



Case Report

Giant primary intracranial multi-fossa leiomyosarcoma involving the frontal sinus, ethmoid air cells, anterior fossa, middle fossa, and intraventricular space: A case report and literature review

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ABSTRACT

Background: Leiomyosarcomas (LMSs) is a type of sarcoma that arises from smooth muscle and generally presents in the abdomen. Although intracranial LMS has been identified before, most reported presentations have been in immunocompromised patients. Here, we present an intracranial LMS in an immunocompetent patient.

Case Description: A 22-year-old male with a history of an atypical pineal parenchymal tumor of intermediate differentiation resected by suboccipital craniotomy at the age of 12 followed by adjuvant radiation therapy, presented with 3 weeks of decreased appetite, weight loss, and lethargy. He subsequently underwent transbasal approach skull base tumor resection. Histologic examination of the mass along with the patient's history of radiation was supportive of a low-grade, radiation-induced LMS arising from the anterior fossa of the skull or meninges and extends to the frontal sinus and ethmoid air cells.

Conclusion: Primary intracranial LMS is an extremely rare diagnosis and presenting symptoms vary with the location and size of the tumor. Due to the poor specificity of clinical symptoms, diagnosis is often based on histology. The most common treatment is surgical resection. Adjuvant chemotherapy with various agents has been found to be somewhat effective outside the central nervous system. When LMS does occur, a history of immunocompromised state or previous radiation exposure is often present. Pathological confirmation is required for an appropriate diagnosis.

Keywords: Anterior fossa, Ethmoid air cells, Frontal sinus, Leiomyosarcoma, Middle fossa, Transbasal approach

INTRODUCTION

Leiomyosarcoma (LMS) is a rare subtype of soft-tissue sarcoma that originates from smooth muscle cells.^[15] These tumors can occur in various parts of the body, typically presenting in the abdomen, retroperitoneum, and peritoneum^[45] with the uterus being a common site of occurrence.^[15] Representing only 2% of all histologically confirmed tumors in adults, LMS is a

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subset of sarcomas, which are themselves rare tumors arising from mesenchymal tissue.^[45]

The incidence of LMS increases with age, peaking in the seventh decade of life. There are no clear predisposing factors for the development of LMS, although patients with hereditary retinoblastoma or TP53 mutations are at risk. Histologically, LMS is characterized by intersecting, sharply marginated fascicles of spindle cells with abundant eosinophilic cytoplasm and elongated, hyperchromatic nuclei. These features are indicative of the smooth muscle lineage from which these tumors arise. Treatment options for LMS include surgery, radiation therapy, and chemotherapy.^[15]

The incidence of LMS in the central nervous system (CNS), specifically intracranial LMS, is extremely rare. Most cases of intracranial LMS are secondary, meaning they have metastasized or spread from tumors located elsewhere in the body.^[2,25,39,44] Primary intracranial LMS is a much rarer entity.^[18] Historically, these tumors have been found to arise from the blood vessels of the brain, particularly the walls of the cerebral veins and sinuses, which contain smooth muscle tissue.^[2,25,39,44]

Intracranial LMS has been reported in the literature, but most cases have been identified in immunocompromised patients, particularly those with human immunodeficiency virus (HIV).^[11,39] The demographic distribution of intracranial LMS is skewed toward males with a wide age range reported, from teenagers to the elderly.^[2,25,41,44,46] Risk factors for intracranial LMS include a history of immunocompromised state, such as HIV infection, and previous radiation exposure.^[39,41,44,46] The etiology of intracranial LMS is not well understood, but it is believed to arise from the mesenchymal cells of the dura mater or cerebral blood vessels. Intracranial LMS traditionally presents in the left lateral ventricle or originates from the skull.^[2,25,44] Common symptoms vary with the location and size of the tumor but often include persistent headaches, nausea, vomiting, decreased appetite, weight loss, and lethargy.^[2,18,25,39,41,44,46] Management of intracranial LMS is challenging due to its rarity. However, gross total resection has been found to be a significantly favorable factor for increased progression-free survival and overall survival. Postoperative radiation is also suggested to help increase the survival of patients with intracranial LMS.^[44]

In this study, we present a case of low-grade, radiation-induced LMS arising from the anterior skull base in a patient with a history of an atypical pineal parenchymal tumor of intermediate differentiation. This case adds to the limited literature on primary intracranial LMS and may serve as a potential reference for clinicians and clinical studies.

CASE DESCRIPTION

A 22-year-old male presented with a history of atypical pineal parenchymal tumor of intermediate differentiation resected

by suboccipital craniotomy at the age of 12 with adjuvant radiation and chemotherapy, followed by a large, atypical meningioma resected through the left parietal craniotomy at the age of 14. The patient reported 3 weeks of altered mental status, visual deficits, fatigue, decreased appetite, weight loss, and lethargy.

On physical examination, he had dysarthria with the paucity of speech without receptive or expressive aphasia – there was some difficulty with repeating complex sentences. There were no focal neurological deficits, the sensation was intact, and there was full strength and spontaneous movement of all extremities. The patient was afebrile and preliminary laboratory results were unremarkable.

Imaging showed a giant – 9.7 cm anteroposterior × 7.5 cm transverse × 6.6 cm craniocaudal – bilobed, heterogeneous, partially cystic mass centered in the anterior cranial fossa, extending to the middle fossa, and intraventricularly with marked mass effect including regional erosion and involvement of the ethmoid air cells and frontal sinus [Figures 1 and 2].

Given the patient's history, differential diagnostic considerations included atypical/malignant radiation-induced meningioma or metastatic pineal parenchymal tumor. He subsequently underwent extensive tumor resection through bifrontal craniotomy with a skull base approach to the left orbit and ethmoid sinus for microsurgical resection.

Surgical description

A right parietal ventriculostomy was placed and a bicoronal incision was done for a bifrontal stereotactic craniotomy with pericranium preservation. The bifrontal craniotomy was done with multiple bur holes, one above the glabella, two frontal bilateral, and then intermediate around the superior sagittal sinus. Afterward, the epidural dissection was performed, and the bone flap was removed. An extended transbasal approach was, then, added. The tumor was extending

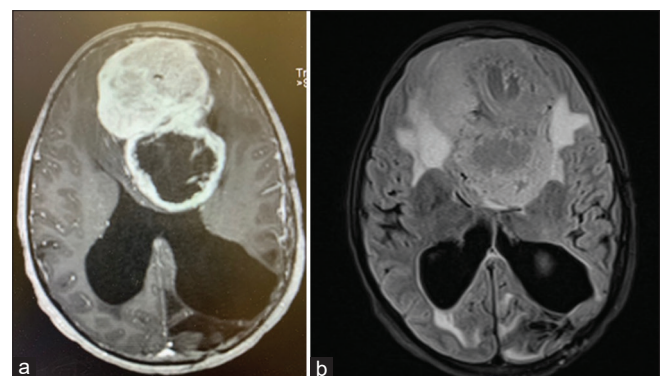


Figure 1: (a) Axial magnetic resonance imaging (MRI) with contrast showing tumor extending from anterior fossa to intraventricular space. (b) Axial MRI with fluid-attenuated inversion recovery sequence showing the significant perilesional edema.

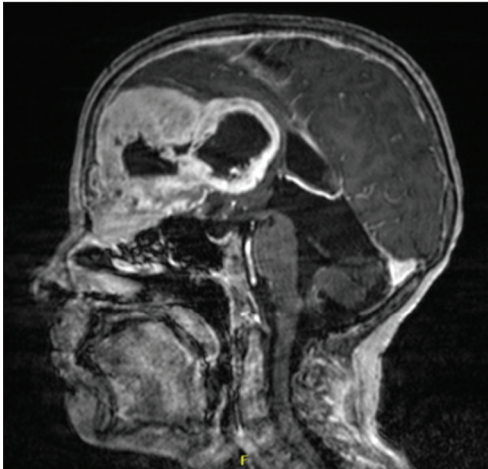


Figure 2: Sagittal magnetic resonance imaging with contrast showing a large mass centered on the left side of anterior cranial fossa/anterior falx with invasion of the skull base, medial left orbit, and left frontal sinus. There was notable erosion of the lamina papyracea on the left with extensive regional mass effect and effacement of the bilateral frontal horns and possible entrapment/developing hydrocephalus of the right occipital horn.

toward the ethmoid sinus and into the left orbit. Using bipolar electrocoagulation, the tumor was devascularized from the anterior and posterior ethmoid arteries bilaterally. We proceeded to debulk the orbital segment of the tumor, followed by the ethmoidal segment, and then finally the dura and anterior fossa. The anterior aspect of the superior sagittal sinus was ligated just behind the tumor using silk sutures as well as hemo-clips. Debulking was done with the ultrasonic aspirator. After adequate central debulking, we were able to access the posterior lobule of the tumor and collapsed the capsule, dissecting it away from the third ventricle and lateral ventricle. When approaching the segment of the tumor that was attached to the pericallosal artery on the right side, a small opening of the pericallosal artery was encountered. A temporary clip was used proximally, and (9 0) sutures were placed to close the area of hemorrhage adequately. The temporary clip was removed, and adequate flow was restored after this. Hemostasis was carefully achieved. An absorbable hemostatic sponge and surgical glue were used to isolate the ventricle from the surgical cavity in the anterior fossa. An additional ventriculostomy was placed with direct visualization. We used a fat graft that was harvested from the abdomen to seal the anterior fossa around the ethmoid sinus and the orbit. We placed fibrin glue and then a titanium plate was used to reconstruct the anterior aspect of the floor of the anterior fossa. We continued to perform the suturing of the pericranium down to the dura at the level of the titanium mesh. At this point, we placed a nonsuturable dura substitute and fibrin glue followed by a suturable dura substitute in

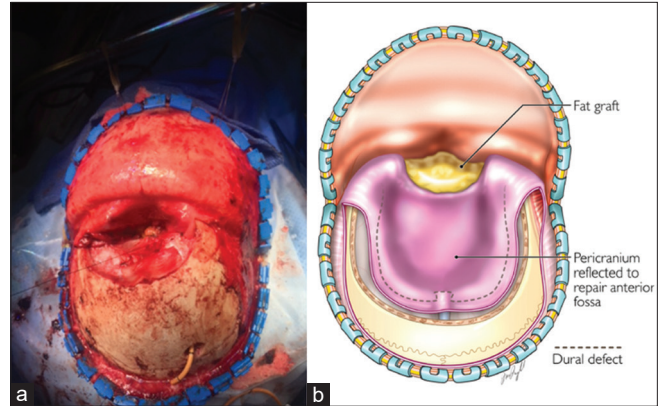


Figure 3: (a) Surgical photo from pericranial graft already sutured to available dura of the skull base and fat graft harvested. The defect of the transbasal approach is evident. (b) Illustration of the surgical reconstruction.

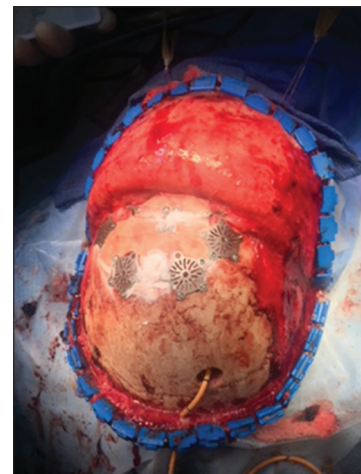


Figure 4: Surgical image of bone flap replacement, including the transbasal approach bone flap.

the convexity [Figure 3]. The bone flap was replaced with miniplates and screws, and a subgaleal drain was placed as well [Figure 4].

Pathology

Histologic examination of the mass revealed a spindle-cell neoplasm with fasciculate architecture and strong diffuse reactivity to caldesmon, supporting smooth vessel origins. The tumor specimen was negative for STAT-6 and e-cadherin staining. These findings, along with the patient's history of radiation, were supportive of an LMS arising from the base of the skull or meninges.

Outcome

The patient required a ventriculoperitoneal shunt and was discharged on postoperative day 20 to acute inpatient

Table 1: A literature review of primary intracranial LMS.

Author, publication year	Location	Age	Sex	Tumor size (mm)
Anderson et al., ^[4] 1980	Sella turcica	35	Male	N/a
Asai et al., ^[5] 1988	Right temporal region	73	Male	30 (Diameter)
Louis et al., ^[25] 1989	Lateral ventricle	72	Female	N/a
Skullerud et al., ^[40] 1995	Pineal region	33	Male	N/a
Lee and Page, ^[22] 1997	Parietal region	8	Male	N/a
Mierau et al., ^[29] 1997	Left temporal region	14	Female	30×30×20
Litofsky et al., ^[24] 1998	Right occipital region	50	Male	32×14×10
Kleinschmidt-DeMasters et al., ^[21] 1998	Left dural transverse sinus	14	Female	N/a
Bejjani et al., ^[7] 1999	Lateral sphenoid wing	38	Male	N/a
Brown et al., ^[9] 1999	Ventral left pons	34	Female	20×30
Merimsky et al., ^[28] 2000	Temporo-occipital region	33	Male	N/a
Oliveira et al., ^[33] 2002	Temporal region	58	Female	N/a
Eckhardt et al., ^[10] 2004	Left parietal region	13	Male	80×70×40
Hussain et al., ^[18] 2006	Right parieto-occipital region	26	Male	N/a
Mathieson et al., ^[27] 2009	Frontal region	5	Male	N/a
Fujimoto et al., ^[12] 2011	Left cerebellopontine angle	45	Female	N/a
Aeddula et al., ^[1] 2011	Left anteromedial temporal lobe	58	Male	16×13
Almubaslat et al. ^[3] 2011	Left frontoparietal lobe	47	Female	63×50×30
Zhang et al., ^[45] 2012	Body of corpus callosum	26	Female	N/a
Alijani et al., ^[2] 2013	Right parieto-occipital region	19	Male	50 (diameter)
Takei et al., ^[42] 2013	Right anterior frontal lobe	27	Male	26×40×33
Saito et al., ^[36] 2014	Cavernous sinus	75	Female	N/a
Gulwani and Garg, ^[16] 2014	Cavernous sinus	55	Female	N/a
Aumüller et al., ^[6] 2014	Torcular herophili	12	Female	40×60
Maslehaty et al., ^[26] 2016	Right posterior fossa	43	Male	N/a
Polewski et al., ^[35] 2016	Left tentorium	41	Female	60×50×46
Gautam and Meena, ^[14] 2017	Left frontotemporal and basal ganglia region	45	Male	59×44×52
Kawabata et al., ^[20] 2018	Right parietal region	76	Female	50 (diameter)
Torihashi et al., ^[43] 2018	Left frontal region	41	Female	40×40×50
Gallagher et al., ^[13] 2018	Left tentorium cerebelli	43	Female	60×60×50
Francisco et al., ^[11] 2018	Left parietal lobe	29	Male	33.6×26.4×20.0
Francisco et al., ^[11] 2018	Left occipital lobe	36	Male	20×11×16
Li, ^[23] 1987	Pineal gland	47	Male	30×25×20
Kamian et al., ^[19] 2020	Frontal region	31	Male	34×28
Morales et al., ^[30] 2020	Left cavernous sinus	23	Male	32×35×46
Bregy et al., ^[8] 2020	Right frontal region	65	Female	32×22
Zhao et al., ^[46] 2021	Right temporal lobe	38	Male	31×25 and 40×18×37
Zhang et al., ^[44] 2021	Parieto-occipital region	19	Female	70×50×30
Zhang et al., ^[44] 2021	Sellar region	48	Female	30×30×20
Zhang et al., ^[44] 2021	Frontal region	51	Female	50×45×30
Zhang et al., ^[44] 2021	Cerebellopontine angle	13	Female	35×30×40
Zhang et al., ^[44] 2021	Medulla oblongata	67	Male	20×10×10
Zhang et al., ^[44] 2021	Parieto-occipital region	51	Female	65×60×50
Zhang et al., ^[44] 2021	Sellar region	50	Male	40×35×30
Zhang et al., ^[44] 2021	Frontal region	58	Female	40×40×30
Zhang et al., ^[44] 2021	Thalamus	57	Female	33×35×40
Zhang et al., ^[44] 2021	Middle and posterior fossa	24	Male	59×37×57
Zhang et al., ^[44] 2021	Frontoparietal region	56	Female	45×45×45
Zhang et al., ^[44] 2021	Fourth ventricle	35	Male	24×25×20
Zhang et al., ^[44] 2021	Temporal region	63	Female	55×52×45
Selbi et al., ^[37] 2023	Right middle cranial fossa	60	Female	N/a
Tabor et al., ^[41] 2023	Left middle cranial fossa	40	Male	58×55×44

LMS: Leiomyosarcoma, N/A: Not applicable

rehabilitation in improved and stable condition. At the patient's most recent follow-up (33 months after), there was the presence of a small area of enhancement at the anterior fossa, some ventricular enlargement, and mild enlargement of left parietal enhancing lesions on imaging. However, due to the patient's clinical condition, it was felt that further treatment was not necessary. Clinically, the patient had improved physical, visual, and cognitive abilities. In addition, the patient demonstrated the ability to independently ambulate.

DISCUSSION

Within the context of intracranial tumors, metastatic LMS is rare, and primary intracranial LMS is rarely reported.^[38] Table 1 illustrates a cumulative literature review of LMS as well as the unique manifestation of LMS in the ethmoid air cells and frontal sinus within the context of an immunocompetent patient presented in this case report.^[1,3,5,6,8,10,13,16,19,20,22-24,26,29,33,35-37]

Examination of Table 1 highlights the fact that no reported LMS has been reported with the same spatial distribution. Furthermore, the total number of reported primary intracranial LMS is 52 and the average age of these patients was 41.98 ± 17.99 years. Of these patients, 25 were male, and 26 were female exhibiting approximately a 1:1 male-to-female ratio. In addition, these tumors were found to be of increased incidence in immunocompromised individuals,^[7,11,27] Epstein-Barr virus infected individuals^[9,21,41,42] as well as individuals previously operated on or treated with radiation therapy.^[12,40]

While the histogenesis of intracranial LMS has not been fully elucidated, several theories pertaining to the origin of such tumors have been proposed. These theories are based on the idea that LMS tumors are mesenchymal-derived tumors. Therefore, conjectures have been proposed claiming transformation from the inner layers of the arachnoid, pia-mater,^[2,7,21,44] and the intracranial vasculature.^[4,7,12,28,43] The significant number of intracranial LMS in association with the dura lends support to the theory of neoplastic transformation of the arachnoid and pia.^[2,7,21,44] The theory of vascular smooth muscle transformation was one of the first theories proposed as to the histogenesis of LMS by Anderson *et al.* in 1980 and has remained popular in the literature.^[4,12,28] With regard to the LMS described in this case report, both theories may be applicable. The ethmoid sinus is well known to be vascularized and the spongy bone predominating the area would give a large surface area and thus opportunity for the LMS to develop from osseous mesenchymal precursors as have been described by others.^[2] We would like to suggest an additional theory describing the etiology of LMS. Within the paranasal sinuses, the lamina propria is a fundamental feature of the histological landscape. The lamina propria is

well described in the literature and is known to be composed of a connective tissue layer with blood vessels, nerves, and of note inflammatory cells. This may be of special interest as LMS has been noted to develop at an increased frequency in immunocompromised patients,^[7,11,27] and perhaps some association lies between the balance of immune cells and the development of mesenchymal tissues of the lamina propria.

An alternative etiologic theory is vested in the association of LMS with HIV. Pang *et al.* demonstrated that mesenchymal stem cells play a role in macrophage, astrocyte, and T lymphocyte-mediated neuroinflammation.^[34] It was found that these mesenchymal stem cell precursors migrate from sources through the bloodstream to areas of injury largely through the expression of CXCR4.^[17,32] CXCR4 and CCR5 are known to be the two major coreceptors for HIV entry into cells.^[31] As HIV is a known risk factor for primary intracranial LMS,^[11,14,30,44] it, therefore, stands to reason that perhaps some mechanism exists between previous prior intracranial insult, leading to upregulation of CXCR4 and thus migration of mesenchymal tissues within the context of HIV that is responsible for the rare development of intracranial LMS. This is all of course conjecture, and further research is necessary as no mechanism has been defined for the histogenesis of LMS.

CONCLUSION

LMSs are rare tumors that seldom present in the CNS, therefore leading to a low index of suspicion in the preoperative differential diagnosis. When LMSs do occur, a history of immunocompromised state or previous radiation exposure is often present. Pathological confirmation is required for an appropriate diagnosis. Microsurgical resection of intracranial lesions when mass effect is present is highly recommended. Adjuvant chemotherapy and radiation appear to be effective treatment modalities; however, further research is necessary in CNS LMS.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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