DISCUSSION

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PERICALLOSAL LIPOMAS
Rare congenital malformative lesions occurring in the interhemispheric fissure closely related to the corpus callosum which is usually abnormal.

Constitute almost half of all intracranial lipomas and are commonly associated with corpus callosal anomalies.
PATHOGENESIS

• Result of an abnormal resorption of the primitive meninges (meninx primitiva).

• Usually, this resorption occurs between the eighth and the 10th week of development to create sub-arachnoid spaces.

• When the primitive meninges persists longer, instead of being resorbed, it differentiates into mature lipomatous tissue.
Such lipoma may develop in all the cerebral cisternae, but they are much more frequent in the area of the corpus callosum where it interferes with its normal growth between the 11th and 20th weeks.

The lipomas of brain are mal-formations rather than a true neoplasm.
• Therefore, anomalies of the development of the corpus callosum (complete or partial agenesis, hypoplasia) almost always coexist.

• The degree of anomaly seems to be in relation with the size and location of the lipoma.

• The normal sub-arachnoid nerves and vessels may also become traversed through these lipomas.
CORPUS CALLOSAL
DYSGENESIS SPECTRUM
TERMINOLOGY

- **Agenesis** = Complete absence of corpus callosum (CC)
- Hippocampal commissure (HC) absent
- Anterior commissure (AC) often present

- **Tricommissural agenesis** = All 3 absent

- **Hypogenetic, dysgenetic CC**
- Rostrum, splenium often absent in partial agenesis
- Partial posterior agenesis = splenium, ± posterior body.
Etiology and Pathology

- Embryonic guiding mechanisms fail
  - Axons may fail to form
  - Molecular guidance fails
  - Glial sling fail to develop normally
  - Failure to guide axons across midline

- Multiple genes implicated - genetic aberration
IMAGING
Racing car sign of corpus callosum agenesis
Associated Anomalies

Malformations

✓ Chiari 2
✓ Dandy-Walker
✓ Frontonasal dysplasia, clefts
✓ Cerebellar hypoplasia/dysplasia
✓ Hypothalamic-pituitary axis malformations
✓ Malformations of cortical development
Syndromes

Nearly 200 inherited syndromes

- Aicardi syndrome
- Apert syndrome
- CRASH syndrome
- 22q11.2 deletion syndrome (DiGeorge)
- Morning glory syndrome
DYSGENESIS WITH PCL
As mal-development of primitive meninx occurs before the development of the inter-hemispheric commissural fissures, there is a frequent association of agenesis of corpus callosum with lipoma of CC.
Extension into ventricles?

The development of lateral ventricular choroid plexus includes invagination of a portion of the inter-hemispheric cistern and tela choroidea of the ependymal roof plate through the choroidal fissure with infolding of the primitive meninx.
• The developing choroid plexus is attached to it and thus enters into the ventricle as well.

• In the absence of corpus callosum, pericallosal lipoma would use the same path to get into the lateral ventricles.
TYPES

• 1 - consists of anteriorly situated round or cylinder-shaped lipomas, termed **tubulonodular** - have a high incidence of corpus callosum dysgenesis and fronto-facial anomalies and can extend into the choroid plexus/lateral ventricles.

• 2 - comprises thin, elongated lipomas measuring less than 1cm in diameter, posteriorly situated curving around the splenium and are termed **curvilinear** - have a low incidence of associated anomalies.
• Dystrophic calcification occurs in both types but is more common in bulky tubulonodular lesions
CLINICAL ISSUES
EPIDEMIOLOGY AND DEMOGRAPHICS

- CC dysgenesis is the most common CNS malformation and is found in 3-5% of individuals with neurodevelopmental disorders.
- It has a prevalence of at least 1:4,000 live births.
- Non syndromic CC dysgenesis is found in patients of all ages.
PRESENTATION

• Minor CC dysgenesis/hypogenesis is often discovered incidentally on imaging studies or at autopsy.

• Major commissural malformations are associated with seizures, developmental delay, and symptoms secondary to disruptions of the hypothalamic-pituitary axis.
GROWTH

• Because of their malformative origin, pericallosal lipoma usually do not become hyperplasic, whereas it might occur when the patient gains weight, as well as other sites of fat storage.
TREATMENT

No specific treatment for pericallosal lipoma is usually required, although seizures if present need medical management and the prognosis depends on the presence of additional central nervous system abnormalities.
Surgical therapy is usually not indicated because the risks of surgical intervention outweigh the potential benefits in most cases and may result in high morbidity and mortality rates, given the incorporated neurovasculaarity and strong adhesion to surrounding tissue.
REFERENCES

INTERNATIONAL JOURNAL OF RADIOLOGY

Pericallosal Lipoma and Agenesis of Corpus Callosum with Associated Lipoma of Choroid Plexus: A Rare Case Report

Ovais A. Hassan

THANK YOU
• Current thinking suggests that lipomas represent a.

• Recent fluorescence in situ hybridization (FISH) and comparative genomic hybridization (CGH) studies have identified clonal cytogenetic aberrations in nearly 60% of ordinary lipomas. The 12q13-15 region is the most commonly involved site. Between 15-20% of lipomas show rearrangements or deletions of the long arm of chromosome 13, particularly 13q12-22.