CAVERNOMA IN THE LEFT TEMPORAL LOBE OF BRAIN - A RARE CASE REPORT

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ABSTRACT
Vascular malformations of the central nervous system are rare to occur and they are the products of abnormal angiogenesis of cerebral vasculature. Cavernous types of angiomas are most common in the region of rolandic fissure; next common site is temporal lobe. They commonly present with seizures and focal neurological manifestations. Here in we present this case of ours a 60 years old female who presented with recurrent seizures, giddiness and headache since 6 months. Histopathology revealed the diagnosis of cavernous angioma.

Key Words: Arteriovenous Malformation, Cavernous Type, Hemorrhages, Hemosiderin Laden Macrophages and Vascular Channels

INTRODUCTION
Vascular malformations of the central nervous system are non-neoplastic benign lesions which arise as developmental anomalies of the cerebrospinal circulation. There are four categories: Capillary telangiectasias, Angiomas (venous and cavernous type), Arteriovenous malformation and Transitional type or hybrid type. Cavernoma also known as Cavernous angioma or cavernous malformation (CM), is a vascular malformation characterized by the presence of sinusoid like capillary vessel containing blood in a very sluggish circulation (Curling et al., 1991).

Usually they are small having a diameter of at least 25mm but giant cavernous malformation having a diameter of greater than 6cm can also be found. Cavernomas account for approximately 5-10% of vascular malformation. Usually presents in 2nd and 4th decade with male and female in equal preponderance (Rigamonti et al., 1987).

The cavernous angioma, the other name is cavernoma, is different from other vascular malformations because its constituent vessels are arranged in compact, globose mass, devoid of intervening neural tissue. It consists of ectatic and fibrous walled vascular channels, without intervening neuroglial tissue, hemosiderin laden macrophages, dystrophic calcification and metaplastic ossification and thrombosis can also occur (Rosai & Ackerman, 2011).

CASE REPORT
A 60 year’s old female presented with recurrent seizures, giddiness and headache since 6 months. Vomitings for the past one week. Patient attained menopause 6 years back. Past history not significant. Central nervous system examination: Motor, sensory and cranial nerves examination were within normal limits. No focal neurological deficits. Routine laboratory investigations including Serum electrolytes were within normal limits. Non-reactive for HIV and HbS Ag. Ophthalmological examination revealed no field defects. Ultrasound abdomen revealed no organomegaly. Electroencephalogram (EEG) showed normal awaked record.

Figure 1: MRI showing well defined lesion in the left temporal lobe of 2x2cm
Radiological Findings
Computerized Axial Tomography (CT scan) imaging of brain – Showed subtle ill-defined hyper dense lesion in left temporal lobe measuring 12x12mm with CSF left around it. Magnetic Resonance Imaging T1W1&T2W1 and FLAIR with a fluid level noted in left temporal lobe-hyper intense well defined single lesion in the left temporal lobe measuring 2x2x1cm below Sylvain fissure, highly vascular tumor with a diagnosis of left temporal space occupying lesion - vascular tumor.

Figure 2 & 3: Haematoxylin and Eosin (10x) Sections showed numerous dilated and congested vascular channels with adjacent gliosis

Figure 4 & 5: Haematoxylin and Eosin (10x and 40x) Sections showed hemosiderin laden macrophages and hyalinised walls

Patient underwent went temporal craniotomy, transcortical approach was done. The mass appeared brownish and had multiple cysts, “Caverns”. Each cavern had brownish liquid blood product and contents were removed by suction, mass removed en mass. Post-Operative status of the patient is uneventful. Gross-we received multiple soft tissue masses altogether measuring 5 cc volume. Cut sections-grey brown areas.

Microscopy
Multiple sections studied show numerous anastomosing vascular channels, some enlarged and filled with blood; some of them showed hyalinised walls and flattened endothelium in some. Focal areas showed hemosiderin laden macrophages and reactive gliotic changes in the glial tissue.

DISCUSSION
Cavernomas are rare neurovascular lesions and poorly described in the literature for their rarity. Most common sites are above tentorium in cerebrum. Posterior fossas, intraventricular cauda equina, spinal cord, sometimes around cranial nerves are rare sites. It can also occur as multifocal lesions (Kivilev, 2010). Usually manifests in childhood but symptomatic lesions are encountered in 3rd and 4th decade of life. Familial cases have autosomal dominant transmission and linked to the mutation of genes involved in integrin mediated angiogenesis. KRIT 1 abnormalities common. They can occur in combination with cavernous angiomas of retina, skin, vertebra and café lait spots. Seizures are the dominant clinical manifestation, head ache, progressive or
transient neurological deficits and sometimes may not produce any neurological deficits. 25% patients present with a hemorrhage and this is a serious complication and death can also occur (Porter et al., 1997). Temporal lobe cavernomas are analyzed to better understand its proliferative factors. Favourable seizure outcome is expected in their sites after lesionectomy. First report of brain cavernoma in 1854 by Luschka. Later descriptions by Rokitansky, Dandy, Krayenbuh and Yasargil were available up to the year 1957. They may be inherited and can present as bilateral and multiple SOL. Autosomal dominant inheritance are also documented. The molecular biology of multiple cavernomas suggest, chromosome 7 abnormalities (Gunel et al., 1996). The mass appears brownish, blackberry like and had multiple cysts, “Caverns”. Each cavern had brownish liquid blood product. T2 imaging – Irregular hyperdense nodules unassociated with edema with minimal mass effect. They may be slightly hyper attenuating or calcified, peripheral hemosiderin produce a black hollow and may be associated with a blooming effect (Griffin et al., 1987).

The differential diagnosis includes a very large number of conditions, such as hemorrhagic neoplasms, inflammatory lesions, and mixed lesions, capillary telangiectasias, and developmental venous anomalies. Treatment is primarily surgical for the cases with non-eloquent locations and the results have been best where complete excision is achieved. Radio surgery may be an alternative for treatment of deep and eloquent area located cavernomas and for the patients who do not accept surgical treatment (Hakan et al., 2004).

CONCLUSION
Microsurgical treatments of cavernoma have significant improvement in symptoms as such in our case. Advances in micro neuro surgery and intra operative monitoring have allowed for safe resection of lesions inside vital structures of the central nervous system.

REFERENCES