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Case Report

High-resolution magnetic resonance imaging in isolated, traumatic oculomotor nerve palsy: A case report ☆

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ABSTRACT

Traumatic, isolated oculomotor nerve palsy is a rare clinical finding and only few reports demonstrate associated magnetic resonance imaging (MRI) findings. Here, we present the case of a 70-year-old woman with left-sided oculomotor nerve palsy following a mild head trauma due to an e-bike accident. Post-traumatic cerebral computed tomography revealed punctiform hemorrhage in the left interpeduncular cistern and the following MRI confirmed an intraneural hemorrhage of the left oculomotor nerve. Nine weeks later, the follow-up MRI showed progressive atrophy and contrast-enhancement of the left oculomotor nerve. To support functional recovery, a treatment with intravenous corticosteroids was started. Six months later, the patient presented with improved oculomotor nerve function and partial recovery of ptosis and diplopia. In accordance, MRI demonstrated recurrent contrast-enhancement of the atrophic nerve. In conclusion, high-resolution MRI allows the reliable delineation of the oculomotor nerve and can support diagnosis in trauma patients with isolated oculomotor nerve palsy.

Introduction

Isolated, traumatic cranial nerve injury is a rare clinical finding as severe head trauma is usually associated with further intracranial pathologies, for example, subarachnoid hemorrhage, basal skull or orbital fractures [1–3]. Therefore, there are only few case reports regarding a distinct isolated oculomotor nerve palsy (ONP) after head trauma [1,4–7].

In general, patients with ONP present with mydriasis, ptosis, diplopia as well as abduction, slight depression, and intorsion of the affected eye [8–10]. In addition to direct ONP following head trauma (often after high-speed car or motorcycle accidents and thus mostly affecting young adult males [1]), there are various other etiologies of ONP: microvascular

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Fig 1 – Initial clinical presentation and follow-up 6 months later. Seventy-year old patient with left oculomotor nerve (ON) palsy after an e-bike accident with mild head trauma. The patient presented with mydriasis, ptosis, abduction and depression of the affected eye (upper line). There were no other pathological findings in neurological and ophthalmological testing. Intravenous corticosteroid treatment was started to support ON recovery. Six months later, the woman presented with partial recovery of ptosis, mydriasis and recurrent diplopia (basal line).

ischemia, aneurysms, tumors, infection, and inflammation [11,12]. Consequently, detailed neurological and ophthalmological testing as well as laboratory analysis is required to enable a reliable diagnosis of ONP and its underlying cause [13]. Furthermore, there is no standard therapy for patients with ONP: Based on the underlying pathology, therapeutic approaches include the surgical decompression of a sphenoid fracture with superior orbital fissure compression, the application of oral or intravenous corticosteroids to support functional oculomotor nerve recovery and prism lenses, injection of botulinum toxin into the lateral rectus muscle of the affected eye as well as low-dose pilocarpine drops to mitigate the patient's symptoms [3,5].

Due to its overall low incidence and its diminutive anatomy, visualization of pathologic alterations of the oculomotor nerve in patients with traumatic, isolated ONP is challenging: Besides the immediately post-traumatic performed cerebral computed tomography (CCT) to exclude intracranial hemorrhage or skull fractures, high-resolution magnetic resonance imaging (MRI) is the modality of choice for detection and follow-up of cranial nerve injury [14].

Here we present the rare case of a patient with isolated ONP following mild head trauma and demonstrate the associated, initial and follow-up radiologic findings.

Case report

A 70-year-old woman without prior neurological or ophthalmological medical history presented in the neurological emergency room of an external hospital after an e-bike accident with a mild head trauma (Glasgow coma score 15): Following a short period of unconsciousness, the patient was not able to open her left eye and thus taken to hospital. The initially performed neurological (including lumbar puncture) and ophthalmological testing revealed an isolated, left-sided ONP. There were no other pathological findings (see also Fig. 1). Immediately performed external CCT and MRI were reported unobtrusive, especially regarding the cranial nerves.

Because ONP has not improved nine weeks later, the woman presented at our department of neurology. The following, detailed re-evaluation of the initial imaging revealed punctiform intraneural hemorrhage of the left ON (see also Fig. 2). To confirm this finding, high-resolution 3 T MRI was repeated: Here, the left oculomotor nerve showed pronounced atrophy and contrast enhancement in the cisternal segment (see also Fig. 3). The intraneural hemorrhage was completely reabsorbed. Furthermore, right ON was normal and there was no intraparenchymal or subarachnoid hemorrhage or other post-traumatic brain injury. To support the patient's clinical rehabilitation, the woman was treated with intravenous corticosteroids (500 mg methylprednisolone) over the course of 5 days. A further clinical and radiological follow-up was scheduled.

Six months later, the patient presented with partial recovery of left-sided ptosis and improved diplopia. In this context, the performed 3 T MRI demonstrated recurrent contrast-enhancement of the constantly atrophic ON (see also Fig. 3).

Discussion

We presented the rare case of traumatic, isolated ONP following a mild head trauma. Initial imaging revealed intraneural ON hemorrhage. The follow-up, high-resolution MRI performed 9 weeks later demonstrated a pronounced atrophy and contrast-enhancement of the ON consistent with a

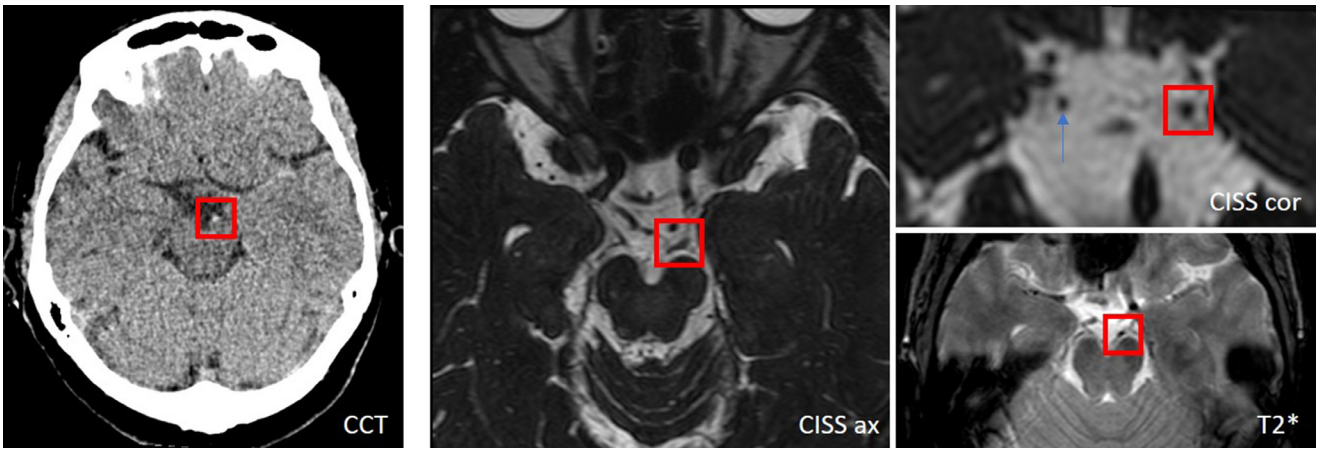

Fig 2 – Initial cerebral computed tomography (CCT) and magnetic resonance imaging (MRI). Initial CCT (left) and MRI (right) demonstrated punctiform hemorrhage of the swollen left oculomotor nerve (red square). There were no other pathological alterations of the brain, right ON was normal (blue arrow).

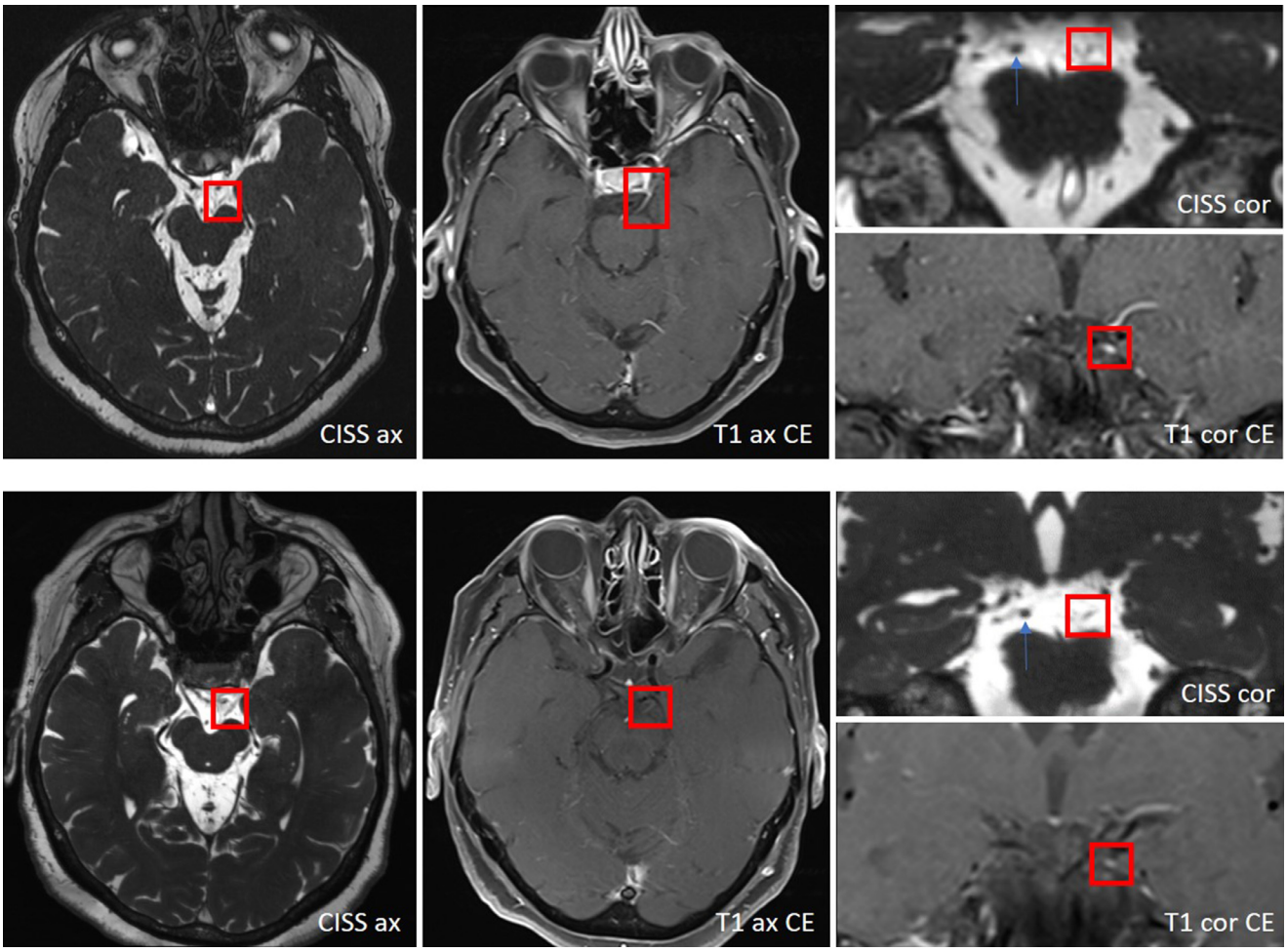

Fig 3 – Follow-up MRI after 2 and 6 months. Follow-up MRI after two (upper line) and six (basal line) months: Two months after the trauma, high-resolution MRI revealed a pronounced atrophy and contrast-enhancement of the left ON (red square); right ON was normal (blue arrow). Six months later, MRI demonstrated recurrent contrast-enhancement of the constantly atrophic ON.

Table 1 – Selected sequences for ONP imaging at 3 T MRI

	T1 coronal	T2 coronal	T2 CISS
TE (ms)	11	87	2.26
TR (ms)	534	3490	5.16
flip angle (degree)	180	165	50
FOV (mm)	190	210	180
matrix	256 × 256	384 × 306	320 × 320
slice thickness (mm)	3.0	3.0	0.6
voxel size (mm x mm x mm)	0.7 × 0.7 × 3	0.5 × 0.5 × 3	0.6 × 0.6 × 0.6
acquisition time (min:sec)	1:52	2:18	3:51

traumatic neural damage. To support functional recovery of ON, intravenous corticosteroid treatment was started. 6 months later, the woman presented with partial recovery of ptosis and improved diplopia. In accordance, high-resolution MRI demonstrated recurrent contrast-enhancement of the impaired, atrophic ON.

Isolated, traumatic injury of the third cranial nerve is rare and only few case reports are available:

In 1984, Elston et al. report 20 patients with traumatic ONP usually caused by high-speed car or motorcycle accidents and consequently often affecting young adult males. Furthermore, ONP is regularly associated with skull fractures and additional palsy of both the fourth and sixth cranial nerves due to their close spatial relation to the ON in the cavernous sinus. Elston et al. conclude, that ONP patients with severe head trauma frequently suffer from additional, permanent neurological deficits [1]. In contrast, Chen et al. report a case of isolated, traumatic ONP. In their study, initial CCT and MRI including MR angiography (MRA) were normal; yet, 4 months after the injury, the patient still suffered from disabling diplopia and mydriasis. The authors conclude, that prognosis of traumatic third nerve palsy is poor and full recovery uncommon [6].

In the largest series yet, Lin et al. followed 26 patients with ONP after mild head trauma. In accordance with our course of action, the authors recommend performing CCT to exclude skull fractures followed by MRI with MRA to detect underlying pathologies responsible for ONP. To reduce post-traumatic neural swelling and to improve functional recovery, the authors recommend treatment with corticosteroids. Furthermore, patients with a sphenoid fracture and consequent compression of the ON in the superior orbital fissure should be considered for early surgical decompression. Lin et al. report recovery rates of 95% for ptosis, 83.3 % for external ophthalmoplegia and 50% for internal ophthalmoplegia [4]. In 2013, Kim et al. presented the first ONP case report demonstrating the associated imaging findings in high-resolution, 3-dimensional MRI: The authors report strong contrast-enhancement and swelling of the affected ON two weeks after the trauma. The authors hypothesize, that the linear force of a high-speed trauma causes a downward move of the brainstem with consequent stretching and contusion of the oculomotor nerve at the nearby posterior petroclinoid ligament. Following treatment with oral corticosteroids, follow-up MRI demonstrated completely resolved ON contrast-enhancement and the patient had fully recovered [5]. This is in accordance with our findings.

In summary and in accordance to the previous case reports, we would recommend the following radiological algorithm in ONP after mild head trauma: First, CCT should be performed to exclude skull fractures with the need for surgical decompression, to detect acute intraparenchymal or subarachnoid hemorrhage and to diagnose acute or subacute ischemia. If CCT is unobtrusive, next high-resolution 3 T MRI including an axial and coronal T1 weighted and T2 weighted sequences, a MRA (to exclude an aneurysm or dissection), diffusion weighted imaging (to detect ischemia), a T2* weighted sequence (to detect hemorrhage) and an isotropic, high-resolution, T2 weighted constructive interference steady state (CISS) sequence as well as contrast-enhanced, axial and coronal T1 sequences (see also Table 1) should be performed to detect traumatic alterations of the ON (eg, swelling, disruption, or hemorrhage) and to exclude indirect reasons for an ONP (eg, brainstem ischemia).

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