A 15 year old boy was admitted with history of alleged fall with head hitting the ground while playing. The friends gave a history of transient loss of consciousness immediately after the fall from which he recovered completely within minutes. There was no history of tongue biting, froth coming from the mouth, convulsive movements or incontinence of urine. There was no past history of epilepsy.

On examination the patient was conscious, oriented, pupils were equal and reacting to light, moving all 4 limbs and no focal neurological deficit. Plantars were down going. Fundi were normal. There was evidence of soft tissue contusion in the occipital region. BP 110/80, PR 78/mt. No abnormality was detected in the cardiovascular, respiratory and gastrointestinal system.

Computerized Tomography of head showed a hypodense lesion -83 to -108 Hounsefield unit (7.8 x 1.0 x 1.3cm in the Antero posterior x Cranio caudal x transverse) with focal anterior calcification seen along the corpus callosum. No other focal neuro parenchymal lesion seen. C.T. findings were suggestive of lipoma of corpus callosum. Since it was an incidental asymptomatic lesion patient was left alone and advised to come for periodic follow up.

Discussion

Lipomas can occur in any part of the body but intracranial lipomas are extremely rare and are developmental in origin. Abnormal resorption of primitive meninges is said to be responsible for the development of these lipomas. During embryological development, resorption of primitive meninges usually takes place between 8th and 10th week of intrauterine life. If the primitive meninges persist longer, it may eventually differentiate into fatty tissue. This is considered to be the pathogenesis of intracranial lipomas.

Intracranial lipomas have been described in the ambient cistern, quadrigeminal plate, chiasmatic cistern, cerebellomedullary cistern, tela-choroidea of lateral and third ventricles, choroid plexus, velum interpositum and callosal cistern (Kazner.et.al. 1980)

Intracranial lipomas frequently occur in the region of corpus callosum (40-50%) and are associated with varying degrees of dysgenesis of corpus callosum. Lipoma of corpus callosum was first described by Rokitansky(2) in a case of necropsy on 1856.

Review of literature revealed that 50% of the corpus callosum lipomas were asymptomatic and were diagnosed during routine neuro-radiological investigations. The present case is an incidental C.T. finding while investigating a case of head trauma. In symptomatic cases 50% presented with fits, 20% with mental retardation, and 13% with hemi paresis. We have reported a case of lipoma of corpus callosum with review of literature in Neurology India 1988 which was excised totally(3).
In pre C.T. era, intracranial lipomas were rarely encountered. Peripheral calcification of lipoma in plain x-rays is reported to be diagnostic. With the advent of C.T. and M.R.I scanning more and more cases of lipomas have been reported.

C.T. appearance of lipoma of corpus callosum was first described by New and Scott (1975) (4) followed by Wallace (1976), Fuerber and Wolpert (1978). A homogenous low density area with attenuation values between -60 and -200 Hounsefield unit, no contrast enhancement, regular margins and often peripheral calcification are the main characteristics of the lesion on the C.T. scan. Dermoids and teratomas should be considered in the differential diagnosis of low density lesions but they have a non homogenous low density which could be detected at a low window level.

Corpus callosum lipomas are very slow growing lesions. Only very rarely they attain a large size sufficient to cause mass effect. Hence they rarely need surgery. Radical excision of the corpus callosum lipoma is almost impossible because of the firm adhesion to the surrounding brain tissue and high vascularity of the lesion. More over the lesion may encompass the anterior cerebral arteries or their branches. Hence during radical removal we may have to sacrifice both anterior cerebral arteries which may lead to compromise of anterior circulation with attendant complications.

References
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Fig. 3. Coronal section of CT brain shows midline hypodense lesion with attenuation values of -83 to -108 characteristic of lipoma.

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Consultant Pulmonologist.

Advanced Pulmonary Function Laboratory, Whole Body Plethysmography, Gas Diffusion Study, Sleep Laboratory, Asthma Clinic, Allergy Clinic, COPD Clinic, Quit Smoking Clinic

Consultation by prior appointment: Tel: 9349523331, 0484 2323331, E mail: chestclin@sancharnet.in