Case Report

A rare case of gastric carcinoid presenting as subacute combined degeneration of spinal cord

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Abstract

Gastric carcinoids originate from the foregut and are derived from enterochromaffin-like (ECL) cells, which are the main neuroendocrine cells in the gastric mucosa. Gastrin acts directly on ECL cells to induce hyperplasia, dysplasia, and, eventually, neoplasia or carcinoid. In patients with chronic atrophic gastritis, decreased gastric acid secretion stimulates the secretion of gastrin, and ECL cells transform into a carcinoid. Herein we report a case of type I gastric carcinoid presenting as SACD.

Keywords: Gastric carcinoid, Subacute combined degeneration, spinal cord.

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Introduction

Gastric carcinoids are rare tumors, representing 4% of all carcinoid tumors [1]. They are of three types of which type I gastric carcinoids are associated with atrophic gastritis, hypergastrinemia and pernicious anemia. Because of atrophic gastritis, these patients may develop vitamin B12 deficiency [2,3] The most common clinical scenario is that these lesions are found incidentally on endoscopy.

However, some patients present with nonspecific symptoms such as nausea, vomiting, dyspepsia, or with complications such as gastrointestinal bleeding. The classic carcinoid syndrome characterized by flushing, diarrhea, and right-sided heart failure present in small percentage of patient. Carcinoids typically appear as polypoid lesions or nodules with normal-appearing overlying mucosa on endoscopy [4]. The stomach is the least common site of gastrointestinal carcinoids [5].

Subacute combined degeneration of spinal cord (SACD) is a rare neurological complication of Vitamin B12 deficiency characterized by demyelination of dorsal and lateral columns of spinal cord.

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Case

A 37 year old Hindu Businessman from Jaipur presented with complaints of tingling sensations in both lower limbs for two years associated with weakness in both lower limbs and abdominal pain for one and half years. Patient was a strict vegetarian, nonsmoker, non alcoholic. On general physical examination, marked pallor was present. On neurological examination, he had reduced motor power, exaggerated knee jerk, absent ankle jerk and extensor plantar responses in both lower limbs. His gait was ataxic, high steppage type, patient looked to the ground while walking. He had loss of vibratory sensation from toes up to the D4 dermatome; proprioception and joint position sense in both lower limbs; pain, touch and temperature in L5 and S1 dermatomes on both sides and Romberg's sign was present. On investigation, he had anemia (Hb 5.6 g%), thrombocytopenia (Platelets 1.25 lakh/cumm) with raised MCV (115.1 fL). PBF showed macrocytic normochromic red cells with hypersegmented neutrophils. Vitamin B12 levels were low (104 pg/ml). MRI of spine showed ill-defined hyperintensities in posterior column at D3 to D9 vertebral levels s/o non compressive myelopathy likely SACD [Figure 1 and Figure 2]. Nerve Conduction Studies revealed axonal sensory neuropathy of Sural and Superficial peroneal

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nerves. Gastroscopy showed complete absence of rughae s/o atrophic gastritis and a fundus submucosal nodule. Biopsy of gastric mucosa showed mild chronic inflammation with one fragment showing nest of monotonous round cells of neuroendocrine origin which were positive for Chromogranin A [*Figure 3 and Figure 4*]. Serum gastrin levels were elevated to 932 pg/mL. Anti-parietal cell antibody was positive. Contrast enhanced CT scan of abdomen and octreotide

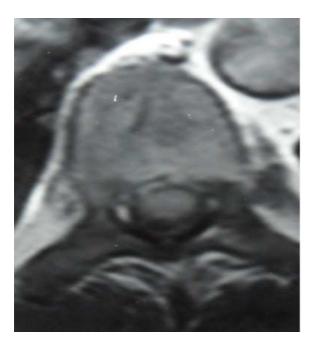
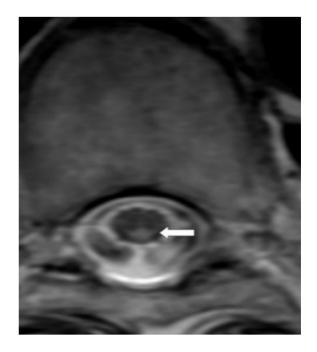


Figure 1: Axial T1 Weighted MRI Image of Thoracic Vertebra which shows no abnormality

scintigraphy was normal. Based on history, physical examination and investigations, a diagnosis of type I gastric carcinoid with chronic atrophic gastritis with pernicious anemia with SACD was kept. Patient was offered endoscopic removal of tumor and was given Vitamin B12 injections. In follow up visits, he shows a good neurologic recovery after 6 month and his serum gastrin levels are within normal limits.



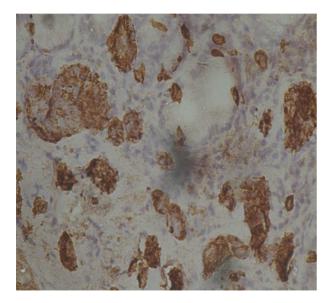


Figure 3: Chromogranin positive Carcinoid tumor cells in the biopsy specimen (40x Magnification)

Figure 2: Axial T1 Weighted Image of Thoracic Vertebra showing Hyperintensity in Posteior Column

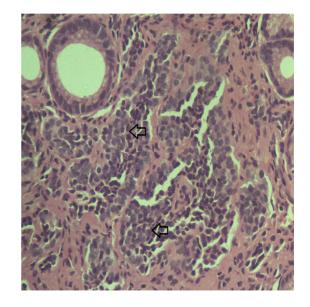


Figure 4: Arrows showing nests of round cells in the biopsy specimen (40x Magnification)

Discussion

Type I gastric carcinoids arise from ECL cell hyperplasia, which in turn, is stimulated by hypergastrinemia in the background of pernicious anemia or atrophic gastritis [6].

Autoimmune gastritis is a disorder in which antibodies are directed towards parietal cells, resulting in their destruction. This destruction causes hypochlorhydria and intrinsic factor deficiency leading to Vitamin B12 deficiency and macrocytic anemia [7].

A loss of negative feedback mechanism causes increased production of gastrin from antral G cells resulting in hypergastrinemia. As stated previously, gastrin itself stimulates ECL proliferation.

The natural history of type I gastric carcinoid is rather indolent. The tumor tends to remain small and localized in stomach and incidence of metastasis is <5% [1]. Gastric carcinoids present with GI complaints in the form of nausea, vomiting and abdominal pain.

The typical carcinoid syndrome characterized by diarrhea, flushing, bronchospasm and right heart failure is rare in gastric carcinoids [8].

Type I gastric carcinoids are associated with atrophic gastritis and Vitamin B12 deficiency [2]. However SACD is rare and from our search of literature only one case has been reported as yet.

Conclusion

Gastric carcinoids are rare tumour. Type I gastric carcinoid are associated with atrophic gastric which may cause vitamine B12 deficiency anemia and rarely neurological manifestation like SACD which is reversible with B12 administration. Type I gastric carcinoid has indolent course and rarely metastasized.

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