

# Wrong side oculomotor nerve palsy

## Introduction

This article presents a woman who was admitted to the authors' emergency department after suffering a head trauma while skiing. She had initially lost consciousness for a few minutes and, as she presented with left oculomotor nerve palsy, multimodal computed tomography and magnetic resonance imaging was performed. This revealed a clinically asymptomatic right internal carotid artery dissection as a relevant additional pathology, which had not been expected as the clinical presentation was on the contralateral side.

## Discussion

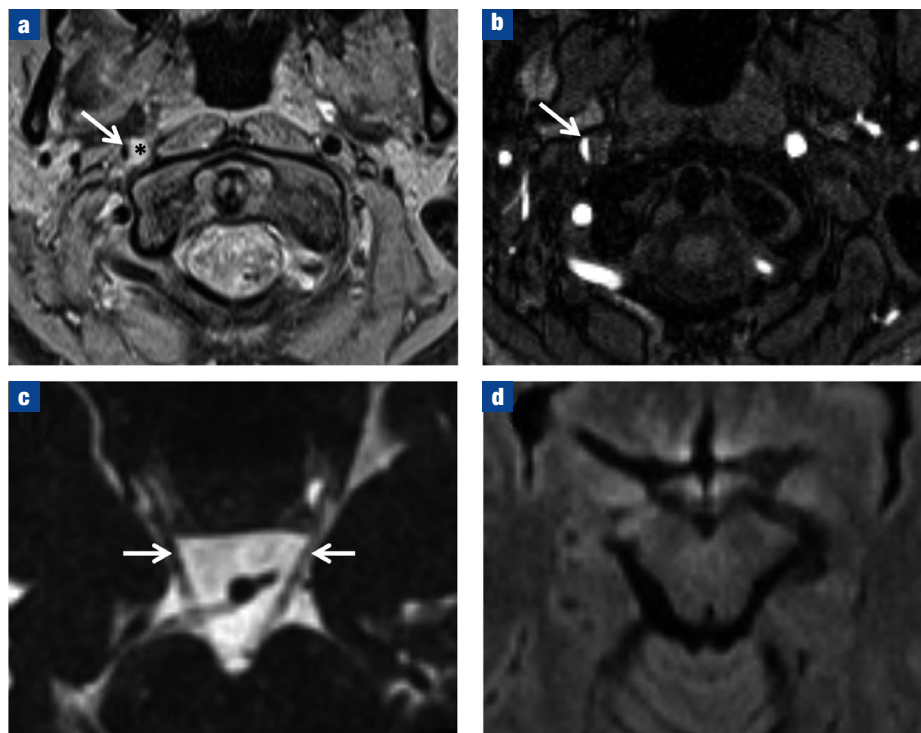
### Internal carotid artery dissection

Various degrees of head and neck trauma may be associated with internal carotid

artery dissection. Intrinsic and/or extrinsic factors compromising integrity of the

vessel wall can cause internal carotid artery dissection (Engelter et al, 2013). Intramural

**Figure 2. a.** Axial T1-weighted fat-suppressed image and **(b)** time-of-flight magnetic resonance angiography show the right internal carotid artery dissection; the mural haematoma (**a** - asterisk) and the narrowed true lumen (white arrow). **c.** No pathology was noted along the oculomotor nerves (white arrows) as depicted on the heavy T2-weighted CISS (constructive interference in steady state) image. **d.** The diffusion-weighted images excluded recent ischaemia.



**Figure 1.** The totally closed eyelid of the affected left eye.



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## CASE REPORT

A 50-year-old woman lost consciousness for a few minutes as a result of a head trauma while skiing, which caused a mild headache. After coming round she could not open her left eye, her left pupil was dilated and unreactive, and her eye position was down- and outward – signs of left oculomotor nerve palsy (*Figure 1*). She was transferred to the authors' emergency unit.

Body computed tomography scan ruled out relevant trauma. Multimodal computed tomography and magnetic resonance imaging did not show visible compression of the oculomotor nerve or subarachnoid haemorrhage. However, it revealed a clinically asymptomatic right internal carotid artery

dissection (*Figure 2*). Neurovascular sonography was also performed, demonstrating the internal carotid artery dissection causing a cervical high-grade stenosis. Anticoagulation was initiated as primary stroke prevention.

During hospitalization, clinical findings remained stable, transient ischaemic attacks did not occur and control magnetic resonance imaging on day 7 did not show new changes, especially no silent ischaemia. On day 8 the patient was discharged home. One year later, the right internal carotid artery was recanalized and the traumatic left oculomotor nerve palsy partially recovered. A surgical intervention was initiated because of disabling diplopia.

haematoma occurs, causing stenosis, occlusion or pseudoaneurysm.

With regard to clinical features, head or neck pain is frequently seen, Horner's syndrome and tinnitus in around 25% of cases, and rarely ipsilateral cranial neuropathies as ipsilateral oculomotor nerve palsy (Nizam et al, 2011). Transient ischaemic attack or stroke occur in >50% of cases (Lee et al, 2006). If present, combined symptoms may indicate internal carotid artery dissection.

The diagnosis is made by neurovascular imaging. Multimodal computed tomography and magnetic resonance imaging show luminal or arterial wall abnormalities and expansion, intramural haematoma and surrounding structures. Digital subtraction angiography is invasive, but can illustrate typical signs of internal carotid artery dissection such as intimal flap, double lumen, string sign and tapered stenosis or occlusion. Carotid duplex can evaluate haemodynamic parameters but has lower sensitivity, especially if internal carotid artery dissection is located near the skull base (Jauch et al, 2013; Kernan et al, 2014).

Data are limited on optimal treatment. For patients with no ischaemic symptoms as in this patient, timely primary stroke prevention should be initiated (evidence grade 2C) with antiplatelets or anticoagulation, followed by routine clinical examination and neurovascular imaging to see the evolution and adjust treatment (Jauch et al, 2013; Kernan et al, 2014).

**Oculomotor nerve palsy**

Lesions anywhere on the path between the oculomotor nerve nuclei in the midbrain,

the fascicular course within the brainstem, the subarachnoid space, the margin of the tentorium, the cavernous sinus and the orbit lead to oculomotor nerve palsy. If it is complete and pupil-involving, patients have ptosis, a large unreactive pupil, paralysis of eye adduction, elevation and depression. The eye rests in a down- and outward position.

Causes vary according to the degree and presence of (non)-pupil-sparing pattern of nerve involvement, associated symptoms, age and historical features such as trauma.

After head trauma, a complete and pupil-involving pattern may present, if loss of consciousness (as in this patient) and/or skull fracture occurred. If not, prompt neuroimaging should be performed, particularly to search for subarachnoid haemorrhage. The use of digital subtraction angiography should be considered on a patient-by-patient basis.

Head trauma damages the oculomotor nerve by compression, deformation, rootlet avulsion, distal fascicular damage and/or defective blood supply.

Traumatic oculomotor nerve palsy, especially if incomplete, may recover spontaneously. Deficits still present after 6 months are generally persistent and surgical interventions may be required to alleviate disabling diplopia and/or ptosis (Bruce et al, 2007). **BJHM**

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**LEARNING POINTS**

- Clinicians should perform neurovascular imaging in the emergency department in a patient with loss of consciousness and oculomotor nerve palsy after head trauma.
- In case of detected internal carotid artery dissection, primary stroke prevention should be initiated.

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# Forthcoming case reports

- Recurrent hypoglycaemia and cognitive impairment: a 14-year follow-up
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- Rhabdomyolysis caused by knee push-ups with whole body electromyostimulation
- Cardiac sarcoidosis: a diagnostic puzzle
- Diagnosed late cardiac sarcoidosis in a patient with atrioventricular block
- A gynaecological cause for spontaneous haemopneumothorax
- Incidental non-functional ectopic thyroid in a returning traveller

**Case Report**

**A feverish ju with a diagn**

**Acute interstitial nephritis caused by two different proton pump inhibitors**

**Case Report**

**Acute interstitial nephritis is a sequela**

**Introduction**

Acute interstitial nephritis is a sequela of acute inflammation of the kidney, which is caused by immune-mediated injury to the renal tubules and interstitium. It is characterized by fever, rash, eosinophilia, and renal dysfunction. The condition is often self-limiting and usually responds to discontinuation of the offending agent. However, in some cases, it can be severe and require dialysis. The condition is most commonly caused by drugs, particularly antibiotics and proton pump inhibitors (PPIs). PPIs are used to reduce stomach acid and are commonly prescribed for a variety of conditions, including gastroesophageal reflux disease (GERD), peptic ulcer disease, and Barrett's esophagus. The use of PPIs has increased significantly in recent years, and this has led to an increase in the incidence of acute interstitial nephritis. In this case report, we describe a patient who developed acute interstitial nephritis after the sequential use of two different PPIs. The patient had a fever, rash, and renal dysfunction, which were resolved after discontinuation of the PPIs. This case highlights the importance of recognizing the signs and symptoms of acute interstitial nephritis and the need for prompt discontinuation of the offending agent. It also emphasizes the need for careful monitoring of patients who are taking PPIs, particularly those who are taking multiple PPIs or who are taking PPIs for a long period of time.

**Discussion**

This case highlights the importance of recognizing the signs and symptoms of acute interstitial nephritis. The patient presented with a fever, rash, and renal dysfunction, which are classic signs of this condition. The sequential use of two different PPIs is a common scenario in clinical practice, and this case demonstrates that the combination of these two drugs can lead to acute interstitial nephritis. It is important to be aware of this potential complication and to monitor patients who are taking PPIs for signs and symptoms of acute interstitial nephritis. If these symptoms are present, the PPIs should be discontinued immediately, and the patient should be treated with supportive care. In some cases, corticosteroids may be used to reduce inflammation and improve renal function. The prognosis for acute interstitial nephritis is generally good, and most patients recover completely within a few weeks. However, in some cases, the condition can be severe and lead to permanent renal damage. Therefore, it is important to be vigilant for the signs and symptoms of acute interstitial nephritis in patients who are taking PPIs.

**Conclusion**

Proton pump inhibitors are a class of drugs that are used to reduce stomach acid. They are commonly prescribed for a variety of conditions, including gastroesophageal reflux disease (GERD), peptic ulcer disease, and Barrett's esophagus. The use of PPIs has increased significantly in recent years, and this has led to an increase in the incidence of acute interstitial nephritis. In this case report, we describe a patient who developed acute interstitial nephritis after the sequential use of two different PPIs. The patient had a fever, rash, and renal dysfunction, which were resolved after discontinuation of the PPIs. This case highlights the importance of recognizing the signs and symptoms of acute interstitial nephritis and the need for prompt discontinuation of the offending agent. It also emphasizes the need for careful monitoring of patients who are taking PPIs, particularly those who are taking multiple PPIs or who are taking PPIs for a long period of time.