Acute mesenteric vein thrombosis after glue injection for Gastric varices – a case report

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Citation

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Abstract

Mesenteric vein thrombosis (MVT) is a relatively rare condition. Here we report a case of MVT following glue injection for fundal varix. MVT is usually characterized by the presence of abdominal pain, which is out of proportion with the physical findings. The etiology of this condition is not known. Embolism can occur through a large porto- venous shunt following glue injection. Mesenteric venous thrombosis (MVT) accounts for 5-10% of patients presenting with acute mesenteric ischemia (AMI)[1]. The etiology of this entity is not clearly defined. Several hypercoagulable states such as antithrombin III, protein C and protein S deficiencies may play a role in pathogenesis of this entity. Here we report a case of acute mesenteric venous thrombosis following the glue injection therapy for gastric varices.

CASE HISTORY

A 19 year old boy, admitted with two days history of passage of bloody diarrhea and crampy abdominal pain. Abdominal Pain was sudden in onset, severe periumbilical pain, radiating to back. He had yellow color watery stools for 12 hrs which was followed by passage of mucus stools mixed with altered blood about 7-8 times/day. He also had vomiting of 250 ml altered blood once prior to hospitalization. He had no abdominal distension, leg swelling or Jaundice. His past history was significant; he is a known case of extra hepatic portal vein obstruction (EHPVO) since 6 years of age. He underwent Tanner's procedure for growth retardation, anemia and massive splenomegaly. Three years after the procedure he had one episode of upper GI bleed. He received EVL or EST for eradication of varices. Forty eight hours prior to onset of all his symptoms, he received 1 ml of glue without any lipidol mixture into fundal varices with a nipple sign on regular follow up endoscopy. He does not smoke and takes alcohol.

On clinical examination, he was afebrile, pale with no icterus or pedal edema and not in respiratory distress. The vital signs showed a blood pressure of 120/80 mm Hg, a regular pulse of 88 per minute, a respiratory rate of 22 breaths/min and body core temperature of 37 °C. The jugular venous pressure was not elevated, the heart wounds were normal. Examination of the chest was normal. Abdominal examination showed mild abdominal distension, moderate tenderness over the epigastrium and periumbilical area with normal bowel sounds. There was no hepatosplenomegaly or free fluids. Rectal examination showed blood stained finger stalk.

Baseline investigations showed hemoglogin (Hb) 6 g/L, white blood counts 15.7 X109/L with differential count of N 84, L 14, E 2 and platelets counts 3.06X109/L. The blood chemistry and liver profile was as follows: glucose 86 mg/dl, creatinine 9 mg/dl, urea 16 mg/dl, sodium 140 mmol/l, potasium 3.9 mmol/l, alkaline phosphatase 52 IU/L (normal 40-128), alanine transaminase 17 IU/L (normal 10-60), aspartate transaminase 22 IU/L (normal 10-42) and total bilirubin was 0.6 mg/dl. The ECG and chest X-ray was normal. Plain abdominal X-ray showed dilated small bowel loops without air fluid levels and with any evidence of air under the diaphragm. A contrast enhanced CAT scan showed Thickening and enhancement of bowel wall of small bowel loops with proximal dilatation and peri-mesenteric edema. Superior mesenteric vein and artery showed no evidence of thrombus (Fig. 1). Upper gastrointestinal endoscopy showed evidence of Grade I esophageal varices with gastroesophageal varices I & II, evidence of portal hypertensive gastropathy with no blood in the lumen. (Fig. 2). He developed severe abdominal pain and progressive abdominal distension. Abdomen showed guarding with

sluggish bowel sounds over 12 hours.

Figure 1

Fig 1. Endoscopic picture showing fundal varices after glue injection

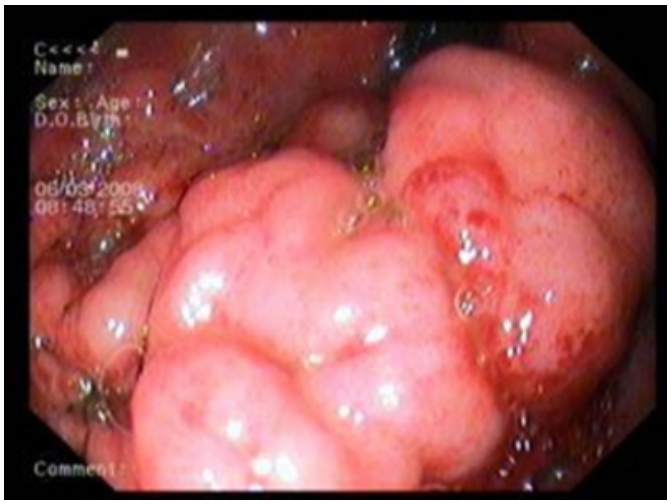
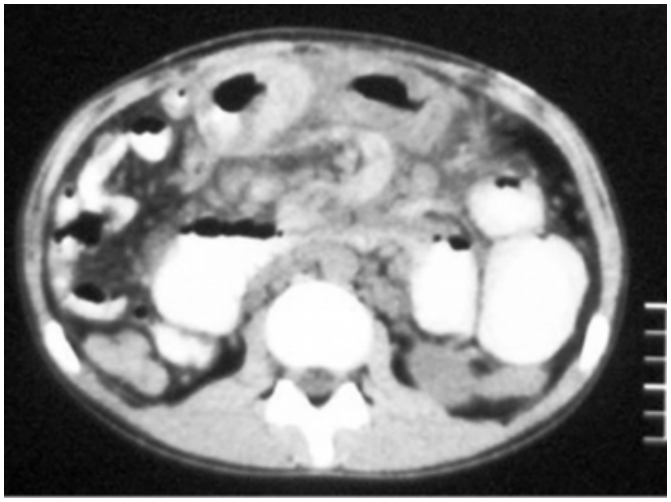


Figure 2

Fig 2. Contrast enhanced CT abdomen showed bowel and mesenteric edema



Emergency laparotomy showed gangrene of small bowel from duodeno -jejunal flexure up to 30 cm from ileo-cecal valve. Gangrene portion of small bowel resected with proximal Jejunostomy and terminal Ileostomy done. Intraoperatively, mesenteric vessels found to be thrombosed and it was confirmed at Histopathological examination. Patient was started on total parenteral nutrition. Subsequent jejuno-ileal anastomoses done.

DISCUSSION

MVT is a relatively rare condition. On autopsy studies, MVT was found in 0.2-2% of the population 2 . MVT is most common in the 6-7th decade of life and it involves the SMV in 95% of the cases reported 2. MVT is usually characterized by the presence of abdominal pain, which is out of proportion with the physical findings. Gross gastro intestinal bleeding, an indication of bowel infarction, was reported in less than 15% 3. Physical findings range from being normal to abdominal tenderness, distension and decreased bowel sounds 4. The etiology of this condition is not known but several secondary causes have been linked to MVT such as portal hypertension, malignancy, myeloproliferative disorders, intraperitoneal inflammation, dehydration, trauma and hypercoagulable states 5.

Common complication following glue injections includes acute ulceration, thrombo-embolic phenomena, septicemia and liver cell failure. With an increasing use of cyanoacrylate glue for the treatment of gastric varices, many cases have been reported with thrombo-embolic manifestation. Embolism can occur through a large porto-venous shunt. The following factors decide the occurrence of thromboembolism that includes. 1) Amount of glue; when it exceeds more than 1ml at a single session. A study 6 showed six patients of 140 with pulmonary emboli received a mean volume of 4.16 mL vs 1.76 mL for those without pulmonary emboli. 2) Ratio of lipidol mixed with glue; normally Glue hardens within 4 sec on contact with blood. When the lipidol concentration exceeds the limit posses delayed polymerization of glue and increases the risk of embolic complications. 3) Number of injections per varix; 2-3 injection per varix increases the risk. Route of embolic phenomena occurs cited in the table 1.

Figure 3

Table 1. Possible route of embolic phenomena following glue injection

Type	Source	Site of involvement
Mesenteric Vein thrombosis	Gastroepiploic vein	Superior mesenteric vein
Pulmonary embolism	Short Gastric/ Gastroepiploic vein	Lt Renal vein – IVC
Splenic infarct	Gastroepiploic vein to portal vein	Centrifugal blood flow
Arterial Ischemia	From systemic veins through Atrial septal defect	Heart, brain and kidney

Figure 4

Table 2. Embolic complications reported in the literature

Studies	No. of cases	Pulmonary Embolism	SMV thrombosis	Others
PJ Belletrutti CDDW 2006 Abstracts	29	-	1	-
Hwang et al. J Comput Assist Tomogr 2001	140	6	-	-
Gin-Ho-Lo, et al. Hepatology 2001.	31	-	-	CVA
Joo HS et al Korean J Gastroenterol. 2007	85	2	-	Splenic infarct -1
Upadhyay AP et al. Endoscopy 2005 [7]	1	-	-	Acute MI
Rickman, Mayo Clin Proc. 2004[8]	1	1	-	-

In symptomatic acute MVT, treatment is based on the presence or absence of peritoneal signs. Signs of peritonitis mandate laparotomy and resection of infarcted bowel. If long segments of questionably viable bowel are found, papaverine is infused and planned for thrombectomy. Heparinization for 7 to 10 days has been shown to diminish recurrence and progression of thrombosis in the postoperative period 9. Duration of anticoagulation are not known. In the presence of hypercoagulable lifelong treatment with warfarin is advised. In our patient, gangrene portion of small bowel resected with proximal Jejunostomy and terminal Ileostomy done. He was started on total parenteral nutrition. Subsequent jejuno-ileal anastomoses done and he is doing well.

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