

SIGNET RING CELL GASTRIC CARCINOMA- A CASE REPORT

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PRESENTATION OF THE CASE

This 39-year-old female patient came to surgical outpatient department with the complaints of non-radiating upper abdominal pain and non-bilious vomiting for 1 week duration. There was history of loss of appetite, loss of weight and melena for past 1 month.¹⁻³ On examination, the patient was found to be poorly nourished, dehydrated and pale. There was no icterus/ no pedal oedema and no significant supraclavicular lymphadenopathy.

Blood investigations showed Hb as 10.3 g/dL, PCV- 34%, WBC- 6,400 cells/cmm, Platelets- 2,13,000 cells/cmm and ESR- 02 mm. The blood grouping was A positive. Serum electrolytes assessment showed sodium levels of 135 mEq/L, Potassium- 3.9 millimoles/lit, Chloride- 103 mEq/L and Bicarbonate levels of 26 mEq/L. Renal function tests revealed Urea level as 27 mg/dL and Serum creatinine level as 0.9 mg/dL.^{4,5,6}

Upper gastrointestinal scopy done in this patient showed an Ulcerative growth involving the whole circumference of the antrum from the incisura to 1 cm start-up pylorus and lumen appeared narrowed (Figure 1).

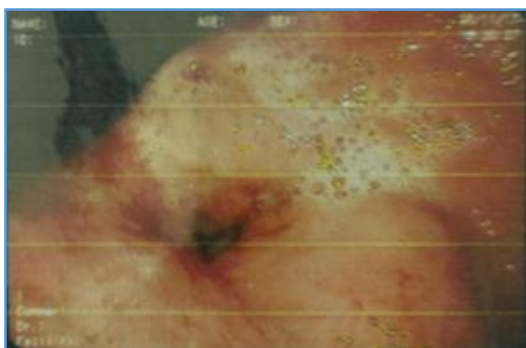


Figure 1. Upper Gastrointestinal Scopy showing Proliferative Growth in the Antrum

Multiple biopsies taken from the proliferative growth and sent for histopathological examination. Biopsy findings revealed poorly differentiated Adenocarcinoma involving antrum. Medical oncologist's opinion was obtained.

USG Abdomen showed gastric antral wall thickening of length 1.7 cm. Liver was normal and there was no free fluid. No evidence for distant spread. CECT abdomen and chest revealed circumferential wall thickening noted in antropyloric region extending for a length of 6.6 cm with a wall thickness of 2 cm causing lumen narrowing. There was no retroperitoneal involvement and no perigastric nodes.^{7,8} Preoperative workup was done. Patient was taken up for D2 subtotal gastrectomy.

Figure 2 shows intraoperative findings. There was gastric antral wall thickening and proliferative growth in the antrum.



Figure 2. Intraoperative Findings showing Proliferative Growth and Antral Thickening

Figure 3 shows the excised growth sent for histopathological examination. Histopathology showed focus of intramucosal signet ring cell carcinoma invading the lamina propria (T1a) (A) and Signet ring cell carcinoma (T1a) invading muscularis propria. Tumour cells had predominant cytoplasmic mucin and eccentrically placed crescent-shaped nucleus.

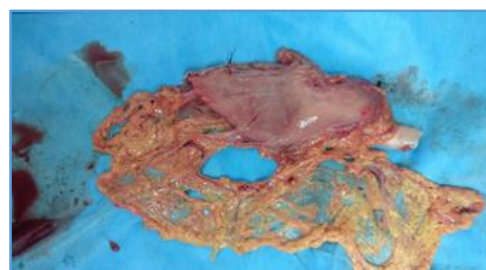


Figure 3

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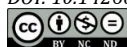
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Post-operative status of the patient was found to be uneventful.

DIFFERENTIAL DIAGNOSES

1. Acute Gastritis.
2. Chronic Gastritis.
3. Atrophic Gastritis.
4. Gastric Lymphoma.
5. GIST.
6. Peptic Ulcer Disease.
7. Gastric Outlet Obstruction.

CLINICAL DIAGNOSIS

Abdomen examination revealed that there was no tenderness, no mass, no free fluid and no visible gastric peristalsis. With this history the provisional diagnosis was made, as this is a case of gastric malignancy with gastric outlet obstruction.

PATHOLOGICAL DISCUSSION

In this study, we are reporting about a 39 years old female patient presenting with the complaints of non-radiating upper abdominal pain and non-bilious vomiting. There was loss of appetite and weight. Clinical examination revealed that the patient was pale. There was no tenderness, no mass, no free fluid in the abdomen and no visible gastric peristalsis. Upper GI scopy showed ulcerative growth in the antral region.^{9,10} Subtotal gastrectomy was done in this patient. Histopathological examination reported Signet ring gastric carcinoma.

Similar case has been reported from Nigeria¹¹ where an 84-year-old local lady presented with upper, central abdominal ache of months duration with history of vomiting, loss of weight and loss of appetite. Scopy findings revealed that the prepyloric and pyloric portions of the stomach were completely taken over by a huge amorphous, fungating mass which had multiple ulcerating areas and had led to the near complete destruction of the normal architecture. The corpus and fundal regions were free of abnormality. The histopathology report showed the antral lesion came back as Signet ring cell carcinoma.¹¹

As per WHO's classification, Signet ring cell carcinoma is defined as a poorly cohesive carcinoma composed predominantly of tumour cells with prominent cytoplasmic mucin and a crescent shaped nucleus eccentrically placed.¹² SRCC is the most aggressive in nature. A California-based study found that SRCA were more likely to be found in women and in the distal stomach.¹³

SRCC has a specific oncogenesis that differ from that of tubular gastric adenocarcinoma. Two main pathologic processes at a cellular level are loss of cell-cell adhesion molecules and accumulation of mucin in large vacuoles. E-cadherin which is encoded by CDH1 gene is a cell-cell adhesion molecule and seems to play a key role in carcinogenesis. Other adherence molecules also involve such as somatic mutations of β -catenin/ APC genes or dysregulation of the Wnt/ β -catenin pathway. In SRCC, accumulation of mucins like MUC1, MUC2, MUC4, MUC5AC, MUC6 results in large vacuoles, which could therefore play a role in carcinogenesis. MUC4 has been reported to increase activation of ErbB2/ErbB3 complexes. Finally, hormonal theory in which oestrogen is involved in tumour initiation or

progression or both has been developed to explain the increased incidence in women of SRCC.

DISCUSSION OF MANAGEMENT

SRCC is highly resistant to chemotherapy and radiotherapy. Paradoxically, SRCC is more frequent in early gastric cancer than in advanced gastric cancer. High chances of recurrence have been reported with SRCC. Better survival rates have been reported for SRCC than for non-SRCC tumours.¹⁴

FINAL DIAGNOSIS

This is a case of Signet ring gastric carcinoma.

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