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Case Presentation

IDH-WILDTYPE GLIOBLASTOMA WITH LEPTOMENINGEAL CARCINOMATOSIS: A CASE REPORT

LEPTOMENİNGEAL KARSİNOMATOZLU IDH-WILDTYPE GLIOBLASTOM: BİR VAKA SUNUMU

Muhammet Kırkgeçit[1] **Hasan Türkoğlu**[2] **Recai Engin**[3]

[1] Department of Neurosurgery, Kahramanmaraş Sütçü İmam University Faculty of Medicine, Kahramanmaraş, Türkiye.
[2] Department of Neurosurgery, Gaziantep City Hospital, Gaziantep, Türkiye.
[3] Department of Neurosurgery, Samsun University Faculty of Medicine, Samsun, Türkiye.

***Corresponding Author:** Muhammet Kırkgeçit mkirkgecit46@gmail.com

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ABSTRACT

Glioblastoma is the most common malignant tumor of the central nervous system. Leptomeningeal dissemination is rare and usually occurs in the late stages of the disease. We report a 26-year-old man diagnosed with IDH-wildtype, WHO grade 4 glioblastoma located in the right thalamus. Despite radiological regression of the tumor after chemoradiotherapy, the patient developed paraparesis seven months later. Spinal magnetic resonance imaging showed diffuse leptomeningeal enhancement extending from C1 to T9, consistent with leptomeningeal carcinomatosis. The patient received palliative treatment and subsequently died due to pneumonia-related complications. Although leptomeningeal dissemination of glioblastoma is uncommon, it is associated with rapid progression and poor prognosis. Therefore, spinal dissemination should be considered in patients with new-onset back pain or limb weakness, even in the presence of radiological regression of the primary tumor.

Keywords: Glioblastoma, Leptomeningeal carcinomatosis, Spinal seeding, Hydrocephalus

ÖZET

Glioblastoma, merkezi sinir sisteminin en sık görülen kötü huylu tümörüdür. Leptomeningeal yayılım nadir görülür ve genellikle hastalığın geç evrelerinde ortaya çıkar. Bu olguda, sağ talamusta yerleşimli IDH-yabanıl tip, WHO derece 4 glioblastoma tanısı alan 26 yaşındaki erkek hasta sunulmaktadır. Kemoradyoterapi sonrasında yapılan manyetik rezonans görüntülemede tümörde gerileme izlenmesine rağmen, yedi ay sonra hastada paraparezi gelişti. Spinal manyetik rezonans görüntüleme, C1–T9 seviyeleri boyunca uzanan yaygın leptomeningeal kontrastlanma ile uyumlu leptomeningeal karsinomatozisi gösterdi. Hasta palyatif tedavi aldı ve daha sonra pnömoniye bağlı komplikasyonlar nedeniyle kaybedildi. Glioblastomanın leptomeningeal yayılımı nadir olmakla birlikte hızlı klinik ilerleme ve kötü prognoz ile ilişkilidir. Bu nedenle, primer tümörde radyolojik gerileme saptansa dahi, yeni başlayan sırt ağrısı veya ekstremite güçsüzlüğü gelişen hastalarda spinal yayılım olasılığı göz önünde bulundurulmalıdır.

Anahtar Kelimeler: Glioblastom, Leptomeningeal karsinomatozis, Spinal yayılım, Hidrosefali

Introduction

Glioblastoma is the most common primary malignant tumor of the central nervous system (1, 2). Leptomeningeal metastases from intracranial high-grade gliomas, including glioblastoma, are rare. Autopsy series show that approximately 25% of intracranial glioblastoma patients have spinal subarachnoid extension, but the exact incidence is unknown because postmortem examination of the spine is not routinely performed (1, 2). In this case report, we describe a patient with isocitrate dehydrogenase (IDH)-wildtype, World Health Organization (WHO) grade 4 glioblastoma in the right thalamus who developed spinal extension with paraparesis approximately seven months after chemotherapy and radiotherapy (RT).

Case Report

A 26-year-old man was admitted to the hospital with complaints of headache, nausea and vomiting for three weeks and intermittent diplopia for three days. Contrast-enhanced brain magnetic resonance imaging (MRI) revealed an 18 × 16 mm lesion with heterogeneous contrast enhancement in the right thalamus (Figure 1). The lesion was interpreted as a high-grade glial tumor after a choline peak was observed on MRI spectroscopy. The patient underwent stereotactic brain biopsy. Histopathological examination confirmed IDH-wildtype WHO grade 4 glioblastoma according to WHO 2021 CNS tumor classification. Immunohistochemical examination revealed loss of alpha-thalassemia/mental retardation syndrome X-linked (ATRX) expression and glial fibrillary acidic protein (GFAP) positivity. The patient received temozolomide chemotherapy and postoperative radiotherapy. Control brain MRI showed shrinkage of the mass in the right thalamic region and marked decrease in contrast enhancement, which was interpreted as tumor regression (Figure 2). Four months after the initial diagnosis, the patient presented with nausea, vomiting, and headache and was found to have obstructive hydrocephalus due to the lesion. A ventriculoperitoneal (VP) shunt was placed in the patient whose funduscopic examination revealed papilledema (Figure 3). The patient was discharged after improvement in general condition.

Seven months after the initial diagnosis, the patient presented to the hospital with the complaint of inability to walk and weakness in both legs. On examination, it was found that bilateral muscle strength in the lower extremities was 3/5, deep tendon reflexes were increased, bilateral Babinski signs and ankle clonus were present. In the whole spinal MRI examination of the patient, hypointensity on T1-weighted images and hyperintensity on T2-weighted images were seen in the spinal cord along the C1–T9 vertebral level, causing enlargement of the spinal cord. Contrast enhancement was observed in the posterior part of the spinal cord between the thoracic second and third vertebrae (T2–T3) and between the thoracic fourth and fifth vertebrae (T4–T5). In addition, intense contrast enhancement was seen throughout the cerebrospinal fluid (CSF) spaces observed after contrast and this was considered to be compatible with leptomeningeal carcinomatosis (Figure 4). Surgery was not planned for the current condition, and the patient was discharged after consultation with medical oncology and radiation oncology.

Three months after discharge, the patient died in the intensive care unit from complications of SARS-CoV-2-related pneumonia.

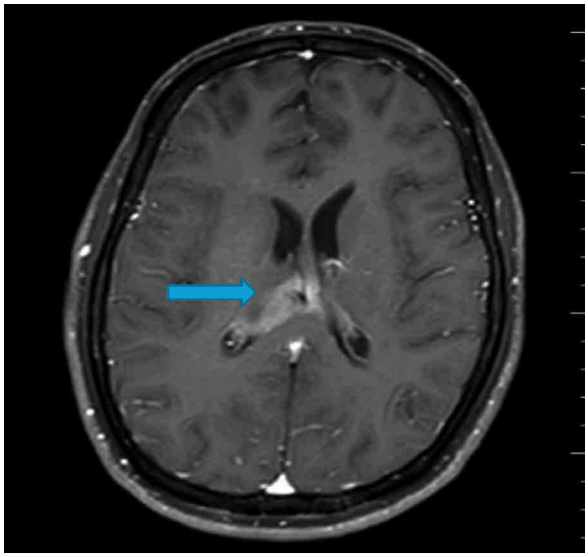

Figure 1. Preoperative contrast-enhanced axial T1-weighted MRI showing an 18 × 16 mm right thalamic mass. MRI: magnetic resonance imaging.

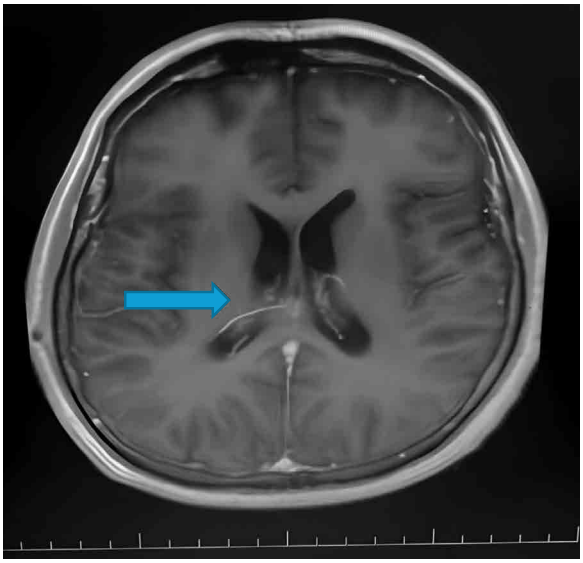

Figure 2. Contrast-enhanced axial T1-weighted MRI obtained three months after radiotherapy, showing decreased enhancement of the right thalamic mass. MRI: magnetic resonance imaging.

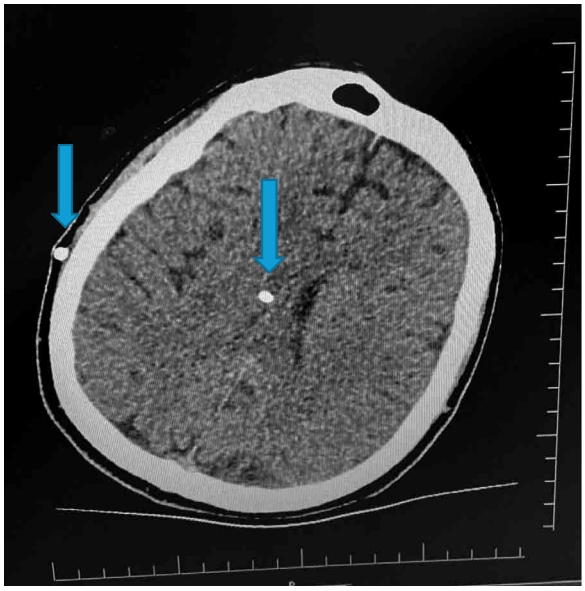

Figure 3. Cranial computed tomography image obtained after ventriculoperitoneal shunt placement for hydrocephalus. VP: ventriculoperitoneal.

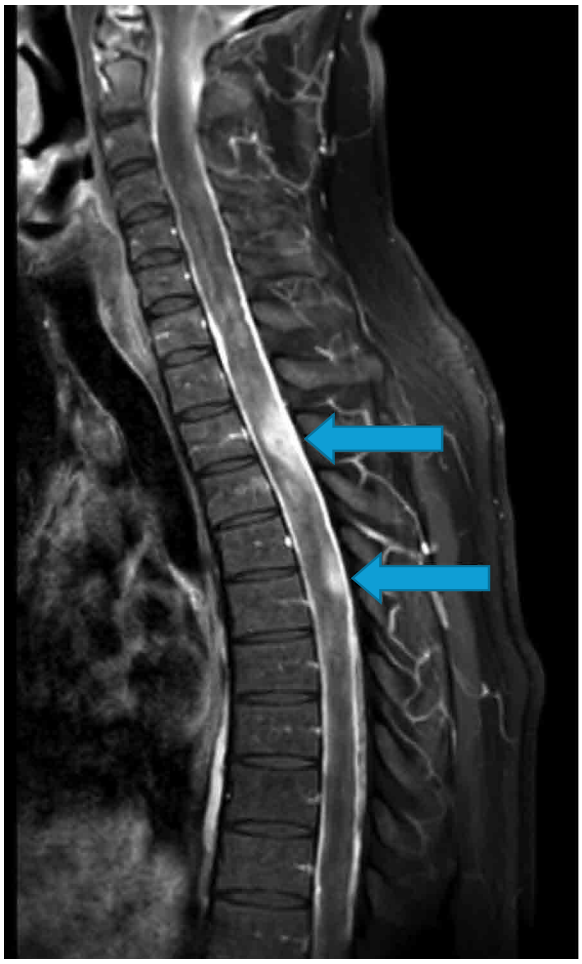

Figure 4. Spinal MRI showing diffuse leptomeningeal enhancement and spinal cord enlargement from C1–T9. MRI: magnetic resonance imaging.

Discussion

Spinal spread of high-grade gliomas was first described in 1931 (3). Spinal spread is observed in 15-25% of glioblastoma cases (1, 4). However, a higher incidence of spinal spread (up to 60%) has been recorded for infratentorial glioblastoma. The rate of leptomeningeal dissemination in glioblastoma cases varies (5). In a study of 600 patients with intracranial glioblastoma, only 2% reported symptomatic spinal dissemination (6). Intramedullary spinal metastases are rare (7). These results suggest that symptomatic involvement occurs relatively late during glioblastoma.

In a study by Grabb et al. on supratentorial gliomas in children, opening to the ventricle at surgery, repeated surgical procedures and male gender were associated with a statistically significant increased incidence of dissemination to CSF (7). Ependymal invasion, fissuring of the ependymal area due to hydrocephalus and tumor disintegration in contact with CSF are also risk factors for dissemination to CSF (3). Repeated tumor resection poses an even greater risk of CSF dissemination due to repeated tumor manipulation, more aggressive tumor cell types, and suppressed immune function after radiotherapy and chemotherapy (8).

In our case, the patient was being followed up with intracranial IDH-wildtype WHO grade 4 glioblastoma and presented with symptomatic spinal dissemination seven months after the initial diagnosis. The reason for this spread suggested that the mass tended to spread from the moment it was first diagnosed. Because spinal spread was detected in the follow-up of the patient despite a decrease in contrast uptake in the contrast-enhanced brain MRI examination requested for control after chemotherapy and RT treatments.

The most common sites of spinal glioblastoma metastases are the lower thoracic, upper lumbar and lumbosacral regions (7, 8). The most common clinical features of leptomeningeal disseminations are radiculopathy and back pain. This is often followed by paraparesis or rarely quadriparesis (8). These symptoms were also observed in our case.

Most large series describe patients with meningeal and spinal dissemination discovered at autopsy (1, 2). Involvement associated with pain and/or neurologic deficit is usually confirmed by imaging studies.

Recommended treatment options for leptomeningeal spread are limited. Surgical intervention may be attempted if there is a symptomatic, large mass causing spinal cord compression but generally leptomeningeal dissemination is not amenable to surgery due to the diffuse nature of the disease (9). The treatment modality for these patients is mainly palliative. Spinal RT is the most commonly used treatment modality with a total dose range of 25-40 Gy (10).

When leptomeningeal spread is detected, it almost always results in death. The median time from diagnosis of primary intracranial glioblastoma to diagnosis of spread to the CSF is 8 to 14 months. The median survival ranges from 11 to 17 months. The mean interval between the diagnosis of leptomeningeal spread and death is 2 to 3 months (8). In our case, leptomeningeal dissemination occurred in a shorter time (7 months) compared to the literature.

Conclusion

Leptomeningeal symptomatic spinal disseminations from supratentorial high-grade gliomas are relatively rare. In the case of high-grade gliomas with recent onset of limb and back pain and/or associated spinal root nerve symptoms should alert clinicians to consider leptomeningeal and/or spinal spread of glioblastoma. Treatment of spinal seeding metastases remains palliative and generally the prognosis is not good.

Disclosures

Peer-review: Externally peer-reviewed.

Informed Consent: Written informed consent for publication of this case report and accompanying images was obtained from the patient's legal next of kin following the patient's death.

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