Recurrent Peptic Ulcer due to Carcinoid Tumour of Jejunum

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Abstract Carcinoid Tumours are classified as Neuro-endocrine tumours. Commonly known ulcerogenic neuro-endocrine tumour is Gastrinoma of the Pancreas and Duodenum. It secretes hormone Gastrin which causes hyperplasia of the gastric parietal cells with excessive secretion of hydrochloric acid resulting in multiple ulcerations in the stomach, duodenum and upper jejunum. Carcinoid tumours is not known to cause peptic ulceration.

Keywords Peptic ulcer · Carcinoid tumour

Case Report

A 17 year old male patient was admitted to the Department of Surgery in March 1997 with signs and symptoms of gastric outlet obstruction. In the preceding 2 years he was admitted to the same hospital 4 times with pain abdomen, vomiting, and malaena on 2 occasions. On each occasion upper G.I. Endoscopy showed ulcers in the stomach, 1st and 2nd part of Duodenum. He was treated conservatively with H₂ antagonist, proton pump inhibitors, sucralfate with relief of symptoms. When presented with malaena and low haemoglobin blood transfusion was given.

On admission he was haemodynamically stable. Routine blood tests including liver function tests, B.T, C.T, prothrombin time and serum gastrin were done. Hb% was 9 Gm/dl and serum Gastrin 160 pg/L, twice the normal value. Rest of the tests were within normal limits. Upper G.

I. Endoscopy and Barium studies confirmed gastric outlet obstruction and he was operated. As on earlier endoscopic examinations, this time also ulcers were found to extend up to 2nd part of Duodenum.

On laparotomy, tail of pancreas had two tiny nodules and was removed. There were about a dozen fleshy enlarged lymph nodes in the mesentery, largest one about 3 cms. A hard nodule of about 1.5 cm was found on the anti-mesenteric border of the jejunum about 15 cm from the D-J flexure. The jejunal nodule and two enlarged lymph nodes were removed for histopathological examination. Truncal vagotomy and posterior gastrojejunostomy was done, utilizing the enterotomy in the jejunum created while removing the jejunal nodule. Rest of the abdomen including liver was normal. The patient had an uneventful recovery.

Histopathological examinations of the Jejunal nodule showed picture of carcinoid tumour, mainly in the submucosa infiltrating the muscle and ulcerating the mucosa at one site only. Lymph nodes showed metastatic deposits; no pathology was found in the resected tail of the pancreas. Urinary 5HIAA was within normal limits. MEN Syndrome I was excluded from a normal skull X-ray.

He was put on oral PPI and remained symptom free. He experienced occasional mild reflux symptoms well controlled by added antacid preparations. Check upper G.I. Endoscopy after 2 and 4 months showed complete healing of ulcers and healthy G-J stoma and PPI was stopped. He remained asymptomatic on regular follow up.

After 11 months USG of abdomen showed doubtful lymphadenopathy. CT confirmed retroperitoneal lymphadenopathy and a doubtful jejunal mass. Second laparotomy was carried out in February, 1998. Appendix was removed and multiple biopsies taken from thickened jejunal wall,

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mesenteric nodes and tissue from root of mesentery. Mesenteric tissue showed presence of neuroendocrine tumour. Other biopsies were negative showing picture of nonspecific hyperplasia.

He was regularly followed up every 6 months with routine blood tests, LFTs, serum gastrin, urinary 5HIAA, Upper G.I. Endoscopy and USG for 3 years. Serum gastrin level gradually came down to normal. Urinary 5HIAA remained normal. During follow up after the second laparotomy, USG showed few marginally enlarged lymph nodes in the para-aortic and mesenteric region. Subsequent sonography were normal. Last CT done in 2000 showed few small nodes in para-aortic region. Since then he has been reviewed annually with clinical examination, blood tests and USG and remained completely asymptomatic leading a healthy normal life.

Discussion

Recurrent peptic ulcerations due to Carcinoid tumours of gut is almost unknown in the extensive medical literature on carcinoid. Neuroendocrine tumours as a group are known to secrete various hormones, commonest is Gastrin secreted by Gastrinoma, a tumour that commonly arises in Pancreas and Duodenum and causes multiple ulcerations in stomach, 1st part of duodenum; and also in unusual sites like 2nd part of duodenum and jejunum and occasionally in Meckel's diverticulum, a condition known as Zollinger-Ellison Syndrome. This may be due to single or multiple gastrinoma at various sites or in association with Multiple Endocrine Tumours (MEN type I). Carcinoid tumours of the gastrointestinal tract differ in their clinical and histopathologic features, depending on the site of origin. There are only a few clinico-pathological studies that specifically describe jejuno-ileal carcinoids in association with peptic ulcer. In a retrospective study of 167 ileal and jejunal carcinoids only one had Zollinger Ellison syndrome [1].

Carcinoid is the most common endocrine gut tumour. Consistent with their endodermal origin, carcinoid tumours are historically classified according to foregut, midgut or hindgut derivation. But most carcinoid develops from embryonic midgut. A meta-analysis of 3,718 collective cases of carcinoid tumours, 45% of the tumours were found in appendix and 28% in jejunum and ileum. Conversely, 34% small intestinal neoplasms and 77% neoplasms and appendix are carcinoids. Although Meckel's diverticulum is relatively uncommon, among Meckel's diverticular tumours, carcinoid is the commonest and may occur in up to 13% of diverticula [2].

Foregut carcinoid arise in the respiratory tract, the stomach, first part of duodenum and pancreas. Midgut

carcinoids appear in the second part of duodenum through to the ascending colon and appendix, where as hindgut tumours arise in the transverse and descending colon and rectum. Bronchial carcinoid may be asymptomatic or may cause severe flushing due to its secretory behaviour and also pulmonary symptoms due to tumour causing obstruction to the bronchial tree. Flushing is usually atypical and due to secretion of Kallikrein which includes Bradykinin like peptides and hypersecretion of histamine. Midgut tumours produce serotonin and tachykinin and cause systemic symptoms of diarrhoea, flushing, wheezing, right sided valvular disease and cutaneous telangiectasia, but only after metastasis to the liver. Appendicular carcinoid is relatively benign and rarely produce serotonin. Hindgut carcinoids are known to synthesize and store several hormones and prehormones like somatostatin, tachykinin, glycentin and peptide YY [3]. But their release into the circulation is doubtful and so there is no systemic effect.

Most gut related carcinoids are diagnosed incidentally at laparotomy or when surgery is done in case of undiagnosed G.I.bleeding and bowel obstruction. Midgut carcinoids are generally advanced at diagnosis unless they are discovered incidentally. Mucosal lesions may ulcerate and present with bleeding. Bowel obstruction due to the tumour blocking the lumen of the gut is very rare as the tumours are always small in size. Obstruction develops due to fibrosis of the bowel wall and cicatrization of the surrounding tissues caused by the secretory products of these tumours [4]. CT can pick up metastatic lesions in liver and lymph nodes and a pre-operative diagnosis is possible by FNAC, but primary tumour is often missed due to its small size. Somatostatin receptor scintigraphy can also make a diagnosis of carcinoid tumour; but this requires very high degree of suspicion on the part of the clinician and when all other conventional tests do not lead to a diagnosis. Serum gastrin level estimation should be done in all cases of recurrent peptic ulcers and particularly if the ulcers are refractory to modern medical management and also in cases where ulcers are detected in unusual site of the G.I. tract. Estimation of 24 h urinary 5HIAA is generally done after a diagnosis of carcinoid is established by histopathology. Raised level is only found in cases of carcinoid syndrome and is not high in all cases.

Carcinoid tumour of the gut may occasionally present with massive gastrointestinal haemorrhage [5]. Bleeding occurs from mucosal ulceration at the site of the tumour [6] and not due to peptic ulcerations in the stomach and duodenum. In the present case there was only a tiny ulcer on the mucosa at the site of the tumour. It is most likely that the boy bled from the multiple ulcerations in the stomach and duodenum when he presented with malaena. In the pathophysiology of peptic ulcer, histamine is known to be a

causative factor and this may be the agent responsible for causing recurrent ulcers in this patient.

Many carcinoid tumours behave like benign tumours and appendicular carcinoids are almost always benign. If detected and treated early patient can have a complete cure of the disease. The size of the primary tumour determines the occurrence of metastasis. Carcinoids 1 cm or less rarely metastasize. One case reported from Japan where a 54 year old man having occult blood in stool was diagnosed to have 8 mm carcinoid in ileum with regional lymph node metastasis [7]. Larger tumours often metastasize to the lymph nodes as in the present case. Carcinoid tumours are in general very slow growing tumour and even in presence of metastasis patient can have a longer survival than adenocarcinoma of a similar stage. The present case proves this well documented fact. But the uniqueness of this case is the complete cure of peptic ulcerations and also the disappearance of the metastatic carcinoid disease of the gut.

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