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Research Article

A RARE CASE OF GASROINTESTINAL STROMAL TRUMOUR PRESENTING AS MESENTRIC CYST

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A R T I C L E I N F O

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A B S T R A C T

A gastrointestinal stromal tumor commonly occours in gastrointestinal tract of stomach or intestine. The tumour cells are thought to arise from specialised cells found in gastro intestinal tract for interstitial cells of cajal(ICCs) or precursors to these cells. GISTs are usually found in adults between 40 and 70; rarely, children and young adults develop these tumours.

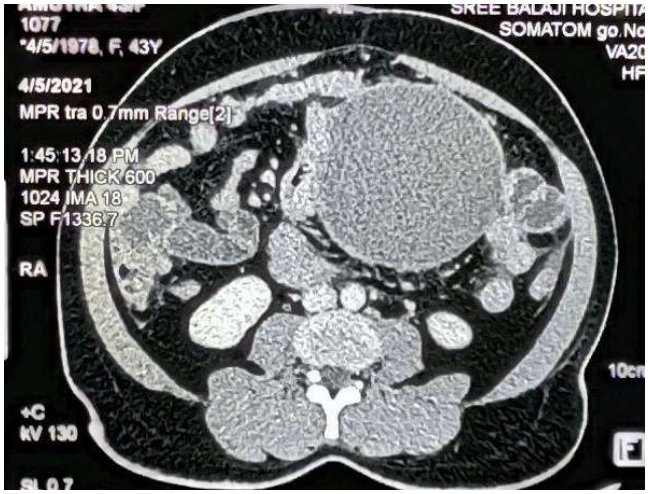
INTRODUCTION

GIST is most common mesenchymal tumour of gastro intestinal tract but accounts for less than 1% of all primary tumours found in GIT. After surgical resection of tumour there is risk of local serosal spread with metastasis to liver[1].Incidence of GIST of is 6.5-14.5 per million population .It is equally found in males and females. It can occur at any age but mean is around 62 yr . The tumours can be malignant or benign. Small tumours may cause no signs or symptoms. However some people with GIST may experience pain or swelling in the abdomen, nausea, vomiting, loss of weight, loss of appetite. Sometimes tumour cause bleeding which may lead to low RBC count (anaemia) and consequently weakness and tiredness. bleeding into the interstitial tract may cause black and tarry stools and bleeding into the throat or stomach may cause vomiting of blood. Affected individuals with no family history of GIST typically have only one tumour (called a sporadic GIST) people with a family history of GIST (familial GIST) often have multiple tumours and additions sign or symptoms including non-cancerous over growth (hyperplasia) of other cells in the GI tract and patches of dark skin on various area of the body

CASE REPORT

A 43 yrs old lady presented with chief complaints of lower abdominal pain for 1 month.pt developed pain on left side radiating to right. It was dull aching, continuous, mild to moderate in intensity no aggravating or relieving factors. H/o of nausea seen for 1 week. No h/o vomiting O/E-patients vitals stable general condition was fair. Pallor +, B/L pedal oedema Single mass palpable of size 15*15 cm over the umbilicus, suprapubic and LIF region. The mass is firm, no free fluid palpable.

CECT CONTRAST showed large well defined thin walled abdominopelvic cyst measuring 15.8*11.3*14.1cm predominantly on left side occupying pubic, umbilical, left iliac and left lumbar region. A portion of jejunal loop is seen related to superomedial aspect. Right inferolateral wall of cyst not defined suggestive of site of rupture .E/O rupture mesenteric cyst.



Patient was posted for emergency laparotomy and proceed.
Intra op findings:

[1]asctic fluid + in POD[2]15*15 cm cyst present in mesentery of jejunum adherent to 2 loop of jejunum [3]cyst wall ruptured with haemorrhagic fluid inside measuring around 2

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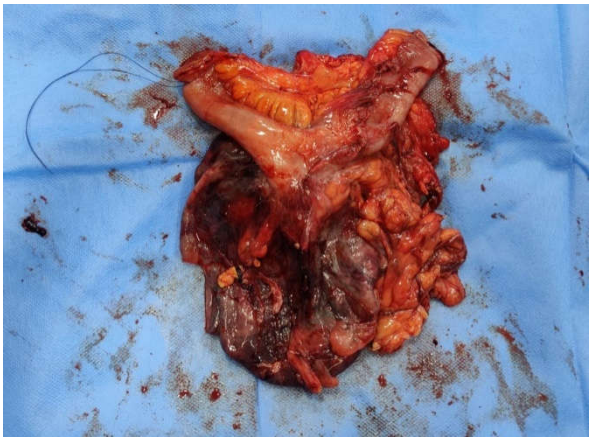
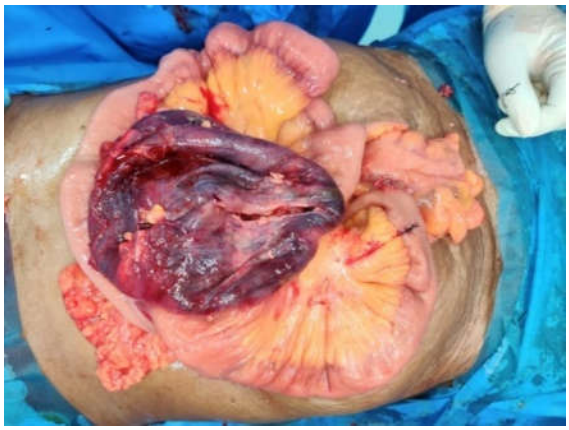
litres [4]multiple adhesion present due to previous laproscopic pps [5]1*1 cm fimbrical cyst in left ovary remaining structures found to be normal

Procedure: Resection of Jejunal Gist with Side To Side Jejunojejunal Anastomosis





HPE revealed spindle shaped cells arranged in broad fascicles. Cells have neoplastic pleomorphic hyperchromatic nuclei with moderate eosinophilic cytoplasm. no omental deposits. Mitosis > 5/50 HPF. Lymph node showed reactive changes. Highly suggestive of Gastrointestinal Stromal Tumours (Spindle Type).

IHC was positive for DOG-1, CD117, SMA in favour of GIST. Patient was started on IMATINIB 400MG.

Postoperative period was uneventful. Patient was discharged on day 10.

DISCUSSION

Most of GIST are sporadic cases but sometimes can be associated with clinical syndromes: most common is neurofibromatosis 1.Most common sites are gastric, intestinal, rectal, extra GIT (mesentery, omentum, retroperitoneum, oesophagus)[8]. GIST are mainly formed due to mutation in c-kit receptor, a proto-oncogene, mutation forming activated c-kit proteins. C-kit is responsible for proliferation and development of interstitial cells of Cajal (ICC) for normal intestinal peristalsis[4]. PDGFRA mutation is seen in gastric GIST.C-kit shows exon 11 frame deletion and exon 9 duplication .This is essential to determine the response to Imatinib. Diagnosis is confirmed with histopathology and IHC (positive for CD117, DOG-1,SMA,CD34).

Treatment of GIST is complete resection (Ro) followed with adjuvant Imatinib. In case of large GIST neoadjuvant Imatinib is tried before resecting[13]. Imatinib is tyrosine kinase inhibitor. Prognosis is based on mitotic rate and tumour size(risk stratification). It is divided into very low risk(<2cm,<5/50 HPF), low risk(2-5 cm,<5/50 HPF), intermediate risk(<5 cm,6-10/HPF or 5-10cm,<5/50 HPF), high risk (>10cm,>10/50HPF)[11]. The specific *KIT* exon in which the GIST mutation resides affects the molecular and clinical phenotype. Exon 13 shows s response to Imatinib while exon 9(found in small bowel GIST) is resistant to Imatinib[15].Other TKI used are sumatinib and regorifab in cases resistant to imatinib. Imatinib was associated with overall improvement in the survival rate and better disease free recurrence[17].Response to adjuvant therapy is determined by Modified CT Response Evaluation suggested by CHOI *et al* following resection. GIST is graded based on tumour density and size[14].In one study,1 year of adjuvant imatinib was compared with placebo. It showed significant improvement in the recurrence-free survival after 1 yr therapy versus placebo (98% versus 83%, respectively)[17]. In another follow-up study demonstrated a persistent improvement in recurrence-free survival but did not demonstrate any improvement in overall survival[19].In a separate trial, patients were randomized to 1 versus 3 years of adjuvant imatinib after resection of c-kit–positive GIST. The 3-year duration of therapy was associated with improvements not only in recurrence-free survival but also in overall survival. These trials showed even after 3 yr Imatinib therapy nongastric GIST and high mitotic index have bad prognosis.

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