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Content Pages

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EDITORIAL: END IN SIGHT

Tubonye C. Harry, FRCOG, FRCP, FWACS
Editor-in-Chief

Niger Delta Medical Journal 2021;**5**(2):5-6

The continuing roll out of the COVID-19 vaccine globally is a signal for increasing herd immunity, which will be the harbinger for the relaxation of the current draconian lockdown in most countries. The major setback to this roll out is vaccine hesitancy. Vaccine hesitancy of 31.1% was recently reported[1] in Emilia-Romagna region of Italy, with past refusal of vaccination being a key discriminating variable. Other significant predictors of hesitancy were ages between 35 and 54 years, female gender, low educational level, low income, and absence of comorbidities. Among adults, the highest COVID-19 vaccine acceptance rates[2] were in Ecuador (97%), Malaysia (94.3%), Indonesia (93.3%) and China (91.3%). However, the lowest rates[2] were found in Kuwait (23.6%), Democratic Republic of Congo (27.7%) & Jordan (28.4%).

In this issue, Odeyemi and colleagues[3] revisits the old chestnut of pulmonary tuberculosis in a cohort of HIV patients in Oshogbo, Western Nigeria. Bello and colleagues[4] report on the causes and predictors of under 5 mortality in Nasarawa State, Northern Nigeria.

The high mortality associated with peptic ulcer disease is explored by Tabowei and colleagues[5], a noteworthy impact of tertiary healthcare centre to its catchment population. The challenge is translating the lessons learnt to improving the outcomes of peptic ulcer disease.

The historical "six-hour rule" as a golden hour for timing to debridement has been refuted in modern literature as a standard of care for timely debridement of trauma patients needing orthopaedic intervention[6]. In this issue Ibrahim and colleague[7] has also shown from their experience in Jos, that it does not matter.

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Original Article:
PULMONARY TUBERCULOSIS IN A COHORT OF HIV PATIENTS IN A TERTIARY HOSPITAL IN OSOGBO, SOUTHWESTERN NIGERIA

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Abstract

Background: *Despite the gains made in the fight against HIV, particularly with the introduction of combination antiretroviral therapy (cART), tuberculosis (TB) remains common among people living with HIV (PLWH). In addition to being common in this group of people, pulmonary tuberculosis (PTB) is reported to be the most common cause of death (being responsible for 37% of deaths) among PLWH.*

Objectives: *To determine the prevalence of PTB and its associated factors in a cohort of PLWH on cART in a Nigerian tertiary hospital.*

Methods: *This was a prospective study carried out at the HIV clinic of Ladoke Akintola University of Technology (LAUTECH) Teaching Hospital Osogbo, Nigeria. The study involved 130 participants aged 18 to 65 years with a confirmed HIV-positive serology who were randomly selected. The participants were followed up for a period of one year while looking out for symptoms and signs suggestive of PTB.*

Results: *One hundred and twenty-two participants had their data analysed with a mean age of 41.9±10.02 years and a male to female ratio was 0.4:1. Six (4.9%) of the participants developed PTB during the study period and this was significantly associated with CD4+ T-cell count and cigarette smoking history.*

Conclusion: *PTB is common among PLWH in Osogbo, and it occurs more frequently in those with low CD4+ T-cell count and those with a history of cigarette smoking.*

KEYWORDS: Pulmonary Tuberculosis, HIV, Southwest Nigeria.

Introduction

The introduction of combination antiretroviral therapy (cART) in the 1990s has drastically reduced the morbidity and mortality associated with Human Immunodeficiency Virus (HIV) infection.[1] Despite the achievements made in the fight against the virus, the epidemic remains a major challenge worldwide, particularly in sub-Saharan Africa. As of the end of 2019, about 38 million people worldwide were said to be living with HIV, 25.6 million of these were in sub-Saharan Africa. In the same year, 1.7 million people became newly infected with the virus worldwide and there were 690,000 acquired immune deficiency syndrome (AIDS) related deaths.[2]Nigeria has the second-highest number of people living with HIV/AIDS in the world after South Africa. As of the end of 2019, about 1.8 million people were said to be living with the virus in the country.[3]Approximately 44,830 people died in Nigeria of AIDS in 2019.[3]

The respiratory system is the most commonly affected organ system by HIV infection and respiratory pathology is most commonly associated with morbidity and mortality among people living with HIV (PLWH).[4] Although the spectrum of respiratory diseases includes infectious and non-infectious types, infectious diseases are the most common.[4,5] Approximately 70% of PLWH develop a pulmonary complication during the course of the disease, mainly of infectious aetiology.[6] One of the most common infectious pulmonary complications of HIV infection is tuberculosis (TB). TB is one of the oldest diseases of humans and despite the successes achieved so far in the control, it continues to be of major public health concern. It is one of the top 10 causes of death and the leading cause from a single infectious agent all over the world.[7]It is estimated that about 10 million people developed TB disease worldwide in the year 2018, out of which 429,000 were in Nigeria, representing 4% of the global total.[7]About 21,000 people had multi-drug resistant TB in Nigeria in the same year.[7]About 1.451 million people were said to have died of TB in the year 2018 with about 157,000 of these deaths occurring in Nigeria.[7]About 24% of the total number of TB cases in Nigeria are found in the Southwest region of the country.[8]

PLWH are 16 to 27 times more likely to develop tuberculosis than those without the virus. In addition to being common in this group of people, pulmonary tuberculosis (PTB) is reported to be the most common cause of death (being responsible for 37% of deaths) among PLWH.[9] Although TB may occur at relatively high CD4+ T-cell counts, it tends to become increasingly common with lower CD4+ T-cell counts. Globally, TB is said to be responsible for the death of an estimated 251,000 PLWH in the year 2018 and about 32,000 PLWH in Nigeria in the same year.[7]

Despite having the second-highest number of people in the world with HIV/AIDS, and despite the high incidence and mortality associated with PTB among PLWH, only a few studies have evaluated the prevalence of PTB and its associated risk factors among PLWH in our environment (South-western Nigeria). This study was therefore conceived with the aim of determining the prevalence of PTB and its associated factors in a cohort of PLWH on cART in a Nigerian tertiary hospital.

Methodology

Study area

The study was carried out at the clinic dedicated to PLWH in Ladoke Akintola University of Technology (LAUTECH) Teaching Hospital (LTH) Osogbo, Osun state.

Study design
The study was a prospective study during which the participants were followed up for a period of one year from July 2015 to June 2016 while looking out for symptoms and signs suggestive of PTB.

Study population
Adult HIV positive people attending the clinic dedicated to PLWH in LTH formed the study population. The clinic has an estimated population of 2000 adult patients and it runs twice in a week with an attendance of about 30 to 40 patients.The study participants were aged 18 to 65 years with a confirmed HIV-positive serology. Excluded were those with Diabetes Mellitus, those who did not give consent and those with a pre-existing respiratory illness such as chronic obstructive pulmonary disease, bronchial asthma, lung fibrosis and bronchiectasis.

Sample size determination
Since the total population of the patients at the clinic is less than 10,000, the sample size for the study was obtained using the formula:[10]

nf (minimum sample size) = n/ 1+(n/ N)

$$n = \frac{Z^2PQ}{D^2}$$

The minimum sample size was determined to be 82, at 95% confidence level using a prevalence of 5.9%,[11] obtained from a previous study. However, 130 patients were recruited for the study.

Sampling method
Participants were selected using the simple random (balloting) technique. A list of the clinic attendees was obtained on each clinic day and each of the attendants were assigned a serial number. Each of these numbers was then written on a separate piece of paper (each wrapped to conceal the number) and put in a box from where 18 participants were subsequently randomly selected on each clinic day. Participants were only recruited into the study if they satisfied the inclusion criteria. This was continued on every clinic day till the required number of participants was recruited. Recruitment was done over four weeks.

Study protocol
The participants were seen in the clinic monthly during the follow-up period and anytime they developed symptoms suggestive of PTB. Phone numbers were exchanged between the researcher and the participants (and their next of kin) to facilitate the follow-up. The study objectives and procedures were explained to participants and informed consent (verbal and written) was obtained. Ethical approval was obtained from the Ethics and Research Committee of LTH, Osogbo.

Socio-demographic and clinical data of the study participants were collected using a structured interviewer-administered questionnaire. All the study participants had a general physical examination and chest examination done at the commencement of the study, during the monthly follow up clinic visits and anytime during the study when any of the participants developed symptoms suggestive of PTB. The weight and height were measured using a GIMA® ASTRA weighing scale with an inbuilt height meter. A baseline Chest X-ray, Full blood count (FBC) and Erythrocyte sedimentation rate (ESR) was done for all the participants at the start of the study. A random blood sugar was also done to exclude diabetes mellitus. The CD4+ T-cell count of the participants was done at the commencement of the study to assess their immune status. In addition, the participants also had Chest X-ray, FBC and ESR done anytime within the study period when they developed any symptom suggestive of PTB.

Definition of terms

Pulmonary tuberculosis was defined by the occurrence of cough with sputum production of more than 2 weeks with or without other respiratory symptoms (shortness of breath, chest pain, haemoptysis) and/or constitutional symptoms (weight loss, night sweats, loss of appetite, fever, and fatigue).[12]

In addition to the above, the patient must have one or more sputum positive for Mycobacterium tuberculosis (either by sputum smear microscopy or Gene Xpert assay) or at least two negative sputum specimens but with radiographic abnormalities consistent with active pulmonary TB.[12]

Cigarette smoking history

Cigarette smoking history was positive if participants were either former cigarette smokers or current cigarette smokers. Former smokers were defined as those who had smoked at least 100 sticks of cigarettes in their lifetime, but currently do not smoke while current smokers were defined as those who had smoked at least 100 sticks of cigarettes in their lifetime and currently smoke cigarettes every day or some days.[13]

Data Management and Analysis

Data were entered into the computer and analysed using the Statistical Package for Social Sciences (SPSS) version 23.0 (SPSS Chicago Inc., IL, U.S.A). The relationship between categorical variables was determined using Chi-square and student T-test was used to compare means of independent groups. The level of significance was set at a p-value less than 0.05.

Results

Eight of the 130 participants recruited for the study dropped out of the study, giving a dropout rate of 6.2%. Hence, only 122 participants had their data analysed. All the participants were on cART and none of them was on isoniazid preventive therapy. The mean age of the participants was 41.9±10.02 years and they had a male to female ratio of 0.4:1. The mean duration since diagnosis of HIV infection and use of cART was 3.9±2.84 years and 2.8±2.33 years respectively. (Table 1) Six (4.9%) of the participants developed PTB during the study period. All of them had drug-susceptible TB. The development of PTB was significantly associated with CD4+ T-cell count and cigarette smoking history. (Table 2) A summary of the characteristics of the six study participants who developed PTB is given table 3.

Discussion

The study has shown that 4.9% of a cohort of adults with HIV infection developed PTB within one year of follow-up and this was significantly associated with low CD4+ T-cell count and a positive history of cigarette smoking. Our finding of 4.9% is less than the 9.6% and 11% observed by Dravid et al and Gunda et al respectively,[14,15] but higher than the 2.0% and 2.4% reported by Karo et al and Kufa et al respectively.[16,17] The reason for this difference may be due to the varying patient population used, stage of HIV infection and the different methodologies employed in the various studies. For instance, while Gunda et al did a retrospective study in Tanzania, Karo et al did a cohort study in Germany, a low incidence country for TB. In our study, we observed that a low CD4+ T-cell count was significantly associated with the development of PTB. This is not unexpected as this has been established by several earlier studies.[18-20] Although TB can occur at any CD4+ T-cell count, the incidence increases as the CD4+ T-cell count declines. This is due to the fact that CD4+ T lymphocytes play an integral role in the coordination of the body's cell-mediated

immune response against *Mycobacterium tuberculosis*.[21] Our study also revealed that the development of PTB was not significantly associated with the duration on cART. This finding is similar to earlier studies.[16,22] Although cART has been shown to reduce the incidence of TB among PLWH, the incidence fails to drop to the level of the HIV-uninfected population.[23,24] Hence, over time, there may be no further drop in the incidence TB. This is due to the fact that cART is unable to fully normalize immune cell phenotype and function.[23,24]

We also observed that a history of cigarette smoking was significantly associated with the development of PTB. This finding is similar to earlier studies.[25,26] Several studies have consistently demonstrated that smoking increases the risk of TB by approximately two-fold regardless of HIV infection.[27-29] This is because the lungs of current and former smokers are unable to respond adequately to infection by *Mycobacterium tuberculosis*. The poor response to the organism has been shown to be due to direct impairment of the macrophage function and cell-mediated immunity, integral to the host defence against the organism.[27]

Conclusion

This study has shown that PTB remains a common problem among PLWH in our environment and it occurs more frequently in those with low CD4+ T-cell count (<200 cells/mm^3) and those with a history of cigarette smoking. We recommend the incorporation of smoking cessation therapies into the care of PLWH and increased surveillance for PTB in this category of people, particularly those with a low CD4+ T-cell count.

Acknowledgement

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Nil

Conflict of Interest

There are no conflicts of interest.

Note: This study has not been published or submitted in part in the past in any journal.

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Table 1: Basic characteristics of respondents

Socio-demographic profile	Frequency (n) n=122	Percentage (%)
Age (in years) Mean±SD	41.9±10.02	
Sex		
Male	35	28.7
Female	87	71.3
BMI		
Underweight	8	6.6
Normal weight	84	68.9
Overweight	19	15.6
Obesity	11	9.0
Mean duration since diagnosis (in years)	3.9±2.84	
CD4 count (cells/mm^3)	25	20.5
<200	64	52.5
200 - 500	33	27.0
>500		
Duration on ART (in years) Mean±SD	2.8±2.33	
Currently on co-trimoxazole		
Yes	36	29.5
No	86	70.5
Cigarette smoking history		
Yes	9	7.4
No	113	92.6

Table 2: Relationship between the patients' characteristics and the development of PTB

Variables	Development of PTB		Test statistics	P-value
	Yes n(%) n=6	No n(%) n=116		
Age (in years) Mean±SD	43.3±12.57	41.8±9.93	t = 0.356	0.723
Sex				
Male	3 (8.6)	32 (91.4)	χ2=1.401	0.237
Female	3 (3.4)	84 (96.6)		
BMI (Kg/m^2)				
Underweight	0 (0.0)	8 (100.0)	χ2=8.333	0.842
Normal weight	4 (4.8)	80 (95.2)		
Overweight	1 (5.3)	18 (94.7)		
Obesity	1 (9.1)	10 (90.9)		
Mean duration since diagnosis of HIV (in years)	2.3±2.53	3.9±2.84	t = -1.410	0.161
CD4 count (cells/mm^3)	4 (16.0)	21 (84.0)	LRχ2=8.064	**0.018**

Variables	Development of PTB		Test statistics	P-value
	Yes n(%) n=6	No n(%) n=116		
<200	2 (3.1)	62 (96.9)		
200 - 500	0 (0.0)	33 (100.0)		
>500	1.8±2.38	2.8±2.33	t = -1.007	0.316
Mean Duration on ART				
Currently on *CTX	3 (8.3)	33 (91.7)	LRχ2=1.166	0.280
Yes	3 (3.5)	83 (96.5)		
No				
Cigarette smoking history	5 (55.6)	4 (44.4)	χ2=53.282	**0.000**
Yes	1 (0.9)	112 (99.1)		
No				

*CTX = Co-trimoxazole,

Table 3: Characteristics of the study participants who developed Pulmonary Tuberculosis*

Variables	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6
Age (years)	30	44	50	64	40	32
Sex	F	M	F	M	M	F
Occupation	Trading	Trading	Trading	Artisan	Artisan	Trading
BMI (kg/m^2)	18.59	27.56	34.41	21.36	20.80	21.33
Cough	Yes	Yes	Yes	Yes	Yes	Yes
Dyspnoea	No	No	No	No	No	No
Haemoptysis	No	No	No	No	No	No
Duration of HIV diagnosis	1 year	6 years	3 months	5 years	6 months	1 year
ART duration	3 months	6 years	2 months	3.5 years	6 months	9 months
CD4 (cells/mm^3)	124	455	73	245	176	156
Use of CTX**	No	Yes	Yes	No	No	Yes
Cigarette smoking	No	Yes	Yes	Yes	Yes	Yes
Anaemia***	Yes	Yes	Yes	Yes	Yes	No

*All six participants were on ART regimen containing a combination of zidovudine, lamivudine and evavirenz. **CTX = Co -trimoxazole. ***Anaemia is defined as haematocrit of <36% in females and <40% in males.[28]

ASSESSMENT OF THE CAUSES AND PREDICTORS OF UNDER-FIVE MORTALITY IN NASARAWA STATE.

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ABSTRACT

Background: *There are concerns about the rising burden of childhood mortality in Nigeria, especially among infants and under five children. This is because childhood mortality is an important indicator of measuring a country's socio-economic development and remains a major challenge confronting the nation.*

Objectives*: To determine the causes and predictors of under five deaths in Nasarawa State.*

Materials and Methods*: This was a cross-sectional study using secondary data from selected Health facilities in the State. A multi staged sampling technique was used in selecting the health facilities from the three senatorial zones. At least, One local government area (LGA) was selected from each of the zones. The Northern senatorial zone had the least number of LGAs (three) compared with the other two zones which had five LGAs each. The General Hospitals were then selected in the ratio of 2:1:2 based on the last National Population census delineation for Nasarawa West, North and South respectively. Admission registers and*

patient folders were used to gather data of all admissions and deliveries in the year 2018. Proforma was completed by the researcher/research assistants in the respective health facilities.

***Results**: Children below five years of age accounted for three quarter (74.2%) of childhood mortalities. There were slightly more male deaths with a M:F of 1.3:1. Under five mortality rate in Nasarawa state is 187 per thousand live births (60.4%). The commonest causes of childhood mortalities are severe malaria, sepsis, diarrhea disease and pneumonia. The predictors of death are duration of illness and number of children.*

***Conclusion**s: Under five mortality rates in Nasarawa State was 60.4% (187 per thousand live births). Preventable causes (such as severe malaria, diarrhea and pneumonia) remain the leading causes of deaths. Duration of illness and large family size predicts poor outcome.*

KEYWORDS: Assessment, causes, infant, mortality, predictors.

INTRODUCTION

There are persistent concerns about the rising burden of childhood mortality in Nigeria.[1] This is because childhood mortality is an important indicator of measuring a country's socio-economic development and remains a major challenge confronting the nation.[2] It is a reflection of a country's health care system and overall quality of life.[1] The trend of deaths among infants and children aged below five years in Nigeria is such that a higher proportion of such deaths occur in rural areas.[3] Significant progress are being attained by developed countries in curbing the tide of childhood mortality, but this is not so in Africa which accounted for almost fifty percent of these deaths.[4] Under five mortality in Nigeria remains unacceptably high with an higher proportion occurring among infants.[5] Contributing factors to this poor outcome includes low maternal age at first birth, high birth rate, cultural practices, low education and household income etc.[2,4,6-8]

Infancy is the period from birth to the first year of life, while infant mortality is death occurring in a child born alive up to the first twelve months.[9] Achieving appreciable reduction in childhood mortality is a panacea to attaining sustainable development goals.[7] In Nigeria, one in every five children dies before their fifth birthday.[10,11] The infant and under-five mortality rates in Nigeria as at 2013 were 69 and 128 per 1000 live birth respectively.[12] Another report (longitudinal report between 2003 – 2013) from Nigeria showed the prevalence of infant and under five mortality rates were 88 and 143 per thousand live birth respectively.[11] These high trend of childhood mortality in Nigeria were largely due to preventable diseases and are of infectious agents.[13]

Despite progress made around the world in curbing the trend of infant and under five mortality rates.[13] There are (if any) insignificant reduction in childhood deaths in the developing countries, Nigeria inclusive. In addition, there is dearth of data on infant and under-five mortality rates from Nigeria and this is worse in the Northern part of the country to which Nasarawa State belong. This study is therefore aimed at determining the under five mortality rate, the common causes of deaths and the predictors of deaths among under five on Nasarawa State. The study will answer the following research questions**:** What is the Infant Mortality Rate (IMR) in Nasarawa State?, what is the Under-five Mortality Rate (UMR) in Nasarawa State?, what are the common causes of childhood deaths in Nasarawa State?, what are the predictors of childhood mortality in Nasarawa State?

MATERIALS AND METHODS

STUDY DESIGN

This is a cross-sectional study conducted using secondary data from the selected health facilities.

STUDY SITE

The study was conducted at the Emergency Paediatrics Unit (EPU) / Children's ward as well as the delivery / Labour wards in the selected Health facilities in Nasarawa State.

Nasarawa State has boundaries with the Federal Capital Territory (FCT) Abuja in the West, Kaduna State in the North, Plateau and Taraba States in the East, Benue and Kogi States in the South. The state is made up of thirteen Local Government Areas (LGA) and Development Areas (DA). Lafia, the capital has a landmass of 27,117km^2 (10,470 sq m). Its total population according to the 2006 National Population Census was 1,869,377.[14] There are three Senatorial Zones namely; Nasarawa West, Nasarawa North and Nasarawa South respectively. There are over seven hundred Primary Health Care Centres, Seventeen General Hospitals and a State Specialist Hospital (DalhatuAraf Specialist Hospital) all across the State.

Nasarawa West, North and South senatorial zones are made up of five, three and five Local Government Areas respectively. The population of Nasarawa West is 716,802, Nasarawa North is 335, 453 and Nasarawa South is 811,020 respectively. In view of this disparity in population and LGA, both the Western and Southern senatorial zones which are at least twice the size of the Northern senatorial in proportion to size had two General Hospital selected from each. Thus, the General Hospitals were selected in the ratio of 2:1:2 i.e., two each from both Nasarawa West and South and one from Nasarawa North.

STUDY POPULATION

The study population consisted of data from children aged 29 days to less than five years in Nasarawa State Nigeria who were admitted in the selected health facilities over a year.

SAMPLING TECHNIQUE

A multi staged sampling technique was used. The State was divided into the three Senatorial Zones. The list of Local Government Areas (LGA) gotten from the State Ministry of Local Government and Chieftaincy Affairs was arranged alphabetically, and two were chosen (except in Nasarawa North where only one was selected) using simple random sampling based on proportion of numbers of LGA as well as population. The General Hospital in the chosen LGAs were then arranged in alphabetical order and one was chosen using simple random sampling. In the chosen secondary health facility, the record of all deliveries in the year 2018, all childhood admissions in the year 2018, all childhood mortalities in the same study period was obtained. These data was arranged based on age, gender, diagnosis, cause of deaths, duration on admission and social status of parents.

All deliveries in the year 2018 irrespective of the mode of deliveries, all children aged 29 days to less than five years admitted and mortality recorded in children less than five years in the same period were included.

PROCEDURE FOR RECRUITMENT

Admission registers, medical records and patient folders were used to gather data of all children admitted or delivered within the study period. The delivery register and other medical records at the delivery suite / labour ward were used to get all data of all live birth within the study period. A proforma was

completed by the researcher or research assistants in the respective health facilities.

ETHICAL CONSIDERATION

Ethical approval was sought and gotten from the State Research Ethics Committee. Approval was also sought from the State Ministry of Health and leadership of the Hospital Management Board. Confidentiality was maintained strictly.

DATA ANALYSIS

The data was coded and enter into an excel spreadsheet before transferring to a Statistical Package for Social Sciences (SPSS) version 20. Categorical variables were presented using frequency and percentages. The means and standard deviation of continuous variables were presented. Associations between categorical variables were determined using chi square test. Results were arranged in tables. The p value was considered significant at $p < 0.05$.

RESULTS

Overview of admissions and mortalities across the state

A total of 9,292 patients were admitted and managed in the selected health facilities within the state in the year 2018. Medical center MararabaGurku (MCMG) and General Hospital (GH) Keffi both contributed more than fifty percent to the overall admission while MCMG had the highest (26.2%) childhood mortality and GH N/Eggon the least (15.1%). Two hundred and fifty two children aged less than 18 years died across the state in the year 2018 which accounted for 43.4% of the overall mortalities **(Table 1).**

Table 1: Overview of admissions and mortalities across the state

	Admissions			Deaths		
Variables	Adults n (%)	Children n (%)	Total	Adults n (%)	Children n (%)	Total
MCMG	1279 (22.2)	1111 (31.4)	2390	80 (24.3)	66 (26.2)	146
GHKeffi	1684 (29.2)	1199 (33.9)	2883	72 (22.0)	55 (21.8)	127
GH N/Eggon	1323 (23.0)	558 (15.8)	1881	66 (20.1)	38 (15.1)	104
GH Doma	804 (14.0)	318 (9.0)	1122	56 (17.1)	48 (19.0)	104
GH Obi	669 (11.6)	347 (9.8)	1016	54 (16.5)	45 (17.9)	99
Total	5759 (100.0)	3533 (100.0)	9292	328 (100.0)	252 (100.0)	580

MCMG=Medical Centre MararabaGurku, GH=General Hospital, N/Eggon=Nasarawa Eggon

Distribution of childhood mortalities across gender, age group and health facilities

There are more deaths among males compared to females with a M:F of 1.3:1. Under-five children accounted for about three quarter (74.2%) of childhood mortalities while infants were about a third (35.3%). There was a total of 3097 deliveries in 2018 of which approximately half (49.5%) these deliveries were at the Medical Centre MararabaGurku. The Nasarawa State infant mortality rate in year 2018 was 28.7% (89 per thousand of 3097 live births). Under five mortality on the other hand was 60.4% (187 per thousand of 3097 live births) **Table 2.**

Table 2: Distribution of childhood mortalities by gender, age group and health facilities

Deaths among children	N	Percentages
Males	142	56.3
Females	110	43.7
Total	252	100
Deaths		
< 1year	89	35.3
< 5 year	187	74.2
Deliveries		
MCMG	1534	49.5
GH Keffi	651	21.1
GH N/Eggon	335	10.8
GH Doma	300	9.7
GH Obi	277	8.9
Total	3097	100

Mean age 49.7 ± 46.9 months (4.14 ± 3.90 years).

Causes of deaths among children across the state

Severe malaria is the commonest cause of childhood mortality in the state. Others include sepsis, diarrheal diseases, pneumonia and enteric fever **(Table 3).**

Table 3: Causes of deaths among children across the state

Variables	N	Percentage
Severe malaria	98	38.9
Sepsis	49	19.5
Diarrhea disease	36	14.2
Pneumonia	21	8.3
Enteric fever	15	5.9
Meningitis	11	4.4
Sickle cell disease	3	1.2
Multiple injuries from road traffic accident	4	1.6
Asphyxia	2	0.8
Urinary tract infection	2	0.8
Others	11	4.4
Total	252	100

Determinants of socio-demographic factors on childhood mortality in Nasarawa State

Duration of illness prior to presentation as well as the number of children in a family was significant statistically to the eventual outcome (childhood mortalities). Number of children in a household is a significantly strong determinant of childhood deaths. It is however worthy to note that there were lots of missing data due to incomplete documentation in this study **(Table 4).**

Table 4: Determinants of socio-demographic factors on childhood deaths in Nasarawa State

Variables	Under-five death	Total	χ^2	*p value*	*B*	*p value*
Socio-economic status						
Upper	5	109	*12.927*	*0.114*	*2.786*	*0.356*
Middle	8					
Lower	96					
Marital status						
Married	21	32	*0.970*	*0.914*	*3.957*	*0.534*
Single	11					
Family type						
Monogamous	11	26	*3.694*	*0.449*	*4.738*	*0.347*
Polygamous	15					
Number of children						
1 – 2	4	109	***26.244***	***0.001****	***8.647***	***0.032****
3 – 4	6					
≥ 5	99					
Duration of illness						
< 24 hours	10	109	***23.559***	***0.003****	***0.460***	*0.881*
24–72 hours	19					
> 72 hours	80					
Duration of admission	22	109	*8.906*	*0.350*	*2.788*	*0.256*
< 24 hours	12					
24 – 72 hours	75					
> 72 hours						
Residence						
Urban	27	64	*1.451*	*0.247*	*2.560*	*1.275*
Rural	37					

*=Statistically significant

DISCUSSION

Children aged less than eighteen years accounted for approximately a third of hospital admissions in the study and almost half of the mortalities. Of the childhood mortalities in this study, three out of four (three quarter) occurred among the under five children.

Infant mortality rate for Nasarawa State in this study is 28.7 per thousand live births. This is lower compared to the 69 per thousand live births found by Morakinyo et al[12] in 2015. It is also lower to the 70 per thousand live births and the 65 per thousand live births reported by the National Bureau of Statistics (NBS) and the United Nation Children Emergency Fund (UNICEF) respectively.[15,16] The difference may be attributed to the fact that the present study was limited to Nasarawa State in Nigeria unlike the later studies that are National data. Similarly, this study is more recent when compared with the above mentioned study.

The under-five mortality rate for Nasarawa State in the current study is 60.4 per thousand live births. This is lower to the 100.2 per thousand live births, 109 per thousand live births and 120 per thousand live births found by the United Nation Children Emergency Fund, Morakinyo and colleagues as well as the National Bureau of Statistics respectively.[12,15,16]
The major causes of admission and death from this study includes severe malaria, sepsis, pneumonia, acute diarrhea diseases, and typhoid. Similar finding was reported at a neighbouring state (Benue) by Abu et al[17] in 2015. This is also similar to the report by George and colleagues from Port-Harcourt.[18] These are preventable diseases stemming from infectious agents which have persisted due to lack of potable water, poor hygiene and sanitation among others.[13] The contributors to mortality were duration of illness before presentation as well as the number of children in a households. This is similar to the reports of other authors.[19,20] Those presenting to the health facility later than 72 hours, households with five or more children and single parenthood all worsens the patient's outcome. Delay in presentation to the health facility and high number of children in household can be attributed to poverty, ignorance, cultural reasons, non empowerment of women, poor access to health centre, and or attitude of health workers. These are similar to earlier findings were high birth rates, low education, low income as well as rural dwellers were associated with higher childhood mortalities.[2,7,8]

CONCLUSIONS

1. Infant mortality rate in Nasarawa State in the year 2018 is 28.7%.
2. Under five mortality rate in Nasarawa State in the year 2018 is 60.4%
3. The predominant causes of childhood death include severe malaria, sepsis, pneumonia and diarrhea diseases.
4. Predictors of mortality in this study are duration of illness before presentation, number of children in an household and marital status.

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CONFLICT OF INTEREST
There is no conflict of interest.

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PERFORATED PEPTIC ULCER DISEASE: THE BAYELSA STATE EXPERIENCE.

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ABSTRACT

Peptic ulcer disease (PUD) is a chronic condition affecting mankind. Perforation of a peptic ulcer is one of the most common complications of this disease. Perforated peptic ulcer is a common cause of the acute abdomen, requiring urgent surgical intervention. The aim of the paper is to determine the pattern of perforated peptic ulcer in our hospital. The specific objectives are to determine the risk factors, clinical presentation and the treatment outcomes in these patients. All patients who had surgery for acute abdomen with a finding of perforated duodenal or gastric ulcer from 2012 to 2017were included in this retrospective study. Variables analysed were the biodata, risk factors, clinical features, chest xray findings, duration of symptoms, type of surgery done and complications. There were fifty patients meeting the inclusion criteria. There were 28 males and 22 females. The 41 to 50 age group was the most affected. The use of Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) and alcohol consumption were seen in 30% and 76% of patients respectively. Ninety percent had an abdominal massage, an unorthodox practice in our environment, after the onset of symptoms but before presentation in hospital. Ninety six percent of our patients presented after 24 hours of the onset of symptoms. Severe abdominal pain was present in all our patients. All patients were treated surgically with simple closure reinforced with a Graham's patch and lavage with warm saline. Wound infection was the commonest complication post operatively. The mortality rate was 40%. Late presentarion and a high mortalty rate was seen in our series. Mass education on the dangers of procuring NSAIDs and other drugs procured over the counter without a doctor's prescription should be embarked on. The importance of early health seeking behaviour should also be stressed.

KEY WORDS; perforated peptic ulcer disease, duodenal and gastric perforation, Bayelsa State, Nigeria,

INTRODUCTION.

Peptic ulcer disease (PUD) is a common chronicgastrintestinal condition affecting human populations.[1] Before the establishment of the causal relationship between H.Pylori and peptic ulcerby Warren and Marshal,[2] the definitive treatment of this disease was surgical.[3] Currently, the cure for peptic ulcer disease is from the use of the H-pylori eradication regimen which was first patented in 1984 by Thomas Borody.[4] Nowadays elective surgery for PUD has virtually disappeared.[3] Surgery is now almost exclusively reserved for some of the complications of PUD which include perforation, bleeding and gastric outlet obstruction.[3,5] Perforated peptic ulcer is one of the commonest causes of the acute abdomen.[6] Despite the improvement in medical practices and technology, perforations of peptic ulcers still pose a challenge to the clinician especially in the third world.[7] as between 2-14 percent of patients who have peptic ulcer disease develop this complication.[6,8] As was noted by other colleagues[9,10,11], perforation may be the first clinical presentation of peptic ulcer disease in some patientsespecially in developing countries.

The perforation results in spillage of gastric/duodenal contents into the free peritoneal cavity with resultant chemical peritonitis and later, purulent infection."[12] Hirschowit et al13reported that two thirds of the patients with perforated gastric ulcer present with severe sudden pain that radiates to the back and with features of spreading peritonitis. Other experts also noted that patients with perforated peptic ulcer have a typical history of sudden onset of acute sharp pain usually located in the epigastric region with shoulder pain indicating free air under the diaphragm[14]. However, in the elderly or in patients who are immunosuppressed, the signs of perforation may be insidious or equivocal.

A study from Benin city, Nigeria, had reported some risk factors implicated in perforatedpeptic ulcer[15]. These include; alcoholconsumption, smoking, and the intake of non-steroidal anti-inflammatory drugs (NSAIDS) such as ibuprofen and diclofenac. In recent years, *Helicobacter pylori* infection and ingestion of NSAIDs have been identified as the two main causes of peptic ulcer perforation.[16] Others had also observed that the use of cracked cocaine leads to increased incidence of perforation in patients with peptic ulcer disease.[17]

The diagnosis of peptic ulcer perforation is usually made from the history, clinical examination and in 75% of cases, an erect chest X-ray radiography showing air under the diaphragm[18]. However, recently computerized tomographic scan is the gold standard for detecting and making a diagnosis of a perforated peptic ulcer.[19]

Delay before surgical treatment is instituted, amongst other factors,isa strong determinant for increased complicationrates, hospital cost and mortality[20].

The mainstay of treatment of perforated peptic ulcer is usually surgical after adequately resuscitating the patient.[21] Conservative treatment consisting of nasogastric aspiration, antibiotics, intravenous fluid and nowadays the use of proton pump inhibitors and eradication of *Helicobacter pylori* using the triple therapyregime can be useful in few selected cases.[22]

In developing countries, patients with perforated peptic ulcer disease often present late to health facilities for manangement.[10] This has led to the high morbidity and mortality observed in patients with perforated peptic ulcer disease in this environment. This study

aims to determine the risk factors, clinical presentation and the treatment outcomes of patients with perforated peptic ulcer disease in our facility.

PATIENTS AND METHODS.

This is a descriptive retrospectivestudy carried out over a 5 year period at the Niger Delta university Teaching Hospital (NDUTH) Okolobiri between June 2012 to July 2017. NDUTH Okolobiri is a 200-bed capacity tertiary Bayelsa State owned hospital. Allconsecutive patients who had surgery for acute abdomen with the confirmation of a duodenal or gastric perforation were included in the study. The patients case notes, anaesthetic notes and the nursing notes were obtained, entered into a proforma and analysed. Data were analyzed for age, sex, occupation, past history of pepetic ulcer disease, drug history, associated risk factors, clinical features, investigations done, treatment given, type of surgery, operative findings, complications seen, and the treatment outcome. Other variables analysed includelength of hospital stay, morbidity and mortality. Patents who had perforated appendicitis, typhoid perforation and traumatic perforation of the gut were excluded from the study. Data obtained were analysed using the SSPS 16 version package. Ethical committee approval was sought and obtained for the study.

RESULTS.

A total of 55 case notes of patients operated for perforated peptic ulcer disease were obtained from the hospital database.Five cases were excluded because of incomplete documentation. Of the 50 cases analysed, 28(56%) were males and 22(44%) were females; giving a male to female ratio of 1.3 to 1. Their ages ranged between 15 to 82 years, the mean age was 44 years ± 7. Majorityof the patients (72%) had no previous history of peptic ulcer disease.

Table 1. The age and sex distribution of patients.

	Number	Males	Female	Percentage.
0-10	0	0	0	0
11-20	3	2	1	6
21-30	6	3	3	12
31-40	11	6	5	22
41-50	18	10	8	36
51-60	7	4	3	14
61-70	3	2	1	6
>70	2	1	1	4
Total	50	28	22	100

Table 2. Occupation of patients.

Occupation of patients.	Number	Percentage
Farmers.	12	24
Civil servants	5	10
Unemployed	8	16
Fishermen	13	26
Business	6	12
Students	2	4
Others	4	8
Total.	50	100

Table 3. Interval between onset of symptoms and presentation at the hospital

Onset of Symptoms	Number	Percentage
0-24 hours	2	4
25-48 hours	7	14
49-60 hours	13	26
61-72 hours	20	40
>72 hours	8	16
Total	50	100

Table 4. Risk factors.

Risk factor	Number	Percentage
Alcohol	38	76
NSAIDs+ alcohol	42	84
NSAIDs Alone	15	30
Smoking, Alcohol, NSAIDs	30	60
Herbal concoction ingestion	7	14
Fasting	2	4
Massaging the abdomen	45	90
None	11	22

Table 5. Clinical features.

Clinical Feature	Number of patients	Percentage
Pain	50	100
Nausea	29	58
Vomiting	20	40
Fever	36	72
Constipation	5	10
Diarrhea	3	6
Abdomenal rigidity	50	100
Abdominal distension	38	76
Silent abdomen	43	86

The most reliable investigation done to make a diagnosis of perforated peptic ulcer diseae was erect plain abdominal X-Ray which showed air under the diaphragm in 45(90%) of patients, while the ultrasound scan revealed free peritoneal fliud and debris in 46(92%)of the patients.

All the patients had surgery with simple closure of the perforation with omental patch. Irrigation of the peritoneal cavity with warm normal saline and insertion of a tube drain was carried out in all patients. No patient hadany definitive vagotomy and drainage procedures.

Table 6. Complications seen in our patients.

Type of complication	Number	Percentage
Wound infection	26	52
Burst abdomen	16	32
Septicemia.	18	36
Intra-abdominal abscesses	10	20
Prolong Ileus	24	48
Pneumonia	16	32
Electrolyte imbalance	18	36
Death	20	40

DISCUSSION.

It is one of the commonest causes of surgical emergency and hospitalization. Despite the wide spread use of gastric antisecretory agents and eradication therapy, the incidence of peforated peptic ulcer has changed little.[23]

Perforated peptic ulcer used to be a disorder mainly of younger patients (predominantly males), but recently, studies have shown that the age of patients with perforated peptic ulcer disease is increasing and many more females are being affected.[24] Others had noted that perfortated peptic ulcer disease occur most commonly in the fourth decade of life and that it affects more males than females[25]. Males and females were almost equally affected in the ratio of 1.3:1 by the disease. This is in sharp contrast with the report of other authors who had documented that males predominated in the number of patients that had perforation of peptic ulcer disease.[23,24,25] Smoking, the use of alcohol and ulcerogenic drugs such as NSAID shave been implicated in causing peptic ulcer perforation as documented by others[26]. While risk factors such as alcohol consumption and the use of NSAIDs are common among males and females in our environment, not many females smoke amongst our catchment population. This may also account for the slight differences in incidence in the ratio between male and females who have perforation of peptic ulcer in our series.

Although, perforation of a peptic ulcer cuts across all strata of human populations, 50% of our patients were fishermen and farmers and are assumed to be of low socio-economic class as these activities are done in a small scale in this semi-urban environment. This observation is consistent with those reported by Dongo et aland other workers who observed this complication affected people of a kower socioeconomic status."[12]

Typically, patients with peptic ulcer disease present with a sudden onset of severe, sharp pain usually located in the epigastric area and sometimes with pain under the shoulder[14]. In our study, all(100%) of our patients had severe abdominal pains at presentation, similar to those reported in the literature[27,28].

The diagnosis of peptic ulcer diseasein many developing countries is first made following a perforation[11].In our study, 36(72%) of the patients had no previous history suggestive of peptic ulcer disease, similar to that reported in the literature[1,10,11,25].

In ourseries, 72% had no previous history of peptic ulcer disease. The reasons cannot be accurately inferred as this was a retrospective study.
Others had noted that in developing countries such as Nigeria, patients with perforated peptic ulcer present late to health facilities[10].In Port Harcourt which is 80km from our center,a studyshowed that 88.9% of the patients presented late to the hospital. In our series, 96% of the patients presented late to health facilities after pepetic ulcer perforation., comparable to those reported elsewhere[10,15,25,29].

Ignorance, religious beliefs, lack of easy assessibility to health facilities, use of alternative/ traditional medicine practitioners and charlartans, poverty and the high cost of treatment are contributory factors.
The presence of air under the Diaphragm in an erect position is diagnostic of perforated peptic ulcer18. In our study, air under the diaphragm was seen in 92% of the cases.
Delay before surgical treatment is a strong determinant for increased complication rates, hospital costs and mortality rates[20]. Shock on admission, associated pre-existing medical illness and prolonged perforation has been shown to be a useful tool in predicting outcome in patients with perforated peptic ulcer disease[22,31].

The high mortality rate (40%)was due to delay in instituting medical/surgical treatment as most of the patients presented late to the health facilities. To decrease the high morbidity and mortality associated with perforation of the peptic ulcer as was noted in our study, it is necessary to educate and sensitise patients on the etio-pathology and complications of peptic ulcer disease and the need to present to health facilities as soon as symptoms of abdominal pain begin.

Non-operative treatment in the form of intravenous fluids, nasogastric tube suction, intravenous antibiotics and the use of proton inhibitors is safe and effective in selected cases as perforations frequently get sealed off spontaneously by omentum and adjacent organs[32].

In our series, most of the patients presented late, some were in a state of shock and their vital signs were unstable. They were actively resuscitated with fluids, antibiotics, analgesics, and therefore none was offered the conservative or non operative mode of treatment. Intraoperatively, only four(8%) patients had their perforation sealed off by omentum and surrounding organs. Although, conservative management in few selected cases may be beneficial, in our environment, this may be fraught with difficulties due to inadequate intensive care facilities. Most of our patients do not meet the criteria for conservative management due to complications associated with late presentation for specialist care. It is our opinion therefore, that the open operative management of the patients be adopted in this environment as soon as the diagnosis of perforation of the peptic ulcer is made.

Closure of the perforation with omental patch(Graham's procedure) and the use of proton pump inhibitors and antibiotics for the eradication of *Helicobacter Pylori*is now the standard of care[5]. In our series, simple closure with omental patch and copious irrigation of the peritonel cavity with warm normal saline was carried out in all the cases. Tube drains

were inserted at both the left and right paracolic gutters. No patient had the traditional definitive vagotomy and drainage procedure done but all had proton-pump inhibitors and antibiotics to eradicate *Helicobacter pylori*.

Peritonitis with resultant shock, severe infection, electrolyte imbalance, late presentation and delay before intervention had been noted to be the commonest cause of death in most series[11,12,15,29], as was the case in our series.

CONCLUSION.

Perforation is a common complication of peptic ulcer disease and remains a major life-threatening surgical condition. The high mortality rate recorded was as a result of late presentation, effect of massaging and delay in instituting surgicalintervation in patients who presented with perforated peptic ulcer disease. There is need for sensitization, medical education and creating awareness among the populace on the importance of early health seeking behaviour. On a general note, health care should be made more accessible and affordable and the general hygienr and living standards of the populace should be improved.

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ORIGINAL PAPER

RELATIONSHIP BETWEEN TOTAL INJURY TIME TO OPERATIVE DEBRIDEMENT AND RATE OF INFECTION IN OPEN LONG BONE FRACTURES.

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Abstract

Background*:Open fractures are orthopaedic emergencies and are associated with increased rate of bone infection, which can cause significant morbidity. This study sought to determine the relationship of time delay to operative debridement of open long bones fracture and rate of wound infection.*

Materials & Method: *Fifty-six patients with open lower limb long bone fractures who consented and met the inclusion criteria for the study were prospectively recruited between 1st August 2015 and 31st July 2016 at the Jos University Teaching Hospital. Following debridement, a correlation was sought between infection rate and time to operative debridement.*

Results: *The mean time interval between injury and presentation at the Accident & Emergency (A&E) was 8.3±4.8 hours. Thirty (53.6%) patients had a time delay of 6-12 hours in the A&E before debridement. The total injury time to operative debridement in 39(69.7%) patients was between 13-24 hours, while 17(33.3%) patients had debridement < 12 hours from time of injury. Gustilo-Anderson (G-A) grade IIIA fractures were the most common in 18(32.1%) patients followed by G-A IIIB in 16(28.6%) patients. The infection rate amongst the various group with total injury time to debridement of < 6 hours, 6-12 hours, 13-18 hours and 19-24 hours groups were 5.4%, 14.3%,14.3% and 28.6% respectively.*

Conclusion: *There was no statistically significant difference between total injury time to operative debridement and wound infection in open lower limb long bone fractures once done within 24 hours*

KEYWORDS: Total injury time, Operative debridement, open lower limb fracture, wound infection rate, Gustilo-Anderson (G-A) grade.

Introductions

Open fractures refer to osseous disruption in which a break in the skin and underlying soft tissue communicates directly with the fracture haematoma. They are orthopaedic emergencies due to the risk of infection secondary to contamination. Adequate surgical debridement thus remains an important principle.

Surgical debridement should be performed within six (6) hours from time of injury to reduce the risk of post-operative wound infection. However, this proclaimed "golden 6-hour rule" is not based on rigorous scientific evidence but thought to have originated from a study[2] conducted in1973, where it was reported that 10^5organism per gram of tissue was the infection threshold in open fractures reached in 5.17hours.

Studies have challenged this rule and reported that the timing of surgical debridement in open fractures may not play such a critical role in the prevention of infection[3], particularly with the introduction of antibiotics[4]. Despite the lack of scientific evidence, Gustilo and Anderson's classic article concluded that open fractures require emergency treatment, including adequate debridement and copious irrigation but the article did not specifically assess the relationship between surgical delay and wound infection rate[5]. A prospective study[6] showed that the time of injury to debridement is not a significant independent risk factor for infection rate; whilst another[7]concluded that the strongest predictor for deep seated infection was the grade of the fracture and not the time to debridement.

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There is a high incidence of open lower limb fractures resulting from high rate of motor vehicular accident(MVA), communal and sectarian crises in our catchment population, yet there is no protocol developed locally for the management of these fractures especially regarding timing to operative debridement. Injured patients present late to the hospital because of factors such as poor ambulance services andsocio-cultural beliefs. Further delays are also encountered in the A&E due to patient's instability or logistics such as operative room availability.

The aim of this study was to determine the relationship between total injury time to operative debridement and rate of infection in open lowerlimb long bone fractures.

Materials and methods

This was a prospective longitudinal study that was conducted in the Orthopaedic Department of Jos University Teaching Hospital(JUTH) and recruited patients presenting, with open long bone fractures. All patients presenting within 24 hours after sustaining open lower limb long bone fractures at the A&E of JUTH, who were 18 years of age and above and had consented participated in the study. Patients with Gustilo-Anderson IIIC fracture, intra-articular fracture, uncontrolled immunosuppressive illness and those on steroid were excluded from the study.

The Ethics Committee of the Jos University Teaching Hospital gave ethical approval for the study. Research Assistants were trained amongst the Resident Doctors of the department. Training was on patient recruitment and protocol of the study. They participated in recruitment of patients from the A&E.

Consecutive patients who have met the inclusion criteria of the study were recruited and their bio data and fracture data were recorded. The data included injury time to presentation, time delay in A/E, total injury time to operative debridement, mechanism of

injury, long bone affected and presence of wound infection within 30 days of debridement.

Each patient was resuscitated with intravenous fluids at presentation. The fluid therapy was dictated by the state of the circulation. Blood samples were taken for investigations (blood glucose and full blood count) and patients with abnormal parameters such as low haemoglobin arising from haemorrhage were optimized appropriately. Wound cultures were not taken at presentation nor at the time of debridement.Tetanus prophylaxis (0.5ml of *tetanus toxoid*, 1500IU of anti-tetanus serum) was administered based on the patient's immunization status. *Pentazocine 30mg*was administeredparenterally for pain control.

Debridement was done in the theatre under aseptic conditions and spinal anaesthesia within the shortest possible time following proper resuscitation. Sometimes there were delays due to the nature of concomitant injuries or operating room logistics.

Debridement and irrigation was done with gauze, *5% chlorohexidine* solution using a minimum of 3 litres of normal saline, and 6-9 litres in severely contaminated wounds. Wounds were closed primarily in G-A I, G-A II and some G-A IIIA fractures. Delayed wound closure was done where primary wound closure was not possible, especially in G-A IIIA and most G-A IIIB fractures. Grafts and local flaps were subsequently used for G-A grade III, where soft tissue defects existed. Initial fracture stabilization was done with cast application and where necessary with external fixator or skeletal traction depending on the site of fracture and its Gustilo-Anderson grade.

The total injury time to operative debridement was considered as the sum of injury time before presentation and time delay in the A/E.

Patients received broad spectrum parenteral antibiotics for five days (*Ceftriaxone and Metronidazole*). Fractures were classified based on Gustilo-Anderson system,including grade III A and B.

Wound infection was defined as presence of positive operative wound discharge or clinical evidence of purulence or wound break down requiring operative debridement recorded from 72 hours after first debridement to four [4] weeks after debridement. Gustilo and Anderson stated that "infections were usually evident during the first month after surgery". Spectrum of wound infection included; cellulitis, wound breakdown, stitch abscess and purulent discharge. Where the dressing of the debrided wound became soaked with offensive odour before 72 hours post debridement, a "second look" was indicated and wound swab was then taken for microscopy, culture and sensitivity.

Statistical analysis

Total injury time to operative debridement was divided into the following groups ;< 6 hours, 6-12 hours, 13-18 hours and 19-24 hours.

All data generated were entered and analyzed usingthe statistical package of social science (*SPSS*)*IBM version 23*. The characteristics of respondents were described using frequencies, tables and charts.

The relationship between injury time to operative debridement and wound infection was evaluated using chi-square test and logistic regression analysis.

Results

A total of 56 patients were recruited within the stated duration of the study. All the patients completed the study with at least four completed weeks of post-debridement follow-

up for wound infection. All patients were included in the final analysis of the study. The mean age of the patients were 39.5(±14.3) years. Males accounted for 71.4% of the study population with a M:F ratio of 2.5:1.The age range was 18-73 years with 46.5% of the patients within the age of 18-37 years (Table 1). The most common mechanism of injury in 87.5% of the patients was MVA while 8.9% had fractures due to gunshot (Table 2).

Gustilo-Anderson grade IIIA fractures were the most common occurring in 18 (32.1%) patients followed by G-A type IIIB (28.6%). Seven (12.5%) patients presented with G-A type I fractures (Figure 1).

The total injury time to operative debridement was considered as the sum of time before Presentationand time delayin the A&E. The total injury time to operative debridement for 39 (61.7%) patients was between 13-24 hours with 17 (30.3%) patients having debridement within 12 hours of total injury time. About 42.9% of patients had debridement 19-24 hours after injury despite early presentation at the A/E (Table 3).

Wound infection was assessed using clinical signs and laboratory evidence of positive wound swab culture. Thirty five (62.5%) patients had evidence of post-operative wound infection with wound breakdown and purulent discharge being the common signs, 17.9% and 33.9% respectively (Table 4). From the study wound infection following debridement was noticed between the 4^{th} to 7^{th}days in 25 (71.4%) patients.

Inferential statistical analysis using chi-square, to determine the relationship between total injury time to operative debridement and wound infection was done. There was no statistically significant relationship between total injury time to debridement and wound infection. The rate of wound infection amongst those who had debridement within a total injury time of <6 hours was 5.4%, and 14.3% in those who had debridement after 6 - 12 hours. Of the 24 patients who had debridement after a total injury time of 19-24 hours, wound infection rate was 28.6% (Table 5).

Twelve (21.4%) with G-A grade IIIA had wound infection while the rate of infection in G-A type IIIB was 28.6%. Rate of infection in G-A grade I and II was 1.8% and 10.7% respectively. A statistically significant difference was found between rate of wound infection and Gustilo-Anderson types of fracture ($p< 0.05$) with rate of wound infection proportionate to severity of open fracture (Table 6).

Table 1: Age group distribution of study participants

AGE GROUP(years)	FREQUENCY	PERCENT
18-27	17	30.4
28-37	9	16.1
38-47	13	23.2
48-57	12	21.4
58-67	2	3.6
68-77	3	5.4
TOTAL	56	100

Table 2: Mechanism of Fracture

MECHANISM OF INJURY	FREQUENCY	PERCENT
MVA	49	87.5%
GUNSHOT	5	8.9%
ASSAULT	2	3.6%
TOTAL	56	100%

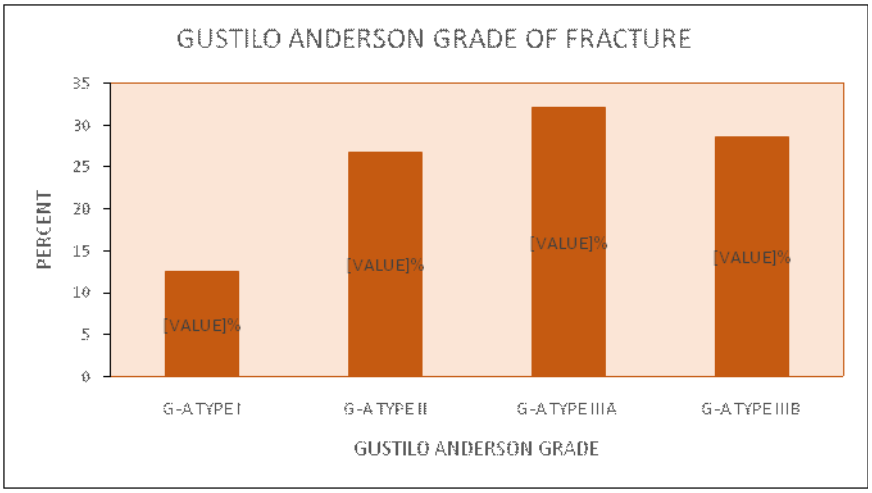

Figure 1: Bar chart showing frequency of Gustilo-Anderson grade of fracture

Table 3: Frequency table of Total injury time before debridement

TOTAL INJURY TIME BEFORE DEBRIDEMENT	FREQUENCY	PERCENT
<6HOURS	4	7.1
6-12HOURS	13	23.2
13-18HOURS	15	26.8
19-24HOURS	24	42.9
TOTAL	56	100%

Table 4: Frequency of post debridement wound infection (N=56)

WOUND INFECTION	FREQUENCY	PERCENT
INFECTION	35	62.5
NO INFECTION	21	37.5
TOTAL	56	100

Table 5: Relationship between total injury time to debridement and wound infection (N=56)

TOTAL INJURY TIME TO DEBRIDEMENT (HOURS)	WOUND INFECTION	
	PRESENT (%)	ABSENT (%)
<6 HOURS	8(14.3)	1(1.8)
6-12 HOURS	8(14.3)	5(8.9)
13-18 HOURS	8(14.3)	7(12.5)
19-24 HOURS	16(28.6)	8(14.3)
TOTAL	35(62.5%)	21(37.5)

Chi square (λ^2) =0.993, P value of 0.803

Table 6: Relationship between G-A grade of fracture and wound infection (N=56)

GUSTILO-ANDERSON GRADE OF FRACTURE	WOUND INFECTION		
	PRESENT (%)	ABSENT (%)	
G-A I	1(1.8)	6(10.7)	
G-A II	6(10.7)	9(16.1)	
G-A IIIA	12(21.4)	6(10.7)	
G-A IIIB	16(28.6)	0(0.0)	
TOTAL	35(62.5%)	21(37.5%)	100%

Chi square=21.634, P value of 0.00

Discussion

The proclaimed "golden 6 hour rule" for surgical debridement[2] to be performed in order to prevent or minimize post-operative wound infection does not appear to be based on rigorous scientific evidence. Studies have shown that the timing of debridement in open fractures should not be cast in stone, particularly with introduction of antibiotics[3,4].

This study was designed to determine the relationship between injury time to operative debridement and incidence of post-operative wound infection in open lower limb fractures. The rate of wound infection was also evaluated in relation to Gustilo-Anderson grade IIIA and B open fractures and the site of lower limb fracture.

The main finding in this study was that there was increased infection rate, the longer the time to debridement. However, this was not statistically significant. The study however showed that the factors that determine wound infection in open fractures are; the G-A grade of fracture, site of long bone fracture and choice of method of stabilization.

A retrospective study[7] of 215 open tibia fractures over a 6 years period concluded that, there was no statistically significant relationship between injury time to operative debridement and wound infection. In the study[4] of 315 patients with severe high energy lower extremity injuries, the time from injury to operative debridement is not a significant independent predictor of the risk of infection.

One study[8], found wound infection rates of 10.1% and 10.8% in debridement carried out <6 hours and >6 hours from injury time respectively. There was no significant infection rate in patients that had debridement > than 6 hours after the injury. Following a prospective study of 63 patients with open fractures of the lower limbs, in Ile-Ife[9], the authors concluded that the time interval between injury and wound debridement was a major prognostic factor in infection. While another study[10], found that delay from injury to surgery had no statistically significant effect on rate of infection.

The mean total injury time to debridement in this study was 16.2±6.1 hours. A group[11], reported a mean time to debridement of approximately 13 hours, while others reported 10.6 hours and 8 hours respectively[12,13]. The higher total injury time to debridement in this study was due to the delays in A&E, with 53.6% of patients having a time delay of 6-12 hours despite a relatively early presentation. The delays in A&E were due to reasons such as lack of urgency in wheeling patient for radiologic investigations, theatre unavailability, patient's physiological instability and co-existing injuries. The exact effect of some of these factors may be beyond the scope of this study. The rate of wound infection in relation to total injury time before debridement showed a 5.4%, 14.3% and 28.6% incidence in the <6 hours, 6-12 hours and 19-24 hours group respectively. Others have reported an infection rate of 6.8% in those debrided <12 hours.[10]Others have found infection rates of 10.1% and 10.8% in debridement carried out < 6 hours and > 6 hours respectively[8]. Similarly others showed an infection rate of 10.8%, 9.5% and 5.6% for injury time delay to operative debridement of 0-6 hours, 6-12 hours and 12-24 hours respectively[7].Another study[14]also showed that the incidence of perioperative infection in patients with open fractures has little to do with the time of first debridement, which is mainly related to the level of the Gustilo-Anderson classification. The relatively higher infection rates in our study might be due to the relatively smaller sample size, standardization weakness

and probably the effect of the method of initial fracture stabilization; which future research topics should address if absolute conclusions are to be made. The absence of a statistically significant relationship between total injury time to debridement and wound infection agrees with the findings in the other studies.[7,8,10] Some of the limitations in our study included; difficulty in establishing the exact injury time and transportation time as reported by patients or relations and operating theatre logistic challenges.

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