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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

Hannah Choi[1], Jorrdan N. R. Bissell[1], Brandon Michael Edelbach[1], Joel Paea[1], Emmanuel Omosor[1], Ravi Raghavan[1], Vadim Gospodarev[2], Miguel Angel Lopez-Gonzalez[2]

[1]Department of Neurosurgery, Loma Linda University Medical School, [2]Department of Neurosurgery, Loma Linda University Medical Center, Loma Linda, California, United States.

E-mail: Hannah Choi - hchoi@llu.edu; Jorrdan N. R. Bissell - jbissell@llu.edu; Brandon Michael Edelbach - bedelbach@students.llu.edu; Joel Paea - jpaea@students.llu.edu; Emmanuel Omosor - eomosor@students.llu.edu; Ravi Raghavan - rraghavan@llu.edu; Vadim Gospodarev - vgospodarev@llu.edu; *Miguel Angel Lopez-Gonzalez - mlopezgonzalez@llu.edu

***Corresponding author:**
Miguel Angel Lopez-Gonzalez, Department of Neurosurgery, Loma Linda University Medical Center, Loma Linda, California, United States.

mlopezgonzalez@llu.edu

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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

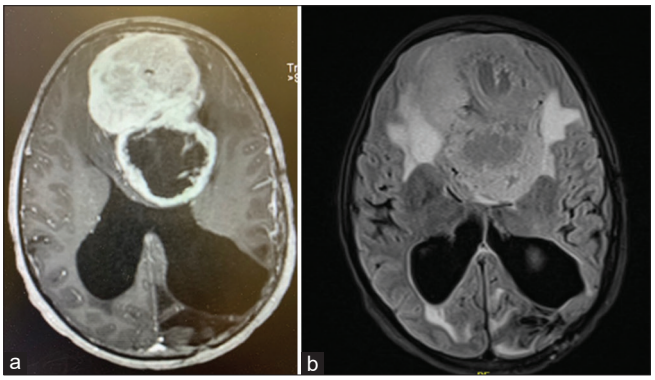

Figure 1: (a) Axial magnetic resonance imaging (MRI) with contrast showing tumor extending from anterior fossa to intraventricular space. (b) Axial MRI with fluid-attenuated inversion recovery sequence showing the significant perilesional edema.

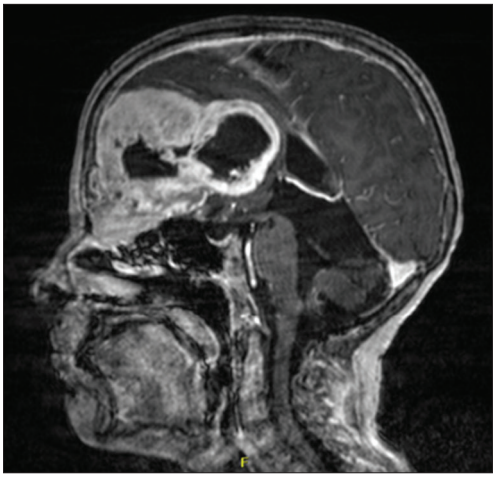

Figure 2: Sagittal magnetic resonance imaging with contrast showing a large mass centered on the left side of anterior cranial fossa/anterior falx with invasion of the skull base, medial left orbit, and left frontal sinus. There was notable erosion of the lamina papyracea on the left with extensive regional 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

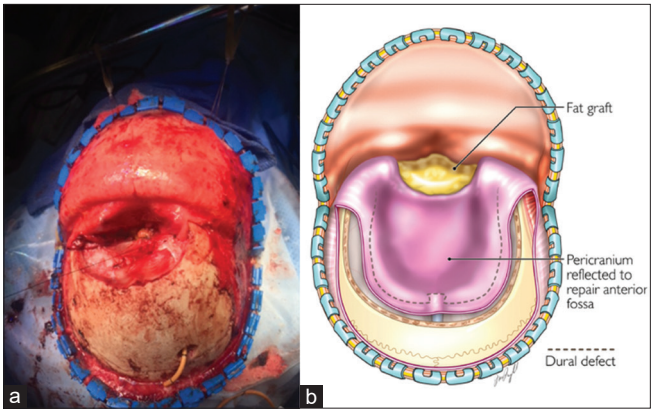

Figure 3: (a) Surgical photo from pericranial graft already sutured to available dura of the skull base and fat graft harvested. The defect of the transbasal approach is evident. (b) Illustration of the surgical reconstruction.

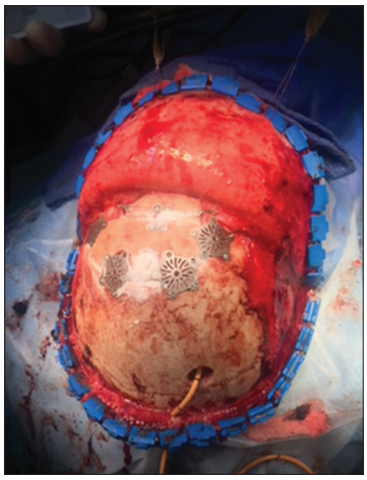

Figure 4: Surgical image of bone flap replacement, including the transbasal approach bone flap.

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.

REFERENCES

1. Aeddula NR, Pathireddy S, Samaha T, Ukena T, Hosseinnezhad A. Primary intracranial leiomyosarcoma in an immunocompetent adult. J Clin Oncol 2011;29:e407-10.
2. Alijani B, Yousefzade S, Aramnia A, Mesbah A. Primary intracranial leiomyosarcoma. Arch Iran Med 2013;16:606-7.
3. Almubaslat M, Stone JC, Liu L, Xiong Z. Primary intracranial leiomyosarcoma in an immunocompetent patient. Clin Neuropathol 2011;30:154-7.
4. Anderson WR, Cameron JD, Tsai SH. Primary intracranial leiomyosarcoma. Case report with ultrastructural study. J Neurosurg 1980;53:401-5.
5. Asai A, Yamada H, Murata S, Matsuno A, Tsutsumi K, Takemura T, *et al*. Primary leiomyosarcoma of the dura mater. Case report. J Neurosurg 1988;68:308-11.
6. Aumüller M, Sykora KW, Hartmann C, Hermann EJ, Krauss JK. Primary intracranial leiomyosarcoma of the torcular Herophili associated with Fanconi anemia and allogenic stem cell transplantation. Childs Nerv Syst 2014;30:1613-6.
7. Bejjani GK, Stopak B, Schwartz A, Santi R. Primary dural leiomyosarcoma in a patient infected with human immunodeficiency virus: Case report. Neurosurgery 1999;44:199-202.
8. Bregy A, Lim J, Lohman R, Kane J, Prasad D, Qiu J, *et al*. Primary leiomyosarcoma of the calvarium with intracranial extension: A case report. Indian J Surg Oncol 2020;11:165-9.
9. Brown HG, Burger PC, Olivi A, Sills AK, Barditch-Crovo PA, Lee RR. Intracranial leiomyosarcoma in a patient with AIDS. Neuroradiology 1999;41:35-9.
10. Eckhardt BP, Brandner S, Zollikofer CL, Wentz KU. Primary cerebral leiomyosarcoma in a child. Pediatr Radiol 2004;34:495-8.
11. Francisco CN, Alejandria M, Salvaña EM, Andal VM. Primary intracranial leiomyosarcoma among patients with AIDS in the era of new chemotherapeutic and biological agents. BMJ Case Rep 2018;2018:bcr2018225714.
12. Fujimoto Y, Hirato J, Wakayama A, Yoshimine T. Primary intracranial leiomyosarcoma in an immunocompetent patient: Case report. J Neurooncol 2011;103:785-90.
13. Gallagher SJ, Rosenberg SA, Francis D, Salamat S, Howard SP, Kimple RJ. Primary intracranial leiomyosarcoma in an immunocompetent patient: Case report and review of the literature. Clin Neurol Neurosurg 2018;165:76-80.
14. Gautam S, Meena RK. Primary intracranial leiomyosarcoma presenting with massive peritumoral edema and mass effect: Case report and literature review. Surg Neurol Int 2017;8:278.
15. George S, Serrano C, Hensley ML, Ray-Coquard I. Soft tissue and uterine leiomyosarcoma. J Clin Oncol 2018;36:144-50.
16. Gulwani HV, Garg N. Primary extradural leiomyosarcoma involving cavernous sinus in an immunocompetent patient. Indian J Neurosurg 2014;3:115-7.
17. Hassanshahi G, Roohi MA, Esmaeili SA, Pourghadamyari H, Nosratabadi R. Involvement of various chemokine/chemokine receptor axes in trafficking and oriented locomotion of mesenchymal stem cells in multiple sclerosis patients. Cytokine 2021;148:155706.
18. Hussain S, Nanda A, Fowler M, Ampil FL, Burton GV. Primary intracranial leiomyosarcoma: Report of a case and review of the literature. Sarcoma 2006;2006:52140.
19. Kamian S, Ebrahimi A, Zadeh KE, Behzadi B. Primary intracranial leiomyosarcoma presenting with frontal bone mass: A case report. Radiat Oncol J 2020;38:282-6.
20. Kawabata Y, Aoki T, Yamamoto T, Yasui H, Sawai S, Fukuda S, *et al*. Pazopanib-mediated long-term disease stabilization after local recurrence and distant metastasis of primary intracranial leiomyosarcoma: A case report on the efficacy of pazopanib as a salvage therapy. NMC Case Rep J 2018;5:1-7.
21. Kleinschmidt-DeMasters BK, Mierau GW, Sze CI, Breeze RE, Greffe B, Lillehei KO, *et al*. Unusual dural and skull-based mesenchymal neoplasms: A report of four cases. Hum Pathol 1998;29:240-5.
22. Lee TT, Page LK. Primary cerebral leiomyosarcoma. Clin Neurol Neurosurg 1997;99:210-2.
23. Li NY. Primary leiomyosarcoma of the pineal gland--a case report. Zhonghua Zhong Liu Za Zhi 1987;9:463-4, 22.
24. Litofsky NS, Pihan G, Corvi F, Smith TW. Intracranial leiomyosarcoma: A neuro-oncological consequence of acquired immunodeficiency syndrome. J Neurooncol 1998;40:179-83.
25. Louis DN, Richardson EP Jr., Dickersin GR, Petrucci DA, Rosenberg AE, Ojemann RG. Primary intracranial leiomyosarcoma. Case report. J Neurosurg 1989;71:279-82.
26. Maslehaty H, Nabavi A, Mehdorn HM. Primary intracranial leiomyosarcoma: Case report and principles for treatment. J Neurol Neuromed 2016;1:16-20.
27. Mathieson CS, St George EJ, Stewart W, Sastry J, Jamal S. Primary intracranial leiomyosarcoma: A case report and review of the literature. Childs Nerv Syst 2009;25:1013-7.
28. Merimsky O, Lepechoux C, Terrier P, Vanel D, Delord JP, LeCesne A. Primary sarcomas of the central nervous system. Oncology 2000;58:210-4.
29. Mierau GW, Greffe BS, Weeks DA. Primary leiomyosarcoma of brain in an adolescent with common variable immunodeficiency syndrome. Ultrastruct Pathol 1997;21:301-5.
30. Morales RL, Álvarez A, Noreña MP, Torres F, Esguerra J. Low-grade leiomyosarcoma of the cavernous sinus in an HIV positive patient: Case report. Cureus 2020;12:e6758.
31. Murakami T, Yamamoto N. Role of CXCR4 in HIV infection and its potential as a therapeutic target. Future Microbiol 2010;5:1025-39.
32. Nitzsche F, Müller C, Lukomska B, Jolkkonen J, Deten A, Boltze J. Concise review: MSC adhesion cascade-insights into homing and transendothelial migration. Stem Cells 2017;35:1446-60.
33. Oliveira AM, Scheithauer BW, Salomao DR, Parisi JE, Burger PC, Nascimento AG. Primary sarcomas of the brain and spinal cord: A study of 18 cases. Am J Surg Pathol 2002;26:1056-63.
34. Pang QM, Chen SY, Fu SP, Zhou H, Zhang Q, Ao J, *et al*.

Regulatory role of mesenchymal stem cells on secondary inflammation in spinal cord injury. J Inflamm Res 2022;15:573-93.
35. Polewski PJ, Smith AL, Conway PD, Marinier DE. Primary CNS leiomyosarcoma in an immunocompetent patient. J Oncol Pract 2016;12:827-9.
36. Saito N, Aoki K, Hirai N, Ishii M, Fujita S, Hiramoto Y, *et al*. Primary intracranial leiomyosarcoma of the cavernous sinus: Case report. Austin J Med Oncol 2014;1:2.
37. Selbi W, Sims-Williams H, Ince P, Carroll TA. Skull base angiomatous leiomyoma: A case report and review of literature. Br J Neurosurg 2023;37:668-70.
38. Serrano C, George S. Leiomyosarcoma. Hematol Oncol Clin North Am 2013;27:957-74.
39. Sivendran S, Vidal CI, Barginear MF. Primary intracranial leiomyosarcoma in an HIV-infected patient. Int J Clin Oncol 2011;16:63-6.
40. Skullerud K, Stenwig AE, Brandtzaeg P, Nesland JM, Kerty E, Langmoen I, *et al*. Intracranial primary leiomyosarcoma arising in a teratoma of the pineal area. Clin Neuropathol 1995;14:245-8.
41. Tabor JK, Lei H, Morales-Valero SF, O'Brien J, Gopal PP, Erson-Omay EZ, *et al*. Epstein-Barr virus-associated primary intracranial leiomyosarcoma in an immunocompetent patient: Illustrative case. J Neurosurg Case Lessons 2023;5:CASE22532.
42. Takei H, Powell S, Rivera A. Concurrent occurrence of primary intracranial Epstein-Barr virus-associated leiomyosarcoma and Hodgkin lymphoma in a young adult. J Neurosurg 2013;119:499-503.
43. Torihashi K, Chin M, Yoshida K, Narumi O, Yamagata S. Primary intracranial leiomyosarcoma with intratumoral hemorrhage: Case report and review of literature. World Neurosurg 2018;116:169-73.
44. Zhang GJ, Weng JC, Huo XL, Ma JP, Wang B, Wang L, *et al*. Surgical management and long-term outcomes of primary intracranial leiomyosarcoma: A case series and review of literature. Neurosurg Rev 2021;44:2319-28.
45. Zhang H, Dong L, Huang Y, Zhang B, Ma H, Zhou Y, *et al*. Primary intracranial leiomyosarcoma: Review of the literature and presentation of a case. Onkologie 2012;35:609-16.
46. Zhao L, Jiang Y, Wang Y, Bai Y, Sun Y, Li Y. Primary intracranial leiomyosarcoma secondary to glioblastoma: Case report and literature review. Front Oncol 2021;11:642683.

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