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Case Report

Germinoma of the Pineal region in a Paediatric Immunocompromised Host: A Case Report

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ABSTRACT:

Pineal region tumours constitute 0.4–1% of all intracranial neoplasms, with germinomas being the most common subtype, predominantly affecting adolescent males. Their occurrence in young children with concurrent immunocompromise is exceptionally rare and diagnostically challenging.

A 9-year-old HIV-positive male on antiretroviral therapy (ART) presented with a two-month history of intermittent holocranial headache. Neuroimaging revealed a 2.5 × 3.1 × 3.2 cm heterogeneous pineal region mass with obstructive hydrocephalus. FDG-PET/CT demonstrated intense focal uptake (SUVmax 24.2) without systemic metastatic disease. Cerebrospinal fluid (CSF) analysis showed lymphocytic pleocytosis with markedly elevated lactate dehydrogenase (LDH: 77 IU/L). Histopathological examination of biopsy tissue revealed large polyhedral cells with prominent nucleoli arranged in sheets, associated with dense fibrous stromal lymphocytosis. Immunohistochemistry (IHC) confirmed positivity for c-KIT (CD117) and podoplanin (D2-40), establishing a final diagnosis of intracranial/pineal germinoma.

This case highlights the diagnostic complexity of pineal germinoma in an immunocompromised paediatric patient, where overlapping pathologies must be carefully excluded. Integration of advanced neuroimaging, CSF biochemistry, histopathology, and IHC is essential for definitive diagnosis. The radiosensitive nature of germinoma, if confirmed on molecular staging, offers a favourable prognosis with appropriate chemoradiotherapy.

Keywords: Germinoma; HIV; Pineal gland-tumor, Pediatric-oncology

INTRODUCTION

Pineal region tumours are rare, constituting approximately 0.4–1% of all intracranial neoplasms in Western populations and 2–8% in Asian populations.[1] Among these, germ cell tumours (GCTs) are the most common, with germinomas accounting for nearly 65% of all pineal GCTs.[2] They show a striking male predominance and typically present in the second decade of life. Clinical manifestations are primarily related to obstructive hydrocephalus due to aqueductal compression and Parinaud syndrome from tectal plate involvement.[3]

Diagnosis of pineal germinoma requires a multi-modal team-work approach encompassing clinical

evaluation, neuroimaging, CSF tumour markers (α-fetoprotein and β-human chorionic gonadotropin), and histopathological confirmation.

Immunohistochemistry plays a pivotal role in characterising germ cell lineage, with c-KIT (CD117) and podoplanin (D2-40) being hallmark markers of germinomas.[4]

The co-occurrence of pineal germinoma with paediatric HIV infection is “exceedingly rare’ in the published literature. Immunocompromise introduces additional diagnostic challenges, as opportunistic CNS

infections, primary CNS lymphoma, and immune reconstitution inflammatory syndrome (IRIS) can mimic or co-exist with primary brain tumours.[5]

We present a case of pineal germinoma in a 9-year-old HIV-positive boy, with a detailed clinico-pathological correlation and discussion of differential diagnoses.

CASE HISTORY

A 9-year-old male child was brought to the Neurosurgery outpatient department with a two-month history of headache. The headache was described as intermittent, holocranial, and moderate in intensity, without associated vomiting, seizures, loss of consciousness, fever, giddiness, slurring of speech, or ear-nose-throat bleeding. The child had a known history of hypertension (diagnosed one month prior) and HIV positivity on antiretroviral therapy (ART) for four months.

On examination, the patient was conscious and oriented. Glasgow Coma Scale (GCS) was 15/15 (E4V5M6). Vital signs were stable: temperature 37°C, pulse rate 80 beats/min, blood pressure 120/80 mmHg. Pupils were bilaterally equal, 2 mm, and reactive to light.

Cardiovascular, respiratory, and abdominal systemic examinations were unremarkable. Neurological examination did not reveal any focal deficits or signs of cerebellar dysfunction.

Haematological investigations revealed haemoglobin of 16 g/dL, total leucocyte count of 12,700/mm³, and platelet count of 2,76,000/mm³. Prothrombin time and INR were 12 seconds and 1.0 respectively. Random blood sugar was 84 mg/dL. Renal and liver function tests were within normal limits.

CSF analysis performed on 03 January 2026 demonstrated clear, colourless fluid with a total leucocyte count of 20/mm³ (100% lymphocytes), RBC 0–3 per high-power field, protein 23.1 mg/dL (normal 15–40 mg/dL), glucose 72 mg/dL, and LDH 77 IU/L (notably elevated; normal <40 IU/L). Microbiological investigations including Ziehl–Neelsen (ZN) stain for acid-fast bacilli and Gram stain were negative; 48-hour culture was sterile.

The elevated CSF LDH with lymphocytic pleocytosis in this immunocompromised host raised the suspicion of high metabolic tumour activity.

CT Brain demonstrated burr hole defects in the right frontal bone indicative of prior surgical access. An ill-defined hypodensity was noted in the right frontal lobe tracking to the frontal horn. A midline, heterogeneously hyperdense, lobulated mass measuring approximately 2.5 × 3.1 × 3.2 cm was identified in the pineal region, with engulfment of pineal calcifications and areas of internal necrosis (Figure 1). The mass exerted significant mass effect on the cerebral aqueduct resulting in obstructive hydrocephalus. Evans Index was 0.30, indicating ventriculomegaly with periventricular ooze (Fig. 1).

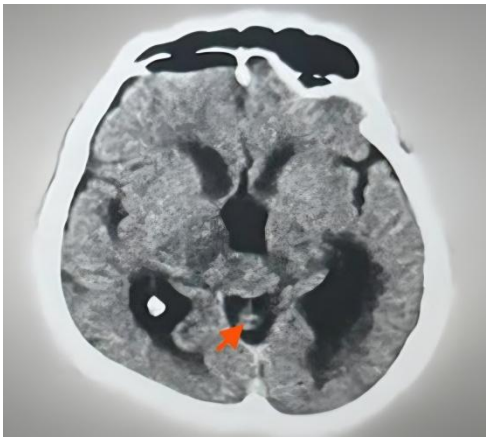

Figure-1: CT brain - Radiological coronal section showing a midline, heterogeneously hyperdense, lobulated mass measuring approximately 2.5 × 3.1 × 3.2 cm in the pineal region (*red arrow*),

MRI Brain demonstrated a well-defined mixed solid-cystic lesion measuring 2.2 × 2.8 × 2.7 cm epicentred in the pineal region. The lesion was heterogeneously hypointense on T2/FLAIR sequences and hypointense on T1-weighted images. Diffusion-weighted imaging (DWI) revealed foci of restricted diffusion. Susceptibility-weighted imaging (SWI) demonstrated no evidence of blooming artefact. Post-contrast sequences showed heterogeneous enhancement with a central non-enhancing cystic core. Mild peri-lesional oedema was observed involving the bilateral thalami, midbrain, and superior cerebellar peduncles. The lesion was noted to abut the tectal plate of the midbrain.

Whole-body FDG PET-CT demonstrated an intensely FDG-avid, heterogeneously enhancing mass in the pineal region extending posteriorly to involve the splenium of the corpus callosum, measuring 30 × 29 × 31 mm with SUVmax 24.2 (Figure 2). The lesion was

observed to abut the bilateral thalami and midbrain with suspicious infiltration, causing mass effect on the cerebral aqueduct with resultant hydrocephalus. Post-operative burr hole changes were identified in the right frontal bone, with a linear hypodensity extending from the right frontal lobe to the lateral ventricle showing no FDG uptake, consistent with a surgical tract. No significant FDG-avid cervical or supraclavicular lymphadenopathy was identified. Physiological FDG uptake was noted throughout the remainder of the body with no evidence of extracranial metastatic disease, establishing a localized staging (Fig. 2).

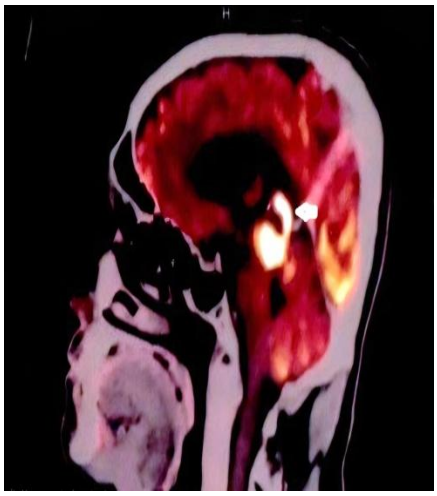

Figure-2: PET-CT brain's sagittal section reveals intensely FDG-avid, heterogeneously enhancing mass in the pineal region extending posteriorly to involve the splenium of the corpus callosum, measuring 30 × 29 × 31 mm (*white arrow*).

Surgical biopsy yielded three irregular, tiny grey-white to grey-brown, soft-to-firm tissue fragments, with the largest piece measuring 0.4 × 0.3 × 0.1 cm (Histopath.-Ref. No. 8695/2025).

Microscopic examination revealed large round to polyhedral neoplastic cells arranged in sheets and clusters. The cells exhibited large round vesicular nuclei with prominent nucleoli and moderate clear-to-pale eosinophilic cytoplasm. Nuclear pleomorphism, occasional binucleation, and mitotic activity were noted. The stromal background demonstrated fibrous septae with dense lymphocytic infiltrate (stromal lymphocytosis), foci of psammomatous calcification, and areas of necrosis and haemorrhage. Notably absent were papillary architecture, atypical mitoses, tumour rosettes, and giant cells (Fig. 3).

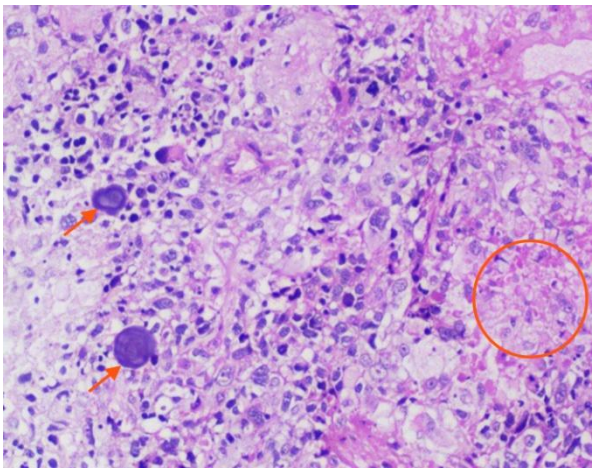

Figure-3: Microphotograph showing round malignant tumor cells in clusters, psammoma bodies (*red arrow*) with tumor necrosis (*red circle*) and scattered small lymphocytes within stromal fibrosis (H&E, x 400)

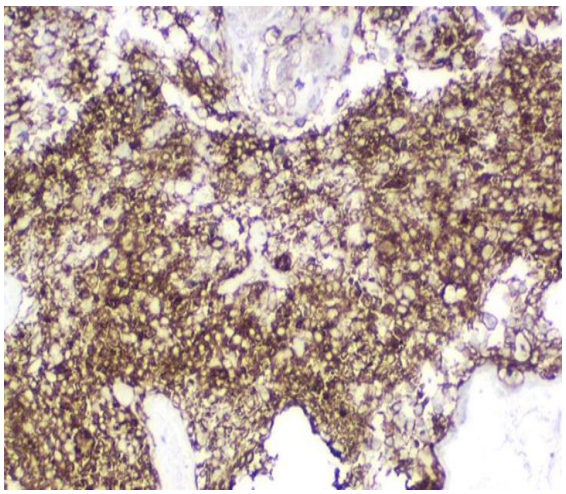

Figure-4: IHC microphotograph showing strong membranous positivity of c-KIT (CD117) stain in all tumor cells which is the hallmark of germinoma (CD117, x 400).

Immunohistochemistry (IHC): c-KIT (CD117) showed strong membranous positivity, the hallmark of germinoma, expressed in >90% of cases (Fig. 4). Podoplanin (D2-40) was positive, further supporting the germinoma lineage. Focal cytokeratin (CK) positivity was noted, acceptable in germinoma and serving to exclude pure PNET. SALL4, synaptophysin, OCT4, CD30, and glypican-3 were all negative, effectively excluding embryonal carcinoma,

yolk-sac tumour, pineoblastoma/pineocytoma, and other PNETs.

Immunohistochemistry (IHC) panel results are summarized in Table 1.

Table 1: IHC results and clinicopathological significance

Marker	Result	Significance
c-KIT (CD117)	POSITIVE	Hallmark of germinoma; expressed in >90% of cases. Strong membranous staining.
Podoplanin (D2-40)	POSITIVE	Supports germinoma lineage; expressed in seminoma/ germinoma family.
Cytokeratin (CK)	POSITIVE (focal)	Focal positivity acceptable in germinoma. Rules out pure PNET.
SALL4	NEGATIVE	Rules out embryonal carcinoma and yolk-sac tumour.
Synapto physin	NEGATIVE	Argues against pineoblastoma /pineocytoma and other PNETs.
OCT4	NEGATIVE	Unexpected in germinoma; may reflect sampling or tissue preservation artefact.
CD30	NEGATIVE	Effectively excludes embryonal carcinoma.
Glypican-3	NEGATIVE	Argues against yolk-sac tumour (AFP-secreting).

DISCUSSION

Pineal germinoma is a CNS germ cell tumour histologically identical to testicular seminoma and ovarian dysgerminoma.[1] It most commonly arises in the second decade of life and shows a male-to-female ratio of approximately 2–3:1.[2] The occurrence in a 9-year-old child, while uncommon, falls within the reported paediatric spectrum. The concurrent presence of HIV infection in this patient adds a clinically significant dimension, as immunocompromise substantially alters the diagnostic probability landscape of intracranial mass lesions.[5]

The triad of obstructive hydrocephalus, pineal region mass, and elevated CSF LDH in a paediatric male strongly favours a diagnosis of germinoma. Elevated CSF LDH is a recognised indicator of high metabolic tumour activity and has been associated with intracranial GCTs, particularly germinoma and non-germinomatous germ cell tumours (NGGCTs).[3] The lymphocytic pleocytosis observed in this case is consistent with the classical stromal lymphocytosis that characterizes germinoma histologically, a feature that may be reflected in the CSF cytology.

On neuroimaging, germinoma classically presents as a well-defined, hyperdense (on CT), homogeneous mass with avid contrast enhancement. The heterogeneous character with central cystic necrosis observed in this case, while atypical for pure germinoma, can occur in larger lesions and has been reported in mixed GCTs.[4] The absence of SWI blooming artefact argues against haemorrhagic lesions such as cavernomas or haemorrhagic metastases. The FDG-avidity (SUVmax 24.2) is characteristic of germinoma's high proliferative index.

The differential diagnosis of a pineal region mass in a paediatric immunocompromised patient includes pineoblastoma, pineocytoma, primary CNS lymphoma (PCNSL), CNS tuberculoma, toxoplasma abscess, and cryptococcoma.

Table 2 summarizes the microscopic features differentiating the three principal diagnoses considered in this case. Pineoblastoma (WHO Grade IV) was excluded by the absence of Homer Wright rosettes, the low-to-moderate mitotic rate, and negative synaptophysin staining. Pineocytoma (WHO Grade I) was excluded by the high-grade histological features and absence of pineocytomatous rosettes. PCNSL, particularly relevant in HIV-positive patients, was excluded by the absence of angiocentric large B-cell infiltrates and negative CD30.

Table 2: Microscopic differential diagnosis of principal pineal region tumours

Feature	Germi-noma	Pineo-blastoma	Pineo-cytoma
Cellularity	High	Very high, densely packed	Moderate
Cell morpho-logy	Large, round, clear cytoplasm	Small round blue cells, scant cytoplasm	Uniform small mature cells
Nuclei	Prominent nucleoli	Hyperchro matic, frequent mitoses	Round, minimal atypia
Back-ground	Fibrous septa + 'lymphoc ytes'	Necrosis & apoptosis	Neuropil-like
Mitoses	Moderate	Very high	Low
Rosettes	Absent	Homer Wright rosettes	Pineocytom atous rosettes
IHC	PLAP+, c-KIT+, D2-40+	Synaptophy sin+, NSE+	Synaptophys in+, NF+
WHO Grade	Grade IV (radiosens itive)	Grade IV	Grade I

IHC confirmation with c-KIT and podoplanin is consistent with established diagnostic criteria for intracranial germinoma.[6] The absence of OCT4 positivity, while unexpected, can occur in cases with suboptimal tissue preservation or when sampling the peripheral reactive zone of the tumour rather than the viable tumour core; this finding should prompt repeat IHC on additional sections if clinically indicated. The negative SALL4 effectively excludes embryonal carcinoma and yolk-sac tumour, the two most aggressive differentials in the GCT spectrum.[4]

Germinomas are 'highly radiosensitive' tumours. Standard treatment for localised intracranial germinoma involves reduced-dose whole-ventricular irradiation (WVI) combined with platinum-based chemotherapy, with reported five-year overall survival rates exceeding 90%.[7]

In the setting of HIV positivity, careful multidisciplinary coordination between neurosurgery, oncology, and infectious disease is essential to optimize ART regimens and minimize treatment-related immunosuppression.

CONCLUSION

We report a rare case of pineal germinoma in a 9-year-old HIV-positive child, a combination that is exceptionally uncommon and presents unique diagnostic challenges. The case underscores the importance of an integrated multimodal approach encompassing clinical assessment, neuroimaging including FDG-PET/CT, CSF biochemical profiling, meticulous histopathological analysis, and a comprehensive IHC panel for definitive characterization of pineal region masses. Accurate histopathological diagnosis is critical as germinoma, unlike the other high-grade differentials, carries an excellent prognosis with appropriate chemo-radiotherapy. Management in an immunocompromised host requires close multidisciplinary collaboration to tailor treatment while optimizing immune function.

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