EJCRIM
European Journal of Case Reports in Internal Medicine

A RARE CAUSE OF ACUTE DYSPHAGIA: LISTERIA RHOMBENCEPHALITIS

Hirannya Karunadasa[1], Robbie Chan[1], Ronan O'Connor[1], Alexander Hodge[1,2]

[1] Department of Gastroenterology, Box Hill Hospital, Eastern Health, Melbourne, Australia
[2] Eastern Health Clinical School, Monash University, Melbourne, Australia

Corresponding author's e-mail: Hirannya.karunadasa@monash.edu

Received: 22/04/2026
Accepted: 11/05/2026
Published: 24/08/2026

Conflicts of Interests: The Authors declare that there are no competing interests.
Patient Consent: The patient has provided written consent for the publication of this case report.
This article is licensed under a CC Attribution-NonCommercial-NoDerivatives 4.0 License

How to cite this article: Karunadasa H, Chan R, O'Connor R, Hodge A. A rare cause of acute dysphagia: Listeria rhombencephalitis. EJCRIM 2026;13:doi:10.12890/2026_006814

ABSTRACT

Background: Acute dysphagia is a common presentation to hospital and requires a structured diagnostic approach to distinguish between oropharyngeal and oesophageal causes. Although oesophageal pathology is often suspected, central neurological causes must be considered, particularly in atypical presentations. *Listeria monocytogenes* is a rare but important cause of central nervous system infection, including rhombencephalitis, which may present with progressive cranial neuropathies and bulbar dysfunction, including acute dysphagia. Early diagnosis is challenging but critical, as delayed recognition is associated with significant morbidity and mortality.

Case report: A 37-year-old male presented with acute onset dysphagia and an inability to manage oral secretions following a short prodrome of fever and gastrointestinal symptoms. Initial evaluation suggested oesophageal obstruction; however, urgent endoscopy was unremarkable. His condition deteriorated with aspiration pneumonitis and persistent dysphagia. In the presence of reduced lingual sensation and impaired gag reflex, neuroimaging and lumbar puncture were performed, confirming Listeria rhombencephalitis. Despite targeted antimicrobial therapy, he developed multiple cranial nerve deficits and required a tracheostomy for airway protection. With prolonged intravenous antibiotics and multidisciplinary care, he demonstrated gradual neurological recovery and was discharged for ongoing rehabilitation.

Conclusion: This case highlights the importance of a systematic diagnostic framework in acute dysphagia and maintaining a high index of suspicion for alternative causes to avoid diagnostic anchoring. Early recognition of oropharyngeal features and neurological signs should prompt timely neuroimaging and lumbar puncture. Central causes including Listeria rhombencephalitis should be considered early, as prompt targeted therapy is essential to improve outcomes.

KEYWORDS

Rhombencephalitis, *Listeria monocytogenes*, dysphagia

LEARNING POINTS

- Acute dysphagia requires a structured and careful diagnostic approach to differentiate between oropharyngeal and oesophageal dysphagia.
- Investigation of acute dysphagia should occur in parallel across differential diagnoses, with a high index of suspicion maintained in atypical presentations.
- Listeria rhombencephalitis should be considered in immunocompetent patients with suggestive clinical signs and symptoms.

EFIM
European Federation of Internal Medicine

Publisher
smc media srl

INTRODUCTION

Dysphagia is a common clinical presentation with a broad differential, requiring a systematic approach to distinguish between oropharyngeal and oesophageal pathologies to guide identification of underlying structural, neuromuscular or functional aetiologies.

Oropharyngeal dysphagia refers to impairment in the transfer of oral contents from the mouth to the oesophagus via the pharynx. It is often typified by symptoms of difficulty initiating swallowing, coughing and choking immediately after swallowing and nasal regurgitation[1]. Oropharyngeal dysphagia is most often associated with neurological pathology involving the brainstem, cranial nerves or neuromuscular junction[2]. In contrast, oesophageal dysphagia is characterised by sensation of food 'sticking' within the oesophagus, often accompanied by retrosternal pain, heartburn and oral regurgitation. Oesophageal dysphagia is most commonly caused by obstruction or motility disorders[1]. The differential diagnosis is further refined by temporal profile, with acute and chronic causes differing significantly in aetiology.

Importantly, dysphagia may represent a clinical masquerade of more insidious neurological disease, particularly when accompanied by cranial nerve deficits, ataxia or altered sensorium. Central causes, including brain stem stroke, demyelinating disease, neoplasia and encephalitis, should be considered early, as delayed recognition may lead to significant morbidity. Although less common, infectious aetiologies are critical to identify given their potential reversibility with timely initiation of antimicrobial therapy.

Listeria monocytogenes, a facultative intracellular Gram-positive rod, is a rare but important cause of central nervous system infection. Listeria rhombencephalitis, a manifestation of invasive listeriosis, affects the brainstem and cerebellum and may present with progressive cranial neuropathies and bulbar dysfunction, including dysphagia[3]. Given its rarity and often non-specific prodrome, early diagnosis can be challenging.

We describe a case of Listeria rhombencephalitis presenting with dysphagia, illustrating the importance of a structured diagnostic framework and early consideration of central causes in atypical presentations.

CASE DESCRIPTION

A 37-year-old previously well male presented with a 12-hour history of sudden acute onset dysphagia and an inability to manage oral secretions. This occurred against a background of a three-day prodrome of nausea, vomiting and fever, following consumption of potentially contaminated smoked salmon. He reported retrosternal chest discomfort and had intermittent coughing. Notably, he had tolerated oral intake several hours prior to the onset of the dysphagia. He had no past medical history and was not taking any regular medications. On examination, he was febrile and was unable to tolerate oral secretions, with frequent expectoration of saliva. Neurological examination demonstrated reduced lingual sensation and an impaired gag reflex. He did not demonstrate features of meningism and was oriented. Pathology revealed a mild leucocytosis (13.0×10^9/l) with neutrophilia and C-reactive protein of 3.6 mg/l. Chest radiography was unremarkable.

Given the acute presentation, retrosternal discomfort and an inability to handle secretions, an obstructing oesophageal food bolus was suspected. His fever and gastrointestinal symptoms were thought to be related to a concurrent viral illness. As such the patient underwent urgent upper gastrointestinal endoscopy, which demonstrated no food or foreign body impaction within the oesophagus or alternative cause for his symptoms *(Fig. 1)*.

Following this, his clinical course was complicated by severe hypoxaemic respiratory failure, with computed tomography demonstrating aspiration pneumonitis *(Fig. 1)*, necessitating admission to the intensive care unit and initiation of intravenous antibiotics (amoxicillin and clavulanic acid 1.2 g intravenously, twelve-hourly).

In the absence of clinical improvement and with emerging concern for bulbar dysfunction, further

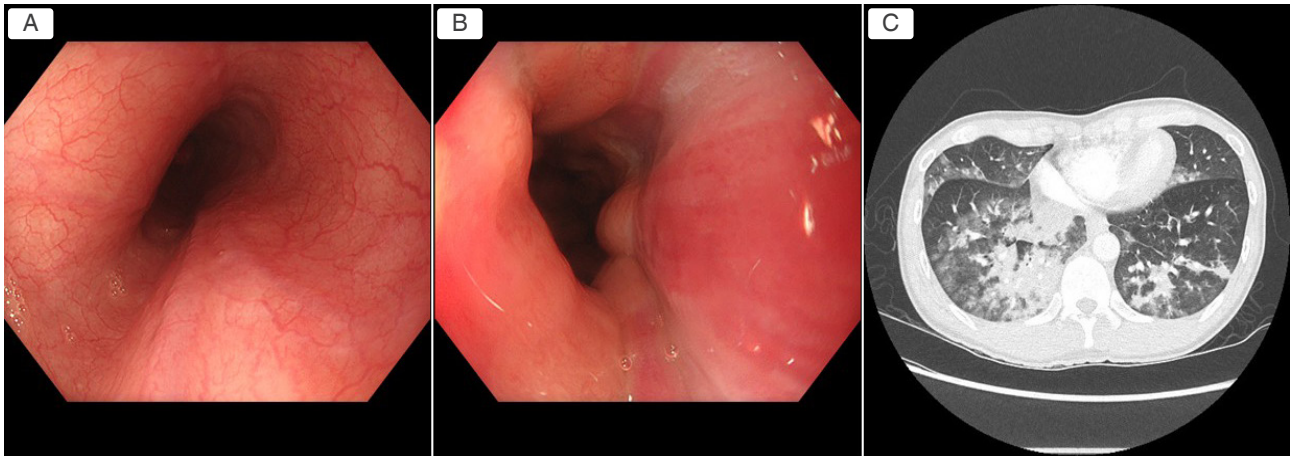


Figure 1. *Endoscopic images of mid oesophagus (A) and gastro-oesophageal junction (B) without appearance of stricture, food bolus or foreign body. Computed tomography of chest (C) demonstrating significant features of aspiration pneumonitis with extensive consolidation involving bilateral lower lobes.*

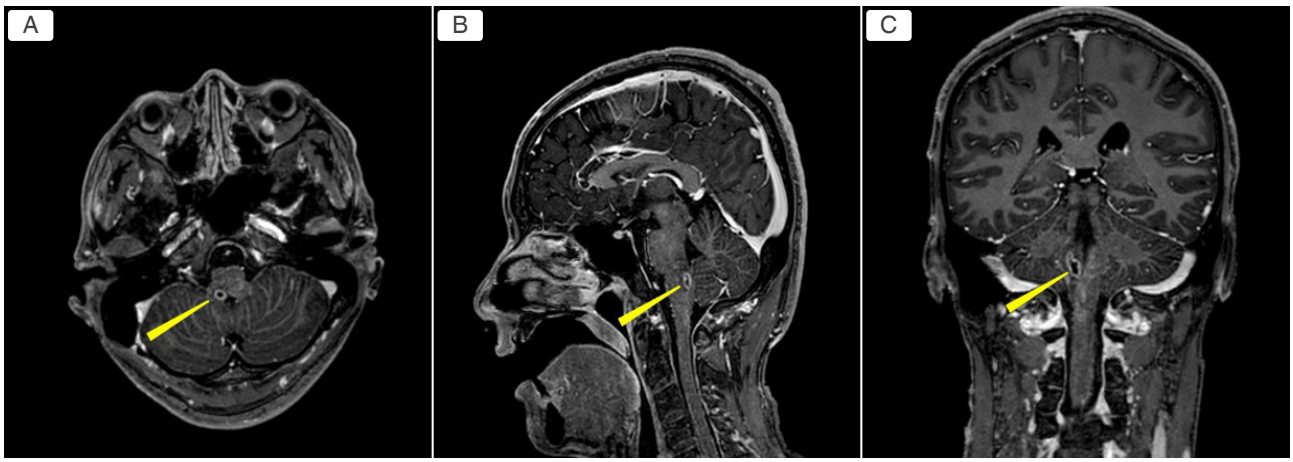


Figure 2. *Axial (A), sagittal (B) and coronal (C) slices of initial magnetic resonance imaging of brain demonstrating a 23 × 5 × 7 mm right posterior medullary abscess (yellow arrow) extending into the cerebellar peduncles consistent with rhombencephalitis.*

neurological investigation was pursued on the second day of admission. The patient was commenced on empiric meningoencephalitis coverage with high dose benzylpenicillin (2.4 g intravenously four-hourly), ceftriaxone (2 g intravenously twelve-hourly) and aciclovir (10 mg/kg intravenously eight-hourly). Urgent lumbar puncture and magnetic resonance imaging (MRI) of the brain were performed. Multiplex polymerase chain reaction assay (BioFire®, FilmArray®, bioMérieux®) was completed on the cerebrospinal fluid (CSF) and detected *Listeria monocytogenes*. The MRI demonstrated a right posterior medullary abscess extending into the inferior cerebellar peduncle, measuring 23 × 5 × 7 mm *(Fig. 2)*, consistent with Listeria rhombencephalitis. High dose benzylpenicillin was continued. Despite targeted therapy, the patient developed progressive neurological deterioration. This included new left-sided cranial nerve XI and XII palsies, a right-sided internuclear ophthalmoplegia and multiple unresponsive episodes likely reflecting ongoing brainstem irritation. He demonstrated persistent loss of protective airway reflexes, consistent with involvement of cranial nerves IX and X. With resolution of respiratory failure but ongoing bulbar dysfunction, a tracheostomy was performed on day 10 of his admission to facilitate airway protection. A flexible nasendoscopy confirmed bilateral vocal cord palsy and persistent inability to initiate swallowing.

Serial MRI imaging over four weeks demonstrated interval improvement in the medullary abscess. The patient was stepped down from the intensive care unit and exhibited partial recovery of neurological function. After a hospital stay of five weeks, he was discharged to the inpatient rehabilitation service for ongoing neurological and functional recovery, supported by multidisciplinary allied health input. A six-week course of intravenous benzylpenicillin was completed and a final MRI demonstrated resolution of the intracerebral abscess and surrounding inflammation.

DISCUSSION

Listeria rhombencephalitis is a manifestation of listeriosis which affects the brainstem and cerebellum, occurring in approximately 17% of patients with listeria infection[4]. *L. monocytogenes* most commonly affects immunocompromised individuals, pregnant patients, neonates and the elderly[5], although invasive disease, including neurolisteriosis, can occur in immunocompetent individuals. Invasive listeriosis is defined by the presence of *L. monocytogenes* within the bloodstream, central nervous system, placenta or uterus and is associated with a high case fatality rate of 20–30%[6].

The high mortality associated with invasive listeriosis is multifactorial. It partly reflects the organism's pathogenic mechanisms, including an intracellular lifecycle enabling immune evasion and actin-mediated cell-to-cell spread facilitating dissemination of infection. Additionally, *L. monocytogenes* is neurotropic and invades the central nervous system via retrograde axonal transport along cranial nerves, resulting in severe complications including meningitis, meningoencephalitis and rhombencephalitis[7].

However, delayed diagnosis represents a significant modifiable predictor of morbidity and mortality, particularly in neurolisteriosis[8]. Clinically, Listeria rhombencephalitis classically presents with a biphasic course, with a prodrome of flu-like illness and gastrointestinal symptoms followed by progressive brainstem and cerebellar dysfunction[9]. Hallmark features include cranial nerve palsies (particularly cranial nerves V, VII, IX and X), cerebellar signs and dysphagia. These manifestations may initially be subtle, evolve over several days and mimic alternative diagnoses such as a stroke, demyelinating disease or neoplastic process[3]. Blood cultures, CSF culture, and polymerase chain reaction assay and MRI imaging are the mainstay of diagnosis of neurolisteriosis and should be obtained promptly when clinical suspicion is present. Computed tomography of the brain is often normal early in the disease course and should not be relied upon to exclude diagnosis. Early recognition of *L. monocytogenes* as the causative pathogen has important therapeutic implications. Standard empirical regimens for meningoencephalitis may not reliably

provide adequate *Listeria* coverage, which requires high dose intravenous penicillin therapy, often in combination with gentamicin. Distinguishing between oropharyngeal and oesophageal causes of dysphagia is critical. In this case, the acuity and presence of retrosternal chest pain initially suggested an oesophageal pathology; it is noted that acute onset dysphagia, in isolation, often suggests a diagnosis of oesophageal food or foreign body impaction[10]. However, several features in this case were suggestive of an oropharyngeal process, including frequent coughing and symptom onset immediately after swallowing[2]. In combination with an impaired gag reflex and lingual paraesthesia, this suggests a central cause and early neuroimaging could have been prioritised. In the early assessment of dysphagia, clinicians should aim to minimise diagnostic anchoring and confirmation biases and allow concurrent evaluation of multiple differential diagnoses.

In this case, endoscopy was performed within six hours of presentation and excluded common oesophageal causes of acute dysphagia. However, reliance on endoscopy in acute dysphagia may delay other investigations and contribute to diagnostic delay.

CONCLUSION

This case underscores the importance of considering central neurological causes in patients presenting with acute dysphagia, particularly in the presence of systemic features of infection or cranial nerve abnormalities. Early neurological examination, prompt neuroimaging and timely lumbar puncture are critical in avoiding diagnostic delay. Importantly, *L. monocytogenes* should be considered even in immunocompetent individuals, as early targeted therapy is essential to reduce morbidity and mortality.

REFERENCES

1. Zerbib F, Omari T. Oesophageal dysphagia: manifestations and diagnosis. Nat Rev Gastroenterol Hepatol 2015;12:322–331. doi: 10.1038/nrgastro.2014.195
2. Rommel N, Hamdy S. Oropharyngeal dysphagia: manifestations and diagnosis. Nat Rev Gastroenterol Hepatol 2016;13:49–59. doi: 10.1038/nrgastro.2015.199
3. Karlsson WK, Harboe ZB, Roed C, Monrad JB, Lindelof M, Larsen VA, et al. Early trigeminal nerve involvement in Listeria monocytogenes rhombencephalitis: case series and systematic review. J Neurol 2017;264:1875–1884. doi: 10.1007/s00415-017-8572-2
4. Hue CD, Bullrich MB, Lam Shin Cheung VC, Sharma AK, Syed AS, Pandey SK, et al. Pearls & Oy-sters: Trigeminal Nerve Dysfunction as the Key Diagnostic Clue to Listeria Rhombencephalitis. Neurology 2021;97:e1457-e60. doi: 10.1212/WNL.0000000000012273
5. Mo T, Wu F, Dou X, Wang D, Xia H, Li X. A Retrospective study of rare listeria meningoencephalitis in immunocompetent children in China. Front Neurol 2022;13:827145. doi: 10.3389/fneur.2022.827145
6. Choi MH, Park YJ, Kim M, Seo YH, Kim YA, Choi JY, et al. Increasing incidence of listeriosis and infection-associated clinical outcomes. Ann Lab Med 2018;38:102–109. doi: 10.3343/alm.2018.38.2.102
7. Vázquez-Boland JA, Kuhn M, Berche P, Chakraborty T, Domínguez-Bernal G, Goebel W, et al. Listeria pathogenesis and molecular virulence determinants. Clin Microbiol Rev 2001;14:584–640. doi: 10.1128/CMR.14.3.584-640.2001
8. Arslan F, Meynet E, Sunbul M, Sipahi OR, Kurtaran B, Kaya S, et al. The clinical features, diagnosis, treatment, and prognosis of neuroinvasive listeriosis: a multinational study. Eur J Clin Microbial Infect Dis 2015;34:1213–1221. doi: 10.1007/s10096-015-2346-5
9. Uldry PA, Kuntzer T, Bogousslavsky J, Regli F, Miklossy J, Bille J, et al. Early symptoms and outcome of Listeria monocytogenes rhombencephalitis: 14 adult cases. J Neurol 1993;240:235–242. doi: 10.1007/BF00818711
10. Redd WD, Anderson C, Barlowe TS, Aleman S, Dellon ES. Prevalence of esophageal foreign body and food impaction in the United States. Gastro Hep Adv 2026;5:100805. doi: 10.1016/j.gastha.2025.100805