

Painful Ophthalmoplegia Mimicking Tolosa-Hunt Syndrome in A 46 Years Old Patient: A Diagnostic Challenge

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ABSTRACT

Background: Painful ophthalmoplegia is an uncommon neuro-ophthalmic syndrome with a wide differential diagnosis, including inflammatory, infectious, vascular, and neoplastic disorders. Tolosa-Hunt syndrome (THS) is a rare cause of painful ophthalmoplegia; however, because it is a diagnosis of exclusion, intracranial space-occupying lesions (SOL) and other structural abnormalities must be carefully ruled out before establishing the diagnosis. This case report aims to remind clinicians of rare conditions that may still be encountered in everyday clinical practice.

Case Presentation: A 46-year-old man presented with a 10-day history of left painful ophthalmoplegia accompanied by blurred vision, diplopia, eyelid swelling, excessive tearing, and left upper extremity weakness. Ophthalmologic examination demonstrated severe visual impairment, marked limitation of extraocular movements, conjunctival hyperemia, and eyelid edema. Neurological examination revealed left upper extremity monoparesis and left peripheral facial nerve palsy. Laboratory investigations showed mild leukocytosis. Non-contrast head computed tomography demonstrated soft tissue swelling at retrobulbar sinistra and left maxilaris sinusitis but was insufficient to exclude an underlying intracranial structural lesion. Based on the clinical presentation, suspected THS was considered; however, the presence of additional neurological deficits raised concern for an intracranial SOL. Intravenous methylprednisolone (125 mg every 12 hours) resulted in rapid improvement of orbital pain, eyelid edema, and conjunctival hyperemia, although severe visual impairment persisted.

Conclusion: This case illustrates the diagnostic dilemma of painful ophthalmoplegia, in which suspected Tolosa-Hunt Syndrome and intracranial Space Occupying Lesions share overlapping clinical manifestations. Although an early response to corticosteroid therapy favored an inflammatory process, persistent neurological deficits and visual impairment underscored the necessity of contrast-enhanced magnetic resonance imaging and further diagnostic evaluation before confirming THS. Early recognition of structural mimics is essential to avoid delayed diagnosis and inappropriate treatment.

Keywords: ophthalmoplegia, Tolosa-Hunt syndrome, space-occupying lesion

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BACKGROUND

Tolosa-Hunt syndrome is an uncommon idiopathic granulomatous inflammatory disorder that primarily involves the cavernous sinus, superior orbital fissure, or orbit, typically affecting one side. It is characterized by intense periorbital pain accompanied by ophthalmoplegia resulting from dysfunction of the third, fourth, or sixth cranial nerves. This syndrome is considered part of the spectrum of idiopathic orbital inflammatory diseases, which also encompasses orbital pseudotumor (Yu et al., 2023; Mahmood et al., 2023; Booroah et al., 2018).

Tolosa-Hunt syndrome occurs globally and does not show any predilection for specific geographic regions or racial groups. It is rarely diagnosed in younger individuals, with the National Organization for Rare Disorders reporting a mean age of onset of approximately 41 years (Amrutkar et al., 2025).

Painful ophthalmoplegia is the characteristic clinical feature of Tolosa-Hunt syndrome. Patients commonly present with diplopia that is more pronounced when viewing distant objects, accompanied by headache, dizziness, nausea, neck stiffness, photophobia, blurred vision, and deep, persistent orbital pain. In severe or extensive cases, the condition may progress to complete unilateral ophthalmoplegia, also referred to as a "frozen globe." (Mullen et al., 2018; Bowman et al., 2022). It mainly involves cranial nerves III, IV, and VI (Mahmood et al., 2023).

The diagnosis of Tolosa-Hunt syndrome is established through a combination of clinical presentation and radiological findings after excluding other potential causes of painful ophthalmoplegia. Magnetic resonance imaging (MRI), particularly when combined with magnetic resonance angiography (MRA) and diffusion-

weighted imaging (DWI), is the preferred imaging modality because it offers superior visualization of the cavernous sinus and orbital apex compared with computed tomography (CT). MRI may reveal granulomatous inflammatory changes that support the diagnosis; however, its findings are not always definitive. Histopathological examination obtained through biopsy can confirm the presence of granulomatous inflammation and provides stronger diagnostic evidence, although the procedure is invasive and may be technically challenging (Mullen et al., 2018).

Response to glucocorticoid therapy is an important feature in both the diagnosis and management of Tolosa-Hunt syndrome. Patients typically experience rapid relief of orbital pain within 48–72 hours after initiation of high-dose systemic corticosteroids. Improvement in cranial nerve palsies generally follows over the subsequent weeks and is often accompanied by regression of inflammatory changes on magnetic resonance imaging (MRI) (Amrutkar et al., 2025; Almokdad et al., 2023).

Although a favorable clinical response to corticosteroids supports the diagnosis of THS, it should not be considered diagnostic in isolation. Similar clinical and radiological improvement may also occur in other conditions, including neoplastic, infectious, and vasculitic disorders. Therefore, a comprehensive diagnostic evaluation and exclusion of alternative etiologies remain essential before establishing a definitive diagnosis of Tolosa-Hunt syndrome. (Falsaperla et al., 2022; Frattini et al., 2023). There are currently no established guidelines regarding the optimal dose, route, or duration of steroid therapy for THS (Sasi et al., 2023).

The recurrence of THS with more severe symptoms underscores the importance of informing patients about the risk of

recurrence and the need for continued follow-up. (Neupane et al., 2025). A rare case of recurrent THS affecting the contralateral eye (Vallinayagam et al., 2021). Previous case reports have suggested that immunosuppressive agents, including infliximab and azathioprine, may serve as alternatives to corticosteroids in THS patients who have an inadequate response to corticosteroid therapy or develop corticosteroid-related adverse effects (Gondim et al., 2020).

Intracranial Space Occupying lesions (SOL) are among the major causes that lead patients to seek neurological and ophthalmological consultations worldwide. Neurological symptoms may arise due to the mass effect exerted on surrounding structures, increased intracranial pressure, secretion or suppression of various hormones by the tumor, or secondary effects such as hydrocephalus (Gotecha et al., 2022).

Ophthalmological manifestations of intracranial tumors, including decreased visual acuity, visual field defects, ophthalmoplegia, papilledema, and optic nerve atrophy, may appear as initial presentations in approximately 46.8–88.6% of patients. These manifestations can result from increased intracranial pressure, compression of the visual pathways, cranial nerves, orbital or ocular structures, or infiltration of the tumor into intraocular structures or ocular adnexa (Gotecha et al., 2022).

Lesions involving the cavernous sinus may affect cranial nerves III, IV, and VI, leading to impaired function of these nerves. Involvement of cranial nerves III, IV, and VI is more frequently observed in tumors located in the supratentorial region (Gotecha et al., 2022; Kumar et al., 2017).

Ophthalmic manifestations may represent one of the earliest clinical features of intracranial space-occupying lesions (SOL). Early recognition of these neuro-

ophthalmic findings is essential, as it may facilitate timely diagnosis, appropriate management, and improved clinical outcomes. Common ocular manifestations of intracranial tumors include decreased visual acuity, diplopia, ophthalmoplegia, pupillary abnormalities, proptosis, nystagmus, and optic disc abnormalities. The pattern and severity of these manifestations depend largely on the location, size, and pathological characteristics of the underlying lesion (Gotecha et al., 2022).

The primary objectives of diagnostic imaging in patients with suspected intracranial masses are to detect the presence of a mass, determine its location and extent of spread, assess involvement of critical anatomical structures, evaluate secondary changes such as edema, herniation, and hemorrhage, and characterize the nature or underlying pathological process (Jindal et al., 2016).

Establishing a definitive diagnosis and accurately characterizing intracranial mass lesions based solely on conventional structural magnetic resonance imaging (MRI) can be challenging. In situations where imaging findings are inconclusive, advanced noninvasive techniques, such as proton magnetic resonance spectroscopy (MRS), can provide additional metabolic information that improves diagnostic accuracy and facilitates differentiation between various intracranial lesions (Dawoud et al., 2016).

CASE PRESENTATION

A 46 years old man presented to the Neurology Outpatient Clinic at Hermina Solo Hospital on Tuesday, July 7, 2026 presented with painful ophthalmoplegia in the left eye, blurred vision in the left eye, and inability to elevate his left arm for 10 days. However, he was still able to move his left leg. He also reported difficulty opening

his mouth for the past 10 days. The symptoms initially began with pain from a decayed tooth, which radiated to the left jaw and left eye. The patient had experienced fever for 10 days. He also complained swelling, excessive tearing, diplopia, and photophobia. Occasionally, he described a throbbing sensation in the left eye. Eye discharge was denied. The patient also reported headache for 10 days, while nausea and vomiting were denied. The patient denied any history of diabetes mellitus, hypertension, asthma, or heart disease.

On general physical examination, the patient appeared moderately ill and was fully conscious. Vital signs were as follows: blood pressure 125/79 mmHg, heart rate 80 beats/minute, respiratory rate 20 breaths/minute, and oxygen saturation 99% on room air.

Neurological examination revealed cranial nerve II with pupils that were isochoric (3 mm/3 mm) and reactive to light bilaterally. There was left-sided palsy of cranial nerves III, IV, and VI, left upper motor neuron facial nerve (cranial nerve VII) palsy, and left upper limb monoparesis with muscle strength of 5/5 on the right and 2/5 on the left. Following evaluation by a neurologist, the patient was admitted for

inpatient care and referred to an ophthalmologist for further evaluation

Ophthalmologic examination showed a visual acuity of 3/60 in the right eye (OD) and 1/300 in the left eye (OS). Intraocular pressure was 14.6 mmHg in the right eye and 17.3 mmHg in the left eye. Extraocular movements were full in the right eye, whereas severe limitation of movement in all directions (−3) was noted in the left eye. Examination of the left eyelid revealed edema, tenderness, and erythema (Figure 1). The tarsal conjunctiva was normal in the right eye but hyperemic in the left eye, while the bulbar conjunctiva was normal in the right eye and showed conjunctival injection in the left eye. Both corneas were clear, the anterior chambers were well formed, and the pupils measured 3 mm bilaterally with intact direct and consensual light reflexes. Examination of the left jaw demonstrated edema, tenderness on palpation, and grade III trismus with marked limitation in mouth opening. No erythema was observed. Funduscopy examination revealed blurred margins of the left optic disc. The optic disc maintained a normal color, and the remainder of the fundus appeared normal.



Figure 1. Hyperemia of the left eyelid (palpebra) on the first day of examination

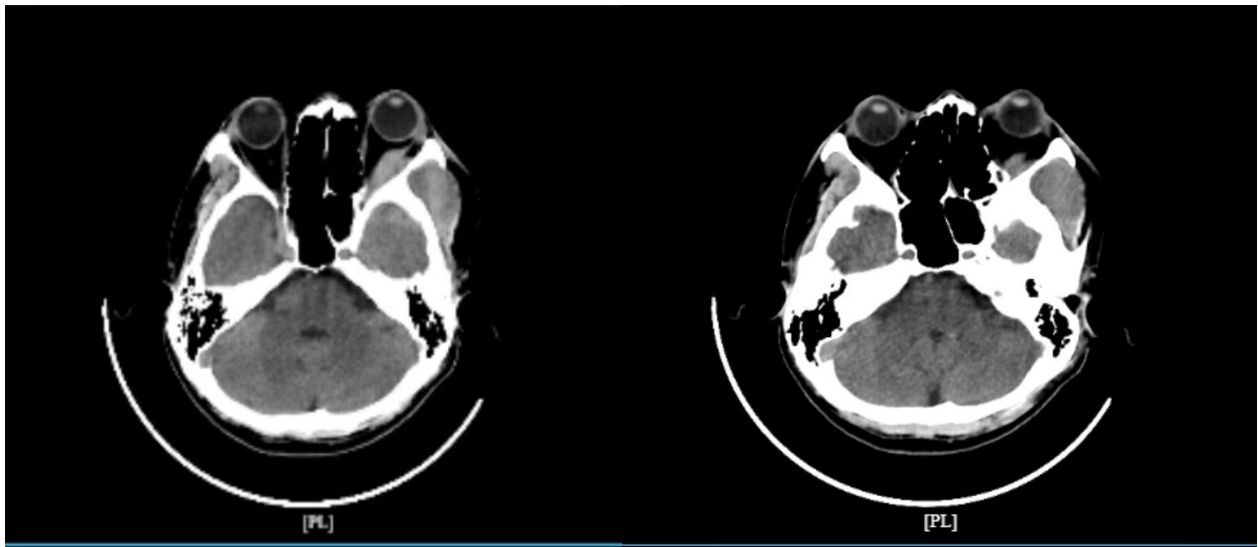


Figure 2. The non-contrast 16-slice head CT scan

Laboratory investigations revealed the following hematological findings: hemoglobin 14.0 g/dL, hematocrit 40.7%, leukocyte count $11.52 \times 10^3/\mu\text{L}$, and platelet count $406 \times 10^3/\mu\text{L}$. Clinical chemistry results showed a blood urea level of 27 mg/dL, serum creatinine of 0.79 mg/dL, random blood glucose of 98 mg/dL, and serum sodium level of 135.2 mmol/L.

The non-contrast 16-slice head CT scan demonstrated soft tissue swelling at retrobulbar sinistral, left maxillary sinusitis, and leftward deviation of the nasal septum. No intracerebral hemorrhage was identified within the brain parenchyma. There was no evidence of fractures involving the calvarium, maxillofacial bones, or cervical spine (Figure 2).

The patient received treatment from the neurology team, including intravenous Ringer's acetate solution at 20 drops per minute, intravenous methylprednisolone 125 mg every 12 hours, intravenous citicoline 500 mg every 12 hours, intravenous omeprazole 40 mg once daily, and folic acid 400 μg twice daily. Ophthalmologic treat-

ment included Prednisolone eye drops, one drop in the left eye three times daily, and Ofloxacin eye drops, one drop in the left eye six times daily.

On the second day of follow-up, the hyperemia of the left eye had decreased, and the patient reported reduced pain. However, visual acuity remained at 1/300, with persistent limitation of extraocular movements (-3). Muscle strength in the left upper extremity still at 2/5.

On the third day of hospitalization, the patient reported improvement in left eye pain after received treatment from the first day of hospitalization; however, visual acuity remained unchanged at 1/300 left eye. Hyperemia of the left eyelid had also decreased. On the third day of hospitalization, a contrast-enhanced magnetic resonance imaging (MRI) examination was recommended to establish the diagnosis. The patient was subsequently referred on Friday, July 10, 2026, to a hospital with MRI facilities for further diagnostic evaluation (Figure 3).



Figure 3. Clinical photograph on the third day of examination demonstrating reduced left eyelid hyperemia, while severe limitation of extraocular movements in all directions persisted.

DISCUSSION

Painful ophthalmoplegia is an uncommon but clinically significant neuro-ophthalmic syndrome with a broad differential diagnosis, including inflammatory, infectious, vascular, and neoplastic disorders. Tolosa-Hunt syndrome (THS) is one of the most recognized inflammatory causes. (Nosaki et al., 2020). However, because it is a diagnosis of exclusion, alternative etiologies must be carefully investigated before a definitive diagnosis can be established.

The present case demonstrated several clinical features compatible with THS, including unilateral orbital pain, severe ophthalmoplegia involving cranial nerves III, IV, and VI, diplopia, eyelid edema, conjunctival hyperemia, and marked visual impairment (Mahmood et al., 2023). The rapid improvement in orbital pain and

eyelid inflammation following initiation of high-dose intravenous methylprednisolone also supported an inflammatory process. Response to corticosteroid therapy is considered a characteristic feature of THS, with orbital pain typically improving within 48–72 hours after treatment ((Amrutkar et al., 2025; Almokdad et al., 2023).

THS is primarily treated with corticosteroids, although standardized recommendations regarding the specific drug, formulation, administration route, and optimal dose are lacking. Clinical improvement typically occurs rapidly, within one to three days of initiating corticosteroid therapy (Kim et al., 2021; Siddhanta et al., 2021; Oghene et al., 2021)

Nevertheless, steroid responsiveness alone is insufficient to confirm the diagnosis, as transient clinical improvement may

also occur in other inflammatory, infectious, neoplastic, or vasculitic disorders. The diagnosis of THS is based on a combination of clinical, laboratory, and imaging findings, while excluding other potential causes of painful ophthalmoplegia (Bennett, 2024). The severe reduction in visual acuity (BCVA 1/300) suggested possible optic nerve involvement or impaired retinal and optic nerve perfusion secondary to orbital inflammation. Combined palsy of cranial nerves III, IV, and VI indicated involvement of structures within the cavernous sinus, superior orbital fissure, or orbital apex, where these nerves travel in close anatomical proximity. Although this pattern is characteristic of THS, similar findings may also occur in intracranial tumors involving the sellar or parasellar region, cavernous sinus, or orbital apex. Consequently, the presence of multiple cranial neuropathies should prompt consideration of both inflammatory and structural lesions (Gotecha et al., 2022; Kumar et al., 2017)

An important feature distinguishing this case from typical THS was the presence of left upper motor neuron facial nerve palsy and left upper-extremity monoparesis. These focal neurological deficits are not characteristic of isolated orbital inflammatory disease and raised concern for an intracranial structural lesion. In particular, isolated upper-limb weakness suggests involvement of the contralateral motor cortex or adjacent subcortical pathways rather than pathology confined to the cavernous sinus or orbital apex. Furthermore, ophthalmic manifestations may represent the earliest presentation of intracranial space-occupying lesions (SOLs). Visual impairment, diplopia, ophthalmoplegia, pupillary abnormalities, proptosis, and optic disc changes have all been described in patients with intracranial tumors, with the clinical presentation depending on the

location, size, and histopathological characteristics of the lesion. Therefore, the coexistence of painful ophthalmoplegia with focal neurological deficits should increase suspicion for an intracranial SOL.

The patient's history of severe dental pain, trismus, fever, and facial swelling initially suggested an odontogenic infection with possible orbital extension. Although the leukocyte count was only mildly elevated at $11.52 \times 10^3/\mu\text{L}$ and the patient showed improvement after corticosteroid therapy. Infectious etiologies could not be completely excluded because corticosteroids may temporarily reduce inflammatory manifestations while masking the progression of an underlying infection. The non-contrast head CT scan demonstrated soft tissue swelling at retrobulbar sinistra and left maxillary sinusitis but showed no evidence of intracerebral hemorrhage or obvious intracranial mass. However, conventional non-contrast CT has limited sensitivity for evaluating lesions involving the cavernous sinus, orbital apex, and skull base.

Contrast-enhanced magnetic resonance imaging (MRI) remains the imaging modality of choice for evaluating painful ophthalmoplegia and distinguishing THS from structural lesions affecting the cavernous sinus and orbital apex. When conventional MRI findings are inconclusive, advanced imaging techniques such as proton magnetic resonance spectroscopy may provide additional metabolic information that improves lesion characterization. Because persistent severe visual impairment, facial nerve palsy, and upper-extremity monoparesis remained despite partial clinical improvement, referral for contrast-enhanced CT and MRI was warranted to exclude an intracranial SOL and establish a definitive diagnosis. (Mullen et al., 2018)

The absence of a definitive contrast-enhanced MRI result in this case represents

an important diagnostic limitation. Although the clinical presentation and early response to corticosteroid therapy were suggestive of THS, the diagnosis could not be established with certainty without adequate exclusion of alternative causes. Further neuroimaging, particularly contrast-enhanced MRI of the brain, cavernous sinus, orbit, and skull base, would be necessary to evaluate for an intracranial space-occupying lesion, infectious extension, or other structural pathology.

This case highlights the diagnostic challenge posed by painful ophthalmoplegia. Although the patient's clinical presentation and early response to corticosteroid therapy favored an inflammatory disorder such as THS, the presence of atypical neurological deficits necessitated thorough investigation for intracranial structural pathology. Careful integration of clinical findings, neurological examination, laboratory results, and comprehensive neuroimaging is essential to achieve an accurate diagnosis and guide appropriate management.

This case highlights the diagnostic challenge of painful ophthalmoplegia, particularly in distinguishing suspected Tolosa-Hunt syndrome from intracranial space-occupying lesions. Although the patient's clinical presentation and early response to high-dose corticosteroid therapy suggested an inflammatory process, the presence of atypical neurological deficits, including central facial nerve palsy and upper-extremity monoparesis, raised concern for an underlying intracranial structural lesion. Because Tolosa-Hunt syndrome is a diagnosis of exclusion, comprehensive neuroimaging, particularly contrast-enhanced magnetic resonance imaging, is essential to exclude alternative etiologies before establishing a definitive diagnosis. Early recognition of atypical clinical features is

crucial to avoid delayed diagnosis and ensure appropriate management.

This case report aims to remind clinicians of rare conditions that may still be encountered in everyday clinical practice, encouraging greater awareness and a broader differential diagnosis when evaluating patients with similar presentations.

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AUTHORS CONTRIBUTIONS

Dini Arintawati: Conceptualization, clinical data collection, investigation, and manuscript drafting.

Heru Prasetyono: Clinical investigation, interpretation of clinical and imaging finding, critical review of the manuscript, and final approval of the version to be published.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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