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A Rare Case of Intraparenchymal Haemorrhage with Secondary Intraventricular Extension Due to Ruptured Berry Aneurysm with Out Subarachnoid Haemorrhage

KEYWORDS: Berry Aneurysm and Anterior Communicating Artery.

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ABSTRACT *Berry aneurysm is a saccular dilatation of a localized segment of an artery. The incidence or berry aneurysm is 2%.This is a rare case report of intracerebral rupture of anterior cerebral artery aneurysm and secondary intraventricular extension with out subarachnoid haemorrhage. The patient clinical history, examination, findings, investigation findings, the epidemiology of this pathology and treatment options Prognosis are discussed.*

INTRODUCTION:

Dilatation of a localized segment of the arterial system is called aneurysm. Morphologically it can be saccular also known as Berry aneurysm, fusiform or dissecting. [1]Aneurysms occur most commonly at the junction of two arteries in the circle of Willis. Deficiency in the tunica media leads to weakness of the vessel wall resulting in its local dilatation.(2)Incidence of Berry aneurysm is 2% and the incidence of aneurysmal rupture is 6-12/100000 persons per year. The female: male ratio is 3:2 and the risk factors include atherosclerotic disease, family history, and polycystic kidney disease. The size of berry aneurysm ranges from a few mm to cm. A berry aneurysm above 2.5 cm is called giant Aneurysm. [3] aneurysms are thin walled and at risk of rupture.

Berry aneurysms frequently rupture into subarachnoid space accounts for 70-80% of spontaneous subarachnoid hemorrhage may also rupture results in intraparenchymal, intraventricular, or subarachnoid hemorrhage.Subarachnoid haemorrhage(SAH) is characterized by sudden severe thunder clap headache, vomiting, neck stiffness, loss of consciousness, focal signs, epilepsy, coma, and sudden death. In 95% of cases CT scan of head is diagnostic [4,5].Though intracerebral(ICH) and intraventricular haemorrhage(IVH) are not uncommon after an aneurysmal rupture ,ICH and IVH with out SAH are rare(<2%).In such cases high index of suspicion is needed to deliver correct management.

CASE REPORT:

A 50-year-old middle aged man presented to emergency services department with sudden loss of consciousness and two episodes of GTCS type of seizures. He has H/O similar episode one month back but after regained consciousness but patient had persistent severe headache. He is not a known hypertensive or diabetic but known smoker since 30 years. On examination patient was unconscious with GCS-5, PR 80 /min, BP 160/100 mmHg, RR: 18/min, other systems are normal. CNS: patient was unconscious, neck stiffness present, planters are bilateral extensors. He was investigated initially with non contrast computerized tomography, plain CT brain as shown intra cerebral bleed in right frontal lobe with secondary intraventricular extension following rupture of aneurysm anterior cerebral artery territory followed by a CT angio was planned after three days and findings were anterior communicating artery – absent, right anterior cerebral artery - Absent or non visualized, left anterior cerebral artery -? Duplication of A2 segment and left ACA – small Berry aneurysm measuring 9.8 x 10.0 x 10.4mm noted at the junction of left A1 and A2 segment. Narrowing of M3, A3 segments bilaterally remaining vertebrobasilar and cavernous and supraclinoid portions and internal carotid artery has normal course and-Caliber other investigations U/S abdomen – normal.ECG – Normal, 2D-Echo – Normal.

As the patient condition was poor, he was managed conservatively with anti-seizure medication, to prevent vasospasm calcium channel blocker, nimodipine was used, to decrease raised intracranial tension mannitol and other medication for symptomatic relief are given. Initially, patient condition improved and he regained consciousness but the patient condition suddenly deteriorated probably due to re-bleed and the patient died.

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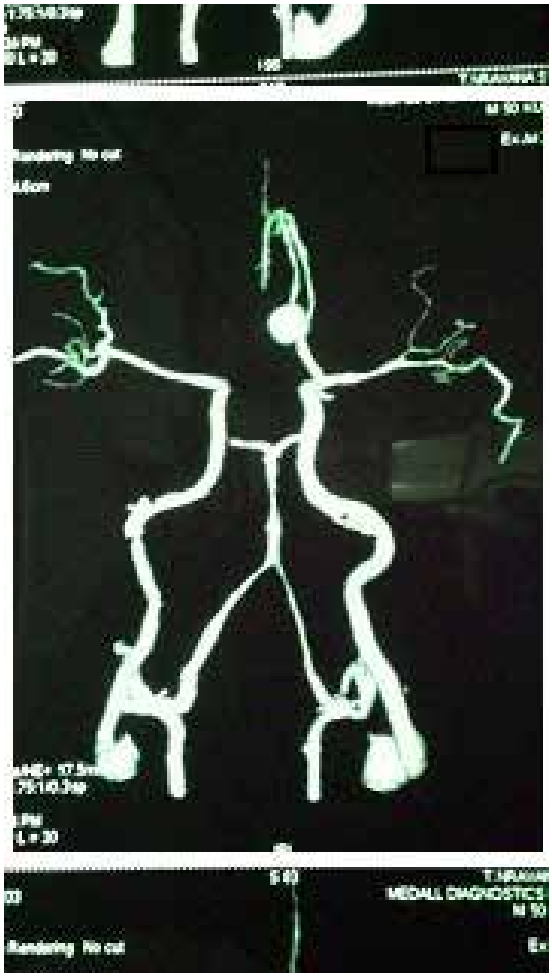

Fig: CT angio showing no anterior communicating artery & right anterior cerebral artery - Absent or non visualized, left anterior cerebral artery -? Duplication of A2 segment and left ACA – small Berry aneurysm measuring 9.8 x 10.0 x 10.4mm noted at the junction of left A1 and A2 segment.

Discussion:

Berry aneurysm is by far the commonest of all cerebral aneurysms, 25% of cerebrovascular deaths are due to ruptured Berry aneurysm. Risk factors for rupture of aneurysm are hypertension, alcohol abuse, drug abuse, smoking, size of aneurysm. < 7 mm has less risk of rupture. Giant aneurysm > 25 mm and large aneurysm 12 – 25 mm produce eye symptoms due to compression over optic nerve. In 25% of persons older than 55 years the Berry aneurysms are silent. [4]

Berry aneurysms can rupture at any time during exertion or at rest. Ruptured Berry aneurysms leads to sudden death due to subarachnoid hemorrhage.[5] The incidence of SAH is 6 – 8/10000 persons years, peaking in the sixth decade. Aneurysmal rupture may present with intraparenchymal hemorrhage intra ventricular hemorrhage (13 – 28%). Presenting symptoms of aneurysmal rupture are headache, typically described as thunder clap headache, stiffness of neck, brief lapse of consciousness, vomiting, hypertension, impaired state of consciousness seizures[6] Sentinal warning headache results from an aneurysm leaks for days to weeks prior to rupture only in minority of individuals has headache prior to rupture.Neurological deficits in aneurysmal rupture may reflect intraventricular hemorrhage [79%].Intraparenchymal hemorrhage [69%], acute hydrocephalus [25%] or frontal lobe stroke syndromes [20%]. Prevalence of saccular cerebral aneurysms in the asymptomatic general population has been reported over a wide range of 0.2-8.9 % when examined angiographically and in 15-30 % of these patients multiple aneurysms are found. [7]

A familial tendency to aneurysms were also recognized with patients who have more than one first degree relative affected having a 17-44 % chance of themselves having an aneurysms.Numerous associations have been identified more relative to abnormal connective tissue. Associations include Elhers Danlos syndrome (type IV), Marfans syndrome (controversial)[8],Autosomal dominant polycystic kidney disease[5],Coarctation of aorta[6]'Bicuspid aortic valve,Neurofibromatosis type1[8],Alfa-1 antitrypsin deficiency,cerebral arteriovenous malformation,fibromuscular dysplasia.Approximately 90% of aneurysm arise from anterior circulation and 15-30% of the patients have multiple aneurysms[9].Anterior circulation :anterior cerebral artery/Anterior communicating artery:30-40%,supraclinoid Internal carotid artery and ICA/posterior communicating artery :30%,Middle cerebral artery[M1-M2] junction bifurcation:20-30%,posterior circulation:10% consists basilar artery tip,superior cerebellar artery ,posterior inferior cerebellar artery.

Berry aneurysms can be imagined in a variety of methods such as CT angiography [CTA]/MR Angiography (MRA),Digital substraction angiography [DSA].

In CT appearance depends upon the presence of thrombosis within the aneurysm.Aneurysm appears as well defined round slightly hyperattenuated lesions.Calcifications can be present.Post contrast patent aneurysm seen as bright and uniform enhancement ,thrombosis in aneurysm :Rim enhancement due to filling defect.On MRI also patent and thrombosed aneurysm display different imaging features.On T1 most of the patent aneurysm appears as flow void,they may show heterogenous signal intensity.In thrombosed aneurysms appearance depends on the age of the clot with in the lumen. In T2 typically hypo-intense, laminated thrombosed may show a hyper-intense rim.

Treatment of large or symptomatic aneurysm should be considered with either endovascular coiling or surgical clipping. Management of small aneurysms is controversial. Less than 7 mm in maximal diameter aneurysms are statisticallylow risk to rupture.

Five year cumulative risk of rupture of anterior circulation aneurysms [10]. < 7 mm :0%, 7-12 mm : 2.6%, 13-24 mm : 14.5%, > 25 mm : 40% Posterior circulation aneurysm [5]. < 7 mm : 2.5%, 7-12 mm : 14.5% , >13-24 mm : 18.4%, > 25 mm : 50%.

Prognosis for a person who's aneurysm has ruptured is largely dependent on age, general health, pre-existing neurological condition. 40% of individuals who's aneurysm has ruptured do not survive the first 24 hours, up to another 25% die or complications within six months.

Conclusion:

Rupture of Berry aneurysm can cause a subarachnoid hemorrhage, cerebral hematoma, and/or intraventricular hemorrhage .In our case ruptured berry aneurysm presented as cerebralhemorrhage with secondary intraventricular exten-

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sion with out subarachanoid hemorrhage . As in our case calcified wall of aneurysm is seen in non contrast CT and after rupture of right sided Berry aneurysm. Right sided anterior cerebral artery is not visible and left side intact Berry aneurysm is seen in CT angiogram. Even in the absence of subarachnoid blood, intra ventricular bleed on CT deserves prompt investigation of aneurysmal rupture.

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