An unusual cause of lower gastrointestinal bleeding: A case report and a review of literature

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Summary

This is an unusual case report of a 60-year-old man who presented with massive rectal bleeding due to angiomatous formation.

He was also found to be cirrhosis and to have an ectopic left kidney in the midline over the roof of the mesenteric vessel.

He was treated successfully by performing a right hemicolectomy.

Key words: ***Massive rectal bleeding, Angiomatous malformation, Ectopic kidney, Liver cirrhosis.***

Résumé

Il s'agit d'un cas d'un rapport peu ordinaire d'un viel homme âgé de 60 ans atteint du saignant massif du rectal attribuable à la formation angiomateuse. On a decouvert qu'il avait également la cirrhose et atteint d'un ectopique dans le rein du côté droite dans le milieu de ligne au dessus du palais du vaisseau mesenterique.

On l'avait soigné connu du succès à travers une intervention chirurgicale d'hemicolectomie du côté droite.

Introduction

Massive lower gastrointestinal bleeding can be a challenging experience to the treating team. Diverticulosis, internal haemorrhoids and angiodysplasia are the usual causes [1, 2]. However, rare causes of massive lower gastrointestinal bleeding, from unusual lesions in the colon or terminal ileum, can offer much diagnostic problem.

A case of portal hypertension associated with an ectopic kidney, presenting with lower gastrointestinal bleeding, is reported.

Case report

A 60-year-old, Saudi male was referred to Asir Central Hospital, with a history of intermittent episodes of melena for two years. There was no history of change in bowel habits. Systemic review was essentially normal. Examination on admission revealed a relatively healthy man with severe pallor, but neither jaundiced, nor cyanosed. Vital signs were stable. Spleen was palpable 2-cm below the costal margin. Liver was not palpable. Rectal examination showed second degree haemorrhoids, but no active bleeding was detected. Haemoglobin was as low as 4.5 gm\dl before transfusion. White blood cell count was 6.2 X 10^9/L, platelet count was 549 X 10^9/L (normal is 140-440X10^9/L). Electrolytes, urea and liver function tests were within normal limits. Stool analysis

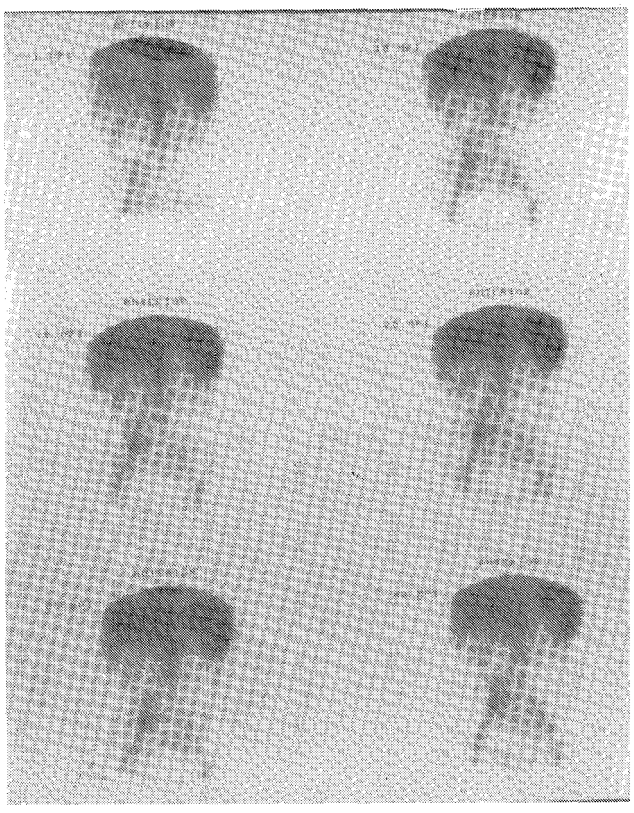

Fig. 1 ***Radio isotope scanning with 99m T c-labelled red blood cell***

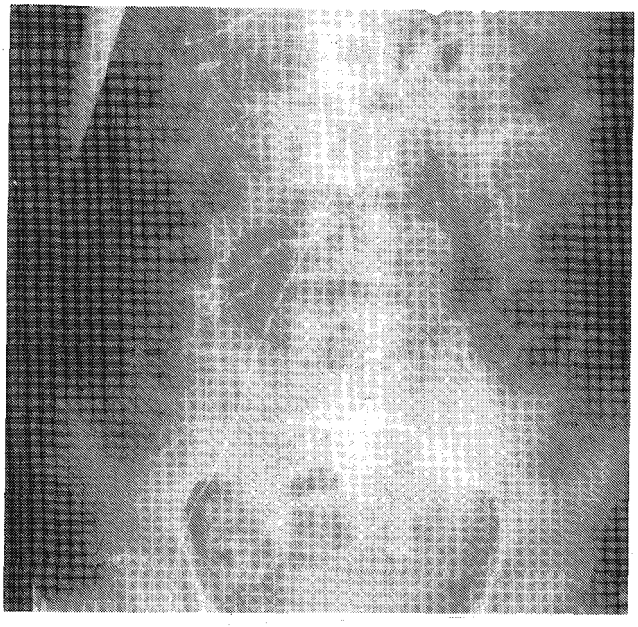

Fig. 2 ***Mesenteric arteriogram***

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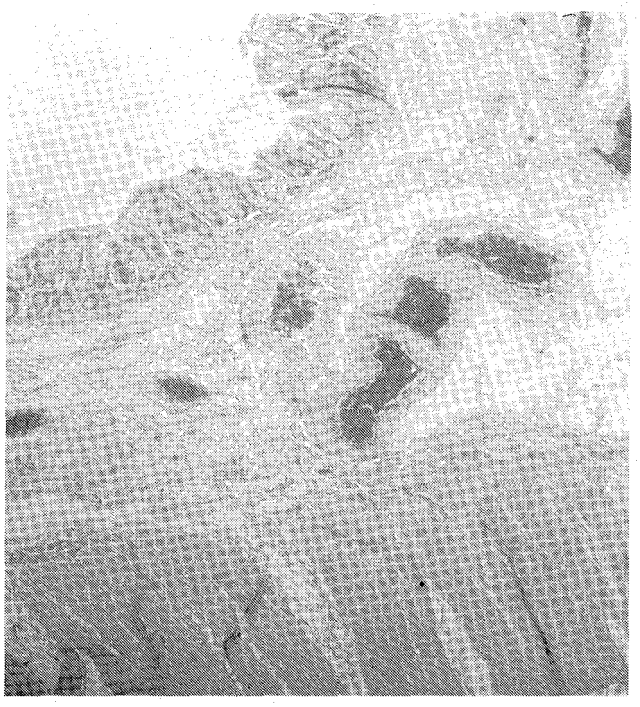

Fig. 3 *Photomicrograph showing increased number of dilated, tortuous, thick walled submucosal vascular channels with histologically unremarkable overlaying colonic mucosa (Haematoxylin and Eosin; original magnification X 80).*

was unremarkable for ova and parasites examination. Upper gastrointestinal endoscopy and flexible sigmoidoscopy were not remarkable.

Ultrasonography of the abdomen showed splenomegaly and dilated epigastric vessels suggestive of varices. The left kidney was also shown to be located in the midline over the roots of the mesentery.

Radio isotope scanning with 99mTc-labelled red blood cells showed an area of bleeding in the right iliac fossa (Fig 1). Patient had mesenteric arteriogram which confirmed angiomatous malformations in the caecum and ascending colon (Fig 2).

After resuscitation and blood transfusion, laparotomy was performed. The liver was found to be cirrhotic. Intraoperatively, the left kidney was found in the midline over the root of the mesenteric vessels. Right hemicolectomy was performed.

Post-operative course was uneventful except that the patient started to have troublesome prolapsed haemorrhoids. About 8 days post-operatively he had haemorrhoidectomy for 4-th degree, prolapsed haemorrhoids.

Gross examination of the removed colon showed multiple, flat, dark-red mucosal lesions ranging from 2-4 mms. The mucosal lesions were seen in the caecum and ascending colon. Fig. (3) Shows the histology of these mucosal lesions as focal increase of dilated, submucosal, thick walled tortuous vascular channels. Dilated thin-walled mucosal venules and capillaries were not seen.

He was finally discharged home 11 days post-operatively, in good condition. He was followed in the outpatient clinic for more than one year, in good health, and without any more episode of rectal bleeding.

Discussion

Massive lower gastrointestinal tract bleeding from obscure causes can be a diagnostic night-mare to the doctor and a frightening experience to the patient. Nevertheless it has been shown that in all cases of lower gastrointestinal massive bleeding, a standard approach in investigation will identify the source of bleeding in about 98% of such cases [3].

Most cases of massive bleeding originating from the colon tend to produce bright red blood, but some, as in this case and in some cases of ulcerative colitis, may produce only melaena [4].

In spite of all efforts, the source of bleeding may still be elusive [3]. In such cases exploratory laparotomy with intraoperative enteroscopy guided by the surgeon may locate the area of bleeding.

The classification of vascular anomalies in the gut is confusing. Congenital types include arterio-venous malformations whereas, histologically identical, but acquired lesions are usually termed angiodysplasia. The vascular alterations in angiodysplasia range from small focal, mucosal vascular ectasia to large, dilated, tortuous, submucosal veins associated with extensive dilatation of thin-walled mucosal venules[5]. Some authors require the presence of dilated, thin-walled venular channels in the mucosa for the diagnosis of angiodysplasia and consider the presence of dilated, tortuous, thick-walled, submucosal vascular channels as a type of arterio-venous malformation[5,6]. The thin-walled, mucosal venular channels were not seen in our case. Upon resection of the colon the ectatic vessels tend to collapse and might be missed by the pathologist. Their demonstration is facilitated by post-resection vascular injection with silicone rubber or radiographic contrast material.

The aetiology of colon varices is unknown. This may include congenital vascular abnormalities, portal hypertension, obstruction of mesenteric vein circulation by thrombosis, extrinsic pressure, adhesions, or kinking, and congenital cardiac malformations [7-9]. Familial varices of the colon have also been reported [10]. There was no report of any gastrointestinal bleeding in the family members of our patient.

Typhoid ileal perforation [11] has also been reported to present in some cases with massive rectal bleeding. This is usually due to erosion of a terminal branch of the ileocolic artery by the ulcer which usually occurs at the terminal ileum. This should be suspected when the rectal bleeding follows a week's history of fever, malaise and diarrhoea.

Surprisingly colonic varices are only rarely encountered in patients with portal hypertension, despite the presence of the meso-systemic collaterals. Therefore, extrinsic pressure by the ectopic left kidney on the mesenteric vein could have played a significant role, in addition to the portal hypertension, in either the development or augmentation (or both) of the colonic vascular lesions in this patient.

Acknowledgement

We would like to thank Professor Nader Murad for his help in preparing the histopathological figure and for his valuable comments.

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