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Ocular Salvage in *Serratia marcescens*-Associated Odontogenic Orbital Abscess: Unmasking Latent Diabetes Mellitus and the Role of Immunoparesis

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A B S T R A C T

Odontogenic sinusitis accounts for up to 40 percent of chronic maxillary sinusitis cases. While typically polymicrobial, the isolation of Serratia marcescens, an opportunistic Enterobacteriaceae, is an aberration in community settings and often signals profound underlying immunodeficiency. This report documents a rare case of Serratia-induced subperiosteal orbital abscess that unmasked latent type 2 diabetes mellitus. A 64-year-old male farmer presented with unilateral proptosis, ophthalmoplegia (frozen globe), and severe orbital pain following chronic odontogenic symptoms. Initial assessment revealed a random blood glucose of 500 mg/dL and glycated hemoglobin of 11.5 percent, confirming undiagnosed type 2 diabetes mellitus. Computed Tomography demonstrated a right maxillary abscess with osteolytic destruction of the lamina papyracea and an extraconal collection. Microbiological analysis of deep tissue biopsy via Vitek 2 confirmed Serratia marcescens while histopathology ruled out invasive fungal sinusitis. Management involved a dual-front strategy: rapid glycemic stabilization and broad-spectrum antibiotics, followed by functional endoscopic sinus surgery and dental extraction on Day 3. Post-operative follow-up showed resolution of proptosis from 24 mm to 16 mm, restoration of intraocular pressure from 32 mmHg to 14 mmHg, and improvement in visual acuity from light perception to 6/18. In conclusion, the isolation of Serratia marcescens in odontogenic sinusitis serves as a sentinel marker for metabolic dysregulation. This case highlights the synergistic lethality of neglected dental pathology and diabetic immunoparesis. Early recognition, exclusion of fungal mimics, and aggressive multidisciplinary intervention are paramount for ocular salvage.

1. Introduction

Rhinosinusitis constitutes a prevalent and heterogeneous inflammatory condition affecting the nasal cavity and paranasal sinuses, representing a significant burden on global healthcare systems. While often perceived as a singular disease entity, rhinosinusitis is etiologically bifurcated into rhinogenic and odontogenic origins, a distinction that is critical for therapeutic success yet frequently overlooked in primary care settings. Within this spectrum, odontogenic sinusitis represents a distinct, pathological entity that accounts for a substantial and

increasing proportion of maxillary sinusitis cases. Historically underestimated, contemporary literature suggests that odontogenic sources are responsible for approximately 25 to 40 percent of all chronic maxillary sinusitis cases, a prevalence that underscores the necessity for interdisciplinary vigilance between otolaryngologists and dental practitioners.[1,2]

Unlike the more common rhinogenic sinusitis, which typically stems from viral upper respiratory infections leading to ostial obstruction, ciliary dyskinesia, and subsequent bacterial superinfection, the pathogenesis of odontogenic sinusitis is fundamentally different. It arises when dental pathogens breach the Schneiderian membrane, the delicate pseudostratified ciliated columnar epithelium that lines the maxillary sinus. This breach is not merely a mucosal event but is deeply rooted in the osteological relationship between the dentition and the antrum. The apices of the maxillary posterior teeth, particularly the first and second molars and the second premolar, share an intimate anatomical proximity to the sinus floor. In many individuals, the intervening alveolar bone thickness can be negligible or entirely absent, leaving only the mucoperiosteum to separate the dental pulp from the sinus cavity. Consequently, periapical pathology, such as chronic periodontitis, apical granulomas, or iatrogenic displacement of dental roots, can facilitate the direct translocation of oral microbiota into the sterile environment of the sinus. Once the Schneiderian membrane is violated, the sinus effectively becomes a reservoir for infection, perpetuated not by ostial failure, but by a continuous bacterial load from the oral cavity.[3,4]

While the majority of odontogenic sinusitis cases remain confined to the maxillary antrum, presenting with unilateral foul-smelling rhinorrhea and facial pressure, a subset of these infections progresses to catastrophic extrasinus complications. The anatomical boundaries of the paranasal sinuses are not impervious barriers; rather, they are shared walls with vital intracranial and orbital structures. The orbit is particularly vulnerable to contiguous infectious spread due to the tenuous nature of the lamina papyracea, the paper-thin bony plate separating the ethmoid air cells and the maxillary sinus from the orbital contents. Furthermore, the venous drainage of the midface involves a valveless network of veins, including the ophthalmic veins and the pterygoid plexus, which allows for bidirectional flow and the retrograde propagation of septic thrombophlebitis.[5,6]

The progression of orbital complications is clinically stratified by the Chandler classification system, which categorizes severity from preseptal cellulitis to cavernous sinus thrombosis. Within this hierarchy, a subperiosteal abscess represents a critical Stage III emergency. Pathologically, this involves the accumulation of purulent material between the periorbita and the bony orbital wall, leading to a rapid increase in intraorbital pressure. Because the orbit is a confined osseous compartment with limited compliance, this pressure elevation can quickly compromise the vascular supply to the optic nerve and the retina. Without urgent surgical intervention and medical decompression, a subperiosteal abscess can precipitate irreversible sequelae, including compressive optic neuropathy, central retinal artery occlusion, and permanent blindness, as well as intracranial extension leading to meningitis or brain abscess.[7]

The microbiological landscape of odontogenic sinusitis further complicates its management and distinguishes it from rhinogenic forms. It is predominantly a polymicrobial infection, reflecting the complex ecology of the oral cavity. The typical microbiome is dominated by strict anaerobes and facultative anaerobes such as *Peptostreptococcus*, *Fusobacterium*, and *Prevotella* species, often in synergistic association with microaerophilic streptococci. However, the isolation of *Serratia marcescens*, a Gram-negative bacillus of the *Enterobacteriaceae* family, represents a significant and

alarming aberration in the context of community-acquired head and neck infections. *Serratia* species are ubiquitous in the environment, thriving in soil and water, but clinically, they are notoriously associated with nosocomial infections, particularly in catheterized or ventilated patients. The organism possesses intrinsic resistance mechanisms, including the production of inducible chromosomal AmpC beta-lactamases, conferring resistance to many first-line antibiotics. The identification of *Serratia marcescens* in a community-acquired odontogenic infection is highly atypical and strongly suggests a complex interplay between the pathogen's virulence factors—such as biofilm formation and the production of cytotoxic proteases like serralysin—and a profound failure of host immune surveillance.[8-10]

This intersection of rare microbiology and severe pathology points inevitably toward the status of the host. The geriatric population presents a unique clinical challenge characterized by immunosenescence, a gradual deterioration of the immune system that accompanies aging. This decline is frequently compounded by undiagnosed or poorly controlled metabolic disorders, most notably Type 2 diabetes mellitus. Diabetes acts as a silent accelerator of infectious processes through the mechanism of diabetic immunoparesis. The hyperosmolar environment inherent in uncontrolled hyperglycemia fundamentally alters leukocyte biology; it impairs neutrophil chemotaxis, reducing the ability of immune cells to migrate to the site of infection, and depresses phagocytosis and intracellular killing via the respiratory burst. Furthermore, the presence of Advanced Glycation End-products compromises the integrity of the vascular endothelium and basement membranes, facilitating bacterial invasion. In such a compromised host, typically low-virulence opportunistic organisms like *Serratia* can escape immune containment and cause fulminant tissue destruction, such as the rapid osteolysis of the orbital floor.[9]

This manuscript aims to document an exceptionally rare presentation of *Serratia marcescens*-induced subperiosteal orbital abscess secondary to odontogenic sinusitis in a sexagenarian with undiagnosed type 2 diabetes mellitus. While odontogenic sinusitis is a known entity, the convergence of this specific nosocomial pathogen with an odontogenic source in a community setting is nearly unprecedented in the literature. We provide a comprehensive analysis of the synergistic lethality between chronic dental neglect and latent metabolic dysregulation, illustrating how this triad can precipitate a surgical emergency. Beyond the clinical narrative, this report specifically highlights the diagnostic necessity of ruling out invasive fungal mimics, such as Mucormycosis, in diabetic orbital pathology, as the clinical presentation often overlaps. Ultimately, this study underscores the critical public health implication that dental practitioners act as the first line of defense in preventing vision-threatening orbital complications and that the discovery of an atypical pathogen in the sinus should serve as a sentinel marker prompting immediate metabolic screening.

2. Case Presentation

Written informed consent was obtained from the patient for the publication of this case report and any accompanying clinical data and images. The patient was fully briefed on the educational purpose of this publication, and all personal identifiers have been anonymized to protect patient privacy in accordance with the Declaration of Helsinki.

A 64-year-old male farmer from a rural agricultural district in Gianyar, Indonesia, was referred to the Emergency Department of a tertiary academic medical center. The patient presented with a chief complaint of progressive, painful swelling of the right eyelid persisting for 6 days, associated with a sudden decline in vision. The clinical trajectory began 8 days prior to admission with high-grade fever and right maxillary

pain. This progressed to periorbital erythema and mechanical ptosis. The patient reported a specific symptom of cacosphonia, a subjective foul smell, strongly suggestive of an anaerobic process. He admitted to a 12-month history of untreated chronic caries involving the right upper molars, for which he had not sought dental care due to socioeconomic constraints. As a rice farmer, the patient reported frequent exposure to soil and the use of untreated well water for oral hygiene, a potential vector for environmental pathogens. Before referral, he received 4 days of intravenous ceftriaxone at 2 grams daily at a district hospital. This treatment failed to halt orbital progression, raising suspicion of a resistant organism or source control failure.

On general survey, the patient appeared septic with tachycardia at 110 beats per minute and tachypnea at 22 breaths per minute, though afebrile at 36.8 degrees celsius, likely due to prior antipyretic administration. Ophthalmologic assessment revealed; (1) Right eye: Visual acuity was severely compromised to light perception only, with inaccurate projection. There was marked proptosis measuring 24 mm on exophthalmometry, severe chemosis, and total ophthalmoplegia or frozen globe. The pupil was fixed and mid-dilated. Intraocular pressure measured at 32 mmHg, significantly higher than the normal range of 10 to 21 mmHg, indicating acute orbital compartment syndrome threatening the optic nerve; (2) Left eye: Normal findings with visual acuity of 6/9 and intraocular pressure of 14 mmHg. A draining fistula on the right buccal mucosa was noted. Intraoral inspection revealed gangrenous radices at positions 13, 14, 16, and 26, with evident periodontal disease.

Comprehensive laboratory analysis unmasked a profound metabolic crisis underlying the acute presentation, fundamentally altering the risk stratification of the patient. The metabolic profile revealed severe hyperglycemia with a random blood glucose of 500 mg/dL, accompanied by a glycated hemoglobin level of 11.5 percent. These values confirmed a diagnosis of uncontrolled, long-standing type 2 diabetes mellitus, which had likely remained latent until unmasked by the substantial physiological stress of the active infection. Fortunately, renal function was preserved with a creatinine level of 0.9 mg/dL, which permitted the safe administration of intravenous contrast media for urgent radiological assessment. Concurrently, hematological parameters signaled a robust systemic inflammatory response indicative of severe bacterial sepsis. This was evidenced by a significant leukocytosis of 17.07 times 10 to the power of 3 per microliter, characterized by a profound left shift with 94.2 percent neutrophilia, and a marked elevation in C-reactive protein at 150 mg/L, underscoring the severity of the infectious burden.

Following metabolic stabilization, contrast-enhanced computed tomography of the paranasal sinuses was performed to delineate the extent of the pathology. The imaging revealed total opacification of the right maxillary sinus consistent with empyema (Figure 1). Of particular diagnostic significance was the visualization of a periapical radiolucent halo surrounding the roots of the first molar, specifically tooth 26. This radiological finding demonstrated a direct communication with the sinus floor, thereby confirming the odontogenic etiology of the infection. The scan further identified extensive osteolytic destruction involving the erosion of the lamina papyracea and the orbital floor. This osseous breach facilitated the extension of the infection into the orbit, manifesting as an extraconal fluid collection measuring 3.2 by 2.1 cm. This abscess displaced the medial rectus muscle laterally, a finding consistent with a subperiosteal abscess classified as Chandler Stage III, necessitating immediate surgical decompression.

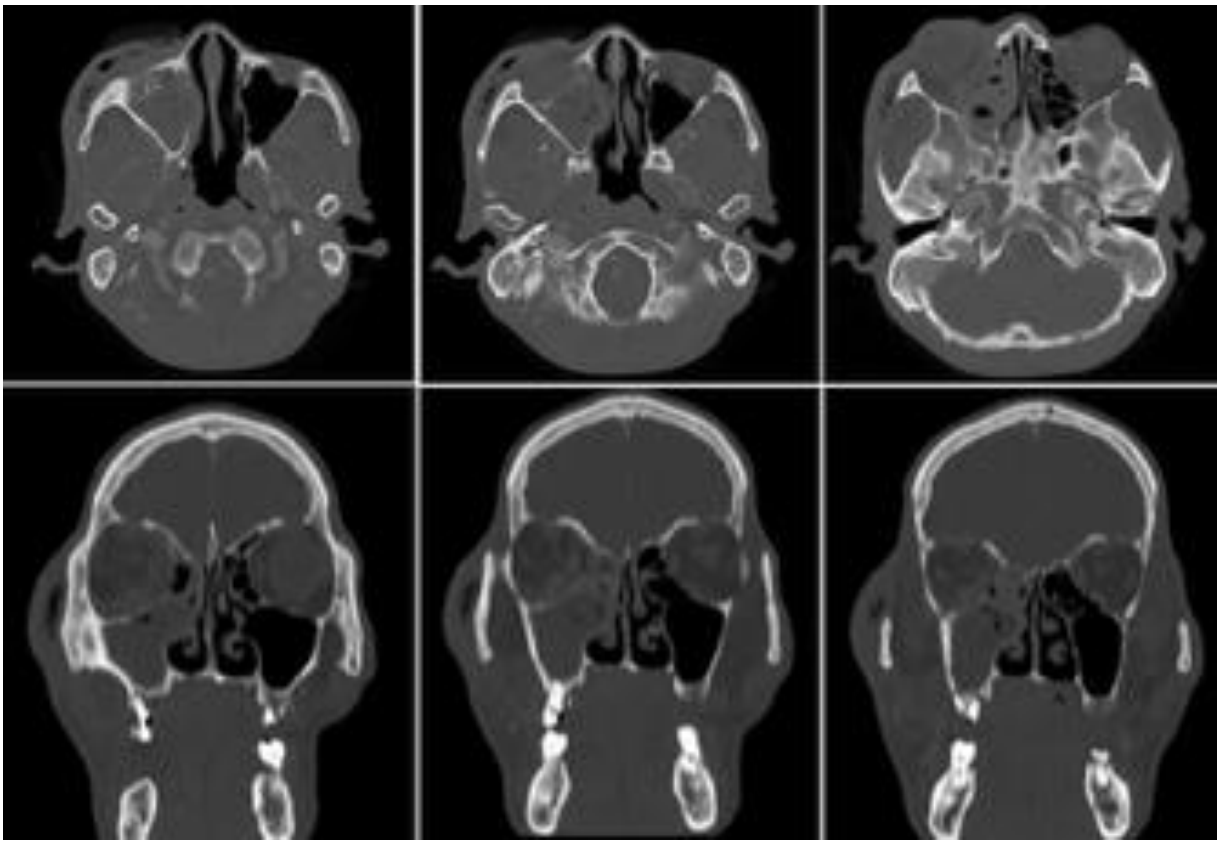

Figure 1. CT-scan of paranasal sinuses.

To ensure high diagnostic validity and mitigate the risk of contamination from commensal oral flora, microbiological samples were rigorously harvested from deep tissue sequestered within the maxillary sinus during the surgical procedure, distinct from the superficial drainage of the buccal fistula. Bacterial identification was subsequently performed utilizing the Vitek 2 Compact automated system, which yielded a pure growth of *Serratia marcescens*. Susceptibility testing revealed a resistance profile characteristic of inducible chromosomal AmpC beta-lactamase production, demonstrating resistance to ampicillin, cefazolin, and cefuroxime. However, the isolate retained sensitivity to ceftriaxone, ceftazidime, and moxifloxacin, guiding the definitive antimicrobial regimen. Given the patient's compromised immunometabolic status and the intraoperative finding of necrotic mucosa, a critical component of the diagnostic workup involved the rigorous exclusion of invasive fungal sinusitis. Histopathological evaluation utilized Grocott-Gomori Methenamine Silver and Periodic Acid-Schiff stains. Both stains were negative for fungal hyphae, definitively ruling out fungal pathology such as Mucormycosis and confirming the bacterial nature of this aggressive orbital infection.

Table 1. Summary of Clinical Findings on Admission

Category	Parameter	Finding / Value	Clinical Interpretation
Patient Profile	**Demographics**	64-year-old Male, Farmer	*Rural exposure (soil/water)*
	Chief Complaint	R. Eyelid swelling (6 days), Vision loss	*Progressive worsening*
Vital Signs	**Hemodynamics**	HR: **110 bpm**, RR: **22 bpm**, Temp: 36.8°C	*Signs of sepsis (afebrile likely due to antipyretics)*
Ophthalmology (Right Eye)	**Visual Acuity (VA)**	**Light Perception (LP)**	*Severe visual compromise*
	External Exam	Proptosis: **24 mm**, Severe Chemosis	*Orbital volume expansion*
	Motility & Pupil	Frozen Globe (Total Ophthalmoplegia), Pupil Fixed	*Orbital Apex Syndrome / Muscle entrapment*
	Intraocular Pressure (IOP)	**32 mmHg**	*Acute Orbital Compartment Syndrome*
Dental & Oral	**Maxillary Examination**	Gangrenous Radices: #13, #14, #16, #26	*"Cacosphonia" (foul smell), Buccal fistula present*
Metabolic Profile	**Glycemic Status**	Random Glucose: **500 mg/dL**	*Severe Hyperglycemia / HHS Risk*
	HbA1c	**11.5 %**	*Uncontrolled, Long-standing T2DM (Undiagnosed)*
Inflammatory Markers	**Leukocytes (WBC)**	17.07 × 10^3/µL (94.2% Neutrophils)	*Severe bacterial leukocytosis with left shift*
	C-Reactive Protein (CRP)	**150 mg/L**	*Acute systemic inflammation*
CT Imaging (Contrast Enhanced)	**Sinus Pathology**	Total opacification of Right Maxillary Sinus	*Consistent with Empyema*
	Bone Involvement	Osteolytic destruction of lamina papyracea & orbital floor	*Periapical halo at tooth #26 communicating with sinus*
	Orbital Extension	Extraconal abscess (3.2 x 2.1 cm)	*Chandler Stage III; Medial rectus displacement*

Abbreviations: HR: Heart Rate; RR: Respiratory Rate; VA: Visual Acuity; LP: Light Perception; IOP: Intraocular Pressure; T2DM: Type 2 Diabetes Mellitus; HHS: Hyperosmolar Hyperglycemic State; WBC: White Blood Cells.

A complex, dual-front therapeutic strategy was implemented to navigate the perilous intersection of sight-threatening orbital pathology and life-threatening metabolic derangement (table 2). The management protocol was stratified into two distinct phases, prioritizing immediate physiological stabilization followed by definitive surgical eradication of the septic focus. Phase 1, spanning days 0 through 2, focused on metabolic resuscitation and provisional decompression. Given the patient's precipitous admission state—characterized by a random blood glucose of 500 mg/dL and the impending risk of hyperosmolar hyperglycemic state—immediate general anesthesia was deemed an unacceptable

physiological risk. Consequently, an aggressive insulin protocol was initiated, utilizing a continuous intravenous infusion at 0.1 units per kg per hour to rapidly correct the hyperosmolarity. This was subsequently transitioned to a basal-bolus subcutaneous regimen employing Glargine and Aspart, successfully titrating blood glucose levels to a target range of 140 to 180 mg/dL.

Concurrently, broad-spectrum antimicrobial coverage was established to address the presumed polymicrobial etiology. Empirical intravenous ceftriaxone at 2 grams every 24 hours was administered to cover Gram-negative facultative anaerobes, while metronidazole at 500 mg every 8 hours targeted the obligate anaerobes typical of odontogenic infections. To mitigate the risk of exposure keratopathy due to proptosis and lagophthalmos, topical moxifloxacin was applied for corneal protection. Recognizing that medical therapy alone could not reverse the compartment syndrome, a strategic decompression bridge was performed at the bedside. Under local anesthesia, a targeted incision was made using a number 11 blade, followed by the placement of a Penrose drain. This temporizing maneuver yielded 30 mL of purulent material, effectively reducing the intraorbital volume and relieving critical pressure on the optic nerve while the patient was metabolically optimized for the operating theater.

Phase 2, the definitive surgical intervention, was executed on day 3 once the patient achieved metabolic stability with glucose levels consistently below 180 mg/dL. The patient underwent Functional Endoscopic Sinus Surgery (FESS) to re-establish physiological drainage and ventilate the sinuses. The procedure entailed a wide middle meatal antrostomy and total ethmoidectomy, extending to the removal of the compromised lamina papyracea to facilitate maximal orbital decompression. In a coordinated multidisciplinary effort, the oral surgery team simultaneously performed extractions of the offending dentition—specifically teeth 13, 14, 26, and 27—followed by alveoloplasty to eliminate the primary odontogenic source.

The clinical efficacy of this staged approach was evidenced by the robust post-operative recovery. Proptosis and inflammatory signs resolved rapidly, allowing for hospital discharge on Day 7 with oral cefixime. At the 3-month follow-up, the functional outcomes were substantial. The patient's visual acuity, which had been compromised to mere Light Perception on admission, significantly improved to 6/18 on the Snellen chart. Furthermore, the Intraocular Pressure normalized from a critical 32 mmHg to a physiological 14 mmHg, confirming the resolution of the compartment syndrome. Nasal endoscopy revealed a patent antrostomy with healthy, well-mucosalized tissue, indicating a complete resolution of the infectious process without recurrence.

Table 2. Diagnosis, Treatment Protocol, Follow-up, and Outcome

Phase / Domain	Component	Details & Regimen	Rationale / Result
Final Diagnosis	**Primary Pathology**	**Orbital** Right Subperiosteal Orbital Abscess (Chandler Stage III) **Sinus** Right Odontogenic Maxillary Sinusitis (Empyema)	*Confirmed by CT (Osteolytic destruction of lamina papyracea)*
	Etiology & Microbiology	**Pathogen:** *Serratia marcescens* (Pure growth) **Source:** Gangrenous Radices (Teeth #13, #14, #16, #26)	*Rare pathogen; Likely environmental inoculation or selection pressure*
	Systemic Comorbidity	Newly Diagnosed Type 2 Diabetes Mellitus (Presentation: Hyperosmolar Hyperglycemic State)	*Unmasked by sepsis (HbA1c 11.5%)*
Phase 1: Stabilization (Day 0-2)	**Medical Optimization**	**Glycemic:** IV Insulin (0.1 u/kg/hr) → Subcutaneous Basal-Bolus (Glargine/Aspart) **Antibiotics:** IV Ceftriaxone (2g q24h) + IV Metronidazole (500mg q8h) + Topical Moxifloxacin	*Target Glucose < 180 mg/dL; Cover Serratia + Anaerobes*
	Bridge Intervention	Bedside Incision & Drainage (Buccal approach) Local Anesthesia + Penrose Drain	*Urgent IOP reduction; Avoided GA risks during acute HHS*
Phase 2: Definitive Surgery (Day 3)	**Surgical Procedures**	**ENT** Functional Endoscopic Sinus Surgery (FESS) with Orbital Decompression **Dental** Multiple Extractions (#13, #14, #16, #26) + Alveoloplasty	*Clearance of septic foci; Restoration of sinus drainage*
	Histopathology	Tissue biopsy stained with GMS (Grocott-Gomori) and PAS	***Result:** Negative for fungal hyphae (Ruled out Mucormycosis)*
Outcome & Follow-up (3 Months)	**Ocular Metrics**	**Visual Acuity:** Improved from LP (Light Perception) to **6/18** **IOP:** Normalized (32 mmHg → **14 mmHg**)	*Successful ocular salvage; Optic nerve recovery*
	Anatomical Status	**Proptosis:** Resolved (24 mm → 16 mm) **Motility:** Full range of motion (Frozen globe resolved)	*Resolution of orbital compartment syndrome*
	Systemic Status	Glycemic control maintained on oral agents. Sinus ostia patent on endoscopy.	*No recurrence of abscess or sinusitis*

Abbreviations: IV: Intravenous; GA: General Anesthesia; HHS: Hyperosmolar Hyperglycemic State; IOP: Intraocular Pressure; GMS: Grocott-Gomori Methenamine Silver; PAS: Periodic Acid-Schiff; LP: Light Perception.

3. Discussion

The case presented illustrates a catastrophic convergence of three distinct but synergistic pathological vectors: a neglected chronic odontogenic infection, the opportunistic emergence of the rare pathogen *Serratia marcescens*, and a profound, undetected state of immunocompromise. This triad did not merely coexist but interacted to accelerate disease progression, transforming a benign toothache into a sight-threatening orbital abscess and a life-threatening metabolic crisis. The rapidity with which this patient deteriorated underscores the vulnerability of the orbital compartment to contiguous spread when host defenses are compromised by metabolic dysregulation.

The isolation of *Serratia marcescens* from the

maxillary sinus represents the pivotal microbiological finding of this report. While the organism is a well-documented nosocomial pathogen, frequently implicated in catheter-associated urinary tract infections and ventilator-associated pneumonia, its identification as the primary etiological agent in community-acquired sinusitis is exceptionally rare. In standard clinical practice, community-acquired odontogenic sinusitis is dominated by a polymicrobial flora of oral streptococci and anaerobes such as *Fusobacterium* and *Peptostreptococcus*. The deviation from this expected microbiological profile necessitates a rigorous examination of the inoculation pathways.[11-12]

We hypothesize two primary mechanisms driving this anomalous colonization. The first is environmental inoculation rooted in the patient's occupational hazards. As a farmer in rural Gianyar, the patient had daily contact with soil and potentially untreated water sources. *Serratia* is a ubiquitous saprophyte in these environments, thriving in damp conditions. It is plausible that the patient introduced the pathogen into the oral cavity through the use of contaminated well water for oral hygiene or direct contact with soil. The patient's chronic dental pathology, characterized by crumbly teeth and necrotic pulp, provided an ideal, nutrient-rich nidus for the organism to establish a biofilm and proliferate, protected from mechanical clearance. The second, and perhaps more clinically significant mechanism, is the role of antibiotic selection pressure. Prior to his referral to our tertiary center, the patient received a four-day course of intravenous Ceftriaxone at a district hospital. *Serratia* species are intrinsically resistant to many beta-lactams due to the presence of a chromosomal *ampC* beta-lactamase gene. While the initial infection likely began as a standard polymicrobial odontogenic abscess, the administration of a cephalosporin may have effectively eradicated the susceptible streptococcal and anaerobic competitors. This "clearing of the field" would allow the resistant *Serratia* clone to surge in population density, transitioning from a passive colonizer to a dominant, invasive pathogen capable of overwhelming local tissue barriers. This phenomenon highlights the double-edged sword of empirical antibiotic therapy in complex, source-control-deprived infections.[13-15]

The radiological evidence of osteolytic destruction, particularly the erosion of the lamina papyracea and the orbital floor, warrants a specific discussion regarding the virulence profile of Serratia (Figure 2). While pressure necrosis from a rapidly expanding abscess is a known mechanism of bone resorption in sinusitis, the speed and extent of destruction observed in this case suggest an enzymatic contribution. *Serratia marcescens* is unique among *Enterobacteriaceae* for its production of extracellular metalloproteases, most notably serralysin. This zinc-dependent protease possesses potent caseinolytic and gelatinolytic activity, enabling it to degrade vital components of the extracellular matrix, including collagen and proteoglycans.[16,17]

Furthermore, serralysin has been shown to degrade immunoglobulins (IgG) and complement factors, effectively blinding the host's immune system to the bacterial presence. We hypothesize that the local concentration of serralysin in the confined space of the maxillary sinus, combined with the organism's known capacity for robust biofilm formation on necrotic bone (sequestrum), accelerated the erosion of the thin bony partition separating the sinus from the orbit. This enzymatic "burrowing" facilitated a rapid breach into the subperiosteal space, converting a sinus empyema into an orbital compartment syndrome much faster than would be expected from pressure alone. This virulence factor may explain why the clinical presentation was so fulminant despite the patient initially appearing afebrile on admission.[18]

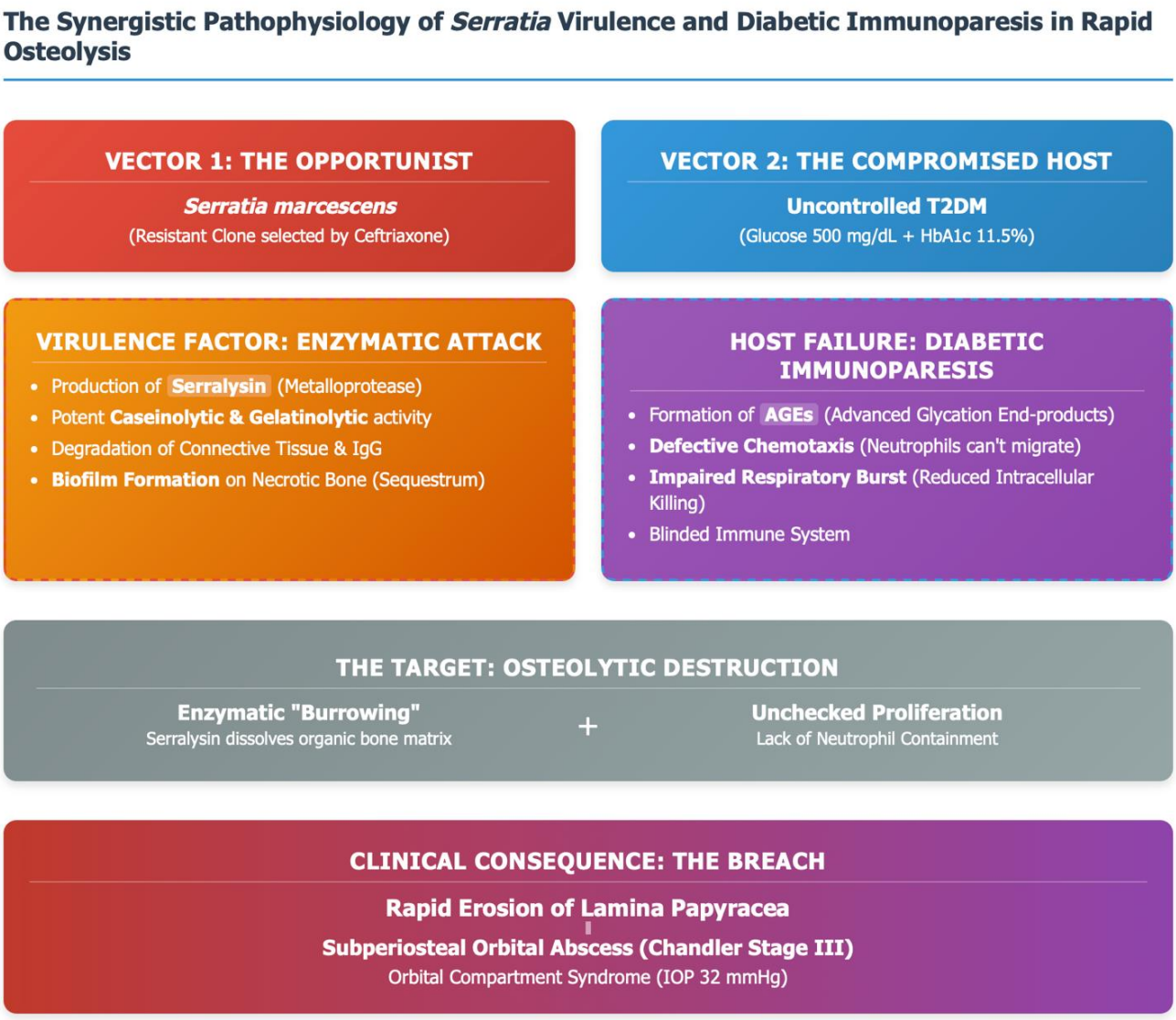

Figure 2. Pathophysiology of osteolysis and virulence.

The unmasking of latent type 2 diabetes mellitus, evidenced by a random blood glucose of 500 mg/dl and a glycated hemoglobin (HbA1c) of 11.5 percent, is central to understanding the severity of this case. The patient's hyperglycemic state was not merely a comorbidity; it was a fundamental driver of the pathology. Chronic hyperglycemia induces a state of diabetic immunoparesis, a multifaceted dysfunction of the innate immune system. Elevated glucose levels lead to the non-enzymatic glycation of proteins, forming advanced glycation end-products (AGEs). These AGEs impair neutrophil function in several critical ways. First, they reduce cellular deformability, hindering the ability of neutrophils to diapedese through the vascular endothelium and migrate to the site of infection. Second, they impair chemotaxis, leaving the host unable to mount an effective cellular response at the point of bacterial invasion. Third, and most critically, hyperglycemia depresses the respiratory burst, the mechanism by which neutrophils generate reactive oxygen species to kill phagocytosed bacteria.19 In this patient, this immune paralysis created a "perfect storm": the host was unable to wall off the infection, unable to kill the

pathogen, and provided a glucose-rich environment that fueled bacterial growth. This explains how a typically low-virulence environmental organism like *Serratia* could act with the aggression of a high-grade pathogen, causing extensive tissue destruction and orbital invasion.

From a methodological and safety perspective, the management of this case hinged on the rigorous exclusion of invasive fungal sinusitis, specifically *Mucormycosis*. In any diabetic patient presenting with sinusitis and orbital apex signs—such as the total ophthalmoplegia and fixed pupil seen here—*Mucormycosis* is the primary differential diagnosis until proven otherwise. The presence of necrotic mucosa and black turbinates, often described in Mucor, can mimic severe bacterial infections. This distinction is not academic; it is a matter of life and death. The standard adjunctive treatment for bacterial orbital cellulitis involves systemic corticosteroids to reduce orbital edema and optic nerve compression. However, administering steroids to a patient with undiagnosed *Mucormycosis* is akin to adding fuel to the fire, as it further suppresses the immune response and accelerates fungal invasion, often leading to fatal intracranial extension. In this case, the clinical team adhered to a strict safety algorithm: despite the pressure to reduce orbital swelling, steroids were withheld until histopathological analysis using Grocott-Gomori Methenamine Silver (GMS) staining confirmed the absence of fungal hyphae. This disciplined approach highlights a critical clinical pearl: in the diabetic orbit, fungus must be ruled out histopathologically before any immunosuppressive therapy is entertained.[18,19]

The management of Chandler Stage III complications requires a nuanced balance between the urgency of orbital decompression and the safety of the patient. Immediate major surgery (Functional Endoscopic Sinus Surgery - FESS) is the gold standard for source control. However, subjecting a septic patient with a blood glucose of 500 mg/dL to general anesthesia carries immense risks of intraoperative ketoacidosis, hemodynamic collapse, and malignant arrhythmias. This case validates the efficacy of a bridge therapy approach. Recognizing the high surgical risk, the team utilized a bedside incision and drainage of the buccal collection under local anesthesia as a temporizing measure. This procedure yielded 30 mL of pus, mechanically reducing the orbital counter-pressure and lowering the intraocular pressure (IOP) from a critical 32 mmHg. This bought valuable time—a safety window—during which the medical team could aggressively hydrate the patient and titrate insulin to achieve metabolic stability.20 Definitive FESS was then performed on Day 3, once glucose levels were below 180 mg/dL. This staged strategy aligns with recent consensus indicating that while orbital compression is an ocular emergency, physiological optimization prevents perioperative mortality. The successful visual outcome (recovery to 6/18) confirms that this brief delay for medical optimization did not result in permanent optic nerve damage.

The antibiotic selection in this case warrants careful scrutiny. The use of Ceftriaxone for *Serratia* infections is controversial. While the isolate was sensitive *in vitro*, *Serratia* species often carry an inducible ampC gene that can be derepressed by third-generation cephalosporins, leading to treatment failure. However, the successful outcome here likely relied on the synergistic use of Moxifloxacin. Fluoroquinolones like Moxifloxacin exhibit excellent bioavailability and, crucially, superior penetration into bone and sinus tissues compared to beta-lactams. They are also highly active against *Enterobacteriaceae* and do not induce *AmpC* beta-lactamases. This double-coverage strategy—combining a cephalosporin with a fluoroquinolone—was a prudent decision in the context of a sight-threatening infection where tissue penetration is paramount. It serves as a reminder that in severe head and neck infections, pharmacokinetic properties

(tissue penetration) are as important as in vitro susceptibility.

4. Conclusion

This case report establishes *Serratia marcescens* as a formidable, albeit rare, pathogen in the etiology of odontogenic subperiosteal orbital abscesses. It challenges the conventional assumption that community-acquired sinusitis is invariably caused by streptococci or anaerobes. Instead, it serves as a potent clinical alert: the presentation of severe, destructive sinusitis in a geriatric patient should trigger an immediate and aggressive screening for metabolic dysregulation. The presence of such an atypical, resistant Gram-negative pathogen is not merely a microbiological curiosity; it is a sentinel marker of a deeper systemic failure. The isolation of Serratia or other atypical opportunists in the sinus should be viewed as a red flag suggesting underlying immunodeficiency (such as undiagnosed diabetes) or specific environmental inoculation pathways. In the context of diabetic orbital pathology, clinical examination is insufficient to exclude fungi. Invasive fungal sinusitis must be explicitly ruled out via biopsy and histopathology in all such cases before steroid therapy is initiated.

This case underscores the catastrophic downstream costs of dental neglect. A preventable dental pathology evolved into a vision-threatening emergency due to socioeconomic barriers to care. Dental screening must be integrated into routine diabetic care protocols to identify and treat these reservoirs of infection before they breach critical anatomical barriers. Future research efforts should focus on characterizing the changing microbiome of odontogenic sinusitis in the aging population. As the prevalence of diabetes and immunosenescence rises, so too may the incidence of these atypical, resistant infections. Updating empiric antibiotic guidelines to account for the potential presence of fluoroquinolone-susceptible Gram-negative rods in this specific demographic could prove vital in preventing permanent visual disability.

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