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# Dural metastasis at medulla oblongata: a rare cause of vocal fold paralysis

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**Abstract**
Malignancy is a major cause of vocal fold paralysis. Nevertheless, metastatic disease at the brainstem leading to high vagal paralysis is rarely encountered. We illustrate an unusual case of unilateral vocal fold paralysis caused by dural metastasis directly compressing on the nucleus ambiguus. The median position of the paralysed vocal fold is inconsistent with the Wagner and Grossman theory predicting the location of the lesion.

**Key words: Vocal fold paralysis; Brainstem; Magnetic resonance imaging**

## Introduction

Vocal fold paralysis is a commonly encountered clinical entity. It represents an underlying pathology situated anywhere from the cerebral cortex to the neuromuscular junction of the larynx. As malignant disease is the principal cause, thorough investigation is mandatory. The correct diagnosis relies on clinical assessment and proper choice of investigations. To date magnetic resonance imaging (MRI) is the best investigation to locate the primary lesion in the central nervous system if a high vagal lesion is suspected. We demonstrate a rare cause of vocal fold palsy confirmed by MRI.

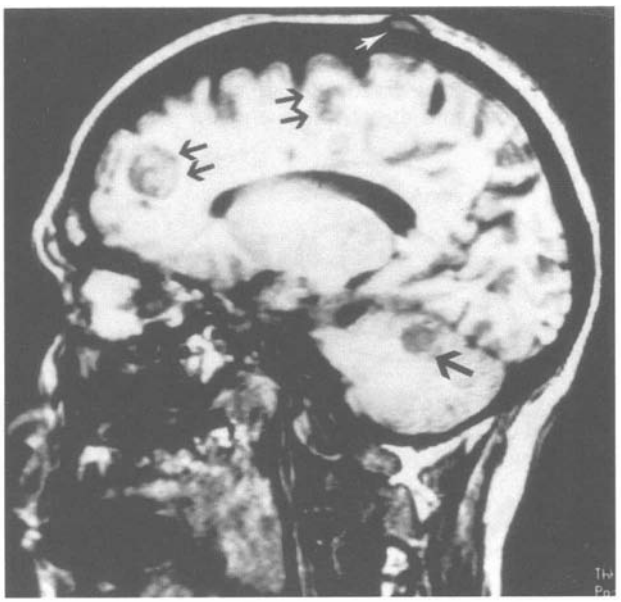

FIG. 1

3D GE T1W sagittal scan of brain.
Multiple spherical nodules shown within the cerebral hemisphere (double arrows), cerebellum (black arrow) and scalp (white arrow). These lesions were heterogeneous in nature with brighter signal on T1- than T2-weighted images, consistent with haemorrhagic metastasis.

## Case report

A 52-year-old woman was referred for investigation of hoarseness. The patient had been diagnosed as having carcinoma of lung three months earlier. Her general condition had deteriorated gradually with symptoms of dizziness, morning headache and dysphagia. Although she

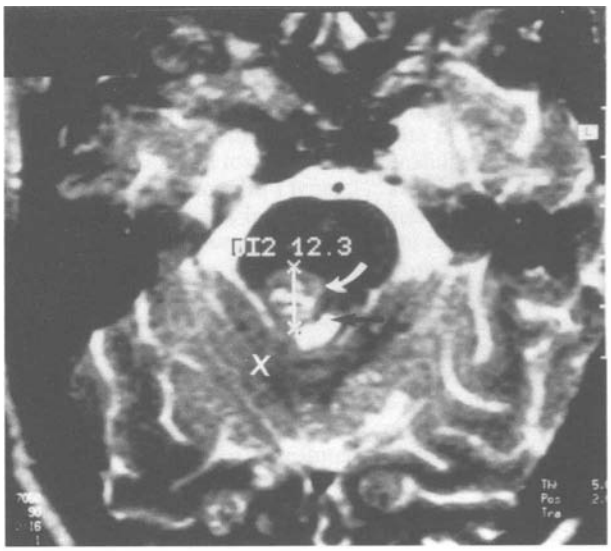


FIG. 2

Fast SE T2W axial scan at the level of the pons.
A metastatic lesion is seen in the posterior part of the pons (white curved arrow) abutting the right cerebellar hemisphere (X) causing mild indentation of the fourth ventricle (black arrow).

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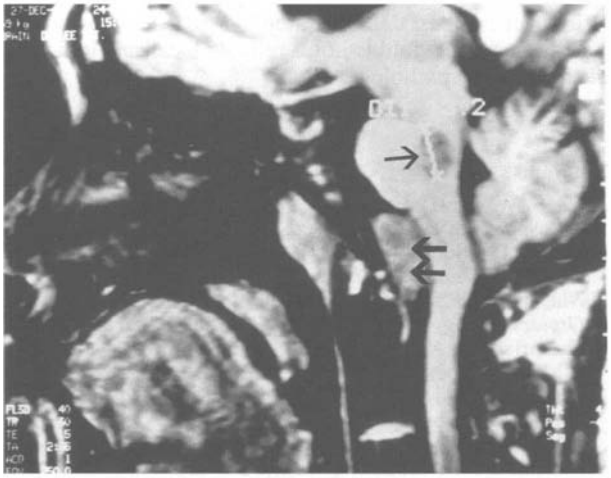

FIG. 3

3D GE T1W sagittal scan of the brainstem showing a metastasis on the dura in front of and compressing the medulla oblongata (double arrows). The metastasis within the pons is also visible (single arrow).

could tolerate a soft diet, she occasionally choked while drinking. One month prior to admission, she developed right-sided weakness. Physical examination demonstrated right hemiplegia, left abducent nerve palsy as well as left-sided palatal paralysis. Indirect laryngoscopy revealed that the left vocal fold was paralysed and situated in the median position. The right vocal fold was mobile and compensating well. In view of adequate airway protection, injection medialization of the vocal fold was not implemented.

MRI of the brain showed multiple small nodular masses in both cerebral hemispheres as well as in the brain stem and cerebellum. A heterogeneous nodule was also noted in the scalp. These nodules appeared hypointense on both T1- and T2-weighted images. They were better visualized on the T1 sequence than T2 sequence (Figures 1 and 2). Since these deposits were small in size (0.5 to 2 cm), no significant oedema could be seen. In view of known pulmonary carcinoma, a diagnosis of multiple intracranial metastases was made.

Especially notable was the dural metastatic lesion situated in front of the brainstem. It was noted to exert pressure on the ventral part of the medulla oblongata where the nucleus ambiguus is located (Figure 3).

During the course of hospitalization, the patient's condition rapidly deteriorated. She died of respiratory failure one week later, before palliative radiotherapy had been commenced.

## Discussion

Although it has been known that voice is generated from the larynx since the time of Hippocrates (460 to 377 B.C.), it was not until the end of the 17th century that the knowledge of anatomy and physiology of the larynx was understood (Weir, 1990). Vocal fold lesions could only be recognized after the introduction of indirect laryngoscopy by Manuel Garcia in 1854 and Gerhardt was credited as being the first clinician to diagnose a vocal fold paralysis (Garcia, 1881; David and Philip, 1991).

Vocal fold paralysis is a sign of disease rather than a diagnosis. Thorough clinical assessment and investigation are required to identify the nature and site of the primary pathology. The lesion may be situated anywhere from the cerebral cortex to the nerve endings in the end organ.

Recurrent laryngeal nerve lesions are the major cause of this condition. The left recurrent laryngeal nerve is affected in 78 per cent of cases mainly due to its longer path. One third of these are due to malignant disease of which 50 per cent arise from lung carcinoma, 20 per cent oesophageal carcinoma and 10 per cent from thyroid neoplasms (Stell and Maran, 1978). High vagal paralysis caused by a brain stem metastasis is remarkably rare when compared to peripheral causes.

In the case reported above, the presence of pharyngeal dysfunction in addition to the unilateral vocal fold palsy was suggestive of a high vagal lesion and this was confirmed by MRI. In view of its multiplanar capability, MR imaging is versatile in its ability to identify intracranial lesions by virtue of their unique signal characteristics. In this case, the MR features were those of haemorrhagic metastases. The persistent presence of intracellular deoxyhaemoglobin secondary to intra-tumoural haemorrhage results in magnetic susceptibility that causes these metastatic lesions to appear more hypointense on T2- rather than T1-weighted images (Atlas, 1991). Furthermore, the sagittal view clearly showed that a dural metastasis was compressing the ventral surface of the medulla oblongata. Dural metastases usually do not transgress the dural boundaries to involve adjacent brain parenchyma unless they are aggressive. Symptomatology from dural metastases, therefore, is usually due to the direct compressive effects on the underlying brain parenchyma (Atlas, 1991). The ventral location of nucleus ambiguus within the medulla oblongata renders it more susceptible to dural compression at this site. Any lesion affecting this nucleus is likely to cause laryngeal and pharyngeal dysfunction including vocal fold paralysis, palatal paralysis, impaired gag reflex as well as dysphagia (FitzGerald, 1985). The presentation is usually unilateral in the early stage but may later become bilateral. Although we were unable to assess

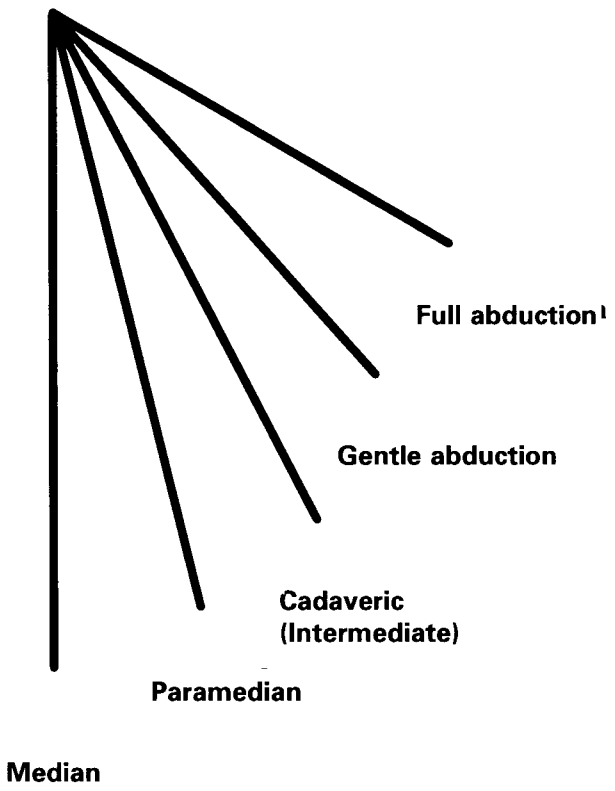


FIG. 4

Different positions assumed by paralysed vocal fold.

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the degree of damage to the nucleus ambiguus, considering the consistency of clinical and radiological features, it is reasonable to assume that the vagal dysfunction was caused by the pressure effect from the dural metastasis.

Extracerebral intracranial metastases are not uncommon, with leptomeningeal and calvarial metastases being more frequent than dural or pachymeningeal metastases. Dural metastases in adults are invariably secondary to metastatic tumour in the adjacent skull, and are usually associated with primary carcinoma of the breast, lung, prostate or kidney (Posner and Chernik, 1978; Sze *et al.*, 1989; Davis *et al.*, 1991). To our knowledge, although metastasis to the brain and dura have been well demonstrated by contrast-enhanced MRI, no previous report has correlated the clinical finding of ipsilateral vocal fold palsy with radiologically proven dural metastasis.

The position of the paralysed vocal fold has been studied in an attempt to predict the location of the causative lesion. Five positions from full abduction to complete adduction have been described in unilateral vocal fold paralysis (Clerf and Suehs, 1941) (Figure 4). There have been several theories put forward to account for this.

Semon's Law states that during the course of a gradually advancing organic lesion affecting the recurrent nerve, three separate stages can be observed. In the first stage, only the abductor fibres are damaged. In the second stage, the vocal fold is immobilized in the median position. In the third stage, the adductors become paralysed and the fold resumes the cadaveric (intermediate) position (Semon, 1881). However, this theory has not been supported by clinical and experimental results and has declined in popularity (Wyke and Kirschner, 1976).

Wagner and Grossman have proposed the most popular and widely accepted theory which suggests that if only the recurrent laryngeal nerve is affected, the vocal fold will remain in a paramedian position because of the unopposed adductive force from the intact cricothyroid muscle. However, if the superior laryngeal nerve is also affected as in a high vagal lesion, the fold will assume the intermediate position because of the loss of this adductive force (Wagner, 1890; Grossman, 1987).

Evidence suggests that the final position of a paralysed vocal fold results from gross atrophy of the intrinsic muscles rather than the unbalanced tension of the individual muscles as suggested by Wagner and Grossman (Quiney and Michaels, 1990). The position of the fold is indeed a function of the duration between the onset and presentation of symptoms. In the case described above, the atypical position of the paralysed fold may be explained by the short duration of the presenting symptoms. This oberservation suggests that the position of the paralysed vocal fold is not a reliable localizing sign of the primary disease. Instead, careful correlation between clinical features and investigation findings is the key to diagnosis.

## Conclusion

The presentation of palatal and pharyngeal dysfunction in addition to vocal fold paralysis indicates a high vagal lesion which is best demonstrated by MRI. The position of a paralysed vocal fold is not a reliable localising indicator of the primary pathology.

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