INCIDENTALLY FOUND LARGE PERICALLOSAL LIPOMA FOLLOWING TRAUMATIC HEAD INJURY: A CASE REPORT AND REVIEW OF THE LITERATURE

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Introduction

Lipomas are the most common soft tissue neoplasms of adults, typically subcutaneous in location especially in trunk and proximal extremities. Apart from this typical superficial location, deeper lipomas located in the intramuscular, intraosseous, intrathoracic, retroperitoneal or intracranial regions can also be encountered. Intracranial lipoma (ICL) which was firstly described by von Rokitansky[1] in 1856, constitutes about 0.1- 0.5% of all intracranial lesions.[2,3] They are not true neoplasms but a congenital malformations resulted from persistence and abnormal differentiation of meninx primitiva to mature adipose tissue instead of being completelly resorbed during the development of subarachnoid cisterns between the 8th and 10th weeks of gestation.[4,5] Due to their meningeal origin, ICLs are located at subarachnoid spaces usually at midline. The most frequent location is the pericallosal cistern and these ICLs are called as pericallosal lipoma (PCL). Neurovascular structures in the subarachnoid space may traverse the lesion.[6] Anterior cerebral vessels can be seen inside or around the PCL. ICLs are frequently associated with various CNS malformations. The most frequent is dysgenesis of the corpus callosum (CC).[7] In addition, vascular abnormalities including aneurysms, arteriovenous malformations and abnormal branches may also occur in association with ICL.[8,9] In this report, we present a case of incidentally found PCL extending into the right lateral ventricle in a patient with traumatic head injury.

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Case Report

A 37 year old female patient presented to our emergency department with trauma to the occipital region after a traffic accident. There was a history of trauma to the occipital region and unconsciousness that occured at the time of accident but recovered shortly after it. In the general clinical examination and neurological examination performed in our emergncy department, the patient was conscious and cooperative and no neurological deficit was detected. She had computed tomography (CT) scan of head and there was no finding associated with trauma but there was an incidentally found hypodens lesion in the interhemispheric fissure with its large component extending into the right lateral ventricle. The size of the lesion was approximatelly 32x16x12 mm. The attenuation value of the lesion was average-94 hounsfield units (HU) and peripheral nodular calcifications were present. There was no ventricular dilatation. Because of the typical location and diagnostic homogenous fat attenuation of the lesion on CT image, the lesion was diagnosed as anterior tubulonodular type PCL and she referred to MRI to look for concomitant abnormalities especially for dysgenesis of the CC.

Discussion

ICLs are rare congenital malformations which are mostly asymptomatic and often found incidentally in

neuroimaging studies. However, depending on their size, location and concomitant malformations,they may present with various symptoms including headache, epileptic seizure, local mass effects, psychomotor retardation and cranial nerve deficits,[10,11,12] Although the most reported presentation of the patients with ICL is headache, it is also reported that this is mostly not atributable to the ICL.[10,13] Presentation with seizure is frequent, and is reported as sometimes unresponsive to antiepileptics.[8,14,15] Carlos et al.[11] reported an undiagnosed massive lipoma associated with sudden death after convulsion as an autopsy finding. Behavioral abnormalities can be present dueto accompanying CC dysgenesis. Yaxiong Li et al[12] reported a case of PCL with severe dysgenesis of the CC presenting with psychotic manifestations that recovered after microsurgical management.

Based on location and morphological appearance, PCLs are classified as tubulonodular type and curvilineer type. The former which is the more common variant, is located anteriorly and rounded or tubular in shape having more than 1 cm in thickness and is frequently associated with dysgenesis of the CC. Rarely, the anterior tubulonodular PCLs can extend into the lateral ventricles as in our case.[16] The curvilineer type located posteriorly at the margins of the CC, is elongated in shape, having less than 1 cm in thickness and is generally not together with other cerebral anomalies. In some cases this classification may not be made and a mixed type may be encountered. Paraget al.[17] reported a case of PCL which occupy both anterior and posterior locations. Dysgenesis of the CC in varying degree is frequently reported in association with PCL. Yilmaz MB. et al.[10] reported 40% rate of CC hypoplasia in their 10 patients, Gomez Gosalvez et al. reported 30% rate of CC anomalies in their 20 patients[17] andSeidl et al.[13] reported an 11.8% rate of CC disgenesis in their 17 patients.[13] The incidence of dysgenesis of the CC is known to be more frequent in the anterior tubulonodular type.[2,4,5,6,7,19] In the study of Tart et al.[19] CC dysgenesis was present with most of the cases (90%) with anterior PCL but in some cases (30%) with posterior PCL. However, in the study of Yilmaz MB et al.[6] 3 of 4 patients having coexisting hypoplisa of CC had a curvilinear PCL while the remaining one patient had tubulonodular PCL. The tubulonodular varient may also be associated with frontofacial defects and in a minority of cases a connection with extracranial subcutaneous lipomas may be seen. There are reported cases of PCLs associated with subcutaneous lipoma with a direct continuity through a cranium bifidium by a fibrolipomatosis stalk or without any connection.[20,21,22,23] Diagnosis of ICLs is made by radiological imaging, Their CT and MR imaging is characteristic and pathognomonic. On noncontrast CT scan, they appear as smoothly circumscribed homogenously hypodens lesions with charactheristic attenuation value of addipose tissue ranging from -80 to -110 HU.[24] They do not show contrast enhancement. Peripherally located lineer or nodular calcifications are commonly seen in tubulonodular PCL.[25] Since there are reported cases of associated vascular malformations CT angiograhy may also be indicated in some cases. On magnetic resonance (MR) imaging, ICLs follow usual fat signal intensity and appears as markedly hyperintense on T1w MR images and show signal loss following fat saturation. These lesions have high signal intensity also on T2w MR images, but lower than cerebrospinal fluid. They are homogenous lesions but there may be low signal ares at the periphral regions due to the calcifications. If there are pericallosal arteries coursing through the lesion, central flow voids can be seen. MRI is especially valuable in the evaluation of concomitant CC dysgenesis. In rare atypical cases biopsy may be rarely necessary in order to make diagnosis, for example mixed intensity lesions with perifocal edema.[26]

All fat containing midlinelesions including fatty falx cerebri, and dermoid cysts are among the differential diagnosis. Fatty falx cerebri is the intracranial fat located between the two visceral layers of the falx cerebri and may be considered in the differential diagnosis of curvilineer PCL. Dermoid cysts have frequently heterogenous apperance different from the very homogenous nature of ICL. Although differs with its clincal presentation, subacute stage hematoma may be considered among the differantial diagnosis in terms of imaging, with T1w shortening effect of intracellular methemoglobin resulting in increased signal intensity on T1w MR image. Fat suppression resolves this confusion.In addition, ICL may also be mistaken for pnemocephalusin patients with head trauma, which appears as completelly black lesion

like ICL on routine brain windows, but the attenuation value is much more lower (about -1000 HU) than that of fat attenuation.

Since ICL are benign and mostly asymptomatic lesions they are managed conservativelly. Radical surgical resection is not indicated and should be avoided in asymptomtic cases due to incorporated neurovasculer structures and adhesions to the neighbouring tissues.[21] However, surgical management should be considered in some cases presenting with for example uncontrolled seizures, signs of increased intracranial pressure, hydrocephalus with progressive dementia.

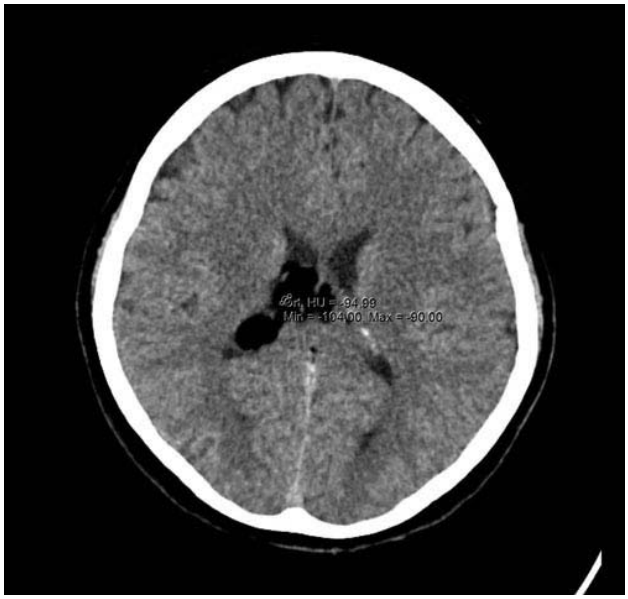

Figure 1: Axial CT scan of head shows a hypodens pericallosal lesion extending into right lateral venticle with ateenuation value of average -94 HU.

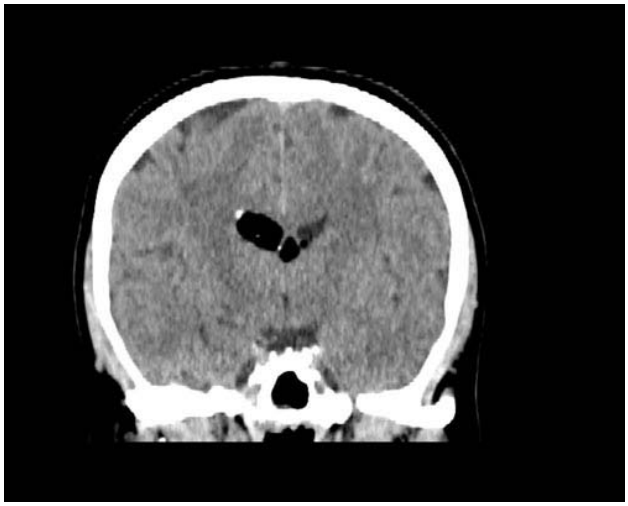

Figure 2: Coronal CT scan of head shows a hypodens pericallosal lesionextending with nodular periferal calcification.

Conclusion

PCL s are frequently asymptomatic benign lesions.. Because of the close relationship of PCLs with important neighboring structures and the fact that anterior neurovascular structures in the subarachnoid distance can pass through the lesion, their surgical treatment is very difficult and dangerous. Therefore, in symptomatic cases, conservative approaches are preferred. However, in certain cases with atypical imaging findings that may mimic malign lesions surgery may be inevitable for histological diagnosis. Careful radiological imaging is important in order to prevent unnecessary interventions and to diagnose rare atypical PCLs which require surgery. In addition, searching for associated anomalies and vascular malformations like aneurysms is very important.

Conlict of Interest: Declared none by authors.

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