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Case report

SYNCHRONOUS POORLY DIFFERENTIATED GASTRIC ADENOCARCINOMA WITH GASTROINTESTINAL STROMAL TUMOR: A CASE REPORT

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ABSRACT

Gastrointestinal stromal tumor (GIST) is categorized as a mesenchymal tumor. In the abdomen more than half occur in the stomach. Adenocarcinoma is the most common epithelial malignancy of stomach comprising over 90% of all gastric cancers. The simultaneous occurrence of both these tumors together is rare. This is an interesting case report of a 54 year old lady with synchronous occurrence of GIST and poorly differentiated adenocarcinoma of intestinal type. A brief review of literature is done regarding the reported cases and proposed hypothesis.

Keywords: Poorly differentiated carcinoma, GIST, Synchronous

INTRODUCTION

The most common gastric tumors are epithelial tumors. Adenocarcinomas constitute the most common type of epithelial gastric tumors. Gastrointestinal stromal tumors (GIST) are non epithelial tumor which can occur in the stomach. In the gastrointestinal tract 1% of all malignancies[1, 2] and 5.7% of sarcomas[3] are accounted by GIST. GISTs and adenocarcinomas have two separate histogenesis. It is extremely rare for the co-existence of GIST and adenocarcinoma. GISTs have been reported in the literature to coexist with tumors of different histogenesis such as adenocarcinomas, carcinoids, MALT lymphomas and Burkitt's lymphomas[4,5,6], as well as with different mesenchymal tumors.[7-13] Here is a case report of a 54 year old lady with poorly differentiated adenocarcinoma and synchronous gastrointestinal stromal tumor which was incidentally detected.

CASE REPORT

A 54 year old lady was admitted with vomiting, following food intake, of 3weeks duration. There was associated abdominal discomfort and belching. She gave a history of loss of appetite and weight loss which was of 3 months duration. On general examination the patient was emaciated and pale. The systemic examination was unremarkable. Blood routine was normal except for the low hemoglobin level (5gm%). Urine routine was normal. Ultrasonograghy showed diffuse thickening of the gastric pyloric antral wall.

Oesophagogastroduodenoscopy showed multiple ulcerations with hypertrophied margins in the lesser curvature and antrum and the impression was gastric outlet obstruction. Endoscopic biopsy done showed microscopic features of a poorly differentiated carcinoma. A lower radical gastrectomy was done and the specimen was received in the histopathology lab. An ulcerating infiltrating neoplasm measuring 30x25mm was detected in the lesser curvature adjacent to which another nodular firm grey white mesenchymal neoplasm measuring 25x20mm was also

noted [Figure1]. The microscopy of the ulcerating tumor showed features of a poorly differentiated intestinal type adenocarcinoma [Figure 2]. The tumor was infiltrating the full thickness of the gastric wall and extended to the serosal fat of the lesser curvature. Four out of nine lymph nodes showed evidence of metastasis.

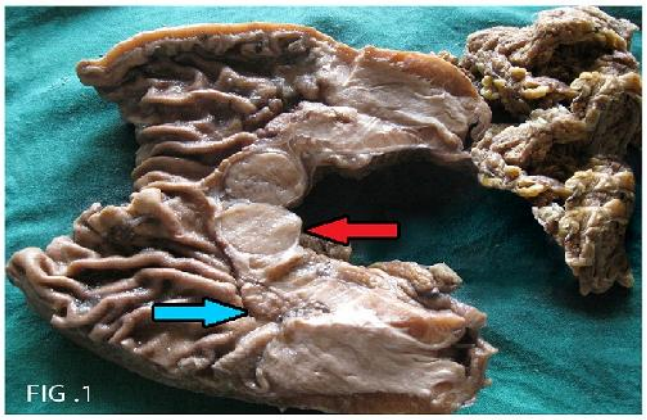

Fig 1: Ulcerating infiltrating gastric carcinoma in the lesser curvature of stomach (blue arrow) with adjacent nodular firm grey white gastrointestinal stromal tumor (red arrow)

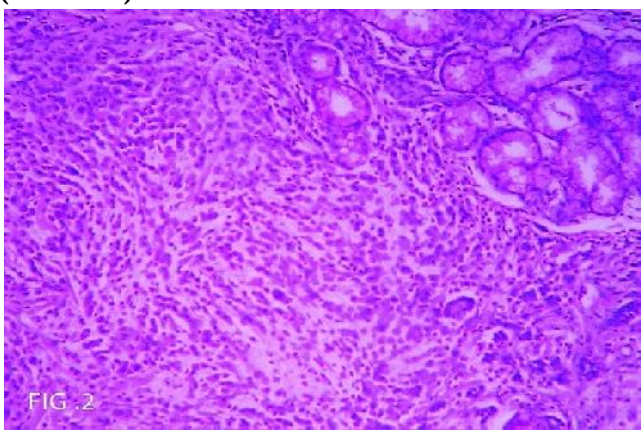

Fig 2: Microscopy of poorly differentiated intestinal type adenocarcinoma in the gastric wall (H&E X 10x)

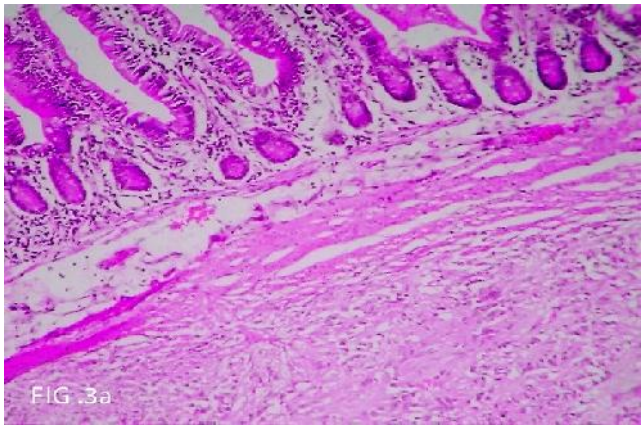

Fig 3a: Microscopy of circumscribed submucosal spindle cell gastrointestinal stromal tumor (H&E X 10x)

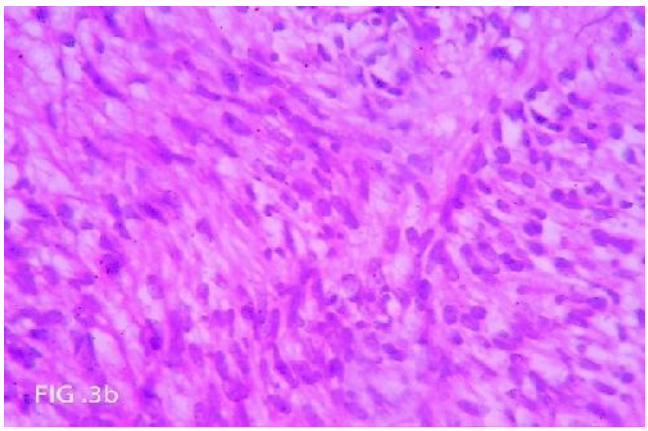

Fig 3b: Microscopy of gastrointestinal stromal tumor showing the sheet- like and fascicular arrangement of spindle shaped cells (H&E X 40x)

The adjacent nodular firm white neoplasm showed microscopic feature of a submucosal spindle cell mesenchymal neoplasm which was circumscribed. A fascicular and sheet like arrangement of plump spindle shaped cells were noted [Figure 3a,b]. Mitotic activity was less than 5/50 HPF. No areas of necrosis were found. Based on the mitotic count of less than 5/50 HPF and size of the tumor less than 5 cm, the histological diagnosis was GIST-low risk type. Immunohistochemical markers were advised for confirmation of GIST.

Immunohistochemical markers were done for the identification of origin of the tumors. The mesenchymal neoplasm was CD 117 (c-kit protein) moderately to strongly positive demonstrating a combined membranous and diffuse cytoplasmic staining pattern. Additionally, CD 34 protein was observed to be membranous stain positive, whereas S-100, desmin and SMA demonstrated negative or very weak reactivity. The poorly differentiated intestinal type adenocarcinoma was cytokeratin positive. A final report of synchronous poorly differentiated intestinal type adenocarcinoma and GIST (low risk type) was arrived. Since intestinal type adenocarcinomas could be due to Helicobacter pylori they were searched in the gastric mucosa but were not detected microscopically.

The resected gastric margins and omentum were free of neoplasm. The postoperative course was uneventful. Following surgery the patient received adjuvant chemotherapy, but unfortunately died of progressive disease 16 months later.

DISCUSSION

GIST was named in the earlier literature as leiomyomas, schwannomas, leiomyosarcomas and leiomyoblastomas. Electron microscopy and immunohistochemical stains recognized it as a distinct entity.[14] Mazur and Clark[15] introduced the term GIST in 1983.

The synchronous occurrence of GIST and gastric carcinomas are rare. A few reports of simultaneous presence of poorly differentiated adenocarcinoma and GIST have been reported.[7-13] Most of the adenocarcinomas were detected after endoscopic biopsies. GIST was diagnosed as an incidental finding. No high risk types of GIST have been reported in association with gastric carcinoma .The tumors were mostly less than 5cm.Recently Karahan N et al.[16] have reported in a neurofibromatosis type-1 patient with development of simultaneous multiple GIST and signet ring cell carcinoma. The synchronous occurrence of these two tumors has excited many and it raises the question as to why they occur together.

The reason for the simultaneous origin of GIST and adenocarcinoma may be due to coincidence. Gene mutations were another reason that was proposed. Recently Yan Y et al [17] conducted molecular analysis and clinicopathological profile of KIT/PDGFRA in both these tumors. No relationship was obvious according to this study.

H. pylorus is another cause that may be considered. H. pylori can cause simultaneous development of gastric carcinoma and lymphoma [7.] Such a relationship with GIST is not proved yet. In the present case study no H. pylori could be detected.

Another hypothesis is the role of carcinogenic agent. It may act on neighboring tissues and may lead to the development of tumors in the same organ with different histogenesis.[18, 19]

CONCLUSIONS

The synchronous occurrence of a GIST with gastric carcinoma is rare, and little is known about this association. Coexisting GISTs are in most cases small, asymptomatic tumors and are detected incidentally during surgery for gastric carcinomas. Hence specimens should be handled cautiously to detect associated lesions. Since most of the cases were poorly differentiated adenocarcinomas further studies are needed to know whether the associated GIST influenced the differentiation of the tumor. Molecular studies are also further needed to explain the simultaneous development of tumors of different histogenesis.

Disclosure of conflicts of interest: The author declares that there is no financial relationship with any organization in this case study and that there is no conflict of interest.

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