

Case Report

# Intraparenchymal subependymoma: Case report and literature review

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## ABSTRACT

**Background:** Intracranial subependymomas are rare slow-growing benign tumors typically located in the ventricular system, accounting for 0.07–0.7% of all intracranial neoplasms. Intraparenchymal subependymoma is extremely rare lesions, imposing a challenging diagnosis and management.

**Case Description:** We describe a case of a supratentorial intraparenchymal mass on left occipital lobe in a 26-year-old woman with progressive headache and visual impairment. Differential diagnosis mainly included gliomas, neuronal-glia tumors, ependymoma, and subependymoma. Complete surgical resection was performed and histopathology analysis confirmed diagnosis of subependymoma. Despite its benign behavior the Ki67/MIB-1 labeling index assessed by immunohistochemistry was 5%. After 1 year of follow-up she was free of tumor recurrence.

**Conclusion:** Intraparenchymal subependymoma is extremely rare tumors and literature review showed only 11 cases reported. In general, they are misdiagnosed as other tumors, so careful attention on clinical and radiological features must be taken when looking at a tumor close to the ventricular system, even though it does not have any obvious direct connection to it. Despite its benign nature, total removal must be attempted given that there are reports of recurrence, especially in partially removed tumors with high proliferation index. The role of adjuvant therapy is still limited and new treatment options are being developed as our knowledge on biological and molecular characteristics advances.

**Keywords:** Atypical, Intraparenchymal, Recurrence, Subependymoma

## INTRODUCTION

Subependymomas were first described by Scheinker in 1945,<sup>[31]</sup> and more than 200 cases have been described since then, typically on case reports or small case series.<sup>[1-4,7,8,14,23,30,32,39,43]</sup> A recent SEER-based analysis revealed a total of 466 intracranial subependymomas from 2004 to 2013, and probably is the larger analysis made on this kind of tumors.<sup>[26]</sup>

This subtype of ependymal tumors comprehend a rare slow-growing glial neoplasm, histologically corresponding as a World Health Organization (WHO) Grade I.<sup>[22]</sup> The true precursor cell is still debated; suggestions include astrocytes of the subependymal plate, subependymal glia,

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ependymal cells, and a mixture of astrocytes and ependymal cells.<sup>[19,22,24,29]</sup>

Due to its silent behavior, the true incidence of subependymomas remains unclear, and they usually appear as an incidental finding on imaging or autopsies of asymptomatic patients.<sup>[14,29]</sup> The presence of symptoms depends on the location and size of the tumor. Most symptomatic patients will present with symptoms related to obstructive hydrocephalus. Less commonly, focal deficits, seizures and subarachnoid hemorrhage have been reported.<sup>[30,36]</sup>

It is estimated that intracranial subependymomas account for only 0.07–0.7% of all intracranial neoplasms,<sup>[4]</sup> and due to the rarity and variable imaging characteristics of these tumors, preoperative diagnosis remains challenging, and many intracranial subependymomas are misdiagnosed as other diseases.<sup>[1]</sup>

It can be present on intracranial compartment or less frequently on the spine.<sup>[13,42]</sup> They typically presents as an intracranial mass within the ventricular system.<sup>[4]</sup> The most frequent location is the fourth ventricle accounting for 50–60% of cases, followed by the lateral ventricles accounting for 30–40% of cases. Less common sites include the spine, third ventricle, septum pellucidum, and brainstem.<sup>[1,22]</sup>

Even less frequently, subependymomas can be present as an intraparenchymal mass presenting with focal deficits and frequently being misdiagnosed as other glial tumors. This kind of presentation can be challenging for the surgeon and lead to improper management, thus it is paramount to recognize all the features these tumors can have.

## CLINICAL CASE

A 26-year-old female, previously healthy, without any remarkable medical history, presenting with left side progressive headache in the last few weeks, was admitted to the emergency department, reporting worsening of the headache and noticing visual impairment on the left eye. She was alert, calm and oriented, without other complaints. Physical examination showed right homonymous quadrantanopia on confrontation visual field testing. Fundoscopy showed no abnormalities.

She was submitted to a computed tomography (CT) scan that revealed an intracranial hypodense mass on the left occipital lobe, measuring 5.6 × 3.8 cm. Post contrast CT was then performed and the lesion showed no enhancement. Patient, therefore, was led to a brain magnetic resonance imaging (MRI), revealing a heterogeneous intra axial mass, on the left occipital lobe, hypointense on T1-weighted sequence relative to normal gray matter, hyperintense on T2-weighted sequence relative to normal gray matter, hyperintense on

FLAIR, with mild heterogeneous enhancement on central part of the lesion, and no enhancement on peripheral part of the lesion, on gadolinium T1-weighted image. Diffusion-weighted image showed no diffusion restriction while the same lesion was increased ADC relative to brain parenchyma. Susceptibility weighted imaging (SWI) showed hypointense signal dots within the lesion, corresponding to small amounts of hemorrhage. No peritumoral edema was evident. Careful analysis revealed the lesion was adjacent and related to the occipital and atrial portions of the left lateral ventricle [Figure 1]. Based on clinical and radiological findings, our main diagnostic hypotheses were glioma, neuronal-glial tumor, and subependymoma.

Patient was placed on a park bench position and underwent occipital craniotomy, with navigation guidance. During the final steps of the dissection a direct communication between the tumor and the ventricular system was evident, being possible to identify the occipital horn and atrium of the left lateral ventricle. After total removal, an external ventricular drainage was placed under direct view for better managing the postoperative care [Figure 2].

Postoperative CT showed no signs of surgical complications. Postoperative MRI was performed on the 1<sup>st</sup> day after surgery and revealed a complete resection [Figure 3].

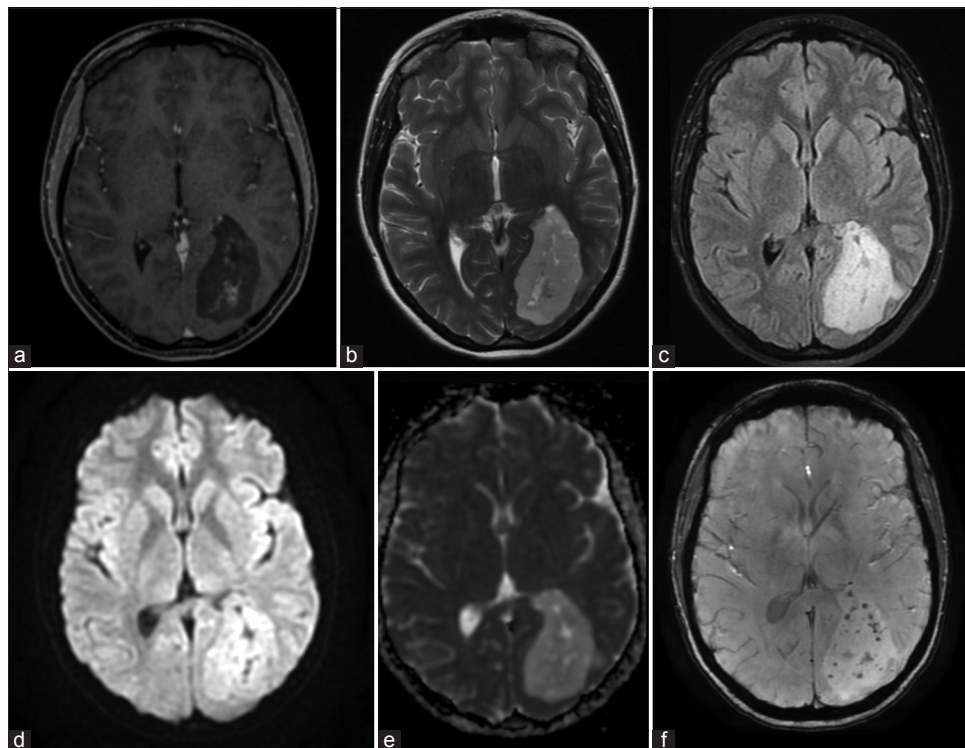
Histopathological analysis revealed hypocellular neoplasm composed of clustered cellular proliferation with round isomorphic nuclei, embedded in dense fibrillar matrix, and large acellular zones. There was no evidence of active mitosis, necrosis, or vascular endothelial proliferation [Figure 4].

Based on previous hypotheses and histopathological findings, immunohistochemical evaluation focused on the expression of neuronal and glial biomarkers, and revealed that the neoplastic cells were positive for glial fibrillary acidic protein (GFAP), and S100 protein. There was no immunoreactivity to epithelial membrane antigen (EMA), no loss of ATRX, no mutation on IDH1/2, and intact 1p-19q. Ki67/MIB-1 labeling index were 5% on hot spots [Figure 4]. No other immunohistochemical testing was made.

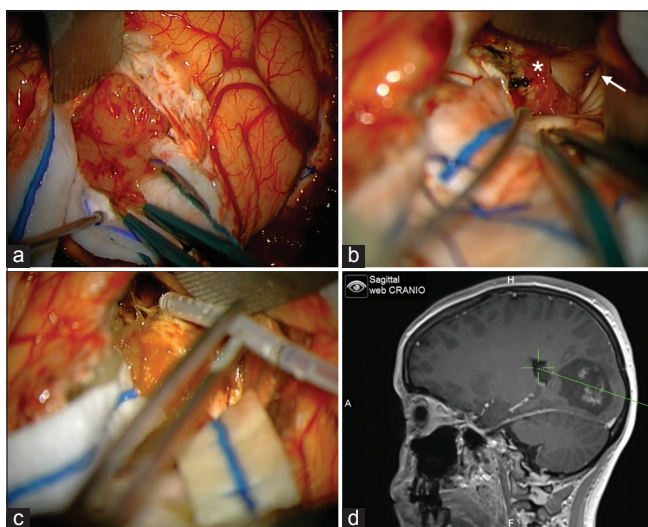
Based on imaging, histopathological and immunohistochemical evaluation, the diagnosis of an intracranial subependymoma was reached.

Patient was discharged without any additional neurological deficits 5 days after surgery, referring improvement on headache and remaining with preoperative visual impairment.

One year postoperative MRI was performed on long-term follow-up, and showed no signs of recurrence [Figure 3]. Computed visual field testing was made on immediate and long-term follow-up and showed right homonymous quadrantanopia without progression.



**Figure 1:** Imaging features of intraparenchymal subependymoma. (a) Axial gadolinium T1-weighted image showing a hypointense mass on the left occipital lobe, with mild contrast enhancement. (b) Axial T2 sequence showing a hyperintense heterogeneous mass. (c) Axial FLAIR sequence with hyperintense evident signal. (d) Axial diffusion-weighted sequence, with no diffusion restriction. (e) Axial ADC with increased signal compared to brain parenchyma. (f) Axial susceptibility weighted imaging showing small dots with hypointense signal within the lesion.



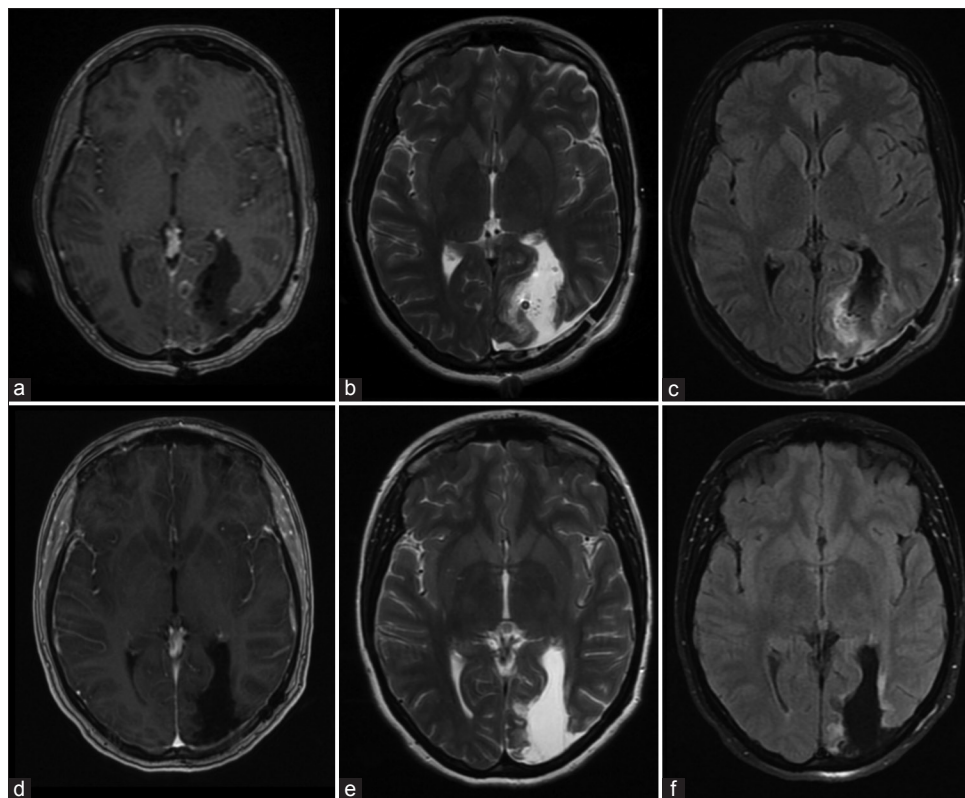
**Figure 2:** Intraoperative images. (a) A 4 cm corticectomy was made and a soft, smooth-contoured, and lobulated mass was evident. (b) At the final steps of the resection it was possible to see the tumor corridor connected the walls of the ventricular system, choroid plexus (white asterisk) and the atrium of the left lateral ventricle (white arrow). (c) Ventricular drainage was placed under direct view for better managing the postoperative care. (d) Use of navigation to confirm the extent of the resection and confirming positioning of ventricular drainage.

## DISCUSSION

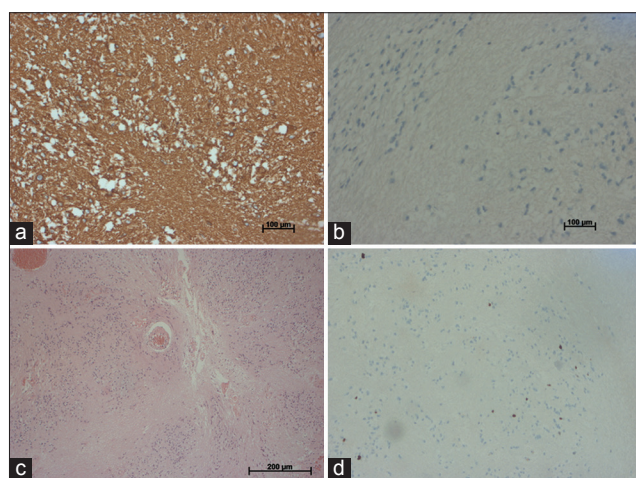
Subependymomas account for only 0.07–0.7% of all intracranial neoplasms,<sup>[4]</sup> and 8.3% of all ependymal tumors.<sup>[33]</sup> The true pathogenesis of subependymomas remains unclear, and many potential precursor cells are still debated. Suggestions include subependymal glia, astrocytes of the subependymal plate, ependymal cells, and a mixture of astrocytes and ependymal cells.<sup>[19,22,24,29]</sup>

Since the current literature regarding intracranial subependymoma is limited, its natural history and management remains unclear. Literature review showed more than 200 cases reported since first described by Scheinker in 1945, and a recent SEER-based analysis revealed a total of 466 intracranial subependymomas from 2004 to 2013, being probably the larger analysis on this kind of tumor.<sup>[26]</sup>

The WHO classification of CNS tumors (2016) has defined subependymomas as a subclassification of ependymomas, being considered a WHO Grade I tumor.<sup>[22]</sup> Very recent study published in 2020 by cIMPACT-NOW group proposed a new classification of ependymal tumors, based on anatomic site, biological and molecular features, but diagnosis



**Figure 3:** Imaging of postoperative MRI. (a) Day 1 after surgery axial gadolinium T1-weighted image showing no signs of contrast enhancement or presence of hypointense lesion previously visualized. (b) Day 1 after surgery axial T2 sequence without evidence of tumor. (c) Day 1 after surgery axial FLAIR sequence with slight hyperintense signal on tumor borders, compatible with recent postoperative imaging and no evidence of residual tumor. (d) One year after surgery axial gadolinium T1-weighted image showing no signs of contrast enhancement or tumor recurrence. (e) One year after surgery axial T2 sequence without evidence of tumor recurrence. (f) One year after surgery axial FLAIR sequence without evidence of tumor recurrence and improvement on hyperintense signal on tumor borders.



**Figure 4:** (a) Positivity for glial fibrillary acidic protein. (b) Negativity for epithelial membrane antigen. (c) Clusters of cellular proliferation embedded in dense fibrillar matrix and large acellular zones. (d) MIB-1 staining showing labeling index of 5%.

of subependymoma is still based only on histological examination.<sup>[6,22]</sup>

Histologically, subependymomas are composed of clusters of small uniform nuclei embedded in a dense fibrillar matrix of glial cell processes associated with large acellular zones, with frequent occurrence of small cysts. Tumor nuclei are isomorphic but occasionally pleomorphic nuclei may be seen. Calcifications and hemorrhage also may be present.<sup>[22,30,37]</sup>

Ki67/MIB-1 labeling index is an important and well recognized marker of cell proliferation, and mitotic activity. In subependymomas, they are usually low or absent and immunohistochemical studies of MIB-1 labeling index have found proliferation values of <1%, compatible with the slow growth pattern of this entity.<sup>[22,28]</sup>

Although considered an atypical feature, some authors reported levels of Ki-67 index higher than 1%, reaching 15% in some cases.<sup>[4,18,27,37]</sup> Our case has a proliferation index of 5%, posing an important challenge in postoperative management.

Immunohistochemical studies typically show immunoreactivity for neuronal markers as GFAP, neuronal cell adhesion molecule and neuron specific enolase. EMA is rarely expressed, unlike

**Table 1:** Literature review of intraparenchymal subependymomas; Note: N/A = Not available; w/o = without.

Case	Year reference	Location	Relationship with ventricle	Resection follow up		Radiological hypothesis
1	1989 Hankey <i>et al.</i> <sup>[10]</sup>	Parietal lobe	N/A	N/A	N/A	N/A
2	2005 Shuangshoti <i>et al.</i> <sup>[35]</sup>	Parietal lobe	N/A	N/A	3.5 years w/o recurrence	N/A
3	2005 Shuangshoti <i>et al.</i> <sup>[35]</sup>	Frontal lobe	N/A	N/A	N/A	N/A
4	2006 Ragel <i>et al.</i> <sup>[29]</sup>	Right temporal lobe	None	Total	3 years w/o recurrence	N/A
5	2006 Ragel <i>et al.</i> <sup>[29]</sup>	Right occipito-parietal lobe	Atrium of right lateral ventricle	Total	N/A	N/A
6	2012 Natrella <i>et al.</i> <sup>[25]</sup>	Left frontal lobe	None	Total	N/A	Pleomorphic xanthoastrocytoma, metastasis
7	2014 Kim <i>et al.</i> <sup>[16]</sup>	Left cerebellar hemisphere	None	Total	N/A	Pilocytic astrocytoma, hemangioblastoma
8	2015 Bi <i>et al.</i> <sup>[11]</sup>	Right thalamus, midbrain & temporal lobe	None	Partial	N/A	Glioma
9	2015 Bi <i>et al.</i> <sup>[11]</sup>	Left parietal lobe	None	Subtotal	N/A	Glioma
10	2015 Bi <i>et al.</i> <sup>[11]</sup>	Left frontal lobe	None	Total	N/A	Glioma
11	2018 Hanashima <i>et al.</i> <sup>[9]</sup>	Right occipital-temporal lobe	Right posterior horn of lateral ventricle	Total	1 year w/o recurrence	High grade glioma
12	2020 Lopes <i>et al.</i> , (Current case)	Left occipitoparietal lobe	Left atrium and occipital horn	Total	1 year w/o recurrence	Glioma, neuronal-glioma tumor

seen on ependymomas,<sup>[22,41]</sup> and no mutation on IDH1/2, loss of ATRX or codeletion of 1p/19q is typically found, unlike seen in gliomas.

On imaging they appear as a heterogeneous mass, with hypointense to isointense signal on MRI T1 sequence compared to gray matter. In the T2 sequence, they are hyperintense compared to gray matter. Contrast enhancement is generally mild to moderate. Low signal may be seen on SWI sequence, corresponding to calcifications or small hemorrhages. Peritumoral edema is typically not seen.<sup>[29,30,40]</sup>

The most common location of intracranial subependymomas is the fourth ventricle, accounting for 50–60% of cases, followed by lateral ventricles accounting for 30–40% of cases.<sup>[26,30]</sup> Less common sites include the spine, third ventricle, septum pellucidum, and brainstem.<sup>[1,22]</sup>

Rarely, intracranial extraventricular sites have been reported.<sup>[9]</sup> They present as an intraparenchymal mass with focal deficits and being misdiagnosed as other glial tumors. Typically, they have at least a small connection to the ventricular system, but in some cases they are exclusively intraparenchymal. This kind of presentation can be challenging for the surgeon and lead to improper management.

We performed a review of the English literature about subependymomas in intraparenchymal locations and found

only 11 cases reported, three on case reports and eight on case series. Most cases were misdiagnosed as glial tumors prior surgery. Tumor characteristics are summarized in [Table 1].

Our case had an obvious relationship to the ventricular system but there are some case reports where there are no direct connections between tumor and ventricular ependyma.<sup>[25,29]</sup>

Another interesting fact is that most subependymomas occur in the fourth ventricle; however, there is only one case report of intraparenchymal cerebellar subependymoma.<sup>[16]</sup> These facts raise questions about the underlying biology and precursor cells of this variant of subependymomas.

Differential diagnosis is usually made between typical tumors of ventricular system, such as ependymomas, medulloblastomas, central neurocytomas, astrocytomas, and meningiomas.<sup>[1]</sup> Concerning an intraparenchymal lesion, differential diagnosis will include other tumors more frequent than subependymomas, especially if there is no relation to the ventricular system.

There are no typical radiological features that allow us to elucidate the differential diagnosis between these tumors and gliomas, neuronal-glioma tumors, or other ependymal tumors; therefore, histopathological and immunohistochemical analysis is focused on this fact.

From our literature review, authors with intraparenchymal subependymomas report difficulty in distinguishing it from other tumors and patients underwent surgery with other diagnostic hypothesis such as gliomas, pilocytic astrocytoma, hemangioblastoma, pleomorphic xanthoastrocytoma, and metastasis [Table 1]. As it happened with other authors, our case was misdiagnosed as a glioma before surgery, and all the surgical strategy and management was made based on this diagnosis. This literature review brings to mind this rare differential diagnosis when looking into an intraparenchymal lesion close to the ventricular system.

Classical management of symptomatic subependymomas is surgery with maximum safe tumor resection.<sup>[15,38]</sup> Due to its benign behavior and slow growing pattern, a watchful management with serial imaging is reasonable in asymptomatic patients;<sup>[29]</sup> however, prospective series with observation on the natural history of these tumors has not been performed.

Some authors argue in favor of surgery even in asymptomatic cases because of the good general outcome after surgical resection, potential surgical cure, and avoiding the risk of clinical and neurological deterioration. In most series, surgery for subependymoma carries a good outcome, with gross total resection rates >70% and good outcomes after surgery.<sup>[1,11,12,15,29,38,40]</sup> Although rare, a rapid progression on an asymptomatic patient has been reported, and raises doubts about the safety of the conservative management.<sup>[20]</sup>

There are reports of tumor recurrence after subtotal removal, especially in tumors with atypical high Ki67/MIB-1 labeling index.<sup>[5,15,30,34]</sup> Bi *et al.* reported 10 years follow-up of 35 patients with recurrence on three patients.<sup>[1]</sup> Some authors argued that the cause of recurrence would be probably related to the high levels of proliferation index in tumors subtotally removed. This rationale is proven to be wrong since some reports of recurrence on tumors that were totally removed has been published.<sup>[1,5,18,37]</sup>

Today, we still do not have enough data to elucidate this question but it seems that recurrence rates are low but it can happen, even after gross total removal, especially in patients with high levels of ki-67 proliferation index.

Radiotherapy is reported for treatment of recurrent or residual tumors but the true role in the management of these lesions is still unclear.<sup>[1,35]</sup> Lombardi *et al.* reported seven patients who underwent radiotherapy, showing good results on follow-up especially on doses of 5,000 cGy or greater.<sup>[21]</sup> A recurrent subependymoma who underwent six resections has been treated with stereotactic radiosurgery with follow-up of 54 months and no tumor recurrence.<sup>[5]</sup>

There are some reports of radiotherapy with good results, but the limited number of patients and follow-up period preclude any conclusions. Statistical analysis of the SEER database

showed that the use of radiation therapy is not a significant prognostic factor in treatment of subependymomas,<sup>[26]</sup> suggesting the limited role of this adjuvant therapy in these tumors.

Molecular, immune and genetic studies have gained great prominence in the neuro-oncological field, trying to discover more effective and less harmful treatment methods. A study of potential therapeutic targets reported that TOP2B, HIF-1 $\alpha$ , MDM2, nucleolin, and phosphorylated STAT3 are frequently expressed in subependymomas, and also that a topoisomerase inhibitor and a p-STAT3/HIF-1 $\alpha$  inhibitor, demonstrated a growth inhibition of the subependymoma cell proliferation.<sup>[17]</sup>

This data suggest that these agents may have clinical impact in subependymoma treatment, but further research is necessary to reach any conclusions.

## CONCLUSION

Intraparenchymal subependymomas are extremely rare tumors, generally misdiagnosed as other tumors before surgery, so careful attention on clinical and radiological features must be taken when looking at a tumor close to the ventricular system.

They usually have a benign nature, with low Ki67/MIB-1 labeling index, but high levels of proliferation index may be found and seems to have impact on tumor recurrence, notably on subtotally removed tumors.

Surgical management is usually recommended, and due to the possibility of cure, total removal must be attempted. New adjuvant treatment options are being developed as our knowledge on biological and molecular characteristics advances, but to this date there are no proven effective adjuvant therapy on this type of tumor.

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## Declaration of patient consent

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

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Nil.

## Conflicts of interest

There are no conflicts of interest.

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