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Colonic inflammatory fibroid polyp: A differential for gastrointestinal stromal tumour

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Abstract
Inflammatory fibroid polyp (IFP) presents as a polypoidal finding protruding into the gastrointestinal tract lumen. On the basis of HPE study IFP is differential for GIST. The line of management changes on the basis of IHC report. IFP at colonic location found rarely.

Keywords: Inflammatory fibroid polyp (IFP), gastrointestinal tumours (GISTs), Histopathological examination (HPE), Immunohistochemistry (IHC).

Introduction

Case presentation

A 52-year-old female presented with cramping abdominal pain and hematochezia for 5 months. Patient admitted 5 years back for abdominal pain and became asymptomatic thereafter. There was no history of vomiting, constipation, melena, jaundice. Clinical examination of the patient was within normal limit. Per abdomen was soft, nontender, no guarding, no rigidity & bowel sounds were present. No palpable mass felt per abdomen. In per rectal examination yellow coloured faeces present & palpable growth felt.

Colonoscopy showed lumen occluding polypoidal growth 5x5cm with necrosis and haemorrhagic lesions in descending colon (Fig1). Ultrasound of abdomen was suggestive of approximately 5*4cm hypoechoic solid growth in descending colon with colo-colic intussusception without intestinal obstruction. CECT (A+P) was suggestive of either a neoplastic lesion or a lymphoma involving the wall (thickness 18-20mm) of distal descending colon of length 7cm.Patient underwent laparotomy. Intraoperative decision of left hemicolectomy was taken because polypoidal growth was hard in consistency.

Microscopic examination (Fig2) revealed ulcerated intestinal mucosa and tumour mass. Tumour is comprising of sheets of cells with ill-defined clear and eosinophillic cytoplasm and oval to spindle bland nuclei. Bands of collagen are seen between the tumour sheets. No e/o cellular atypia, increased mitosis. On the basis of HPE study diagnosis was given low risk GIST. IHC study was advised for confirmation of diagnosis. Further IHC workup with CD34, SMA (patchy) (Fig 4), CD10, Cyclin D1 were positive and suggestive of IFP. CD117 (kit), DOG1 & CD35 were negative in IHC study. Patient discharged after IHC report confirmation and advised followup.

Discussion

IFPs were first described in 1920 as smooth, solitary, submucosal proliferating growths with inflammatory eosinophilic and fibroblastic infiltration [1]. In 1952 Kofler reported the first colonic IFP case [2]. IFP is rare; clinically and histologically benign condition [3]. IFP might be associated with Helicobacter infection or Type A gastritis [4]. IFP represent true neoplasms which are driven by activating mutations in the PDGFRA gene [5]. Trauma, allergic reaction, genetic tendency, bacterial, physical, chemical and even metabolic stimuli have been suggested as initiators and etiological factors for IFPs. It could be a consequence of chronic irritation, inflammation, extreme reaction of the body to an intestinal trauma or a localized variant of eosinophilic gastroenteritis with marked increased eosinophilic infiltration [4]. They are mostly found in the stomach (70%) and the small intestine (20%). Colonic IFPs are rare [6]. They are most commonly located in the proximal colon, especially in the cecum [7].

They can be sessile or pedunculated. Biopsies can be challenging because the epicenter of the lesion is often in the submucosa and the polyp is often covered by epithelial mucosa [6]. Microscopically, IFPs exibit vascular & fibroblastic proliferation accompanied by an inflammatory response which is predominantly eosinophillic in nature is characteristic [8]. IFP are clearly distinct from gastrointestinal stromal tumors by their morphology, submucosal origin and clinical behaviour, although both entities share common mutational subtypes of the PDGFRA gene [9]. Using immunohistochemistry studies, spindle cells of IFPs are generally positive for CD34, α-SMA [10]. The spindle cells of IFPs are negative for KIT, DOG1, S100 protein, ALK, nuclear β-catenin and STAT6 [11]. GISTs is the major differential diagnosis of IFPs on the basis of HPE study. Here, immunohistochemistry plays vital role to rule out other conditions and to make definitive diagnosis.

All spindle cell type GISTs express KIT strongly and DOG1 also. CD34 shows over 90% of gastric spindle cell type GISTs. GISTs other than gastric origin do not apparently express CD34. Expression of CD34 is inversely related to that of α-SMA in GISTs. Thus, positive rate of α-SMA is not so high in gastric GISTs, but it is high in GISTs other than gastric GISTs [11].

The existence of c-kit-negative GIST points to the need for additional markers, including PDGFRA (6), PKC-theta (5,13) and DOG-1 [12]. In our case, immunohistochemistry is negative for DOG1 gene.

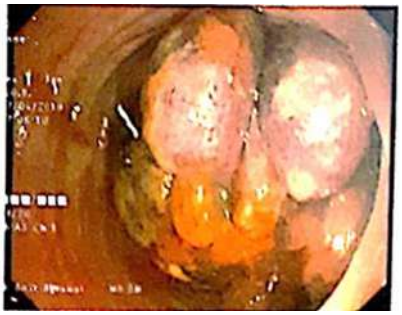

Fig 1: Colonoscopic polypoidal growth in colon

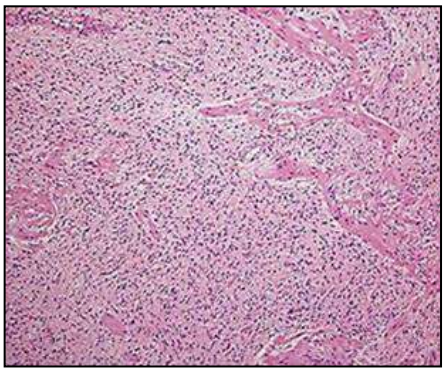

Fig 2: Eosinophillic cytoplasm & oval to spindle bland nuclei (HPE)

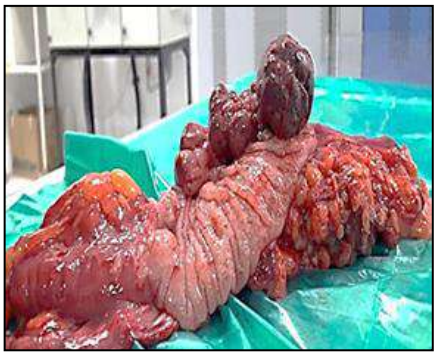

Fig 3: Macroscopic image (resected specimen)

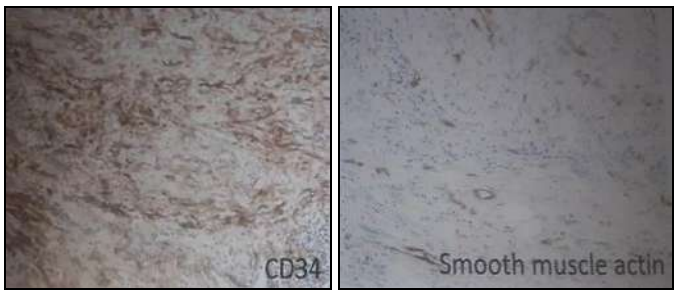

Fig 4: CD34&SMA positive (IHC study)

Table 1: Differentiating factors between IFPs & GISTs [13]

Inflammatory Fibroid Polyp	GIST (spindled,bland)
Submucosal	Intramural
Spindled and stellate cells	Spindled but no stellate cells
Abundant stromal eosinophils	Eosinophils infrequent
Perivascular concentric cuffing common	Lacks concentric cuffing
Fibromyxoid background with regular vascular pattern	May be myxoid but lacks regular vascular pattern
CD117 negative	CD117 74-95%
DOG1negative	DOG1 87-94%

Both are frequently CD34 positive

In clinical presentation tumours situated above the level of the ligament of treitz are usually asymptomatic. Patient may present with abdominal discomfort, upper abdominal pain, vometing and bleeding. Tumours situated below the ligament of treitz usually present as acute colicky abdominal pain, lower GI bleeding. In certain cases it presents as complicated intussusception.[14]Once definitive diagnosis made, line of management is decided. As IFPs is benign neoplasm. Removal of polypoidal growth either endoscopically or surgical intervention is done. And it is sufficient. Patient symptomatically relieved.

In GISTs proven cases on the basis of high mitotic activity, tumour size, tumour site & KIT gene mutation; GISTs classified as low risk and high-risk category. The standard treatment of localised GISTs is complete surgical excision of the lesion, with no dissection of clinically negative lymph nodes. R0 excision is the goal. R1 (microscopically positive) margins acceptable for low-risk lesions. If R1 excision was already carried out, re-excision may be an option. As per histological profile patient with intermediate, moderate or high risk and those with R1 & R2(microscopic & macroscopic tumour residue) or tumour rupture should receive long term adjuvant therapy with Imatinib [15].

Thus, in differential diagnosis of IFPs; IHC study plays vital role to rule out GISTs. As if IHC study suggestive of GISTs only surgical intervention does not sufficient. Adjuvant Imatinib therapy has to be given to lower recurrence of GISTs. And hence it affects overall survival of patient.

Conclusion

HPE findings of spindle cells in eosinophilic cytoplasm creates confusion between IFP and GIST. IHC study is mandatory to decide between the two and hence the line of management.

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