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SPONTANEOUS RUPTURE OF INTRACRANIAL DERMOID WITH DISSEMINATED SUBARACHNOID FAT DROPLETS: CLINICAL RADIOLOGICAL REVIEW OF THIS UNUSUAL COMPLICATION

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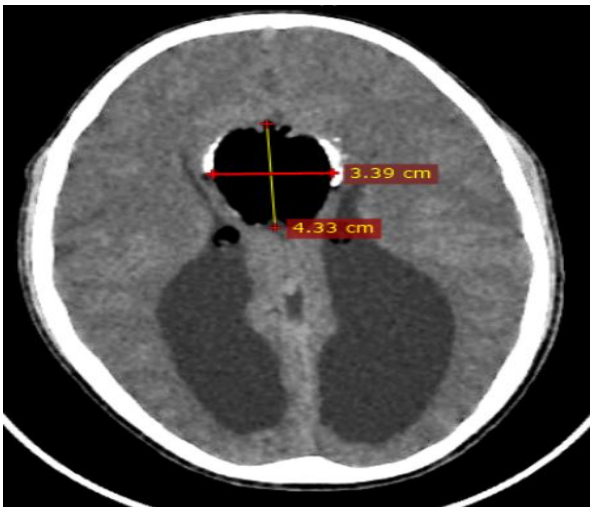
ABSTRACT
Intracranial dermoid cysts are uncommon mass lesions that arise from ectopic cell rests amalgamated in the closing neural tube. Rupture of the intracranial dermoid cyst is a rare complication and is associated with significant morbidity and mortality. We present a case of 14 year old girl with history of refractory seizures who was referred for CT Brain to look for underlying cause.

KEYWORDS: Intracranial dermoid, Seizures, Headache, Computed tomography, Magnetic resonance imaging.

INTRODUCTION
Dermoid cysts are congenital, non neoplastic ectodermal cysts with different ectodermal derivatives. They are similar to teratoma in some aspects, but difference is that, teratomas are true neoplasms and have contents derived from all the three embryonic germ cell layers.[1,2] Intracranial dermoid cysts constitute less than 1% of all intracranial masses.[3] In brain, dermoid cysts most commonly occur in midline suprasellar, posterior fossa. They also occur in spine. Dermoid cysts are non neoplastic and their malignant transformation into squamous cell carcinoma is very rare.[2] Dermoid cysts are more common in first three decades of life and are more common in females.[3,4] There is usually history of symptoms like headache, seizure. These lesions usually enlarge over time due to glandular secretions, epithelial desquamation. Due to increased internal pressure they rupture spontaneously and sometime they rupture following trauma. Their rupture and dissemination of internal contents lead to chemical meningitis which cause vasospam leading to infarction and even death.[5]

Case
A 14 year girl came to hospital with history of repeated episodes of the seizures, headache for the past 4 days. She had complaint of decrease in vision also. On examination, she had decreased visual acuity of 6/18 and bilateral papilledema. Cranial nerve examination was normal. Rest of systemic examination was normal. She was referred for CECT head for further evaluation. CT scan showed 4.3x3.4x4.1 cm well circumscribed, fat attenuation, midline anterior cranial fossa lesion in the supra sellar region with peripheral coarse, discontinuous calcifications. This lesion was causing mass effect on b/l lateral ventricle, leading to effacement of the anterior horn and dilatation of body, occipital horn, temporal horn of the b/l lateral ventricle. There was presence of fat attenuation content in the b/l lateral ventricles, anterior inter hemispheric fissure and in the left sylvian fissure along the left middle cerebral artery. On post contrast images, no abnormal intra lesional enhancement seen. No abnormal meningeal enhancement seen.



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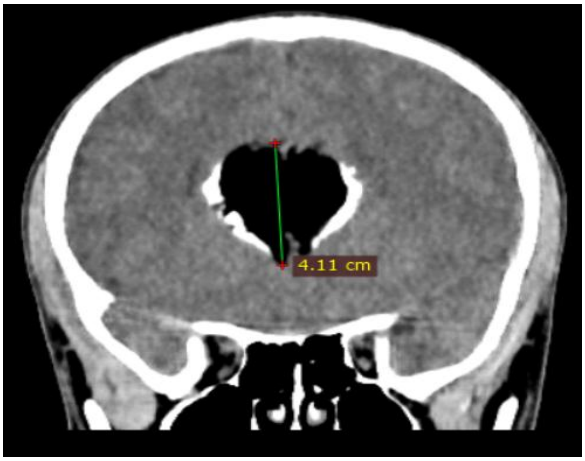

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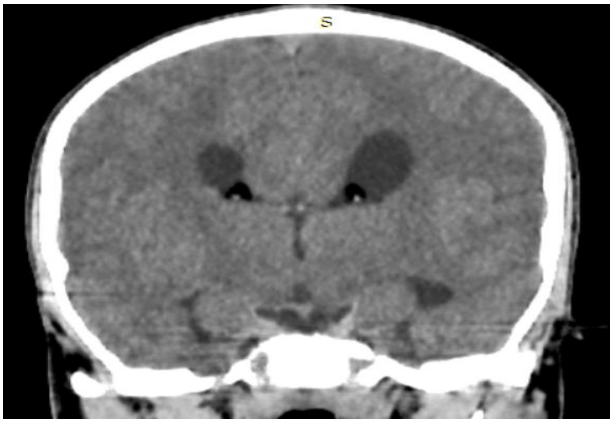
Figure 1: NCCT Brain axial (a), Coronal (b) Image showing well circumscribed, fat attenuation , midline anterior cranial fossa lesion in the supra sellar region with peripheral coarse, discontinuous calcifications. This lesion is causing mass effect on b/l lateral ventricle, leading to effacement of the anterior horn and dilatation of body, occipital horn of the b/l lateral ventricle.



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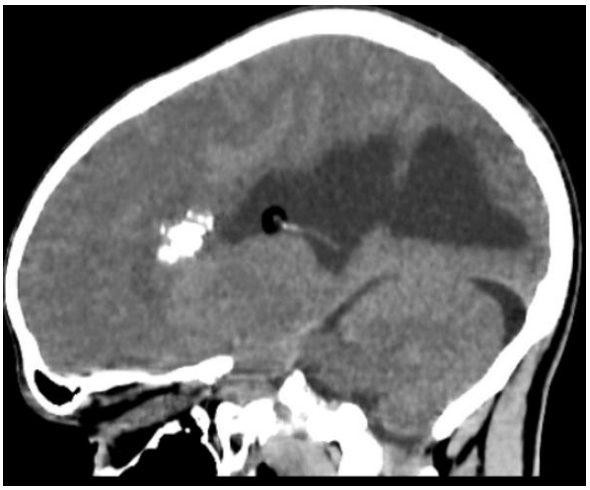

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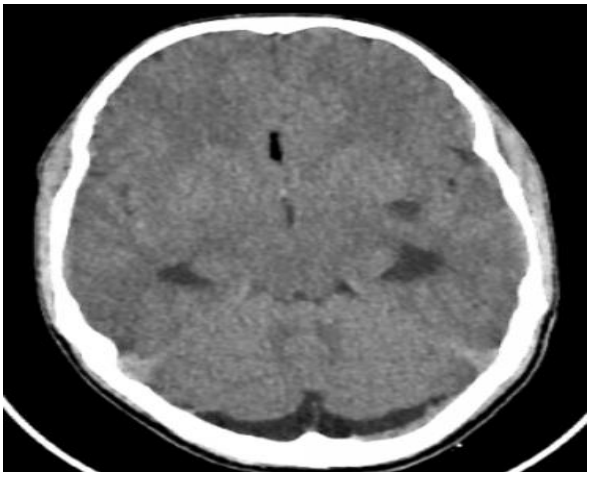
Figure 2: NCCT Brain coronal (a), Sagittal (b) Images showing presence of fat attenuation content in the b/l lateral ventricles.



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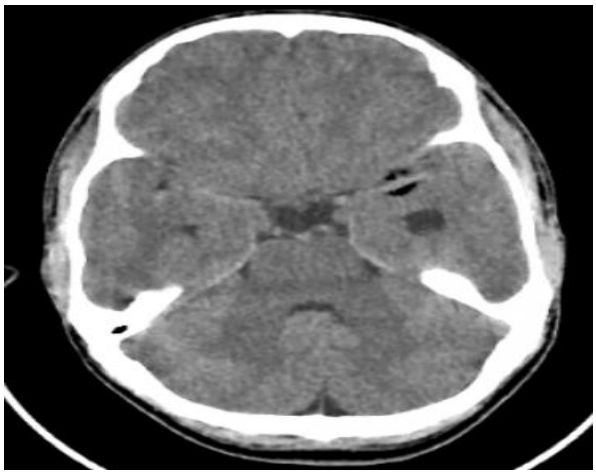

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Figure 3: NCCT Brain axial images showing presence of fat attenuation content in anterior inter hemispheric fissure (a) and in the left sylvian fissure along the left middle cerebral artery (b).

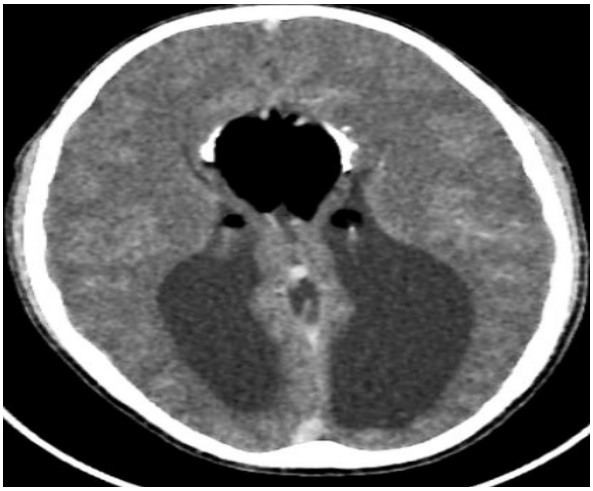

Figure 4: CECT Brain axial image shows no evidence of meningeal enhancememt.

DISCUSSION

Intracranial dermoid cyst is a congenital non neoplastic lesion that occurs due to incorporation of the ectodermal

components at the time of neural tube closure and due to this dermoid cysts have predisposition for midline location. According to Lunardi et al. intracranial dermoid cyst arise from ectodermal nest cells which become adherant to primitive veins during the process of embryogenesis.[6] They can be described as squamous epithelial cyst containing dermal elements including hair follicles, sebaceous and sweat glands. When the embryonic ectodermal tissue get entrapped in the neural tube between 5th and 6th week of fetal life, these lesions occur in midline and if they get entrapped during secondary cerebral vesicle formation, they usually occur at site other than midline.[27] Most common sites include sellar, suprasellar, frontonasal regions. They can also occur in posterior cranial fossa, but they are rare. Sporadically, they can be associated with Goldenhar (oculoauriculo-vertebral dysplasia) and Klippel-Feil syndromes. They show progressive growth because of sebum, sweat secretion leading to mass effect on adjacent organs, compression of neurovascular structures and symptoms like headache, seizures. Due to progressive enlargement they rupture leading to chemical meningitis.

Radiological investigations play an important role in diagnosis and monitoring of these lesions. On computed tomography (CT), they appear as well defined non enhancing hypodense lesions with average CT attenuation value ranging from 0 to -100 HU. They can have variable proportion of fat, hair and epidermal debris. Calcification are seen in 20 % cases.[8]

In MRI (magnetic resonance imaging) dermoid cysts show hyperintense signal on T1, T2, and FLAIR sequences. There is presence of the fat signal intensity in the subarachnoid space or the ventricles in case of ruptured dermoid cysts. Leptomeningeal enhancement can be seen in ruptured dermoid cyst which is suggestive of chemical meningitis. Dermoid cysts do not demonstrate restricted diffusion on DWI whereas epidermoid show diffusion restriction.

CONCLUSION

Incidence of rupture of intracranial dermoid cyst is very less and there is minimal literature on it, nevertheless this can be a very grave condition. Apart from headache and seizures, rupture of intracranial dermoid can cause hydrocephalus, chemical meningitis and even death .Computed tomography and magnetic resonance imaging helps in diagnosis of these lesions, especially in cases of their rupture, so that early appropriate intervention can be done.

REFERENCES

1. Muçaj S, Ugurel M, Dedushi K, et al. Role of MRI in Diagnosis of Ruptured Intracranial Dermoid Cyst, Case Report. Acta Inform Med, 2017; 25: 141-144.
2. A. G. Osborn and M. T. Preece. Intracranial cysts: radiologic pathologic correlation and imaging approach, Radiology, 2006; 239(3): 650–664.
3. Mehta MP, Chang SM, Guha A, et al. Principles and Practice of Neuro-Oncology: A Multidisciplinary Approach. Demos Medical, 2010.
4. Runge VM, Smoker WRK, Valavanis A. Neuroradiology: The Essentials with MR and CT. Thieme, Berlin, 2014.
5. Kim KS, Weinberg PE. Dermoid tumor. Surg Neurol, 1981; 15: 375–376.
6. P. Lunardi, P. Missori, A. Rizzo, and F. M. Gagliardi. Chemical meningitis in ruptured intracranial dermoid. Case report and review of the literature, Surgical Neurology, 1989; 32(6): 449–452.
7. J. G. Smirniotopoulos and M. V. Chiechi. Teratomas, dermoids, and epidermoids of the head and neck, Radiographics, 1995; 15(6): 1437–1455.
8. Erasmus LJ, Hurter D, Naude M, et al. A short overview of MRI artifacts. SA J Radiol, 2004; 8: 13-17.