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Microvascular decompression for a patient with oculomotor palsy caused by posterior cerebral artery compression: A case report and review of the literature

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Abstract

BACKGROUND

Aneurysm compression, diabetes and traumatic brain injury are well-known causative factors of oculomotor nerve palsy (ONP), while ONP induced by neurovascular conflicts are rare entities. Here, we report a typical case of ONP caused by right posterior cerebral artery (PCA) compression to increase neurosurgeons' awareness of the disease and reduce misdiagnosis and recurrence.

CASE SUMMARY

+ADw-html+AD4APA-p+AD4-A 54-year-old man without known medical history presented with right ONP for the past 5 years. The patient presented to the hospital with right ptosis, diplopia, anisocoria (rt 5 mm, lt 2.5 mm), loss of duction in all directions but abduction and impaired pupillary light reflexes. Magnetic resonance angiography (MRA), computed tomography venography (CTV) examinations showed no phlebangioma, aneurysm or intracranial lesion. The diabetes and myasthenia gravis were excluded through oral glucose tolerance test and prostigmine test. The cranial nerve Magnetic resonance imaging (MRI) showed that the right posterior cerebral artery (PCA) loop was in direct contact with the cisternal segment of the right oculomotor nerve (ON). Microvascular decompression (MVD) of the culprit vessel from

the ON through a right subtemporal craniotomy was carried out, and the symptoms of ONP was significantly relieved at 3 mo follow-up.+ADw-/p+AD4APA-/html+AD4-

CONCLUSION

Vascular compression of the ON is a rare pathogeny of ONP that may be refractory to drug therapy and ophthalmic strabismus surgery. MVD is an effective treatment for ONP induced by neurovascular compression.

INTRODUCTION

Oculomotor nerve palsy (ONP) are well-known caused by aneurysm, intracranial space-occupying lesion and diabetes mellitus. Few studies were available on ONP induced by neurovascular conflicts. In 1988, Kojo *et al* described the first case of ONP due to vascular compression of the ON, which was successfully relieved by microvascular decompression (MVD)[1]. Subsequently, only anecdotal cases have been documented in the literatures. Early diagnosis of such atypical neurovascular compression became quite simple since the development and extensive application of cranial nerve magnetic resonance imaging (MRI) contributes to classifying nervous system diseases[2, 3]. Here, we report a case of ONP caused by neurovascular conflict and conducted a systematic review of the literature on ONP caused by neurovascular compression is performed.

CASE PRESENTATION

Chief complaints

A 54-year-old man presented with worsening symptoms of right ptosis, diplopia and ophthalmoplegia for 5 years.

History of present illness

Patient's symptoms started 5 years ago with progressive ptosis, diplopia and ophthalmoplegia of the right eye. The patient had once undergone neostigmine intravenous injection and ophthalmic strabismus surgery in an outside hospital.

History of past illness

The patient was diagnosed with hyperlipidaemia 3 years ago and received no treatment.

Personal and family history

The patient had a free personal and family history.

Physical examination

The patient's width of right palpebral fissure was significantly larger than that of contralateral eye, and his right ocular motility weakened in primary position, adduction, medial-to-upper left, elevation, medial-to-upper right (Figure 1A-E) except abduction (Figure 1F). Additionally, the right pupil was 5 mm and showed a sluggish response to direct and consensual pupillary light reflexes, while the left pupil was 2.5 mm and reacted sensitively.

Laboratory examinations

The neuro-ophthalmologic evaluation, funduscopy and blood glucose level were negative.

Imaging examinations

There were no aneurysm and phlebangioma to be detected through Magnetic resonance angiography (MRA) and computed tomography venography (CTV), but a cranial nerve MRI (Figure 2A-B) and a three-dimensional (3D)-model of the cisternal segment (Figure 3A-C) revealed the right posterior cerebral artery (PCA) loop was in direct contact with the cisternal segment of the right ON.

FINAL DIAGNOSIS

The diagnosis of ONP caused by neurovascular conflict was based on laboratory findings and typical imaging findings.

TREATMENT

+ADw-html+AD4APA-p+AD4-MVD surgical to decompress the neurovascular compression was performed. The incision mark was labeled from 1 cm below anterior ear zygomatic arch to the forepart of the parietal tuber, and then we performed a right-sided subtemporal craniotomy for an approach to the cisterna ambiens. When concerning the tentorial notch, the interpeduncular cistern and ventrolateral brainstem were identified and then the overlaying arachnoid was sharply opened (Figure 4A). And we observed that the right PCA compressed on the ON with a distinct indentation (Figure 4B), indicating that the right PCA and its bifurcation were the culprit vessels. The results of intraoperative exploration were consistent with that preoperative cranial nerve MRI.+ACY-nbsp+ADsAPA-/p+AD4APA-/html+AD4-

OUTCOME AND FOLLOW-UP

We observed the patient's symptoms were not altered immediately after MVD. However, the right eyestrain and diplopia were relieved by the time the patient discharged. At 1 year neuro-ophthalmologic follow-up, the patient’s ocular symptoms were significantly relieved at 3 mo after surgery, and the results of postoperative cranial nerve MRI (Figure 2C-D) and 3D-model imaging showed a small cerebrospinal fluid space between the right ON and the ipsilateral PCA (Figure 3D-F). Furthermore, the right pupil diameter and pupillary light reflexes returned to normal again. The right ocular motility to different directions and the symmetry of palpebral fissures in ONP patient recovered too (Figure 1G-I, K), but mild deficits in elevation (Figure 1J).

DISCUSSION

+ADw-html+AD4APA-p+AD4-We described the successful treatment by vascular decompression of the ON to improve ocular movements in a 54-year-old male patient. The patient has a protracted course of the disease and was recalcitrant to conservative treatment and ophthalmic strabismus surgery, ultimately the MVD has acquired an overall satisfactory outcome for him. The definite pathophysiology of ONP caused by vascular compression has not been investigated clearly so far. Some studies suggested that mechanical compression caused by abnormal structures such as aneurysms, intracranial lesions can result in axonal membrane instability and nerve ischemia, which would further induce segmental demyelination and ephaptic neural transmission hyperexcitability+AFs-4+AF0-. Additionally, an acute reaction of pulsations on the ON may clarify the acute presentation of ONP+AFs-5+AF0-, whereas the ischemic effect of pulsations and the atherosclerotic deformation associated with aging may explain the chronic and persistent forms+AFs-6, 7+AF0-.+ACY-nbsp+ADsAPA-br /+AD4-Reviewing the related literatures, ONP caused by non-aneurysmal vascular compression has been defined as persistent palsy or as oculomotor neuromyotonia (ONM) with an intermittent presentation+AFs-7+AF0-. We identified 43 cases+AFs-1, 3, 5, 8-37+AF0- of oculomotor involvement that attributable to isolated neurovascular conflict, while only 12 cases including the current case were treated with MVD (Table 1)+AFs-1, 8-16, 37+AF0-. Among these 12 cases, only 5 cases+AFs-12, 13, 15, 16+AF0- including the current case involved compressive effects by the simple neurovascular conflict without vascular variation or atherosclerotic changes, and the responsible vessels of other 7 cases+AFs-1, 8-11, 14, 37+AF0- were confirmed to have atherosclerotic changes or accompanied by vascular tortuosity or dilation (Table 1). According to statistical analysis, we observed that the culprit vessels were PCA in 17 cases (17/43, 39.53+ACU-), 11 cases (11/43, 25.58+ACU-) were posterior communicating artery (PcomA), 6 cases (6/43, 13.95+ACU-) were basilar artery (BA), 2 cases were (2/43, 4.65+ACU-) superior cerebellar artery (SCA), 1 case (1/43, 2.33+ACU-) was trigeminal artery. This statistical result is consistent with the anatomy of the ON, previous study has shown that neurovascular conflict occurs most often in the cisternal segment of the

ON, and especially the branches arising from the P1 and P2 segments of the PCA has a close intimate relationship with the dorsal surface of the ON+AFs-8, 38+AF0-. Interestingly, Liang +ADw-i+AD4-et al+ADw-/i+AD4- found that SCA is most likely to be in direct contact with the ON, followed by PCA, accounting for 58.9+ACU- and 55.1+ACU- respectively, and only 7 of 392 nerves were found to be compressed by PcomA+AFs-3+AF0-. This may be different from our statistics that PCA and PcomA are the most common culprit vessels to induce ONP, and perhaps the following points account for these differences. Firstly, the PCA (7/216, 3.24+ACU-) might be more easily formed a +ACI-curve+ACI- induced by neurovascular conflict than that of SCA (5/231, 2.16+ACU-)+AFs-3+AF0-. A study suggested that there are about 55+ACU- elderly patients+ACY-rsquo+ADs- PCA was +ACI-curve+ACI-, and the +ACI-curve+ACI-PCA generally develop to tortuosity and dilation, which easily leads to neurovascular conflict+AFs-39+AF0-. Secondly, the ON originates from the midbrain and traverses between the PCA and the SCA, then runs parallelly to the PcomA, it should be noted that ON often contacts with the medial trunk of the PCA when the it travels to the interpeduncular fossa, while the SCA spreads out beneath the oculomotor nerve and has little contact with the nerve+AFs-40+AF0-. Additionally, the ON, PcomA and anterior choroidal artery were firmly surrounded by arachnoid membrane at the interpeduncular cistern segment+AFs-41+AF0-, which makes PcomA more easily generate a pulsation to the nerve. Hence these anatomical features may be able to increase the incidence of vascular compression by the PcomA and PCA, especially when atherosclerotic changes or vascular variations occur. Thirdly, the incidence of PcomA concomitant with infundibular dilatation is as high as 24.6+ACU-(64/260)+AFs-41, 42+AF0-, previous study suggests that the infundibular dilatation of PcomA should be regarded as a normal anatomical condition but not aneurysm because it is generally considered to be a congenital or acquired anatomical variation+AFs-43+AF0-. And we followed this principle in the process of counting the number of blood vessels, which might lead to differences in the number of PcomA between our study and Liang +ADw-i+AD4-et al+ADw-/i+AD4-'s. Other reasons may be related to

the selected cases. Further clinical researches with a larger number of cases are required to illuminate these problems.+ADw-br /+AD4-Through literature review, the diagnosis of ONP caused by neurovascular conflict should be performed with neuro-imaging examinations including cranial nerve MRI, MRA, angiography and CTV. Surgical treatment and conservative medication are reported as options of administration, but there is not an expert consensus or guidelines in the treatment of such cases. And how to correctly make the indications and opportunity for surgery is still controversial. However, the therapy method is relatively uniform in the prevailing views. For intermittent presentation, such as ONM, Stockman +ADw-i+AD4-et al+ADw-/i+AD4- demonstrated that an attempt of conservative medication with carbamazepine should be performed, and the response rate could be more than 87.8+ACUAWw-44+AF0-. For ONP patients with acute onset, Shimizu +ADw-i+AD4-et al+ADw-/i+AD4- reported that administration of steroids could relieve the inflammatory state associated with vascular compression to make the patient's recovery+AFs-35, 45+AF0-. MVD is only considered to apply for some patients with ONP who failed to conservative treatment. Additionally, MVD is also available for ONM, but it should be avoided when risks exceed benefits+AFs-7+AF0-. Until now, just anecdotal evidences recommended that MVD is an effective operative method+AFs-8-11, 13-15+AF0-, and there is still some disagreement about the surgery opportunity choice. According to some studies, MVD probably should be considered once the neurovascular conflict is identified in MRI for the old patients+AFs-8, 11, 13, 46+AF0-. Although the specific reasons are not mentioned in their studies, we speculate that it is related to cerebral arteriosclerosis changes of culprit vessels in elderly patients with ONP. It is well known that the cerebral arteriosclerosis is a predisposing factor to cause ONP. The severity of arteriosclerosis increases with age, which is irreversible+AFs-9+AF0-. Therefore, surgical intervention is ultimately needed to solve the problem. Interestingly, there were 4 ONP patients with culprit vascular atherosclerosis who achieved clinical recovery after MVD, indicating that the vascular arteriosclerotic changes contribute to ON dysfunction and MVD is an effective therapy approach+AFs-8-11+AF0-.+ACY-

nbsp+ADsAPA-br /+AD4-Although the arteriosclerotic changes are not observed in our case, the patient has a history of hyperlipidemia that is a risk factor for atherosclerosis. Does it remind us that the surgical indications of MVD to treat ONP should be more broadly? Prior study observed that longer intervals between diagnosis and surgical intervention were associated with a lower possibility of neurofunctional rehabilitation+AFs-16+AF0-, and a review of 319 cases with isolated ONP induced by aneurysms and administered with surgical decompression revealed that 64+ACU- of patients obtained complete restoration of neurological function when surgical intervention was carried out within 2 wk of symptom onset, 30+ACU- within 2 - 4 wk and 14+ACU- after 1 mo+AFs-47+AF0-. These studies convey a significant message to us that earlier detection and surgical intervention should be followed in patients with ONP.+ADw-br /+AD4-About choice of surgical approach for patients with ONP, both subtemporal craniotomy and orbitozygomatic craniotomy are documented+AFs-8, 11, 12, 16+AF0-. Compared with orbitozygomatic craniotomy, we believe that subtemporal craniotomy provides a wide surgical access to arrive at the base of middle cranial fossa, superior petroclival region and their adjacent structures, which is more suitable for detecting complex vascular malformations in interpeduncular cisterns+AFs-8, 11, 12+AF0-. The patients with ONP treated by MVD does not acquire timely relief of ocular symptoms and usually need about 3 mo recover+AFs-1, 8, 9, 11, 13, 14+AF0-, which is different from those suffering from trigeminal neuralgia and hemifacial spasm that can be alleviated immediately after MVD. Similar results are achieved in our case. The reasons may be attributed to the following reasons after we refer to the causes of delayed healing of hemifacial spasm and the anatomical features of ON+AFs-40, 48-50+AF0-. Firstly, the culprit vessels usually give off arterioles to innervate ON. And these arterioles are easily stimulated by intraoperative traction or subarachnoid blood oozing to constrict, which may result in transient nerve ischemia. The process of ischemia recovery will take some time and go through several periods for changing gradually. Secondly, the neurovascular conflict contributes to local inflammation and demyelination. However, the inflammation and demyelination will be alleviated after

MVD, although it will take some time to regain neuro-potentials from hyperexcitability state+AFs-51, 52+AF0-. Thirdly, a visible indentation, indicating serious neuropathy, can be observed on the surface of ON induced by long-term vascular compression in some cases, including our case+AFs-13, 14+AF0-, and we infer that the delay restoration is related to the time of indentation disappear.+ADw-/p+AD4APA-/html+AD4-

CONCLUSION

+ADw-html+AD4APA-p+AD4-ONP caused by neurovascular conflict is rare and special in clinical practices. To further study the etiology of ONP will provide a reliable basis for subsequent precise treatment. We recommend that any patient with a pupil-involving palsy should be conducted high-quality cranial nerve MRI and angiographic analysis timely to detect the microanatomical relationship between vessels and nerves. The MVD should be considered if the neurovascular conflict is identified in MRI, but the correlation between surgical timing and clinical prognosis should be further investigated.+ADw-/p+AD4APA-/html+AD4-

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