PYLORIC STENOSIS ASSOCIATED WITH CHRONIC DUODENAL ULCERATION:- REVIEW OF SEVENTY EIGHT CASES TREATED AT KENYATTA NATIONAL HOSPITAL

A dissertation presented in part fulfilment of Master of Medicine (Surgery) Part II.

BY

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DECLARATION

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

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SUPERVISOR

This dissertation has been submitted for examination with my approval as University Supervisor.

.....

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INTRODUCTION

Duodenal ulcer is a chronic disease characterised by ulceration. The part of the duodenum affected is the duodenal bulb, the part just beyond the narrow pyloric channel through which gastric contents enter the duodenum. In a few individuals the ulcer heals without further recurrence but in most patients it recurs over the years, some often and in others infrequently. (Hallenbeck 1976)

Incidence and prevalence

Prior to the turn of the twentieth century duodenal ulcer was rare in the United States of America and Western Europe. The prevalence was then 0.1-0.3 per cent of the population (Ivy, 1950). In the period 1900- 1910 the prevalence had risen to 1.0% of the population. After 1910 the incidence of duodenal ulcer increased and by 1950 the prevalence of duodenal ulcer was 2.2 - 3.3% of the population. After 1950 the incidence of duodenal ulcer in Western countries has steadily declined by about 8% per annum, (Mandeloff, 1974, Susser 1967).

The incidence of duodenal ulcer in Africa and rural Indian Sub-Continent has been low until the early sixties (Cleave 1962, Tovey, 1975). In 1935, Brainsbridge, the first surgical specialist in Kenya reported that during his ten years of practise in Nairobi he saw only two cases of duodenal ulcer in Kenyan Africans. Wright in Kenya (1954) could only recall two cases of duodenal ulcer seen among the Africans over a period of eighteen years.

Miller (1966) in Nairobi reported that duodenal ulcer was being diagnosed among the Kenyan Africans at the rate of sixty cases per year at Kenyatta National Hospital. Whittaker (1966) in a series of one thousand cases examined radiologically reported a prevalence of 22.2% of positive duodenal ulcer among the Africans. Gatumbi and Roy (1970) in a prospective study among the rural Kenyan Africans observed a prevalence of 13.7%.

It seems therefore that what happened in the Western societies (United States of America and Western Europe) at the turn of the century is now happening in the developing countries like Kenya and that duodenal ulcer is on the increase in the black population of Kenya. In 1967, Susser suggested that urbanization was one of the causes of duodenal ulcer and as the rural population was urbanised the incidence of duodenal ulcer increased. When the population acclimatized to the urban life the incidence of duodenal ulcer reduced. Cleave (1962) attributed the rise of duodenal ulcer solely to refined food low in protein and argued that incidence of duodenal ulcer is low in rural communities eating unrefined foods with relatively high protein content. When the rural communities start eating refined food low in protein, the incidence of duodenal ulcer in these communities rises.

Clinical presentation of duodenal ulcer

Duodenal ulcer first appear in significant numbers in patients in their early twenties. The incidence of the disease then increases in frequency and reaches a peak incidence and prevalence in the age group 40 - 50 years. Occasionally cases of duodenal ulcer are reported below 20 years. Also rarely does a duodenal ulcer start after the age of sixty years (Walker 1973). The male to female ratio at the age group 20 - 30 years is 4:1 and in the age group 30 - 50 the male to female ratio is 3:1 (Fry 1964).

Uncomplicated duodenal ulcer is characterised by fluctuating pain deep to the abdominal wall, poorly localised in the epigastrium. The pain may be burning, boring, aching, gnawing or just a vague epigastric discomfort. It begins one to three hours after a meal and it is relieved by eating food,(especially milk or other protein rich food) taking antacids or vomiting. The pain is not present upon waking up in the morning or after a long fast. Pain within one to two hours after a meal is common in both duodenal ulcer and gastric ulcer. However, pain within the first thirty minutes after a meal occur in 20% of gastric ulcer patients. Only very rarely does a duodenal ulcer produce pain within thirty minutes after a meal. This is because acid produced in the body

of the stomach bathes the ulcer in the body of the stomach earlier than the one in the duodenum. The pain of duodenal ulcer occur in bouts followed by periods of remission. Each bout of attack of pain last from one to eight weeks and the period of remission may be upto three months only to be followed by another bout of pain. Vomiting occur infrequently in patients with uncomplicated duodenal ulcer (Friedman 1948, Radloff 1947). When it does occur it is in those who learn that by vomiting the epigastric pain is relieved. Weight loss occurs in 50% of patients with duodenal ulcer and this is usually not marked (Smith, Boles and Jordan 1950). On examining the patient epigastric tenderness is elicited. The tenderness is modest, not rebound and without rigidity.

Diagnosis of duodenal ulcer

The diagnosis of duodenal ulcer is based on history and physical findings described above and radiological and endoscopic findings.

Barium meal is the most important investigation and its accuracy ranges between 85 - 90 per cent. (Belbe 1971) A niche in the duodenal bulb confirms the diagnosis of active duodenal ulcer and a deformed duodenal bulb is pathgnomonic of previous duodenal ulcer disease but does not necessarily indicate an active ulcer is present.

Whenever possible duodenoscopy should be done particularly among patients with symptoms of duodenal ulcer but have negative or inconclusive radiological findings.

The duodenal bulb is entered and inspected. On inspection of the duodenal bulb an active ulcer or deformity may be visualised.

Treatment of duodenal ulcer

This is both medical and surgical. Medical therapy for duodenal ulcer is directed at relieving symptoms, hastening healing and decreasing the frequency of relapse, surgical treatment at present has relatively low mortality and morbidity and effect a cure in 80 - 90 per cent of duodenal ulcer patients. Nevertheless, this is achieved at the expense of a significant side effects in a troublesome minority of patients and a recurrence rate of 5 - 15 per cent of the operated cases (Walker 1973).

Medical treatment

1. Drugs, Alcohol, smoking and diet

Patients are advised to avoid noxious agents such as aspirin and alcohol. Aspirin damages gastric mucosa and other analgesics should be given when analgesia is necessary. Alcohol similarly produces gastritis and stimulates gastric acid secretion without buffering it, but alcohol has never been shown to cause duodenal ulcer (Needham, Kyle, James 1971). Caffeine like alcohol stimulates gastric acid secretions and therefore strong coffee and tea should be avoided. Reserpine, phenylbutazone and Glucocorticoids perpetuate duodenal ulceration and should not be prescribed to duodenal ulcer patients (Menguy and Max 1970). Smoking is

associated with delay in healing of gastro-duodenal ulcer and therefore the patient should be advised to stop it altogether. The patient is advised to take frequent meals. This lowers the stomach acidity and relieves pain.

2. Antiacids

Neutralization of the stomach acid would best be achieved by giving hourly antiacids but this is both impractical and inconvenient to the patient. Antacid given one hour after a meal will start to neutralize the acid at a time when acid secretion is at its maximum and this will lower the gastric P^H achieved after a meal. A second dose given three hours after a meal lowers the gastric acidity until the time for the next meal. The larger the dose of antiacid taken the longer the period of reduced acidity (Fordhan and Collins 1966). Most readily available antacids contain a mixture of aluminium and magnesium salts. Given in large doses, magnesium preparations are complicated by diarrhoea while aluminum hydroxide causes constipation.

3. Histamine $^H2$ - Receptor antagonists

This group of drugs inhibit gastric acid secretion and enhance healing of duodenal ulcer. Cimetidine is the commonly used preparation. When it is given for four to six weeks about 75 to 90 per cent of the duodenal ulcers heal but recurrence of the ulcer is

observed in 75 per cent of the patients within the next six months. The recurrence rate can be reduced by giving a maintenance dose of 400 mg twice daily. Sometimes, cimetidine binds at other sites in the body producing important side effects. The best known of these are:-

(i) In the liver cimetidine binds cytochrome enzymes responsible for inactivation of drugs like propranalol, diazepam and warfarin. Inhibition of these enzymes result in prolonged action by these drugs.

(ii) It binds androgen receptor occasionally producing gnaecomastia and impotence in the male.

(iii) In the brain, and particularly among the elderly, it occasionally binds neurons producing mental confusion (Russell and Lopez 1980).

Other H_2-receptor antagonists are metianide and Ranitidine (Zantac, Glaxo). Studies on Ranitidine done in United Kingdom, Australia, South Africa, Italy and Poland have shown duodenal ulcer healed in 71 to 87 per cent of cases following 150mg twice daily treatment for four weeks. Ranitidine has been found to heal duodenal ulcers which have resisted Cimitidine (Black et al, 1972).

4. Carbenoxolone Sodium

This has anabolic effect and produces protection of the gastric mucosa. It is prepared in capsule form for release in the duodenum. It has been shown to produce healing of duodenal ulcer when given for a period of 8 to 12 weeks. Side effects include salt and water retention, occasional hypokalaemia and raised blood pressure. Therefore it is limited to patients with no cardio-pulmonary, liver or renal diseases.

Surgical Treatment

The commonest indication for surgery in duodenal ulcer is intractability. This is defined as persistent symptoms inspite of adequate medical therapy. Other indications for surgery in duodenal ulcer include development of complications such as uncontrolled haemorrhage, acute perforation and development of pyloric stenosis. The operations carried out for duodenal ulcer are:

(i) Drainage procedure (pyloroplasty or gastrojejunostomy).

(ii) Partial gastrectomy.

(iii) Vagotomy.

In the beginning of this century when duodenal ulcer was becoming more prevalent in western countries simple gastrojejunostomy used to be done alone. The aim was to divert the irritating gastric juice from ulcerated duodenum. This procedure was accompanied by up to 50 per cent recurrence rate and has been abandoned.

Partial gastrectomy.

In this operation the distal two thirds of the stomach is excised. This reduces gastric acid secretion by reducing the parietal cell mass and by excising the gastrin producing cells of pyloric antrum. After excision of the distal two thirds of the stomach, the remnant is anastomosed to the duodenum (Billroth I operation) or the stomach remnant is anastomosed to the jejunum (Billroth II operation). In Billroth II operation retro-colic anastomosis is done and the defect formed in the transverse meso-colon closed after the anastomosis. This prevents herniation of small gut through the opening.

Vagotomy

Afferent stimuli of stress or food are received in the higher centres of brain and these are transmitted along the vagus resulting in increased gastric acid secretion. If the vagus is divided, this gastric acid secretion mediated by external stimuli is abolished. Division of the vagus is accompanied by decreased gastric motility.

Three types of vagotomy may be done. They are truncal, selective and proximal gastric vagotomy.

Truncal vagotomy is the simplest of the three. Both trunks of the vagus lying anterior and posterior to the abdominal oesophagus are divided between ligatures to avoid regeneration. In selective vagotomy the hepatic branch

of the anterior trunk is identified and division of the nerve is done distal to this branch. For the posterior trunk the coelic branch is identified and division of the trunk done distal to it.

Proximal gastric vagotomy (highly selective vagotomy) consists of excising the vagal branches innervating the cardia, the fundus and body of the stomach. The branches innervating the antrum are spared. Following highly selective vagotomy gastric peristalsis and emptying is preserved and it is not necessary to do an additional drainage procedure.

If truncal vagotomy and selective vagotomy are done a drainage procedure is necessary since gastric emptying is impaired. The drainage procedures done are gastrojejunostomy and pyloroplasty. Gastrojejunostomy is frequently done since it is reversible and can be done easily where there is excessive duodenal scarring. The gastrojejunostomy done is retro-colic and the window made in the transverse mesocolon is closed.

Pyloroplasty is considered most physiological than gastrojeunostomy since no blind loop is left behind (Hoerr 1971 But is is not reversible and can be difficult to perform if there is marked fibrosis in the duodenum. The commonly done pyloroplasty is Heinek-Mikulicz pyloroplasty.

It consists of a longitudinal incision extending 3.5 cm. into the stomach and 2.5 cm. in the duodenum and the incision is closed transversely in a single layer of interrupted stitches. If two layers are used the lumen achieved is too narrow and not satisfactory (Weinberg 1956).

COMPLICATIONS

Bleeding

This occurs in approximately 25% of the patients with duodenal ulcer. Ivy (1974), Stolle (1944) in Amsterdam found that the incidence of bleeding increased steadily with the duration of the ulcer symptoms. It was 37 per cent in those who had the ulcer for 5 years increasing steadily to 86 per cent in those who had the ulcer for 30 years Chinn (1956) determined the risk of subsequent bleeding in patients with duodenal ulcer. He found that subsequent bleeding increased gradually from about 23 per cent after 3 years of ulcer symptoms to 48 per cent after 10 years of ulcer symptoms. The chance of having a second bleeding was not affected by age but bleeding is more often lethal in the aged than in the young. Bleeding duodenal ulcer frequently occur among patients of blood group O (Horwich, Evans, McConnel 1966).

Acute perforation

This occurs when the ulcer extend through the wall of the duodenum very rapidly and there is no time for adhesions to develop to seal the ulcer to surrounding structures. Duodenal contents leak into the general peritoneal cavity and there is a sudden severe abdominal pain with abdominal rigidity and pneumoperitoneum. The perforation usually occurs anteriorly since posteriorly the ulcer is protected by attachment of the pancreas.

The reported prevalence of acute perforation varies between 5 and 10 per cent in patients with duodenal ulcer (Hanbrick 1974) and 7.5 per cent of patients with acute free perforation have not had a previous history suggestive of duodenal ulcer (Tolley 1967). After simple closure of the acute perforation 70 per cent of the patients have further trouble of ulcer symptoms later (Jordan, DeBakey, Dunkan, 1974). It is advisable to carry out a definitive therapeutic procedure in those patients who have acute perforation as an elective operation at a later date (Hallenbeck 1976).

Intractability

This is the most frequent indication of surgical treatment in duodenal ulcer. It is defined as failure of symptoms to be controlled to a degree that permits a satisfactory life despite adequate medical treatment. The ulcer pain looses periodicity, there are no remissions and the patients life revolves about a continuous ulcer activity. The designation intractability is subjective

and those patients who are unwilling to get involved in their work and other activities appear to have intractable symptoms. These include patients with depression, anxiety neuroses and alcoholics. When these are operated upon the results are very poor (Jorgensen, Christen, Fischer 1971).

Pyloric Obstruction

Gastric outlet obstruction occuring during the course of duodenal ulcer diathesis may be due to either oedema or cicatricial obliteration of the duodenal lumen. In the former the patient usually has a good deal of pain from concommitant active ulcer while in the latter, pain is often absent. When obstruction is due to oedema operation is avoided whenever possible.

Chronic ulcer is characterized by its chronicity associated with period of excercebations followed by periods of remissions. Over a duration of years, with repeated episodes of duodenal ulceration, and healing duodenal bulb become relatively scarred and stenotic with increasing gastric outlet obstruction. Long standing pyloric stenosis leads to gastric dilatation and hypertrophy. First there is sufficient residue in the stomach for the gastric splash to be elicited. As the stomach hypertrophies visible peristalsis becomes obvious. Finally the thickened stomach full of inssipated content becomes

palpable. Due to the obstruction the stomach produces giant peristaltic waves in an attempt to overcome the obstruction. When this is continued for a long time there is decompensation due to gastric muscular failure and the stomach becomes atonic with marked dilatation and gastric retention. The actual cause of the gastric muscular failure is not clear but it is postulated to be due to decreased intracellular potassium which occur as a result of vomiting (Kreel and Ellis 1965). In this way the gastric muscular failure may be similar to cardiac muscular failure in stenotic valvular heart disease. The gastric failure is reversible and when the stomach is rested by gastric suction and electrolyte deficit replacement the tone of the stomach is regained. The gastric dilatation subsides and peristalsis is regained.

Presentation of pyloric obstruction

The reported incidence of pyloric stenosis is 10 to 23 per cent of hospitalised cases with duodenal ulcer (Dwoken and Roth 1962). The wide range in frequency is attributable to differences in criterion for diagnosis. In most reported series patients with pyloric stenosis are aged over 50 years and have a long history of ulcer symptoms. Their distress differs from that of usual pain of duodenal ulcer in that the pain is not relieved by ingestion of antiacids. Vomiting generally relieves

the discomfort and occurs once or twice daily. The vomiting is voluminous and may be projectile and often contain food eaten 12 to 24 hours earlier. Some patients manage to avoid vomiting by decreasing food intake, this leading to weight loss.

Pallor and anaemia occur in about 20 per cent of the patients with pyloric stenosis. When encountered it is usually the result of associated bleeding from the ulcer (Kreel 1965). At the time of admission about 10 per cent of the cases are found to be dehydrated particularly in those with a long history of vomiting (Howe 1964). Consequently in such cases the levels of haemoglobin are usually high due to hemo concentration. Dehydrated patients have marked electrolyte imbalance,elevated blood urea and may have mental confusion.

Gamble and Ross, in 1925, first demonstrated the metabolic changes produced by pyloric stenosis. They showed that serum chloride is markedly lowered and serum sodium and potassium are moderately lowered. Kennedy and Dunning (1949) showed that the p^H of body fluids is elevated and renal function is impaired. Howe and Leguine (1964) also analysed the metabolic disturbances of pyloric stenosis. They found that body fluid p^H was always elevated above 7.5 and serum sodium was usually within normal limits of 135 - 145 milli-equivalents per litre.

The serum potassium was usually below normal limits, that is under 3.5 milli-equivalents per litre. Blood urea was frequently elevated above 50 milli equivalents per litre. They also observed that the higher the blood urea the greater the initial deficit of plasma volume due to vomiting. In their analysis serum chloride was always below normal limits of 95 milli equivalents per litre.

On analysing the body deficit of these electrolytes at the time of admission, Howe and Lequene (1964) made a number of observations following correction of water and electrolytes deficits and after estimating the amount of ions lost in urine and gastric aspirate. They found that the greatest deficit was that of chloride, with a mean deficit of 820 milli equivalent. This was followed by sodium with a deficit of 465 milli equivalent. The potassium deficit was much smaller with a mean of 72.5 milli equivalents. By analysing the electrolytes lost in gastric aspirate and in urine they found that the gastric aspirate contained a high content of chloride in excess of the sum of sodium and potassium. The loss of chloride was accompanied by a proportionate loss of hydrogen ions and an accompanying rise in serum bicarbonate. As the chloride was lost in the vomit there was a diminished

amount of urine chloride until when the serum chloride was below 95 milli equivalent per litre no chloride was lost in urine. They showed that although serum sodium is usually within normal limits a large sodium deficit develops:- Sodium being lost both in urine and in the vomit. As the sodium is lost water is lost with it and plasma volume falls. In severe cases peripheral circulatory failure occurs which can be life threatening.

The potassium is lost mainly in urine and also in the vomit. In pyloric stenosis there is a heavy loss of potassium in urine but this is replaced by potassium from tissue breakdown produced by wasting seen in pyloric stenosis.

Diagnosis of pyloric stenosis due to duodenal ulcer

The diagnosis is made from history, physical findings and radiological findings. Endoscopic findings supplement the radiological findings. The patient gives a classical history of duodenal ulcer symptoms dating for a long time back. The patient indicates that recently the epigastric pain has been replaced by a constant discomfort which is relieved by vomiting. The most distressing symptom of pyloric stenosis is vomiting which is comparatively recent in onset. The vomiting occur once or twice daily, is copious, containing food eaten several hours back.

On examination dehydration is found in 10 to 30 per cent of the patients, pallor in 20 per cent of the patients and succussion splash is positive in 64 per cent of the patients(Kozzol and Meyer 1964). Visible peristalsis and mass in the epigastrium are occasionally detected.

After admission stool is taken for occult blood and parasitic ova. Kaustam, in 1970, working in Nigeria reported a close association between pyloric stenosis due to duodenal ulcer and hookworm infestation. Positive occult blood in stool may be due to an active bleeding duodenal ulcer. Blood is taken for haemoglobin concentration assessment and serum for urea and electrolytes. If in a remote hospital with no radiological facilities the saline load test can be done to verify gastric retention. The test was described by Goldstein and Boyle in 1965. The stomach is emptied and a load of 750 ml of normal saline is placed in the stomach via a nasogastric tube. Thirty minutes later the saline is aspirated from the stomach. Recovery of more than 400 ml of saline indicates gastric obstruction. This test is positive in 90 per cent of cases with pyloric stenosis confirmed at operation.

The patient is started on intravenous fluids and nasogastric suction. The nasogastric suction is continued for 72 hours. The intravenous fluids are replaced with normal saline and when the patient is passing adequate

amount of urine, potassium chloride is added to the infusion at a rate of 10 milli equivalents per hour. Until the hypokalaemia has been corrected then the patient is maintained on 40 to 80 milli equivalents to replace the loses by nasogastric suction and in urine.

After 72 hours of nasogastric suction, if the obstruction was due to oedema this will have subsided and there will be no gastric retention at barium meal. The patient is started on oral fluids then soft diet and slowly graduated to a normal diet. If there is obstruction due to fibrosis, barium meal will still show obstruction after 72 hours of nasogastric suction and this patient will need surgery.

Radiological features of pyloric stenosis

When a plain film of the abdomen is taken it shows a large gastric shadow with retained food particles producing partcy translucencies. The erect film show a large high gastric fluid level. At barium meal several signs of obstruction are evident. They include:-

1. Presence of excessive fasting gastric juice. This is immediately apparent on the first mouthful of barium reaching the stomach. Instead of the bolus descending along the lesser curve and parting the opposed gastric walls the bolus of barium sink through a layer of fluid and come to rest at the bottom of the greater curve giving a sourcer like appearance.

2. Before gastric failure with resultant gatric atony develops ingestion of the barium is followed by two to three giant peristaltic waves which indent both the greater and lesser curves simultaneously. These waves identify the active stomach of pyloric obstruction from the atomic decompensated stomach which requires a period of rest (by gastric suction) and electrolyte replacement before peristalsis can be resumed.
3. A 24 hour residue barium is diagnostic of pyloric stenosis but this is not necessary. A six hour film using barium sulphate as a contrast medium normally show less than 25% retention whereas in pyloric stenosis there is more than 50 per cent retention of barium.
4. The duodenal ulcer (which is usually active in pyloric stenosis) is seen as an irregular diverticulum which remain constant in size and position and most frequently in the postero-superior aspect of the duodenum.

The barium meal features definitely exclude a carcinoma of the stomach if this happens to be the cause of the pyloric obstruction.

Duodenoscope

This fibreoptic instrument is passed into the stomach and the duodenum. The stomach is examined to rule out a carcinoma of the pyloric antrum as a cause of the obstruction or any other tumour of the stomach that might accompany the pyloric stenosis. The duodenum is then

entered and the fibrosed ulcer is examined.

In this study an attempt was made to evaluate the incidence of complication of duodenal ulcer disease among patients seen at Kenyatta National Hospital with particular emphasis on pyloric stenosis. The clinical features, laboratory and radiologic findings, treatment and complications of surgery among patients with pyloric stenosis were analysed. It was also the purpose of this study to compare the findings at Kenyatta National Hospital with those reported elsewhere and to summarise the present knowledge of duodenal ulcer disease with particular emphasis on recent developments.

PATIENTS AND METHODS

The clinical notes of cases of proved duodenal ulceration treated at Kenyatta National Hospital between 1970 - 1980 inclusive were reviewed. Attention was paid to the presenting features, methods of investigation, mode of treatment and complications of pyloric stenosis.

This information extracted was recorded on a special form (Appendix I). The cases with clinical features of duodenal ulcer disease but had no radiologic, endoscopic or operative confirmation of the disease were excluded from this study. The findings are presented.

RESULTS

Over the eleven year period a total of 511 cases had evidence of duodenal ulcer disease. Two hundred and twenty (42.6%) of them presented with no major complication. Nine patients (1.7%) presented with stomal ulcer. Of The remainder, one hundred and thirty five patients (22.3%) had bleeding duodenal ulcer, seventy eight patients (15.3%) had pyloric stenosis, and sixty nine (13.5%) had perforated duodenal ulcer (Table 1).

TABLE 1 PREVALENCE OF DIFFERENT COMPLICATIONS OF DUODENAL ULCER

STATE OF DUODENAL ULCER	NUMBER OF CASES	PERCENTAGE
Uncomplicated duodenal ulcer	220	42.6%
Pyloric stenosis	78	15.3%
Bleeding duodenal ulcer	135	22.3%
Perforated duodenal ulcer	69	13.5%
Stomal ulcer	9	1.7%
TOTAL	511	100%

AGE, SEX AND TOTAL DISTRIBUTION

There were 63 males and 15 females presenting with pyloric stenosis (male:female ratio = approx. 4:1). The ages ranged between 15 years and 64 years with an average of approximately 40 years. The peak was in the 20 - 29 year old age group (Table 2). All females with pyloric stenosis were aged above 39 years (Table 2).

TABLE 2 PREVALENCE OF PYLORIC STENOSIS DISTRIBUTED ACCORDING TO AGE AND SEX

AGE IN YEARS	NUMBER OF CASES		TOTAL	PERCENTAGE
	Male	Female		
10 - 19	6	-	6	7.7%
20 - 29	21	-	21	25.6%
30 - 39	12	-	12	15.3%
40 - 49	6	6	12	15.3%
50 - 59	12	6	18	23.1%
60 and above	6	3	9	10.3%
TOTAL	63 (80.8%)	15 (19.2%)	78	100%

Table 3 shows the tribal distribution. Slightly over fifty per cent of patients belonged to Kikuyu tribe. The second commonest tribe was Kisii (11.7%).

TABLE 3 PREVALENCE OF PYLORIC STENOSIS IN DIFFERENT TRIBAL GROUPS

TRIBAL GROUP	NUMBER OF PATIENTS	PERCENTAGE OF TOTAL
Kikuyu	42	53.9%
Kisii	9	11.7%
Kamba	6	7.9%
Luhya	6	7.9%
Kalenjin	6	7.9%
Luo	3	3.9%
Meru	3	3.9%
Somali	3	3.9%
TOTAL	78	100%

All the seventy eight patients gave a history of epigastric pain or discomfort and had vomiting. Only nine patients complained of weight loss, seven of them males (Table 4).

TABLE 4 PRESENTING SYMPTOMS

SYMPTOM	NUMBER OF PATIENTS		TOTAL	PERCENTAGE
	MALES	FEMALES		
Pain	63	15	78	100%
Vomiting	63	15	78	100%
Weight loss	7	2	9	11.6%

DURATION OF SYMPTOMS (TABLES 5 and 6)

Forty five per cent of patients presented with duration of epigastric pain or discomfort of under ten years, the majority having had pain for 5 to 9 years. The duration of vomiting was under four weeks in 51 patients (65%) table 6. Nearly one in five cases had been vomiting for eleven or more weeks.

TABLE 5 DURATION OF EPIGASTRIC PAIN RELATED TO PREVALENCE OF PYLORIC STENOSIS

DURATION IN YEARS	NUMBER OF PATIENTS	PERCENTAGE
0 - 4	12	15.4%
5 - 9	33	39.9%
10 - 14	12	15.4%
15 - 19	12	15.9%
20 and above	18	23.0%
TOTAL	78	100%

TABLE 6 DURATION OF VOMITING

DURATION IN WEEKS	NUMBER OF PATIENTS	PERCENTAGE
0 - 2	6	7.9%
3 - 4	45	57.1%
5 - 6	6	7.9%
7 - 8	6	7.9%
9 - 10	0	0
11 and above	15	19.2%
TOTAL	78	100%

The presenting physical findings were dehydration, pallor and positive succussion splash.

Thirty patients (38.5%) were dehydrated, succussion splash was positive in fifty one patients (65.4%), fifty seven patients (73.1%) had haemoglobin above 10 gm% and twenty one patients (26.1%) had haemoglobin concentration below 10 gm%.

RESULT OF INVESTIGATIONS

The serum urea and electrolytes of most of the patients was obtained at admission. The results of serum electrolytes analysis are shown in table 7. Forty eight patients had their serum sodium, potassium and bicarbonate assessed soon after admission. Only 42 patients had their chloride assessed soon after admission. The electrolytes recorded are before the electrolyte replacement was started on the patient.

Blood urea nitrogen (BUN) was obtained in forty eight patients. Twenty one had blood urea nitrogen above normal (6.7 milli moles per litre), eighteen patients had blood urea nitrogen within normal limits (2.5 to 6.7 milli moles per litre) and nine patients had blood urea nitrogen below normal.

TABLE 7 SERUM ELECTROLYTE LEVELS IN MILLI EQUIVALENTS PER LITRE

Electrolyte	Number of patients with level below normal	Number of patients with levels within normal range	Number of patients with levels above normal	Total number of patients assessed for each electrolyte
Sodium	(under 135) 10	(135-145) 38	(above 145) 0	48
Potassium	(under 3.5) 15	(3.5-5.0) 33	(above 5.0) 0	48
Chloride	(under 95) 36	(95-105) 6	(above 105) 0	42
Bicarbonate	(under 24) 12	(24-32) 24	(above 32) 12	48

Of the seventy eight patients recorded, thirty four (43.6%) had been admitted before with pyloric stenosis, underwent medical treatment, became better and discharged. They were re-admitted again after a period ranging from one month to two years with pyloric stenosis and this occasion they had surgical treatment. Fourty four patients (56.4%) did not respond to medical treatment on their first admission and they underwent surgical treatment.

All the 78 patients had truncal vagotomy done with an accompanying drainage procedure (either gastro-jejunostomy or pyloroplasty). The drainage procedure done most frequently was gastrojejunostomy and this was done in 75 patients. Only three patients had pyloroplasty. The result of surgery in these three patients were just as good as those who had gastrojejunostomy. One of the three had features of early dumping syndrome which subsided after 6 months of follow up. The other two reported complete satisfaction with this operation two months after surgery.

RESULTS OF TREATMENT AND COMPLAINTS

One patient died within ten days after operation, a mortality of 1.3 per cent. Seventy seven patients were discharged within ten days after operation. Their complaints at 3 months, six months and one year were recorded (Table 8).

It is noted that the number of patients with the gastro-intestinal symptoms after operation increased with passage of time until after one year when 66 per cent of the patients had no gastrointestinal symptoms.

TABLE 8 RESULTS OF OPERATION

RESULTS	AT THREE MONTHS	AT SIX MONTHS	AT O YEA
(A) No gastro intestinal symptoms			
(B) Minor gastrointestinal symptoms. No evidence of recurrence. Patient satisfied with results and is pursuing his/her occupation.			
(C) The patient is dissatisfied with function of his G.I.T. but there is no evidence of recurrent ulcer			
(D) A proved or probable ulcer has recurred or the patient is disabled from other G.I.T. symptom			
(E) Not classifiable	10 (13%)	5 (7%)	(5%
TOTALS	77 (100%)	77 (100%)	7 (1(

Table 9 indicates the complication of surgery from the symptoms described by the patients one year after surgery. Fifty five (71.4%) had no complication of surgery noted one year after operation. Nine (11.8%) reported billous vomiting one year after surgery.

TABLE 9 COMPLICATIONS OF SURGERY

COMPLICATION	NUMBER OF PATIENTS	PERCENTAGE
Early dumping	9	11.2%
Late dumping	4	5.2%
Billous vomiting	9	11.2%
None significant	55	71.4%
TOTALS	77	100%

DISCUSSION

Dworken and Roth (1962) reviewed 3515 cases of duodenal ulcer seen at Cleveland Hospital over the period 1951-1961 inclusive. Out of these patients 186 (7%) developed pyloric stenosis. In their study two thirds of patients with pyloric stenosis were over fifty years of age and male to female ratio was 3:1. Moody and Connel (1962) obtained a male to female sex ratio of 3.5:1. The present study has revealed that at Kenyatta National Hospital the males outnumber the female in a ratio of 4:1. In the study done here the total number of patients diagnosed positively as duodenal ulcer in the period 1970 to 1980 was 511 cases. Out of these 78 (15.3%) developed pyloric stenosis. This is twice the incidence recorded by Dworken and Roth in 1962. In their study 66 per cent of the patients with pyloric stenosis were over fifty years of age while in this study only 33% of the patients were over fifty years of age.

In the present series the peak incidence of pyloric stenosis was in the age group 20 to 29 years accounting for 26.6 per cent of the cases. This is a relatively young age group and may be due to the short period between developing duodenal ulcer symptoms and development of pyloric stenosis as seen in Table 5 in which it is noted that 45

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per cent of the patients had had their symptoms for less than ten years before they developed pyloric stenosis. The average duration of symptoms in the Dworken study was 12 years. Hence it can be said from this study that Kenyan African patients develop duodenal ulcer at an early age group and develop pyloric stenosis after a shorter duration of symptoms.

From this study it emerged that the majority of the patients with pyloric stenosis due to duodenal ulcer are Kikuyu who accounted for 53.9 per cent (Table 3). This may partly be due to the fact that Nairobi borders Kikuyu land and the patients may be coming from the neighbouring districts to seek medical treatment at Kenyatta National Hospital. However, Nairobi city has a multi-tribal society and the Luo Community who are quite numerous in Nairobi and come second to the Kikuyu according to the 1979 National Census accounted for only 3.9 per cent of the patien The Luhya who border the Luo in the Western Kenya accounted for only 7.9 per cent of the cases. There may be something in the way of life of the Kikuyu and possibly the Kisii which make them prone to developing pyloric stenosis. Both the Kisii and the Kikuyu are bantus and have the banana (matoke) as a staple food. Possibly there is something in the 'matoke' which predisposes to

development of pyloric stenosis in duodenal ulcer patients.

Weight loss was not frequently reported by patients studied in this series. Only nine patients (11.6%) reported noticing that they have lost weight. So, if a patient presents with features of pyloric obstruction and gross weight loss, gastric malignancy in the pyloric atrium should be suspected.

Vomiting is the most important symptom in patients with pyloric stenosis. All the patients in the present series had had a period of vomiting. In the Kreel (1962) study vomiting was reported in 81.4% of the cases and in Kozzo and Meyer study (1964) it was reported in 90% of the cases. Moody and Connel (1962) reported vomiting in 80% of their cases and described it to be projectile containing food which was eaten 12 to 24 hours earlier. In this study 35 per cent of the patients presented to hospital after they had been vomiting for more than four weeks.

Vomiting leads to dehydration and electrolyte imbalance Dehydration was seen in 38.5 per cent of the patients in this series on admission. Moody and Connel (1962) found 28.5 per cent of their patients dehydrated while Kozzol and Meyer (1964) found dehydration in only 10 per cent of their patients. These figures are lower than that obtained here. This comparatively high rate of dehydration at Kenyatta National Hospital is most likely the result of delay in reporting to hospital in a significant number of cases. Succussion splash was elicited in 65 per cent

of our cases and this was in agreement with findings by Kozzol and Meyer (1964) and Dworken and Roth (1962) who respectively reported positivity of 64 per cent and 60 per cent.

Anaemia is a rare feature of pyloric stenosis and was present in 26.1 per cent of the cases under review. In Moody and Connel's series (1962) only 20 per cent of their patients had haemoglobin concentration less than 10 gm%. Kreel and Ellis (1965) also found 20 per cent of their patients anaemic.

In the present study majority of patients'serum potassium and sodium levels were within normal range. By contrast in 85.7 per cent of cases serum chloride levels had dropped below normal at the time of admission.

Elevated serum urea occurs as a result of reduced glomenular filtration rate due to hypovolaemia which occurs as a result of loss of sodium in the renal tubules. Sodium is lost in urine accompanying bicarbonate ions. Bicarbonate ions are produced in excess in gastric parietal cells as chloride ions are lost in the vomitus. In this way excess of bicarbonate reaches the renal glomerulus where it is filtered. Sodium accompany it to maintain electrical neutrality of urine. Water is lost with the sodium ions. Hypovolaemia so produced activate the renin-angiotensin system and aldoterone is secreted by the adrenal glands leading to renal conservation of sodium. Hydrogen ions and potassium are as a result, exchanged for sodium in the distal convoluted tubules of the nephron and the

patient, although alkalotic secretes an acid urine (Burrows and Commons 1950).

The management of pyloric stenosis consists of medical treatment lasting 72 hours and those who do not respond to medical treatment surgery is recommended. The medical treatment consist of fluid and electrolyte replacement. Fifteen per cent of those who respond well to medical treatment will have recurrence of obstruction within a year and another ten per cent will have a recurrence of obstruction within the next eight years (Goldstein and Boyle 1968). In this study 43.6% of the patients had had medical treatment before being re-admitted with recurrence of obstruction. Medical treatment succeeds in relieving gastric retention in those with obstruction due to oedema of the ulcerating duodenum. It rarely succeeds in those with marked fibrosis of the ulcerated area.

However it is necessary for all patients with pyloric stenosis to undergo 72 hours of continuous nasogastric suction and replacement of fluids and electrolytes. The nasogastric suction decompresses the stomach and restores peristalsis. It also relieves gastric oedema and makes gastric wall suitable for surgery. When the patient is severely wasted on admission, he has a contracted volume which may not appear so because of their present low weight. The haematocrit is also normal. These patients develop

severe hypotension during surgery or in the immediate post-operative period and it is advisable to tranfuse 1000 ml. of whole blood over 24 hours just before surgery(Goldstein and Boyle 1968). With adequate preparation for surgery outlined above Goldstein, Janin, Schapiro and Boyle, in 1966 reported a mortality of 1.3 per cent while the mortality for those inadequately prepared was 19 per cent. Only one patient in this series died.

Surgical treatment of pyloric stenosis is that of the causative duodenal ulcer. In pyloric stenosis due to duodenal ulcer adequate drainage of the stomach is necessary and this is best achieved by gastrojejunostomy (Herman 1976). Davis and Williams in 1971 obtained equally good results with vagotomy and pyloroplasty. In the present series 75 patients had vagotomy and gastrojejunostomy and three patients had vagotomy and pyloroplasty. The result of operation were the same in the two operations. Pyloroplasty can be difficult to perform in a patient with pyloric stenosis due to duodenal ulcer because the pylorus is markedly fibrosed and dissection of the pylorus can be difficult and dangerous (Hoerr 1976).

COMPLICATIONS OF SURGERY

These can be grouped into early complications and late complications:-

Early complications are:-

(i) Haemorrhage from the anastomatic site.

(ii) Paralytic ileus.

(iii) Stomal obstruction.

(iv) Acute pancreatitis.

Haemorrhage from the anastomotic site is particularly likely to occur when Connel's suture is used during the anastomosis. The patient vomits blood and the blood pressure is noticed to be falling. Conservative treatment usually succeeds. The patient is transfused with blood and is given adrenaline solution 1 ml. of 1:1000 solution orally every hour. If bleeding gets severe, which is unusual unless the patient has a bleeding diathesis, re-exploration is done and the bleeding points coagulated.

Paralytic ileus results from handling of the gut during operation. The patient presents with abdominal distension and vomiting. It is particularly likely to be most marked if the patient's serum potassium is low. It is managed by nasogastric suction, intra-venous fluids and electrolyte replacement.

Stomal obstruction occur due to oedema of the gastric wall. It is particularly likely to occur if the patient has been inadequately prepared and the stomach wall is still oedematous during operation. The patient presents with vomiting and severe electrolyte imbalance can occur. Nasogastric suction is done and electrolyte imbalance corrected. This continued for 72 hours relieves the obstruction.

Acute pancreatitis result from trauma to the pancreas during surgery. The serum amylases are elevated above 1000 somogyi units. The serum calcium may be low and in severe cases the low calcium level produces electrocardiogic changes similar to those of myocardial infarction. Acute pancreatitis is managed by intravenous fluids and nasogastric suction. If hypocalaemia occurs calcium gluconate is given.

In this study none of the above early complications of surgery were recorded.

Late complications of surgery include:-

(i) Stomal ulcers.

(ii) Early and late dumping syndromes.

(iii) Post-vagotomy diarrhoea.

(iv) Jejuno-gastric intussusception.

(v) Post-operative alkaline reflux gastritis (bile gastritis).

Stomal ulcer

The incidence of stomal ulcer following vagotomy and drainage is 5% (ranging from 3 to 25 per cent in different studies), that following vagotomy and removal of the antrum is 1 per cent, following partial gastrectomy alone the incidence is 3 per cent (Stampen 1971). Selective vagotomy with drainage has had lower incidence of stomal ulcer than truncal vagotomy with drainage (Kennedy and Connel 1969). In 33% of patients done vagotomy the

Hollander test is positive but only a very low percentage of these develop stomal ulcer. Those with negative Hollander test never develop stomal ulcer (Cleator, 1975). Hence incomplete vagotomy is a major cause of stomal ulcer. Gastrin secreting tumour of the pancreas, duodenum or pyloric antrium is a more rare cause of stomal ulcer. These patients have a high serum gastrin (Kavlie and White 1972). The majority of stomal ulcers occur within five years of operation but they can occur many years after operation (Cleator 1974, Largache, 1965, Stammers, 1963).

In this series with patients followed for 1 year after operation stomal ulcer was not recorded.

Early dumping syndrome

The term 'early dumping' refers to the time after a meal and not to the interval after surgery. It occurs in most of the patients soon after surgery but persists in only 10 per cent of them after ten years of follow up. Early dumping was seen in 11.2 per cent of the patients in this series. Early dumping is defined as:-

(i) Distension and fullness due to mechanical effect of food in the stomach and intestine.

(ii) Dizziness, faiting, sweating and pallor due to hypovolaemia, secondary to ingestion of such hypertonic foods as thick soups, milk shakes and other drinks with sugar. These cause transfer of fluid from circulation into the intestine. The symptoms are due to transfer of extracellur fluid into the intestinal lumen and release of humoral factors

serotonin and brandykinins into the general circulation.

The main factor that play a role in the pathogenesis of early dumping are dilatation of the proxiaml intestine by bolus of chyme followed by release of vaso-active kinin and a sharp fluid shift of extracellur fluid into the jejunal lumen with subsequent changes in the circulating volume and serum potassium (Jessep 1968).

If time is allowed most of the patients recover on medical treatment alone. In this, the patient takes small, frequent, high protein, dry meals. Fluid and sugarly meals are avoided. Belladoma and Codein reduce intestinal activity. Brandykinin and serotinin blockers such as methylsergide maleate and periactin are also useful. Most of the patients respond to this treatment and report marked improvement with passage of time (Eldth, 1974).

If the patient has severe symptoms surgery is done with the aim of delaying gastric emptying and increase reservour capacity. In this a reversed segment of jejunum 10 cm. long is inserted 70 to 90 cm. beyond the ligament of Treutz (Keller, 1971, Leonard, 1967). A similar operation is done for post-vagotomy diarrhoea which is characterised by unpredictable urgency.

Late dumping syndrome.

This occurs in only five of the patients (Brown and Cleator 1965). In this series late dumping syndrome was

seen in 5.2 per cent of the cases one year after operation. It is characterised by symptoms similar to those of early dumping which occur 2 to 3 hours after a meal. These are due to hypoglycaemia subsequent to post-prandial hyperglycaemia with excess release of insulin. Part of the hypoglaycaemia is due to insulin releasing effect of gastric inhibitory peptide (G.I.P.) which is released by duodenum following introduction of chyme into the intestine (Brown and Cleator 1965).

Post Operative alkaline reflux gastritis (Bile gastritis)

The patients with this complication complain of unremitting burning epigastric pain, unrelieved by alkali, made worse by eating food and associated with considerable weight loss. The patient also complain of billous vomiting. The vomiting is not projectile or copious. The symptoms may start soon after surgery or many years later. Some get massive gastric bleeding with resultant haematemesis. Gastroscopy in these patients show considerable regurgitation from the stoma with fiery red oedema of the mucosa which bleed easily on touch.

When pain and billous vomiting is troublesome the patient is prepared for theatre. The procedure of choice is Roux-en-Y diversion of duodenal contents to a point 25 cm. distal to the gastro-jejuno anastomosis (Drapanos and Bethea 1974).

In the present series billous vomiting was noted in 11.2 per cent of the patients one year after the operation.

Jejuno-Gastric Intussusception

This is a rare complication and occur in those who have had vagotomy and gastrojejunostomy. It may occur early in the post operative period but is usually a late complication (Conchin and Markowtz 1965). It was not noted in any of the patients in the present series. There is an acute onset of severe epigastric pain with haematemesis. The main finding is a palpable mass in the epigastrium which can be shown by barium swallow to be stomach filled with jejunum. The patient is operated promptly to avoid gangrene of the jejunum. Most usually the distal loop has intussuscepted into the stomach. The intussuscepted loops are reduced. If they are viable side to side jejeno-jejunostomy is performed between the affarent and efferent loops to prevent recurrence of the intussusception. Gangrenous loops are resected and gastrojejunostomy re-constructed (White and Common 1976).

CONCLUSIONS

1. Duodenal ulcer at present is a common disease among the Kenyan Africans. It occurs at a younger age group as compared to the western countries and it is complicated by pyloric stenosis after a shorter duration of symptoms. The male to female ratio here is the same as in Western countries.
2. Pyloric stenosis, due to duodenal ulcer, from this study appear to be most frequent among the Kikuyu and the Kisii, but this needs to be pursued further with prospective studies among the different tribal groups in the country.
3. A large proportion of our patients wait for a long time after developing symmptoms of pyloric obstruction before they present in hospital and this lead to a high proportion of our patients being dehydrated at the time of admission.
4. Weight loss is not a common feature of the patients seen here with pyloric stenosis due to duodenal ulcer. It is suggested that if a patient with features of pyloric obstruction presents with marked weight loss, carcinoma of the pyloric antrum should be excluded first.

RECOMMENDATIONS

1. If a patient is admitted with pyloric stenosis due to duodenal ulcer, responds well to medical treatment, but barium meal show marked duodenal cicatricion it is advisable to call the patient back soon and have an elective operation done than to wait until he has another attack of pyloric obstruction.
2. Prevalence of duodenal ulcer and pyloric stenosis due to this disease among the different tribal groups in this country should be studied prospectively to confirm or refute the apparent high incidence of pyloric stenosis due to duodenal ulcer among the Kikuyu and the Kisii seen in this study. A study of the different eating habits of the tribes should be done to see if there is any food type which enhance development of duodenal ulcer and pyloric stenosis.

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APPENDIX I

NAME: _____ UNIT NUMBER: _____

AGE: _____ SEX: _____ TRIBE: _____

HISTORY OF ILLNESS

EPIGASTRIC PAIN: YES ☐ NO ☐

If yes duration _____

VOMITING: YES ☐ NO ☐

If yes duration _____

WEIGHT LOSS YES ☐ NO ☐

ADMITTED BEFORE WITH PYLORIC STENOSIS YES ☐ NO ☐

If yes how long ago _____

PHYSICAL FINDINGS

DEHYDRATION YES ☐ NO ☐

PALLOR YES ☐ NO ☐

SUCCUSSION SPLASH POSITIVE ☐ NEGATIVE ☐

INVESTIGATIONS

HAEMOGLOBIN CONCENTRATION _____ gm%

SERUM ELECTROLYTES CONCENTRATION IN MILLI EQUIVALENTS PER LITRE

NA^+ _____ K^+ _____ Cl^- _____

HCO_3^- _____

SERUM UREA (BUN) _____ MILLI MOLES PER LITRE

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.../cont.

RESULTS OF OPERATION (REFER TABLE 8 FOR THE CODE A,B,C,D,E).

AT 3 MONTHS	A	☐	B	☐	C	☐	D	☐	E	☐
AT 6 MONTHS	A	☐	B	☐	C	☐	D	☐	E	☐
AT 1 YEAR	A	☐	B	☐	C	☐	D	☐	E	☐

COMPLICATIONS OF OPERATION AT 1 YEAR

Early dumping Syndrome ☐

Late dumping Syndrome ☐

Billous vomiting ☐

None ☐