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CASE REPORT

SUCCESSFUL MANAGEMENT OF RECURRENT ORBITAL INFLAMMATION WITH PAINFUL OPTHALMOPLEGIA CAUSED BY TOLOSA HUNT SYNDROME MULTIDISCIPLINARY APPROACH

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ABSTRACT

Background: Orbital inflammatory disease (OID) is a diagnosis of exclusion, one of the differential diagnoses is Tolosa hunt syndrome. Even though corticosteroids are effective, this disease has the potential for recurrence.

Case Presentation: A 22-year-old male presented with left eye pain, proptosis, incomplete eyelid closure, decreased visual acuity, and limited eye movements, with symptoms recurring three times in one year. CT scan findings were consistent with Tolosa-Hunt Syndrome, and biopsy revealed a non-specific inflammatory process. Diagnosis was based on clinical presentation, surgical findings, and imaging. The patient was treated with high-dose corticosteroids and subsequently received immunomodulatory therapy to prevent recurrence, resulting in a symptom-free period over the past year.

Tolosa-Hunt syndrome is a rare condition caused by inflammation of the cavernous sinus, characterized by recurrent orbital pain and cranial nerve palsies involving nerves III, IV, V, and VI. In addition to clinical manifestations, imaging plays a critical role in confirming the diagnosis. Corticosteroids are considered the first-line therapy for this condition. However, in this case, corticosteroid therapy alone failed to prevent recurrence. The administration of immunomodulatory agents such as methotrexate has been shown to effectively reduce the risk of relapse.

Conclusion: Tolosa-Hunt syndrome is an important differential diagnosis of orbital inflammatory disease, especially in recurrent cases with cranial nerve palsies. High-dose corticosteroids are the first-line treatment but have long-term side effects. Immunomodulators like methotrexate may help prevent recurrence. This case highlights the need for a multidisciplinary approach and adequate patient education to achieve lasting remission.

Keywords : *Orbital inflammatory disease, Tolosa Hunt syndrome, Painful Opthalmoplegia*

INTRODUCTION

Orbital inflammatory disease (OID, aka orbital inflammatory pseudotumor, idiopathic orbital inflammatory syndrome, nonspecific orbital inflammation) [1-3] was first described by Gleason in 1903 and accounts for 6% of diseases involving the orbit [4]. It is the third most common orbital disease after Grave's orbitopathy and lymphoproliferative diseases [5]. OID is most commonly unilateral with symptoms and clinical findings depending on the site involved as well as the degree of inflammation, fibrosis, and any mass effect. Generally, acute OID presents with proptosis, extraocular motility disturbance, pain, erythema, and chemosis [2]. As OID is a diagnosis of exclusion, patients must be evaluated to rule out any malignancy, infection, systemic inflammatory process, or other concomitant medical conditions [6]. The differential diagnosis includes local and systemic inflammatory conditions caused by neoplasm, infection, vascular malformation, and trauma [5].

Many orbital inflammations may be associated with systemic conditions or remote organ dysfunction. Categories of orbital pseudotumor according to location include anterior, diffuse, posterior, or apical. Other classifications include myositis, dacryoadenitis, periscleritis, perineuritis, and focal mass [7].

Understanding the clinical spectrum and diagnostic criteria of orbital pseudotumor is essential for accurate diagnosis and effective treatment. Patients commonly present with unilateral proptosis, discomfort, diplopia, and visual abnormalities, which can be either acute or subacute [8].

These symptoms often resemble other orbital diseases such as neoplasms, infections, or thyroid eye disease. Clinical assessment, imaging modalities (such as

computed tomography [CT] and magnetic resonance imaging [MRI]), and occasionally histological examination acquired via biopsy are utilized to differentiate orbital pseudotumor from these conditions [9].

In this case, the patient has experienced recurrence 3x in this 1 year period. Establishing a correct diagnosis will help in determining therapy. Multidisciplinary therapy is needed to provide optimal therapeutic results and reduce the potential for disease recurrence. Patients, families, opthalmologists, internists have a very important role in this case.

CASE PRESENTATION

December 2023

A 22-year-old male presented with complaints of left eye pain for the past three months. The pain was recurrent and accompanied by left eye protrusion, eyelid closure, decreased vision, and difficulty in eye movement. Visual acuity was 6/6 in the right eye and 5/60 in the left eye. On examination of the left eye, there was proptosis, ptosis, reduced pupillary reflex (Figure 1), and restricted ocular movement (Figure 2).

Figure 1. There was ptosis, and the globe appeared more proptotic.
(Picture taken with patient's consent, *Courtesy : Poli Room RSUD Dr. Soetomo*)

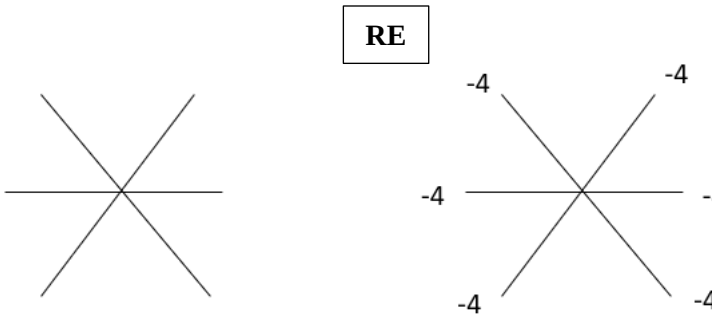

Figure 2. Ocular motility in the left eye showed a -4 restriction in all directions, without any pain.

CT scan revealed findings suggestive of rhabdomyosarcoma, with differential diagnoses including thyroid-associated orbitopathy and isolated primary amyloidosis. The patient subsequently underwent an incisional biopsy of the orbital mass.

Histopathological examination showed a non-specific inflammatory process, with no evidence of malignancy and no features supporting amyloidosis.

Table 1. The patient's clinical condition is based on the treatment

Date	April 2024	July 2024	September 2024
Complaint	The same complaint	Sudden loss of vision in both eyes	Right eye vision has returned, no pain
Visual Acuity	RE : 5/5 LE : 5/12 ph 5/10	RE : Light Perception LE : Light Perception	RE : 5/5 LE : 5/60 phni
Diagnose	LE Orbital tumor susp NSOID Susp Psoriatic Arthritic	RLE orbital inflammation dd tolosso hunt syndrome RLE Optic Perineuritis PSA	RLE Orbital Inflammation with Painful Opthalmoplegia ec Tolosa Hunt Syndrome PSA
Planning	Medicamentosa (Corticosteroid High dose IV)	Medicamentosa (Corticosteroid High dose IV)	Medicamentosa (Tappering off Corticosteroid)

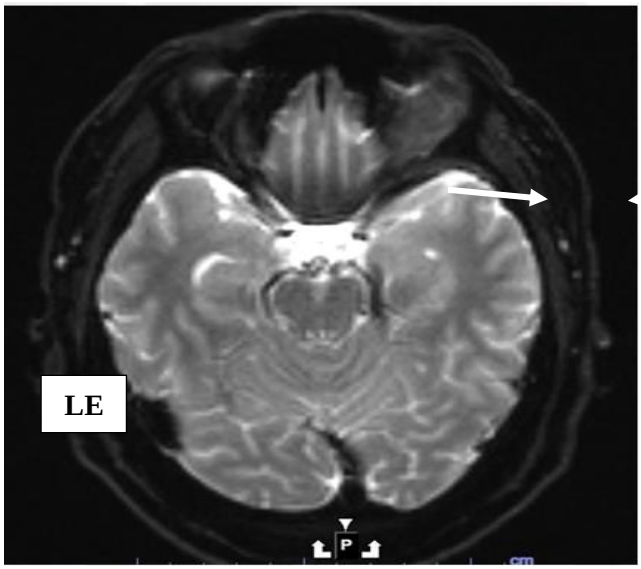

Figure 3. The contrast-enhanced head MRI of the patient in June 2024 demonstrated findings consistent with Tolosa-Hunt syndrome, with a differential diagnosis of en plaque meningioma. (Picture taken with patient's consent, *Courtesy : Poli Room RSUD Dr. Soetomo*)

We reduced corticosteroid therapy to 5mg/day. Other therapies are appropriate for internists and neurologists. Patients and families are educated regarding prognosis, recurrence prevention, rehabilitation, visual optimization and regular health control.

Based on the results of previous multidisciplinary therapy conclusions. The diagnosis in this patient was based on anamnesis, clinical examination, supporting examinations and the results of multidisciplinary consultations. Our patient was diagnosed with RLE recurrent orbital inflammation with painful opthalmoplegia ec tolosa hunt syndrome, Psoriatic Arthritis, Pre Obesity (BMI 27.43).

The following are clinical photographs of the patient during the initial visit prior to the incisional biopsy, and the most recent visit in September 2024 (Figure 4).

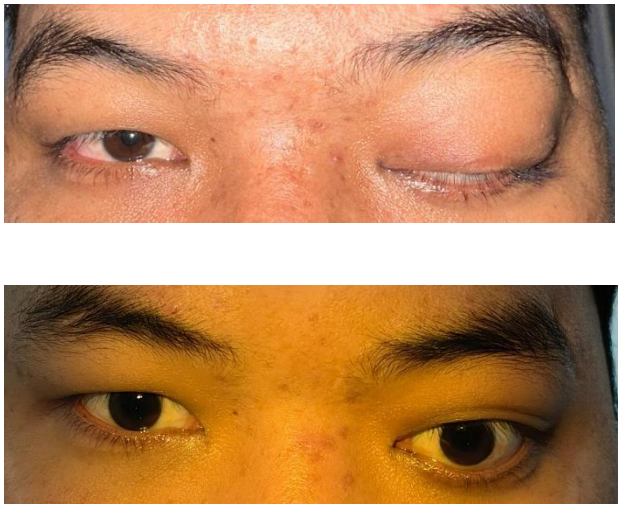

Figure 4. A. shows the clinical photograph of the patient during the initial visit prior to the incisional biopsy. **B.** shows the clinical photograph during the most recent visit in September 2024. Notable differences are observed, including incomplete closure of the left eyelid and the absence of left eye proptosis.
(Picture taken with patient's consent, *Courtesy : Poli Room RSUD Dr. Soetomo*)

DISCUSSION

Orbital inflammatory disease (OID, aka orbital inflammatory pseudotumor, idiopathic orbital inflammatory syndrome, nonspecific orbital inflammation) [1-3] was first described by Gleason in 1903 and accounts for 6% of diseases involving the orbit [4]. As OID is a diagnosis of exclusion, patients must be evaluated to rule out any malignancy, infection, systemic inflammatory process, or other concomitant medical conditions [6]. The differential diagnosis includes local and systemic inflammatory conditions caused by neoplasm, infection, vascular malformation, and trauma [5].

The anterior OID affects the globe, conjunctiva, eyelids, neural, and adjacent muscular structures. Pain and periorbital swelling are the most frequently encountered presenting features (Figure 4). Other common features include conjunctival chemosis and limited ocular motility. Rarely, proptosis, uveitis, papillitis, and exudative retinal detachment can also be seen[10].

In the patients we treated, the patient first came with clinical symptoms of acute OID, such as pain in the left eye accompanied by decreased vision, proptosis, swollen and closed eyelids. As explained that OID is a diagnosis of exclusion, the differential diagnosis in our patient at that time was Rhabdomyosarcoma if we looked at the MRI results from the area of origin. The patient underwent a tumor biopsy incision and the results were inflammatory process, non specific. Does not appear malignancy, does not support amyloidosis. The patient has experienced recurrence 3 times during 1 year. The MRI results after the biopsy incision stated that there was a high possibility of Toloso Hunt syndrome (most likely), Enplaque meningioma (less likely).

Apical or posterior OID, while less common, is associated with a poorer visual outcome[4]. Clinically, apical OID presents with orbital pain, restricted eye movement, visual loss, and minimal proptosis[11]. Inflammatory lesions of the orbital apex may extend intracranially through superior orbital fissure, optic canal, and inferior orbital fissure. The cavernous sinus and the middle cranial fossa are the two most common locations for intracranial involvement[12]. Tolosa–Hunt syndrome is a rare clinical condition caused by idiopathic granulomatous inflammation in the region of cavernous sinus and/or superior orbital fissure. Tolosa–Hunt syndrome presents with relapsing/remitting partial/complete ophthalmoplegia, visual loss, unilateral headache, and paralysis of cranial nerves III, IV, V1, and VI[13]. Neuroimaging may show an enhancing mass within the cavernous sinus (Figure 5).

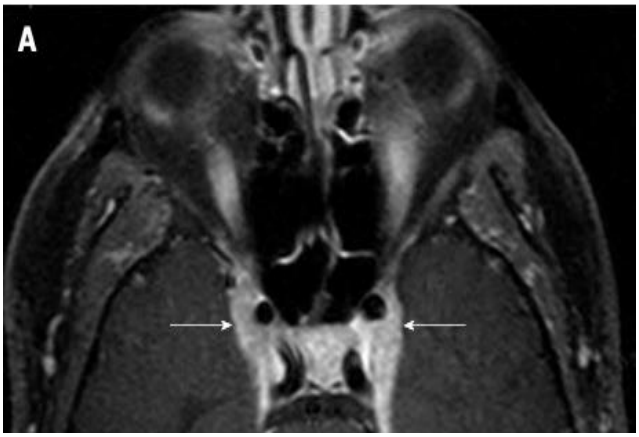

Figure 5. Tolosa-Hunt syndrome. Axial fat suppressed contrast-enhanced T1 showing bilateral cavernous sinus infiltration and enhancing tissue along the lateral margins of the cavernous sinus

Tolosa Hunt Syndrome also finds a place in the IHS Classification ICHD-3 Beta, in part three, under Painful cranial neuropathies and other facial pains. The IHS lays down diagnostic criteria for THS which have

high sensitivity (approximately 95% to 100%) but low specificity (approximately 50%). They are summarized as follows (Table 3)[16]:

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Table 3. Diagnostic criteria for Tolosa Hunt syndrome[16].

International Headache Society (IHS) Diagnostic Criteria
• Unilateral headache
• Includes both of the following: 1. Presence of granulomatous inflammation of the cavernous sinus, superior orbital fissure or orbit, as seen on MRI or biopsy 2. Palsies of one or more of the oculomotor nerves (cranial nerves III, IV, and/or VI) on the same side
• Corroboration of the cause as evidenced by both of the following: 1. Palsies of cranial nerves III, IV, and/or VI have followed headache in two weeks or less, or have developed simultaneously with a headache 2. Localization of a headache around the eye on the same side
• Not better explained by any other headache etiology

In this case, the patient's complaints and clinical symptoms similiarly Tolosa Hunt syndrome. This is supported by the results of the post-operative MRI, namely that there is enhancement in the sinus covernosus and orbit. The patient also had weakness in nerves III, IV and VI in the left eye with painful and restricted eye movements. Frequently, the cause of a painful ophthalmoplegia in patients initially diagnosed with Tolosa- Hunt syndrome is later discovered to be neoplastic. Therefore, Tolosa- Hunt syndrome is a diagnosis of exclusion[14].

Patients with suspected Tolosa Hunt Syndrome will have a large risk of recurrence, as is the case in this case. The patient has experienced recurrence 3 times in a 1 year period. The vision in the left eye slowly began to decrease even though therapy had been given. The patient had no control and stopped taking medication, which increased the risk of disease recurrence and worsened the prognosis of the disease. Ideally, holistic management is needed involving ophthalmologists, internal medicine, neurologists for rehabilitative efforts and the role of the patient and family as a preventive effort.

Current therapeutic agents available for OID are: corticosteroids, nonspecific steroid-sparing agents such as (Methotrexate, Cyclosporin-A, Cyclophosphamide), biologic agents such as Etanercept, radiation therapy if Intolerant to cortisone or unresponsive therapy and surgery for nonresponders. Systemic corticosteroids, such as prednisone or methylprednisolone, are the cornerstone of early medical treatment for ocular pseudotumor[14].

Corticosteroid are considered first-line treatment in patient with tolosa hunt syndrome, over 75% of patients showing rapid improvement. Corticosteroid given the advantages of being inexpensive, relatively effective, readily available, and providing rapid onset of clinical improvement of signs and symptoms. But there is no specific data to give recommendations about dose, duration, or route of administration. Spontaneous remission of symptoms is known to occur. Although orbital pain drastically improves with steroid treatment, there is no evidence to suggest cranial nerve palsies improve faster with it. Treatment for Tolosa Hunt syndrome involves initial high-dose therapy for few days. Imaging studies like MRI can be repeated for follow-up, but usually, lag behind a few weeks as compared to symptomatic improvement[20].

Corticosteroid treatment includes prednisone, methylprednisolone, and dexamethasone orally or intravenously. The usage of methylprednisolone IV, 250 mg every 6 hours for 3 days. For adults, initial steroid treatment doses are typically 1 mg/kg of prednisone. High-dose oral corticosteroids of 1.0 to 1.5 mg/kg/day are the preferred protocol for children. The reported total dose ranges from 60 to 100 mg daily for 1 to 2 weeks with a 5- to 6-week taper[15].

Long-term use of corticosteroids is associated with various systemic and ocular side effects, such as immunosuppression, diabetes, hypertension, osteoporosis, secondary glaucoma, drug induce cataract and dry eye syndrome, which calls for careful monitoring and the use of steroid-sparing medications in some situations. To achieve a quick remission of inflammation, high-dose corticosteroids are frequently used, followed by a stepwise tapering regimen designed to minimize adverse effects and preserve disease control[15].

The steroids can begin tapering as soon as the clinical response is complete[18]. Despite beneficial effects, they also have side effects that depend on the dose, type of steroid and length of treatments. Many of the severe complications

occur in long-term use at doses greater than 20 mg of prednisone per day for three weeks or more causes tertiary adrenal insufficiency due to the HPA-axis (Hypothalamic Pituitary Adrenal Axis) suppression. Tapering dosage is needed when administering in longterm corticosteroids. The following is a reference for reducing the dose of corticosteroids in long-term use[20].

Table 4. Guideline Recommendation of Glucocorticoid Tapering[20].

Article	Type of Disease	Recommendation
Dasgupta et al, 2010	Giant cell arteritis (GCA) or temporal arteritis	• Continue prednisolone 40-60 mg (not < 0,75 mg/kg) for 4 weeks, then • Reduction dose by 10 mg every 2 weeks to 20 mg, then • Reduction dose by 2.5 mg every 2-4 weeks to 10 mg, then • Reduction dose by 1 mg every 1-2 months, provided no relapse occurs
Robinson, 2014	Immune-related toxicity	• Initiate corticosteroid taper over 3-6 weeks • Reduction prednisolone dose by 10 mg every three days (as toxicity allows) until 10mg/day, then • reduce by 5 mg every five days, then stop
Furst & Saag, 2018	Rheumatoid	• Initial dose above 40 mg/day of prednisone, reduction 5 to 10 mg/day every one to two weeks. • If prednisone dose 20-40 mg/day, reduction 5 mg/day every one to two weeks. • If prednisone dose 10-20 mg/day, reduction 2,5 mg/day every two to three weeks. • If prednisone dose 5-10 mg/day, reduction 1 mg/day every two to four weeks.

Steroid-sparing medications may be considered as an additional or alternative therapy in situations of corticosteroid dependency, intolerance, or refractoriness to preserve disease management and reduce corticosteroid-related comorbidities. Immunomodulatory drugs such as methotrexate, azathioprine, mycophenolate mofetil, and cyclosporine are frequently utilized to treat orbital pseudotumor. These medications facilitate corticosteroid tapering and avoid disease recurrence by lowering inflammatory cell infiltration inside the orbit and regulating aberrant immune responses[18].

While OID is generally benign, its course can be clinically fulminant with vision loss and severe oculomotor dysfunction. In cases where medical therapy is ineffective, such as refractory illness, compressive optic neuropathy, or severe vision-threatening consequences, surgical measures may be necessary. Orbital decompression, achieved through either transcutaneous or endoscopic methods, aims to reduce orbital pressure and alleviate optic nerve compression, thereby preserving visual function and relieving symptoms such as proptosis or diplopia[7].

Surgical excision or debulking of ocular lesions may also reduce tumor burden and relieve compressive symptoms in situations of mass effect or localized inflammation. Patients with optic nerve involvement may require admission for aggressive intravenous steroid therapy to prevent long-term visual impairment. Thus, prompt diagnosis and treatment are imperative[17].

While orbital pseudotumor resolves without treatment in some patients, steroids are the mainstay

of treatment. Over 75% of patients show improvement within 24 to 48 hours of steroid treatment. As mentioned previously, the reported cure rate is 37%. Recurrence is approximately 52%, even with corticosteroid treatment. Incomplete resolution of inflammation, corticosteroid dependency, insufficient tapering of immunosuppressive medication, and underlying autoimmune diseases are factors linked to an increased chance of recurrence. Repeated orbital pseudotumor episodes may exacerbate visual results and functional impairment by causing increasing orbital fibrosis, tissue scarring, and cumulative damage to orbital components[18].

Currently the main focus for this case is how to prevent recurrence. The patient has an autoimmune disease (psoriatic arthritis) so there is potential that if there is no good treatment synergy, the psoriatic arthritis could trigger inflammation in the patient's eyes. Several studies have shown that decreased vision due to recurrence of OID has the potential to increase the risk of permanent vision loss. This will have an impact on the patient's quality of life.

The patient is entering productive age (22 years old), and the patient works to help his parents. If a relapse occurs, the patient cannot work, and requires parental

assistance in his daily life so that the family does not get any income. This is in accordance with research conducted by Cockerham. The patient's quality of life will be greatly influenced by chronic OID[19].

Patients with complaints that have recurred 3 times since 1 year. The loss of visual acuity in these cases
is unpredictable and can be permanent. Patients with complexity and high risk of recurrence require special attention. Patient care will be more challenging in the presence of an autoimmune disease as in this case. Giving corticosteroids over a long period of time will cause new problems. Combination therapy can provide benefits to minimize the side effects of long-term corticosteroid use. Immunomodulatory drugs such as methotrexate and biologic agents such as Etanercept have been proven to reduce the effects of recurrence[14]. The patient's understanding of the disease they are suffering from and compliance with treatment will greatly influence the prognosis of the disease. Patients should be aware of clinical signs of relapse periods such as decreased vision, pain in the eyes, or even swelling of the eyes. This will really help the opthalmologist in treatment.

CONCLUSION

Orbital inflammatory disease is a diagnosis of exclusion, with the differential diagnosis including infection, systemic inflammatory conditions, and neoplasms, among other conditions. Tolosa–Hunt syndrome is a rare clinical condition caused by idiopathic granulomatous inflammation in the region of cavernous sinus and/or superior orbital fissure. Tolosa–Hunt syndrome presents with relapsing/remitting partial/complete ophthalmoplegia, visual loss, unilateral headache, and paralysis of cranial nerves III, IV, V1, and VI. Neuroimaging may show an enhancing mass within the cavernous sinus. Tolosa Hunt syndrome good responds with corticosteroids, but not prevent recurrence.

The loss of visual acuity in these cases is unpredictable and can be permanent. Patients with complexity and high risk of recurrence require special attention. Patient care will be more challenging in the presence of an autoimmune disease. Giving corticosteroids over a long period of time will cause new problems. Combination therapy of immunomodulatory drugs such as methotrexate and biologic agents such as Etanercept has been proven to reduce the effects of recurrence. Multidisciplinary synergy and good patient education are needed to improve the quality of life of patients with Tolosa Hunt syndrome.

DECLARATIONS

Data availability

The corresponding author (asmaa.reda@dentistry.cu.edu.e.g.)can provide access to the datasets utilized or examined in this study upon a reasonable request.

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare no conflict of interest.

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