clinical signs of shock such as hypotension, tachycardia or bradycardia, no heart rate, or loss of consciousness are present, or when one or both of the following groups of symptoms are also present: (1) itching, erythema, urticaria, angioedema and (2) laryngeal edema or spasm or bronchospasm.[10]

Haeney[2] reported on a patient who repeatedly developed angioedema and urticaria two hours after ingestion of omeprazole 20-mg capsules but not after use of enteric-coated granules without the capsule shell (which in Europe consists of gelatin, red iron dioxide, and titanium dioxide), suggesting a causal relationship with the capsule shell and not with omeprazole. The triggering constituent was not identified.

Bowl by and Dickens [3] reported a patient who developed angioedema and urticaria immediately after taking oral omeprazole 20 mg, which was confirmed by rechallenge. Challenge with omeprazole granules without the capsule shell was also positive, which suggests an allergy to the drug and not to the capsule.

Galindo et al.[5] reported on a patient who developed anaphylaxis a few minutes after infusion of omeprazole 40 mg; evidence of cross-reactivity was provided by skin tests to omeprazole and lansoprazole.

The available evidence indicates that the PPIs currently in use can cause anaphylactic reactions. These benzimidazole derivatives are chemically related, observations in a few patients suggest that cross-sensitivity may occur. However, further study is needed to provide more precise information regarding the frequency of anaphylactic reactions during the use of these drugs. Because anaphylaxis is a potentially serious reaction, healthcare professionals need to be aware when prescribing these agents that PPIs can occasionally cause anaphylactic reactions.

Conclusion

This case report suggests that the PPI can cause anaphylactic reactions and that one should be aware of its life threatening adverse reaction while prescribing PPIs.

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Giant villous adenoma of rectum mimicking an infiltrating adeno-carcinoma

Introduction

Giant villous adenoma (GVA) is a rare mass forming lesion of the gastrointestinal tract, which is often difficult to differentiate

from an infiltrating adenocarcinoma based on clinical presentation and radiological investigations. These lesions however can be suspected in an elderly patient if a gastroenterologist is aware of its typical location and presentation, with knowledge that conventional radiological investigations may not give sufficient information regarding its benign nature. If diagnosed peroperatively endoscopic or laparoscopic limited excision procedures can reduce morbidity of extensive surgical resection. Herein, we report a case of a GVA with literature review, citing examples, where they were mistaken for an invasive carcinoma.

Case report

This 55 year old male, presented with history of passing blood and mucus in stool along with weakness since last one year. There was no history of pain during defecation or alteration of bowel habits. No significant family or past history of such illness was there. On clinical examination his performance status was ECOG 2 with pallor and anasarca. Digital rectal examination revealed an ulcero-proliferative growth starting at 1 cm from the anal verge on left lateral wall of rectum and becoming circumferential at 4 cm, with no palpability proximal to it. Growth was mobile and gloved finger was stained with blood. Rest of the systemic examination was unremarkable. Laboratory investigations showed significant anaemia (Hemoglobin 5.2 gm/dL) and hypo-albuminaemia (Albumin 1.2 gm/dL) with rest of the parameters within normal limits, including electrolyte levels. Colonoscopy revealed a circumferential polypoidal growth starting at 1 cm from anal verge and extending up to 10 cm proximally. Computed tomography scan (CT) of abdomen showed a bulky soft tissue mass with ulceration, arising from anal canal and rectal wall extending from anal verge to recto-sigmoid junction [**Figure 1**] with normal liver and no

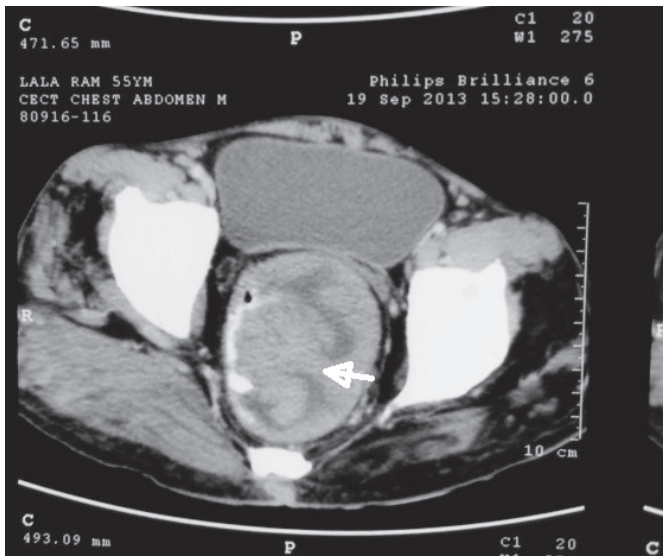

Figure 1: Computer tomogram (CT) of abdomen shows a bulky soft tissue mass arising from anal canal and rectal wall extending from anal verge to recto-sigmoid junction. polypoidal stalk is identified (arrow).

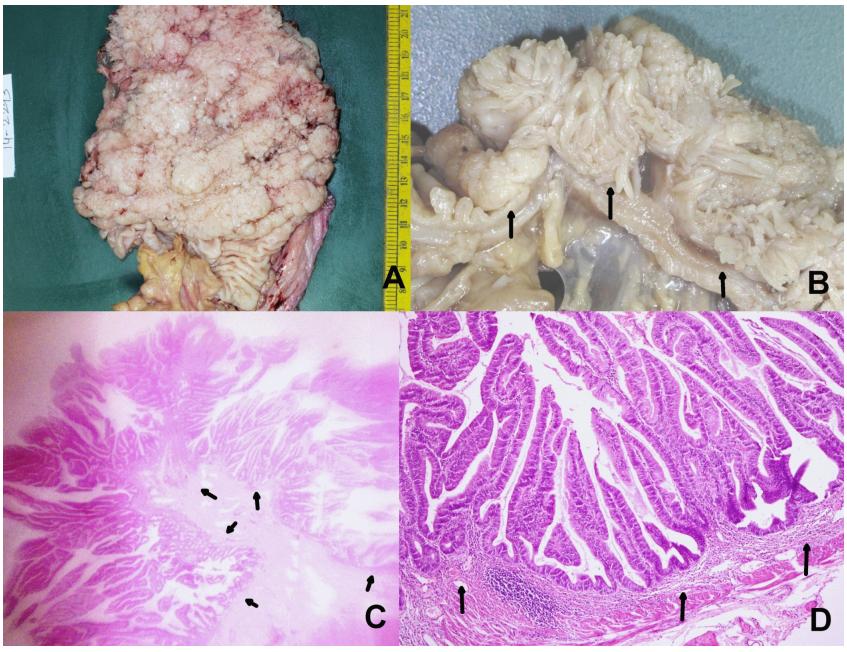

Figure 2: Gross photographs show a large adenoma with villous surface, filling the whole rectal lumen. Cut surface shows no define invasion as the boundary between the lesion and underlying subcutaneous tissue and muscularis propria is clean (arrows) [Figures 2A & B, gross]. Photomicrographs show a villous adenoma with low grade dysplasia and no evidences of stromal infiltration (arrows) [Figs 2C, H& E x 20; Figure 2D, H&E x 40].

ascites.The lesion was biopsied twice and in both occasions superficial fragments from a villous adenoma (VA) with low grade dysplasia were reported. However as clinical and radiological suspicion of an adenocarcinoma was strong an abdominoperineal resection (APR) was performed. Grossly a 14 centimetres (cm) long APR specimen was received and there was a large villiform lesion measuring 8.5x6x4 cms, lying 5cm away from the proximal resection margin [**Figure 2A & B**]. The cut surface showed a polypoidal lesion comprising predominantly of villous structures. Grossly infiltration into underlying layers was not identified. Multiple sections examined from all over the lesion showed a villous adenoma with low grade dysplasia. No infiltration into muscularis mucosae or stalk was noted. No displaced glands were identified. No lymph node was found in the APR specimen [**Figure 2**]. Based on overall features diagnosis of a giant villous adenoma with low grade dysplasia was suggested. The patient is under regular follow up and is now asymptomatic.

Discussion

Though, it has been stressed in the published literature that, while the proximal colonic adenomas are polypoidal, and the distal ones are sessile, this is not always true. While most of the right colonic lesions present with features of anaemia, the left colonic lesions show altered bowel habit, blood in stool and intestinal obstruction. Amongst the adenomas, VA is least common (5-10%) than the tubular (70%) and tubulo-villous adenomas (10%). Though the VAs are Commonly 2-4 cms in size, rarely they can be more than 5 cm in size and even can be upto 18 cms in size [GVA].[1-3] Giant polyposis causing intestinal symptoms have also been described in inflammatory bowel diseases (IBD), especially in ulcerative colitis.[4] The GVAs are usually seen in the fifth decade and present with features of secretory diarrhoea and electrolyte loss.[1,2] Rarely rectal bleeding can be seen when it is large and ulcerated.[2] Rare reports of GVA presenting as McKittrick–Wheelock syndrome are there, characterized by large volume diarrhoea, sever electrolyte dysfunction and pre-renal acute renal failure.[3] Occasionally irreducible rectal prolapse can be a complication of GVA. The index case presented with diarrhoea admixed with mucus and blood and there was radiological suspicion of an infiltrating carcinoma. No clinical, colonoscopic and radiological feature of IBD was there. Radiological investigations often cannot differentiate a GVA from carcinoma and conventional computer tomography (CT) is non-specific in this regard. Attenuation of conventional CT is equal to the adjacent soft tissue, and hence determination of invasion is difficult. Magnetic resonance imaging (MRI) and CT colonography are considered superior to CT scan.[2] secretagouges, especially the prostaglandin E2 (PGE2) is thought to induce the goblet cells in VAs leading to excess mucus secretion. As these lesions are mostly situated distally, presence of very less absorptive mucosa beyond the lesion may result in diarrhoea and loss or sodium and potassium.[5] Unless there is infiltration of rectal wall resulting in edema and rigidity, intestinal obstruction is not usual presentation in GVA.[2] As any adenomas of GI tract have malignant potential, they should be removed endoscopically. However GVA is difficult to remove endoscopically and require segmental excision by parasacral approach or anterior mucosal eversion techniques. Trans-anal endoscopic microsurgery is effective in some cases, but may lead to stenotic lesion if the GVA is circumferential, like the index case.[5]

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