

Giant Vertebrobasilar Fusiform Aneurysm as a Cerebellopontine Angle Mass

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SUMMARY

Introduction According to the literature, a fusiform aneurysm located in the cerebellopontine angle (CPA) is an extremely rare condition.

Case Outline We report a case of a 59-year-old patient with initial dizziness and left-sided sensorineural hearing loss that had gradually developed over six months. Vertebrobasilar fusiform aneurysm, with intraluminal thrombus, which was displaced to the right cerebellopontine angle, creating mass effect, was diagnosed using brain magnetic resonance imaging and magnetic resonance angiography.

Conclusion Atherosclerosis may be the essential factor in the pathogenesis of a fusiform aneurysm of the basilar artery, especially in elderly patients. The best treatment option is yet to be determined, but in spite of numerous previous large studies, personalized approach is probably the best.

Keywords: fusiform vertebrobasilar aneurysm; basilar artery; cerebellopontine angle

INTRODUCTION

The predominant lesions in cerebellopontine angle (CPA) are different benign tumors, and schwannoma are the commonest [1, 2]. Vascular lesions are not so frequent, and vertebrobasilar fusiform aneurysm, with estimated incidence of 0.06–5.8%, are rarely located in the CPA [3]. Unruptured intracranial aneurysms usually present with symptoms of raised intracranial pressure, such as headaches, nausea, vomiting and blurring of vision, or with cranial nerve deficits, embolic ischemia and mass effects. Otological symptoms are extremely rare and include pulsatile tinnitus, vertigo, and progressive and sudden sensorineural hearing loss [4]. Treatment of fusiform aneurysms is divided into conservative and surgical approach. We report a case of a patient with unruptured giant vertebrobasilar fusiform right-sided aneurysm with intraluminal thrombus, diagnosed using brain magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA), which raises several questions related to the pathogenesis of recurrent ischemic events and medical management. Written informed consent was obtained from the patient who participated in this case.

CASE REPORT

The patient was a 59-year-old man with past medical history of treated hypertension. He was suffering from chronic headache and occasional dizziness. Two months before hospital admission, the patient had right side facial hypoesthesia and difficulties with eating.

On admission, the patient's blood pressure was 130/70 mmHg with a sinus heart rhythm. Neurological tests revealed evidence of left-sided nystagmus, truncal ataxia, vertigo, dysarthria and dysphagia. He also presented right-sided facial hypoesthesia, absent right corneal reflex, loss of taste sensation and hearing loss in the left ear. Caloric testing with ice water revealed a normal response in the right ear, but no response in the left one. Cerebellar tests were positive and there was right deviation of uvula.

Brain MRI (1.5 TE Siemens, Avanto, Erlingen, Germany) confirmed the presence of an elongated, tubular, tortuous, hyperdense, fusiform, extra-axial, 35 mm long mass lesion, which compressed the porus of the left internal auditory canal and extended into the CPA causing compression of the left cerebellar hemisphere, rotation of the brainstem to the right and minimal compression of the fourth ventricle (Figures 1 and 2). MRA (1.5 TE Siemens, Avanto, Erlingen, Germany) showed an ectatic basilar artery markedly displaced to the left (Figures 3 and 4), which coincided with the lesion in the left CPA. The patient was diagnosed with having a fusiform vertebrobasilar aneurysm with thrombus formation. There was a double lumen and recent hemosiderin deposition. We decided to treat the patient with anticoagulation therapy.

DISCUSSION

Interest in studying fusiform aneurysms has increased recently because little is known about their pathogenesis and the best way of their management. The fusiform type is the rarest

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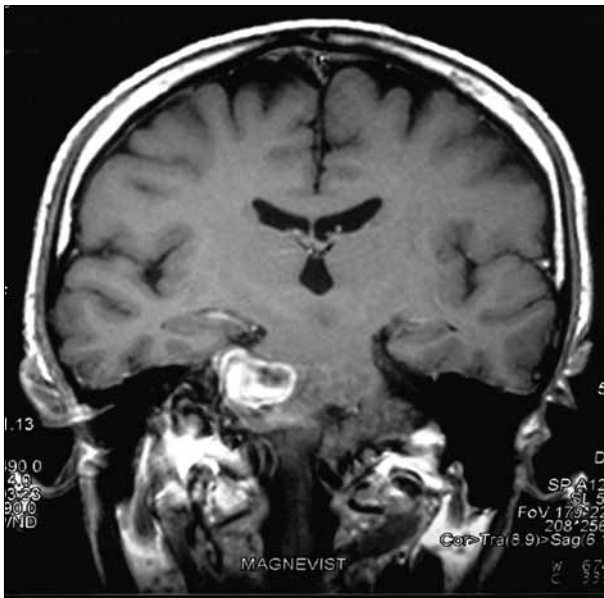


Figure 1. Coronal brain MRI showing a giant hyperdense tubular extra-axial lesion in the right CPA with significant compression



Figure 2. Axial MR images with gadolinium show a partial thrombosed fusiform aneurysm of the basilar artery and a double lumen of the fusiform basilar dissection. The external wall of thrombosed aneurysm is with recent hemosiderin deposition.

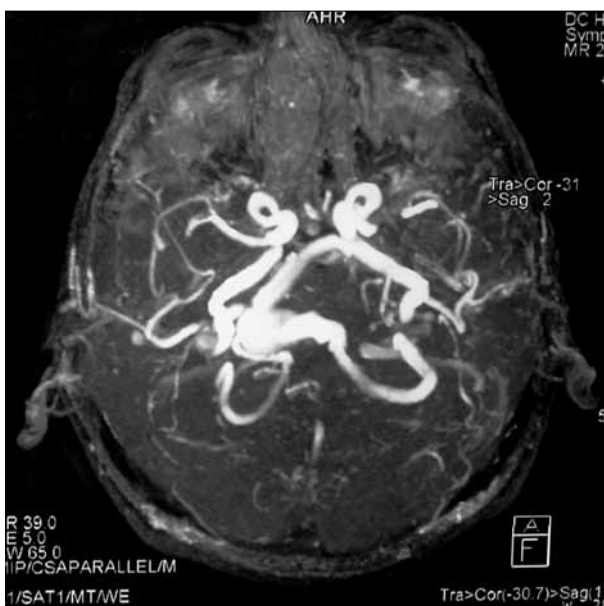


Figure 3. MRA showing an ectatic basilar artery markedly displaced to the right



Figure 4. Digital subtraction angiography image

form of vertebrobasilar aneurysm, characterized by dilatation and elongation of an artery [5]. The origin of fusiform aneurysms is unclear and several hypotheses exist. They are most common in elderly patients with advanced atherosclerosis and hypertension, and are believed to be the result of a degenerative process of the arterial wall [6]. Contrary, other authors stated that out of 120 patients with giant fusiform aneurysms, who underwent surgical treatment, atherosclerosis was found in only six of them. A congenital anomaly, mechanical injury by post-stenotic turbulence, intimal disruption from arterial dissection and severe reticular fiber deficiency in the muscle layer have been proposed as alternative explanations for the formation of fusiform aneurysms [7]. It was speculated that initial event in the formation of

a fusiform aneurysm is lipid deposition in and beneath the intima that disrupts the internal elastic lamina and infiltrates the muscular wall. The resultant atrophy of the elastic substance and the musculature then leads to tortuosity of the vessel due to high intravascular pressure causing the ectatic vessel to expand in diameter and length. Our patient was a 59-year-old man with past medical history of treated hypertension. He had no history of mechanical trauma or congenital coronary illness in his family.

The natural tendency for a fusiform aneurysm is to slow down the circulation, because of this increased luminal diameter, and to expand and produce mass effects on nearby structures. Repeated thrombosis near the wall also makes the vessel stiff and thick. The expanding fusiform aneurysm

can distort vascular branches, reducing distal flow, or can even serve as a nidus for clot formation and distant embolization. This clinical evolution was also seen in our patient.

Otological symptoms are rarely seen but our patient initially had a unilateral hearing loss, as well as a characteristic form of nystagmus called Bruns nystagmus, caused by lateral brainstem compression. Right-sided hypoesthesia could be explained by the aneurysm's compressive effect, which displaced the brainstem contralaterally. This characteristic form of nystagmus is a combination of low-frequency, horizontal nystagmus on looking ipsilaterally and high-frequency, small amplitude on looking contralaterally, but it is observed in patients with large tumors located in the CPA [8].

Two types of fusiform cerebral aneurysms are reported: acute type, presented as subarachnoidal hemorrhage or stroke, and chronic type that rarely bleeds [9]. Basilar aneurysms can be graded according to their diameter into small (<12 mm), large (12–25 mm) and giant (>25 mm). Considering that our patient had a long history of treated hypertension, headaches, and dizziness, we presume that he had a small intracranial aneurysm with slow progression to a giant aneurysm of 35 mm. According to the literature, this is also one of the biggest fusiform aneurysms in this region.

Treatment of fusiform aneurysms is divided into conservative and surgical approach, but there is no general agreement regarding the treatment, nor have consistently successful results been reported in the literature. Small advantage is given to conservative therapy, since surgery and endovascular embolization are very risky. Antithrombotic medication is as effective as anticoagulation in stroke prophylaxis in the setting of fusiform aneurysm. Median survival rate of patients with intracranial fusiform aneurysms who were treated conservatively was 7.8 years and death was most commonly caused by ischemia [10]. Considering the abovementioned data, we chose conservative therapy for our patient, with anticoagulants and a policy of vigilant follow-up.

We emphasize the need to think of a giant fusiform aneurysm as differential diagnosis when mass in the CPA is presented, diagnosed using brain MRI and MRA. We speculate that a fusiform aneurysm was the final outcome of dynamic pathological process of the arterial wall. Hypertension and atherosclerosis may be the essential factors in the pathogenesis of a fusiform aneurysm of the basilar artery, especially in elderly patients. More prospective, randomized studies are needed to find the best treatment for this pathological vascular change.

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Џиновска вертебробазиларна фузиформна анеуризма као маса у понтоцеребеларном углу

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КРАТАК САДРЖАЈ

Увод Према подацима из литературе, фузиформна анеуризма која се налази у понтоцеребеларном углу је изузетно ретко стање.

Приказ болесника Приказујемо 59-годишњег болесника с почетним вртоглавицама и губитком слуха на лево уво који су се постепено развили током шест месеци. Вертебробазиларна фузиформна анеуризма с интралуминалним тромбом, који је расељен у десни понтоцеребеларни угао, стварајући мас-ефекат, дијагностикован је применом магнетне

резонанције мозга и магнетнорезонантне ангиографије. Атеросклероза може бити суштински фактор у патогенези фузиформне анеуризме на базиларној артерији, посебно код болесника старијег животног доба.

Закључак Најбоља опција лечења тек треба да се утврди, али упркос бројним ранијим великим студијама, персонализовани приступ је вероватно најбољи.

Кључне речи: фузиформна вертебробазиларна анеуризма; базиларна артерија; понтоцеребеларни угао