CASE REPORTS

Childhood Glioblastoma Multiforme Without Peritumoral Edema

Çocukluk Çağında Peritümöral Ödemi Bulunmayan Glioblastoma Multiforme

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Abstract: Glioblastoma multiforme comprises 15-20 % of all intracranial tumours. It makes a peak between the fifth and seventh decades of life but it is rare in childhood. One of the radiological and clinical properties of glioblastoma multiforme is perifocal vasogenic oedema associated with the tumour. In the radiological evaluation of an 8 -year-old boy with glioblastoma multiforme, perifocal oedema did not accompany the lesion. The case is discussed with the cases in the literature.

Key Words: Computed tomography, glioblastoma multiforme, magnetic resonance imaging, peritumoral oedema.

Özet: Glioblastoma multiforme tüm kafa içi tümörlerin %15-20'sini oluşturur, beşinci ve yedinci onyılllar arasında sık rastlanır, çocuklukta nadirdir. Glioblastoma multiforme'nin radyolojik ve klinik özelliklerinden biri de tümöre eşlik eden değişik derecelerdeki perifokal vazojenik ödemdir. Glioblastoma multiforme'si olan 8 yaşındaki bir erkek çocuğun radyolojik değerlendirilmesinde perifokal ödemin bulunmaması dikkati çekmiş ve olgu literatürdeki benzer olgularla birlikte tartışılmıştır.

Anahtar sözcükler: Bilgisayarlı tomografi, glioblastoma multiforme, manyetik rezonans görüntüleme, peritümöral ödem

INTRODUCTION

Glioblastoma multiforme (GM) is the most common tumour of the central nervous system (CNS). It characteristically invades the white matter and is rare in childhood (8).

GM is generally surrounded with a peritumoral vasogenic oedema (8), caused by an abnormal increase in the vascular permeability of the tumour by neovascularisation (9).

An important portion of GMs have perifocal vasogenic oedema in computerized tomography (CT) and generally magnetic resonance imaging (MRI) shows perifocal oedema in T2 weighted images (9).

CASE REPORT

An 8-year- old boy was admitted with a 3 months history of headache and vomiting.

Neurological examination revealed right papilloedema, slight left hemiparesis and extensor plantar response on the left side. There was no pathological changes in the skull x-rays. CT revealed a heterogeneous right temporal mass with a hyperdense lesion in the center consistent with intratumoral haemorrhage (Figure 1). After intravenous contrast injection a 74x62.5 mm mass, contrast enhancing especially at the periphery was observed (Figure 2). An MRI was performed. T1- weighted images revealed a right temporal mass with a large

hyperintense haemorrhagic center and hypointense peripheric solid compartments (Figure 3). Proton density- weighted axial images revealed a right temporal solid mass with heterogeneous center and homogeneous hyperintense periphery. The central hyperintense lesion in T1- weighted and proton-density weighted images was concluded to be intratumoral haemorrhage and the homogeneous hyperintense periphery was the solid portion of the tumour (Figure 4).

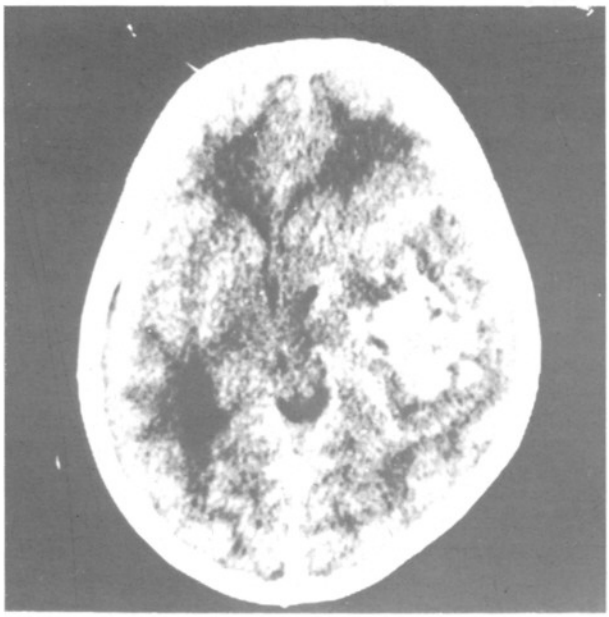

Figure 1: Non-contrast CT showing a right temporal mass with an haemorrhagic hyperdense center. The mass is heterogeneous in density and causing midline shift.

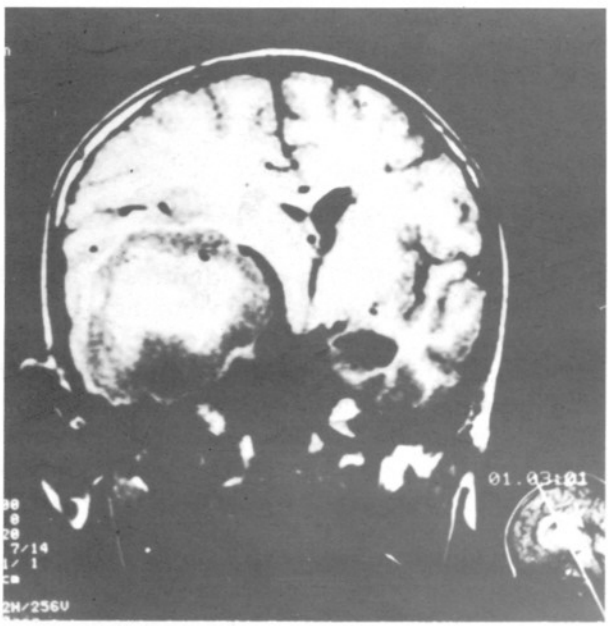

Figure 3: T1-weighted coronal MRI image showing a right temporal mass with an haemorrhagic hyperintense center and causing a midline shift.

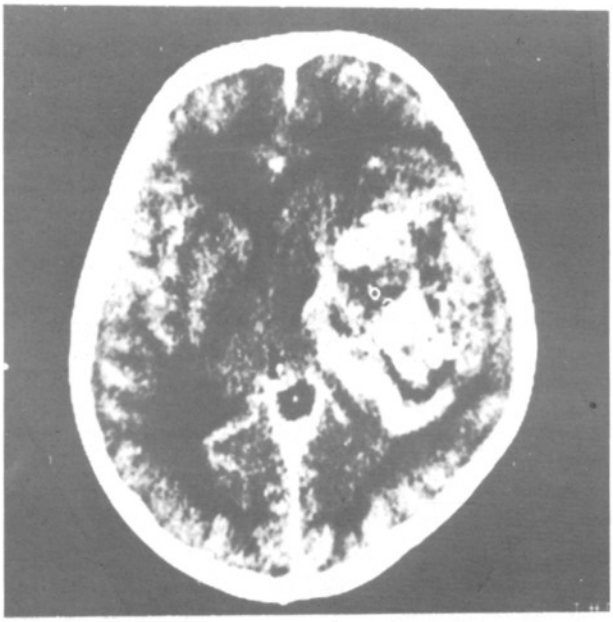

Figure 2: Contrast enhanced CT showing prominent contrast enhancement at the tumour periphery. The tumour shows heterogeneous contrast enhancement and there are some non-enhanced parts at the center.

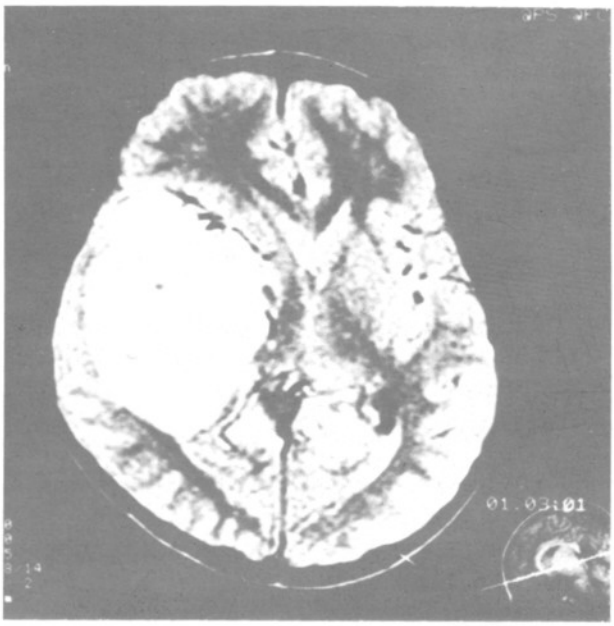

Figure 4: Proton density weighted axial MR image showing a right temporal mass with heterogeneous hyperintense haemorrhagic center. The peripheral zone of the tumour surrounding the haemorrhagic and necrotic center is homogeneously hyperintense.

With CT and MRI findings and considering the patient's age, an initial diagnosis was astrocytoma or ganglioglioma. The tumour was excised subtotally. Giant cells, vascular proliferation and necrosis were observed in the tumoral tissue and histopathological diagnosis was GM (Figure 5a). In the transition zone between the tumour and brain parenchyma no prominent oedema was observed microscopically (Figure 5b).

The patient was discharged at the tenth post-operative day in good condition and transferred to an oncology department for post-operative radiotherapy.

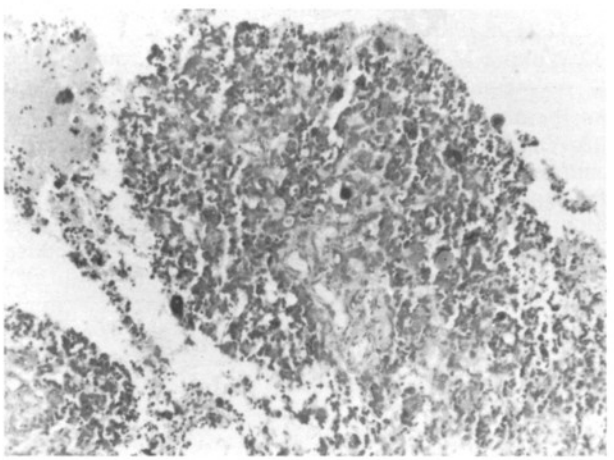

Figure 5a: Microphotograph of the tumour showing giant cells, vascular proliferation and necrosis. (H&E,X200)

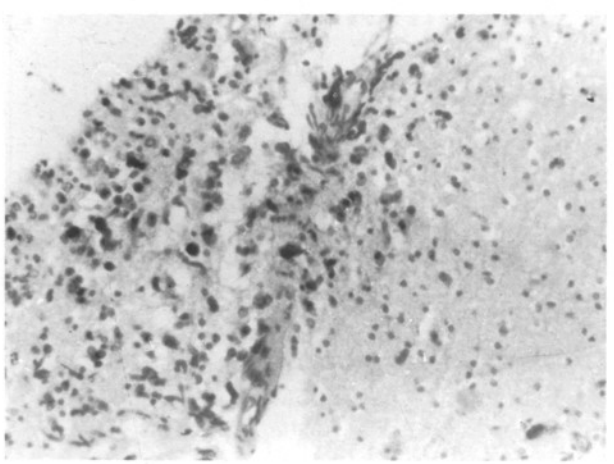

Figure 5b: Microphotograph showing the transition zone between the tumour and brain parenchyma. No prominent oedema was observed microscopically. (H&E,X 400)

DISCUSSION

GM is the most common primary CNS tumour (5). It peaks between the fifth and seventh decades of life and is more rare in childhood (6,8).

GMs are surrounded with a heterogeneous rim in CT. Necrotic fields are common in the center. Cysts and haemorrhagic fields are more rare. The tumour enhances except the necrotic fields. In more vascular tumours peripheral enhancement can be observed. This is usually heterogeneous and more than 5 mm. This feature can be helpful in radiological differential diagnosis of GMs with cerebral abscess (9). In 5% of cystic tumours a thin rim enhancement can be observed but contrast enhancement is rarely seen in the center of the cysts (8).

GMs have big, thin-walled, thrombotic and/or haemorrhagic vessels. Small vessels, especially capillaries show characteristic hyperplastic changes especially prominent in the tumour periphery and in parallel with the contrast enhancement at the tumour's periphery at CT (3).

Vasogenic edema accompanying mass lesions of the brain is a known condition. The responsible physiopathological mechanisms are; a) permeability of the new tumoral microvessels occurring with tumoral angiogenesis, b) changes in the microvascular permeability, by the excreted tumoral substances, c)immunological changes, d) increase of the microvascular permeability by the inflammation (2).

By the mechanisms explained above vasogenic oedema widens the subcortical white matter and causes "digital pseudopod or garland edema". This mild or prominent peritumoral edema is characteristic for GM (1,8-11), but was not observed in our case (Figures 1, 5b).

In CT, there is perifocal vasogenic oedema in 88% of GMs and it is usual in T2- weighted images (9). In glioma cases peritumoral edema may show a correlation with the tumoral malignancy (4,11).

Our case can be concluded to be a rare case of GM because of the age of the patient, absence of prominent peritumoral edema on CT and MRI, and also histopathological examination revealed no prominent edema microscopically. The absence of peritumoral edema can be attributed to changes in the vasogenic edema mechanisms.

In a review of the literature we could not find a case of childhood GM without peritumoral oedema (1,6,7,10,12,13).

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