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Online Only Clinical Record

Stridor: an unusual presentation of lateral medullary syndrome

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Abstract

We report an unusual case of lateral medullary syndrome which presented with symptoms of acute upper airway obstruction mimicking angioneurotic oedema. Although dysphonia and dysphagia are common symptoms of lateral medullary syndrome, we have found no other reports of this condition presenting as stridor. This case highlights the importance of maintaining a high index of suspicion for central causes of common otolaryngological symptoms in the absence of local signs.

Key words: Lateral Medullary Syndrome; Voice Disorders; Vocal Cord Paralysis

Case report

A 53-year-old man presented to the emergency department with a history of sudden onset of a choking sensation in his throat lasting a few minutes, together with difficulty in breathing and complete loss of voice. He had felt extremely unwell, with dizziness and profuse sweating. His voice had returned, but it remained very hoarse and his throat felt tight. He reported transient visual blurring and a frontal headache and felt unsteady on his feet, but denied any limb weakness or numbness. His wife had noticed that his neck appeared swollen. There was no history of allergen exposure, medication use, insect bite or alcohol consumption. His past medical history included ischaemic heart disease, hypertension and a coronary artery bypass operation one year previously.

Given the dramatic history, a presumptive diagnosis of angioneurotic oedema was made. He was immediately administered intravenous chlorphenamine 10 mg and hydrocortisone 200 mg and was referred for airway management and further evaluation.

Fibre-optic laryngoscopy demonstrated a left vocal fold paralysis, but there was no erythema or pooling of saliva and no mass was present. Computerised tomography of the head, neck, chest and abdomen was performed to rule out malignancy, but the only abnormality identified was a deformed and calcified left vocal fold. The patient was alert and oriented throughout his stay and his vital signs were stable, with no further airway problems, but the persistence of impaired balance prompted a neurological referral.

A formal neurological examination revealed left-sided facial anhidrosis and ptosis, suggestive of left Horner's syndrome. The patient also had diminished pinprick and temperature sensation on the right side. The possibility of a posterior circulation stroke in the distribution of the posterior inferior cerebellar artery was raised.

A magnetic resonance imaging (MRI) scan (Figure 1) was performed, along with magnetic resonance angiography (MRA) of the brain (Figure 2). These investigations confirmed infarction in the lateral medulla, with occlusion of the left vertebral artery.

The patient underwent neuro-rehabilitation and was successfully discharged home a week later.

Discussion

Lateral medullary syndrome, also known by its eponym Wallenberg's syndrome, is a constellation of signs caused by infarction of a wedge of lateral medulla lying posterior to the inferior olivary nucleus. It usually presents with the acute onset of nausea, vomiting, vertigo, ataxia, dysphagia and hoarseness of the voice, with hemisensory changes (Table I).[1] Atherosclerotic occlusion of the vertebral artery is the most common cause, although occlusion of the posterior inferior cerebellar artery and, more rarely,

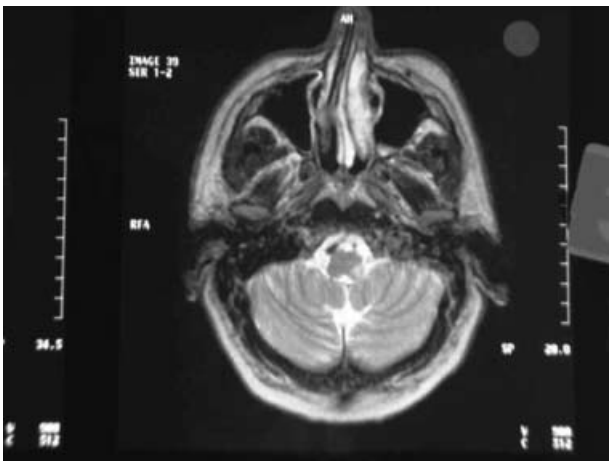

Fig. 1

Magnetic resonance imaging scan showing a wedge-shaped infarct in the left medullary region.

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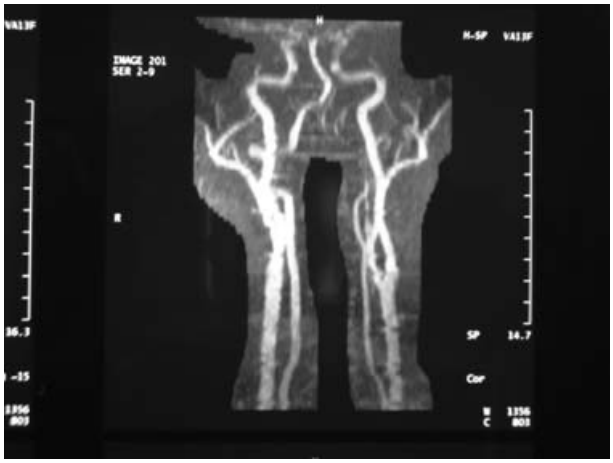

FIG. 2
Magnetic resonance angiogram showing an occlusive thrombus in the left vertebral artery.

TABLE I
SYMPTOMS OF LATERAL MEDULLARY SYNDROME

Clinical features	Anatomical source
Ipsilateral	
Pain, numbness, impaired sensation over half the face	Descending tract & nucleus of Vth nerve
Ataxia of limbs, falling to side of lesion	Uncertain
Vertigo, nausea, vomiting	Vestibular nuclei & connections
Nystagmus, diplopia, oscillopsia	Vestibular nuclei & connections
Horner's syndrome (miosis, ptosis, anhidrosis)	Descending sympathetic tract
Loss of taste (rare)	Nucleus & tractus solitarius
Numbness of ipsilateral arm, tingling in leg	Cuneate & gracile nuclei
Contralateral	
Decreased pain & thermal sense over half the body, sometimes face	Spinothalamic tract

Adapted with permission.[1]

dissection of the vertebral artery have also been described as causes.[1,2]

Vocal fold paralysis, as a direct result of acute ischaemic stroke, is most commonly associated with brainstem stroke and lateral medullary syndrome.[3] Otolaryngological features include ipsilateral vocal fold paralysis and palatal and pharyngeal paresis, leading to varying degrees of dysphonia, dysphagia and nasal regurgitation. Lateral medullary syndrome has also been reported as a cause of obstructive sleep apnoea, and airway obstruction has occurred following cortical stroke.[4,5] A diligent literature search of the Medline® database using PubMed® and Dialog Datastar® interfaces, and the Cochrane Library did not produce any reports of this syndrome causing a compromised airway.

Unilateral vocal fold paralysis is the sine qua non of lateral medullary syndrome, and it usually presents as slowly worsening dysphonia rather than as stridor. In a prospective study of acute ischaemic stroke patients, 100 per cent of patients with lateral medullary infarction had unilateral vocal fold paralysis, while dysphonia was seen in 80 per cent of cases and was considered a reliable diagnostic sign.[6] In the largest reported series of lateral medullary syndrome documented by MRI and MRA, dysphonia was reported in 63 per cent of patients and was commonly associated with dysphagia.[7] Often, the functioning vocal fold may even cross over the midline, such that phonation is preserved. In such circumstances, stridor is more likely to be due to bilateral vocal fold paralysis.

In our patient, the delay in diagnosis was attributed to the unusually subtle neurological signs and the overwhelming evidence in favour of laryngeal pathology. In stroke cases, early diagnosis and multi-specialty involvement can result in significant changes in management and consequently in prognosis.[8]

This case illustrates the need for otolaryngologists to be aware of the more unusual causes of vocal fold palsy and dyspnoea. A prompt and thorough search for central causes must be made in the absence of local findings on fibre-optic laryngoscopy.

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