

## CASE REPORT

Veronica Macchi,<sup>1</sup> M.D.; Andrea Porzionato,<sup>1</sup> M.D.; Romeo Bardini,<sup>2</sup> M.D.; Anna Parenti,<sup>3</sup> M.D.; and Raffaele De Caro,<sup>1</sup> M.D.

# Rupture of Ascending Aorta Secondary to Esophageal Perforation by Fish Bone

**ABSTRACT:** Perforations of both esophagus and aorta after swallowing foreign bodies have been described, with aorto-esophageal fistulas at the level of the descending aorta or aortic arch. We present the case of a 48-year-old man with esophageal perforation by fish bone, mediastinitis, and evidence of perforation of the ascending aorta during surgical drainage of the mediastinum. At autopsy, a fish bone was found under the aortic arch. Serial histological macrosections of the mediastinic block allowed reconstruction of the type of injury suffered and a thorough analysis of mediastinic structures, with preservation of topographic relationships. Direct demonstration of the perforation of the posterior wall of ascending aorta was provided. Histological examination ascribed aortic perforation to migration of the fish bone and direct injury. This is the first anatomical and pathological study of an autopsy case of perforation of the ascending aorta by fish bone.

**KEYWORDS:** forensic science, forensic pathology, histological macrosection, morphological findings, vascular injury, foreign body ingestion, esophageal perforation, endovascular stent

Perforation of both esophagus and aorta after swallowing foreign bodies is reported in the literature (1–8), involving the descending aorta or aortic arch, with aorto-esophageal fistulas. In this report, we present an autopsy study of a delayed death, due to perforation of the esophagus by a fish bone, which had migrated to and penetrated the ascending aorta.

### Case History

A 48-year-old man with a history of hypertension and smoking presented to an emergency department with fever and marked dyspnoea. Symptoms had begun 9 days before hospital admission, starting during a fish meal and consisting of violent back pain in the middle thoracic region, with a single episode of hematemesis. On physical examination, the patient appeared restless. Blood pressure was 150/100, heart frequency 90 beats/min, respiratory rate 25 breaths/min, and temperature 38.8°C. Decreased breath and heart sounds were found. Other examination findings were normal. The ECG was unremarkable. The white blood count was 18,500 cells/mL, with 90% neutrophils. An ear-nose-throat visit was unremarkable. A chest radiogram showed a mild opacity, which enlarged the superior profile of the mediastinum on the right side. No evidence of contrast leak was found on esophagogram. An enhanced computed tomography (CT) scan of the chest showed a flow of hypodense, uneven tissue, which infiltrated the mediastinum on the right side, from the base of the neck to the tracheal bifurcation, and contained numerous air bubbles. The patient was admitted for mediastinic phlegmon and probable esophageal

perforation due to foreign body (fish bone). The mediastinic phlegmon was drained via the right cervical approach and antibiotics were immediately administered. An echocardiogram was performed, followed by pericardiocentesis of 500 cc of cloudy yellow liquid. On the second day, via right posterior thoracotomy, the phlegmon between the superior vena cava and the trachea was drained. During the operation, hemorrhage from a perforation of the ascending aorta was found and was sutured with three knots. Search for a presumed fish bone or other foreign body was unsuccessful. Purulent discharge was cultured for microbiological study. After 2 days, digital angiography revealed an outpouching of 5 × 5 mm in the posterior wall of the ascending aorta, 20 mm proximally to the origin of the brachiocephalic artery. No perforation image could be obtained. On the fourth day a CT check of the chest showed uneven material with air bubbles still present. On the fifth day the patient underwent thoracic esophagectomy with pharyngostomy and placement of a gastrostomy tube for enteral feeding. On the twelfth day, a CT scan of the chest revealed enlargement of the outpouching of the aortic arch (Fig. 1D). Digital angiography showed rupture of the wall of the ascending aorta, and an endoluminal stent graft was inserted into the ascending aorta via the right femoral artery. However, the patient died a few hours after this procedure.

### Autopsy Findings

At autopsy, it was noted that the deceased was well nourished for his age and build, with a height of 176 cm, a body weight of 82 kg, and a body-mass index of 26.5 kg/cm<sup>2</sup>. External examination of the body revealed left pharyngostomy, gastrostomy, bilateral cervicotomy, xifo-umbelical laparotomy, thoracotomy at the level of the fourth intercostal space, and an inguinal access. Two thoracic drainage tubes were present in the right hemithorax and one in the left side.

Examination of the brain was unremarkable. The esophagus was absent. Both lungs showed augmentation of volume, weight (right:

<sup>1</sup>Department of Human Anatomy and Physiology, University of Padova, Via Gabelli 65, 35127 Padova, Italy.

<sup>2</sup>Department of Surgical and Gastroenterological Sciences, University of Padova, Via Giustiniani 2, 35127 Padova, Italy.

<sup>3</sup>Department of Oncological and Surgical Sciences, University of Padova, Via Gabelli 61, 35127 Padova, Italy.

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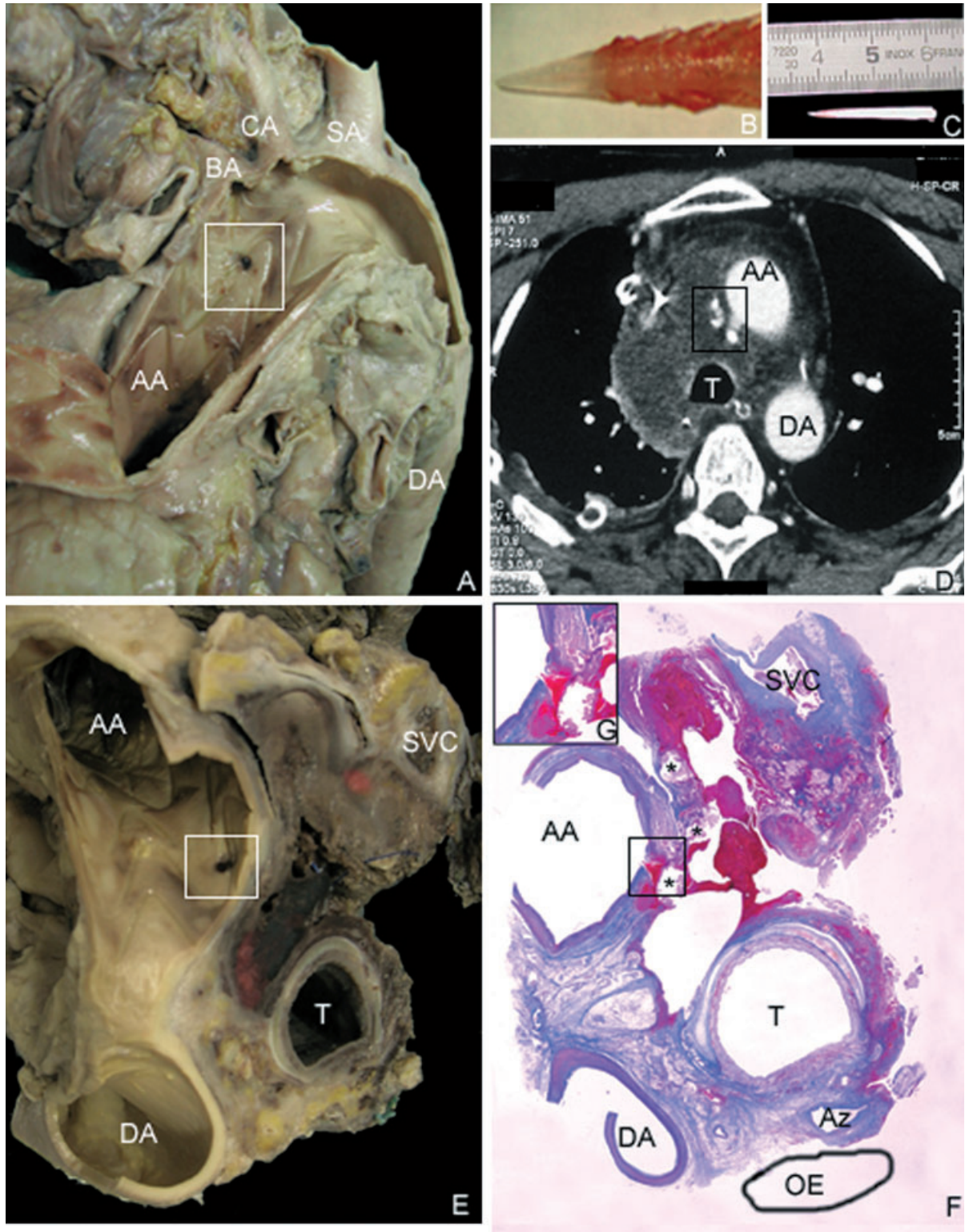


FIG. 1—(A) Left view of mediastinic block. A window on left wall of aorta was opened, showing a small dark area (boxed) on right posterior wall of ascending aorta (AA). (B–C) Whitish fish bone found under aortic arch, with evidence of its saw-shaped edges. (D) Enhanced CT scan of chest, performed after suture of aortic perforation. Outpouching of posterior wall of AA is visible, near three surgical knots (boxed). (E) Transverse slice of mediastinic block performed 2 cm above tracheal carina. Aortic rupture is boxed. (F) Histological transverse macrosection of slice of Fig. E, showing rupture of AA (boxed and magnified in G), with quite regular edges, and position of surgical knots (asterisks) (azan-Mallory, original magnification  $\times 1$ ). Position of removed esophagus (OE) is drawn (BA, brachiocephalic artery; CA, left common carotid artery; SA, left subclavian artery; DA, descending aorta; T, trachea; SVC, superior vena cava; Az, azygos vein).

780 g, left: 740 g), and consistency. Pulmonary surfaces presented multiple dark petechiae, 2–4 mm in diameter. On cut section, both lungs were moderately edematous, with patchy areas of consolidation. A small quantity of mucus was present within the bronchi. Pulmonary arteries were patent. The heart weighed 400 g and its diameters were 9 × 9 cm. Left and right ventricular wall thicknesses were 15 and 5 mm, respectively. Cardiac valves were normal. Coronary arteries showed marked calcific atherosclerotic lesions, mainly in the anterior descending branch of the left coronary artery, with 60% stenosis. Black coagulated blood was found in the mediastinum below the tracheal bifurcation. A whitish spine with saw-shaped edges (a fish bone: length 35 mm, width 2 mm) (Fig. 1B,C) was found in the mediastinic tissue under the aortic arch. A window on the left wall of the aortic arch was opened. After removal of the endoprosthesis, a dark area (3 × 2 mm) of hemorrhagic infiltration was appreciable on the right posterior wall of the ascending aorta, 2 cm proximally to the origin of the brachiocephalic artery (Fig. 1A,E). Mediastinic structures (aorta, trachea, bronchi, azygos vein, superior vena cava) were fixed *en bloc* in 10% formalin for 2 weeks for subsequent histological examination. Postmortem radiological examination of the fish bone confirmed that it was radiotransparent.

Examination of the abdomen did not show synechiae nor abnormal fluid in the peritoneal cavity. The stomach showed a gastrostomy of 1.4 cm diameter in its anterior antral wall, through which the feeding tube was still inserted. The stomach was empty and its mucosa was congestive, but no signs of inflammation were detected. Examination of the other organs revealed only lipidic striae within the descending aorta.

## Histology

Multiple transverse macrosections of the mediastinic block were performed. Slices were embedded in paraffin and cut into 10- $\mu$ m thick sections, which were stained with hematoxylin and eosin and azan-Mallory. On macrosections, the right posterior wall of the ascending aorta showed a transmural rupture, with diameters of 1 and 2 mm on the intimal and adventitial sides, respectively (Fig. 1F,G). The edges of the aortic perforation were quite linear and regular, and showed mild hemorrhagic and granulocytic infiltration. Apart from this injury, the aortic wall showed only mild intimal thickening. Fibroadipose tissue adjacent to the ascending aorta contained blood and granulocytic exudate. The other mediastinic structures, i.e., trachea, bronchi, azygos vein, superior vena cava, and descending aorta, did not show damage.

Histological examination of sections of internal organs taken during autopsy revealed: in brain, mild edema; in lungs, edema; in myocardium, thickening of vascular walls; in stomach, congestion; in liver, mild steatosis; in kidney, a few postinflamed scars in the cortex.

The autopsy conclusion was that the cause of death was a concurrence of hemorrhage and severe mediastinitis.

## Discussion

Several cases of perforation of esophagus and aorta by foreign bodies have been reported (1–8). In most cases, an aorto-esophageal fistula develops, characterized by the three signs of Chiari (9), i.e., painful esophageal injury, asymptomatic interval, and “signal hemorrhage,” leading to exsanguination hours to days later. The esophageal perforation is due to the impact of the foreign body with the anatomical constrictions of the esophagus, located at the level of the cricopharyngeal muscle, aortic arch, and inferior

esophageal sphincter (1). Vasculo-esophageal fistulas are usually located in the aortic arch, descending aorta, or left subclavian artery (10), due to their anatomical relation with the thoracic esophagus. In our case, aortic perforation involved the ascending aorta, which is quite distant from the esophagus (Fig. 1G).

The clinical course between swallowing of the fish bone, with hematemesis, and death, was quite long. The clinical presentation of our subject was typical, with an immediate signal hemorrhage, not taken seriously by the subject himself, which subsequently led to the development of a mediastinic phlegmon, with marked leukocytosis and fever. After perforating the esophagus, the fish bone in the mediastinic adipose tissue developed a phlegmon, due to its high degree of contamination by the esophageal flora. The absence of anatomic-pathological injuries of the esophagus after esophagectomy may be explained by the rapid migration of the bone through the wall, with subsequent wall repair over the following days. On the basis of clinical data, aortic perforation was ascribed to direct injury by the fish bone or to damage of the aortic wall caused by adjacent sepsis.

From the forensic point of view, Byard (11) has recently emphasized that a detailed autopsy investigation is required for evidence of systemic or remote disease when lesions are found within the esophagus. In our case, serial histological macrosections proved very useful in reconstructing the type of injury, permitting thorough analysis of mediastinic structures, with preservation of topographic relationships. Macrosections have usually been applied to anatomical studies (12) and in validation studies of radiological findings in staging of tumors (13). In our case, macrosections directly demonstrated the perforation of the posterior wall of the ascending aorta and the location of the surgical knots, but did not reveal other direct injuries to mediastinic structures. Histological examination ascribed the aortic perforation to migration of the fish bone and direct injury, as the edges of the laceration were linear and regular. Inflammatory infiltration of the aortic wall was minor, excluding secondary erosion.

Migration of fish bones or other foreign bodies has been described in the literature. A fish bone was reported to have injured the thyroid gland after perforation of the cervical esophagus (14) and other structures, reaching the liver after gastrointestinal perforation (15,16). A swallowed sewing needle was also reported to have migrated from the stomach, through the diaphragm and pericardium, up to the left heart ventricle (17). In our case, the fish bone moved anteriorly, without injuring interposing structures such as trachea, bronchi, or descending aorta, and perforated the posterior wall of the ascending aorta. Its saw-shaped edges (Fig. 1C) probably favored its migration. To the best of our knowledge, this is the first report of perforation by fish bone of the posterior wall of the ascending aorta.

As regards the timing of aortic perforation, contemporary perforation of esophagus and ascending aorta may be excluded, due to the distance between the two. The perforation did not show giant cells or fibrous reaction, indicating quite recent chronology. This finding is in accordance with identification of perforation during surgical drainage of the mediastinic phlegmon and subsequent hemorrhagic relapse before death.

From the clinical and medico-legal points of view, it is important to note that treating perforation of the esophagus depends on the cause, location, and severity of the perforation, the interval between perforation and treatment, the patient's characteristics, damage to surrounding tissues, and any concomitant esophageal pathology or injury. If a foreign body is suspected to be in the mediastinum after esophageal perforation, rapid surgical intervention is essential, because of possible migration. Therapy in aorto-esophageal fistula

ranges from open surgery with direct suture, to use of the Sengstaken-Blakemore tube or endovascular treatment (18). In our case, surgical operation and suture of the aortic perforation were timely, but the fish bone could not be located and removed, due to its further migration and the onset of mediastinitis. In addition, its radiotransparency, also confirmed by postmortem radiology, prevented it from being found. An endovascular approach by placing a stent graft in the ascending aorta was also performed, due to radiological and clinical findings of hemorrhagic relapse. From a review of the literature, 18 patients with diagnosis of aorto-esophageal fistula have been treated by endovascular surgery, one of which was caused by ingestion of a foreign body (8). In our case, unfortunately, the stent graft could not prevent death, due to concurrence of hemorrhage and severe mediastinitis.

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Additional information and reprint requests:  
 Prof. Raffaele De Caro, M.D.  
 Department of Human Anatomy and Physiology  
 Università di Padova  
 Via Gabelli 65, 35127 Padova  
 Italy  
 E-mail: rdecaro@unipd.it