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Case Report

Transient Vocal Cord Palsy Caused by Hypoperfusion of Unilateral Hemisphere

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Abstract We report a 68-year-old man who exhibited mild dysarthria and mild right hemiparesis resulted from hypoperfusion of the left hemisphere. An MR angiography showed a severe stenosis at the second portion of left middle cerebral artery (MCA). After the beginning of treatment, the patient suffered from hoarseness, followed by breathing failure. The laryngeal fiber exhibited right vocal cord paresis. Unilateral cortico-bulbar tract dysfunction does not typically cause vocal cord palsy. However, several cases indicate the involvement of a dominant projection from the contralateral cortico-bulbar tract to the vocal cord. In the present case, hypoperfusion of the left hemisphere might have temporarily produced right vocal cord palsy, considering the stenosis of the left MCA.

Key words : Vocal cord palsy, Breathing failure, MCA hypoperfusion, Recurrent laryngeal nerve, Vagal nerve, Corticobulbar tract

Case report

A 68-year-old man, exhibiting left vocal cord palsy of unknown etiology for one year was brought to our hospital by ambulance because of an acute development of right hemiparesis. Neurological examination on admission revealed mild tongue deviation to the left, mild dysarthria, mild right hemiparesis. The hemiparesis predominantly affected the leg, but also the face, and the right leg exhibited hypoesthesia. Tendon reflexes were brisk in all extremities and the Chaddock reflex was elicited on the right. Anemia (red blood cell [RBC] 3450 X 10³, Hb 10.3 g/dl, Ht 31.1 %), elevated blood urea nitrogen (82 mg/dl) and creatinine (5.38 mg/dl), reduced creatinine clearance (22.7 ml/min), hyperglycemia

(200 mg/dl), and hyper triglyceride (200 mg/dl) were observed. A diffusion weighted image (DWI) of the brain magnetic resonance image (MRI) revealed hyperintensity lesions in the left anterior cerebral artery (ACA)-supplying area (Fig. 1a). An MR angiography revealed an occlusion at the second portion of the left ACA and severe stenosis in the second portion of left middle cerebral artery (MCA) (Fig. 1b). The patient had suffered from hypertension, diabetes mellitus type II and chronic renal failure for several years. He was diagnosed with a left-side cerebral infarction due to left ACA occlusion and left MCA hypoperfusion. Intravenous administration of argatroban together with glycerol and edaravone was introduced. On the day after the treatment began, the patient developed hoarseness, followed by breathing failure. The laryngeal fiber exhibited right vocal cord paresis in addition to the pre-existent left vocal cord palsy. He was intubated immediately. Two weeks later, the patient's right vocal cord palsy

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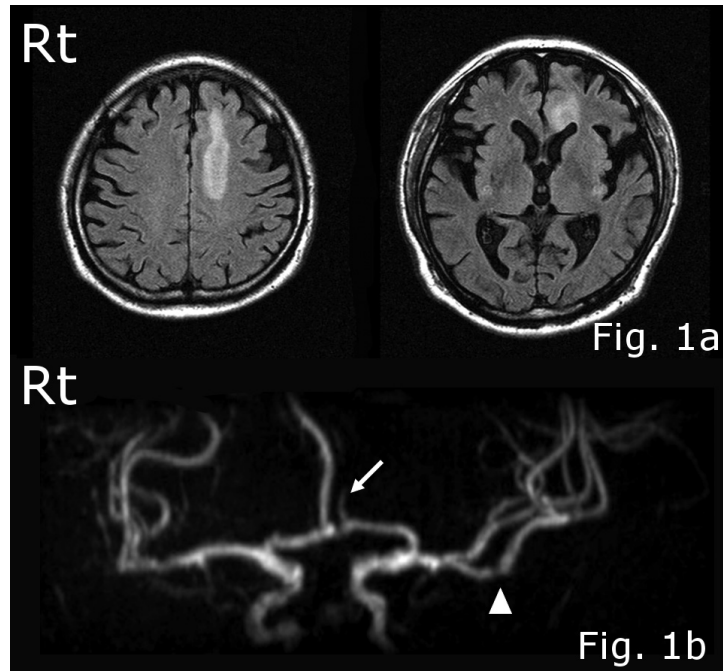


Fig. 1 a) MRI FLAIR image. Hyperintensity lesions are demonstrated in the left anterior cerebral artery-supplying area. b) MR angiography reveals the occlusion at the A2 portion of the left anterior cerebral artery (an arrow) and severe stenosis at the M2 portion of the left middle cerebral artery (an arrowhead).

improved completely and the endotracheal tube was removed, although the left vocal cord palsy was not changed.

Discussion

The recurrent laryngeal nerve is one of the vagal nerve branches originating from the nucleus ambiguus. This nerve controls both the abduction and adduction of the vocal cord¹⁾, and is primarily innervated by the corticobulbar tracts bilaterally^{1)~4)}. Therefore, unilateral corticobulbar tract dysfunction does not typically cause vocal cord palsy. However, several cases have previously presented unilateral vocal cord palsy caused by the infarction of the contralateral corticobulbar tract⁵⁾⁶⁾. These cases indicated a dominant projection from the contralateral corticobulbar tract to the vocal cord. The rate of such dominance was approximately 55%⁵⁾. Although single photon emission computed tomography (SPECT) was not performed in this case, it may be possible that hypoperfusion of the

left hemisphere temporarily produced right hemiparesis involving the right vocal cord, considering the stenosis of his left MCA. In combination with pre-existing left vocal cord palsy, right vocal cord paresis produced breathing failure. The cause of pre-existent left vocal cord palsy was unclear, however, diabetes mellitus type II which this patient had suffered from for several years might be related to it⁷⁾. The present report suggests that hemispheric infarction can produce contralateral vocal cord paresis. As such, the possibility of vocal cord paresis should be considered in cases of breathing failure.

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(和文抄録)

左大脳半球梗塞による声帯麻痺の1例

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左大脳半球の血流低下による, 軽度の構音障害と右半身脱力で発症した68歳男性を報告する. MRアンギオグラフィでは, 左中大脳動脈(MCA)のsecond portionに高度狭窄を認めた. 治療開始後, 嗄声が出現しその後呼吸困難となった. 喉頭鏡による検査では右声帯の完全麻痺を認めた. 通常片側の皮質球路障害では片側の声帯麻痺を生じることはないとされている. しかし, 対側の皮質機能低下により片側の声帯麻痺を生じる症例報告が散見され, 皮質球路は完全な両側支配ではなく左右いずれかの優位側が存在する可能性が示唆されている. 本症例では, 左MCAの高度狭窄による左大脳皮質の機能不全が一時的に存在し, そのため対側の右声帯麻痺を来した可能性があると考えられた.