Obstructive hydrocephalus caused by giant basilar artery aneurysm

Hulagu Kaptan, *MD,* ***Ender Koktekir,*** *MD,* ***Fahri Recber,*** *MD,* ***Gokhan Akdemir,*** *MD, PhD.*

ABSTRACT

أن القاعدة العملاقة لأم الدم المصاحبة لاستسقاء الدماغ نادرة الحدوث وعند حدوثها يقدم علاج استقساء الدماغ أكثر من أم الدم نفسها وخاصة أن كانت مصابة بالخثار. تشمل خيارات العلاج على تحويلة بطينية صفاقية وأشعة منظار البطين الذي يكون ملازم للمضاعفات الشديدة ولكن خفض إرتفاع ضغط الدم داخل الفحص قد يؤدي إلى زيادة أم الدم. في هذا التقرير نستعرض حالة مريضة مصابة باستقساء الدماغ نظراً لوجود قاعدة عملاقة لأم الدم، وقد راجعت المريضة المستشفى وهي تشتكي من ارتفاع ضغط القحف الداخلي والتي تمت السيطرة علية بتحويلة بطينية صفاقية مع تطور وتحسن ملحوظ. وبعد 15 يوماً بعد العملية توفيت المريضة بأزمة دماغية وعائية. استعرضنا هذه الحالة التي تدهورت بسبب احتشاء الدماغ لزيادة أم الدم بعد تحويلة بطينية صفاقية. كما قمنا بمناقشة طرق العلاج لهذه الحالات.

Giant basilar artery aneurysms are rarely associated with hydrocephalus. When it occurs the treatment usually addresses the hydrocephalus rather than the aneurysm itself, especially if it is already thrombosed. The treatment options include ventriculoperitoneal shunting and endoscopic third ventriculostomy, which may be related to high complication rates. However, reducing the intracranial hypertension may produce aneurysmal growth. We report a patient with obstructive hydrocephalus due to thrombosed giant basilar artery aneurysm. The patient initially presented with symptoms of increased intracranial pressure, and was managed by ventriculoperitoneal shunting with significant symptomatic improvement. Fifteen days after operation, the patient died due to a cerebrovascular event. We report a case that deteriorated because of cerebral infarction due to aneurysmal growth after ventriculoperitoneal shunting. We also discuss the treatment options in such cases.

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From the Department of Neurosurgery, Medical School, Selcuk University, Selcuklu, Konya, Turkey.

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Address correspondence and reprint request to: Dr. Hulagu Kaptan, Assistant Professor in Neurosurgery, Department of Neurosurgery, Medical School, Selcuk University, Alaeddin Keykubat Campus, Selcuklu, Konya, Turkey. Tel. +90 (332) 2415000 Ext. 40232. Fax. +90 (332) 2411550. E-mail: hulagukaptan@yahoo.com

Aneurysms are classified according to their shape and pathogenesis. The most common type is saccular, which is berry-shaped, whereas fusiform aneurysms are less common and characterized by dilatation and elongation of an artery.[1] Giant fusiform aneurysms are uncommon, but most frequently involve the internal carotid artery, and middle cerebral artery.[1,2] Giant aneurysms arising from the basilar artery trunk and branches are extremely rare.[1-4] Patients with basilar artery aneurysms often have symptoms and signs of occlusion of the main artery or perforating branches, aneurysmal rupture, or mass effect.[1-3] Although most patients present with symptoms due to subarachnoid hemorrhage, rarely, hydrocephalus may be the initial finding.[1,3-6] In these situations, hydrocephalus is caused by obstruction of the CSF pathways or transmission of the ectasic artery pulsations.[1] Our objective in presenting this particular case is to highlight the risk of aneurysmal growth after ventriculoperitoneal shunting.

Case Report. A 56-year-old woman presented to the emergency department with complaints including vertigo with nausea, gait disturbance, impaired balance and disturbed level of consciousness for 3 days. The Glasgow Coma Scale (GCS) was 12 (E2, V5, M5). Computed tomography and MRI revealed a noncontrast

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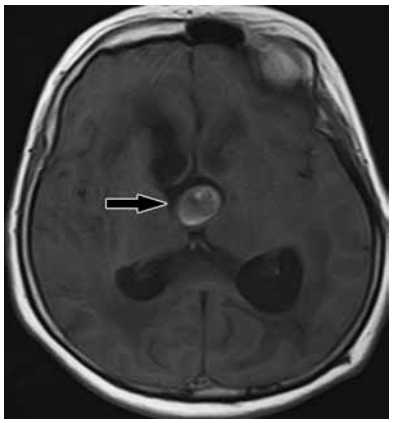

Figure 1 - Axial T1 weighted MRI showed a mass lesion at the third ventricle.

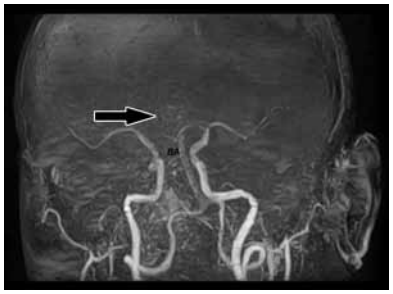

Figure 2 - Magnetic resonance angiography showed no filling of the aneurysm (arrow indicates the thrombosed basilar tip aneurysm).

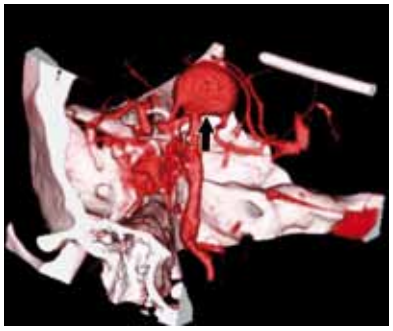

Figure 3 - Computed tomography angiography showing giant basilar tip aneurysm.

enhancing mass lesion (27 x 18 x 17 mm) in the third ventricle, which caused obstructive hydrocephalus (**Figure 1**). Magnetic resonance angiography (MRA) showed a totally thrombosed basilar artery aneurysm (**Figure 2**). Computed tomography angiography (CTA) showed a thrombosed basilar artery aneurysm, 2.5 cm in diameter (**Figure 3**). A ventriculoperitoneal shunting procedure was performed to treat the hydrocephalus. Postoperatively her clinically situation showed

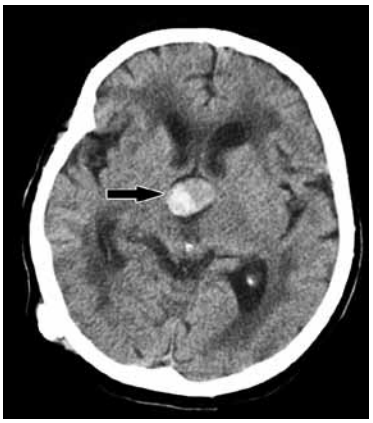

Figure 4 - A CT scan on the second admission showing aneurysm growth.

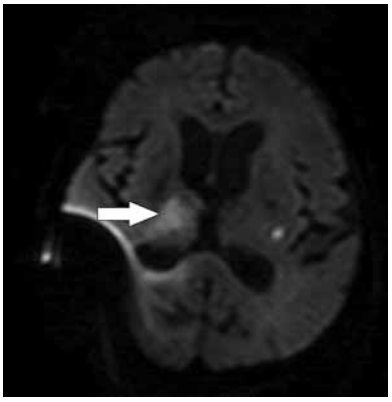

Figure 5 - Diffusion MRI showing acute infarction at the right thalamus.

remarkable improvement (GCS: 15) and she was discharged on the postoperative fifth day. Fifteen days later she was admitted to the emergency department with sudden loss of consciousness; her GCS was 4 (E1, V2, M2). The CT scan showed an increase in the size of the aneurysm without any signs of hydrocephalus or subarachnoid hemorrhage (**Figure 4**). Diffusion MRI showed an acute thalamic infarction at the right thalamus (**Figure 5**). She died 3 hours after admission.

Discussion. Aneurysms larger than 2.5 cm in diameter are defined as giant aneurysms.[1,4,5] They constitute around 5% of all aneurysms, and only a few are located in the basilar artery. They usually act as space occupying lesions and may lead to obstructive hydrocephalus.[4-6] Treatment can be addressed directly to reduce mass effect or indirectly to the hydrocephalus. Treatment options that attempt to reduce mass effect include direct neck clipping of the aneurysm, by-pass surgery, or endovascular occlusion of one vertebral artery.[4,6]

Microsurgical management of giant basilar artery aneurysms remain the most challenging condition treated by neurosurgeons. The importance of the surrounding neurovascular tissue, the complex arrangement of perforating vessels distorted by mass lesions, and the constricted corridors of surgical access all contribute to this complexity.[1,6] Direct microsurgical clipping of the giant basilar artery aneurysms are associated with high mortality and morbidity. For these reasons, endovascular coiling has become the most popular technique for treatment of giant basilar artery aneurysms. In our presented case, we did not perform microsurgical aneurysm clipping or endovascular treatment because the aneurysm was already thrombosed and the symptoms were related to hydrocephalus rather than the aneurysm itself.

Aneurysms presenting as third ventricular masses are uncommon, and the most common is a giant aneurysm arising from the basilar artery.[5,6] However, anterior cerebral artery, posterior communicating artery, anterior communicating artery, and internal carotid artery aneurysms, which cause third ventricle mass lesions have also been reported.[5-7] Diagnosis of the aneurysm at the third ventricle may be difficult especially if it is unruptured and thrombosed. In our patient, there was no subarachnoid hemorrhage seen on CT and MRI, and the lesion had a regular shape. Although MRA could not demonstrate the aneurysm because of thrombosis, the CTA clearly showed a giant basilar artery aneurysm.

Because of the anatomic location of the basilar artery, obstruction of the CSF pathways due to basilar artery aneurysms would be seen at the third ventricle floor, or at the level of the aqueductus sylvii. The treatment options of hydrocephalus in such cases include endoscopic third ventriculostomy and ventriculoperitoneal shunting.[1,4-7] There are only a few reports existing in the literature that used endoscopic third ventriculostomy in such cases.[7-10] Oertel et al[9] performed an endoscopic third ventriculostomy in 3 of their patients who had obstructive hydrocephalus due to giant basilar artery aneurysm, and concluded that this procedure should not be performed unless sufficient prepontine cistern for perforation of third ventricle floor is present. In our patient the cause of hydrocephalus was a high lying basilar artery thrombosed aneurysm, which occluded the third ventricle floor, so the endoscopic approach was not performed because of the high risk of aneurysmal rupture during the procedure. For this reason we performed a ventriculoperitoneal shunting procedure and the patient improved clinically.

Theoretically, decreasing the intracranial pressure by lumbar puncture or ventriculoperitoneal shunting increases the risk of the aneurysmal growth and rupture.[10] Kim et al[10] reviewed the literature and found that 2 of 23 cases who had undergone ventriculoperitoneal shunt operation died because of brain stem failure due to the mass effect of aneurysm growth. They also reported 2 aneurysm cases with hydrocephalus that deteriorated because of aneurysm growth after ventriculoperitoneal shunt operation.[10] Here, we report the fifth case in the literature. In our case, cranial CT and diffusion MRI, which were taken on the second admission of the patient, showed an increase in the size of aneurysm and acute thalamic ischemia without any signs of aneurysm rupture or progressive hydrocephalus. In this case, acute thalamic infarction might be caused by occlusion of the perforating branches of the basilar artery as a consequence of growth of the aneurysm.

In conclusion, our case demonstrates that aneurysmal growth, which caused the clinical deterioration, may be induced by the ventriculoperitoneal shunting. Direct microsurgical clipping of the aneurysm should be considered whenever possible in such cases.

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