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Research Article

Primary Anaplastic Pleomorphic Xanthoastrocytoma with Brainstem Dissemination: A Case Report from West Africa

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Abstract

Background

Anaplastic pleomorphic xanthoastrocytoma (APXA) is a rare primary brain neoplasm with a poor prognosis.

Case Description

An adult male presented with a sixteen-day history of headaches. His neurologic examination revealed a left upper limb pronator drift. Neuroimaging showed a right frontoparietal tumour for which he had craniotomy and complete tumour excision. Histology revealed features of an APXA. The patient initially declined adjuvant chemoradiotherapy. He presented three months post-surgery with clinical features of raised intracranial pressure. Repeat neuroimaging showed evidence of tumour recurrence. He defaulted follow-up, subsequently re-presenting six months post-operatively with seizures, personality changes and urinary incontinence. Brain magnetic resonance imaging revealed a significant increase in his tumour size, with transcallosal spread to the contralateral frontal lobe. He later had a re-opening craniotomy and subtotal tumour excision. Histopathological evaluation of the resected specimen confirmed recurrence of the initial tumour. He received adjuvant chemoradiotherapy. At his follow-up review sixteen months post-initial diagnosis, he had optimal seizure control and complete resolution of all symptoms. He remained well until eighteen months post-diagnosis, when he developed a gait imbalance with a neuroimaging correlation of medullary tumour dissemination. He progressively deteriorated until his demise twenty-three months post-diagnosis.

Conclusion

We have described a rare APXA (adult-onset) in a Nigerian man with an aggressive clinical course and brainstem dissemination. The lack of timely adjuvant therapy may have contributed significantly to his poor treatment outcome. It is noteworthy that chemoradiotherapy afforded him a brief period of tumour remission. This case further highlights the challenges of neuro-oncological practice in lower- to middle-income countries where ignorance, distrust in healthcare facilities, and unregulated, unorthodox care are pervasive.

Keywords: kepes tumour; high-grade glioma; neuro-oncology

Introduction

Pleomorphic xanthoastrocytoma (aka Kepes tumour) is a rare form of primary brain tumour first described by Kepes et al. in 1979. [1,2] This tumour accounts for < 1% of all astrocytic tumours, typically manifesting in childhood and young adulthood (first and third decades of life). [2-4] It exhibits no gender predilection and has the highest frequency of occurrence in the temporal lobe. [3] The criteria for the diagnosis of the entity; 'PXA with anaplastic features' were initially proposed by Giannini et al. in 1999, preceding the recognition of the tumour by the World Health Organisation (WHO) as a distinct histopathological sub-type in the 2016 edition of the classification of brain tumours. [5,6] APXA constitutes about 9-20% of all cases of PXA, is usually associated with a worse outcome (particularly the adult-onset variety) and can rarely undergo malignant transformation into a glioblastoma. [7-10] We report an adult-onset supratentorial APXA with brainstem dissemination in a Nigerian male.

Case Presentation

An adult male presented to our facility with a sixteen-day history of headaches of increasing severity. He had no clinical features of phakomatosis. His neurologic examination findings were normal save for a left upper extremity pronator drift. Cranial computerized tomography scan/ brain magnetic resonance imaging (MRI) revealed a right frontoparietal mixed solid and cystic tumour with a contrast-enhancing medial solid portion (Figure 1).

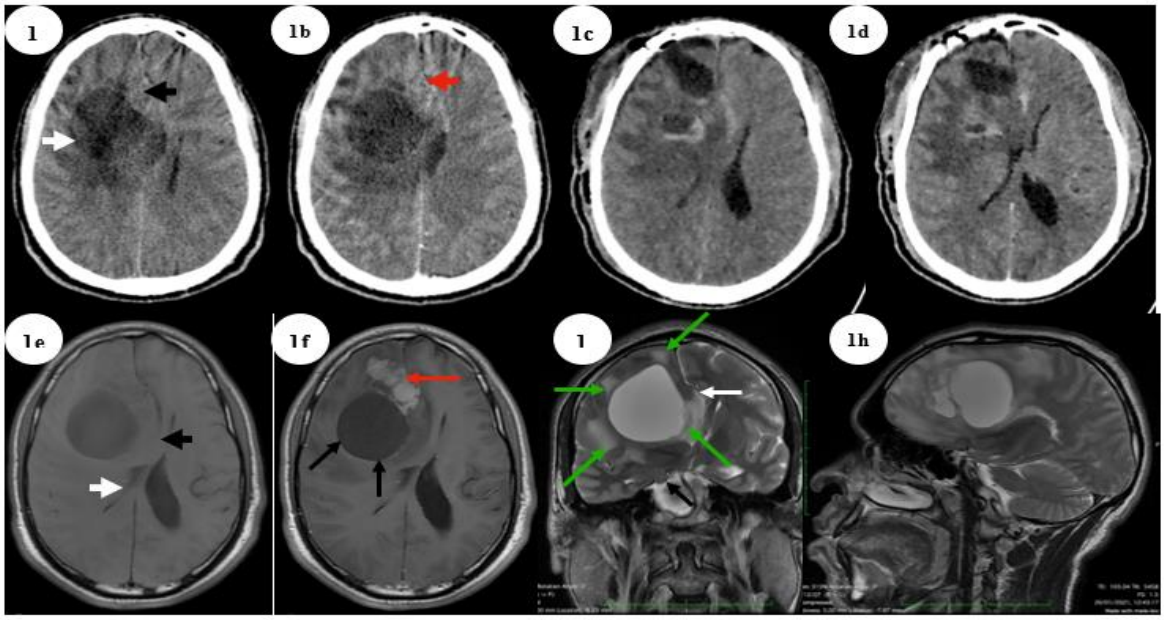

Figure 1 (a-d): Cranial computerised tomography (CT) scan (Pre/post-operative).
(e-h): Pre-operative brain magnetic resonance imaging.

a: Pre-operative non-contrast axial CT scan showing a predominantly cystic right frontoparietal lesion (white arrow) with an isodense solid component (mural nodule, black arrow).

b: Pre-operative contrast image showing heterogeneous enhancement of the mural nodule (red arrow).

c: Post-operative non-contrast axial CT scan showing gross total tumour excision.

d: Post-operative contrast image showing no residual tumour.

e: Pre-operative non-contrast axial T1-weighted image. Note the mass effect evidenced by partial effacement of the right lateral ventricle (white arrow) and a left-ward midline shift (black arrow).

f: Pre-operative contrast-enhanced axial T1-weighted image. Note the leptomengeal attachment of the contrast-enhancing mural nodule (red arrow). The wall of the cystic portion of the tumour shows minimal contrast enhancement (black arrows).

g: Pre-operative non-contrast coronal T2-weighted image. Note the large tumour size, sub-falcine (white arrow)/ uncal herniation (black arrow) and the extensive peritumoural oedema (green arrows).

h: Pre-operative non-contrast sagittal T2-weighted image.

He subsequently underwent a right frontal craniotomy and gross total excision of this tumour, with no new neurological deficit. The patient had an uneventful post-operative course and was discharged home after five days of hospitalization. Histology revealed features of an anaplastic pleomorphic xanthoastrocytoma, WHO grade III (Figure 2).

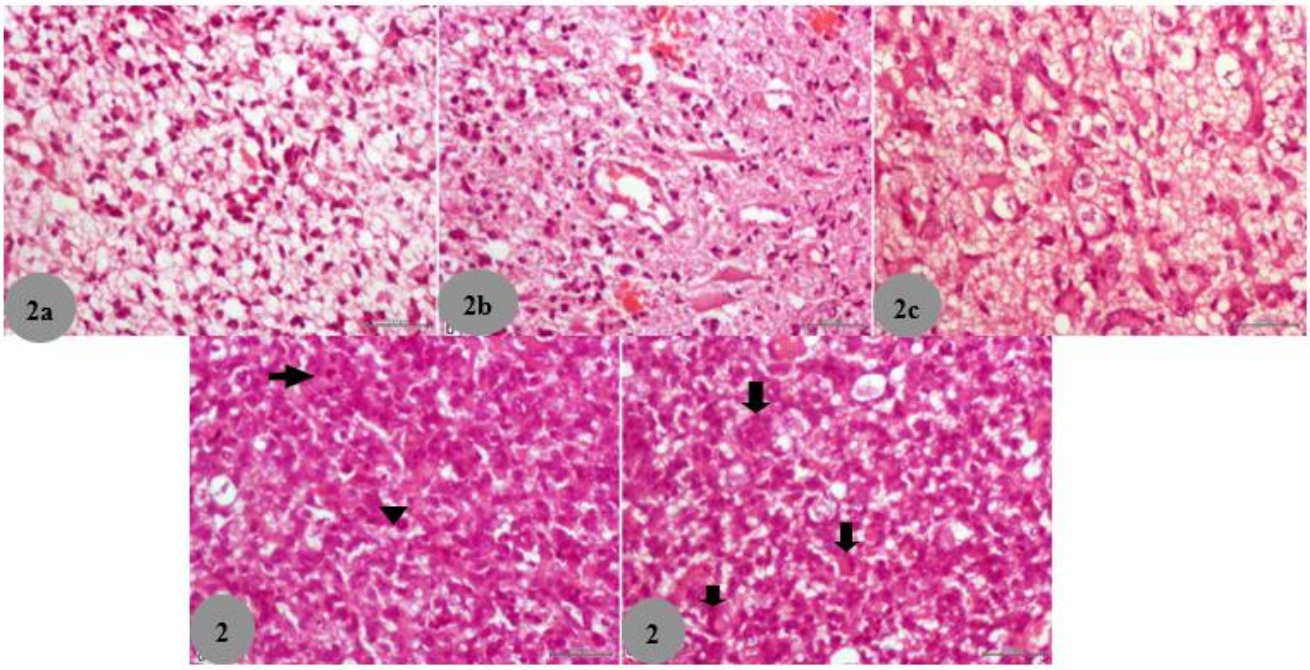

Figure 2 (a-e): Hematoxylin and Eosin staining photomicrographs of the primary tumour specimen. These show a cellular astrocytic neoplasm with marked pleomorphic cells and a background of microcystic spaces (a, d, e). Some cells have foamy cytoplasm with markedly pleomorphic tumour giant cells (arrows; e). There are foci of endothelial proliferation (c). Also seen are eosinophilic granular bodies and Rosenthal fibres (a and b). There are frequent mitotic figures (arrowheads; d).

The patient initially declined adjuvant chemoradiotherapy, instead opting for unspecified over-the-counter medications. He presented three months post-surgery with recurrent headaches, vomiting, and elevation of the bone flap. The neurological examination remained unremarkable. Repeat neuroimaging showed evidence of tumour recurrence (Figure 3a-d).

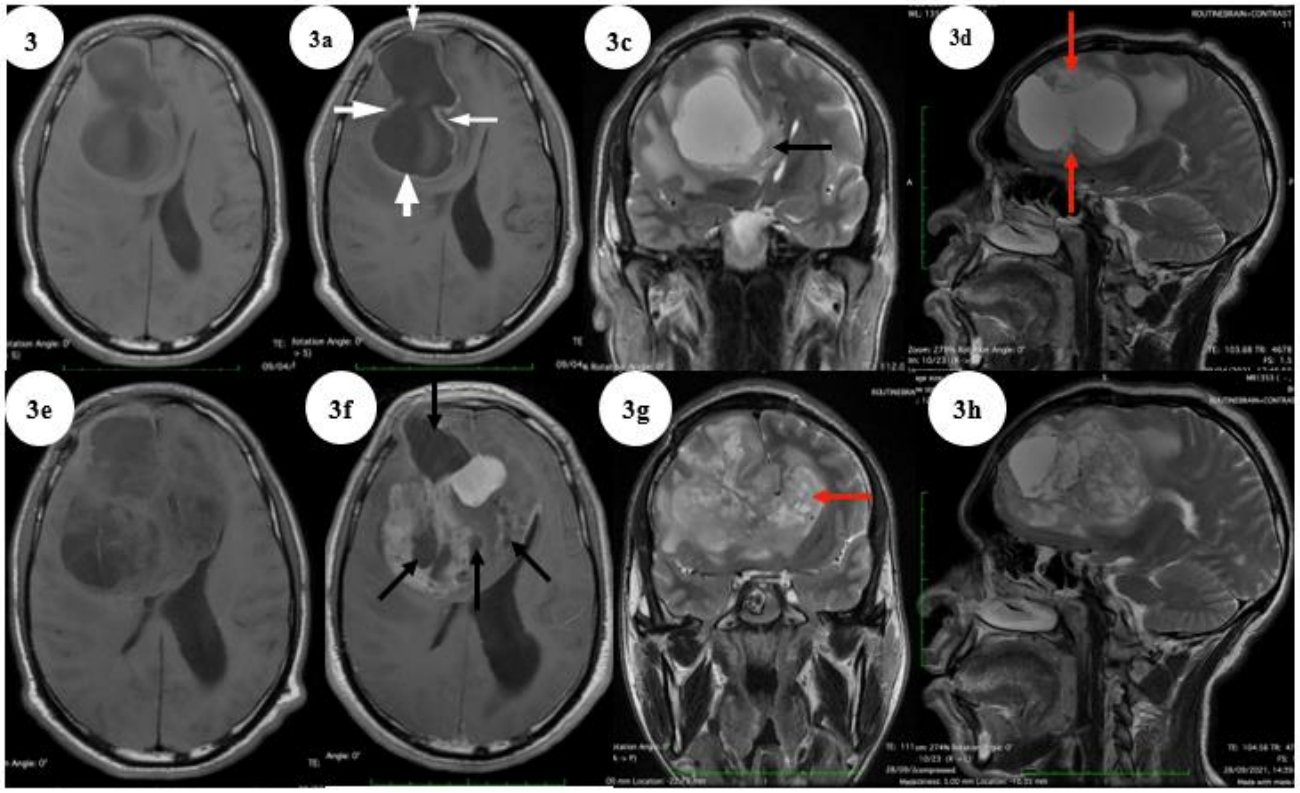

Figure 3 (a-d): Brain Magnetic Resonance Imaging (4 months post-surgery).
(e-h): Brain Magnetic Resonance Imaging (7 months post-surgery).

a: Pre-contrast axial T1-weighted image showing tumour recurrence in the right frontal lobe.

b: Post-contrast axial T1-weighted image. Note the contrast enhancement of the wall of the tumour cavity (white arrows).

c: Pre-contrast coronal T2-weighted image showing a medial solid portion of the tumour (black arrow).

d: Pre-contrast sagittal T2-weighted imaging: Note the solid components along the superior and inferior aspects of the wall of the tumour cavity (red arrows).

e: Pre-contrast axial T1-weighted image showing progressive increase in tumour size.

f: Post-contrast axial T1-weighted image. Note the multi-cystic nature of the tumour (black arrows) and its heterogenous contrast enhancement.

g: Pre-contrast coronal T2-weighted image showing an extension of the tumour to the contralateral side (red arrow) via the corpus callosum. Note the multicystic nature of the tumour.

h: Pre-contrast sagittal T2-weighted imaging showing a change in the morphology of the tumour to a predominance of the solid component.

He was counselled for surgery and adjuvant chemoradiotherapy but defaulted from the clinic. He re-presented six months post-operatively with complaints of seizures, personality changes and urinary incontinence, suggesting further tumour progression. Brain MRI revealed a significant increase in his tumour size, with a change in its morphology to a predominantly solid tumour with extension to the contralateral frontal lobe via the corpus callosum (Figure 3e-h). Although he consented to further surgical treatment, this was delayed for two weeks due to a respiratory tract infection. He developed expressive aphasia, spastic quadriparesis and altered sensorium (Glasgow coma score of 11) during this period. A repeat MRI showed a further increase in the tumour size, for which he had a re-opening craniotomy and subtotal tumour excision. He regained full consciousness and complete resolution of the motor and speech impairments. He, however, had residual frontal lobe deficits (mood changes, poor self-care, and urinary incontinence). Histopathological evaluation of the resected specimen confirmed a recurrence of the initial tumour (Figure 4).

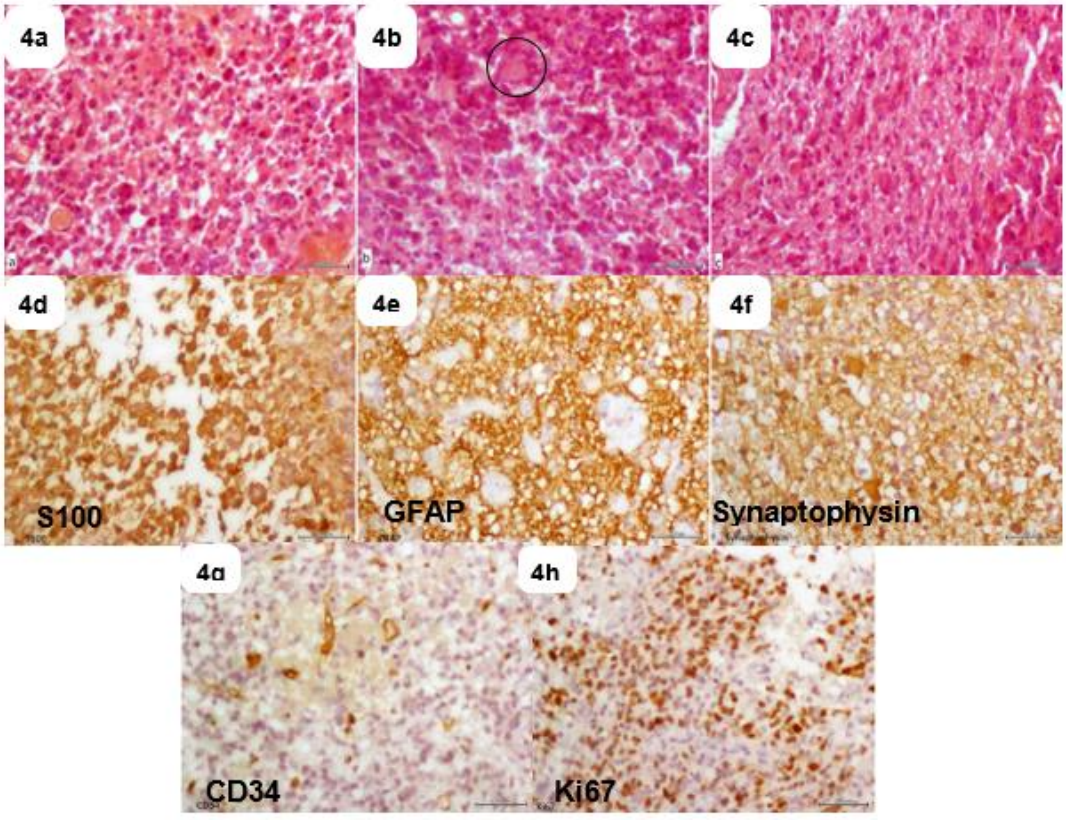

Figure 4 n(a-c): Hematoxylin and Eosin staining photomicrographs of the recurrent tumour biopsy specimen showing similar pleomorphic cells as seen in the initial tumour (a) and multinucleated giant cells, some resembling 'Touton' type giant cells (circled; b). Rosenthal fibres and eosinophilic granular bodies are also seen in this tumour (c). The tumour exhibited a strong S100, GFAP, and Synaptophysin staining (d-f), and its cells showed focal CD34 (g) staining and a 45% Ki67 proliferation index (h). These features are consistent with a recurrent APXA.

He received adjuvant Temozolomide chemotherapy and whole-brain radiotherapy (at a dose of 25 Grays in ten fractions, followed by a second phase at 24 Grays in twelve fractions over five weeks), which were well tolerated. At his follow-up evaluation sixteen months post-initial diagnosis, he had optimal seizure control and complete resolution of all other symptoms. His neuroimaging showed radiological evidence of significant tumour size reduction and brain decompression (Figure 5).

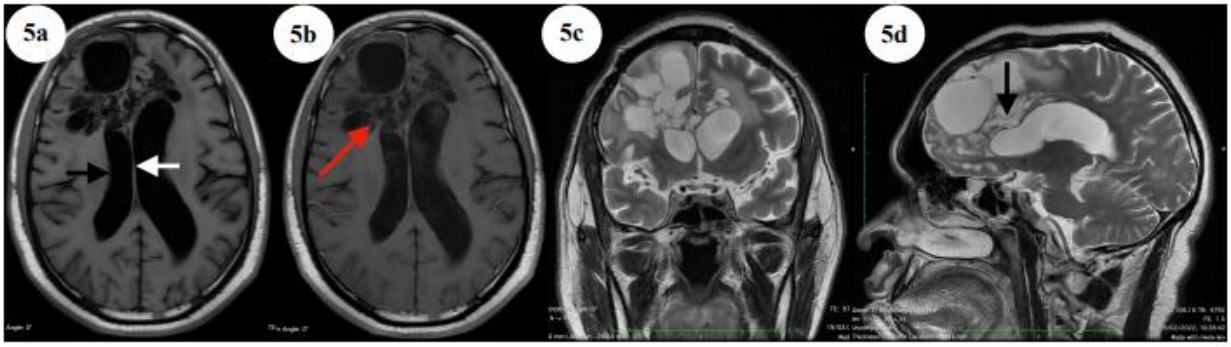

Figure 5 (a-d): Brain Magnetic Resonance Imaging (18 months post-initial operation; 6 months post re-operation; 3 months post-chemoradiotherapy).

a: Pre-contrast axial T1-weighted image showing a significant reduction in the tumour size, re-expansion of the right lateral ventricle (black arrow), and restoration of the midline (white arrow).

b: Post-contrast axial T1-weighted image. Note the non-contrast enhancement and the multicystic nature of the residual tumour (red arrow).

c: Pre-contrast coronal T2-weighted image. There are features of diffuse encephalomalacia (likely radiotherapy-induced).

d: Pre-contrast sagittal T2-weighted image showing involvement of the anterior half of the corpus callosum (black arrow). Note the significant tumour size reduction/ brain decompression.

He remained well until two months later, i.e. eighteen months after the first diagnosis, when he developed gait imbalance and a tendency to fall to the left side, with an MRI correlation of medullary and fourth ventricular tumour dissemination. There was another lesion in the medial wall of the occipital horn of the left lateral ventricle, suggesting spread via the cerebrospinal fluid (Figure 6). He progressively deteriorated until his demise twenty-three months post-diagnosis.

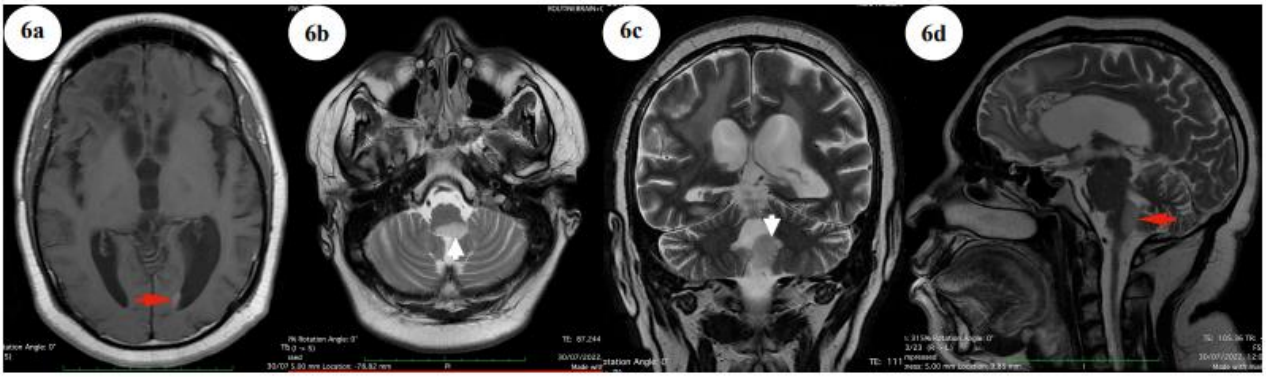

Figure 6 (a-d): Brain Magnetic Resonance Imaging (18 months post-initial diagnosis).

a: Post-contrast axial T1-weighted image. Note the tumour deposit on the medial wall of the occipital horn of the left lateral ventricle (red arrow).

b: Axial T2-weighted image showing the medullary tumour dissemination (white arrow).

c: Coronal T2-weighted image. Note the fourth ventricular extension of this tumour (white arrow).

d: Sagittal T2-weighted image showing the dorsal location of the tumour on the medulla (red arrow).

Discussion

Anaplastic pleomorphic xanthoastrocytoma, now known as grade III pleomorphic xanthoastrocytoma, is a rare form of high-grade glioma, with very few reported cases in the literature. [11] These tumours may infrequently occur de novo or, more commonly, from malignant transformation of pre-existing grade II PXAs. [2,8,12] The histopathological features of anaplasia in PXAs include hypercellularity, increased invasiveness, loss of pericellular reticulin, necrosis, microvascular proliferation and a mitotic index of >5 mitotic figures per high power field. [7-9,13] Several genetic mutations have been linked to the occurrence of APXAs, the most frequent of which is the BRAF V600E mutation. [2,8,12] Other genomic aberrations implicated in the malignant transformation of PXAs include loss of heterozygosity of chromosome 10 and alterations in the TERF (amplification or promoter mutation), IDH2, P16 and P53 genes. [2,12-14] A rare association of APXA with H3G34 mutation has been reported by previous authors. [15] Phillips *et al.* in 2019 conducted a study on the genetic profiling of 23 PXAs (15 of which were APXAs) wherein they found that the key genetic characteristic of PXAs/ APXAs is the concurrence of RAF mutations and CDKN2A homozygous deletion. [14] Previous reports of Neurofibromatosis I-linked APXAs exist in neurosurgical literature. [16] Although the typical age of onset of APXAs is in childhood or early adulthood, they have been reported in older individuals. [13] Our patient, who presented in the third decade of life, had histologic features consistent with anaplastic pleomorphic xanthoastrocytoma and no clinical finding of phakomatosis. Whereas APXAs are often solitary/ supratentorial, multicentricity and infratentorial location of these tumours have been documented. [17,18]

Previous authors have profiled rare reports of sellar, thalamic, hypothalamic, callosal, quadrigeminal, intraventricular and pineal APX. [8,10,19] Meningeal dissemination of anaplastic PXAs as well as widespread metastasis of these tumours at the initial presentation have likewise been described. [4,20] Clinical manifestations of APXAs are usually related to tumour location/size and onset of raised intracranial pressure (RICP).2 Recurrent seizures from cortical irritation (attributable to the superficial tumour location) are the most common symptom of APXAs. [21,22] Atypical presentations of APXA with musical hallucination and as a spontaneous intracerebral bleed have been previously reported. [3,8] The index patient had a solitary deep frontoparietal lesion with falcine attachment at the time of his initial diagnosis when the only symptom he had was a headache. As the lesion progressively increased in size and became more superficial, he experienced seizures, features of intracranial hypertension and focal neurologic deficits. He later developed clinical manifestations of cerebellar deficits following infratentorial tumour dissemination. The short duration of his initial symptom may suggest a de novo occurrence of APXA, rather than a secondary evolution of anaplasia in a primary PXA.

Radiological features of PXAs include their mixed consistency (with a variable predominance of the solid and cystic components), superficial location, leptomeningeal contact and temporal lobe predilection.[8,19,23] She D *et al*., in their novel study investigating the distinguishing magnetic resonance imaging characteristics of PXAs/ APXAs, found features similar to those of high-grade gliomas to occur at a higher frequency in APXAs than PXAs.[19] These include heterogenous contrast-enhancement, extensive vasogenic peritumoural oedema, a relatively larger size at the first diagnosis on conventional magnetic resonance imaging (MRI), and lower minimum relative apparent diffusion coefficient/ higher maximum relative cerebral blood volume on advanced MRI. [19] Our patient had a predominance of the cystic component with a contrast-enhancing solid portion at the initial diagnosis, which gradually transformed into a predominantly solid tumour with disease progression. The radiological characteristics of his tumour were suggestive of a high-grade glioma. Interestingly, he also exhibited aggressive spread via the corpus callosum to the contralateral cerebral hemisphere. Although glioblastomatous transformation has been reported in recurrent APXAs, the histopathological diagnosis of the recurrent lesion in the index patient was concordant with that of the initial tumour. There may be molecular signatures of glioblastoma in the recurrent tumour, as a butterfly glioma pattern is typically not seen in APXA. It is also established that BRAF V600E mutations, CDKN2A homozygous deletions and TERF gene alterations could occur in the epithelioid variant of both glioblastoma multiforme and APXA.[3,13,24,25] As such, it is sometimes difficult to distinguish between these two histologic tumour subtypes, which may rarely co-exist and have been suggested to represent the same entity or be closely related.[8,24] However, the tumour in the index patient showed eosinophilic granular bodies and Rosenthal fibres, which are reported to be uncommon in epithelioid glioblastoma. Similarly, glial fibrillary acid protein (GFAP), which was strongly positive in our patient, is typically patchy in glioblastoma. [26] The patient also exhibited tumour dissemination via the cerebrospinal fluid pathway, evidenced by transventricular metastasis from the supratentorial to the infratentorial compartment (Figure 5). This mode of tumour spread has been documented in the literature in association with APXAs. [27]

Due to the rarity of APXAs, standard treatment protocols are yet to be strictly defined. [8,11,21] The extent of surgical resection is an important prognostic indicator for PXAs/APXAs, and complete surgical excision of APXAs is advocated whenever possible. [2,8,11,22] However, aggressive tumour resection has the potential for debilitating neurological sequelae in patients with butterfly gliomas. The role of adjuvant chemotherapy and radiotherapy is not well established, with variable documented treatment responses. [8,21] Primary anaplastic pleomorphic xanthoastrocytomas are known to have an early recurrence pattern and a poor prognosis. [2,21] The average interval from the initial diagnosis to recurrence in a literature review of 24 cases of primary APXAs (1979-2016) by Choudry *et al*. was fourteen months (the range being one month to thirty-six months) with significantly shorter disease-free survival compared to PXAs. [21] BRAF V600E-inhibitors (like Dabrafenib and Vemurafenib) are potential therapeutic options for RAF mutated-APXAs, and previous authors have detailed their usefulness in patients with poor response to radiotherapy and alkylating chemotherapy. [8] MEK inhibitors (trametinib) may be employed for APXA patients with resistance to BRAF inhibitors. [11,28] This underlines the importance of genetic studies, which are presently unavailable in our practice environment, in patients diagnosed with APXAs.

In conclusion, we have described a rare APXA (adult-onset) in a Nigerian man with an aggressive clinical course and brainstem dissemination. Aside from the tumour biology, the patient's hesitance to have timely surgical intervention and adjuvant therapy contributed significantly to his poor treatment outcome. It is noteworthy that chemoradiotherapy afforded him a brief period of tumour remission. This case further highlights the challenges of neuro-oncological practice in lower- to middle-income countries where ignorance, distrust in healthcare facilities, and unregulated, unorthodox care are pervasive.

Conflict of Interests

The authors declare none.

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