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Case Report

AVID triad: a case report

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ABSTRACT

Asymmetric ventriculomegaly, interhemispheric cyst and dysgenesis of the corpus callosum (AVID) constitutes a rare imaging triad. Additional findings include subcortical and subependymal heterotopia, polymicrogyria, fused thalami, deficient falx, and hydrocephalus. The knowledge of this triad helps us to diagnose prenatally by sonography and fetal MRI. In this case report authors present MRI Imaging findings in a case of AVID syndrome in a 6year old male child presenting with history of seizures and delayed milestones.

Keywords: Asymmetric ventriculomegaly, Agenesis, AVID, Corpus callosal dysgenesis, Interhemispheric cyst

INTRODUCTION

The imaging triad of ventriculomegaly, interhemispheric cyst, and callosal malformation has been reported in the literature.[1] However, many cases have been reported postnatally in children and adults. The knowledge of this triad helps us to diagnose prenatally by sonography and fetal MRI.[2]

CASE REPORT

A 6 year old male child presented with H/o episodes of seizures since birth, H/o delayed mile stones and poor school performance. MRI Brain with contrast was done for further evaluation. MRI Brain with contrast shows partial agenesis of corpus callosum with complete absence of the splenium, thinning of the body with normally appearing Genu and Rostum (Figure 1). Asymmetric dilation of the supratentorial ventricle are seen (Figure 2). Two large interhemispheric cystic lesions (in left parafalcine location) in the cerebral hemispheres (along medial left fronto -parietal lobe). One of the cystic lesion shows mild T1 iso intensity and T2 hyperintensity contents and shows mild peripheral enhancement on post contrast study. The other cystic lesion follow CSF intensity (Figure 3).

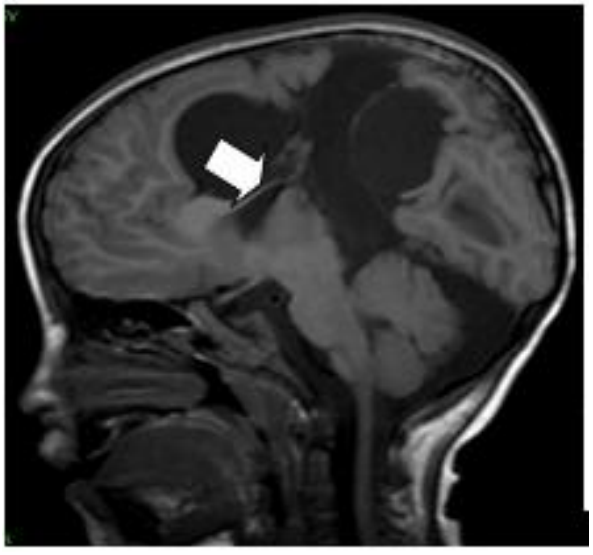

Figure 1: Sagittal T1 weighted image showing thinning of the body of corpuscallosum (arrow).

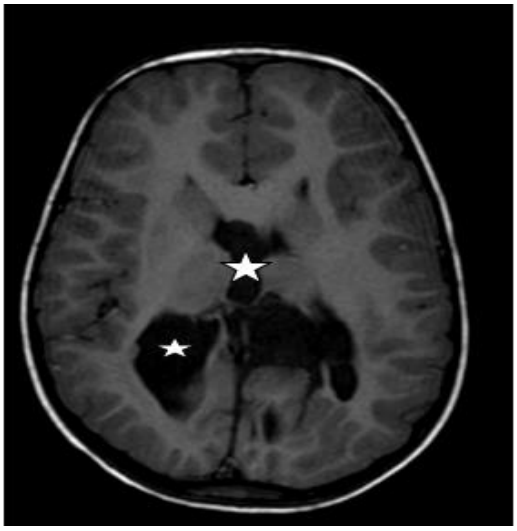

Figure 2: Axial weighted T1W image showing assymetrical dilatation of supratentorial ventricles(*).

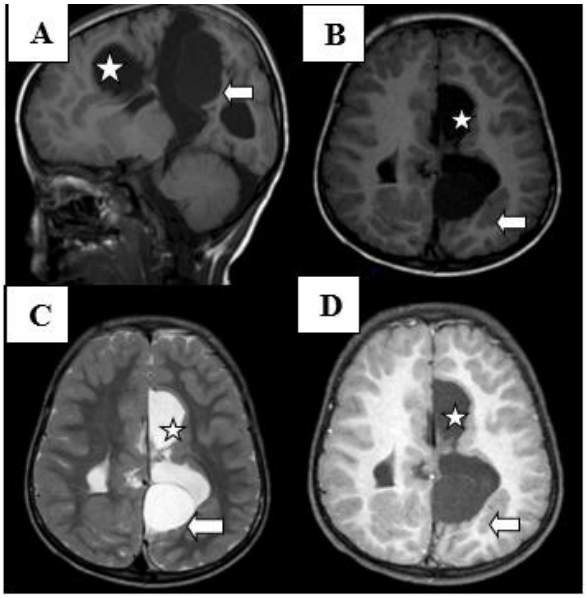

Figure 3: (a) T1W saggital, (b) T1W, (c) T2W and (d) T1 post contrast axial image show two large interhemispheric cystic lesions (in left parafalcine location) in the cerebral hemispheres (along medial left frontoparietal lobe).

No obvious communication of the cystic lesions with the ventricular system - Type 2 cyst in Barkovich et al, classification. Retrocerebellar cystic leison with thin membrance - possibility of arachnoid cyst (Figure 4).

Deformed brain parenchyma in the right parafalcine lobe (medial to the right lateral ventricle) which shows heterogenous enhancement on post contrast study - Gliosis enhancement - sequelae of previous parenchymal insult (Figure 5).

Small nodular heterotropia (measuring ~ 4mm) in the occipital horn of right lateral ventricle (Figure 6).

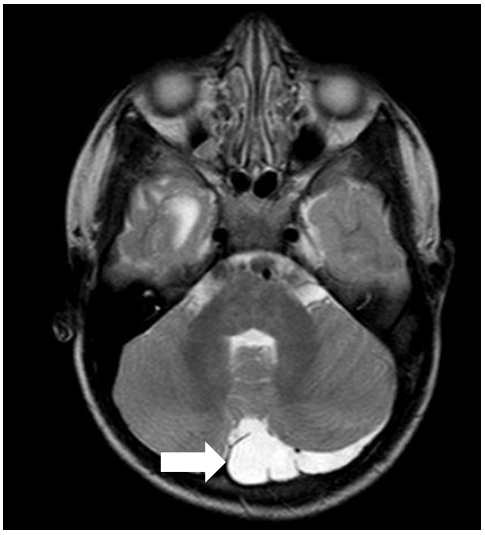

Figure 4: T2W axial image showing Retrocerebellar cyst which is not communicating with the lateral ventricle (Arrow).

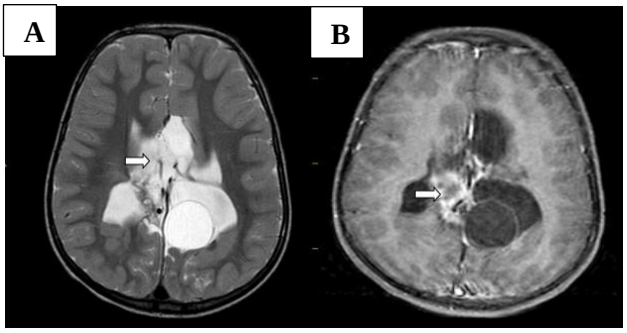

Figure 5: (a) T2W and (b) T1 post contrast axial images show sequelae of previous parenchymal insult (arrows).

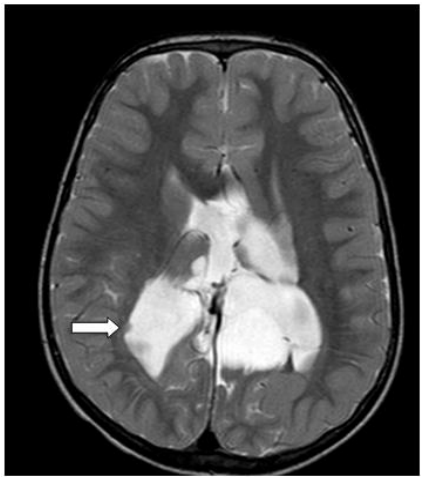

Figure 6: T2W axial image showing nodular heterotropia in the occipital horn of the right lateral ventricle (arrow).

Multiple intracranial cysts associated with partial agenesis of the corpus callosum and assymetrical

ventriculomegaly- AVID TRIAD (Asymmetric ventriculomegaly, interhemispheric cyst and dysgenesis of the corpus callosum) was the final diagnosis

Differential diagnosis includes arachnoid cyst, porencephalic cyst, dermoid/epidermoid cyst, low grade cystic neoplasm (eg, ganglioglioma, DNET).

DISCUSSION

Intracranial cysts and Callosal dysgenesis/agenesis are the congenital anamolies of the brain which can be associated together. Older infants and children develop seiures developmental delay and macrocephaly.[1] The callosal deformity may be either complete or segmental. The cysts may not become apparent by imaging until late in gestation. Two basic types of cysts have been described by barkovich. Type 1 cysts communicate with third and lateral ventricles. Type 2 cysts do not have communication with the ventricles. Type 2 cysts developmental delay and subcortical heterotropia are classically seen in childhood with seizures and developmental delay.[2]

Due to advances in fetal ultrasonography and MRI these abnormalities are frequently identified in utero.[3] CT cannot fully characterize the associated abnormalities like nodular heterotropia. MRI is the preferred modality for complete characterization of the congenital anomalies

With MRI Type 1 cysts typically appear isointense to CSF on all pulse sequences, while type 2 cysts are often hyperintense to CSF on both T1 and T2WI. Type 2 cysts may also be multiloculated, and communication between locules and ventricles may be difficult to visualize.[4] The imaging triad AVID (Asymmetric Ventriculomegaly, Interhemispheric cyst, and Dysgenesis of the corpus callosum) has been proposed to distinction from aqueductal stenosis and porencephaly.[4,5] Associated subependymal and subcortical heterotopia, polymicrogyria, deficient falx, fused thalami and hydrocephalus may also be associated.[1]

Developmental delay and focal neurologic deficits are now shown to vary significantly in both type 1 and 2 cysts, ranging from severe delay and medically refractive seizures to average aptitude, normal social interactions and no clinical findings.[2,6] This patient was seen for a seizures since birth and exhibited mild developmental delay and poor performance in school. Overall, seizures are seen in about half of patients, and lifelong epilepsy is common.

Management includes cyst fenestration or shunting which may result in significant clinical improvement in appropriately selected individuals.

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