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A rare presentation of extra-axial supratentorial ependymoma with subdural hematoma mimicking a parasagittal meningioma

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ABSTRACT

Introduction: Primary extra-axial ependymomas, though rare, the majority of such lesions are identified as WHO grade III ependymomas. These ependymomas may mimick a meningioma on radiological investigations which can be confirmed by histopathology.

Research Question: We show in this case report a rare presentation of extra-axial supratentorial ependymoma with concomitant subdural hematoma mimicking a parasagittal meningioma.

Material and Methods: A 59 years lady with no known comorbidities presented with weakness of right half of body and decreased speech for 2 days. She was aphasic. Contrast MRI brain revealed an extra-axial dural-based homogenously enhancing lesion in the left anterior 1/3rd parasagittal area with left frontotemporoparietal chronic subdural hematoma. With a provisional diagnosis of meningioma, the patient was subjected to bifrontal open-book craniotomy with gross total excision of lesion with periosteal graft duraplasty and acrylic cranioplasty. Left sided frontotemporal subacute SDH with thin greenish yellow membrane was present. In post operative period, patient quickly became E4V5M6 with power of 4/5 in the right half of body which was same as compared to the preoperative period.

Results: The biopsy of the mass, however, revealed features suggestive of extra-axial supratentorial ependymoma (WHO Grade III). Immunohistochemistry supported the diagnosis of supratentorial ependymoma, NOS. The patient was then referred for further chemoradiation.

Discussion and Conclusion: We report the first case of extra-axial supratentorial ependymoma mimicking a parasagittal meningioma occurring with adjacent subdural hematoma. Clinical and imaging background along with a complete pathological examination with immunohistochemical study is essential to confirm the diagnosis of rare brain tumours.

1. Introduction

The incidence of ependymomas range from 2% to 9% of all intracranial neoplasms. They are more common in children and the majority occur in the infratentorial region (Youkilis et al., 2001). Supratentorial ependymomas are more common in adults and constitute less than one third of total ependymoma cases. The supratentorial ependymoma can be either intra-axial/intraparenchymal or extra-axial. Although most supratentorial ependymomas are believed to arise from the ventricular system, a small number of these lesions are primarily intra-axial without clear-cut ventricular involvement. The origin of such intra-axial ependymomas is controversial with certain authors postulating that these intraparenchymal lesions develop from extensions of the ventricular surface that have subsequently regressed (Hayashi et al., 1994). It is believed that the intra-axial, extra-ventricular ependymomas originate from ependymal embryologic remnants (Cosgrove et al., 1985).

Primary extra-axial ependymomas, however, very rarely occur. In adults the majority of such lesions are identified as WHO grade III ependymomas (Niazi et al., 2009). Here, we report a case of extra-axial supratentorial ependymoma mimicking a parasagittal meningioma with subdural hematoma.

2. Case report

A 59 years female patient with no known comorbidities presented to the emergency room with complaints of weakness of right half of body

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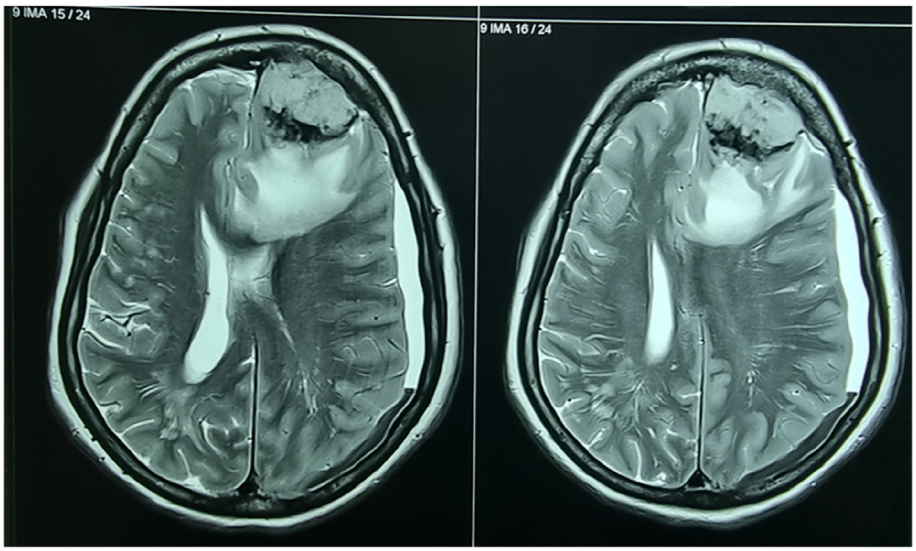

Fig. 1. jpg: T2 axial MRI brain showing left frontal hyperintense lesion with surrounding perilesional edema with left frontotemporal subdural hyperintense lesion suggestive of chronic hematoma.

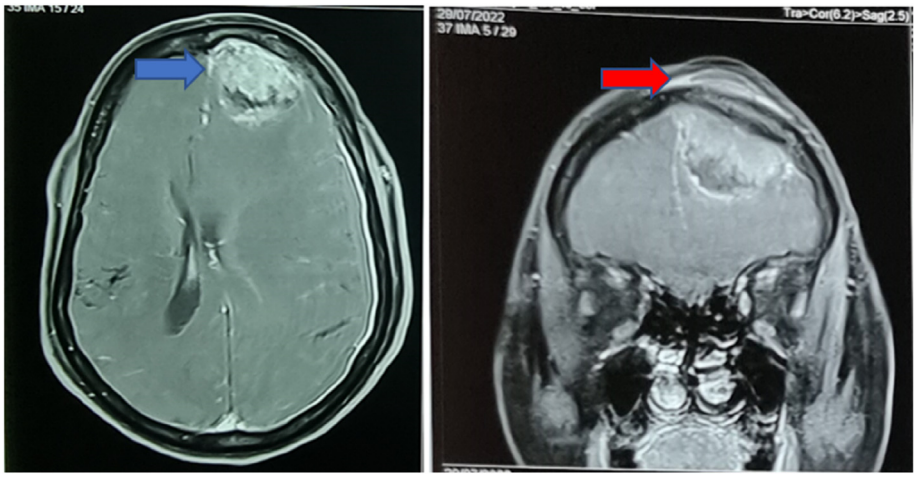

Fig. 2. jpg: Contrast axial (left) and coronal (right) MRI brain showing homogeneously contrast enhancing left frontal lesion (blue arrow) with involvement of overlying bone (red arrow). (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

and decreased speech for two days. The patient also had swelling over the left frontal scalp since 10 days which was gradually increasing in size. There was also associated vomiting one episode on the day of presentation. There was no history of loss of consciousness, seizure or trauma. The patient was also not on any blood thinners.

On examination, her GCS was E4V1M6 (aphasic). Her pupils were 3 mm bilaterally reactive to light and intact ocular movements. Her cranial nerve examination was within normal limit. On motor examination, she had power of 4/5 in right upper and lower limb with intact power in left upper and lower limb. Her reflexes were exaggerated in the right half of body. Sensory examination and cerebellar function were within normal limit.

Plain computed tomography (CT) scan revealed a left frontal space occupying lesion with diffuse perilesional edema with left frontotemporoparietal chronic subdural hematoma with mass effect with midline shift. The lesion was hyperdense with attachment to anterior third superior sagittal sinus and peripheral-based, corresponding to a meningioma. The patient was admitted and planned for contrast magnetic resonance imaging (MRI) brain. The patient was started on intravenous steroids. Contrast MRI brain revealed an extra axial dural based homogenously enhancing lesion of size 5.6 cm × 3.5 cm X 3.2 cm in the left anterior 1/3rd parasagittal area with hyperostosis of overlying bone and spread of tumour to the subgaleal layer. There was marked perilesional edema with significant mass effect as evidenced by effacement of ipsilateral lateral ventricle and subfalcine herniation. Magnetic resonance venography (MRV) revealed involvement of anterior 1/3rd superior sagittal sinus as evidenced by absence of blood flow. There was also presence of T2 high signal intensity lesion over the left frontotemporoparietal region with thickness of approximately 7 mm causing midline shift towards right suggesting chronic subdural hematoma. Thus, a diagnosis of left anterior one third parasagittal meningioma with left frontotemporal subdural hematoma was made.

After 48 hours of intravenous steroid, the patient was taken up for surgery. The patient underwent bicoronal skin flap and bifrontal open book craniotomy with gross total excision of lesion with synthetic dural patch duraplasty and acrylic cranioplasty under neuronavigation in supine position with head fixed in 3 pin fixation system. Intraoperatively, 5 × 4x3 cm extracranial, greyish white, moderately vascular, CUSA suckable lesion was present in the left frontal lobe involving left frontal bone and anterior third of superior sagittal sinus. The tumour was seen eroding through the bone and involving subgaleal tissue. Left sided frontotemporal subacute SDH with thin greenish yellow membrane was present

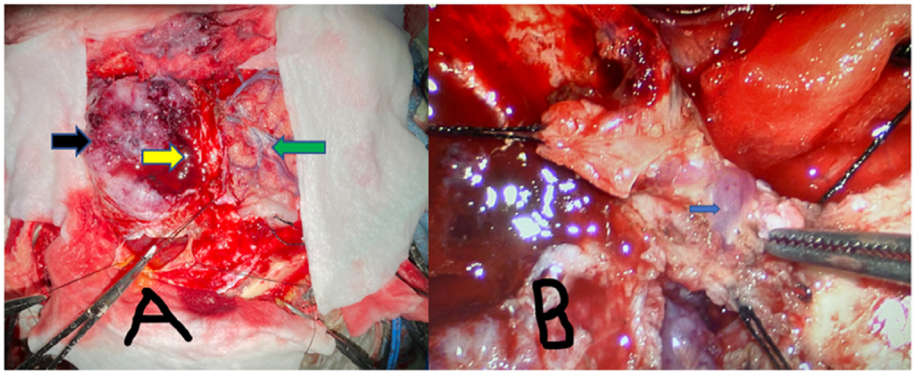

Fig. 3. jpg: (A) Intraoperative picture showing extracranial, greyish white, moderately vascular, CUSA suckable lesion (black arrow) in left frontal lobe (engorged, marked with black arrow) involving left frontal bone and anterior third of superior sagittal sinus (yellow arrow). The tumour was seen eroding through the bone and involving subgaleal tissue. Contralateral right frontal lobe was normal and not involved (green arrow). (B) Intra-operative picture showing infiltration of lumen of anterior one third of superior sagittal sinus with tumour (blue arrow). (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

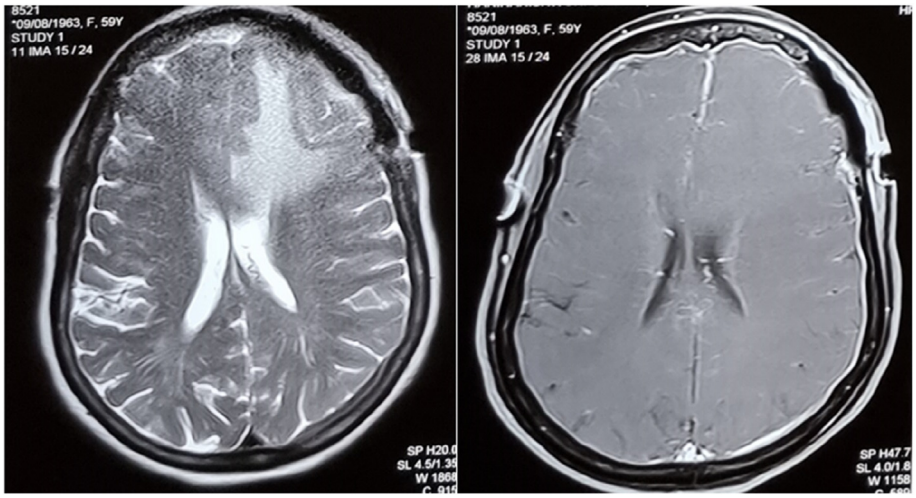

Fig. 4. jpg: Axial T2 (left) and post contrast (right) MRI brain showing complete excision of tumour.

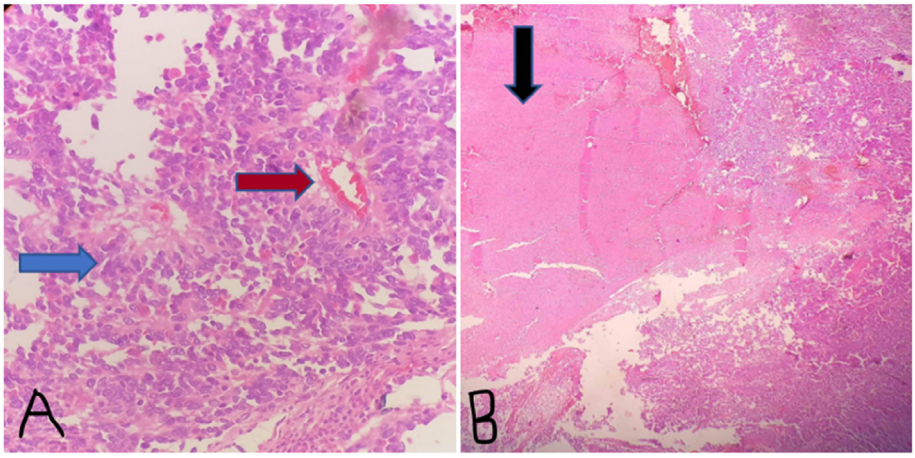

Fig. 5. jpg: (A): Histopathological HPF (40X) picture of the tumour specimen showing small blue round cell (dark blue nucleus with scant cytoplasm) (blue arrow) with perivascular pseudorossettes (red arrow). (B): Histopathological LPF (4X) picture of tumour specimen showing perivascular pseudo-rossettes with necrosis (geographical necrosis) (black arrow). (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

which was excised. The patient was extubated in the postoperative period. Her GCS was E4V5M6 with power of 4/5 in the right half of body which was same as compared to the preoperative period. She was shifted to the ward from ICU on the 3rd postoperative day. Postoperative MRI was done which revealed no residual tumour.

Biopsy of the mass revealed cellular tumour with cells arranged in syncytial pattern and diffuse pattern with tumour cells showing mild to moderate nuclear pleomorphism with round to oval cells and spindle shaped cells with speckled chromatin. Perivascular pseudo-rosettes, vascular proliferation and concentric calcifications with areas of necrosis were also seen suggestive of extra axial supratentorial ependymoma (WHO Grade III). Immunohistochemistry was sent which showed GFAP negative, EMA positivity, IDH-1 wild type, Olig 2 focally positive, p53 wild type and 70% Ki 67 index and supported the diagnosis of supratentorial ependymoma, NOS. The patient was then referred for further chemoradiation (see Figs. 1-5).

Table 1
Summary of the case reports on supratentorial extra axial ependymoma misdiagnosed as meningioma published in the literature till date.

S. No.	Author/Year	Patient age/Sex	Location	Preoperative Diagnosis	Surgery	Dural attachment	Histology	Adjuvant treatment	Follow up
1	Hanchey et al. (Hanchey et al., 1976), 1976	29 years/ male	Anterior interhemispheric falx	Meningioma	GTR	Yes	Ependymoma, WHO Grade II	Radiotherapy, Chemotherapy	Not reported
2	Hayashi et al. (Hayashi et al., 1994), 1994	13 years/ male	Right occipitoparietal convexity	Meningioma	GTR	Yes	Clear cell ependymoma, WHO Grade II	None	Not reported
3	Youkilis et al. (Youkilis et al., 2001), 2001	20 years/ male	Left middle one third parafalcine	Meningioma	GTR	Yes	Ependymoma, WHO Grade III	None	No recurrence at 12 months
4	Goto et al. (Goto et al., 2003), 2003	29 years/ male	Left frontal convexity	Meningioma	GTR	No	Ependymoma, WHO Grade II	Not reported	No recurrence at 9 months
5	Miyazawa et al. (Miyazawa et al., 2007), 2007	33 years/ male	Left parietal convexity	Meningioma	GTR	Yes	Anaplastic ependymoma, WHO Grade III	Radiotherapy and chemotherapy	Recurrence at 10 months
6	Salunke et al. (Salunke et al., 2011), 2011	43 years/ female	Left posterior third parasagittal	Meningioma	GTR	Yes	Ependymoma, Who Grade II	Radiotherapy	No recurrence at 6 months
7	Singh et al. (Singh et al., 2012), 2012	35 years/ male	Left middle third parafalcine	Meningioma	GTR	No	Anaplastic ependymoma, WHO Grade III, MIB-1: 40%	Radiotherapy	No recurrence at 12 months
8	Ma et al. (Ma et al., 2012), 2012	10 years/ male	Left frontotemporal convexity	Meningioma	GTR	No	Ependymoma, WHO Grade II	None	No recurrence at 2 years
9	Seo et al. (Seo et al., 2013), 2013	25 years/ male	Petroclival region	Meningioma	GTR	No	Ependymoma, WHO Grade II	Radiotherapy and Chemotherapy	Not reported
10	Elsharkawy et al. (Elsharkawy et al., 2013), 2013	25 years/ male	Right frontal region	Meningioma	GTR	No	Anaplastic ependymoma, WHO Grade III	Radiotherapy	No recurrence at 6 months
11	Yang et al. (Yang et al., 2014), 2014	35 years/ female	Left petroclival region	Meningioma	GTR	No	Ependymoma, Grade II	None	Recurrence at 4 months
12	Puduru et al. (Puduru et al., 2014), 2014	18 years/ female	Right middle third parasagittal	Meningioma	GTR	Yes	Ependymoma, WHO Grade II, MIB-1: 2–3%	None	No recurrence at 2 years
13	Satyarthee et al. (Satyarthee and Moscote-Salazar, 2016), 2016	9 years/ female	Right middle third parafalcine	Meningioma	GTR	Yes	Anaplastic ependymoma, WHO Grade III	Radiotherapy	No recurrence at 16 months
14	Yang et al. (Yang et al., 2016), 2016	47 years/ male	Left middle third parafalcine	Meningioma	GTR	Yes	Anaplastic ependymoma, WHO Grade III	Radiotherapy	No recurrence at 53 months
15	Gupta et al. (Gupta et al., 2016), 2016	9 years/ male	Right frontoparietal convexity	Meningioma	GTR	Yes	Anaplastic ependymoma, WHO Grade III	None	No recurrence at 15 months
16	Nambirajan et al. (Nambirajan et al., 2016), 2016	9 years/ female	Right middle third parafalcine	Meningioma, Hemangiopericytoma	GTR	Yes	Anaplastic ependymoma, WHO Grade III	Radiotherapy	No recurrence at 6 months
17	Berhili et al. (Berhili et al., 2017), 2017	46 years/ male	Right parietooccipital convexity	Meningioma	Subtotal resection	Yes	Anaplastic ependymoma, WHO Grade III	Radiotherapy	Stable residual tumour at 1 year
18	Ahn et al. (Ahn et al., 2017), 2017	33 years/ female	Right parietal convexity	Meningioma	GTR	No	Clear cell ependymoma, WHO Grade III	None	Not reported
19	Karthigeyan et al. (Karthigeyan et al., 2017), 2017	33 years/ female	Right frontoparietal convexity	Atypical meningioma, Gliosarcoma	GTR	Yes	Anaplastic ependymoma, WHO Grade III	Radiotherapy	No recurrence at 1 year
20	Nagayasu et al. (Nagayasu et al., 2022), 2022	26 years/ male	Left temporoparietal convexity	Meningioma	GTR	Yes	Anaplastic ependymoma, WHO Grade III	Radiotherapy	Recurrence at 1 year postsurgery

3. Discussion

Intracerebral ependymomas constitute 5% of all intracranial tumours in adults with one third of these ependymomas being supratentorial. Supratentorial ependymomas occur more frequently in the adult population (Schwartz et al., 1999). About 50% of supratentorial ependymomas originate in the ventricular system and the remaining ependymomas occur in the parenchyma without obvious association with the ventricular system (Youkilis et al., 2001). The patient in our case presented with an extra-axial supratentorial ependymoma which mimicked a parasagittal meningioma on pre-operative imaging and had concomitant subdural hematoma which has not been published in the literature before.

Extra-axial location of supratentorial ependymomas is extremely rare. In our review of the literature, we found few reported cases of extra-axial ependymomas. No definitive mechanism for the development of these

extra-axial ependymomas has been postulated. One hypothesis involves the extension of subcortical, subependymal rests extra-axially with the subsequent growth of tumour (Hayashi et al., 1994). Necrosis and calcification of the originating subependymal rests would then follow, leaving a predominately extra-axial ependymoma. Donich et al. reported the case of an extra-axial cerebellopontine angle ependymoma extending to the cavernous sinus without obvious connection to the ventricular system (Donich et al., 1999). The authors in their case report stated that a microscopic cellular tract existed between the ventricular system and the extra-axial ependymoma. Another possible mechanism was the heterotopic placement of ependymal cell rests during fetal development with subsequent growth of tumour (Lyons and Kelly, 1991). Radiographically, extra-axial ependymomas can be difficult to differentiate from other dural-based lesions as was in our case. The lesion in our case mimicked a parasagittal meningioma with involvement of superior sagittal sinus and concomitant subdural hematoma. Even classic lesions in the posterior fossa can be variable in appearance. As with meningiomas, ependymomas can present with isointense signal on T1-and T2-weighted MRI images. However, contrast enhancement of ependymomas, unlike meningiomas, is usually inhomogeneous. The lesions are generally well circumscribed with cystic regions and various degrees of calcification. Consequently, the location of the lesion is of significant importance in distinguishing the differential diagnosis (Mangalore et al., 2015).

Extra-axial ependymomas often exhibit unusual structural features that they may be neglected during pathology study. Histologically, anaplastic ependymomas WHO grade III are defined by the presence of 2 or more of the following characteristics: 4 mitoses per 10 high-power fields, hypercellularity, endothelial proliferation, and necrosis (Van Gompel et al., 2011). The importance of the immunohistochemical evaluation is utmost. GFAP is an important marker that eliminates major differential diagnosis when positive. It highlights glial processes in perivascular rosettes, unlike occasional schwannomas that express GFAP but not perivascular rosettes. Oligodendroglioma resemble ependymoma, but they lack EMA and perivascular rosettes whereas central neurocytomas express neuronal markers and lack GFAP (Berhili et al., 2017). The immunohistochemical profile used in our case confirmed that the lesion was an ependymal tumour.

Till date, 20 case reports of extra-axial supratentorial ependymoma have been published in the literature which were initially misdiagnosed as meningioma (Youkilis et al., 2001; Hayashi et al., 1994; Berhili et al., 2017; Hanchey et al., 1976; Goto et al., 2003; Miyazawa et al., 2007; Salunke et al., 2011; Singh et al., 2012; Ma et al., 2012; Seo et al., 2013; Elsharkawy et al., 2013; Yang et al., 2014, 2016; Puduru et al., 2014; Satyarthee and Moscote-Salazar, 2016; Gupta et al., 2016; Nambirajan et al., 2016; Ahn et al., 2017; Karthigeyan et al., 2017; Nagayasu et al., 2022). The first such case was reported by Hanchey et al., in 1976. They had reported a case of 29 years male with anterior interhemispheric meningioma as diagnosed with the help of conventional carotid angiography. Intraoperative findings also revealed an encapsulated, sift, reddish brown tumour extending beneath and firmly adherent to the sides of falx, thus, supporting the diagnosis of meningioma. However, the histology revealed moderately cellular ependymoma (Hanchey et al., 1976). Extra-axial ependymomas are rare occurrence as the ependymoma is a cortical tumour and is expeted to be intra-axial. The last such case was reported by Nagayasu et al., in 2022. They had reported a 26 years male with left temporoparietal mass which was diagnosed as meningioma based on the preoperative imaging and as rhabdoid meningioma based on frozen section biopsy. However, the histology revealed RELA fusion positive ependymoma, WHO Grade III (Nagayasu et al., 2022). This signifies the importance of all diagnostic modalities, particularly the histology (see Table 1).

Anaplastic ependymoma with intratumoral hemorrhage is a usual occurrence. The hemorrhage usually occurs from the abnormal and extensive neovascularization seen in high grade tumours (A KN YS OM KJ HY N, 2003). According to Ernestus et al., the factor that most commonly predisposes tumours to bleeding is extensive and abnormal vascularity, and endothelial proliferation or dilated, thin-walled vessels, which were common in ependymal tumours with spontaneous hemorrhages (Ernestus et al., 1992). Our case was different in that the hemorrhage was extra-axial, occurring in the subdural space. In reviewing the literature, we could not find any case report with extra-axial hemorrhage. In our case the subdural hematoma was chronic with presence of membranes. Maximal surgical resection is the initial treatment of ependymomas. Surgery alone may be sufficient in selected cases; however, the standard of care for most patients is postoperative radiation to improve the five-year survival rate (Salazar et al., 1983; Donahue and Steinfeld, 1998; Garrett and Simpson, 1983). Palma et al. concluded that radical surgery alone was satisfactory for selected supratentorial ependymomas (Palma et al., 1993). These ependymomas were completely resected and were not of high grade. In our case, a complete resection was accomplished and, the patient was referred for postoperative radiation in view of high grade of tumour. The tumour response is related to radiation dose, and tumour control increases with doses >50 Gy. Thus, doses of 54–59.4 Gy at 1.8–2 Gy per fraction are typically prescribed (Halperin et al., 2013).

Various prognostic factors have been presented in the literature such as age of patient, tumour location, histology, and the extent of resection. Many studies have shown increased survival with gross total resection (Lyons and Kelly, 1991). Tumour location (supratentorial vs infratentorial) significantly impacts survival, although there have been case reports in literature showing decreased survival with a supratentorial location (Armington et al., 1985; Reni et al., 2004). Schwartz et al. concluded that the most significant negative predictors for survival were association with the third ventricle and metastatic disease (Schwartz et al., 1999).

4. Conclusion

We report the first case of extra-axial supratentorial ependymoma mimicking a parasagittal meningioma occurring with adjacent subdural hematoma. Rare brain tumours are difficult to diagnose based on clinical and imaging background. A complete pathological examination with immunohistochemical study is essential to confirm the diagnosis.

Declaration of competing interest

We certify that this manuscript is a unique submission and is not being considered for publication, in part or in full, with any other source in any medium. There is no conflict of interest.

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