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# Secondary glioblastoma with abdominal metastasis: Case report

*Ana María Granados PhD*[a], *Natalia Zuñiga MD*[b], *Camila Ospina MD*[c,*], *Lina Merchancano MD*[b]

[a] *Neuroradiology, Fundación Valle del Lili, Cali, Colombia*
[b] *Radiology department, Fundación Valle del Lili, Cali, Colombia*
[c] *Center for Clinical Research, Fundación Valle del Lili, Av. Simón Bolívar, Carrera 98 # 18-49, Cali 760032, Colombia*

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ABSTRACT

Extracranial glioblastoma (GB) metastasis is an uncommon entity, rarely described in the literature, representing 0.2% of cases of GB. Several theories have been proposed to explain the extracranial dissemination of GB, such as surgical interventions, ventriculoperitoneal shunt, and radiation therapy. We present a case of a 15-year old adolescent girl, with an initial diagnosis of low-grade glioma and later transformation to a high-grade glioma. In the final phase of the disease, the patient presented with distention and abdominal pain, secondary to peritoneal compromise of GB metastasis. The use of new therapies has increased survival times, leading to a rise in the probability of developing extracranial metastasis.


## Introduction

Glioblastoma (GB) accounts for 15% of all primary brain tumors. It has a poor prognosis with a 5-year survival rate of 4.7%. The cause of death is usually due to tumoral intracranial compromise, rather than metastasis. Only 0.2% of patients with GB develop extracranial metastasis with a 2-year median time of presentation [1].

It is believed that GB metastasis is very unlikely because of the presence of the blood-brain barrier, the absence of brain lymphatic system, and poor prognosis. However, treatment improvements, such as complete surgical resection and concomitant radiotherapy-chemotherapy, have increased the survival time, therefore increasing the probability of extracranial dissemination [2].

Regarding the physiopathology leading to extracranial metastasis, several hypotheses have been proposed, including direct access to the dural vessels during surgical interventions, invasion of the dura mater and bone, and migration through the ventriculoperitoneal shunt [2]. Another aspect that must be taken into consideration is the count of circulating tumoral cells, which are an extravasation of GB cells to vascular space, originating from hematogenous dissemination and implantation of GB on extracranial areas. The presence of tumoral circulant cells has been detected in 20% of GB cases; however, not all patients develop metastases [3]. Additionally, it has been proposed that there are enzymatic factors that predispose to

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* *Corresponding author.*
E-mail address: cami8491@gmail.com (C. Ospina).

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extracranial spreading: gelatinase A and B are proteolytic enzymes that degrade the extracellular matrix of the central nervous system, promoting spreading; these enzymes also degrade the basal membrane of the blood vessels [4]. Forsyth et al. found higher expression of the active form of these enzymes in GB with extraneural metastasis [5,6].

A significant theory is the generation of radiation-induced glial cell sarcomatous metaplasia, which develops extracellular matrix proteins associated with vascular and hematogenous invasion. Several molecular characteristics have also been found, such as P53 mutations, gene *PTEN*, P16 deletion, and MDM2 and CDK4 amplifications [7].

We present the case of a 15-year-old adolescent girl with an initial diagnosis of low-grade glioma, which subsequently transformed to a high-grade glioma, with later development of abdominal metastasis.

## Case presentation

A 15-year-old adolescent girl presented to our institution with a 1-month history of a mild global headache, more predominantly in the mornings. Her physical examination was unremarkable with no neurologic findings. A brain magnetic resonance imaging (MRI) was performed, revealing a mass localized on the right basal ganglia, predominantly in the thalamus, associated with midline deviation and perilesional

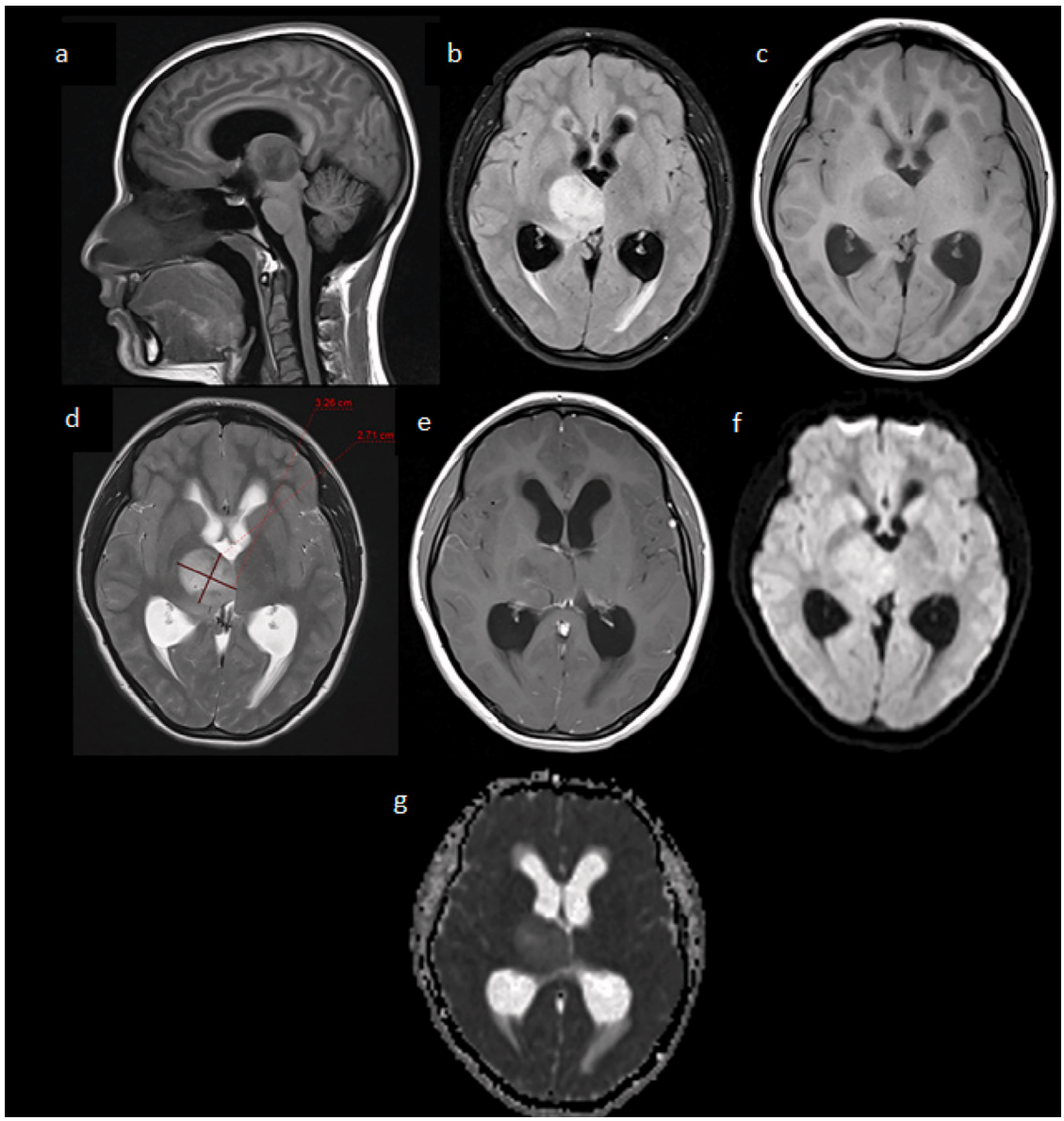


**Fig. 1 – Sagittal T1-FLAIR-weighted image (A), axial FLAIR-weighted image (B), axial T1-weighted image (C), axial T2-weighted image (D), axial T1 contrast-enhanced image (E), axial diffusion-weighted image (F), and axial apparent diffusion coefficient image (G). A right basal ganglia mass-type lesion is shown, with compressive effect over the third ventricle and midline deviation. The mass lesion is hyperintense on T2 and FLAIR images, hypointense on T1, and no restriction with diffusion-weighted imaging and mild postcontrast enhancing. FLAIR, fluid-attenuated inversion recovery.**