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(CASE REPORT)

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Recurrent adamantinomatous craniopharyngioma: Case report of an uncommon tumor, and a brief review of the literature

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Abstract

Adamantinomatous craniopharyngioma (ACP) is a rare, WHO type I, benign but aggressive tumor, associated with a known challenging diagnosis. It overwhelmingly occurs in the pediatric population. ACP is known for being locally aggressive, with a high recurrence rate and long-term morbidity. We present a case of a 12-year-old boy who was diagnosed with ACP after a long journey with symptoms lasting three years. On physical examination, there was evidence of raised intracranial pressure, delayed puberty, and signs of central diabetes insipidus. MRI and CT scans revealed a huge multicystic, focally calcified suprasellar mass leading to hydrocephalus. The imaging features, in addition to the clinical and laboratory data, indicated a craniopharyngioma.

The patient was subjected to endoscopic transsphenoidal resection that was partially successful since the tumor was firmly adherent to the surrounding structures. Histopathologically, the tumor features were consistent with the diagnosis of ACP. Immunohistochemistry (IHC) and molecular analysis showing the WNT signaling pathway activation due to the mutation of CTNNB1 confirmed the definitive diagnosis. The postoperative treatment involved external beam radiotherapy and a multidisciplinary follow-up of endocrine and visual sequelae. The patient developed recurrence of the tumor after three years of treatment, with a subtotal resection being done once again. He was still under active follow-up at the four-year follow-up, with the emphasis on long-term disease control and quality of life.

This case highlights the significance of early detection of silent neuroendocrine symptoms, precision in imaging analysis, and the multidisciplinary management strategy in the treatment of pediatric ACP, which has a high recurrence rate and long-term consequences.

Keywords: Adamantinomatous craniopharyngioma; Aggressive tumor; Pituitary; Adherent Tumor; Recurrence

1. Introduction

Craniopharyngiomas are classified into two main types: adamantinomatous (ACP) and papillary (PCP). ACP is more common in children and tends to be less solid with cystic components, while PCP is more common in adults and is characterized by a more solid tumor structure. [1] ACP is a rare, benign, epithelial subtype of craniopharyngioma tumor that arises from the fragments of Rathke's pouch. [2] Craniopharyngioma was first described by Zenker in 1857 and introduced as a term in 1932 by Cushing. [3] This tumor arises along the pituitary stalk within the suprasellar region

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alongside the optic chiasm. [2] ACP is characterized by its activation of the Wnt signaling pathway. This activation leads to mutations in the CTNNB1 gene, which is responsible for encoding β-catenin, leading to tumorigenesis. [4]

The incidence of craniopharyngioma follows a bimodal distribution, with peaks between 5 and 14 years and again in adults aged 40 to 70 years. They account for 1 to 5% of all primary intracranial tumors and represent one of the most common non-glial tumors in children. The environmental or inherited risk factors for ACP are difficult to find. [5]

ACP tumor growth progresses slowly with insidious development of symptoms, with a range of 1-2 years of symptom development to diagnosis. [3] Typical presentation involves a range of symptoms from hypothalamic/pituitary deficiencies, visual impairment, and increased intracranial pressure. [5] Compression of the pituitary structures leads to endocrine abnormalities such as hormone deficiencies of thyroid-stimulating hormone, growth hormone, adrenocorticotropic hormone, and vasopressin. [2] Visual symptoms develop due to compression of the optic chiasm or nerves, resulting in visual deficits with variation depending on the size of the tumor. [1] Intracranial pressure increases from mass effect due to a tumor or secondary hydrocephalus due to obstruction of the ventricular system, with symptoms of headaches, nausea, and vomiting. [3] ACP can also present with more generalized symptoms, slowed cognition, and depression due to the broadening of the tumor into the frontal cortex, thalamic, or limbic system. [2]

Diagnosis of ACP involves neuroimaging with detection of a suprasellar enhancing mass in the region of the pituitary stalk. It must be distinguished from other tumors that can be present in the same area. [6] Treatment methods involve aggressive total resection or conservative partial resection followed by radiotherapy. [7] We present a rare case of adamantinomatous craniopharyngioma with silent neuroendocrine symptoms to highlight the importance of early detection of ACP in pediatric patients to prevent long-term consequences and reduce recurrence.

2. Case presentation

A 12-year-old boy was referred to the pediatric neurology clinic because of a 3-year history of progressive, frequent headaches, eyesight problems, and fading energy. The mother, who brought him, was concerned with his slow physical development compared with his peers. She also noticed slight cognitive deterioration and personality changes that had taken place recently. The symptoms had developed gradually and insidiously and had led to various assessments over the last three years without any definitive diagnosis. The patient described his frequent headaches as occurring in the early morning with nausea and vomiting, and they were not related to meal time or food intake.

The patient had a history of well-controlled type 1 diabetes mellitus. His mother remembered that one of the blood tests ordered earlier showed irregularities in some hormone levels. She recalled the abnormalities included growth hormone, luteinizing hormone, and thyroid-stimulating hormone. She reported that there was no follow-up as she felt it was due to discrepancies in the laboratory results. She reported that she noticed he recently developed a loss of peripheral vision and was hitting objects or hitting the room wall. Additionally, it was reported that he had frequent urination and gradually started to have phases of confusion, loss of energy, and instability of gait. Most recently, he had reported bilateral temporal hemianopsia, which had a major effect on his daily functioning.

Physical examination revealed that the boy was short in proportion to his age and had growth hormone deficiency indicators and delayed puberty. He was significantly obese and had polydipsia and polyuria, raising suspicion of central diabetes insipidus. The fundoscopic examination showed papilledema, which denotes elevated intracranial pressure. Neurological assessment revealed a visual field defect and loss of acuity. Laboratory studies favored pituitary dysfunction, and there were numerous hormonal deficiencies and laboratory findings of central diabetes insipidus. His diabetes was well controlled.

Considering the combination of the symptoms, the clinical manifestations, and the endocrine disturbances, a mass lesion in the central nervous system was presumed. CT revealed gross calcifications in the lesion and obstructive hydrocephalus. Brain MRI showed a large, multilobulated, multicystic lesion of about 3 cm with irregular rim enhancement in the suprasellar area with mass effect on the nearby structures. The cystic elements were hyperintense on the T1-weighted images, indicating a proteinaceous nature. The cyst walls and solid parts were heterogeneously enhanced. These results were best indicative of a craniopharyngioma, especially of the adamantinomatous type. Differential diagnoses were optic pathway/hypothalamic glioma, germ cell tumors, and, more rarely, Rathke cleft cyst or xanthogranuloma.

The case was discussed in a multidisciplinary tumor board. Most of the recommendations were for a surgical resection through an endoscopic transsphenoidal surgery (ETS), and postoperative radiotherapy was only to be used in case of residual disease. Surgery showed a complex lesion that firmly adhered to surrounding neurovascular structures, and

total removal was not possible, resulting in a subtotal resection of the tumor confirmed by postoperative imaging. Intraoperative frozen section consultation identified bland squamous cells and foamy histiocytes, but final diagnosis was deferred to the permanent sections. Multiple fragments of tan tissue measuring 1.5 cm in total aggregate were reported by gross pathological examination, which was intermingled with greasy, oily material. Microscopic examination showed features compatible with adamantinomatous craniopharyngioma. These characteristics were lobules and trabeculae of well-differentiated squamous epithelium with peripheral palisaded cells and a central stellate reticulum. Nuclear morphology was bland, and there was minimal atypia and no mitotic activity. The predominant finding was cystic degeneration and xanthogranulomatous inflammation, with occasional multinucleated giant cells. The tumor formed a loose, stellate reticulum, a star-shaped network of cells that gave the impression of a "wet keratin" or "ghost cell" appearance. (Figure 1 A, B)

Immunohistochemistry (IHC) studies showed strong nuclear positivity of the tumor cells for β-catenin. In addition, other positive markers included: EMA, CK7, CK8, CK5/6, CK19, vimentin, and pancytokeratin. The mutant BRAF (V600E) markers, neuroendocrine differentiation, and pituitary hormones were negative. Ki-67 proliferation index was low (about 6 %) and was localized to the squamous basal palisading layer. Definitive diagnosis was established after molecular testing and identification of the activating CTNNB1 mutations of the WNT signaling pathway. The final reported diagnosis was adamantinomatous craniopharyngioma (WHO Grade I).

The patient was later subjected to external beam radiotherapy. There were no postoperative complications, and it was uneventful. Hormonal replacement therapy was used in long-term management, and a multidisciplinary team was used to follow up the patient to monitor recurrence and to manage endocrine, visual, and cognitive sequelae.

Surveillance MRI at three years showed recurrence of the tumor as a multiseptated cystic lesion of about 9 cm in the suprasellar region, and a second subtotal resection was done. Follow-up imaging showed persistence of disease, and the patient was closely followed by radiologic and clinical monitoring. Four years after initial diagnosis, the patient was still alive and still in regular endocrinologic care at the time of the last visit before being lost to follow-up. The patient needed constant hormonal therapy and neuro-ophthalmic observation. This follow-up was made on the balance between the tumor control and adjusting to the quality-of-life factors, which testifies to the complexity and chronic character of this rare pediatric tumor.

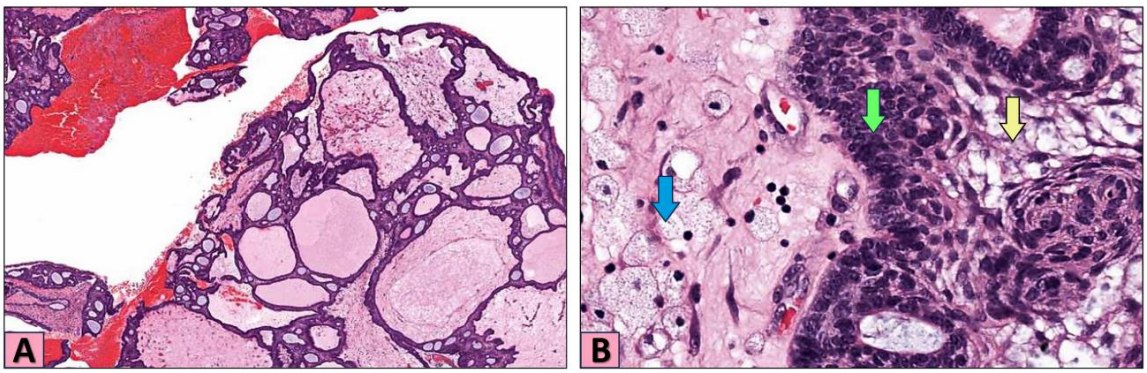

Figure 1 Histomorphology of the resected adamantinomatous craniopharyngioma (ACP); 1A Low power view showing nodular, fragmented, predominantly cystic tumor (H&E stain X20); 1B: High power view showing characteristic cellular components: Xanthomatous cells (Blue arrow), squamous basal cell palisading (Green arrow), and central stellar reticular cells (Yellow arrow) (H&E stain X40)

3. Discussion

3.1. History, epidemiology, risk factors, and WHO classification

Craniopharyngiomas are rare epithelial tumors arising from remnants of Rathke's pouch. They were first described by Jakob Erdheim in 1904. [5] These neoplasms are histologically benign yet clinically aggressive due to their proximity to vital neurovascular structures and their propensity for recurrence. Craniopharyngiomas are broadly categorized into two histological subtypes: adamantinomatous craniopharyngioma (ACP) and papillary craniopharyngioma (PCP), with ACP being more commonly encountered in the pediatric population. [8]

Craniopharyngiomas account for approximately 1.2% to 4.6% of all intracranial tumors, and 5% to 10% of pediatric central nervous system (CNS) tumors, with an estimated incidence of 0.5 to 2 cases per million population per year. [7] [8] The incidence of craniopharyngioma follows a bimodal distribution, with peaks between 5 and 14 years and again in adults aged 40 to 70 years. They account for 1 to 5% of all primary intracranial tumors and represent one of the most common non-glial tumors in children. The environmental or inherited risk factors for ACP are difficult to find. [5] The typical clinical presentation includes signs of increased intracranial pressure, visual disturbances, and endocrinopathies such as delayed puberty, growth hormone deficiencies, or central diabetes insipidus.

While the precise etiology of ACP remains unclear, mutations in the CTNNB1 gene, leading to activation of the WNT/β-catenin signaling pathway, have been identified in nearly all cases of ACP. [9] These somatic mutations are not inherited and occur sporadically. No established familial and environmental risk factors have been identified. However, the tumor's slow-growing and locally invasive nature contributes to delays in diagnosis, as initial symptoms may be insidious and attributed to other conditions. [9]

According to the 2021 World Health Organization (WHO) Classification of Tumors of the Central Nervous System, adamantinomatous craniopharyngioma is defined as a WHO Grade I tumor, consistent with a low-grade tumor displaying non-malignant histologic features and limited cellular proliferation [10]. Despite this, ACP presents a substantial clinical challenge due to its anatomical location, high recurrence rate, and the requirement for long-term multidisciplinary care, especially in pediatric populations.

Early identification using imaging features such as calcified, multicystic, suprasellar masses combined with hormonal and visual disturbances should prompt further evaluation. As our case illustrates, delayed diagnosis can result in considerable neuroendocrine sequelae and a more complicated treatment trajectory.

3.2. Imaging features

Usually, both CT and MRI are used together; they have complementary, often mutually exclusive roles in imaging of ACP. CT is the best at picking up calcification, which is a typical feature of this form of tumor. However, MRI will provide superior detail of the soft tissues and is essential in planning surgery. [11]

CT is very good at identifying the calcified area of the tumor. On CT scan, they show up as bright, dense foci and are seen in up to 90 percent of ACP in children. CT is also effective at demonstrating the overall structure of the tumor, both cystic and solid, and can demonstrate any accompanying hydrocephalus by demonstrating ventricular dilation. [6] The main imaging modality used in the diagnosis of craniopharyngiomas is MRI since it gives a lot more detail of the soft tissues and how they relate to surrounding structures. MRI is the best way to look at both new and old ACP tumors. T1-weighted MRI with gadolinium contrast shows solid tumor parts better and finds cystic changes. T2-weighted MRI can distinguish between hydrocephalus, edema, and cystic parts. [1]

Magnetic resonance imaging (MRI) is also the best way to look at both new and old ACP tumors. T1-weighted MRI with gadolinium contrast shows solid tumor parts better and finds cystic changes. T2-weighted MRI can distinguish between hydrocephalus, edema, and cystic parts. [11] The cysts are commonly proteinaceous and cholesterol-rich in content and may be bright on T1-weighted images (a finding often called motor oil cysts). [12] These cysts are normally bright on T2-weighted images. Enhancement of the solid tumor parts is bright when a contrast agent (gadolinium) is injected. [11] MRI is also invaluable in determining the exact location of the tumor about life-threatening structures such as the optic chiasm, pituitary gland, and hypothalamus, which is very necessary in planning the surgical removal and the minimal damage to these structures. [6] DWI and FLAIR are two types of imaging that can help tell the differences between the properties of cystic fluid. [13] Volumetric MRI can accurately track the growth or return of a tumor over time. It is still the best way to detect recurrence, particularly post-surgery. [14]

Monitoring hormones and clinical conditions is also beneficial. Functional evaluation is important because ACP often comes back near the hypothalamic-pituitary axis. [17] Endocrine testing should include measuring levels of TSH, ACTH, GH, prolactin, LH/FSH, cortisol, and other hormones. [6] Additionally, neuro-ophthalmologic exams that include visual field testing are essential because initial or recurrent tumors can affect the optic chiasm. [12] Advanced imaging techniques are often used as an extra tool or for research purposes. MR spectroscopy can tell the difference between recurrence and necrosis or gliosis. [15] PET (Positron Emission Tomography) scans are helpful when an MRI is inconclusive. Examples are 18F-FDG or 11C-methionine. [16]

3.3. Differential diagnosis Key features

ACP is often seen in children (5–15 yrs), unlike papillary CP (adults). Imaging shows common coarse calcifications in ACP (especially on CT). Cysts and solid components are classical features in ACP (machinery oil fluid signal). There is usually an irregular rim enhancement in MRI contrast images of ACP. Histopathology, "wet keratin", and “palisading squamous epithelium” are characteristic of ACP. β-catenin shows strong nuclear staining in ACP. Molecular studies show ACP with CTNNB1 mutation, and papillary CP with BRAF V600E mutation. Table 1 summarizes the key features of the differential diagnosis and how to differentiate from ACP.

Table 1 Differential Diagnosis Key Feature How to Differentiate from ACP

Differential Diagnosis	Key Features	How to Differentiate from ACP
Papillary Craniopharyngioma	Occurs in adults (40–60 y), solid > cystic, lacks calcification	No "wet keratin" or calcification; lacks β-catenin nuclear staining; BRAF V600E mutation positive
Rathke’s Cleft Cyst	Benign cystic lesion, often asymptomatic; located in the sellar/suprasellar region	No calcification; simple cyst on MRI (T1 hyperintense if proteinaceous); lacks palisading epithelium or "wet keratin"
Optic Pathway/Hypothalamic Glioma	Pediatric tumor; associated with NF1; solid, T2 hyperintense	Lacks calcification and cysts; no epithelial lining; GFAP+, not β-catenin+
Germinoma	Midline tumors in children/young adults, often in the pineal/suprasellar regions	Iso/hypodense on CT, no calcification; elevated β-hCG/AFP in serum/CSF; CD117+, PLAP+
Pituitary Macroadenoma	Common in adults; solid, expansile sellar mass	Homogeneous enhancement; no calcification or cysts; hormonally active or compressive symptoms; pituitary origin on MRI
Langerhans Cell Histiocytosis (LCH)	Systemic disease affecting bones, pituitary; diabetes insipidus is common	MRI shows thickened pituitary stalk; CD1a+, S100+, Birbeck granules (EM); systemic LCH features
Epidermoid Cyst	Benign, slow-growing lesion in the basal cisterns	Follows CSF on MRI but shows restricted diffusion (DWI); no calcification or enhancement; keratinous debris.
Arachnoid Cyst	Arachnoid Cyst	Purely cystic with no enhancement or calcification; follows CSF on all MRI sequences.
Hypothalamic Hamartoma	Pediatric, associated with precocious puberty or gelastic seizures	Non-enhancing, isointense to gray matter on all sequences; no cyst or calcification

*Compiled from References 1,2,4,6,10,15, and 21

3.4. Treatments and outcomes

Because of its vascularity, adherence to surrounding structures, and morphologic resemblance to other sellar and suprasellar neoplasms, the management of ACP is challenging. The management of ACP is similar to that of other posterior pituitary tumors, with the endoscopic transsphenoidal surgery (ETS) procedure being the first line of management. [17] ETS is preferred due to its direct access, lower morbidity, and reduced risk of neurovascular injury when compared to transcranial methods. [1]

Complete resection can be difficult to perform, as these tumors are hypervascular and often infiltrative, particularly when the diagnosis is delayed. In this case, the intraoperative findings of vascularity and adherence limited the extent of resection, resulting in subtotal removal. [17] Residual tumor volume is an established indicator of recurrence, and therefore, adjuvant radiotherapy is recommended when subtotal resection is achieved to improve local control. [18]

While there is no consensus on the timing or modality of radiotherapy due to the rarity of these tumors, fractionated stereotactic radiotherapy (FSRT) and intensity-modulated radiotherapy (IMRT) have demonstrated efficacy in controlling tumor progression in cases of residual or recurrent disease, as observed in our patient. [2] Some studies reported radiologic stability in over 80% of patients receiving adjuvant therapy following subtotal resection. [18]

Prognosis is generally favorable, consistent with the tumor's low-grade classification and low mitotic index, with most patients experiencing long-term survival. However, recurrence is not uncommon, especially after incomplete resection, with time-to-recurrence ranging from 6 months to several years. [17] The case we present illustrates this trend as the tumor relapse took place not later than 9 months after which the tumor had to be removed again using surgical excision and subsequent radiotherapy.

Another long-term effect is endocrinologic complications, especially hypopituitarism. They can be caused by pressure of the tumor, manipulation of the pituitary gland during surgery, or radiation damage. In our case, the patient acquired hypopituitarism after recurrence and radiotherapy, and thus, the patient would have to use hormonal replacement therapy as long as she lives. It is thus important to have close longitudinal endocrinologic monitoring. [2]

Surveillance strategies commonly involve serial MRI scans every 3 to 6 months during the first two years after surgery, followed by annual imaging thereafter if the patient remains stable. Continued follow-up with a multidisciplinary team, including neurosurgery, endocrinology, and neuro-oncology, is crucial for detecting any recurrence early and preventing potential treatment-related complications. [19]

In conclusion, ACP is histologically benign but has a propensity to recur, may have endocrine issues, and surgical management is complicated and, thus, needs multidisciplinary, multimodal management. The timely detection of the condition and the development of individual treatment plans are also crucial to the positive patient outcomes and long-term quality of life.

3.5. What does the future hold for the diagnosis and treatment of adamantinomatous craniopharyngioma

Diagnosis and treatment of ACP are experiencing a paradigm shift with the rapid advancement in technology and molecular research. The ACP treatment has traditionally been done using surgery and radiotherapy, with the treatment being restricted by the sensitive site of the tumor, the recurrence of the tumor, and the long-term effect that the treatment has on neuroendocrine and visual functions. However, the future is looking promising due to a greater knowledge of the molecular biology of tumors and the advent of precision medicine.

New molecular profiling, particularly the identification of CTNNB1 mutations and aberrant WNT/beta-catenin signaling, provides new opportunities to target therapy. Active clinical trials are investigating WNT pathway inhibitors, immune modulation, and epigenetic regulators, which one day may complement or substitute standard treatment. [20] Also, liquid biopsy approaches, via cerebrospinal fluid or plasma, can detect recurrence or treatment response at an earlier time point, potentially reducing the number of imaging studies.

Imaging-wise, high-resolution MRI, diffusion tensor imaging (DTI), and functional MRI (fMRI) are becoming more precise in terms of preoperative planning and enabling neurosurgeons to maneuver through intricate tumor-to-tumor relations without destroying essential structures. [21] The use of intraoperative neuro navigation and augmented reality is becoming more widespread in the field to enhance surgical accuracy and minimize morbidity. [22] Likewise, the current methods of proton beam therapy and conformal radiotherapy facilitate the ability to target remaining tumor tissue more effectively and without damaging the healthy parts of the brain. [23]

The field of artificial intelligence (AI) is set to transform diagnostics and assist in the planning of treatment, with initial machine-learning models demonstrating the potential to perform tumor segmentation, growth prediction, and even histological classification. The use of AI in clinical practice can contribute to the personalization of treatment plans, tracking cognitive and hormonal trends, and detecting the first manifestations of a relapse. [24]

Concisely, there is more hope for less invasive, more individualized, and biologically oriented methods in the future that can turn ACP into a more multidisciplinary, precision-based disease as opposed to a surgically centered disease.

3.6. What did we learn from this case?

This case is a good illustration of the difficulties of the diagnosis and treatment of ACP in children, particularly when the tumor has subtle and progressive symptoms, which resemble more common non-neoplastic illnesses. Among the most

important lessons is the near-essential role of early neuroimaging of children with unexplained endocrine dysfunction, cognitive alterations, and visual complaints, even where these symptoms occur insidiously over time.

The case also supports the shortcomings of the existing surgical alternatives. Although expert care and multidisciplinary decision-making were utilized, full resection was not possible because of the tumor's firm adherence to the nearby neurovascular structures, which is a characteristic of ACP. The recurrence of the tumor following initial subtotal resection and later radiotherapy proves the chronic and recurring nature of ACP, whereby close surveillance and treatment are crucial over a long period.

Notably, the case also shows the importance of molecular diagnosis, especially the detection of CTNNB1 mutations and immunohistochemical markers such as one beta-catenin, which not only serves to diagnose, but possibly to direct subsequent treatment. Moreover, it highlights the necessity of lifelong endocrine, ophthalmological, and cognitive support, so it is obvious that ACP is not a simple tumor that should be eliminated, but a chronic disease that demands constant, caring, and coherent care.

Finally, this case is a reminder that quality of life should be at the forefront of management. The experience of the child is one of careful oncologic management and functional preservation of well-being, which is a major dilemma in pediatric neuro-oncology.

4. Conclusion

The present case of recurrent adamantinomatous craniopharyngioma highlights the diagnostic and treatment challenges of a rare but significant pediatric brain tumor. It shows the necessity of early identification, the insufficiency of the existing treatment options, and the significance of long-term multidisciplinary follow-up. With further development of technology, it is hoped that molecular knowledge and precision therapy will eventually lower the recurrence rate and improve outcomes. With this, the management of ACP will shift to a proactive approach, focusing on personalized care and long-term quality of life.

Compliance with ethical standards

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Disclosure of conflict of interest

All authors make the following declarations:

- **Payment/services information:** All authors have declared that they received no financial support from any organization for the submitted work.
- **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 be interested in the submitted work.

Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

Statement of informed consent

Consent for publication of this case was obtained from the patient’s mother.

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