EJCRIM
European Journal
of Case Reports
in Internal Medicine

SERRATIA MARCESCENS SINUSITIS PRESENTING AS LEFT ORBITAL APEX SYNDROME

Yusuf Kagzi[1], Murtaza Khambati[1], Varshini Thiruvadi[1], Tulika Chatterjee[2]

[1] Department of Internal Medicine, University of Illinois College of Medicine, Peoria, USA
[2] Edward Via College of Osteopathic Medicine Louisiana Campus, Monroe, USA

Corresponding author's e-mail: ykagzi@uic.edu

Received: 30/08/2025
Accepted: 01/09/2025
Published: 26/09/2025

Conflicts of Interests: The Authors declare that there are no competing interests.
Patient Consent: Written informed consent was obtained from the patient for publication of this case report and accompanying clinical details.
Acknowledgments: The authors would like to thank the medical and nursing staff involved in the care of the patient for their support and contributions.
This article is licensed under a Commons Attribution Non-Commercial 4.0 License

How to cite this article: Kagzi Y, Khambati M, Thiruvadi V, Chatterjee T. Serratia marcescens sinusitis presenting as left orbital apex syndrome. *EJCRIM* 2025;**12**:doi:10.12890/2025_005814

ABSTRACT

Background: Orbital apex syndrome (OAS) is a vision-threatening condition characterized by optic neuropathy, ophthalmoplegia, and cranial nerve dysfunction at the orbital apex. While fungal and neoplastic aetiologies are well recognized, bacterial causes are uncommon. *Serratia marcescens*, an opportunistic Gram-negative bacillus primarily associated with nosocomial infections, has rarely been implicated in sinusitis or orbital apex involvement.

Case report: A 73-year-old woman with type 2 diabetes presented with progressive unilateral headache, diplopia, and vision loss. She was empirically treated for allergic and bacterial sinusitis, including steroids, without improvement. Imaging showed left sphenoethmoid sinus opacification with orbital apex involvement, and initial endoscopy suggested fungal sinusitis. Repeat biopsy ruled out fungal disease and malignancy, while cultures identified *S. marcescens* susceptible to cephalosporins and fluoroquinolones. Antifungals were discontinued and completed levofloxacin with rapid clinical recovery. At 2-month follow-up, imaging confirmed resolution of orbital apex inflammation and sinus disease.

Conclusion: This case highlights *S. marcescens* as a rare cause of OAS in the setting of sphenoid sinusitis. Awareness of atypical bacterial pathogens, avoidance of premature steroid or antifungal therapy, and reliance on repeat endoscopy with cultures are critical for timely diagnosis and vision preservation.

KEYWORDS

Orbital apex syndrome, *Serratia marcescens*, sphenoid sinusitis

LEARNING POINTS

- In immunocompromised patients presenting with orbital apex syndrome, the differential diagnosis should be broadened to include rare Gram-negative pathogens.
- Repeat endoscopy and biopsy are essential in atypical, progressive, or refractory cases.
- *Serratia marcescens* should be recognized as a potential emerging pathogen beyond its traditional nosocomial associations.

EFIM
European Federation of Internal Medicine

Publisher
smc media srl

INTRODUCTION

Orbital apex syndrome (OAS), or Jacob's syndrome, is characterized by dysfunction of cranial nerves II, III, IV, V_1, and VI as they traverse the orbital apex. It may result from infectious, inflammatory, neoplastic, or traumatic processes at the junction of the optic canal and superior orbital fissure[1]. Typical features include vision loss, pain, ophthalmoplegia, diplopia, proptosis, mydriasis, and hypoesthesia. While fungal infections, particularly Aspergillus and Mucor species, are well recognized, bacterial causes are rare and underreported[2]. *Serratia marcescens*, a Gram-negative opportunistic bacillus usually linked to nosocomial infections, has only rarely been implicated in sinusitis or orbital apex involvement[3]. Given the diagnostic challenges and risk of rapid visual loss, early identification and targeted therapy are essential. This case contributes to the limited evidence of *S. marcescens* sinusitis as a cause of OAS.

CASE DESCRIPTION

A 73-year-old woman with a past medical history of type 2 diabetes mellitus presented with a 4-month history of constant, unilateral headaches localized to her left eye. Initially, she had no visual or neurological deficits and was treated empirically with 2-week course of prednisone 40 mg daily followed by 10-day course amoxicillin for presumed sinusitis, without improvement. Shortly after, she developed diplopia and vision loss in the left eye. She denied right-eye symptoms, systemic complaints, or substance use, and had no medication allergies. Her history included remote head trauma 3 years earlier.

On physical examination, the left eye demonstrated periorbital swelling, ptosis, reduced visual acuity in the upper left quadrant, a relative afferent pupillary defect with sluggish pupillary response, and cranial nerve III, IV, and VI palsies. There was no proptosis or loss of corneal sensation. In contrast, the right eye and the remainder of her neurological and sensorimotor exam were normal.

Laboratory studies demonstrated white blood cells 14 ×10^9/l (reference 4–12 ×10^9/l) with neutrophilic predominance along with elevated inflammatory markers (erythrocyte sedimentation rate 27 mm/h, C-reactive protein 2.4 mg/dl). A non-contrast computed tomography (CT) scan of the sinuses revealed opacification of the left posterior sphenoethmoid sinus and a soft tissue polyp in the right maxillary sinus. Brain magnetic resonance imaging (MRI) showed inflammatory changes involving the left optic nerve sheath, while orbital MRI demonstrated persistent enhancement extending from the left orbital apex through the inferior orbital fissure into the cavernous sinus, with mild extension into the left pterygopalatine fossa *(Fig. 1)*. MR venography and angiography were unremarkable, with no evidence of thrombosis or vascular stenosis.

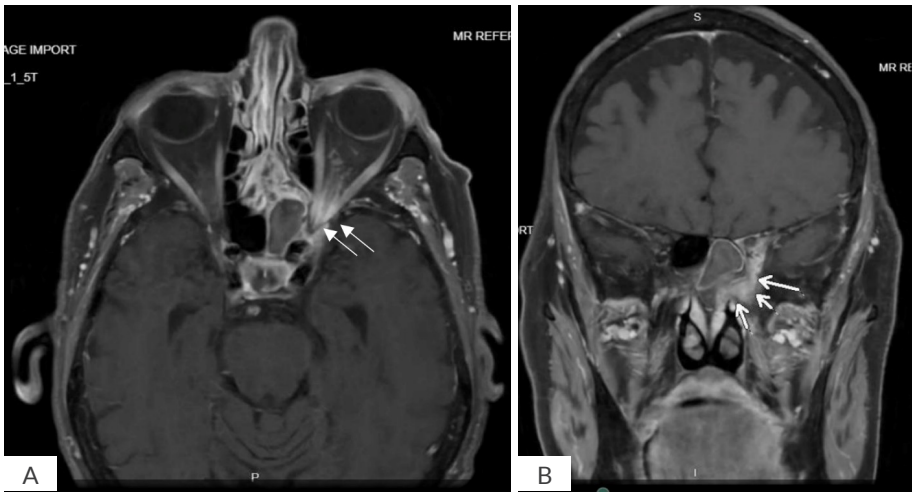


Figure 1. A) Post-contrast magnetic resonance imaging (MRI) demonstrates asymmetric enhancement at the left orbital apex with extension along left optic nerve sheath (arrows) may represent infectious aetiology related to invasion from the left sphenoid sinusitis. B) Coronal MRI of paranasal sinuses showing opacification of left sphenoid sinus (arrows).

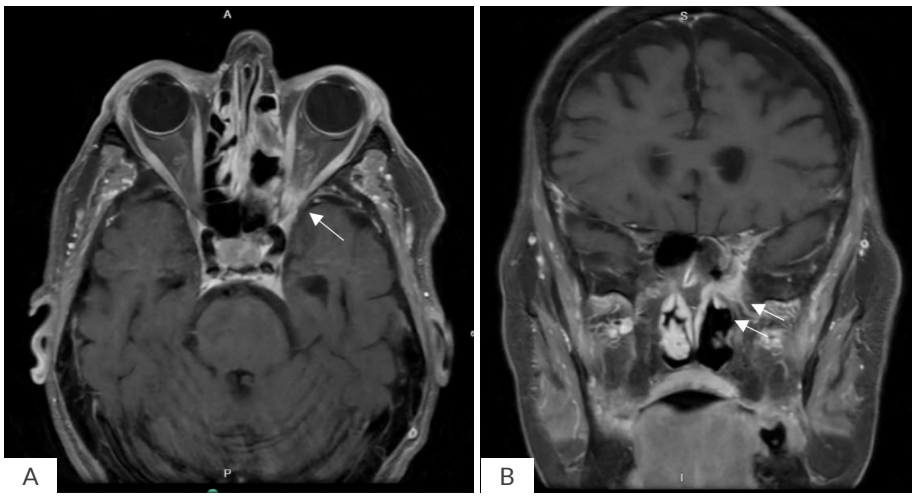


Figure 2. A) Post-contrast magnetic resonance imaging (MRI) showing stable asymmetric enhancement at the left orbital apex with no worsening. B) Coronal MRI of paranasal sinuses showing postsurgical changes of the left paranasal sinuses.

Initial nasal endoscopy revealed mild purulent debris with negative fungal cultures. Repeat endoscopy at our centre showed no necrosis or features of invasive fungal sinusitis, and an extended fungal workup was negative. Gram stain demonstrated Gram-negative bacilli, and cultures from nasal swab and biopsy grew *S. marcescens*, pan-susceptible to multiple antibiotics. Surgical pathology showed benign chronic inflammation without vasculitis or malignancy.

The initial differential included sphenoid mycetoma with optic neuritis, considering recent steroid use and early endoscopic findings. The patient was started on intravenous amphotericin B for suspected fungal sinusitis but developed infusion reactions and was switched to voriconazole. As symptoms progressed, repeat endoscopy and biopsy excluded fungal disease and malignancy. Cultures grew *S. marcescens*, and she was treated with intravenous ceftriaxone and metronidazole, leading to rapid headache relief and improvement in ocular function with declining inflammatory markers. Antifungals and metronidazole were discontinued after culture confirmation, and she completed a 21-day course of oral levofloxacin.

On day 12, repeat contrast-enhanced MRI of the brain and orbits demonstrated improvement in left orbital apex enhancement without new abnormalities *(Fig. 2)*. At 4-week neuro-ophthalmology follow-up, fundoscopic examination and optical coherence tomography (OCT) were normal, with no evidence of retinal or optic nerve pathology. A follow-up MRI at 2 months showed partial resolution of the orbital apex abnormality and improved sphenoid sinus opacification *(Fig. 3)*. Clinically, the patient remained asymptomatic, with no residual neurological deficits.

DISCUSSION

Fungal infections, particularly *Aspergillus* and *Mucor*, and neoplastic processes are well-recognized causes of OAS, whereas bacterial aetiologies are far less common. *S. marcescens* has rarely been reported in this anatomical region[1-3]. Our case highlights an unusual presentation of OAS secondary to *S. marcescens* in the setting of isolated sphenoid sinusitis.

S. marcescens is a Gram-negative, facultative anaerobic bacillus of the *Enterobacteriaceae* family and an opportunistic pathogen found in soil, water, plants, insects, animals, and various hospital reservoirs, including sinks, nebulizers, and equipment. It is primarily associated with nosocomial infections such as urinary tract and pulmonary infections but has also been implicated in osteomyelitis, endocarditis, and soft tissue infections. Rarely, it has been isolated from ocular tissue or cerebrospinal fluid[4-5].

Although *S. marcescens* is an uncommon pathogen in sinus infections, its occurrence in this patient is better understood in the context of underlying comorbidities and treatment exposures. Long-standing type 2 diabetes mellitus is a well-recognized risk factor for impaired neutrophil function, reduced chemotaxis, and impaired phagocytosis, all of which predispose to opportunistic and atypical infections. In addition, corticosteroid therapy likely compounded immune suppression by blunting both the inflammatory response and host defence mechanisms, thereby facilitating pathogen proliferation and masking early disease progression[6-8]. Chronic rhinosinusitis itself carries the risk of serious complications, including orbital cellulitis, abscess formation, venous thrombosis, bony erosion, OAS, and even intracranial spread. A remote history of left periorbital trauma may have further compromised local sinus architecture[9]. *S. marcescens* is known to produce β-lactamases and form biofilms, enhancing both virulence and antibiotic resistance. Although isolated reports exist of *S. marcescens* associated panophthalmitis and orbital cellulitis, typically in intravenous drug users, contact lens wearers, or immunocompromised hosts, this case is unique in demonstrating *S. marcescens* sphenoid sinusitis with direct extension to the orbital apex, resulting in an inflammatory cranial neuropathy that closely mimicked invasive fungal infection[10].

In this patient, progressive unilateral retro-orbital headache with transient steroid response, followed by vision loss and cranial nerve III, IV, and VI involvement, raised concern for sphenoid fungal sinusitis, orbital pseudotumor, or optic neuritis. Empiric steroid therapy likely suppressed inflammatory markers and complicated diagnosis. Repeat

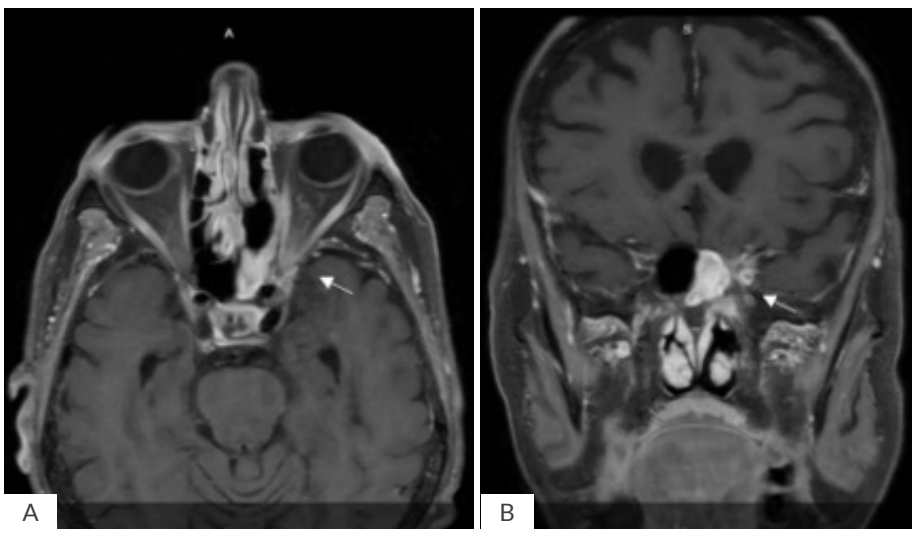


Figure 3. A) Post-contrast magnetic resonance imaging (MRI) showing partial interval resolution of previously demonstrated left orbital apex signal abnormality with no new orbital abnormality. B) Coronal MRI of paranasal sinuses showing improvement in left sphenoid sinus opacification.

endoscopy and biopsy excluded invasive fungal sinusitis and malignancy, and the findings were most consistent with OAS[3,7-8]. Orbital pseudotumor was not supported by biopsy or IgG4 studies, and optic neuritis was unlikely given absent infraorbital enhancement, advanced age, lack of autoimmune history, and multiple cranial neuropathies. IgG4-related disease, common variable immunodeficiency, and cavernous sinus thrombosis were also excluded through normal IgG4 levels and negative vascular imaging. Ultimately, infection was confirmed with *S. marcescens* cultures.

Treatment of Serratia infections poses challenges due to the organism's inherent resistance to multiple antibiotics, including first-generation cephalosporins and aminopenicillins. Current recommendations support the use of third- and fourth-generation cephalosporins (e.g., ceftazidime, cefepime), fluoroquinolones (e.g., ciprofloxacin, moxifloxacin), and carbapenems (e.g., meropenem) based on susceptibility profiles. Although no standardized regimen exists for infections with orbital apex involvement, extended antibiotic courses are generally preferred when there is concern for deep-seated infection, such as orbital cellulitis or osteomyelitis. In our patient, the extent of orbital apex enhancement on MRI raised suspicion for early orbital osteomyelitis, which justified prolonged therapy. Our patient responded well to 21 days of oral levofloxacin guided by culture sensitivity, consistent with literature supporting fluoroquinolones as effective therapy for *S. marcescens* infections[11].

In conclusion, OAS is a vision-threatening emergency, and *S. marcescens*, though rare, should be considered in the differential. This case highlights the value of repeat cultures and imaging to distinguish bacterial from fungal or neoplastic causes, and the importance of early targeted therapy to preserve vision.

REFERENCES

1. Badakere A, Patil-Chhablani P. Orbital Apex Syndrome: A Review. *Eye Brain* 2019;**11**:63-72. doi: 10.2147/EB.S180190
2. Lee PH, Shao SC, Lee WA. Orbital Apex Syndrome: A Case Series in a Tertiary Medical Center in Southern Taiwan. *Front Med (Lausanne)* 2022;**9**:845411. doi: 10.3389/fmed.2022.845411
3. Khanna A, Khanna M, Aggarwal A. *Serratia marcescens*- a rare opportunistic nosocomial pathogen and measures to limit its spread in hospitalized patients. *J Clin Diagn Res* 2013;**7**:243-246. doi: 10.7860/JCDR/2013/5010.2737
4. Goyal P, Lee S, Gupta N, Kumar Y, Mangla M, Hooda K, Li S, Mangla R. Orbital apex disorders: Imaging findings and management. *Neuroradiol J* 2018;**31**:104-125. doi: 10.1177/1971400917740361
5. Tavares-Carreon F, De Anda-Mora K, Rojas-Barrera IC, Andrade A. *Serratia marcescens* antibiotic resistance mechanisms of an opportunistic pathogen: a literature review. *PeerJ* 2023;**11**:e14399. doi: 10.7717/peerj.14399
6. Sharma M, Bundogji N, Azar NS, Lin A, Nakatsuka AS. A Case of Fulminant *Serratia marcescens* Panophthalmitis After Penetrating Keratoplasty. *Eye Banking and Corneal Transplantation* 2024;**3**:e0031. doi:10.1097/ebct.0000000000000031
7. Lee SH, Woo SJ, Park KH, Kim JH, Song JH, Park KU, et al. Serratia marcescens endophthalmitis associated with intravitreal injections of bevacizumab. *Eye (Lond)* 2010;**24**:226-232. doi: 10.1038/eye.2009.86
8. Peeters A, Vandercam B, Sindic CJ, Hantson P, Mahieu P. Community-acquired Serratia marcescens meningitis. *J Infect* 1997;**35**:303-304. doi: 10.1016/s0163-4453(97)93384-3
9. Richter AL, Gallagher KK. Chronic invasive fungal sinusitis causing a pathologic Le Fort I fracture in an immunocompetent patient. *Ear Nose Throat J* 2016;**95**:E1-3. doi: 10.1177/014556131609500904
10. Breazzano MP, Jonna G, Nathan NR, Nickols HH, Agarwal A. Endogenous *Serratia marcescens* panophthalmitis: A case series. *Am J Ophthalmol Case Rep* 2019;**16**:100531. doi: 10.1016/j.ajoc.2019.100531
11. Zivkovic Zaric R, Zaric M, Sekulic M, Zornic N, Nesic J, Rosic V, et al. Antimicrobial Treatment of *Serratia marcescens* Invasive Infections: Systematic Review. *Antibiotics (Basel)* 2023;**12**:367. doi: 10.3390/antibiotics12020367