Case report

Massive lower gastrointestinal bleeding in a young postpartum lady: A management dilemma

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ABSTRACT

Introduction: Lower gastrointestinal (GI) bleeding secondary to bleeding colonic angiodyplasia in a young population is rare. The prevalence of angiodyplasia in healthy asymptomatic adult was 0.8% and increases in elderly population, chronic renal failure, pulmonary disease, aortic stenosis, and von Willebrand disease.

Aim: We are reporting a case of a young, postpartum woman who presented to us with massive lower GI bleeding secondary to bleeding angiodyplasia. Angiodyplasia is rare in this group of patients, making the detection and treatment challenging.

Case study: A fit 25-year-old woman presented with type IV hypovolemic shock due to massive lower GI bleeding on postpartum day 15. Emergency colonoscopy was attempted with no evidence of active bleeding detected until splenic flexure. The patient was rushed to the operation theatre for exploratory laparotomy due to unstable condition. Segmental colonic clamping was performed and due to persistent blood pooling at different segments throughout the colon, subtotal colectomy and end ileostomy were performed. Patient recovered well post-operatively.

Results and discussion: Physiological changes during pregnancy may has aggravated the risk of bleeding in GI angiodyplasia. Following resuscitation, a lesion causing lower GI bleed can be identified and treated with a variety of endoscopic procedures, radiological and surgical interventions. In our case, the role of surgery comes in place due to massive life-threatening lower GI bleeding not responding to resuscitation.

Conclusions: Postpartum gastrointestinal bleeding from multiple colonic angiodyplasia is rare. It poses a challenge in diagnostic and treatment strategies. A timely individualized intervention either via endoscopy, interventional radiology or surgery is crucial and life-saving.

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1. INTRODUCTION

Lower gastrointestinal (GI) bleeding secondary to bleeding colonic angiodysplasia in a young population is rare. Its prevalence in the overall population is not known. The prevalence of angiodysplasia in healthy asymptomatic adult was 0.8%\textsuperscript{1,2} The prevalence increases with age more than 60 years old, chronic renal failure, pulmonary disease, aortic stenosis, and von Willebrand disease.\textsuperscript{1,2} Those with chronic renal failure have an increased prevalence of 19%–32% compared to a healthy individual.\textsuperscript{1}

2. AIM

We are reporting a case of a young, postpartum woman who presented to us with massive lower GI bleeding secondary to bleeding angiodysplasia. Angiodysplasia is rare in this group of patients, making the detection and treatment challenging.

3. CASE STUDY

A fit 25-year-old woman presented with type IV hypovolemic shock due to massive lower GI bleeding on postpartum day 15. She denied any previous abdominal pain, altered bowel habit, or any family history of similar presentation. On presentation, she was in hypovolemic shock and was transfused 13 units of packed cells. The abdominal examination was unremarkable. On proctoscopy examination, there was blood clots and no mass seen. The vaginal and perineal examination by the gynaecology team were also unremarkable.

Emergency colonoscopy was attempted with no evidence of active bleeding detected until splenic flexure. However, there were blood clots identified coming from the proximal part of the colon. Computed tomography (CT) angiography and embolisation were planned. However, her condition deteriorated with labile haemodynamics, and we decided on an emergency exploratory laparotomy.

Intraoperatively, segmental colonic clamping was performed and noted persistent blood pooling at different segments throughout the colon. Hence, subtotal colectomy and end ileostomy were performed. There were multiple punctate bleeding lesions throughout the entire colon suggestive of bleeding angiodysplasia (Figure 1). She recovered well with no recurrence of bleeding and was discharged after 7 days.

Histopathological examination revealed numerous haemorrhagic spots on the mucosa throughout the colon.
Microscopically, there were small blood vessels with foci of haemorrhage on the mucosa (Figure 2). Within the submucosa, there were also many congested and ectatic medium-sized blood vessels (Figure 3).

4. RESULTS AND DISCUSSION

GI angiodysplasia is an intestinal vascular malformation that usually appears macroscopically as a slightly elevated lesion on the mucosal surface, red with sizes ranging between 2–10 mm.4

The pathogenesis of angiodysplasia remains unclear.1 Chronic intermittent low-grade GI tract obstruction led to vessels dilatation at the submucosal level causing tortuosity of the submucosal veins and then the venules and capillaries on the mucosa.1,5 An increased level of angiogenic vascular endothelial growth factor (VEGF) due to mucosal hypoxia was also a contributing factor to angiodysplasia formation.1,5

The clinical presentation of angiodysplasia may range from an asymptomatic lesion incidentally found from the colonoscopy, unexplained iron deficiency anaemia, positive occult stools, to melena and life-threatening lower GI bleeding.1,6 Haemorrhage was usually painless with spontaneously halted in at least 90% of cases but it may rebleed in up to 34%–40%.2,4

There is no literature to date reporting on the relationship between postpartum and haemorrhagic colonic angiodysplasia. A few physiological changes during pregnancy, such as increased level of progesterone and the enlarging uterus which may contribute to constipation.4

The increase in aldosterone level in pregnancy also causes an increase in water and sodium reabsorption resulting in reduced stool volume leading to increase colonic transit time.7 As a result of constipation, more pressure was exerted towards the colonic mucosa leading to more prominent tortuous vessels formation, therefore, increasing the risk of bleeding. Furthermore, the third trimester of pregnancy was also associated with thrombocytopenia due to haemodilution and accelerated clearance.7 However, these were hypothetical deductions and was never proven on the currently available evidence. Up to date, there was no specific pharmacological agent recommended as treatment or prevention for bleeding GI angiodysplasia.8

Therapeutic endoscopy such as colonoscopy is the first line modality since it allows detection and intervention simultaneously. It can accurately identify the source of bleeding in as high as 82% of the patient.8 During the colonoscopy, the lesion are often seen at the caecum (49%), ascending colon (17%) and sigmoid colon (16%).2 Majority (40%–60%) of the patients will have more than one lesion with 20% of them having lesions at different locations.2 In our case, the lesions were detected throughout the length of the large bowel starting at the caecum until the sigmoid colon. This findings were rarely reported in the literature.

A timely colonoscopy may vary between different centres subject to the availability of the team. American Society of Gastrointestinal Endoscopy (ASGE) recommended that the colonoscopy should be performed following bowel preparation within the first 24 h of presentation for evaluation of a patient with severe GI bleeding.9

Endoscopic argon plasma coagulation (APC) is one of the choices to treat angiodysplasia. APC is a non-contact electrosurgical monopolar thermal procedure that delivers thermal energy up to a depth of 2–3 mm into the mucosa.9,10

Care should be taken given the risk of bowel perforation by putting probe 1–3 mm away from the mucosa and using a lower power setting of 30 Watts/L/min of argon flow.9,10

The non-contact coagulation effect of APC produced minimal tissue penetration and destruction, which therefore proven to be safe with acceptable complications.9,11 Combination treatment can also be done by applying APC followed with injection with 1 : 10000 diluted adrenaline.2

There is always a risk of rebleeding since angiodysplasia can occur throughout the colon and may be obscured by bowel mucosa on initial endoscopic assessment. One meta-analysis demonstrated the efficacy of initial endoscopic intervention with bleeding recurrence rate of 34%.12 During rebleeding, the therapeutic endoscopy was the recommended immediate treatment of choice. Few studies highlighted a few factors which were statistically proven to predict rebleeding rate such as low haemoglobin during the presentation, advanced age, those on anticoagulant treatment, findings of more than 2 angiodysplasias during endoscopy, and lesion at left side of colon.2,13

Angiography should also be considered in a stable patient where an angiodysplasia appeared as dense opacified and dilated early filling veins.14 This imaging modality has widely replaced by CT angiography (CTA) with its superiority of being less invasive and the ability to detect slow bleeding rates of 0.3–0.5 mL/min.9,15 Generally, CTA is performed before catheter angiography which requires at least 1 mL/min to pre-locate the site of bleeding. Findings of angiodysplasia in CTA are an accumulation of vessels in the colonic wall, early-filling veins and enlarged supplying arteries to colonic walls.9,15 In case of insidious occult bleeding, radionuclide red blood cell scan by Technetium (99mTc)-based tracers can detect bleeding as low as 0.1–0.5 mL/min.9,15

In our case, catheter angiography was not attempted due to the haemodynamic instability of the patient. However, if it was attempted, catheterisation and embolisation using gel foam or coils were reported to achieve a cessation of bleeding in 85%–97% of patient, but it also carries risk such as intestinal ischaemia, contrast-related nephropathy and radiation.9,15

There was also reported the role of minimally invasive surgery in managing haemorrhagic GI angiodysplasia but limited to haemodynamically stable patient. In a case report by Hitoshi, a successful laparoscopic surgery was done in a bleeding jejunal angiodysplasia by initially localising the lesion with selective trans-arterial embolisation then laparoscopic resection of the involved segment of the small bowel.10

Open exploratory surgery is indicated in those haemodynamic instabilities, not responding to blood product resuscitation. Apart from achieving immediate haemostasis
by resection, surgery also is more effective at a lower rate of rebleeding than endoscopic procedures (53% against 24%). This procedure involved either segmental resection or subtotal colectomy. Localization of bleeding site is of paramount importance as subtotal colectomy leads to morbidity. This could be achieved by either intraoperative colonoscopy or angiography or even ileostomy to help differentiate small intestinal or colonic bleed.

In our case, subtotal colectomy with end ileostomy was performed due to persistent blood pooling from multiple sites of bleeding angiodysplasia throughout the colon. She has an uneventful post-operative recovery. She was well during follow up and we have successfully reversed her ileostomy after 6 months.

5. CONCLUSIONS

Postpartum gastrointestinal bleeding from multiple colonic angiodysplasia is rare. It poses a challenge in diagnostic and treatment strategies. A timely individualized intervention either via endoscopy, interventional radiology or surgery is crucial and life-saving.

Conflict of interest
None declared.

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References


