

Recurrent Sinonasal Fibromyxoid Tumor with Orbital and Intracranial Extension in a 75-Year-Old Woman: A Case Report and Narrative Review of the Literature

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Abstract

Case Report

Sinonasal myxoid tumors are rare and may be difficult to classify, particularly in adults, because much of the recent literature concerns infantile sinonasal myxoma. We report the long-term course of a 75-year-old woman followed since 2016 for a right sinonasal fibromyxoid tumor. Despite several surgical procedures, the tumor recurred repeatedly. During a new resection in 2024, histopathological examination showed a moderately cellular mesenchymal proliferation arising within abundant myxoid stroma. The tumor cells were predominantly spindle shaped and displayed only mild atypia, with no necrosis or atypical mitotic figures; foci of calcification and ossification were also present. Immunohistochemically, the tumor cells showed diffuse vimentin expression, whereas smooth muscle actin and S100 protein were only focally expressed. CD34 and MUC4 were negative, and the Ki-67 proliferation index was approximately 4%. In 2025, the lesion progressed to approximately 70 × 80 × 95 mm, with skull-base destruction, orbital extension, right optic-nerve involvement, frontal intracranial extension, and a midline shift of about 23 mm. A later pathology report referred to the lesion as an “ossifying fibromyxoid tumor,” introducing uncertainty in the final classification. Given the repeated recurrences and local extent, radiotherapy was planned, but the patient was lost to follow-up before treatment delivery could be confirmed. A focused narrative review shows that adult sinonasal myxoid tumors remain less well characterized than infantile forms and that orbitocranial extension is rarely reported. This case illustrates the discrepancy that may occur between bland histologic morphology and markedly destructive local behavior and highlights the value of expert pathologic review when terminology evolves during follow-up.

Keywords: fibromyxoid tumor; sinonasal myxoma; orbit; skull base; intracranial extension; recurrence; narrative review.

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INTRODUCTION

Myxomas are rare mesenchymal tumors characterized by bland spindle or stellate cells dispersed within abundant myxoid extracellular matrix. In the craniofacial region, the historical literature has focused mainly on odontogenic myxoma of the jaws. True sinonasal lesions are far less common, and their nomenclature has long been inconsistent, with overlap among the terms “myxoma,” “fibromyxoma,” and, more broadly, “myxoid tumor” [1–5].

Recent studies have mainly clarified infantile sinonasal myxoma. This form typically arises during the first years of life and has distinctive clinical,

immunohistochemical, and molecular features, including alterations involving the WNT/β-catenin pathway in a substantial proportion of cases [1–4]. Sinonasal myxoid tumors arising in older patients appear more heterogeneous and should not automatically be considered equivalent to the infantile entity [1,2].

The present case is unusual for several reasons: the patient’s age, the nearly 10-year course, repeated recurrences, focal ossification, and, most importantly, extensive orbital and intracranial progression despite relatively bland histologic features. We describe the clinical course while preserving the diagnostic uncertainties that emerged during follow-up and place

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the case in the context of a targeted narrative review of the literature.

CASE REPORT

Clinical history

A woman with no major documented comorbidities first presented in 2016 with progressively worsening nasal obstruction, initially unilateral and later bilateral, associated with episodes of epistaxis. Surgical excision led to a diagnosis of fibromyxoma of the right nasal cavity. An early local recurrence required a second operation in 2017. The slides and paraffin blocks from

the 2016 and 2017 procedures were not available for the present reassessment.

After several years of relative clinical stability, symptoms recurred in 2024. Imaging demonstrated an infiltrative nasoethmoidal mass with bone destruction and extension toward the orbit, nasopharynx, and intracranial compartment. A new surgical procedure was performed in June 2024. The subsequent course was not marked by durable disease control; further radiologic progression was documented during 2025.

Clinical timeline

Table 1: Clinical timeline of the patient's disease course.

Date	Main findings	Management / outcome
2016	Progressive nasal obstruction and epistaxis. Fibromyxoma of the right nasal cavity diagnosed after excision.	First surgery.
2017	Early local recurrence.	Second surgical procedure.
May–June 2024	Infiltrative nasoethmoidal mass with bone destruction and orbital and intracranial extension.	Repeat surgery with endonasal and intracranial specimens.
Dec 30, 2024	IHC: diffuse vimentin+, focal SMA+, weak/focal S100+, CD34–, MUC4–, Ki-67 approximately 4%.	Findings interpreted as supporting the previously diagnosed fibromyxoma.
Apr 3, 2025	Craniofacial CT: lesion approximately 57 × 62 × 83 mm versus 41 × 53 × 63 mm on Oct 21, 2024.	Radiologic progression confirmed.
Oct 7, 2025	MRI: lesion approximately 70 × 80 × 95 mm versus 58 × 64 × 88 mm on Mar 20, 2025, with orbital and intracranial extension.	Further multidisciplinary reassessment.
Oct 16, 2025	Pathology report: “mesenchymal proliferation in a myxoid background, in favor of an ossifying fibromyxoid tumor.”	Terminologic discrepancy with previous reports.
Mar 10, 2026	ECOG performance status 1; persistent naso-maxillo-orbital swelling and right exophthalmos.	Radiation-oncology assessment; imaging update and local-treatment discussion.
After Mar 2026	No further clinical or radiologic data available.	Radiotherapy planned; patient lost to follow-up before treatment delivery could be documented.

Imaging findings

Craniofacial CT performed on April 3, 2025 showed a poorly defined, multilobulated, expansile and infiltrative lesion centered on the nasal cavities and predominantly right sided. It measured approximately 57 × 62 × 83 mm, compared with 41 × 53 × 63 mm on the nasosinus CT of October 21, 2024. Superiorly, the lesion eroded the ethmoid cells, nasal bones, cribriform plate, and crista galli. It extended intracranially, producing frontal mass effect and mild perilesional edema. On the right, the lesion destroyed the lamina papyracea and orbital roof, infiltrated the orbit, displaced the globe, and reached the optic nerve. Posteriorly, it extended toward the choanae, nasopharynx, and right sphenoid sinus.

MRI performed on October 7, 2025 confirmed further progression, with the mass measuring approximately 70 × 80 × 95 mm, compared with 58 × 64 × 88 mm on March 20, 2025. There was extensive frontal intracranial extension with infiltration of the frontal lobes, mild surrounding edema, and a large postoperative hydro-aeric/hemorrhagic collection. The mass effect produced an approximately 23-mm right-to-left midline shift with subfalcine herniation. The right orbit was extensively involved, with globe deformity, grade 3 exophthalmos, and optic-nerve involvement. On the left, the medial orbital wall was involved, although a preserved fat plane remained around several extraocular muscles.

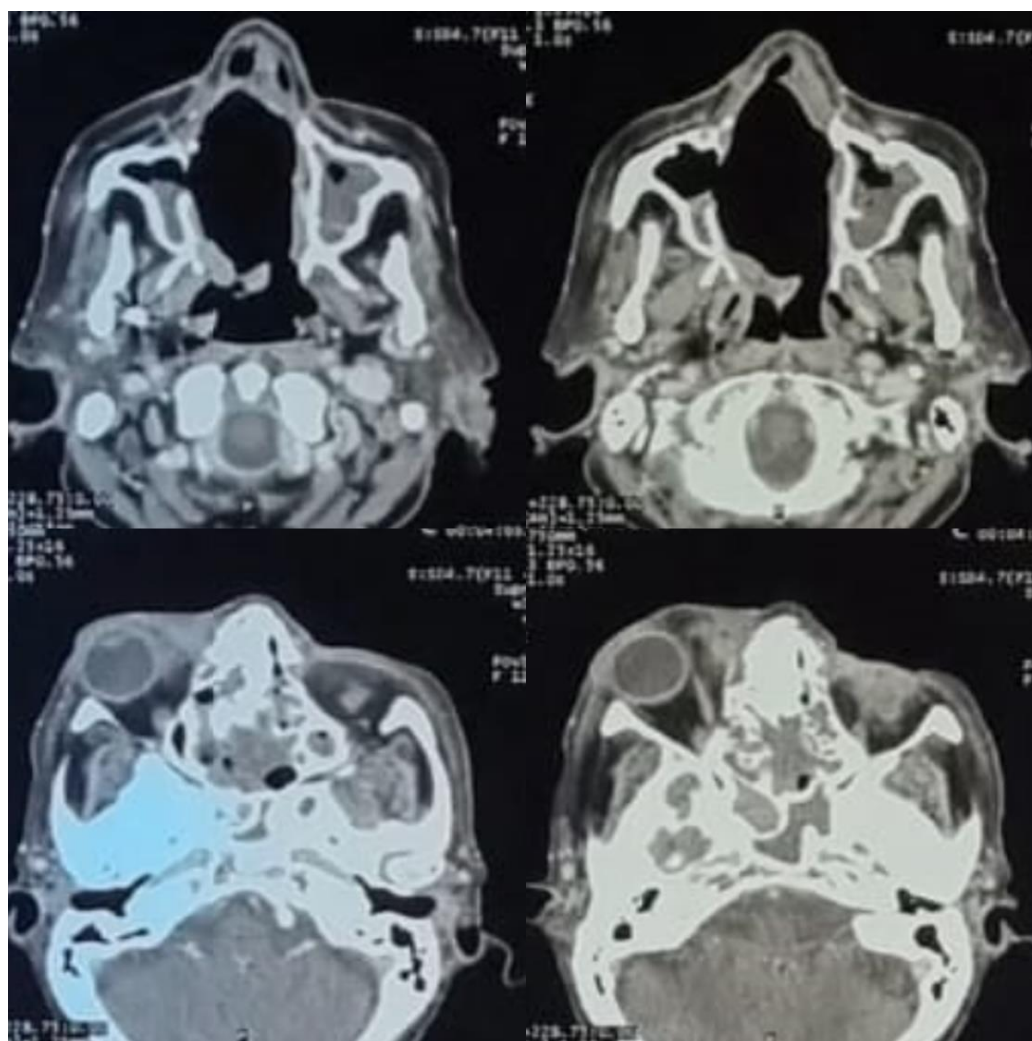


Figure 1: Representative craniofacial CT images showing the recurrent nasoethmoidal mass with bony destruction and orbital extension. Peripheral metadata were removed for de-identification

Histopathology and immunohistochemistry

The June 2024 procedure yielded two fragmented specimens, one intracranial and one endonasal. Microscopically, the intracranial component consisted of a moderately cellular mesenchymal proliferation within abundant myxoid stroma. The tumor cells were arranged in variable-sized lobules and diffuse sheets and were predominantly spindle shaped, small to medium in size, with oval-to-elongated nuclei that were occasionally irregular and hyperchromatic. No significant nuclear pleomorphism, atypical mitoses, or tumor necrosis was identified. The stroma was richly vascularized, with thin branching vessels described in places as hemangiopericytoma-like, and contained foci of calcification and ossification. The endonasal specimen showed the same proliferation widely dissecting the respiratory mucosa.

Immunohistochemically, the tumor cells showed moderate diffuse cytoplasmic expression of

vimentin and moderate focal expression of smooth muscle actin. CD34 was negative. S100 protein showed weak focal nuclear and cytoplasmic expression. MUC4 was negative, and the Ki-67 proliferation index was approximately 4%. Anti-laminin staining was not available. Based on the morphology and immunophenotype, the 2024 interpretation remained in favor of the previously diagnosed fibromyxoma.

A pathology report dated October 16, 2025 subsequently used the wording “ossifying fibromyxoid tumor.” This distinction is clinically relevant because ossifying fibromyxoid tumor (OFMT) is a separate soft-tissue entity rather than a synonym for fibromyxoma. The available records did not include INI1/SMARCB1 assessment, PHF1 rearrangement testing, other dedicated molecular studies, or an external specialist pathology review.

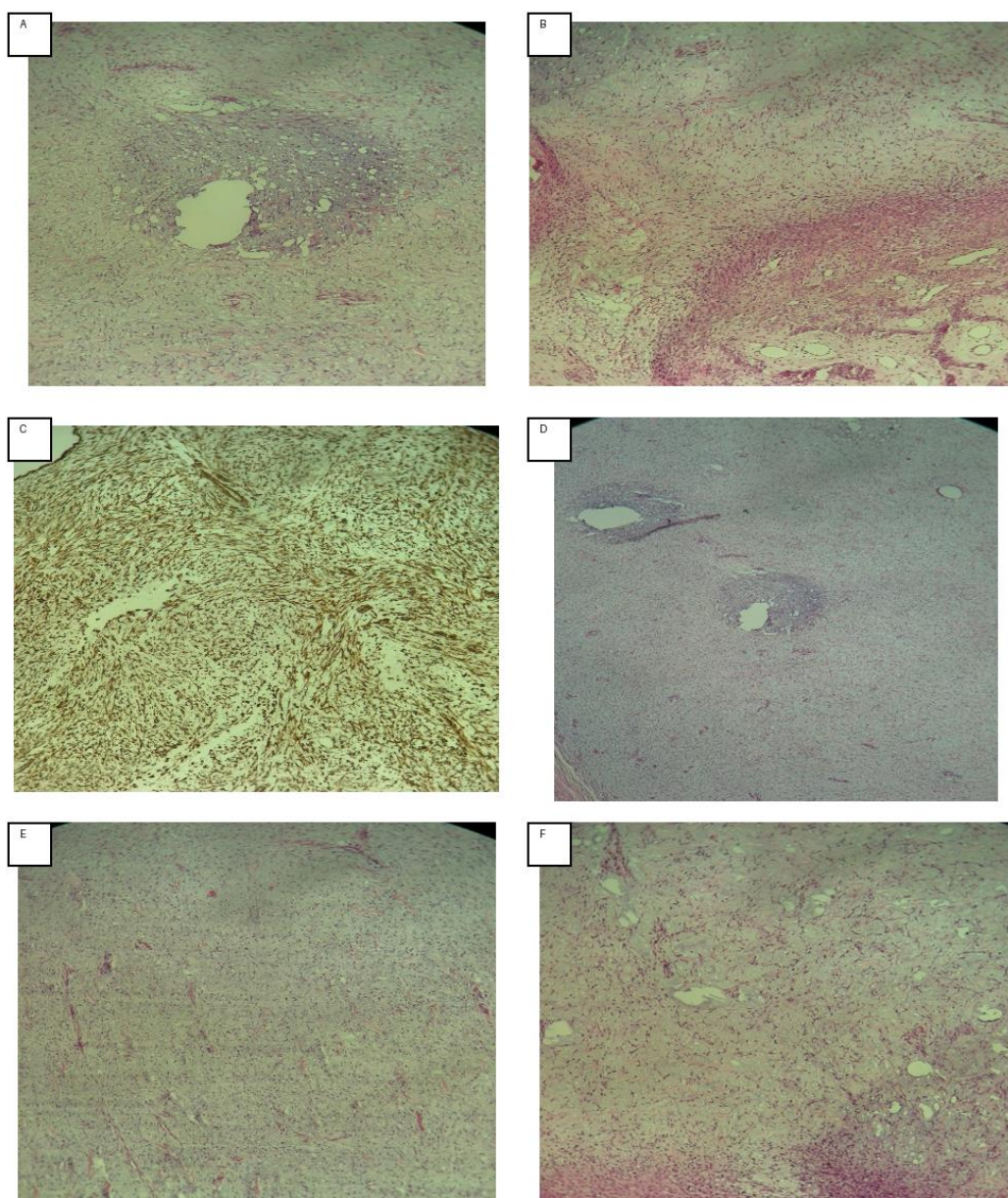


Figure 2: Representative histologic images from the 2024 surgical specimen showing a spindle-cell mesenchymal proliferation in abundant myxoid stroma with prominent vascularity. Original magnifications should be verified from the pathology archive before final production if requested by the journal

Therapeutic decision and follow-up

At the radiation-oncology assessment on March 10, 2026, the patient was conscious and clinically stable, with an ECOG performance status of 1. Examination showed swelling of the nasal dorsum extending toward the right maxillary and orbital regions, associated with right exophthalmos. Given the repeated recurrences, tumor volume, and orbitocranial extension, a further complete resection was considered particularly complex and potentially morbid. The case was discussed in a multidisciplinary setting, and radiotherapy was planned as an individualized local-control strategy.

The patient did not return for the planned treatment and was subsequently lost to follow-up. The available records therefore do not establish that

irradiation was started or delivered, and no post-treatment response, toxicity, subsequent imaging, or survival outcome can be reported.

DISCUSSION

To contextualize this case, we performed a targeted narrative search of PubMed/MEDLINE through September 24, 2026. Search terms, used alone or in combination, included “sinonasal myxoma,” “nasal myxoma,” “paranasal sinus myxoma,” “sinonasal fibromyxoma,” “ethmoid myxoma,” “orbital myxoma,” “intracranial myxoma,” and “ossifying fibromyxoid tumor.” We prioritized reports of sinonasal, ethmoidal, orbital, and skull-base lesions, together with series and reviews relevant to diagnostic classification. Studies of

odontogenic myxoma were used only to inform discussion of biology and recurrence, with explicit recognition that direct extrapolation to adult primary sinonasal tumors is imperfect. This was a focused narrative review rather than a systematic review.

Contemporary literature increasingly separates infantile sinonasal myxoma from other craniofacial myxoid tumors. Mewar *et al.*, proposed that it represents a distinctive lesion of early childhood [4]. Molecular studies by Velez Torres *et al.*, and Odintsov *et al.*, further supported this concept by identifying WNT/ β -catenin pathway alterations in a substantial proportion of infantile cases and by demonstrating differences from myxomas arising in craniofacial bones of older patients [1,2]. Burns *et al.*, in a review including 48 infantile cases younger than 3 years, confirmed the strong pediatric predominance and showed that orbital extension, although possible, remains an unusual presentation [3]. A further pediatric case with orbital involvement was reported in 2026 [19].

In adults, the literature is dominated by isolated case reports and older publications using heterogeneous terminology. Some nevertheless document striking local aggressiveness. Hunchaisri described a nasal and paranasal sinus myxoma invading both the orbits and brain and causing blindness, treated by three operations including transbasal craniotomy [12]. Yilmaz *et al.*, reported a nonodontogenic maxillary sinus myxoma extending into the sphenoid sinus and pterygopalatine fossa in a 48-year-old woman [13]. Ryu *et al.*, described a giant ossifying myxoma arising from the ethmoid region with intracranial extension in a 50-year-old man, managed by subtotal resection because complete removal was expected to be highly morbid [14]. Matsuo and Tanaka later reported an orbital myxoma with probable ethmoidal origin, with residual disease remaining radiologically stable for 12 years [15]. In 2025, Jahandideh *et al.*, described an inferior turbinate myxoma in a 47-year-old woman that was completely removed endoscopically with no recurrence at 1 year [16].

Selected adult and orbitocranial cases

Table 2: Selected adult or orbitocranial cases reported in the literature. This table is a narrative synthesis and not an exhaustive systematic review.

Reference	Age/sex	Primary site	Extension	Treatment	Outcome
Hunchaisri, 2002 [12]	Adult	Nasal cavity and paranasal sinuses	Orbits and brain; blindness	Three resections including transbasal craniotomy	Disease-free at publication
Yilmaz <i>et al.</i> , 2011 [13]	48 y, F	Maxillary sinus	Sphenoid sinus and pterygopalatine fossa	Combined endoscopic and gingivobuccal approach	Not specified in abstract
Ryu <i>et al.</i> , 2015 [14]	50 y, M	Ethmoid/anterior skull base	Intracranial extension; marked ossification	Subtotal resection	Limited follow-up data
Matsuo & Tanaka, 2023 [15]	41 y, F	Orbit; probable ethmoidal origin	Along the medial orbital wall	Resection at age 49; residual disease	Residual stable for 12 years
Jahandideh <i>et al.</i> , 2025 [16]	47 y, F	Inferior nasal turbinate	No orbitocranial extension reported	Complete endoscopic resection	No recurrence at 1 year
Present case	75 y, F	Right nasoethmoidal region	Orbits, right optic nerve, skull base, frontal lobes	Repeated surgery; radiotherapy planned	Lost to follow-up before radiotherapy documentation

Nosologic and diagnostic considerations

The main challenge in this case is not simply rarity but classification. The historical diagnosis of fibromyxoma was repeatedly maintained and is supported by the 2024 morphology. However, recent literature shows that the term “sinonasal myxoma” does not necessarily refer to the same biological entity across age groups and anatomic settings. The better-characterized infantile form occurs almost exclusively in very young children and shows a distinctive molecular profile [1–4]. Our patient clearly falls outside this setting.

For this reason, we deliberately use the descriptive term “sinonasal fibromyxoid tumor” in the title. It remains faithful to the available pathology while avoiding a level of diagnostic certainty that the existing investigations cannot fully support. This caution is particularly important because the 2025 report introduced the term “ossifying fibromyxoid tumor,” although OFMT is a distinct soft-tissue entity with its own morphologic, immunohistochemical, and molecular features [10,11].

Pathologic interpretation

The 2024 specimen showed a low-grade-appearing mesenchymal myxoid proliferation, with bland spindle cells, no necrosis, no atypical mitoses, and a low Ki-67 index. Diffuse vimentin positivity supports mesenchymal differentiation but is nonspecific; contemporary series of odontogenic myxoma also report consistent vimentin expression [8]. MUC4 negativity is informative because diffuse strong MUC4 expression is highly sensitive and specific for classic low-grade fibromyxoid sarcoma, making that diagnosis substantially less likely in the present context [9].

Foci of calcification and ossification and weak focal S100 expression nevertheless justify retaining OFMT in the differential diagnostic discussion. Series by Folpe and Weiss and by Graham *et al.*, show that OFMT encompasses conventional, atypical, and malignant forms and that recurrent molecular alterations, including those involving PHF1, can occur [10,11]. In the present case, the absence of molecular testing and expert external pathology review prevents further resolution. If archival material remains available, specialist review of the 2024 and 2025 specimens would be the most useful next diagnostic step.

Clinicoradiologic aggressiveness

The most striking feature of this case is the discrepancy between relatively bland histology and extreme local aggressiveness. Over nearly a decade, the lesion recurred after several operations, destroyed the sinonasal and skull-base structures, invaded the orbit, involved the right optic nerve, and extended intracranially into the frontal region with major mass effect. This behavior does not by itself establish histologic malignancy. It does, however, illustrate how a myxoid tumor considered benign or low grade can become clinically devastating because of infiltrative growth and the difficulty of obtaining complete resection in a constrained anatomic region.

Published adult cases illustrate a similar phenomenon. Hunchaisri reported combined brain and orbital involvement, while Ryu *et al.*, described a giant ossifying lesion arising from the ethmoid region with intracranial extension [12,14]. The present case is distinguished by the patient's advanced age, repeated recurrences over almost 10 years, bilateral orbital involvement predominantly on the right, and the degree of intracranial mass effect.

Surgery, recurrence, and the uncertain role of radiotherapy

Surgery remains the best-supported treatment for craniofacial myxomas when complete resection can be achieved with acceptable morbidity. In odontogenic myxoma, pooled analyses generally show higher recurrence after more conservative procedures than after wider resection [6,7]. These findings cannot be transferred uncritically to an adult nasoethmoidal tumor

because the origin, biology, and anatomic constraints differ.

In our patient, the extent of disease in 2025 made complete resection particularly challenging: bilateral orbital involvement, proximity to or involvement of the right optic nerve, skull-base destruction, and frontal intracranial extension. Radiotherapy was therefore selected in multidisciplinary discussion as an individualized local-control option. This decision must be interpreted cautiously, because there is no validated radiotherapy standard or established dose for a true sinonasal fibromyxoma. Evidence from desmoid fibromatosis or other locally aggressive benign mesenchymal tumors represents biological extrapolation rather than direct evidence [17]. In addition, optic-nerve involvement and the proximity of the chiasm and frontal brain would have created major dosimetric constraints [18].

Our observation cannot evaluate this strategy because the patient was lost to follow-up before treatment delivery could be confirmed.

Strengths and limitations

A major strength of this report is the length of follow-up, which documents the evolution of a rare myxoid tumor over nearly 10 years through repeated surgery, detailed histopathology and immunohistochemistry, and serial imaging showing progressive local extension. The case also provides a practical example of how diagnostic terminology may evolve when a lesion recurs and additional morphologic features become evident.

The limitations are equally important. The 2016 and 2017 specimens were not available for review; no molecular characterization was performed; the 2025 terminology was not reconciled through external expert pathology review; and follow-up ended before the planned radiotherapy. Finally, the literature review was targeted and narrative rather than systematic and was intended to contextualize the case rather than generate pooled estimates of incidence, recurrence, or treatment efficacy.

CONCLUSION

This case is less the story of a simply "rare benign tumor" than that of a myxoid lesion whose local behavior progressively exceeded what its initial morphology would have suggested. In this 75-year-old woman, repeated recurrences culminated in major orbital and intracranial involvement despite persistently bland histologic features and a low proliferative index.

Three practical points emerge. First, adult sinonasal myxoid tumors should not be automatically equated with the better-defined infantile sinonasal myxoma. Second, a change in terminology here from fibromyxoma to ossifying fibromyxoid tumor should

prompt specialist pathologic reassessment rather than automatic continuation of a historical diagnosis. Third, when disease extent makes surgery highly morbid, management must remain multidisciplinary and proportional to the limited evidence base. In the present case, radiotherapy was planned, but loss to follow-up prevented any assessment of treatment outcome.

INFORMED CONSENT

Written informed consent was obtained from the patient for publication of this case report and the accompanying clinical, radiologic, and histopathologic information and images.

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