A giant-sized pilocytic astrocytoma mimicking a brain abscess: A case report and literature review

Jenty Parata[1,2] and Kenneth Sube[1,3,4]

Author Affiliation:

1. Department of Paediatrics and Child Health, School of Medicine, University of Juba, South Sudan
2. Al Sabah Children's Hospital, Juba, South Sudan
3. Departments of Biochemistry and Ophthalmology, School of Medicine, University of Juba, South Sudan
4. South University of Medicine, Science and Technology, Juba, South Sudan

Correspondence:
Jenty Parata
jentyparata@gmail.com

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ABSTRACT

Pilocytic astrocytoma (PA) is a rare but common low-grade glioma affecting children and young adults. It has a favourable prognosis and a high survival rate with appropriate management. Despite slow growth, PA often presents with significant symptoms due to its tendency to reach a large size, especially in paediatric patients, where compensatory mechanisms can delay detection. PA mimics a brain abscess. However, a brain abscess presents as an intracerebral infection localised to a specific area, which frequently develops into collections of pus surrounded by a well-demarcated, vascularised capsule. With advanced MRI imaging, PA tumours show free diffusion-weighted imaging with a high Apparent Diffusion Coefficient (ADC) value, while an abscess shows restricted diffusion-weighted imaging with a low ADC. This case report describes a six-year-old male child who presented with fever, focal seizures, and altered consciousness. CT scan results revealed a right parietal mass that was consistent with a PA and hydrocephalus. Even though this case report highlights the value of neuroimaging for PA diagnosis, the unique presentation displays challenges in managing PA, emphasizing the importance of timely intervention to optimize neurological outcomes.

Keywords: pilocytic astrocytoma, brain abscess, CT scan, paediatric, South Sudan

Introduction

Pilocytic astrocytoma (PA) most commonly occurs in paediatric patients. It is a type of glioma that accounts for 15.6% of central nervous system (CNS) tumours in children.[1] Even though PA can arise anywhere in the CNS, the most frequent sites in descending order are cerebellum, supratentorial compartment, optic pathway, hypothalamus, brainstem, and the spinal cord, with 42%, 36%, 9%, 9%, and 2%, respectively.[2] In children, the most affected site is the cerebellum (6%).[2] It is mainly found in children aged 5 to 14 years, with the highest incidence between 6 and 8 years, and is slightly more common in males.[2,3,4] The ten-year survival rate exceeds 85-95%.[1]

Neuroimaging (CT scan and MRI) plays a crucial role in diagnosis, treatment, and monitoring. CT scanning shows a hypodense cystic component, an isodense/hypodense nodule, enhancement after contrast, and possible calcification. MRI (T1) shows a cyst isodense with CSF and a nodule as iso- to hypodense to

grey matter. However, MRI (T2/FLAIR) shows a cyst as hyperdense (like CSF) and a nodular hyperdense. Post-contrast T1: Dense, homogeneous enhancement of the mural nodule (and sometimes cyst wall). Diffusion Weight Imaging (DWI) and Apparent Diffusion Coefficient (ADC) show a high signal (bright) on ADC maps (no restricted diffusion).

In contrast a brain abscess (BA) appears on CT scan as a rim-enhancing lesion with a central low-density (necrotic/fluid) area with extensive surrounding oedema. MRI (T1) demonstrates a hypodense central core with a capsule that may show an isodense signal. MRI (T2/FLAIR) shows a bright central core an isodense capsule and significant oedema. Post-contrast T1 reveals a thick, irregular, "ring-like" enhancement of the capsule. DWI/ADC: Restricted diffusion (dark) in the central core (pus), contrasting with the bright ADC signal of PA.[5] In resource-limited settings, CT scan can be used for screening purposes because it is fast, accessible, and detects hydrocephalus. On the other hand, MRI has high resolution but is expensive and can be used after screening.

Complete surgical removal is the gold standard treatment.[6] Total resection (TR) is usually the preferred approach, with a ten-year survival rate of about 95%,[7] but others use minimally invasive techniques and focus on hydrocephalus management.[8] However, craniotomy has challenges ranging from complex anatomy and proximity to vital structures: the cerebellum is adjacent to the brainstem and cranial nerves, and the risk of damage to these structures is very high. When the tumour is large, and part is left behind, the risk of recurrence increases. Postoperative neurological deficits include gait ataxia, sensory disturbances, cranial nerve palsies, dysphagia, Cerebellar Mutism Syndrome (Posterior Fossa Syndrome), hydrocephalus, and CSF leaks. There are also intraoperative limitations involving patient positioning, intraoperative imaging, a lack of a clear field of view, and an inability to accurately assess the extent of tumour infection and bleeding.[7,9,10]

Hydrocephalus can be managed with an external ventricular drain (EVD) insertion, ventriculoperitoneal shunt (VP shunt), or ventriculostomy.[11] Radiotherapy and chemotherapy should be considered for recurrent or progressive tumours involving the cerebellar peduncle or the brainstem. Patients may have permanent cerebellar dysfunction and cognitive disturbance after surgery.

Case report

A six-year-old male was referred from Yafa paediatric centre to Al Sabah Children's Hospital with a persistent high-grade fever of four weeks' duration. He was previously healthy with normal developmental milestones and had been fully immunised, including BCG for tuberculosis. The fever was temporarily relieved by diclofenac, associated with abnormal body movements characterized by sustained flexion of the right arm and numerous daily episodes lasting less than one minute over the previous three weeks. He became unconscious two days prior to admission and was unresponsive to voice and touch. He had no history of head trauma, vomiting, headache, family history of seizures, weight loss, night sweating, cough, upper respiratory or gastrointestinal symptoms, and no contact with a known TB patient. Moreover, there was no history of preceding upper respiratory and gastrointestinal symptoms.

On arrival, he was looking ill, in moderate respiratory distress (Respiratory Rate=34/min) with a Glasgow Coma Scale (GCS) of 6/15. The hospital does not have an Intensive Care Unit, and hence he was admitted to a high care unit where he was maintained on continuous oxygen until he was referred for neurosurgical evaluation in one of the private hospitals. The pupils were fixed, dilated, and unresponsive to light. Head circumference= 58cm (normal 53-55 cm), anterior fontanel 1cm x 1cm, and the posterior fontanel admitted the tip of a finger. He was pale and febrile (38^0 C). Laboratory results revealed mild normocytic normochromic anaemia with the haemagram of the followings: Hb 10.4g/dl, RBC 4.3x106/µL, HCT 32%, MCV 81fl, MCH 27 pg, MCHC 34g/dl, RDW 13%, platelets 340,000/µL, WBC 7200/µL with differential of: neutrophils 50%, lymphocytes 38%, monocytes 6%, basophils 0.6%, and eosinophils 2%, elevated erythrocyte sedimentation rate 95mm/hr, positive blood film for Plasmodium falciparum 4 parasites, per 100 HPF. Electrolytes, liver function tests, urea, and creatinine were normal. Chest X-ray was normal. GeneXpert was performed and was negative for TB.

A provisional diagnosis of hydrocephalus with coma secondary to cerebral malaria was made. Our differential diagnoses included a space-occupying lesion (e.g., tuberculoma, brain abscess, tumour). He was infused with a calculated dose of quinine, 20mg/kg as a loading dose, then 10mg/kg 8 hourly; an anti-meningitis dose of cefepime 50mg/kg 12 hourly, and vancomycin 20kg/kg 8 hourly; and dexamethasone 0.6 mg/kg in four divided doses. Due to the high ESR, he was given an anti-tuberculous regimen of isoniazid, rifampicin, ethambutol, and pyrazinamide at the dose of three tabs dissolved in clean water and administered via a nasogastric tube. However, there was no improvement.

A brain CT scan was requested on the tenth day since

admission, and this showed a left parietal cerebral hypodense, well-circumscribed lesion, demonstrating marginal enhancement measuring 6.5x5.4x4.1cm. There was compression and dilatation of the left lateral ventricle. The right cerebral hemisphere, cerebellum, and brainstem were normal, with no midline shift.

It was concluded that there was an obstructive hydrocephalus with a left parietal cerebral focal lesion, most likely an astrocytoma or abscess. However, based on the history, physical examination, and brain CT scan, we suspected a pilocytic astrocytoma. The patient was referred for neurosurgical assessment in one of the private hospitals in Juba. The parents were unable to afford the cost of the surgery, and hence, a definitive histological diagnosis was not possible. Unfortunately, we learnt that, three weeks later, our patient was operated upon in a private hospital and two days later died.

Discussion

Pilocytic astrocytoma (PA) is a slow-growing glioma classified by the World Health Organization (WHO) as a grade I tumour.[12] It is commonly found in children aged 5 to 14 years, with the highest incidence between 6 and 8 years, and slightly more males than females.[2,3,4] Our patient was a male aged six years.

A PA is slow-growing with insidious presenting features. The symptoms depend on the tumour's location.[1] Most PAs are diagnosed when they are large, compressing adjacent structures and increasing intracranial pressure.[1] Headache is the commonest symptom,[12] although our patient had not complained of a headache, bearing in mind the impaired consciousness.

He presented with fever and impaired consciousness. The fever appeared to be caused by malaria and subsided following quinine infusion, but the convulsions persisted. A cerebral abscess was considered, but there was no response to antibiotics, and the differential WBC was normal. Therefore, it was highly likely that the seizures and impaired consciousness were caused by the pressure effect of a PA on the surrounding tissues and the obstructive hydrocephalus. Neuroimaging plays a crucial role in the diagnosis and management of PA.[13] On CT scans PAs usually appear as round/oval lesions with well-defined iso- or slightly hypo-dense areas, markedly enhanced with contrast media.[13] On the other hand, magnetic resonance imaging (MRI) displays hypodense (darker than the surrounding tissues), isodense (the same brightness as the surrounding tissues), or hyperdense (whiter than the surrounding tissues), with clear or diffuse enhancement.[1,13] The masses may have cysts or "tumour nodules in a cyst," an appearance particularly seen in cerebellar and cerebral hemisphere tumours.[13] The CT scan of our patient showed the left parietal cerebral hemisphere with a hypodense area of a well-circumscribed lesion with marginal enhancement. The size of the PA in our case (6.2x5.4x4.1cm) was within the previously reported range (4–10cm).[14]

PAs occur most commonly in the cerebellum and regions around the third ventricle, followed by, the optic nerve and chiasm, hypothalamus, brainstem, thalamus, basal ganglia, and cerebral hemispheres.[1] Sixty-seven percent of childhood PAs arise in the cerebellum. In our case, it was in the left parietal cerebral lobe. Our patient had an obstructive hydrocephalus, which occurs in 90% of PA cases.[6] The histopathological characteristics show loose microcystic areas interspersed with compact, fibrillary regions. Rosenthal fibres and eosinophilic granular bodies are common.[15]

Surgical resection is the ideal approach for PAs. In cases that are inoperable because of a very high-risk location or the patient is unfit for surgery, alternate approaches like partial resection with adjuvant therapies, which include chemotherapy with carboplatin, vincristine, and temozolomide. Radiotherapy may also be considered.[16]

Conclusion

This case demonstrates the challenges in referral, diagnosis, and management of PA in low-resource settings such as South Sudan, and especially with fragile health systems.

A thorough clinical assessment and prompt referral to a tertiary children's hospital (Al Sabah Children's Hospital) improve outcomes. Greater financial support for the paediatric hospital is needed to facilitate the development of a comprehensive CT and MRI imaging unit. Furthermore, a paediatric surgical unit with a well-equipped theatre is needed since specialized human resources are available. There is also a need to establish a well-equipped paediatric ICU, complemented by capacity-building for staff at different levels, with appropriate specialists.

There is a great need to support family members in accessing expensive medical services while they wait for the government to create national health insurance.

Declarations: Written informed consent for publication was obtained from the patient's parent.

Competing interests: None.

Author's contributions: JS drafted the case report, literature review, and conclusion with recommendations,

while KS reviewed, expanded it, and edited. All reviewed the final manuscript and agreed on the final version.

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References

1. Nugraha HG, Yoasta E, Sobana M, Usman HA. Pilocytic astrocytoma: A rare case report. Radiol Case Rep. 2025 Feb 1;20(4):2175-2179. https://doi.org/10.1016/j.radcr.2025.01.025. PMID: 39975648; PMCID: PMC11835623.
2. Collins VP, Jones DTW, Giannini C. Pilocytic astrocytoma: pathology, molecular mechanisms and markers. Acta Neuropathol 2015;129(6):775–88 . https://doi.org/10.1007/s00401-015-1410-7.
3. Milde T, Rodriguez FJ, Barnholtz-Sloan JS, Patil N, Eberhart CG, Gutmann DH. Reimagining pilocytic astrocytomas in the context of pediatric low-grade gliomas. Neuro-Oncol 2021;23(10):1634–46. https://doi.org/10.1093/neuonc/noab138.
4. Tabash MA. Characteristics, survival and incidence rates and trends of pilocytic astrocytoma in children in the United States; SEER-based analysis. J Neurol Sci 2019;400:148–52. https://doi.org/10.1016/j.jns.2019.03.028. Epub 2019 Mar 29.
5. Resende LL, Alves CAPF. Imaging of brain tumors in children: the basics-a narrative review. Transl Pediatr. 2021 Apr;10(4):1138-1168. doi: https://doi.org/10.21037/tp-20-285. PMID: 34012860; PMCID: PMC8107871.
6. Bonfield CM, Steinbok P. Pediatric cerebellar astrocytoma: a review. Childs Nerv Syst 2015;31(10):1677–85. https://doi.org/10.1007/s00381-015-2719-1.
7. Knight J, Karsonovich T, De Jesus O. Pilocytic Astrocytoma. [Updated 2024 Aug 7]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK560614/.
8. Core techniques in operative neurosurgery (2nd edition). 2020, pages 21- 24 https://doi.org/10.1016/B978-0-323-52381-3.00005-8.
9. Santos AN, Dieckmann C, Rauschenbach L, Oppong MD, Dinger TF, Deuschl C, Tippelt S, Fleischhack G, Schmidt B, Pierscianek D, Jabbarli R, Wrede KH, Müller O, Sure U, Dammann P. Long-term outcome after management of pilocytic astrocytoma in the posterior fossa in a pediatric population. IBRO Neurosci Rep. 2022 Oct 7;13:388-392. doi: https://doi.org/10.1016/j.ibneur.2022.10.001. PMID: 36340442; PMCID: PMC9630615.
10. Ruella M, Giovannini S, Christian Pirozzi Chiusa CP et al. Cerebellar pilocytic astrocytoma. Retrospective cohort study assessing postoperative functional outcome, cerebellar mutism and hydrocephalus, World Neurosurgery: X, Volume 19, 2023,100180, ISSN 2590-139 https://doi.org/10.1016/j.wnsx.2023.100180.
11. Ronsley R, Bouffet E, Dirks P, Drake J, Kulkarni A, Bartels U. Successful management of symptomatic hydrocephalus using a temporary external ventricular drain with or without endoscopic third ventriculostomy in pediatric patients with germinoma. Journal of Neurosurgery 2022; 137(3), 807-812. https://doi.org/10.3171/2021.8.JNS211443.
12. Bell D, Chitnavis BP, Al-Sarraj S, Connor S, Sharr MM, Gullan RW. Pilocytic astrocytoma of the adult--clinical features, radiological features and management. Br J Neurosurg. 2004 Dec;18(6):613-6. https://doi.org/10.1080/02688690400022896. PMID: 15799194.
13. Nikam RM, Yue X, Kaur G, Kandula V, Khair A, Kecskemethy HH, Averill LW, Langhans SA. Advanced Neuroimaging Approaches to Pediatric Brain Tumors. Cancers. 2022; 14(14):3401. https://doi.org/10.3390/cancers14143401.
14. Oktama V, Abdurrahman M, Thoriq A, Rais F, Syekh H. Giant Size Pilocytic Astrocytoma in Pediatric Patient: A Case Report. Asian Australasian Neuro and Health Science Journal (AANHS-J) 2022; 4(2). https://doi.org/10.32734/aanhsj.v4i2.8937.
15. Bhangale P, Kashikar S, Parihar PH, Shinde D, Kumari A, Nimodia D. Chronic headache and obstructive hydrocephalus: A case of pilocytic astrocytoma in the third ventricle with detailed radiological and histopathological findings. Radiol Case Rep. 2025 Jan 25;20(4):1993-1997. https://doi.org/10.1016/j.radcr.2024.12.058. PMID: 39926270; PMCID: PMC11802356.
16. Muhsen BA, Aljariri AI, Elayyan M, Hirbawi H, Masri MA. Insight about the characteristics and surgical resectability of adult pilocytic astrocytoma: tertiary center experience. CNS Oncol. 2022 Apr 6;11(1):CNS81. https://doi.org/10.2217/cns-2021-0014. Epub ahead of print. PMID: 35382555; PMCID: PMC8988253.