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Adult BRAF V600E-Mutant Pilocytic Astrocytoma Extending Into the Fourth Ventricle and Progressing Toward the Cerebellopontine Angle: A Case Report and Literature Review

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Shinsuke Abe [1], Fumihiro Matano [1], Chihiro Horii [2], Shinya Kojima [2], Yasuo Murai [1]

1. Neurological Surgery, Nippon Medical School, Tokyo, JPN 2. Diagnostic Pathology, Nippon Medical School, Tokyo, JPN

Corresponding author: Fumihiro Matano, s00-078@nms.ac.jp

Abstract

Pilocytic astrocytoma (PA) is an astrocytic tumor that primarily occurs in children and is relatively uncommon in adults. Among these, cases with adult-onset PA extending into the fourth ventricle and progressing toward the cerebellopontine angle are particularly rare. Our case involved a 27-year-old male who presented with headache and underwent surgical resection. Histopathological diagnosis was PA, and genetic panel testing detected a *BRAF* V600E point mutation. *BRAF* V600E mutations are uncommon in PA, particularly in adult posterior fossa tumors. The patient showed no evidence of tumor recurrence or regrowth at 1.5 years after surgery. This case highlights the anatomical and molecular heterogeneity of adult-onset PA and emphasizes the importance of integrated radiological, histopathological, and genetic evaluation. As demonstrated in this case, uncommon genetic alterations may be identified. Therefore, comprehensive molecular testing may be useful, particularly in cases of subtotal resection or recurrence, to identify potential molecular targets.

Categories: Oncology, Neurosurgery
Keywords: adult-onset, braf v600e mutation, fourth ventricular tumor, pilocytic astrocytoma (pa), subtotal resection

Introduction

Pilocytic astrocytoma (PA) is one of the most common brain tumors in children and typically arises in the cerebellum, optic pathways, hypothalamus, and brainstem. However, the incidence of PA decreases with age, and PA accounts for only 0.8% of central nervous system (CNS) tumors in patients aged ⩾19 years [1]. A Surveillance, Epidemiology, and End Results (SEER) study of 865 cases of adult-onset PA reported that cerebral tumors accounted for 29.7% of cases, representing the most common tumor location [2]. In the same study, intraventricular tumors accounted for only 9.5% of adult-onset PA cases, suggesting that tumors extending into the fourth ventricle are even rarer. Although there are reports of adult-onset PA originating in the cerebellum and extending into the fourth ventricle, as well as adult-onset PA arising in the cerebellopontine angle, to our knowledge, only one case involving a lesion in the fourth ventricle and a lesion in the cerebellopontine angle at the time of recurrence has been described in the literature [3]. We found no reports of a primary tumor extending simultaneously into the fourth ventricle and progressing toward the cerebellopontine angle. Herein, we report a case of primary adult-onset PA extending into the fourth ventricle and progressing toward the cerebellopontine angle. In addition, genetic testing in this case revealed a *BRAF* V600E point mutation; we also present the potential for molecularly targeted therapy.

Case Presentation

A 27-year-old man with intermittent headaches presented to our hospital with a persistent headache lasting one week. His medical history was notable for childhood asthma. Magnetic resonance imaging (MRI) revealed a cystic and solid lesion extending into the fourth ventricle and expanding toward the cerebellopontine angle. The lesion was hypointense on T1-weighted imaging (T1WI) and hyperintense on T2-weighted imaging (T2WI) and fluid-attenuated inversion recovery imaging, with minimal contrast enhancement on gadolinium (Gd)-enhanced T1WI (Figures *1A*-*1C*).

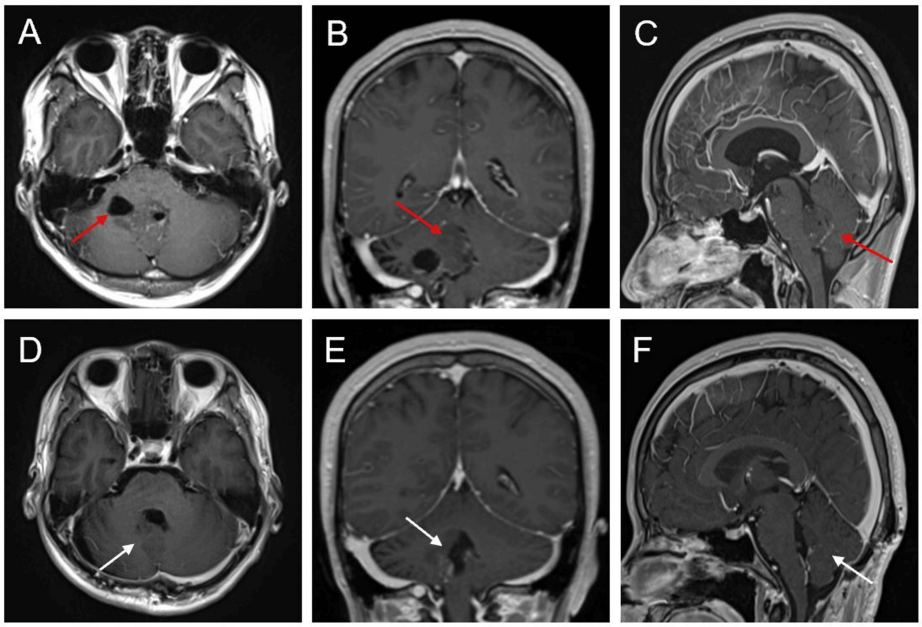


FIGURE 1: Preoperative gadolinium-enhanced MRI and postoperative gadolinium-enhanced MRI of this case

Preoperative gadolinium-enhanced MRI revealed a slightly hypointense solid lesion within the fourth ventricle, and axial imaging (A) showed that the cystic lesion was expanding toward the cerebellopontine angle. Coronal imaging (B) confirmed that the lesion was present throughout the fourth ventricle, and sagittal imaging (C) showed that the lesion was causing mild compression of the dorsal brainstem. (Red arrows in each figure indicate tumor.)

In the 19-day postoperative gadolinium-enhanced MRI, axial imaging (D) showed a residual lesion in the posterior region of the fourth ventricle, and coronal images (E) revealed a residual lesion above the right upper portion of the fourth ventricle. Sagittal images (F) demonstrated attachment of the residual lesion to the medullary velum. (White arrows in each figure indicate residual tumor.)

The lesion in the fourth ventricle measured 20 × 22 × 28 mm. A typical mural nodule, which is often associated with cystic PA, was absent. No spinal lesions were detected on MRI. Digital subtraction angiography revealed no tumor staining. Imaging showed that the tumor extended into the cerebellopontine angle; therefore, ependymoma was considered the primary preoperative diagnosis. Rosette-forming glioneuronal tumor (RGNT), which commonly occurs in young adults and frequently involves the fourth ventricle, was also considered in the differential diagnosis. Because of the persistent headache and the risk of obstructive hydrocephalus associated with tumor growth, surgical resection was performed. Surgery was performed via a midline suboccipital (telovelar) approach with the patient in the park-bench position because of the location of the lesion. After the arachnoid was incised and the cerebellar tonsil was retracted, the lesion was visualized. The fourth ventricle was filled with the lesion, which contained a partially cystic component. The lesion was resected using a central decompression technique. Intraoperative frozen section examination suggested PA. Although gross total resection (GTR) was attempted, subtotal resection (STR) was performed because of strong adhesion between the lesion and the middle cerebellar peduncle. A small portion of the tumor adherent to the lower cranial nerves was intentionally left in place to avoid neurological injury. Postoperatively, the patient developed delayed-onset right-sided hearing loss and right facial paralysis corresponding to House-Brackmann grade II [4]. The facial paralysis gradually improved with mecobalamin and corticosteroid treatment. The patient also experienced unsteadiness due to cerebellar ataxia, which improved with rehabilitation. He was discharged 23 days after surgery and continued regular outpatient follow-up. Contrast-enhanced MRI performed 19 days after surgery demonstrated complete resection of the cerebellopontine angle/cerebellar component, with a small residual tumor along the roof of the fourth ventricle (Figures *1D*-*1F*). The residual volume of the tumor was approximately 0.8 cm^3. MRI performed 1.5 years after surgery showed no change in the volume of the residual tumor and no evidence of residual tumor growth or recurrence, although the relatively short follow-up period remains a limitation. The postoperative right facial paralysis completely resolved; however, the right-sided hearing loss persisted at the latest follow-up.

Histopathological examination confirmed the diagnosis of PA. Neoplastic cells with round-to-oval nuclei and clear cytoplasm were observed, with occasional eosinophilic granules (Figure *2A*). Rosenthal fibers were also observed sporadically (Figure *2B*). Piloid cells with bipolar processes were present, and the processes of the neoplastic cells were diffusely positive for glial fibrillary acidic protein (Figure *2C*). Additionally, the neoplastic cells were diffusely positive for S100 protein (Figure *2D*), and their nuclei were positive for

oligodendrocyte transcription factor 2 (Olig2) (Figure 2E). The Ki-67 labeling index was 2%-3% (Figure 2F).

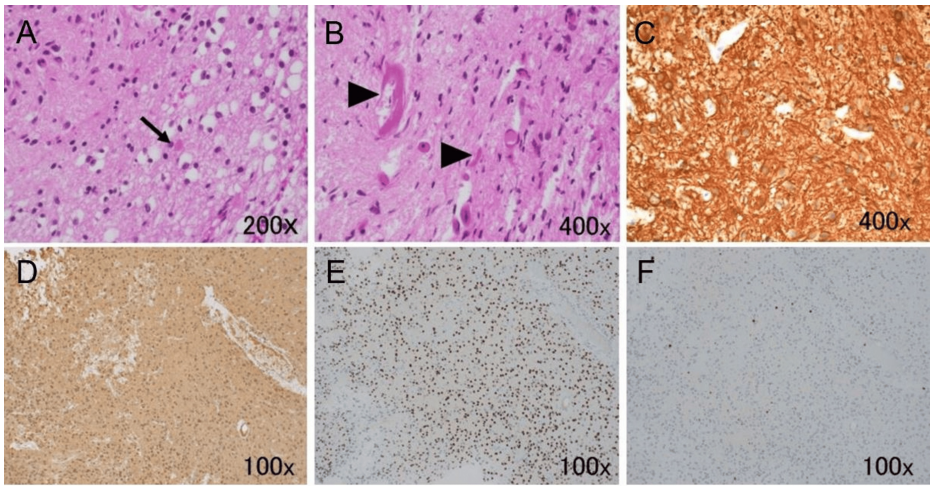


FIGURE 2: Histological analysis of this case

A: Acidophilic granules were observed in the hematoxylin and eosin stain. (black arrow, original magnification ×200)

B: Rosenthal fibers were observed in the hematoxylin and eosin stain. (black thick arrow, original magnification ×400)

C: The processes of the neoplastic cells were diffusely positive for glial fibrillary acidic protein (GFAP). (original magnification ×400)

D: Neoplastic cells were diffusely positive for S100 protein. (original magnification ×100)

E: Neoplastic cell nuclei were positive for oligodendrocyte transcription factor 2 (Olig2). (original magnification ×100)

F: Ki-67 labeling index was 2%-3%. (original magnification ×100)

Based on these histopathological findings, ependymoma, which is typically negative for Olig2, and RGNT, which demonstrates characteristic rosette formation, were excluded. Furthermore, the characteristic angiocentric pattern of pilomyxoid astrocytoma was not observed. Overall, the histopathological findings were consistent with PA. Subsequently, genetic panel testing was performed using the K-Panel, developed by Kagoshima University, which screened 88 genes, including those relevant to the diagnosis of glioma [5]. RNA and DNA were extracted from unstained histological specimens and analyzed using next-generation sequencing. DNA analysis revealed a *BRAF* V600E point mutation with a variant allele frequency of 17.8%. No other notable genetic alterations were detected. Detailed information on the genetic alterations identified is presented in Figure 3.

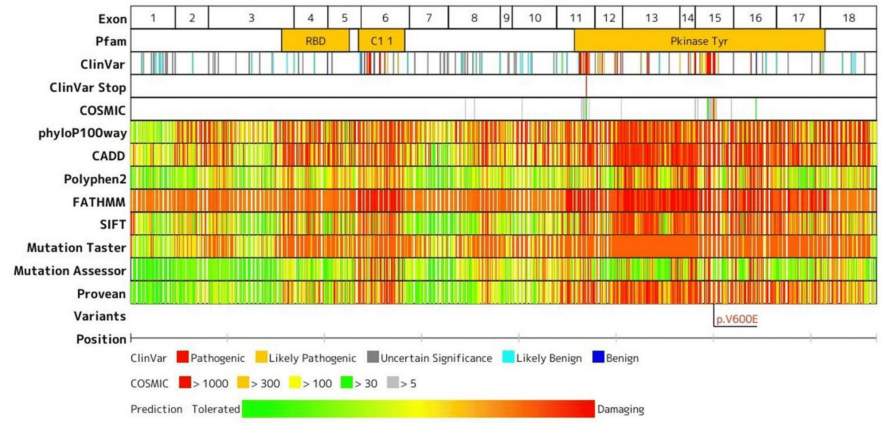


FIGURE 3: Molecular characterization of the brain tumor

Genetic analysis was performed using next-generation sequencers. In the *BRAF* gene, exon 15, a substitution was detected at the 1799th position in the nucleotide sequence (c.1799T>A), confirming the presence of the *BRAF* V600E point mutation.

Fusion genes were assessed using an RNA fusion panel designed to comprehensively detect fusion genes in CNS tumors and identify potential diagnostic markers and therapeutic targets; however, no fusion genes were detected. Thus, the *BRAF* V600E point mutation was the only alteration identified in the RAS/mitogen-associated protein kinase (MAPK) pathway.

Discussion

This case highlights several important clinical and molecular considerations in adult-onset PA. First, it underscores the importance of recognizing the characteristic imaging features of PA. PA can arise in various regions of the CNS, including the cerebellum, cerebral hemispheres, optic nerve, brainstem, optic chiasm, hypothalamus, basal ganglia, and spinal cord. In the present case, the tumor was presumed to originate in the cerebellar parenchyma and extend into the fourth ventricle based on surgical findings of adhesions to the wall of the fourth ventricle.

Previous studies have reported diverse MRI features of PA [6,7]. Cerebellar PA typically appears hypointense on T1WI, hyperintense on T2WI, and enhances on Gd-enhanced T1WI. In the present case, similar signal characteristics were observed on noncontrast MRI; however, contrast enhancement was minimal. Although cystic PA is often accompanied by a mural nodule, this feature was absent in the present case. Previous studies have also described PA as frequently comprising a mixture of cystic and solid components [8], consistent with the imaging findings in this case.

A previous clinical study reported a median Ki-67 labeling index of 2.8% in adult-onset PA [1], suggesting that the Ki-67 index observed in the present case was not particularly high. Numerous genetic alterations have been reported in PA. Alterations involving the MAPK pathway have been identified in many cases and may play an important role in the pathogenesis of PA. Among adult-onset PAs arising in the posterior fossa, the *KIAA1549-BRAF* (BK) fusion (*BRAF-KIAA1549* fusion) has been reported at a relatively high frequency [9].

In the present case, genetic panel testing revealed a *BRAF* V600E point mutation rather than a BK fusion (Figure *3*). Systematic reviews of gliomas harboring *BRAF* V600E mutations have reported a prevalence of only 3%-9% in PA [10,11], indicating that this alteration is relatively uncommon. Furthermore, in adult-onset PA, as in pediatric PA, *BRAF* V600E mutations are less common than BK fusions. *BRAF* V600E-positive adult-onset PAs have also been reported to occur more frequently in supratentorial than in infratentorial locations [9], making lesions arising within the fourth ventricle particularly rare. Conversely, posterior fossa PAs are more commonly associated with BK fusion. Therefore, the present case represents both an anatomical and molecular outlier. Although adult-onset *BRAF* V600E-positive PA has been described in sporadic case reports [12], relatively few cases have been reported. We searched PubMed for reports published over the past 25 years describing PA involving the fourth ventricle in patients aged ⩾20 years. The search terms included “adult pilocytic astrocytoma in the fourth ventricle”, “cerebellar pilocytic astrocytoma in adults”, “intraventricular pilocytic astrocytoma”, and “cerebellopontine angle pilocytic astrocytoma.” The last date of the literature search was October 1, 2026. Cases involving the fourth ventricle were extracted from the systematic review of adult-onset intraventricular PA by Woodiwiss et al. [13]. We additionally searched for cases of PA involving the fourth ventricle in patients aged ⩾20 years that were not included in that systematic review. In this process, we reviewed the references cited in the systematic review to ensure there were no duplicate cases. Of the cases involving the fourth ventricle identified in the systematic review,

only one was excluded because the patient was aged <20 years. As shown in Table *1*, 11 cases of adult-onset PA involving the fourth ventricle have been reported in the literature [3,8,13-17]; however, to our knowledge, none have been reported to harbor a *BRAF* V600E point mutation. Also, since genetic test results have not been reported for some cases in the past, it is important to appreciate that there are limitations to comparing them with this case.

Author, year	Age, years	Sex	Extension to the CPA	Genetic alteration	Extent of resection	Adjuvant treatment	Recurrence*	Follow-up, years**	Outcome***
Ellis et al., 2009 [14]	22	M	NR	NR	GTR	RT	□	3.5	NR
Trabelsi et al., 2015 [15]	58	M	□	BK fusion	GTR	NR	□	2	Dead
Xia et al., 2016 [8]	30	F	NR	NR	NR	NR	□	NR	NR
Xia et al., 2016 [8]	32	F	NR	NR	NR	NR	□	NR	NR
Xia et al., 2016 [8]	29	M	NR	NR	NR	NR	□	NR	NR
Rodriguez et al., 2019 [16]	68	M	NR	NF1	STR	□	NR	NR	Dead
Khan et al., 2021 [3]	26	F	□	□	NTR	□	□	0.8	Dead
Korkmazer et al., 2023 [17]	20	F	□	NR	NR	NR	□	NR	NR
Korkmazer et al., 2023 [17]	26	M	NR	NR	NR	NR	□	NR	NR
Woodiwiss et al., 2024 [13]	69	M	□	□	NTR	□	□	0.5	Dead
Woodiwiss et al., 2024 [13]	71	M	□	□	NTR	RT	□	6	Dead
Present case	27	M	+	*BRAF* V600E	STR	□	□	1.5	Alive

TABLE 1: Review of previously reported cases of adult pilocytic astrocytoma involving the fourth ventricle

CPA, cerebellopontine angle; GTR, gross total resection; NR, not reported; NTR, near-total resection; RT, radiotherapy; STR, subtotal resection; +, present; −, absent.

*Presence or absence of recurrence based on the information reported in the literature.

**Time from treatment to the last follow-up or recurrence.

***Patient status (alive or dead) at the last reported follow-up.

Recent studies have indicated that pediatric patients with *BRAF* V600E-positive PA do not have a significantly different prognosis from those with *BRAF* wild-type PA [18]. However, the prognostic significance of this mutation in adult-onset PA remains unclear and warrants further investigation. In recurrent tumors harboring actionable genetic alterations, molecularly targeted therapies have shown therapeutic potential. Combination therapy with dabrafenib and trametinib has demonstrated efficacy in recurrent *BRAF* V600E-positive low-grade gliomas [19], and vemurafenib has also been reported to be effective in tumors harboring *BRAF* V600E mutations [20]. These findings highlight the potential role of molecularly targeted therapies in recurrent tumors and in cases in which surgical resection is difficult. However, the number of patients with PA included in previous clinical trials has been limited. Given the rarity of adult-onset PA, multicenter prospective studies are needed to establish the efficacy and safety of molecularly targeted therapies in this population. In the present case, careful follow-up is required to monitor for recurrence within the cerebellar parenchyma and regrowth of the residual tumor in the fourth ventricle. If recurrence or tumor regrowth occurs, molecularly targeted therapy may represent a potential treatment option. Future studies should also compare the outcomes of molecularly targeted therapy with

those of radiotherapy for recurrent or progressive disease and evaluate the potential role of combined treatment approaches.

Previous studies have reported that PA in adults is associated with a poorer prognosis than PA in children [1,21]. Clinical studies have also demonstrated higher mortality rates among adults with PA than among children and adolescents, with survival decreasing with increasing age at diagnosis [1]. Although overall survival rates among patients diagnosed with PA at 20-39 years of age are relatively high (93% at two years and 89.5% at five years), they remain lower than those among patients diagnosed at ⩽19 years of age.

GTR has been considered an important prognostic factor in adult PA. A previous meta-analysis suggested that GTR is important for reducing the risk of recurrence and improving prognosis [22]. However, a recent study reported that although GTR was associated with prolonged progression-free and recurrence-free survival in pediatric patients, no such association was observed in adults [23]. As shown in Table *1*, recurrence within five years has been reported even after GTR. Therefore, long-term follow-up is necessary in patients with adult-onset PA, regardless of whether GTR or STR is achieved. Previous studies have recommended a follow-up period of at least 40 months after GTR [24]. In patients undergoing STR, as in the present case, the possibility of recurrence or residual tumor growth should be carefully considered. Given the young age of the patient, long-term surveillance with MRI is essential. No randomized controlled trials have evaluated the efficacy of adjuvant radiotherapy following STR or GTR for PA [2]. Prospective studies have demonstrated favorable outcomes with surgery alone, including both STR and GTR [25], whereas a 2021 study reported poorer overall survival among patients who received postoperative radiotherapy [26]. Considering the available evidence, adjuvant radiotherapy was not administered in the present case. However, it is important to note that the results of this meta-analysis may be attributable to patient selection rather than treatment-related adverse events.

GTR is an important prognostic factor but is not always achievable. When complete resection cannot be achieved, the role of adjuvant radiotherapy remains limited, and its clinical benefit is uncertain; therefore, its use should be carefully considered on a case-by-case basis. Additionally, the development of novel adjuvant strategies, particularly molecularly targeted therapies, may provide more effective treatment options and improve outcomes in such cases.

Conclusions

We report a case of adult-onset *BRAF* V600E-mutant PA extending into the fourth ventricle and progressing toward the cerebellopontine angle. This case highlights the anatomical and molecular heterogeneity of adult-onset PA and emphasizes the importance of integrated radiological, histopathological, and molecular evaluation. As demonstrated in this case, uncommon genetic alterations may be identified. Comprehensive molecular profiling may be particularly valuable in adult patients with residual, recurrent, or surgically challenging PA, as it may identify potentially actionable alterations and inform targeted therapeutic strategies.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Shinsuke Abe, Yasuo Murai

Acquisition, analysis, or interpretation of data: Shinsuke Abe, Fumihiro Matano, Chihiro Horii, Shinya Kojima

Drafting of the manuscript: Shinsuke Abe

Critical review of the manuscript for important intellectual content: Fumihiro Matano, Chihiro Horii, Shinya Kojima, Yasuo Murai

Supervision: Yasuo Murai

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Nippon Medical School issued approval R1-11-1212. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** This work was supported by KAKENHI (Grant Number 22K16671) from the Japan Society for the Promotion of Science (JSPS). . **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other

relationships or activities that could appear to have influenced the submitted work.

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