INTRODUCTION

Brain tumor apoplexy has often been associated with intratumoral bleeding, with reported hemorrhage rates ranging from 1.4% to 10.0%. We report a case of cerebral anaplastic astrocytoma presenting with intratumoral bleeding and acute subdural hematoma. We reviewed the pathological characteristics and mechanism of massive tumor bleeding.

CASE REPORT

A 62-year-old woman had an intermittent dull nature headache for the last few days. She had a sudden bursting headache, nausea, vomiting and lost her consciousness 2 hours before arriving at emergency room. Neurologic examination revealed the left hemiparesis (Grade IV) and right third cranial nerve palsy. She was semicomatose. Brain enhanced computed tomography showed a heterogenous enhancing space occupying lesion at right temporal lobe (4.5×4.5×4.5cm) (Fig. 1). Transfemoral cerebral angiogram showed a typical vascular displacement pattern caused by a large temporal lobe mass. But there was no vascular abnormality (Fig. 2). She did not have any anticoagulant therapy nor coagulopathy. After magnetic resonance imaging (MRI) with a contrast media (Fig. 3), she underwent emergency brain surgery. The brain tumor and subdural hematoma was totally removed. The patient recovered immediately after craniotomy. But, right 3rd cranial nerve palsy was recovered 3 months later.

Histologic examination of tissue sections revealed anaplastic astrocytoma (WHO grade III) (Fig. 5). Hematoxylin and Eosin staining showed the old and fresh hemorrage. It also showed obscure luminal structures invaded by tumor cells. There were thombi in the lumen of these structures (Fig. 6-A). Immunohistochemistry for factor VIII showed these
obscure structures were vessels (Fig6-B). These structures were disrupted by tumor cells.

Fig. 1. Initial CT shows intracerebral hemorrhage at right temporal lobe and acute subdural hemorrhage at right froto-temporo-parietal convexity.

Fig. 2. Transfemoral cerebral angiogram shows a typical vascular displacement pattern caused by a large temporal lobe mass. But there was no vascular abnormality.

Fig. 3. MRI shows space-occupying lesion at right temporal lobe.

Fig. 4. Intraoperative photography shows the massive tumor bleeding(A) and we clearly removed the tumor and subdural hematoma(B).

Fig. 5. Histologic examination of tissue sections reveals anaplastic astrocytoma (WHO grade III). A. Immunohistochemistry for glial fibrillary acidic protein shows positive immunoreactivity (×400). B. Immunohistochemistry for Ki-67 (×400) reveals high proliferative activity (Ki-67 index: 10%). C. Immunohistochemistry for Epithelial Membrane Antigen shows negative immunoreactivity (×400). D. Hematoxylin and Eosin staining (×200) reveals the old and fresh hemorrhage. It means repeated hemorrhage.
Fig. 6. Hematoxylin and Eosin staining reveals obscure luminal structures invaded by tumor cells (A). There are thrombi in the lumen of these structures. Immunohistochemistry for Factor VIII confirms these obscure structures are vessels (B). These structures are disrupted by tumor cells.

Radiation therapy with total 6840 cGy spread over 7 weeks (5 treatments per week, total 38 treatments) was performed. 6 month after surgery, she has no recurrence. A longer period of clinical follow-up is needed.

DISCUSSION

The most common presenting symptoms of gliomas are headache, focal neurologic deficits, and seizures. Symptoms depend on tumor location. However, sudden loss of consciousness is unusual presenting symptoms of gliomas. Apoplexy is usually due to a cerebrovascular accidents. But, occasionally apoplexy is due to a tumoral bleeding, too. Brain tumor apoplexy has often been associated with tumoral hemorrhage, with reported hemorrhage rates ranging from 1.4% to 10.0%. Spontaneous macroscopic tumor bleeding has been reported to occur in 6.1% of malignant astrocytoma.

Regardless of the pathology, apoplexy needs a prompt accurate diagnosis and a proper management. In this case, the prompt emergency craniotomy saved her life.

The first impression of her initial CT scan was traumatic brain injury. But there was no evidence of external scalp wound, skull fracture and trauma history. Therefore, the second impression was cerebrovascular malformation. We performed catheter angiography. The angiography revealed a typical vascular displacement pattern caused by a large temporal lobe mass. But there was no vascular abnormality. After angiography, we confirmed this space-occupying lesion by MRI.

Common histological features of tumors that bled microscopically or macroscopically include: tumor necrosis as well as the vascular changes of hyalinization, degeneration or necrosis of vessel walls, thrombosis, the presence of many thin-walled vessels, and ruptured vessels. In this case, histologic examination showed the vascular invasion by tumor cells and ruptured vessels.

Russel et al reported that the presence of abnormal vascularity correlated with both shortened survival and higher grade of malignancy. Catheter angiography was performed on patients with low and high-grade gliomas in an effort to characterize vascularity as a reliable predictor of outcome, as well as an additional adjunct to histologic diagnosis and malignancy grading. In this case, vascularity is grade 1, i.e. the tumor has no abnormal vascularity evident on angiography. So, we respect that this patient will survive a longer period.

Hentschel et al reported a case of a neoplasm that presented exclusively with subarachnoid hemorrhage (SAH). The final cause of bleeding is a malignant
oligodendroglioma. The authors informed that patients experiencing negative SAH angiographically should undergo MRI, occasionally on a serial basis, to exclude other etiologies for hemorrhage, include neoplasia. In this case, preoperative accurate evaluation was made by MRI.

Glioma progression is strongly dependent on the development of a new vascular network that occurs primarily by angiogenesis. But cerebral angiography revealed no vascular abnormality in this case. It may be due to the locational properties, i.e. temporal lobe is more abundant vascularity than any other lobe in normal brain.

Treatment of high grade astrocytoma has remained one of the most challenging field in cancer therapy. Standard-of-care treatment include resection, radiation therapy, and adjuvant chemotherapy. Locoregional therapeutics and molecularly targeted agents represent new promising approaches for malignant astrocytoma treatment.

CONCLUSION

We report a case of cerebral anaplastic astrocytoma presenting with intratumoral bleeding and subdural hematoma. We confirm that the vascular invasion by tumor cells made massive tumor bleeding. Regardless of the pathology, apoplexy needs prompt accurate diagnosis and proper management.

References