CASE REPORT

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Internal Carotid Artery Dissection Presenting as Isolated Hypoglossal Nerve Palsy – A Case Report

ABSTRACT

As compared to other cranial nerves, hypoglossal nerve palsy is rare and is often associated with other lower cranial nerve palsies. The most common causes of hypoglossal nerve palsies are tumors followed by trauma, stroke, surgery, infection, and multiple sclerosis. We report a case of a 55-year-old male presenting with dysphagia, dysarthria, and difficult mastication. He was diagnosed to have isolated hypoglossal nerve palsy secondary to compression by internal carotid artery dissection (ICAD). He was treated conservatively with antiplatelets and showed improvement on a 6-month follow-up. ICAD is a dangerous entity with possible endangering complications and hence recognition of it is critical for appropriate treatment and prevention of possible complications. ICAD should be considered in differential diagnosis for isolated hypoglossal nerve palsy.

Key words: Internal carotid artery dissection, Hypoglossal nerve palsy, Cranial nerve palsy, Magnetic resonance angiography, Antiplatelet therapy

INTRODUCTION

Hypoglossal nerve palsy is most seen post head-and-neck surgeries, trauma, infective, and secondary to compressive etiologies like tumors such as chordoma, lymphoma, and glomus.[1] Usually, it is associated with other lower cranial nerve involvement and isolated hypoglossal nerve palsy is very rare. Hypoglossal nerve palsy presents with more signs rather than symptoms, but when it does it can present as dysphagia, dysarthria, difficulty in mastication, and other symptoms associated with other lower cranial nerve involvement.

Spontaneous dissection of carotid or vertebral arteries is one of the common causes of ischemic strokes in the young and middle-aged population.[2] Lower cranial nerve palsies are frequently associated with internal carotid artery dissection (ICAD), particularly the hypoglossal nerve. We present an unusual case of isolated hypoglossal nerve palsy as a presentation of ICAD.

CASE PRESENTATION

A 55-year-old male presented with complaints of dysphagia for 1 week and difficulty in mastication (moving the bolus of food in mouth) for 3–4 days. He also complained of slurring of speech which made few of his pronunciation difficult to understand. He had no history of trauma. Neurological examination revealed tongue deviation to the left on protrusion and dysarthria suggesting left hypoglossal nerve palsy [Figure 1a]. No other neurological findings were noted. Blood pressure on admission was 140/80 mmHg.

No acute infarct was noted on diffusion-weighted magnetic resonance images (MRI) to explain the hypoglossal nerve palsy. Magnetic resonance angiography (MRA) demonstrated left ICAD with a false lumen. T1 fat-saturated sequence images demonstrated a false lumen of ICAD that compressed the outgoing hypoglossal nerve tube with hyperintensity in the hypoglossal canal [Figure 1b and c]. These findings suggested that the false lumen of ICAD was causing compression of the hypoglossal nerve hence causing 12th nerve palsy.

The patient was admitted and was started on tablet aspirin 150 mg/day to prevent thromboembolism from the dissected portion of the artery. On follow-up after 6 weeks, tongue deviation persisted with atrophy on the left hemi-tongue, but the patient had significant improvement in the tongue function and dysarthria had completely resolved with some residual difficulty in mastication.

DISCUSSION

Spontaneous ICAD is a relatively rare disease with the annual incidence of carotid dissection is 2.5–3/100,000.[2,3] The

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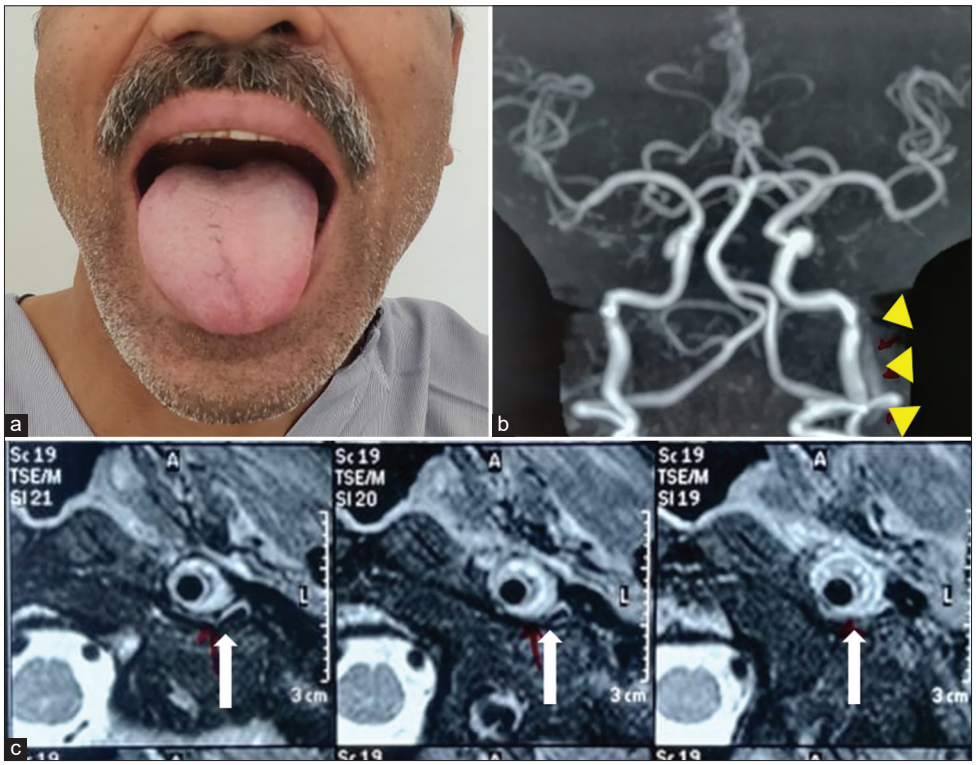

Figure 1: (a) Demonstration of deviation of the tongue toward the left. (b) Magnetic resonance (MR) angiogram: The left distal cervical internal carotid artery (ICA) is dilated and shows an elongated hyperintense signal on MR angiography within the wall of the artery likely hematoma/thrombosed false lumen (yellow arrows). (c) Proton Density Axial (PD TRA sequence) showing dilated ICA and eccentric hyperintense signal likely thrombosed false lumen compressing the hypointense true lumen and resulting in compression of the hypoglossal nerve coursing along its posterior wall (white arrows)

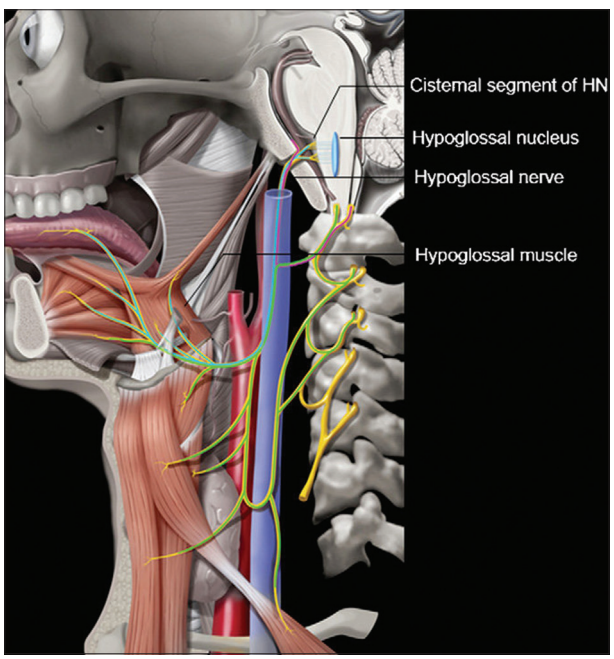

Figure 2: Course and different segments of the hypoglossal nerve

extracranial segments of the carotid as well as vertebral arteries are much more prone to dissections than their intracranial segments due to the free mobility of the extracranial segment. The intimal tear allows blood under arterial pressure to enter the wall of the artery and form an intramural hematoma, the so-called false lumen. Most ICAD occurs spontaneously and may be due to minor subclinical trauma (e.g., sneezing, coughing, vomiting, neck manipulation). Additional etiologies include arterial hypertension, fibromuscular dysplasia, autosomal dominant polycystic kidney disease, internal carotid artery (ICA) redundancy, infectious diseases, or connective tissue disorders.[4]

The main symptoms of ICAD can be divided into (a) local manifestations such as acute-onset unilateral neck pain, unilateral facial and orbital pain, occipital headache, and cranial nerve palsies and (b) ischemic manifestations present as neurological deficits, blurring of vision, or blindness.[5] MRA or computed tomography angiography is the most important investigation for diagnosis of the ICAD. Proton density axial imaging and fat-saturated MRI sequences demonstrate a crescentic T1 high signal within the vessel wall (mural hematoma), with associated luminal narrowing.

The hypoglossal nerve is the motor nerve controlling the intrinsic muscles of the tongue and palate except for palatoglossus. The inferolateral precentral gyrus cortical center for tongue movement sends fibers to the contralateral medullary hypoglossal nucleus. The medullary segment of CNXII travels anterolaterally from the hypoglossal nuclei, exiting the pre-olivary sulcus. The cisternal segment travels in the pre-medullary cistern posterolateral to the vertebral artery making it susceptible to vascular or neoplastic lesions. The extracranial course is divided into the carotid segment and the final sublingual segment [Figure 2]. The carotid segment extends from the hypoglossal canal through the carotid space at the level of the nasopharynx, passing between the internal jugular vein and the ICA. The hypoglossal nerve is vulnerable at this point to compression secondary to ICA pseudoaneurysm or dissection. The sublingual segment is susceptible to floor-of-mouth neoplasms, infections, and inflammation.

Lower cranial neuropathies as a presentation of ICAD is rare with various case reports described in literature. Sturzenneger *et al*. in 1993 analyzed 36 cases with ICAD and multiple cranial nerve palsies and they concluded that 6% of patients present with isolated hypoglossal nerve palsy.[6] Murakami *et al*. have summarized 29 cases of ICAD with lower cranial nerve palsy.[7] The most common nerve to be involved was the hypoglossal nerve (11 of 37 patients – 29.7%), followed by IX, X, and XI nerves.[8] To date, approximately 40 cases of ICAD presenting with lower cranial nerve palsy have been reported in the literature.

The most common treatment for ICAD is anticoagulant or antiplatelet therapy to prevent thromboembolic complications of dissection.[9] In patients with no benefit from antiplatelets, carotid artery stenting is preferred. A review of 201 ICAD patients that underwent CAS showed that the success rate of surgery was 99.1%. The rate of major cardiovascular events in the perioperative period was 4%.[10]

CONCLUSION

While hypoglossal nerve palsy is a rare manifestation of ICAD, it may be the only clinical sign at presentation. Hence, high index of suspicion for ICAD should be kept in mind for prompt to facilitate prompt and appropriate therapy for ICAD, and radiologists must be aware of the imaging findings of hypoglossal nerve palsy.

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