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Gemistocytic astrocytoma mimicking hypertensive haemorrhage. A rare case of tumour disguised as intracerebral haemorrhage

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craniotomy, gemistocytic astrocytoma, intracerebral haemorrhage, tumour mimicking stroke, brain gliomas, ICH

ABSTRACT

Gemistocytic astrocytoma is a rare variant of diffuse astrocytoma, characterized by a high proportion of gemistocytic cells, which exhibit aggressive behaviour and an increased risk of malignant transformation. Its clinical and radiological presentation can be misleading, especially when it mimics other intracerebral pathologies, such as hypertensive intracerebral haemorrhage (ICH). Differentiating between a primary haemorrhagic event and a haemorrhagic tumour remains a significant diagnostic challenge.

We report the case of a 56-year-old male with no prior medical history who presented with sudden-onset right-sided hemiparesis and severe speech disturbances. Initial neuroimaging revealed a deep intraparenchymal hematoma in the left internal capsule, and lenticular nucleus, strongly suggestive of a hypertensive haemorrhagic stroke. Despite intensive medical management, the patient's condition deteriorated, prompting further imaging studies, which raised suspicion of an underlying neoplastic process. Subsequent MRI findings indicated features atypical for a purely haemorrhagic lesion, necessitating neurosurgical intervention for definitive diagnosis.

The patient underwent a left fronto-temporo-parietal craniotomy, during which a tumour-like mass was encountered and completely resected. Histopathological analysis confirmed the diagnosis of gemistocytic astrocytoma. Postoperatively, the patient showed gradual neurological improvement, though residual deficits persisted.

This case highlights the complexity of differentiating a gemistocytic astrocytoma from a spontaneous hypertensive haemorrhage, particularly in patients without a prior oncological history. While intracerebral haemorrhage is commonly associated with chronic hypertension, intratumoural haemorrhage remains an important differential diagnosis, especially when imaging findings suggest a mass effect, perilesional oedema, or progressive neurological deterioration despite optimal medical therapy. MRI characteristics, such as hyperintense T2-weighted and FLAIR signals, can provide critical clues, but histopathological confirmation remains the gold standard.

Gemistocytic astrocytomas, though rare, should be considered in cases of unexplained intracerebral haemorrhage, particularly when imaging findings or clinical progression are atypical. This case underscores the importance of a

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multidisciplinary approach involving neurology, neuroradiology, and neurosurgery to ensure timely diagnosis and appropriate management. A high index of suspicion is crucial to prevent delays in the recognition and treatment of haemorrhagic brain tumours, which can significantly impact patient outcomes.

Introduction

Astrocytomas are common primary brain tumors originating from astrocytes, glial cells that maintain neuronal function and brain homeostasis. Gemistocytic astrocytoma, a rare subtype of diffuse astrocytoma (WHO grade II), is distinguished by a high proportion of gemistocytic cells-large, eosinophilic astrocytes with eccentric nuclei [1]. These tumors typically arise in adults in their fourth and fifth decades, predominantly in the cerebral hemispheres, and exhibit a higher tendency for malignant transformation into glioblastoma, necessitating early detection and intervention [2].

While gemistocytic astrocytomas usually present with progressive neurological symptoms such as headaches, seizures, or focal deficits, they can occasionally mimic hypertensive intracerebral hemorrhage (ICH), creating diagnostic challenges. Although hypertensive ICH is the leading cause of spontaneous brain hemorrhage, affecting deep structures like the thalamus and basal ganglia, hemorrhagic tumors account for only 2-5% of spontaneous ICH cases. Tumor-related hemorrhage may result from fragile neovascularization, necrosis, or vascular invasion, leading to an acute presentation [3].

Imaging plays a crucial role in differentiating these conditions. While CT scans may reveal hyperdense lesions, MRI-particularly contrast-enhanced sequences-can indicate irregular enhancement, perilesional edema, and mass effect, raising suspicion of an underlying tumor. However, initial imaging may fail to detect tumors; in a study of 193 patients with primary malignant brain tumors, 9 had normal initial MRI scans, while 8 had abnormalities misinterpreted [4]. In gemistocytic astrocytomas, hyperintense signals on T1-weighted MRI, especially in cases with necrosis or infiltration, further complicate differentiation from ICH [5]. Follow-up imaging or histopathological confirmation remains the gold standard for diagnosis.

At the molecular level, gemistocytic astrocytomas frequently harbor TP53 mutations and IDH1/IDH2 pathway alterations, contributing to their aggressive behavior and poor prognosis. Despite their WHO grade II classification, they carry a significant risk of progression to glioblastoma, underscoring the importance of early diagnosis [6].

Delays in identifying tumor-related hemorrhages can impact treatment strategies and outcomes. While hypertensive hemorrhages are managed conservatively, hemorrhagic tumors often require surgical intervention, biopsy, or oncological treatment. Misdiagnosis may lead to delayed tumor detection, allowing disease progression. Even when imaging suggests hypertensive ICH, a neoplastic etiology should be considered, particularly if the clinical course is atypical.

We present the case of a 56-year-old male initially diagnosed with hypertensive ICH, whose progressive neurological decline and atypical imaging findings ultimately led to the diagnosis of gemistocytic astrocytoma. This case highlights the need to consider tumor-related hemorrhage in select spontaneous ICH cases and emphasizes the role of advanced neuroimaging and a multidisciplinary approach in distinguishing hemorrhagic strokes from brain tumors to ensure timely and accurate management.

Case Presentation

A 56-year-old male with no prior medical history experienced a sudden onset of right-sided hemiparesis and severe speech disturbances. His spouse reported a rapid neurological decline, prompting immediate transportation to the Emergency Department (ED). Upon arrival, the patient was somnolent and minimally cooperative, with a Glasgow Coma Scale (GCS) score of 7 and a National Institutes of Health Stroke Scale (NIHSS) score of 15, indicating a major neurological deficit.

The neurological examination revealed complete flaccid hemiplegia on the right, upper motor neuron (central) facial paresis on the right, right homonymous hemianopia, and profound mixed aphasia affecting both receptive and expressive language functions. Given the acute presentation and severe deficits, an intracerebral hemorrhage was highly suspected.

Consequently, an emergent non-contrast cranial CT scan was performed immediately upon admission. The imaging revealed a large deep intraparenchymal hematoma in the left internal

capsule and lenticular nucleus, measuring approximately 30-40 mL. The hematoma was accompanied by mild perilesional edema and moderate mass effect, although no signs of herniation were observed at that time. These findings strongly suggested a hypertensive intracerebral hemorrhage (ICH), particularly in the context of the patient's elevated blood pressure at presentation (Figure *1*).

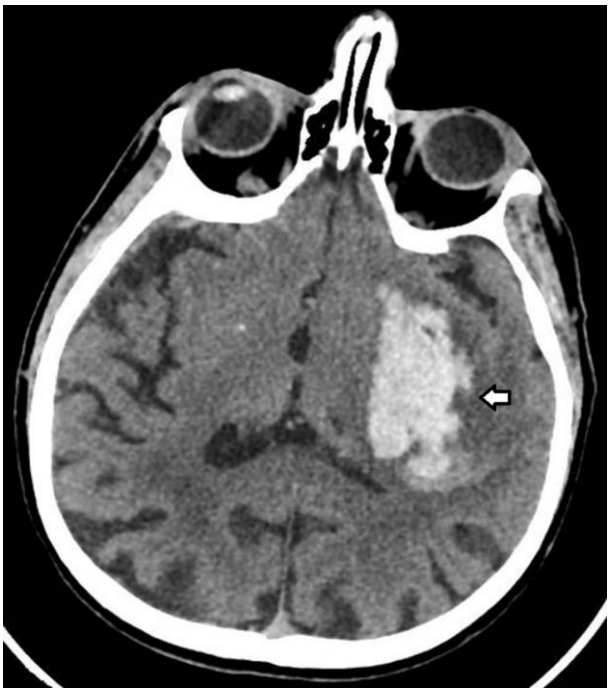

Figure 1. Initial cranial CT scan showing a deep intraparenchymal hematoma in the left internal capsule and lenticular nucleus

During the hospital course, while under conservative neurological management, the patient exhibited a slight deterioration in his neurological status. A repeat cranial CT scan was performed (Figure 2), which revealed an expansion of the intracerebral hemorrhage, accompanied by perilesional edema and a slightly mass effect on the midline structures.

Conservative management was continued over the following week as the patient's clinical status remained stable. A third cranial CT scan showed a cerebral hematoma in the process of resolving, without evidence of recent bleeding, but with significant cerebral edema and a midline shift (Figure 3).

This unfavorable imagistic evolution raised the possibility of surgical intervention. Consequently, an intravenous contrast-enhanced cranial MRI was performed for further evaluation (Figure 4).

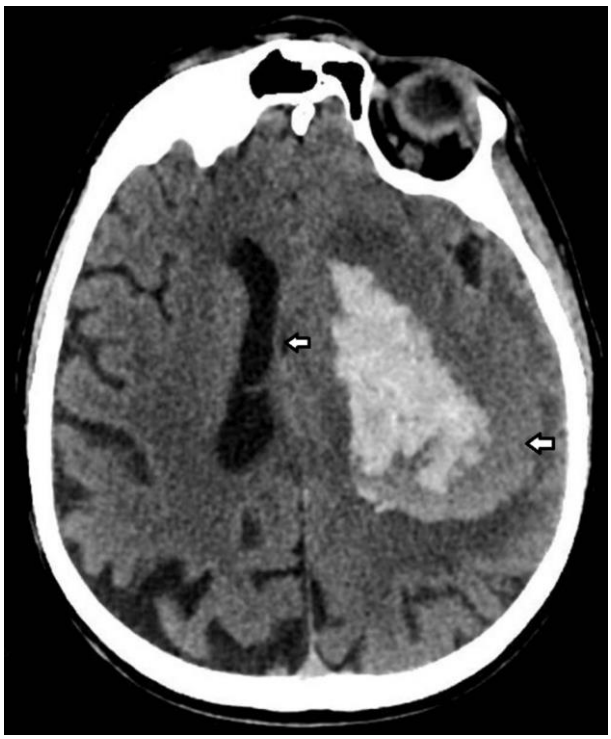

Figure 2. Follow-up CT scan showing an increase in hematoma size, extension into the left frontal and temporal lobes, and mass effect on the left lateral ventricle. Arrows indicating the expanding hematoma and accompanying midline shift.

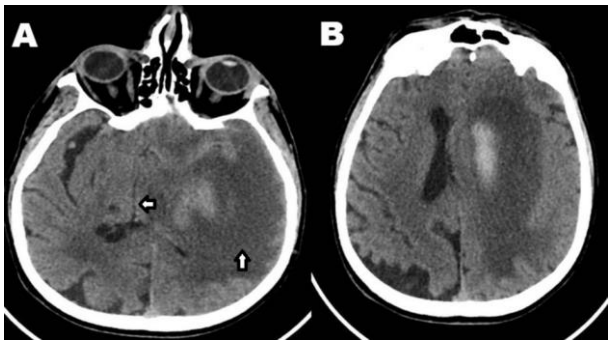

Figure 3. Follow-up CT scan showing a decrease in hemorrhagic density with extended perilesional edema, presenting a peritumoral-like appearance. (A) - large mass effect, (B) - compression of the left lateral ventricle.

Due to the worsening mass effect and persistent neurological deficits, the patient was transferred to the Neurosurgery Department for surgical evaluation. A left fronto-temporo-parietal craniotomy was performed under general anesthesia. Intraoperatively, a tumor-like mass with

hemorrhagic components was identified and completely resected along with hematoma evacuation. The tumor appeared well-vascularized and infiltrative, which raised concerns for a primary brain neoplasm rather than a simple hematoma.

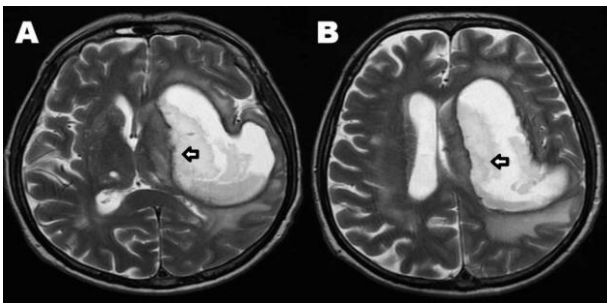

Figure 4. MRI scan showing a large hematoma with hyperintense signals on T2-weighted and FLAIR sequences, raising suspicion of a tumor. (A) sylvian valley - insular level; (B) left lateral ventricle compression

Following surgery, the patient was transferred to the Neurosurgery ICU for close monitoring. A postoperative CT scan showed a porencephalic cavity in the left temporo-insular region, with minimal adjacent hematoma and moderate perilesional edema, but no signs of progressive bleeding or infection (Figure 5). The resolution of mass effect was evident, confirming the successful removal of the underlying pathology.

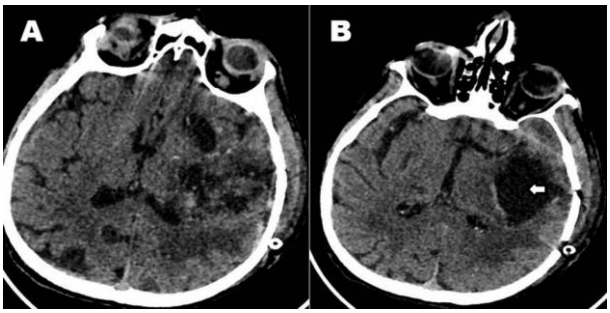

Figure 5. Postoperative CT scan showing porencephalic cavity and resolution of mass effect. (A) lateral ventricle level; (B) 3rd ventricle level

Over the next few days, the patient's condition gradually improved. He regained partial consciousness and became more cooperative, though severe mixed aphasia and right-sided hemiplegia persisted. Rehabilitation, including speech therapy and physical therapy, was initiated to aid neurological recovery.

At discharge, the patient was conscious and cooperative, with persistent right-sided hemiplegia and motor aphasia, but early signs of motor recovery were observed. He was referred for intensive neurological rehabilitation, with close outpatient follow-up scheduled to monitor his progress and assess for potential tumor recurrence.

Histopathological examination (Figure 6) of the excised tissue confirmed gemistocytic astrocytoma (WHO Grade II), characterized by abundant gemistocytic cells with eosinophilic cytoplasm and eccentric nuclei. The presence of tumor-associated hemorrhage explained the misleading presentation and radiological findings.

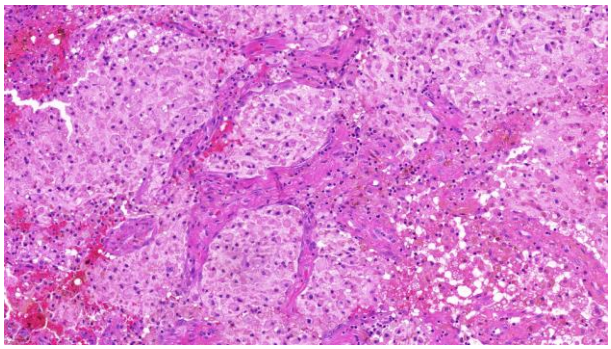

Figure 6. Histopathology 35.1x - Gemistocytic astrocytoma

This case highlights the diagnostic challenge posed by gemistocytic astrocytomas when they present as intracerebral hemorrhage, leading to an initial misdiagnosis of hypertensive stroke. The patient's progressive neurological deterioration and MRI findings raised suspicion of a neoplasm, ultimately confirmed through histopathological analysis after neurosurgical intervention. Recognizing such cases is crucial to avoid delays in diagnosis and ensure timely, appropriate management.

Discussion

The case of a gemistocytic astrocytoma presenting as a spontaneous hypertensive intracerebral hemorrhage (ICH) highlights the complexity of diagnosing brain tumors with atypical hemorrhagic presentations. While hypertensive hemorrhages typically occur in deep brain structures such as the thalamus, basal ganglia, and brainstem, hemorrhagic brain tumors are rare and can be misinterpreted as primary vascular events. Distinguishing between a hypertensive ICH and a

hemorrhagic tumor is challenging due to overlapping clinical and radiological features [7].

Tumor-associated hemorrhages account for 2-5% of spontaneous ICH cases, with gliomas, metastases, and vascular malformations being the most common causes. High-grade gliomas, particularly glioblastomas, have a higher propensity for bleeding due to fragile neovascularization and tumor necrosis. Low-grade gliomas, such as gemistocytic astrocytomas, rarely present with hemorrhage, making this case unusual. This suggests that hemorrhagic risk is influenced not only by tumor histology but also by factors such as location, vascular infiltration, and tumor biology [8].

The initial diagnosis of hypertensive hemorrhage was plausible given the capsulo-thalamic localization and history of hypertension. However, the patient's unfavorable clinical progression under conservative treatment and MRI findings raised suspicion of a tumor. Intracerebral hematomas and brain tumors share common imaging characteristics, including perilesional edema, mass effect, and secondary structural compression. Initial imaging can be inconclusive, as studies have shown that in 193 patients with primary malignant brain tumors, 9 had normal initial MRIs, and 8 had abnormalities that were not initially diagnosed [9]. This suggests that some tumors may grow rapidly and escape early detection. In this case, it cannot be ruled out that the hemorrhage was initially hypertensive and that the tumor developed later in the same location.

Neuroimaging is crucial for differentiation, but CT scans, while effective for detecting hematomas, may not reveal underlying neoplasms. MRI, particularly T2-weighted and FLAIR sequences, can provide critical clues such as heterogeneous signal intensities, irregular borders, and residual enhancement, suggestive of an underlying tumor. Perfusion-weighted imaging (PWI) and MR spectroscopy can aid differentiation, though they are not always available in emergency settings. Intraoperative findings confirmed the presence of a tumor, and histopathological analysis established the diagnosis of gemistocytic astrocytoma (WHO Grade II). Given the high risk of malignant progression, reported in 60-80% of cases within 2-5 years, early diagnosis and intervention are essential [9].

The role of surgery in hemorrhagic gemistocytic astrocytomas is debated. While gross total resection (GTR) is associated with better survival outcomes, complete excision is often challenging due to tumor infiltration. In cases where total resection is not feasible, adjuvant radiotherapy and temozolomide-based chemotherapy are standard treatment options. In this case, craniotomy and total tumor resection were justified due to mass effect, worsening neurological symptoms, and imaging findings suggestive of neoplasm [10].

Misdiagnosing a tumor-related hemorrhage as a primary stroke can lead to delays in appropriate treatment. Patients with suspected hypertensive ICH typically receive blood pressure management and supportive care, with hematoma evacuation in select cases. However, in cases of underlying tumors, delayed recognition may allow disease progression and worsen prognosis. A retrospective study by Nozaki et al. demonstrated that misdiagnosis of tumor-associated hemorrhages as strokes correlated with poorer survival outcomes [11]. Given that glioma-related hemorrhages occur in 3.7% to 12% of cases, even when imaging suggests a hypertensive ICH, an underlying tumor should remain a differential diagnosis. In cases of atypical evolution or poor response to conservative therapy, histological sampling during hematoma evacuation and post-operative MRI are critical for detecting hidden neoplasms.

Anticoagulation can exacerbate tumor-associated hemorrhages, as fragile tumor vasculature increases bleeding risk. The role of anticoagulation reversal in ICH raises concerns about undiagnosed gliomas presenting with early hemorrhage, potentially delaying tumor detection [12]. Further research should explore whether coagulation status influences glioma-associated hemorrhage and clinical progression.

Gliomas, including gemistocytic astrocytomas, have high malignant potential, requiring long-term imaging follow-up. Even low-grade tumors can progress to glioblastoma, emphasizing the need for MRI surveillance every 3-6 months [13, 15]. Recognizing hemorrhage as an early sign of tumor instability could refine treatment and monitoring strategies.

Long-term management includes close surveillance with MRI every 3-6 months due to the high risk of recurrence and malignant transformation. Neurorehabilitation, including speech and physical therapy, is essential for

functional recovery. Despite persistent aphasia and hemiplegia, early signs of motor improvement were observed, reinforcing the benefits of intensive rehabilitation [11, 14].

This case underscores the importance of a multidisciplinary approach involving neurologists, neurosurgeons, neuroradiologists, and pathologists to ensure accurate diagnosis and optimal treatment. While spontaneous ICH is often attributed to vascular causes, an underlying tumor should be considered, particularly when neurological deterioration is progressive, mass effect persists, or imaging features are atypical. Vigilance in cases of unexplained hemorrhage can facilitate timely intervention and improve patient outcomes.

Conclusions

This case underscores the diagnostic challenges of differentiating hemorrhagic tumors from primary intracerebral hemorrhage (ICH), particularly when initial imaging suggests a vascular origin. While spontaneous ICH is frequently attributed to hypertensive causes, this case highlights the need for a high index of suspicion in atypical presentations. The stepwise diagnostic approach, including MRI evaluation, ultimately led to the identification of an underlying neoplasm, which was confirmed intraoperatively and histopathologically.

Our findings reinforce the importance of multimodal imaging and clinical vigilance in cases where neurological deterioration persists despite conservative management. Early differentiation between tumor-associated hemorrhage and primary ICH is crucial, as treatment strategies differ significantly. A multidisciplinary approach integrating neurology, neurosurgery, and neuroradiology is essential to ensuring timely intervention and optimizing patient outcomes.

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