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Arteriovenous Malformation Leading to Triple Aneurysm of the Posterior Inferior Cerebellar Artery: A Rarity

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Abstract: ***Aneurysmal rupture is the most common cause of spontaneous non traumatic subarachnoid and intraventricular haemorrhage. Saccular aneurysms (aka Berry aneurysm) are the most common phenotype of intracranial aneurysm that arise from branch points of major cerebral arteries with maximal hemodynamic stress. 10% of these aneurysms are seen to involve the posterior circulation. Posterior inferior cerebellar artery (PICA) is a branch of vertebral artery and is an uncommon site for intracranial aneurysm. The present report describes a 50-year old chronic alcoholic and hypertensive with history of sudden onset altered sensorium presenting with multiple aneurysms in the PICA associated with arteriovenous malformation on CT angiography scan.***

Keywords: triple aneurysm, posterior inferior cerebellar artery, arteriovenous malformation, subarachnoid haemorrhage

1. Introduction

Non-traumatic subarachnoid haemorrhage and intraventricular bleed are most commonly the result of ruptured aneurysm. Intracranial aneurysms are classified by their phenotypical appearance. Most common intracranial aneurysm is saccular or “berry” aneurysm. They are true aneurysms and arise from the branch points of major cerebral arteries in which hemodynamic stress is maximal. Most aneurysms are acquired while few can be congenitally present. Conditions such as Autosomal dominant polycystic kidney disease, inherited connective tissue disorders like Marfan’s syndrome, Ehler-Danlos syndrome II and IV, neurofibromatosis type I and anomalous vasculature like bicuspid aortic valve, coarctation of aorta, persistent trigeminal artery, congenital anomalies of the anterior cerebral artery, all carry an increased risk of saccular aneurysm. Upto 20 % of patients have a family history of intracranial aneurysms.

90% of the saccular aneurysms occur in the anterior circulation. Approximately one-third of saccular aneurysms occur in the anterior communicating artery with another one-third arising from the junction of the internal carotid artery and posterior communicating artery. 10% saccular aneurysms arise from the posterior circulation. The basilar artery bifurcation is the most common site with the posterior inferior cerebellar artery being the second most common.

2. Clinical History

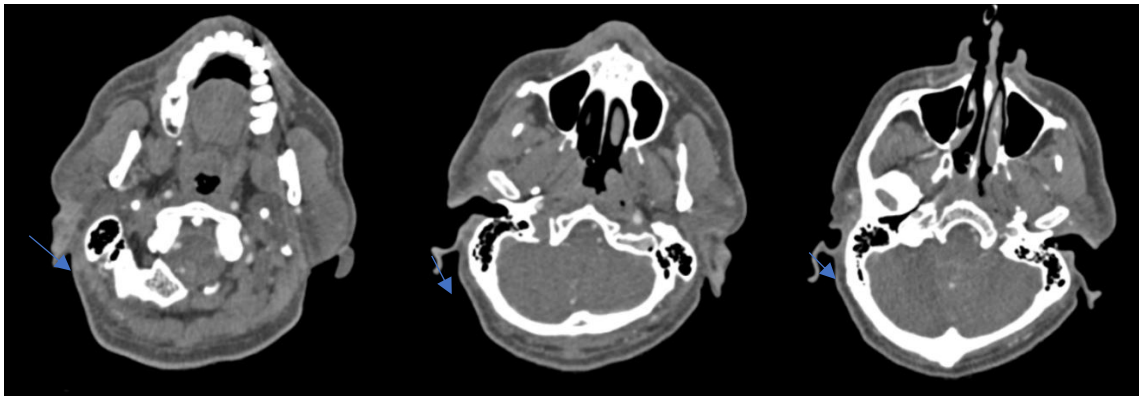
50 year-old chronic alcoholic and known case of hypertension on treatment. Patient was found unresponsive at home and was brought to emergency services. On examination he was drowsy and intermittently obeyed command. Pupils were sluggishly reactive. Rest of the neurological examination was within normal limits.

On performing CT scan of brain and CT Angiography an intraparenchymal bleed in cerebellar vermis measuring 8.6 × 5.5 × 8.3 cm (AP × TR × SI) in size was noted with intraventricular extension into the fourth ventricle, third ventricle and bilateral lateral ventricles, cerebello-medullary cisterns and posterior subarachnoid space. Early hydrocephalus was noted. There was a focal outpouching at the distal segment of right posterior inferior cerebellar artery (PICA), arising from telovelo-tonsillar segment (P4 segment). Another focal outpouching was seen arising from the tonsillo-medullary segment (P3 segment). Third focal out pouching was seen arising from the cortical branches (P5 segment). The left vertebral artery was seen to arise directly from the aorta.

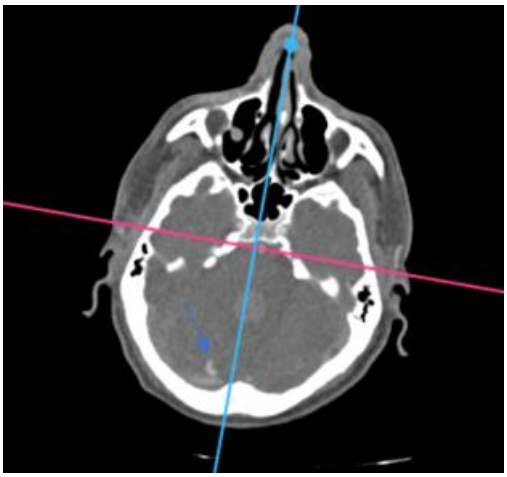
MRI of Brain was performed which showed an acute infract in right cerebellar hemisphere and right high parietal region. Intraventricular haemorrhage was seen into occipital horns of bilateral lateral ventricles and third ventricle.

Digital subtraction angiography revealed a small right cerebellar AV malformation fed by feeders from right posterior cerebral artery and right PICA draining via an early draining vein into right transverse and sigmoid sinus. Multiple feeding artery aneurysms were seen in the right PICA (p3, p4, p5 and p6 segments).

Embolization of the right cerebellar arterio-venous malformation was performed with coiling of the feeding right posterior inferior cerebellar artery aneurysms.



The above images display focal contrast filled outpouchings at the p5, p4 and p3 segments of the right postero-inferior cerebellar artery (blue arrows).



Right cerebellar AV malformation (blue arrow).

3. Discussion

A subarachnoid hemorrhage due to the rupture of an intracranial aneurysm is a devastating event associated with high rates of morbidity and mortality. Approximately 12 percent of patients die before receiving medical attention, 40 percent of hospitalized patients die within one month after the event, and more than one third of those who survive have major neurologic deficits. [1]

The prevalence of aneurysms is increased in certain genetic diseases; the classic example is autosomal dominant polycystic kidney disease (ADPKD), Ehlers-Danlos syndrome, neurofibromatosis, and a1-antitrypsin deficiency and Marfan's Syndrome. Aneurysms also run in families with a prevalence of 7% to 20% in first or second-degree relatives of patients who have suffered a SAH. [2-3]

The symptoms of a ruptured intracranial aneurysm include headache, unilateral third cranial nerve palsy (from a posterior communicating artery aneurysm), bilateral temporal hemianopsia (from an anterior communication artery aneurysm impinging on the optic chiasm) ischemic cerebrovascular disease, poorly defined spells, and seizures. [4-5]The gold standard for diagnosis of intracranial aneurysms is currently IADSA, but a diagnosis can also be provided by CTA and MRA. On CTA, aneurysms will appear as a rounded, spherical mass with attenuation equal to that of the large vessels. On MRA, the lumen of the aneurysm will appear as a flow void, meaning that it does produce a magnetic signal because blood flow has slowed due to the larger cross-sectional area of the aneurysms compared to the nearby normal vasculature. Areas of high signal intensity surrounding the flow void area of the aneurysm on MRA likely represent a rupture with subsequent hemorrhage: the high signal pattern is produced by stagnant blood. [6]

As mentioned above, the gold-standard for diagnosis of cerebral aneurysms is IADSA because it remains the test with the highest spatial resolution. However, IADSA is costly and invasive with a 2–4% transient complication rate and about 0.5% of patients undergoing catheterization are left with permanent neurological complications. [7,8,9]

The parameters suggesting that a patient should not undergo intervention include: lack of symptoms, aneurysm size <7mm, lesion in the anterior circulation, age older than 64 years, and no personal or family history of SAH. Patients younger than 50 years with symptomatic aneurysms >25mm located in the posterior circulation and a personal or family history of SAH should undergo intervention. [4]

Endovascular treatment consists of guiding a catheter from the femoral artery to the cerebral vasculature via the ICA or vertebral artery, depending on the location of the aneurysm. The procedure is guided by fluoroscopy, and when the catheter has reached the aneurysm, several soft platinum coils are deployed in the lumen of the lesion. These coils completely fill the lumen and induce the formation of a thrombus to occlude the aneurysm, preventing future rupture. [4]

Complete occlusion is achieved in 80% to 90% of patients. At post-treatment follow-up, however, small neck remnants are common, and some degree of thrombus recanalization is observed in 50% of all patients and up to 90% of patients with giant aneurysms. Both neck remnants and recanalization are associated with a risk of rupture, and 20% of patients may require more than one coiling procedure. [10]

Surgical clipping involves placing a surgical clip at the junction of the healthy artery and the neck of the aneurysm. This treatment is very effective, demonstrated by annual risks of rupture following clipping reported from 0% to 0.9%. [4]

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