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Rapid formation of aorto-esophageal fistula complicated by mycotic thoracic aortic aneurysm secondary to infective endocarditis

Maki Setake¹, Yasuhiro Uechi¹, Yuzuru Kinjo¹, Masaru Miyazato¹, Noriya Nakachi¹, Ryosaku Tomiyama¹, Namio Higa², and Akira Hokama³

¹Department of Gastroenterology, ²Department of Cardiovascular Medicine, and ³Department of Medical Checkup, Naha City Hospital, Naha, Okinawa, Japan

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Corresponding Author: Maki Setake, MD
Department of Gastroenterology, Naha City Hospital,
2-31-1 Furujima, Naha, Okinawa 902-8511, Japan.
E-mail: setakemaki@gmail.com

Dear Editor,

A 73-year-old man was admitted with four weeks of intermittent fever. He had a history of total aortic arch replacement for aortic arch aneurysm four years prior. Transthoracic echocardiography and CT scans (Fig. 1A) showed no abnormalities before admission. Laboratory tests showed a white blood cell of 7400/ μ L and C-reactive protein of 4.3 mg/dL. Repeated blood cultures yielded *Streptococcus anginosus* and *Prevotella melaninogenica*, suggesting infective endocarditis (IE). On day 4, transesophageal echocardiography revealed a vegetation on the aortic valve (Fig. 1B), confirming IE. His fever persisted despite antibiotics. On day 8, he suddenly presented with massive hematemesis and hypotension, and hemoglobin was 7.3 g/dL. Endoscopy revealed an elevated lesion with a laceration but no active bleeding in the

esophagus (Fig. 1C, 1D). We first speculated it was a possible mechanical laceration caused by the transesophageal echocardiography because no abnormalities were detected on the CT three weeks prior. The massive hematemesis recurred the next day, and the Sengstaken-Blakemore tube was inserted. CT scans showed a thoracic aneurysm involving the esophagus (Fig. 1E, 1F). A diagnosis of aorto-esophageal fistula (AEF) complicated by mycotic thoracic aortic aneurysm (MTAA) was made, and he was transferred to a university hospital, where he underwent stent graft interpolation followed by minimally invasive esophagectomy. Gastric tube reconstruction was later performed, and the patient recovered uneventfully.

MTAAs are more prone to rupture than arteriosclerotic aneurysms as they are usually not true but pseudoaneurysms that lack layers of the aorta (1). Antecedent infection, including endocarditis, sepsis, predisposes to MTAA. AEF is a rare but life-threatening cause of gastrointestinal bleeding characterized by Chiari's triad, consisting of chest pain, followed by a herald hemorrhage and fatal hematemesis after an asymptomatic period (1), thus early diagnosis and treatment are critical. The AEF related MTAA has been reported to be 7.8%. Hemorrhagic shock, sepsis and multiorgan failure were risk factors for death in AEF (2). Although we performed endoscopy for herald hemorrhages, we failed to recognize the presence of AEF because of the rapid formation of an aneurysm within three weeks. Several cases with rapid expansion (10 days to 3 weeks) of MTAAs have been described (3); however, there have been no reports of rapid formation of AEF after the graft replacement, as shown here. A recent article reported a rapid formation (16 days) of AEF after thoracic endovascular aortic repair, emphasizing prosthetic infection as the most important risk factor (4). Our case underscores the importance of suspecting AEF and conducting repeated examinations even if initial examinations do not reveal any aneurysms.

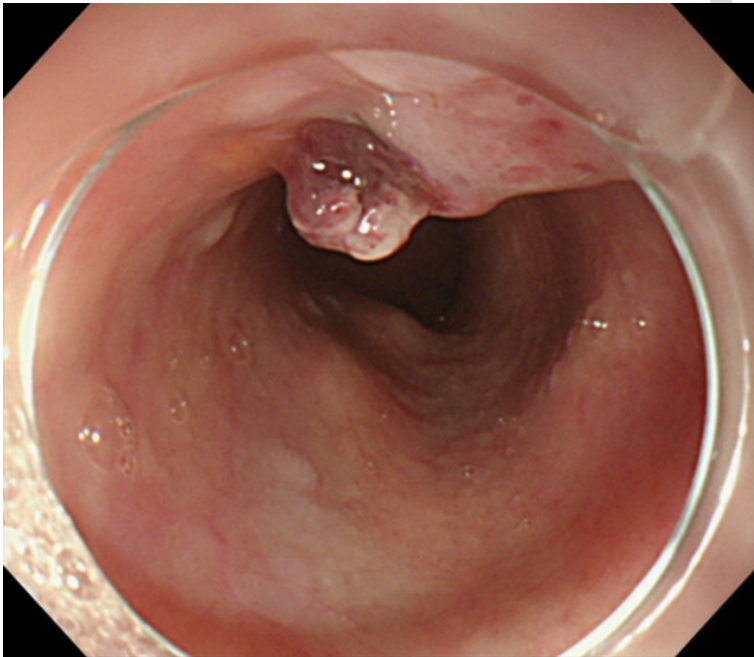
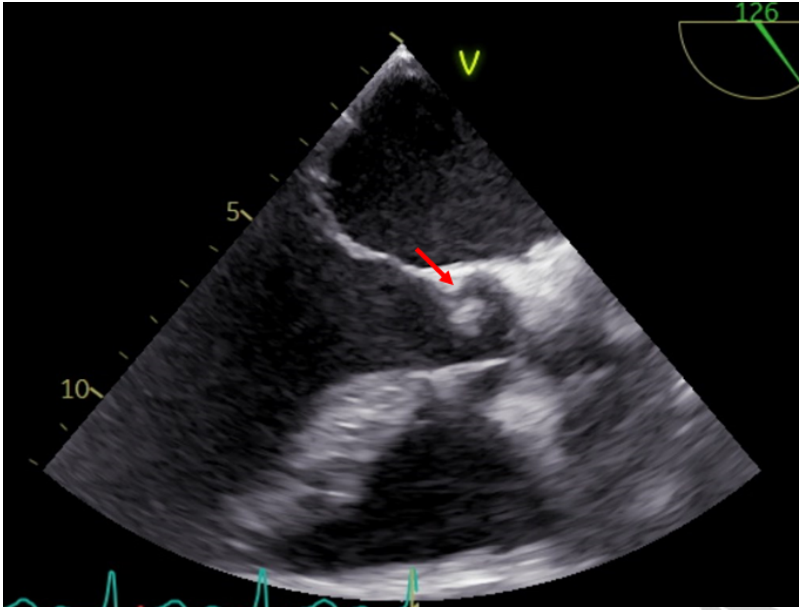
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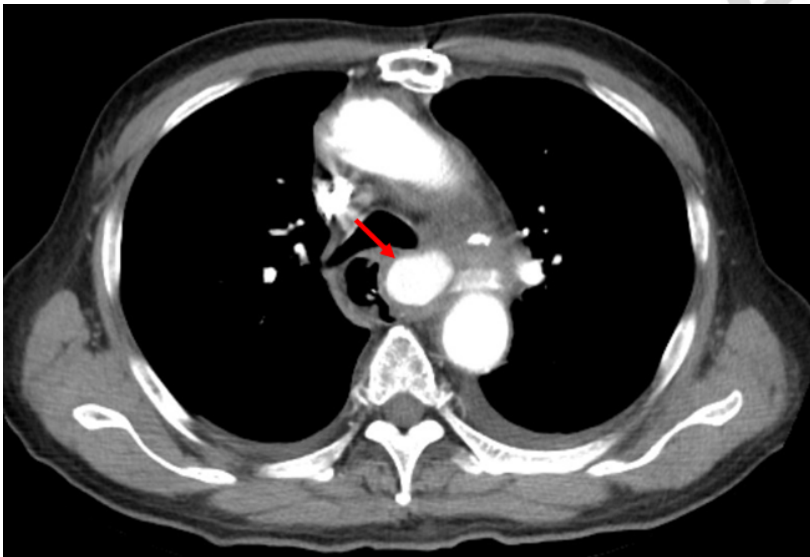
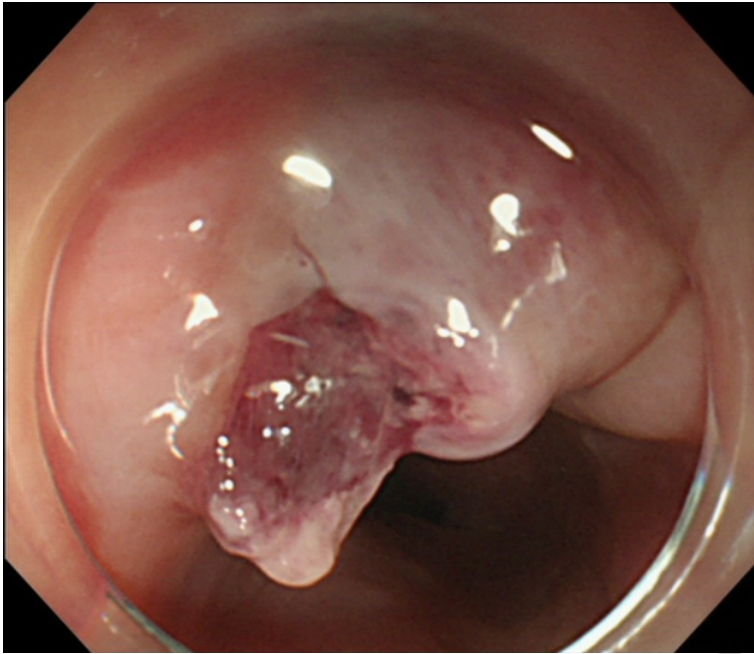
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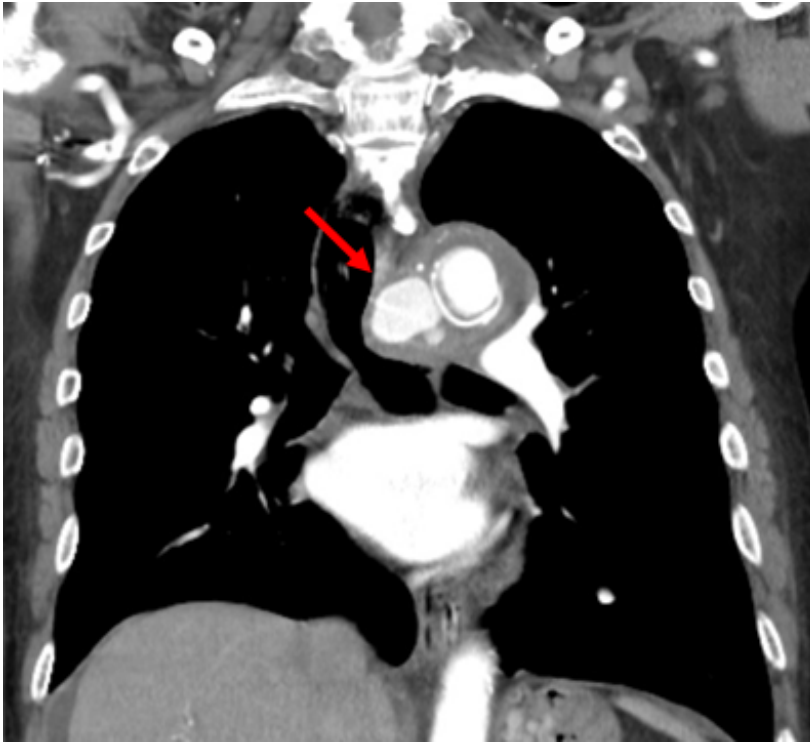


Fig. 1. A. An axial CT scan showed the aortic arch graft but no aneurysm.

B. A transesophageal echocardiography disclosed a vegetation measuring 1 cm in size on the aortic valve (arrow).

C. Endoscopy revealed an elevated lesion with adherent clots 28cm from incisors but no active bleeding in the esophagus.

D. Closer observation showed blood clots arising from the laceration.

E. An axial CT scan showed a saccular thoracic aneurysm (arrow) at the distal end of the aortic graft in communication with the esophageal wall.

F. A coronal CT scan showed the saccular thoracic aneurysm (arrow) with a maximum diameter of 28 mm compressing the trachea.