Glioblastoma Masquerading as a Hypertensive Putaminal Hemorrhage: A Diagnostic Pitfall
—Case Report—

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Abstract

A 58-year-old man presented with a rare case of glioblastoma masquerading as intracerebral hemorrhage (ICH). He had been medicated for hypertension and diabetes for 10 years before collapsing at home. Brain computed tomography (CT) showed ICH in the right putamen, but CT with contrast medium showed no underlying lesion. He was treated initially with intravenous administration of anti-hypertensive agent under a diagnosis of hypertensive putaminal hemorrhage. ICH aspiration surgery was performed, and serial CT showed ICH resorption. However, he was again admitted for unstable gait and mildly altered mental status 3 months after discharge. Magnetic resonance (MR) imaging with gadolinium showed an enhanced ring-shaped mass around the hematoma cavity. Open biopsy was performed. The histological diagnosis was glioblastoma multiforme, and he was treated with radiation therapy and oral chemotherapy with temozolomide. MR imaging showed marked shrinkage of the tumor, but he died of pneumonia 3 months after the second surgery. In this case, the cause of the hemorrhage was not identified after the seemingly successful hematoma evacuation surgery, and no definitive diagnosis was made until tumor regrowth. Brain tumor should be suspected as a cause of ICH even if the patient has a history of hypertension and the location is typical for hypertensive ICH. Clinical/radiological follow up is essential for detecting subtle neurological deterioration to avoid diagnostic delay.

Key words: brain tumor, hypertension, putaminal hemorrhage, intratumoral hemorrhage, diagnosis

Introduction

Brain tumor is a relatively rare but important cause of intracerebral hemorrhage (ICH).2,4,7) Brain tumor accounted for 4.4% to 7.2% of non-traumatic ICHs,6,9) whereas major intratumoral hemorrhage occurred in 1.5% to 2.9% of patients with brain tumor.5,9) The incidence of intratumoral hemorrhage depends mainly on the tumor histology and location.2,4,10,11) Intratumoral hemorrhage can usually be identified preoperatively by imaging studies, as the border between the tumor and hemorrhage is usually clear,2,8) but if the hemorrhage is large and expansive enough, the tumor may be compressed and thus not visualized, even if contrast material is used. Therefore, intratumoral hemorrhage may be indistinguishable from spontaneous ICH, and any delay in diagnosis may adversely affect the prognosis for the patient.

We describe a case of glioblastoma masquerading as hypertensive putaminal hemorrhage, which resulted in diagnostic delay.

Case Report

A 58-year-old man who had been medicated for hypertension and diabetes for 10 years was brought to the emergency department after collapsing at home. He lived alone so the timing was unclear. He showed altered mental status with a Glasgow Coma Scale score of E3V4M5, and left hemiparesis. Blood pressure was 183/111 mmHg. Brain computed tomography (CT) showed ICH in the right putamen (Fig. 1A). CT with contrast medium was performed because of the presence of mild brain edema, but no underlying lesion was identified (Fig. 1B). He was treated initially with intravenous administration of anti-hypertensive agent under a diagnosis of hypertensive putaminal hemorrhage.

ICH aspiration surgery was performed the next day by inserting a drainage tube into the hematoma cavity, with
the use of a Komai stereotactic frame. He made a steady recovery, and serial CT showed ICH resorption (Fig. 1C). He could walk with the aid of brace and cane after rehabilitation, and was transferred to a local rehabilitation facility 40 days after surgery. However, he was referred back to our institution because of unstable gait and mildly altered mental status 3 months after discharge. Magnetic resonance (MR) imaging with gadolinium showed an enhanced ring-shaped mass around the hematoma cavity (Fig. 2A). He was readmitted, and open biopsy was performed. The histological diagnosis was glioblastoma multiforme, and he underwent a combination of radiation therapy and oral chemotherapy with temozolomide. MR imaging showed marked shrinkage of the tumor (Fig. 2B), but he died of pneumonia 3 months after the second surgery. Autopsy was not performed.

Discussion

In retrospect, our approach to the present case had been biased by the preexisting hypertension. Preoperative CT had demonstrated brain edema around the ICH, an indication of an unusual etiology, but this was falsely attributed to his delayed admission. Only CT with contrast medium was performed to exclude the presence of underlying pathologies that might cause ICH. Postoperatively, only CT was performed because we were convinced that hypertension was the only cause of the ICH, and also because the surgery was apparently successful.

The present case shows that only preoperative CT with or without contrast medium cannot exclude intratumor hemorrhage as the cause of ICH, even if the patient has a history of hypertension and the location is typical for hypertensive ICH. Postoperatively, only CT with contrast medium is typical for hypertensive ICH. Histological investigation of surgical specimen is essential, even if stereotactic aspiration surgery is performed. Clinical/radiological follow up in the subacute to chronic stage is also essential for detecting subtle neurological deterioration of the patient to avoid diagnostic delay.

References


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