The Occurrence of a High-grade Glioma Secondary to Vascular Malformation: A Case Report

Qiao Wang[1], Haogeng Sun[2], Zhikuan Yu[2], and Houfu Deng[1]
[1]Department of Nuclear Medicine, West China Hospital of Sichuan University, Chengdu, Sichuan China
[2]Department of Neurosurgery, West China Hospital of Sichuan University, Chengdu, Sichuan China
Email: denghfy@126.com

***Abstract*—The authors reported a rare case of the occurrence of a high-grade glioma secondary to a vascular malformation (VM) lesion in the same location in a 32-year-old man. Initial brain CT scan revealed a left frontal hematoma with perifocal edema. Furthermore, the Susceptibility weighted imaging (SWI) revealed many tortuous vessels around the hemotoma. But the Digital Subtraction Angiography (DSA) showed no obvious VM and aneurysm. At last, the histological section examination showed a VM with haemorrhage after hematoma evacuation. Sixteen months later, the Magnetic Resonance Imaging (MRI) revealed a multiple cystic space-occupying lesion in the left frontal lobe involving in the left lateral ventricle, with midline shift, and the contrast enhanced area around the cystic lesion. The patient underwent excision of the lesion. The final histopathological examination of the specimen revealed a high-grade glioma with haemorrhage, necrosis and cystic degeneration. A vicious circle of aberrant neoangiogenesis and hypoxia or/and an excessive inflammatory might explain this situation. Routine imaging reexamination might be suggested for some patients suffering with VM.**

***Index Terms*—arteriovenous malformation, vascular malformation, glioma**

I. Introduction

Rare cases of a glioma associated with an arteriovenous malformation (AVM) were reported in recent years, and the pathogenesis of which the two different lesions of central nervous system occur in the same location remains unclear. We reported the occurrence of a high-grade glioma secondary to VM in the same location and discussed the potential pathogenesis as well as monitoring of such condition. Comparing with some cases of AVMs coexisting with gliomas in the brain[1-3], we believe that the presented case may provide a better explanation for the relations between glioma and VM. Primary brain tumors are cancers that originate in the brain. These tumors are very different from secondary brain tumors, which originally developed elsewhere in the body and spread (metastasized) to the brain.Primary brain tumors develop from glial cells. Glial cells provide the structural backbone of the brain and support the function of the neurons (nerve cells), which are responsible for thought, sensation, muscle control, and coordination. Gliomas cause symptoms by invading (growing) into and/or creating pressure in nearby normal brain tissue. The most common symptoms include: Cognitive symptoms like memory loss, personality change, confusion, speech problems, Headache and Seizures — Seizures occur in more than one-half of patients with grade III gliomas and about one-fourth of patients with grade IV gliomas. Seizures are caused by disorganized electrical activity in the brain. Medications can help to control seizures. Other common symptoms of brain tumors include muscle weakness, visual symptoms, and changes in sensation. The only way to determine the type of tumor with certainty is for a neurosurgeon to remove a piece of the tumor (biopsy), usually during surgery. A pathologist will then examine the biopsy under a microscope.However, a biopsy may be done without surgery; this approach is preferred if the tumor is located within a critical area of the brain or if you are too sick for surgery. In these circumstances, a procedure called a stereotactic needle biopsy is used to take a sample of the tumor by inserting a needle through the skull into the brain itself[4].

II. Case Report

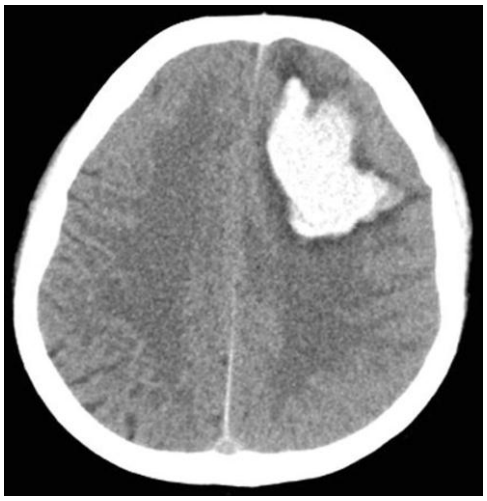

Figure. 1. Axial CT scans demonstrating a left frontal hematoma

Manuscript received May 8, 2014; revised July 20, 2014.

doi: 10.12720/jomb.4.3.259-262

A 32-year-old man was admitted to our neurosurgery unit in March 2012 with a two-day history of sudden severe headache and vomit. His neurological examination revealed unremarkable and no prior medical history was informed. Initial CT scan revealed a left frontal hematoma with perifocal edema with 5.5 × 3.9 cm in maximal transverse section Fig. 1.

The brain SWI revealed a left frontal and basal ganglia region hemotoma surrounded with numerous tortuous vessels Fig. 2.

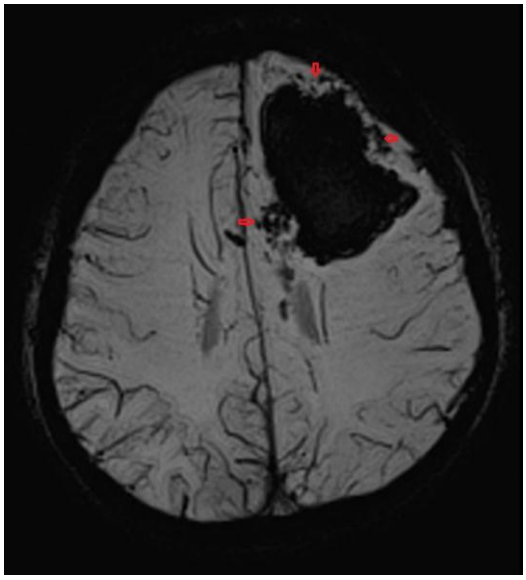

Figure 2.The brain SWI demonstrating many tortuous vessels around the hemotoma.(red arrows).

The DSA showed no obvious VM, aneurysm and tumor stain but a very small abnormal sign locating in the basal ganglia region belonging to the branch of A2 segment of the left anterior cerebral artery(ACA) Fig. 3.

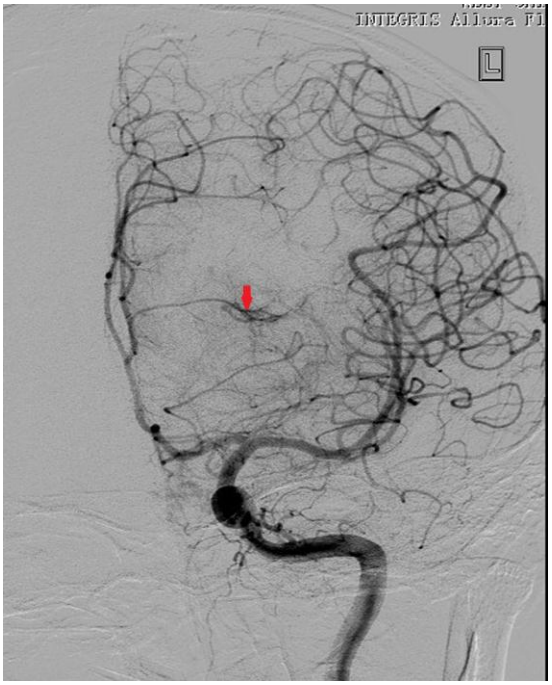

Figure 3.The DSA showing a very small abnormal sign belonging to the branch of A2 segment of the left ACA(red arrow)

Maybe it was a low-flow malformation or a hemotoma leading to the thrombogenesis of the feeding arteries. As a result, the initial diagnosis was VM with bleeding. Due to the request of the patient and his relatives, we performed a left frontal hematoma evacuation. In the operation, we found a minute lesion resembling an AVM and didn't see any abnormal tissues like tumour or other lesions in and around the hemotama. The following histological section examination showed a VM with haemorrhage and didn't find any tumour cells Fig. 4.

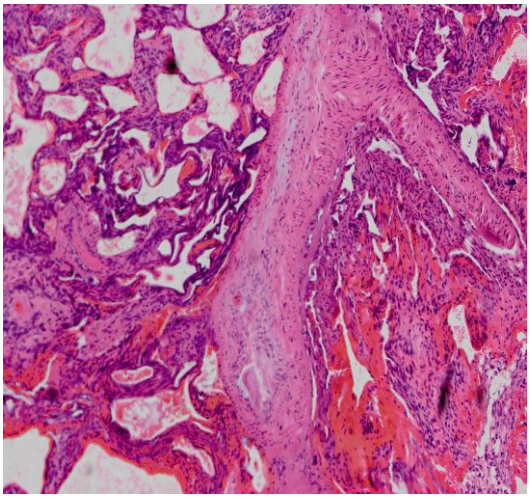

Figure 4.The histological section derived from the first operation revealing the numerous enlarged blood vessels with irregular walls in varying caliber, some of which were thrombosed, and the vascular endothelial cells was incomplete (Hematein Eosin;magnification ×100).

The patient resumed well without any significant neurologic deficits when he left our unit.

Sixteen months later, the man was admitted with an episodic headache to our neurosurgery unit again without any significant neurologic deficits. A brain MRI scan with contrast showed a multiple cystic space-occupying lesion in the left-sided frontal lobe involving in the left lateral ventricle with midline shift, and the contrast enhanced area around the cystic lesion Fig. 5.

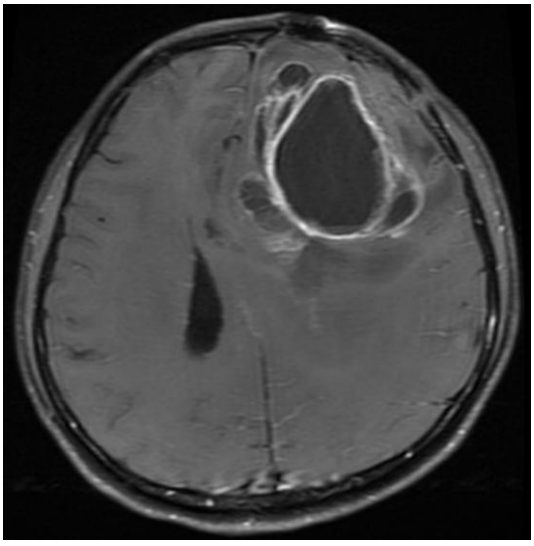

Figure 5.A brain MRI scan with contrast(16 months after the first operation) showing a multiple cystic space-occupying lesion in the left frontal lobe and the contrast enhanced area around the cystic lesion

Finally the patient underwent excision of the lesion. Intraoperatively, we performed a gross total excision of the cystic lesion. The seprated tissues for frozen section examination revealed that the cyst wall cells were malignant. The final histopathological examination of the specimen revealed a high-grade glioma with haemorrhage, necrosis and cystic degeneration Fig. 6.

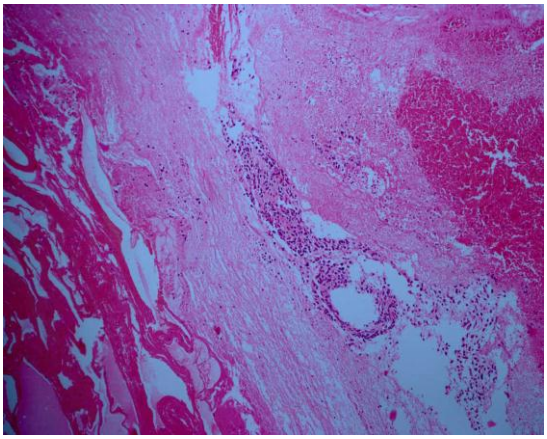

Figure 6. The final histopathological examination of the specimen showing a high-grade glioma with haemorrhage, necrosis and cystic degeneration (Hematein Eosin; magnification ×100).

The patient knew that the prognosis of his disease was really bad. So he requested discharge.

III. Discussion

The occurrence of a high-grade glioma secondary to a VM lesion at the same location of the central nervous system is rare. It is still unclear whether this represents two pathologies or a combined lesion. Some authors provided evidence that the tumor could induced VM growth[4], while other articles could be speculated that the tumor might be induced by VM [6, 7]. Additionally, other authors suggested that the highly vascularized tumors should be named as angiogliomas [8, 9]. Above hypotheses are disputed and could not fully explain which one is original lesion. Our case presented an AVM-like lesion appearing before a high-grade glioma and no visible glioma tissue in and around the hemotama at the first time by histological section examination. Therefore, we may speculate the AVM was original lesion.

A vicious circle of aberrant neoangiogenesis and hypoxia may provide the conditions for the growth of tumors. Neoangiogenesis, which results from overexpression of vascular endothelial growth factor (VEGF) and other pro-angiogenic factors provides oxygen and metabolites to the tumor tissue [10]. Upregulation of hypoxia-associated factors due to perfusion disturbances connected with AV shunt and deficiently oxygenate related to the rapidly growing tumor tissue causes the overexpression of various angiogenic factors and induces neoangiogenesis[1]. Thus, this circle may provided a well explanation for our case.

Excessive inflammation, which produces a space-occupying lesion, is induced by some foreign substances in the vicinity of the surgical site[11]. Haemostatic as foreign substances are widely used in neurosurgery. Fibrillar and gelatin sponge had been left in place for haemostasis at the first operation and foreign body reactions should be included in the differential diagnosis of this patient.

This patient was not performed any imaging examinations until admitted to hospital again after resection of AVM-like lesion. The patient might own a good outcome if the space-occupying lesion had been diagnosed by imaging reexamination early and recieved a positive treatment. Therefore, it is worthwhile recommending that routine imaging reexaminations should be more frequently performed for following prognosis evaluation after patients discharge.

IV. Conclusion

A VM and high-grade glioma, as two different lesions of the central nervous system, discovered at different time but in the same location is really rare. A vicious circle of aberrant neoangiogenesis and hypoxia or/and an excessive inflammatory may explain this situation. In addition, to reduce the influence of lesions secondary to VM, we suggest that imaging examination should be more frequently performed on patients suffering with VM.

References

[1] J. Aucourt, P. Jissendi, O. Kerdraon, and M. Baroncini, "Neuroimaging features and pathology of mixed glioblastoma--AVM complex: A case report," *J. Neuroradiol* vol. 39, pp. 258-262, 2012.

[2] M. Gmeiner, M. Sonnberger, G. Wurm, and S. Weis, "Glioblastoma with the appearance of arteriovenous malformation: Pitfalls in diagnosis," *Clin Neurol Neurosurg*, vol. 115, pp. 501-506, 2013.

[3] I. M. Ziyal, K. Ece, B. Bilginer, G. G. Tezel, and O. E. Ozcan, "A glioma with an arteriovenous malformation: An association or a different entity?" *Acta Neurochir (Wien)*, vol. 146, pp. 83-86, 2004.

[4] T. Batchelor, Patient Information: High-grade Glioma in Adults (Beyond the Basics),The Oncologist, April 2014

[5] M. Onishi, T. Ichikawa, K. Kurozumi, and I. Date, "Angiogenesis and invasion in glioma," *Brain Tumor Pathol*, vol. 28, pp. 13-24, 2011.

[6] M. Ehtesham, P. Kabos, W. H. Yong, W. I. Schievink, K. L. Black, and J. S. Yu, "Development of an intracranial ependymoma at the site of a pre-existing cavernous malformation," *Surg Neurol*, vol. 60, pp. 80-82, 2003.

[7] J. S. McKinney, T. Steineke, D. Nochlin, and J. L. Brisman, "De novo formation of large arteriovenous shunting and a vascular nidus mimicking an arteriovenous malformation within an anaplastic oligodendroglioma: Treatment with embolization and resection," *J. Neurosurg*, vol. 109, pp. 1098-1102, 2008.

[8] J. M. Bonnin, C. E. Peña, and L. J. Rubinstein, "Mixed capillary hemangioblastoma and glioma. A redefinition of the 'angioglioma'," *J Neuropathol Exp Neurol*, vol. 42, pp. 504-516, 1983.

[9] E. Matyja, W. Grajkowska, A. Taraszewska, A. Marchel, P. Bojarski, and P. Nauman, "Advanced reactive astrogliosis associated with hemangioblastoma versus astroglial-vascular neoplasm 'angioglioma'," *Folia Neuropathol*, vol. 45, pp. 120-125, 2007.

[10] M. Preusser, C. Haberler, and J. A. Hainfellner, "Malignant glioma: Neuropathology and neurobiology," *Wien Med Wochenschr*, vol. 156, pp. 332-337, 2006.

[11] S. K. Dinesh, S. Y. Lee, and J. Thomas, "A case of mistaken identity: Intracranial foreign body reaction after AVM

embolisation mimicking a glioma," *J. Clin Neurosci*, vol. 15, pp. 463-465, 2008.

Wang Qiao, male, 26 years old, was born on November 15, 1987 in chengdu, sichuan province, China, 2006-2011 he was majoring in medical imaging, chongqing medical university, bachelor of medicine, 2011-2014 study in sichuan university huaxi medical college clinical nuclear medicine, with a master's degree in medicine

Hao Gen Sun, Male, 26 years old, born in nanchong, sichuan province, China, 2006-2011 study in luzhou medical college, get a bachelor's degree in medicine, huaxi clinical medical school of sichuan university, 2011-2014, received a master's degree.

Zhi Kuan Yu, male, 26 years old, born in dalian in liaoning province, China, 2006-2011 study in dalian medical university clinical medical professional, get a bachelor's degree in medicine, 2011-2014 study in sichuan university huaxi medical college clinical neurosurgery, received a master's degree.

Hou Fu Deng, born in Sichuan Jin Tang, presently for west China hospital of sichuan university professor, imaging and nuclear medicine doctor (master) graduate student tutor, the state council special allowance from the government.

Pay attention to the communication with clinical departments with learning, with a pragmatic spirit and multidisciplinary knowledge. In the domestic first conducted three diagnosis and treatment projects, the nuclear medicine has produced important influence on the popularization and enhancement. In nuclear medicine experiment, in the development and application of the imaging agent, organ imaging, has deep attainments function determination, etc. In the treatment with nuclide thyroid function hyperfunction, treatment of thyroid cancer; Treatment of bone tumors, multiple metastases of cancer to bones, huge bone metastatic carcinoma, multiple myeloma, etc have in-depth study and has accumulated rich experience.