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VASOGENIC EDEMA AND HYDROCEPHALUS AS COMPLICATIONS OF GLIOBLASTOMA

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Abstract

Glioblastoma is the most aggressive and common primary brain tumor in adults, characterized by rapid growth, infiltrative behavior, and poor prognosis despite multimodal treatment. Among its most significant complications are vasogenic edema and hydrocephalus, which exacerbate intracranial hypertension and contribute substantially to morbidity and mortality. We report the case of a 59-year-old male who presented with progressive decreased consciousness, severe headaches, right-sided weakness, and visual disturbances. Neuroimaging revealed a left temporoparietal mass with cystic, solid, and necrotic components, accompanied by extensive vasogenic edema, midline shift, and obstructive hydrocephalus. Histopathological examination confirmed glioblastoma, WHO grade IV. The patient was treated with intravenous dexamethasone and acetazolamide, resulting in improved consciousness and partial symptomatic relief. This case illustrates the pathophysiological role of blood–brain barrier disruption in vasogenic edema and cerebrospinal fluid flow obstruction in hydrocephalus. Dexamethasone remains the cornerstone of symptomatic management for edema, while acetazolamide may provide temporary benefit in hydrocephalus, although its long-term efficacy is limited. The coexistence of these complications significantly worsens prognosis, and treatment primarily focuses on symptom control and quality of life. Prompt recognition and appropriate management are essential to stabilize patients, even though overall survival in glioblastoma remains limited..

Keywords: vasogenic edema, hydrocephalus, neurological disorders, glioblastoma

INTRODUCTION

As the most aggressive form of glioma, glioblastoma is classified by the World Health Organization (WHO) as grade IV (Louis et al., 2021) and represents the most common primary brain tumor in adults (Li et al., 2023). This neoplasm has an incidence of 3–4 cases per 100,000 population annually, with a median survival of only about 15 months (Thakkar et al., 2014). Its invasive growth and high progression rate limit therapeutic responses to multimodal treatment, including surgery, radiotherapy, and chemotherapy (Jiang et al., 2023).In glioblastoma, vasogenic edema is a frequent complication caused by increased capillary permeability around the tumor. The resulting extracellular fluid accumulation leads to raised intracranial pressure, causing headaches, nausea, vomiting, and focal neurological deficits. This makes vasogenic edema one of the major contributors to morbidity, and a key therapeutic target through corticosteroids or decompressive procedures (Ohmura et al., 2023). In addition, glioblastoma can also lead to obstructive hydrocephalus due to impaired cerebrospinal fluid (CSF) flow, further aggravating intracranial hypertension, reducing consciousness, and potentially causing brain herniation if untreated (Alfayate et al., 2025). Understanding the clinical manifestations and management of vasogenic edema and hydrocephalus is essential in glioblastoma care, as these complications often determine both patient quality of life and survival. This case report discusses a patient with glioblastoma complicated by vasogenic edema and hydrocephalus, along with the diagnostic and therapeutic approaches undertaken.

CASE REPORT

We present the case of a 59-year-old male who presented with decreased consciousness for six days prior to admission. Initially, the patient appeared somnolent but was still arousable by loud verbal stimuli. Three days before admission, his level of consciousness further declined, and he could only open his eyes without attending to

interlocutors. Communication became impaired, and he occasionally produced moaning sounds without forming words. The patient also reported headaches for four months, initially intermittent and relieved by over-the-counter medications, but becoming persistent during the past month, particularly in the mornings. The pain was localized to the left side of the head, throbbing in nature, progressively worsening, and associated with vomiting. Family members reported progressive weakness of the right extremities beginning one month earlier, eventually leading to complete immobility. The patient also exhibited communication difficulties, including incoherent speech, forgetfulness, and word-finding difficulty. Additionally, he complained of right-sided visual field loss, requiring head turning to view objects on that side. Elimination (urination and defecation) remained normal, and there was no diaphoresis. However, he experienced unintentional weight loss of approximately 5 kg over one month. He denied anosmia, diplopia, hearing loss, tinnitus, history of head trauma, ear or dental infection, seizures, fever, chronic cough, hemoptysis, or dyspnea. On arrival at the emergency department, he was stuporous with a Glasgow Coma Scale (GCS) score of 10, a Karnofsky score of 30, right-sided motor deficits, and intact doll's eye reflex. Blood pressure was 167/102 mmHg, and pulse rate was 62 bpm. A non-contrast head CT scan (Figure 1) revealed an inhomogeneous mass (predominantly cystic with solid components and prominent calcifications in both peripheral and central regions) in the left parietal lobe. The mass caused midline shift of approximately 1.17 cm to the right, narrowing of the left ventricle, and trapped right ventricular effusion. The patient was started on intravenous dexamethasone 10 mg every 6 hours (tapered) and acetazolamide 250 mg twice daily.

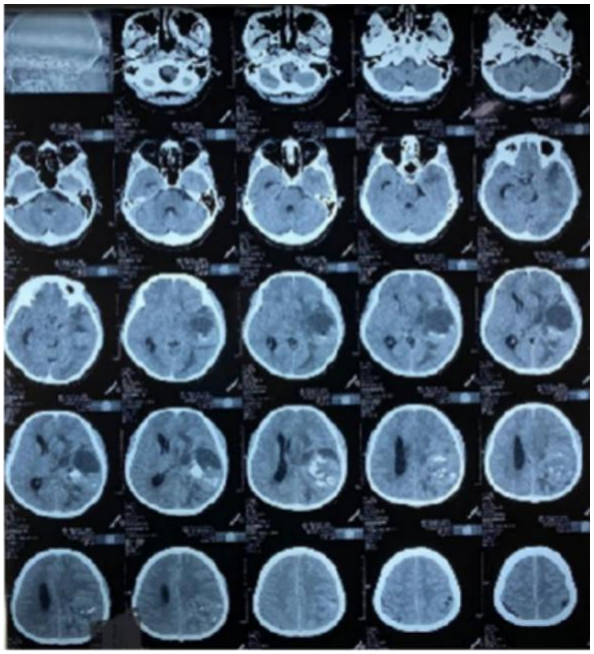

Figure 1. CT SCAN

On day 3, contrast-enhanced brain MRI (Figure 2) showed a well-defined but irregular supratentorial intra-axial lesion in the left temporoparietal lobe with cystic, solid, hemorrhagic, and necrotic components, surrounded by tentacle-like edema compressing the left lateral and third ventricles, and causing 1.8 cm rightward subfalcine herniation. The lesion was supplied by branches of the left MCA (segment 2), consistent with high-grade astrocytoma. Right lateral ventriculomegaly was also noted. Histopathological analysis confirmed glioblastoma, WHO grade IV. By day 5, the patient had regained full consciousness (GCS 15). Intravenous dexamethasone 5 mg every 12 hours and acetazolamide 250 mg twice daily were continued.

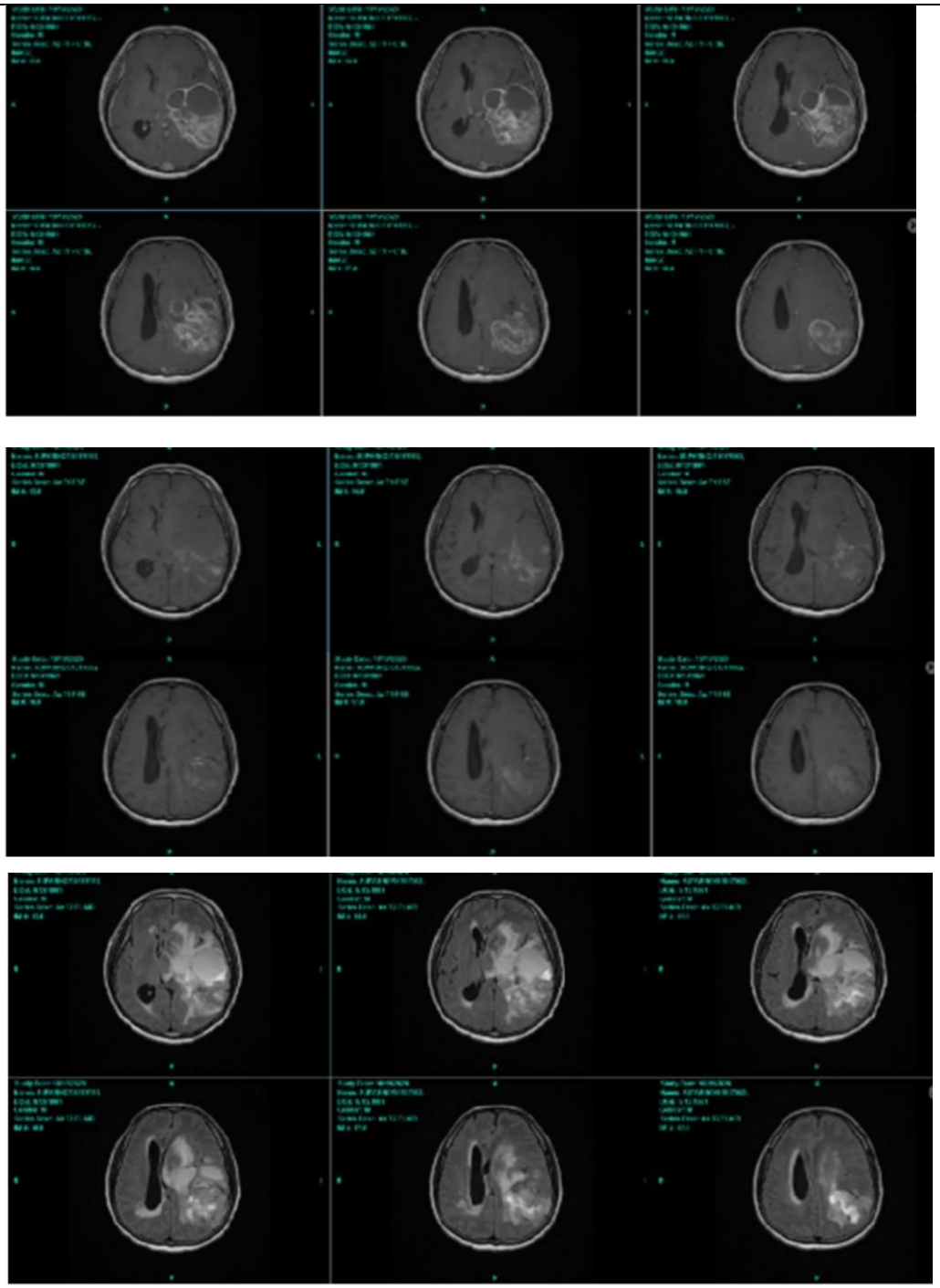

Figure 2. MRI brain

RESULTS AND DISCUSSION

Glioblastoma is a primary brain tumor with a dismal prognosis, characterized by infiltrative behavior and rapid growth, frequently leading to complications (Louis et al., 2021). Vasogenic edema is a major complication caused by disruption of the blood–brain barrier by tumor cell infiltration, allowing plasma fluid to leak into the extracellular space. This leads to increased tissue volume and mass effect, ultimately raising intracranial pressure and producing focal neurological symptoms (Ohmura et al., 2023). Vasogenic edema in glioblastoma is commonly managed with corticosteroids such as dexamethasone, which reduces capillary permeability and provides rapid symptomatic relief. However, this treatment does not alter tumor progression. In this patient, dexamethasone successfully reduced symptoms of intracranial hypertension (Scheffler et al., 2024). Shahrabi et al. (2025) recommend an initial intravenous bolus of dexamethasone 10 mg, followed by maintenance dosing of 16–20 mg

daily, tapered according to clinical response. In addition to edema, obstructive hydrocephalus may occur due to impaired CSF flow caused by tumor mass effect or ventricular infiltration. Hydrocephalus worsens intracranial pressure and can be managed pharmacologically with acetazolamide, which reduces CSF production and helps prevent further neurological damage (Del Bigio, 2016). Clinical studies have shown improvement with oral acetazolamide at daily doses of 250–500 mg (Alperin, 2014). Nonetheless, acetazolamide is not effective for long-term management of chronic hydrocephalus and carries risks of metabolic complications (Barbuskaite et al., 2022). The coexistence of vasogenic edema and hydrocephalus significantly exacerbates clinical outcomes in glioblastoma patients. Management of these complications aims primarily to improve quality of life, but overall prognosis remains poor, with median survival of approximately 15 months despite multimodal treatment (Thakkar et al., 2014).

CLOSING

Conclusion

Glioblastoma remains a highly aggressive brain tumor with limited survival despite multimodal treatment. Vasogenic edema and hydrocephalus are critical complications that aggravate intracranial hypertension, worsen neurological function, and significantly influence patient outcomes. In this case, timely administration of dexamethasone and acetazolamide provided symptomatic improvement, underscoring the importance of early recognition and supportive management. Although these interventions do not alter the natural course of glioblastoma, they play a crucial role in stabilizing patients and improving short-term quality of life. Future studies are needed to explore more effective strategies to address these complications and optimize care for glioblastoma patients.

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