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CASE REPORT

Bilateral vocal cord palsy: An uncommon late complication of cortical ischaemic stroke

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Abstract

Vocal cord palsy in stroke patients is an uncommon manifestation and occurs when an area of the cerebral cortex or brainstem suppling the recurrent laryngeal nerve branch of the vagus is damaged. A stroke which affects the motor cortex does not usually cause paresis of the vocal cords as the vagus nerve nucleus is innervated by the corticobulbar tract from both sides of the brain. We present a unique case of sudden onset of bilateral vocal cord palsy due to left fronto-parieto-insular stroke presenting on the eighth day of ictus.

Background

Middle cerebral artery (MCA) stroke describes the sudden onset of a focal neurological deficit resulting from brain infarction or ischaemia in the territory supplied by the MCA. The MCA is by far the largest cerebral artery and is the vessel most commonly affected by a cerebrovascular accident. The MCA supplies most of the outer convex surface of the brain, nearly all the basal ganglia, and the posterior and anterior internal capsules. Infarcts that occur within the vast distribution of this vessel lead to diverse neurological sequelae.

Primary dysfunction of the larynx following stroke is most frequently the result of an insult to the nucleus ambiguus and the nucleus solitarius; however, some authors have described insults to the cortical and subcortical pathways that result in loss of the laryngeal cough reflex.[1] One of the most common causes of bilateral vocal cord palsy is recurrent laryngeal nerve damage from thyroid surgery or compression by a tumour. Brainstem strokes and cortical strokes are rare causes of bilateral and/or unilateral vocal paralysis as the solitary nucleus of the vagus nerve is innervated from the corticobulbar tracts from both sides of the brain.

Case presentation

We present the case of a 67-year-old male with complaints of sudden transient loss of consciousness followed by weakness of the right upper and lower limb with slurring of speech. The patient had no lifestyle comorbidities. Non-contrast CT and CT perfusion of brain (activated as per stroke protocol) showed early ischaemic changes in the left MCA territory and hypodensity in the left insular cortex was noted. CT angiography of the brain and neck vessels showed that the left MCA was occluded at the proximal M1 segment. The left bulbar internal carotid artery showed luminal thrombus.

Because the patient presented outside the time window for intravenous thrombolysis, he underwent intra-arterial recanalisation via mechanical thrombectomy. The clot was crossed using a Rebar-18 microcatheter and Traxcess micro guidewire, good distal flow was confirmed and two passes were done using a Solitare stent. At the end of the procedure, recanalisation was achieved. No complications occurred during the procedure and post-procedure CT showed no intracerebral haemorrhage. However, post thrombectomy, there was little neurological improvement. He was conscious but globally aphasic and power continued to be 0/5 in right upper limb and 1/5 in lower limb.

Non-contrast CT brain 2 days later showed an acute infarct with slight haemorrhage in the left middle and inferior frontal gyrus and in the subjacent white matter, insula and peri-insula frontotemporal region and ipsilateral basal ganglia; mass effect was evident in the form of effacement of the adjacent cortical sulci and compression of the ipsilateral lateral ventricle. A midline shift of 7 mm was seen, which had increased by 2 mm compared with the previous scan.

With regular physiotherapy, the patient gradually improved neurologically. His global aphasia improved to incomprehensible sounds and power improved to 2/5 in right upper limb and 3/5 in lower limb. He remained fully conscious and alert. Non-

contrast CT brain on day 5 showed a mild interval decrease in the midline shift from 7 mm (2nd day of admission) to 5 mm.

On day 8, the patient developed intermittent stridor with noisy breathing and was again aphasic. His power, however, was preserved and repeat CT brain (along with CT perfusion as part of the stroke protocol) showed no fresh ischaemic changes *(figure 1)*. As the respiratory distress worsened, despite nebulisation and steroids, the patient was started on high flow oxygen therapy. He maintained a saturation of 100%, the PaCO2 was 32, with a respiratory rate of 25-28 breaths per minute but the breathing was noisy with stridor. The ear, nose and throat team was consulted. On flexible fibre-optic laryngoscopy examination, bilateral abductor palsy was detected. The patient was immediately taken up for emergency surgical tracheostomy. By the next day, the patient's breathing had improved. He was transferred to the ward on day 11. Swallowing assessment showed no immediate aspiration so the nasogastric tube was removed on day 13.

CT brain, repeated on day 15, revealed interval reduction in the left MCA infarct and haemorrhagic transformation and patient was discharged home with a tracheostomy tube in situ.
Follow-up at one month after discharge showed persistent vocal cord palsy and decannulation was deferred as he did not pass the swallowing assessment.

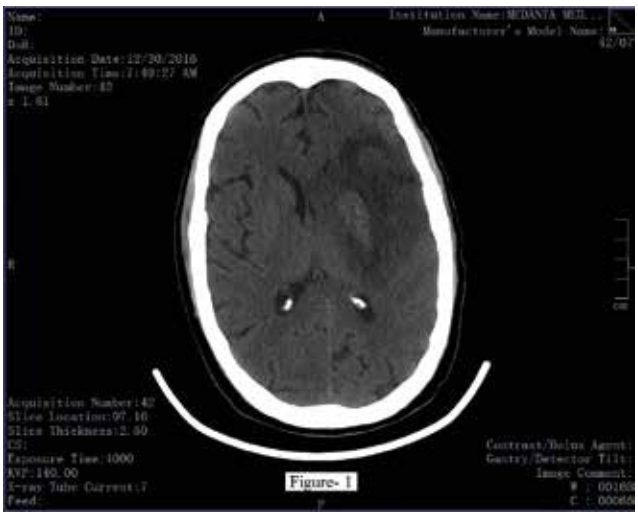

Figure 1. CT brain on day 8

Discussion

Vocal cord palsy means the vocal cord lies in an immobile position and may cause dysphonia. Bilateral vocal cord palsy may lead to failure to adequately protect the airway and in turn there is a risk of regurgitation of gastric contents into the lower airway and lungs leading to aspiration pneumonia; this may become life threatening secondary to upper airway obstruction.[2,3]
The vagus nerve originates in the nucleus ambiguus, situated in the reticular formation in the lateral medulla at the lower end of the brainstem, it passes between the inferior cerebellar peduncle and olive on the lateral aspect of medulla and descends through the jugular foramen to enter the neck. Further down the fibres divide and innervate the cricothyroid muscle as the superior laryngeal nerve and recurrent laryngeal nerve from the ipsilateral vagus.[4] Vocal cord palsy in patients with a cortical stroke is an unexpected finding as the lesion lies above the nucleus ambiguus. Our patient had a left fronto-parieto-insular stroke and developed bilateral vocal cord palsy on the 8th day.
The insular cortex is a portion of the cerebral cortex folded within the lateral sulcus, separating the temporal lobe from the parietal and frontal lobes. Insular stroke is usually associated with an increase in levels of metanephrine and sympathetic hyperactivation due to decreased vagus nerve activity; Walter et al. proposed a connection between the vagus nerve and insular cortex.[5]

Venketasubramanian et al. found vocal cord palsy in 16.6% of the patients who had a stroke in the cortical or large subcortical areas.[6] However, in their study, vocal cord palsy developed within 48 hours of presentation in all patients and none had bilateral vocal cord palsy. They questioned whether the nucleus ambiguus had bilateral cortical innervation, as was seen in other previous studies, meaning that involvement of the nucleus ambiguus in stroke may result in unilateral or bilateral vocal cord palsy. Shaw also believed the hypothesis that bilateral innervation of the nucleus ambiguus may not be seen in all individuals.[3]

Nasrat et al.[7] presented a similar case of bilateral vocal cord paralysis due to right insular stroke but the presentation was on the 5th day and the cause was also unknown. They indicated the possibility of a dominant vocal cord centre in this area to be affected by the stroke with a bilateral paralysis presentation.

Conclusion

To summarise, with this case report we would like to highlight two points. Firstly, in selected patients there can be a dominant unilateral cortical projection to the bilateral nucleus ambiguus. Secondly, sudden bilateral vocal cord palsy, a potentially life-threatening situation, can present even at >1 week of ictus with an improving clinical and radiological condition of the patient. The cause of delayed presentation remains unclear. Since the morbidity and mortality associated with this condition is high, all patients with acute ischaemic stroke with dysphonia should be regularly screened to rule out vocal cord palsy.

Disclosure

Informed written consent was obtained from the patient's son for publication of this case report.
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