



GASTRIC NEUROENDOCRINE TUMOUR A RARE PRESENTATION MURUGADASAN

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Abstract : A 50 years old male patient was admitted in our ward with the complaints of upper abdominal pain for the past 10 years and haematemesis for the past two months. Endoscopy was done and an ulceroproliferative growth was noted in the greater curvature of the stomach from which biopsy was taken and the histopathological examination revealed moderately differentiated adenocarcinoma. Subsequently contrast enhanced CT scan of abdomen was done which showed irregular circumferential wall thickening of body of the stomach with perigastric lymph node enlargement. Subtotal gastrectomy was done and the resected specimen was sent for histopathological examination. Histology revealed type IV gastric neuroendocrine tumour with lymph node metastases. This case is reported for the rarity of this tumour and for its rare histological pattern of presentation.

Keyword : Neuroendocrine tumor, stomach carcinoma, carcinoid syndrome

INTRODUCTION:

Neuroendocrine tumours arise from neuroendocrine cells found in mucosa of bronchi, stomach, ileum, jejunum, biliary tract, pancreas and urogenital tract. They secrete neuroendocrine markers and produces carcinoid syndrome. Jejunioileum is the commonest site for neuroendocrine tumours. 5% of the neuroendocrine tumours arises from the stomach. Gastric neuroendocrine tumours accounts for less than 1% of all gastric neoplasms. There are four types of gastric neuroendocrine tumours. Type I is benign and type II is benign or low grade malignant tumours. Type III is low grade malignant and type IV is high grade malignant tumour similar to adenocarcinoma. Treatment depends on the type of the tumour and the presence of metastasis.

CASE REPORT:

Mr. Selvaraj, a 50 years old male patient presented with ten years history of upper abdominal pain. He had more pain for the past two months with vomiting on and off for the past two months. He did not have fever, dysphagia, jaundice or change in bowel habits. He had loss of weight and loss of appetite. He completed antitubercular treatment one year back for pulmonary tuberculosis.

On physical examination the patient was anaemic. He did not have any generalized or supraclavicular lymphadenopathy. Examination of the respiratory, cardiovascular, central nervous system showed normal findings. On examination of the abdomen epigastric tenderness was present, there was no mass, no organomegaly, no ascites, no visible gastric or intestinal peristalsis. Per rectal examination was found to be normal. Upper gastrointestinal endoscopy was done which revealed ulceroproliferative growth involving the body and greater curvature of the stomach and biopsy was taken and sent for histopathological examination. Histology revealed moderately differentiated adenocarcinoma of the stomach. Subsequently ultrasonogram of abdomen was done which showed irregular wall thickening of body and antrum of the stomach with enlargement of multiple perigastric lymph nodes.

Contrast enhanced CT scan of abdomen was done which showed irregular circumferential wall thickening of the body of stomach with enlargement of multiple perigastric lymph nodes. There was no free fluid in the abdomen. Two units of packed cells were transfused preoperatively and the patient was taken up for surgery. Intraoperatively there was a growth in the stomach extending from the body to 2 cm proximal to the pylorus. There was enlargement of multiple perigastric lymph nodes. There was no liver metastases, ascites or pancreatic infiltration. Subtotal gastrectomy with roux-en-y gastrojejunostomy and jejuniojejunostomy was done. Feeding jejunostomy was done to improve the nutritional status. Resected specimen sent for histopathological examination. Histopathological examination revealed type IV gastric neuroendocrine tumour (mixed glandular pattern). Proximal and distal cut margins were free of tumour invasion and nineteen out of twenty seven lymph nodes showed metastatic deposits. Immunohistochemistry was positive for chromogranin A, neuron specific enolase and cytokeratin (marker for adenocarcinoma). Post operative period was uneventful. Oral feeds were started on the 8th post operative day. Etoposide 100mg and 5-fluorouracil 750mg were given for three days as post operative chemotherapy as per oncologist's opinion. Patient was discharged and advised to review after 3 weeks.

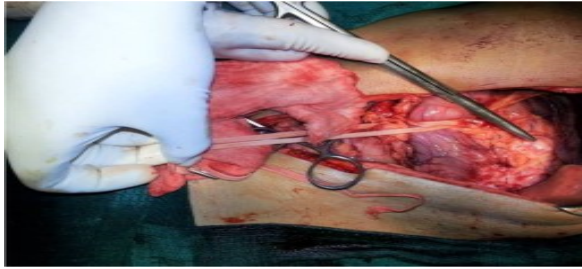


fig 1



fig 2

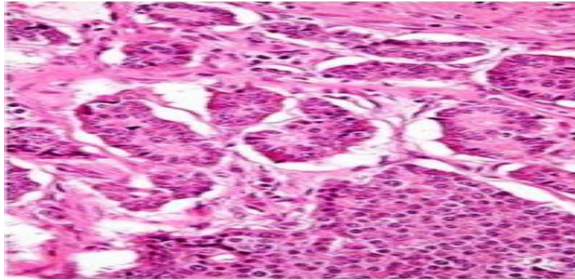


fig 3

REVIEW OF LITERATURE Neuroendocrine tumors arise from the neuroendocrine cells found in mucosa of bronchi, stomach, ileum, pancreas, biliary tree, and genitor urinary tract^[1]. Majority of the neuroendocrine tumors occur within the gastro-entero-pancreatic axis and further subdivided into foregut, midgut, hind gut neuroendocrine tumors^[3]. Only midgut neuroendocrine tumors are argentaffin positive and secrete serotonin and hence they are referred to as carcinoid tumors^[3]. 10% of neuroendocrine tumors arise from lungs, 5% arise from stomach, 2% from duodenum, 25% from small bowel, 6% from colon and 15% from rectum^[1].

The neuroendocrine tumors secrete neuron specific enolase, serotonin, chromogranin A and C, synaptophysin, insulin, growth hormone, melanocyte stimulating hormone and pancreatic polypeptides^[3]. When the neuroendocrine tumors metastasize to liver they produce carcinoid syndrome^[1].

Gastric neuroendocrine tumors account for 5% of all neuroendocrine tumors and 1% of all gastric neoplasms^[3]. Because of the modern endoscopic facilities the detection of gastric neuroendocrine tumors increases three fold^[6]. There are four types of gastric neuroendocrine tumors^[2]. Chronic atrophic gastritis type A, pernicious anemia, Zollinger-ellison syndrome with MEN-type I are the conditions associated with neuroendocrine tumors^[4]. Chromosomal abnormalities, mutation in MEN-type I gene, p53 gene mutation and chromosomal loss of 5q21 are associated with more aggressive tumors and poor survival. But majority of the tumors occur as sporadic forms^[5].

Type-I gastric neuroendocrine tumors are called ECLomas^[7]. They account for 80% of all gastric neuroendocrine tumors. The gastric mucosa contains enterochromaffin like cells.

They are usually benign, non-functional and well differentiated tumors. Elderly women are most commonly affected^[1]. They have a linear nodular pattern and are detected by routine endoscopy. Type I tumors are associated with hypergastrinemia due to atrophic gastritis^[3].

Type II tumors are benign or low grade malignant tumors. They are similar to type I tumors but the only difference is hypergastrinemia is due to gastrinomas associated with MEN-I^[4].

Type-III tumors are low grade differentiated malignant tumors. Serum gastrin level is normal. They occur sporadically and are usually solitary tumors. Tumors more than 2 cm are almost always malignant with lymph node metastases^[3].

Type-IV tumors are high grade malignant tumors with small cell types. Macroscopically they are large ulcerative malignancies similar to adenocarcinoma. Grading of gastric neuroendocrine tumors depends on mitotic rate and % of cells.

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