Clinical Case Report

Medicine®

OPEN

Neurological symptoms and spinal cord embolism caused by endoscopic injection sclerotherapy for esophageal varices

A case report and literature review

Shiqian Liu, MD, Nannan Wu, MD, Mingkai Chen, MD, PhD*, Xi Zeng, MD, Fang Wang, MD, Qian She, MD

Abstract

Rationale: Spinal cord embolism is a rare complication of endoscopic injection sclerotherapy (EIS).

Patient concerns: We report a case of a 56-year-old man who presented neurological symptoms and spinal cord embolism caused by EIS on esophageal varices. Clinical signs and symptoms, laboratory tests, thoracic magnetic resonance imaging (MRI), and related treatment supported its diagnosis.

Diagnoses: spinal cord embolism.

Interventions: We stopped the hemostatic and anti-coagulation treatment, and switched to nerve nutrition, microcirculation, and hormone therapy, along with administering gastric mucosal protective agents.

Outcomes: The all patient's signs and symptoms and signs of spinal cord embolism were all relieved within 3 months after the clinical treatment.

Lessons: We recommend that neurological symptoms after EIS in patients with esophageal varices should be considered a rare complication. Life-threatening conditions could be avoided by an accurate and timely diagnosis.

Abbreviations: EIS = endoscopic injection sclerotherapy, MRI = magnetic resonance imaging.

Keywords: esophageal gastric varices, sclerotherapy, spinal cord embolism

1. Introduction

Portal hypertension is a progressive complication of liver cirrhosis which causes variceal bleeding in different parts of the body, especially the esophagus and other gastric organs, and is associated with considerable mortality. Randomized controlled trials[1] show that endoscopic injection sclerotherapy (EIS) can reduce re-bleeding and hospitalization time. Crafoord and Frenckner[2] first reported in 1939 that EIS could control active variceal bleeding, and this approach was subsequently adopted by clinicians to treat patients with heavy varicose veins and high risk of hemorrhage. However, EIS may lead to a range of neurological complications, including the rare spinal cord embolism. In addition to presenting our case report, we have reviewed existing literature on this rare complication associated with esophageal varices after EIS.

2. Case presentation

A 56-year-old man with a history of hepatitis B cirrhosis was admitted to our hospital's emergency department on account of upper gastrointestinal hemorrhage. He was eventually treated with endoscopic tissue glue injection. Following the second and third operations spaced across monthly intervals, endoscopic examination was conducted, and the patient underwent ligation of esophageal and gastric varices with tissue glue injection under intravenous anesthesia. Post-ligation, 5 check-ups were conducted under endoscopy and bleeding was only seen during the first examination.

The cirrhotic condition of the patient before operation was classified as grade A based on the Child-Pugh classification. The endoscope used was Olympus GIF-Q260J (Olympus Corporation, Tokyo, Japan) and Empilan KB (Shaanxi Tianyu Pharmaceutical Co., Ltd, Xian, China) (10 mL: 100 mg per bottle) was selected as the sclerosing agent. After intravenous anesthesia, the endoscope was removed and then re-inserted with an NM-400L-0423

Editor: N/A.

SL and NW shared co-first authorship.

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee. Written informed consent was obtained from the patient for publication of this case report and accompanying images. For this type of study formal consent is not required. This article does not contain any studies with animals performed by any of the authors.

Pro. Mingkai Chen is a consultant to this paper. He received research funding from Wuhan University. The other authors have no conflicts of interest to disclose.

Department of Gastroenterology, Renmin Hospital of Wuhan University, Wuhan, Hubei Province, P.R. China.

* Correspondence: Mingkai Chen, Department of Gastroenterology, Renmin Hospital of Wuhan University, No. 238 Jie Fang Road, Wuhan 430060, Hubei Province, P.R. China (e-mail: kaimingchen@163.com).

Medicine (2018) 97:18(e0622)

Received: 22 December 2017 / Accepted: 10 April 2018

http://dx.doi.org/10.1097/MD.0000000000010622

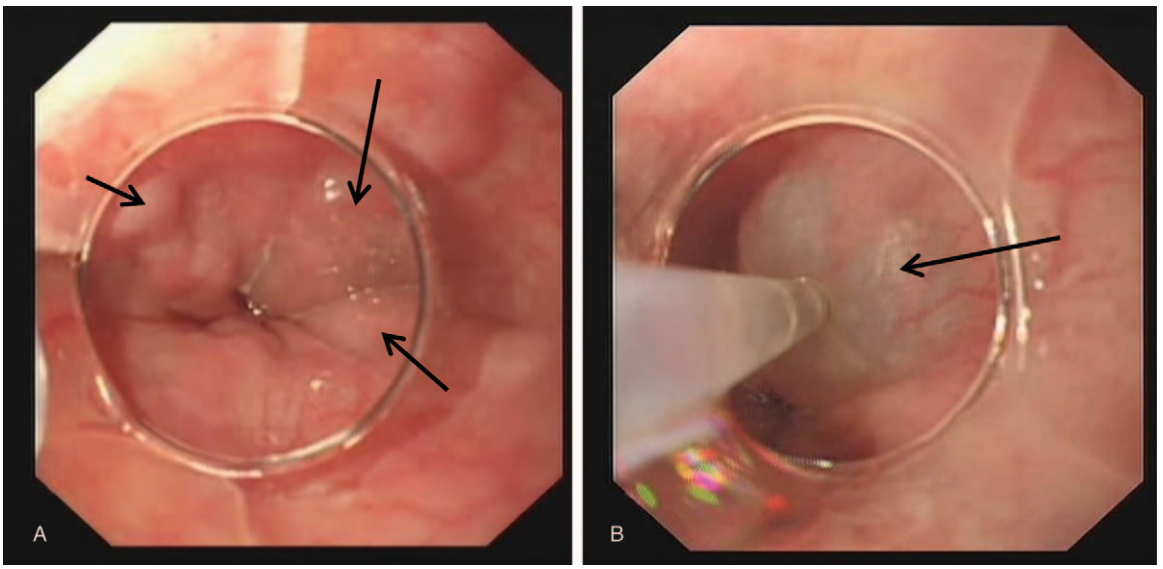

Figure 1. A, Illustration of 3 blue varices was seen in the lower esophagus. B, Displays that sclerosing agents were injected by syringe needle.

needle attached to the tip. Endoscopy revealed coarse esophageal mucosa with a scar formation in the middle, and 3 blue varices in the lower esophagus (Fig. 1). Red Wale signs were absent in the fundus of stomach. Around 20 mL of the sclerosing agent was injected intravenously and paravenously at each position of the esophagus. After the operation, the varices paled and active bleeding ceased. Although a small amount of erosion was seen at the prior injection site, the varicose vein group and bleeding point were not found. The patient was safely shifted back to the ward after the operation, and was recommended complete bed rest. At the same time, a series of palliative treatments such as acid suppression, hemostasis, and somatostatin injection were given.

The patient complained of weakness and numbness in the lower limbs 6 hours after operation, along with abdominal distension and slight dysuresia. His vital signs were stable, lower extremities were normal, the right limb muscle strength was IV level, and bilateral Babinski signs were positive (Table 1). On the basis on these symptoms, we conducted a magnetic resonance imaging (MRI) of the thoracic and lumbar spine within 12 hours postoperation and detected spinal cord embolism. Therefore, we stopped the hemostatic and coagulation regimen immediately and started nerve nourishment and microcirculation therapies, along with dexamethasone injections. Three months after this treatment, cervical and thoracic MRI (Fig. 2) did not show any embolism in the spinal cord.

3. Discussion

Patients who survive an episode of acute variceal hemorrhage have a very high risk of re-bleeding and even death. Although EIS can control variceal bleeding, it is associated with a range of complications such as esophageal stricture, perforation, bleeding,

Table 1

Relative physical sign and neurologic examination after EIS.

Days	Condition of urination and defecation	The left lower extremity	The right lower extremity
0	Retention	Accurate positioning; Babinski(+)	Accurate positioning; myodynamia grade for IV; Babinski(+)
7	Retention	Myodynamia grade for IV; temperature sense was poor; patellar tendon reflex and deep tendon reflex(–)	Myodynamia grade for III; temperature sense was normal; patellar tendon reflex and deep tendon reflex(–)
20	Retention	Myodynamia grade for V; the abnormal temperature sense was above knee about 10 cm	Myodynamia grade for V; temperature sense was normal
24	Retention	Myodynamia grade for V; the abnormal temperature sense level was around knees	Myodynamia grade for II+; temperature sense level was normal
25	Retention	Myodynamia grade for V; the abnormal temperature sense level was under knee about 10 cm	Myodynamia grade for III; temperature sense level was normal
27	Press the belly to assist urinate by himself	Myodynamia grade for V; the abnormal temperature sense level was under knee about 15 cm	Grade for III; temperature sense level was normal
30	Press the belly to assist urinate by himself	Myodynamia grade for V; the abnormal temperature sense level was above ankle about 5 cm	grade for IV; temperature sense level was normal
90	Normal	The double lower limbs felt numb once in a while but can walk freely	The double lower limbs felt numb once in a while but can walk freely

EIS = endoscopic injection sclerotherapy.

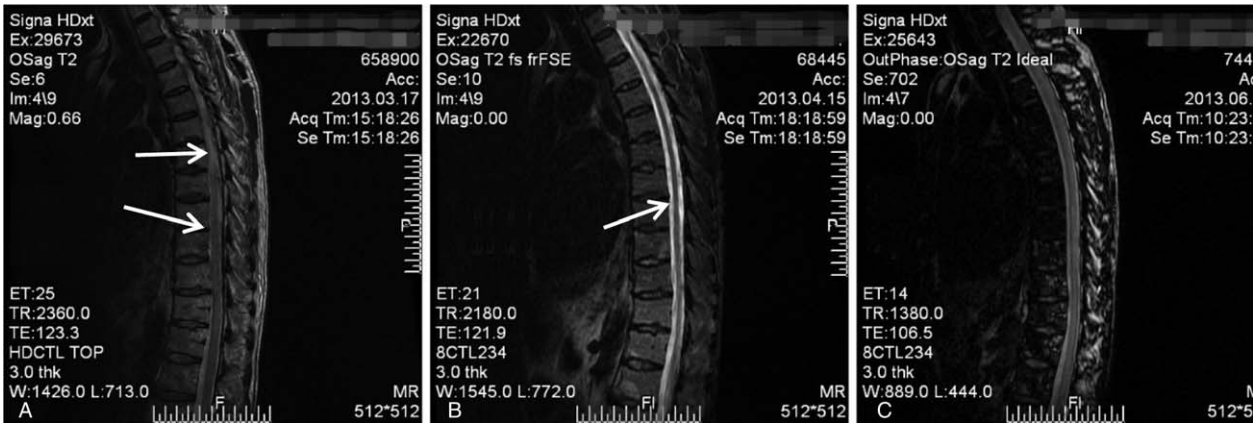

Figure 2. A, T2-weighted magnetic resonance imaging (MRI) of the spine 48 hours after EIS, demonstrating high-signal intensity within the spinal cord from approximately T4 through T11 levels. B, T2-weighted MRI 20 days after the episode, revealing slight persistence of the hyperintense change within the spinal cord, though with clear decrease in intensity compared with 48 hours after EIS. C, Thoracic spine is normal after treatment of 3 months. EIS = endoscopic injection sclerotherapy, MRI = magnetic resonance imaging.

mediastinitis, hemolysis (sodium morrhuate sclerotherapy), and ectopic embolism. The latter commonly takes place in the portal vein, spleen, spinal cord etc. Spinal cord embolism is rare and to the best of our knowledge only 3 cases have been reported so far.[3–5] The pathogenesis of spinal cord embolism induced by EIS is unclear. In 1984, Seidman et al[3] reported a case of a 4-year-old child with cryptogenic cirrhosis who was injected ethanolamine oleate in the varices at the mid-esophagus level. Irreversible paraplegia was documented within 8 hours postoperation, and 2 years later she died from gastrointestinal bleeding. Various hypotheses have been put forward to explain the passage of the sclerosing agent from the esophagus to the anterior spinal artery. In 1996, Heller et al[4] reported a case of a middle-aged woman with primary biliary cirrhosis who showed vein embolism after endoscopic sclerotherapy. Clinical examination 1 month later revealed a right Brown-Sequard syndrome with mild weakness in the right lower-extremity and sensory ataxia with diminished pinprick sensation. The suspected etiology was liver cirrhosis progressing to portal hypertension, and the associated vascular, anatomical, and hemodynamic changes. In 2003, Debette et al,[5] described a 65-year-old patient who suffered from alcoholic cirrhosis and received EIS for active bleeding from esophageal varices. On the third day postoperation, he developed a rapidly progressive motor deficit in the left leg. Three months later, the patient was still in poor condition and the recovery was limited to very faint distal movements of the toes. Considering that this patient also suffered from limb weakness and urine retention 6 hours after the operation, the most likely reason was spinal cord embolism.

The spinal cord artery mainly supplies to the anterior, posterior, and radicular spinal arteries. A segment of the radicular spinal artery, specifically the thoracic spinal artery, is supported by intercostal arteries. Spinal cord veins empty into the vertebral venous plexus via anterior and posterior spinal veins, with the latter ascending to the veins of medulla oblongata and contacting with the portal vein. The low pressure and the blood flow direction of the internal vertebral venous plexus, part of a network of valveless spinal veins, are often affected by thoracic and abdominal cavity pressure. They are susceptible to thrombus or particulate emboli when they are pressed to the spinal cord during weight lifting, coughing, or bowel movements. Moreover, arteriovenous fistula may also exist. The intravenous and paravenous injection of the sclerosing agent may also injure vascular endothelium and stimulate vascular inflammation. In fact, the aim of injecting the sclerosing agent into the submucosal tissue of varices is to injure the tissue and produce a localized inflammation, which proceeds to fibrosis that can protect the varices. Under these circumstances, esophageal mucosal surface tension is increased, which inhibits local capillary proliferation and prevents the recurrence of varicose veins.

Esophageal varices are supplied to the left gastric vein towards the direction of the head and split up above the stomach, and its pre-ramus branches out in the front of the stomach. It enters into the esophageal vein through cardia, and its posterior branch moves upwards to form para-esophageal varices that run through intercostal arteries and azygos vein. Therefore, the sclerosing agent enters into the spinal cord arteriovenous system through intercostal arteries and azygos, and forms ectopic embolism which leads to spinal cord ischemia, subsequently resulting in the symptoms of spinal cord damage. In addition, our patient went through 2 endoscopic treatments, which disrupted the portal system circulation and opened some previously closed communicating branches. Therefore, the sclerosing agent got into these spinal cord vessels and lead to myelitis and related symptoms. As the patients' clinical manifestation clearly pointed to spinal cord embolism, we stopped using hemostatic and blood-clotting-enzymes and switched to nerve nourishment, improved microcirculation, hormone therapy, and injection of gastric mucosal protective agents. Unlike the 3 reported cases of spinal cord embolism, our patient fully recovered after 3 months of treatment. His muscular strength and tone of bilateral lower limbs were normal. Although the complication of spinal cord embolism caused by EIS is rare, early detection and treatment can improve prognosis.

4. Conclusion

The severity of spinal cord embolism is directly proportional to the dose of sclerosing agent. In addition, intravascular injection results in a higher risk of embolism compared with paravasal injection. Therefore, we recommend paravenous as opposed to direct injection of the sclerosing agent into esophageal varices. Excessive manipulation of the esophagus and stomach should

also be avoided, and adhesive injection is not appropriate for some gastric varices of the big shunt branch.

Author contributions

Data curation: Shiqian Liu.
Investigation: Nannan Wu.
Project administration: Fang Wang.
Resources: Xi Zeng, Qian She.
Writing—original draft: Shiqian Liu.
Writing—review and editing: Mingkai Chen.

References

[1] The Copenhagen Esophageal Varices Sclerotherapy ProjectSclerotherapy after first variceal hemorrhage in cirrhosis. A randomized multicenter trial. N Engl J Med 1984;311:1594–600.
[2] Crafoord C, Frenckner P. New surgical treatment of varicose veins of the oesophagus. Acta Otolaryngol 1939;27:422–9.
[3] Seidman E, Weber AM, Morin CL, et al. Spinal cord paralysis following sclerotherapy for esophageal varices. Hepatology 1984;4:950–4.
[4] Heller SL, Meyer JR, Russell EJ. Spinal cord venous embolism following endoscopic sclerotherapy for esophageal varices. Neurology 1996;47:1081–5.
[5] Debette S, Gauvrit JY, Desseaux G, et al. Paraplegia after ligation of esophageal varices. Neurology 2003;60:879–80.