

Death Resulting from Pneumocephalus Complicating Endoscopic Food Bolus Retrieval in a Patient with Eosinophilic Esophagitis

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ABSTRACT

Pneumocephalus is a rare complication of esophagogastroduodenoscopy (EGD), but existing literature does not discuss pneumocephalus surrounding endoscopic food bolus retrieval. We present a death involving pneumocephalus complicating endoscopic food removal from the esophagus.

A 40-year-old man presented with dysphagia and suprasternal discomfort 12 hours following chicken ingestion. On flexible endoscopy, chicken was visualized in the distal esophagus. After successful retrieval, a mucosal laceration was noted where the chicken had been lodged. He was unarousable following the procedure and was emergently transported to a hospital, where computed tomography scanning showed pneumocephalus. He was later declared brain dead. The case was referred for medicolegal autopsy.

The brain was examined first, revealing rare air bubbles within meningeal vessels and numerous, diffuse petechiae-like hemorrhages within the brain parenchyma. The esophageal mucosa had focal discoloration and a partial thickness laceration; microscopic examination revealed eosinophilic esophagitis.

Eosinophilic esophagitis is a known risk factor for food bolus impaction and should be suspected in such patients. Pneumocephalus is a rare possible complication of EGD for food bolus retrieval. In patients unresponsive after endoscopy, radiographic detection of potential pneumocephalus should be encouraged to enable timely therapy and improved outcomes, or to supplement autopsy in the event of patient death. Forensic pathologists should understand that pneumocephalus is a potential mechanism of injury/death in patients experiencing esophageal trauma, including injury incurred during EGD. *Acad Forensic Pathol.* 2016 6(4): 703-708

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INFORMATION

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INTRODUCTION

Cerebral air embolism is a rare but life-threatening endoscopic complication. In the context of mucosal disruption, insufflated air may enter the venous and then arterial circulations due to a shunt (e.g., intracardiac or intrapulmonary) or insufficient filtration by pulmonary capillaries, leading to cerebral infarction (1). Its recognition and diagnosis can be difficult, as patients may present with cardiopulmonary instability and neurologic symptoms potentially attributable to sedation-related problems or a stroke of alternate etiology (e.g., thrombotic, hemorrhagic), respectively (2).

Previous studies have described cerebral air embolism and pneumocephalus during endoscopic procedures, including esophageal balloon dilatations, endoscopic retrograde cholangiopancreatography, and esophagogastroduodenoscopy (EGD). These patients have been of all ages, and the majority, but not all (3), have had mucosal disruption either as part of the intended pro-

cedure (e.g., biopsy) or prior to it (e.g., gastric ulcer) (2, 4). Their complications have ranged from seizures and left-sided hemiparesis to coma and brain death (1, 2, 4–6). To our knowledge, there are no existing reports of air embolism during endoscopy for food bolus impaction.

In this report, we present a death related to pneumocephalus occurring as a complication of endoscopic food removal from the esophagus.

CASE REPORT

A 40-year-old previously healthy male with history of dysphagia to meat presented with dysphagia and suprasternal discomfort 12 hours following chicken ingestion. On flexible endoscopy performed under intravenous sedation at an endoscopy center, the chicken was seen lodged in the lower third of the esophagus (**Images 1 and 2**), and the esophageal mucosa was more friable than usual. The food bolus was difficult to snare, and the patient experienced tremendous gag-



Image 1: Foreign body (chicken meat) visualized in mid to distal esophagus on endoscopy.



Image 2: Closer view of meat on endoscopy. Note friable appearance of adjacent mucosa.

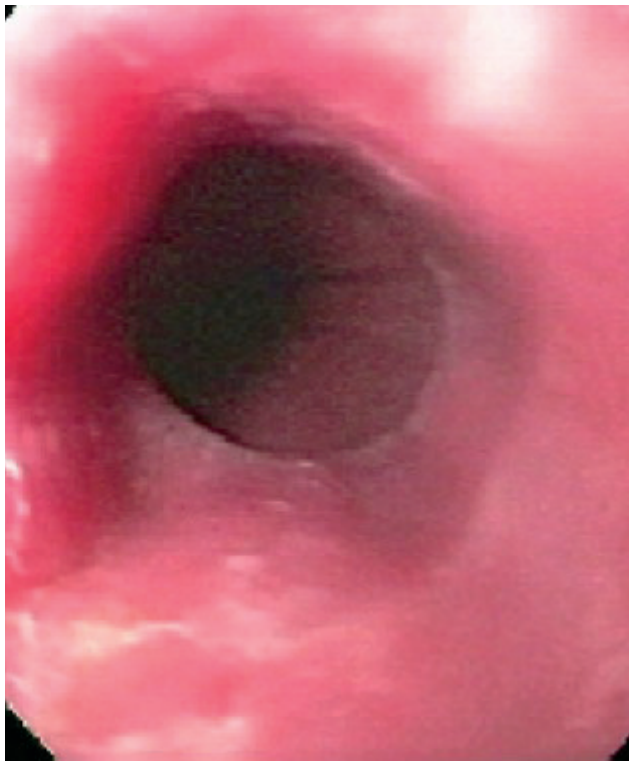


Image 3: Normal distal esophagus, following food bolus retrieval.



Image 4: Esophageal tear in the mid- to distal esophagus, following food bolus retrieval.

ging and coughing during the procedure. After successful retrieval of the food bolus, a mucosal laceration was noted just proximal to the site where the chicken had been lodged (**Images 3 and 4**).

Following the procedure, the patient was unarousable and was emergently transported to a hospital. A subsequent head computed tomography (CT) scan revealed pneumocephalus (**Image 5**), while a chest CT showed pneumomediastinum. The patient was later declared brain dead and the case was referred for medicolegal autopsy.

At autopsy, the brain was examined first. It was diffusely edematous. There were rare air bubbles within meningeal vessels (**Image 6**) and numerous petechiae-like hemorrhages throughout the grey and white matter on sectioning (**Image 7**).

Upon chest plate removal, severe pneumomediastinum was evident by the presence of adipose tissue emphysema (**Image 8**). The esophageal mucosa was focally discolored and contained a 5.5 cm long jag-

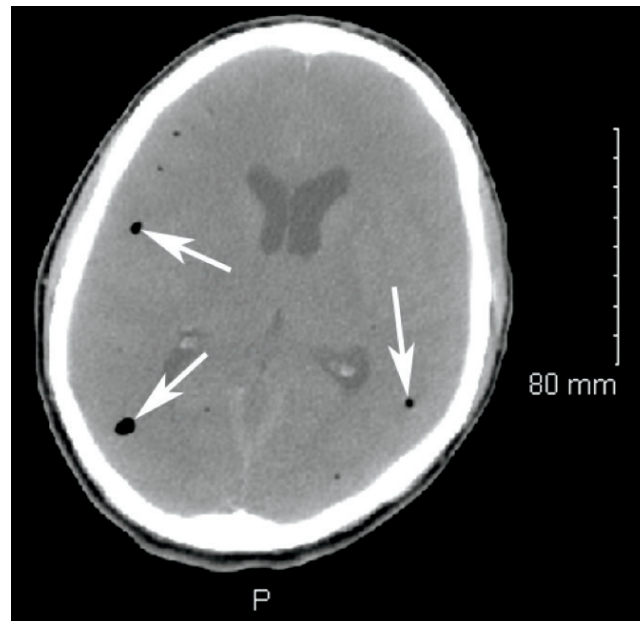


Image 5: Antemortem computed tomography scan of the head, demonstrating the presence of numerous air emboli within the cerebral vasculature (arrows).

ged, vertically oriented, partial-thickness laceration in its anterior aspect, ending 5.0 cm from the gastroesophageal junction (**Image 9**). There was underlying soft tissue hemorrhage. Microscopic examination of the esophagus revealed intense intraepithelial eosinophilia (average 14 eosinophils/high power field [hpf], maximum 41/hpf) throughout the length of the esophagus (**Image 10**), but no eosinophilic microabscesses. There was focal ulceration with abundant columnar-lined epithelium, with focal areas of gastric epithelium. Focal submucosal eosinophilic, hyaline-like material (Congo-red/polarized light-negative) was observed.

Examination of the heart revealed normal myocardium and valves and a closed foramen ovale. The lungs were moderately congested but without emboli. There was mild emphysema and mild mixed bronchial inflammation.

Toxicology testing was noncontributory. A serum tryptase utilizing hospital blood was within normal limits. The cause of death was certified as complications of eosinophilic esophagitis, including esophageal laceration during endoscopic removal of entrapped food bolus, with subsequent air embolism/pneumocephalus. The manner of death was ruled accident.

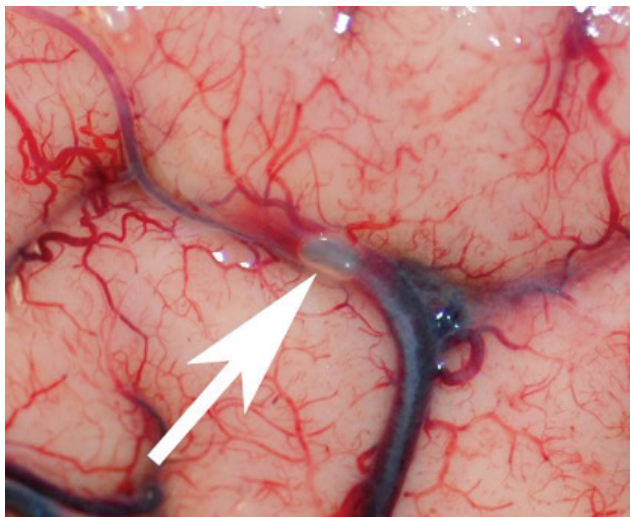


Image 6: Air bubble in meningeal blood vessel (arrow), seen after careful removal of calvarium at autopsy.

DISCUSSION

Eosinophilic esophagitis (EE) is an increasingly prevalent immune/antigen-mediated condition characterized clinically by esophageal dysfunction and pathologically by eosinophilic infiltration; both clinical and microscopic criteria are required for diagnosis (7). Esophageal tissue from patients with EE typically also demonstrates a thickened mucosa with basal layer hyperplasia and papillary lengthening (8).

Dysphagia and food bolus impaction are the predominant presenting symptoms of EE in adults (9), and up to 54% of people with EE require endoscopic food bolus removal (10). Since the chronic inflammation of EE results in a weakened, structurally altered wall, EE is a risk factor for esophageal laceration and complications during endoscopy (11).

A high index of suspicion should be maintained for air embolism and pneumocephalus in cases of cardiopulmonary instability or neurologic symptoms surrounding endoscopy, especially in cases of possible underlying EE or mucosal disruption. Rapid radiographic detection of air embolism by head CT and echocardiography should be encouraged. However, while head CT is highly sensitive, it is only diagnostic if obtained early, since air is rapidly reabsorbed from cerebral arterioles; further, echocardiography and chest CT may not detect a right-to-left shunt (4).

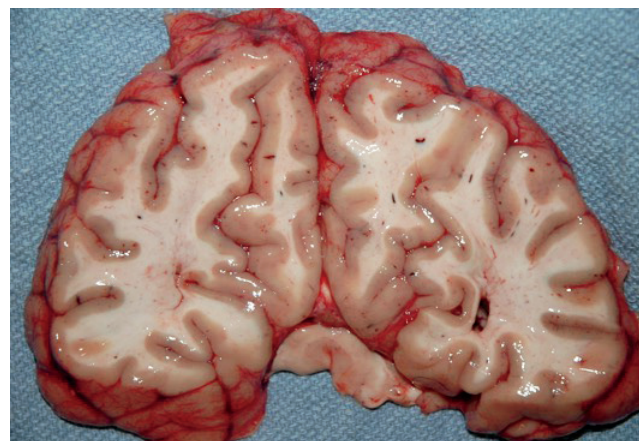


Image 7: Numerous petechiae-like hemorrhages present within white and grey matter on brain sectioning.

Temporizing measures should be initiated immediately if air embolism is suspected, including stopping the procedure, administering hyperbaric or high-flow 100% O₂, starting a high volume normal saline infusion, and placing the patient in Trendelenburg and left lateral decubitus position. In the event of death associated with endoscopy, forensic pathologists should consider cerebral air embolism as a potential mechanism, even in the absence of full thickness esophageal perforation, anatomic shunt (4), or air in the cerebral or meningeal vessels.

CONCLUSION

Eosinophilic esophagitis is a known risk factor for food bolus impaction and should be suspected in such patients. This case demonstrates that pneumocephalus is a rare possible complication of EGD for food bolus retrieval, where EE was a contributing risk factor. In patients unresponsive after endoscopy, radiographic (CT scan) detection of potential pneumocephalus should be encouraged to enable timely therapy and improved outcomes or to supplement autopsy in the

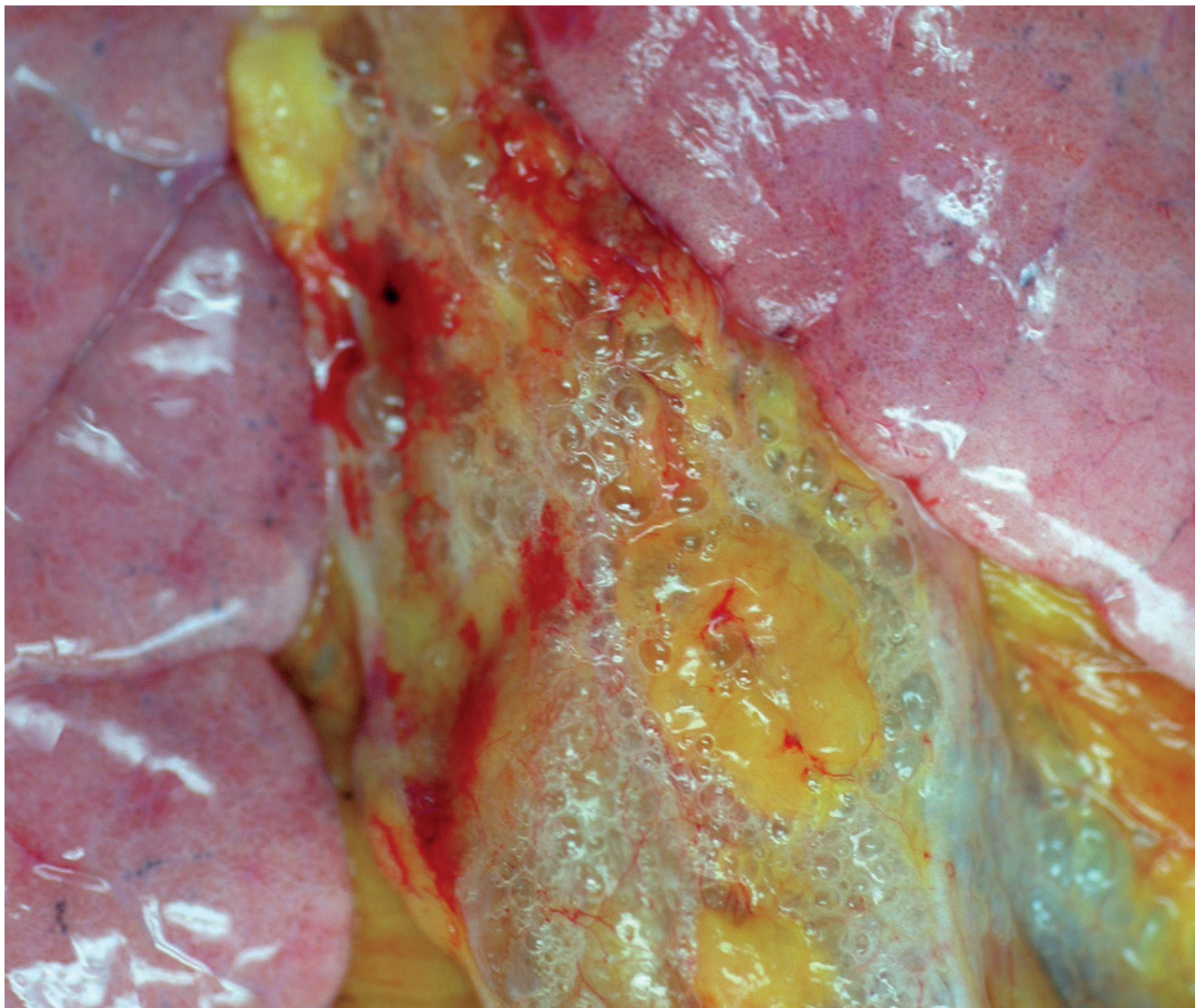


Image 8: Abundant air bubbles within adipose tissue of anterior mediastinum.



Image 9: Longitudinal, partial-thickness, laceration of the mid esophagus (proximal end to left; distal end to right).

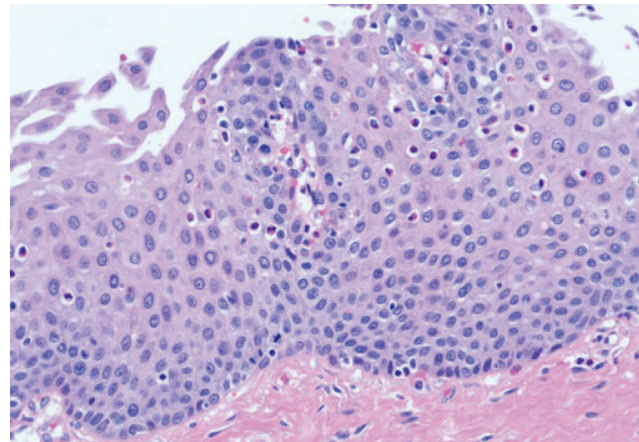


Image 10: Microscopic section of esophagus, near the laceration, showing a mucosal infiltrate of eosinophils (H&E, x200).

event of patient death. Forensic pathologists should understand that pneumocephalus is a potential mechanism of injury/death in patients experiencing esophageal trauma, including injury incurred during EGD.

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