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Clinical reports

Ruptured superior mesenteric artery aneurysms during treatment of tetanus: a case report

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Introduction

Tetanus is an acute neurologic disease characterized by general muscle rigidity, spasm, and autonomic dysfunction. Because of autonomic cardiovascular disturbances, severe tetanus is accompanied by hypertension and tachycardia alternating with hypotension, bradycardia, and cardiac arrest [1,2]. Moreover, sedative drugs used in conjunction with antihypertensive agents make the interpretation of hemodynamic change difficult [1,2]. Aneurysm of the superior mesenteric artery (SMA) is a rare disease, and rupture may induce hemorrhagic shock. Sun et al. [3] reported a patient who died of uncontrolled bleeding from ruptured intestinal aneurysm during treatment of severe tetanus. We report a patient who had ruptured SMA aneurysms while being treated for severe tetanus, with both diagnosis and treatment being ultimately successful.

Case report

A 56-year-old man was transferred to our hospital for treatment of tetanus. The diagnosis of tetanus was based on muscle rigidity and an episode of dirty-nail injury to his toe 7 days before. There was no remarkable medical history except essential hypertension for more than 10 years. On admission, blood pressure was 164/94 mmHg, heart rate was 130 beats per minute, and body temperature was 38.2°C. He received antitetanus toxoid, human tetanus immunoglobulin, and penicillin G intravenously as the usual treatment. Although he was conscious and alert, he exhibited signs of lockjaw, which progressed to opisthotonos 10 h after admission. The patient's muscle spasms became so severe that it was necessary to intubate his trachea, using intravenous midazolam and vecuronium bromide to allow controlled ventilation.

After the onset of severe muscle spasms, cardiovascular instability occurred frequently. Despite the administration of intravenous diltiazem hydrochloride 5 mg hourly and nicardipine 1 mg as required, the patient's hemodynamic state remained unstable, and his systolic blood pressure exceeded 200 mmHg on several occasions. His condition was classified as very severe tetanus, as graded by modified Ablett's classification [1,2].

One week following admission, the patient suddenly developed hypotension and tachycardia (Fig. 1). Although he had often shown the alternate hypertension and hypotension caused by sympathetic storm, we suspected hemorrhagic hypotension in this situation. This suspicion was confirmed by a decrease in hemoglobin concentration from 12.5 to 10.7 $g \cdot dl^{-1}$ after fluid resuscitation. A massive intraperitoneal hematoma was detected by abdominal ultrasonography and computed tomography. The emergency arteriography showed bleeding from multiple SMA aneurysms (Fig. 2). We used a 5-Fr catheter (Mallinckrodt) via the right femoral artery. Embolization was then performed with eight coils (MWCE-35-4-3, MWCE-35-5-8, and MWCE-35-2-3; Cook), gelatine (Gelfoam), and 3-0 silk strings. After embolization for the aneurysms of the main trunk, the patient's hemodynamic state returned to a normal range (Fig. 2).

Temporary paralytic ileus resulted from the embolization and the peritoneal hematoma, requiring treatment with sodium picosulfate orally, panthenol and dinoprost ($PGF_{2\alpha}$) intravenously, and glycerin

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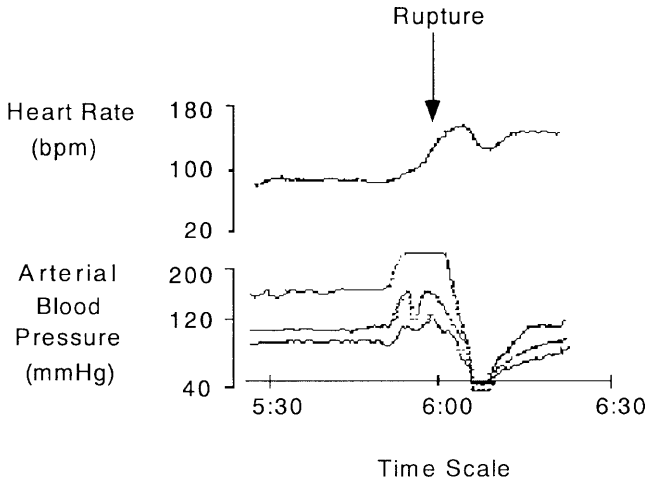

Fig. 1. Hemodynamic changes after rupture of aneurysms (*arrow*). Heart rate increased and blood pressure decreased immediately after rupture

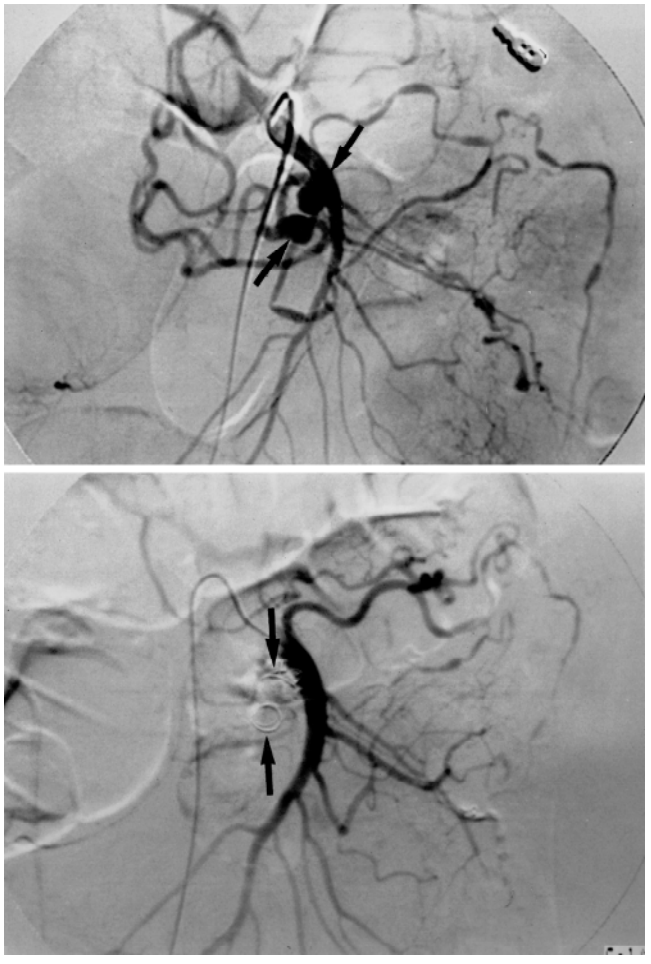

Fig. 2. **a** Main superior mesenteric artery aneurysms on angiogram (*arrows*). **b** After embolization using coils, gelatin, and silk strings, the main superior mesenteric artery aneurysms (between the *arrows*) disappeared

enema. Because his hypertensive state due to tetanus persisted after the episode of the rupture of SMA aneurysms, we used intravenous diltiazem hydrochloride 25 mg·h^{-1} and phentolamine mesylate 5 mg·h^{-1} continuously. In addition, nicardipine or propranolol hydrochloride was given intravenously as required. Three months after admission, the patient was discharged from the hospital and has been symptom-free for three years, with no recurrence of mesenteric ischemia or peritoneal bleeding.

Discussion

The combination of severe tetanus and SMA aneurysms made management difficult. First, uncontrolled hypertension might have caused rupture of aneurysms. Second, the diagnosis of rupture was complex because of autonomic instability and treatment with sedative agents.

On the basis of the degree and extent of muscle spasms and involvement of respiratory and autonomic functions, tetanus in patient was graded III-B, the most severe grade. Autonomic instability could not be controlled, despite the use of a combination of drugs, including sedative, muscle relaxant, and antihypertensive agents. Sudden hypotension is observed during severe tetanus because of the withdrawal of sympathetic activity with autonomic nervous system dysfunction [1–3]. However, when prolonged severe hypotension occurs, we need to consider the differential diagnosis of shock status, such as pump failure (myocardial infarction, etc.), hypovolemia (hemorrhage, etc.), or failure of vascular tone. In this case, we first excluded acute heart failure and myocardial infarction by echocardiography and electrocardiography. Hypovolemia was then demonstrated by the small cardiac size on echocardiography, and acute anemia suggested bleeding of unknown origin. We presumed that the origin of the bleeding was impending rupture of the aorta, gastrointestinal bleeding, or abdominal hemorrhage. Immediately, ultrasonography showed massive intraperitoneal hematoma. In previous reports, ultrasonography was important in identifying the SMA aneurysm itself [4–6]. However, the aneurysms themselves were too small to be detected by ultrasonography in this case. Therefore, acute intraperitoneal bleeding of unknown origin indicated the need for emergency angiography [7].

Treatment of patients with ruptured SMA includes surgical procedures, either ligation, endoaneurysmorrhaphy, or bypass, with or without bowel resection [7–10]. However, Bindman et al. [11] reported the first application of transcatheter embolization for treatment of a ruptured SMA aneurysm. In the present case, the

number, location, and small size of the aneurysms suggested that any surgical procedure would be difficult. Furthermore, owing to excessive sympathetic nervous system activity, anesthetic management was expected to be even more challenging. Therefore, transcatheter embolization was chosen as the best option available to treat this patient's SMA aneurysm.

SMA aneurysm may occur in several ways. A blunt traumatic injury and arteriosclerosis with hypertension have occasionally provoked aneurysm. Several reports [7–10] have revealed mycotic aneurysm to be associated with infectious endocarditis. SMA aneurysms have been reported with congenital etiology [12] and in association with Ehlers-Danlos syndrome [13]. The patient in this case had no predisposing disease except tetanus and hypertension. Sun et al. [3] reported one patient who died of a ruptured mycotic aneurysm in the jejunum during treatment of severe tetanus. However, evidence of a causal relationship between tetanus and SMA aneurysm is still lacking.

In summary, we have reported a 56-year-old man with ruptured multiple SMA aneurysms during treatment of severe tetanus. Although diagnosis of ruptured multiple SMA aneurysms in a patient with severe tetanus may be difficult, transcatheter embolization of ruptured SMA aneurysms may be performed successfully.

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