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Case Report

Carotid artery stenting for spontaneous internal carotid artery dissection presenting with hypoglossal nerve palsy: A case report

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ABSTRACT

Background: Some studies reported cases of internal carotid artery (ICA) dissection (ICAD) that was treated by carotid artery stenting (CAS). Symptoms of ICAD resulting from the lower cranial nerve palsy are rare and the treatment strategy is not clearly defined. We report a patient with ICAD showing hypoglossal nerve palsy alone that was treated by CAS.

Case Description: A 47-year-old man presented with headache, dysphagia, dysarthria, and tongue deviation to the left. He had no history of trauma nor any other significant medical history. Axial T2-CUBE MRI and MRA showed dissection of the left ICA accompanied with a false lumen. These findings indicated that direct compression by the false lumen was the cause of hypoglossal nerve palsy. Although medical treatment was continued, symptoms were not improved. Therefore, CAS was performed to thrombose the false lumen and decompress the hypoglossal nerve. His symptoms gradually improved after CAS and angiography performed at month 6 showed well-dilated ICA and disappearance of false lumen.

Conclusion: CAS may be an effective treatment for the lower cranial nerve palsy caused by compression by a false lumen of ICAD.

Keywords: Carotid artery stenting, Hypoglossal nerve palsy, Internal carotid artery dissection, Lower cranial nerve palsy

INTRODUCTION

Carotid artery stenting (CAS) has become the definite treatment for internal carotid artery (ICA) stenosis. Moreover, some studies reported cases of ICA dissection (ICAD) that was treated by CAS. CAS is performed for patients with ICAD whose symptoms are not controlled by antithrombotic drugs or who are at high risk of stroke.[32] Symptoms of ICAD resulting from the lower cranial nerve palsy are rare and the treatment strategy is not clearly defined. Furthermore, CAS for ICAD patients with the lower cranial nerve palsy is rarely performed. In this report, we present a case of ICAD showing hypoglossal nerve palsy alone that was treated by CAS and discuss radiological findings and management of such cases.

CASE PRESENTATION

A 47-year-old man was initially diagnosed as dysphagia. On the next day, he developed dysarthria and tongue deviation to the left, and he visited our hospital. He had no history of trauma nor any other significant medical history. Blood pressure was 184/126 mmHg. Neurological examinations revealed leftward deviation of the tongue on protrusion and dysarthria, suggesting a left hypoglossal nerve palsy. There were no other neurological deficits. Diffusion-weighted magnetic resonance images (MRI) did not demonstrate any lesions that could cause left hypoglossal nerve palsy [Figure 1a]. Axial T2-CUBE MRI demonstrated a false lumen of ICAD that compressed the outlet portion of the hypoglossal nerve tube and dilation of the perineural space in the hypoglossal canal [Figure 1b]. The false lumen of ICAD was adjacent to the hypoglossal canal at its distal side and protruded toward the hypoglossal neural tube. These findings indicated that direct compression by the false lumen was the cause of hypoglossal nerve palsy [Figure 1c]. MR angiography (MRA) showed dissection of the left ICA accompanied with a false lumen [Figure 1d]. The patient was admitted and we started transoral administration of amlodipine (5 mg/day) and azilsartan (20 mg/day) for antihypertensive treatment. On the 2nd day, he felt headache, and transoral administration of loxoprofen was started. On the 7th day, he was started on aspirin (100 mg/day) and clopidogrel (75 mg/day) for prevention of thromboembolism from the dissected portion. Cerebral angiography performed on the 8th day showed an ICAD consistent with the findings on MRI and MRA [Figure 1e]. Cone-beam computed tomography (CBCT) showed protrusion of the false lumen to the hypoglossal canal [Figure 1f].

Although medical treatment was continued, his headache worsened, and the neurological symptoms were not improved. Therefore, CAS was performed on the 19th day to thrombose the false lumen and decompress the hypoglossal nerve. A 6Fr guiding catheter (Axcelguide MSK, Medikit, Tokyo, Japan) was inserted through the right brachial artery and a 4-6Fr catheter (Dymon catheter, Silux, Saitama, Japan) was placed in the left common carotid artery. An embolic protection device (FilterWire EZ, Boston Scientific, MA) was advanced through the lesion and a filter was deployed in the ICA at the petrous portion. A carotid stent (Wallstent, Boston Scientific) was advanced to the dissected portion and deployed. Angiography immediately after stenting showed dilatation of the true lumen and congestion of contrast medium in the false lumen [Figure 2]. There were no neurological symptoms or vital changes throughout the procedures. Pre- or post-dilation was not performed because the vessel was sufficiently dilated and there was a risk of enlargement or rupture of the false lumen.

His headache gradually improved after CAS and disappeared on the 25th day. His postoperative course was uneventful and he was discharged on the 26th day. No improvement of

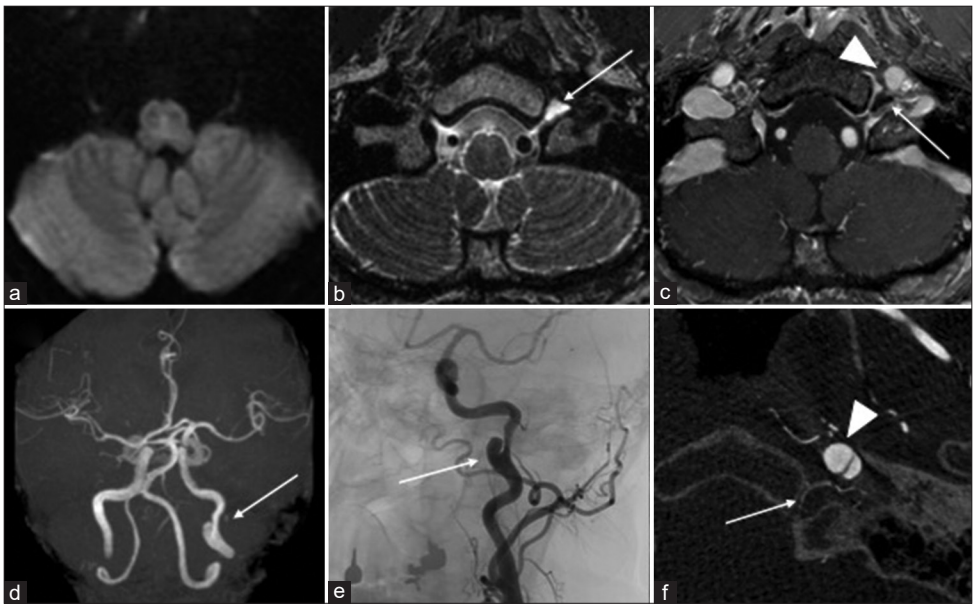

Figure 1: (a) Diffusion-weighted MRI showing no lesion in the medulla oblongata including the hypoglossal nerve nucleus. (b) T2-CUBE MRI showing false lumen of ICAD compressing the hypoglossal nerve tube, and the perineural space in the left hypoglossal canal is dilated (arrow). (c) T1-weighted MRI with contrast-enhancement showing dissected false cavity (arrowhead) compressing the distal side of the hypoglossal canal (arrow). (d) MRA showing a left ICAD with a false lumen (arrow). (e) Angiography showing a left ICAD (arrow). (f) Cone-beam CT showing that the false lumen (arrowhead) is protruding to the direction of the hypoglossal canal (arrow). Direct compression is thought to be causing hypoglossal nerve palsy.

hypoglossal nerve palsy was observed at the time of discharge. At 1 month after discharge, neurological symptoms, such as tongue deviation and dysarthria, improved. Cerebral angiography performed at month 6 showed well-dilated ICA and disappearance of false lumen [Figure 3].

DISCUSSION

Spontaneous ICAD is a relatively rare disease and incidence is reported to be 1.72–2.9/100,000 population.[6,16] In a study of 32 patients with spontaneous ICAD, the main symptoms were headache and neck pain (78%), ischemic symptoms (59%), and Horner's syndrome (25%).[16] On the other hand, symptoms of the lower cranial nerve palsy are rare. In another report, only two of 31 ICAD patients (6%) were reported to show lower cranial nerve palsy.[29] Sturzenegger and Huber[29] summarized 38 cases of ICAD presenting with cranial nerve palsy. Among them, 27 cases were accompanied with the lower cranial nerve palsy (IX, X, XI, and XII). Murakami *et al*.[25] summarized 29 cases of ICAD with the lower cranial nerve palsy. To the best of our knowledge, a total of 37 cases of ICAD presenting with the lower cranial nerve palsy have been reported until 2018 [Table 1].[1-5,7-15,17,18,20-31,33] Of the 37 cases, 34 were male. The side of dissection was left (n = 23), right (n = 11), or both (n = 3). The mean age was 46.7 years, occurring in a relatively young age group. The most common neuropathy was XII nerve palsy, which was observed in 11 of 37 patients (29.7%). IX, X, XI, and XII nerve palsy was the second most frequent pattern (n = 10, 27.0%). Similar to the previous reports, our patient was a relatively young male, and he presented symptoms due to hypoglossal nerve palsy.

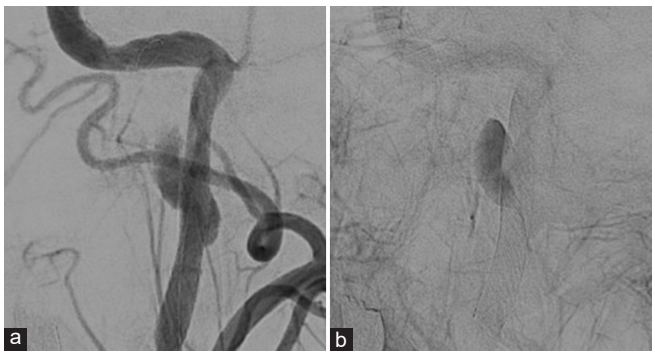

Figure 2: (a) Post-CAS angiography demonstrating dilatation of the true lumen of ICA. (b) Congestion of contrast medium in the false lumen is observed immediately after stenting.

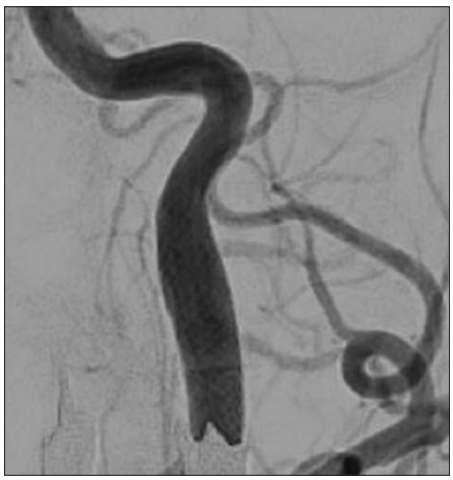

Figure 3: Cerebral angiography performed at month 6 demonstrating sufficient dilatation of ICA and absence of the false lumen.

MRI, MRA, CT angiography (CTA), and cerebral angiography have been used for diagnosis of ICAD. However, in cases of ICAD presenting with the lower cranial nerve palsy, nerve pathways cannot be visualized, and the diagnosis is often based on clinical symptoms. In the present case, angiography showed ICAD with a false lumen, and CBCT was used to visualize the hypoglossal canal and ICAD. CBCT showed ICAD at the level of the hypoglossal canal [Figure 1f]. MRI and CBCT demonstrated a relationship between the hypoglossal canal and the false lumen of ICAD. Furthermore, MRI demonstrated a compressed neural tube and perineural space. The perineural space was demonstrated as having a triangle shape, suggesting compression by the dissected ICA cavity [Figure 1b]. The high intensity region in [Figure 1b] may reflect retention of cerebrospinal fluid in the compressed perineural space.

The most common treatment for ICAD is anticoagulant or antiplatelet treatment and there is no significant difference in the outcome between these two treatments.[19] In some patients, symptoms were not improved with medical treatment, and CAS was additionally performed. A review of 201 ICAD patients that underwent CAS showed that the success rate of surgery was 99.1%.[32] The rate of major cardiovascular events in the perioperative period was 4%. Moreover, intimal hyperplasia, in-stent restenosis, and occlusion occurred in 3.3% of cases, and transient ischemic attack occurred in 2.1% of cases. Among them, cases of ICAD that was accompanied with the lower cranial nerve palsy were treated with antithrombotic drugs. To the best of our knowledge, CAS has only been performed for three cases, including the present case. Of the 20 patients that received conservative treatment, 18 patients (90%) showed improvement. For these 18 patients, the mean observation period from onset to improvement was 61.1 days. On the other hand, two patients who underwent CAS showed improvement in neurological symptoms within 1 month without complications. In the present case, both headache and neurological symptoms due to hypoglossal nerve palsy improved within 30 days. In the false lumen, little thrombus was found and it was filled with circulated blood, which is similar to a pseudoaneurysm. Thrombosis of the false lumen induced by CAS may immediately decrease the direct

Table 1: Patients with spontaneous internal carotid artery dissection presented with the lower cranial nerve palsy.

Case	Age/Sex	Side	Palsy	Pain	ischemia	HS	Treatment	Outcome	Observation Period
Havelius *et al.*[11]	44/M	Right	IX X XI XII	No	No	No	N/A	Improve	Mostly improved within 1 y partial X palsy persistent
Goodman *et al.*[8]	49/M	Right	XII	Temporal	Yes	Yes	Anticoagulant	Improve	Recovery within 6 m
Mokri *et al.*[22]	47/M	Bilateral	IX X XI XII	Occipital	No	Yes	N/A	Improve	Recovery within 8 m
Davies[5]	63/F	Left	X	No	No	No	Anticoagulant	Improve	X palsy mostly improved within 6 m
Panisset *et al.*[28]	36/M	Right	IX X XI XII	Occipital, eye	No	No	None	Improve	Recovery within 2 m
Panisset *et al.*[28]	53/M	Left	V VII IX X XII	Occipital, neck	No	No	None	Improve	Recovery within 6 m
Mokri *et al.*[23]	41/M	Left	IX X XI XII	No	No	No	Antiplatelet	Improve	Recovery within 1 m
Bradac[2]	28/M	Right	XII	Neck right	No	Yes	Antiplatelet	Stable	XII palsy persistent after 4 m
Bradac[2]	41/F	Left	XII	Head	Yes	Yes	N/A	Stable	XII palsy persistent after 6 m
Bradac[2]	53/M	Left	XII	Head, neck	No	No	Antiplatelet	Stable	XII palsy persistent after 1 y
Hommel[13]	55/M	Left	XII	Eye, temple left	No	No	N/A	Improve	Recovery within 2 m
Hommel[13]	54/M	Right	X XII	Temporal, occipital	No	No	N/A	Improve	Recovery of XII palsy within 3 d recovery of X palsy within 6 m
Hommel[13]	44/M	Left	IX X XII	Jaw, ear face	Yes	No	N/A	Stable	XII palsy persistent
Goldberg[7]	49/M	Left	IX X XI XII	Face	No	Yes	N/A	Improve	Recovery within 7 w
Waespe[31]	41/M	Bilateral	IX X XII	Neck, face	No	No	EC-IC bypass	Improve	Recovery within 2 w
Lieschke[17]	42/M	Right	X XII	Eye	No	No	None	Improve	Recovery within 5 m
Pozzo[4]	58/M	Left	IX X XI XII	Neck	No	No	N/A	Stable	Palsy persistent after 13 m
Sturzenegger and Huber[29]	42/M	Right	IX X	jaw, ear, face	Yes	No	Anticoagulant	Improve	Recovery within 10 w
Sturzenegger and Huber[29]	45/M	Left	XII	Nuchal	No	No	Anticoagulant	Improve	Recovery within 3 w
Klossek *et al.*[15]	49/M	Left	IX X XI XII	Head	No	Yes	Anticoagulant	Improve	Recovery within 4 m
Nusbaum *et al.*[27]	40/M	Right	X	Temporal	No	No	Anticoagulant	Improve	Recovery within 6 d
Guy *et al.*[9]	60/M	Left	XII	Head	No	Yes	Anticoagulant antiplatelet	Improve	Recovery of XII palsy within 3 w Horner's syndrome persistent
Guy *et al.*[9]	49/M	Left	IX X XII	Head	No	Yes	Anticoagulant antiplatelet	Improve	Recovery within 1 w
Arnoldner *et al.*[1]	52/M	Left	X XII	No	No	No	Anticoagulant	Improve	Recovery within 4 w
Mizutani *et al.*[21]	50/M	Left	IX X XI XII	Neck	Yes	Yes	Anticoagulant antiplatelet	Improve	Recovery within 1 m
Moussouttas *et al.*[24]	40/M	Right	Xi	No	No	No	N/A	N/A	N/A
Vaes[30]	57/M	Left	Xe	No	No	No	Anticoagulant	N/A	N/A
Ishildak[14]	40/M	Bilateral	IX X XII	No	No	No	Anticoagulant	N/A	N/A
Nguyen *et al.*[26]	35/M	Left	X	N/A	No	No	Antiplatelet	Improve	Recovery within 3 w
Zeleňák *et al.*[33]	46/M	Left	IX X XI XII	Jaw, head	No	No	Antiplatelet, stent	Improve	Recovery within 2 w after surgery

(Contd...)

Table 1: *(Continued).*

Case	Age/Sex	Side	Palsy	Pain	ischemia	HS	Treatment	Outcome	Observation Period
Majeed *et al.*[18]	55/M	Right	VIIiX XII	No	No	Yes	Anticoagulant	Improve	Recovery of X and XII palsy within 6 w: partial Horner's syndrome and y within 6 w: parti
Hanyu[10]	40/M	Right	XII	N/A	No	No	N/A	N/A	N/A
Heckmann *et al.*[12]	44/F	Left	IX X XI XII	Temporal, jaw	No	No	Anticoagulant	N/A	N/A
Murakami *et al.*[25]	42/M	Left	X XII	No	No	No	Anticoagulant antiplatelet, stent	Improve	X XII palsy mostly improved within 1 w after surgery
Cruciata *et al.*[3]	56/M	Left	XII	No	No	No	Anticoagulant coiling	Improve	Recovery of XII palsy within 8 w
Mes *et al.*[20]	42/M	Left	XII	No	No	No	Antiplatelet	Improve	Recovery within 15 w
Present case	47/M	Left	XII	Occipital	No	No	Antiplatelet stent	Improve	Recovery within 1 m after surgery

F: Female; HS: Horner's syndrome; M: Male; m: Month; N/A: Not applicable; w: Week; y: Year

compression force of the hypoglossal nerve. As the results of medical treatment were good, medical treatment should be the first choice of the treatment for ICAD with the lower cranial nerve palsy; however, it often takes a long time for the improvement of neuropathy and it is difficult to predict the period of improvement. In patients with severe headaches, early surgical treatment improves the patient's quality of life. In such cases, CAS should be considered as treatment. Furthermore, for patients presenting with the lower cranial nerve palsy, CAS may be a choice of the treatment to decrease compression of the nerve by false cavity thrombosis. Limitation of this report is that the number of cases is small. Therefore, further investigations to evaluate the efficacy of CAS for such cases are mandatory.

CONCLUSION

CAS may be an effective treatment for the lower cranial nerve palsy caused by compression by a false lumen of ICAD. Decompression of the nerve by CAS leads to early improvement of symptoms.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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