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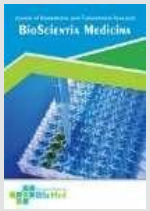

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Masquerading as an Orbital Malignancy: A Rare Presentation of Pott's Puffy Tumor with Intraorbital Extension in a Diabetic Adult

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A B S T R A C T

Background: Pott's puffy tumor (PPT) is a rare, life-threatening clinical entity characterized by frontal bone osteomyelitis and subperiosteal abscess, typically resulting from untreated frontal sinusitis. While predominantly a pediatric diagnosis, adult presentation is exceptionally rare and often associated with immunocompromised states. The clinical mimicry of PPT, particularly when presenting with bone destruction and orbital extension, frequently leads to misdiagnosis as a malignant neoplasm. This study aims to report a rare and instructive case of Pott's puffy tumor in a 52-year-old diabetic female. **Case presentation:** We report a case of a 52-year-old female with uncontrolled Type 2 Diabetes Mellitus presenting with progressive left ocular proptosis, globe displacement, and blurred vision persisting for two months. Imaging revealed a heterogeneous mass in the frontoethmoidal sinus with extensive osteolytic destruction of the orbital roof and frontal bone, initially raising strong suspicion of a sinonasal malignancy or metastasis. The patient underwent a bicoronal craniectomy and debridement. Intraoperative findings revealed a purulent subperiosteal collection and necrotic bone, confirming the diagnosis of PPT with intraorbital extension. The defect was repaired via craniofacial reconstruction using bone cement. Post-operative culture analysis confirmed a polymicrobial infection. **Conclusion:** This case underscores the necessity of maintaining a high index of suspicion for PPT in diabetic adults presenting with proptosis and osteolytic radiographic findings. Although rare, PPT can masquerade as a malignancy. Early recognition and a multidisciplinary approach combining aggressive surgical debridement with targeted antibiotic therapy are imperative to prevent catastrophic intracranial and orbital complications.

1. Introduction

The clinical entity known as Pott's puffy tumor (PPT) represents a fascinating and formidable intersection of otolaryngology, ophthalmology, and neurosurgery.[1] First elucidated by the eminent British surgeon Sir Percivall Pott in 1768, the condition was originally described in the context of direct cranial trauma. In his seminal work, Pott detailed a phenomenon whereby a "puffy, circumscribed, indolent tumor" of the scalp would manifest days or weeks following a blow to the forehead, signaling the separation of the pericranium from the skull due to underlying osteomyelitis. While Pott's original cohort primarily comprised trauma victims, the post-antibiotic era has seen a paradigm shift in the etiology of this disease.[2] Today, PPT is almost exclusively recognized as a complication of untreated or partially treated frontal sinusitis, transforming it from a surgical curiosity of the battlefield to a complex infectious disease challenge in modern rhinology and ophthalmology.[3] Despite the precipitous decline in its incidence following the advent of broad-spectrum

penicillin and cephalosporins in the mid-20th century, PPT remains a diagnosis of critical importance due to its potential for catastrophic intracranial sequelae.

The distinct pathophysiology of PPT distinguishes it from routine sinusitis. The frontal sinus, unlike the maxillary or ethmoid sinuses, shares a unique and perilous vascular relationship with the marrow of the frontal bone. The physiological driver of this condition is the retrograde spread of infection through the valveless diploic veins of Breschet.[4] These venous channels, which traverse the diplöe (the spongy cancellous bone between the inner and outer tables of the skull), facilitate a bidirectional flow of blood. In the setting of acute or chronic frontal sinusitis, mucosal inflammation can lead to septic thrombophlebitis within these mucosal venules. Because these veins lack valves, increased intrasinus pressure—often exacerbated by obstructed ostia—forces septic thrombi to propagate retrograde into the diploic system. This process instigates a suppurative osteomyelitis that necroses the anterior table of the frontal sinus. Once the infection breaches the outer cortex, purulent material accumulates beneath the periosteum, creating the pathognomonic boggy, fluctuating swelling on the forehead—the "puffy tumor". Crucially, this same valveless venous network communicates posteriorly with the dural venous sinuses, placing the patient at immediate risk for epidural abscess, subdural empyema, and cavernous sinus thrombosis.[5]

Epidemiologically, PPT has long been considered a disease of adolescence. This age predilection is rooted in the developmental anatomy of the frontal sinus and cranial vascularity.[6] The frontal sinus begins to pneumatize around the age of two but does not reach full maturation until late adolescence. During this period of rapid growth, the vascularity of the diploic venous system is at its peak to support osteogenesis. Consequently, the high blood flow facilitates the rapid hematogenous dissemination of bacteria from the sinus mucosa to the bone marrow. In contrast, the adult frontal bone undergoes a process of marrow conversion, becoming less vascular and more sclerotic, which theoretically confers a resistance to osteomyelitic spread. This physiological maturation explains why adult-onset PPT is an epidemiological paradox, accounting for a small fraction of reported cases globally. When PPT does manifest in the adult population, it is rarely an isolated event; rather, it is almost invariably a signal of systemic immune failure or significant local anatomical disruption.[7]

The rarity of adult PPT necessitates a rigorous search for predisposing comorbidities that impair host defense mechanisms. Among these, diabetes mellitus (DM) emerges as a potent and prevalent risk factor, yet its specific role in the pathogenesis of PPT is often underappreciated in general practice.[8] The diabetic microenvironment creates a "perfect storm" for complicated sinusitis. Chronic hyperglycemia induces a state of immunosenescence, characterized by distinct defects in neutrophil chemotaxis, phagocytosis, and intracellular bactericidal activity. This impairment prevents the effective containment of mucosal pathogens within the sinus cavity. Furthermore, diabetes-associated microangiopathy compromises the perfusion of the sinus periosteum and bone, reducing the local bioavailability of systemic antibiotics and fostering a hypoxic tissue environment. This hypoxia is significant because it selects for anaerobic pathogens—such as *Bacteroides*, *Fusobacterium*, and *Peptostreptococcus*—which are notorious for causing indolent, destructive osteomyelitis that is resistant to standard aerobic antibiotic regimens. Thus, in the diabetic adult, what begins as a routine sinus infection can smolder subclinically for months or years, slowly eroding bone architecture before presenting as a catastrophic structural failure.

The clinical presentation of PPT in adults can be deceptive, often lacking the florid acute signs seen in children. While the classic triad consists of headache, fever, and frontal swelling, adult cases may present indolently with vague, chronic headaches or orbital complaints, masking the severity of the underlying bone infection. This diagnostic ambiguity reaches its apex when the infection erodes the orbital roof, leading

to intraorbital extension. In such scenarios, the radiographic appearance of PPT can be indistinguishable from aggressive sinonasal malignancies. The lytic destruction of the frontal bone and orbital roof, combined with a soft tissue mass extending into the orbit and compressing the globe, mimics the behavior of squamous cell carcinoma, esthesioneuroblastoma, or metastatic disease. Radiologists and surgeons viewing a Computed Tomography (CT) scan showing "bone destruction" and "soft tissue mass" are conditioned to prioritize neoplasia in the differential diagnosis, particularly in older adults. This "mimicry of malignancy" is a dangerous pitfall; mislabeling a subperiosteal abscess as a tumor can lead to delays in source control, inappropriate biopsy attempts that risk disseminating infection, or the initiation of oncological protocols that fail to address the urgent need for drainage.[9]

The distinction between an infectious "pseudotumor" and a true neoplasm is not merely academic; it dictates the surgical urgency and approach. Malignancies typically require extensive staging and planned resection with wide margins, whereas PPT demands immediate, aggressive debridement and drainage to prevent blindness or death. The orbital extension of PPT places the optic nerve at risk of compression ischemia or septic neuritis, while the proximity to the anterior cranial fossa keeps the threat of meningitis or cerebral abscess ever-present. Differentiating these entities requires a nuanced understanding of radiological signs—such as the "rim-enhancement" of abscesses versus the solid enhancement of tumors—and a heavy reliance on clinical correlation. However, in resource-limited settings or complex presentations, the diagnosis is frequently confirmed only intraoperatively, underscoring the need for surgical adaptability.[10]

Against this background of historical significance and diagnostic complexity, the present study aims to report a rare and instructive case of Pott's puffy tumor in a 52-year-old diabetic female. Unlike the classic pediatric presentation, this case is notable for its extensive osteolytic destruction of the orbital roof and frontal bone, which masqueraded clinically and radiologically as a malignant orbital neoplasm. The novelty of this report lies in three key areas: (1) Demographic rarity: It documents the occurrence of PPT in a demographic (older adult) where the disease is statistically improbable, highlighting the critical role of diabetes in altering the natural history of cranial osteomyelitis; (2) Diagnostic mimicry: It provides a detailed analysis of the "malignancy masquerade," illustrating how severe bone destruction in chronic infection can deceive even experienced clinicians and how to navigate this diagnostic dilemma; (3) Surgical management: It demonstrates the efficacy of a single-stage, multidisciplinary approach utilizing immediate radical debridement and antibiotic-impregnated bone cement reconstruction, challenging the traditional dogma of delayed reconstruction in the setting of active infection. By dissecting the intersection of chronic immunosuppression, anatomical vulnerability, and diagnostic mimicry, this report seeks to propose a heightened index of suspicion and a refined diagnostic algorithm for adult patients presenting with destructive fronto-orbital lesions.

2. Case Presentation

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A 52-year-old female presented to the Oculoplastics and Orbital Oncology clinic at Dr. M. Djamil General Hospital with a primary complaint of progressive protrusion of the left eye (proptosis) over the preceding two months. The patient reported an associated dull, throbbing headache localized to the left frontal region and a gradual blurring of vision in the affected eye. The patient's history was significant for recurrent episodes of epistaxis and chronic nasal congestion persisting for approximately 15 years, for which she had self-medicated with over-the-counter decongestants without seeking formal otolaryngologic evaluation. Two months prior to admission, she noticed swelling of the left upper eyelid, which she initially attributed to a minor trauma she could not

recall. The swelling progressively hardened and displaced the globe inferiorly and outwardly. She denied current fever, chills, or purulent nasal discharge at the time of presentation. The patient had a 10-year history of type 2 diabetes mellitus and hypertension. Her adherence to medication was poor. She admitted to irregular use of oral hypoglycemic agents (metformin 500mg) and antihypertensives (amlodipine 10mg).

On general examination, the patient appeared non-toxic but anxious (Table 1). Vital signs were stable; Blood Pressure 140/90 mmHg, Heart Rate 88 bpm, Respiratory Rate 20/min, and Temperature 37.2°C (afebrile). Ophthalmologic examination revealed; (1) Visual Acuity (VA): Right Eye (OD) was 1/300 (Hand Motion) due to a mature senile cataract. Left Eye (OS) was 20/50, corrected with a posterior chamber intraocular lens (pseudophakia); (2) Inspection: Gross non-axial proptosis of the left eye was evident with significant inferolateral displacement (exotopia). A fluctuating, tender, doughy swelling was palpable over the left brow and frontal region, measuring approximately 4 cm x 3 cm (Figure 1); (3) Hertel Exophthalmometry: Base 105mm; OD 14mm, OS 24mm, indicating 10mm of relative proptosis; (4) Extraocular Movements (EOM): Significant restriction was noted in the left eye, particularly in supraduction (-3) and adduction (-2), consistent with a mass effect in the superomedial orbit; (5) Anterior Segment: OS conjunctiva was hyperemic (chemosis grade 2). The cornea was clear. Pupil reflexes were brisk and equal; (6) Posterior Segment: Dilated fundus examination of the left eye revealed tortuous veins and mild disc edema, suggestive of orbital congestion. No signs of proliferative diabetic retinopathy were observed.

Hematological analysis revealed a leukocytosis (White Blood Cell count: 14,500/µL) with a neutrophilic shift (82% neutrophils). Inflammatory markers were elevated: Erythrocyte Sedimentation Rate (ESR) was 65 mm/hr, and C-Reactive Protein (CRP) was 42 mg/L. Metabolic profile indicated uncontrolled diabetes with a random blood glucose of 285 mg/dL and Glycated Hemoglobin (HbA1c) of 9.8%. Renal and liver function tests were within normal limits.

Figure 1. Clinical photo of patient.

A non-contrast and contrast-enhanced computed tomography (CT) scan of the orbit and paranasal sinuses was performed. The scan demonstrated a heterogeneous, non-enhancing soft tissue mass occupying the left frontal and ethmoid sinuses. Crucially, there was frank destruction of the anterior and posterior tables of the frontal sinus, as well as erosion of the superior orbital roof. The mass extended into the superomedial aspect of the left orbit, compressing the superior rectus muscle and displacing the optic nerve sheath complex inferiorly. The presence of aggressive bone destruction led to a primary radiological differential diagnosis of a sinonasal malignancy such as squamous cell carcinoma or a metastatic lesion. However, the presence of rim-enhancing fluid collections within the

frontal sinus raised the possibility of a complex abscess. Pre-operative diagnosis in this patient was; (1) primary: tumor of the left frontoethmoidal sinus with orbital extension (suspect malignancy versus mucocele); (2) secondary: uncontrolled type 2 diabetes mellitus, hypertension; (3) differential: Pott's puffy tumor, sinonasal carcinoma, metastatic lesion.

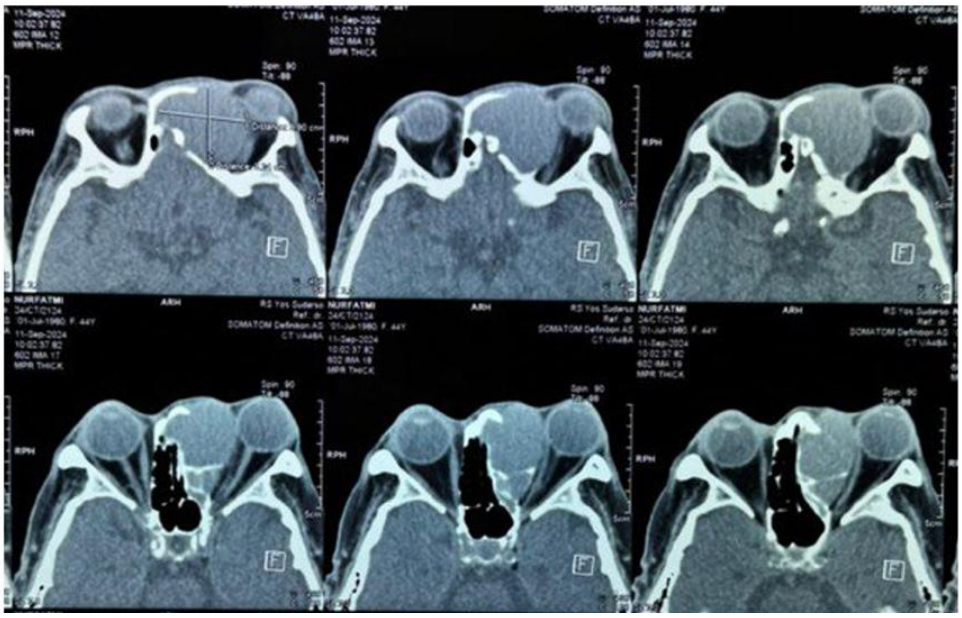

Figure 2. CT scan of orbit and paranasal sinuses.

Given the orbital compression and the ambiguity of the diagnosis (tumor versus infection), surgical intervention was deemed urgent. A multidisciplinary team comprising Ophthalmology and Neurosurgery was assembled (Table 2). The patient underwent a combined trans-cranial approach under general anesthesia. A bicoronal skin incision was made to expose the frontal bone. Upon reflecting the scalp flap, a purulent collection was encountered immediately beneath the periosteum, confirming a subperiosteal abscess. The anterior table of the frontal sinus was necrotic and friable. Upon removal of the necrotic bone (craniectomy), copious purulent material (approx. 30cc) was drained from the frontal sinus. The sinus mucosa was thickened and polypoid. The infection had eroded the posterior table (exposing the dura, which was intact) and the orbital roof. The purulent material extending into the superior orbit was carefully evacuated, relieving pressure on the globe. After thorough irrigation with antibiotic saline and hydrogen peroxide, the sinus was cranialized. The defect in the frontal bone was reconstructed using polymethyl methacrylate (PMMA) bone cement to restore the cosmetic contour of the forehead and protect the intracranial contents. Tissue samples and bone fragments were sent for histopathological examination and microbiological culture. On histopathology evaluation, sections showed fragments of lamellar bone showing lacunae empty of osteocytes (bone necrosis) surrounded by dense inflammatory infiltrates composed of neutrophils, plasma cells, and lymphocytes. Fibrosis and neovascularization were present. No evidence of malignant cells or granulomas was found; conclusion: chronic osteomyelitis. Culture of the purulent material yielded *Staphylococcus aureus* sensitive to Ceftriaxone and Clindamycin, and *Bacteroides* species.

Table 1. Summary of patient clinical findings on admission

DOMAIN	DETAILED FINDINGS
PATIENT PROFILE & VITALS	
Demographics	52-year-old Female
Chief Complaint	Progressive **proptosis (left eye)** for 2 months, dull frontal headache, blurred vision.
Vital Signs	BP: **140/90 mmHg** (Elevated) | HR: 88 bpm | RR: 20/min | Temp: 37.2°C (Afebrile)
Comorbidities	• Type 2 Diabetes Mellitus (Uncontrolled, 10-year history) • Hypertension (Poor adherence to medication)
OPHTHALMOLOGIC EXAMINATION (LEFT EYE - OS)	
Visual Acuity	20/50 (Corrected with IOL)
External Inspection	Gross **non-axial proptosis** with inferolateral displacement (exotopia). Palpable mass: Fluctuating, tender, doughy swelling over left brow/frontal region (approx. 4cm x 3cm).
Hertel Exophthalmometry	**OS: 24mm** (vs OD: 14mm) **Relative Proptosis: 10mm** (Base: 105mm)
Extraocular Movements (EOM)	Significant restriction detected: • Supraduction: **-3 restriction** • Adduction: **-2 restriction**
Anterior & Posterior Segment	**Anterior:** Chemosis (Grade 2), Cornea clear. **Posterior:** Tortuous veins, mild disc edema (suggestive of orbital congestion).
LABORATORY & RADIOLOGICAL INVESTIGATION	
Hematology & Inflammatory Markers	• WBC: **14,500/µL** (Leukocytosis) • Neutrophils: **82%** (Left shift) • ESR: **65 mm/hr** (Elevated) • CRP: **42 mg/L** (Elevated)
Metabolic Profile	• Random Blood Glucose: **285 mg/dL** • HbA1c: **9.8%** (Indicates poor long-term glycemic control)
CT Scan Findings (Orbit & Sinus)	• **Mass:** Heterogenous, non-enhancing soft tissue mass in left frontoethmoidal sinus. • **Bone Destruction:** **Frank lysis** of anterior/posterior frontal tables and superior orbital roof. • **Extension:** Intraorbital extension compressing superior rectus muscle and displacing optic nerve inferiorly. • **Specific Sign:** Rim-enhancing fluid collections suggestive of abscess.

The patient was started on empirical intravenous Ceftriaxone 2g daily and Metronidazole 500mg every 8 hours, which was continued based on culture sensitivity. Post-operative CT scans showed successful evacuation of the abscess and restoration of the frontal contour. Clinically, the proptosis resolved significantly by Day 5 post-op (Hertel OS: 18mm). Visual acuity remained stable at 20/50. The eyelid edema subsided, and extraocular motility improved to the full range of motion. The patient was discharged on Day 14 with oral Ciprofloxacin and Clindamycin to complete a 6-week course. At the 3-month follow-up, the patient remained asymptomatic with no recurrence of swelling, and glycemic control was optimized (HbA1c 7.5%) in collaboration with internal medicine.

Table 2. Diagnosis, treatment, followup and outcome

MANAGEMENT PHASE	CLINICAL DETAILS & ACTION
	DIAGNOSTIC CONFIRMATION
Pre-operative Diagnosis	Suspected Tumor of Left Frontoethmoidal Sinus with Orbital Extension (DDx: Malignancy vs. Mucocele) + Uncontrolled Type 2 DM.
Definitive Diagnosis	**Pott's Puffy Tumor** with Intraorbital Abscess and Chronic Osteomyelitis.
Histopathology	Chronic Osteomyelitis: Lamellar bone necrosis, dense inflammatory infiltrates (neutrophils/plasma cells), fibrosis. **Negative for malignancy.**
Microbiology	**Polymicrobial Infection:** 1. *Staphylococcus aureus* (Aerobe) 2. *Bacteroides* species (Anaerobe) *Sensitivity:* Ceftriaxone, Clindamycin.
	SURGICAL INTERVENTION
Procedure	Combined Trans-cranial Approach (Bicoronal Incision).
Intra-operative Actions	• **Drainage:** Evacuation of 30cc subperiosteal purulence from frontal sinus and superior orbit. • **Debridement:** Removal of necrotic anterior/posterior frontal tables and orbital roof. • **Reconstruction:** Sinus cranialization + Frontal bone defect repair using **PMMA Bone Cement**.
	MEDICAL MANAGEMENT
In-patient Regimen	• IV Ceftriaxone 2g daily • IV Metronidazole 500mg q8h • Insulin therapy for strict glycemic control.
Discharge Regimen	• Oral Ciprofloxacin + Clindamycin • Duration: Total 6-week course to treat osteomyelitis.
	FOLLOW-UP & OUTCOME
Post-op Day 5	• **Significant resolution** of proptosis (Hertel OS: 18mm). • Full Extraocular Motility (EOM) restored. • Eyelid edema subsided.
3-Month Follow-up	• **Anatomical:** Excellent cosmetic contour of forehead; no recurrence of swelling. • **Functional:** Vision stable (20/50); asymptomatic. • **Systemic:** Glycemic control improved (**HbA1c reduced to 7.5%**).

3. Discussion

The case presented herein highlights a classical pathology, Pott's puffy tumor (PPT), manifesting in an atypical demographic with features that deceptively mimicked a high-grade orbital malignancy.[11] To fully appreciate the complexity of this case, one must revisit the unique vascular anatomy that underpins the pathogenesis of this condition. The frontal sinus is not merely an air-filled cavity; it is intimately connected to the cranial marrow space via the diploic veins of Breschet. These veins are anatomically distinct because they are devoid of valves and possess thin, non-contractile walls.[12] This structural characteristic allows for bidirectional blood flow, which is dictated primarily by pressure gradients rather than hemodynamic regulation. In the physiological state, these veins drain the frontal sinus mucosa into the dural venous sinuses. However, in the setting of acute or chronic frontal sinusitis, the obstruction of the nasofrontal duct leads to the accumulation of purulent secretions and a subsequent rise in intramedullary pressure. This pressure reversal forces septic emboli—clusters of bacteria and inflammatory debris—to propagate retrograde from the sinus mucosa into the diploic venous system. The ensuing process is a septic thrombophlebitis that occludes the nutrient vessels of the frontal bone. Deprived of its blood supply and overwhelmed by bacterial invasion, the anterior table of the frontal sinus undergoes septic necrosis. Once the infection erodes through the outer cortex, it creates a subperiosteal abscess, clinically visible as the "puffy tumor" on the forehead.[13]

However, the danger of this valveless system lies in its posterior communication. The same veins that drain anteriorly also communicate with the superior sagittal sinus and the cavernous sinus. Consequently, a patient with a seemingly localized frontal swelling is at immediate risk for catastrophic intracranial extension, including epidural abscess, subdural empyema, and meningitis. Our patient's presentation with orbital symptoms (proptosis) indicates that the infection followed an inferior trajectory, exploiting the thinness of the orbital roof—a pathway of least resistance—to breach the orbital periosteum (periorbita). This multidirectional spread underscores why PPT must be regarded not as a localized abscess, but as a potential precursor to pansinusitis and craniocerebral sepsis.[14]

The rarity of PPT in the adult population is a well-documented epidemiological phenomenon. A previous study noted that less than 20% of PPT cases occur in patients over the age of 50. This predilection for the pediatric and adolescent population is largely attributed to the vascular dynamics of the developing skull.[15] In children, the diploic venous system is highly vascularized to support rapid cranial growth and sinus pneumatization. This hypervascularity acts as a superhighway for hematogenous bacterial spread. Conversely, as the skull matures into adulthood, the marrow undergoes fatty conversion and sclerosis, and the diploic channels become less prominent, theoretically obliterating the pathway for osteomyelitic spread.

Therefore, the emergence of PPT in a 52-year-old female, as described in our case, represents an "Adult Paradox" that necessitates the presence of a significant systemic disruptor. In this patient, uncontrolled type 2 diabetes mellitus (with a critically elevated HbA1c of 9.8%) served as the catalyst that reactivated this pediatric pathway. The pathophysiology of diabetes-related osteomyelitis is multifactorial. First, chronic hyperglycemia induces a state of cellular immunodeficiency. It impairs the chemotaxis (migration) of neutrophils to the site of infection and reduces their phagocytic and intracellular bactericidal capabilities. This failure of the innate immune response allows commensal sinus flora to proliferate unchecked. Secondly, and perhaps more critically, diabetes is characterized by microangiopathy—a thickening of the basement membrane of capillaries. This vascular compromise reduces perfusion to the sinus mucosa and the frontal bone, creating a distinct microenvironment: the "hypoxic sinus." This hypoxia has two profound consequences: it limits the bioavailability of systemic antibiotics at the tissue level, rendering standard

medical therapy ineffective, and it creates an ideal anaerobic culture medium. This explains the isolation of *Bacteroides* species in our patient. Anaerobes are notorious for their osteolytic potential; they produce enzymes that aggressively demineralize bone, leading to the "moth-eaten" destruction seen on this patient's CT scan. The chronicity of the patient's symptoms—15 years of recurrent epistaxis and congestion—combined with this immune dysfunction, allowed a smoldering, low-grade infection to slowly erode the dense cortical bone of the adult skull over years, culminating in the acute presentation of proptosis.[15]

One of the most instructive and perilous aspects of this case was the radiologic and clinical masquerade. The patient presented with a palpable mass, bone destruction, and displacement of the globe—a triad that classically screams "malignancy" in an adult patient. In the context of a chronic mass in a 52-year-old, the differential diagnosis is heavily weighted toward neoplastic entities such as squamous cell carcinoma of the sinus, esthesioneuroblastoma, lymphoma, or metastatic disease from a distant primary (such as breast or lung).[16] The extensive osteolysis involving the orbital roof and frontal tables is typically interpreted as a hallmark of aggressive neoplastic invasion.

This mimicry creates a cognitive bias known as the "malignancy first" algorithm. Radiologists and surgeons, trained to rule out the worst-case scenario, may misinterpret the lytic bone changes of chronic osteomyelitis as tumor erosion. However, subtle cues on computed tomography (CT) can aid in differentiation. A subperiosteal abscess typically presents as a rim-enhancing fluid collection with adjacent soft tissue stranding (cellulitis), whereas tumors tend to show solid, heterogeneous enhancement. Furthermore, the presence of air bubbles within the mass or the intracranial collection is pathognomonic for infection by gas-producing organisms. As noted by a previous study, differentiating osteomyelitis from neoplasia on CT alone is fraught with difficulty. Magnetic resonance imaging (MRI) is the gold standard modality in equivocal cases.[17] On MRI, an abscess will demonstrate restricted diffusion (bright on DWI sequences) due to the high cellularity of pus, a feature not typically seen in necrotic tumors. Additionally, MRI is superior in detecting dural inflammation (pachymeningitis) and delineating the abscess capsule. In our resource-limited setting, where MRI was not immediately available or cost-prohibitive, clinical acumen combined with the CT findings guided the decision. The presence of fluctuance on palpation (the "puffy" sign) rather than the hard, fixed nature of a carcinoma was a critical physical sign that pointed toward an infectious etiology, justifying the decision for urgent surgical exploration rather than a diagnostic biopsy. A biopsy in this setting carries the risk of disseminating infection or creating a sinocutaneous fistula if the diagnosis is indeed PPT.

Orbital involvement in PPT is a complication that is less common than intracranial extension but equally debilitating.[18] The orbit is a rigid, bony cone with limited volume. Any space-occupying lesion, whether purulent or neoplastic, rapidly increases intraorbital pressure, leading to a compartment syndrome. The infection in our patient spread to the orbit through a dehiscence in the orbital roof—a direct extension from the frontal sinus. Alternatively, infection can travel via the valveless superior and inferior ophthalmic veins, which communicate with the sinus vasculature.

The clinical consequences of this invasion were evident in our patient's significant proptosis (10mm relative) and ophthalmoplegia. The limitation of extraocular movement was mechanical, due to the mass effect of the abscess compressing the superior rectus muscle, but also potentially inflammatory, due to myositis. The gravest risk in such scenarios is Compressive Optic Neuropathy. The increased orbital pressure can compromise the vascular supply to the optic nerve or cause direct compression, leading to irreversible ischemia and blindness. Furthermore, the proximity of the abscess to the cavernous sinus poses a risk of septic thrombosis, a condition with high mortality. The immediate surgical decompression of

the orbit performed in this case likely salvaged the patient's vision and prevented the development of orbital apex syndrome. This highlights that PPT is not just a neurosurgical emergency but an ophthalmological one; timely decompression is vision-saving.

The management of PPT remains a subject of surgical debate, fluctuating between minimally invasive techniques and radical open approaches. In the modern era, Endoscopic Sinus Surgery (ESS) has gained popularity as a first-line treatment for uncomplicated frontal sinusitis. It preserves the frontal recess and avoids external scarring. However, the literature suggests that ESS is often insufficient for "complicated" PPT cases—specifically those with significant anterior table destruction, extensive subperiosteal collections, or inaccessible lateral extensions. In our case, the extensive osteolysis and the intraorbital component mandated a bicoronal approach (open craniectomy). This approach provided wide exposure, allowing for the complete evacuation of the abscess, thorough debridement of necrotic bone (sequestrectomy), and direct visualization of the dura to rule out macroscopic tear or epidural collection.[19]

Figure 3. Pathogenesis, diagnostic and management of adult Pott's puffy tumor.

A critical and controversial decision in this case was the immediate (single-stage) reconstruction of the frontal defect using bone cement (polymethyl methacrylate - PMMA). Historically, the dogma in osteomyelitis surgery was to perform a two-stage procedure: initial debridement and drainage, followed by delayed cranioplasty months later, once the infection was confirmed eradicated. This caution was driven by the fear that implanting foreign material into an infected field would form a biofilm, perpetuating the infection. However, recent literature challenges this conservative paradigm. Previous studies demonstrated that single-stage reconstruction using antibiotic-impregnated bone cement is safe and effective if thorough debridement is achieved. The exothermic reaction of PMMA curing releases local heat that may have bactericidal properties, and the material can be loaded with heat-stable antibiotics (like Tobramycin or Vancomycin) to provide high local concentrations of antimicrobial activity. In our patient, immediate reconstruction offered immense psychological and physiological benefits. It prevented the significant cosmetic deformity of a sunken forehead, which can be stigmatizing, and it restored the protective barrier for the intracranial contents, eliminating the "syndrome of the trephined" (headaches and dizziness due to atmospheric pressure changes on the brain). Our successful outcome—with no recurrence of infection at 3 months—supports the viability of aggressive, single-stage management in select adult patients, provided that the debridement is radical and systemic antibiotic coverage is adequate.

While this report provides a detailed examination of adult-onset PPT, it is limited by its design as a single case report. The findings regarding the safety of primary reconstruction cannot be generalized without larger cohort studies. Additionally, while we identified *Bacteroides* and *Staphylococcus*, molecular sequencing (PCR) of the pathogen was not performed. Molecular techniques could have provided further insight into the resistance genes and the specific microbiome of the diabetic sinus. Future research should focus on the role of biofilm formation on the frontal bone in diabetic patients and the comparative efficacy of antibiotic-eluting implants versus autologous bone grafts in infected fields.[20]

4. Conclusion

In summary, this report illuminates the persistence of Pott's puffy tumor as a formidable clinical entity that, while historically tethered to the pre-antibiotic era, continues to manifest with deceptive complexity in the modern adult population. The case of a 52-year-old diabetic female serves as a critical pedagogical example of how chronic immunosuppression can fundamentally alter the natural history of frontal sinusitis, transforming a common mucosal infection into a destructive osteomyelitis that mimics high-grade malignancy. The "malignancy masquerade"—characterized by extensive osteolytic destruction of the frontal table and orbital roof—poses a significant cognitive trap for clinicians, potentially delaying life-saving drainage in favor of oncological staging.

This case underscores that the adult diabetic patient represents a distinct vulnerability phenotype, where the confluence of hyperglycemia-induced immunosenescence and microangiopathic hypoxia creates a sanctuary for anaerobic pathogens. Consequently, we advocate for a recalibration of the diagnostic threshold: in any adult presenting with frontal swelling, proptosis, and radiographic evidence of bone erosion, the differential must remain broad enough to encompass chronic invasive osteomyelitis, regardless of the patient's age. The utilization of contrast-enhanced imaging is non-negotiable in differentiating avascular necrosis from neoplastic enhancement.

Furthermore, the successful outcome achieved herein validates the efficacy of a decisive, multidisciplinary surgical strategy. The collaboration between neurosurgery and ophthalmology allowed for the safe navigation of the anterior cranial fossa and the orbit, ensuring complete source control while preserving visual function. Crucially, this report challenges the traditional trepidation surrounding

immediate reconstruction in infected fields; it demonstrates that single-stage cranialization with antibiotic-impregnated bone cement is not only a viable limb of management but an essential step in restoring patient dignity and preventing the physiological sequelae of cranial defects. Ultimately, the management of adult PPT requires a departure from conservative antibiotic-only regimens in favor of aggressive surgical debridement, ensuring that this "puffy tumor" does not evolve into a fatal intracranial catastrophe.

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