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Case Report

Rare presentation of angiomatosis in the paranasal sinuses mimicking juvenile nasopharyngeal angiofibroma in a 16 year old male ☆

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ABSTRACT

Rare presentation of pediatric angiomatosis of the paranasal sinus and skull base presenting mimicking juvenile nasopharyngeal angiofibroma (JNA). This is a 16-year-old male who presented to the emergency room with acutely worsening headaches, decreased visual acuity, subjective diplopia on lateral gaze, and a skull base mass centered in the sphenoid cavity. Endoscopic biopsy at an outside facility was aborted due to profuse bleeding. Upon transfer to a tertiary care center, contrast MR demonstrated a heterogeneously and avidly enhancing vascular mass centered around the sphenoid and skull base originating from the internal maxillary artery with significant bilateral extension into the adjacent paranasal sinuses, sella, and cavernous sinus. History of presentation and imaging was suggestive of JNA. Patient underwent preoperative embolization followed by endoscopic endonasal transphenoidal resection with a skull base trained otolaryngologist and neurosurgeon. Final pathology confirmed angiomatosis. This is only the second reported case of paranasal sinus angiomatosis in the literature. Angiomatosis has a high rate of recurrence and failure of timely diagnosis can lead to requirement of repeated surgical intervention. Re-operations are asso-

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ciated with increased costs, patient dissatisfaction, and poorer surgical/clinical outcomes. Because angiomatosis can mimic JNA, hemangiomas, or other vascular tumors, it is essential to maintain a broad differential diagnosis that includes angiomatosis when evaluating sinonasal tumors.

Introduction

Angiomatosis

Angiomatosis is a rare vascular lesion that extends diffusely along a contiguous segment of multiple different tissue types (muscle, bone, adipose, etc.). It presents usually within the first two decades of life and displays a female predilection. This condition is commonly seen in the lower extremities, with additional cases reported in other sites such as the heart, forearm, abdominal wall, genitalia, and retroperitoneum [1–5]. Cases in the head and neck region are rare however, with singular case reports demonstrating involvement of the paranasal sinuses, cheek/lip, malar region, masseter, and mandible [1,6,7].

The etiologies for angiomatosis include congenital and acquired forms. Congenital forms may arise sporadically or with an accompanying syndrome, such as Klippel-Trenaunay-Weber syndrome, Sneddon's syndrome, or Gorham disease [8–10]. Sporadic cases have been shown to display a predominance for the extremities, while the associated syndromes have demonstrated a potential to develop lesions in any tissue type with an extensive and continuous pattern [2]. The acquired forms may arise due to infection with Bartonella henselae, in the setting of HIV infection or an immunocompromised state, or from iatrogenic sources such as AV fistula formation and trauma [2,11,12].

Although benign, it is often confused with other malignancies and vascular lesions (juvenile nasopharyngeal angiofibroma [JNA], hemangioma, arteriovenous malformation, etc.) due to its aggressive growth and infiltrative nature [1]. Because of the latter, achieving complete surgical resection with negative margins has proven to be challenging with recurrence rates of greater than 90% [6]. Additionally, it is reported that around 40% of patients experience more than one recurrence every 5 years [7]. Imaging/clinical presentation for the diagnosis of angiomatosis is not precise, and definitive diagnosis is confirmed by histopathology. Early histopathologic diagnosis is critical to achieving curative resection on the first operation and may reduce the risk of recurrence. Nevertheless, it is imperative to schedule routine postoperative surveillance for these patients with biannual nasal endoscopy and annual magnetic resonance imaging (MRI) [1].

According to updated International Society for the Study of Vascular Anomalies (ISSAVA) classifications, the differential diagnosis for benign/locally aggressive vascular anomalies of the head and neck include arteriovenous malformations, hemangioma, tufted angioma, spindle cell hemangioma, kaposiform hemangioendothelioma, hemangioendothelioma, and papillary intralymphatic angioendothelioma [13]. Additionally, requiring consideration due to its similar benign vasoproliferative profile is JNA.

Juvenile Nasopharyngeal Angiofibroma

JNA is a rare, benign, highly vascular tumor that presents almost exclusively in male adolescents around the ages of 14-25 years. It is the most common benign nasopharyngeal neoplasm, yet only represents 0.5% of all head and neck neoplasms [14]. JNA typically arises from the sphenopalatine artery near the sphenopalatine foramen and can quickly expand. It is notorious for its locally infiltrative nature. JNAs grow into the pterygopalatine fossa and infratemporal fossa laterally, as well as medially into the nasopharynx and skull base. This infiltrative potential is secondary to JNA's hypervascular and unencapsulated characteristics. The presenting symptoms are due mainly to obstruction and impingement of adjacent structures from the rapid growth, mass effect, and pressure resorption. Initial presentation is typically painless progressive unilateral nasal obstruction often accompanied by epistaxis, congestion, and rhinorrhea. More severe complications include cranial nerve palsies, facial deformities, proptosis, vision loss/changes, and even chronic otomastoiditis from eustachian tube obstruction [15,16].

Although mostly found in the nasopharynx, JNA also commonly affects the sinuses. Unlike JNA, angiomatosis is exceedingly rare in the head and neck and most reported cases have been limited to the oropharyngeal cavity [7]. Only one other case of angiomatosis in the paranasal sinuses has been reported in the literature [6]. In this case, we report a 16 year old male with sinonasal and skull base angiomatosis which mimicked the clinical and radiographic presentation of JNA.

Case presentation

This is a 16-year-old male with a history of headaches who was transferred to a tertiary care center for management of a large sphenoid sinus/skull base mass. Two weeks prior to presentation, the patient presented to an outside ED for reduced visual acuity, subjective diplopia on lateral gaze, and progressively worsening frontal headaches. Radiographic imaging at that time showed a left intracranial frontal dural lesion as well as a large sphenoid sinus mass. MRI image of the left frontal dural lesion was characteristic of meningioma (Fig. 1). Prior to transfer, patient underwent successful craniotomy and resection of the frontal dural lesion with pathology confirming grade II meningioma. After recovery, patient underwent endonasal biopsy of the large sphenoid sinus mass at the outside

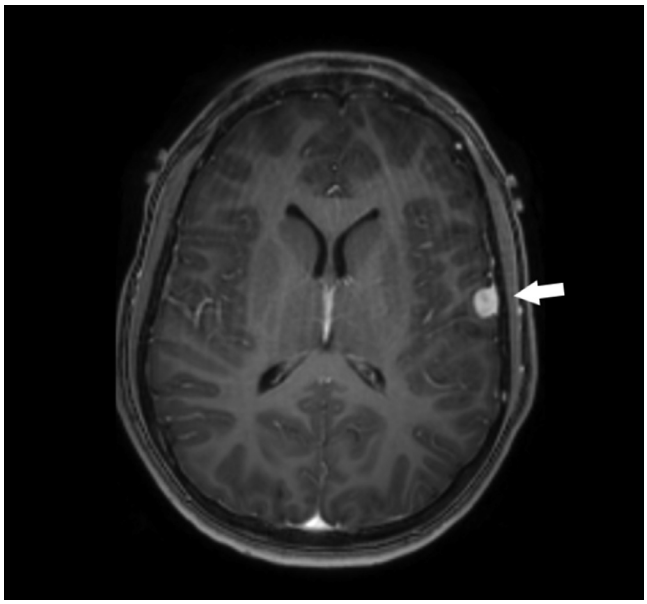

Fig. 1 – Meningioma. Axial MRI. Left frontal dural-based lesion (white arrow) with subsequent biopsy confirmation of meningioma grade II.

facility. Biopsy was aborted due to significant bleeding requiring nasal packing and transfer to a tertiary care center for further management.

Upon transfer to our hospital, the patient endorsed persistent headaches, poor visual acuity, and diplopia. He denied a history of epistaxis prior to his biopsy. CT demonstrated a large expansile, irregular destructive mass centered in the sphenoid sinus obliterating the sphenoid sinus cavity with involvement of the clivus, pterygopalatine fossa, sphenopalatine foramen, and erosion of the planum sphenoidale, floor of the sella and optic canal with sellar extension and erosion of the clivus and skull base. No bowing was noted of the posterior maxillary sinus wall (Fig. 2A-D). MRI showed an avidly heterogeneously enhancing lesion with multiple cystic areas/necrosis without fluid levels involving the central skull base with extension along the planum sphenoidale and posterior ethmoid sinus, nasal cavity and nasopharyngeal soft tissues and encasement of the internal carotid arteries and optic nerves. There were no prominent flow voids in the lesion and no anterior displacement of the posterior maxillary wall (Holman-Miller sign) that are commonly seen in JNA (Fig. 3A-D).

Based on clinical presentation and radiographic characteristics, a preliminary diagnosis of JNA was made. Aneurysmal bone cyst or giant cell tumors were felt to be less likely possibilities. Patient underwent preoperative angiograms demonstrating hypervascular blush from the left internal maxillary artery (Fig. 4A, B). Postembolization angiograms of the left external carotid artery demonstrated no evidence of hypervascular blush (Fig. 5A, B). The next day, the patient underwent uncomplicated endoscopic endonasal trans-sphenoidal resection with a skull base trained rhinologist and neurosurgeon. Intraoperative findings were notable for a friable submucosal mass along the course of the sphenopalatine and posterior nasal septal artery, filling the sphenoid sinuses and right posterior ethmoid sinus. Initial frozen section biopsy was suspicious for angiofibroma with hemorrhagic components. Postoperatively, the patient reported improvement in his diplopia and visual acuity. Postoperative course was complicated by cerebrospinal fluid (CSF) rhinorrhea on postoperative day 3 which was conservatively managed with a lumbar drain, acetazolamide, and bed rest. The lumbar drain was removed on postoperative day 6 with no more evidence of CSF rhinorrhea and the patient was subsequently discharged in good condition.

Histologic examination showed numerous nonanastomosing blood vessels as well as endothelial proliferation (Fig. 6A-D). The vessels were lined by a bland single-layer endothelium. In some foci, the lesion is composed of thick-walled vessels and an intraosseous component is also identified. The morphology was consistent with venous vessels, and this is further corroborated using elastin stain confirming the absence of an arterial component. ERG immunostain shows numerous vascular spaces in degenerated areas. The endothelial cells were negative for D2-40, confirming the absence of a lymphatic portion. Lastly, the specimen was also Glut-1 negative and Desmin negative to rule out hemangiomas. Thus, these pathology findings are consistent with angiomatosis of the head and neck.

Postdischarge course was complicated by epistaxis one month from discharge requiring control in the operating room. Patient was noted to have a large hematoma with post-surgical changes which was controlled endoscopically. There was no evidence of disease recurrence or progression. Patient was discharged the following day. One-year post-treatment follow up with nasal endoscopy has demonstrated no recurrence.

Discussion

JNA is the most common benign nasopharyngeal tumor worldwide, thus extensive literature has been published on epidemiology, clinical presentation, and imaging/diagnosis.

JNA is nearly always seen in adolescent males, with only a handful of case reports having described female JNA [17–19]. Studies hypothesize that JNA is androgen receptor dependent, explaining the prevalence in males, but this is still controversial [20]. However, angiomatosis displays a slight female predilection, but also typically presents in the first 2 decades of life [1]. The incidence of JNA is much higher than that of angiomatosis worldwide [6,14]. Given that our patient is a 16 year old male with a mass centered in the sphenoid and pterygopalatine fossa, the preoperative diagnosis of JNA was most likely.

JNA often presents in the paranasal sinuses and arises from the internal maxillary artery with extension into the nasal cavity along the sphenopalatine artery (branches of the external carotid artery). With more aggressive growth, sellar, cavernous sinus, and diffuse skull base extension can occur from pressure resorption. A recent study showed that 10%-37% of JNA cases display intracranial extension [21]. Our patient also presented with skull base dehiscence and sellar extension.

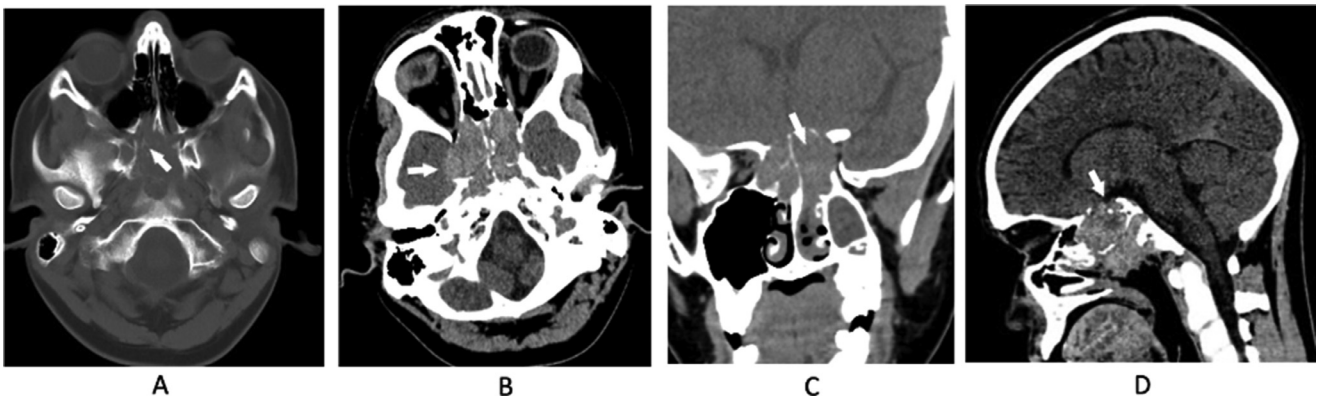

Fig. 2 – CT. (A) Axial CT without contrast demonstrates an expansile destructive lesion with calcifications involving the sphenoid bone and clivus with soft tissue in the pterygopalatine fossa and sphenopalatine foramen (white arrow). No bowing of the posterior maxillary sinus wall. (B) Axial CT without contrast demonstrates the expansile heterogeneous hyperdense soft tissue component centered in the sphenoid bone involving the clivus, extending along the posterior ethmoid sinus and intracranial extension (white arrow). (C) Coronal CT demonstrates involvement of the optic canal (white arrow) with extension to the left nasal cavity and post obstructive changes in the left maxillary sinus. (D) Sagittal CT shows the irregular, expansile destructive lesion of the central skull base, involving the planum sphenoidale and floor of the sella (white arrow) with a lobulated hyperdense soft tissue component with few hypodense foci extending into the posterior nasal cavity and nasopharyngeal soft tissues.

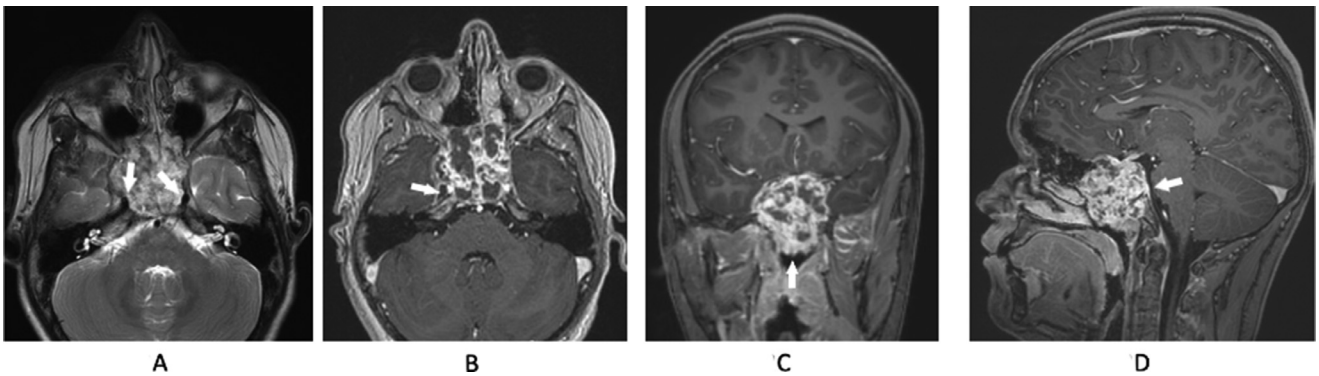

Fig. 3 – MRI. (A) Axial T2 weighted image demonstrates a heterogeneous, expansile mass lesion with scattered low T2 signal intensity areas involving the central skull base extending into the sphenopalatine foramen, the posterior ethmoid sinus and displacing the paraclival internal carotid arteries (white arrows). (B) Axial T1 with contrast demonstrates a heterogeneously, enhancing lesion with non-enhancing cystic areas with extension to Meckel's cave (white arrow), cavernous sinuses and extension into the medial temporal lobes. (C) Coronal T1 demonstrates the mass involvement of the optic canal and encasement of the internal carotid arteries and extending to the nasopharynx (white arrow). (D) Sagittal T1 image demonstrates the involvement of the sphenoid bone and clivus (white arrow), floor of the sella and the anterior cranial fossa with a lobulated soft tissue component extending into the posterior nasal cavity and nasopharyngeal soft tissues.

However, angiomatosis has rarely ever been reported in the head and neck; a handful of case reports have mentioned oropharyngeal involvement specifically, describing lesions of the cheek/lip, masseter, mandible/malar region [1,7]. Only one other case report in the literature has described angiomatosis affecting the paranasal sinuses [6].

Nasal endoscopy for JNA's will show a firm, friable, polypoid mass ranging in color from pale to red-blue depending on extent of hemorrhage/vascularity. In office biopsy for the diagnosis of JNA is generally avoided due to the risk of epistaxis. Thus, intraoperative biopsy and imaging is important in the initial preoperative diagnosis and staging of JNA [16].

CT, MRI, and angiography are gold standard imaging modalities for the diagnosis and preoperative management of JNA. On CT with contrast, JNA shows significant postcontrast enhancement. CT imaging is also effective at detecting the bony erosions of the pterygoid lamina/medial surrounding posterior maxillary wall, pterygoid plates, and skull base from the local destruction of the tumor. A pathognomonic sign of JNA on CT is the Holman Miller sign characterized by anterior bowing of the posterior maxillary sinus wall due to tu-

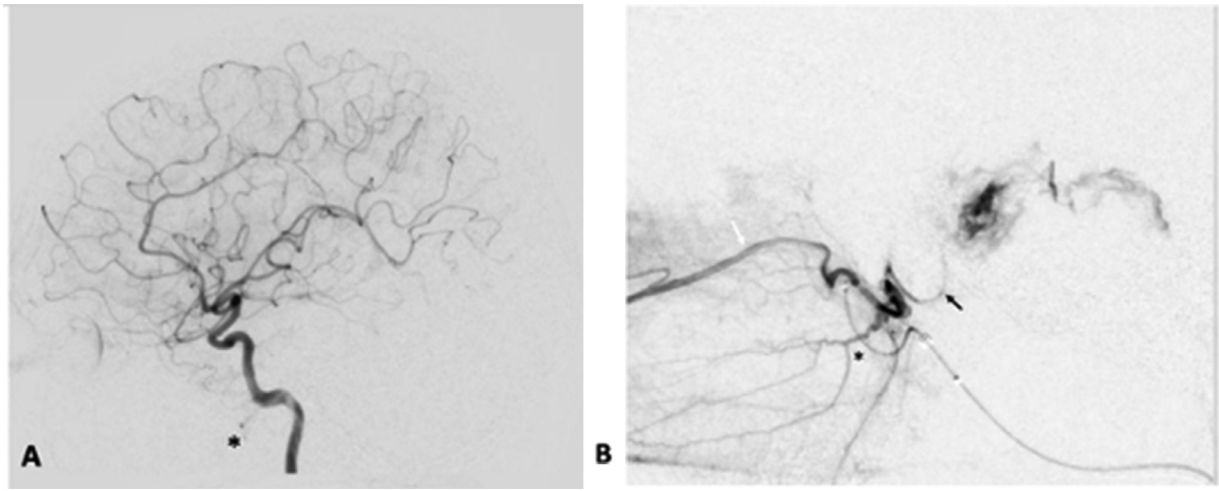

Fig. 4 – Preoperative angiogram. (A) Preoperative imaging from diagnostic cerebral angiography. Lateral view of left internal carotid artery (ICA) showing blush from vidian artery branch of petrous ICA (*). (B) Superselective angiograms (*). Lateral view of microcatheter angiographic run at distal left internal maxillary artery showing infraorbital artery, sphenopalatine artery, and a recurrent branch (black arrow) supplying hypervascular blush.

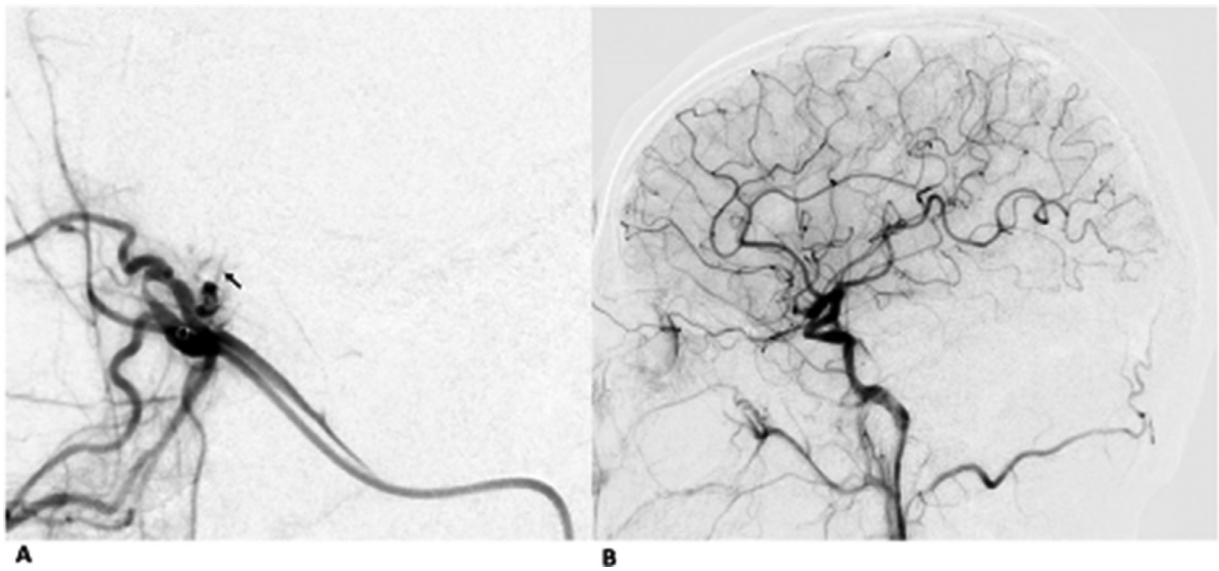

Fig. 5 – Postembolization angiogram. (A) Magnified lateral view of distal left external carotid artery showing embolic material (black arrow) without any hypervascular blush. (B) Unmagnified left common carotid artery angiogram, lateral view, showing reduction of preoperative blush without any off-target embolization.

mor expansion of the pterygomaxillary space. MRI is useful in identifying any soft tissue (eg, orbit) or intracranial extension of tumor [22]. Contrast enhanced MRI shows pathognomonic prominent flow voids with a "salt and pepper" appearance. Angiography has utility in delineation of the feeder vessels of the tumor, showing a highly vascular tumor with a prominent central feeding artery. This is particularly useful for preoperative embolization of feeder vessels to limit intraoperative blood loss and improve endoscopic visualization. The caveat of embolization, however, is increased risk of residual tumor; a lesion that does not bleed can often be missed. While the most common feeding artery for JNA is the internal maxillary artery (branch of the external carotid artery), the ascending pharyngeal artery, also a branch of the external carotid artery has been reported. Rarely, branches of the internal carotid artery, namely the sphenoidal branches, ophthalmic artery, and the cavernous internal carotid have been described [23].

In our case, preoperative CT and MR imaging suggested an aggressive destructive lesion with heterogenous enhancement and numerous cystic/necrotic areas without fluid levels involving the central skull base, pterygopalatine fossa, sphenopalatine foramen with intracranial extension. However, there was no Holman Miller sign typical of JNA's. While JNAs are favored in the differential for vascular tumors of the

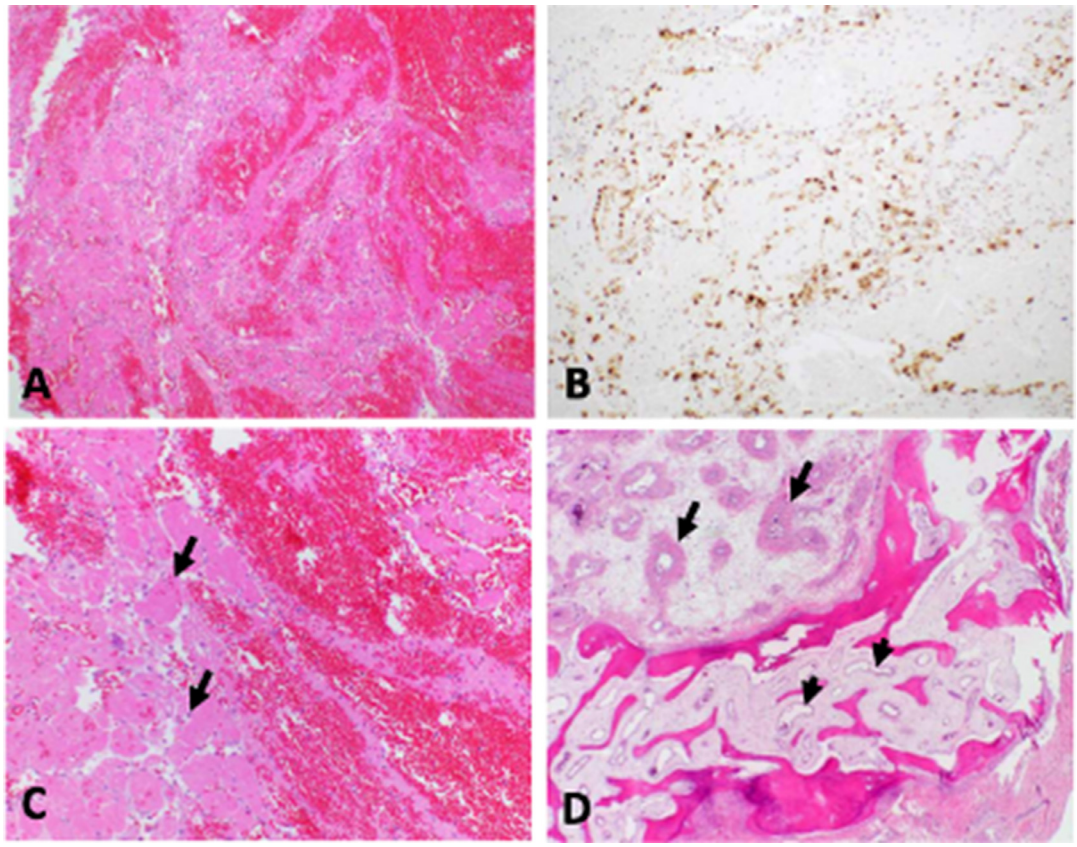

Fig. 6 – Histopathology. (A) The lesion shows poorly formed vascular spaces with degenerative change due to hemorrhage (hematoxylin and eosin, 100X). (B) ERG immunostain shows numerous endothelial cells corresponding to degenerated vascular spaces (immunostain, 100X). (C) In many foci, endothelial papillary proliferation is noted (black arrows) next to large vascular spaces (hematoxylin and eosin, 100X). (D) In relatively preserved foci, the lesion shows thick-walled blood vessels (black arrows) as well as an intraosseous component (black arrowheads) (hematoxylin and eosin, 100x).

nasopharynx, other vascular lesions are to be considered. The International Society for the Study of Vascular Anomalies (ISSAVA) updated classification divides vascular lesions into 2 groups: tumors (proliferative neoplasms) and malformations (abnormal morphogenesis). Following this classification, potential benign vascular tumors of the head and neck include infantile/congenital hemangioma, tufted angioma, spindle cell hemangioma, and pyogenic granuloma [13]. The differential diagnosis further includes locally aggressive/borderline tumors of the head and neck such as kaposiform hemangioendothelioma, hemangioendothelioma, and papillary intralymphatic angioendothelioma [13].

While angiomatosis and JNA are both highly vascular lesions with similar radiographic characteristics, histopathological diagnosis via biopsy is the gold standard to differentiate the two [16]. Angiomatosis has been demonstrated to present in 2 histopathological patterns. The most common pattern consists of a disorganized proliferation of varying sized vessels, with characteristic clusters of capillary-sized vessels residing within, or adjacent, to vein walls. The second, yet uncommon, pattern is the presence of clusters of capillary sized vessels demonstrating soft tissue invasion. A feature commonly present in both patterns of angiomatosis is the presence of large amounts of adipose tissue [2]. In contrast, characteristic histopathological findings of JNA are defined as the presence of highly vascular fibrous proliferation within a densely collagenized and cellular stroma, with characteristic angular to plump stellate cells, identified as fibroblasts. In a study aimed at examining immunohistochemical features of JNA, the authors identified high levels of Ki-67 expression among proliferating endothelial cells, stromal cell expression of vimentin and factor XIIIa, and lymphatic presence at the periphery [24].

This case describes a patient with histologically proven angiomatosis of the head and neck whose presentation was similar to JNA with respect to clinical presentation and radiographic findings. Notably, the patient did not present with epistaxis or a Hollman Miller sign commonly seen in patients with JNA.

Adelson et al. described the first and only other reported case of paranasal sinus angiomatosis in the literature [6]. Their patient was a 34 year old male who was referred for evaluation of a multiply recurrent left middle meatal mass. Preoperative imaging with postcontrast MRI similarly demonstrated a submucosal vascular lesion with bony remodeling. Patient underwent endoscopic resection with ligation of the left sphenopalatine artery. Histopathology showed diffuse vascular proliferation and immunohistochemical stain-

ing positive for CD31 and factor VIII consistent with angiomatosis. This case was similar to that presented in this report with a lesion with characteristic vascular that required tissue diagnosis to differentiate from angiomatosis.

Surgical treatment for JNAs and angiomatosis are similar with respect to complete endoscopic surgical resection. Nonsurgical treatment of JNAs include tumor irradiation for nonsurgical candidates or those with incomplete surgical resection [25]. Published nonsurgical treatment of angiomatosis include radiotherapy or interferon alpha 2a [1]. Despite treatment, both have high recurrence rates with JNAs recurring in up to 20-57% of patients and angiomatosis recurring in up to 90% [26]. Biannual nasal endoscopy with annual MRI is recommended for post-op surveillance of recurrence/residual tumor. Because both angiomatosis and JNA are locally aggressive vascular lesions with high recurrence rates, timely diagnosis, accurate staging, complete resection with negative margins, and routine postoperative surveillance are critical to reduce recurrence [16,23].

Lastly, since our patient presented with both a meningioma and angiomatosis, concern exists for possible genetic syndromes. Congenital angiomatosis syndromes include: Von-Hippel Lindau, Sturge Weber, Kippel-Trenaunay-Weber, Sneddon syndrome, and Gorham disease [6]. However, there are no reports in the literature describing synchronous or metachronous meningiomas in the context of any of the aforementioned congenital syndromes with angiomatosis.

Conclusion

This case presents an extremely rare presentation of angiomatosis of the paranasal sinuses and skull base. Patient demographics, location/extension of tumor, clinical presentation, and imaging were all suggestive of JNA. Angiomatosis can mimic JNA and other vascular tumors, so it must be included in the differential diagnosis of sinonasal tumors. Histopathological diagnosis is needed for diagnosis. Timely accurate diagnosis, complete surgical resection, and regular post-op surveillance are essential to reduce recurrence rates.

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Patient consent

Was obtained from all individual participants included in the study. The participants have consented to the submission of the case report to the journal.

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