

Glioblastoma Multiforme Mimicking Arteriovenous Malformation

Arteriyovenöz Malformasyonu Taklit Eden Glioblastoma Multiforme

ABSTRACT

Glioblastoma multiforme is the most common intracranial neoplasm of all primary central nervous system tumors. Glial tumors can present in different forms. Intracranial hemorrhage may occur in all central nervous system tumors to a varying degree and extent and may even be massive. A 58-year-old man presented with intraparenchymal hemorrhage manifesting as severe headache and vomiting. Cranial computed tomographic scans revealed a right posterior temporoparietal intraparenchymal hemorrhage. Cerebral angiography revealed a 3x2 cm right inferior parietal arteriovenous malformation. The patient underwent surgical treatment with a diagnosis of arteriovenous malformation. Postoperatively, the histological diagnosis was glioblastoma. Glioblastoma may mimic an arteriovenous malformation. Close follow-up of such patients is essential.

KEYWORDS: Glioblastoma multiforme, Arteriovenous malformation, Temporoparietal, Intraparenchymal hemorrhage

ÖZ

Glioblastoma multiforme santral sinir sisteminin primer tümörleri arasında en sık görülen tümör cinsidir. Glial tümörler beyinde farklı formlarda ortaya çıkabilmektedirler. Intraparankimal kanamalar ise, santral sinir sisteminin tüm tümörlerinde değişik derecelerde ve büyüklükte ortaya çıkabilmektedirler. 58 yaşında erkek hasta intraparankimal kanama nedeniyle şiddetli baş ağrısı ve kusma şikayetiyle kliniğimize başvurmuştur. Hastanın çekilen bilgisayarlı beyin tomografi görüntülerinde sağ posterior temporoparietal bölgede intraparankimal kanama tespit edilmiştir. Takiben yapılan serebral anjiyografisinde sağ inferior parietal bölge yerleşimli 3X2 cm boyutlarında arteriyovenöz malformasyon tespit edilmiştir. Hasta arteriyovenöz malformasyon tanısıyla cerrahi tedavi ile tedavi edilmiştir. Cerrahi sonrası histolojik tanı glioblastoma olarak gelmiştir. Bu tür hastaların yakın takiplerinin yapılması gerekmektedir.

ANAHTAR SÖZCÜKLER: Glioblastoma multiforme, Arteriyovenöz malformasyon, Temporoparietal, Intraparankimal hematoma

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Received : 01.12.2008

Accepted : 29.04.2009

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INTRODUCTION

Glioblastoma multiforme (GBM) is the most common intracranial neoplasm, accounting for 15% to 20% of all primary central nervous system tumors (1). Imaging characteristics of GBM are variable. However, most GBM lesions have revealed a pronounced mass effect and heterogeneous enhancement with centrally necrotic regions on computerized tomography (CT) (4). GBM is easily identified on MRI, but there are some non-characteristic findings that may be misinterpreted as non-neoplastic disease. Here we describe a case of glioblastoma multiforme with imaging findings mimicking arteriovenous malformation (AVM).

CASE REPORT

A 58-year-old man presented with sudden development of severe headache and vomiting. He was admitted to our emergency center immediately after the onset. His neurological examination was normal on admission. The patient's cranial computed tomographic scan (Figure 1) revealed a right posterior temporoparietal intraparenchymal hemorrhage upon the sylvian fissure. Cerebral angiography revealed a 3x2 cm right inferior parietal AVM fed exclusively by the right middle cerebral artery and draining into the right transverse sinus (Figure 2). There was no evidence of deep venous



Figure 2: Lateral cerebral angiogram of the right internal carotid artery revealing an AVM fed by the middle cerebral artery.

drainage. The patient was taken to the operating room, where he underwent a right temporoparietal craniotomy. The intraparenchymal hemorrhage and a hypervascular mass were resected via standard microsurgical techniques. Postoperative CT revealed no residual mass or hemorrhage (Figure 3), and the patient had no neurological deficits.



Figure 1: Computed tomography scan of the brain performed at admission showing a right posterior temporoparietal lobar hemorrhage.

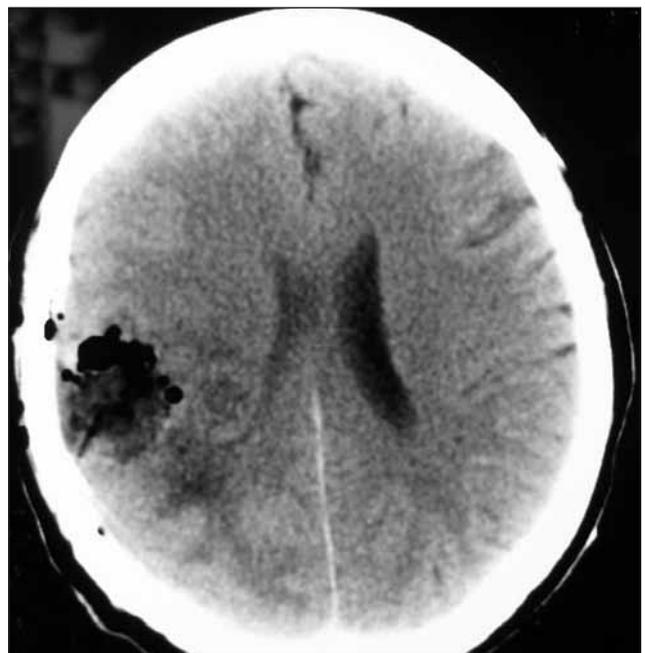


Figure 3: Postoperative CT image reveals successful evacuation of the lesion.

Histological examination of the hemorrhagic tissue revealed a tumor mass composed of neoplastic astrocytic cells along with blood vessels of various sizes and shapes. The neoplastic cells showed a high mitotic activity and Ki-67 proliferation index. Both large ischaemic necrosis and pseudopalisading necrosis were observed. Microvascular proliferation was also noted (Figure 4 A,B). The findings were compatible with the diagnosis of GBM, grade IV according to the WHO 2007 classification criteria.

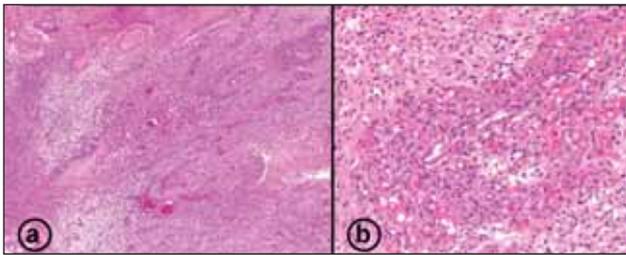


Figure 4: (A) Necrotic and hemorrhagic tumor tissue consistent with GBM (H&E, x40). (B) Highly cellular tumor tissue was composed of neoplastic astrocytic cells and microvascular proliferation was present (H&E, x200).

DISCUSSION

The frequency of intracranial hemorrhage in patients with intracranial neoplasms varies from 2% to 5% in different series. It may occur in all central nervous system tumors to a varying degree and extent and may even be massive. Massive hemorrhage mostly occurs in more malignant tumors, such as glioblastoma or metastases. Previously, Can SM et al. reported a case where traumatic hemorrhage was found to originate in an underlying giant cell glioblastoma; and proposed that traumatic intracerebral hemorrhage occurring in an uncommon site should indicate a search for underlying non-traumatic causative factors such as brain tumor, even in the absence of history and clinical findings suggestive of tumoral growth preceding the onset of hemorrhage (2). Nonetheless, benign tumors, such as pituitary adenoma, pilocytic astrocytomas, and meningiomas may also bleed to the extent of a massive hemorrhage (9, 7).

Several mechanisms have been advocated to explain the pathogenesis of brain tumor related hemorrhages: endothelial proliferation with subsequent obliteration of the lumen, thin walled or poorly formed vessels, perivascular necrosis with

subsequent loss of vessel support, and presence of intratumoral arteriovenous fistulae (11,18). In our case, the hemorrhage could have resulted from the abnormal hyalinized vessel wall in the papillary zones or from the presence of numerous thin walled vessels in the solid areas.

Brain digital subtraction angiography is the most precise and reliable radiological examination for diagnosing AVMs. However, MRI offers additional details about the primary lesion, the type of vascular lesion present, and its differential diagnosis (6). AVMs characteristically have low intensity on T1-weighted MRIs and high intensity with a signal void on T2-weighted MRIs. The lesion can be enhanced intensely on contrast-enhanced MRI (19). On the other hand, MRI imaging characteristic of glioblastoma is usually infiltration with less well-defined borders, associated with significant surrounding edema. Glioblastoma is more likely to show heterogeneous enhancement, reflecting the histological heterogeneity associated with the typical cystic and necrotic changes, as demonstrated in the abnormal hypointensity on diffusion-weighted images (17, 21).

Lesions with a combination of anomalous vasculature and glioma are designated as angiogliomas. Intracranial angioma and glioma may arise either as two separated lesions or be present within the same mass (10). Ziyal IM et al. reported a case report where preoperative images of the right temporo-parietal lobe-localized lesion resembled a typical glioma. Following the operation, the final histopathological examination of the specimen revealed an arteriovenous malformation surrounded by a high-grade glioma (22). Angiogliomas have been carefully differentiated from cases of glioma with intensive microvascular proliferation or vascular tumors such as hemangioblastoma with prominent stromal astrogliosis mimicking astrocytoma (16). Vascular anomalies involved in the lesions are AVM (12) or cavernous hemangioma (13). The types of glioma in angioglioma are also diverse. They can be astrocytoma (13), pilocytic astrocytoma (10), pleomorphic xanthoastrocytoma (5), oligodendroglioma (15), mixed oligoastrocytoma (3), and subependymal giant-cell astrocytoma (8). Because the term angioglioma is a histological term and simply represents a low-grade highly vascular glioma, histopathological findings of our case were not matched with the angioglioma.

In the setting of acute hemorrhage, as observed in the patient presented in this report, these characteristic neuroimaging findings of GBM become obfuscated and a vascular lesion seems more likely on the basis of the initial imaging. In a 58-year-old man, the presence of acute hemorrhage on CT scans and an AVM appearance on angiography scans are suggestive of an arteriovenous vascular malformation. The diagnosis could be made histopathologically in our case. GBM has mimicked non-neoplastic cerebral lesions and primary brain lymphoma in the literature (14, 20). GBM's presentation in hemorrhage as AVM is very rare entity and has not been previously reported in the literature.

In conclusion, glial tumors can present in different forms. GBM should be considered in the differential diagnosis of intracranial hemorrhage, especially in the absence of predisposing factors. The association of tumors and vascular malformations should always be taken into consideration and preoperative radiological investigation should be performed explicitly in cases that have intraparenchymal hemorrhage.

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