ScientificScholar®
Knowledge is power
Publisher of Scientific Journals

www.surgicalneurologyint.com

Surgical Neurology International

Editor-in-Chief: Nancy E. Epstein, MD, Clinical Professor of Neurological Surgery, School of Medicine, State U. of NY at Stony Brook.

SNI: Neuropathology

Editor
Istvan Vajtai, MD
Service of Clinical Pathology, University Hospital of Geneva, Switzerland

SNI
Open Access

Case Report

Pediatric sellar solitary fibrous tumor/hemangiopericytoma: A rare case report and review of the literature

Hammad Ghanchi[1], Tye Patchana[1], Eisha Christian[2], Chao Li[3], Mark Calayag[4]

[1]Department of Neurosurgery, Riverside University Health System, Moreno Valley, [2]Department of Neurosurgery, Kaiser Permanente, Los Angeles, [3]Department of Neurosurgery, Desert Regional Medical Center, Palm Springs, [4]Department of Neurosurgery, Kaiser Permanente, Fontana, California, United States.

E-mail: Hammad Ghanchi - hammad.ghanchi@gmail.com; *Tye Patchana - tyepatchana@gmail.com; Eisha Christian - eisha.christian@kp.org; Chao Li - clarkchaoli@gmail.com; Mark Calayag - mark.calayag@kp.org

***Corresponding author:**
Tye Patchana,
3rd Year Neurosurgery Resident,
Department of Neurosurgery,
Riverside University Health System, 26520 Cactus Ave,
Moreno Valley 92555,
California, United States.

tyepatchana@gmail.com

Received : 30 April 2020
Accepted : 16 July 2020
Published : 08 August 2020

DOI
10.25259/SNI_234_2020

Quick Response Code:

ABSTRACT

Background: Solitary fibrous tumor (SFT)/hemangiopericytoma (HPC) is a rare tumor which originates from the walls of capillaries and has historically been thought to be able to occur anywhere in the body that blood vessels are found. It is rarely found in the sellar region.

Case Description: InS this report, we present the first case of this tumor occurring in the sellar region of a pediatric patient. This 12-year-old male presented with progressive vision loss which prompted surgical resection after a sellar lesion was discovered on imaging. The initial transsphenoidal approach resulted in subtotal resection and the patient experienced reoccurrence within 3 months. He underwent an orbitozygomatic craniotomy to achieve gross total tumor resection.

Conclusion: We conducted a literature review of intracranial SFT/HPC in the pediatric population and found it to be an extremely rare occurrence, with <30 cases reported. The incidence of SFT/HPC occurring in the sellar region for any age group was also found to be a rare entity. Treatment recommendations for this tumor are also scarce, based on retrospective chart reviews from the adult population. The role for adjuvant radiation has mixed results.

Keywords: Orbitozygomatic craniotomy, Pediatric hemangiopericytoma, Pediatric sellar tumor, Pediatric tumors

INTRODUCTION

Solitary fibrous tumor (SFT)/hemangiopericytoma (HPC) is a rare tumor derived from the wall of capillaries and has historically been thought to be able to occur anywhere in the body, though it is rarely seen in the sellar region. The term "hemangiopericytoma" was first introduced in 1942 by Stout and Murray as a vascular tumor originating from Zimmermann's pericytes, modified smooth muscle cells within the capillary walls that form endothelial tubes and sprouts.[22] Since that time, HPCs have undergone various iterations of nomenclature; ultimately extracranial HPCs have been moved within the spectrum of "solitary fibrous tumors" with neuropathologists retaining the term "hemangiopericytoma."[13] Furthermore, a theory of origination from

translocation occurring at chromosome 12, resulting in chromosomal fusion has replaced the theory of origination from Zimmerman's pericytes.[26]

Head-and-neck cases make up approximately 25–33% of total HPC with <1% of these lesions occurring intracranial. HPC is rare in adults, and even more so in children, as less than 10% of HPC occur in children:[17] Intracranial HPC for pediatric patients has rarely been reported in the literature. In this report, we describe a rare entity in the pediatric population; a sellar region lesion mimicking a nonfunctional pituitary adenoma on imaging, with a determined final pathology of SFT/HPC. To the best of our knowledge, this is the first reported sellar SFT/HPC in the pediatric population. Furthermore, we performed a literature search of pediatric intracranial HPC and general reports of SFT/HPC located in the sellar region.

CASE PRESENTATION

Our patient is a 12-year-old male who was initially being evaluated by optometry for headaches and progressive visual impairment over a 2-month period. On assessment of the patient's vision, a diagnosis of bitemporal hemianopsia was made, prompting magnetic resonance imaging (MRI) of brain and orbits. As demonstrated in [Figure 1], it revealed a sellar lesion with suprasellar extension exhibiting homogenous enhancement. At this time, a broad differential diagnosis was considered, including those typified by sellar lesions (including sarcoid, aneurysm, teratoma, craniopharyngioma, hypothalamic glioma, meningioma, metastasis, or optic nerve glioma).

On neurological examination, the patient was neurologically intact with the exception of bitemporal visual field loss. Visual acuity was noted to be 20/20 on the right and 20/100 on the left. Serum laboratory assessment for hormonal irregularities was unremarkable. The patient was also noted to have polydipsia without overt signs of diabetes insipidus (i.e., stable serum sodium levels and remaining euvolemic). He subsequently underwent transnasal transsphenoidal craniotomy for resection and decompression of the optic chiasm. Postoperatively, the patient recovered well and was subsequently discharged home without any evident complications, with plan for possible further surgery after pathology finalized.

Intraoperatively, the tumor was noted to be firm and fibrous. Complete resection was not feasible from the trans-nasal approach as the tumor was noted to be extremely adherent to surrounding structures. Frozen section pathology from the initial surgery was inconclusive. However, final pathology from this surgery was reported to be SFT/HPC, World Health Organization (WHO) Grade II.

On 3-month follow-up, imaging demonstrated progressive and enlarging lesion, as demonstrated in [Figure 2]. Furthermore, the patient also complained of worsening vision in the left eye, with visual acuity now at 20/200. The remainder of cranial nerves were intact. At this time, the patient returned to the operating room for a left-sided orbitozygomatic craniotomy. Tumor was visualized in the suprasellar region and resected in a piecemeal fashion with the aid of an ultrasonic aspirator. The tumor has a similar consistency to the 1st time – firm and fibrous with regions of high vascularity. Microsurgical techniques were used to dissect the tumor capsule from all neural elements. The surgery proceeded without complications.

Postoperative imaging, as visualized in [Figure 3], demonstrated gross total resection (GTR) of the mass. This time, the patient's postoperative course was complicated with the development of diabetes insipidus requiring desmopressin. The patient also had transient loss of vision in the left eye which returned to preoperative baseline before discharge from the hospital. [Figure 4] demonstrates the pathology obtained from the patient; [Figure 4a-c] displays the hematoxylin and eosin staining which demonstrates a mesenchymal neoplasm with moderate cellularity and nuclear pleomorphism. Tumor cells were noted to form into sheets and clusters with fibrous stroma, as well as abundant vasculature. Furthermore, staghorn appearance and additional strong positivity for CD34 was noted along endothelial cells with no definitive staining of tumor cells [Figure 4d] as well as strong nuclear staining for

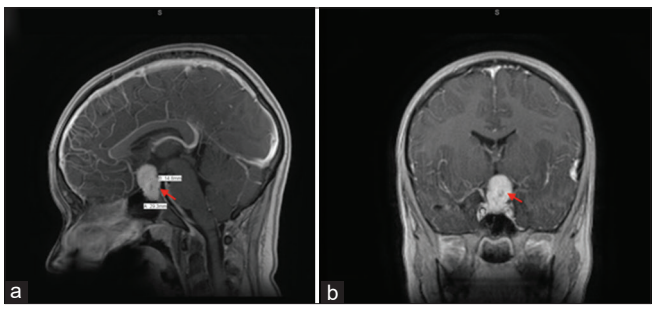

Figure 1: Magnetic resonance imaging brain (a) sagittal, (b) coronal demonstrating a sellar lesion with suprasellar extension exhibiting homogenous enhancement. Red arrows demonstrate a sellar lesion with suprasellar extension with homogenous enhancement.

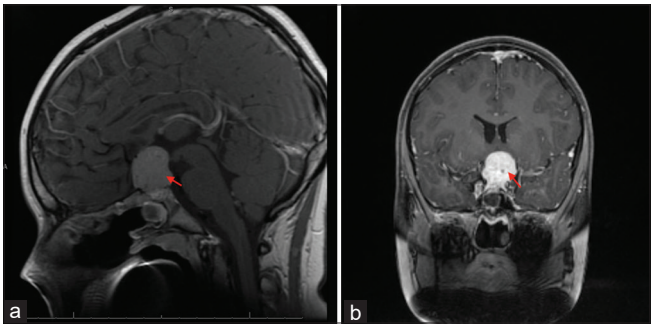

Figure 2: On 3-month follow-up, magnetic resonance imaging (a) sagittal and (b) coronal sequences demonstrated progressive and enlarging lesion. Red arrows demonstrate the progressively enlarging lesion.

signal transducer and activator of transcription 6 (STAT6) [Figure 4e], confirming the diagnosis of SFT/HPC. The final diagnosis of HPC was agreed on by outside consultation to the Children's Hospital of Los Angeles, who reviewed the samples.

DISCUSSION

WHO classification

Extracranial SFT/HPCs have been reclassified as a spectrum of SFTs, whereas the HPC continues to be used among neuropathologists. Both entities share the 12q3 inversion and fusion of the NGFI-A-binding protein 2 (NAB2) and STAT6 genes with the STAT6 nuclear expression visible on immunohistochemistry.[20] This inversion and fusion juxtaposes NAB2 and STAT6 in an initiating event found to be a disease-specific gene fusion.[23] This finding may have contributed to the change to the 2016 WHO classification of central nervous system tumors, assigning three grades to the "SFT/HPC" class, breaking the tradition of assigning names to the classes (e.g., glioblastoma as Grade IV);[8] the specific descriptions of each grade is displayed in [Table 1]. In the prior 2007 WHO classification, "Hemangiopericytoma" was distinct from SFT and anaplastic HPC.[12] For our case, a WHO Grade II SFT/HPC is the same as the prior "hemangiopericytoma" classification, thus the literature search was limited to this term.

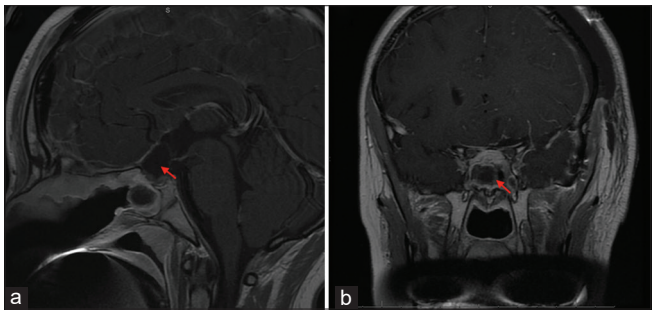

Figure 3: Magnetic resonance imaging brain imaging (a) sagittal and (b) postoperative imaging, as visualized in demonstrating gross total resection of the mass. Red arrows demonstrate removal of the previously enlarging lesion.

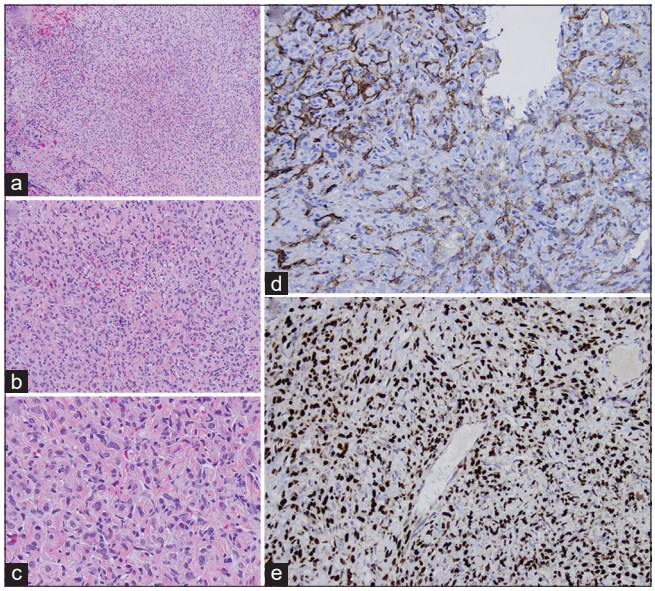

Figure 4: The histopathology obtained from the patient; (a-c) the hematoxylin and eosin staining demonstrating staghorn appearance with additional positivity for CD34 (d) and signal transducer and activator of transcription 6 (e), confirming solitary fibrous tumor/hemangiopericytoma.

Pediatric SFT/HPC

HPCs have the potential to occur anywhere in the body as they originate from blood vessels but the most common location is in the lower extremity in the pediatric population.[9] Intracranial SFT/HPC is a rare entity in the pediatric population. Limited cases were identified in the literature as can be found in [Table 2]. Moreover, from

Table 1: Three grades to the "solitary fibrous tumor/hemangiopericytoma" class, breaking the tradition of assigning names to the classes (e.g., glioblastoma as Grade IV). The specific descriptions of each grade are depicted above.

WHO Grade (2016)	Description	Prior WHO (2007) Name
I	Highly collagenous, relatively low cellularity, spindle cell lesion	Solitary fibrous tumor
II	More cellular, less collagenous tumor with plump cells and "staghorn" vasculature	Hemangiopericytoma
III	Five or more mitoses per 10 high-power fields	Anaplastic hemangiopericytoma

Table 2: Limited cases of intracranial solitary fibrous tumor/hemangiopericytoma identified in the literature.

Pediatric cases of solitary fibrous tumor/hemangiopericytoma				
References	**Year**	**Patients**	**STAT6**	**CD34**
Herzog *et al.*	1995	2	N	N
Cole and Naul	2000	1	N	N
Bunai *et al.*	2008	1	N	Y (pos)
Fernandez-Pineda *et al.*	2011	5	N	N
Kerl *et al.*	2011	1	N	Y (pos)
Chen *et al.*	2012	9	N	N
Trabelsi *et al.*	2015	1	Y	Y (neg)
Pang *et al.*	2015	2	N	N
Silva da Costa *et al.*	2017	1	N	N
Yilmaz Semerci *et al.*	2017	1	N	Y (neg)
Roth and Constatini	2017	1	N	N
Total		25		

the available literature, there are no reports that were of HPC located in the sellar region. The largest case series for pediatric HPC reported nine cases with the cranial convexity and parasagittal region being the most common locations.[10]

Two forms of HPC have been identified during childhood; infantile type which occurs during the first 12 months of life and the adult type which occurs after the age of 12 months old.[4,17] Pediatric HPC consists of both entities and distinction between the two which is essential for treatment strategies. Infantile HPCs are considered congenital and have a favorable prognosis. Similar to adult HPC, infantile HPC has been classified at times in the literature as "infantile myofibromatosis" with studies suggesting both as different stages of the same entity.[16,25] Adult HPCs behave similar in the pediatric population as the adult population. This distinction is made from the clinical standpoint rather than histological.[9]

A recent study[24] from three pediatric hospitals reevaluating prior pathology from the past 30 years found that 18 tumors total were classified as SFTs. However, when immunohistochemistry was performed on the tissue, only three tumors from two patients were found to be STAT6 positive; the remainder tumors required reclassification. This demonstrates that the diagnosis of SFT, and HPC, may be rarer than previously thought. Indeed, in the past, both SFTs and HPCs were referred to histologically as "angioblastic meningiomas" or "hemangiopericytic variant of meningioma," before the advent of STAT6 immunopositivity studies.[1] Our patient's tissue was confirmed to be STAT6 positive [Figure 4e]. Furthermore, the implications of this study are vast as much of the prior epidemiological studies may have included false-positive diagnosis.

STAT6 for diagnosis of SFT/HPC

STAT6 immunohistochemistry has been found to be an exclusively nuclear immunostaining signal with a strong ability to verify SFTs and HPCs, with recent studies showing all non-SFT/HPCs unanimously STAT6 negative and 98.7% agreement among observers.[3] In the past, CD34 has been the most characteristic immunohistochemical stain for SFTs, however, it is also nonspecific.[3,20] Very little was known about the molecular genetics of this tumor. Around 2013, immunohistochemical staining for STAT6 was presented in the literature as a more specific marker for SFTs and HPC.[3,11,19] A NAB2-STAT6 gene fusion, results in an inversion at the 12q13 locus, produces a chimeric protein in which a repressor domain of NAB2 (EGR1-binding protein 2) (NAB2) is replaced with a carboxyterminal transactivation domain from signal transducer. The activator of transcription 6 and interleukin-4 induced (STAT6) acts as a transcriptional activator through early growth of the tumor and has been recently believed to be highly sensitive and specific for SFTs. Several journal articles have confirmed the validation of the diagnosis of intracranial and extracranial SFT.[6]

Sellar location SFT/HPC

To date, a total of 15 cases of sellar region SFT/HPC were identified in the English literature, as listed in [Table 3]. However, none have been reported in the pediatric population. The youngest patient in literature was an 18-year-old female who also had similar imaging – homogenously enhancing mass concerning for pituitary adenoma.[2] The surgical approach utilized in these cases was transsphenoidal, transcranial, or the former followed by latter after diagnosis was revealed and significant residual tumor or recurrence occurred. Many of these patients presented with endocrinopathies that would result from pituitary compression, as well as visual change from compression of the optic apparatus. The most common pediatric tumor in this location is a craniopharyngioma (6–9%), followed by hypothalamic glioma (4–8%), germ cell tumors (1–2%), and pituitary adenoma (0.5–2.5%).[10,25]

Treatment

Given the rarity of HPC, information about management specific to intracranial HPC for pediatric population is scarce. Moreover, the distinction between infantile and adult HPC is paramount to the clinical course. The infantile variant, occurring <12 months of age, is chemoresponsive. There are reports of extracranial infantile HPC exhibiting spontaneous regression.[4,14] Moreover, this variant has a propensity to mature into benign neoplasms, such as hemangioblastoma, and has been shown to be responsive to neoadjuvant chemotherapy.[7] One report of a neonate with intracranial HPC was treated with neoadjuvant anthracycline-based chemotherapeutic agent which was able to decrease tumor size before surgical resection.[5] Another series of two infantile HPCs were treated with surgical resection without adjuvant therapy with no evidence of recurrence at time of report.[9,18,21] Large series specific for intracranial infantile HPC are lacking given limited cases.

On the contrary, adult HPC requires a more aggressive approach. Surgical resection followed by adjuvant radiation therapy has been recommended in the past for treatment from adult literature.[21] In a 29-patient series report for intracranial adult HPC, the 5-, 10-, and 15-year overall survival rates were 85%, 68%, and 43%, respectively, for patients undergoing this treatment route.[15] Another 43-patient series reported 1-, 5-, and 10-year overall survival at 100%, 94.4%, and 72.2%, respectively.[21] Outcome is directly related to the presence of metastatic disease and adequacy of local control; GTR is advocated as the 5-year local disease control rates were 84% when compared to only 38% for subtotal resection (STR).[18]

Table 3: To date, a total of 15 cases of sellar region solitary fibrous tumor/hemangiopericytoma were identified in the English literature.

Sellar location solitary fibrous tumor/hemangiopericytoma					
References	**Year**	**Age**	**Sex**	**Location**	**Surgical approach**
Koml *et al.*	1968	24	F	Sellar	Transsphenoidal
Mangiardi *et al.*	1983	59	F	Sellar	Transsphenoidal
Yokota *et al.*	1985	35	F	Suprasellar	Transcranial
Kumar *et al.*	1986	22	M	Sellar	Transcranial and transphenoidal
Morrison and Bibby	1997	35	F	Sellar and Suprasellar	Transsphenoidal
Gharbi *et al.*	2001	44	M	Sellar and Parasellar	Not listed
Kanda *et al.*	2001	60	F	Sellar	Transcranial and transphenoidal
Juco *et al.*	2007	18	F	Sellar	Not listed
Han *et al.*	2007	44	M	Sellar	Transcranial
Yin *et al.*	2009	32	M	Sellar	Not listed
Das *et al.*	2010	47	M	Sellar	Transsphenoidal
Sanchez	2011	72	F	Sellar and suprasellar	Not listed
Gibson *et al.*	2017	34	M	Sellar and suprasellar	Not listed

However, a recent meta-analysis of 563 cases found that GTR alone provided superior survival, with or without adjuvant radiation therapy; patients receiving >50 Gy of radiation had worse survival outcomes.[15] This study included pediatric patients, with age ranging from 1 month to 80 years, however, the distinction between pediatric and adult population was not made in their analysis nor was the number of patients under age 18 reported. A single-institution retrospective review of 43 patients reported adjuvant radiation can hinder tumor progression but appears to have no effect on overall survival.[17] Patients with GTR alone were compared to patients with STR + radiation and the latter group had longer progression-free interval. However, when comparing overall survival for the same groups, GTR alone had better results.

Data specific to pediatric population for intracranial disease is lacking; however, given the similar clinical behavior of extracranial disease,[20] we believe that these results can be extrapolated to the pediatric population with intracranial adult HPC. Surgical intervention with GTR should be the goal with conflicting evidence for adjuvant radiation therapy. Given the benefit of improved recurrence-free interval with radiation therapy, there is likely some benefit to adjuvant radiation therapy if total dose is kept below 50 Gy. This dose limit may need to be decreased in younger population given proximity to optic apparatus, further research is needed in this area.

CONCLUSION

The authors present a report of a SFT/HPC occurring in a 12-year-old male patient in the sella. This lesion mimicked a nonfunctioning pituitary adenoma both clinically and on imaging, with a surprising final pathological result; this is the first report of HPC occurring in the sellar region in the pediatric population. Moreover, recent literature with regard to STAT6 positivity suggests that many prior diagnoses of HPC may be false positives, making it an even rarer entity. Data are lacking for evidence-based treatment recommendations for pediatric HPC; from adult data, goals favor GTR for best overall survival for adult-type HPC, which occurs in the pediatric population after the age of 1 year. The role for adjuvant radiation therapy is unclear at this time, with benefits seen with in patients only able to undergo STR with regard to the progression-free interval. Prospective trials are needed to further characterize specific treatment modalities.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Adesina AM. Histopathology of primary tumors of the central nervous system. In: Mahajan A, Paulino A, editors. Radiation Oncology for Pediatric CNS tumors. Cham: Springer International Publishing; 2018. p. 21-53.
2. Deopujari CE, Kumar A, Karmarkar VS, Biyani NK, Mhatre M, Shah NJ. Pediatric suprasellar lesions. J Pediatr Neurosci 2011;6:S46-55.
3. Doyle LA, Vivero M, Fletcher CD, Mertens F, Hornick JL. Nuclear expression of STAT6 distinguishes solitary fibrous tumor from histologic mimics. Mod Pathol 2014;27:390-5.

4. Fernandez-Pineda I, Parida L, Jenkins JJ, Davidoff AM, Rao BN, Rodriguez-Galindo C. Childhood hemangiopericytoma: Review of St Jude Children's research Hospital. J Pediatr Hematol Oncol 2011;33:356-9.
5. Herzog CE, Leeds NE, Bruner JM, Baumgartner JE. Intracranial hemangiopericytomas in children. Pediatr Neurosurg 1995;22:274-9.
6. Juco J, Horvath E, Smyth H, Rotondo F, Kovacs K. Hemangiopericytoma of the sella mimicking pituitary adenoma: Case report and review of the literature. Clin Neuropathol 2007;26:288-93.
7. Kerl K, Sträter R, Hasselblatt M, Brentrup A, Frühwald MC. Role of neoadjuvant chemotherapy in congenital intracranial haemangiopericytoma. Pediatr Blood Cancer 2011;56:161-3.
8. Kim BS, Kim Y, Kong DS, Nam DH, Lee JI, Suh YL, *et al*. Clinical outcomes of intracranial solitary fibrous tumor and hemangiopericytoma: Analysis according to the 2016 WHO classification of central nervous system tumors. J Neurosurg 2018;129:1384-96.
9. Kim YJ, Park JH, Kim YI, Jeun SS. Treatment strategy of intracranial hemangiopericytoma. Brain Tumor Res Treat 2015;3:68-74.
10. Kumar R, Corbally M. Childhood hemangiopericytoma. Med Pediatr Oncol 1998;30:294-6.
11. Li XL, Fu WW, Zhang S, Chen DY, Chen YP, Wu J, *et al*. Solitary fibrous tumor/hemangiopericytoma of central nervous system: A clinicopathologic analysis of 71 Cases. Zhonghua Bing Li Xue Za Zhi 2017;46:465-70.
12. Louis DN, Ohgaki H, Wiestler OD, Cavenee WK, Burger PC, Jouvet A, *et al*. The 2007 WHO classification of tumours of the central nervous system. Acta Neuropathol 2007;114:97-109.
13. Louis DN, Perry A, Reifenberger G, von Deimling A, Figarella-Branger D, Cavenee WK, *et al*. The 2016 World Health Organization classification of tumors of the central nervous system: A summary. Acta Neuropathol 2016;131:803-20.
14. McHugh BJ, Baranoski JF, Malhotra A, Vortmeyer AO, Sze G, Duncan CC. Intracranial infantile hemangiopericytoma. J Neurosurg Pediatr 2014;14:149-54.
15. Melone AG, D'Elia A, Santoro F, Salvati M, Delfini R, Cantore G, *et al*. Intracranial hemangiopericytoma-our experience in 30 years: A series of 43 Cases and review of the literature. World Neurosurg 2014;81:556-62.
16. Mentzel T, Calonje E, Nascimento AG, Fletcher CD. Infantile hemangiopericytoma versus infantile myofibromatosis. Study of a series suggesting a continuous spectrum of infantile myofibroblastic lesions. Am J Surg Pathol 1994;18:922-30.
17. Rodriguez-Galindo C, Ramsey K, Jenkins JJ, Poquette CA, Kaste SC, Merchant TE, *et al*. Hemangiopericytoma in children and infants. Cancer 2000;88:198-204.
18. Rutkowski MJ, Sughrue ME, Kane AJ, Aranda D, Mills SA, Barani IJ, *et al*. Predictors of mortality following treatment of intracranial hemangiopericytoma. J Neurosurg 2010;113:333-9.
19. Savary C, Rousselet MC, Michalak S, Fournier HD, Taris M, Loussouarn D, *et al*. Solitary fibrous tumors and hemangiopericytomas of the meninges: Immunophenotype and histoprognosis in a series of 17 Cases. Ann Pathol 2016;36:258-67.
20. Schweizer L, Koelsche C, Sahm F, Piro RM, Capper D, Reuss DE, *et al*. Meningeal hemangiopericytoma and solitary fibrous tumors carry the NAB2-STAT6 fusion and can be diagnosed by nuclear expression of STAT6 protein. Acta Neuropathol 2013;125:651-8.
21. Soyuer S, Chang EL, Selek U, McCutcheon IE, Maor MH. Intracranial meningeal hemangiopericytoma: The role of radiotherapy: Report of 29 Cases and review of the literature. Cancer 2004;100:1491-7.
22. Stout AP, Murray MR. Hemangiopericytoma: A vascular tumor featuring zimmermann's pericytes. Ann Surg 1942;116:26-33.
23. Tai HC, Chuang IC, Chen TC, Li CF, Huang SC, Kao YC, *et al*. NAB2-STAT6 fusion types account for clinicopathological variations in solitary fibrous tumors. Mod Pathol 2015;28:1324-35.
24. Tan SY, Szymanski LJ, Galliani C, Parham D, Zambrano E. Solitary fibrous tumors in pediatric patients: A rare and potentially overdiagnosed neoplasm, confirmed by STAT6 immunohistochemistry. Pediatr Dev Pathol 2018;21:389-400.
25. Toren A, Perlman M, Polak-Charcon S, Avigad I, Katz M, Kuint Y, *et al*. Congenital hemangiopericytoma/infantile myofibromatosis: Radical surgery versus a conservative "wait and see" approach. Pediatr Hematol Oncol 1997;14:387-93.
26. Tsirevelou P, Chlopsidis P, Zourou I, Valagiannis D, Skoulakis C. Hemangiopericytoma of the neck. Head Face Med 2010;6:23.

How to cite this article: Ghanchi H, Patchana T, Christian E, Li C, Calayag M. Pediatric sellar solitary fibrous tumor/hemangiopericytoma: A rare case report and review of the literature. Surg Neurol Int 2020;11:238.