Brief Communication

A Case of a Non-Specific Orbital Inflammation

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Abstract

Non-specific orbital inflammation (NSOI) is a complex diagnosis marked by inflammation of ocular tissues without an identifiable aetiology. This case report describes a 37-year-old female with recurrent left-sided NSOI. Her early presentation included pain, proptosis, chemosis, and limited ocular movements. Imaging showed oedema of the left optic nerve, hypertrophy of the extraocular muscles, enlargement of the lacrimal gland, and periorbital soft tissue oedema. Although she initially exhibited a positive response to high-dose intravenous corticosteroids, her symptoms reemerged during the tapering process, requiring extended oral corticosteroid treatment. This case highlights the significance of a multidisciplinary approach, personalized treatment, and additional study into the fundamental processes and optimal management of this condition.

Keywords: Non-specific orbital inflammation, Steroid, Orbit, Proptosis.

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INTRODUCTION

NSOI, also called idiopathic orbital inflammation, is a rare orbital inflammatory disorder that has no known cause. It can have a focal or diffuse effect on a variety of orbital tissues, including adipose tissue, lacrimal glands, and extraocular muscles.[1] Periocular discomfort, orbital oedema, proptosis, and restricted extraocular movement are among the symptoms that clinically differentiate NSOI.[2] When NSOI is diagnosed, other infectious, neoplastic, and specific inflammatory disorders must be ruled out.

The primary treatment for NSOI is systemic corticosteroids, which often produce a rapid symptom relief.[1]A significant percentage of patients experience recurrence, and long-term steroid use can lead to systemic side effects and a low cure rate; some studies indicate a 52% recurrence rate.[3] This emphasizes how challenging it is to treat steroid-dependent or recurring cases of NSOI.

This case report details a complicated case of steroid-dependent non-specific orbital inflammation, focusing on the patient's management options, diagnostic factors, and clinical pattern. The patient had recurrent symptoms that required several high-dose steroid treatments.

Case Presentation

A 37-year-old female presented to emergency department with stabbing pain in her left eye, radiating to her head. The eye was swollen and protruded for the last two days, followed by visual impairment. The patient also experienced nausea and vomiting. She had similar symptoms a month earlier, was hospitalized and received intravenous methylprednisolone 250mg four times daily for three days. Two weeks prior, the patient underwent a CT orbit and brain with contrast, which indicated oedema in the left optic nerve with a sagittal diameter of 0.5 cm. The scan also indicated heterogeneous echo-parenchymal intensity and hypertrophy of the left medial rectus muscle. The largest muscle diameter measured 3.7 × 0.9 cm. No cerebral abnormalities or masses were present. The CT scan indicated a potential case of left retrobulbar

neuritis, prompting a differential diagnosis of Graves' ophthalmopathy **(Figure 1).**

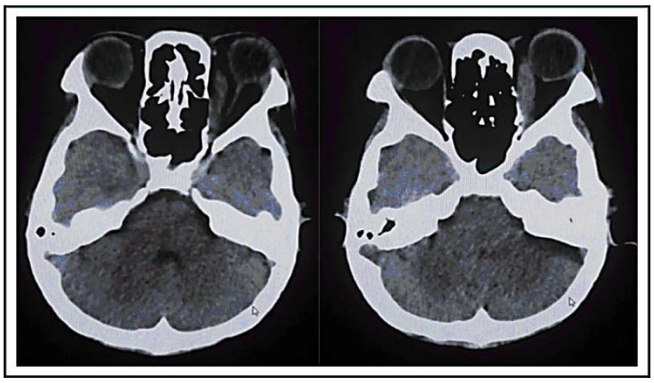

Figure 1: CT scan with left proptosis.

TSH, total T3, and free T4 levels were within normal ranges. The patient had experienced involuntary eye movements since childhood but have not been accompanied by any symptoms other than blurred vision and felt better when wearing glasses. The patient was married and not pregnant. There is no history of similar diseases in the family.

The visual acuity of the right eye was 6/60, which improved to 6/40 with a pinhole, however the visual acuity of the left eye remained at 1/60 with no improvement on pinhole test. Intraocular pressure was 20 mmHg and 29 mmHg, respectively, during the initial emergency department presentation and throughout her hospitalization.

Both eyes showed manifest spontaneous nystagmus. The right eye was normal, whereas the left eye showed proptosis, chemosis, and restricted extraocular movement in all directions. A positive RAPD was observed on the left eye. Fundus was normal. All other systems were unremarkable.

There was leucocytosis of 20,290 uL, an increase in neutrophils of 76%, and a decrease in lymphocytes of 18%. MRI and MRA revealed left proptosis. The left optic nerve was dilated, extraocular muscles appeared thickened and enhanced with contrast, the lacrimal gland was enlarged, and soft tissue oedema was noted in the periorbital tissues. The impression was NSOI. Other findings suggested chronic cerebral small vessel ischemic disease (Fazekas grade I) and differential diagnoses of multiple sclerosis. MR Angiography revealed left A1 ACA segment stenosis, right and left P1 PCA segment stenosis, maxillary and ethmoidal sinusitis, and left mastoiditis **(Figure 2).**

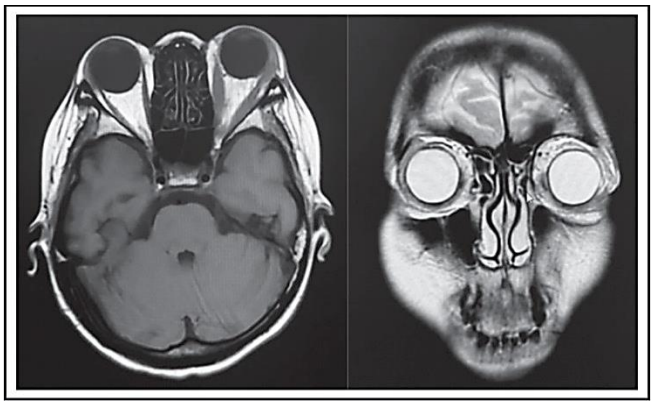

Figure 2: MRI and MRA with contrast.

The patient was hospitalized and received intravenous methylprednisolone, Metamizole injection 500 mg three times daily, Ranitidine injection twice daily, acetazolamide orally 250 mg four times daily, potassium chloride orally 600 mg once daily, timolol 0.5% eye drops one drop twice daily in the left eye for controlling high IOP, and gentamicin eye ointment for lubrication. After three days, the patient was shifted to oral methylprednisolone at a dosage of 64 mg once daily but condition aggravated **(Figure 3),** prompting the re-administration of intravenous methylprednisolone at a dosage of 250 mg three times daily, followed by a tapering of 125 mg per day over three days. Subsequently dose was reduced to 62.5 mg daily for two days.

Another relapse occurred on day 10, when the dose of intravenous methylprednisolone was reduced. Leukocytes were 18,300 uL. Ceftriaxone 1 gram was added twice daily. Upon improvement intravenous methylprednisolone was replaced with oral prednisone 60 mg once a day for 4 consecutive days. The patient had multiple relapses after every attempt to reduce the dose of steroids or to shift to oral prednisolone (Figure 4).

During the second to third week, the patient improved, and drugs were reduced **(Figure 4).** We plan to continue evaluating the patient weekly and gradually tapering down the steroid dose.

DISCUSSION

The 37-year-old female patient aligns with the common demographic for NSOI, which affects women accounting for approximately 68.1% of cases in some studies. NSOI can occur across a wide age range and in both sexes. In this case, NSOI is

Figure 3: Evaluation of the patient's left eye on day 0 of treatment, day 3, day 4, and 3 days after discharge from the hospital (third week).

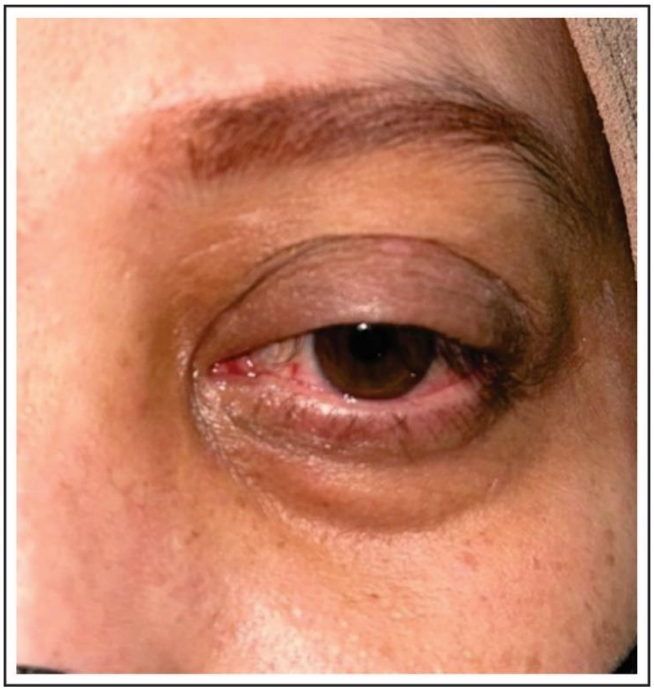

Figure 4: Three weeks after a steroid tapering dose.

characterized by a unilateral presentation that affects the left eye, consistent with the more common presentation observed in adult cases.[2] Bilateral involvement can occur, particularly in paediatric patients, with an incidence ranging from 8% to 20%.[1]

Our patient exhibited intense pain, pronounced proptosis, oedema, visual impairment, and limited extraocular movements, aligning with the standard clinical presentations reported in the literature.

The observed leucocytosis with neutrophilia and lymphopenia is a common laboratory finding in NSOI, reflecting the underlying inflammatory process.[4] However, it is non-specific and does not differentiate NSOI from other inflammatory conditions. The initial CT scan findings of left optic nerve oedema and medial rectus muscle thickening, along with subsequent MRI findings of thickened extraocular muscles, enlarged lacrimal gland, and periorbital soft tissue oedema, were consistent with NSOI. The MRI findings of sinusitis and mastoiditis raise the possibility of an infectious component, which should be considered in the differential diagnosis.

NSOI is a diagnosis of exclusion.[3,5] The normal thyroid function tests exclude Graves' ophthalmopathy. The other differentials include neoplastic, inflammatory, autoimmune, and infectious conditions.[2] The incidental findings of chronic cerebral small vessel ischemic disease and possible multiple sclerosis on MRI warrant further investigation, although they are unlikely related to the NSOI.

Systemic corticosteroids are the mainstay of NSOI treatment, often leading to a rapid and dramatic response, typically within 48 hours of administration.[3] High-dose corticosteroids is gradually tapered off over months. Quick tapering may result in a "relapse" of orbital inflammation.[6]

In a similar case of NSOI, clinical symptoms improved after 48 hours of intravenous methylprednisolone but a relapse occurred during the process of tapering down the steroid dose.[7] However, there are cases that improved after 6 weeks of hospitalization with a slowly tapered dose of prednisone, and complete resolution after 4 weeks of discharge.[8]

There should be a delicate balance between inflammation control and steroid-related side effects

which necessitates careful corticosteroid titration and the use of steroid-sparing agents in patients with recurrent disease.

Steroid-sparing agents can be used when corticosteroids alone are not enough or when long-term steroid use is undesirable because of possible side effects. Various immunosuppressants and other drugs customized to each patient's requirements and reaction are among these agents. In certain situations, especially when dealing with localized or resistant disease, radiotherapy and surgery may also be taken into consideration.[9]

The addition of antibiotics in this case was based on concerns about potential contributing infections. Supported by the patient's laboratory results showing leucocytosis. The importance of a multidisciplinary approach involving ophthalmologists, radiologists, and other specialists in the diagnosis and treatment of NSOI is demonstrated by this example. This case emphasizes the necessity of a comprehensive evaluation, customized treatment plans, and ongoing monitoring. The optimal strategies for treating recurrent NSOI while reducing the long-term morbidity linked to the illness and its treatment require further investigation.

CONCLUSION

This case report discusses the challenge to treat recurrent NSOI. The difficulties in achieving sustained remission are highlighted by the patient's initial presentation, imaging results, and response to high-dose corticosteroids, which was followed by relapse upon tapering. In-depth evaluations, customized treatment regimens, close recurrence monitoring, and additional research into steroid-sparing treatments and the underlying mechanisms of NSOI are all necessary in this case.

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Conflict of Interest: Authors declared no conflict of interest.

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Authors Designation and Contribution

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Nur Khoma Fatmawati; Consultant Ophthalmologist: *Concepts, Design, Data Acquisition, Manuscript Editing, Manuscript Review.*