

## True- true- unrelated? a delayed onset, complete third-nerve palsy after traumatic subarachnoid hemorrhage

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### Abstract

A 61-year-old male presented to the emergency department (ED) with painless diplopia, left-ptosis, and left downward gaze, 3 days after sustaining a fall from standing height with subsequent lumbar and head trauma. Prior to the ED consult, his only symptom was persistent generalized high intensity headache. On physical examination, no other neurological deficit was found. Computed tomography (CT) scan showed Fisher 4 subarachnoid hemorrhage (SAH). Cerebral angiogram and brain magnetic resonance imaging (MRI) were negative. Screening for possible secondary causes of isolated third-nerve palsy (TNP) were all negative. To our knowledge, this is the first report of a traumatic SAH with delayed onset of an isolated complete TNP as its manifestation.

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CN	cranial nerve
DSA	digital subtraction angiogram
GCS	Glasgow Coma Scale
PCOM	posterior communicating artery
SAH	subarachnoid hemorrhage
TNP	third nerve palsy
tSAH	traumatic SAH

### Keywords

subarachnoid; hemorrhage; complete third-nerve palsy

### Introduction

Isolated complete third-nerve palsy (TNP) in the setting of a subarachnoid hemorrhage (SAH) is most commonly seen secondary to compression by a posterior communicating artery (PCOM) aneurysm or by damage to the

nerve itself caused by blood in the subarachnoid space [1]. In fact, an isolated TNP is one of the clinical hallmarks of a PCOM aneurysm [3]. A CT angiogram (CTA) or digital subtraction angiography (DSA) can

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Figure 1. a CT head without contrast at presentation showing hyperdense subarachnoid hemorrhage with blood in the interhemispheric fissure and basal cisterns. b Digital Subtraction angiogram (DSA)-posterior circulation view demonstrating no evidence of AVM or PCOM aneurysm.

detect a PCOM aneurysm in a high percentage of cases [3]. In this setting, TNP has a relatively good prognosis with a high probability of complete resolution of signs and symptoms within 3- to 6 months [2,10]. However, this same clinical picture with a negative angiogram and otherwise negative imaging studies becomes extremely rare [7]. In contrast, traumatic, isolated TNP tends to have a poor prognosis with a low probability of complete recovery. Although trauma has been described as one of the most common causes of isolated TNP [10,11], concomitant post-traumatic SAH and late onset isolated complete TNP has never been reported. The most common ocular motor cranial neuropathy after trauma is cranial nerve (CN) IV or a trochlear palsy which manifests in hypertropia, contralateral head tilt to offset the intorsion lost from the superior oblique, and diagonal subjective diplopia. In the case of bilateral CN

IV palsies, anterior head tilt can be seen as a clinical sign after significant head trauma.

### Case report

A 61-year-old male presented to an outside hospital Emergency Department with a 3-hour history of painless diplopia and left eye ptosis. He recalled falling with closed-head injury without loss of consciousness 3 days prior to his presentation. He described a generalized 8/10 intensity headache that was partially relieved by ibuprofen since the fall and denied a true thunderclap headache onset. His non-contrast head computed tomography (CT) scan showed a Fisher 4 grade subarachnoid hemorrhage (SAH) (Figure 1a). He was transferred to our hospital due to the concern about a potential posterior communicating artery aneurysm (PCOM) given his CT scan and third-nerve palsy (TNP) at the request of

**Table 1.**

Differential of isolated TNP and cranial imaging with intracranial bleeding or hyperdensities

Diagnosis	Clinical features	Angiogram
Perimesencephalic SAH [4-]	Sudden onset headache. No history of trauma. Painless/painful simultaneous/delayed onset TNP	Negative
Aneurysmal SAH	Sudden onset headache and concomitant TNP	Positive. Most common PCOM aneurysm
Traumatic SAH	History of moderate-to-severe head trauma immediately prior to the onset of TNP	Negative
AML [8]	Painless, complete, bilateral TNP during chemotherapy. No headache or meningeal irritation signs	Negative

the outside hospital neurosurgeon. The patient's past medical history was significant for type 2 diabetes mellitus, obesity (body mass index = 35.5), obstructive sleep apnea, depression, and hyperlipidemia. He was not taking any aspirin, clopidogrel, or anticoagulant medications such as warfarin at the time of his fall.

Upon arrival, the patient was alert, oriented with a Glasgow Coma Scale (GCS) of 15. His neurological exam revealed a dilated 6 mm unreactive left pupil, with impairment of left eye adduction, depression, and elevation consistent with isolated pupil-involved or complete TNP. His complete neurological examination was otherwise normal. He denied any neurological symptoms such as numbness, weakness, or speech difficulty.

Diagnostic DSA was performed and showed no evidence of a PCOM or any other intracranial aneurysm (Figure 1b), vascular anomaly, or other source of bleeding. Brain magnetic resonance imaging (MRI) failed to demonstrate any findings of compression or tumor along the midbrain nucleus or the tract of the third nerve or its fascicles. Based on his neuro-ophthalmologic examination, he had no impairments of visual acuity, visual fields, and dilated fundoscopic examination. His glycosylated hemoglobin was 6.1% (normal being 3.5% to 5.5%) being elevated consistent with his history of diabetes, but his coagulation prothrombin (PT), activated thromboplastin time (aPTT), and platelet count were normal. Causes for secondary vasculitic or ischemic causes of TNP were tested and were normal, including a sedimentation rate of 40, CRP of 7.8, normal angiotensin converting enzyme (ACE) serum level for sarcoidosis, antimyeloperoxidase, and antiproteinase antibodies. After reviewing the MRI, it was evident that the pattern of SAH was traumatic given falcine and tentorial patterns of SAH and very small bilateral subdural hematoma (not shown). Therefore, a repeat DSA was not considered necessary given the patient's clear history of trauma prior to the initiation of symptoms. The patient remained hospitalized in the intensive care unit (ICU) for 5 days with stable, serial neurologic examinations,

and transcranial Doppler (TCD) examinations which were negative for vasospasm. He was discharged 5 days after admission with no further complications but without any improvement of TNP signs and symptoms.

## Discussion

This case illustrates an isolated, complete TNP from traumatic SAH (tSAH). From the presentation of symptoms and image findings, it is most likely that TNP was secondary to direct traumatic injury rather than indirect injury from SAH expansion or aneurysm compression. It is an atypical case for a number of reasons. First, the concomitant presentation of SAH and TNP is rather uncommon, with only a few case reports in the literature. Further on, the late onset of TNP signs and symptoms, being evident up until 3 days after trauma, has never been reported. Finally, the nature of the traumatic episode makes this clinical scenario even more unlikely, since tSAH is usually a consequence of moderate-to-severe head trauma. No case reports of tSAH and late onset TNP have been described so far.

Traumatic SAH has a high incidence (61%) in patients with TBI. It can carry a poor prognosis; however, prognosis can vary significantly according to the grading of tSAH and the severity of CT findings. It is important to consider the traumatic etiology of the TNP in this setting because traumatic palsy has a worse prognosis and less favorable clinical course than palsy secondary to direct compression from an aneurysm, bleeding, or ischemic origin [7].

Given the patient's mild-to-moderate trauma, we feel it is important to consider other etiologies as potential differential entities for TNP, although lower on the list. Spontaneous perimesencephalic SAH is a clinical entity in which no traumatic episode precedes initiation of symptoms and vascular imaging fails to show a source of the bleeding. Actually, in 15–20% of patients with spontaneous SAH, vascular imaging fails to show a source of bleeding [4,9]. These patients have a favorable

outcome compared to those with aneurysmal SAH [9]. They also show a characteristic pattern of bleeding on CT scan, which is typically in front of the pons and mid-brain. Our patient, however, had a traumatic SAH pattern unlike the perimesencephalic SAH pattern. Perimesencephalic SAH with isolated TNP as the only neurologic manifestation is also a rare situation with only three case reports in the literature [4–6]. In these cases, patients later had complete resolution of the TNP.

Another cause in the differential diagnosis of a TNP is an ischemic TNP. Although this has been described as one of the most common causes of isolated TNP in patients with negative angiogram [10–12], these patients usually have a long history of poorly controlled diabetes mellitus, which was not the case of our patient. Additionally, the fact that his pupil was significantly involved would seem atypical for vascular, or ischemic diabetic TNP. Therefore, the case illustrates a rare case of concomitant post-traumatic SAH and complete TNP from trauma. His atypical presentation may represent the combination of both diabetes and traumatic injury to the cranial nerve III in the subarachnoid space, rather than either etiology by itself.

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