

Pneumosinus Dilatans: A Case Report and Bone Analysis of a Rare Observation of its Relationship with Meningioma

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Abstract: Pneumosinus dilatans (PD) is an abnormal expansion of the paranasal sinuses, frequently associated with anterior skull base meningiomas. Despite its potential clinical significance, the etiology remains unclear, with theories ranging from mechanical obstruction to biochemical stimulation. We report a case of a 30-year-old male with a tuberculum sellae meningioma who developed sphenoid PD after years of surveillance, concurrent with continuous tumor growth. The patient underwent successful endoscopic resection, with histopathology confirming a World Health Organization (WHO) grade I meningioma infiltrating bone. This temporal association supports the theory that PD arises from active, tumor-induced bone remodeling rather than obstructive mechanisms. PD should be recognized as a significant radiographic marker of underlying meningioma activity, warranting thorough investigation and influencing management decisions.

Keywords: Bone Remodeling; Meningioma; Pathogenesis; Pneumosinus Dilatans; Skull Base.

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1. Introduction

Pneumosinus dilatans (PD) is a rare condition characterized by the abnormal expansion of one or more paranasal sinuses containing only air, lined by normal mucosa, and without evidence of bony destruction or mucosal disease [1,2,3]. First described by Meyes in 1898, it is now an established clinical entity and a possible marker of underlying pathology [4,5]. Often asymptomatic and incidental on neuroimaging [6], PD may cause deformity, headache, and visual or neurological issues. It predominates in males under 35 years old and has various presentations: frontal PD (48–63%) causes forehead bossing, while sphenoid PD (24–43%) causes visual symptoms [7,8], and rare maxillary PD causes proptosis and orbital signs [9].

The etiology remains unclear and multifactorial, with proposed mechanisms including mass compression, biochemical stimulation by tumor-derived growth factors, "ball-valve" obstruction, and congenital/developmental anomalies [4,10-13]. Diagnostic Computerized Tomography (CT) shows sinus expansion with thinned walls, while Magnetic Resonance Imaging (MRI) better delineates associated soft tissue [14]. Ricci proposed a recent classification and algorithm to guide clinical conduct based on sinus involvement and associated symptoms [2]. Management ranges from observation in asymptomatic

cases to complex multidisciplinary approaches for symptomatic or complicated cases [2,9].

Sweatman et al. [8] performed a search using Medline, EMBASE, and PubMed, reviewing 66 articles (111 cases) plus 16 CT cases, and identified 22 literature cases and 10 dataset cases specifically linked to meningioma. Our subsequent search of PubMed and other databases (using search terms such as pneumosinus dilatans, meningioma, and brain tumor) identified 20 additional articles. While these combined findings demonstrate an established link between PD and meningioma, the condition is rare and the current literature consists predominantly of isolated case reports and small series rather than large-scale studies.

Regarding meningioma, two main theories explain the development of PD. The ball-valve mechanism suggests mechanical sinus expansion, but this lacks support since no secondary sinus changes (such as mucocoele or sinusitis) are typically observed [15,16,17], and surgical exploration generally shows no anatomical obstruction [8,18,19]. The chemical theory proposes local stimulation by growth factors (IGF-1, IGF, PDGF), as meningiomas express these factors and induce local bone remodeling [11,20]. Furthermore, PD patients show increased F-NaF uptake, and bone analysis reveals abundant active osteoblasts [17]. Currently, local factors represent the most reliable theory regarding the formation of pneumosinus dilatans [4]. When different sinuses are affected, they display close anatomical correlations, indicating that local pathophysiological factors are more critical than systemic factors. Furthermore, skull base tumors (81.3%) are far more frequently associated with pneumosinus dilatans than non-skull base tumors [8].

This review and case report highlights the diagnostic and therapeutic challenges of this condition and strengthens the current formation theories of PD. Informed consent was obtained from all participants, and the confidentiality of patient information was strictly ensured.

2. Case Report

A 30-year-old immunocompetent male from Manaus, Brazil, presented with an initial incidental diagnosis of an extra-axial, dural-based sellar tubercle lesion radiologically consistent with a meningioma, discovered during neuroimaging evaluation following a traumatic brain injury (TBI). The baseline MRI performed in May 2019 (Figure 1) using a 1.5T scanner demonstrated a $1.3 \times 1.4 \times 0.7$ cm extra-axial nodule with a broad dural attachment to the sellar tubercle. The lesion appeared isointense on T1-weighted images, showed intermediate signal intensity on T2/FLAIR, and exhibited homogeneous gadolinium enhancement.

Anatomically, it was located anteriorly to the sphenoid planum, causing posterosuperior displacement of the suprasellar cistern and maintaining intimate contact with the right paraophthalmic internal carotid artery and the cisternal segment of the optic nerve. Notably, despite the lesion's anatomical extension and intimate contact with the optic nerve, the patient presented with completely intact vision and no visual field defects. Consequently, although surgical intervention was formally indicated at that time given the tumor's location, the risks and potential benefits of the procedure were extensively discussed with the patient, who declined to undergo surgery at that moment. Based on this shared clinical decision-making, a conservative approach with close prospective clinical and neuroimaging surveillance was adopted.

In December 2020, a follow-up 1.5T MRI (Figure 2) recorded tumor dimensions of $1.5 \times 1.2 \times 1.3$ cm. This numerical increase, particularly along the craniocaudal axis compared to the baseline 0.7 cm, was followed by a similar expansion of the PD, but did not show an aggressive biological progression. After discussion between the board, patient and family members, and given that we were going through the pandemic, it was decided to continue surveillance and to repeat a new controlled MRI. Throughout this surveillance period, the possibility of surgery was reassessed, but the patient remained strictly asymptomatic with completely preserved visual acuity and intact visual fields.

In September 2024, a follow-up 3T MRI (Figure 3) demonstrated an increase in the dimensions of the extra-axial lesion at the tuberculum sellae, with a contact with the optic nerves bilaterally and a displacement of the optic chiasma. Crucially, the examination revealed prominent signs of hyperostosis and pneumosinus dilatans, explicitly characterized by the abnormal expansion of the sphenoid sinus extending up to the sphenoid roof. Given the confirmed radiological progression of the tumor combined with the significant progressive expansion of the pneumosinus dilatans in a young patient, a definitive therapeutic decision was made to proceed with surgical resection to prevent impending and irreversible visual compromise.

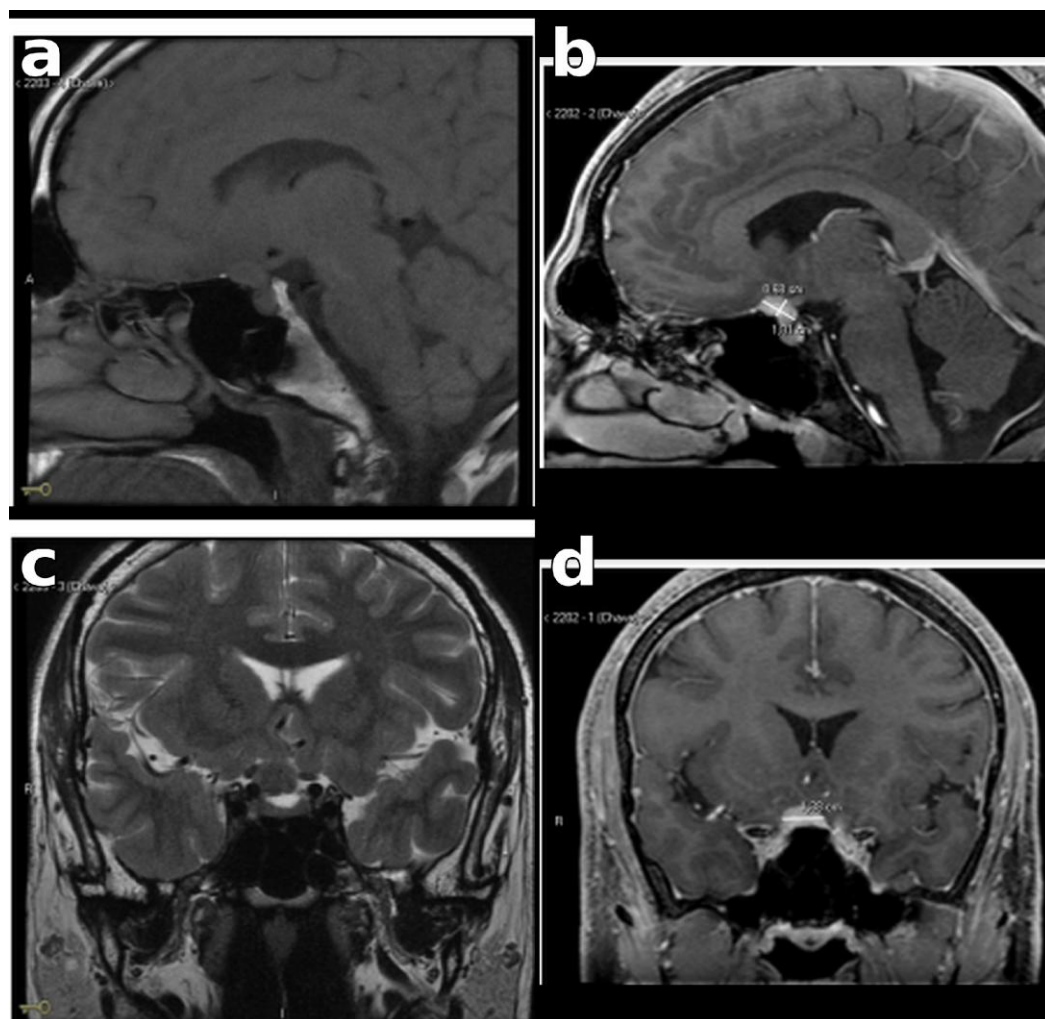


Figure 1. Baseline MRI at initial diagnosis (2019, 1.5 T). Sagittal T1-weighted images before (A) and after (B) gadolinium, coronal T2-weighted image (C), and coronal post-contrast T1-weighted image (D). A small extra-axial nodule ($1.3 \times 1.4 \times 0.7$ cm) with a broad dural base at the tuberculum sellae is isointense to grey matter on T1, shows intermediate signal on T2/FLAIR, and enhances homogeneously after gadolinium (B, D). The lesion lies on the tuberculum sellae and planum sphenoidale, displaces the suprasellar cistern posterosuperiorly, and abuts the right paraophthalmic internal carotid artery and the cisternal segment of the optic nerve. The sphenoid sinus is normally aerated, without expansion.

The patient subsequently underwent a skull base resection via an endoscopic transnasal approach. Notably, the superior and angulated displacement of the sphenoid roof caused by the pneumosinus dilatans paradoxically modified and facilitated the surgical corridor, providing excellent transnasal exposure and a wider operative trajectory to the

lesion. The anterior wall of the sella, the tuberculum sellae, and the planum sphenoidale were completely removed. A dural opening was performed in the shape of an inverted trapezoid, bounded laterally by the optic nerve canals, successfully exposing all anatomical limits of the tumor. This optimal exposure allowed for a gross total resection of the meningioma via meticulous arachnoid dissection.

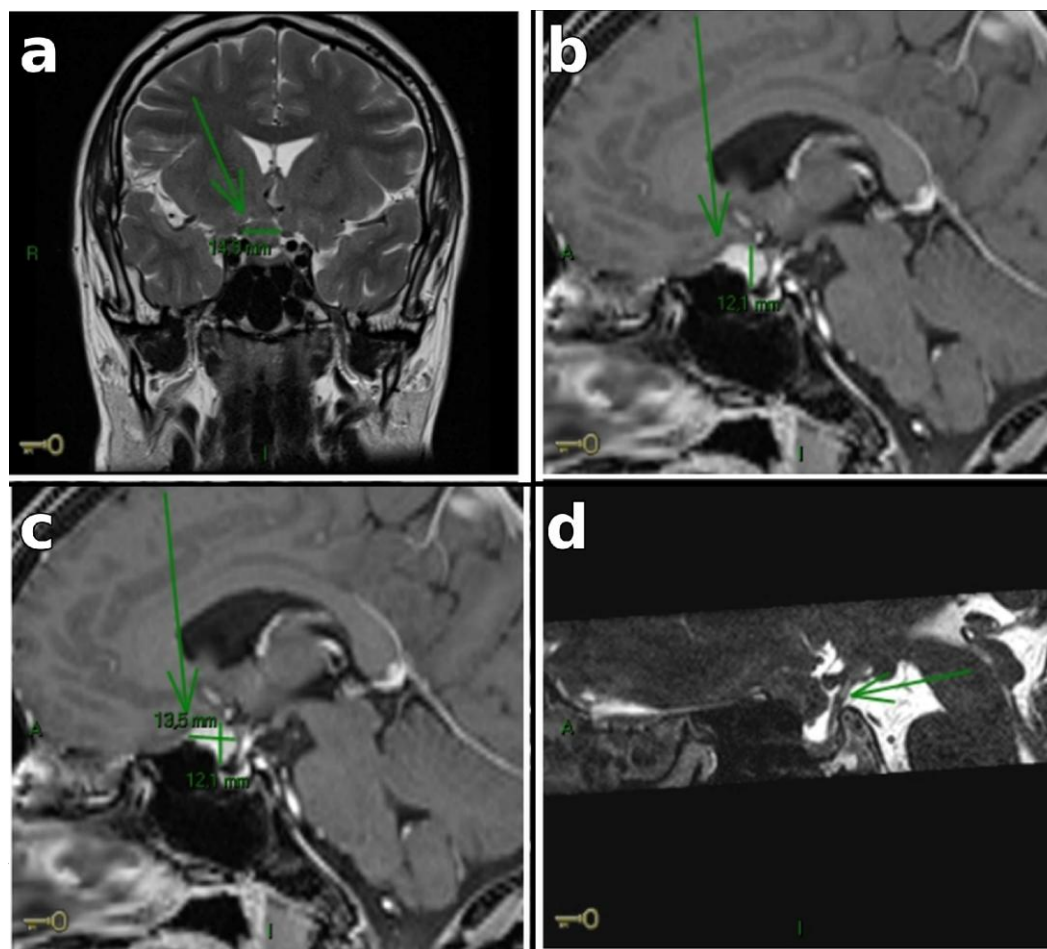


Figure 2. Follow-up MRI (December 2020, 1.5 T). Coronal T2-weighted (a) and sagittal post-contrast T1-weighted (b, c) images, together with a sagittal thin-section steady-state (CISS) image (d), show the tuberculum sellae meningioma, which measures $1.5 \times 1.2 \times 1.3$ cm (calipers, a–c), reflecting a slight, predominantly craniocaudal increase relative to baseline, consistent with an indolent, slow-growing lesion. A subtle new bony prominence is seen beneath the tumour, but there is still no overt expansion of the sphenoid sinus.

Skull base reconstruction was accomplished using a double layer dural substitute (DuraGen) and a vascularized nasal septal mucosal flap, secured with anterior and posterior nasal packing. The procedure was completed successfully without any perioperative complications, and postoperative images were obtained (Figures 4A and 4B).

2.1 Pathological Findings

Macroscopic examination of the fragment destined for pathological analysis revealed a tan-colored, lobulated tumor specimen measuring $1.8 \times 1.7 \times 1.2$ cm, along with an osseous fragment (0.5 cm) indicating bone infiltration. Microscopic sections (Figure 5) revealed a meningotheial architecture composed of syncytial cells with oval nuclei, occasional pseudoinclusions, and eosinophilic cytoplasm. These cells were arranged in a lobular pattern with whorls and sparse psammomatous calcifications (A). Mitotic figures and necrosis were absent. The neoplastic cells were immersed in a fibro-edematous stroma

with vascular ectasia. The adjacent bone showed permeation of sclerotic lamellar trabeculae by tumor infiltration into the marrow spaces, accompanied by reactive sclerosis and focal osteoblastic rimming. According to WHO criteria, the absence of mitoses, necrosis, and brain invasion confirmed a Grade 1 lesion (B and C).

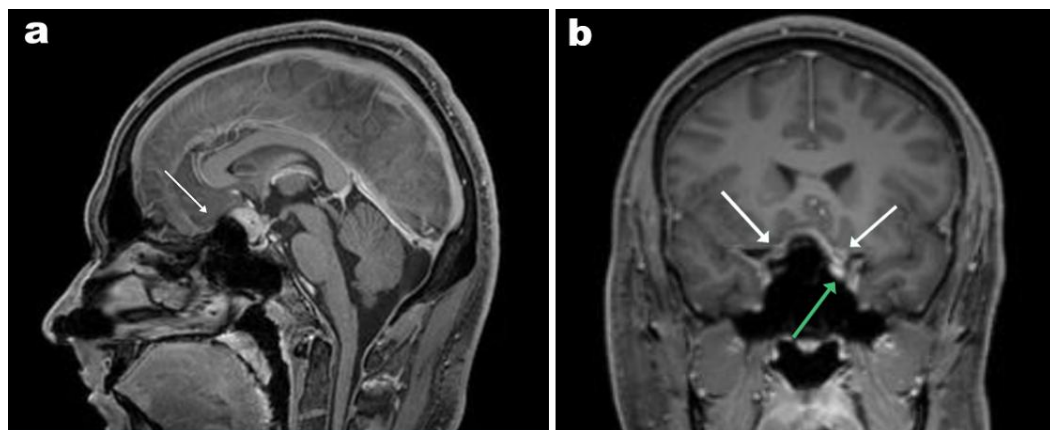


Figure 3. Follow-up MRI (December 2020, 1.5 T). Coronal T2-weighted (a) and sagittal post-contrast T1-weighted (b, c) images, together with a sagittal thin-section steady-state (CISS) image (d), show the tuberculum sellae meningioma, which measures $1.5 \times 1.2 \times 1.3$ cm (calipers, a-c), reflecting a slight, predominantly craniocaudal increase relative to baseline, consistent with an indolent, slow-growing lesion. A subtle new bony prominence is seen beneath the tumour, but there is still no overt expansion of the sphenoid sinus.

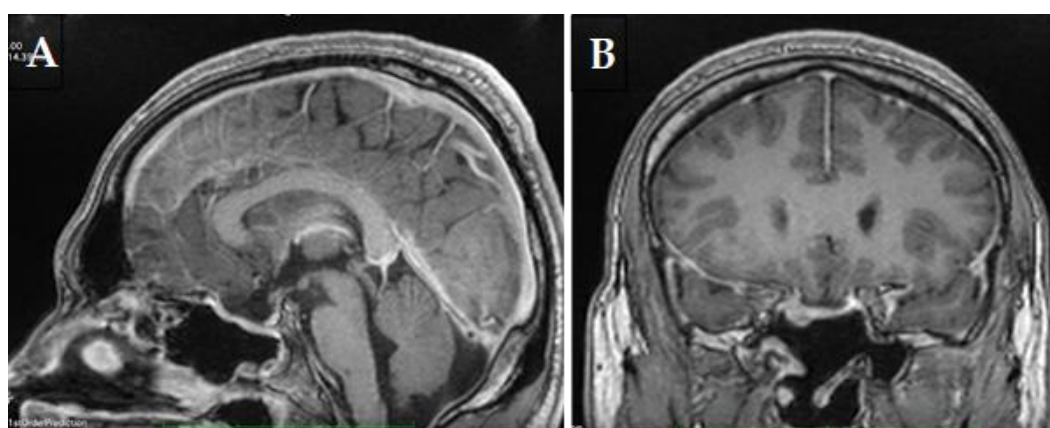


Figure 4. A. Postoperative MRI (2025, 3 T), sagittal post-contrast T1-weighted image after endoscopic endonasal resection. Gross total resection of the meningioma with no residual enhancing tumour. The planum sphenoidale, tuberculum sellae and anterior sellar wall were removed, and the skull base was reconstructed with a dural substitute and a vascularised nasoseptal flap. B. Postoperative MRI (2025, 3 T), coronal post-contrast T1-weighted image. Complete resection of the tuberculum sellae meningioma with a clean resection cavity and no residual or recurrent enhancing tumour; the optic apparatus is decompressed.

Immunohistochemical staining revealed tumor cells diffusely positive for EMA/MUC1 (d) and progesterone receptor (e). S100 was negative, and the Ki-67 proliferation index was 2% (f). The combined findings were consistent with the diagnosis of a meningotheial meningioma, central nervous system (CNS) WHO grade 1 (Table 1).

While standard hematoxylin and eosin (H&E) staining clearly demonstrated tumor infiltration, reactive bone sclerosis, and focal osteoblastic rimming, it is important to acknowledge that these findings inherently represent generic reactive hyperostosis. Without specialized spatial transcriptomics or targeted immunohistochemistry for specific

growth factors (such as IGF-1 or PDGF), we cannot definitively prove that this bone expansion was biochemically driven by paracrine signaling rather than a purely mechanical traction process. Nonetheless, the clear temporal correlation, where the sphenoid PD emerged strictly in tandem with progressive tumor growth over the years of surveillance, strongly supports the hypothesis of a dynamic, tumor-induced osteolytic-osteoblastic interaction.

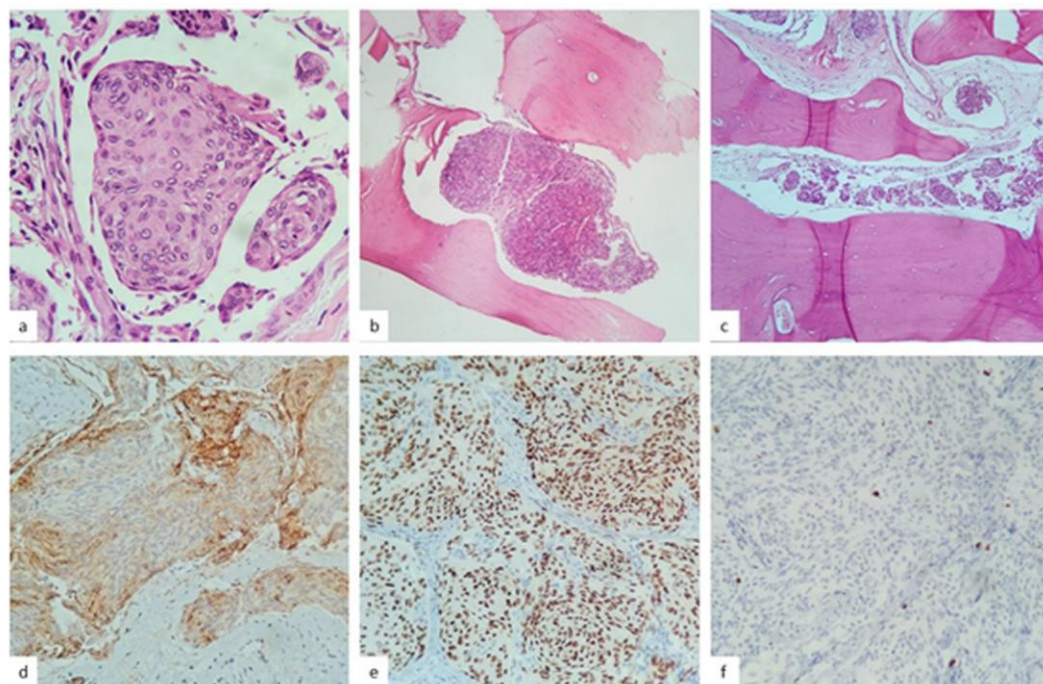


Figure 5. Histological sections reveal a lesion composed of cells with oval nuclei with occasional pseudoinclusions and eosinophilic cytoplasm, arranged in a lobular, syncytial architecture with whorls (A). Mitotic figures and necrosis were not seen. The neoplastic cells were immersed in an edematous and fibrous stroma with sparse psammomatous calcifications and infiltrated adjacent bone, which showed sclerotic trabeculae (B and C). Immunohistochemical staining revealed tumor cells diffusely positive for EMA/MUC1 (D) and progesterone receptor (E). S100 was negative and the Ki-67 proliferation index was 2% (F). The findings were consistent with the diagnosis of meningothelial meningioma, central nervous system (CNS) WHO grade 1.

Table 1. Immunohistochemical profile of the lesion and its diagnostic and phenotypic significance.

Marker	Result	Pathological / Clinical Significance
EMA / MUC-1	Diffuse (+)	Confirms meningothelial lineage.
S100	Negative (−)	Excludes schwannoma and metastatic carcinoma.
Ki-67	2%	Indicates a low proliferative index, consistent with indolent behavior.
PR (PgR636)	Diffuse (+)	Evidences a hormone-responsive phenotype (progesterone), characteristic of benign meningiomas.

Note: (+) positive; (−) negative; % percentage of stained nuclei. Abbreviations: EMA, epithelial membrane antigen; Ki-67, cellular proliferation marker; MUC-1, mucin 1; PR / PgR636, progesterone receptor, clone PgR636; S100, S100 calcium-binding protein.

3. Discussion and Conclusion

The leading theory in PD associated with meningioma involves local tumor-induced bone remodeling. Meningiomas near the tuberculum sellae or planum sphenoidale cause chronic pressure, thinning, and outward sinus expansion [4]. This "blistering" effect is clearly evident in our case of sphenoid PD, where the bulging of the sphenoid roof was intrinsically linked to the tumor's focal footprint [14]. PD is an active process, frequently accompanied by increased osteoblastic and osteoclastic activity in the affected bone [6].

The one-way valve hypothesis, initially proposed by Sugita et al. [19], posits that sinus ostium obstruction traps air, leading to progressive expansion. However, this theory fails to explain why many PD cases lack secondary inflammatory changes (e.g., mucoceles) or why surgical explorations often reveal patent ostia [12]. Instead, emerging evidence supports a biochemical mechanism, wherein meningioma-secreted factors, such as IGF-1, IGF-2, and PDGF, stimulate bone turnover [5]. This aligns with observations that PD-associated bone exhibits heightened metabolic activity on F-NaF PET imaging [6]. Our case demonstrated PD progression over five years associated with a slow-growing meningioma. While our standard histological findings cannot isolate the precise paracrine pathways, the temporal parallel between the tumor's volumetric expansion and the sphenoid blistering strongly reinforces the concept that meningiomas dictate active local bone alterations leading to PD [6].

PD demonstrates a striking anatomic specificity, most frequently involving the frontal (48–63%) and sphenoid (24–43%) sinuses [8]. This distribution parallels the predilection of meningiomas for the anterior skull base, where tumor proximity directly influences sinus pneumatization [7]. Intriguingly, PD is not merely an incidental finding but often a crucial diagnostic clue. In sphenoid sinus PD, 83% of cases are associated with meningiomas, arachnoid cysts, or visual loss [6]. These associations underscore the importance of systematic imaging evaluation in patients, particularly when new neurological or ophthalmologic symptoms are present.

From a surgical perspective, while PD can signal underlying pathology, it can also paradoxically assist the operative approach. The advent of endoscopic techniques has revolutionized access to skull base lesions. In this specific patient, the abnormal superior and angulated displacement of the sphenoid roof directly facilitated the endoscopic transnasal corridor, providing wider access and improved visualization of the dural limits around the optic nerves.

In conclusion, PD should be regarded as a dynamic indicator of underlying pathology, particularly anterior skull base meningiomas, warranting comprehensive evaluation and tailored surgical planning. Future studies should investigate molecular drivers and specific immunohistochemical markers to definitively bridge the gap between mechanical dural traction and biochemical bone remodeling.

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Research Ethics Committee Approval: All procedures performed in studies that involve human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the Declaration of Helsinki of 1964 and its subsequent amendments or comparable ethical standards. Free and informed consent was obtained from the individual included in this study.

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Conflicts of Interest: All other authors declare no conflicts of interest.

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