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Case Report

Chronic Maxillary Osteomyelitis Mimicking Invasive Fungal Sinusitis in a Woman With Uncontrolled Diabetes Mellitus: A Case Report

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ABSTRACT

Background: Unilateral destructive sinonasal disease in a patient with uncontrolled diabetes immediately raises concern for rhino-maxillary mucormycosis. Because invasive fungal infection can progress rapidly, early recognition and tissue diagnosis are essential. However, chronic maxillary osteomyelitis may present with a very similar clinical and radiologic picture.
Case Presentation: A 55-year-old woman with poorly controlled type 2 diabetes mellitus presented with left-sided facial discomfort, unilateral nasal obstruction, foul oral odor, and leakage of fluids from the mouth into the nose. Examination revealed unhealthy mucosa over the left maxillary palate, exposed necrotic bone, and an oronasal fistula. Non-contrast CT of the paranasal sinuses showed unilateral left-sided sinonasal soft tissue thickening with destructive bony changes involving the maxillary antrum, hard palate, nasal septum, and adjacent skull-base region, raising strong concern for invasive fungal sinusitis. She was admitted and managed with broad-spectrum intravenous antibiotics, insulin therapy, and urgent tissue sampling. Histopathology showed necrotic and sclerotic bony trabeculae, empty osteocyte lacunae, chronic inflammatory infiltrate, and bacterial colonies, while fungal stains were negative. A deep tissue culture obtained before antibiotics showed mixed aerobic and anaerobic oral flora. These findings supported a diagnosis of chronic maxillary osteomyelitis with secondary palatal breakdown rather than tissue-proven fungal invasion.
Conclusion: In diabetic patients, destructive maxillofacial lesions should not be assumed to represent mucormycosis without tissue confirmation. Chronic maxillary osteomyelitis may closely mimic invasive fungal sinusitis on clinical examination and imaging, and histopathologic evaluation is essential to guide appropriate treatment.

1. Introduction

Osteomyelitis is an inflammatory infection of bone that may arise from hematogenous spread, contiguous extension from adjacent infection, or direct inoculation after trauma or surgery. In the jaws, odontogenic sources, periodontal disease, poor oral hygiene, and local anatomic factors can contribute to infection. The maxilla is less commonly affected than the mandible. Still, systemic compromise such as poorly controlled diabetes may predispose to infection by impairing neutrophil function, delaying healing, and reducing host resistance [1].

Unilateral destructive disease of the maxilla and paranasal sinuses in a patient with uncontrolled diabetes is often treated as mucormycosis until proven otherwise [2]. This approach is justified because mucormycosis is an angioinvasive fungal infection that can rapidly cause tissue necrosis, orbital extension, cavernous sinus thrombosis, and death [3]. At the same time, chronic bacterial osteomyelitis of the maxilla can present with a nearly identical appearance on clinical examination and computed tomography, including bone destruction, soft-tissue thickening, palatal erosion, and fistula formation [4].

The distinction between these two entities is clinically important. Mucormycosis requires urgent antifungal therapy and often surgical debridement, while chronic osteomyelitis is managed with prolonged antibacterial therapy, metabolic optimization, and selective removal of devitalized bone [5, 6]. Tissue diagnosis is therefore decisive. The following case illustrates how an initially alarming CT picture in a diabetic woman proved to be chronic maxillary osteomyelitis rather than mucormycosis. This case report is reported in line with the CARE guidelines [7], and a CARE checklist is provided as supplementary file S1. **(Figure 1)** and **(Figure 2)** show the saggital while **(Figure 3)** show the coronal CT scan images of the patient.

2. Case Presentation

A 55-year-old woman with known type 2 diabetes mellitus for the past 20 years presented to the outpatient department with progressive left-sided facial and oral symptoms. Over the preceding two weeks, she had developed dull pain and fullness over the left maxillary region, unilateral nasal obstruction, foul-smelling oral discharge, a bad taste in the mouth, reduced appetite, and leakage of fluids from the oral cavity into the nose while drinking. Oral discomfort worsened during mastication.

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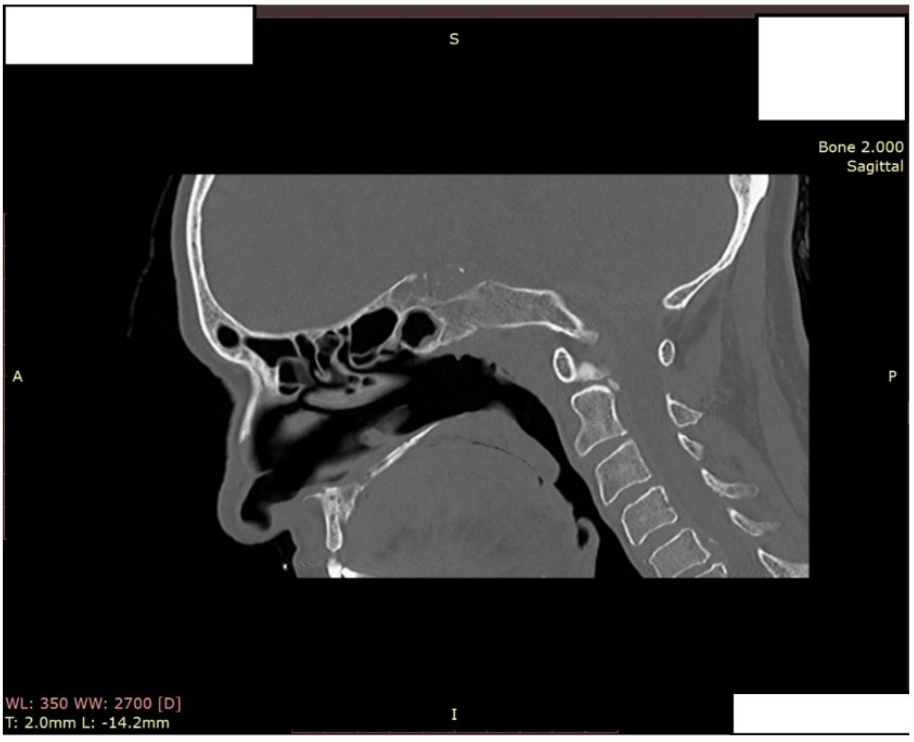

Figure 1: Sagittal CT paranasal sinus bone-window image showing destructive involvement of the left maxillary region and adjacent hard palate with associated soft tissue thickening.

There was no history of epistaxis, diplopia, proptosis, facial numbness, black nasal discharge, severe headache, altered consciousness, trauma, radiotherapy, corticosteroid use, or recent hospitalization. She denied visual blurring and trismus. Her diabetes had been poorly controlled for several months, and she admitted irregular medication adherence. The patient did not have a recent dental extraction, periodontal disease, denture use, smoking history, or antiresorptive medication exposure.

On examination, she was alert, afebrile, and hemodynamically stable. Mild fullness was present over the left midface. Intraoral examination revealed unhealthy mucosa over the left maxillary palatal region with focal ulceration, exposed necrotic bone, and a small oronasal fistula. The surrounding tissue was indurated and tender, with a foul odor. Bedside examination did not reveal proptosis, extraocular movement restriction, facial weakness, or gross sensory loss. Cranial nerve function was grossly intact on bedside testing. Gross dental instability was not appreciated, although formal dental mobility charting was not documented. Visual acuity and detailed neuro-ophthalmic testing were grossly normal. Because uncontrolled diabetes in the setting of destructive unilateral maxillofacial disease raises concern for invasive fungal sinusitis, mucormycosis was considered early in the diagnostic workup.

3. Investigations

The available imaging and pathological findings were most consistent with chronic maxillary osteomyelitis with secondary bacterial infection. **(Table 1)** shows the baseline investigation results and **(Table 2)** shows diagnostic studies which **(Table 3)** shows the clinical timeline

4. Histopathology

Urgent biopsy of the palatal/maxillary lesion was performed. Microscopic examination showed necrotic and sclerotic bony trabeculae with empty osteocyte lacunae, areas of reactive bone change, and a chronic inflammatory infiltrate composed predominantly of lymphocytes and plasma cells, with scattered macrophages and osteoclastic activity. Small bacterial colonies were identified within the specimen. No broad aseptate fungal hyphae or angioinvasion were seen on routine histology **(Figure 4)**. Periodic acid-Schiff and Gomori methenamine silver stains were negative for fungal organisms. Taken together, these findings supported a diagnosis of chronic maxillary osteomyelitis with secondary bacterial infection and palatal destruction, rather than tissue-proven mucormycosis.

5. Therapeutic Intervention

The patient was admitted under internal medicine with input from ENT and oral – maxillofacial surgery. No formal infectious diseases consultation was documented. Because invasive fungal sinusitis was a serious initial consideration, urgent tissue sampling was performed promptly to establish a diagnosis before further labeling of the lesion.

Empiric treatment was started with broad-spectrum intravenous antibiotics to cover mixed oral flora and anaerobic organisms: Piperacillin-tazobactam 4.5 g IV every 8 hours and Metronidazole 500 mg IV every 8 hours.

This regimen was continued for 14 days, after which she was transitioned to oral amoxicillin-clavulanate 875/125 mg twice daily for 4 weeks. The total 6-week antibacterial course was selected because the lesion represented chronic osteomyelitis rather than necrotizing fungal infection, the biopsy had already removed loose devitalized tissue, and the patient showed early clinical improvement

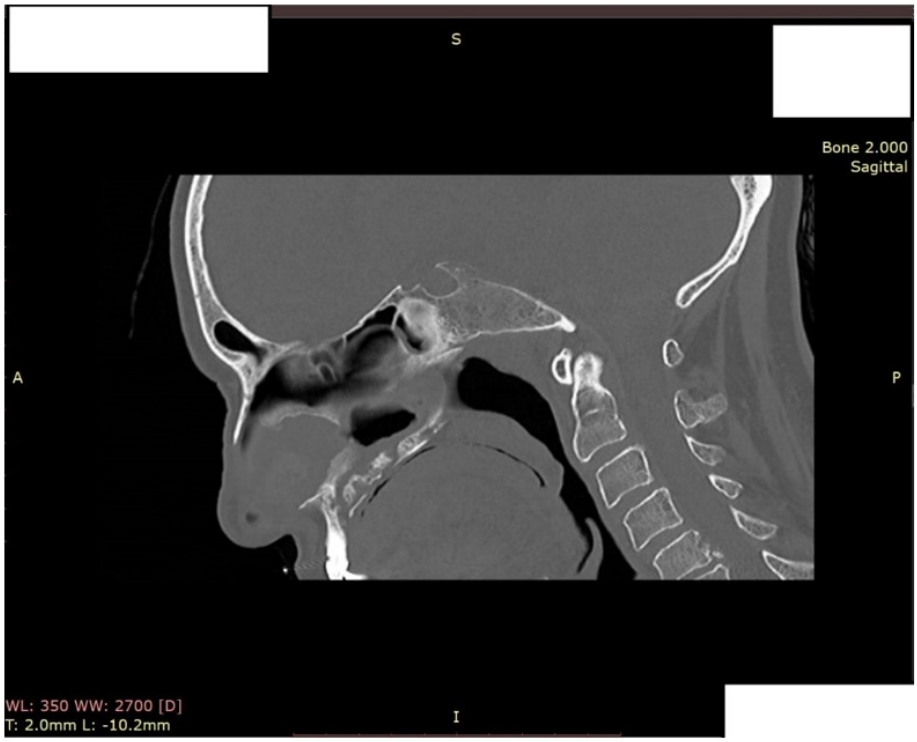

Figure 2: Sagittal CT paranasal sinus bone-window image demonstrating more conspicuous palatal destruction with communication toward the oral cavity, consistent with an oronasal defect.

without persistent fever, orbital deterioration, cranial neuropathy, or hemodynamic instability. Glycemic control was optimized with regular insulin, 8 units subcutaneously three times daily before meals, and basal insulin, 24 units subcutaneously at night.

Antifungal therapy was not started because biopsy and stains did not support fungal invasion. She also received frequent bedside glucose monitoring, saline mouth rinses, nutritional support, and local wound care. Loose necrotic fragments were removed during biopsy, which likely provided only limited debridement. Findings that would have prompted escalation included worsening facial pain or swelling, progressive exposure of bone or fistula enlargement, persistently elevated inflammatory markers, orbital signs, cranial nerve deficits, or repeat imaging showing ongoing extension; any of these would have prompted reconsideration of the regimen and formal surgical debridement.

6. Outcome, Follow-Up, and Timeline

The patient's pain began to improve within the first several days of treatment. The foul odor diminished, oral intake became easier, and fluid leakage between the mouth and nose gradually lessened. Over the next two weeks, the local inflammatory changes continued to regress, and the exposed bone became less prominent. By the end of treatment, the palatal soft tissues appeared healthier, and the patient was more clinically comfortable.

Overall improvement was observed after a combination of antibacterial therapy, glycemic control, and removal of loose necrotic fragments during biopsy. Because tissue sampling also had a limited debridement effect, the favorable course should not be attributed solely to medical therapy. Follow-up was limited to the period covered in this report, and longer-term assessment of fistula healing, recurrence, or the need for further debridement was not available in the chart, as shown in **(Figure 5)**.

7. Discussion

Osteomyelitis is an infective-inflammatory process of bone that may originate within the medullary spaces and extend through the Haversian canals to involve cortical bone and periosteum [8]. In the jaws, infection may arise from odontogenic sources, contiguous spread from adjacent soft tissue or sinus disease, or direct introduction of microorganisms following trauma or surgical disruption. The maxilla is less commonly affected than the mandible, but systemic compromise, such as poorly controlled diabetes, can predispose to infection by impairing host defenses and delaying healing [9–11].

This case is clinically important because the initial presentation and imaging strongly suggested rhino-maxillary mucormycosis, a diagnosis that must be considered urgently in diabetic patients. Unilateral sinonasal soft-tissue thickening, maxillary and palatal destruction, septal perforation, and extension into adjacent spaces are concerning findings that appropriately warrant urgent evaluation for invasive fungal disease [12]. At the same time, these findings are not specific for mucormycosis. Advanced bacterial osteomyelitis can produce a similar destructive pattern, particularly when diagnosis is delayed.

The differential diagnosis in this patient also included odontogenic infection, malignancy, granulomatous disease, actinomycosis, medication-related osteonecrosis, invasive fungal disease, and other necrotizing processes. Odontogenic infection was considered because chronic dental infection is a common route to maxillary osteomyelitis, but the patient had no recent extraction, periodontal

Table 1: Baseline Laboratory Investigations

Investigation	Result	Normal range	Interpretation
Hemoglobin	10.6 g/dL	12.0–15.5 g/dL	Mild anemia
Hematocrit	32.8%	36–46%	Low
RBC count	3.85 $\times 10^6$/μL	4.0–5.2 $\times 10^6$/μL	Slightly reduced
Total leukocyte count	13.9 $\times 10^3$/μL	4.0–11.0 $\times 10^3$/μL	Leukocytosis
Neutrophils	82%	40–75%	Neutrophilia
Lymphocytes	12%	20–45%	Relative lymphopenia
Platelets	421 $\times 10^3$/μL	150–450 $\times 10^3$/μL	Reactive thrombocytosis
ESR	74 mm/hr	0–20 mm/hr	Markedly elevated
CRP	68 mg/L	<5 mg/L	Elevated
Random blood glucose	298 mg/dL	70–140 mg/dL	Poor glycemic control
Fasting blood glucose	184 mg/dL	70–99 mg/dL	Elevated
2-hour postprandial glucose	286 mg/dL	<140 mg/dL	Elevated
HbA1c	10.1%	<5.7%	Chronic uncontrolled diabetes
Urine glucose	Positive	Negative	Hyperglycemia
Urine ketones	Negative	Negative	No ketoacidosis
Urea	28 mg/dL	15–40 mg/dL	Normal
Creatinine	0.9 mg/dL	0.6–1.2 mg/dL	Normal
Sodium	136 mmol/L	135–145 mmol/L	Normal
Potassium	4.2 mmol/L	3.5–5.0 mmol/L	Normal
Chloride	101 mmol/L	98–107 mmol/L	Normal
Bicarbonate	24 mmol/L	22–28 mmol/L	Normal
ALT	24 U/L	7–56 U/L	Normal
AST	28 U/L	10–40 U/L	Normal
ALP	118 U/L	44–147 U/L	Normal
Total bilirubin	0.7 mg/dL	0.2–1.2 mg/dL	Normal
Serum albumin	3.3 g/dL	3.5–5.0 g/dL	Mildly low

Abbreviations: ALT, alanine aminotransferase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; HbA1c, glycated hemoglobin; RBC, red blood cell.

Table 2: Diagnostic Studies

Investigation	Result
Non-contrast CT of the paranasal sinuses	Lobulated soft tissue thickening in the left nasal cavity and left paranasal sinuses with widening of the osteomeatal unit; erosion of the lateral wall of the left maxillary antrum; extension into the retroantral space, infratemporal fossa, and inferior orbital fissure; multifocal erosions of the hard palate with oronasal fistula; ill-defined lucencies in the greater wing and adjacent body of the left sphenoid bone; perforation of the posteroinferior nasal septum
ENT nasal endoscopy	Inflamed, unhealthy sinonasal mucosa with destructive changes on the left side
Tissue fungal smear (KOH mount)	Negative for fungal elements
PAS stain	Negative
GMS stain	Negative
Histopathology	Necrotic and sclerotic bone, empty lacunae, chronic inflammatory infiltrate, bacterial colonies
Tissue culture	Deep tissue biopsy culture obtained before antibiotics: mixed aerobic and anaerobic oral flora
Blood culture	No growth

Abbreviations: CT, computed tomography; ENT, ear, nose, and throat; GMS, Grocott methenamine silver; KOH, potassium hydroxide; PAS, periodic acid–Schiff.

disease, or other clear odontogenic trigger. Malignancy was considered because destructive maxillary lesions may represent squamous cell carcinoma or lymphoma, yet the biopsy lacked malignant cells and instead showed necrotic bone with chronic inflammation. Granulomatous disease and actinomycosis were less likely because histology did not show granulomas or sulfur granules. Medication-related osteonecrosis was unlikely because she denied antiresorptive therapy exposure. Invasive fungal disease was initially a major concern given uncontrolled diabetes and the destructive imaging findings; however, fungal stains were negative, and no hyphae or angioinvasion were identified. Taken together, chronic maxillary osteomyelitis with secondary bacterial infection remained the most likely diagnosis.

Table 3: Clinical Timeline of Presentation, Diagnosis, and Management

Time point	Clinical event
Approximately 2 weeks before presentation	Onset of left maxillary pain, unilateral nasal obstruction, foul oral odor, and oral–nasal leakage while drinking.
Presentation / Day 0	Outpatient assessment, clinical suspicion of destructive unilateral maxillofacial disease, and admission for workup.
Day 0	Non-contrast CT of the paranasal sinuses demonstrated destructive unilateral left maxillary–palatal sinonasal disease with skull-base extension.
Day 0–1	An urgent endoscopic assessment and biopsy of the palatal/maxillary lesion were performed; cultures were obtained from deep tissue before antibiotics were administered.
Day 1–2	KOH, PAS, and GMS stains were negative for fungal elements; histopathology supported the diagnosis of chronic osteomyelitis; culture yielded mixed aerobic and anaerobic oral flora.
Days 0–14	Intravenous piperacillin-tazobactam plus metronidazole, with insulin-based glycemic optimization and local wound care.
After IV phase	Transition to oral amoxicillin-clavulanate for 4 additional weeks.
Discharge and last available follow-up	Discharged after clinical improvement and step-down to oral therapy; later follow-up in the available chart showed ongoing symptomatic improvement, but longer-term outcome was not documented.

Abbreviations: CT, computed tomography; ENT, ear, nose, and throat; IV, intravenous.

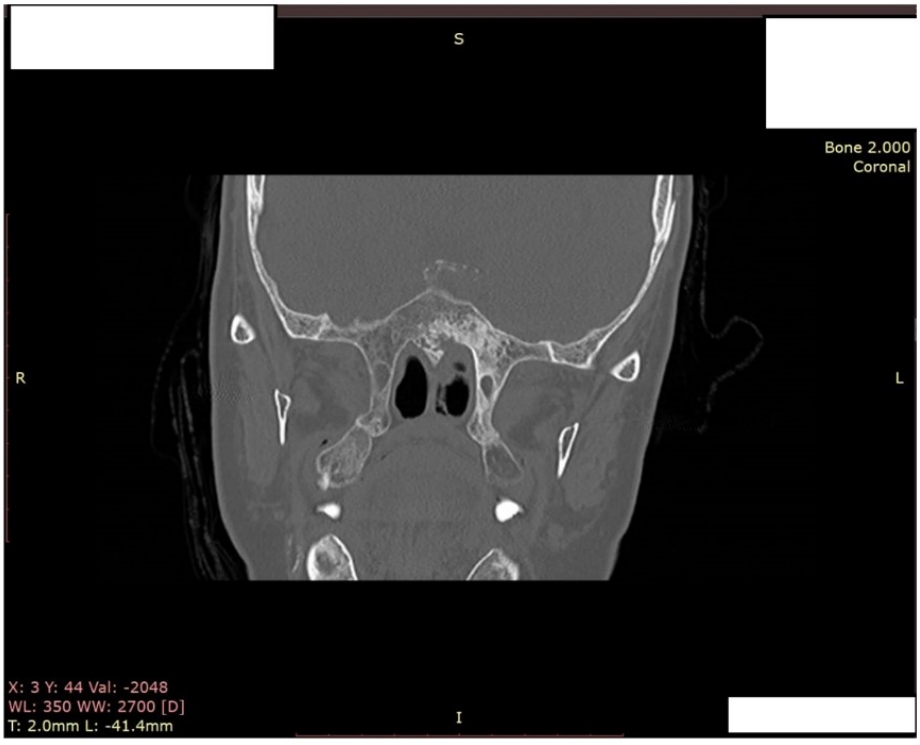

Figure 3: Coronal CT paranasal sinus bone-window image showing unilateral left-sided sinonasal soft tissue thickening and extensive osseous erosion of the maxillary-palatine complex.

The maxilla is less commonly affected by osteomyelitis than the mandible because of its comparatively rich blood supply [13]. Even so, uncontrolled diabetes creates a favorable environment for chronic infection by impairing neutrophil function, delaying healing, and reducing tissue resistance [14]. Once established, osteomyelitis may extend across the maxillary alveolus, hard palate, sinus walls, and adjacent spaces, producing the impression of invasive fungal disease [15].

The diagnostic distinction in this case depended on tissue examination. Histopathology showed necrotic and sclerotic bony trabeculae, empty lacunae, chronic inflammatory infiltrate, and bacterial colonies, while fungal stains did not demonstrate hyphae. Taken together, these findings supported a diagnosis of chronic maxillary osteomyelitis with secondary bacterial infection rather than tissue-proven mucormycosis. The biopsy also removed loose necrotic fragments, which may have contributed to local improvement alongside medical therapy.

The extent of radiologic destruction in this patient was substantial, involving the maxillary sinus, palate, nasal septum, and adjacent spaces. Such spread is worrisome and justifies close clinical monitoring, multidisciplinary input, and a low threshold for escalation if orbital, neurologic, or skull-base features emerge. In this case,

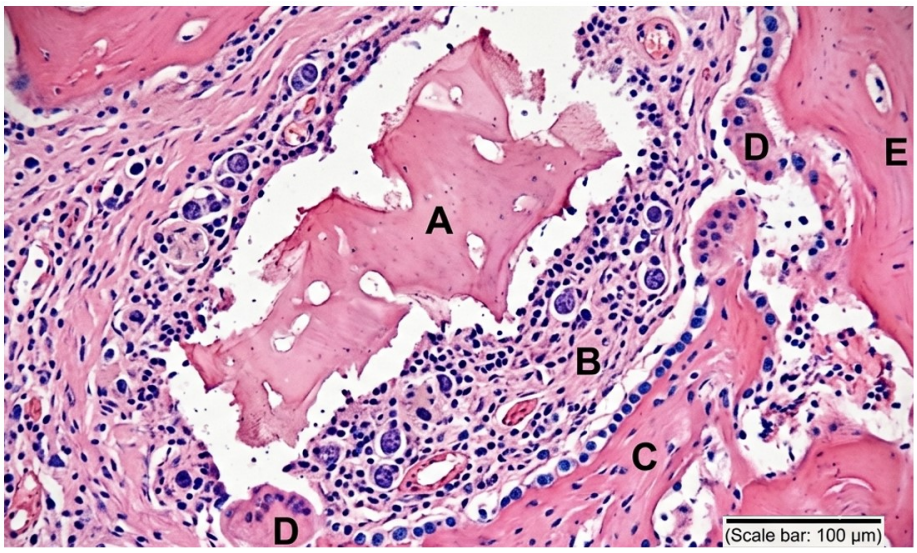

Figure 4: Histopathology of chronic osteomyelitis on H&E stain: (A) sequestrum showing a devitalized necrotic bone fragment lacking viable osteocytes; (B) chronically inflamed stroma with dense infiltrate of plasma cells, lymphocytes, and macrophages; (C) reactive new bone formation (involucrum) surrounding the infected area; (D) osteoclast-type multinucleated giant cells indicating active bone resorption; (E) residual viable bone with preserved cellular architecture.

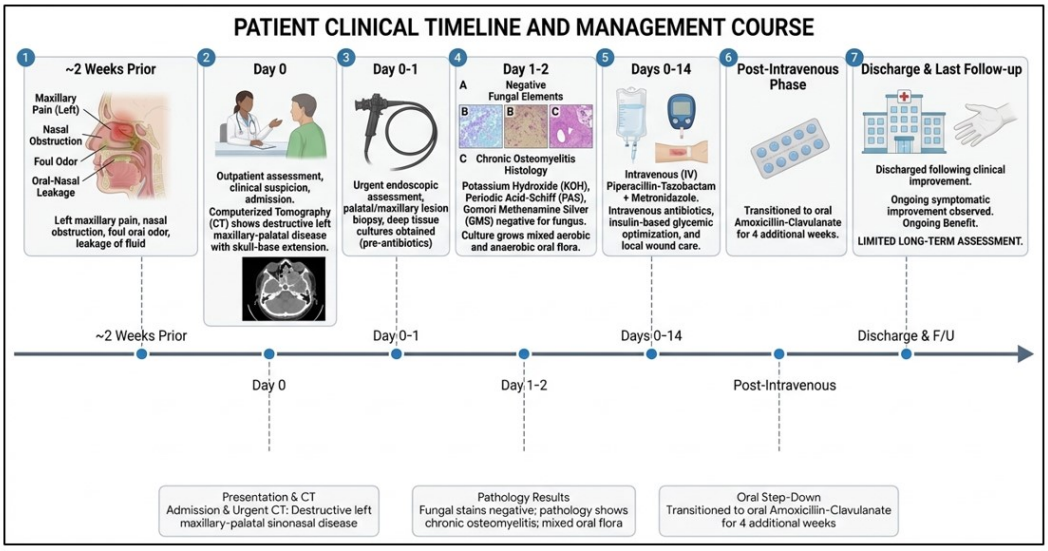

Figure 5: Patient Clinical Timeline and Management Course. The central horizontal arrow illustrates the clinical timeline from symptom onset to final follow-up. The seven distinct panels across the top outline the sequential diagnostic findings and therapeutic interventions. (1) Symptom onset 2 weeks before presentation, including detailed facial pain, odor, and oral-nasal leakage locations. (2) Day 0 outpatient assessment and admission with non-contrast computerized tomography (CT) imaging. (3) Day 0-1 urgent endoscopic assessment and deep-tissue biopsy. (4) Day 1-2 pathologic results, including negative fungal stains (KOH, Potassium Hydroxide; PAS, Periodic Acid-Schiff; GMS, Gomori Methenamine Silver) and mixed oral flora. (5) Days 0-14 intensive inpatient phase with intravenous (IV) piperacillin-tazobactam plus metronidazole, insulin-based glycemic optimization, and wound care. (6) Post-IV step-down phase to oral amoxicillin-clavulanate for 4 additional weeks. (7) Discharge status and last documented follow-up. Summary boxes below the linear timeline summarize key findings.

bedside examination did not show gross orbital involvement, and the patient improved without extensive ablative surgery. Nevertheless, the extent of radiological destruction should be interpreted in light of its clinical context and the limits of the available follow-up. The patient did not have additional MRI or formal neuro-ophthalmic staging, which is a limitation of this report.

Management focused on the most likely bacterial and metabolic contributors. Broad-spectrum antibiotics were selected to cover mixed oral aerobes and anaerobes, and glycemic control was optimized because uncontrolled diabetes can impair host defense and delay healing. The favorable response supports the final clinicopathologic diagnosis, but the improvement cannot be attributed to antibiotics alone, as the biopsy also removed some devitalized tissue. For that reason, it is more accurate to describe the procedure as having a limited debridement effect rather than as purely diagnostic.

The main teaching point from this case is that destructive unilateral maxillary disease in a diabetic patient should prompt urgent evaluation for mucormycosis. Still, the final diagnosis must rest on tissue confirmation. Overcalling mucormycosis on imaging alone risks unnecessary antifungal therapy, while under-recognizing invasive

fungal disease can be dangerous. This case therefore reinforces the need for multidisciplinary assessment, careful clinicopathologic correlation, and balanced interpretation of imaging, microbiology, and histology.

8. Conclusion

A destructive unilateral maxillary lesion in a patient with uncontrolled diabetes is not synonymous with mucormycosis. Chronic maxillary osteomyelitis can closely resemble invasive fungal sinusitis clinically and radiologically, particularly when palatal destruction and sinonasal extension are present. In this case, biopsy findings supported chronic osteomyelitis with secondary bacterial infection and no fungal invasion on special stains. The patient improved with antibacterial therapy, glycemic control, and limited removal of loose necrotic fragments during biopsy. This case highlights the importance of prompt tissue diagnosis before committing to a specific etiologic label in aggressive maxillofacial disease.

9. Patient Perspective

At the time my symptoms began, I was very worried because the pain, bad smell, and leakage of liquids from my mouth into my nose were getting worse each day. When I was told that a serious fungal infection was being considered, I became frightened about what might happen. During my admission, the doctors explained each test clearly and started treatment quickly. I received intravenous medicines for the infection and insulin injections to control my diabetes. The staff regularly checked on me, managed my pain, and helped me understand the importance of keeping my blood sugar under control.

After several days of treatment, I noticed that the pain had reduced and the unpleasant smell had started to improve. Eating and drinking gradually became easier, and I felt more comfortable overall. I was relieved when the biopsy showed it was not the fungal infection initially suspected. I am thankful to the medical team for identifying the real cause of my illness and helping me recover. This experience also made me realize how important it is to take diabetes treatment regularly and attend follow-up appointments.

Conflicts of Interest

The authors declare no competing interests that could have influenced the objectivity or outcome of this research.

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Informed Consent

Written informed consent was obtained for publication of clinical details and imaging in anonymized form.

Large Language Model

None.

Author Contributions

UFK conceived the case report, managed the patient, supervised the work, and critically revised the manuscript for important intellectual content. MS conducted the literature review, interpreted the clinical findings, drafted the manuscript, prepared the figures and tables, coordinated the submission process, and served as the corresponding author. JA contributed to data collection, literature review, manuscript revision, and interpretation of clinical findings. AA contributed to pathological interpretation, critically reviewed the manuscript, and approved the final version for publication. All authors read and approved the final manuscript and agree to be accountable for all aspects of the work.

Data Availability

None.

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