CASE REPORT

VERTEBROBASILAR AND BILATERAL CAROTID DOLICOECTASIA: A RARE ENTITY

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ABSTRACT

Dolichoectasia is a term used to describe marked widening, tortuosity and elongation of an artery. Intracranial vertebral and basilar arteries are most commonly involved. Verteobasilar dolichoectasia is usually asymptomatic. Both verteobasilar and bilateral carotid dolichoectasia has been reported very rarely in the literature. Cranial nerve compression and cerebral ischemia findings are frequently seen in symptomatic patients. We reported a 67-year-old female, without medical or family history for cerebrovascular disease, presented with verteobasilar and bilateral carotid dolichoectasia and subarachnoid hemorrhage, manifesting as reduced level of consciousness and weakness, and left abducens palsy.

Key Words: Verteobasilar, Bilateral Carotid, Dolichoectasia.

INTRODUCTION

Intracranial arterial dolichoectasia (IAD) is defined as an increase in the length and diameter of the intracranial arteries (1). It is characterized by dilation and elongation of cranial arteries. The prevalence of dolichoectasia has been estimated to be 0.05%-0.06% (2). IAD most frequently involves the vertebral and basilar arteries. Involvement of both the verteobasilar and carotid systems is extremely rare (3) Dolichoectasia is frequently associated with ischemia and rarely with intracranial hemorrhage (2). We present a rare entity with abducens paresis and subarachnoid hemorrhage, also with fatal dolichoectasia involving both the verteobasilar and carotid artery systems.

CASE

A 67-year-old woman was admitted to the emergency department of a local hospital with headache, consciousness and weakness. Computed cerebral tomography (CT) was performed at local hospital and revealed subarachnoid hemorrhage signs at posterior fossa and right temporal lobe sulci and also showed dilatation of the verteobasilar and bilateral carotid artery (Figure 1a). Then the patient was taken to our neurology clinic for evaluation. She had essential hypertension. Neurologic examination revealed tetraplegia and left abducens palsy. Respiratory functions, body temperature, the blood count, C-reactive protein, serum glucose, erythrocyte sedimentation rate, thyroid function tests and the

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Figure 1a: Cerebral Tomography shows subarachnoid hemorrhage signs at posterior fossa and right temporal lobe sulci and also shows dilatation of the bilateral carotid and posterior cerebral artery.

Figure 1b: Control Cerebral Tomography: Subarachnoid hemorrhage signs were disappeared.

other hematological investigations were all within normal limits. We performed firstly CT angiography and then catheter angiography in our interventional neurology clinic. Angiogram shows symmetric fusiform dilation of the cavernous and supraclinoid segment of both carotid artery and severe dilated distal segment of the left vertebral artery and basilar fusiform aneurysmal dilatation. (Figure 2a and 2b). Both carotid, left vertebral and basilar arterial diameters were over 4.5 mm. Cerebral infarcts of the left parieto-occipital lobe, left basal ganglion and the brainstem were demonstrated in diffusion-weighted ADC images (Figure 3). After 20 days later, control CT was performed again and it revealed that subarachnoid hemorrhage signs were disappeared (Figure 1b). Patient was treated conservatively, any surgical or antiagregan and low molecular weight heparin was administered. However there was not enough improvement in neurological examination during hospitalization. We have treated and followed up the patient in Neurologic Intensive Care Unit nearly two months. During this period the patient developed pneumonia and respiratuary failure, then mechanical ventilation and antibiotic therapy were applied. But we did not observed any improvement and the patient died 2 months after admission, due to respiratuary complications.

DISCUSSION

Atherosclerotic degeneration of the vascular wall, either alone or associated with arterial hypertension, has been proposed as the initial pathogenic factor in the development of IAD

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Ischemic lesions may possibly be caused by emboli derived from thrombi or fragments of plaques in the walls of the enlarged arterial segment (2). These patients are usually elderly and there is a left-sided predominance of symptoms. In our patient cerebral infarcts of the left parieto-occipital lobe, left basal ganglion and the brainstem were demonstrated in diffusion-weighted images.

Subarachnoid hemorrhage is not a common issue in vertebrobasilar dolichoectasia of the atherosclerotic type, whereas the incidence of subarachnoid hemorrhage increases in congenital or dissecting type vertebrobasilar dolichoectasia. Hemorrhage is associated with the degree of ectasia and the elongation of the basilar artery and may be favoured by hypertension and use of antiplatelet or anticoagulant agents (4). As mentioned above, we observed subarachnoid hemorrhage especially at posterior fossa in CT images and in angiography, basilar fusiform aneurysmal dilatation was detected. Subarachnoid hemorrhage disappeared after twenty days later.

Dysfunction of one of the ocular motor cranial nerves due to vertebrobasilar dolichoectasia is very rare (4). Like our patient, it has been rarely associated with abducens paresis. When vertebral artery is dolichoectatic, it deviates from its course ventral to the brainstem and may compress the cranial nerves, most frequently as they emerge from the brain stem (root entry zone). The facial and trigeminal nerves are the mostly affected ones. Isolated abducens nerve palsy related to dolichoectatic vertebral artery (DVA) compression is very rare (6,7). In addition DVA can produce ischemic stroke, transient ischemic attacks, and intracerebral hemorrhage. Abducens nerve palsy usually results from brainstem ischemia, hemorrhage, infiltration of tumor or vascular compression (7).

Death was likely a result of cerebral infarction due to obstruction of the basilar artery and compression of the brainstem (3). The prognosis for patients with vertebrobasilar ectasia may depend mainly on the pathological changes in the basilar artery (5).

Traditionally, the diagnosis of dolichoectasia has been based on catheter angiography. In our patient catheter angiography was used to diagnose dolichoectasia. Currently, there is no generally
accepted method to quantitatively diagnose IAD. The most commonly used criteria for the diagnosis of IAD includes an arterial diameter of over 4.5 mm at any location along its course, and deviation of any portion by over 10 mm from the shortest expected course. In our patient both carotid, left vertebral and basilar arterial diameters were over 4.5 mm (1).

In conclusion, we have presented a case of a very rare form of IAD that involves the vertebrobasilar and both carotid artery systems with isolated abducens palsy and also with subarachnoid hemorrhage and cerebral ischemia. The diagnosis of such rare entities may have importance in the treatment of vascular lesions of the brain.

REFERENCES