CARCINOMA OF THE PANCREAS:

A retrospective study of cases seen at Kenyatta National Hospital from January 1977 to December 1985.

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A thesis submitted in part fulfillment for the degree of Master of Medicine (Surgery) in the University of Nairobi 1988.
This thesis is my original work and has not been presented for a degree in any other University.

Signed

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CONTENTS

INTRODUCTION 1
LITERATURE REVIEW 2
HISTORICAL BACKGROUND 2
EPIDEMIOLOGY 3
PATHOLOGY 4
CLINICAL PRESENTATION 8
LABORATORY DIAGNOSIS 11
PANCREATIC BIOPSY TECHNIQUES 15
RADIOLOGICAL DIAGNOSIS 16
TREATMENT 22

MATERIALS AND METHODS 28

TABLES AND CHARTS NOS. 1 - 16

RESULTS 29
DISCUSSION 35
CONCLUSION 41

REFERENCES 43

APPENDIX I PROFORMA
INTRODUCTION:

Out of the various organs in the body the pancreas is still treated with "great respect" by surgeons. This is mainly due to the high morbidity and mortality resulting from diseases of the pancreas, the surgical complications after pancreatic surgery - Mainly pancreatitis and pancreatic fistula are the major causes of this "great respect" coupled with the very poor prognosis in Carcinoma of the exocrine pancreas.

It is thus not until 1935 that Whipple and associates performed the first successful pancreatecoduodenectomy for a carcinoma of the head of pancreas, that other surgeons also started performing various surgical procedures on the pancreas, but even then majority of pancreatic related surgery was palliative bypass surgery.

In Kenya no study has been done to document the condition as it presents locally. It is the aim of this study to have a retrospective review of cases seen at the Kenyatta National Hospital over a Nine year period and study the epidemiology, Clinical features, Methods of diagnosis and management of pancreatic Exocrine, cancer.
LITERATURE REVIEW:

HISTORICAL BACKGROUND:

The earliest recognition of cancer of the pancreas has been attributed to Morgagni who mentioned 5 cases believed to have been cancer of pancreas in 1679. A proper description was later given by Mondière 1836.

The first successful resection for a periampullary tumour in the United States literature was in 1898 by Halstead where the patient died 7 months later of a recurrence.

Whipple and Colleagues in 1935 described the first pancreaticoduodenectomy which they performed in two stages, a Biliary bypass followed later by resection of the tumour.

Trimble and associates (1941) modified the operation and performed it in one stage and thereafter pancreaticoduodenectomy became the operation Choice in patients with operable carcinoma of head of pancreas distal bile duct, ampulla and duodenum.

Crile (1970) advocated abandonment of the Whipple procedure due to the high operative mortality with no significant long term survival compared to palliative biliodigestive Bypass operation.
Traverso and Longmire (1978) introduced pylorus preserving pancreatico - duodenectomy in attempt to preserve the physiological function of the stomach and reduce the risk of ulcerations.

EPIDEMIOLOGY:

In the United states, Carcinoma of the pancreas is the 4th commonest cause of death from cancer (Silverberg 1986). It is reported to be increasing; but this could be due to refinement in the diagnostic methods and increased awareness among patients and physicians (Levin 1981). The incidence increases with increasing age with a peak incidence in the 6th and 7th decades of life (Cubilla 1978). Sirinek (1986) reports a peak incidence in the 5th decade.


Gordis (1984) reported a forty fold rise in incidence in blacks compared to whites, but this has not been verified by other workers.

Various aetiological agents have been implicated Smoking (Wynder 1975), alcoholism and diet but no direct evidence is available as yet DI Magno (1977).
Late onset diabetes may be due to an underlying pancreatic carcinoma. Bell (1957) reported a 30% association. The proportion is variable but most of the patients with pancreatic carcinoma may have abnormal glucose tolerance (Cubilla 1974).

PATHOLOGY:

MICROSCOPIC APPEARANCE:

In primary non endocrine cancer of the pancreas, adenocarcinomas form up 90% of the tumours seen. The main histological types are:

1. Duct cell adenocarcinoma 75%
2. Giant cell carcinoma 4%
3. Adenosquamous carcinoma 4%
4. Mucinous cystadenocarcinoma 1%
5. Acinar cell adenocarcinoma 1%
6. Others 15%

(Cubilla 1975)

DUCT CELL ADENOCARCINOMA

This is the commonest type, originating from the duct epithelium. Tumours from the large ducts stain positive for mucin while those from ductules are negative. The tumour shows a high desmoplastic reaction
with dense bands of collagen and pancreatitis surrounding the tumour, thus on biopsy if tumour not included, only areas of pancreatitis may be seen. Some glands so well differentiate especially in the periampullary region that it may be difficult to differentiate from normal gland.

**Giant Cell Carcinoma:**

Is characterised by presence of huge cells with very large pleomorphic often vacuolated nuclei containing prominent nucleoli and abundant coarse nuclear chromatin. Phagocytosis is occasionally seen. The features are not specific for the pancreas.

**Adenosquamous Carcinoma:** has two components: adenocarcinoma and Squamous cell carcinoma features.

**Mucinous Cystadenocarcinoma:**

This is quite rare accounting for about 1%. It is a malignant Variation of the benign cystadenoma. Common in females (Sleisenger 1978) and occurs mainly in varying sizes. Surgical resection may result in long term survival in most of the patients.
Acinar Cell Adenocarcinomas:

These arise from the acini and may have systemic manifestation simulating connective tissue disorders e.g. arthritis.

Other histological features rarely presenting include the anaplastic carcinoma, microadenocarcinoma and other rarer forms.

The location of a tumour in the pancreas is of marked significance in the entire problem of pancreatic cancer. It is the major determinant of the symptomatology the patient presents with, the investigations to be undertaken and the mode of treatment.

The tumour is located predominantly in the head of pancreas in over 65%, to the ampulla of vater in 7%; body of pancreas in 25% and the tail in about 3% (LUND 1968). Similar proportions have been reported by many workers (CUBILLA 1978).

Tumour Spread:

Occurs early, both locally and distantly to mainly the duodenum, liver, bone, adrenals and lung (Macdonald 1982). Spread is enhanced by the rich lymphatic network draining the pancreas, presence of short wide veins and proximity of many visceral organs.
around the pancreas. Five main groups of lymph nodes draining the pancreas have been identified (Cubilla 1978).

1. Superior group consisting of:
   Superior head, Superior body and Gastric nodes.
2. Inferior group consisting of inferior head and inferior body groups.
3. Anterior group consisting of the anterior pancreaticoduodenal, pyloric and Mesenteric nodes.
4. Posterior group comprising of the posterior pancreaticoduodenal and common bile duct nodes and the
5. Splenic group comprising of nodes at the hilum of the spleen and tail of pancreas.

The superior and posterior head groups are most commonly involved (Nagai 1986, Cubilla 1978).

STAGING:

Two methods are in use.

A). The national survey of cancer staging, where three stages are described.
   i) Tumour localised to the pancreas
   ii) Regional lymph node involvement.
   iii) Distant spread outside the pancreas and lymph nodes.
B. The T.N.M. Classification as proposed by the American joint Committee on cancer (1983).

None of these methods has much value in the prognostication because even in stage I tumour there is a low survival rate due to early distant metastases (Nagai 1986).

Pancreatic cancer has a grave prognosis mainly due to the late presentation. Herter 1982 found a Median survival of 2 months after biopsy only 6 months for patients with bypass, 9 months with total pancreatetomy and 14 months after a Whipple procedure. The five year survival is almost nil 0% (Cohen 1982); 4.5% (Herter 1982); 0.4-3% (Gudjohnson 1978).

Only the cystadenocarcinoma has a better prognosis (Cullen 1963) after resection with resultant long term survival in 30 - 60% of patients.

CLINICAL PRESENTATION:

Carcinoma of the pancreas presents with various signs and symptoms depending on the size of the primary tumour in the gland and the extent of spread. Tumours in the head of pancreas present commonly with jaundice; hence present earlier than those in the body and tail of pancreas where the features are non specific in the initial stages.
Less than 15% of patients consult a physician within one month from the onset of symptoms (Warren 1967) but about 85% will have done so within six months. 13% may present after more than two years.

The triad of weight loss, Jaundice and abdominal pain is the commonest form of presentation and is highly indicative of carcinoma of the head of pancreas.

Weight loss in the earliest and commonest symptom irrespective of tumour site. The cause is unknown but is partly attributed to reduced food intake due to nausea and anorexia; or the indigestion associated with pancreatic exocrine insufficiency (Go 1977).

In tumours located in the head and periampullary area, Jaundice is the main feature occurring in 75-100% of patients (Dencker 1972), (Go 1977). For tumours in the body and tail Jaundice is rare.

Abdominal pain is also common. About 63% of patients present with abdominal pain (Cubilla 1974). The localisation and radiation is variable, but mainly in the mid epigastric region (Gambie 1970). Other areas include the right upper quadrant, the left upper quadrant and the lower abdomen.

Pain referred to the back is a sign of poor prognosis as it is commonly associated with tumour metastases to th coeliac plexus.
Non specific gastrointestinal symptoms of anorexia, nausea and vomiting are also common (Bowden 1965) and occasionally manifest infiltration of tumour into the duodenum.

Weakness and fatigue are symptoms related to anaemia and occult bleeding (Ansari 1968). Infiltration of the duodenum and pancreatobiliary tract may also be a cause of blood loss in the stool (Ponka 1971).

Lower gastrointestinal complaints particularly diarrhoea may cause distress in about 25 - 50% of patients (Dencker 1972) due to malabsorption caused by fat indigestion.

Psychiatric symptoms, mainly depression, anxiety and frightening premonitions may be the only early symptoms (FRAS 1968) and some patients may even have been referred to psychiatrist and treated for depression.

The pancreas is inaccessible to palpation unless the tumour is large in advanced disease. The palpable gall bladder of Courvoiser's law is found in a majority of patients with tumour in the head of pancreas who have not had a previous cholecystectomy.

The liver edge is usually palpable due to obstruction to biliary outflow.

Thrombophlebitis is rare-less than 4% (Sack 1977) and most often seen in post mortems (Douglass 1974).
A bruit may be heard in the left upper quadrant in 3 -6% of patients due to stenosis of the splenic vein or a major artery by tumour encroachment (Hardcastle 1969).

The tumour may also cause portal vein obstruction causing splenomegaly and portal hypertension manifested as bleeding varices.

Ascites and positive Trossiers sign are also signs of advanced disease.

LABORATORY DIAGNOSIS:

The clinical laboratory is of minimal value in the establishment of a diagnosis of pancreatic cancer. (Melenyk 1978). Serum amylase may be elevated in a few patients due to secondary pancreatic duct obstruction by tumour but the presentation is subclinical (Macdonald 1980).

Liver function tests show features of obstructive jaundice in patients with periampullary and head of pancreas tumours. The bilirubin levels may be highly elevated mainly the direct bilirubin. Alkaline phosphatase levels are also significantly elevated.
The enzymes Aspartate aminotransferase (AST) and Alanine aminotransferase (ALT) are also elevated but only indicate bile duct obstruction. The serum protein may be reduced due to impaired liver function.

The prothrombin time is prolonged in obstructive jaundice and responds readily to Vitamin K injections.

Glucose tolerance test may be abnormal in 25-50% of patients. (Melenyk 1980)

Pancreatic exocrine function tests using the pancreatic Secretin test are also non-specific (Kawaii 1980, Drelling 1975) and of no value in differentiating chronic pancreatitis from pancreatic cancer.

Leucine Aminopeptidase (LAP) a Material secreted into pancreatic juice has been found elevated in most obstructive cholangiopathies. It is also elevated in many icteric conditions not associated with obstruction. Upon relief of obstruction, levels fall to normal but it is not specific for cancer of pancreas.

Tumour Markers:

A number of circulating tumour markers have been examined in patients with pancreatic cancer. These include
i) Pancreatic oncofetal antigen

ii) Carcino embryonic antigen

iii) Ferritin

iv) Alpha - Feto protein

v) Human serum Ribonuclease

vi) CA 19 - 9 Antigen

Carcino Embryonic Antigen:

CEA levels are elevated in majority of patients with adenocarcinoma of pancreas (Ona 1973). With metastases up to 70% have high levels. However the tumour marker is also elevated in many other conditions including pancreatitis. It is useful only in monitoring for recurrence after tumour resection.

Pancreatic Oncofetal Antigen:

This is also non specific as it is also found in colonic cancer, Billiary tract cancer and pancreatitis. It is a tumour associated antigen and only useful for follow up (Gelder 1974).

Ferritin

Serum ferritin levels are usually high preoperatively and after resection they fall drastically to normal. It can thus be used to differentiate
pancreatitis from cancer of the pancreas and also for follow up. False positive results are seen with other conditions especially lung cancer, breast cancer, hepatocellular cancer, haemachromatosis and chronic inflammation (Kawaii 1980).

**ALPHA - FETO PROTEIN:**

This is also elevated in a proportion (25%) (Mcintire 1975); but is non specific.

**HUMAN SERUM RIBONUCLEASE:**

This is still under evaluation but offers no added advantage over the rest of tumour markers. Elevated levels are seen in pancreatitis pancreatic cancer and even in normal subjects (Kawaii 1980).

**SERUM CA 19 - 9 ANTIGEN:**

This is carbohydrate antigenic determinant defined by a monoclonal antibody. High levels are observed in pancreatic lesions, 90% in carcinoma of pancreas (Malesci, 1987). But a few positives in chronic pancreatitis also seen. It is thus more specific for pancreatic cancer compared with the other available markers.
PANCREATIC BIOPSY TECHNIQUES:

The correct surgical treatment of localised tumour in the pancreas demands an exact histological diagnosis. The error by palpation is about 3 - 25% (Gudjohnson 1978). The tumour is usually surrounded by a large area of pancreatitis which makes palpation of tumour difficult and increases the risk of complications, especially Bleeding, fistula formation and pancreatitis (Stromby 1972).

Use of frozen sections intraoperatively causes considerable difficulty in interpretation especially with well differentiated adenocarcinoma.

Aspiration biopsy can be obtained using a fine needle or during E.R.C.P.

FINE NEEDLE BIOPSY:

This can be done intraoperatively or preoperatively. Preoperatively this is done if a mass is palpable. Intraoperatively, the mass is palpated and fine needle - gauge 21 used. (Shorey 1975). A fранzen needle (Forsgren 1973) may also be used, 3 to 5 aspirates are taken and the biopsy stained with Glemsa (Stromby 1972). Use of intraoperative ultrasonic guidance greatly increases the success rate (Mittly 1981). If combined with C.T. scan the depth of the lesion can be precisely defined.
ASPIRATION DURING E.R.C.P.

This is done before injection of contrast. 2 - 10cc of pancreatic duct contents are obtained and centrifuged then a papanicolau smear is done.

DUONENAL ASPIRATION:

Has a low yield (Sato 1967), (Gregg 1972). PAP smear is then done.

RADIOLOGICAL DIAGNOSIS:

This is the mainstay of diagnosis.

PLAIN X - RAYS:

Plain Xrays may show calcification in patients with cystadenoma or cystadenocarcinoma in 10% of patients (Becker 1965). Calcification is also seen in chronic pancreatitis. Presence of calcification has been linked to carcinoma of pancreas in 2 - 4% (Eaton 1973). Plainfose 1987) reports a 6 - 10% association.

ULTRA SOUND:

Ultrasonic examination of the pancreas is useful in patients who have unexplained upper abdominal pain and is worth considering whenever pancreatic carcinoma is a diagnostic possibility. Overall accuracy in pancreatic pathology is between 80 - 90% (Doust 1976)
Pancreatic carcinoma is seen as an enlargement generally confined to the area of the gland with fairly informal structural and irregular outlines. Lower limit that can be diagnosed is 2 - 3 cm diameter. Tumours in the body and tail of the pancreas are difficult to define. Also differentiation between carcinoma and Chronic pancreatitis is impossible (Melenyk 1978, Eaton 1973).

Ultrasound can also be used Intraoperatively to locate the tumour mass and do a preoperative aspiration biopsy. Arteries and veins are avoided under ultrasound guidance and the needle guided into the mass (Plainfosse 1987).

Ultrasound is useful in identifying the dilated bile ducts, gall bladder with associated pathology and also the common bile duct, thus clearly defining the site of biliary obstruction. It is of much help in carcinoma head of pancreas which mainly presents with biliary obstruction.

False positive results can result from the air, bone or barium overlying the pancreas. Thus ultrasound should be used in combination with roentgenography and computerised tomography in the evaluation of a patient with suspected carcinoma of the pancreas.
COMPUTERISED TOMOGRAPHY:

This gives more information than ultrasound and should be the initial procedure of choice. The entire pancreatic bed can be visualised in nearly all cases. It has an accuracy rate of up to 90% (Rubenstein 1984). It is also useful in the assessment of tumour operability. Extension into lesser sac, mesocolon and around the coeliac axis and superior mesenteric artery can be detected if there is sufficient fat around the structures. Infiltration of the fat is a sign of inoperability (Whalen 1979).

Opening up of the collateral circulation can also be visualised and this implies obstruction to the superior mesenteric and splenic vein outflow by tumour. A dilated pancreatic duct and pancreatic calcification and also visible, thus with C.T. Barium studies are not necessary.

In combination with ultrasonography, C.T. is useful in assessment of the depth of lesion with high precision and the aspiration biopsy thus taken under ultrasonic guidance (Haaga 1978).

However like ultrasound it is difficult to differentiate between chronic pancreatitis and pancreatic carcinoma.
SELECTIVE ARTERIOGRAPHY:

This is valuable for the localisation of the tumour as well as identifying the tumour feeding vessels preoperatively. With selective coeliac and superior mesenteric angiograms, accuracy in diagnosis of pancreatic carcinoma is 60% (Rosch 1979). To detect small tumours superselective angiography is needed.

Arterial narrowing and encasement is seen and this suggests compression or invasion by tumour. Angiography is contradicted in atherosclerosis. It also requires a highly experienced radiologist as the success rate depends on the clinical experience of the radiologist.

ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY: (E R C P)

This is useful in obtaining material for cytology and in the evaluation of the pancreatic and distal bile ducts. Once the ampulla of vater is cannulated aspiration is done and material sent for cytology, contrast material is then injected. Carcinoma of the pancreas may cause pancreatic duct stenosis or obstruction. There may be delayed contrast outflow, ductal displacement, necrotic cavity formation and deformity of common bile duct by tumour in the head of pancreas. Visualisation and Cannulation of the ampulla may not be achieved in up to 10%
or more as the success parallels the experience of the endoscopist. (Moosa 1979).

PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY:

This is useful for patients with obstructive post hepatic jaundice. It demonstrates abnormalities along the distal common bile duct part of which lies in the head of pancreas. There may be complete or partial obstruction of the bile duct. In tumours localised to the body and tail, PTC is of no value.

UPPER GASTROINTESTINAL CONTRAST STUDIES:

These have been replaced by the more sophisticated an informative techniques due to their low diagnostic accuracy, most often only for detection of large tumours.

The barium meal and follow through may show extrinsic compression, mucosal abnormalities with metastatic infiltration or varices in portal hypertension. In the duodenum, direct invasion of the duodenal wall may cause deformity of the duodenal wall and in some cases the inverted - 3 signs of Frostberg may be seen (Melenyk 1978).

"A mass effect on the medial aspect of the C-loop, thickened mucosal folds; post bulbar duodenal ulceration and motility abnormalities may be seen."
Hypotonic duodenography using anticholinergic drugs into the duodenum or intravenous secretin has no added diagnostic value in detecting pancreatic cancer (Sirinek, 1986, Shirley 1974).

MAGNETIC RESONANCE IMAGING:

This has no further advantage over CT scan (Sarner 1986).

PANCREATIC SCANNING:

Was performed using Se-Selenomethionine. In most cases both conventional and subtracted photoscans were obtained (Eaton 1968) but due to increased activity in the liver the pancreas is not well visualised hence the method has been abandoned (Rubenstein 1984).
TREATMENT:

Treatment of carcinoma of the pancreas is rife with controversies. Mainly because of the outcome after surgery. Cure of the disease is only possible after resection of tumour but this is rarely achieved due to the late presentation of patients with the tumour coupled with poor post operative results even in those in whom it is discovered early.

Palliative bypass operations for the biliary and gastric obstruction are performed in non resectable tumours. The poor post operative quality and duration of life require careful evaluation of the surgical risk since the incidence of mortality and morbidity after both operations is high (Pedrazoli 1987).

Pancreatic resections should not be performed without a histological diagnosis. Palpation and inspection have a diagnostic error up to 25% (Gudjohnson 1978).

The important risk factors are weight loss, duration of jaundice, serum bilirubin, total protein and patients age (Pedrazolli 1987). (Obertop 1982) noted that pain was a poor prognostic sign as it indicated metastases to the coeliac plexus especially if it was radiating to the back. (Kuchita & Cohen (1982) on their 40 year experience identified the poor prognostic factors as:
a) Age greater than 51 years
b) Serum Bilirubin more than 6mg/dl
c) SGOT greater than 100 iu/l
d. Previous drainage procedure.

Preoperatively, correction of the prothrombin time index using intramuscular Vit K injection, correction of anaemia, control of diabetes and fluid and electrolyte balance are mandatory as in any major surgical procedure.

PANCREATODUODENECTOMY:

This was popularised first by Whipple in 1935. Initially it was performed in two stages: Biliary bypass and later pancreateoduodenectomy was performed. This was later performed in a single stage by (Trimble 1941). It involves en bloc removal of the distal portion of stomach, duodenum, head of pancreas, common bile duct and upper jejunum and then reconstruction by choledochojejunostomy, pancreatojejunostomy and gastrojejunostomy. The biliary and pancreatic anastomosis precede the gastric anastomosis to avoid reflux of acid into the bile duct or the pancreatic duct. Truncal vagotomy is done as a prophylaxis to the high incidence of gastric and jejunal
ulcers. (Traverso and Longmire 1986). Cholecystectomy is also advocated as a prophylactic measure against gall stone formation (Melenyk 1978).

The mortality rate is very high, (Dencker 1972) reports a mortality rate of up to 20%. Early complications include pancreatic fistula, wound infection, intrabdominal haemorrhage and Biliary fistula.

Less common complications are gastrointestinal haemorrhage thromboembolism and gastric fistula.

The management of the pancreatic duct determines the success rate of the pancreaticoduodenectomy (Denker 1972)

The experience of the surgeon is a major determinant of the morbidity and mortality. (Howard 1968) performed 41 Whipple resections without any operational mortality.

Due to the high incidence of gastrointestinal bleed following a Whipple procedure, a pylorus preserving procedure is now advocated. (Traverso 1979, Braasch 1986). In these patients there is a delay in recovery of normal gastric function in up to 50%.

Marginal ulcers are also seen despite the vagotomy but the operation preserves most of the anatomy (distal stomach and pylorus). The rate of ulceration is about 3%. 

TOTAL PANCREATECTOMY:

Due to the multifocal nature of the tumour, and the high rate of fistula formation, total pancreatectomy appears a better alternative. Splenectomy and Lymph node dissection is also done.

This operation is associated with a brittle diabetes (Obertop 1982) which requires very high doses of insulin. Also the patients require supplements of pancreatic enzymes.

Fortner (1973) advocates a regional pancreatectomy with vascular reconstruction. The operation takes up to 14 hours.

PALLIATIVE TREATMENT:

Due to the late presentation of the disease in most patients, palliative treatment in the form of bypass surgery, radiotherapy and chemotherapy is given.

Even in resectable tumours, the high morbidity and mortality after resection coupled with the poor prognosis, even after successful resection form the basis of the controversies in the treatment of pancreatic carcinoma.
Crile (1970) found that the duration of survival after bypass was as long as 4 years. This is because the tumour is slow growing. (Hertzberg 1974) found that patients undergoing pancreatoduodenal resection had a shorter life span than those with bypass procedures.

The choice of biliodigestive procedure to relieve the jaundice depends on the extent of the disease and surgeons preference. Cholecystojejunostomy is easier and is most preferred, but if the gall bladder is diseased or patient had had cholecystectomy before then Choledechojejunostomy is only alternative.

Occasionally the gall bladder and the extraphepatic ducts may not be available due to tumour infiltration hence a hepatoduodenostomy with a Roux - en-Y is performed.

A concommitant gastroenterostomy should be performed as a palliation to gastroduodenal obstruction. This is because after biliary bypass up to 40% of patients later present with duodenal obstruction (Blievernicht 1980).

Internal biliary drainage by endoprosthesis is also used in poor risk patients with inoperable tumour (Bucharath 1981). The tube is inserted percutaneously and transhepatically. It requires an experienced radiologist. The tube may dislocate. Cholangitis and subphrenic abscess may also complicate the endoprosthesis.
RADIATION THERAPY

This has been tried in inoperable tumours but with minimal success in improvement of survival. It also increases the risk of Gastrointestinal haemorrhage (Weiss 1985)

CHEMOTHERAPY

Combination Chemotherapy using 5 - Fluorouracil, Doxorubicin and mitomycin C has been tried and is reported to be superior than single drug therapy with 5 - FU. However the effect on improved survival is doubtful (Macdonald 1982).
MATERIALS AND METHODS

1. Records of operations performed at Kenyatta National Hospital for pancreatic carcinoma.

2. Follow up clinical records from the patients case notes.


Only patients with an operational diagnosis of carcinoma of the pancreas were entered in the study. The data was then analysed and presented in the form of tables and bar charts.
<table>
<thead>
<tr>
<th>YEAR</th>
<th>NO. OF CASES</th>
<th>TOTAL NO. OF INPATIENTS</th>
<th>INCIDENCE PER 100,000</th>
</tr>
</thead>
<tbody>
<tr>
<td>1977</td>
<td>7</td>
<td>28,119</td>
<td>24.9</td>
</tr>
<tr>
<td>1978</td>
<td>6</td>
<td>32,144</td>
<td>18.7</td>
</tr>
<tr>
<td>1979</td>
<td>7</td>
<td>53,221</td>
<td>13.2</td>
</tr>
<tr>
<td>1980</td>
<td>8</td>
<td>64,303</td>
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</tr>
<tr>
<td>1981</td>
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<td>1983</td>
<td>3</td>
<td>68,186</td>
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<tr>
<td>1984</td>
<td>4</td>
<td>65,254</td>
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</tr>
<tr>
<td>1985</td>
<td>6</td>
<td>62,802</td>
<td>9.6</td>
</tr>
</tbody>
</table>
FIGURE 1

INCIDENCE OF PANCREATIC CANCER PATIENTS SEEN BETWEEN 1977 AND 1985 PER 100,000 INPATIENTS

YEAR

'77  '78  '79  '80  '81  '82  '83  '84  '85

24.9  18.7  13.2  12.4  9.9  3.1  4.4  6.1  9.6
### Table 2

**Age Distribution of Patients Diagnosed of Pancreatic Cancer at Kenyatta National Hospital 1977 to 1985**

<table>
<thead>
<tr>
<th>Age (Years)</th>
<th>No. of Patients</th>
<th>%</th>
</tr>
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<tbody>
<tr>
<td>20-29</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>30-39</td>
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<td>18.4</td>
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<tr>
<td><strong>Total</strong></td>
<td><strong>49</strong></td>
<td><strong>100.0</strong></td>
</tr>
</tbody>
</table>
FIGURE 2

BAR CHART TO SHOW AGE DISTRIBUTION IN PANCREATIC CANCER PATIENTS AT KENYATTA NATIONAL HOSPITAL BETWEEN 1977 AND 1985

AGE IN YEARS
TABLE 3

AGE-SEX DISTRIBUTION OF PANCREATIC CANCER PATIENTS SEEN AT KENYATTA NATIONAL HOSPITAL FROM 1977 TO 1985

<table>
<thead>
<tr>
<th>AGE (Years)</th>
<th>No of males</th>
<th>No of females</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>20-29</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>30-39</td>
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<td>3</td>
<td>6</td>
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<td>8</td>
<td>3</td>
<td>11</td>
</tr>
<tr>
<td>70-79</td>
<td>3</td>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>80-89</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>No Records</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>TOTAL</td>
<td>31</td>
<td>18</td>
<td>49</td>
</tr>
</tbody>
</table>

Male:Female = 1.7:1

Mean age in males = 52.4 years

Mean age in females = 56.4 years
FIGURE 3

BAR CHART TO SHOW AGE-SEX DISTRIBUTION IN PANCREATIC CANCER PATIENTS AT KENYATTA NATIONAL HOSPITAL BETWEEN 1977 AND 1985

AGE IN YEARS

NUMBER OF PATIENTS

Female
Male
### Table 4

**Distribution of Patients According to District**

<table>
<thead>
<tr>
<th>District</th>
<th>No. of Patients</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>KIAMBU</td>
<td>13</td>
<td>26.5</td>
</tr>
<tr>
<td>MACHAKOS</td>
<td>6</td>
<td>12.2</td>
</tr>
<tr>
<td>NYERI</td>
<td>4</td>
<td>8.1</td>
</tr>
<tr>
<td>KISUMU</td>
<td>4</td>
<td>8.1</td>
</tr>
<tr>
<td>KERU</td>
<td>3</td>
<td>6.1</td>
</tr>
<tr>
<td>MURANGA</td>
<td>2</td>
<td>4.1</td>
</tr>
<tr>
<td>KAKAMEGA</td>
<td>2</td>
<td>4.1</td>
</tr>
<tr>
<td>NAIROBI</td>
<td>2</td>
<td>4.1</td>
</tr>
<tr>
<td>NAKURU</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>SIAYA</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>BUSIA</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>KITALE</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>KISII</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>DISTRICT</td>
<td>NO. OF PATIENTS</td>
<td>%</td>
</tr>
<tr>
<td>------------</td>
<td>-----------------</td>
<td>-----</td>
</tr>
<tr>
<td>EMBU</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>KAJIADO</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>KITUI</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>KIRINYAGA</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>BARINGO</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>MOMBASA</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>NO RACORDS</td>
<td>2</td>
<td>4.1</td>
</tr>
<tr>
<td>TOTAL</td>
<td>49</td>
<td>100</td>
</tr>
</tbody>
</table>
TABLE 6

DURATION BETWEEN ONSET OF ILLNESS AND PRESENTATION TO MEDICAL PERSONNEL

<table>
<thead>
<tr>
<th>DURATION</th>
<th>NO. OF PATIENTS</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>MONTHS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 1</td>
<td>8</td>
<td>16.3</td>
</tr>
<tr>
<td>1 - 3</td>
<td>28</td>
<td>57.1</td>
</tr>
<tr>
<td>4 - 7</td>
<td>9</td>
<td>18.4</td>
</tr>
<tr>
<td>8 - 11</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>YEARS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>3</td>
<td>6.1</td>
</tr>
<tr>
<td>2</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>&gt; 2</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>TOTAL</td>
<td>49</td>
<td>100</td>
</tr>
</tbody>
</table>
### TABLE 6

DURATION BETWEEN CLINICAL DIAGNOSIS AND OPERATION FOR PANCREATIC CANCER AT KENYATTA NATIONAL HOSPITAL DURING THE PERIOD 1977 TO 1985

<table>
<thead>
<tr>
<th>DURATION (WEEKS)</th>
<th>NO. OF PATIENTS</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>LESS THAN 1</td>
<td>3</td>
<td>6.1</td>
</tr>
<tr>
<td>WITHIN 1</td>
<td>4</td>
<td>8.1</td>
</tr>
<tr>
<td>WITHIN 2</td>
<td>15</td>
<td>30.6</td>
</tr>
<tr>
<td>WITHIN 3</td>
<td>6</td>
<td>12.2</td>
</tr>
<tr>
<td>WITHIN 4</td>
<td>12</td>
<td>24.5</td>
</tr>
<tr>
<td>WITHIN 5 - 8</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>WITHIN 9 - 12</td>
<td>2</td>
<td>4.1</td>
</tr>
<tr>
<td>NO RECORDS</td>
<td>6</td>
<td>12.2</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>49</strong></td>
<td><strong>100</strong></td>
</tr>
<tr>
<td>SYMPTOM</td>
<td>NO. OF PATIENTS</td>
<td>%</td>
</tr>
<tr>
<td>---------------------------------</td>
<td>----------------</td>
<td>-----</td>
</tr>
<tr>
<td>Weight loss</td>
<td>15</td>
<td>30.6</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>47</td>
<td>95.9</td>
</tr>
<tr>
<td>Pain radiating to back</td>
<td>12</td>
<td>24.5</td>
</tr>
<tr>
<td>Pain worsened by feeding</td>
<td>7</td>
<td>14.2</td>
</tr>
<tr>
<td>Anorexia</td>
<td>22</td>
<td>44.8</td>
</tr>
<tr>
<td>Acholic stools</td>
<td>26</td>
<td>53.0</td>
</tr>
<tr>
<td>Itching</td>
<td>36</td>
<td>73.4</td>
</tr>
<tr>
<td>Dark urine</td>
<td>39</td>
<td>79.6</td>
</tr>
<tr>
<td>Nausea</td>
<td>19</td>
<td>38.7</td>
</tr>
<tr>
<td>Constipation</td>
<td>7</td>
<td>14.2</td>
</tr>
<tr>
<td>Jaundice</td>
<td>39</td>
<td>79.6</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>5</td>
<td>10.2</td>
</tr>
</tbody>
</table>
### TABLE 8

**DISTRIBUTION OF ABDOMINAL PAIN IN PANCREATIC CANCER PATIENTS AT KENYATTA NATIONAL HOSPITAL BETWEEN 1977 AND 1985**

<table>
<thead>
<tr>
<th>SITE</th>
<th>NO. OF PATIENTS</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epigastric</td>
<td>21</td>
<td>44.7</td>
</tr>
<tr>
<td>Right Upper Quadrant</td>
<td>18</td>
<td>38.3</td>
</tr>
<tr>
<td>Left Upper Quadrant</td>
<td>3</td>
<td>6.4</td>
</tr>
<tr>
<td>Epigastric and Left Upper Quadrant</td>
<td>4</td>
<td>8.5</td>
</tr>
<tr>
<td>Right Upper Quadrant and Left Upper Quadrant</td>
<td>1</td>
<td>2.1</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>47</strong></td>
<td><strong>100</strong></td>
</tr>
<tr>
<td>PHYSICAL SIGN</td>
<td>NO. OF PATIENTS</td>
<td>(X/49)%</td>
</tr>
<tr>
<td>----------------------</td>
<td>----------------</td>
<td>---------</td>
</tr>
<tr>
<td>Hepatomegaly</td>
<td>25</td>
<td>51.0</td>
</tr>
<tr>
<td>Palpable Gall bladder</td>
<td>13</td>
<td>26.5</td>
</tr>
<tr>
<td>Abdominal mass</td>
<td>14</td>
<td>28.6</td>
</tr>
<tr>
<td>Jaundice</td>
<td>39</td>
<td>79.6</td>
</tr>
<tr>
<td>Ascites</td>
<td>4</td>
<td>8.1</td>
</tr>
<tr>
<td>Stool +ve for blood</td>
<td>5</td>
<td>10.2</td>
</tr>
<tr>
<td>Splenomegaly</td>
<td>2</td>
<td>4.1</td>
</tr>
<tr>
<td>INVESTIGATION</td>
<td>NO. OF PATIENTS</td>
<td>%</td>
</tr>
<tr>
<td>-------------------------------------</td>
<td>----------------</td>
<td>-----</td>
</tr>
<tr>
<td>Anaemia (Hb &lt; 10g/dl)</td>
<td>11</td>
<td>22.4</td>
</tr>
<tr>
<td>Elevated Alkaline Phosphatase</td>
<td>30</td>
<td>61.2</td>
</tr>
<tr>
<td>Elevated SGOT</td>
<td>23</td>
<td>46.9</td>
</tr>
<tr>
<td>Elevated SGPT</td>
<td>16</td>
<td>32.7</td>
</tr>
<tr>
<td>Elevated Serum Bilirubin</td>
<td>39</td>
<td>79.6</td>
</tr>
<tr>
<td>Elevated Blood Glucose</td>
<td>9</td>
<td>18.3</td>
</tr>
<tr>
<td>Elevated Serum Lipase</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Elevated Serum Amylase</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Prolonged Prothrombin Time</td>
<td>29</td>
<td>59.1</td>
</tr>
<tr>
<td>INVESTIGATION</td>
<td>NO. OF PATIENTS</td>
<td>%</td>
</tr>
<tr>
<td>---------------------------------------------------</td>
<td>----------------</td>
<td>----</td>
</tr>
<tr>
<td>Plain Abdominal X-ray</td>
<td>3</td>
<td>6.1</td>
</tr>
<tr>
<td>Barium meal</td>
<td>23</td>
<td>46.9</td>
</tr>
<tr>
<td>Ultra sound</td>
<td>15</td>
<td>30.6</td>
</tr>
<tr>
<td>Percutaneous Transhepatic Cholangiogram</td>
<td>5</td>
<td>10.2</td>
</tr>
<tr>
<td>Hypotonic Duodenography</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>Endoscopic retrograde Cholangiopancreatogram</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Computerised Tomography Scan</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Coeliac Angiography</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>NO RECORDS</td>
<td>2</td>
<td>4.1</td>
</tr>
<tr>
<td>BIOPSY SITE</td>
<td>NO. OF PATIENTS</td>
<td>NO. +VE FOR TUMOUR</td>
</tr>
<tr>
<td>------------------------------</td>
<td>-----------------</td>
<td>---------------------</td>
</tr>
<tr>
<td>Primary tumour</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>Liver Metastases</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Omental tumour</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Virchows node</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Mesenteric lymph node</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Metastatic tumour (site not indicated)</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>26</strong></td>
<td><strong>18</strong></td>
</tr>
</tbody>
</table>
**TABLE 13**

LOCATION OF TUMOUR IN THE PANCREAS IN PATIENTS WITH PANCREATIC CANCER AT KENYATTA NATIONAL HOSPITAL FROM 1977 TO 1985

<table>
<thead>
<tr>
<th>SITE</th>
<th>NO. OF PATIENTS</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head</td>
<td>39</td>
<td>79.6</td>
</tr>
<tr>
<td>Head and body</td>
<td>4</td>
<td>8.2</td>
</tr>
<tr>
<td>Body</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>Tail</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>Diffuse</td>
<td>4</td>
<td>8.2</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>49</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>
TABLE 14

SURGICAL PROCEDURES PERFORMED ON PANCREATIC CANCER PATIENTS AT KENYATTA NATIONAL HOSPITAL FROM 1977 TO 1985

<table>
<thead>
<tr>
<th>SURGICAL PROCEDURE</th>
<th>NO. OF PATIENTS</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exploratory laparotomy</td>
<td>11</td>
<td>22.4</td>
</tr>
<tr>
<td>Palliative procedures:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1) Gastrojejunostomy</td>
<td>2</td>
<td>4.1</td>
</tr>
<tr>
<td>2) Cholecystojejunostomy</td>
<td>12</td>
<td>25.0</td>
</tr>
<tr>
<td>3) Jejunojejunostomy and Cholecystojejunostomy</td>
<td>13</td>
<td>26.5</td>
</tr>
<tr>
<td>4) Gastrojejunostomy and Cholecystojejunostomy</td>
<td>9</td>
<td>18.3</td>
</tr>
<tr>
<td>Partial resection of tumour</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>No records</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>49</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>
### TABLE 15

**POST OPERATION COMPLICATIONS AFTER PALLIATIVE SURGERY IN PANCREATIC CANCER PATIENTS AT KENYATTA NATIONAL HOSPITAL FROM 1977 TO 1985**

<table>
<thead>
<tr>
<th>COMPLICATION</th>
<th>NO. OF PATIENTS</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nil complication</td>
<td>32</td>
<td>65.3</td>
</tr>
<tr>
<td>Wound sepsis</td>
<td>3</td>
<td>6.1</td>
</tr>
<tr>
<td>Burst abdomen</td>
<td>3</td>
<td>6.1</td>
</tr>
<tr>
<td>Faecal fistula</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>Biliary leak</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>Haemorrhage</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>Hypoglycaemic coma</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>Renal failure</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Anastomotic leak</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Peritonitis</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>NO RECORDS</td>
<td>6</td>
<td>12.2</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>49</strong></td>
<td><strong>100</strong></td>
</tr>
<tr>
<td>HISTOLOGICAL TYPE</td>
<td>NO. OF PATIENTS</td>
<td>%</td>
</tr>
<tr>
<td>--------------------------------------</td>
<td>----------------</td>
<td>-----</td>
</tr>
<tr>
<td>Adenocarcinoma</td>
<td>12</td>
<td>24.5</td>
</tr>
<tr>
<td>Anaplastic Carcinoma</td>
<td>4</td>
<td>8.2</td>
</tr>
<tr>
<td>Papillary Carcinoma</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>Leiomyomatous Carcinoma</td>
<td>1</td>
<td>2.0</td>
</tr>
<tr>
<td>of Pancreas</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No Histological Diagnosis</td>
<td>31</td>
<td>2.0</td>
</tr>
<tr>
<td>TOTAL</td>
<td>49</td>
<td>100</td>
</tr>
</tbody>
</table>

TABLE 16
PATHOLOGICAL DIAGNOSIS
RESULTS:

A. EPIDEMIOLOGY:

A total of 49 patients with an operative diagnosis of carcinoma of the pancreas were studied.

1. INCIDENCE:

Table 1 and Figure 1 show an incidence between 3.1 to 24.9 per 100,000 admissions. The average incidence from 1977 to 1983 is 11.4 per 100,000 admissions at Kenyatta National Hospital.

2. AGE SEX DISTRIBUTION

Male: Female ratio is 1.7:1 (Table 3). The age distribution shows a normal distribution curve (FIGURE 2) with a peak at 50 - 59 years. (Table 2). The youngest patient was 21 years old and oldest 80 years. The mean age in males was 52.4 years while in females it was 56.4 years. In males there were two peaks of age presentation; at 40 - 49 years and 60 - 69 years, while in females only one peak at 50 - 59 years was seen (FIGURE 3).
DISTRIBUTION ACCORDING TO DISTRICT OF ORIGIN

Kiambu district had the highest proportion 26.5% followed by Machakos with 12.2%. Overall, central province had 40.7% of the total. The distribution was widespread in all provinces except North Eastern province where no case was reported (TABLE 4).

B. HISTORY OF THE DISEASE:

1. Duration between onset of illness and Presentation to Medical Personnel.

73% of the patients had sought Medical attention by 3 months after onset of illness. The peak duration was 1 - 3 months. No patients were seen after 2 years of disease (TABLE 5)

2. Duration between diagnosis and treatment

Only 6.1% were operated on in less than one week, but majority 81% had been operated on within 4 weeks (Table 6). The longest waiting period was 9 - 12 weeks in 4.1%.
C. CLINICAL FEATURES

1. Presenting Symptoms

Abdominal pain was the commonest presenting symptom occurring in 95.9% (TABLE 7) followed by jaundice (in 79.6%) with its attendant symptoms of jaundice, itchiness, dark urine and acholic stools. Weight loss occurred in 30.6% of patients.

The abdominal pain was localised mainly in the epigastrium in 44.7% (Table 8), Right upper quadrant in 38.3% and other sites as shown. The pain radiated to the back in 24.5% and was worsened by feeding in 14.2% of patients.

2. Physical Findings:

Jaundice was the main presenting feature in 79.6% (TABLE 9). Hepatomegaly was seen in 51% and a palpable gall bladder in 26.5% a palpable abdominal mass was found in 28.6% of patients. Splenomegaly was found in 4.1%.

Ascites in 8.1% and stool positive for blood in 10.2%.
D. INVESTIGATIONS:

1. Laboratory:

The main findings were those of obstructive jaundice in 79.6% of patients. Elevated alkaline phosphatase was seen in 61.2%; and the other liver enzymes as shown (Table 10). Anaemia was not a main feature seen in only 22.4%. Diabetes mellitus was seen in 18.3% and serum amylase levels were normal. 59.1% presented with a prolonged prothrombin time.

2. Radiological:

6.1% had plain abdominal X-rays which were normal. A Barium meal was the commonest investigation done (46.9%) Table 11, and this showed obstruction at pylorus, or duodenal infiltration. Ultra sound was done in 30.6% and showed extrahepatic biliary obstruction with a mass at the porta hepatis. A pancreatic mass was seen in 93% of the Ultrasound examinations. Hypotonic duodenography was performed in 1 patient and was normal. The other radiological examinations were not performed as shown in Table 11.
3. **Tumour Biopsy:**

26 patients (53%) had a biopsy taken, of these 34.6% had a biopsy of primary tumour. Biopsy from primary tumour and liver metastases yielded a 100% positive result (Table 12). Most of the lymph node biopsies were reported as either normal or reactive lymph nodes.

E. **LOCATION OF TUMOUR IN Pancreas:**

79.6% were located in the head of pancreas (Table 13). Head and body of pancreas in 8.2% body of pancreas in 2% tail in 2% and diffuse in 8.2% (Table 13).

F. **SURGICAL PROCEDURES PERFORMED:**

Majority (75.5%) had palliative bypass procedures. Cholecystojejunostomy was performed in 25%; jejunoojejunostomy and Cholecystojejunostomy in 26.5%, while gastrojejunostomy with cholecystojejunostomy was performed in 18.3% (Table 14). Exploratory laparatomy with no bypass procedure was performed in 22.4%.

Partial resection was performed in only one patient.
H. **POST OPERATIVE COMPLICATIONS:**

Wound sepsis and a burst abdomen occurred in 6.1% while faecal fistula, bile leakage, pneumonia, haemorrhage and hypoglycaemic coma were seen in 2.0%. Most of the patients (65.3%) had no post operative complications. (Table 15).

J. **SURVIVAL RATES:**

This was impossible to assess due to poor follow up. Most of the patients after diagnosis were repatriated to their district hospitals for follow up.

Of those followed up at Kenyatta National Hospital:

- 4(8.1%) died within two weeks.
- 1(2.0%) died 4 months post operatively.
- 1(2.0%) died one year post operatively.
- 4% were followed up for 2 years
- 4% for 3 years and 2% for 4 years

K. **PATHOLOGICAL DIAGNOSIS:**

Adenocarcinoma formed 24.5; Anaplastic 8.2% papillary 2.0% and leiomyomatous carcinoma of the pancreas 2.0% (Table 16).

63.3% had no Histological diagnosis.
DISCUSSION

A. EPIDEMIOLOGY:

1. INCIDENCE:

An average incidence of 11.4 per 100,000 admissions is recorded from 1977 to 1985. This is similar to reports from other workers. (Gudjohnson (1978) reports an incidence of 12.1 per 100,000 men. There is a steady increase in incidence from the lowest 2 per 100,000 in 1982 to 3 per 100,000 in 1985. It would be interesting to see whether the trend is maintained in the following years.

2. AGE - SEX DISTRIBUTION

A sex ratio of 1.7:1 compares well with other reports Sirinek (1986) 2:1; Lund (1986) 3:2. The mean age in males (52.4 years) is lower than in females (56.4 years). Males have two peak ages of presentation 40 - 49 years and 60 - 69 years while females have only one peak at 60 - 69 years. The reason for this difference is not clear.
3. DISTRIBUTION ACCORDING TO DISTRICT OF ORIGIN

Proximity to the referral hospital has a great effect on the distribution, thus Kiambu district which is very near to Kenyatta National Hospital had the higher proportion (26.5%). Similarly central Province had 40.7% of the total referrals. Except for North Eastern Province where no case was reported, the cases appeared in all other provinces.

B. PATHOLOGY:

Most of the tumours were located in the head of pancreas 79.6%, with 8.2% involving the whole pancreas. Only 2% involved the tail. This trend is similar to other reports (Cubilla 1974) except the figures for head of pancreas which is slightly high.

Obstructive Jaundice draws the attention of both patients and physician early hence the highest proportion of tumour in the head of pancreas in this study.
Tumour biopsy was done in only 53% of cases. Of the histology reports available, 66.7% were Adenocarcinomas of duct cell origin. Anaplastic carcinomas comprised 22.2%. This contrasts with other reports (Cubilla 1975) where anaplastic carcinoma forms less than 1% of the tumours and is considered rare.

In this study, a clinical staging for tumour had not been done by the surgeon at operation but from the clinical notes; only one patient was in stage I, the rest were in II or III (National survey of Cancer staging).

C. CLINICAL FEATURES:

Abdominal pain was the commonest symptom seen (95.9%) while weight loss which is reported as the commonest and earliest symptom (Go 1977) occurred in only 30.6%. Second Commonest were features of obstructive jaundice. This is because most of the cases had tumours localised to the head of pancreas.

Most patients were jaundiced (79.6%). With an enlarged liver (51%) or palpable gallbladder (26.5%). An abdominal mass was palpated in 28.6% - these had advanced disease and only a laparatomy and Biopsy were done.
C. Most patients presented early after onset of the disease (75%) within three months but this was still late as they had inoperable tumour. Diabetes mellitus of Maturity onset type was a common association in 18.3%. The diabetes was poorly controlled on insulin with some requiring high doses of insulin. Bell (1957) reported a 30% association.

D. INVESTIGATIONS:

1. LABORATORY INVESTIGATIONS:

This was of minimal value in the diagnosis of pancreatic cancer. With obstructive jaundice, high alkaline phosphatase levels, high bilirubin levels and liver enzymes were seen. However in the preoperative management Blood Sugar and prothrombin time were estimated serially. No assays of tumour markers were done.

2. TUMOUR BIOPSY:

Biopsy of primary tumour by incisional method, and excision of liver metastases yielded 100% positive results. Lymphnodes were mainly reported as reactive. Omental tumour was also useful but with low yield.
2. Aspiration biopsy was not done despite its high safety margin and high yield. After biopsy of primary tumour no major complications were reported.

3. RADIOLOGICAL INVESTIGATIONS:

These were the mainstay of diagnosis. Ultrasound showed features of extrahepatic biliary obstruction and/or a pancreatic mass in 93%, hence should be the first radiological Investigation of choice. Barium contrast studies were useful in cases of duodenal or gastric outlet obstruction. Computerised Tomography was not available hence no experience is reported. Percutaneous Transhepatic cholangiography was done in 8% and was useful in demonstrating the site of Biliary obstruction.

TREATMENT

E. Upon presentation to hospital most patients were operated on within four weeks (81%). The delay was mainly caused by the waiting for investigations and control of Diabetes and prothrombin time to safe levels where the patient was fit for surgery.
The surgical procedures were mainly palliative bypass in 75.5%; Cholecystojejunostomy only was performed in 25%; while a combined biliary and duodenal bypass was performed in 45%. In 22.4% only laparotomy was done. This is because the tumour was inoperable. Only in one patient was the tumour resected.

No duodenal obstruction occurring after Cholecystojejunostomy was observed (Blevernicht 1980).

The complications seen after palliative surgery were mainly burst abdomen in 6.1%. In 65.3%, no complications were reported. After the resection a biliary leak was observed.

F. **SURVIVAL RATES:**

Despite poor follow up, there is a high mortality rate (10% within 4 months) Most of the patients were sent to the referring hospitals for follow up and terminal care hence making it difficult to obtain figures for follow up.

For those followed in Kenyatta National Hospital, there were 2% survivors at 4 years after palliative bypass, operation, thus the tumour is slow growing (Crile 1970).
CONCLUSION:

Pancreatic cancer in Kenya is a disease of middle age commonest in males. The patients usually present late. Up to 18% are associated with maturity onset diabetes, especially those who are poorly controlled on insulin.

Ultrasound has a high diagnostic yield and should be done first when computerised tomography is not available.

Fear to biopsy the organ should be overcome as it can be safely done using a small bore needle for aspiration. For patients with liver metastases, biopsy of liver nodule is quite representative.

Due to the early metastases of the tumour palliative biliodigestive bypass should be the operation of choice for the surgeon who has little experience in pancreatic resection.

RECOMMENDATION:

Pancreatic cancer as seen at Kenyatta National Hospital is a surgical problem of similar magnitude to that reported in other centres; the surgeons involved in the management of these patients should therefore refine their techniques.

Aspiration biopsy which as per this study is not used should be encouraged because it is reliable and safe. Also patients who present early with
resectable tumours should have tumour resection done.
REFERENCES:


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APPENDIX 1
CARCINOMA OF THE PANCREAS AT THE KENYATTA NATIONAL HOSPITAL

PROFORMA

A. PATIENT'S DETAILS

NAME
AGE
SEX
PLACE OF BIRTH (DISTRICT)
PRESENT RESIDENCE

B. HISTORY OF DISEASE

DURATION BETWEEN NOTICING THE DISEASE AND PRESENTATION TO MEDICAL PERSONNEL:

DAYS
WEEKS
MONTHS
YEARS

DURATION BETWEEN DIAGNOSIS AND TREATMENT:

DAYS
WEEKS
MONTHS

C. SYMPTOMS

WEIGHT LOSS
ABDOMINAL PAIN
SITE EPIGASTRIC
RUQ
LUQ
RADIATION TO BACK
RELIEVED BY BENDING FORWARD
WORSENED BY FEEDING
ANOREXIA
VOMITING
ACHOLIC STOOLS
ITCHING
DARK URINE
JAUNDICE
NAUSEA
CONSTIPATION
DIARRHOEA

D. PHYSICAL FINDINGS
HEPATOMEGALY
ABDOMINAL MASS
EPIGASTRIC
RUQ
OTHER
JAUNDICE
ASCITES
STOOL POSITIVE FOR BLOOD

E. INVESTIGATIONS

I. LABORATORY
ANAEMIA HB<10/dl
ELEVATED ALKALINE PHOSPHATASE
ELEVATED SGOT
ELEVATED SGOT
ELEVATED SERUM BILIRUBIN
ELEVATED BLOOD GlUCOSE
ELEVATED SERUM LIPASE
ELEVATED SERUM AMYLASE
PROLONGED PROTHROMBIAIN TIME

2. RADILOGICAL
PLAIN ABDOMINAL XRAY
BA MEAL
COELIAC ANGIOGRAPHY
ERCP
CAT SCAN
REPORT

3. BIOPSY /Yes / No /
SUPRACLAVICULAR L. N.
PARACENTESIS
PERCUTANEOUS LIVER BIOPSY
LAPARATOMY
BIOPSY OF METASTATIC TUMOUR
BIOPSY OF PRIMARY TUMOUR

F. LOCATION OF TUMOUR IN THE GLAND (PANCREAS)
HEAD
HEAD AND BODY
BODY
BODY AND TAIL
TAIL
DIFFUSE
G. SURGICAL PROCEDURE PERFORMED AT LAPARATOMY

EXPLORATORY LAPARATOMY
PALLIATIVE PROCEDURES
BILIARY BYPASS ONLY
GASTRIC BYPASS ONLY
GASTRIC AND BILIARY BYPASS
RESECTION OF THE TUMOUR
PARTIAL PANCREATECTOMY
TOTAL PANCREATECTOMY

H. COMPLICATIONS POST - OPERATIVE

1. AFTER PALLIATIVE SURGERY

LEAKAGE OF ANASTOMOSIS
BILIARY
GASTRIC
PERITONITIS
RENAL FAILURE
PNEUMONIA
WOUND SEPSIS
OTHERS

2. COMPLICATIONS AFTER RESECTION OF PANCREATIC TUMOUR

CARDIOPULMONARY
HAEMORRHAGE
ANASTOMOTIC LEAK
BILIARY
GASTRIC
PANCREATIC
HYPOGLYCAEMIA
INFECTION

WOUND SEPSIS

HEPATIC FAILURE

RENAL FAILURE

OTHERS

I. SURVIVAL

FROM ONSET OF ILLNESS

DAYS WEEKS MONTHS YEARS

FROM TIME OF SURGERY


J. PATHOLOGICAL DIAGNOSIS

DUCT CELL ADENOCARCINOMA

GIANT CELL CARCINOMA

ADENOSQUAMOUS CARCINOMA

ACINAR CELL ADENOCARCINOMA

OTHERS