Here, we report a rare case of an anaplastic astrocytoma masquerading as a hypertensive basal ganglia hemorrhage. A 69-year-old woman who had been under medical management for hypertension during the past 3 years suddenly developed right hemiparesis with dysarthria. Brain computed tomography (CT) scans with contrast and CT angiograms revealed an intracerebral hemorrhage (ICH) in the left basal ganglia, without an underlying lesion. She was treated conservatively, but underwent a ventriculoperitoneal shunt operation 3 months after the initial attack due to deteriorated mental status and chronic hydrocephalus. Three months later, her mental status deteriorated further. Magnetic resonance imaging (MRI) with gadolinium demonstrated an irregular enhanced mass in which the previous hemorrhage occurred. The final histological diagnosis which made by stereotactic biopsy was an anaplastic astrocytoma. In the present case, the diagnosis of a high grade glioma was delayed due to tumor bleeding mimicking hypertensive ICH. Thus, a careful review of neuroradiological images including MRI with a suspicion of tumor bleeding is needed even in the patients with past medical history of hypertension.

Key Words: Basal ganglia · Intracerebral hemorrhage · Tumor bleeding · Brain tumor · Hypertension · Anaplastic astrocytoma.
Intratumoral hemorrhage is thought to originate from abnormal newborn vessels that traverse necrotic areas or from tumoral invasion of large vessels, leading to thinning and rupture of the vessels walls. Another potential mechanism would be relatively weak tumor vessels, which are not well invested with a glial meshwork; this may contribute to reduced resistance to the shearing forces of the brain. Endothelial proliferation with subsequent obliteration of the lumen or presence of intratumoral arteriovenous fistulae are alternate explanations for intratumoral bleeding. Thus, hemostasis often cannot be easily achieved at hematoma removal; this finding indicates the possibility of brain tumors as a cause of bleeding.

There have been known risk factors of intratumoral hemorrhage. Hemorrhage more often develops in malignant tumors such as glioblastoma and metastatic brain tumors. The incidence of tumor bleeding in malignant astrocytoma in one study was 6% while that in glioblastoma and metastatic brain tumors were 6.5-8% and 7-9%, respectively. Among benign neuroepithelial tumors, the incidence of hemorrhage from mixed glioma and oligodendroglioma was much higher than the other tumors. On the other hand, pituitary adenoma and meningiomas have the high risk for developing intratumoral hemorrhage among benign non-neuroepithelial tumors. The location of bleeding depends on the different site of brain tumor even though intratumoral hemorrhage usually develops in the atypical location of hypertensive intracerebral hemorrhage and the patients often have no history of hypertension.

Radiological studies with contrast material usually distinguish tumors from hemorrhage, as the border between the tumors and hemorrhage is usually clear. In contrast, if the tumors are compressed by a large hemorrhage, or the border between the tumors and hemorrhage is unclear, intratumoral hemorrhage may be indistinguishable from spontaneous ICH, even though contrast material is used. Thus, a CT with contrast cannot exclude underlying pathologies that may cause ICH, even though contrast material is used. Radiological studies with contrast material usually distinguish tumors from hemorrhage, as the border between the tumors and hemorrhage is usually clear. In contrast, if the tumors are compressed by a large hemorrhage, or the border between the tumors and hemorrhage is unclear, intratumoral hemorrhage may be indistinguishable from spontaneous ICH, even though contrast material is used. Thus, a CT with contrast cannot exclude underlying pathologies that may cause ICH, especially if the patient has a history of hypertension, and the location is typical for hypertensive ICH. MRI with gadolinium in the early follow-up period would likely have lead to earlier detection of the tumors in the present case. However, Inamasu et al. suggested that in terms of cost effectiveness, it is controversial to have every patient presenting with typical hypertensive small ICH undergo MRI with gadolinium to rule out intratumoral bleeding.

Histopathological examination of hematoma specimens with brain parenchyma may have detected the brain tumor, if hematoma evacuation or aspiration surgery had been performed in the current case. However, the relatively small volume of hematoma and patient’s age led us to choose conservative treatments, rather than surgical options. It is controversial to undergo surgical treatments for histological diagnosis in typical hypertensive small ICH, though potential surgical modalities include stereotactic hematoma aspiration or endoscopic hematoma evacuation. However, if surgical treatment is indicated, a considerable hematoma specimen with adjacent brain parenchyma would be preferred to rule out underlying pathologies through histological examination.
Interestingly, normal initial neuroimaging including MRI is not uncommon among patients with malignant primary brain tumors. Thaler et al.\(^8\) reported that among 193 patients with malignant primary brain tumors, initial imaging preceding diagnosis with a short interval was normal in nine patients and abnormal but non-diagnostic in an additional eight patients. They conclude that dramatic, rapid tumor growth is possible\(^8\). Thus, in our case, it cannot be excluded that basal ganglia hemorrhage was a hypertensive hemorrhage which was not related with brain tumor, and then anaplastic astrocytoma rapidly developed in the site of hemorrhage.

In the present case, there was an opportunity to detect the underlying high-grade glioma during outpatient follow-up. The patient suffered from physical malaise and a deteriorated mental status 6 weeks after discharge. However, only a CT was performed as the etiology of the ICH was presumed to be hypertension. A follow-up CT revealed asymmetric ventriculomegaly and periventricular edema, with shunt operation performed without additional radiological studies. As a result, the final diagnosis of the tumor was delayed. However, in retrospect, asymmetric ventriculomegaly suggests an underlying pathology in the region of the pre-existing hemorrhage. Thus, follow-up MRI in the chronic stage of ICH seems to be necessary for detecting underlying pathologies masquerading as hemorrhage if the patient undergoes subtle neurological deterioration during follow-up in order to avoid diagnostic delay, even in cases typical for hypertensive hemorrhage.

**CONCLUSION**

The present case indicates that some brain tumors, masquerading as a hypertensive ICH, may be difficult to diagnose in the acute phase. A diagnostic delay for highly malignant tumors leads to progression without proper treatment and resultant poor outcomes. Thus, although the morphology and location of the hematoma and patient medical history are typical for a hypertensive ICH, brain tumors should be suspected as a cause of spontaneous hemorrhage. If needed, aggressive histological investigation at hematoma evacuation and MRI follow-up should be considered to avoid hidden, underlying pathologies.

- **Acknowledgements**

This study was supported by a grant of the Korean Health Technology R&D Project, Ministry of Health & Welfare, Republic of Korea (A1203920300).

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