UNIVERSITY OF NAIROBI

A STUDY OF SOME CLINICAL AND LABORATORY ASPECTS OF THE AFRICAN SUFFERING FROM DUODENAL ULCERATION

BY

DR. GODFREY WASHINGTON ENOCH NSEREKO LULE
MB. chB (Makerere)

A THESIS SUBMITTED IN PART FULFILL MENT FOR THE DEGREE OF MASTER OF MEDICINE (Medicine) AT THE UNIVERSITY OF NAIROBI 1982

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DECLARATION

CANDIDATE

This thesis is my original work and has not been presented for a degree in any other University.

DR. GODFREY LULE MB ChB (Makerere)

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SUPERVISORS

This thesis has been submitted for examination with our approval as University Supervisors.

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CONTENTS

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Lastly but not least I would like to thank everybody who helped me in the preparation of this manual; without their help nothing would have been possible.

SUMMARY

This is a prospective study of 50 patients with duodenal ulcer proved by endoscopy. The clinical and laboratory features of these patients were analysed and where possible compared to a group of 30 control subjects.

It was found that a significant number of duodenal ulcer patients do not present with the classical clinical picture. The frequency of blood group O was more in the duodenal ulcer patients as compared to the controls and duodenal ulcer patients had higher basal and maximal acid output values. An attempt at interpreting these results in the Kenyatta Hospital set up has been made.

INTRODUCTION

A duodenual ulcer may be defined macroscopically as a round, oval, elongated or elliptical defect in the mucosa of the duodenum. The edges of the ulcer are often indurated and hyperemic with the ulcer base covered by an adherent granula or slimy whitish gray exudate. The surrounding mucosa is normally seen as a soft, pliable and normal coloured fold though occasionally oedematous and hyperemic. Microscopically it is a defect in the duodenal mucosa which extends through the muscularis mucosae. Normally an ulcer is up to 1 cm or less in diameter but occasionally giant ulcers occur which measure 2 - 3 cms in their greatest dimension. In western Europeans more than 95% of duodenal ulcers occur in the first part of the duodenum and approximately 90% of these are located within 3 cm from the pylorus. (Mc Guigan, 1980). Ulcers may be located distal to the duodenal bulb; in this situation they are referred to as Postbulbar ulcers but their symptoms do not differ significantly from those of ordinary duodenal ulcers.

Clinical and epidemiological studies of duodenal ulcers have been hampered by the lack of reliable set of criteria for indentification of the affected individuals. Amongst factors that have been used are symptom patterns, radiographic and endoscopy findings, surgical reports and examination of tissues obtained at autopsy. Hirschowitz et al (1958) were the first people to introduce the fibreoptic gastroduodenoscope with a hope that endoscopy would be useful adjunct in the management of duodenal ulcer disease. However this was not realised in the early period until 1968 when new endoscopes came into use. In 1971 Belber compared results of endoscopic examination of the duodenal bulb to Xray and found that 25 to 35% of the

radiologically diagnosed ulcers were not found on endoscopy.

He suggested endoscopy as the major diagnostic tool in ulcer disease. Other workers following his have tended to share the same view. Rogers in 1976 compared endoscopy, routine and double contrast barium meal study in diagnosis of gastric and duodenal disorders and showed the endoscope as the leading tool in giving the diagnosis followed by double contrast and lastly routine barium study. In Kenyatta National Hospital (K.N.H) work was done to try and establish the most reliable diagnostic aid (Wankya et al 1979) and endoscopy was recommended as the most reliable diagnostic procedure in upper gastrointestinal disorders.

In his series of 15,000 consecutive endoscopicexaminations of the duodenum, Ottenjam in 1979 noticed only 3 cases (0.2%) of malignant ulceration in the duodenal bulb. Thus the occurence of malignancy is extremely rare and there has been no routine necessity to biopsy the duodenal bulb. However ulcerative lesions in Crohn's disease of the duodenum seem to be becoming more common so that the general opinion that it is not necessary to biopsy duodenal ulcers may need to be revised in the future. (Classen, 1980).

The familial aggregation of peptic ulcer and its association with clear cut genetic factors such as blood group O and nonsecretor status is well established in the Western world but not in Kenya. (Edwards, 1965, Roberts 1965, Langman 1973). It has also been proposed that peptic ulcer is not one disease but a group of disorders with different genetic and environmental causes, (Rotter, 1980. McConnel 1966). This unravelling of the genetic heterogeneity of peptic ulcer has important clinical and aetiological implications, for if what is termed a disease is in reality a group of disorders put together due to some clinical features, these distinct disorders may differ markedly in genetic, pathophysiology,

interaction with environmental agents, prognosis and response to therapy.

Amongst the many factors studied in the genetic predisposition to duodenal ulcer have been the blood groups. Aird et al, 1954 showed that duodenal ulcer was associated with blood group O. This was later worked out by Langman in 1973 when he found that individuals with blood group O have a 30 - 40% greater incidence of peptic ulceration than those with other blood groups. Many other genetic markers have been studied apart from blood group O. There is a small but significant increased risk of duodenal ulcer in subjects who fail to secrete ABO blood group antigens in saliva (Cowan, 1973). The effect of both blood group O and nonsecretor status is multiplicative and has been claculated to about 2.5 times that of a normal individual in predisposing to duodenal ulceration.

Workers in the Western world and elsewhere in the developed countries have found that secretion of acid and pepsin is necessary for duodenal ulceration to occur. The average rates of basal and stimulated secretion of gastric acid were found by Wormsley, 1965 to be higher in patients with doudenal ulcer than in controls although some duodenal ulcer patients have acid secretions that fall within the normal range. This was further established by Goldberg in 1969 when he studied the role of acid and pepsin in the aetiology of duodenal ulceration and found both factors involved. . He found that pepsin played a signigicant role at pH 1.6 - 2.0 but that at higher levels of acidity, which are often present in the stomach of human subjects, acid alone can produce injury. From results obtained by Wormsley and Grossman in 1965 duodenal ulcers not uncommonly develop in patients who secrete less acid/pepsin than the mean value for normal subjects. It is also true that although a large area of mucosa is exposed to acid/pepsin, only a small cir cumscribed area develops ulceration. Therefore localised impairment

must be postulated in virtually every patient to explain the focal rather than diffuse lesions produced. This observation implies that decreased mucosal resistance plays a role in at least some cases of ulceration.

Cox, 1952 demonstrated that patients with duodenal ulcer have a higher than normal number of parietal cells in their stomachs. This would explain the higher basal acid output and histamine or pentagastrin reponses in the duodenal ulcer subjects. Factors that tend to change the acid-pepsin load or lower the mucosal permeability barrier would lead to increased ulcer risk. These are the enviromental factors an individual is exposed to and over the recent years this has gradually gained prominence. The association between cigarrette smoking and peptic ulcer was first documented by Friedman in 1974 when he found that smokers have more ulcers (Gastric and duodenal) than non-smokers and they have higher death rates from ulcers than non-smokers. Although Bordemar et al 1981 concluded that smoking delays ulcer healing, no definite relationship has been found between amount smoked and the risk of ulcer.

Coffee stimulates gastric acid secretion although the evidence linking coffee drinking to ulcer is doubtful. In his study Friedman did not find any association between alcohol and coffee consumption and the prevalence of peptic ulcer. However in contrast, Paffenbarger et al (1974) found in college students that ingestion of coffee or other beverages, mainly colas, increased the risk of later development of ulcers whereas ingestion of milk decreased the risk.

It is the purpose of this paper to study and report on the importance of all these observations in the pathogenesis and existence of the duodenal ulcer in our Kenyan African enivornment.

MATERIALS AND METHODS

The study was carried out between September, 1980 and May, 1981. During this period, the author together with two experienced endoscopists examined all patients suspected to be suffering from duodenal ulceration using the fibreoptic Olympus GF_1 P_2 panendoscope and lecturescope in the minor theatre of surgical outpatients. The patients studied were a select group, chosen ater interviews relating to symptomatology by either the author assisted by the two supervisors or referred from the medical wards outpatient clinics and medical and surgical wards of Kenyatta National Hospital. For each patient the age, sex, tribe and weight were recorded. The major presenting symptom was abdominal pain and patients were interviewed about the site of pain, its nature whether burning, gnawing , boring, or aching. They were further asked about association of this pain with food and the time interval following ingestion of the food. The duration of this pain, whether continuous or intermittent with remissions and relapses was also noted. The severity of this pain, relieving and aggravating factors specifically mentioning maize grain, milk smoking and alcohol were asked for. The subjects were also asked whether the pain radiated to an extra site and whether they only had day pain and/or night pain. After recording the information they would then be subjected to endoscopy.

ENDOSCOPY

Patients were booked for endoscopy every Tuesday during the period of the study. They were required to fast overnight preceeding the examination. On presenting to the minor theatre in the surgical outpatients each was given a gown and then given premedication.

Pentocaine 2% was used topically for pharyngeal anaethesia. Atropine 0.6 mg was given intravenously 10 minutes prior to introducing the endoscope. Hyoscine Butylbromide (Buscopan) 40 mg was given and in addition 5 - 10 mg of diazepam. The patient was placed on the examination couch lying in the left lateral position. A Dental guard to protect against damage to the instrument and to facilitate easy passage of the examining scope was put in the mouth of the patient. It would be passed right down into the stomach and following the method advised by Wormsley et al (1965) the duodenal opening was identified and the duodenum entered.

A careful examination of mucosa was done and the findings collaborated by another observer via the lecturescope. The duodenal bulb was always thoroughly examined followed by the duodenal tunnel. The endoscope was then carefully withdrawn observing the mucosa of the duodenum, stomach and oesophagus. The subjects with proven duodenal ulce were then advised to undergo further testing. The few patients who declined were given the appropriate advice and sent back to the referring clinics.

PENTAGASTRIN AND STIMULATION TESTS

These were carried out in the department of medicine and patients were given appointments by the technician who helped to carry out the tests. They were asked to fast after midnight on the night preceeding the day of the test.

The following procedure was then followed.

(a) The patient was weighed and the weight in Kilogrammes used to calculate how much pentavlon (pentagastrin) would be required. Each patient was given a total dose of 8 ug per Kg. body weight.

(b) The patient would then lie on the couch in a position of comfort with the head rest propped up at an angle of 30^{o}. A standard nasogastric tube was then selected, its lower end lubricated with jelly and with no local anaesthetic it was passed into the stomach. Radiological proof that the tube was in the stomach was not possible but using a 5 ml syringe 2-3 mls of air were introduced in the stomach and using a stethoscope auscultation over abdomen was done. Whenever the tube was in the stomach the air would be heard entering the stomach.

(c) The tube was then anchored to the nose with adhesive tape and a small gastric fluid aspirated to check on the position of the tube. It was then connected to the pump and via interconnecting tubes, to the collecting flasks. This is a method similar to that by Tucker et al (1980). The tube was then connected to an electric low flow pump to enable suction of gastric aspirate at a steady rate. Collection of gastric aspirate was done every 15 minutes for the subsequent 45 minutes.

The samples thus obtained were referred to as Basal I and 2 respectively. After Basal 2 an intramuscular injection of pentavlon 8ug/kg body weight was given and 15 minutes collections of gastric aspirates made for I hour. These were then labelled as gastric 1, 2, 3, and 4 respectively and results tabulated as below:-

15 minutes Specimens	*volume Ml.*	*concn. Meq/litre*	*output Meq*	*pH*
Fasting				
Basal 1				
Basal 2				

Intramuscular injection of pentavlon at 8ug/kg body weight was given at this point and result tabulated as below:-

Penta-gastrin	*volume ml*	*concn. Meq/litre*	*output Meq.*	*pH*
1				
2				
3				
4				

TITRATION

10 mls of distilled water was put in a porcelain jar and 1 ml of gastric fluid added. 2 drops of indicator (0.1% Bromothymol blue dissolved in menthanol) were then added to obtain an orage colour. Titration with 0.IN sodium hydroxide was then carried out until a greenish blue end point was reached and the volume of sodium hydroxide used was recorded from the burette. The process was then repeated for all samples collected as shown in the table above.

CALCULATION

Base used (NaOH) is 0.0IN.

Milliequivalents of acid neutralised

= (vol. of base used x 10) milliequivalents/litre

Meq. output per specimen =

= volume obtained x meq/litre

= Meq/specimen obtained.

INTERPRETATION

Basal 1 and 2 were used and double this would give the basal Acid output per hour (BAO/HR) . The 2 highest responses to 8ug of pentagastrin were added and multiplied by 2 to obtain maximal acid output per hour (MAO/Hr). The results obtained were compared between doudenal ulcer patients and normal control group.

DISTRIBUTION OF BLOOD GROUPS IN THE PROVED DUODENAL ULCER PATIENTS AND NORMAL CONTROLS

The tube method was the procedure used. About 5 mls. of blood were collected from each patient. The red blood cells were washed 3 times in saline and then a thin cell suspension was made. Three tubes were labbelled A, B, and D. Using a pasteur pipette one drop of the patients washed red blood cells was added to each of the 3 tubes.

In tube A 1 drop of anti A serum was added.

In tube B 1 drop of anti B serum was added.

In tube D 1 drop of anti D serum was added.

INTERPRETATION OF RESULTS

If there was no agglutination in both tubes A,B and there was agglutination in tube D, the blood group was O positive.

If there was agglutination in tube A and D, if was A positive.

If there was agglutination in tube B and tube D the blood group was B positive.

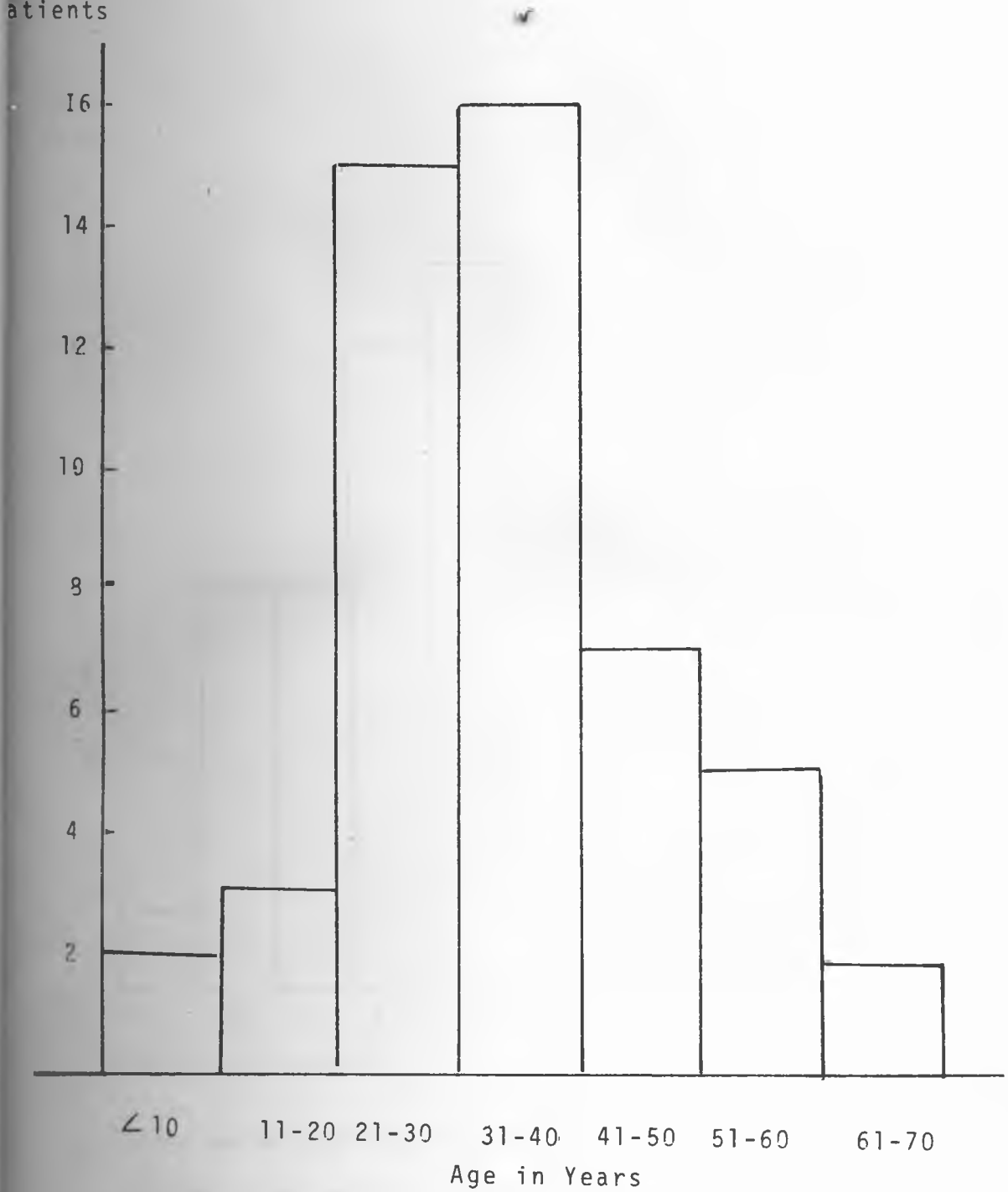
Where there was no agglutionation in tube D in the above steps then the blood group was either A, B or O negative.

CONTROL GROUP

This group consisted of 30 subjects with no symptoms or signs suggestive of duodenal ulcer and who were negative for duodenal ulcer on endoscopy. They underwent clinical and laboratory investigations similar to endoscopically proved duodenal ulcer patients in this study.

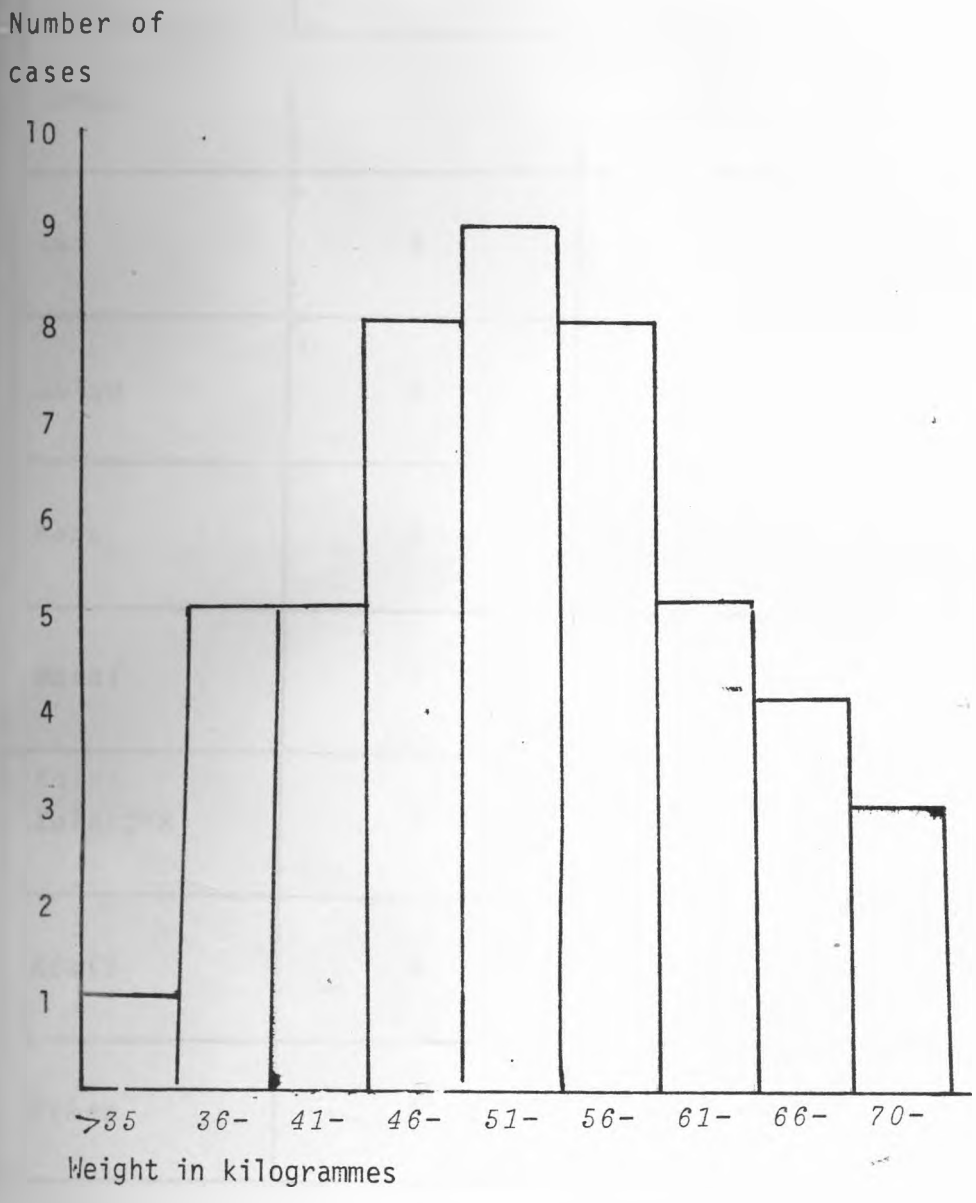
RESULTS

Histogram I showing the age distribution of 50 patients with duodenal ulcer.



The majority of patients were aged 21 to 50 years age (76% of total number studied.)

Histogram 2 showing distribution of patients with duodenal ulcer by weight.



There was no significant change in age corrected weights in the duodenal ulcer patients as compared to the normal group.

Table 1 showing the distribution of patients with duodenal ulcer by tribe:-

Tribe	Number of Duodenal patients	perc %	KNH % Admission Rate
Kikuyu	22	44	33.9
Kamba	6	12	19.8
Luo	8	16	17.2
Luhya	5	10	17.2
Meru	3	6	3.9
Masai	0	0	3.3
Kalen Kalenjin	1	2	1.0
Kisii	2	4	2.2
Other	2	4	—

Note: 1. Admission rates for KNH have been used and thus do not reflect the actual outpatient attendance which has not been worked out.

Table 2 showing distribution of patients with duodenal ulcer by sex

Sex	Number of Patients	Pencentage
Male	34	68
Female	16	32

The ratio of male to female patients with duodenal ulcer in this series is 2:1

Table 3 showing the site of abdominal pain in 42 duodenal ulcer patients

Qbdominal Site	Number of patient	percentage
Epigastrium	27	64.3
Right upper quadrant	10	23.8
Other	5	11.9

Table 4 showing radiation of the pain

Di rection	Number of patients	percentage of total
Retrosternal	16	38
Back	6	14.3
other side	2	4.7
No radiation	18	43.0

The majority of the patients with radiation had their pain radiating retrosternally. (38%)

Table 5 Duodenal Ulcer patients with Night pain

Parameter	Number of patients	percentage
Night pain	26	62
No night pain	10	24
Not Known	6	12

A significant number of patients had night pain (62%) which awakened them occasionally.

Table 6 Relationship of/maize ingestion and day pain whole/

Pain	Number of patients	Percentage
Aggravated by maize	15	36
Not Aggravated	21	50
Do not know	6	14

Table 7 - Response of pain to milk ingestion

Pain	Number of patients	percentage
pain improved	28	67
No effect	14	33

Milk improved the pain in 67% of cases

Table 8 - Distribution of Nausea and Vomiting in the Duodenal ulcer patients

Nausea and Vomiting	Number of patients	Percentage
Present	29	69
Absent	12	31

Table 9 - The prevalence of smoking in the group of Duodenal ulcer patients

Cigarrette smoking	Number of Patients	percentage of total
smokers	15	36
Non smokers	21	50
Non answer	6	14

36% of the patients were smokers

Table 10 - showing Alcohol consumption in Duodenal Ulcer patients

Alcohol	Number of patients	pertcentage
Use Alcohol	17	40
Do not use	25	60

40% of the patients used alcohol.

Table 11 - Showing Distribution of blood groups in 50 duodenal ulcer patients compared to 30 controls

Blood group	Number of patients with Duodenal Ulcer	Number of Controls
A	8	8
B	10	8
O	31	15
AB	1	0

(see next table for percentage and significance).

Table 12 - Distribution of 49 patients with duodenal ulcer by blood groups compared with the distribution in the control group.

Blood group	number of patients	percentage	no. of controls	percentage
A	8	16.3	8	25.8
B	10	20.4	8	25.8
O	31	63.3	15	48.4
Total	49	100	31	100.0

$X^2 = 1.82$ $df=2$ $n=49$ $0.5 > p > 0.2$

1. There was no significant difference betwen ABO blood group distribution of duodenal ulcer patients compared with that of the controls.

2. Comparing blood group O in the duodenal ulcer subjects

to the normal control group there were more duodenal ulcer patients with blood group O than normals.

Table 13-showing distribution of the basal and maximal Acid output in each of the 47 duodenal ulcer patients:-

Patients	B.A.O Meq/Hr	M.A.O Meq/Hr	Patients	B.A.O Meq/Hr	M.A.O. Meq/Hr.
1	6.2	21.0	24	1.6	12.2
2	4.6	29.4	25	1.9	15.0
3	3.6	30.6	26	2.1	18.4
4	2.1	19.0	27	1.6	12.2
5	8.2	41.0	28	3.0	19.6
6	0.8	13.4	29	1.6	24.6
7	16.4	49.6	30	2.2	8.4
8	3.0	33.4	31	3.0	33.4
9	1.8	35.8	32	4.2	42.0
10	2.2	29.4	33	3.5	28.4
11	9.2	24.0	34	2.8	36.0
12	10.4	36.2	35	10.4	18.8
13	1.6	17.8	36	20.6	35.6
14	2.6	6.8	37	2.6	30.4
15	5.8	34.6	38	3.4	45.6
16	14.4	84.0	39	12.0	34.6
17	5.8	22.6	40	19.0	42.4
18	20.8	31.4	41	14.0	46.2
19	22.0	59.0	42	5.1	38.0
20	6.8	15.8	43	5.8	50.6
21	5.2	14.6	44	1.0	16.6
22	2.0	45.8	45	1.4	16.8
23	3.8	21.4	46	7.0	32.0
24	1.6	12.2	47	8.6	44.4

B.A.O. - Basal Acid Output

M.A.O. - Maximal Acid Output

See table 15 for the mean and standard deviation.

Table 14 - showing distribution of the Basal and maximal Acid Outputs in each of 30 normal control subjects:-

Patients	B.A.O. Meq/Hr	M.A.O Meq/Hr	Patients	B.A.O. Meq/Hr	M.A.O Meq/Hr
1	3.6	32.6	16	2.9	9.3
2	1.6	12.2	17	3.2	10.0
3	1.8	14.0	18	3.2	28.2
4	4.2	12.2	19	3.2	27.0
5	0.8	8.0	20	1.1	8.4
6	1.6	3.0	21	2.4	27.0
7	1.4	3.8	22	2.1	16.8
8	1.6	3.8	23	3.1	14.2
9	6.8	35.6	24	2.1	8.6
10	7.6	21.2	25	1.8	9.0
11	3.2	10.0	26	1.0	3.0
12	4.8	1.8	27	1.9	3.2
13	0.6	0.8	28	1.8	3.6
14	2.4	5.4	29	4.8	10.8
15	1.7	5.6	30	1.6	9.0

B.A.O - Basal Acid output

M.A.O - Maximal Acid Output

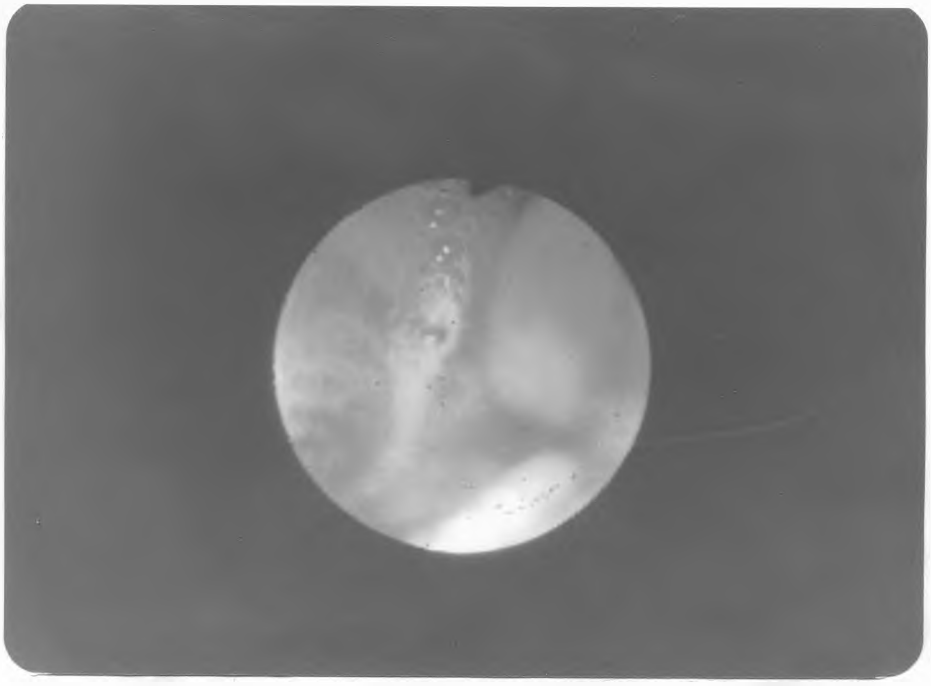
See table 15 for the mean and standard deviation.

Table 15 - Basal and Maxinal Acid output in patients with duodenal ulcer and normal controls

Measurement	*Patients with duodenal ulcer n-47*	*Controls n-30*	*t value*	*p value*
Basal Acid Output ± 1SD (Meq/Hr)	*6.30 ± 5.80*	*2.80 ± 1.60*	*3.89*	*<0.001*
Maximal Acid Output Mean + 1.S.D Meq/1Hr	*30.20 ± 14.80*	*11.90±9.50*	*6.54*	*<0.001*

Note: *Students t test for the analysis of the paired data used for evaluating the results.*

Photograph 1- showing an active duodenal ulcer as seen at endoscopy



Large duodenal ulcer with Fibrin deposit base (Yellow) and areas of variable healing in between

Large ulcer extending from about 4 o'clock to 10 o'clock. Note the apparent fibrinoid deposition (Yellow).

DISCUSSION

AGE

Histogram I shows that although duodenal ulcer can occur at any age, the majority of our patients were between 20 - 40 years of age. Correcting these ages for the duration of the disease which ranged between 5-10 years in most cases, it was found that for the majority of our patients the onset of the disease was at a very early age, in most cases less than 25 years. This would tend to mean that the Kenyan African is predisposed to developing ulcer at a fairly early age. In 1976 Lam and Ong classified duodenal ulcer into two subgroups on the basis of age of onset of the disease. They found that their early onset group (onset below age 20 years) was characterised by a significant family history, no relationship to blood group O and was less prone to major complications. In contrast they had a late onset group (onset after age 30 years) who had a significantly infrequent family history of ulcer disease, had an increase of blood group O, and was prone to more complications. In this present study most patients tended to be young falling in their early onset category. This could have been probably due to a relatively small sample of 50 compared to theirs or probably due to the fact that the general population of patients attending Kenyatta National Hospital is predominantly a young population in relation to the young Kenyan Society.

SEX

As shown in table 2 ratio of male to female patients with duodenal ulcer was approximately 2 to 1. This compared favourably with work done else where. Workers in Western Europe have shown the incidence of new cases as about 1.8 per 1000 adults males and about 0.8 per 1000 adult women per year (Pulvertaft 1959, Bonnevie 1975) although these figures appear to be getting less. This would put the male to female ratio as 2:1. In 1963 Crean found that women have a relatively low incidence of peptic ulceration especially prior to menopause and during pregnancy. Although our group was a select group of patients it was evident that the male sex is more predisposed to duodenal ulceration with no difference between the Kenyan African and other people in the rest of the world. This would tend to implicate sex hormones in the aetiolgical pathogenesis of these ulcers. At least two studies have shown that Oestrogen treament hastens ulcer healing (True love 1960 Parbhoo et al 1966). Acid and pepsin secretion are not decreased by oestrogen treatment in humans although Parbhoo et al has suggested that mucus production is increased during such therapy. There is no fall in acid secretion during pregnancy although progesterone given intramuscularly lowered acid secretion modestly in patients with peptic ulcer in the study quoted above. Thus sex hormones appear to play a role in duodenal ulcer although the true mechanism by which female sex protects against ulcer is not yet clear.

TRIBAL DISTRIBUTION

In this study the Kikuyu tribe had the majority of patients (44% of the total) who had duodenal ulcer, closely followed by the Luo, Kamba and Luhya (table 1). Although it was not possible to work out the tribal outpatient attendance of Kenyatta National Hospital, the tribal distribution was compared to the admission rates to Kenyatta National Hospital in 1979 and did not differ significantly. Thus there was no tribe which was more predisposed to develop duodenal ulcer, the results just reflected the attendance at Kenyatta National Hospital.

SYMPTOMATOLOGY

In this study 64% of patients had their pain in the epigastrium while 24% had it in the right upper quadrant. The pain radiated retrosternally in 24% of the cases and 62% of the cases had night pain. Boiled maize grain made the pain worse in 36% of the cases and the pain was improved or disappeared after ingestion of milk in 67% (table 3-10). Comparing our results with that of other workers there were major differences in how these patients presented. In 1976 Earlam obtained data by questionnire from 100 consecutive hospitalised duodenal ulcer patients. At least 81 reported their pain as being epigastric, 88 had night pain and the pain radiated to a retrosternal location in 59. The ulcer patients weighed less than an age and sex matched control group with 79 reporting that the pain caused them to eat less. In a follow up study Harrocks and De Dombal (1978) studied 360 patients and their findings generally agreed with those of Earlam dispelling the notions that there is a prominent

relation of ulcer pain to ingestion of food and that patients with duodenal ulcer eat more often to feed their ulcer. Although we only studied 50 patients the difference in presentation between the Kenyan African and white duodenal ulcer patients appeared to differ.

This could have been due to the methods of investigations used. In Earlams study he sent out a questionnaire relying heavily on the patients interpretation of his questionnaire. He also used radiological proof other than endoscopic results in his diagnosis of the duodenal ulcers. In this study it appears that many patients presented atypically than those in Earlam and De Domba'ls study quoted above. However, it agreed well with Peterson who in 1980 emphasized that it appears that the classic symptoms do not ensure the diagnosis of duodenal ulcer nor do their absence in anyway exclude the diagnosis. Thus a patient who has no epigastriumpain at all can present as an emergency due to complications in a silent active duodenal ulcer.

Although Earlam's study group had showed some increase in weight which he had attributed to eating frequently, there was no significant change in age corrected weights in the duodenal ulcer group as compared to the normal population (Histrogram 2). Various studies all over the world have tended to differ on this point, others stressing increase in weight due to frequent feeding to relieve the pain while other like De Dombal (1978) found loss of weight which they explained as due to frequent vomiting. Only 69% of the cases in my series had nausea and vomiting at least at one time of their illness despite the fact that there was no significant gastric outlet obstruction nor of duodenal stenosis, but even these did not present with signifcant weight loss.

Hence the mode of presentation of our patients in Kenyatta National Hospital appears to differ from that of the White Europeans and Americans but further work using a large sample of patients needs to be done.

SMOKING

Various workers have shown a strong association between cigarrette smoking and duodenal ulceration. (Doll et al 1958, 1964, Edwards et al 1964, Cooke 1980) More gastric and duodenal ulcers have been found in smokers than in nonsmokers, and smokers have higher death rates than non-smokers.

In this study (table 9) 36% of our duodenal ulcer patients were smoking agreeing with results elsewhere that smoking is associated with duodenal ulcers. However the percentage of the population in Nairobi or Kenya which smokes is not known and so no direct significance can be put on these results. Nicotine may reduce pancreatic bicarbonate secretion and thus might reduce acid neutralisation in the duodenum. There is also some increase in duodenogastric reflux in some patients with peptic ulceration who smoke making available more acid pepsin to cause ulceration.

ALCOHOL

This is widely known for its cause of acute gastric mucosal erosions but no evidence as to whether it directly causes duodenal ulcer has been forthcoming. While discussing environmental aspects of ulcer disease Cooke in 1980 denied any association between alcohol and the duodenal ulcer but made the exception of the association of duodenal ulcer with alcoholic cirrhosis of the liver. In this study 38% of our duodenal ulcer patients agreed to using alcohol in moderation at least once a week (table 10). Our figures were too small and the group too highly selected to make any major conclusion but it is evident that there might be some association

LABORATORY STUDIES

BLOOD GROUP

From table 12 there were more patients with blood group O compared to blood groups B and A in the duodenal ulcer subjects, although there was no significant difference between ABO blood groups distribution in the duodenal ulcer patients and the control. (X^2=1.82 0.5> P >0.2) The control group had a similar distribution of blood groups to that already worked out for the rest of Kenya (Beecher et al 1967). Soon after the original association between blood groups and gastric cancer was described by Aird et al in 1954 the same group of investigators noticed that peptic ulcer was associated with blood group O. It was calculated by McConnell in 1966 and Langman in 1973 that individuals with blood group O have a 30 - 40% greater incidence of peptic ulcer than those of other blood groups. These results agreed with ours showing a higher incidence of blood group O in duodenal ulcer patients.

Lam and Ong classified duodenal ulcer subjects into young and old classes already referred to above. Going by age groups the patients in this study would fall in their young onset group who they claim have no association with blood group O. The patients studied were few and probably more patients would have to be studied to determine the real relationship.

ACID STUDIES

The Basal acid output B.A.O. in our duodenal ulcer patients was much higher than that our control group. (student t test = 3.89 $p > 0.001$) The range of BAO in our doudenal ulcer patients was 0.8 - 22.0 Meq/Hr with 1/3 in the normal secretion range. This agreed with figures by other workers elesewhere such as Cox who in 1952 also found the same relationship. After pentagastrin stimulation the maximal acid output had a mean of 30.20 Meq/Hr. in the duodenal ulcer subjects as compared to 11.90 Meq/Hr. in the control group. (student t test = 6.54, $p > 0.00$) with a range of (12.2 to 84.0 Meq/Hr) in the duodenal ulcer subject compare- to (0.8-32.0 Meq/Hr) in the control group (table 15). Wormsley et al 1967 and Baron, 1963 reported that duodenal ulcer subjects secrete more acid than normal subjects and have a higher response to stimulation than normal subjects. They also concluded that patients with duodenal ulcer have an increased capacity to secrete acid, increased rate of emptying of food in the stomach and duodenum, increased responsiveness to stimulation of acid secretion by pentagastrin and decreased inhibition of acid secretion. Some of these findings were very much shown by our results showing no difference between the Kenyan African Duodenal Ulcer patients and that of the Western European duodenal ulcer patients.

Duodenal ulcers do not occur in the absence of acid, thus the mucosal permeability together with the amount of acid determine whether an ulcer will occur. (Grossman 1979). However if acid pepsin secretion was the sole cause of ulcer disease, one expects that all gastrinoma (Zollinger-Ellison Syndrome) patients with massive amounts of gastric acid and pepsin secretion would develop ulcer. This is not the case. Isenberg in 1973 reported some gastrinoma patients with marked gastric acid hypersecretion, but without evidence of ulcer.

The most likely explanation for this discrepancy may be the net result of interaction of a number of genetic, enviromental, pathophysiological and psychological factors although acid plays a big role. It is believed that once duodenal defence machanisms have been breached, acid from the human diffuses into the mucosa and submucosa setting into sequence a series of events resulting ultimately into the destruction of capillaries causing gastric and duodenal erosion. (Sturdevant, 1979). This would be more likely to occur in those with hypersecretion, a fact very well shown by our results.

CONCLUSIONS

From this study several conclusion were made:

1. The Male Kenyan is twice as predisposed to duodenal ulceration as his counterpart female, and will get his ulcer at an early age.

2. The development of a duodenal ulcer depends both on the genetic predisposition an individual has and the environment he is exposed to. This does not differ from the European set up.

3. There is a significant group of duodenal ulcer patients who do not present with the classical symptoms and one has to keep this group in mind when dealing with upper gastrointestinal problems and emergencies.

4. The ABO blood group distribution in the duodenal ulcer patients taken as a whole does not differ significantly from that of the normal population although more duodenal ulcer patients have blood group O. Further work to verify this is still needed.

5. Duodenal ulcer patients secrete a lot of gastric acid normaly and respond to stimulation with pentagastrin with increased secretion having higher values than normal people.

6. It would appear that whole maize may affect the ulcer subjects in a way that is not clear at the moment and needs further elucidation.

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