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PROGRESSIVE DYSPHAGIA REVEALING GLIOMA: DIAGNOSTIC OVERSHADOWING BY SUSPECTED MYASTHENIA GRAVIS

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ABSTRACT

Progressive dysphagia in older adults presents a diagnostic challenge with a broad differential including neuromuscular, structural and malignant causes. Dysphagia associated with aspiration may lead to significant respiratory compromise requiring intensive care management.

We report the case of a 78-year-old patient with rheumatoid arthritis treated with methotrexate who presented with progressive dysphagia to solids and liquids, weight loss, and cough. She subsequently developed aspiration pneumonia complicated by repeated type 2 respiratory failure and required mechanical ventilation.

Initial investigations focused on aspiration pneumonitis and possible neuromuscular disease, particularly myasthenia gravis, given the presence of bulbar symptoms and an insensate epiglottis on nasendoscopy. However, subsequent magnetic resonance imaging of the brain demonstrated a glioma explaining the progressive bulbar dysfunction.

This case demonstrates diagnostic overshadowing, as recurrent type 2 respiratory failure was repeatedly managed as aspiration pneumonia, delaying the diagnosis of glioma. Early neuroimaging should be considered in patients with progressive dysphagia when initial investigations fail to explain symptoms.

KEYWORDS

Dysphagia, glioma, myasthenia gravis, diagnostic overshadowing, aspiration pneumonia

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LEARNING POINTS

- Progressive dysphagia in an older adult warrants a broad differential - neuromuscular, structural and intracranial causes - not just aspiration-related respiratory disease.
- A bulbar syndrome (insensate epiglottis, dysphagia) can be produced by an intracranial lesion as well as by myasthenia gravis; negative acetylcholine receptor antibodies do not fully exclude either possibility and should not halt the diagnostic search.
- Recurrent type 2 respiratory failure that responds poorly to treatment for presumed aspiration pneumonia should prompt reconsideration of the working diagnosis, rather than repeated management of the same presumed cause - a pattern of diagnostic overshadowing and anchoring bias.
- Early neuroimaging should be considered when dysphagia and respiratory failure evolve atypically or fail to resolve despite appropriate treatment, as magnetic resonance imaging was ultimately pivotal in revealing the underlying glioma in this case.

INTRODUCTION

Dysphagia is a common but potentially serious symptom in elderly patients and may arise from structural, neuromuscular or malignant causes. These in turn are associated with a myriad of complications including aspiration pneumonia, malnutrition and respiratory failure.

Evaluation typically includes clinical assessment, swallow evaluation and targeted imaging. Neuromuscular conditions such as myasthenia gravis (MG) are important considerations in patients presenting with bulbar symptoms including dysphagia, dysarthria and aspiration. However, intracranial structural lesions can occasionally mimic neuromuscular disease. Neuromuscular disorders, such as MG, characteristically produce fatigable weakness that worsens with sustained activity and improves following rest, resulting in a diurnal pattern of symptom fluctuation. During the evaluation of progressive dysphagia, the presence or absence of this diurnal variation is an important clinical discriminator. Its absence should prompt consideration of structural causes, including intracranial neoplasms, in which progressive tissue destruction or replacement produces persistent neurological deficits such as dysphagia which do not exhibit the recovery characteristic of disorders affecting neuromuscular transmission such as MG.

Gliomas are primary brain tumours arising from the supportive glial cells of the central nervous system and represent the most common primary intracranial malignancy. Clinical presentation varies, but commonly includes headache, seizures, focal neurological deficits. Presentation with isolated bulbar dysfunction is rare. This case highlights an unusual presentation of a glioma with predominantly bulbar symptoms and illustrates how diagnostic overshadowing can delay recognition of serious intracranial pathology in patients presenting with progressive dysphagia.

CASE DESCRIPTION

A 78-year-old woman presented to the emergency department with a three-month history of progressive dysphagia to both solids and liquids. This was associated with dysphonia, persistent cough, reduced oral intake and consequent unintentional weight loss. The patient denied fever, chest pain, limb weakness, diplopia or other focal neurological symptoms.

Her past medical history included rheumatoid arthritis treated with methotrexate, bilateral sensorineural hearing loss and seasonal allergic rhinitis. She had previously undergone tonsillectomy in childhood.

One week prior to this admission she had been discharged after treatment for a presumed lower respiratory tract infection, although her cough had not resolved at the time of readmission.

On presentation she was hypoxic on room air requiring supplemental oxygen. Respiratory examination revealed bibasal crackles, more prominent on the right. Oropharyngeal examination was unremarkable with no stridor or cervical lymphadenopathy.

Initial laboratory investigations demonstrated hyponatraemia (sodium 127 mmol/l; baseline 135 mmol/l), leucocytosis (24 $\times 10^9$/l), mildly elevated C-reactive protein (25 mg/l), normal renal function (creatinine 49 µmol/l), and normal liver function tests. Although these findings supported a possible infective process, they were non-specific and did not fully explain the patient's progressive dysphagia and subsequent respiratory deterioration.

Chest radiography demonstrated right basal consolidation, initially interpreted as aspiration pneumonia in the context of oropharyngeal dysphagia. The patient failed a bedside swallow assessment and was kept nil by mouth, with nasogastric tube feeding initiated for nutritional support. Intravenous co-amoxiclav was commenced for presumed aspiration pneumonia. Flexible nasendoscopy performed by the otolaryngology team demonstrated pooling of saliva and an insensate epiglottis with preserved vocal cord mobility, confirming severe bulbar dysfunction but not identifying its underlying cause.

As dysphagia was accompanied by progressive respiratory failure, neuromuscular aetiology was considered. Acetylcholine receptor antibodies were negative, making

myasthenia gravis less likely. Despite antimicrobial therapy, the patient experienced recurrent episodes of hypercapnic (type 2) respiratory failure requiring intubation and mechanical ventilation in the intensive care unit. Computed tomography (CT) pulmonary angiography excluded pulmonary embolism but demonstrated bilateral basal atelectatic change and inflammatory nodules, findings that were insufficient to account for the severity or recurrence of the respiratory failure.

Neuroimaging proved pivotal in establishing the diagnosis. A CT head performed one month before admission following a fall demonstrated only age-related changes and a repeat CT head during the current admission was initially reported as normal, illustrating the limited sensitivity of CT scan for infiltrative brainstem pathology. Ongoing neurological deterioration prompted magnetic resonance imaging (MRI) of the brain, which demonstrated diffuse infiltrative signal abnormality centred within the brainstem with neuroradiological features highly suggestive of a high-grade diffuse glioma *(Fig. 1 and 2)*. These findings provided a unifying explanation for the patient's progressive bulbar dysfunction and recurrent ventilatory failure. Histopathological confirmation was not obtained because of the lesion's eloquent brainstem location and the associated procedural risks; consequently, the diagnosis was based on the characteristic clinical presentation and neuroradiological findings, which represents a limitation of this case.

The diagnostic process was challenging because the patient's initial presentation with progressive dysphagia and recurrent episodes of hypoxia and hypercapnic (type 2) respiratory failure strongly suggested a neuromuscular or aspiration-related disorder rather than a central nervous system lesion. Repeated episodes of respiratory failure were attributed to presumed aspiration pneumonia secondary to oropharyngeal dysphagia, despite the absence of convincing radiological or microbiological evidence of pneumonia, resulting in a degree of diagnostic overshadowing.

MG was also considered given the bulbar symptoms and respiratory compromise; however, acetylcholine receptor antibodies were negative, and neurophysiological testing did not demonstrate findings sufficient to support the diagnosis. Motor neurone disease remained a differential because of progressive bulbar dysfunction but was not supported by the clinical pattern or subsequent neuroimaging. Structural oesophageal pathology was investigated with upper gastrointestinal endoscopy, which was inconclusive and failed to identify a mechanical cause for dysphagia.

MRI of the brain instead demonstrated diffuse infiltrative brainstem abnormalities characteristic of a primary glial neoplasm. Other intracranial pathologies, including metastatic disease, primary central nervous system lymphoma, cerebral abscess, and methotrexate-associated lymphoproliferative disorder (MTX-LPD), were considered. However, the absence of a systemic primary malignancy or infective source, together with the pattern of diffuse brainstem infiltration, favoured a diffuse glioma over these alternative diagnoses. Although MTX-LPD can demonstrate contrast enhancement and diffusion restriction, its radiological appearance is typically more focal, and histopathological confirmation would have been required to definitively exclude this diagnosis.

Ultimately, the diagnosis of diffuse brainstem glioma best unified the patient's progressive dysphagia, recurrent ventilatory failure, and imaging findings, illustrating how an initially plausible diagnosis of aspiration pneumonia

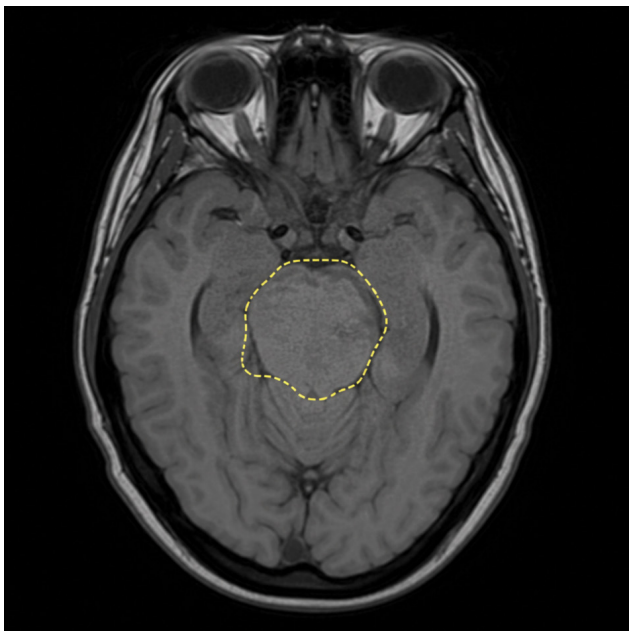


Figure 1. *Axial T1-weighted magnetic resonance image of the brain showing an infiltrative brainstem lesion (dotted line).*

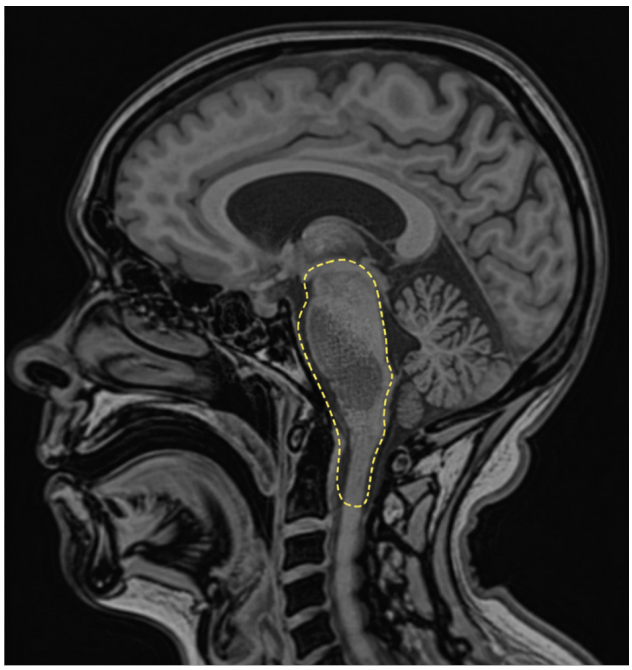


Figure 2. *Sagittal T1-weighted magnetic resonance image demonstrating a diffuse infiltrative brainstem glioma involving the pons and medulla (dotted line).*

delayed recognition of the underlying central neurological pathology. Histological confirmation was not pursued because of the lesion's eloquent brainstem location, the procedural risks of biopsy, and the patient's clinical condition. Consequently, the diagnosis remained a radiological diagnosis of diffuse brainstem glioma, which we acknowledge as a limitation of this case.

The patient was initially treated for aspiration pneumonia with intravenous antibiotics, supplemental oxygen, and supportive respiratory care. She subsequently developed type 2 respiratory failure requiring admission to the intensive care unit, where she underwent invasive mechanical ventilation. Following clinical improvement, she was successfully weaned from ventilator support and extubated.

Given the initial clinical suspicion of MG, empirical treatment with pyridostigmine was commenced. During this period, the patient underwent tracheostomy to facilitate ventilator weaning and respiratory rehabilitation. Although her respiratory status improved, subsequent antibody testing for MG was negative, and the diagnosis was not confirmed

Further neurological assessment, including neuroimaging, identified a glioma as the underlying cause of her neurological presentation. The case was reviewed by the multidisciplinary neuro-oncology team, who concluded that the tumour was not amenable to surgical resection or radiotherapy. Consequently, management was palliative, with 4 mg dexamethasone prescribed for symptomatic control alongside ongoing supportive care and rehabilitation. The patient's neurological condition subsequently declined, and she died within two weeks of the diagnosis, in keeping with the poor prognosis of a high-grade diffuse brainstem glioma.

DISCUSSION

Dysphagia in older adults is common, frequently under-recognised, and carries a broad differential spanning oropharyngeal, oesophageal, neuromuscular and structural intracranial disease[1]. Because presbyphagia and comorbid illness often mask early symptoms, older patients are disproportionately affected by dysphagia-related complications, including malnutrition, aspiration pneumonia and respiratory failure[1,2]. Oropharyngeal dysphagia in particular is a well-established risk factor for aspiration pneumonia and current guidance recommends formal swallow assessment for patients with clinical features of dysphagia or radiologically confirmed community-acquired pneumonia[3]. This framework, while appropriate, can itself become a trap: once a patient is labelled as having "aspiration pneumonia," clinicians may reasonably but incorrectly attribute recurrent respiratory decompensation to an underlying neuromuscular disorder, rather than recognising that the respiratory deterioration may instead reflect the primary disease process responsible for the dysphagia.

Bulbar symptoms in an older adult understandably raise suspicion for MG, a treatable neuromuscular junction disorder that can present with isolated dysphagia, dysarthria and respiratory compromise, sometimes mimicking acute stroke in elderly patients[4]. However, a substantial minority of patients with a genuine clinical syndrome of MG are seronegative for acetylcholine receptor antibodies, and the absence of antibodies does not fully exclude the diagnosis without more specialised testing (clustered AChR, MuSK or LRP4 assays)[5,6]. This diagnostic ambiguity can prolong the search for MG-specific confirmation while a different, and in this case more sinister, structural pathology continues to progress. Critically, unlike MG, the neurological deficits produced by an intracranial mass lesion are typically fixed or progressive rather than fatigable. Although the absence of diurnal variation may prompt reconsideration of a structural cause, as illustrated by this case, this distinction can be difficult to assess in critically ill, intubated patients, in whom reduced consciousness, sedation, mechanical ventilation and the inability to perform serial neurological examinations may obscure the characteristic fluctuation seen in MG. Brainstem gliomas are rare in adults, accounting for well under 2% of adult gliomas, and their clinical presentation is notoriously heterogeneous[7,8]. Most patients presented with a combination of several features rather than a single symptom, meaning bulbar dysfunction typically appeared alongside (not instead of) cranial nerve palsies, ataxia, or raised intracranial pressure. Isolated or predominant bulbar dysfunction as the presenting feature, rather than this more typical combination, is therefore distinctly unusual. A large retrospective series of histologically confirmed adult brainstem gliomas found dysarthria and dysphagia as presenting features in only around 14% of cases, underlining how atypical our patient's presentation was[7]. The same series highlighted a further diagnostic difficulty relevant to our case: even when neuroradiological appearances are highly suggestive of glioma, histopathological confirmation is obtained in only a minority of published adult brainstem series, largely because biopsy of an eloquent brainstem lesion carries substantial risk; a purely radiological diagnosis, as in our patient, therefore remains a recognised and accepted limitation in this tumour location[7,9,10].

Our patient's treatment with long-term methotrexate for rheumatoid arthritis also warranted consideration of methotrexate-associated lymphoproliferative disorder (MTX-LPD), a rare but recognised complication of immunosuppression that can rarely involve the central nervous system and present with new neurological deficits[11,12]. CNS MTX-LPD typically produces multiple enhancing lesions with a more focal, often supratentorial distribution, in contrast to the diffuse infiltrative brainstem signal change seen on our patient's MRI, but the two entities can overlap radiologically, and definitive distinction would have required tissue diagnosis[11,12].

This case illustrates diagnostic overshadowing: the tendency for an existing or initially plausible diagnosis to dominate clinical reasoning and delay consideration of alternative explanations for evolving symptoms[13]. Although the term originated in the context of missed physical illness in patients with pre-existing mental illness or intellectual disability, it is now used more broadly to describe any situation in which a working diagnosis anchors subsequent interpretation of new or worsening symptoms[13,14]. In our patient, the initial and recurrent attribution of hypercapnic respiratory failure to aspiration pneumonia - a plausible and common diagnosis in an elderly patient with confirmed oropharyngeal dysphagia - delayed recognition that a single unifying intracranial process was responsible for both the bulbar dysfunction and the ventilatory failure. This pattern reflects the anchoring and premature closure biases that are increasingly recognised as drivers of diagnostic error across medicine, not only in psychiatric populations[13,14].

This case supports a low threshold for early neuroimaging in older patients with progressive, unexplained dysphagia, particularly when bulbar examination findings (such as an insensate epiglottis) are not readily explained by the initial working diagnosis, when relevant serology is negative, or when the clinical course does not follow the expected trajectory of the presumed diagnosis. Earlier MRI in this case may not have altered the ultimate outcome, given the poor prognosis and inoperable location of high-grade brainstem glioma, but it could plausibly have shortened a prolonged and burdensome diagnostic pathway involving repeated intensive care admissions.

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