Imaging Findings Of Extensive Splenic Infarction After Cyanoacrylate Injection For Gastric Varices – A Case Report

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SUMMARY

Endoscopic injection of N-Butyl-2-cyanoacrylate is a widely accepted treatment for esophagogastric varices. This procedure is commonly associated with minor complications which include transient pyrexia and abdominal discomfort. Serious vascular complications secondary to systemic embolization of cyanoacrylate have rarely been reported. We describe the CT findings of extensive splenic infarction in a patient following cyanoacrylate injection for gastric varices.

KEY WORDS:

Cyanoacrylate injection, gastric varices, splenic infarct

INTRODUCTION

Cyanoacrylates were first used for the treatment of gastric varices by Lunderquist et al. via percutaneous transhepatic approach in 1978. Endoscopic injection of N-Butyl-2-cyanoacrylate for bleeding esophagogastric varices was described by Soehendra et al. in 1986¹.

N-Butyl-2-cyanoacrylate (Histoacryl; B.Braun Dexon, Spangenberg, Germany) is a tissue glue monomer, the treatment of choice for obliteration of gastric varices. Mixture of Histoacryl and Lipiodol during variceal injection aims to prevent Histoacryl from hardening too quickly and to allow radiographic monitoring. However, delayed polymerization of Histoacryl-Lipiodol mixture may result in serious vascular complications².

This case highlighted the uncommon CT findings of extensive splenic infarction after elective obliteration of gastric varices with cyanoacrylate.

CASE REPORT

A 44-year-old Indian woman, with known autoimmune liver cirrhosis and portal hypertension presented with one week history of intermittent low grade fever and left sided abdominal pain. Two weeks prior to her presentation, she underwent endoscopic obliteration of fundal varices with Nbutyl-2-cyanoacrylate using 0.5ml Histoacryl diluted with 0.8ml Lipiodol when fundal gastric varices with portal gastropathy and grade 1 esophageal varices were noted at surveillance endoscopy.

On examination, her blood pressure was 138/91mmHg, pulse rate was 102bpm. There was splenomegaly with mild tenderness in the left hypochondrium. She was afebrile, nontachypnoeic and anicteric. Examination of the other systems was unremarkable.

She was slightly thrombocytopenic (platelet count ranges between $111 \times 10^{\circ}$ /L and 142×109 /L during admission), with a mildly elevated erythrocyte sedimentation rate (32mm/hr) and a low albumin level (28g/L). Total bilirubin level was 33µmol/L. Her hemoglobin level, total white blood count, coagulation profile and renal profile were normal.

An urgent CT abdomen revealed liver cirrhosis with esophageal, splenic hilar and large retroperitoneal varices. Dense particles in keeping with injected cyanoacrylate were seen within the esophageal varices, splenic hilar varices, perihilar splenic parenchyma, posterior stomach wall and the main portal vein. There was gross splenomegaly with a large hypodense area at the upper pole and a smaller hypodense area anteriorly, consistent with areas of infarction. The patient was diagnosed to have splenic infarction secondary to cyanoacrylate injection for gastric varices. Her symptoms gradually resolved following conservative treatment with analgesics, anti-histamine and anti-emetic. She was discharged on the sixth day. She was subsequently followed-up in the gastroenterology clinic and had remained asymptomatic with no further complaint.

DISCUSSION

Gastric varices are found in about 20% of patients with portal hypertension as a complication of liver cirrhosis. Another 10% to 20% of patients with portal hypertension develop gastric varices after endoscopic therapy of esophageal varices. Acute gastric variceal bleeding can be life threatening. Risk factors for gastric variceal bleeding include fundal location, advanced Child-Pugh class, presence of red spots and large variceal size³.

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Fig. 1: There are dense particles with Hounsfield unit ranging from 1500–2500HU within the esophageal varices. There was a large hypodense area with similar foci of high density particles seen within the splenic parenchyma.

Endoscopic variceal sclerotherapy, endoscopic variceal band ligation and transvenous intrahepatic portosystemic shunt have been applied to control bleeding gastric varices. Prophylactic eradication is sometimes practiced in patients who are at high risk for bleeding, as in this case².

Endoscopic injection of sclerosants such as ethanolamine oleate or absolute alcohol, have been very successful in the eradication of esophageal varices but less effective for fundal gastric varices, probably due to the high-volume blood flow ³. Endoscopic variceal band ligation, the preferred treatment of esophageal varices, has a limited role in gastric variceal treatment due to the retroflex position of endoscope leading to difficult deployment and difficulty in suction of large gastric varices. A recent randomized control trial has concluded that cyanoacrylates injection is more effective and safer than band ligation in the management of bleeding gastric varices ⁴. Transvenous intrahepatic portosystemic shunt is used as second-line rescue therapy in acute gastric variceal bleeding ³.

Cyanoacrylates injection has been shown to successfully control acute gastric variceal bleeding in over 90% of cases with a low rebleeding rate and is now the first-line treatment of bleeding fundal gastric varices and prophylactic eradication of gastric varices in many centers around the world^{3,5}.

The overall safety record of cyanoacrylate has been relatively good. Transient pyrexia and abdominal discomfort commonly occur as a result of normal inflammatory response. However, scattered cases of serious complications have been reported³.

Splenic infarction is an uncommon complication which may occur secondary to splenic arterial embolization or retrograde splenic venous embolization from the portal circulation due to forceful injection of a large volume of lipiodol. Other rare, but serious complications that have been reported include near-fatal multiple pulmonary embolisms and splenic



Fig. 2: Reformatted images in coronal section demonstrated curvilinear densities in the upper pole of the spleen and splenic varices. The liver is cirrhotic with nodular margins.

infarction with septicemia, cerebral and portal vein embolism, portal and splenic vein thrombosis and retroperitoneal abscess ^{2, 3}.

Spontaneous portosystemic shunts occur in 60% to 85% of patients with gastric varices. Systemic embolization may probably occur through anomalous arteriovenous pulmonary shunts or gastrorenal shunts. Hepatopulmonary syndrome, characterized by dilated pulmonary microvasculature with consequence right-to-left shunt potentially facilitating passage of Histoacryl-Lipiodol mixture into the systemic circulation, occurs in 20% of patients with cirrhosis. Gastrorenal shunts are portosystemic shunts between splenic vein and gastric varices, via the inferior phrenic or suprarenal vein to the left renal vein³. The majority of cases of distal embolization have occurred when a mixture of Histoacryl and Lipiodol was used. Delayed polymerization of this mixture has been suggested as a possible explanation².

This case report highlighted the CT findings of extensive splenic infarction which is not commonly seen. In this patient, paradoxical splenic embolisation has not resulted in significant morbidity. On the other hand, there was slight improvement in her platelet count due to less sequestration effect caused by hypersplenism.

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