Abducent nerve palsy treated by microvascular decompression: a case report and review of the literature

TOMOSATO YAMAZAKI,[1,2] TETSUYA YAMAMOTO,[2]

TORU HATAYAMA,[3] ALEXANDER ZABORONOK,[2]

EIICHI ISHIKAWA,[2] HIROYOSHI AKUTSU,[2]

MASAHIDE MATSUDA,[2] NORIYUKI KATO,[1]

AND AKIRA MATSUMURA[2]

[1]Department of Neurosurgery, National Hospital Organization, Mito Medical Center, Mito, Ibaraki, Japan

[2]Department of Neurosurgery, Faculty of Medicine, University of Tsukuba, Tsukuba, Ibaraki, Japan

[3]Department of Neurosurgery, Mito Brain Heart Center, Mito, Ibaraki, Japan

Corresponding author:

Tetsuya Yamamoto, MD, PhD

Department of Neurosurgery, Faculty of Medicine

University of Tsukuba

Tennodai 1-1-1, Tsukuba

Ibaraki 305-8575, Japan

Telephone: +81-29-853-3220

Fax: +81-29-853-3214

E-mail: yamamoto_neurosurg@md.tsukuba.ac.jp

Abstract

Too few cases of isolated abducent nerve palsy caused by neurovascular compression syndrome have been reported. We here report on a case of abducent nerve palsy caused by neurovascular compression syndrome that was successfully treated by microvascular decompression (MVD). A 46-year-old man presented with a 6-month history of right-sided persistent abducent nerve palsy. High-resolution magnetic resonance imaging revealed a neurovascular contact of the vertebral artery with the right abducent nerve. MVD was performed via a retrosigmoid craniotomy, with remarkable improvement of the palsy. Our report suggests that MVD might be considered as an optional treatment if the symptoms progress or persist.

Keywords: abducent nerve palsy, microvascular decompression, neurovascular compression syndrome, retrosigmoid approach

Introduction

Neurovascular compression syndromes, which typically manifest as hyperactive dysfunction such as hemifacial spasm or trigeminal neuralgia, are effectively treated by microvascular decompression (MVD) [5, 20]. However, the efficacy of and indication for MVD for cranial nerve hypoactive dysfunction caused by neurovascular compression remain unclear. Only a few cases in which the patient was treated with MVD for these entities, including oculomotor nerve or abducent nerve palsy, have so far been reported [10, 13, 17]. In this case report, we describe a patient with isolated abducent nerve palsy caused by vascular compression of the nerve for whom MVD was performed, and thereby discuss the indications for MVD for isolated abducent nerve palsy.

Clinical Presentation

Patient

A 46-year-old man complained of right-sided diplopia, which had appeared 6 months previously. The diplopia had progressively worsened and was disturbing his daily life. He had no notable past medical history. Hematologic investigations disclosed no abnormalities. Ophthalmologic and neurologic examinations confirmed right-sided abducent nerve palsy. No other neurologic signs were found. The Hess screen test revealed partial right-sided abducent palsy (Figure 1a).

Imaging studies

High-resolution constructive interference in the steady-state (CISS) images using 1.5 Tesla magnetic resonance imaging (MRI) demonstrated a microvascular conflict of the abducent nerve with elongation of the right vertebral artery. The right abducent nerve was clearly compressed by the right vertebral artery. No other abnormalities were observed (Figure 2a).

Surgery

We considered that the vascular compression had caused the progressively worsening abducent nerve palsy, and the patient agreed to undergo MVD and provided signed informed consent for this. A right retrosigmoid craniotomy was performed with the patient in the left lateral park-bench position. The arachnoid membrane around the seventh-eighth nerve complex and the lower cranial nerves was dissected. In the corridor between the seventh-eighth cranial nerve complex and the lower cranial nerves, we subsequently observed that the right abducent nerve was severely compressed by the elongated right vertebral artery (Figure 3). Kinking of the nerve at the entrance to the dura mater was also found. The

right vertebral artery was dissected, transposed away from the right abducent nerve, and fixed to the surrounding dura mater of the petrous bone region with Teflon felt and fibrin glue. The dura mater was closed.

Postoperative course

The postoperative course was uneventful. The abducent palsy had considerably improved by the seventh day after the surgery, and the function of the abducent nerve continued to improve over the next few days. The Hess screen test 7 days after the surgery demonstrated a remarkable improvement of the palsy (Figure 1b). Postoperative high-resolution MRI with a CISS sequence demonstrated decompression of the abducent nerve by the right vertebral artery (Figure 2b). Three months after the surgery, the diplopia had completely disappeared, and the patient did not mention any corresponding disturbance in his daily life. The follow-up Hess screen test 3 months postoperatively revealed that his eye position and movement had almost normalized (Figure 1c).

Discussion

Cranial nerve vascular compression syndromes such as trigeminal neuralgia and hemifacial spasm are typically characterized by paroxysmal hyperactivity likely related to ephatic neurotransmission. These conditions are caused by vascular compression of the root entry/exit zones (REZ) or central segments (glial part) of the cranial nerves [5]. On the other hand, the abducent nerve palsy in the present case is considered a hypoactive dysfunction syndrome caused by vascular compression neuropathy [5, 20]. It is likewise a clinical manifestation supposedly similar to oculomotor nerve palsy caused by posterior communicating artery aneurysms. Several cases with ocular motor dysfunction (oculomotor nerve [1, 4, 10, 17], trochlear nerve [3, 7, 15], and abducent nerve [2, 8, 11-14, 16, 18, 19, 21]) attributed to non-aneurysmal vascular compression have so far been reported. Two cases have been reported in which the patient presented with superior oblique myokymia caused by vascular compression of the trochlear nerve at its REZ as a hyperactivity dysfunction; both cases were successfully treated with MVD [3, 15]. Regarding oculomotor nerve dysfunction caused by non-aneurysmal vascular compression, several cases with oculomotor nerve palsy (hypoactive dysfunction) have been reported. Of those, 3 cases caused by neurovascular compression of the posterior cerebral artery and/or superior cerebellar artery at the cisternal portion of the oculomotor nerve were successfully treated with MVD [1, 10, 17]. On the other hand, we identified only 1 case, reported by Inoue et al, in which the presenting symptom was ocular neuromyotonia (hyperactive dysfunction) caused by pinching between the posterior cerebral and superior cerebellar arteries, that was successfully treated with MVD [4]. In their case also, the cisternal portion of

the oculomotor nerve was found to be the site of the vascular compression. Although it is generally thought that vascular compression for the REZ of the nerves causes hyperactive dysfunction while that for the cisternal portion leads to hypoactive dysfunction, the pathogenetic differences between the hyperactive and hypoactive dysfunctions in the ocular motor nerves remain unclear. They are supposedly associated with severity and/or sites of compression as well as varying vulnerability of the nerves related to the severity of nerve demyelination, length of the central myelin-peripheral myelin transitional zone of the nerves, and so forth [4, 18].

Table 1 summarizes the published cases of isolated abducent nerve palsy due to neurovascular compression of the vertebrobasilar arteries or the anterior inferior cerebellar artery. No report has been published describing vascular compression of the abducent nerve presenting as hyperactive dysfunction. In the reports shown in Table 1, high-resolution MRI with CISS sequences, and so forth, effectively demonstrated a vascular conflict of the abducent nerve with the dolichoectatic vertebrobasilar arteries or the anterior inferior cerebellar artery at the cisternal portion or REZ of the nerve. In 7 of the 10 reported cases, the types of treatment and/or clinical outcomes were described. Four cases were treated with observation and were subsequently clinically unchanged, except for 1 case that showed slight improvement. Zhu et al reported a 68-year-old man who presented with sudden onset of left abducent nerve palsy. Three-Tesla MRI demonstrated a dolichoectatic left vertebral artery compressing the left abducent nerve at the REZ [21]. He underwent left medial rectus recession without transposition 1 year after the onset. Orthophoria was consequently restored, and fortunately he became unaware of the diplopia 3 months after the surgery. Despite evidence supporting neurovascular compression as a possible cause of abducent nerve palsy, only 1 case has been reported of abducent nerve palsy treated by MVD. Ridder et al reported a 56-year-old man with isolated abducent nerve palsy caused by neurovascular contact with a dolichoectatic basilar artery [13]. The patient underwent surgery consisting of a combined supratentorial and infratentorial presigmoid approach and subsequent MVD of the abducent nerve. Intraoperatively, Teflon felt was interposed between the nerve and the basilar artery. Immediately after the surgery, the diplopia was remarkably improved. Likewise, the diplopia in our case was remarkably relieved immediately after MVD surgery. Fortunately, the retrosigmoid approach and the transposition of the vertebral artery that we used carried a lower risk and higher feasibility than did the MVD procedure for a dolichoectatic basilar artery used by Ridder et al. Considering the etiology of neurovascular compression syndrome, we think that MVD has the potential to become a reasonable treatment option.

It is important to discuss the surgical indications for abducent nerve palsy. First, because the natural history of isolated abducent nerve palsy due to non-aneurysmal vascular compression is unknown, we need to infer it from the literature on isolated abducent nerve palsy of undetermined etiology, which

indicates that approximately 70% of the cases resolved spontaneously within 4 to 6 months [9]. However, of the 10 cases caused by neurovascular compression listed in the table of our literature review, 4 cases treated with observation did not demonstrate a favorable functional recovery. Moreover, we think that in some patients, especially in socially active or younger patients, the persistent diplopia is cumbersome and impairs their daily or social activities. Second, it has generally been reported that the MVD procedures for hemifacial spasm and trigeminal neuralgia carry low risks for complications (neurologic complications in 1.7%, hematomas in 0.5%, facial palsies in 0.6% of patients, and so on.) although the surgical risks differ between decompression performed on the vertebral artery and that performed on the basilar artery [6]. The latter may be more risky and more technically demanding, while the former just requires a conventional surgical approach (eg, the retrosigmoid approach). Third, in reference to oculomotor nerve palsy due to cerebral aneurysm, some articles suggested that earlier decompression provides more chances to achieve favorable functional recovery [1]. Therefore, it is reasonable to think that MVD should be a treatment option to consider if the abducent nerve palsy persists for 4 to 6 months or longer.

Conclusions

We have shown that MVD could provide relief in a case of isolated abducent nerve palsy. Bearing in mind the importance of maintaining a balance between the risks of surgery and the severity of the symptoms and disturbance of daily life, we can recommend that if the symptoms progress or persist, MVD is an option to consider in selected cases of abducent nerve palsy.

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Patient Consent

The patient has consented to the submission of the case report to the Journal.

Conflict of Interest Statement

All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speaker’s bureaus;

membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or with any non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

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Figure Legends

Figure 1

a: The Hess screen chart before the surgery demonstrating partial right-sided abducent palsy.

b: The Hess screen chart 7 days postoperatively demonstrating remarkable improvement of the right-sided abducent palsy.

c: The follow-up Hess screen chart 3 months postoperatively revealed that the eye position and movement had normalized.

Figure 2

a: Preoperative high resolution MRI with a CISS sequence revealing compression of the right abducent nerve *(white arrow*) by the right vertebral artery (*black arrow*).

b: Postoperative high-resolution MRI with a CISS sequence revealing decompression of the right abducent nerve (*white arrow*) by the right vertebral artery (*black arrow*).

Figure 3

Intraoperative photographic images.

a: The right abducent nerve is inferiorly compressed by the right vertebral artery, as observed in the corridor between the seventh-eighth nerve complex and the lower cranial nerves.

b: By slightly retracting the right vertebral artery downward with a microdissector, the compression of the right abducent nerve by the right vertebral artery is more visible.

c: The right vertebral artery was transposed and fixed to the surrounding dura mater in the petrous bone with Teflon felt and fibrin glue. The kinking of the nerve at the entrance to the dura mater became more visible after the transposition of the vertebral artery.

Table 1

Summary of reported cases of patients with isolated abducent nerve palsy caused by non-aneurysmal neurovascular compression

ND not described, REZ root exit zone, AICA anterior inferior cerebellar artery, MVD microvascular decompression, * indicates that the affected portion of the nerve was not clearly described in the article.

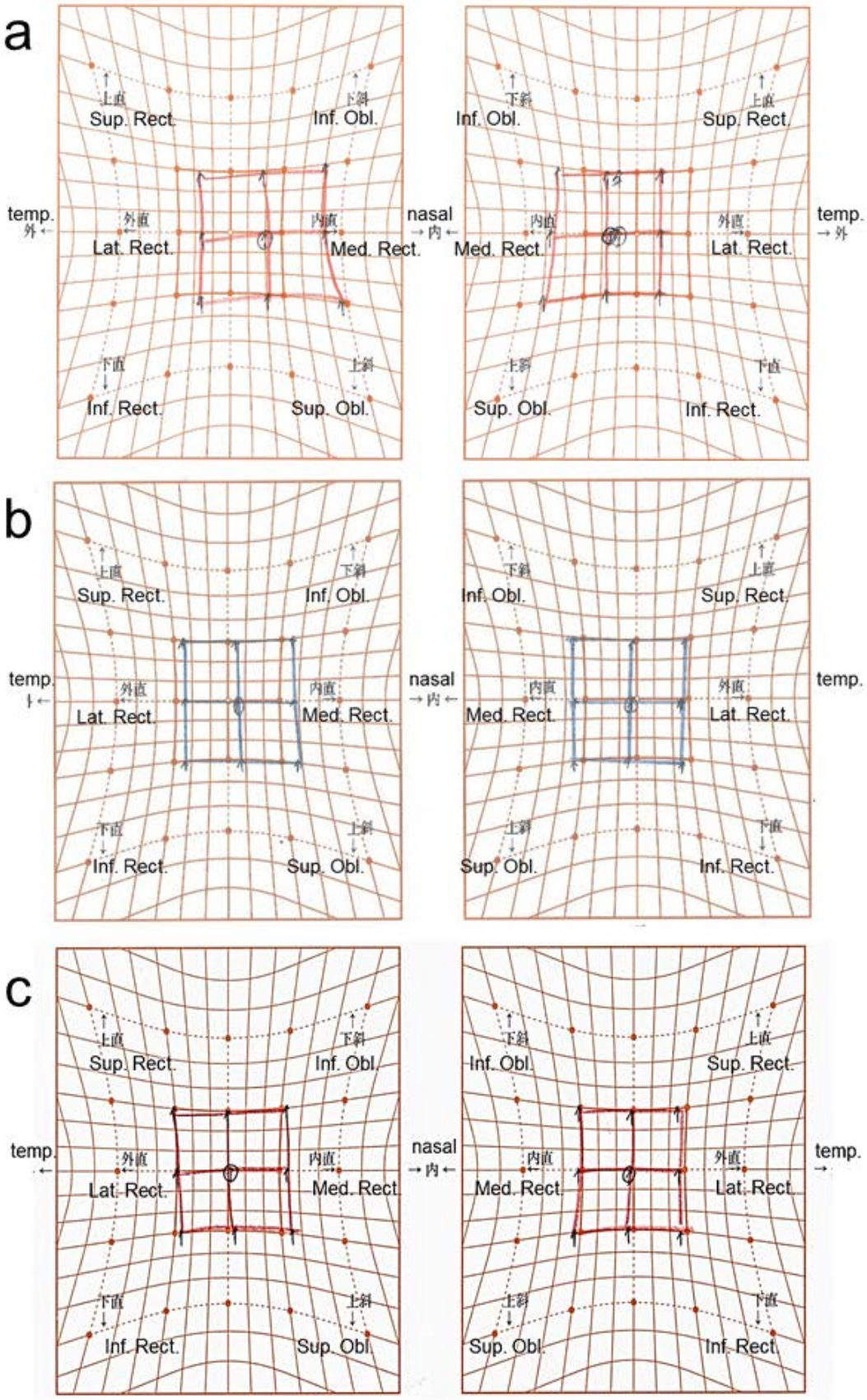
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Figure 1

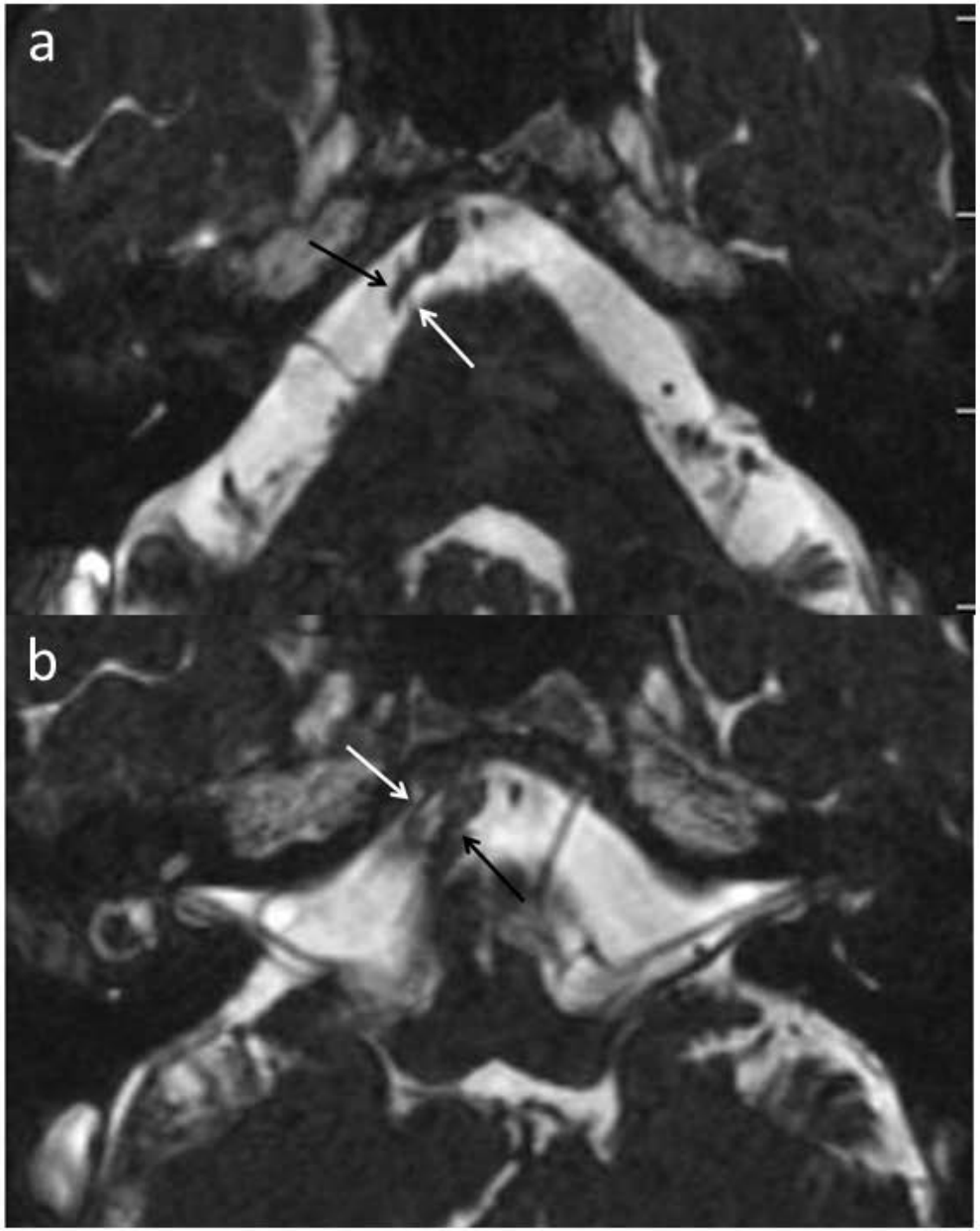

Figure 2

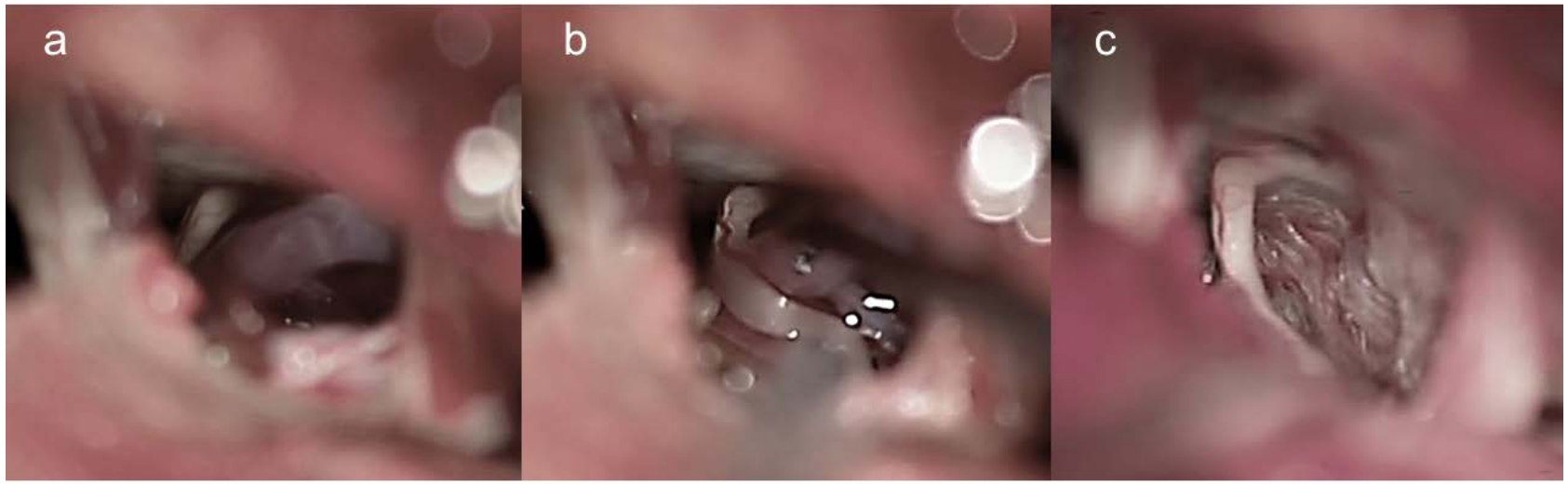

Figure 3

Table 1 Summary of reported cases of patients with isolated abducent nerve palsy caused by non-aneurysmal neurovascular compression

References	Age/sex	Affected portion of abducent nerve	Involved artery	Type of treatment	Outcomes
Smoker et al [16], 1986	59/M	Cisternal portion	Dolichoectatic left vertebral artery	ND	ND
Ohtsuka et al [12], 1996	46/M	REZ	Elongated left vertebral artery	Observation	Unchanged (1-year follow-up)
Narai et al [11], 2000	47/M	Cisternal portion	Elongated left vertebral artery	ND	ND
Goldenberg-Cohen et al [2], 2004	65/M	Cisternal portion or REZ*	Dolichoectatic basilar artery	Observation	Unchanged (4-year follow-up)
Zhu et al [21], 2005	68/M	REZ	Dolichoectatic left vertebral artery	Medical rectus recession	Orthophoria
Ridder et al [13], 2007	56/M	Cisternal portion	Dolichoectatic basilar artery	MVD	Complete resolution
Sandvand et al [14], 2008	38/M	REZ	Right AICA	Prism	ND
Kato et al [8], 2010	50/M	REZ	Basilar artery & AICA	Observation	ND
Taniguchi et al [18], 2011	75/M	REZ for the left Cisternal portion for the right	Bilateral AICA	Observation	Slightly improved (4-month follow-up)
Tsai et al [19], 2012	11/M	Cisternal portion or REZ*	Elongated basilar artery	ND	ND
	52/M	Cisternal portion or REZ*	Elongated basilar artery	ND	ND
Current case	46/M	Cisternal portion	Elongated right vertebral artery	MVD	Complete resolution

ND not described, *REZ* root exit zone, AICA anterior inferior cerebellar artery, *MVD* microvascular decompression, * indicates that the affected portion of the nerve was not clearly described in the article