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PRIMARY DUODENAL CANCER: CASE REPORT

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PRIMARY DUODENAL CANCER: CASE REPORT

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SUMMARY

Primary carcinoma of the duodenum is uncommonly encountered. This is a report of a 64-year-old diabetic/hypertensive who was admitted in our unit with six months history of upper abdominal pain, vomiting on and off and weight loss of greater than 10 kgs. Endoscopy revealed complete obstruction of the third part of the duodenum. Lesion biopsy revealed moderately differentiated adenocarcinoma and associated duodenitis. A staging CT scan showed thickening of the duodenal wall over a span of six centimetres, luminal narrowing, mucosal irregularity and multiple para-vascular large nodes some greater than or equal to two centimetres. Palliative bypass surgery was suggested as the preferred mode of treatment. He underwent cholecysto-jejunostomy/jejunostomy to palliate biliary and intestinal obstruction.

INTRODUCTION

Adenocarcinoma of the duodenum is a rare malignancy accounting for only 0.35% of all gastrointestinal carcinomas. Carcinoma of the third or fourth part constitutes approximately 15% of duodenal carcinomas (1).

Due to infrequency of the condition, it remains a diagnostic and therapeutic uncertainty, and thus diagnosis is often made late, like in our patient who was initially treated for peptic ulcer disease. In addition many duodenal cancers are diagnosed at an advanced stage, because symptoms of these cancers do not normally develop until tumours have invaded adjacent structures, regional lymph nodes, or both. However, occasionally early duodenal cancers are incidentally discovered during endoscopy or on barium studies in patients without symptoms directly attributed to these lesions (2-4). We recently encountered a case of late presentation of the primary duodenal cancer that was diagnosed on a repeat endoscopy and biopsy. Initially the patient had been treated for duodenal ulcers without improvement. This kind of late presentation of duodenal cancer

has not been reported in our local regional literature. Thus the necessity to report this case.

CASE REPORT

A 64 year-old diabetic/hypertensive male, who had been on insulin for 14 years and also on junior aspirin and enalapril and hydro-chlothiazide, presented to us with six months history of diffuse abdominal pains especially after meals. He had induced vomiting on three occasions with some relief. He had noted blood staining of the vomitus in the last two occasions. He also noted progressive weight loss of approximately ten kilograms in six months. He also stated that he could easily get tired when walking briskly for his exercises.

A barium meal done three months earlier had shown gastritis and gastro-oesophageal reflux. The patient was subsequently put on proton pump inhibitors to treat the peptic ulcer disease.

On examination at admission the patient was moderately pale with stable vital signs. He was not jaundiced. There was vague epigastric tenderness but no masses were palpable. An initial

impression of peptic ulcer disease was made. Baseline investigation showed anaemia of 6.2 g/dcl of the microcytic microchromic type, renal and liver functions were reported as normal.

Endoscopy done showed a deformed papilla and a mass in the third part of the duodenum. The biopsy taken showed moderately differentiated adenocarcinoma associated with severe acute on chronic duodentitis (Figure 1 (a) and (b)).

A CT scan of the abdomen was done and it showed a narrowing of the third part of duodenal lumen with thickening of wall over a span of six

centimetres. The mucosal pattern was abnormal with shouldering and irregularity. There were multiple para-pancreatic nodes and para-aortic, para-caval and peri-porta hepatic nodes. However liver was not involved (Figure 2).

The patient was transfused blood to raise his haemoglobin to 10 g/dcl. He was also put on antibiotics, artostatin, insulin, esomeprazole, enalapril and hydrochlorthiazide and domperidone. By pass surgery was done after extensive tumour involving D₂/D₃ parts of duodenum and multiple stations, enlarged lymph nodes were found.

Figure 1

Mass in the duodenum endoscopy

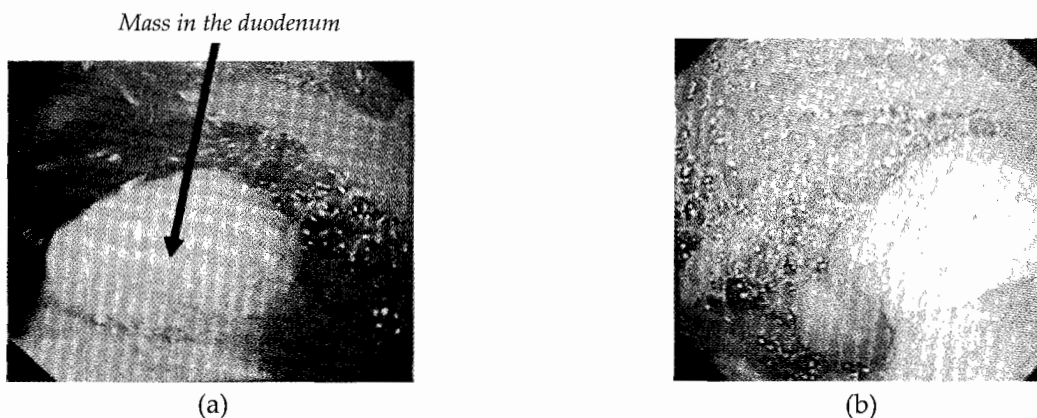
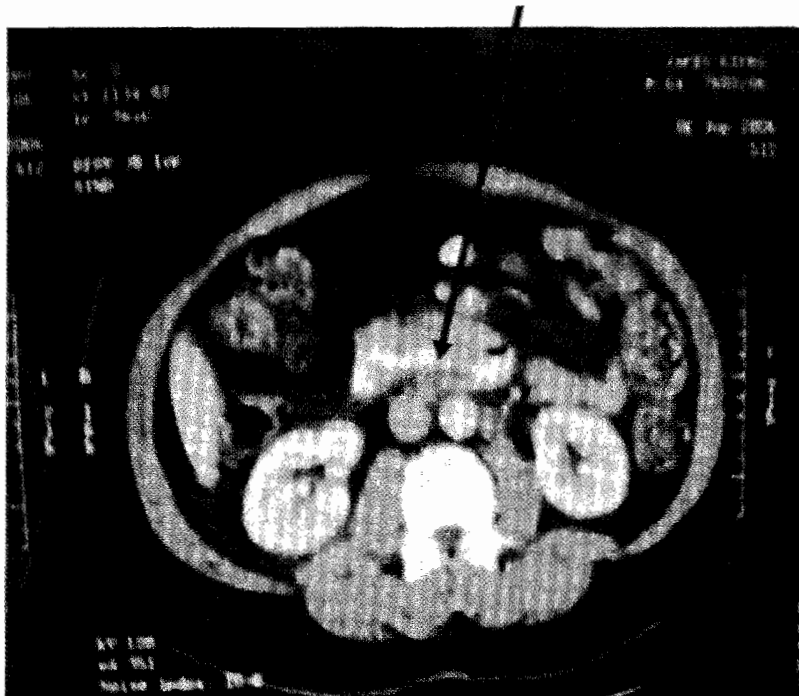


Figure 2

A CT scan of the abdomen showing a narrowing of the third part of duodenal lumen with thickening of wall

Mass in the duodenum



DISCUSSION

Although uncommon, adenocarcinoma of the duodenum accounts for 45-65% of small bowel cancers (1). These tumours may develop de novo in the duodenum or may result from the malignant degradation of pre-existing adenomas polyps (particularly villous adenomas) (5). Duodenal cancers commonly occur in the ampullary or periampullary regions of the descending duodenum; however, these tumours are occasionally found in other portions of the duodenum including the third part (6). The most frequent clinical findings are; nausea, vomiting, post prandial bloating, weight loss and signs of upper gastrointestinal bleeding, such as guaiac-positive stool and iron deficiency anaemia (1,6).

Unfortunately, most symptomatic patients with duodenal cancer have advanced lesions at presentation, like was the case in our patient. It is important for clinicians to suspect presence of duodenal malignancy in the elderly rather than treat empirically for peptic ulcer disease, in cases with additional findings like weight loss, haematemesis, endoscopic evaluation is necessary for early pick-up. These patients have a poor prognosis, with overall five-year survival rates, ranging from 20 to 40% (1,2).

Factors affecting patient survival include the histological grade of the tumour (the degree of differentiation and nuclear grade), depth of invasion, presence or absence of nodal or distant metastases duration of symptoms and location of the tumour in the duodenum. Distal lesions have a better prognosis if discovered early because they are amenable to less mutilative surgery (2,7). The preferable treatment for resectable lesion in the second portion of the duodenum is a pancreatic duodenectomy with en-bloc resection of adjacent tissues including regional lymph nodes. The distal lesions of the duodenum when they present early can be ably managed by resection and duodeno-duodenostomy.

Most authors suggest that the outcome in duodenal carcinoma depends on resectability, lymph node involvement and histological grade. Therefore our patient at his presentation had a poor outcome.

Thus we advised him to have palliative double bypass surgery to obviate his impending biliary obstruction and intestinal obstruction.

The role of post-operative adjuvant therapy is still unclear, and efforts are underway with five fluorouracil based chemotherapy regimens.

CONCLUSION

Primary carcinoma of distal duodenum is rare and diagnosis is often late like in our patient. The disease should be suspected, even if symptomatology is poor. For early disease, segmental resection with regional lymphadenectomy if feasible, offers a good treatment option for patients with early duodenal cancer of distal duodenum. However in late presentation like in our patient, surgical palliation in form of bypass surgery becomes the mainstay of management.

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SCIENTIFIC LETTER

THE MANAGEMENT OF THE ANENCEPHALIC FOETUS

Dear Sir,

The aim of this short communication is to facilitate the understanding of the presentation and management of the anencephalic foetus and a reminder that anencephaly and other neural tube defects (NTDS) are common in the general population but preventable.

Anencephaly is a neural tube defect (NTD) in which the brain and cranial vault are grossly malformed. The cerebrum and cerebellum are reduced or absent, but the hindbrain is present. This defect results when the neural tube fails to close during the third to fourth week of development, leading to foetal loss, stillbirth or neonatal death (1). The incidence of NTDs varies considerably according to geographic and ethnic factors. A figure of one in 1000 to 2000 births is commonly quoted for the general population (2). The aetiology is multifactorial with a recurrence risk after birth of one affected child of 1% to 3%.

The diagnosis of anencephaly is easy and can be made as early as the 12th week on ultrasonography and relies on the demonstration of the absence of the cranial vault (2,4). History of a previous occurrence is important in the diagnosis as the risk of a foetal neural tube defect is increased about ten fold among women, who previously had one affected pregnancy, about twenty fold among women who had two affected pregnancies and about forty fold among those who had three. These women are at sufficiently high risk to justify amniocentesis and a detailed ultrasound examination. The main biochemical diagnostic tests for open neural tube defects such as anencephaly, are amniotic fluid alpha-feto protein and amniotic fluid acetyl cholinesterase measurements (5). A high maternal alpha-feto protein level should alert the clinician to the possibility of the presence of an NTD and the need for ultrasonography.

As anencephaly is an invariably fatal lesion, termination of pregnancy can be offered at anytime in pregnancy (6). Prenatal counselling and informed recommendations should be provided to the affected couple based on the degree of foetal abnormality

and the likely outcome. Many options are available for termination of a pregnancy with an anencephalic foetus depending on the gestational age at the time of diagnosis. Since the diagnosis of anencephaly can be made as early as the 12th week on ultrasonography, termination of a pregnancy with an anencephalic foetus can be carried out in the first trimester using either suction or surgical curettage. Termination of pregnancy with an anencephalic foetus in the second trimester may be effected surgically or medically. Surgical abortion, in which the cervix is dilated sufficiently to allow narrow evacuation forceps to be passed into the uterus to remove the foetus in fragments may be used and is also referred to as a dilatation and evacuation procedure (7). Medical abortion or induction of labour is a method in which uterine contractions and cervical dilation are induced medically with drugs 20mg intravaginal prostaglandin E₂ suppositories may be used every 3-4 hours until abortion occurs. Induction of labour may also be effected by intra-amniotic instillation of hypertonic (20%), sodium chloride solution, hyperosmolar (59.7%) urea, usually with oxytocin or prostaglandin or intra-amniotic prostaglandin F₂ α alone (8). In the third trimester or close to term, cervical ripening prior to induction of labour may be effected using small doses of intravaginal prostaglandin E₂, misoprostol, relaxin, endocervical insertion of hygroscopic dilators such as laminaria tents, lamichel, dilapan or endocervical insertion of a Foley catheter with a 25-50ml balloon following which oxytocin induction should be started (9). Amniotomy followed by the administration of titrated oxytocin infusion could also be used for the induction of labour.

The labour may be prolonged as the presenting part is usually the face, which is a poor dilator of the cervix and soft passage. Shoulder dystocia is a common complication in labour and since anencephaly is incompatible with life, more drastic manoeuvres such as fracturing the foetal clavicle can be made to disimpact the shoulders (10).

Some clinical experience supports the hypothesis that the foetal pituitary gland is involved in the mechanism controlling initiation of labour in the

human (11). Because the pituitary gland may be absent in an anencephalic foetus, spontaneous precipitation of labour may be delayed; therefore, the risk of the pregnancy progressing into the post-term period is significant. In addition, the rate of abnormal foetal presentations during delivery is increased in these anencephalic pregnancies (1). Thus, the absence of the foetal pituitary in the anencephalic foetus may pose a serious challenge during the induction of labour by increasing the induction to delivery interval or causing failed induction of labour altogether especially in the absence of efficient cervical ripening agents such as prostaglandins.

Approximately 60% of affected fetuses die before birth and none survives beyond two weeks postnatally (11). The commonest associated malformations with anencephaly are spinal bifida, cleft lip and palate and club foot.

Anencephaly and other NTDs are preventable. Periconceptual use of folic acid reduces first occurrence as well as recurrence of NTD (3). Patients with history of previous anencephaly or other NTDs should be advised on the need for preconceptual supplementation with high dose folate continued throughout the first trimester. The currently generally accepted recommendation is for supplementation with 4mg of folate daily under these circumstances in contrast to the usual recommendation of 0.4 mg of additional folate daily in those without history of previous anencephaly or other NTDs (3). Proper antenatal care and screening as well as ultrasound evaluation of every pregnancy for a minimum of two ultrasound scans in the first and second trimesters for a normal gestation are necessary for early detection of NTDs and other congenital malformations.

Yours sincerely,

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