ORIGINAL ARTICLE

J Rhinol 2024;31(2):78-85 | https://doi.org/10.18787/jr.2024.00012 pISSN 1229-1498 / eISSN 2384-4361
www.j-rhinology.org

Salvaging Vision: A Study of Non-Traumatic Optic Neuropathies

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Background and Objectives: Various ear, nose, and throat (ENT) conditions can result in vision loss. The purpose of this study is to identify the etiologies, presentations, and radiological findings associated with impaired vision in the context of ENT. Additionally, this article discusses management protocols, including optic nerve decompression and orbital decompression.

Methods: In a retrospective study, we examined the period from 2016 to 2022 at a tertiary care hospital in Mumbai, India. The analysis included 11 patients who presented with progressive diminution of vision. All patients received a regimen of broad-spectrum intravenous antibiotics and high-dose intravenous steroids. This was followed by either endoscopic optic nerve decompression or orbital decompression. Subsequent improvements in vision were documented, and any complications were evaluated.

Results: A total of 11 patients were treated with medical management followed by successful surgery, with 10 patients demonstrating significant vision improvement.

Conclusion: Identifying the etiology of vision loss and managing the condition can present challenges for otorhinolaryngologists. A thorough grasp of the underlying pathophysiology, combined with active surveillance of clinical and radiological indicators, can enable these clinicians to achieve effective and rewarding outcomes.

Keywords: Optic nerve decompression; Non-traumatic; Sinusitis; Mucocele; Fibrous dysplasia.

INTRODUCTION

Vision impairment rarely presents directly to otorhinolaryngologists. In these unusual cases, awareness of etiological causes is helpful for timely intervention, as the ear, nose, and throat (ENT) surgeon can be instrumental in reversing vision loss. However, these situations introduce considerable challenges due to the broad range of potential causes. Additionally, ENT centers typically possess limited data on vision loss due to its rare presentation. The most common cause of sudden vision impairment encountered by otorhinolaryngologists is traumatic optic neuropathy (TON), which usually results from trauma to the intracanalicular segment of the optic nerve [1]. Our study aims to investigate other causes of progressive vision loss, which are currently less well understood. Compressive optic neuropathy is caused by intrinsic or extrinsic compression anywhere along the pathway of the optic nerve [2] and can result from various pathologies in the posterior paranasal sinuses, especially the sphenoid and ethmoid sinuses [3]. Treatment protocols remain a topic of debate, with the main approaches being expectant versus surgical management. The purpose of this study is to identify the causes of reduced vision in patients presenting to an otorhinolaryngologist and to discuss their treatment outcomes.

Received: April 24, 2024 **Revised:** June 6, 2024
Accepted: June 7, 2024

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METHODS

A retrospective study examined a total of 11 patients who presented with progressive optic neuropathy to a tertiary care center between 2016 and 2022. The data were analyzed based on visual acuity, etiology, response to medical management, and significant intraoperative findings. The study included patients with reduced vision and an identifiable cause of optic nerve damage in the intracanalicular portion, who presented with a progressive course of the disease. Patients with involvement of the intracranial course of the optic nerve, those with complete transection of the nerve, and those with a pale optic disc on fundoscopy were excluded. Additionally, patients

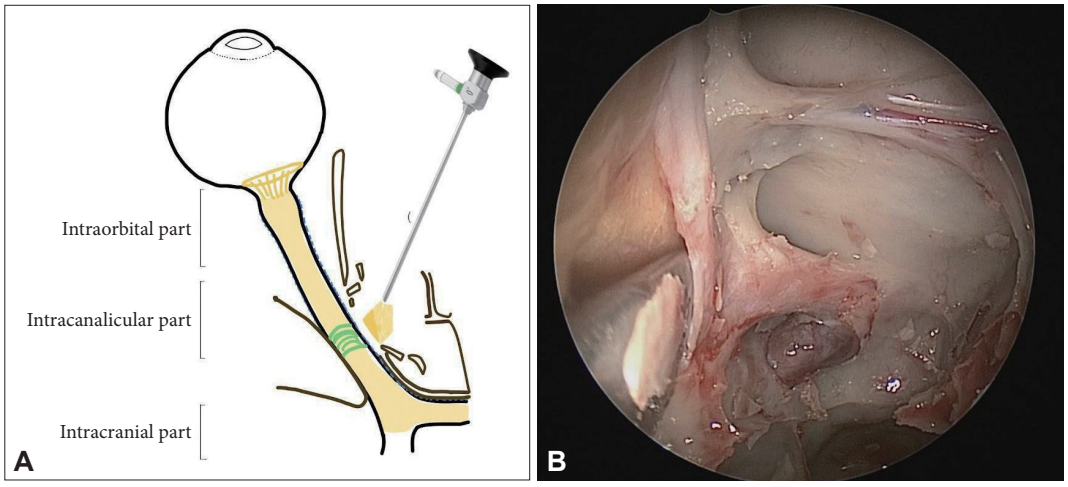

Fig. 1. Endoscopic anatomy of optic nerve. A: Schematic representation of intracanalicular part of optic nerve that can be assessed by transnasal endoscopy. B: Endoscopic anatomical relations of the right optic nerve showing carotid artery, opticocarotid recess and posterior ethmoidal artery in the Onodi cell in cadaveric dissection

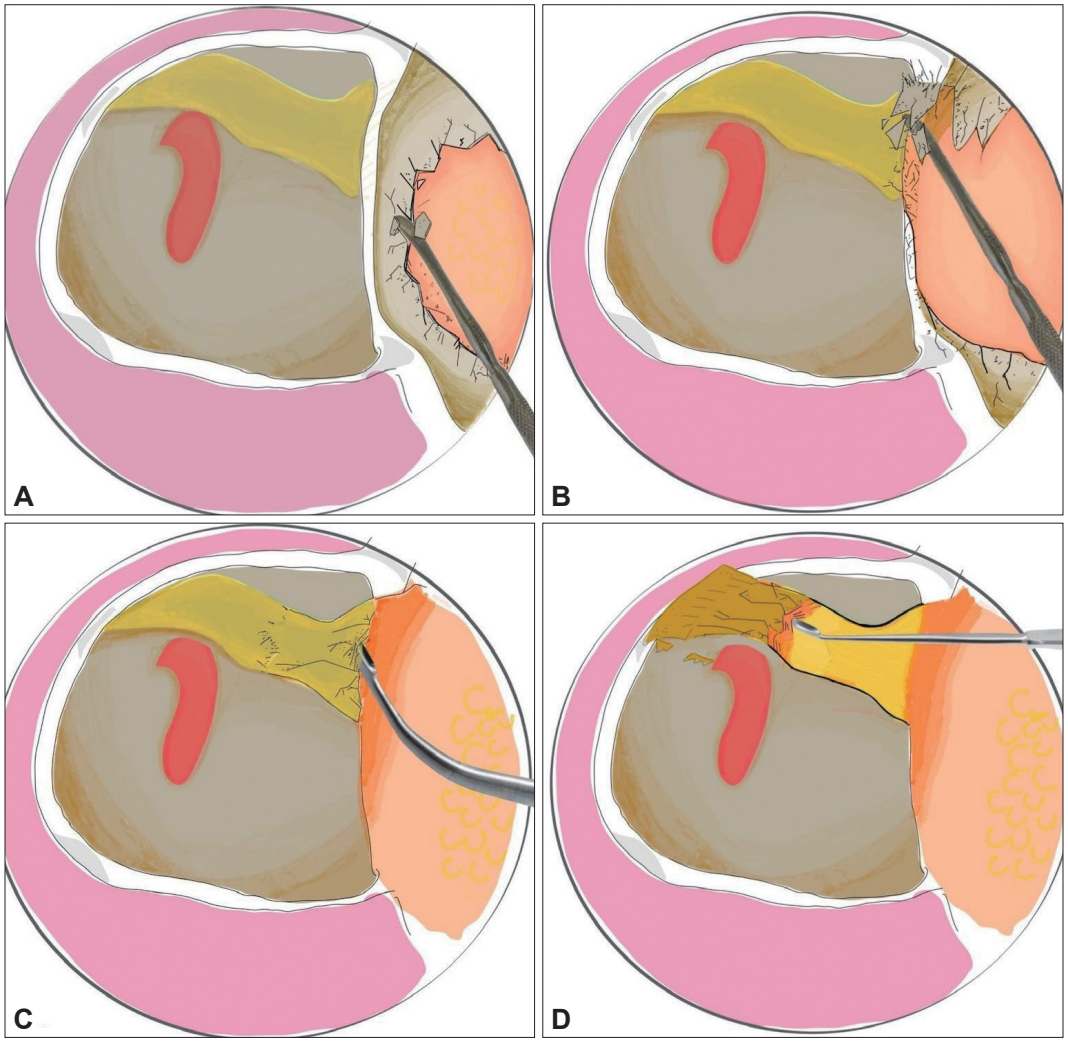

Fig. 2. Schematic diagram showing the steps of left trans-nasal endoscopic optic nerve decompression. A and B: The relationship of the left optic nerve with lamina papyracea and carotid. Removal of the lamina papyracea with a Freer's elevator. C and D: Removal of the optic canal and annulus of Zinn with curette to decompress the optic nerve.

presenting with sudden onset of vision loss following head trauma, indicative of TON, were not included in the research. This retrospective study analyzed the management of patients who previously received treatment at our center, and a waiver of consent was obtained from the institutional ethics committee (ethics approval: IEC(III)/OUT/320/2023).

The diagnosis was established through clinical evaluation and radiological assessment. This was corroborated by a range of clinical tests, including visual acuity testing, assessment of extraocular muscle movements, pupillary evaluation, fundoscopy, and color vision testing. All patients underwent computed tomography of the paranasal sinuses and brain with 1-mm orbital slices, along with magnetic resonance imaging (MRI) of the brain and orbit.

Each patient selected for the study initially received medical management, which consisted of intravenous steroids (1 g of methylprednisolone daily for 3 days), except when contraindicated due to conditions such as severely altered blood sugar levels or active foci of infection. Intravenous antibiotics were administered in cases with infectious etiologies. Patients who experienced an improvement in visual acuity were transitioned to a tapering regimen of oral steroids and monitored. Individuals whose vision did not improve or worsened, as well as those with bony fragments compressing the nerve, underwent transnasal endoscopic surgery under general anesthesia. Only one patient, who exhibited optic nerve perineuritis, responded favorably to high-dose intravenous steroids. The remaining 10 patients required surgical intervention using an endoscopic transnasal approach (Figs. 1 and 2). The surgical procedure for these patients involved comprehensive decompression of the optic nerve's intracanalicular segment, extending from the lamina papyracea along its trajectory into the lateral wall or roof of the sphenoid sinus, or in some cases the Onodi cell. One patient with an orbital abscess also underwent orbital decompression, which included drainage of the pus from the orbit through removal of the lamina papyracea and periorbital puncture. Preoperative scrutiny of radiological investigations provided essential guidance for surgery. Recognizing anatomical variations in the course of the optic nerve is crucial during preoperative assessment. The widely accepted DeLano classification [4], which describes the relationship of the optic nerve to the posterior paranasal sinuses, offers valuable insight into the surgical approach that should be adopted.

Postoperatively, patients were monitored for signs of complications. Visual acuity was documented on postoperative days 1 and 3, as well as after discharge. A postoperative fundoscopic examination was performed. When not contraindicated, patients were prescribed a tapering dose of oral steroids upon discharge.

Table 1. Vision assessment after treatment (n=11)

Assessment	Number of patients
Significant improvement	10
No improvement	1

RESULTS

The results were tabulated based on data regarding visual acuity at the beginning and end of treatment, radiological

Table 2. Comparison of pretreatment and posttreatment assessments (n=11)

Etiology	Age (yr)/ sex	Progression of vision loss	Duration of vision loss	Pretreatment vision	Posttreatment vision
Infectious (n=3)					
1. AFRS	40/M	Worsening	1 month	PL present	HM
2. Bacterial rhinosinusitis	55/F	Worsening	15 days	No PL	FC at 0.5 m
3. Orbital abscess	18/F	Rapidly worsening	5 days	HM	6/36
Inflammatory (n=4)					
4. Sphenoid mucocele	62/M	Worsening	1 month	PL present	FC at 0.5 m
5. Sphenoid mucocele	30/M	Constant	2 months	6/12	6/6
6. Posterior ethmoid mucocele	35/F	Worsening	20 days	HM	FC at 1 m
7. Perineural inflammatory lesion	68/F	Slowly progressive	3 months	No PL	FC at 0.5 m
Neoplastic (n=4)					
8. Fibrous dysplasia	25/F	Slowly progressive	25 days	PL present	6/9
9. Posterior ethmoid chondrosarcoma	28/F	Slowly progressive	2 months	6/36	6/9
10. JNAF	18/M	Sudden	1 day	No PL	6/18
11. Osteopetrosis	20/M	Slowly progressive	6 months	HM	HM

AFRS, allergic fungal rhinosinusitis; M, male; F, female; PL, perception of light; HM, hand movements; FC, finger counting; JNAF, juvenile nasopharyngeal angiofibroma

findings, and etiology-specific outcomes (Tables 1 and 2). The treatment protocol administered to each patient is detailed in Table 3.

Significant improvement from the pretreatment status was defined as an improvement in vision to at least the level of perceiving hand movements, which is functionally useful for the patient.

Of the 11 patients, an infective etiology was identified in three cases. Two patients presented with rhinosinusitis, specifically allergic fungal rhinosinusitis (AFRS) and bacterial sinusitis of the sphenoid sinus, while the third patient had developed an orbital abscess as a complication of sinusitis. Inflammatory causes were found in four patients; three of these had mucoceles in either the sphenoid (Fig. 3) or posterior ethmoid sinus, while one patient exhibited perineural inflammation along the intracanalicular segment of the optic nerve. Neoplastic etiologies leading to optic nerve compression were diagnosed in four patients, with conditions including fibrous dysplasia (Fig. 4), chondrosarcoma, and juvenile nasopharyngeal angiofibroma (JNAF). One patient had the uncommon diagnosis of marble bone disease of the sinuses, also known as osteopetrosis (Fig. 5), which necessitated bilateral optic

Table 3. Summary of treatment protocol for all patients

Etiology	Duration of vision loss	Medical management*	Surgical management
Infectious (n=3)			
1. AFRS	1 month	MPS injections×3 days and IV antibiotics	Followed by FESS with OND
2. Bacterial rhinosinusitis	15 days	IV antibiotics	Emergency bilateral FESS with OND
3. Orbital abscess	5 days	IV antibiotics	Endoscopic orbital decompression
Inflammatory (n=4)			
4. Sphenoid mucocele	1 month	MPS injections×5 days	Mucocele marsupialization with OND
5. Sphenoid mucocele	2 months	MPS injections×5 days	Mucocele marsupialization with OND
6. Posterior ethmoid mucocele	20 days	MPS injections×5 days	Mucocele marsupialization with OND
7. Perineural inflammatory lesion	3 months	MPS injections×5 days	Not done
Neoplastic (n=4)			
8. Fibrous dysplasia	25 days	MPS injections×5 days	Endoscopic OND
9. Posterior ethmoid chondrosarcoma	2 months	MPS injections×5 days	Endoscopic mass excision with OND
10. JNAF	1 day	Preoperative embolization	Emergency mass excision with OND
11. Osteopetrosis	6 months	- Preoperative and postoperative corticosteroids - Postoperative calcium and vitamin D3 supplementation	Endoscopic OND

*medical management was initiated immediately upon presentation of the patient to our OPD and was followed by surgical management. AFRS, allergic fungal rhinosinusitis; IV, intravenous; MPS, methylprednisolone; FESS, functional endoscopic sinus surgery; OND, optic nerve decompression; JNAF, juvenile nasopharyngeal angiofibroma; OPD, outpatient department

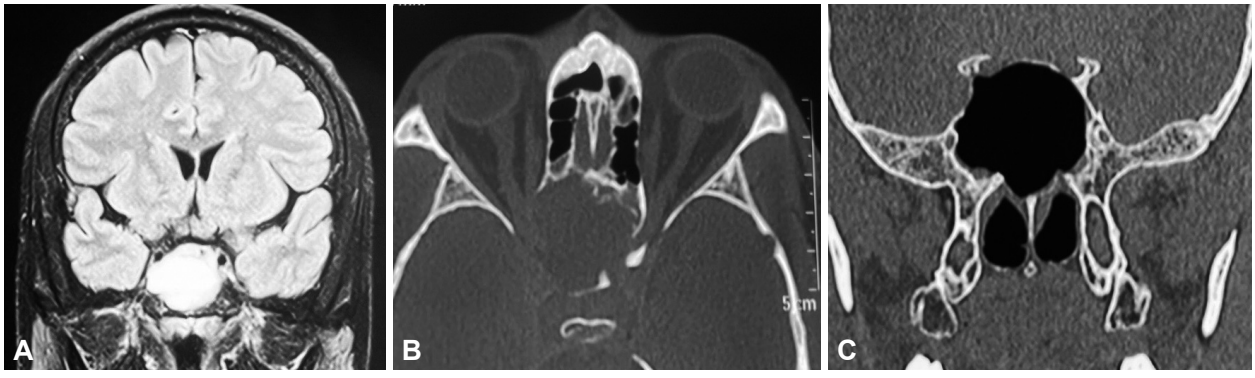

Fig. 3. Contrast-enhanced computed tomography of the paranasal sinuses in a 62-year-old male patient presenting with headache and diminution of vision. A and B: Imaging revealed a sphenoid mucocele causing compression of the optic nerves. C: The patient underwent endoscopic marsupialization of the sphenoid mucocele and a postoperative scan for the same was done.

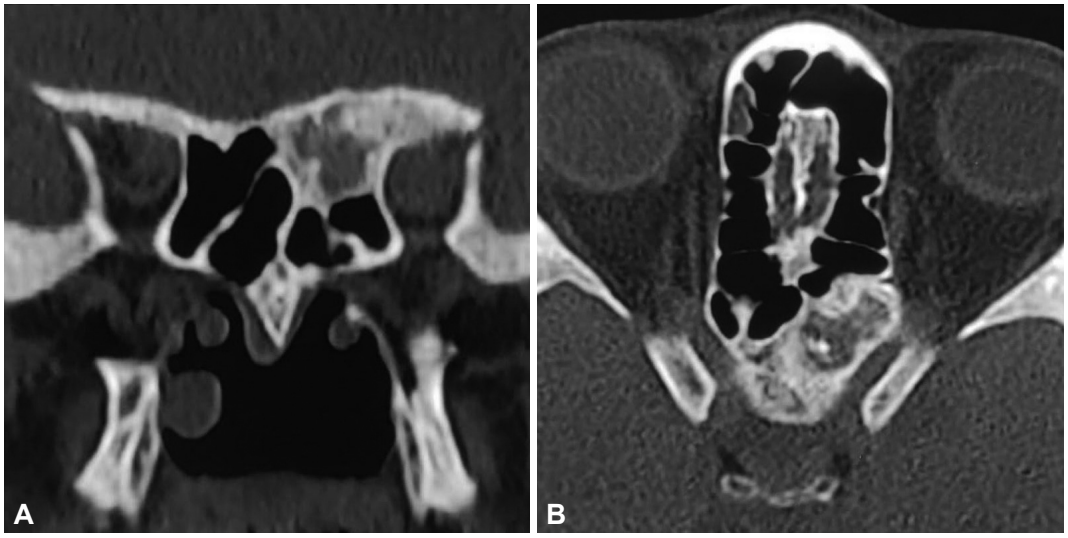

Fig. 4. Coronal (A) and axial (B) computed tomography images of the paranasal sinuses showing ground glass opacity in the Onodi cell. A 25-year-old female patient presented to our outpatient department with left-sided diminution of vision and diplopia and left ophthalmoplegia of insidious onset for 15 days. On starting intravenous steroids, the ophthalmoplegia had improved. CT scan of paranasal sinuses revealed ground glass opacification of the Onodi cell suggestive of fibrous dysplasia with involvement of optic nerve on the left. Fundoscopy was done to check the viability of the optic disc plan endoscopic decompression. The patient required drilling of the dense fibrous dysplasia lesion in order to release the compression from the left optic nerve.

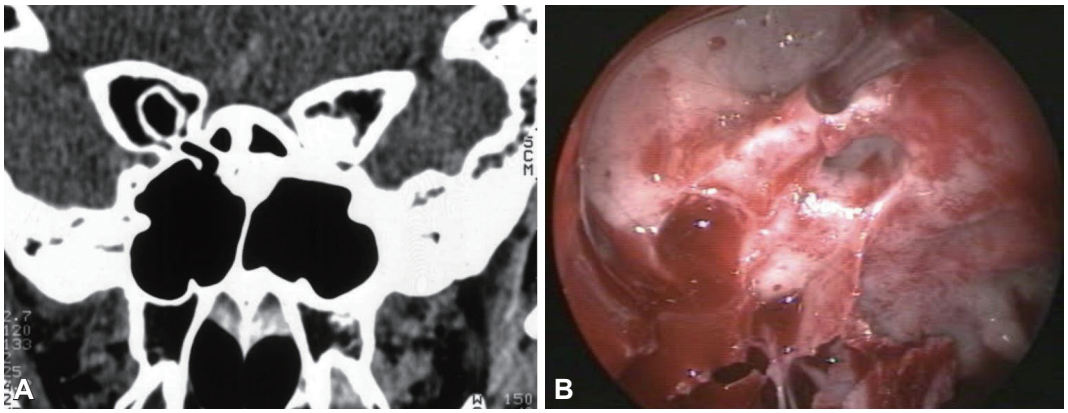

Fig. 5. A rare case of osteopetrosis was identified in our study population (20-year-old male). The patient had presented with a recent onset, progressive diminution of vision on the left side. A: Axial CT scan revealed dense bone around the optic nerve causing optic canal stenosis. B: Endoscopic decompression of the left optic nerve was achievable only with the use of micro-drill. On follow-up, the progression of vision deterioration had halted and the patient was taken for endoscopic decompression of optic nerve on the right side after 3 months.

nerve decompression.

Posttreatment, significant improvement in vision was documented if the individual could appreciate hand movements, a clinically useful outcome observed in 10 of the patients. However, one case of osteopetrosis requiring bilateral optic nerve decompression showed no improvement during the postoperative period.

DISCUSSION

Blindness has been linked to a variety of ENT etiologies, which range from mild compression of the optic nerve by a tumor or pus to complete transection due to severe head trauma. Depending on the underlying cause, optic neuropathy in ENT cases can be classified into several categories: traumatic, neoplastic, inflammatory, infectious, and genetic.

Traumatic impingement of the optic nerve is the most common cause of vision loss necessitating decompression. Initially, this condition was thought to present secondary to vascular compromise due to external compression, but it is now understood to result from conduction block and focal demyelination caused by manual compression. In instances of direct TON, the nerve is either lacerated by bone fragments or completely disrupted. This results in severe visual loss and a lower likelihood of recovery compared to indirect damage. Indirect injuries arise from blunt trauma in which force is

transmitted through the oculofacial soft tissues and skeleton to the nerve [5], yet the integrity of the nerve remains intact. A systematic review article by Karimi et al. [6] estimated the incidence of TON after trauma to be approximately 2.5%, with indirect TON being more prevalent. Overall, factors associated with poor prognosis of endoscopic optic nerve decompression include total blindness at presentation, delayed presentation, and lack of improvement with steroid treatment [7].

In this study, we included all nontraumatic causes of vision loss that fell within the scope of otorhinolaryngology.

Infectious diseases of the paranasal sinuses can cause vision to deteriorate, potentially due to acute optic neuropathy with painful eye movements or slowly progressive neuropathy resulting from compression. Before the advent of antibiotics, sphenoethmoidal sinusitis was the primary cause of optic neuropathy and subsequent blindness [8]. Patients with orbital complications of sinusitis, such as those classified within Chandler's groups 2 to 4 [9], can experience direct infection of the perineural sheath or suffer from infectious processes that cause compressive effects, vasculitis, or thrombosis. Our study included two patients with sinusitis who developed a diminution of vision due to intraorbital complications. Another patient exhibited a gradually progressive deterioration of vision, which, upon endoscopic surgery, was found to be caused by AFRS with fungal concretions compressing the dehiscent optic nerve in the sphenoid sinus. In a study conducted by Kim et al. [10], chronic inflammation of the sphenoid sinus was shown to reduce the thickness of the optic nerve. Furthermore, a positive correlation was noted between optic nerve changes and the inflammation of the posterior ethmoid and sphenoid sinuses. The presence of a highly pneumatized posterior ethmoid sinus (Onodi cell) increases the risk of optic nerve damage in cases of chronic sinusitis.

Inflammatory conditions are associated with an imbalance between various interleukins and interferons, which can cause direct nerve injury or lead to sinus outflow obstruction. Mucoceles are epithelium-lined, mucous-filled cysts in the sinuses that exhibit a multifactorial etiology, including allergy, trauma, or iatrogenic causes. Immunologically, mucoceles are associated with elevated expression of interleukin (IL)-12, IL-2, and interferon-gamma, which can cause the inflammation to spread to the optic nerve sheath [11]. Additionally, compressive optic neuropathy can occur due to the local destructive effects of mucocele expansion. Mucoceles in the frontoethmoidal region typically exert pressure on the ipsilateral orbit, resulting in proptosis, diplopia, and potential periorbital swelling. Conversely, mucoceles located in the posterior ethmoid and sphenoid regions tend to compress the optic canal, leading to visual symptoms [12]. We observed a patient who developed a gradual and progressive loss of vision due to a sphenoid sinus mucocele (Fig. 3). Urgent endoscopic clearance was performed, and the patient's visual acuity improved from no perception of light to near-normal vision postoperatively. Mucoceles causing optic nerve compression are rare, and a high degree of clinical suspicion, supported by appropriate radiology, is necessary to establish the diagnosis. However, timely management can lead to gratifying outcomes.

We encountered one patient with optic perineuritis (OPN), a rare inflammatory condition of the orbit that causes marked thickening of the nerve sheath due to nonspecific fibrosis. The patient presented with a complete loss of light perception and eye pain that worsened with eye movements. The diagnosis was confirmed by MRI, which displayed characteristic contrast enhancement of the optic nerve sheath in the intracanalicular portion, sparing the optic nerve itself and creating a "tram track" appearance in the axial view. The patient was treated with high-dose intravenous steroids, with stark improvements in vision by the end of the treatment course. Subsequently, the patient was transitioned to a tapering dose of oral steroids, after which they were able to count fingers at half a meter. According to a systematic review using Cochrane methodology by Gupta et al. [13], OPN generally has a favorable prognosis with oral steroid treatment. However, the steroids must be gradually tapered to prevent relapse.

Neoplastic lesions can lead to compression of the optic nerve by inducing a hyperostotic reaction, which exerts a mass effect on the nerve. Fibrous dysplasia is a benign fibro-osseous lesion that can be either monoostotic or polyostotic, depending on whether the disease crosses the joint line. It is characterized by defective osteoblastic differentiation and maturation, ultimately leading to the replacement of normal cancellous bone with abnormal fibrous tissue. At our center, we encountered a 25-year-old female patient who presented with a 1-month history of progressively diminishing vision and reduced eye movements. This patient was diagnosed with fibrous dysplasia based on radiological findings and had developed orbital apex syndrome due to narrowing of the optic canal (Fig. 4). Sinonasal masses, such as JNAF and ethmoid chondrosarcomas, can often lead to diminished vision, as observed in our study. This may be due to the destruction of the bony canal or the presence of an intratumor bleed that compresses the dehiscent optic nerve. In such cases, it is crucial to radiologically determine the location of the optic nerve in relation to the sphenoid sinus. In a study by DeLano et al. [4], bony dehiscence over the optic nerve was estimated to be present in 24% of cases. In the present study, two patients had dehiscent optic nerves, and one developed vision impairment due to compression by a lesion in the sphenoid sinus. Chondrosarcomas are malignant but slow-growing cartilaginous tumors that infrequently occur in the sinonasal region. The

patient in this study with a pathologically confirmed chondrosarcoma presented postoperatively with vision of 6/36. Imaging findings showed typical features of chondrosarcoma, such as increased intensity on T2-weighted images and peripheral nodular enhancement on T1-weighted images. We employed a multimodal therapy approach for this patient, in which chemotherapy and radiotherapy were administered after complete endoscopic resection. Subsequently, the patient's vision improved to 6/9 at follow-up. In cases of sinonasal malignancies, early ENT surgical intervention is critical not only for preserving vision but also for preventing mortality due to the tumor. Compressive neoplastic optic neuropathies are among the most treatable forms of optic nerve dysfunction [14], often resulting in recovery following decompression and tumor excision, as demonstrated in our case.

Genetic disorders such as osteopetrosis, characterized by the abnormal and excessively dense growth of bones, can occasionally affect the paranasal sinuses. This condition can lead to the narrowing of the optic canal, resulting in progressive vision loss. In this study, a patient with this complication underwent bilateral optic nerve decompression. Although the patient's vision did not improve after surgery, further deterioration was prevented (Fig. 5). Postoperatively, the patient received calcium and vitamin D supplements to help slow the progression of the disease. In addition to these treatments, the medical management of the malignant, autosomal recessive form of osteopetrosis may include bone marrow transplantation with hematopoietic stem cells or injections of interferon-gamma 1b [15]. Furthermore, high doses of calcitriol are used to promote osteoclast differentiation and slow disease progression.

In our study, 10 patients underwent endoscopic surgery following a high-dose course of intravenous steroids, except when steroids were contraindicated. Only one patient, who had fibrous dysplasia, experienced an intraoperative complication—a cavernous bleed—which was successfully managed. None of the patients experienced postsurgical sequelae. The endoscopic approach offers excellent cosmesis due to the absence of an external scar, provides adequate 180-degree decompression, and allows for end-on visualization of the nerve, all with a shorter operative time compared to external approaches. At our center, we do not routinely perform opening of the optic nerve sheath due to the associated risks of cerebrospinal fluid (CSF) leakage, potential meningitis, and the possibility of iatrogenic injury to the nerve fascicles or ophthalmic artery.

Contraindications for optic nerve decompression include complete disruption of the optic nerve, (as seen in some cases of traumatic head injury), total atrophy of the optic nerve, and the presence of carotid-cavernous fistula [16].

A multidisciplinary approach is essential in cases of vision loss; such collaboration should involve otolaryngologists and ophthalmologists, along with general surgeons in cases of traumatic head injury. Notably, the patient with orbital cellulitis in this study was referred to us by our ophthalmology colleagues, who had examined the scans and requested an ENT evaluation.

The role of an otorhinolaryngologist in the evaluation and management of vision loss is critical, given the proximity of the paranasal sinuses and the presence of various valveless venous communications with the orbit. A high index of suspicion, along with interdisciplinary coordination, is necessary to salvage vision and prevent morbidity. Carefully understanding the radiological data, while correlating the findings with clinical signs, can aid in securing a diagnosis. The judicious use of intravenous steroids must be employed by carefully weighing the benefits against the potential side effects of such high doses. Applying a fixed protocol of management to all cases of vision loss in ENT is impractical due to the varied etiologies; each patient requires a tailor-made clinical strategy. However, the transnasal endoscopic approach is considered the gold standard because of its various benefits, such as quick postoperative recovery, improved cosmesis, and access to the medial, inferior, and superior parts of the optic canal via the lateral wall of the sphenoid sinus. Decompression can be accomplished by removing the bony optic canal and the annulus of Zinn. Additionally, incision of the perineural sheath is an option for further decompression, reducing the risk of compartment syndrome but increasing the risk of CSF leakage. Thus, careful patient selection using simple assessment tools and the use of outcome measures based on varied presentations can serve as a guide for improved results. Timely intervention with steroids and endoscopic decompression can prevent permanent sequelae. Estimating the severe effects of impaired vision on patients, as well as the deterioration in quality of life due to the condition or treatment complications, can help refine our approach to individual patient management.

Availability of Data and Material

The datasets generated or analyzed during the study are available from the corresponding author on reasonable request.

Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

Author Contributions

Conceptualization: Hetal Marfatia. **Data curation:** Ashwathy KP, Monankita Sharma, Juilee Kamble, Anav Rattan. **Formal analysis:** Anoushka Sahai, Ashwathy KP, Monankita Sharma. **Investigation:** Juilee Kamble, Anav Rattan. **Methodology:** Anoushka Sahai, Monankita Sharma, Ashwathy KP. **Project administration:** Hetal Marfatia, Anoushka Sahai. **Resources:** Ashwathy KP, Monankita Sharma, Juilee Kamble, Anav Rattan. **Supervision:** Hetal Marfatia. **Validation:** Hetal Marfatia, Anoushka Sahai, Monankita Sharma. **Writing—original draft:** Anoushka Sahai. **Writing—review &**

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Funding Statement

None

Acknowledgments

None

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