):245-252.
14. Chitaley K, Kupelian V, Subak L, Wessells H. Diabetes, obesity and erectile dysfunction: field overview and research priorities. *J Urol.* 2009; 18(6): S45-S50.
15. Mutagaywa RK, Lutale J, Aboud M, Kamal BA. Re: Prevalence of erectile dysfunction and associated factors among diabetic men attending diabetic clinic at Muhimbili National Hospital in Dar-es-Salaam, Tanzania. *J. Urol.* 2015; 193(4):1325-1326.
16. Oyelade BO, Jemilohun AC, Aderibigbe SA. Prevalence of erectile dysfunction and possible risk factors among men of South-Western Nigeria: a population based study. *Pan. Med. J.* 2016; 20(124):1-8.
17. Chaudhary RK, Shamsi BH, Tan T, Chen H-M, Xing J-P. Study of the relationship between male erectile dysfunction and type 2 diabetes mellitus/metabolic syndrome and its components. *J. Int. Med. Res.* 2016; 44(3):718-727.
18. Ghalayini IF, Al-Ghazo M a, Al-Azab R, Bani-Hani I, Matani YS, Barham a-E, et al. Erectile dysfunction in a Mediterranean country: results of an epidemiological survey of a representative sample of men. *Int. J. Impot. Res.* 2010; 22(3):196-203.
19. Fung MM, Bettencourt R, Barrett-Connor E. Heart disease risk factors predict erectile dysfunction 25 years later: the Rancho Bernardo study. *J. Am. Coll. Cardiol.* 2004; 43(8):1405-1411.
20. Abdullah TIM. Erectile Dysfunction and Other Sexual Activity Dysfunctions among Saudi Type 2 Diabetic Patients. *Int. J. Health Sci Qassim Univ.* 2014; 8(4): 347-359.
21. Skeldon SC, Detsky AS, Goldenberg SL, Law MR. Erectile dysfunction and undiagnosed diabetes, hypertension, and hypercholesterolemia. *Ann. Fam. Med.* 2015; 13(4):331-335.
22. Majzoub A, Arafa M, Al-Said S, Dabbous Z, Aboulsoud S, Khalafalla K, Elbardisi H. Premature ejaculation in type II diabetes mellitus patients: association with glycemic control. *Transl. Androl. Urol.* 2016; 5(2): 248-254.
23. McVary K. Lower urinary tract symptoms and sexual dysfunction: epidemiology and pathophysiology. *BJU Int.* 2006; 2:23-28.
24. Kovac JR, Labbate C, Ramasamy R, Tang D, Lipshultz LI. Effects of cigarette smoking on erectile dysfunction. *Andrologia.* 2015; 47(10):1087-1092.
25. Arrellano-Valdez F, Urrutia-Osorio M, Arroyo C, Soto-Vega E. A comprehensive review of urologic complications in patients with diabetes. *SpringerPlus.* 2014; 3(549):1-8.

26. Salman M, Shehzadi N, Khan MT, Islam M, Amjad S, Afzal O, et al. Erectile dysfunction: Prevalence, risk factors and involvement of

antihypertensive drugs intervention. Trop. J. Pharm. Res. 2016; 15(4):869-876.

Appendix 1: Univariate analysis between clinical data and ED

Characteristics	Erectile dysfunction 78(62.4)	No Erectile dysfunction 47 (37.6)	OR (95% CI)	P-value
HbA1C level				
<7%	18(56.2)	14(43.7)	Ref	
>7%	46(63.8)	26(36.1)	1.38(0.54-3.50)	0.46
Unknown	14(66.6)	7(33.3)	1.56(0.43-5.73)	0.45
Duration of DM				
<=10years	50(56.1)	39(43.8)	Ref	
>10years	28(77.7)	8(22.2)	2.73(1.04-7.35)	0.03
Tobacco use				
Yes	3(75)	1(25)	1.84(0.16-47.34)	
No	75(61.9)	46(38)	Ref	
Alcohol use				
Never	42(76.3)	13(23.6)	Ref	
Monthly or less	9(56.2)	7(43.7)	0.40(0.11-1.48)	0.12
2 to 4 times a month	18(45)	22(55)	0.25(0.009-067)	0.002
2 to 3 times a week	7(58.3)	5(41.6)	0.43(0.10-1.92)	0.21
4 or more times a week	2(100)	0(0.00)	1.59 (indefinite)	0.43
History of HTN				
No	43(58.1)	31(41.8)	Ref	
Yes	35(68.6)	16(31.3)	1.58(0.70-3.58)	0.23
History of stroke				
No	76(62.8)	45(37.1)	Ref	
Yes	2(50)	2(50)	0.59(0.06-6.14)	0.61
Peripheral neuropathy				
No	58(61)	37(38.9)	Ref	
Yes	20(66.6)	10(33.3)	1.28(0.50-3.31)	0.58
Medication of hypertension				
No	47(59.4)	32(40.5)	Ref	
Yes	31(67.3)	15(32.6)	1.41(0.61-3.24)	0.38
History of pelvic surgery				
No	73(62.9)	43(37)	Ref	
Yes	5(55.5)	4(44.4)	0.74(0.16-3.49)	0.66

Appendix 2: Univariate analysis between demographic data and ED

Characteristics	Erectile Dysfunction	No Erectile dysfunction	OR (95% CI)	P-value
	78(62.4)	47 (37.6)		
Age [years]				
20-30	5 (45.4)	6 (54.5)	Ref	
31-40	14 (53.8)	12 (46.1)	1.40 (0.28-7.24)	0.64
41-50	19 (57.5)	14 (42.4)	1.62 (0.34-7.94)	0.49
51-60	26 (74.2)	9 (25.7)	3.47 (0.70-17.97)	0.08
61-70	14 (70)	6 (30)	2.80 (0.48-17.26)	0.19
Residency				
Countryside	15 (60)	10 (40)	Ref	
Urban	63 (63)	37 (37)	1.14 (0.42-3.03)	0.78
Site				
Fraternity clinic	11 (31.4)	24 (68.5)	Ref	
CHUK	15 (78.9)	4 (21)	8.18 (1.90-38.31)	0.001
CHUB	10 (52.6)	9 (47.3)	2.42 (0.67-9.01)	0.13
Masaka DH	42 (80.7)	10 (19.2)	9.16 (3.07-28.31)	0.001
Marital status				
Married	68 (61.2)	43 (38.7)	0.95 (0.17-4.89)	0.96
Single	5 (62.5)	3 (37.5)	Ref	
Divorced	1 (100)	0 (0.00)	1.91 (indefinite)	0.45

Characteristics	Erectile Dysfunction	No Erectile dysfunction	OR (95% CI)	P-value
	78(62.4)	47 (37.6)		
Cohabiting	3 (75)	1 (25)	1.80 (0.07-71.23)	0.67
Widowed	1 (100)	0 (0.00)	1.91 (indefinite)	0.45
Level of education				
Informal 6(75)		2 (25)	1.26 (0.20-10.1)	0.79
Primary	43(70.4)	18 (29.5)	Ref	
Secondary	22(61.1)	14 (38.8)	0.66 (0.25-1.71)	0.34
Postsecondary	7(35)	13 (65)	0.23 (0.07-0.74)	0.006
Employment				
Farmer	13(72.2)	5(27.7)	Ref	
Government	3(60)	2(40)	0.58(0.05-7.03)	0.6
Private sector	14(45.1)	17(54.8)	0.32(0.07-1.29)	0.07
Self-employee	34(64.1)	19(35.8)	0.69(0.18-2.53)	0.53
Others	14(77.7)	4(22.2)	1.35(0.24-7.88)	0.7
Health insurance				
Community based insurance	63(73.2)	23(26.7)	Ref	
Premium	5(31.2)	11(68.7)	0.17(0.004-0.59)	0.002
None	10(43.4)	13(56.5)	0.28(0.10-0.8)	0.009

Prevalence and Risk Factors of Malaria and Human Immunodeficiency Virus Co-Infection among Pregnant Women in Sokoto State, Nigeria

Authors: Chisom. E. Okechukwu¹; Idris. N. Abdullahi^{2,*}; D. Aliyu³; M. Kabiru¹; H. Aderisayo Adekola⁴; Emeka. I. Ikeh⁴; Thompson H.I. Spencer¹; Ngwoke. C. Chinedu¹

Affiliations: ¹Department of Medical Microbiology, Faculty of Medical Laboratory Sciences, Usmanu Danfodiyo University, Sokoto; ²Department of Medical Laboratory Science, Faculty of Allied Health Sciences, Ahmadu Bello University, Zaria, Nigeria; ³Department of Medical Laboratory Science, University of Calabar, Calabar, Nigeria; ⁴Department of Microbiology, Olabisi Onabanjo University, Ogun, Nigeria; ⁵Department of Medical Microbiology, Faculty of Medical Sciences, University of Jos, Jos, Nigeria

ABSTRACT

BACKGROUND: Malaria in pregnant women is a significant cause of obstetric morbidity especially when there is co-infection with human immunodeficiency virus (HIV). This cross-sectional study aimed to determine the prevalence of malaria parasitaemia and associated risk factors among HIV infected pregnant women in Sokoto State, North-Western Nigeria.

METHODS: 103 HIV infected pregnant women attending antenatal clinics of Sokoto state secondary hospitals were enrolled for this study. The socio-demographic variables and risk factors of malaria were assessed from all participants using int questionnaires. Malaria parasitaemia was detected using World Health Organization malaria microscopy protocol while CD4+ T cell count was performed using FASC count analyser.

RESULTS: 58 out of 103 (56.3%) pregnant women were infected with malaria parasites. All were *P. falciparum*. There was no significant association between malaria parasitaemia and all sociodemographic variables and risk factors of participants ($p > 0.05$). The mean (\pm standard deviation) CD4+ T-cell counts for pregnant women with malaria-HIV co-infection and HIV mono-infection were 127 \pm 45 cells/mm³ and 322 \pm 62 cells/mm³, respectively. The CD4+ T-cell counts of subjects with HIV/malaria co-infection were significantly ($p < 0.001$) lower than those with HIV mono-infection.

CONCLUSION: The prevalence of malaria recorded in this study is high, but with negative findings with regards to all socio-demographic variables of participants and risk factors of malaria.

Keywords: Malaria, Coinfection, Lymphopenia, Risk, HIV, Pregnancy

INTRODUCTION

Malaria is a well-known tropical parasitic disease responsible for high morbidity and mortality in sub-Saharan Africa. The Plasmodium species, the etiological agents of malaria are transmitted by the blood meal of female anopheles mosquito at dawn to dusk. So far, six Plasmodium species have been identified to cause human diseases, and these include *P. falciparum*, *P. vivax*, *P. ovale*, *P. knowlesi*, *P. simium* and *P. malariae* [1]. Plasmodium

falciparum is the most virulent species responsible for severe form of disease in humans [2]. Severe malaria is a multi-system disorder, which may arise from several pathological processes which include abrupt hemolysis of infected red blood cells and dyserythropoiesis. In addition, interaction of malaria infection with other infectious agents and nutritional deficiencies encourage severe malaria [2]. Malaria, human immunodeficiency virus infection (HIV) and tuberculosis are the three most important communicable diseases of developing countries [3,4].

***Corresponding author:** Mr. Idris Nasir Abdullahi, Department of Medical Laboratory Science, Faculty of Allied Health Sciences, Ahmadu Bello University, Zaria, Nigeria, Email: inabullahi@abu.edu.ng; **Potential Conflicts of Interest (Col):** All authors: no potential conflicts of interest disclosed; **Funding:** All authors: no funding was disclosed; **Academic Integrity:** All authors confirm that they have made substantial academic contributions to this manuscript as defined by the ICMJE; **Ethics of human subject participation:** The study was approved by the local Institutional Review Board. Informed consent was sought and gained where applicable; **Originality:** All authors: this manuscript is original has not been published elsewhere;

Review: This manuscript was peer-reviewed by three reviewers in a double-blind review process; **Type-editor:** Dennis Hopkinson (USA)

Received: 30th August 2019; **Initial decision given:** 6th September 2019; **Revised manuscript received:** 16th January 2020; **Accepted:** 17th January 2020

Copyright: © The Author(s). This is an Open Access article distributed under the terms of the Creative Commons Attribution License (CC BY-NC-ND) (click here), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. **Publisher:** Rwanda Biomedical Centre (RBC)/Rwanda Health Communication Center, P.O.Box 4586, Kigali.

ISSN: 2079-097X (print); 2410-8626 (online)

Citation for this article: C. E. Okechukwu, I. N. Abdullahi, D. Aliyu et al. Prevalence and Risk Factors of Malaria and HIV Co-infection among Pregnant Women at Sokoto, Nigeria. Rwanda Medical Journal, Vol 77, no 2, pp 12-16, 2020

Malaria and HIV co-infection is a topical clinical issue in sub-Saharan Africa especially in pregnant women and this can lead to poor obstetric outcomes if not properly managed [5, 6]. Malaria cases tend to increase each year in low- and middle-income countries due to poor healthcare delivery systems, emergence of antimalarial and insecticide resistance, and climate change [7].

Both malaria and HIV infection accounted for over 2 million deaths each year [7]. Malaria and HIV co-infections overlap, primarily in sub-Saharan Africa, Southeast Asia and South America [7]. In sub-Saharan Africa alone, an estimated 25 million people harbour HIV and more than 350 million episodes of malaria (in both HIV seronegative and seropositive persons) occur yearly [8]. HIV increases the risk of malaria infection and the development of clinical malaria [8]. Conversely, malaria infection increases HIV replication rates [8].

Nigeria is the most populous country in Africa with a population of over 180 million, and has the highest number of cases of malaria and HIV infection with a prevalence of 1.4% [9, 10]. HIV/AIDS can increase the adverse effects of malaria in pregnancy, which include anaemia, placental malaria and low birth weight [9, 11]. Individuals considered semi-immune to malaria in endemic regions of tropical countries can also develop clinical malaria, especially if they are HIV co-infected [11]. It has been shown that pregnant women experience reduced immunity to malaria, making them more prone to episodes of severe malaria and subsequent anaemia [12]. Another study reported higher susceptibility of malaria in pregnant women who had never or less episodes of malaria prior to pregnancy [13].

The immunopathogenesis of malaria is associated with a pro-inflammatory state, providing an ideal condition for the spread and replication of HIV in CD4+ T cells thus enhancing their destruction and reduction in count [14-16].

Due to the clinical and public health significance of HIV/malaria coinfection, this study sought to investigate the prevalence of malaria parasitemia and associated risk factors among HIV infected pregnant women in Sokoto, North-Western Nigeria

METHODS

Study design and Site: This cross-sectional study was conducted in 103 HIV infected pregnant women attending antenatal clinics of three secondary hospitals in Sokoto State: Specialist Hospital, Maryam Abacha Hospital, and Women and Children Welfare Clinic in Sokoto State.

The Sokoto township is in the dry Sahel zone surrounded by sandy terrain and isolated hills. Rainfall starts in June and ends in September, sometimes extending into October. The average annual rainfall is 55 cm with peak rainfall occurring in August. The highest temperatures (up to 45°C) occur during the hot season in March and April. Harmattan, a dry, cold, and dusty weather phenomenon is experienced between November and February. Malaria transmission is meso-endemic from September to December and hyperendemic from January to August [17, 18]. This study was conducted between the 20th of April and the 20th of

September 2017, which coincides with the wet season, which is presumptively to have high malaria transmission rate [17].

Participants were between the ages of 18 and 50 years. They were all screened for required inclusion criteria, and their HIV status was confirmed using Uni-Gold Recombigen® HIV-1/2 (Trinity Biotech, Ireland) and Determine™ (Alere, New Zealand) proprietary reagents.

Informed consent and ethical approval: The study was explained to the potential participants, and participants who wishes to enroll provided written informed consent. An interviewer-based questionnaire was administered by trained nurses and research assistants to obtain sociodemographic and risk factors variables from study participants. These questions included information on obstetric history, use of insecticide-treated bed nets (ITNs), and malaria chemoprophylaxis. We also reviewed hospital cards and folders of participants to access information such as gravidity and gestational age of the subjects. This study was conducted in accordance with the Declaration of Helsinki. Ethical approval was obtained from the Ethical Research Committee of Sokoto State Ministry of Health, Nigeria. Data generated from participants were anonymously analyzed throughout the study.

Eligibility Criteria: This study included patients who were HIV positive and pregnant. Women with febrile illness who had history of viral hepatitis and pulmonary tuberculosis were excluded.

Sample size calculation: The sample size for this study was calculated using the 10.6% prevalence of malaria parasitemia in pregnant women from a previous cross-sectional study in Sokoto, Nigeria, by Buhari et al [18]. Thus, the minimum sample size required for this study was 100 using a 5% error margin and 95% confidence interval. However, a total of 103 volunteers were enrolled for this study.

Laboratory analytical methods: Two milliliters of peripheral blood were collected through venipuncture from all recruited HIV pregnant women and immediately taken (within 1 hour) to the laboratory at the Specialist Hospital for detection of malaria parasites. Samples were analysed in batches. Using a clean grease-free microscope slide, a small drop of blood was placed to the center of the slide and the blood was spread to create the thick smear. After drying, the slides were stained for 10–15 min with 10% Giemsa solution. When the thick film was dry, a drop of immersion oil was placed to an area of the film which appears mauve colored (usually around the edges). The slides were examined for malaria parasites and malaria pigment. At least 100 high power (x100 objective) microscope fields were examined for parasites. A slide was considered negative when 100 high-power fields were examined under oil immersion objective, as described by Adefioye et al [19]. Quality control slides (positive and negative) were used by trained microscopists during malaria investigation.

Determination of CD4+ cell count: Based on the manufacturer's instructions, the CD4+ cell counts in the whole blood were analyzed using a FASC count analyser BD™ (ThermoFisher, Paisley, UK). This device used the principle of light scattering property (based

on dissimilarity in cell size or granularity) and the fluorescence of cells following staining with monoclonal antibodies to markers on the cell surface bound to fluorescent dyes. Cell populations of interest were then gated after identification. Absolute CD4+ cell counts were subsequently analyzed using a single-platform technique.

Statistical analysis: Data were analyzed using SPSS software version 24 (IBM Corporation, Armonk, NY, USA) and were presented as percentages. Two tailed Chi-square and unpaired T test were used to assess the association and difference between categorical and continuous variables. Univariate logistic regression was used to determine the odds ratio and relative risk of malaria parasitemia. P values ≤ 0.05 at a confidence interval of 95% were considered statistically significant.

RESULTS

Malaria prevalence was 56.3%. All were *P. falciparum*. Almost 50% of the participants in the study were in the age-group of 25-30 (Table 1). The highest prevalence of 71.4 % was found among those in the age group of 37-42. The age groups of 18-24, 25-30, 31-36, 43-48 had prevalences of 45%, 54.9%, 52.9% and 0.0%, respectively (Table 2).

Table 1: Socio-demographic variables of study participants

Variables	Frequency	%	
Age (years)	18-24	20	19.4
	25-30	51	48.5
	31-36	17	16.5
	37-42	14	14.5
	43-48	1	1.1
Household size	1-3	80	77.7
	4-6	18	17.5
	Above 6	5	4.5
Level of education	Primary	12	11.7
	Secondary	58	56.3
	Tertiary	4	3.9
	Religious	29	28.2
Occupation	Farmers	0	0
	Traders	21	21.4
	Civil servants	1	1.0
	Housewife	80	77.7
	Number of previous pregnancies	None	8
	1	34	33.0
	2	34	33.0
	3	10	9.7
	4 and above	17	16.5
Gestational period	1 st Trimester	26	25.2
	2 nd Trimester	16	15.5
	3 rd Trimester	61	59.2

The prevalence of malaria parasitaemia was highest among participants with household size of 4-6 persons (61.1%), followed by those with household size of 1 – 3 persons (56.3%) and least in those with household size of ≥ 6 persons (40%). Over 50%

Table 1: Prevalence of Malaria/ HIV Co-infection by Sociodemographic Variables of Participants

Variables	No. examined	No. infected	% Infected	P-value	
Age group (years)	18-24	20	9	45	0.335
	25-30	51	28	54.9	
	31-36	17	9	52.9	
	37-42	14	10	71.4	
	43-48	1	0	0.0	
Household Size	1-3	80	45	56.3	0.701
	4-6	18	11	61.1	
	Above 6	5	2	40	
Level of education	Primary	12	7	58.3	0.565
	Secondary	58	32	55.2	
	Tertiary	4	1	25.0	
	Religious	29	18	68.1	
Occupation	Traders/Business	22	15	68.2	0.285
	Civil servant	1	1	100.0	
	Housewife	80	42	52.5	
Number of previous pregnancies	None	8	6	75.0	0.228
	1	34	14	41.2	
	2	34	20	58.8	
	3	10	7	70.0	
	4 and above	17	11	64.7	
Gestational age	1 st Trimester	26	19	73.1	0.121
	2 nd Trimester	16	9	56.3	
	3 rd Trimester	67	30	69.3	

of the participants had only secondary education. The prevalence of malaria was higher among those with only religious education, 68.1%, while in those with primary education, secondary and tertiary the prevalences were 58.3%, 55.2% and 25.0% respectively.

Table 3: Risk factor of Malaria parasitemia in HIV infected pregnant women

Variables	No. examined	No. infected (%)	aOR	RR	
Knowledge and use of ART	Yes	1	0 (0.0)	0.234	0.256
	No	102	58 (56.9)		
Insecticide treated net	Yes	31	16 (51.6)	0.76	0.83
	No	72	42 (58.3)		
Use of Sulfadoxine/pyrimethamine (Prophylaxis)	Yes	8	3 (37.5)	0.44	0.47
	No	95	55 (57.9)		
Store water on open containers	Yes	70	37 (52.9)	0.64	0.87
	No	33	21 (63.6)		
Resident in proximity to gutters, refuse dumpsites (≤ 50 meters)	Yes	73	43 (58.9)	1.43	1.11
	No	30	15 (50.0)		

Analyzed by logistics regression analysis (univariate)

Over 75% of participants were housewives, and this group had a prevalence of 52.5% (Table 2). The prevalence amongst primigravidae was high at 75%, while for secundigravidae the prevalence was 41.2% and the prevalence for multigravidae was 64.5%. More than half of the participants were in their third trimesters. Prevalence of malaria seemed to be higher in the first trimester at 73.1% while second and third trimesters malaria prevalence was 56.3% and 49.2%, respectively. Only one participant knew which antiretroviral agents she was prescribed and was adherent. 56.9% of subjects had no knowledge of which antiretroviral agents they were prescribed. There was a significant association between malaria and history of fever ($P = 0.042$). Prevalence of malaria in those with a history of fever was 64.1% and in those without a history of fever was 43.5%. There was a prevalence of 51.6% in those who used ITNs while

those who do not use ITNs had a malaria prevalence of 58.3%.

Prevalence of malaria amongst non-usage of sulfadoxine/pyrimethamine is higher (57.9%) than those who used sulfadoxine/pyrimethamine (Table 3). However, after univariate analysis, subjects who reside in proximity to gutters and refuse dumpsites had higher Odd ratio for malaria parasitaemia than those who do not [OR: 1.43 (95%: 0.61-3.37)] (Table 3).

The mean (standard deviation) CD4+ T-cell counts for pregnant women with malaria-HIV co-infection and HIV mono-infection were 127 ± 45 cells/mm³ and 322 ± 62 cells/mm³, respectively. The CD4+ T-cell counts of subjects with HIV/malaria co-infection were significantly (p -value = 0.000) lower than those with HIV mono-infection (Table 4).

Table 4: Comparison of CD4+ T-lymphocyte count of subjects with and without malaria

Group	CD4+ T-cell counts (cells/mm ³)		T-test	P value
	Range	Mean \pm SD		
Participants with HIV/malaria co-infection (n =58)	59 – 439	127 \pm 45		
HIV infected participants without malaria (n =45)	82 – 654	322 \pm 62	8.49	<0.001*

*Significance determined by student t-test

DISCUSSION

The present study has yielded some important findings in regard of malaria and HIV coinfections among pregnant women in North-western Nigeria. The increased risk for severe malaria in pregnant women and HIV-infected persons already has been reported in some areas in Nigeria [19, 20].

This present study shows 56.3% prevalence of Malaria in HIV infected pregnant woman, which is slightly higher than a similar study conducted by Adeoti et al [21] where they reported prevalence of 30.2%. The prevalence was also higher when compared to the malaria prevalence of 52.2% among apparently healthy pregnant women attending Antenatal clinics in Sokoto, Nigeria [22]. The increase in prevalence from the present study may be because of HIV/AIDS in our subjects. It has been shown that HIV increases the risk of contracting malaria in an endemic setting [22].

Findings from this study showed that no significant association between all the sociodemographic variables, risk factors and prevalence of malaria parasitemia. Hence, this is a negative result. This study shows that the mean CD4+ T cell count of pregnant women with malaria/ HIV coinfection was significantly lower than those who had HIV without malaria. This aligns

with the previous findings of Nasir et al [23]. The relatively low CD4+ T cell count in the HIV/malaria coinfecting subjects could be due to lack of prescribed antiretroviral therapy or lack of adherence to antiretroviral therapy, prolonging the clinical course of malaria and thus reducing the cellular immunity of HIV infected pregnant women [23].

LIMITATIONS

Although the study offers some important findings, it also has limitations: The study was cross sectional, therefore the possibility of sampling error cannot be overruled. Also, the study used ITNs and prophylaxis as the sole indicator of control measures, but the usage of other measures such as indoor residual spraying (IRS), larvicides and mosquito repellent coils was not assessed. In addition, the non-comparison of malaria parasitaemia rate between HIV infected and HIV negative women is another limitation.

CONCLUSIONS

The prevalence of malaria recorded in this study is high, but with negative findings with regards to all sociodemographic variables of participants and risk factors of malaria.

REFERENCE

1. Brasil P, Zalis MG, de Pina-Costa A, Sequira AM, Júnior SB, Silva S, et al. Outbreak of human malaria caused by Plasmodium simium in the Atlantic Forest in Rio de Janeiro: a molecular epidemiological investigation. *Lancet Glob Health* 2017; 5:

e1038–46 [https://doi.org/10.1016/S2214-109X\(17\)30333-9](https://doi.org/10.1016/S2214-109X(17)30333-9)

2. White NJ. Anaemia and malaria. *Malar J*. 2018; 17: 371. doi: 10.1186/s12936-018-2509-9

3. World Malaria Report 2018. Geneva: World Health Organization; 2018. <https://apps.who.int/iris/bitstream/handle/10665/275867/9789241565653-eng.pdf> Last accessed 20th November, 2019

4. Naing C, Sandhu NK, Wai W. The Effect of Malaria and HIV Co-Infection on Anemia A Meta-Analysis. *Med*. 2016; 95 (14): 1-10
5. Rowe AK, Rowe SY, Snow RW. The burden of malaria mortality among African children in the year 2000. *Inter J of Epid*, 2006; 35(3):691–704
6. WHO: Evidence Review Group. Intermittent preventive treatment of malaria in pregnancy (IPTp) with sulfadoxine-pyrimethamine (SP). Geneva: W H O, 2012P; 1–17
7. World Health Organization . Malaria in HIV/AIDS Patients. Geneva: WHO; 2017
8. A. Alemu, Y. Shiferaw, Z. Addis, B. Mathewos, W. Birhan. Effect of malaria on HIV/AIDS transmission and progression. *Parasit & Vect*, 2013; 6:18 doi: 10.1186/1756-3305-6-18
9. Wumba RD, Zanga J, Aloni MN, Mbanzulu K, Kahindo A, Mandina MN, et al. “Interactions between malaria and HIV infections in pregnant women: a first report of the magnitude, clinical and laboratory features, and predictive factors in Kinshasa, the Democratic Republic of Congo”. *Malar J*. 2015; 14: 82. doi: 10.1186/s12936-015-0598-2
10. HIV prevalence in Nigeria. 2019. UNAIDS. https://reliefweb.int/sites/reliefweb.int/files/resources/20190314_PR_Nigeria_en.pdf Last accessed 20th November, 2019
11. González R, Ataíde R, Naniche D, Menéndez C, Mayor A. HIV and malaria interactions: where do we stand? *Expert Rev Anti Infect Ther*. 2012;10(2):153–165.
12. Bouyou-Akotet MK, Adegnika AA, Agnandji ST, Ngou-Milama E, Kombila M, Kremsner PG. Cortisol and susceptibility to malaria during pregnancy. *Microb Infect*, 2005; 7:1217-23.
13. De Cock KM, Fowler MG, Mercier E, de Vincenzi I, Saba J, Hoff E, et al. Prevention of mother-to-child HIV transmission in resource-poor countries: translating research into policy and practice. *J Am Med Assoc*, 2000; 283:1175–1182.
14. Rastogi S, Agrahari S, Singh UP, Singh A, Verma AV. Clinical Stages of HIV. *Biolixir*, 2011; 1(1):29-31
15. World Health Organization. “HIV for surveillance and revised clinical staging and immunological classification of HIV-related disease in adults and children”. August 2006, <http://www.who.int/hiv/pub/vct/hivstaging/en/>
16. Alemu A, Shiferaw Y, Addis Z, Mathewos B, Birhan W. Effect of malaria on HIV/AIDS transmission and progression. *Parasit Vect* 2013; 6(2): 18-24
17. Abellana R, Ascaso C, Aponte J, Saute F, Nhalungo D, Nhacolo A, Alonso P. Spatio-seasonal modeling of the incidence rate of malaria in Mozambique. *Malar J*. 2008; 7: 228. doi: 10.1186/1475-2875-7-228
18. Buhari HA, Erhabor O, Momodu I. Plasmodium falciparum Malaria among Pregnant Women attending Ante-Natal Clinic in Sokoto, North Western, Nigeria. *Sokoto J Med Lab Sci*. 2016; 1(1): 187 – 193
19. Adefionye OA, Adeyeba O, Hassan WO, Oyeniran OA. Prevalence of malaria parasite infection among pregnant women in Osogbo, southwest Nigeria. *Int J Nat App Sci*, 2007; 2(2): 61-64.
20. Onyenekwea CC , Ukibe N, Meludu SC, Ilika A, Aboh N, Ofiaeli N. Prevalence of Malaria as Co-Infection in HIV-Infected Individuals in a Malaria Endemic Area of Southeastern Nigeria. *J Vect Borne Dis*, 2007;44: 250-254.
21. Adeoti OM, Awobode HO, Anumudu CI. Cellular Immune Responses to HIV and falciparum Malaria Co-infection among Pregnant Mothers. *Am J Biomed Res*, 2015; 3(2):13-20
22. Udomah FP, Isaac IZ, Lukman I, Nwobodo D, Erhabor O, Abdulrahman Y, John RT. Plasmodium Parasitaemia Among Pregnant Women Attending Antenatal Clinic in Sokoto, North Western Nigeria. *J Nur Sci*, 2015; 1: 9-14.
23. Nasir IA, Agbede OO, Bakare M, Babandina MM. Severe malaria parasitemia and it’s effects on Haemoglobin and CD4 cells of HIV infected pregnant women at Kaduna state, Nigeria. *Asian Pacific J Trop Dis*. 2016; 6 (12): 943 - 945

Clinical Presentation of Splenomegaly at Kigali University Teaching Hospital, Rwanda - A Retrospective Descriptive Study

Authors: E. Nkusi^{1,*}; E. Nziyomaze¹; R. Salama²; V. Musengamana²; C. Mukamukama²; M.G. Nyirabizimana²; F. Hakizimana²; F. Shikama³

Affiliations: ¹Department of Internal Medicine, Butaro Hospital, Burera District, Rwanda; ²Department of Internal Medicine, Kigali University Teaching Hospital, Kigali, Rwanda; ³Department of Internal Medicine, Butare University Teaching Hospital, Huye, Rwanda

ABSTRACT

INTRODUCTION: Splenomegaly is a common condition in malaria endemic region and is frequently associated with cytopenias. Splenomegaly is usually a clinical finding, but imaging studies have helped to assess for or confirm it. Evaluating the clinico-hematological presentation of splenomegaly at the biggest referral hospital in Rwanda would bring important information to the clinicians.

METHODS: This was a retrospective descriptive study conducted at Kigali University Teaching Hospital, medical department, for a period of one year. We reviewed patients' charts admitted with the conditions known to be associated with splenomegaly referring to the available literature, and were enrolled if found to have it. Demographic, clinical, hematological and radiological information was extracted, then descriptive analyses were performed.

RESULTS: A total of 1950 patients' files were examined, and 117 (6%) patients had splenomegaly. The mean age was 39 years, and women were predominant (55.5%). Many cases of splenomegaly were detected on admission (49.6%), at stages II (33.3%) and III (23.1%). Many patients had no symptoms of splenomegaly (30.7%), others had features of anaemia (22.2%), and infection (25.6%). Abdominal ultrasound was the imaging of choice to evaluate the spleen size, and has discovered splenomegaly in 28.2% of cases after clinical examination was unremarkable. The main causes of splenomegaly were Hyper-reactive Malarial Splenomegaly (21.4%) and portal hypertension (19.6%).

CONCLUSION: Splenomegaly is a frequent condition and more prevalent in the eastern province, it was detected on admission at a moderate stage and associated with hemolysis in many cases.

Keywords: Splenomegaly, Anemia, Stage, Ultrasonography

INTRODUCTION

The human spleen is a dark purple bean-shaped, largest lymphoid organ, located in the left hypochondrium of the abdomen, underlying left ribs 9, 10 and 11, on the posterior to the mid-axillary line. The size is about (12 X 7 X 4 cm) and weighs (130–150 g) [1, 2]. The major functions of the spleen are: sequestration and phagocytosis of red blood cells and platelets, extramedullary hemopoiesis, blood pooling, production of hu-

moral antibodies, production and maturation of B and T cells and plasma cells [1]. Splenomegaly is defined as enlargement of the spleen measured by size or weight [2]. The spleen has to increase in size threefold before it becomes palpable [3]. Causes of splenomegaly are varied and include: infection (among them septic shock, infective endocarditis, typhoid, infectious mononucleosis, tuberculosis, brucellosis, malaria, kala-azar, schistosomiasis), inflammation (rheumatoid arthritis, sarcoidosis), haematological disorders (haemolytic anaemia, haemoglobinopathies, leukaemia, lymphomas

***Corresponding author:** Eugene Nkusi, MD, Butaro Hospital, Burera District, Rwanda, Email: archim2020@gmail.com, Tel: +250788755779; **Potential Conflicts of Interest (Col):** All authors: no potential conflicts of interest disclosed; **Funding:** All authors: no funding was disclosed; **Academic Integrity:** All authors confirm that they have made substantial academic contributions to this manuscript as defined by the ICMJE; **Ethics of human subject participation:** The study was approved by the local Institutional Review Board. Informed consent was sought and gained where applicable; **Originality:** All authors: this manuscript is original has not been published elsewhere;

Review: This manuscript was peer-reviewed by three reviewers in a double-blind review process; **Type-editor:** Dennis Hopkinson (USA)
Received: 31st March 2019; **Initial decision given:** 11th April 2019; **Revised manuscript received:** 7th December 2019; **Accepted:** 24th February 2020

Copyright: © The Author(s). This is an Open Access article distributed under the terms of the Creative Commons Attribution License (CC BY-NC-ND) (click here), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. **Publisher:** Rwanda Biomedical Centre (RBC)/Rwanda Health Communication Center, P.O.Box 4586, Kigali.
ISSN: 2079-097X (print); 2410-8626 (online)

Citation for this article: E. Nkusi, E. Nziyomaze, R. Salama et al. Clinical presentation of splenomegaly at Kigali University Teaching Hospital, Rwanda- a retrospective descriptive study. Rwanda Medical Journal, Vol 77, no 2, pp 17-22, 2020

and myeloproliferative disorders), portal hypertension and congestion (chronic liver diseases, congestive heart failure), storage diseases, amyloidosis, the lymphoid and myeloid neoplasms, tropical splenomegaly syndrome [6, 7]. Massive splenomegaly is seen in myelofibrosis, chronic myeloid leukaemia (CML), chronic malaria, kala-azar or, Gaucher's disease [8, 9].

Splenomegaly is usually associated with increased workload (hypersplenism), leading to cytopenia(s) from haemolysis and a compensatory bone marrow proliferative response [10, 11]. There are many ways to classify and evaluate the magnitude of splenomegaly. Hackett's classification is the most frequently used in cross-sectional studies; it classifies splenomegaly in five classes: from a non-palpable spleen (class I) to a huge spleen with the lowest point in the right iliac fossa (class V) [4].

In some cases, there are no symptoms of splenomegaly, while some patients complain of mild, vague, abdominal discomfort, pain which is referred to the left shoulder, early satiety from gastric displacement, or symptoms and signs that are related to the underlying disorder [20-22]. In the past, splenomegaly was a clinical finding, but in recent years, imaging studies have also helped to assess for or confirm mild splenomegaly [5].

Chronic splenomegaly is a common condition in tropical Africa, and it is only in about 25% that the aetiology is found [6]. In malaria endemic area, approximately 50-80% of its residents have splenomegaly, and Hyper-reactive Malarial Splenomegaly is the main cause [7]. In the United States, one large series reported a palpable spleen in 2% of patients and another in 5.6% of patients [8].

Referring to a study done in Kenya; where aetiologies of chronic splenomegaly were investigated in 131 Kenyan patients, the major causes were hyper-reactive malarial splenomegaly (31%), hepatosplenic schistosomiasis (18%), visceral leishmaniasis (5%), non-schistosomal forms of portal hypertension (20%), while no diagnosis could be established in 12% [9]. One study conducted in the Eastern Province of Rwanda found that the prevalence of hyper reactive malarial splenomegaly in healthy pupils from the selected primary schools was 6.2% (13 out of 210 participants) [10].

Rationale and study objectives

To the best of our knowledge, there are no data about the prevalence of splenomegaly and associated etiologies in the Rwandan population although our preliminary data from the hospital's statistics department show that a significant number of patients admitted in the medical department had splenomegaly. Only one study, without comparator, has evaluated splenomegaly in Butare, focusing only on the therapeutic aspects of HMS [11].

A wide variety of diseases can lead to splenomegaly, and the predominant causes vary with geographical distribution of diseases prevalent in the region. With this study, we hope to bring an important information to the clinicians and researchers about the diseases that are commonly associated with splenomegaly in Rwanda which is not yet known.

In the majority of cases, splenomegaly can be detected clinically by the treating clinician during physical examination, however,

there is still a good number of splenomegaly that are discovered by imaging studies, which were sometimes ordered for other purposes; we want to formally assess this discrepancy and provide preliminary data that can guide policy makers for appropriate quality improvement projects.

Splenomegaly is a subject of considerable clinical concern and warrants thorough clinical and laboratory evaluation; we anticipated that assessing the demography, common clinical presentations and hematological characteristics of patients with splenomegaly referred to the biggest national referral hospital receiving patients from all over the country could bring important information to clinicians and health decision makers that will contribute to improve the clinical assessment of patients with splenomegaly.

This study aims to assess the demographic data, clinical presentation, diagnostic aspects and hematological profile of patients with splenomegaly. In addition, we also document different conditions associated with splenomegaly among hospitalized medical patients at Kigali University Teaching Hospital, observed during the year 2018.

METHODS

Patients and settings: This was a retrospective descriptive study conducted at Kigali University Teaching Hospital (CHUK), Department of Internal Medicine from January 1, 2018 to December 31, 2018. CHUK is a tertiary referral hospital and main public health institution in Rwanda. It is located in the center of Kigali city and receive patients from all the corners of the country via District hospitals. It has a 560 beds capacity.

The study included patients that were admitted in internal medicine to either ward 3 (male), ward 4 (female) ward 6 (Isolation ward) and PCK (prison ward). We did not include patients admitted from the other departments as the most causes of splenomegaly are medical conditions and not directly surgical or obstetrics diseases. Patients' files with various systemic diseases and hematological conditions, where splenomegaly was not present were not enrolled.

Sample size: The estimated sample size was calculated using the following formula [12,13]

$$N = \frac{Z^2 \cdot P \cdot (1-P)}{E^2}$$

N: Sample size **P:** Expected Prevalence in the study population **E:** Absolute standard error (precision), **E**= 0.01

Z is the statistic corresponding to level of confidence, **Z**=1.96 and the level of confidence aimed for is 95%

Given the lack of prior local data in the study population, the prevalence of splenomegaly in hospitalized medical patients was estimated based on figures from California in United States where the prevalence of splenomegaly was 5.6% [8]. Based on a large eligible population of hospitalized medical patients and assuming the estimated prevalence of splenomegaly of 5%, a sample size of 1825 was calculated to achieve a 1% absolute standard error at a 95% confidence level for the prevalence of splenomegaly.

Recrutement of study participants: We reviewed the charts of all patients admitted with the conditions known from the literature to be associated with splenomegaly and include: infection (among them septic shock, infective endocarditis, typhoid fever, infectious mononucleosis, tuberculosis, brucellosis, malaria, kala-azar, schistosomiasis), inflammation (rheumatoid arthritis, sarcoidosis), haematological disorders (haemolytic anaemia, haemoglobinopathies, leukaemia, lymphomas, myeloproliferative disorders), portal hypertension and congestion (chronic liver diseases, congestive heart failure), storage diseases, amyloidosis, neoplasia, myelofibrosis, Hyper-reactive Malarial Splenomegaly, chronic myeloid leukaemia, and we enrolled those with splenomegaly (discovered either on clinical examination or by imaging) for further data analysis. Eligible patients were identified through the patients' registry, where every admitted patient in the respective ward is registered and his/her diagnosis is well mentioned together with his/her hospital identification and archiving numbers which helped to access their medical records.

Study design: Relevant information was extracted from existing medical records, patient's chart, laboratory and radiology registers; and was collected on a predesigned case report form which was anonymous with a unique identifier. Baseline demographic data (age, gender, marital status, place of residency), clinical data including general systemic symptoms such as fever, sweats, weakness, weight loss, nausea, anorexia, presenting signs and symptoms of splenomegaly such as left hypochondrial discomfort, early satiety, abdominal pain and or tenderness, a palpable mass in left upper quadrant, features of underlying condition including lymphadenopathies, hepatomegaly, portal hypertension, congestion, abdominal distension, and associated hematological parameters including leucocytes count, hemoglobin levels and thrombocytes. We collected as well information about when splenomegaly was discovered (before admission, on admission, or incidentally after admission) how splenomegaly was diagnosed (either by physical examination, abdominal ultrasonography, abdominal CT Scan or MRI) and Hackett's clinical stage of that enlarged spleen. All collected data were kept under conditions of strict confidentiality and are rendered anonymous by a unique identification code.

Ethical consideration: This study was approved by Kigali University Teaching Hospital Research and Ethics Committee. We did not request individual consent from the participants of this study as the risks of the study are minimal to the patient. The research involved retrospective review of patient records only;

there are no experimental interventions or additional patient encounters with research/clinical staff outside of routine data collection. Data were abstracted from routine hospital records for patients who met the inclusion criteria, and viewed only by research team members in accordance with data management procedures described above. Since the study is a retrospective review of routine medical records, the study did not directly affect positively or negatively the care and routine management of the patients in the study. The possible risk is inappropriate exposure of patient confidential records, we minimized this risk by ensuring that only de-identified data is stored in database. Also, all study staff involved in data abstraction received appropriate research training to ensure understanding and compliance of data management and confidentiality procedures. The key record identifying the participants will be kept confidential even after publication of the results.

Statistical analysis: Data were initially collected on a case report form and then entered using Epidata version 3.1 software. Descriptive analysis, using standard statistical methods, was performed using the Statistical Package for Social Sciences (SPSS) software, version 16.0. Demographic and clinical characteristics were noted. During our data collection, some patients had incomplete files; they had some missing information for some variables and there were not considered for data analysis; a listwise deletion was performed before analysis. The most commonly affected variables are weight, temperature, lymph node examination, abdominal examination, FBC and final outcome. The analysis was applied for significant variables in new sample size with complete data.

RESULTS

During the study period, a total of 2019 eligible patients' files were examined, and among them 1950 had complete data; and out of them, only 117 (6%) patients were identified to have splenomegaly either on clinical examination or by imaging; they were enrolled in data collection and included in the final analysis. Their mean age was 39 years, with a range between 15 to 79 years. The number of women was slightly higher 65 (55.5%). Many patients were from the Eastern Province 47 (40.2%) followed by Kigali City 30 (25.6%) (Table 1).

Table 1: Demographic and clinical features of splenomegaly

Variables	N (%)
Sex	
Male	52 (44.5)
Female	65 (55.5)
Province of origin	
Kigali city	30 (25.6)
Eastern province	47 (40.2)
Northern province	8 (6.8)
Southern province	23 (19.7)
Western province	8 (6.8)
Outside Rwanda	1 (0.9)
When splenomegaly was diagnosed?	
Before admission	14 (11.9)
On admission	58 (49.6)
After admission	43 (36.8)
Not documented	2 (1.7)
Splenomegaly stage (Hackett's Classification)	
stage I	25 (21.4)
stage II	39(33.3)
stage III	27(23.1)
stage IV	24(20.5)
stage V	2(1.7)
Diagnostic tools	
Already known	14 (11.9)
Physical examination only	16 (13.7)
Abdominal ultrasound	33 (28.2)
Suspected on physical exam and confirmed with abdominal ultrasound	53 (45.3)
CT Scan	1 (0.9)
Total	117(100%)

In the majority of cases, splenomegaly was detected or suspected on physical examination (a palpable left upper quadrant abdominal mass) on admission (49.6%) or in few days post admission (36.8%). Splenomegaly was already documented before admission in 11.9% of cases. Many cases of splenomegaly were diagnosed at stage II (33.3%), stage III (23.1%) and stage IV (20.5%). Abdominal ultrasonography was the imaging tool that was used to evaluate the spleen size in the majority of cases. Splenomegaly was confirmed in 45.3% of cases after being suspected clinically, and it was discovered by ultrasonography in 28.2% of cases while clinical examination was unremarkable. Many patients did not have symptoms associated with an enlarged spleen (30.7%), others complained of abdominal discomfort (19.6%), chest pain similar to pleuritic pain when stomach or bowels are full (17%), complaints of early satiety (16%), symptoms of haemolysis (24.8%), symptoms of anaemia (22.2%), symptoms of infection (25.6%), constitutional symptoms suggestive of malignancy (13.6%) signs and symptoms related to the underlying conditions (17.1%). Splenomegaly

was associated with hepatomegaly in 18.8% and with lymphadenopathies in 30%. Anaemia was present in the majority of patients (70%), followed by leukopenia (49.5% of cases), thrombocytopenia in 45.3% of cases while 30.7% of patients had pancytopenia. Leucocytosis was present in 19.6% and thrombocytosis was observed in 14.5% of the patients (Table 2).

Table 2: Clinico-haematological presentation of splenomegaly

Clinical presentation	N (%)
No symptoms of splenomegaly	36(30.7)
Constitutional symptoms	16(13.6)
Early satiety	19(16.2)
Abdominal pain and or tenderness	23(19.6)
Chest pain	20(17.1)
Features of anaemia	26(22.2)
Features of infection	30(25.6)
Features of haemolysis	29(24.8)
Symptoms of the underlying conditions	20(17.1)
Association with hepatomegaly	22(18.8)
Association with lymphadenopathies	35(30.0)
Haematological findings	
Anaemia (Hb<12g/dl)	82(70.0)
Leukopenia (WBC<4.500 cells/mm ³)	58(49.5)
Thrombocytopenia (PLT<150.000/ μ l)	53(45.3)
Pancytopenia	36(30.7)
Leucocytosis (WBC>12.000 cells/mm ³)	23(19.5)
Thrombocytosis (PLT>450.000/ μ l)	17(14.5)
Normal Haemoglobin	35(30.0)
Normal WBC (4.500 - 12.000 cells/mm ³)	36(31.0)
Normal PLTs (150.000 - 450.000/ μ l)	47(40.2)

The main causes of splenomegaly were HMS (21.4%) and it was prevalent among patients from the Eastern province (19 out of 25 total cases of HMS), followed by cirrhosis and portal hypertension (19.6%), acute infection (16.2%), CML (12%), disseminated tuberculosis (9.4%). No clear etiology of splenomegaly was found in (11%) of patients (Table 3).

Table 3: Clinical diagnosis in 117 cases of splenomegaly

Causes of splenomegaly	N (%)
HMS	25(21.4)
Cirrhosis and portal hypertension	23(19.6)
Acute infection	19(16.2)
Chronic myeloid leukaemia	14(11.9)
Tuberculosis	11(9.4)
Congestive cardiac failure	8(6.8)
Acute Leukemia	9(7.7)
Lymphoma	7(5.9)
Chronic lymphocytic leukaemia	5(4.2)
Myelofibrosis	3(2.5)
Schistosomiasis	1(0.8)
No clear etiology	13(11.1)

The present study is an attempt to find out the frequency of various causes of splenomegaly, its clinical presentation and how it is diagnosed at a referral level hospital in Rwanda. In addition, it highlights the role of hematological parameters as a diagnostic or additional tool in elucidating etiopathogenesis of splenomegaly. The results of this study show that there are no major differences with the existing

findings: the prevalence of splenomegaly was 6%, which is similar with 5.6% found by O'Reilly et al. at a big referral hospital in California [8], and similarly with other studies, there was no age or gender predilection for splenomegaly [5].

The most frequent cause of splenomegaly in present study was hematological diseases followed by Hyper-reactive Malarial Splenomegaly, congestive splenomegaly (associated with liver cirrhosis) and infection; these are quite similar with figures from Kenya [9]. One study revealed that in malaria endemic area, about 11-45% of patients with massive splenomegaly were due to HMS [15, 17]; we got the same findings as the main cause of splenomegaly was HMS (21.4%) and was frequent among patients from the Eastern province (the highest malaria endemic area in Rwanda).

The abdominal ultrasonography was used to confirm splenomegaly in 45.3% of cases after being suspected clinically, and it was discovered by ultrasonography in 28.2% of cases while clinical examination was unremarkable. One study showed that while clinical examination can be convincing in splenic enlargement, radiology is often needed to confirm the diagnosis, and it was found that point-of-care ultrasonography significantly improves examiners' sensitivity in diagnosing splenomegaly [23, 24].

In this study, many patients presented with moderate splenomegaly and cytopenias (mainly anemia) from an enlarged overworking spleen; this is a common finding in patients with chronic splenomegaly especially from developing countries; where infectious diseases and hematological malignancies are prevalent [25, 26]; and routine hematological evaluations may provide an important clue about the etiology of splenomegaly, for example, finding a parasite, evidence of hemolysis, septicemia, leukemia, lymphoma or myeloma.

The present study is an attempt to find out the frequency of various causes of splenomegaly, its clinical presentation and how it is diagnosed at a referral level hospital in Rwanda. In addition, it highlights the role of hematological parameters as a diagnostic or additional tool in elucidating etiopathogenesis of splenomegaly. The results of this study show that there are no major differences with the existing findings: the prevalence of splenomegaly was 6%, which is similar with 5.6% found by O'Reilly et al. at a big referral hospital in California [8], and similarly with other studies, there was no age or gender predilection for splenomegaly [5].

The most frequent cause of splenomegaly in present study was hematological diseases followed by Hyper-reactive Malarial Splenomegaly, congestive splenomegaly (associated with liver cirrhosis) and infection; these are quite similar with figures from Kenya [9]. One study revealed that in malaria endemic area, about 11-45% of patients with massive splenomegaly were due to HMS [15, 17]; we got the same findings as the main cause of splenomegaly was HMS (21.4%) and was frequent among patients from the Eastern province (the highest malaria endemic area in Rwanda).

The abdominal ultrasonography was used to confirm splenomegaly in 45.3% of cases after being suspected clinically, and it was discovered by ultrasonography in 28.2% of cases while clinical examination was unremarkable. One study showed that while clinical examination can be convincing in splenic enlargement, radiology is often needed to confirm the diagnosis, and it was found that point-of-care ultrasonography significantly improves examiners' sensitivity in diagnosing splenomegaly [23, 24].

In this study, many patients presented with moderate splenomegaly and cytopenias (mainly anemia) from an enlarged overworking spleen; this is a common finding in patients with chronic splenomegaly especially from developing countries; where infectious diseases and hematological malignancies are prevalent [25, 26]; and routine hematological evaluations may provide an important clue about the etiology of splenomegaly, for example, finding a parasite, evidence of hemolysis, septicemia, leukemia, lymphoma or myeloma.

Study limitations

This study didn't assess in details all laboratory and hematological investigations that would help to best characterize the different conditions associated with splenomegaly.

The detailed assessment of the spleen (like the structure, echogenicity, contours, etc.), as reported by the radiologists/radiology technicians were not recorded and included in analysis while this information would serve to better characterize the enlarged spleen. Imaging contribution was just to confirm splenomegaly and its stage. Another limitation of this study is listwise deletion that was applied to deal with missing data; this method can introduce a systematic bias; but there was no significant change of our results.

CONCLUSION

Splenomegaly is a frequent finding in a medical department. In our country, especially the Eastern province, there is a high prevalence of tropical splenomegaly. Most cases with splenomegaly are detected on admission by physical examination or by abdominal ultrasonography and found at moderate stages in many cases. Splenomegaly should always be investigated thoroughly as most of the common causes are treatable. There is an exhaustive list of different etiologies of splenomegaly and hematological causes outnumbered the non-hematological cause of splenomegaly. Hematological profile in cases with enlarged spleen are of utmost importance as a diagnostic tool which can also be used to monitor the response to treatment.

DECLARATIONS

Ethics approval: This study protocol was reviewed and approved by the Kigali University Teaching Hospital Research and Ethics Committee. We ensured confidentiality of the study data and maintained the anonymity of the study participants.

Availability of data and materials: The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Authors' contributions: All authors contributed to the design and development of this study. EN, EN coordinated the data collection

process. RS, CM and MGN played an important role in data collection and data entry. FH and FS performed statistical analyses. EN, EN, VM and FS reviewed and corrected the final work. All authors read and approved the final manuscript.

Acknowledgements: The authors would like to thank the nurses, doctors, medical residents and entire staff of the Kigali University

Teaching Hospital, department of internal medicine and radiology for their support and collaboration during this study. We also would like to thank Dr Benoit Seminega for his invaluable advices and comments during this work.

REFERENCES

- [1] J. Van Den Ende, A. Van Gompel, E. Van Den Enden, H. Taelman, G. Vanham, and T. Vervoort, "Hyperreactive malaria in expatriates returning from sub-Saharan Africa," *Trop. Med. Int. Heal.*, vol. 5, no. 9, pp. 607–611, 2000.
- [2] Z. Bisoffi et al., "Chronic malaria and hyper-reactive malarial splenomegaly: A retrospective study on the largest series observed in a non-endemic country," *Malar. J.*, vol. 15, no. 1, 2016.
- [3] E. E. Elmakki, "Hyper-reactive Malarial Splenomegaly Syndrome (HMSS): Review article," *Cureus*, vol. 4, no. 11, 2012.
- [4] S. Leoni, D. Buonfrate, A. Angheben, F. Gobbi, and Z. Bisoffi, "The hyper-reactive malarial splenomegaly: A systematic review of the literature," *Malar. J.*, vol. 14, no. 1, 2015.
- [5] J. Chapman and A. M. Azevedo, *Splenomegaly*. 2019.
- [6] G. Bedu-Addo, J. Sheldon, and I. Bates, "Massive splenomegaly in tropical West Africa," *Postgrad. Med. J.*, 2000.
- [7] G. Bedu-Addo and I. Bates, "Causes of massive tropical splenomegaly in Ghana," *Lancet*, 2002.
- [8] R. A. O'Reilly, "Splenomegaly in 2,505 patients at a Large University Medical Center from 1913 to 1995 - 1913 to 1962: 2,056 patients," *West. J. Med.*, 1998.
- [9] K. M. De Cock et al., "Chronic splenomegaly in Nairobi, Kenya. I. Epidemiology, malarial antibody and immunoglobulin levels," *Trans. R. Soc. Trop. Med. Hyg.*, 1987.
- [10] L. E. G. Mboera, W. R. Mukabana, and K. J. Njunwa, "Integrated Research Partnerships for Malaria Control through an Ecohealth Approach in East Africa : Kenya , Rwanda , Tanzania and Uganda Projects Integrated Research Partnerships for Malaria Control through an Ecohealth Approach in East Africa : Kenya , R," no. January, pp. 1–40, 2014.
- [11] N. Issn, "Rwanda Medical Journal Revue Médicale Rwandaise," vol. 71, no. 1. 2014.
- [12] M. A. Pourhoseingholi, M. Vahedi, and M. Rahimzadeh, "Sample size calculation in medical studies," *Gastroenterol. Hepatol. from Bed to Bench*, vol. 6, no. 1, pp. 14–17, 2013.
- [13] T. Edition, *Biostatistics Wayne Daniel*. .
- [14] T. B. Pepinsky, "A Note on Listwise Deletion versus Multiple Imputation," *Polit. Anal.*, vol. 26, no. 4, pp. 480–488, 2018.
- [15] T. Lumley, "Missing data (Analyzing complex surveys with R)," vol. 334, no. 7590, pp. 424–424, 2007.

Prevalence of Primary Infertility Caused by Chromosomal Abnormalities and Assessment of Clinical Manifestations in Rwandan patients

Authors: B. Mvuyekure¹; C. Mutoni¹; E. A. Murinzi¹; W. Ngizwenayo¹; C. Nsanzabaganwa²; L. Mutesa^{1,*}

Affiliations: ¹ School of Medicine and Health Sciences, University of Rwanda; ² Rwanda Military Hospital, Kigali, Rwanda

ABSTRACT

INTRODUCTION: Infertility affects millions of couples worldwide causing psycho-social problems and conflicts in families. Despite the establishment of multiple causes of infertility in both males and females, there have been no studies carried out in Rwanda about primary infertility caused by chromosomal abnormalities. Thus, the aim of this study is to determine the prevalence of primary infertility caused by chromosomal abnormalities and to assess the clinical manifestations in Rwandan patients.

METHODS: We performed a cross-sectional retrospective assessment of the data extracted from medical files and OpenClinic (an electronic data recording system) of patients transferred to one genetic lab in Huye that works with three main referral hospitals: Kigali University Teaching Hospital (CHUK), Huye University Teaching Hospital (CHUB) and Rwanda Military Hospital (RMH) from June 2009 to June 2019.

RESULTS: This study showed that the overall prevalence of primary infertility caused by chromosomal abnormalities was 25.4% (N=15/59) among the patients who consulted the genetic department. Females were more affected than males with 32% (N=8/25) of females being primarily infertile due to chromosomal abnormalities and 20.58% (N=7/34) of males respectively. Our study also found that the majority (66.1%) of infertile patients had a normal karyotype in both genders with 40.7% of the males (46, XY) and 25.4% of the females (46, XX).

CONCLUSION: Chromosomal abnormalities contribute significantly to primary infertility in the Rwandan population. Thus, clinicians should consider these chromosomal abnormalities in patients attending fertility clinics.

Keywords: Infertility, Primary, Chromosomal Abnormalities, Prevalence, Rwanda

INTRODUCTION

Infertility is a global health burden that affects more than 186 million couples worldwide [1]. By definition, it is a disease of the reproductive system characterized by the inability to conceive naturally for a couple, despite one year of regular unprotected sex [2]. It causes many psycho-social problems such as conflicts in families, divorce or separation of partners, loss of social security and emotional distress [3]. Infertility is classified into primary and secondary infertility [4]. Primary infertility, also named sterility, is a term used for women who have never been pregnant and for men who have never impregnated a woman despite one year of regular unprotected sex. The term second-

ary infertility refers to the inability to conceive in a couple who have had at least one successful conception in the past [4].

There are multiple causes of infertility in both males and females. According to the World Health Organization (WHO), the most common identifiable female factors were menstruation and ovulatory disorders (32%), fallopian tube abnormalities secondary to pelvic adhesions and infections (34%), and endometriosis (15%) [3]. There are also other factors such as genetic disorders, as well as uterine and cervical factors. Male causes of infertility include genetic factors, abnormal sperm production and motility, anatomical defects, endocrine disorders and sexual dysfunction [5].

The statistics from the European Journal of Human Genetics re-

***Corresponding author:** Leon Mutesa, MD, PhD, Email: lmutesa@gmail.com, Director of Center for Human Genetics, College of Medicine and Health Sciences, University of Rwanda; **Potential Conflicts of Interest (CoI):** All authors: no potential conflicts of interest disclosed; **Funding:** All authors: no funding was disclosed; **Academic Integrity:** All authors confirm that they have made substantial academic contributions to this manuscript as defined by the ICMJE; **Ethics of human subject participation:** The study was approved by the local Institutional Review Board. Informed consent was sought and gained where applicable; **Originality:** All authors: this manuscript is original has not been published elsewhere; **Review:** This manuscript was peer-reviewed by three reviewers in a double-blind review process; **Type-editor:** Ahmed (USA)

Received: 21st November 2019; **Initial decision given:** 21th April 2019; **Revised manuscript received:** 10th January 2019; **Accepted:** 17th January 2020

Copyright: © The Author(s). This is an Open Access article distributed under the terms of the Creative Commons Attribution License (CC BY-NC-ND) ([click here](https://creativecommons.org/licenses/by-nc-nd/4.0/)), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. **Publisher:** Rwanda Biomedical Centre (RBC)/Rwanda Health Communication Center, P.O.Box 4586, Kigali. **ISSN:** 2079-097X (print); 2410-8626 (online)

Citation for this article: B. Mvuyekure, C. Mutoni, E. A. Murinzi et al. Prevalence of primary infertility caused by chromosomal abnormalities and assessment of clinical manifestations in Rwandan patients. Rwanda Medical Journal, Vol 77, no 2, pp 23-27, 2020

vealed that genetic abnormalities could be the cause of infertility in 10% of female infertile patients and 15% of male infertile patients [4]. These genetic abnormalities include chromosome aberrations, micro deletions, duplications, single gene mutations, complex conditions and epigenetic disorders.

A study in Iran performed at a government centre for infertility in Fatemeh Hospital during 2010 to 2011, found that genetic factors were amongst the dominant causes of infertility in 29.8% of males [2]. Other male causes of infertility that were found were semen disorders (44.6%), vascular disorders such as varicoceles (17.2%), and anti-spermatogenesis agents (8.4%) [6].

Despite the prevalence of infertility and the growing need of using assisted reproduction treatment such as In Vitro Fertilization (IVF) and Intra-cytoplasmic Sperm Injection (ICSI), many researchers are not examining the genetic factors of infertility in Africa, and Rwanda in particular. And yet, research has found that gametes that contain chromosomal aberrations have a high probability of failing to enter in fertilization. There is also a concern nowadays about assisted reproductive techniques, which might force the formation of a zygote by dominating hidden chromosomal changes in parents, thereby transmitting genetic abnormalities to the next generation or increasing the number of early abortions or foetal abnormalities due to parental genetic defects [7].

Furthermore, the consequences of infertility for couples is very significant in Rwanda and other African countries because children are highly valued for economic and socio-cultural reasons.

Since there have been no studies carried out in Rwanda surrounding these issues, our study aimed to determine the prevalence of primary infertility caused by chromosomal abnormalities and assess the clinical manifestations in Rwandan patients who consulted the genetic department. We believe this study will bring new knowledge and evidence needed to improve the management of infertile patients in Rwandan hospitals.

This study aimed at investigating somatic and sex chromosomal abnormalities that cause primary infertility in Rwandan patients evaluating the contribution of chromosomal abnormalities to decreased fertility in humans and describing the clinical manifestations of patients with primary infertility who consulted the genetic department in Rwanda

METHODOLOGY

Study design: This study used a cross-sectional retrospective assessment of the data extracted from patients' files and OpenClinic (electronic data recording system used in hospitals).

Study population: The study population included 59 patients, both male and female, with primary infertility who consulted the genetic department between June 2009 and June 2019.

Study site: The study was conducted in three referrals hospitals (CHUK, RMH, CHUB) which work with the Genetic Lab located in Huye.

Inclusion criteria: All patients with primary infertility as well as those with associated different forms of Disorders of Sex Development (DSD) who consulted the genetic department and whose files were available were included in this study. Symptoms considered for females were: the inability to conceive for one year despite unprotected and regular sexual intercourses and abnormal menstrual cycles. Symptoms considered for males were: the inability to induce pregnancy regardless of multiple unprotected sexual intercourses.

Exclusion criteria: Patients with incomplete files whose address, hospital identification and karyotype results were missing.

Data collection tool: We used a well-structured paper and electronic questionnaire. Questions were extracted from reliable and valid questionnaires released by different institutions including the American Society for Reproductive Medicine, UNC Fertility and Johns Hopkins Medical Centre.

Data analysis: Data was entered, stored and analysed in IBM SPSS Statistics software version 23. The data is presented in tables and figures and interpreted in percentages and frequencies.

Ethical consideration: Data security, privacy and confidentiality were taken into account based on recommendations from the Research Ethical Committee (REC).

RESULTS

Baseline details of data: In our study, we found 59 patients whose records met the inclusion criteria. They consulted the genetic department for primary infertility on suspicion of chromosomal abnormalities as a cause in a period of 10 years from June 2009 to June 2019.

The majority of patients (57.6%) were males whilst 42.4% of patients were females and the mean age was 32.81. The majority of the patients were aged between 20-34 years and the peak age group was 56 years. We found that most of the patients 52.5% (N=31) came from Kigali City, followed by Eastern Province with 18.6% (N=11). Western Province represented 13.6% (N=8) of patients, Southern Province with 5.1% (N=3) and the last being the Northern Province with no patients. 23.7% (N=14), of patients were from the Kicukiro district, followed by Gasabo having 20.3% (N=12) of patients and Nyarugenge and Kayonza having 8.5% (N=5) of patients each (Table 1).

Clinical features of patients with infertility: In our study, the most predominant clinical feature found in men was gynecomastia (14.7%) followed by micropenis (11.8%). Ejaculatory problems and abnormal reproductive organs were represented by 8.8% of patients whilst ambiguous genitalia were found in 2.9% (Figure 1).

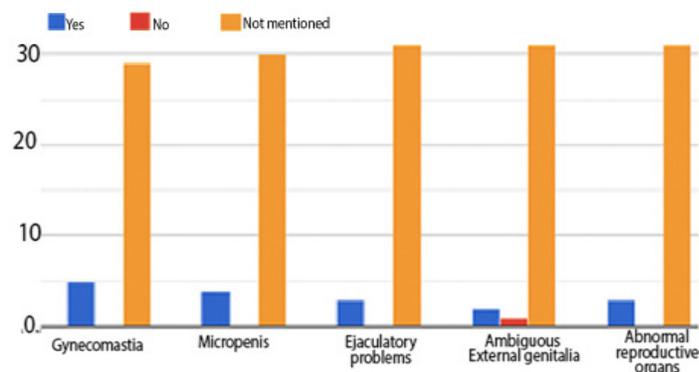


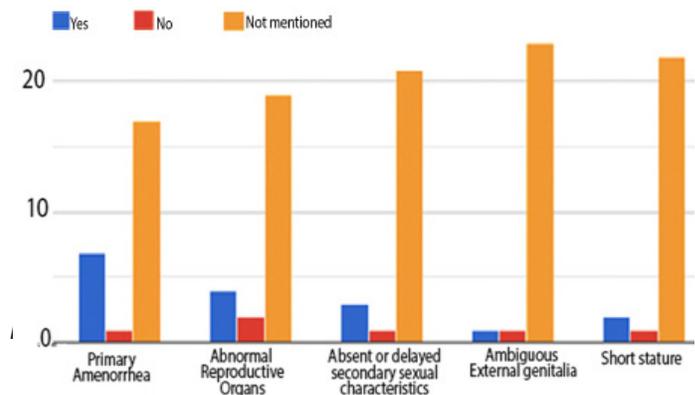
Figure 1: Clinical presentation of men with infertility

Table 1: Baseline details of patients showing gender and age distribution

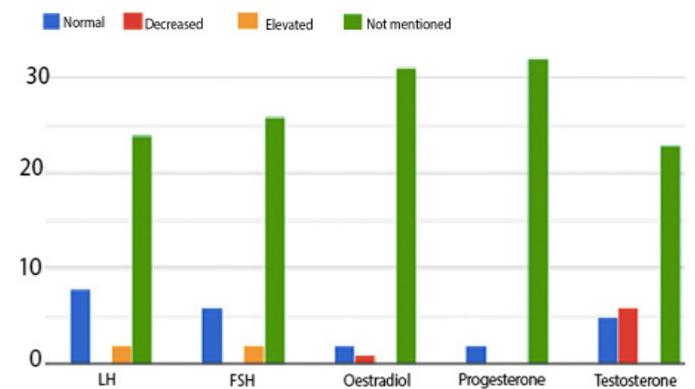
Gender	Frequency	Percentage (%)
Male	34	57.6
Female	25	42.4
Age range	Frequency	Percentage (%)
20 to 34 years	39	66.1
35 to 50 years	18	30.5
51 to 65 years	2	3.4
Province	Frequency	Percentage (%)
Kigali City	31	52.5
Eastern Province	11	18.6
Western Province	8	13.6
Southern Province	3	5.1
Not Recorded	6	10.2
Province	Frequency	Percentage (%)
Bugesera	2	3.4
Gasabo	12	20.3
Gatsibo	2	3.4
Huye	1	1.7
Karongi	2	3.4
Kayonza	5	8.5
Kicukiro	14	23.7
Ngoma	1	1.7
Nyabihu	3	5.1
Nyamagabe	1	1.7
Nyamasheke	1	1.7
Nyanza	1	1.7
Nyarugenge	5	8.5
Rubavu	1	1.7
Rusizi	1	1.7
Rwamagana	1	1.7

Primary amenorrhea was the most common clinical feature (28%) in women, followed by abnormal reproductive organs (16%). Absent or delayed secondary characteristics were seen in 12% of women, followed

by short stature at 8%. Ambiguous external genitalia were found in 4% of female patients (Figure 2).

**Figure 2: Clinical presentation of women with infertility**

Our study results showed that 14.7% of male patients had low testosterone levels, 2.9% (N=1) had low oestradiol levels, whilst LH and FSH were both high in 5.9% of male patients (Figure 3).

**Figure 3: Hormonal studies in male patients**

As shown in figure 4, FSH and oestradiol were both abnormal in 16% of female patients. 12% of female patients had abnormal LH and Testosterone levels, whilst progesterone was low in 4% (Figure 4).

We found that 46.9% of male patients had abnormal semen with azoospermia being the most common cause (29.4%), followed by oligospermia at 8.8% (N=3/34). Necrospermia, hypospermia and oligoasthenoteratozoospermia were found in 2.9% of male patients each (Table 2).

Table 3: Karyotype results according to gender

		Semen Analysis							Total	
Gender	Male	Count	Necrospermia	Hypo-spermia	Oligoasthenoteratozoospermia	Normal	Not Recorded	3	15	34
Total		Count	10	3	1	1	1	3	15	34
% of Total			29.4%	8.8%	2.9%	2.9%	2.9%	8.8%	44.1%	100.0%

In our karyotype results, we found that the abnormal karyotype counted for 25.4% of patients, amongst which numerical abnormalities are responsible for 11.9% (N=7).

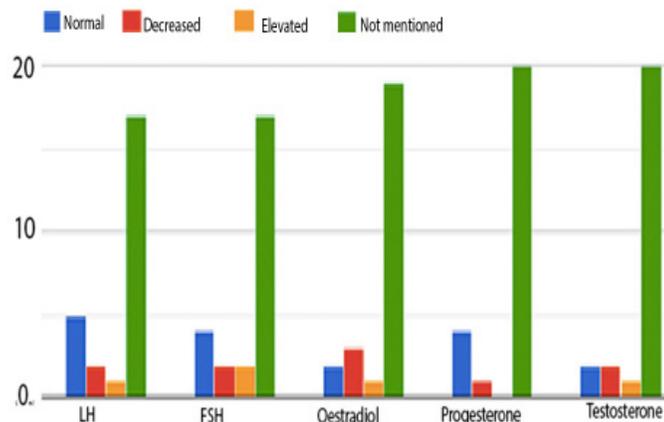


Figure 4: Hormonal studies in female patients

Turner syndrome and Klinefelter syndrome (Figure 5) affected 6.8% and 5.1% of patients respectively, whilst abnormal sex chromosomes equivalently counted for 11.9%. Structural abnormalities were the least prevalent found only in 1.7% of patients. Our study found that 66.1% had a normal karyotype in both genders with 40.7% male 46, XY and 25.4% female 46,XX (Table 3).

DISCUSSION

In our study, we found that the overall prevalence of chromosomal abnormalities was 25.4% of the total number of male and female patients with primary infertility who consulted the genetic department in Rwanda. Our study showed that 20.58% (N=7/34) of all the patients with chromosomal abnormalities were male, whereas 32% were females. A study performed in Croatia also showed a higher proportion of chromosomal abnormalities in infertile females compared to males at 26.4% and 17.7% respectively [8].

Analysis showed that the most common genetic diseases found in the patients had different prevalence in each gender.

In males, we found that the most prevalent chromosomal abnormality is 46, XX DSD, affecting 11.76% of patients. This is a type of DSD where a person appears male phenotypically but the karyotype test reveals a female genotype (masculinized female). The second most common chromosomal abnormality was Klinefelter syndrome which was seen in 8.82% of male patients. There were no males with a structural karyotype abnormality or a Y chromosome micro-deletion.

Our findings are not very different from a study that was carried out in the USA where Klinefelter syndrome was found to be the most common chromosomal aberration in 14% of infertile patients with azoospermia [9].

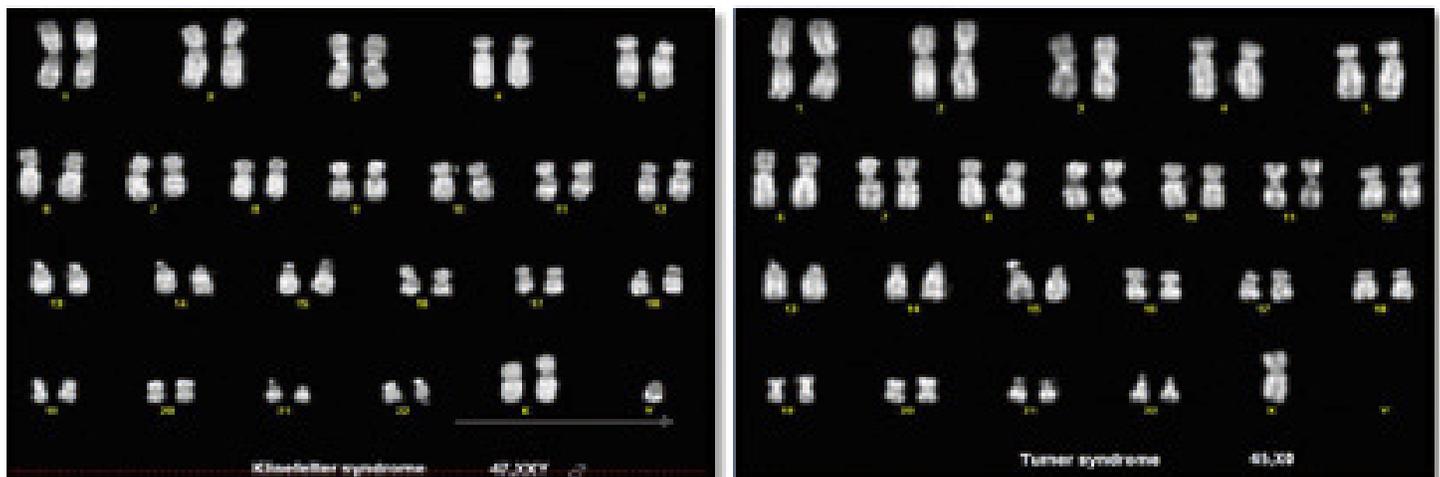


Figure 5: Klinefelter (Left) and Turner (Right) syndrome Karyotypes

Table 3: Karyotype results according to gender

Gender	Karyotype									
		46, XX	46, XX DSD	45, X0	47, XXY	46, X del (Xq)	Not Recorded			Total
Male	Count	24	0	0	4	0	3	0	3	34
	% of Total	40.7%	0.0%	0.0%	6.8%	0.0%	5.1%	0.0%	5.1%	57.6%
Female	Count	0	3	15	0	4	0	1	2	25
	% of Total	0.0%	5.1%	25.4%	0.0%	6.8%	0.0%	1.7%	3.4%	42.4%

In females, we found that most prevalent chromosomal abnormality was Turner syndrome 45, X0 which accounted for 16% of female patients who consulted the genetic department in Rwanda. The least common genetic defect in females was 46, X del (Xq) that accounted for 1.7% of patients.

Semen analysis is one of the cornerstone investigations for an infertile couple. Our study revealed that 46.9% of male patients had abnormal semen, gynecomastia, micropenis, ejaculatory problems and abnormal reproductive organs. In a study by Dohle GR

et al, 40-60% also had abnormal semen, gynecomastia and hypogonadism [10].

In conclusion, this study revealed that chromosomal abnormalities contribute significantly to primary infertility in the Rwandan population. Thus, clinicians should consider these abnormalities in patients attending the fertility clinic. This study highlights the need for patients to consult as a couple, since infertility is a shared problem that affects both men and women.

REFERENCES

1. Arafa MM, Majzoub A, Alsaid SS, El W, Al A, Elbardisi Y, et al. Chromosomal abnormalities in infertile men with azoospermia and severe oligozoospermia in Qatar and their association with sperm retrieval intracytoplasmic sperm injection outcomes. 2018;132–9.
2. Tabong PTN, Adongo PB. Understanding the Social Meaning of Infertility and Childbearing: A Qualitative Study of the Perception of Childbearing and Childlessness in Northern Ghana. *PLoS One*. 2013;8(1).
3. Dhont N. Clinical, epidemiological and socio-cultural aspects of -infertility in resource-poor settings. Evidence from Rwanda. *Facts, views Vis ObGyn*. 2011;3(2):77–88.
4. Benksim A, Ph D, Elkhoudri N, Ph D, Addi RA, Baali A, et al. Difference between Primary and Secondary Infertility in Morocco : Frequencies and Associated Factors. 2018;12(2):142–6.
5. Fallis A.. Vol. 53, *Journal of Chemical Information and Modeling*. 2013. 1689–1699 p.
6. Masoumi SZ, Parsa P, Darvish N, Mokhtari S, Yavangi M, Roshanaei G. An epidemiologic survey on the causes of infertility in patients referred to infertility center in Fatemeh Hospital in Hamadan. *Iran J Reprod Med*. 2015;13(8):513–6.
7. Clementini E, Palka C, Iezzi I, Stuppia L, Guanciali-Franchi P, Tiboni GM. Prevalence of chromosomal abnormalities in 2078 infertile couples referred for assisted reproductive techniques. *Hum Reprod*. 2005;20(2):437–42.
8. Badovinac AR, Buretić-Tomljanović A, Starčević N, Kapović M, Vlastelić I, Randić L. Chromosome studies in patients with defective reproductive success. *Am J Reprod Immunol*. 2000;44(5):279–83.
9. Zorrilla M, Yatsenko AN. The Genetics of Infertility: Current Status of the Field. *Curr Genet Med Rep*. 2013;1(4):247–60.
10. Dohle GR, Colpi GM, Hargreave TB, Papp GK, Jungwirth A, Weidner W. EAU guidelines on male infertility. *Eur Urol*. 2005;48(5):703–11.

Non-Communicable Diseases - Challenging Agenda for The Rwandan Health Sector

Author: C. Mazimpaka^{1,*}

Affiliation: Partners In Health/Inshuti Mu Buzima, Rwanda

EDITORIAL

Worldwide, seven out of the 10 top causes of deaths are from non-communicable diseases (NCDs) [1]. Each year, 41 million deaths, accounting for 71% of all deaths globally, are due to NCDs [2]. In resource-limited countries, NCDs account for 85% of all premature deaths for people aged between 30-69 years [3]. In Rwanda, NCDs account for 44% of all deaths [4]. The estimated cost of treatment and productivity loss associated with NCDs in middle and low-income countries is estimated to be \$7 trillion between 2011 and 2025 [5].

Rwanda has made remarkable progress in rebuilding its health system that enabled the delivery of HIV and TB drugs after many believed it was too complex for resource-limited countries to achieve [6]. For example, with the global fund and the President's Emergency Plan for AIDS Relief (PEPFAR) funding, great progress was made in rebuilding our health system. The funding enabled the country to have almost universal access to HIV, TB, and malaria treatment and the best children vaccination program in the world, with 93% complete vaccination for 11 vaccines for boys and 93% complete vaccination for 12 vaccines for girls [6]. In addition, Rwanda has set up a sound legal framework with policies, strategies, an action plan to tackle major health care challenges, and a decentralized system that brings care to where people live [7]. However, the implementation of NCD prevention and treatment strategies remain an unfinished agenda.

The main challenge lies at the community level where low habit of routine health check-ups do not facilitate the health system in detecting and treating NCDs at the earlier stages. This habit originates from a health system that underwent major reforms at a time when infectious diseases were the most pressing healthcare challenge. During that time, most health systems in resource-limited countries were designed to address infectious diseases mostly in hospital-centered environment and major investments from PEPFAR and the Global Fund were made to educate communities and health care providers on alarming symptoms such as fever, pain, diarrhea, vomiting, headache and many other acute infectious disease signs. With most NCDs however, challenges lie in fact that patients have no symptoms until the diseases have progressed to late stages and that is why clinicians call these diseases "silent killers".

Mass screening, education campaigns and linkage to care are important, however, enhancing health systems strengthening initiatives to facilitate routine community screening and follow-up by community health workers can be a key solution [8, 9]. In addition, strengthening the primary health care facilities to include systematic NCD screening in outpatient departments, can facilitate early diagnosis and timely treatment [10]. This requires optimizing and leveraging resources in primary care delivery achieved in the health sector over decades from infectious diseases funding.

Keywords: Non-Communicable Disease, Premature Death, Early Detection, Linkage to Care, Rwanda

REFERENCES

- [1] R. Factors, "The Lancet : Global Burden of Disease study 2015 assesses the state of the world ' s health," 2015.
- [2] WHO, "The top 10 causes of death," no. May, 2018.
- [3] D. Detection, "Noncommunicable diseases," no. June 2018, pp. 1-4, 2019.
- [4] R. Of, P. Death, and D. U. E. To, "Non-communicable Diseases (NCD) Country Profiles. Rwanda," p. 2018, 2018.
- [5] P. Ncd, T. Who, A. Director-general, and N. Diseases, "Noncommunicable diseases prematurely take 16 million lives annually , WHO urges more action," no. January, 2015.
- [6] Binagwaho Agnes et al, "Rwanda 20 years on: investing

***Corresponding author:** Christian Mazimpaka, Institutions: Partners In Health/Inshuti Mu Buzima, Email: machrist2020@yahoo.fr, Phone: +250783545391; **Potential Conflicts of Interest (Col):** All authors: no potential conflicts of interest disclosed; **Funding:** All authors: no funding was disclosed; **Academic Integrity.** All authors confirm that they have made substantial academic contributions to this manuscript as defined by the ICMJE; **Ethics of human subject participation:** The study was approved by the local Institutional Review Board. Informed consent was sought and gained where applicable;

Originality: All authors: this manuscript is original has not been published elsewhere; **Type-editor:** Sessions (USA)

Review: This manuscript was peer-reviewed by three reviewers in a double-blind review process;

Received: 27th July 2018; **Initial decision given:** 4th October 2018; **Revised manuscript received:** 6th January 2019; **Accepted:** 5th March 2019

Copyright: © The Author(s). This is an Open Access article distributed under the terms of the Creative Commons Attribution License (CC BY-NC-ND) (click here) which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. **Publisher:** Rwanda Biomedical Centre (RBC)/Rwanda Health Communication Center, P.O.Box 4586, Kigali.

ISSN: 2079-097X (print); 2410-8626 (online)

Citation for this article: C. Mazimpaka: Non-Communicable Diseases- Challenging Agenda for The Rwandan Health Sector. Rwanda Medical Journal, Vol 77, no 2, pp 28-29, 2020

in life,” vol. 384, no. 9940, pp. 371–375, 2014.

[7] F. Sayinzoga and L. Bijlmakers, “Drivers of improved health sector performance in Rwanda : a qualitative view from within,” *BMC Health Serv. Res.*, pp. 1–10, 2016.

[8] K. Heidari et al., “Establishment of Health Clinics as Mass Screening and Referral Systems for Chronic Non-commu-

nicable Diseases in Primary Health Care,” vol. 3, no. 3, pp. 173–180, 2012.

[9] D. Maher, A. D. Harries, R. Zachariah, and D. Enarson, “A global framework for action to improve the primary care response to chronic non-communicable diseases : a solution to a neglected problem,” vol. 7, pp. 1–7, 2009.

Disseminated Cysticercosis in Rwanda—Case Report of a Patient Presenting with Difficulty with Walking and Skin Nodules

Authors: J. Tuan^{1,*}; L. Kailani^{2,3}; P. Ngabitsinze³; S. Umuganwa⁴; F. Munyaneza³; E. Musoni^{4,5}; A. L. Canales⁶; M. Nkeshimana^{3,7}

Affiliations: ¹Yale University School of Medicine, New Haven, Connecticut, Department of Internal Medicine, Section of Infectious Diseases; ²Geisel School of Medicine at Dartmouth, Hanover, New Hampshire; ³University of Rwanda College of Medicine and Health Sciences, Kigali, Rwanda, Department of Internal Medicine; ⁴University of Rwanda College of Medicine and Health Sciences, Kigali, Rwanda, Department of Pathology; ⁵University Teaching Hospital of Kigali, Kigali, Rwanda, Department of Pathology; ⁶Brigham and Women's Hospital, Boston, Massachusetts, Department of Dermatopathology; ⁷University Teaching Hospital of Kigali, Kigali, Rwanda, Department of Accident and Emergency Medicine.

ABSTRACT

Human cysticercosis is a parasitic disease caused by larval cysts of the *Taenia solium* tapeworm. The pathogenesis of disseminated cysticercosis involves migration of *Taenia solium* embryos from the hepatportal system to organs and tissues in the body. Symptoms may range anywhere from neurologic sequelae such as seizures, to skin manifestations such as subcutaneous nodules [1-2]. Disseminated cysticercosis is a rare complication of cysticercosis, and globally, fewer than 50 cases of disseminated cysticercosis are documented [1-3]. Human cysticercosis is endemic to Rwanda with a seroprevalence of 7% [4]. An increased prevalence of cysticercosis has been noted among epileptic persons in the southern province of Rwanda [5].

We describe a 46-year-old Rwandan woman from Kamonyi District, in the southern province of Rwanda, who presented with a two-week history of bilateral lower limb weakness, causing difficulty walking. She had associated fevers and headache. She was febrile and tachycardic, with decreased lower extremity strength and subcutaneous nodules on her trunk and extremities. Laboratory data demonstrated leukocytosis with neutrophilic predominance and mild eosinophilia. Excisional biopsy of a subcutaneous nodule revealed a cyst containing a protoscolex with suckers, ramifying cistern, and calcareous bodies; brain magnetic resonance imaging demonstrated diffuse, cystic cerebral, cerebellar, and soft tissue lesions—consistent with disseminated cysticercosis.

The patient received a fourteen-day course of albendazole and prednisolone, and afterwards, noted restored ability to walk independently. In this case, prompt diagnosis and treatment of disseminated cysticercosis led to dramatic clinical improvement.

Keywords (MeSH): Cysticercosis, Neurocysticercosis, *Taenia Solium*, Rwanda

CASE PRESENTATION

A 46-year-old woman from Kamonyi District in the southern province of Rwanda with no significant past medical history presented to University Teaching Hospital of Kigali with a two-week history of acute-onset bilateral lower limb weakness and calf

pain. She rated the pain at a three out of ten in severity, and she stated that this pain had caused her to have difficulty standing. The pain was associated with a two-week history of intermittent fevers, headache, and generalized weakness. She also reported a three-year history of multiple, painless subcutaneous nodules in her neck, chest, and arms. The patient was a farmer who had previ-

***Corresponding author:** Jessica Tuan, MD, MS, Email: Jessica.Tuan@Yale.edu, Phone: 630-532-3325, Address: 333 Cedar Street, PO Box 208022, New Haven, Connecticut, 06510; **Potential Conflicts of Interest (Col):** All authors: no potential conflicts of interest disclosed; **Funding:** All authors: no funding was disclosed; **Academic Integrity:** All authors confirm that they have made substantial academic contributions to this manuscript as defined by the ICMJE; **Ethics of human subject participation:** The study was approved by the local Institutional Review Board. Informed consent was sought and gained where applicable; **Originality:** All authors: this manuscript is original has not been published elsewhere; **Type-editor:** Matthew Cardillo (USA)

Review: This manuscript was peer-reviewed by three reviewers in a double-blind review process;

Received: 21st Sept 2019; **Initial decision given:** 26th Oct 2019; **Revised manuscript received:** 21st January 2020; **Accepted:** 25th January 2020

Copyright: © The Author(s). This is an Open Access article distributed under the terms of the Creative Commons Attribution License (CC BY-NC-ND) (click here) which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. **Publisher:** Rwanda Biomedical Centre (RBC)/Rwanda Health Communication Center, P.O.Box 4586, Kigali.

ISSN: 2079-097X (print); 2410-8626 (online)

Citation for this article: J. Tuan; L. Kailani; P. Ngabitsinze et al. Disseminated Cysticercosis in Rwanda—Case Report of a Patient Presenting with Difficulty with Walking and Skin Nodules Rwanda Medical Journal. Vol 77, no 2, pp 30-33, 2020

ously lived in Nyaruguru District, an area with many pork farmers. Two years prior, she had moved to a rural area in Kamonyi District. She reported that she had been raising pigs on the farm in order to sell them, but denied ingestion of pork meat. She generally had poor hygiene practices.

Two days prior to her presentation, she had presented to the local district hospital in Rwanda with similar symptoms. There, she had laboratory data that demonstrated a leukocytosis with a white blood cell count of 16,550 cells/ μ L. C-reactive protein was elevated at 96 mg/dL. Hepatitis B virus surface antigen and Hepatitis C virus antibody were negative.

At our hospital, on physical examination, she was a well-nourished female in no acute distress. She had a temperature of 39.2°C, heart rate of 110 bpm, blood pressure of 109/63 mmHg, respiratory rate of 22 cpm, and oxygen saturation of 93% on room air. She had several firm, non-tender, mobile nodules, approximately 1.5 cm in diameter. One nodule was located in the sternal area, another in the right anterolateral chest, two in the right neck, and a cluster of three nodules were located in her right forearm (Figure 1). She had tenderness to palpation of the right antecubital region and her bilateral calves. The range of motion of her bilateral lower extremities was slightly limited due to this pain. Aside from strength of 4/5 in the bilateral lower extremities, she was neurologically intact with preserved motor function and sensation. She did not demonstrate clinical signs of muscular pseudohypertrophy. Ophthalmoscopic exam was not performed.

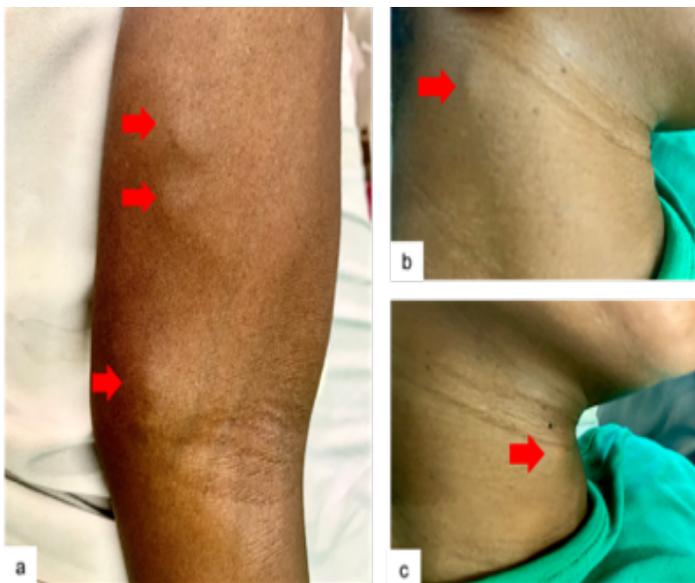


Figure 1. **a)** Right forearm with a cluster of subcutaneous nodules, approximately 1.5 cm in diameter. **b)** Right neck subcutaneous nodule. **c)** Right neck subcutaneous nodule

Laboratory data revealed a leukocytosis with a white blood cell count of 15,520 cells/ μ L with 81% neutrophils, 9% lymphocytes, 6% monocytes, and 4% eosinophils. Hemoglobin was 9.3 g/dL with a mean corpuscular volume of 88.4 fL, and a platelet count of 389,000 cells/ μ L. Blood urea nitrogen was 2.3 mmol/L and creatinine was 40 μ mol/L. She had an aspartate transaminase of 22.2

U/L, alanine transaminase of 12.1 U/L. C-reactive protein was elevated to 101 mg/dL. Rapid Human Immunodeficiency Virus test and malaria thick smear were negative. Blood cultures revealed no growth. Stool ova and parasites and immunological tests for *Taenia solium* were not performed. Chest x-ray demonstrated no acute process.

The patient was admitted to our hospital and given intravenous fluids and started empirically on ceftriaxone. She was also started on empiric prednisolone to reduce inflammation. On day five, a fine needle aspirate of the right anterolateral chest wall nodule demonstrated significant fragments of acellular lamellate membrane, keratinous debris, and lymphocytes, which were suggestive of cysticercosis via Romanowski stain (Figure 2a).

The hematoxylin and eosin stain section from the excisional biopsy of the nodule showed a fibrous cyst containing a protoscolex with identifiable suckers and ramifying cistern. There was a loose channel-rich stroma, and calcareous bodies were readily noted (Figure 2b). Given the histopathologic findings, she was diagnosed with disseminated cysticercosis.

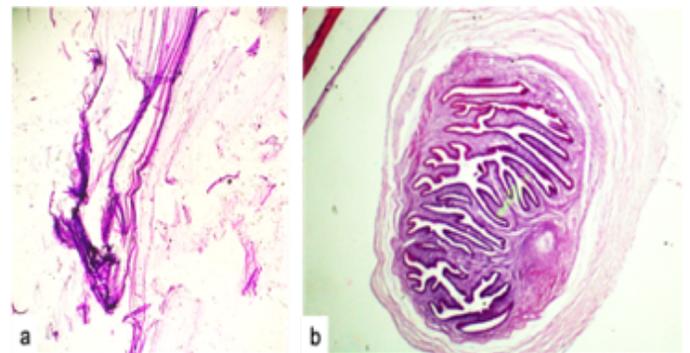


Figure 2. **a)** Fine needle aspirate of the right anterolateral chest wall subcutaneous nodule demonstrating significant fragments of acellular lamellate membrane (Romanowski stain, 100x magnification). **b)** The Hematoxylin and Eosin stain section from the excisional biopsy of the nodule showed a fibrous cyst containing a protoscolex with identifiable suckers and ramifying cistern. There was a loose channel-rich stroma and calcareous bodies were readily noted (Hematoxylin and Eosin stain, 100x magnification).

The patient was discharged with a planned thirty-day course of albendazole, but due to insurance issues and medication availability, only received a fourteen-day course. She received prednisolone starting at 40 mg PO daily for two weeks, followed by a steroid taper. The patient did not have imaging of peripheral extremities or musculature, but she was requested to follow-up outpatient for further brain imaging, which she eventually completed, three weeks post-discharge. Magnetic Resonance Imaging (MRI) of the brain revealed diffuse, cystic lesions with thin, non-enhancing walls with central hyperintense foci in the cerebrum, cerebellum, lateral ventricles, and craniofacial and neck soft tissues (Figure 3). These features were consistent with disseminated cysticercosis with vesicular stage neurocysticercosis with a racemose neurocysticercosis component. There were no features of muscular pseudohypertrophy noted on imaging.

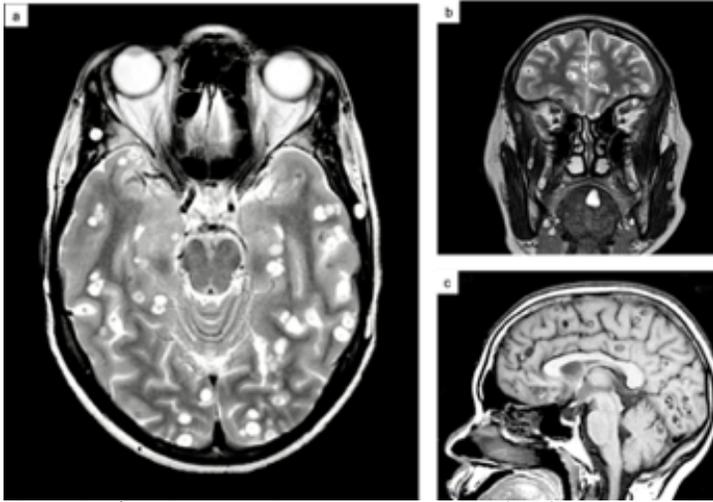


Figure 3. a) Brain Magnetic Resonance Imaging (MRI) transverse view demonstrated diffuse cystic lesions with thin, enhancing walls with central hyperintense foci in the cerebrum, cerebellum, lateral ventricles, and craniofacial and neck soft tissues, consistent with disseminated cysticercosis with vesicular stage neurocysticercosis with a racemose neurocysticercosis component. b) Brain MRI coronal view demonstrated diffuse cystic lesions with central hyperintense foci in the craniofacial and neck soft tissues. c) Brain MRI sagittal view demonstrated features of disseminated cysticercosis with vesicular stage neurocysticercosis with racemose neurocysticercosis component.

At follow-up three weeks after discharge, she denied headaches or fevers, but did report some episodes of intermittent dizziness. Although her physical exam was significant for the persistent subcutaneous nodules, which were similar in character to her initial presentation, she reported clinical improvement in ambulation, and she was able to stand independently.

DISCUSSION

Cysticercosis is a parasitic disease, caused by larval cysts of the *Taenia solium* tapeworm, commonly noted in areas such as Africa, Asia, and Latin America [1]. Cysticercosis can be transmitted via the fecal-oral route by ingestion of the eggs from the *Taenia solium* tapeworm carrier, rather than undercooked pork meat alone, and poor hygiene practices [2]. There has been a noted increased prevalence of cysticercosis in people with epilepsy in the southern province of Rwanda [5]. Prior literature reports that in Rwanda, approximately 21% percent of patients with epilepsy in southern Rwanda were positive for cysticercosis immunoblot testing, which detect glycoproteins of *Taenia solium* [5,6]. Both porcine and human cysticercosis are endemic in Rwanda, and cysticercosis is noted to be prevalent in areas where pigs are farmed and raised in Africa [7,8].

Disseminated cysticercosis is noted to be a rare complication of cysticercosis, and occurs when *Taenia solium* embryos disseminate from the hepatportal system to other organ systems and tissues in the body, including the central nervous system most commonly, as well as skeletal muscle, lungs, liver, eyes, and brain [3,9]. Cardiac muscle is less frequently involved [10]. Based on site of involvement, cyst burden and host immunity, symptoms of disseminated cysticercosis may include seizures, muscular hypertrophy, and dermatologic nodules [2]. Limited case reports describe such complications

in Rwanda. Less than 50 cases of disseminated cysticercosis have been reported worldwide [1-3]. Those cases were predominantly located in India [2,3]. Seroprevalence of human cysticercosis in Rwanda has been reported at 7% [4].

The diagnostic criteria for neurocysticercosis were revised in 2016 [11]. Absolute criteria include histologic parasitic confirmation, subretinal cysts, scolex within a cyst in the brain. Neuroimaging criteria include Major [cystic lesions with no scolex, enhancing lesions, cysts which are multilobulated, calcifications], Confirmative [cyst resolution after cysticidal antiparasitic medications, resolution of single enhancing lesion spontaneously, migrating ventricular cysts on follow-up neuroimaging], and Minor [presence of hydrocephalus, leptomeningeal enhancement]. Clinical criteria include presence of anticysticercal antibodies or cysticercal antigen by standardized tests, systemic cysticercosis, household *Taenia* carrier, compatible clinical presentation, and habitation in an endemic area [11]. For a definitive diagnosis, the following is needed: one absolute criteria, or two major neuroimaging criteria plus any clinical criteria; or, one major and one confirmative neuroimaging criteria plus any clinical criteria; or, one major neuroimaging criteria plus two clinical criteria (including one major clinical criteria) along with other pathologies which yield similar neuroimaging findings excluded. Probable diagnosis involves those with one neuroimaging criteria along with evidence of exposure [11].

Serologic studies such as enzyme-linked immunoelectrotransfer blot should be performed to confirm the diagnosis of neurocysticercosis [12]. Excisional biopsy of skin or muscle lesion can help support a diagnosis of extraneural cysticercosis, although positive testing could reflect prior infection and negative tests do not exclude cysticercosis [13]. Patients with suspected neurocysticercosis should have neurological imaging performed [12]. MRI of the brain should be repeated at least every six months to assess for resolution of cystic components [12].

Anthelmintic drugs are indicated for patients with viable or degenerated cysts on neuroimaging, with the exception of high cyst burden, presence of calcified lesions only, or untreated hydrocephalus [12]. Use of these antiparasitic drugs have been reported to yield an improved prognosis [12]. However, anti-helminthic drugs can increase degeneration of viable cysts, thus leading to inflammation, cerebral edema, hydrocephalus, precipitating seizure activity, and ultimately lead to brain herniation [12]. Steroids are recommended to be administered before and concomitantly with anti-helminthic drug therapy, which has been associated with decreased seizures [12].

Albendazole is the typical treatment and has been shown to have superior central nervous system penetration, compared to praziquantel [1]. In cases from literature review, albendazole (15 mg/kg/day for 30 days) and praziquantel (50 mg/kg/day for a duration of 15-20 days), which are both cysticidal, had also been co-administered for treatment of cysticercosis

[1,2]. Implementation of these antiparasitic drugs decreases parasite load, leading to antigen release, which may cause local swelling and severe inflammation [1]. Thus, there is a role for corticosteroids in reducing inflammation, prior to and during initiation of antiparasitic drugs [1].

The *Taenia solium* tapeworm itself can be ingested from undercooked pork, and it is advised to cook meat thoroughly. Additional measures to reduce *Taenia solium* disease and thus cysticercosis include preventing pigs from scavenging food that may be infected with *Taenia solium* eggs, optimizing slaughterhouse meat inspection and sanitation, and administering anthelmintic medications to entire groups of pigs on farms and even human communities [14].

Enhanced access to clean water as well as improved sanitation and hygiene practices, such as handwashing, should be advised, especially given tapeworm carriers can re-infect themselves. Tapeworm carriers, especially if food handlers, can enhance the risk of cysticercosis acquisition [12, 14].

REFERENCES

- [1] S.Y. Park, M.H. Kong, J.H. Kim, and K.Y. Song, "Disseminated Cysticercosis," *J Korean Neurosurg Soc.*, Vol. 49, No. 3, pp. 190–193, Mar. 2011.
- [2] K.A. Bothale, S.D. Mahore, and S.A. Maimoon, "A rare case of disseminated cysticercosis," *Trop Parasitology*, Vol. 2, No. 2, pp. 138–141, 2012.
- [3] A. Banu, N. Veena, "A rare case of disseminated cysticercosis: Case report and review of literature," *Indian J Med Microbiology*, Vol. 29, pp. 180–3, 2011.
- [4] P. Odeniran and I.O. Ademola, "Zoonotic Parasites of Wildlife in Africa: A Review, *African Journal of Wildlife Research*," Vol. 46, No. 1, pp. 1-13, 2016.
- [5] R. Rottbeck, J.F. Nshimiyimana, P. Tugirimana, et. al., "High Prevalence of Cysticercosis in People with Epilepsy in Southern Rwanda," *PLoS Negl Trop Dis*, Vol. 7, No. 11, pp. 2558, Nov. 2013.
- [6] V.C.W. Tsang and M. Wilson, "*Taenia solium* cysticercosis: an under-recognized but serious public health problem," *Parasitology Today*, Vol. 11, pp. 124-6, 1995.
- [7] A. Zoli, O. Shey-Njila, E. Assana, J.P. Nguekam, P. Dorny, J. Brandt, and S. Geerts, "Regional status, epidemiology and impact of *Taenia solium* cysticercosis in Western and Central Africa," *Acta Trop*, Vol. 87, No. 1, pp. 35-42, Jun. 2003.
- [8] A.C. White, "Neurocysticercosis: A Major Cause of Neurological Disease Worldwide," *Clinical Infectious Diseases*, Vol. 24, pp. 101-15, 1997.
- [9] N. Saeed, A. Ehsan, and S.M. Vasenwala, "Disseminated cysticercosis incidentally diagnosed in a patient of fracture shaft of femur," *BMJ Case Reports*, Volume 2017, pp. 1-3, Feb. 2017.
- [10] B.K. Jain, S.S. Sankhe, M.D. Agrawal, and P.S. Naphade, "Disseminated cysticercosis with pulmonary and cardiac involvement," *Indian Journal of Radiology and Imaging*, Vol. 20, No. 4, pp. 310–313, Nov. 2010.
- [11] O.H. Del Brutto, T.E. Nash, A.C. White Jr, et. al., "Revised diagnostic criteria for neurocysticercosis," *J Neurol Sci* Vol. 372, pp. 202–210, 2017.
- [12] White AC Jr, Coyle CM, Rajshekhar V, et. al. Diagnosis and Treatment of Neurocysticercosis: 2017 Clinical Practice Guidelines by the Infectious Diseases Society of America and the American Society of Tropical Medicine and Hygiene. *Clin Infect Dis*, Vol. 66, No. 8, 2018.
- [13] Gutierrez Y. Cysticercosis, Coenurosis, and Sparganosis. *Diagnostic Pathology of Parasitic Infection with Clinical Correlation*, Gutierrez Y (Ed), Oxford University Press, Oxford, 2000, pp.635.
- [14] Kungu JM, Dione MM, Ejobi F, et. al. Risk factors, perceptions and practices associated with *Taenia solium* cysticercosis and its control in the smallholder pig production systems in Uganda: a cross-sectional survey. *BMC Infect Dis*, Vol. 17, No. 1, 2017.

CONCLUSION

In summary, disseminated cysticercosis is a rare complication of cysticercosis, which has been rarely described in literature from Rwanda. This patient presented with an atypical presentation of cysticercosis, with manifestations involving the skin, skeletal muscles, soft tissues, and brain.

Thus, clinicians in cysticercosis-endemic regions, such as Rwanda, should keep disseminated cysticercosis on the differential diagnosis when patients present with such atypical clinical manifestations, including diffuse organ involvement, in order to recognize and promptly treat affected patients. Implementation of a comprehensive, systematic method to document cases of cysticercosis and its sequelae—particularly in endemic regions, such as Rwanda—would be invaluable in elucidating the epidemiologic patterns and helping to prevent transmission of this important disease.

Severe Anemia by a Leech Infestation in a Pediatric Patient: A Case Report

Authors: D. Rutagumba^{1,*}; B. Niyoyita¹; R. Nyirasafari²

Affiliations: ¹University of Rwanda, Kigali, Rwanda; ²Rwanda Military Hospital, Kigali, Rwanda

ABSTRACT

CASE PRESENTATION: We report a case of a five-year-old female patient who presented to the Rwanda Military Hospital for left ear foreign body. She had a background of hematemesis and body weakness and was found to have severe anemia. The anemia resolved after a blood transfusion and vomiting of a leech parasite.

DISCUSSION: Leeches are rare parasites that can cause life-threatening anemia. Leeches are transmitted by using unfiltered infested water through bathing or drinking or while swimming in contaminated water. Although leech infestation is a rare cause of severe anemia, it should be considered as a possible cause of hematemesis, vaginal, or gastrointestinal bleeding.

Keywords: Leech, Infestation, Anemia, Case Report

INTRODUCTION

Anemia in children is a common worldwide problem which can have serious consequences. Anemia due to active bleeding in the upper respiratory tract or in the gastrointestinal system can be life-threatening. Previous case reports have described cases of anemia associated with leech infestation in children [1,2]. Leeches are hermaphroditic endoparasites which nourish on host blood and live in contaminated water. Leeches vary in color and in length, ranging anywhere from a few millimeters to half a meter [3].

Leech infestation occurs through drinking non-clean infested water from streams, pools, dam, and springs [3]. Leech bodies are composed of thirty-four segments. They attach to their hosts by two muscular suckers, called the anterior and posterior sucker. Leeches then can use three teeth inside their anterior sucker for biting and the posterior sucker is used for leverage [4]. The amount of blood feed can reach up to ten times the leeches body weight [5,6].

The leeches have mechanisms which allow them to continuously receive nourishment from their host without being noticed. They have an anesthetic product in their saliva that reduces the sensation felt by the host. In addition, leech saliva contains a va-

sodilator that causes the blood vessels near the leech to become dilated, and thus provides the leech with a bleeding supply that can reach 150 ml of blood within 48 hours which can cause patients infested to develop severe anemia due to this significant blood loss [7]. Hematophagous leeches are able to attach to their host until when they are full, that is when they will fall and start digestion [5,7].

Leeches can present on the mucosa of the oropharynx, nasopharynx, tonsils, esophagus, nose, trachea or vagina, but rarely in the rectal mucosa. In rare cases, the leech can also be the cause of upper airway obstruction [2,8–10]. The clinical presentation of leech infestation depends on the exact site of the infestation. The nose, especially lateral and medial nasal walls of the nose, and the vagina are the most common sites of living leech infestation. Epistaxis is the most common presenting clinical symptom [10]. Depending on the site of infestation, hematemesis, epistaxis, vaginal bleeding, signs of upper airway obstruction, all these may occur [1]. In our patient, the presenting features include hematemesis, weakness, pallor and severe anemia.

The removal of the leech requires significant attention because of its tight attachment. Removal usually require endoscopic or laryngoscopy-guided removal under general anesthesia [12]. Internal attachment, such as the vagina is more likely to require medical

***Corresponding author:** Dr Dieudonne RUTAGUMBA, CMHS/UR, Email: rdodos2020@yahoo.fr; **Potential Conflicts of Interest (Col):** All authors: no potential conflicts of interest disclosed; **Funding:** All authors: no funding was disclosed; **Academic Integrity.** All authors confirm that they have made substantial academic contributions to this manuscript as defined by the ICMJE; **Ethics of human subject participation:** The study was approved by the local Institutional Review Board. Informed consent was sought and gained where applicable;

Originality: All authors: this manuscript is original has not been published elsewhere; **Type-editor:** Ahmed (USA)

Review: This manuscript was peer-reviewed by three reviewers in a double-blind review process;

Received: 23rd Sept 2019; **Initial decision given:** 10th Oct 2019; **Revised manuscript received:** 18th Nov 2019; **Accepted:** 21st Nov 2019

Copyright: © The Author(s). This is an Open Access article distributed under the terms of the Creative Commons Attribution License (CC BY-NC-ND) (click here) which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. **Publisher:** Rwanda Biomedical Centre (RBC)/Rwanda Health Communication Center, P.O.Box 4586, Kigali.

ISSN: 2079-097X (print); 2410-8626 (online)

Citation for this article: D. Rutagumba; B. Niyoyita; R. Nyirasafari. Severe Anemia by a Leech Infestation in a Pediatric Patient: A Case Report, Rwanda Medical Journal, Vol 77, no 2, pp 34-36, 2020

intervention [11]. For induction, a sevoflurane-oxygen mixture via facemask is used. The leech is paralyzed and detached from its attachment by sevoflurane. Blunt jaw forceps are preferable for removal. Hirudin, an anticoagulant enzyme released by salivary glands of leeches, can continue to cause blood loss, and therefore, the entirety of the leech's body should be removed. Direct endoscopy is the gold standard to obtain an emergency diagnosis and to remove the leech [4].

Though the following case is unusual cause of life threatening anaemia, it should be considered in differential diagnosis of anaemia especially in poor settings.

CASE PRESENTATION

History: A 5-year-old female child presented to Rwanda Military Hospital (RMH) from a district hospital. She was referred to pediatric department with a two-week history of foreign body (stone) in her left ear. Her parents had delayed to consult because they thought the stone would come out by itself. They decided to seek medical care when they saw a purulent discharge from the left ear.

On review of system, it was reported that the child had developed a new-onset of vomiting gastric content mixed with fresh blood, five days prior to the consultation, associated with fatigue. She also reported mild headache and abdominal pain, but there was no diarrhea, no fever, no constipation, no hematochezia nor melena. She was previously healthy, and the past medical history was unremarkable. She lives in a rural area in the eastern province of Rwanda and comes from a family with poor socio-economic level. Her mother reported that the child sometimes drank unsafe water from a dam near their home.

Physical examination: On physical examination at admission, she was noted to be a well-nourished child with normal anthropometric measures plotted on the WHO curves weight: 17kg, Height: 105cm, with weight-for-height 0 z-score, and height-for-age $1 < HA < 0$. She was asthenic, had a temperature of 37.0°C, was tachycardic with a heart rate of 135 beats per minute, the capillary refill time was below two seconds, and the extremities were warm. She had tachypnea with respiratory rate of 28 cycles per minute and oxygen saturation of 97% on room air. She had severe conjunctiva and palmo-plantar pallor, purulent discharge in the left ear canal and we were not able to visualize the tympanic membrane because of pus in the external ear canal. There was no lymphadenopathies, no hepato-splenomegaly, no petechiae and no bruises. The rest of the physical exam was unremarkable.

Investigations: The initial Full Blood Count (FCB) showed severe normocytic anemia with a hemoglobin was 3.4g/dl and mean corpuscular volume of 96.2 fl. The rest of her FBC was normal. The C-reactive protein was 35.7mg/dl, and the malaria smear was negative.

Management: The patient had a clinical picture of significant anemia, and was treated with a transfusion of Packed Red Blood cell (PRBCs) at two consecutive sessions at 10 ml/kg over slow

infusion. The Ear, Nose and Throat (ENT) department was consulted and the patient was scheduled for foreign body removal the following day. Meanwhile the patient was given neomycin ear drops. Twelve hours into her hospital stay the child vomited a brown moving parasite found to be a leech (Figure 1). After vomiting this parasite, hematemesis stopped and there was no other episode of vomiting. After blood transfusion, the fatigue resolved, the vital signs normalized, and the child became playful as usual.



Figure 1: Vomited leech with dark brown color

She was then sent to the Endoscopic department for exploration and removal of any other possible remaining parasites. The upper GI endoscopy (checked oropharynx, esophagus, stomach and duodenum) was done on day three of admission and fortunately the endoscopic exam revealed no other parasites, no varices and there was no gastric or duodenal ulcers, nor stenosis. The nose and nasopharynx were not endoscopically examined for leeches. On the fifth day of admission the patient was then reviewed by an ENT surgeon and the small stone was removed from the left external ear canal and a washout performed. After six days in hospital (three days after vomiting the leech), the FBC was repeated with a satisfactory hemoglobin level of 10.3g/dl. The patient was discharged after seven days in hospital and was given an appointment to come back after one week but she did not attend.

DISCUSSION

Apart from being rare and affecting poor rural patients, anemia associated with leech infestation can easily be mistaken for another cause. It can be mistaken for upper GI bleeding like peptic ulcer disease, or esophageal varices when it affects the upper GI whereas when it affects the respiratory system can be thought to be foreign body, or even other wide range of causes of upper airway obstruction. These challenges in diagnosis have been described in previous case reports [11,13,14].

The leech may also present as bleeding disorders such as platelet disorder or coagulation factors dysfunction as described in previous case report [8,10,15], but the difference exists on presence of isolated anemia in leech infestation induced anemia due to blood loss, whereas if the anemia is associated with other causes abnormal parameters will be found. Another indicator to ensure that it is the leech induced bleeding not something else, the bleeding will stop after the parasite is removed either spontaneously or by medical

intervention [15]. Our case also presented as upper GI bleeding, with clinical and biological findings of anemia and bleeding stopped after the parasite was vomited.

Most previous case reports have described leech infestation to be common in rural areas [6,13,16]. To the best of our knowledge, this is the first case to be reported in Rwanda and could not be used as reference to understand the epidemiology and characteristics of leech infestation in Rwanda rather this can serve as a reminder to clinicians and researchers in this setting.

Hygiene education and improved sanitation infrastructure are the key to preventing this rare but potentially life-threatening complication of drinking unsanitary water [17]. In our case, the child came from a poor family living in a rural area, requiring them to drink and to bath in unclean water from a dam nearby their home, which is thought to be the source of this leech parasite.

REFERENCES

- [1] M. Taşkesen, S. Katar, and H. Başçık, "An unusual cause of gastrointestinal bleeding and severe anemia in a child: Leech infestation," *J. Trop. Pediatr.*, vol. 55, no. 5, pp. 338–339, 2009.
- [2] A. Hossein, J. Rouhi, S. Vegari, S. S. Vahdati, and D. Porhosein, "Nasopharyngeal Bleeding due to Leech Bites in a 9-month-old Infant," *Indian J. Pediatr.*, vol. 77, pp. 573–574, 2010.
- [3] D. Mekonnen, "Leech infestation: the unusual cause of upper airway obstruction," *Ethiop. J. Health Sci.*, vol. 23, no. 1, pp. 65–68, 2013.
- [4] S. N. S. Shibabaw, "Severe anemia due to pharyngeal leech infestation; a case report from Ethiopia," *BCM Surg.*, pp. 1–3, 2017.
- [5] M. A. Behçet AL, Mehmet Emin YENEN, "Rectal bleeding due to leech bite : a case report," *Turkish J. Trauma Emerg. Surg.*, vol. 17, no. 1, pp. 83–86, 2011.
- [6] N. Saki, F. Rahim, S. Nikaghalagh, and G. Saki, "Meta analysis of the Leech as live foreign body: Detection, Precaution and Treatment," *Pakistan J. Biol. Sci.*, vol. 12, no. 24, pp. 1556–1563, 2009.
- [7] N. Shitaye and S. Shibabaw, "Severe anemia due to pharyngeal leech infestation; A case report from Ethiopia," *BMC Surg.*, vol. 17, no. 1, pp. 1–3, 2017.
- [8] A. Hossein et al., "Nasopharyngeal Bleeding due to Leech Bites in a 9-month- old Infant," *J. Trop. Pediatr.*, vol. 77, no. 5, pp. 573–574, 2010.
- [9] M. Saha and S. Nagi, "Intraperitoneal leech: A rare complication of leech bite," *J. Indian Assoc. Pediatr. Surg.*, vol. 16, no. 4, p. 155, 2011.
- [10] A. Muhammad Ashraf, Ruqia Fida, Muhammad Ishaq,

CONCLUSION

Leech infestation is rare but can happen especially in rural areas. Its effects can be severe enough to cause severe anemia, hence it should be considered in the differential diagnosis amongst the causes of unexplained internal bleeding and upper respiratory tract obstruction. History taking should focus on the circumstances surrounding suspicion of leech infestation. Prevention of this disease can be promoted by teaching patients about the necessity of using and consuming clean, sanitized water. Researches are needed to learn more about these parasites in Rwanda.

INFORMED CONSENT: Verbal informed consent was obtained from the mother of the patient for publication of this case report and the image used in the case.

This case report has been described using CARE checklist [18].

Israr Ahmed Akdund, "Leech infestation in upper respiratory tract," *Medical Channel*, vol. 19, no. 2, pp. 60–62, 2013.

[11] M. Rafeey and Y. Jabbari-Mogaddam, "Intermittent gastrointestinal bleeding in a child: Leech infestation," *Iran. J. Pediatr.*, vol. 22, no. 4, pp. 572–573, 2012.

[12] M. J. Hannan and M. M. Hoque, "Leech infestation in children through body orifices: Experience in a hospital in bangladesh," *World J. Surg.*, vol. 36, no. 9, pp. 2090–2092, 2012.

[13] C. Krüger, I. Malleyeck, and O. H. E. Olsen, "Aquatic leech infestation: A rare cause of severe anaemia in an adolescent Tanzanian girl," *Eur. J. Pediatr.*, vol. 163, no. 6, pp. 297–299, 2004.

[14] B. B. A. Estambale, R. Knight, and R. Chunge, "Haematemesis and severe anaemia due to a pharyngeal leech (*myxobdella africana*) in a kenyan child: A case report," *Trans. R. Soc. Trop. Med. Hyg.*, vol. 86, no. 4, p. 458, 1992.

[15] H. Ağin, F. Y. Ayhan, G. Gülfıdan, D. Çevik, and H. Derebaşı, "Severe Anemia Due to the Pharyngeal Leech *Limnatis nilotica* in a Child," *Turkish Soc. Parasitol.*, vol. 32, no. 3, pp. 247–248, 2008.

[16] Tesmegen Kelaye, "Assessment of Prevalence of Exclusive Breast Feeding Practice and Associated Factors among Under Six-Month-Old Children Selected Woreda South Nation Nationality of People Regional State ," *J. Nutr. Food Sci.*, pp. 1–7, 2017.

[17] T. Temesgen and T. Tilahun, "CASE REPORT Vaginal Leech Infestation : A Rare Cause of Hypovolumic Shock In Postmenopausal Woman," *Ethiop. J. Health Sci.*, vol. 25, no. 5, pp. 377–380, 2015.

[18] C. Case Report guidelines, "CARE Checklist of information to include when writing a case report," 2016.

Bedside Ultrasound Scan in the Intensive Care Unit of a Referral Hospital in Kigali, Rwanda: Case reports and a review of the literature—Case Series

Author: K. U. Tobi^{1*}; O. F. Umuhire²; L. Mumporese²; L. Uwamahoro²; G. Mbanjumucyo²; P. R. Banguti²

Affiliation: ¹Department of Surgery and Anesthesiology, Hage Geingob Campus, University of Namibia, Windhoek, Namibia; ²Department of Emergency and Critical Care Medicine, University of Rwanda, Kigali, Rwanda.

ABSTRACT

A bedside ultrasound scan has become an integral part of care in the intensive care unit (ICU). Its advantages include rapid diagnosis and thus management of life-threatening conditions, reduction in the cost of care, and reduced need for transport of patients out of the unit. These advantages have made bedside ultrasound scans one of the best tools in the hands of the critical care physician.

In this case series, we present four patients with different clinical states which include confirmation of central line placement, confirmation of pneumothorax and diagnosis of hemoperitoneum. The management of these patients was positively influenced by using bedside ultrasound scans in the intensive care unit.

Bedside ultrasound in the management of critically ill patients is quick, reliable, and has the potential to influence patient outcomes positively. It is cost-effective, and safe to use for all categories of ICU patients. Deployment of this simple but effective tool is a step in the right direction in the quest to improve patient care in the ICU.

Keywords (MeSH): Bedside Ultrasound Scan, ICU, Case reports, Pneumothorax, Hemoperitoneum

BACKGROUND

Bedside ultrasound scan (USS) has become an integral part in the care of critically ill patients in the intensive care unit [1]. The advantages of Bedside USS include the fact that it is easy to use and fast diagnostic power which allows quick management of life-threatening conditions. In addition, it is inexpensive, and does not require transport of patients out of the unit [2]. These advantages have made ultrasound scans one of the best tools in the hands of the critical care physician.

About three years ago, a large multi-center study involving three different countries in Europe reported the use bedside USS in about 36% of patients daily. About 97% of them were for diagnostic purposes and only 3% were for interventional purposes [3]. Many clinical scenarios have been managed successfully in the intensive care unit with the use of ultrasound scans. Some of these situations include the placement of cen-

tral venous catheters, diagnosis and confirmation of pneumothorax and other lung pathologies, and the diagnosis of haemoperitoneum [4-6].

As with all other diagnostic or interventional tools, bedside ultrasound scan is not without limitations. These include its operator-dependency, its high cost of initial purchase, and the continued maintenance cost, all of which provide challenges in a resource-challenged environment. In addition, there is a lack of documented evidence to prove its effect on improving patients' outcome [7]. This may be due to the fact that despite the use of Beside USS, some of the patients may still die. Despite these limitations, bedside ultrasound scans remain an auspicious and helpful tool in intensive care settings.

In this case series, we present four patients with different clinical states managed at King Faisal Hospital, Kigali in which a bedside ultrasound scans positively influenced their management in the intensive care unit.

***Corresponding author:** Dr. Kingsley Ufuoma Tobi, Email: tobikingsley265@gmail.com; Department of Surgery and Anaesthesiology, Hage Geingob Campus, University of Namibia, Windhoek, Namibia; **Potential Conflicts of Interest (Col):** All authors: no potential conflicts of interest disclosed; **Funding:** All authors: no funding was disclosed; **Academic Integrity.** All authors confirm that they have made substantial academic contributions to this manuscript as defined by the ICMJE; **Ethics of human subject participation:** The study was approved by the local Institutional Review Board. Informed consent was sought and gained where applicable; **Originality:** All authors: this manuscript is original has not been published elsewhere; Type-editor: Hannah King (USA)

Review: This manuscript was peer-reviewed by three reviewers in a double-blind review process;

Received: 06th May 2019; **Initial decision given:** 30th July 2019; **Revised manuscript received:** 03th August 2019; **Accepted:** 101st September 2019

Copyright: © The Author(s). This is an Open Access article distributed under the terms of the Creative Commons Attribution License (CC BY-NC-ND) (click here), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. **Publisher:** Rwanda Biomedical Centre (RBC)/Rwanda Health Communication Center, P.O.Box 4586, Kigali.

ISSN: 2079-097X (print); 2410-8626 (online)

Citation for this article: K. U. Tobi; O. Felix; L. Mumporese, et al; Bedside ultrasound scan in the intensive care unit of a referral hospital in Kigali, Rwanda: case reports and a review of the literature-Case Series. Rwanda Medical Journal, Vol 77, no 2, pp 37-41, 2020

Case report 1: Ultrasound to confirm central venous catheter placement

A 73-year-old morbidly obese female with hypertension and diabetes was admitted to our intensive care unit with altered level of consciousness and shock evidenced by her cold extremities, increased capillary refill time, and hypotension (blood pressure of 78/41 mmHg). Initial resuscitation was performed in the emergency department which included endotracheal intubation, a bolus of one liter of normal saline, and glucose-insulin therapy. A Foley catheter was inserted but this failed to drain urine, which raised suspicion for anuria. Initial blood samples for laboratory examinations were taken and she was immediately sent to our intensive care unit.

On arrival at the ICU, noradrenaline was started to achieve a MAP of 65 mmHg and she was scheduled for urgent hemodialysis. A dialysis catheter was inserted in the right internal jugular vein and immediately following this a left internal jugular catheter was inserted. A post-procedure chest X-ray (Figure 1) was obtained to confirm the correct placement of both central lines and endotracheal tube placement. However, she developed a tension pneumothorax which was confirmed with a bedside ultrasound and a right chest tube was immediately inserted.



Figure 1: Chest X-ray demonstrating correct placement of central venous catheter

A search for the cause of the pneumothorax was subsequently initiated. Among the differentials were the complication of central lines passed since pneumothorax could complicate the procedure and the process of airway management vis-à-vis endotracheal intubation. The supine chest-x-ray obtained after the procedure was not conclusive for the left internal jugular vein due to difficult visualization as a result of morbid obesity. The dialysis catheter was seen entering the right ventricle. We then decided to recheck the left central line with agitated saline using bedside USS with a four-chamber cardiac view, and through this, we confirmed correct placement with ultrasound bubble contrast in the right atrium (Figure 2).



Figure 2: A four-chamber cardiac USS showing a positive bubble test in the RA following infusion of agitated saline

Case report 2: Ultrasound to rule out pneumothorax in an intubated and ventilated patient

A 75-year-old female was admitted straight to the ICU for severe asthma that was refractory to continuous nebulization with salbutamol, ipratropium bromide, and magnesium sulphate. She was intubated and commenced on mechanical ventilation due to severe hypercarbia with a PaCO₂ of 85 mmHg. The following day, she was observed to be hypoxic with a sharp drop in peripheral oxygen saturation (SpO₂) from 100% to 88% with a blood pressure of 210/103 mmHg.

Upon physical examination there was remarkably decreased breath sounds in the right lung zone. A lung ultrasound scan was performed immediately which revealed absence of “lung sliding” in the apex of the same lung (Figure 3). A diagnosis of a right-sided pneumothorax was thus suspected. Urgent chest x-ray was done and while this could not confirm a pneumothorax, it revealed pulmonary infiltrates in the right lung. A chest computer tomography scan could not be obtained because the patient was not stable enough to be moved to the radiological suite. A needle decompression was performed and she improved remarkably. She was successfully taken off mechanical ventilation after about two weeks.

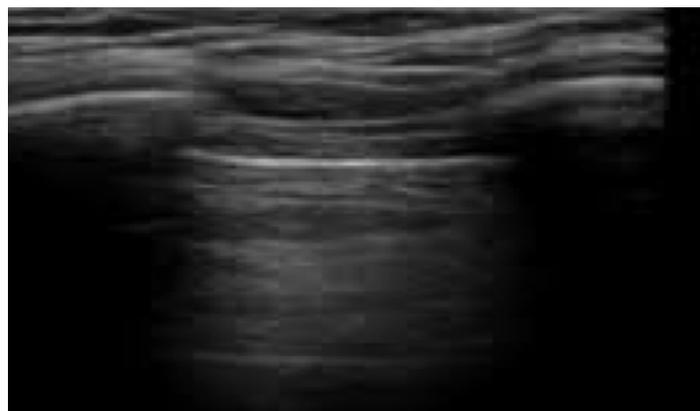


Figure 3: Lung USS showing absent lung sliding

Case report 3: Ultrasound for confirmation of free fluid in the abdomen

A 22-year-old male patient without known comorbidities was admitted to the ICU for respiratory support following severe blunt chest trauma sustained in a road traffic accident. He was subsequently intubated for severe respiratory distress and remained hemodynamically stable. However, on his second day of admission his hemoglobin concentration dropped from 14 g/dl to 7 g/dl and he was subsequently transfused three units of packed red blood cells. This necessitated an urgent abdominal ultrasound which revealed moderate free fluid found in the pelvis and Morison's Pouch. An abdominal CT scan was completed and this confirmed hemoperitoneum.

The patient was evaluated by the surgical team and he was taken to the operating room for an explorative laparotomy where the fluid in the abdomen was evacuated, although the quantity was not available. The patient was transferred back to the ICU and was successfully managed and discharged back to the ward after approximately two weeks.

Case Report 4: Ultrasound confirmation of hemoperitoneum

A 71-year-old male patient presented with severe chest pain, difficulty breathing, and abdominal pain following a fall the previous day. The patient had no associated co-morbidities or other chronic illnesses. On admission to the ICU, he was confused and was hypotensive with a blood pressure of 78/45 mmHg, which was managed with fluid boluses.

Upon general examination, the lungs were clear, regular heart sounds were noted on auscultation, and the abdomen was soft and moved with respiration with no area of tenderness. An abdominal ultrasound was performed which revealed free fluid in Morison's pouch and in the pelvis but the patient had become haemodynamically stable, and we decided to observe closely. On the second day of admission, he was found to be in critical status with signs of shock, a blood pressure of 88/40 mmHg, a heart rate of 132 beats per minute, and a respiratory rate of 44 cycles per minute. On physical exam, he had become pale with cold extremities, had a capillary refill time of 3-4 seconds, and had a weak pulse. A repeat abdominal USS at the bedside showed increased free fluid in all quadrants.

He was immediately transfused with three packed red blood cells (PRBC) and three fresh frozen plasma (FFP) and was brought to the theatre for an emergency laparotomy. Intra-operatively, about two liters of fluid blood was evacuated with an additional one litre of clotted blood. Other abdominal findings included severe liver cirrhosis, but no solid or hollow viscous injury nor bleeding vessel was identified. A diagnosis of spontaneous hemoperitoneum secondary to decompensated chronic liver disease was then made. Postoperatively, the patient underwent massive transfusion but unfortunately subsequently developed multi-organ failure and died in the ICU.

DISCUSSION

Traditionally, chest X-ray (CXR) is used for the confirmation of correct placement of a central venous catheter. The incidence of mal-positioning of a central venous catheter (CVC) has been estimated to range from 3.6% to 14% [8]. CXR has been adopted as a necessity after the passage of a central venous catheter, especially for internal jugular vein or subclavian vein cannulation. This is required both for confirmation of correct placement and to rule out complications such as pneumothorax [9].

Correct catheter position has been defined by positioning of the catheter tip at the distal third of the superior vena cava (SVC). This position is said to be optimal to reduce complications such as catheter migration, malfunction, and vascular perforation [10]. This position corresponds to the location of the carina which is easily identifiable on a CXR. A retrospective study conducted in 2006 suggests that all right-sided internal jugular vein catheterization should be placed with the catheter tip above the carina while all left-sided catheter tips should be located below the carina [11]. However, the upper limit of the pericardial reflection of the SVC cannot be visualized on CXR hence the need for cardiac ultrasound in doubtful situations.

In our patient, the tip of the left internal jugular catheter could not be visualized neither in the expected position in the SVC nor in the right atrium (RA) (Figure 1). This led to doubts about its correct position despite its functionality in terms of aspiration of dark venous blood and free flow of injectate through it. To ascertain its position, a four-chamber cardiac view was performed using agitated saline. The bubble was seen entering the right atrium when agitated saline was injected through the catheter (Figure 2).

Previously, ultrasound-guided CVC tip placement at the SVC-RA junction has been found to have an accuracy of 95-100% [12], but its use in the confirmation of correct placement is another dimension to its usefulness when doubts arise. Chest X-ray has been found to be less accurate and less reliable in the confirmation of correct placement of CVC catheters (13). The use of bedside USS confers about 100% accurate confirmation of catheter placement [13], and its use in this regard adds to its other numerous applications in the critical care setting.

A pneumothorax can rapidly progress to a tension pneumothorax which is a life-threatening emergency with subsequent development of cardiac arrest if not recognized and treated early. Conventionally, a pneumothorax is suspected based on clinical presentation and examination and is confirmed with a chest x-ray. However, an antero-posterior view in the supine position is poorly sensitive and often leads to catastrophic misdiagnosis [14]. Our patient was in a supine position in addition to being morbidly obese, further reducing the reliability of the CXR we obtained. A computer tomography (CT) scan of the chest has been described as the gold standard diagnostic tool (15) for pneumothorax, but it is bedeviled with numerous limitations especially in the ICU such as unavoidable delays, transport of unstable patients, high cost, and exposure to radiation.

Lung ultrasound scan, on the other hand, has emerged as a quick

and reliable tool for the diagnosis of pneumothorax in the ICU. It has been found to be superior to a CXR with a sensitivity and specificity greater than 90% [16]. A pneumothorax is diagnosed on a lung USS by the absence of “lung sliding”, which refers to the back and forth movement of parietal and visceral pleura sliding on each other with normal respiration and the presence of “lung point”, representing the point where the visceral pleura (lung) begins to separate from the parietal pleural (chest wall) at the margin [17]. In addition, the size of a pneumothorax and its consequent implications can also be assessed with the use of “lung point” as a more lateral location suggests a bigger pneumothorax [18]. Our patient had signs of a pneumothorax such as desaturation while being mechanically ventilated and subsequently experienced cardiovascular collapse which prompted confirmation with lung USS. An urgent needle decompression at the second intercostal space and placement of intercostal chest drainage resulted in a positive outcome. Although a chest CT scan could have equally helped in the diagnosis and management of this patient, we did not have the luxury of time to transport this patient hence the importance of the bedside lung USS.

Focused Assessment with Sonography in Trauma (FAST) examinations are an essential component of trauma care to detect the presence of free fluid in the peritoneal cavity with a sensitivity and specificity of 64-98% and 86-100%, respectively [19]. A positive FAST refers to the presence of free fluid either in the Morison’s pouch, in the splenorenal recess, or the pelvic region. A FAST examination is quick to perform, requiring < 20 seconds in positive cases, and about 160 seconds in negative cases in experienced hands [20]. The use of FAST has replaced diagnostic peritoneal lavage (DPL) as a primary method of detecting intraperitoneal fluid.

Furthermore, FAST examination has been contemplated in the evaluation of spontaneous hemoperitoneum in non-trauma patients as it helps in the detection of free intraperitoneal fluid especially in hemodynamically compromised patients [21]. Our third and fourth patients rapidly deteriorated after admission to

the ICU with a sharp drop in hemoglobin concentration with clinical signs of shock. A FAST examination performed at the bedside revealed a large amount of blood in the Morison’s pouch which prompted surgical consultation and review with consequent intervention in the operating theatre.

In the fourth patient, intraoperative findings were suggestive of spontaneous hemoperitoneum secondary to chronic liver disease. Spontaneous hemoperitoneum may result from various causes such as gynecologic, hepatic, splenic, vascular, or coagulopathic conditions [22]. It frequently presents with acute abdominal pain with or without hemodynamic collapse. Like in our patient, hemodynamic collapse may become obvious only after the initial evaluation which implies that spontaneous hemoperitoneum should be detected rapidly during the evaluation. There was no obvious external bleeding or other causes of cardiovascular collapse in this patient. A rapid and reliable bedside USS of the abdomen came to our rescue in identifying the cause of the hemodynamic instability we observed in our patient.

The regular use of bedside USS in the ICU in a resource-limited environment like ours is hampered by relative unavailability of an USS machine in most ICU settings. The presence of skilled operators and reliability of results as compared with CT scan add to the challenges of bedside USS use in our setting. Thus, there is a need to encourage hospital administrators and government in general to invest in the procurement of dedicated USS machine for ICU use. Regular training and retraining of physicians working in the ICU on the use of bedside USS cannot be overemphasized.

CONCLUSION

Ultrasound is quick, reliable, and has the potential to influence patient outcome positively. In addition, it is cost-effective and safe to use for all categories of ICU patients. Deployment of this simple but effective tool is a step in the right direction in the quest for improved patient care in the ICU

REFERENCES

1. CL. Moore, JA. Copel. Point-of-care ultrasonography. *N Engl J Med*;364:749–57. doi: 10.1056/NEJMra 0909487. 2011
2. J. Chacko, G. Brar. Bedside ultrasonography: applications in critical care. *Indian J Crit Care Med*; 18 (5): 301-309. 2014
3. L. Zieleskiewicz, L. Muller, K. Lakhal, Z. Meresse, C. Arbelot, PM. Bertrand, B. Bouhemad, B. Cholley, et al. Point-of-care ultrasound in intensive care units: assessment of 1073 procedures in a multicentric, prospective, observational study. *Intensive Care Med*. 41(9):1638–1647, 2015
4. T. Maecken, T. Grau. Ultrasound imaging in vascular access. *Crit Care Med*. 35 (5 suppl): S 178-185, 2007
5. NT. Mowery, OL. Gunter, BR. Collier, JJ. Diaz Jr, E. Haut, A. Hildreth, et al. Practice management guidelines for management of hemothorax and occult pneumothorax. *J Trauma*. Feb. 70(2):510-8. 2011
6. LJ. Wherrett, BR. Boulanger, BA. McLellan, FD. Brenneman, SB. Rizoli, J Culhane, et al. Hypotension after blunt abdominal

- trauma: the role of emergent abdominal sonography in surgical triage. *J Trauma*. 41:815–20. doi: 10.1097/00005373-199611000-00008. 1996
7. P. Blanco, FM. Aguiar, A. Vallejo. Point-of-care ultrasonography in critical care medicine: a one-way directional road. *Journal of ultrasound*. 19 (2): 157-158, 2016
8. A. Pikwer, L. Baath, B. Davidson, I. Perstoft, J. Akesson. The incidence and risk of central venous catheter malpositioning: A prospective cohort study in 1619 patients. *Anaesth Intensive Care*. 36:30–7, 2008
9. GB. Palepu, J. Deven, M. Subrahmanyam, S. Mohan. Impact of ultrasonography on central venous catheter insertion in intensive care. *The Indian Journal of Radiology & Imaging*. 19(3):191-198. doi:10.4103/0971-3026.54877. 2009
10. TM. Vesely. Central venous catheter tip position: A continuing controversy. *J VasIntervRadiol*. 14:527–34, 2003
11. PA. Stonelake, AR. Bodenham. The carina as a radiological landmark for central venous catheter tip position. *Br J Anaesth*. 96:335–40. 2006

12. W. Schummer, C. Schummer, C. Schelenz, P. Schmidt, R. Fröber, E. Hüttemann. Modified ECG-guidance for optimal central venous catheter tip positioning. A transesophageal echocardiography controlled study. *Anaesthesist*. 54:983–90, 2005
13. DB. Andropoulos, SA. Stayer, ST. Bent, CJ. Campos, LI. Bezold, M. Alvarez, et al. A controlled study of transesophageal echocardiography to guide central venous catheter placement in congenital heart surgery patients. *AnesthAnalg*. 89:65–70. 1999
14. SL. Hill, T. Edmisten, G. Holtzman, A. Wright. The occult pneumothorax: An increasing diagnostic entity in trauma. *Am Surg*. 65:254–8. 1999
15. H R Omar, D Mangar, S Khetarpal, D H Shapiro, J Kolla, R Rashad et al. Anteroposterior chest radiograph vs. chest CT scan in early detection of pneumothorax in trauma patients. *Int Arch Med*. 2011; 4: 30.
16. M. Slama, J. Maizel. Echocardiographic measurement of ventricular function. *CurrOpinCrit Care*. 12:241–8. 2006
17. D. Lichtenstein, G. Mezière, P. Biderman, A. Gepner. The “lung point”: An ultrasound sign specific to pneumothorax. *Intensive Care Med*. 26:1434–40. 2000
18. G. Soldati, A. Testa, S. Sher, G. Pignataro, M. La Sala, NG. Silveri. Occult traumatic pneumothorax: Diagnostic accuracy of lung ultrasonography in the emergency department. *Chest*. 133:204–11, 2008
19. M. Körner, MM. Krötz, C. Degenhart, KJ. Pfeifer, MF. Reiser, U. Linsenmaier. Current role of emergency US in patients with major trauma. *Radiographics*. 28:225–42. doi: 10.1148/rg.281075047. 2008
20. LJ. Wherrett, BR. Boulanger, BA. McLellan, FD, Brenneman, SB. Rizoli, J. Culhane, et al. Hypotension after blunt abdominal trauma: the role of emergent abdominal sonography in surgical triage. *J Trauma*. 41:815–20. doi: 10.1097/00005373-199611000-00008. 1996
21. N M Parmar, M D Patel, S S Negi, C M Savani, N L Desai. Spontaneous haemoperitonium. *Gujarat Medical Journal*. 2015, 70;2:19-26
22. BC. Lucey, JC. Varghese, SW. Anderson, JA. Soto. Spontaneous hemoperitoneum: a bloody mess. *EmergRadiol*. 14:65–75. doi: 10.1007/s10140-007-0594-0. 2007

Rwanda Medical Journal

DISCLAIMER

Opinions expressed in the “Rwanda Public Health Bulletin” are those of the authors and contributors; and do not necessarily reflect those of the Editorial Board or the Publisher. Authors hold sole responsibility of views expressed in their texts. The mention of specific companies or certain manufacturers’ products does not imply that these are endorsed or recommended in preference to other ones of a similar nature.

© June 2020

All rights reserved; no part of this publication may be reproduced, stored in retrieve system, or transmitted in any form or by any means without prior permission of the publisher.