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CASE REPORT

Eosinophilic pleural effusion after gastric variceal obliteration with cyanoacrylate

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Summary
Pleuropulmonary complications after endoscopic injection sclerotherapy for treatment of esophagogastric varices are not uncommon but are usually mild and self-limited. Herein we report a male patient with liver cirrhosis who underwent endoscopic injection sclerotherapy, using a mixture of *n*-butyl-2-cyanoacrylate and ethiodized oil for obliteration of gastric varices. After the procedure, he developed moderate amount of left pleural effusion that persisted for a period of time and required thoracentesis and medical treatment. We believed that the inadvertent retrograde reflux of the embolized glue and ethiodized oil via the portosystemic venous collateral into the left pleura might be the possible mechanism for the development of left pleural effusion as the droplets of ethiodized oil were seen along the left pleura on the imaging studies.

Introduction

Because of its efficacy and safety, endoscopic injection sclerotherapy (EIS) has now become the treatment of choice for obliteration of esophagogastric varices.[1,2] Satisfactory hemostasis in cases of acute variceal bleeding can be achieved by injection of various agents including the tissue adhesive *n*-butyl-2–cyanoacrylate (NBCA), ethanolamine oleate, polidocanol, ethanol, sodium tetradecyl sulfate, and sodium morrhuate.[3] However, NBCA is now considered the best sclerosing agent for EIS for obliteration of bleeding gastric varices.[3–6] Undiluted cyanoacrylate almost immediately polymerizes after contact with blood, resulting in rapid coagulation and vascular occlusion. Because of the risk of gluing of the needle in the varix, a mixture of NBCA and ethiodized oil is used in order to delay polymerization of NBCA and to help fluoroscopic monitoring.[7,8] Nevertheless,

Abbreviations: EIS, endoscopic injection sclerotherapy; NBCA, *n*-butyl-2-cyanoacrylate; ADA, adenosine deaminase; CT, computed tomography

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the delayed polymerization of the NBCA–ethiodized oil mixture may increase the risk of distal glue embolization or reflux into other systemic vessels.[9–11]

Pleural effusion is not an unusual complication following EIS but is usually mild and self-limited. It may occur in association with pulmonary artery embolization and parenchymal infarction after EIS.[7,12] To our knowledge, however, other possible mechanism for the development of pleural effusion after EIS has neither been proposed nor demonstrated by the imaging studies. Herein we report a patient who developed left pleural effusion after EIS for obliteration of gastric varices. We believed that we were the first to be able to demonstrate the deposition of ethiodized oil along the left pleura in the chest radiograph and computed tomography (CT). The mechanism responsible for the development of left pleural effusion was also discussed.

Case report

A 43-year-old man with known hepatitis B-related liver cirrhosis and diabetes mellitus was admitted to our hospital because of low-grade fever, dry cough and left-sided pleuritic chest pain. Three weeks prior to admission, he had upper gastrointestinal bleeding and went to a private hospital. Esophageal and gastric varices were found on endoscopic examination. EIS was then performed by an experienced gastroenterologist for obliteration of gastric varices, using a mixture of 0.5 mL *n*-butyl-2-cyanoacrylate (Histoacryl; B-Braun Sugical GmbH, Melsungen, Germany) and 0.7 mL ethiodized oil (Lipiodol; Laboratoire Guerbet, Aulnay-Sous-Bois, France). The procedure was successfully done without any complication. One week after the procedure, however, he developed a new session of upper gastrointestinal hemorrhage that was successfully controlled by balloon tamponade and continuous intravenous administration of sandostatin. Initial chest radiograph obtained after the balloon insertion revealed multiple radiopaque materials in both hemithoraces and at the gastric fundus (Fig. 1). During that time, he had no fever or respiratory symptoms.

On admission, the patient was in no distress but was febrile with body temperature of 38.8 °C. Physical examination revealed decreased breath sound and dullness on percussion at the left hemithorax. Other physical findings were unremarkable. Oxygen saturation, as detected by pulse oximeter at room air, was 95%.

Complete blood count showed a hematocrit of 32%, white blood cell count of 9400 cells/mm^3 with 78% neutrophils, 17% lymphocytes, 15% monocytes and a platelet count of 127,000 cells/mm^3. Blood sugar was 441 mg/dL. Serum lactate dehydrogenase (LDH) was 422 U/L (range, 100–190). Serum protein was 78 g/L and serum amylase was 54 U/L. Other blood chemistry and coagulation tests were within normal limits.

Initial chest radiograph revealed a moderate amount of left pleural effusion and a tiny radiopaque density overlying the right upper hemithorax (Fig. 2). Thoracocentesis was then performed and yielded straw-colored pleural fluid that was composed of a white blood cell count of 2010 cells/mm^3, with 45% neutrophils, 40% lymphocytes, and 15% eosinophils. The pleural fluid had a glucose level of 394 mg/dL, LDH of 1000 U/L, protein of 43 g/L, adenosine deaminase (ADA) of 52 U/L, and amylase of 19 U/L. Microscopic examination of pleural fluid was negative for gram and acid-fast bacilli. Moreover, there was no organism growth on pleural fluid culture. Pleural fluid cytology revealed only inflammatory cells. Amoxicillin-clavulanate was then given

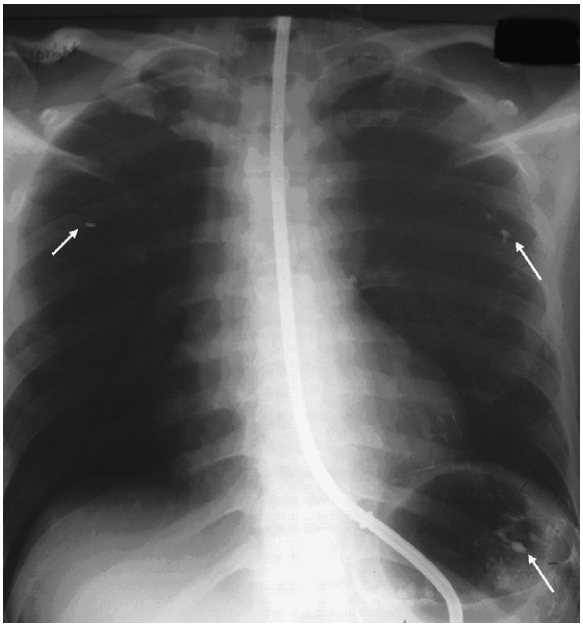

Figure 1 Chest radiograph performed 1 week after endoscopic injection sclerotherapy, after gastric balloon insertion for control of upper gastrointestinal hemorrhage, demonstrating multiple radiopaque materials in bilateral hemithoraces and at the left upper abdominal quadrant (arrows).

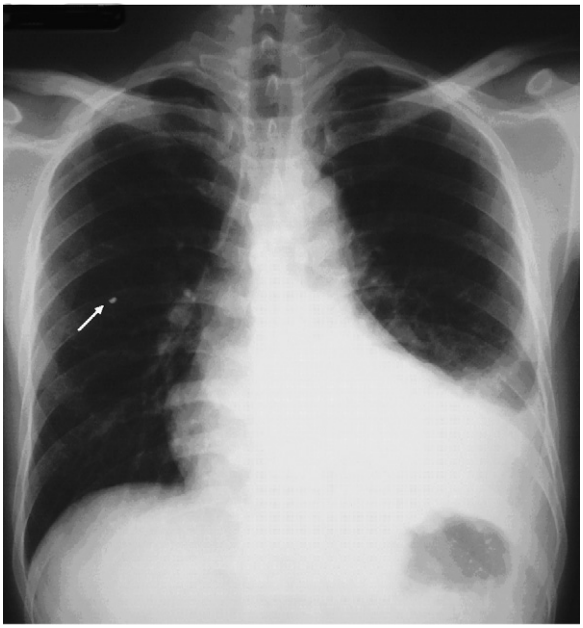

Figure 2 Chest radiograph taken 3 weeks after EIS showing a left pleural effusion. A tubular radiopacity at the right chest is also noted (arrow).