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SHORT REPORT

Revisiting the past: Intra-arterial vasopressin for severe gastrointestinal bleeding in Crohn's disease

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Abstract

Technological advances in the last couple of decades have led to a tremendous improvement in the safety and efficacy of embolization making it the therapeutic intervention of choice in angiogram positive lower gastrointestinal bleeding. Vasopressin has thus been forgotten and it is hardly ever used by the current generation of interventionists. However, coil embolization is technically challenging and requires greater expertise. Difficulty in super-selective catheterization and lack of adequate collateralization can also prevent successful delivery of coils. In this article we present the successful use of intra-arterial vasopressin in a patient with Crohn's disease with severe lower gastrointestinal bleeding. Despite not being the first choice, vasopressin can be safely and effectively used in selected patients who are not candidates for embolotherapy. The purpose of this article is to discuss the relative merits and demerits of vasopressin vis-à-vis embolization and to identify the role of vasopressin in the current era of super-selective embolization. Successful control of massive lower gastrointestinal bleeding by intra-arterial vasopressin infusion has previously been reported only once before in Crohn's disease. We suggest that this technique may be used in an attempt to avoid surgery in these patients.

1. Introduction

The incidence of lower gastrointestinal (LGI) bleeding is approximately 20–30/1,00,000 adults and this increases with increasing age.[1] In a vast majority of cases the bleeding resolves with conservative management. Endoscopic, vascular and surgical interventions are reserved for the small

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minority with significant bleeding not responding to conservative measures.[2] The advent and evolution of coil embolization has replaced and relegated the use of intra-arterial vasopressin to medical history. Herein, we describe the successful use of intra-arterial vasopressin in a patient with refractory LGI bleeding secondary to active Crohn's disease whose anatomy was not favorable for embolization.

2. Case summary

A 60 yr old male was admitted to the hospital with altered mental status. Past medical history was significant for long standing Crohn's disease and multiple abdominal surgeries for fistulae and recurrent intestinal obstruction. He was on Mesalamine maintenance therapy. His recent flare was 2 weeks prior to admission, colonoscopy revealed active ileitis. He also had poorly controlled hypertension, diabetes mellitus and stable coronary artery disease. On evaluation he was found to have malignant hypertension and subarachnoid hemorrhage with intraventricular extension. He was intubated, started on intravenous nicardepin and urgent neurosurgery consult was sought. He subsequently underwent ventriculostomy for worsening hydrocephalus. At the time of admission the patient was on high dose oral steroid, ciprofloxacin and metronidazole for a recent flare of his Crohn's disease and these were continued intravenously. The patient developed a massive gastrointestinal hemorrhage (manifested by malena) with hemodynamic instability a week after hospitalization. He was resuscitated with crystalloid and multiple units of packed RBC. Urgent gastroenterology and surgery consults were called after initial stabilization and the patient underwent evaluation to localize the source of bleeding. CT scan of abdomen, upper and lower gastrointestinal endoscopies were unrewarding and did not reveal any obvious source of bleeding. Nuclear scan revealed a potential source of bleeding in the area of the small bowel. Patient's hemoglobin continued to drop despite frequent RBC transfusions and his hemodynamics remained tenuous. He was deemed to be a poor surgical candidate in view of active Crohn's disease and extensive adhesions from his multiple prior abdominal surgeries. An interventional radiology consult was therefore sought for potential embolization of the culprit vessel. Mesenteric angiogram revealed brisk bleeding from one of the branches of superior mesenteric artery in the region of ileum (Fig. 1). The anatomy was deemed unfavorable for embolization due to lack of adequate collaterals, hence putting a large segment of bowel at risk for infarction. We therefore decided to use local vasopressin infusion to control his bleeding. Selective catheterization of the SMA was done with a Sos Omni 2 microcatheter and intra-arterial vasopressin infusion was started at 0.2 U/min. A repeat angiogram showed persistent bleeding despite uptitrating the dose to 0.4 U/min (Fig. 2). We then proceeded to super-selective catheterization of the culprit vessel and restarted vasopressin infusion. It was started at 0.2 U/min and was titrated in 0.1 U increments every 20 min if there was evidence of continued bleeding on angiogram. Hemostasis was finally secured after 20 min of infusion at 0.4 U/min (Fig. 3). Thereafter, the catheters and sheaths were secured and vasopressin infusion was continued at 0.1 U/min for 12 h.

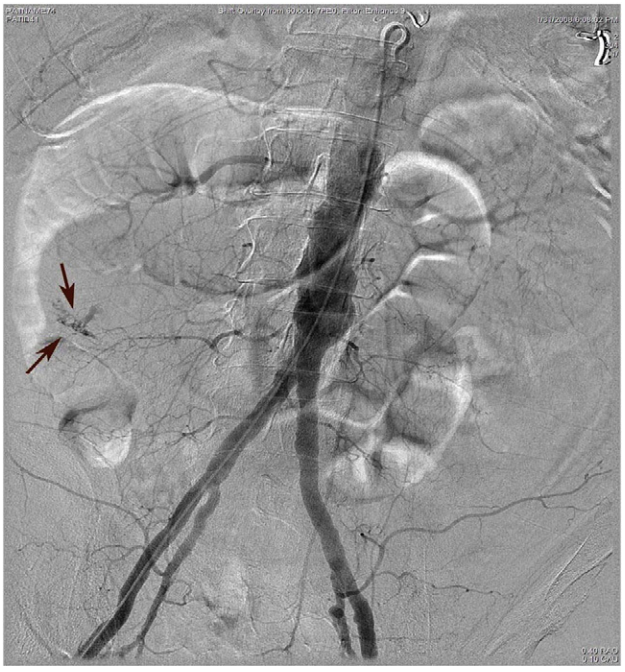

Figure 1 Late phase abdominal aortogram through an omni flush catheter. Arrow denotes active contrast extravasation in the ileal region.

Vasopressin was then discontinued after a repeat angiogram confirmed the absence of any further bleeding (Fig. 4). Total procedure time was 90 min, initial procedure lasted 70 min and the later check angiogram took 20 min. Patient was closely monitored in the intensive care unit for any clinical or laboratory evidence of coronary, mesenteric or limb ischemia. Patient tolerated the procedure well without any peri

Figure 2 Superior mesenteric arteriogram 20 min after vasopressin infusion at 0.4 units/minute. Arrow denotes persistent contrast extravasation in the ileal region. Arrowhead denotes the Omni Sos 2 catheter in the superior mesenteric artery.