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Spontaneous Rupture of a Fourth Ventricular Mature Teratoma Presenting with Aseptic Meningitis in a 10-Year-Old: A Rare Case from a Rural Kenyan Hospital

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Abstract

Background: Mature teratomas of the central nervous system are rare congenital germ cell tumours, accounting for <0.5% of all intracranial neoplasms. Spontaneous rupture with dissemination of fatty material into the ventricles and subarachnoid spaces is uncommon, particularly from the fourth ventricle, and may precipitate aseptic meningitis, complicating diagnosis and management. **Case:** We report a 10-year-old girl with progressively worsening headaches, recurrent vomiting, and new-onset generalised tonic–clonic seizures. Neurological examination showed mild truncal ataxia without focal motor deficits. Cranial computed tomography (CT) demonstrated a heterogeneous fourth ventricular mass with fat–fluid levels and fat droplets layering the ventricular system and basal cisterns. **Intervention:** The patient underwent telovelar microsurgical gross total resection (GTR). The procedure was uneventful, and early postoperative contrast-enhanced CT confirmed complete excision with patent cerebrospinal fluid pathways. No adjuvant chemotherapy or radiotherapy was given, as histopathology confirmed a mature teratoma. **Outcome:** The postoperative course was uncomplicated. At 3- and 6-month follow-up, the child remained neurologically intact and free of headaches, seizures, and other symptoms, with no radiological evidence of residual or recurrent tumour. Long-term follow-up with serial clinical and imaging assessments is planned for at least three years to monitor for recurrence or delayed complications. **Conclusion:** Telovelar gross total resection alone may achieve excellent outcomes in ruptured fourth ventricular mature teratomas. This case, to our knowledge the first report of spontaneous rupture of such a lesion from a rural African neurosurgical centre, underscores the growing capacity for complex neurosurgical care in resource-limited settings.

Keywords: Intracranial teratoma; Fourth ventricle; Spontaneous rupture; Aseptic meningitis; Paediatric neurosurgery; Rural Africa

INTRODUCTION

Intracranial teratomas are rare central nervous system tumours, histologically classified into three subtypes: mature (generally benign) teratoma, immature teratoma, and teratoma with somatic-type malignancy. They account for approximately 0.5% of primary intracranial neoplasms (1–3). Mature lesions comprise well-differentiated tissues derived from all three germ layers (ectoderm, mesoderm, endoderm), whereas immature forms contain foetal or embryonal tissue from the same layers. Most reported intracranial teratomas are congenital, often defined as arising within the first 60 days of life (1,4,5). These tumours typically occur along midline structures—the pineal region, suprasellar cistern, basal ganglia, thalamus, and third ventricle—and present predominantly in infancy or childhood, with a male predominance reported in several series (3,4,17).

Spontaneous rupture is exceptionally rare. When lipid-rich contents escape into the subarachnoid or ventricular spaces, chemical (aseptic) meningitis may ensue (6,8). Documented ruptures most often involve supratentorial or pineal lesions; fourth-ventricular involvement is very uncommon (pineal > suprasellar ≫ fourth ventricle) (7,17). On non-contrast CT, ruptured teratomas may show intraventricular hydrocephalus and characteristic fat–fluid levels, with fat demonstrating negative attenuation on the Hounsfield scale (6,10).

We report a spontaneously ruptured fourth-ventricular mature teratoma in a child, highlighting CT detection of intrathecal free fat and a telovelar resection approach relevant to resource-limited settings.

Case Presentation:

A ten-year-old girl presented to the emergency department in a post-ictal state following a generalised tonic–clonic seizure. Her mother reported a six-month history of progressive gait unsteadiness, global headaches, and recurrent seizures. The seizures were predominantly generalised tonic–clonic, lasted <1 minute, and were preceded by dizziness and malaise. Each episode began with a brief scream, followed by eye-rolling, urinary incontinence, loss of consciousness, tonic stiffening progressing to clonic jerking of all limbs, and a post-ictal somnolent phase of 20–30 minutes. There was no history of focal onset, tongue biting, or trauma. Two days before admission she developed worsening headaches, neck pain, and non-projectile post-prandial vomiting. One week earlier, the headaches had acutely intensified, with recurrent vomiting and reduced oral intake. There was no fever, photophobia, or reported meningeal signs. Empirical antimalarial therapy at a peripheral facility was ineffective. The family reported unsupervised use of over-the-counter anticonvulsants, although documentation was unavailable. She also complained of urinary frequency, occasional frank haematuria, and intermittent left flank pain.

On examination: temperature 36.7 °C, pulse 87 beats/min, blood pressure 110/73 mmHg, respiratory rate 16 breaths/min, oxygen saturation 98% on room air. After recovering from the seizure, her Glasgow Coma Scale (GCS) was 15. Neurological examination showed truncal ataxia and an unsteady gait. Cranial nerves were intact, with no long-tract signs. Fundoscopy was not performed at presentation, so papilloedema could not be confirmed or excluded. There were no signs of meningeal irritation. She was stabilised and commenced on levetiracetam (loading 40 mg/kg, then 10 mg/kg twice daily, weight 29 kg).

Investigations

At presentation, the complete blood count showed marked leucocytosis (29.83 × 10^9/L) with 90% neutrophils; other haematological parameters were normal. Serum biochemistry revealed an isolated elevation of random blood glucose (13.0 mmol/L), with normal renal and liver function. Initial urinalysis demonstrated bacteriuria (+1), white cells 3–4/HPF, glucosuria (2+), and ketonuria (2+). Serum ketones were not measured. HbA1c and arterial blood gas were normal. A malaria rapid test was negative (**Table 1**).

The paediatric team admitted the patient for further evaluation and management. Intravenous levetiracetam was continued for seizure control, and empiric intravenous ceftriaxone was started in view of the abnormal urinalysis. An urgent contrast-enhanced CT brain was planned prior to lumbar puncture. Cerebrospinal fluid (CSF) was clear and colourless: white cells 2/mm^3 (100% lymphocytes), red cells 0/mm^3, and protein 74.6 mg/dL; glucose was not reported due to technical issues (**Table 1**). The opening pressure at the time of external ventricular drain (EVD) placement was not documented, but CSF egress was noted to be under high pressure.

Repeat urinalysis the following day showed worsening urinary tract infection (UTI) features—white cells 15–20/HPF, bacteria 3+, leucocyte esterase 3+—with resolution of the earlier glucosuria and ketonuria (**Table 1**). Urine culture grew *Escherichia coli*, sensitive only to meropenem from the tested panel (ceftriaxone not tested). Antibiotics were switched from ceftriaxone (given empirically for two days) to meropenem for a further five days, after which urinalysis confirmed clearance.

Non-contrast and contrast-enhanced CT demonstrated marked ventriculomegaly with pan-ventricular fat–fluid levels (approximately −110 to −160 Hounsfield units on soft-tissue window), consistent with lipid content (**Figure 2**). Hypodense fat was also seen within the subarachnoid space. A well-defined fourth-ventricular lesion (3.9 × 3.9 × 5.2 cm) contained solid and lipid-rich components with focal high attenuation suggestive of

haemorrhage or calcification. Mild central post-contrast enhancement was present. The overall appearance was highly suggestive of a mature teratoma (**Figures 1–2**). MRI was not available in-house, and the family could not afford referral imaging. CT evidence of a posterior fossa mass obstructing the aqueduct, with partial patency of the aqueduct and foramen of Magendie, supported a telovelar surgical approach to access the fourth ventricle while minimising cerebellar retraction.

Table 1: Summary of patient's laboratory investigations

Category	Investigation	Result	Reference Range	Interpretation
Complete Blood Count (CBC)	White Blood Cell Count	29.83 × 10^9/L	4.0–10.0 × 10^9/L	**High**
	Neutrophils	90%	40–75%	**High**
	Haemoglobin	12.4 g/dL	11.5–15.5 g/dL	Normal
	Platelets	356 × 10^9/L	150–400 × 10^9/L	Normal
Cerebrospinal Fluid (CSF)	Appearance	Clear	–	Normal
	Protein	74.6 mg/dL	15–45 mg/dL	**High**
	Glucose	Not reported	2.5–4.5 mmol/L	-
	WBC	2	<5 cells/µL	Normal
	Culture	No growth	–	Normal
Repeated Urinalysis	Bacteriuria	3+	Absent	**Abnormal**
	White Blood Cells	15-20 /HPF	0–5 /HPF	**High**
	Ketones	Absent	Absent	Normal
	glucose	Absent	Absent	Normal
	Other parameters	Negative	–	Normal

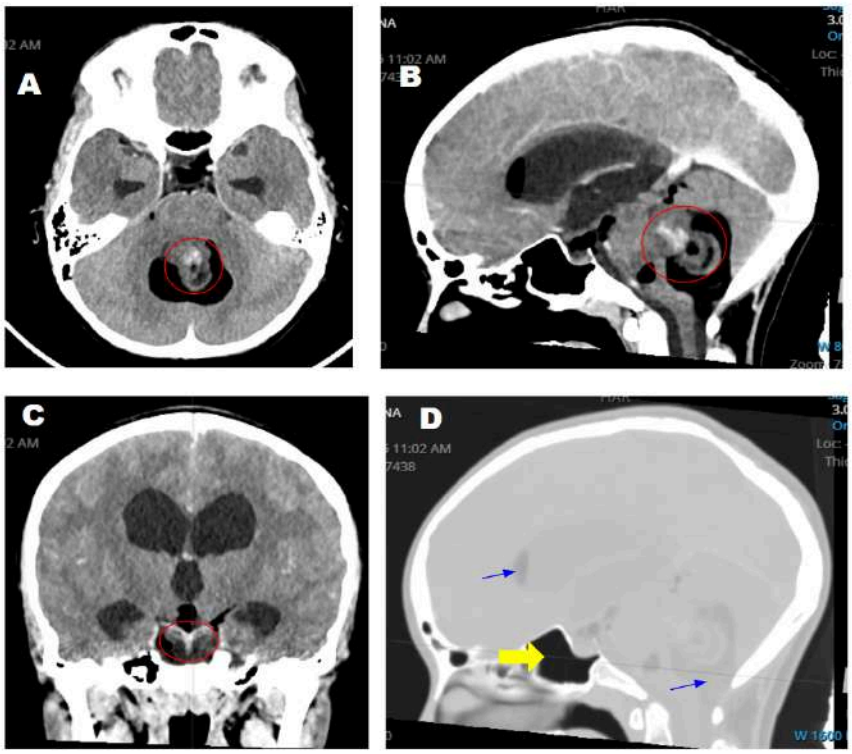


Figure 1: *Pre-operative CT shows a posterior fossa mass (Fig. 1A–C, red circle) in axial, sagittal, and coronal planes, obstructing the aqueduct of Sylvius with resultant obstructive hydrocephalus. A centrally enhancing core is surrounded by hypodense components, with similar density material layering the lateral ventricles, brainstem, and sellar region, suggestive of dissemination. A windowed image (Fig. 1D) highlights the increased density of the tumour and intraventricular seeding (blue arrow) relative to the air-filled paranasal sinuses (yellow arrow).*

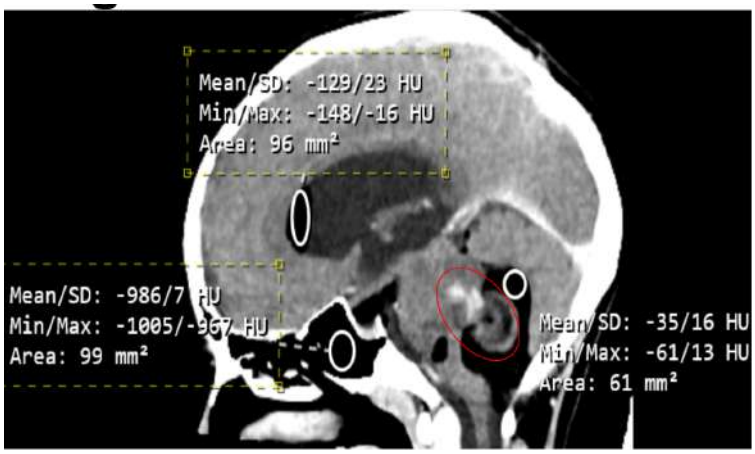


Figure 2: *Midline sagittal post-contrast CT (brain window) demonstrating the solid tumour component (red circle) with mixed attenuation. Areas of fat attenuation are visible both peritumourally and within the frontal horn (approximately −110 to −160 HU; occasionally up to −30 HU depending on composition). Hounsfield units in the sphenoid sinus are shown for comparison.*

Management

Following contrast-enhanced CT confirming obstructive hydrocephalus secondary to a fourth-ventricular lesion, the neurosurgical team placed an external ventricular drain to temporise raised intracranial pressure, alongside concurrent treatment for UTI. Available EVD systems lacked an integrated transducer, making precise height/pressure settings unreliable and increasing the risk of over-drainage through inadvertent adjustment. To mitigate this, controlled intermittent drainage (20 mL every four hours) was employed with close neurological and output monitoring. The opening pressure was within normal limits, and serial assessments over seven days showed stable intracranial dynamics. Fat globules with a glistening appearance were observed in the EVD drip chamber, consistent with lipidaceous CSF after tumour rupture (**Figure 3**).

Definitive surgery was undertaken via a telovelar approach, achieving gross total excision of the fourth-ventricular mass. Intra-operatively, the aqueduct of Sylvius was patent, confirming relief of obstruction. Pre-operative CT did not show convincing parenchymal or leptomeningeal inflammation; scheduled corticosteroids were therefore not indicated. The patient received routine peri-operative dexamethasone (2 mg twice daily for 24 hours, then stopped). Post-operatively, she tolerated an EVD clamp trial without recurrent hydrocephalus, allowing EVD removal on day 2.

Operation(Surgical description)

Under general anaesthesia with endotracheal intubation, the patient was positioned prone in the Concorde position with meticulous padding. The head was secured in a three-point Mayfield clamp, with the occiput positioned uppermost to facilitate venous drainage. WHO surgical safety protocols were observed. Neurophysiological monitoring was not available.

A midline suboccipital incision was made from the inion to the arch of C2. Layered dissection through the avascular midline raphe exposed the suboccipital region and posterior arch of C1. A C1 laminectomy improved inferior exposure. Two burr holes were fashioned adjacent to the midline, and a suboccipital craniotomy was completed, extending to the foramen magnum. A Y-shaped durotomy exposed the inferior cerebellar hemispheres, tonsils, and proximal cervical cord. The arachnoid over the cisterna magna was opened. The tonsils were gently separated along the tonsillomedullary fissure to establish the telovelar corridor, providing a view of the floor of the fourth ventricle. The inferior medullary velum was incised and the uvulotonsillar plane opened, allowing full visualisation of the ventricular cavity.

A yellowish fatty lesion was identified, extending through a markedly dilated obex into the fourth ventricle (**Figure 4**). On opening the arachnoid, the fatty component was carefully aspirated under low suction to decompress the lesion. A firmer, nodular portion within the fourth ventricle was attached by a narrow ventral stalk to the ventricular floor. The stalk was coagulated with bipolar cautery and divided. Circumferential microdissection enabled mobilisation and en bloc delivery of the tumour through the telovelar corridor.

Inspection with a surgical mirror confirmed complete excision, with no visible residual tumour and a patent, freely flowing aqueduct. Small fatty

fragments within the ventricle and cisterna magna were irrigated and aspirated until CSF was clear. Haemostasis was achieved with meticulous bipolar coagulation. Estimated blood loss was ~40 mL, with no intra-operative complications.

The dura was closed watertight, augmented with an autologous pericranial graft. The wound was closed in layers (2/0 Vicryl for muscle/fascia; 3/0 Vicryl for subcutaneous tissue; subcuticular 3/0 Monocryl for skin). The patient was extubated and transferred in stable condition to recovery.

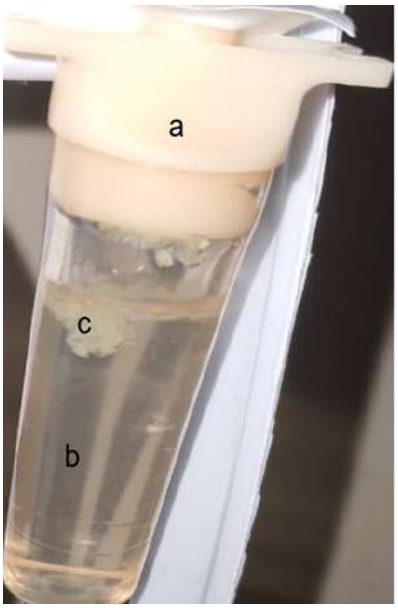


Figure 3. *Fat globules floating within CSF in the EVD drip chamber: (a) EVD chamber, (b) CSF column, (c) suspended fat globules.*



Figure 4: Intra-operative pre-resection view showing the lesion between the cerebellar hemispheres (a), the tumour (b) centrally, and reflected dura (c) superiorly and inferiorly.

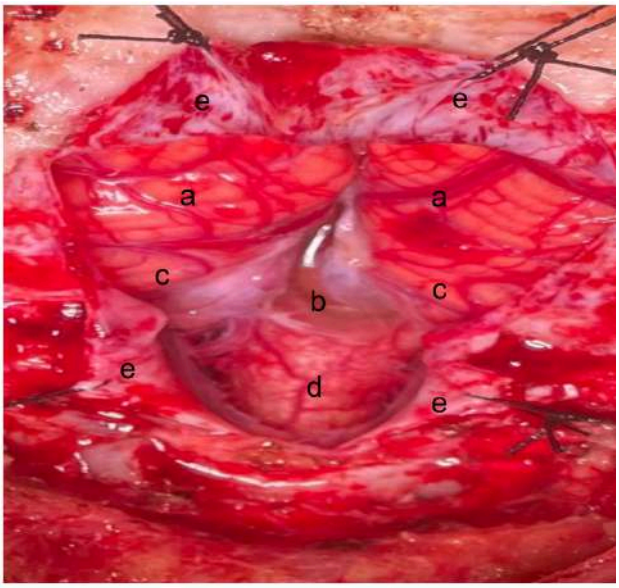


Figure 5. *Post-resection view showing the cerebellar hemispheres (a) flanking the resection cavity (b); cerebellar tonsils (c) inferiorly; medulla oblongata (d) deep in the field; and reflected dura (e) superiorly and inferiorly.*

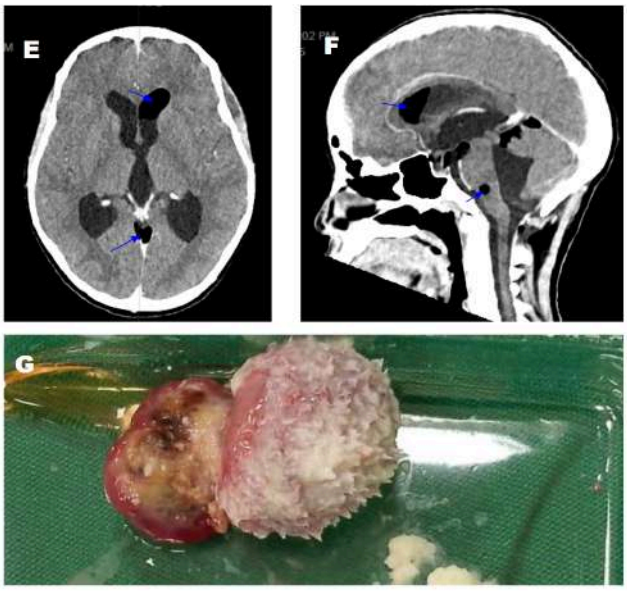


Figure 6. *Post-operative contrast-enhanced CT demonstrates complete resection, interval expansion of the fourth ventricle, and no residual enhancing lesion. Foci of fat density (blue arrows) persist within the ventricular system, medulla, and suprasellar region, consistent with disseminated fat droplets after pre-operative rupture. Image G shows the excised specimen.*

Histopathology:

Tissue biopsy was submitted for assessment. Grossly, the specimen was a firm tan-brown mass measuring 2.5 × 1.5 × 1 cm with an irregular surface; the cut surface was heterogeneous and glistening, with alternating brown and white areas. The tissue was entirely submitted in four blocks. Microscopically, a mature teratomatous lesion was seen, comprising benign keratinising squamous epithelium, dermal adnexal structures, mucin-producing glands, smooth muscle, mature adipose tissue, and cartilage (**Figures 7–9**). No immature or malignant elements were identified. Brain parenchyma was absent, consistent with a fully differentiated mature teratoma. Immunohistochemistry was not performed, as routine H&E was diagnostic.

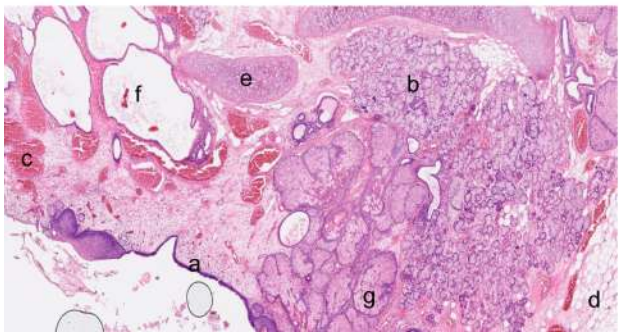


Figure 7. *Low-power (×10) photomicrograph showing stratified squamous epidermis (a) over dermal adnexal structures, with mature adipocytes (b), intravascular blood cells (c), connective-tissue stroma (d), cartilage (e), cystic/glandular spaces (f), and sebaceous glands (g); features of a mature teratoma.*

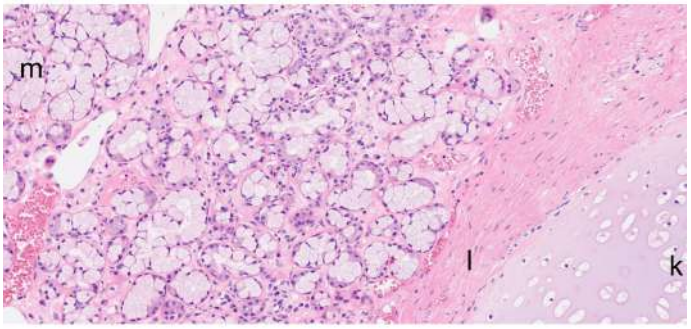


Figure 8. *Photomicrograph (×100) demonstrating mature cartilage (k), fibrous septa (l), and adjacent adipose tissue (m).*

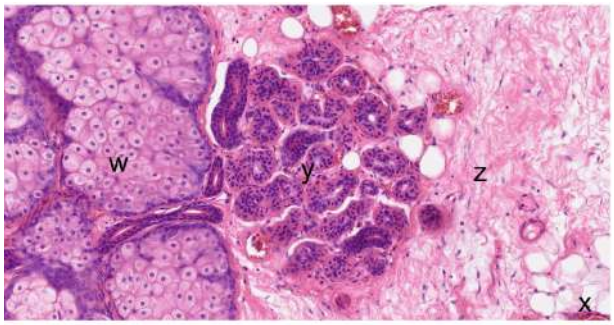


Figure 9. *High-power (×400) photomicrograph showing sebaceous glands (w), adipocytes (x), ductal/basaloid epithelial cells (y), and intervening stroma (z).*

Outcomes & follow-up

Non-contrast and contrast-enhanced head CT on postoperative day (POD) 2 confirmed gross total resection with resolving hydrocephalus (Figure 6). After a successful 48-hour EVD clamp trial, the drain was removed. At discharge (POD 3) the Modified Rankin Scale (mRS) score was 1 (minor symptoms). At three months the mRS was 0 (full recovery). There was no seizure recurrence; levetiracetam is being tapered with discontinuation planned at 12–24 months post-operatively if seizure-free. Recovery has been uneventful. At six months she remains clinically well and is scheduled for repeat contrast-enhanced CT to assess the tumour bed and exclude hydrocephalus. Surveillance imaging is planned at one and three years, or earlier if symptoms recur.

DISCUSSION

Diagnostic considerations

Intracranial mature teratomas are radiologically heterogeneous owing to their mixed tissue constituents—fat, cartilage, calcification, and cystic or solid elements (1,2). Neuroimaging is essential to define lesion composition and guide operative strategy. In our patient, non-contrast CT showed low-attenuation (lipid) areas with fat–fluid levels within the ventricles and subarachnoid spaces; the fat component measured approximately −142 HU, consistent with lipid density (10). Mature teratomas most often arise at midline locations (pineal and suprasellar regions); fourth-ventricular involvement is exceptionally rare. Rupture or leakage of cyst contents can precipitate aseptic meningeal irritation, a mechanism concordant with this presentation (13). Free fat droplets in the

ventricular system and cisterns, while not pathognomonic, are highly suggestive of tumour rupture in the appropriate clinical context (6,8). Reported attenuation values in ruptured teratomas range from −110 to −160 HU (8). The visible fat droplets in the external ventricular drain (EVD) chamber provided corroborative evidence of lipid-laden CSF—an especially practical clue where MRI is unavailable.

Mechanisms of rupture and pathogenesis

Spontaneous rupture of mature teratomas is rare. Proposed mechanisms include progressive weakening of the capsule from intracranial pulsations, raised intracystic pressure, or degenerative change (6,8). Hormonal influences during growth may increase cystic pressure and precipitate rupture, although this remains speculative. Our patient's age may coincide with such physiological changes, but causality cannot be inferred from a single case. Emerging molecular data cite occasional germline or somatic variants (e.g., *CARD11*, *IRS1*) in germ-cell tumours; their relevance to intracranial mature teratomas is uncertain and not demonstrable here (12).

Management and surgical nuances

Gross total resection (GTR) is the cornerstone of treatment for histologically mature teratomas, given their benign nature and limited responsiveness to adjuvant therapy (2,9,16). In this case, histopathology confirmed a purely mature teratoma with no immature or malignant elements. Serum and CSF tumour markers (AFP, β-HCG) were not obtained; importantly, β-HCG has low diagnostic sensitivity in non-germinomatous lesions and may be normal in mature teratomas, so absent markers cannot reliably exclude a teratomatous pathology (11). Accordingly, no adjuvant chemotherapy or radiotherapy was indicated. A telovelar microsurgical approach via the uvulotonsillar and tonsillomedullary fissures was used, enabling exposure of the fourth ventricle while preserving the vermis—an approach supported in paediatric neurosurgical literature for midline posterior fossa lesions with reduced risk of postoperative cerebellar dysfunction or mutism (16,17).

Postoperative care, chemical meningitis, and steroid use

Fat dissemination into CSF spaces may provoke a sterile inflammatory reaction causing chemical meningitis (6,14). In our case, postoperative CSF protein was mildly elevated (74.6 mg/dL), consistent with an inflammatory response (18). Peri-operative dexamethasone (2 mg twice daily for 24 hours) was given to mitigate inflammation; no taper was needed as there were no signs of persistent meningitis. This aligns with reports recommending short-course corticosteroids for lipid-induced irritation, though higher doses with taper are often described (7). Aseptic meningitis is well recognised following teratoma rupture, with lipid dissemination triggering delayed fever, meningitic symptoms, and CSF pleocytosis despite sterile cultures—underscoring the need for proactive monitoring even after complete excision (7).

Prognosis and follow-up

When histology confirms maturity and complete excision is achieved, prognosis is excellent (2,3,9). Nonetheless, microscopic remnants may persist, with potential for recurrence or delayed obstructive hydrocephalus. We therefore advocate interval imaging—ideally MRI, or contrast-enhanced CT where feasible at one and three years postoperatively.

Relevance to resource-limited settings

This case illustrates how careful CT interpretation (including HU quantification) and intra-operative clues such as lipids in the EVD chamber can compensate for limited MRI access. Pragmatic short-course steroid use likewise reflects rational resource utilisation. Such context-specific adaptations are important for safe neurosurgical care in low-resource environments.

Novelty and literature context

Following a search of PubMed and African neurosurgical archives (to 2025), we found no prior report of spontaneous rupture of a fourth-ventricular mature teratoma from a rural African centre. This case therefore contributes a distinctive perspective from a low-resource setting. Most previously published African reports originate from large urban referral hospitals with readier access to MRI, neuroradiology, and comprehensive histopathology (16). In contrast, this patient was managed in a rural tertiary hospital with limited subspecialty resources, underscoring both the diagnostic challenge and the significance of achieving GTR in a constrained environment. The rural–urban distinction is clinically relevant, highlighting disparities in access to advanced neuroimaging and specialist care that may delay diagnosis or complicate management of rare intracranial pathologies (12,15,16). While ruptured fourth-ventricular teratomas have been described elsewhere, we identified no prior published African case documenting this specific presentation (16). In summary, this case refines understanding of ruptured mature teratomas in late childhood,

emphasises the diagnostic utility of CT attenuation and intra-operative findings in MRI-limited contexts, and reaffirms that GTR alone can be curative for histologically mature lesions.

CONCLUSION

Spontaneous rupture of a fourth-ventricular mature teratoma with intrathecal fat dissemination is exceptionally rare in the paediatric population. This case reinforces consideration of ruptured intracranial teratoma in paediatric aseptic meningitis when neuroimaging shows intraventricular fat–fluid levels. In settings where MRI is not readily available, meticulous CT assessment—including recognition of fat–fluid levels and fat layering in the ventricles and basal cisterns—provides crucial diagnostic clues. Oily material in an EVD chamber may further support early suspicion. Gross total resection remains the mainstay of treatment, offering excellent long-term outcomes without adjuvant therapy in mature lesions. The telovelar approach affords safe access to fourth-ventricular tumours with minimal cerebellar disruption. Judicious postoperative corticosteroid use can mitigate inflammatory responses due to chemical meningitis after fat spillage, while avoiding unnecessary prolonged courses. Given the potential for recurrence or delayed complications such as hydrocephalus from residual fat, long-term surveillance with serial imaging is essential. To our knowledge, this is the first documented instance of a spontaneously ruptured fourth-ventricular mature teratoma in rural Africa, highlighting the value of prompt recognition, context-adapted diagnostic acumen, and meticulous microsurgical technique to achieve favourable outcomes in resource-constrained environments.

Ethics approval and consent to participate: Ethical approval was waived by the Institutional Review Board for this single case report.

Consent for publication: Verbal informed consent for publication, including clinical details and anonymised images, was obtained from the patient's parents.

Competing interests: The authors declare that they have no competing interests.

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Availability of data and materials: All data supporting the findings of this report are contained within the manuscript.

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