

Brain Abscess Secondary to *Streptococcus Intermedius* Following an EGD with Biopsy in a Patient with Eosinophilic Esophagitis

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ABSTRACT

Brain abscess is a rare complication of esophagogastroduodenoscopy (EGD) with few reported cases in the literature. In this report, we discuss a patient presenting with altered mental status, headache, and dysarthria due to brain abscess caused by *S. intermedius* shortly after an EGD with an esophageal biopsy showing a new diagnosis of eosinophilic esophagitis. We highlight the rare association of EGD and brain abscess, and discuss the importance of prompt diagnosis and treatment.

KEYWORDS: esophagitis, brain abscess, *Streptococcus intermedius*

INTRODUCTION

Esophagogastroduodenoscopy (EGD) is a relatively safe procedure, and complications resulting from it are uncommon. *Streptococcus intermedius* (*S. intermedius*) is a gram-positive bacterium that is commonly found in various sites as part of the normal flora including the gastrointestinal tract. In this case report, we present a patient who developed altered mental status, headache, and dysarthria due to brain abscess caused by *S. intermedius* shortly after an EGD with an esophageal biopsy that resulted in a new diagnosis of eosinophilic esophagitis.

CASE PRESENTATION

A 53-year-old man with no significant past medical history presented to the emergency department (ED) with complaints of food impaction, dysphagia to solids and liquids, and retrosternal pain of one day. He was eating a piece of boneless chicken when he felt it got stuck in his throat one day prior to his presentation. Remarkably, he had similar symptoms about three years prior that resolved on their own. In the ED, his vital signs were within normal limits and he was afebrile. Initial laboratory work-up was unremarkable. X-ray imaging of the neck, chest and abdomen was negative for acute obstructing foreign body, and no free air was visible. An esophagogram was obtained and showed no evidence of contrast extravasation. EGD was performed and showed a large food bolus in the distal esophagus which was removed easily with a Roth net. In addition, a large 5 cm x 1 cm deep

esophageal ulceration was noted underneath the food bolus without obvious perforation. Multiple linear esophageal furrows with rings were also noted in the esophagus which was suggestive of eosinophilic esophagitis, along with grade B reflux esophagitis. Proximal and distal esophageal biopsies were obtained, and the patient was discharged with pantoprazole 40 mg twice daily without complications. The histological evaluation showed squamous mucosa with basal cell hyperplasia, increased intraepithelial eosinophils (up to 25-30/high power field), and lymphocytes on both proximal and distal esophagus tissue samples consistent with a diagnosis of eosinophilic esophagitis (Figures 1 A,B).

Nine days after his discharge, the patient presented to the ED with altered mental status, oriented to self only, severe headache, word finding difficulties and dysarthria. No signs of meningitis were present. Laboratory work-up showed mild leukocytosis at $13.3 \times 10^9/L$ (3.5–10.1). CRP and ESR were within normal limits. Computed tomography (CT) head with and without contrast was remarkable for 55 x 26 mm ring-enhancing left temporal occipital mass in the area of vasogenic edema with midline shift, consistent with brain abscess. Blood cultures were obtained. Treatment with vancomycin, cefepime, metronidazole, dexamethasone and levetiracetam were initiated. Magnetic resonance imaging (MRI) with and without gadolinium and combined with diffusion weighted images, and magnetic resonance venography of brain were performed, and showed a left temporal occipital mass measuring 61 x 33 x 20 mm with extensive surrounding vasogenic edema, 5 mm of rightward midline shift and effacement of the left lateral ventricle suggestive of abscess versus mass (Figure 2A). Cavernous and paranasal sinuses, and mastoid air cells were intact. A panoramic dental x-ray was obtained and it did not show any evidence of osteomyelitis. Transthoracic echocardiography was obtained and no major valve abnormalities or valvular vegetation were observed. A decision to proceed with urgent craniectomy was made as patient started having worsening headache. Left temporal craniectomy was performed with evacuation of the abscess containing copious purulent yellow-green fluid. Aerobic, anaerobic, fungal, and acid-fast bacillus cultures were collected. Gram stain reported few neutrophils but no organisms were seen. Surgical cultures showed the presence of pan-susceptible *S. intermedius*, and fungal and acid-fast bacillus were negative. Patient remained

Figure 1A,B. Squamous mucosa with basal cell hyperplasia, increased intraepithelial eosinophils and lymphocytes on esophageal tissue samples consistent with a diagnosis of eosinophilic esophagitis. (H&E, **A:** 10x, **B:** 40x magnification.)

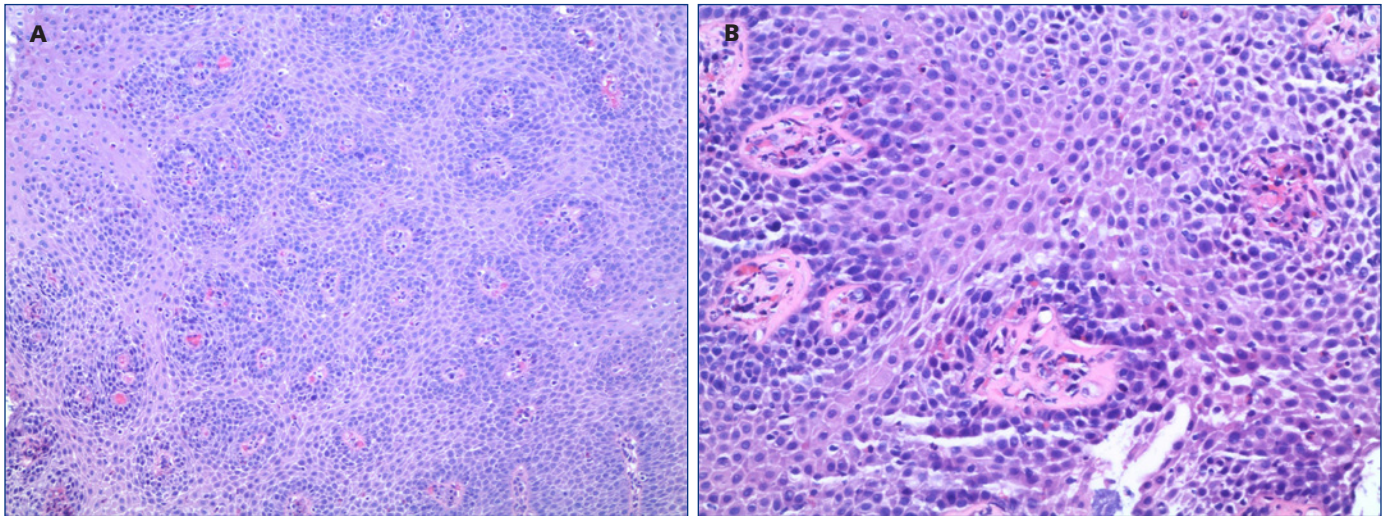
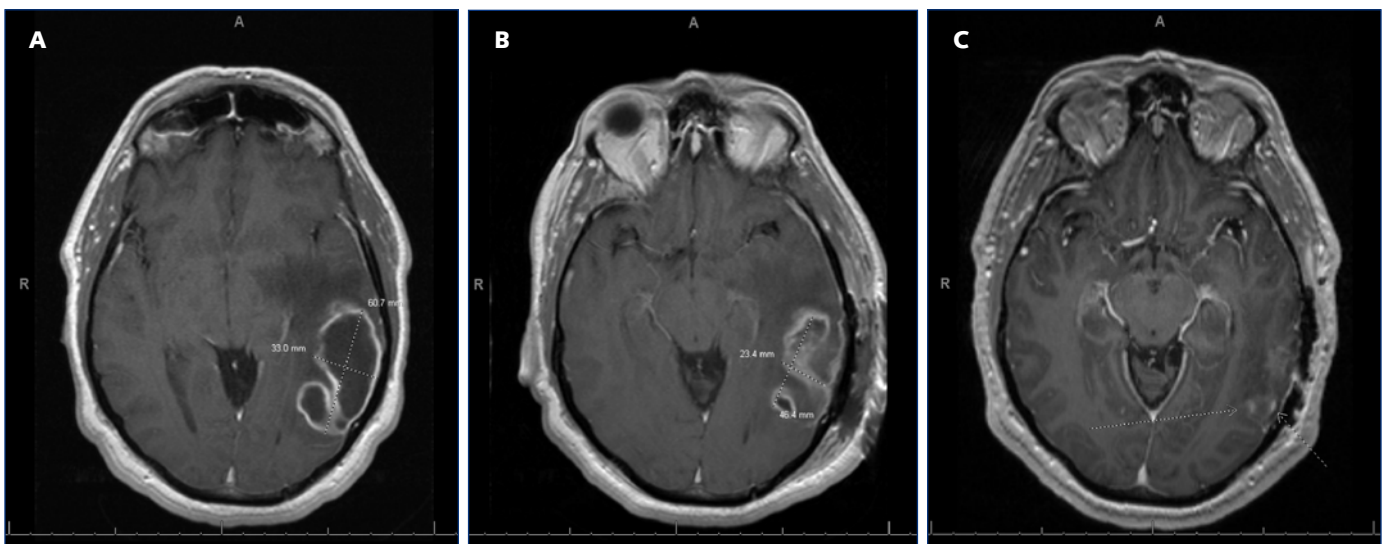


Figure 2. Brain MRI images at presentation (**A:** T1 FLAIR), after craniotomy (**B:** Contrast-enhanced T1), and at 6-week follow-up (**C:** Contrast-enhanced T1 MP-RAGE).



afebrile with normal vital signs while admitted, his mental status improved and dysarthria has resolved. Blood cultures remained with no growth. Peripherally inserted central line was placed, and his antibiotic regimen was modified to Ceftriaxone. Post-surgical MRI of brain with and without gadolinium showed post-surgical changes with no definitive abscess cavity, significantly improved surrounding edema, and absence of mass effect on the left lateral ventricle or midline shift, and marked decrease in the degree of diffusion restriction (**Figure 2B**). Patient remained on Ceftriaxone for six weeks with close follow-up, and repeat MRI showed continued improvement of left temporo-parietal abscess with no discrete abscess cavity, no mass effect on the lateral ventricle or midline shift (**Figure 2C**).

DISCUSSION

Central nervous system infections, such as brain abscess, can occur due to direct extension from nearby structures such as teeth, middle ear, sinuses, mastoid, hematogenous spread from other sites, or as a result of penetrating trauma, and neurosurgical interventions.¹ Prompt recognition and treatment of brain abscesses is crucial as they can be potentially fatal. Despite advancements in diagnostic imaging, neurosurgical interventions and the use of broad-spectrum IV antibiotics, the mortality rate of brain abscess remains high at 10–15%.^{2,3} Brain abscess is a rare complication of EGD, in particular if no esophageal dilation, variceal ligation or sclerotherapy is performed, with only a few reported cases in the literature.

Mucosal or deeper tissue trauma during endoscopic procedures can cause bacteremia consisting of endogenous bacterial flora.⁴ However, this bacteremia rarely leads to clinically significant infections as due to its transient nature and insufficient inoculum to cause an infection in an immunocompetent host.⁵ High-risk endoscopic procedures with the highest rate of bacteremia include dilation of an esophageal stricture, endoscopic sclerotherapy of varices, and endoscopic retrograde cholangiography (ERCP).⁴ Although bacteremia is relatively common after gastrointestinal procedures, infectious complications are uncommon, and routine use of prophylactic antibiotics is not recommended. Routine upper endoscopy and biopsies are considered low-risk procedures, and mean reported rate of bacteremia is estimated to be 4% for both gastroscopy without biopsy and colonoscopy.⁴ Most patients undergoing gastrointestinal procedures, including those with valvular heart disease or prosthetic joints, do not require routine antibiotic prophylaxis.⁴ However, certain procedures may require antibiotic prophylaxis, such as percutaneous endoscopic gastrostomy or jejunostomy tube placement to reduce the risk of peristomal wound infection, ECRP in patients with biliary obstruction that is unlikely to be endoscopically drained, patients with mediastinal cysts undergoing endoscopic ultrasound-fine needle biopsy, patients who have severe neutropenia, and patients with cirrhosis in the setting of ascites and undergoing a procedure with high-risk bacteremia.

Brain abscess after esophageal dilation due to caustic substance ingestion-related esophageal stricture in children has been reported following esophageal dilation in five adults and eight pediatric patients.⁶⁻⁹ Endoscopic sclerotherapy has also been associated with brain abscess development.¹⁰⁻¹³ A case of brain abscess has also been reported after a removal of a foreign object in a pediatric patient.¹⁴ In addition, a case of brain abscess due to streptococcus group F has been reported after endoscopic variceal ligation.¹⁵ Another case was also reported the presence of *S. intermedius* brain abscess in the setting of previously undiagnosed esophageal squamous cell carcinoma.¹⁶

S. intermedius is a beta-hemolytic gram-positive bacterium that is a member of the Streptococcus anginosus group (SAG) and is a part of normal human flora of oropharynx, gastrointestinal tract, and genitourinary tract. SAG consists of three species: *S. anginosus*, *S. intermedius*, and *S. constellatus*. A meta-analysis reporting all 101 cases of *S. intermedius* infections (between years 1996–2019) with available metadata on the literature and it showed that brain abscess was the most common infection (41.6% cases).¹⁷ Dental procedures and sinusitis have been identified as the two most important underlying risk factors for *S. intermedius* infections.¹⁷ In cases where the cause of brain abscess is not evident, investigations to identify the foci of infection should include echocardiography for endocarditis, chest

X-ray to detect lung infections, evaluation for sinusitis and mastoiditis, and examination of teeth.¹

For management, neurosurgical intervention for diagnosis and decompression, along with antimicrobial treatment is recommended.³ The recommended duration of antimicrobial treatment is typically 6–8 weeks.¹ Cranial imaging should be performed post-operatively and at least biweekly until a clinical recovery is observed.³

To the best of our knowledge, this is the first reported case of a brain abscess caused by *S. intermedius* following an EGD and esophageal biopsy in a patient diagnosed with eosinophilic esophagitis. The short time duration between the EGD and the development of brain abscess suggests a possible causal relationship in an otherwise healthy patient. This case highlights the significance of recognizing the risk of serious infections after an EGD, and the urgency of prompt diagnosis and treatment for brain abscesses.

References

1. Brouwer MC, van de Beek D. Epidemiology, diagnosis, and treatment of brain abscesses: *Curr Opin Infect Dis*. 2017;30(1):129-134. doi:10.1097/QCO.0000000000000334
2. Brouwer MC, Coutinho JM, van de Beek D. Clinical characteristics and outcome of brain abscess: Systematic review and meta-analysis. *Neurology*. 2014;82(9):806-813. doi:10.1212/WNL.0000000000000172
3. Brouwer MC, Tunkel AR, McKhann GM, van de Beek D. Brain Abscess. *N Engl J Med*. 2014;371(5):447-456. doi:10.1056/NEJMra1301635
4. ASGE Standards of Practice Committee, Khashab MA, Chithadi KV, et al. Antibiotic prophylaxis for GI endoscopy. *Gastrointest Endosc*. 2015;81(1):81-89. doi:10.1016/j.gie.2014.08.008
5. Shaukat A, Nelson DB. Risks of Infection from Gastrointestinal Endoscopy. *Tech Gastrointest Endosc*. 2007;9(4):225-232. doi:10.1016/j.tgie.2007.08.003
6. Aslan N, Sesli E, Koca T, Şenol N, Akçam M. A rare complication of esophageal dilatation: Brain abscess. *Türk Arch Pediatr Pediatr Arş*. 2017;52(1):50-52. doi:10.5152/TurkPediatr-Ars.2017.2485
7. Gaïni S, Grand M, Michelsen J. Brain abscess after esophageal dilatation: case report. *Infection*. 2008;36(1):71-73. doi:10.1007/s15010-007-6223-8
8. Algoed L, Boon P, De Vos M, et al. Brain abscess after esophageal dilatation for stenosis. *Clin Neurol Neurosurg*. 1992;94(2):169-172. doi:10.1016/0303-8467(92)90077-g
9. Van Even E, Boel A, Van Vaerenbergh K, De Beenhouwer H. Brain Abscesses with Peptostreptococcus: Not Unusual After Oesophageal Dilatation. *Acta Clin Belg*. 2012;67(4):292-294. doi:10.2143/ACB.67.4.2062675
10. Wang WM, Chen CY, Jan CM, Chen LT, Wu DC. Central nervous system infection after endoscopic injection sclerotherapy. *Am J Gastroenterol*. 1990;85(7):865-867.
11. Cohen FL, Koerner RS, Taub SJ. Solitary brain abscess following endoscopic injection sclerosis of esophageal varices. *Gastrointest Endosc*. 1985;31(5):331-333. doi:10.1016/S0016-5107(85)72217-1
12. Shih HI, Lee HC, Chuang CH, Ko WC. Fatal Klebsiella pneumoniae meningitis and emphysematous brain abscess after endoscopic variceal ligation in a patient with liver cirrhosis and diabetes mellitus. *J Formos Med Assoc Taiwan Yi Zhi*. 2006;105(10):857-860. doi:10.1016/S0929-6646(09)60275-8

13. Kovaleva J, Peters FTM, van der Mei HC, Degener JE. Transmission of Infection by Flexible Gastrointestinal Endoscopy and Bronchoscopy. *Clin Microbiol Rev.* 2013;26(2):231-254. doi: 10.1128/CMR.00085-12
14. Louie JP, Osterhoudt KC, Christian CW. Brain abscess following delayed endoscopic removal of an initially asymptomatic esophageal coin. *Pediatr Emerg Care.* 2000;16(2):102-105. doi: 10.1097/00006565-200004000-00011
15. Laviv Y, Ben-Daviv U, Vated M, Rappaport ZH. Brain abscess following endoscopic ligation of esophageal varicose veins. *Acta Neurochir (Wien).* 2010;152(4):733-734. doi:10.1007/s00701-009-0539-3
16. Nayfe R, Ascha MS, Rehmus EH. Esophageal Squamous Cell Carcinoma Presenting with *Streptococcus intermedius* Cerebral Abscess. *Case Rep Pathol.* 2017;2017:e5819676. doi: 10.1155/2017/5819676
17. Issa E, Salloum T, Tokajian S. From Normal Flora to Brain Abscesses: A Review of *Streptococcus intermedius*. *Front Microbiol.* 2020;11. Accessed January 6, 2023. <https://www.frontiersin.org/articles/10.3389/fmicb.2020.00826>

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Disclosures

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