

Effects of intravascular embolization operation on adult VGAD

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Abstract. – OBJECTIVE: To analyze the effects of intravascular embolization on adult vein of Galen aneurysmal dilatation (VGAD) patients.

PATIENTS AND METHODS: Five consecutively selected patients (median age of 56.4 years) were diagnosed with VGAD in our hospital from February 2010 to February 2015 and treated with intravascular embolization.

RESULTS: 2 cases were confirmed with malformed vessels in cerebellum, 2 cases in basal ganglia region and 1 case in brain stem; 3 cases with single branch artery blood supply and 2 cases with multiple branch artery blood supply; malformed vessel was 2.5-5.5 cm in diameter, on average 4.3 (± 1.2) cm; 3 cases were dominated by intracranial hemorrhage, 1 case by a headache and 1 case by seizure; GCS scores ranged from 8-12, on average 10.5 (± 1.6); intraoperative blood loss ranged from 20-80 ml, on average (55.8 $\pm$ 15.9) ml; 1 case died after operation, 1 case was disabled, and the remainder were normal.

CONCLUSIONS: Intravascular embolization was safe and effective for adult VGAD patients.

Key Words:

VGAD, Intravascular embolization, GCS scoring.

Introduction

Vein of Galen aneurysmal dilatation (VGAD) refers to the Galen vein dilation that results from arteriovenous malformations of the midline (AVMs) or a dural arteriovenous fistula converging into the normal Galen vein but obstructed mechanically (occlusion or stenosis) in the downstream outflow tract. The arterialized blood flows back into the veins, leading to a series of clinical symptoms¹. Different from vein of Galen aneurysmal malformation (VGAM), VGAD manifests in real Galen veins, mostly in teenagers or adults, the blood supply is from the internal carotid artery, meningeal branches of internal carotid and external carotid artery, normal sinus², Galen ve-

nous drainage of blood of normal brain tissues, visible mesencephalic or diencephalic AVM under head CT or MRI examinations³, suitable for embolism or embolism knife⁴. In this study, the effects of intravascular embolization operation on adult VGAD patients were studied and proved to be safe and effective.

Patients and Methods

Patients

Of the five patients, three were males and two females; their ages ranged from 48-72 years, median age of 56.4 years; 2 cases were confirmed with malformed vessels in the cerebellum, 2 cases in the basal ganglia region and 1 case in the brain stem; 3 cases with single branch arterial blood supply and 2 cases with multiple branches arterial blood supply; the malformed vessel was 2.5-5.5 cm in diameter, (average 4.3 $\pm$ 1.2 cm); 3 cases were dominated by intracranial hemorrhage, 1 case by a headache and 1 case by seizures; GCS scores ranged from 8-12, (average 10.5 $\pm$ 1.6). This study obtained the approval of the ethics committee of the hospital and the informed consent of the patients and their relatives. All cases conformed to the indications of intravascular embolization and were free from any conflicts of interest.

Operation Procedure

Before the operation, digital subtraction angiography (DSA) was implemented to define the location and size of the malformed mass and the condition of feeding arteries and draining veins. Patients were placed in a supine position, under general anesthesia. Femoral artery puncture was done on the right side by Seldinger method and arterial sheath inserted. Bilateral internal carotid artery and vertebral artery angiography were performed using a single-bend angiography catheter, that defined the location of the malformed mass, and then was withdrawn. After systemic heparini-

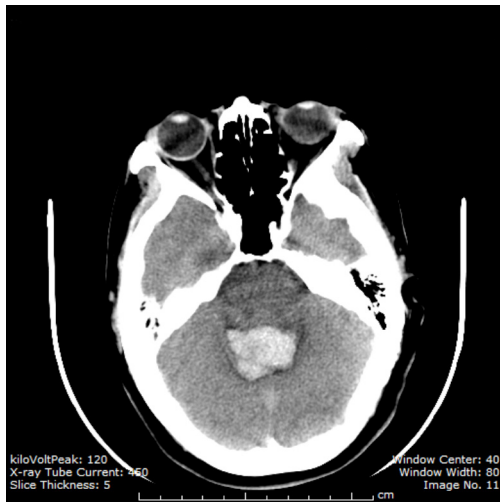


Figure 1. Head CT.

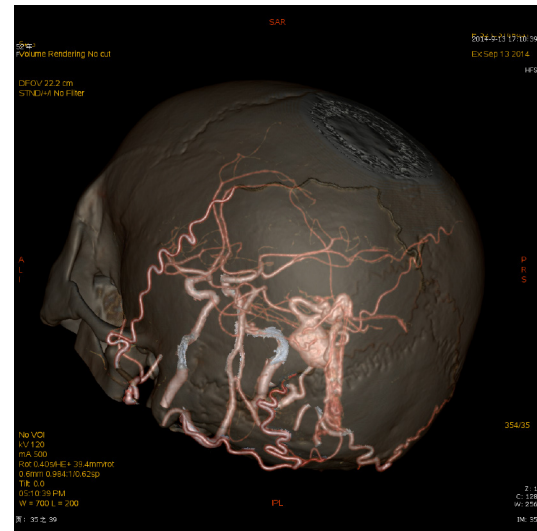


Figure 3. CTA 3D reconstruction.

zation, the guiding catheter was inserted into the corresponding internal carotid artery or vertebral artery using a guide wire. The micro catheter was inserted into the main arterial blood supply of the malformed mass under the guidance of the micro guidewire. This was withdrawn, slowly releasing the embolic materials after confirming that there was no normal perforator vessel.

One Specific Case

Case: female, 52 years, symptomatic for 7h after the sudden onset of a headache.

Physical examination: decreased cognition, opened eyes on calling, alalia. Vitals: temperatu-



Figure 2. Head CTA.

re 36.5°C, pulse 86 bpm/min, respirations 20 per min, blood pressure 120/80 mmHg. Normal head size without malformation, bilateral pupils of equal size and shape, diameter 2.5mm, sensitive to direct and indirect light reflex, slight resistance of neck, flexible four limbs supportive of on-bed autonomic activities, rejection of specific strength tests, no discovery of other pathological signs. No significant abnormalities in heart and lungs. Glasgow Glaucoma Scale (GCS) scoring: 4 points for spontaneous opening eyes, 3 points for unclear speech, 5 points for pain location, in total 12 points.

History: Brucella infection one month ago, no hypertension, diabetes mellitus or other diseases.

Head CT: irregular hyperdensity mass in and around the fourth ventricle, middle-density mass in the middle, increased density in the ambient cistern, bilateral ventricle and the third ventricle normal in shape, no shift in midline structures. Results suggestive of cerebellar hemorrhage (Figure 1).

Diagnosis: 1. Cerebellar hemorrhage; 2. Brucella infection.

Treatments implemented such as intracranial pressure lowering, infection prevention, wake-promoting, and decompression. Three days later, patient awoke and performed early functional rehabilitation exercises. Repeat head CT and CTA results: flake-like hyper-density mass in auda cerebelli, ventricular system normal in size and shape. No abnormality in the cerebral sulcus and cerebral fissure, No shift in midline structures (Figures 2 and 3). MRI and DSA examinations

(Figures 4 and 5), confirmed VGAD. Intravascular embolization was performed; nausea and vomiting appeared but soon disappeared after symptomatic treatment. Repeat CT results: mal-formed vascular mass in auda cerebelli disappeared, embolic material artifact could be seen (Figures 6 and 7). Physical examination: conscious, GCS scoring: 4 points for spontaneous opening eyes, 3 points for clear speech, 6 points for action coordination in total 15 points. When vital signs were stable, the patient was discharged from the hospital.

Results

Intraoperative blood loss ranged from 20-80 ml, (average 55.8 ± 15.9 ml); 1 case died after the procedure, 1 case was disabled, and the remainder recovered.

Discussion

According to the source of abnormal blood, VGAD can be divided into solid type, dura mater type and varicose type. Clinical symptoms include headache, exophthalmos, decreased visual acuity, intracranial murmur, subarachnoid hemorrhage, seizures and ataxia⁵. All of the above symptoms could be explained by the abnormal venous drainage following VGAD. The diagnosis and treatment rely on imaging examinations such as CT, MRI and

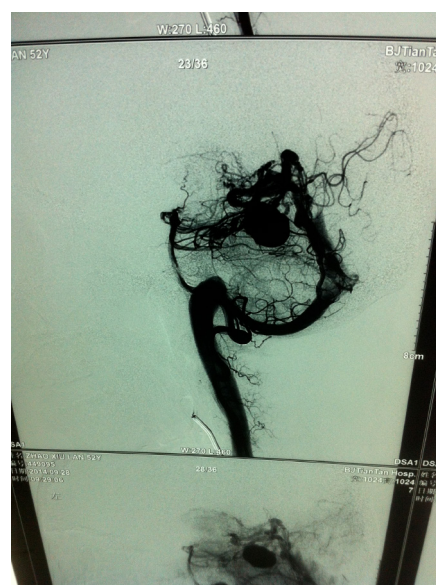


Figure 5. Head DSA.

DSA⁶. CT scanning can show hyper-dense, round masses behind the third ventricle and inside the quadrigeminal cistern, enlarged third ventricle and lateral ventricle. Enhanced scanning can reveal clearer masses, dilated blood supply arteries and draining veins, dilated straight sinus connecting with the round masses in the midline⁷. However, CT plain scanning is incapable of distinguishing BGAD from pineal cyst bleeding, meningioma, and pineal gland tumor⁸. MRI can show quasi-circular sharp-edged ballooning lesions inside cisternae venae magna



Figure 4. Head MRI.

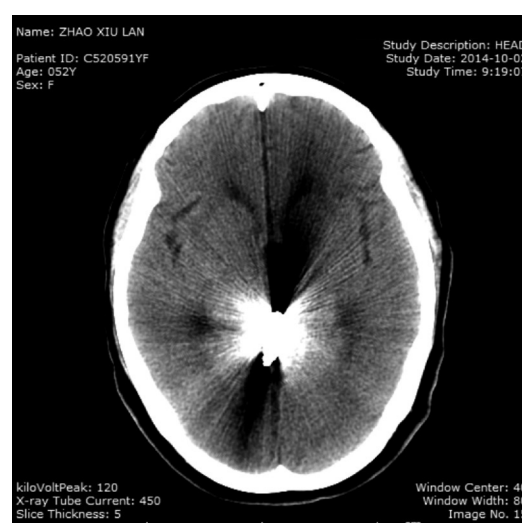


Figure 6. First CT after operation.

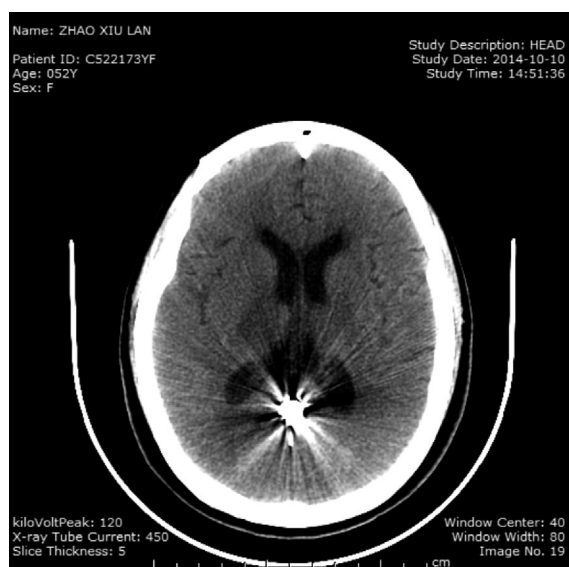


Figure 7. Second CT after operation.

cerebri and multiple abnormal vessel sections, ballooning lesions connecting with superior sagittal sinus, dilated superior sagittal sinus and a dilated straight sinus. Assisted by special imaging technology, dilated arteries and draining veins could be seen clearly, the midline can reveal dilated superior sagittal sinus linked to a round mass⁹. The “golden standard” of diagnosis is DSA, which clearly shows the tumor-like dilated veins and blood vessels and also excludes abnormal interference signals¹⁰.

The Spetzler grading system is the most frequently used system clinically, which divides arteriovenous malformation (AVM) into 5 levels according to the size of the lesion, the mode of draining vein, and the site of the lesion. Clinical experience showed high level AVM was generally supplied by multiple arteries that could hardly be occluded by simple vascular embolization¹¹. Some embolization reduces the blood flow volume of the malformed mass, but after embolotherapy, the low perfusion state of surrounding vessels caused by the blood shunting from the malformed mass would be damaged. After the regulatory functions of vessels were impaired, vascular brain swelling occurred, leading to vessel rupture and hemorrhage. So for high-level AVM, comprehensive treatment, such as intravascular embolization + microsurgery, intravascular embolization + radiotherapy, microsurgery + radiotherapy, intravascular embolization + microsurgery + radiotherapy, are the major treatment methods in the present clinic.

With the development of interventional neuroradiology and the improved understanding of the pathogenesis of VGAD, the key to treat with VGAD has turned to the planned embolization or stereotactic radiotherapy. For low level VGAD, intravascular embolization is the best treatment method because it is safe and effective and suitable for all clinical symptoms¹². The key to treatment is the occlusion of the arteriovenous fistula orifice and prevention of damage to the venous drainage system¹³. In the clinic, the time, route and material of embolization shall be decided according to the clinical state of patients and the characteristics of the lesion vessels shown in DSA. These include: (1) via artery: balloon, spring coil, isobutyl cyanoacrylate (IBCA) or N-butyl-cyanoacrylate (NBCA) to selectively embolize the fistula or AVM lesion. The clinical application of interventional embolization material such as NBCA and ethylene vinyl alcohol copolymer (ONYX) further improves results, reduces the complications, such as death, hemiplegia, aphasia, intracranial infection and cranial nerve dysfunction. (2) Via vein: retrograding via the femoral vein and converging via the sinus to Galen vein or forebrain central venous. The timing for the shunting of hydrocephalus is also very important. Shunting before the procedure might lead the Galen veins to dilate and result in deterioration.

Conclusions

Intravascular embolization was safe and effective for adult VGAD patients, and worthy of clinical application.

Conflicts of interest

The authors declare no conflicts of interest.

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