



Case report

Idiopathic unilateral oculomotor nerve palsy: A case report

Tokunori Kanazawa^{*}, Utaro Hino, Takumi Kuramae, Masayuki Ishihara

Department of Neurosurgery, National Hospital Organization Tochigi Medical Center, 1-10-37, Nakatomatsuri, Utsunomiya, Tochigi, Japan



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ABSTRACT

Cranial nerve III palsy, also known as oculomotor nerve palsy, may result from various causes; however, the etiology remains unknown in some instances. The aim of this case report is to present the authors' experience with two cases of idiopathic cranial nerve III palsy, together with a review of the literature. Case 1 is a 78-year-old woman and case 2 is a 75-year-old man, both having no history of trauma and no vascular risk factors. They presented to the authors' hospital with diplopia and palpebral ptosis and were diagnosed with idiopathic unilateral cranial nerve III palsy. They received oral steroids for treatment. One patient recovered completely within 3 months, while the other patient did not recover regardless of long-term follow-up. Idiopathic cranial nerve III palsy can occur in otherwise healthy individuals and often recover in several months. Careful examinations to rule out other causes and then steroid treatment should be considered after early diagnosis.

1. Introduction

Isolated cranial nerve III palsy is a common neurosurgical presentation in daily practice. There are various causes to cranial nerve palsy, with major causes being diabetes mellitus (DM) and cerebral aneurysms. Recovery can occur within several weeks to months and occurs more frequently in patients with DM. Although there are many known causes for the palsy [1, 2, 3, 4, 5], physicians cannot confirm the etiology in some cases. To our knowledge, however, there have been only a few reported cases associated with the "idiopathic" oculomotor nerve palsy in the literature [6, 7]. Therefore, the authors report two cases of idiopathic unilateral cranial nerve palsy along with the diagnostic workup and a review of the literature.

2. Case presentation

2.1. Case 1

A 78-year-old woman presented to the authors' hospital with acute onset diplopia. The patient was immediately referred to the authors' hospital for careful examination. The patient had no history of trauma. Past medical history included carpal tunnel syndrome and chronic sinusitis. Clinical presentation and examination revealed right upper lid ptosis with impaired levator function, right dilated pupil, and anomalous eye movements when attempting elevation, depression, or adduction of the right eye in motility testing. A detailed workup was performed:

neurological and ophthalmological examination, blood pressure measurement, laboratory tests, and MRI of the brain and orbits including source image of TOF MRA (see Table 1 for the diagnostic workup and the results). Basic laboratory tests consisted of CBC, blood chemistry, coagulation screening, inflammatory markers, urinalysis and CSF. The patient's routine laboratory examination was normal with no evidence of DM, hyperlipidemia, inflammation, and infection. MRI was also unremarkable without intracranial mass lesions and acute infarction (Figure 1 a–c). The authors ruled out various possible underlying causes, including microvascular ischemia, aneurysm, trauma, neoplasm, inflammation, and neurosurgical intervention; thus, the patient was diagnosed with idiopathic unilateral cranial nerve palsy. The recommended therapy was steroid treatment. The patient, therefore, received oral prednisolone with tapering for 2 weeks. However, there were no improvements in diplopia, angle of squint, and ptosis regardless of long-term follow-up. Prism therapy and strabismus surgery were taken into consideration as possible treatment options; however, the patient decided to refuse additional active treatment.

2.2. Case 2

A 75-year-old man presented to the authors' neurosurgery outpatient department with chief complaints of a droopy right upper eyelid and epiphora. The patient had no history of head injury and was otherwise healthy with no significant medical history. On examination, the right pupil was fixed and dilated, with ptosis and adduction palsy of the right

^{*} Corresponding author.

E-mail address: norinori0128jp@yahoo.co.jp (T. Kanazawa).

eye. The other cranial nerves were normal. All the results of routine laboratory tests were within normal limits, and head MRI and MRA revealed no evidence of intracranial aneurysm and acute infarction (Figure 1 d–f); therefore, a presumptive diagnosis of idiopathic unilateral cranial nerve III palsy was made (see Table 1 for the diagnostic workup and the results). The patient immediately received oral prednisolone with tapering for 2 weeks. Complete recovery of his palsy occurred within 12 weeks, and no further treatment was needed.

3. Discussion

Cranial nerve III palsy may result from various causes: microvascular ischemia caused by diseases, including DM, hypertension, and atherosclerosis, aneurysm, trauma, neoplasm, inflammation, neurosurgical intervention, and other known rare causes [1, 2, 3, 4, 5]. Regardless of recent advances in neuroimaging, the etiology of cranial nerve III palsy remains unknown in some cases. The undetermined etiology, also known as “idiopathic,” accounts for around 2.3–26.9% of the cases [1, 3, 4]. In this study, the authors reported two cases of idiopathic unilateral cranial nerve III palsy and reviewed previously published studies associated with cranial nerve III palsy (Table 2).

Isolated cranial nerve III palsy has been reported to occur mainly after the age of 40, with an average age of 39.3–61.4 years [2, 4, 5]. Recovery was noted in 48.3%–70.3% of the cases [1, 3, 4, 5]. Palsies caused by

vascular diseases, such as DM, hypertension, or atherosclerosis, are frequently temporary. Palsies with idiopathic causes also demonstrated significant high recovery rates (50%–72.1%) [1, 3, 4], as previously reported. On the other hand, the recovery rates of cranial nerve impairments due to aneurysm, trauma, and neoplasm, were reported to be low [1, 4]. The mean time of recovery is within 6 months [4]. With regard to the cases of this study, one patient completely recovered within 3 months, while the other did not recover.

Although recent advances in neuroimaging have made early diagnosis easier, management of patients with isolated cranial nerve III palsies still remains challenging. Recommended treatment options vary according to the etiologies. For example, the treatment of palsies with vascular causes largely centers on supportive therapy (i.e., eye patching and prism therapy). In contrast, patients with idiopathic cranial nerve III palsies have been reported to respond well to steroid treatment and to have good prognoses [7], although the mechanism is unclear; indeed, one patient of the two cases in this study showed great improvement after oral steroids for treatment.

In summary, oculomotor nerve palsy can occur in healthy adults in an idiopathic manner but can frequently resolve in several months. It can, however, persist in some cases. The authors emphasize that if a detailed diagnostic workup to avoid misdiagnoses and rule out other causes is performed in otherwise healthy individuals, then steroid treatment should be considered after early diagnosis.

Table 1. The results of diagnostic workup for cranial nerve III palsy.

Underlying Cause	Diagnostic Test	Results
Inflammatory and paraneoplastic process	Lumbar puncture	Case 1: normal, Case 2: normal
Intracranial mass lesions and acute infarction	MRI brain and orbits	Case 1: unremarkable, Case 2: unremarkable
Diabetes	HbA1C	Case 1: normal, Case 2: normal
Inflammatory process or infection	CBC and inflammatory markers	Case 1: normal, Case 2: normal
Aneurysm	MRA	Case 1: unremarkable, Case 2: unremarkable
trauma and neurosurgical intervention	MRI, CT and past history	Case 1: unremarkable, Case 2: unremarkable
Alcohol	Urinalysis	Case 1: negative, Case 2: negative

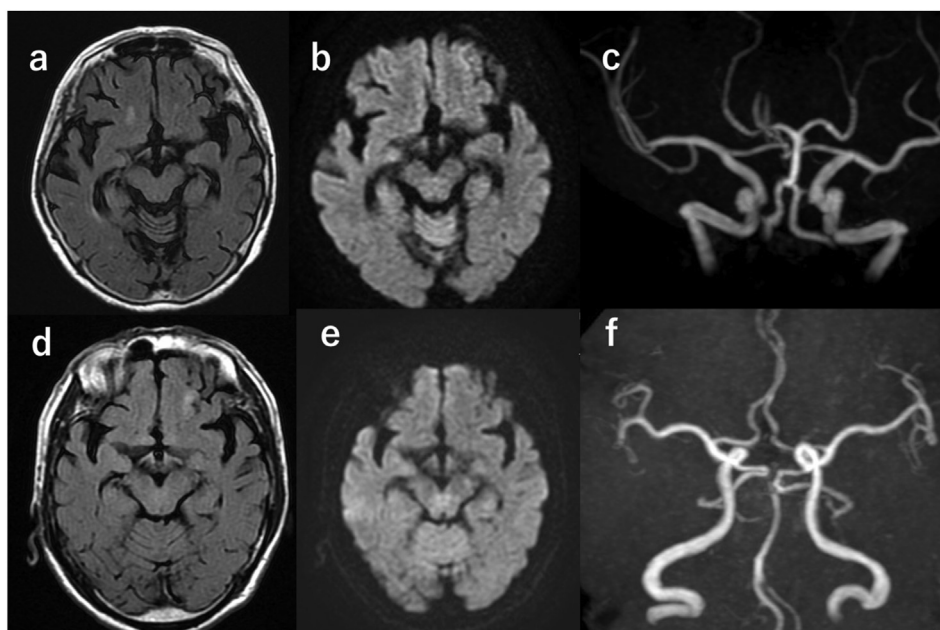


Figure 1. Two cases with right idiopathic unilateral oculomotor nerve palsy. No evidence of intracranial mass lesions (FLAIR; a: case 1; d: case 2), acute infarction (DWI; b: case 1; e: case 2), and intracranial aneurysm (MRA; c: case 1; f: case 2).

Table 2. Selected published reports associated with cranial nerve III palsy.

Published reports [Reference]	Total cases	Age (mean)	Etiology; Cause No. of cases (%)	Recovery (%)
Rush et al. (1981) [1]	290	n/a (not available)	idiopathic 67 (23.1) trauma 47 (16.2) neoplasm 34 (11.7) vascular 60 (20.7) aneurysm 40 (13.8) other 42 (14.5)	total 48.3 idiopathic 50.7
Berlit et al. (1991) [2]	172	53.8	vascular 49 (28.5) inflammation 11 (6.4) tumor 7 (4.1) aneurysm 9 (5.2) trauma 6 (3.5) other 8 (4.7) idiopathic 10 (5.8)	n/a
Fang et al. (2017) [3]	145	n/a	microvascular 61 (42.1) stroke 6 (4.1) compression 25 (17.2) trauma 18 (12.4) post-neurosurgery 14 (9.7) other 8 (5.5) idiopathic 6 (4.1)	total 70.3 idiopathic 50
Kim et al. (2018) [4]	63	61.4	trauma 3 (4.8) neoplasm 2 (3.2) vascular 40 (63.5) aneurysm 1 (1.6) idiopathic 17 (26.9)	total 68.3 idiopathic 72.1
Phuljhele et al. (2020) [5]	129	39.3	ischemia 75 (58.1) trauma 33 (25.6) compressive 9 (7) inflammation/infection 3 (2.3) other 6 (4.7) idiopathic 3 (2.3)	total 69.8 idiopathic n/a

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Author contribution statement

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