

CASE REPORT

A Rare Case of Pericallosal Lipoma Associated with Bilaterally Symmetrical Lateral Ventricular Choroid Plexus Lipomas without Corpus Callosal Anomalies

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ABSTRACT

Lipomas constitute less than 5% of primary brain tumors. Pericallosal lipomas (PCLp) constitute almost half of all intracranial lipomas. Corpus callosal anomalies commonly occur in cases with PCLps. Although PCLp is often described as corpus callosal lipoma, it is most often pericallosal in location. PCLps may have calcification in the periphery and may continue into lateral ventricles, which is a very rare presentation. We observed a case of PCLp with peripheral calcifications associated with PCLp continuing as bilaterally symmetrical lateral ventricular choroid plexus lipomas (CPLp) without any corpus callosal or other central nervous system anomalies, and as this is not been previously reported, we are presenting it. The appearance of PCLp in this case does not correspond to the descriptions of any of the existing morphological types (anterior and posterior) of classification of PCLps; it is rather mixed, where PCLp occupies both anterior and posterior locations around the corpus callosum.

Key words: Corpus callosum, choroid plexus, lipoma, magnetic resonance, pericallosal lipoma

INTRODUCTION

Lipomas constitute less than 5% of primary brain tumors.^[1] PCLps constitute almost half of all intracranial lipomas and corpus callosal anomalies are commonly present in

cases with PCLps.^[2] PCLps may have calcification in the periphery and may continue into the lateral ventricles, which is a very rare presentation.^[3,4] Although, PCLp is often described as corpus callosal lipoma, it is most often pericallosal in location. Intracranial lipomas result from anomalous differentiation of persistent primitive meninges.^[3]

CASE REPORT

A 58-year-old Arab man presented to our hospital with a 12-month history of recurrent headache. Clinical and neurological examinations were unremarkable. Radiological

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evaluation included computed tomography (CT) and Magnetic resonance imaging (MRI) scanning.

The CT and MRI images revealed a PCLp cephalad to the entire antero-posterior extent of the corpus callosum; continuing as bilaterally symmetrical lateral ventricular CPLps [Figures 1-4]. The PCLp continued through the choroidal fissure as bilaterally symmetrical lateral ventricular CPLps [Figures 2 and 4]. Peripheral calcifications were noted within the PCLp [Figure 2], and the corpus callosum was unremarkable [Figure 3]. The fat appeared more hypodense than CSF on CT scans [Figure 1]. PCLp measured approximately 11.2 × 2.1 cm. CPLps measured approximately 1.3 cm in maximum diameter. Discrete

areas of fatty tissue were noted in the subarachnoid space along the anterior interhemispheric fissure [Figure 3] and also in the medial sulcus of the right frontal lobe [Figure 5]. Incidentally, unusually thick dural calcifications and cavum septum pellucidum were also noted with bilateral supraventricular white matter ischemic lesions. No other central nervous system abnormalities were noted. Sagittal unenhanced T1 weighted MRI scans of a normal brain with superimposed black shading (black arrows) show morphological features that classify PCLps into anterior [Figure 6a] and posterior [Figure 6b] groups and is compared to features seen in our case [Figure 6c].

DISCUSSION

PCLps are classified into anterior [Figure 6a] and posterior [Figure 6b] groups indicating their location in relation to the corpus callosum. Anterior ones are tubulonodular in shape, larger than 10 mm and are commonly associated with various intracranial malformations including those affecting the corpus callosum. Posterior ones are

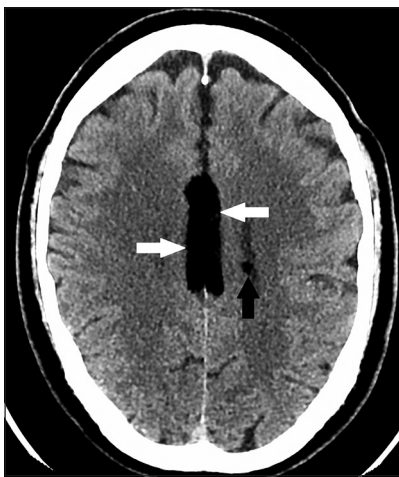


Figure 1: 58-year-old man with a 12-month history of recurrent headaches diagnosed with PCLp associated with bilaterally symmetrical lateral ventricular CPLps without corpus callosal anomalies. Axial unenhanced CT scan of the head shows midline PCLp (white arrows) and left lateral ventricular CPLp (black arrow). The fat appears more hypodense than CSF.

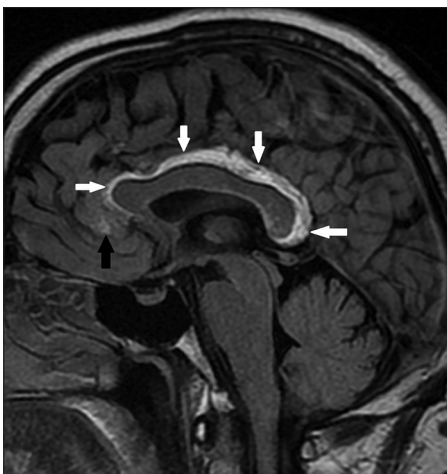


Figure 3: 58-year-old man with a 12-month history of recurrent headaches diagnosed with PCLp associated with bilaterally symmetrical lateral ventricular CPLps without corpus callosal anomalies. Sagittal unenhanced FLAIR MRI scan shows PCLp (white arrows) and discrete areas of fatty tissue around the anterior interhemispheric fissure (black arrow). Also, evident is normal corpus callosum.

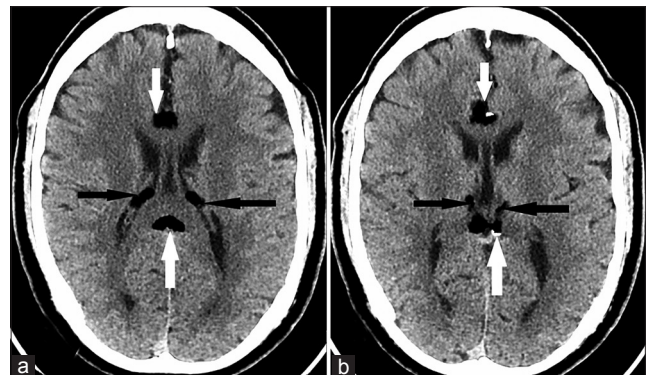


Figure 2: 58-year-old man with a 12-month history of recurrent headaches diagnosed with PCLp associated with bilaterally symmetrical lateral ventricular CPLps without corpus callosal anomalies. (a and b) Axial unenhanced CT scans of the head show PCLp with peripheral calcifications (white arrows) and PCLp continuing as bilaterally symmetrical CPLps (black arrows).

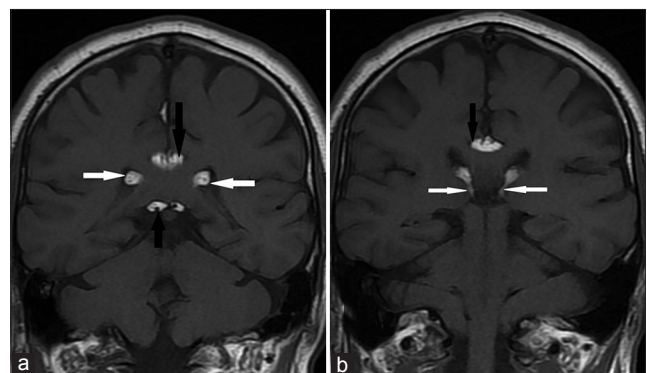


Figure 4: 58-year-old man with a 12-month history of recurrent headaches diagnosed with PCLp associated with bilaterally symmetrical lateral ventricular CPLps without corpus callosal anomalies. (a and b) Coronal unenhanced T1 weighted MRI scans show PCLp (black arrows) continuing as bilaterally symmetrical lateral ventricular CPLps (white arrows).

elongated, thin, and smaller than 10 mm and are mostly located around the splenium. The incidence of the corpus callosal and other intracranial anomalies is significantly less in posterior lipomas.^[2-9] In our case, the PCLp occupies anterior as well as posterior locations and its posterior part is thicker than the anterior part [Figure 6c]. To the best of our knowledge, this appearance has not previously been described.

In 20 to 50% cases, PCLp may occur with CPLp.^[4] PCLps may occur with hypogenesis/agenesis of the corpus callosum in most of the cases (90%) of anterior PCLps and some of the cases (30%) of posterior PCLps.^[7]

Dysgenesis or persistence of the primitive meningeal tissue leads to the formation of the lipomas in the cranium.^[8,9] This process occurs during the first trimester of pregnancy, most likely between the eighth and the tenth weeks of intrauterine pregnancy.^[9] The primitive meningeal tissue

is present along the inner parts of the pia, the arachnoid and the dura mater and is considered as a mesenchymal tissue originating from neural crest.^[9] It occupies the future cisterns in the developing brain of the embryo, and its subsequent resorption leads to the creation of the subarachnoid spaces.^[8] Failure of this resorption and maldifferentiation of the primitive meningeal tissue leads to the development of the mesenchymal tissue including mature adipose tissue, calcifications, and mature bone tissue replacing its space.^[8,9] This mesenchyme may be located at various places in the cranium, which explains the presence of mature adipose tissue or lipomas and calcifications in multiple subarachnoid cisterns.^[8,9] The lipomas in the cranium can thus be considered as malformations and not true neoplasms.^[8,9] The normal structures in the subarachnoid space including nerves and vessels may traverse through these lipomas.^[8] The process of resorption of the primitive meningeal tissue and formation of the subarachnoid cisterns occurs in an orderly fashion, and preferential cisternal sites for development of lipomas in the cranium were described based on it.^[6,9] Mature bone tissue is considered to be an osseous metaplasia occurring after the formation of calcifications, and occurrence of osseous tissue and lipomas in the cranium associated with corpus callosal agenesis is considered to be originating due to disturbances in development of the neural crest.^[9]

The association of corpus callosal agenesis with PCLp is common, because, maldevelopment of primitive meninx occurs before the interhemispheric commissural fissures develop.^[8]

During the embryonic stage of the choroid plexus in lateral ventricles, there is an insinuation of a part of the interhemispheric cistern and the tela choroidea of the ependymal roof plate through the choroidal fissure with infolding of the primitive meninx.^[4,8] The developing choroid plexus is attached to the primitive meninx and thus the

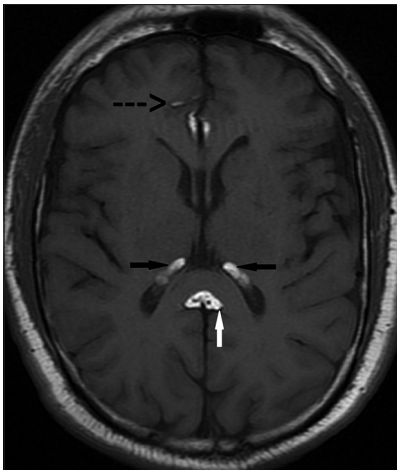


Figure 5: 58-year-old man with a 12-month history of recurrent headaches diagnosed with PCLp associated with bilaterally symmetrical lateral ventricular CPLps without corpus callosal anomalies. Axial unenhanced T1 weighted MRI scan shows PCLp (white arrow) and bilaterally symmetrical lateral ventricular CPLps (black arrows). Also note discrete areas of fatty tissue in the medial sulcus of the right frontal lobe (dashed arrow).



Figure 6: 58-year-old gentleman complaining of recurrent headaches since twelve months diagnosed with PCLp associated with bilaterally symmetrical lateral ventricular CPLps without corpus callosal anomalies. (a, b and c) Sagittal unenhanced T1 weighted MRI scans of a normal brain with superimposed black shading (black arrows) show morphological classification of PCLps (a: Anterior and b: Posterior) and c: Appearance as in our case.

primitive meninx enters into the ventricle.^[4,8] This explains the occurrence of PCLp with CPLp in our case. PCLp would use the same course to get into the lateral ventricles, even in cases with corpus callosal agenesis.^[8] This explains the occurrence of PCLp with CPLp in cases with corpus callosal agenesis.

An isolated PCLp mostly does not cause symptoms. Associated clinical problems are always due to associated central nervous system abnormalities.^[2,9] This is true in our case.

Almost half of the cases of PCLp are detected incidentally. These cases may present with various central nervous system related symptoms like headache, epileptic fits, limb weakness, and psychological or memory related problems. In our case also, the PCLp was detected incidentally during an investigation of recurring headache. It was detected late at the age of 58 years because he had no significant neurological disabilities, possibly due to the absence of any central nervous system anomalies other than the PCLp and CPLps.

Our patient has an intracranial lipoma displaying fat density and peripheral calcifications on CT scans. It lies cephalad to the entire antero-posterior extent of the corpus callosum (PCLp). The PCLp also extends into both lateral ventricles through the narrow neck of the choroid fissure to appear as CPLps.

Management of PCLp is mostly symptomatic, and prognosis depends on the presence of additional central nervous system abnormalities. Patients with PCLp may develop seizures and hydrocephalus. Surgery is not indicated in isolated pericallosal lipomas; however, it is essential to exclude atypical intracranial lipomas which require surgery.^[2,9]

CONCLUSION

The appearance of PCLp in our case does not correspond to the descriptions of any of the existing morphological types (anterior and posterior) of classification of PCLps;

it is rather a mixed variant, where PCLp occupies both anterior and posterior locations around the corpus callosum. To our knowledge, this appearance has not been described in the published literature before. This rare presentation of intracranial lipomas may provide new insights to anatomists or embryologists to confirm or refute various theories related to the origin of intracranial lipomas. Confirmed diagnosis of an isolated and typical PCLp helps prevent any further costly investigations and interventions. Also, it is necessary to diagnose rare and atypical PCLps, which require surgical intervention. It is, therefore, essential for physicians and radiologists to know about this entity and its various atypical presentations.

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