



A Case of Vocal Cord Paralysis Secondary to Vertebral Artery Dolichoectasia: A Rare Presentation

Vertebral Arter Dolikoektazisine Sekonder Vokal Kord Paralizisi: Nadir Bir Sunum

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Dear Sirs;

Vocal cord paralysis may arise from intra and extralaryngeal pathologies. Neurological pathology is a rare cause of unilateral vocal cord paralysis. The most common neurological diseases inducing vocal cord paralysis are meningomyelocoele, hydrocephalus, the Chiari malformation and bulbar palsy. The vocal cords are innervated by the superior and inferior laryngeal nerves (recurrent laryngeal nerve) that are branches of the vagal nerve. Compression of the vagal nerve for any reason at the level it leaves the medulla oblongata can lead to vocal cord paralysis (1).

The cerebral arteries in the subarachnoid space may apply pressure to and distort the brain parenchyma and stretch the cranial nerves. Most intracranial arterial compressive lesions have been attributed to dolichoectasia, which refers to the dilation, enlargement, and tortuosity of vessels (2). Within the cervicocranial arteries, dilatative arteriopathy preferentially involves the vertebrobasilar system (3). Although the clinical findings of pontine compression due to basilar artery dolichoectasia are known, our knowledge on indentation of the medulla oblongata by the vertebral artery is limited.

A 62-year-old male presented to the head and neck surgery clinic with hoarseness. The patient had experienced hoarseness during speaking that was relieved by rest for a long time before the persistent hoarseness. On endolaryngeal examination, the only finding was the left vocal cord fixed in a paramedian position. The surgeon could not find any intralaryngeal or extralaryngeal etiology and directed the patient to the neurology outpatients. Neurological examination was totally normal except the hoarseness.

Cranial MRI and MR angiography showed expansion, elongation, and torsion of vertebrobasilar arteries. Marked compression of medulla oblongata and pons due to vertebrobasilar dolichoectasia was seen (Figure 1,2). Routine biochemical and haematological studies were normal. CT scans of the thorax and neck were normal. EMG study of the neuromuscular junction was normal. After all other etiologies were excluded, it was concluded that the left vocal paralysis had occurred as a result of vagal nerve compression at the level of the medulla oblongata. The patient rejected decompression surgery.

Medullar compression secondary to vertebral artery dolichoectasia is not a well-known clinical presentation. In the case series reported by Savitz et al., which is the most extensive series of medullar compression secondary to vertebral artery dolichoectasia, vocal cord paralysis was seen in only one of the nine patients (4). Interestingly, our patient had experienced paradoxal hoarseness before it became persistent, and it had improved as in neuromuscular junction disorders. Similar clinical finding were present in Savitz et al's case, but dysphagia was also reported. We believe that existing vertebral artery angiopathy and dolichoectasia became permanent in time, leading to persistent hoarseness. In the literature, there is evidence of an effect of aging on the angiopathy (3). However, chronic compression of the vagal nerve can also have the same result. In a case reported by Tomasello et al., dysphagia and hemiparesis were also present in addition to the vocal cord paralysis (5). The fact that the neurological examination was normal except the vocal cord paralysis provided clinical evidence that it was not an intramedullary pathology. The

