Splenic infarction after cyanoacrylate injection for fundal varices

A 43-year-old man with liver cirrhosis due to hepatitis B infection, who had undergone regular endoscopic variceal therapy for fundal varices, was admitted for secondary prophylaxis. The laboratory findings were unremarkable apart from a low platelet count (34 × 10^3/µL) and prolonged prothrombin time (17.7 s, international normalized ratio [INR] 1.46, 59%). Endoscopic examination revealed small esophageal varices and large fundal varices with the red color sign. Two injections of 2 mL of N-butyl-2-cyanoacrylate (Histoacryl; B. Braun, Tuttlingen, Germany) diluted with Lipiodol (Guerbet, Aulnay Sous Bois, France) were administered into the fundal varices. The patient had undergone multiple sessions of endoscopic injection therapy in the past with no occurrence of complications. However, 2 days after the last endoscopic cyanoacrylate therapy, he developed abdominal pain and fever, and his white blood cell (WBC) count rose to 14 × 10^3/µL. Abdominal computed tomographic (CT) angiography revealed remnants of cyanoacrylate in the splenic vein with nonenhancing splenic parenchyma, suggestive of cyanoacrylate-induced splenic infarction (Fig. 1). Intravenous antibiotics were immediately initiated along with supportive management. Both the abdominal pain and fever gradually subsided and the WBC count normalized. The patient was discharged on day 20 in the hospital without any further complications or symptoms. A follow-up CT showed persistent, large, hypoattenuated lesions in the spleen, but the patient remained asymptomatic during the 9 months’ follow-up in the outpatient department (Fig. 2).

Endoscopic injection of cyanoacrylate is generally considered to be the usual treatment for gastric varices, but complications such as fever, deep ulceration, chest pain, and vascular occlusion can occur. Although splenic infarction after administration of cyanoacrylate is rare, it has been reported to occur when large volumes of cyanoacrylate are injected rapidly. This is because of the consequent splenic venous occlusion [1]. With adequate supportive management the splenic infarct can be left in situ. Its clinical course may be self-limiting and the patient may not require surgical treatment, such as splenectomy [2].

Competing interests: None