Review Article

Anomalies of the Corpus Callosum in Prenatal Ultrasound: A Narrative Review for Diagnosis and Further Counseling

CME Credits

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Abstract

The corpus callosum is the major interhemispheric tract that plays an important role in neurological function. Understanding the etiology and embryology development helps the ultrasound diagnosis for disorders of the corpus callosum and further counseling. The nonvisualization of cavum septum pellucidum or dysmorphic cavum septum pellucidum in axial view are indirect signs for beginners to diagnose complete agenesis of corpus callosum (cACC) and partial agenesis of the corpus callosum (pACC). Further coronal view, sagittal view, and fetal magnetic resonance imaging are also important for evaluation. Genetic testing plays an essential tool in anomalies of corpus callosum by revealing the underlying genetic pathophysiology, such as chromosomal anomalies and numerous monogenetic disorders in 30%–45% of ACC. Diagnosis and prediction of prognosis for hypoplasia or hyperplasia of the corpus callosum are more difficult compared to cACC and pACC because of the limited reports in the literature. However, the complex types often had poorer prognostic outcomes compared to the isolated types. Hence, it is important to evaluate and follow fetal conditions thoroughly to rule out intracranial or extracranial anomalies in other systems.

Keywords: Agenesis of the corpus callosum, genetic counseling, hyperplasia of the corpus callosum, hypoplasia of corpus callosum, prenatal ultrasound

Introduction

The corpus callosum is the white matter interhemispheric tract between the two cerebral hemispheres. It can facilitate the integration of motor and sensory information which contribute to human cognitive function.[1] The corpus callosum usually develops from the 10th to 20th weeks of gestation.[2] However, the development of the corpus callosum may be interrupted by many genetic factors and maternal alcohol abuse during the prenatal period.[3] Anomalies of the corpus callosum are one of the common central nervous system structural anomalies detected among prenatal ultrasounds.[4] The prevalence of agenesis of the corpus callosum was about one in 4000–5000 individuals in the past.[5] A study in Hungary calculated the overall prevalence of agenesis and hypoplasia of corpus callosum and revealed 2.05/10,000 live births.[6] The anomalies should not be limited to total absence or partial absence but also included abnormal shape, abnormal echogenicity, and abnormal thickness.[7] Thus, the categories are complete agenesis, partial agenesis, dysgenesis, hypoplasia of the corpus callosum, and hyperplasia of the corpus callosum. Because of the diverse etiology and categories, the sequelae are heterogenous from good to very poor neurological outcomes and make the counseling even more challenging during the prenatal period. This study aimed to focus on the prenatal diagnosis of fetal corpus callosum anomalies in 2D ultrasonography and provide further updated information about prognosis for counseling.

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Development of the Corpus Callosum

In the prenatal period, the corpus callosum could be divided into four defined regions: the rostrum, the genu, the body, and the splenium.[8] The adult corpus callosum included the following regions: rostrum, genu, body, isthmus, and splenium.[9] Although some isolated agenesis of the corpus callosum was noted, some other patients also have defects of the hippocampal commissure.[10]

Brain development started in the first trimester which ventral induction induces the division of the prosencephalon and two lateral telencephalic vesicles and the diencephalon. Furthermore, the mesencephalon develops into the midbrain and the rhombencephalon becomes the metencephalon and myelencephalon. After 8–9 weeks, the choroid plexus and the falx cerebri become visible. Then, the corpus callosum can be identified by color Doppler in the mid-sagittal plane a few weeks later.[11,12] Studies showed that the corpus callosum develops first in the region of the anterior body and grows bidirectionally to form the genu and the rostrum at the anterior portion and the posterior part of the body and the splenium backward.[13]

Only until 20 weeks of gestation does the corpus callosum be visible. The corpus callosum was noted to grow according to the expansion of the hemispheres and develop the frontal segment prenatally, whereas the segment of splenium postnatally.[10]

Etiology of the Corpus Callosum Anomalies

The development of corpus callosum is a complicated process that may involve many pathways such as Semaphorin/Plexin/Neuropilin, Slit/Robo, Eph/Ephrin, Netrin/DCC/Unc5, Wnt/Ryk, and FGF8/MAOK. The special AT-rich sequence-binding protein plays a major role in the development of callosal projections because of its function in transcriptional regulation and chromatin remodeling.[9] Many factors interrupt this complex process leading to corpus callosum anomalies. For example, 52% of agenesis of the corpus callosum is an isolated condition whereas 25% have associated undiscovered causes of anomalies, and the other 23% are based on chromosomal, monogenic, or teratogenic causes.[14]

Prenatal Sonographic Features and Further Counseling

Complete agenesis of the corpus callosum

Discovering the agenesis of the corpus callosum is usually because of the absence of cavum septi pellucidi in the axial plane. The shared embryogenesis leads to the concurrent disappearance of both structures. The other sign is the teardrop shape of the ventricle since the dilation of occipital horns is the result of reduced white matter formation in the absence of callosal axons.[15]

Nonetheless, the golden standard for complete agenesis of corpus callosum (cACC) was diagnosed based on the midsagittal view of the brain which the corpus callosum, the thin band of white-matter fibers, was absent.[16,17] Thus, if the indirect sign such as ventriculomegaly, absence of the cavum pellucidi or widening of the interhemispheric fissure, or discovering other extracranial findings, the patient should be referred for further neurosonogram, including coronal and mid-sagittal planes.[8]

The normal coronal section should demonstrate the anterior horns of the lateral ventricles and cavum septi pellucidi. The cavum septi pellucidi should be oriented as close to vertically as possible.[18] In the coronal view of cACC, the roof of the diamond-shaped third ventricle would be elevated into the interhemispheric space and reach the midline flax. The anterior horns would become parallel and "comma-shaped" or "crescent-shaped" concavity towards the midline.[15]

After checking the coronal plane, rotate the transducer 90°, and an anechoic C-Shaped structure with echogenic outlines would appear as the corpus callosum.[15,18] The pericallosal artery will also appear under the color Doppler around the corpus callosum. The setting for pulse repetition frequency should be as low as the range of 20–40 cm/s.[18] The definitive diagnosis could be made if the corpus callosum is absent in the midsagittal plane and the pericallosal artery is not seen as well.

Counseling for cACC should include the commonly associated conditions. The risk of central nervous system (CNS) malformations is nearly 80%. Interhemispheric cysts with hydrocephalus, neuronal migration disorder, Dandy–Walker spectrum, inferior vermis agenesis, encephalocele, and interhemispheric fissure lipoma are documented in the literature.[19] The risk for other extra-CNS anomalies, such as skeletal, genitourinary, and congenital heart disease, is about 60%.[20] Miscellaneous conditions with inborn errors of metabolism, fetal alcohol syndrome, maternal phenylketonuria, vascular factors, TORCH infection (toxoplasmosis, other agents, rubella, cytomegalovirus, herpes and syphilis), and Zika virus are also noted.[19,21]

Genetic causes account for 30%–45% of ACC and approximately 10% of these cases are related to chromosomal abnormalities.[3] Aneuploidy such as trisomy 18, 13, and 8 are the most common. Other 20%–35% include numerous genetic syndromes and single gene disorders.[3,19] Thus, conventional karyotype could only identify complete or large partial chromosome anomalies in about 4.8% to 7.5% of ACC.[22] Microarray analysis may detect additional pathological copy number variations in 5.7%–6.9% of ACC with normal karyotype.[23] Recently, using whole-exome sequencing revealed genetic mutations such as *MED12* and *EFNB1* in a clinical case report.[24] It is expected that shortly, exome sequencing may be used as a clinical tool to help clinicians deal with such diseases.

A long-term follow-up for 128 pregnancies with fetal anomalies of the corpus callosum in Italy revealed 53 cases with apparently isolated cACC. Most of them (up to 90%) are evaluated by fetal magnetic resonance imaging (MRI) as well. Cases with subsequent termination of pregnancy, intrauterine demise, postnatal detection of 8p21.3q11.21 mosaic duplication, or lost follow-up were excluded. The remaining follow-up for 26 children for their neurodevelopment from 1 to 16 years old was done. The result showed that 88% of the

children do not have severely impaired neurodevelopment even among patients with isolated cACC.[25] Another recent study also disclosed the normal outcome in 65% of children with prenatally diagnosed ACC at the mean age of 3.8 years and equal gender distribution.[26] Another retrospective study revealed that the intelligence quotient level is about 82.3 ± 12.9 (mean ± standard deviation [SD]) in 34 cases with isolated ACC.[27] In summary, studies found that the most important prognostic factors are the presence of associated congenital anomalies and other genetic conditions.

Partial agenesis of the corpus callosum

Partial agenesis of corpus callosum (pACC) was defined when one of the four anatomical structures (rostrum, genu, body, and splenium) could be identified.[28] Generally speaking, the posterior segments are usually missing in patients who have partial agenesis.[15] The underlying etiology is the arrest of development between 12 and 18 weeks of gestation, which involved the dorsal part or the splenium.[29] Although studies cannot confirm whether true malformation or disruptive events result in callosal partial agenesis, it is undeniable that the association of interhemispheric arachnoid cyst, holoprosencephaly, asymmetric ventriculomegaly, and migration disorder has been reported.[30-32]

Under the sonography, the cavum septi pellucidi are detectable with a normal shape in the axial plane. But some of them can appear with dysmorphic shapes with wide and short images. A screening marker is a length-to-width ratio <1.5 for the detection of partial agenesis of the corpus callosum.[18] It is estimated that small cavum septi pellucidi accounts for 80% of partial agenesis of corpus callosum cases after 20 weeks.[33]

A standard checklist suggested that fetal head examination should be performed transabdominally in the axial, coronal, and sagittal planes. The corpus callosum should be measured in its length and thickness in the median plane. An abnormal shape or shorter appearance for gestation age raises the suspicion of partial agenesis. There are few works of literature focusing on partial agenesis of corpus callosum which emphasized the difficulties in diagnosis.[34-36] The most common feature is colpocephaly (the enlargement of the ventricular atria and the occipital horns) in a retrospective case series study. However, the absence of any indirect signs could happen and lead to a lower detection rate in the prenatal period compared to the postnatal period with a discrepancy proportion among callosum anomalies.[35] In addition to grayscale ultrasound, Doppler imaging and the MRI may increase clinicians' confidence in diagnosing such a disease.[29]

Counseling the prognosis of pACC is challenging because of the paucity of case reports and the selection bias from postnatal neurodevelopment children. Most of the literature demonstrates that the neurological outcome in isolated pACC and cACC are similar.[17,29,37] It is difficult to differentiate neurodevelopmental outcome between cACC and pACC because of the overlapping neurological symptoms and imaging studies in the prenatal and postnatal period.[38] A meta-analysis showed that 76.42% (95% confidence interval [CI], 64.3–86.1) of children with isolated cACC in 20 studies has normal neurodevelopmental outcome, whereas 71.42% (95% CI, 53.1–86.7) of children with isolated pACC in 15 studies has normal outcome. The rates of borderline/moderate neurodevelopmental outcome in cACC and pACC were 16.04% and 14.92%, respectively. The rates of severe neurodevelopmental outcomes in cACC and pACC were 8.15% and 12.52%, respectively. These data need to be interpreted with caution because of the limited number of patients.[20] For isolated cases of pACC, the percentage of neurodevelopmental delay could be around 25% in another study.[35] One study claimed that a long-term follow-up is necessary because of the lower median IQ and behavior troubles which are not related to partial or complete ACC.[17] Furthermore, it is important to explain in advance that changing diagnosis (e.g., from isolated-partial ACC to complex-complete or complex-partial) may happen after birth.[39]

Dysgenesis

The definition for this type of corpus callosum abnormality relies on the altered structure with the presence of each of four anatomical segments.[40] However, mixed-used radiology terminology in literature is common and sometimes pACC could be referred to as dysgenesis.[41] Thus, scarce articles are truly related to this kind of malformation.

Hypoplasia or thin corpus callosum

Hypoplasia of the corpus callosum is defined as a thinner corpus callosum whose thickness is below the 10th percentile, or <2 SD.[40] The references to corpus callosum thickness could be found in other literature.[42,43] The prevalence of the diagnosis among all anomalies of the corpus callosum is about 20%.[40] The embryogenesis of hypoplasia can be traced to 11–20 weeks when association fibers failed to enter massa commissuralis. The anatomical manifestation is partly or wholly thinning of the corpus callosum.[44] Callosal hypoplasia is suspected to be the consequence of teratogens (e.g., radiation, maternal alcohol consumption) or compression (e.g., intracranial masses, obstructive hydrocephalus).[45] Genetic syndromes and metabolic disorders such as Apert syndrome, Crouzon Syndrome, Masa Syndrome, Mowat–Wilson syndrome, and nonketotic hyperglycemia are also noted in cases with hypoplasia of corpus callosum.[45-47]

The finding could be isolated, but combined with either intracranial or extracranial abnormalities are usually seen. For instance, the absence of the septum pellucidum, ventriculomegaly, hemimegalencephaly, septal-optic dysplasia, hypoplasia of the cerebellar vermis, microcephaly, hydrocephalus, cardiac disease, maxillofacial, kidney anomalies, and the skeletal anomaly are all the possible concurrent conditions.[29,40,48]

Diagnosis for hypoplasia is more difficult because the axial plane is usually normal without specific sonographic signs. Hence, the diagnosis was often made in the third trimester.[40] The intracranial or extracranial findings usually raise suspicion to discover hypoplasia. Follow-up for isolated mild or moderate ventriculomegaly also confirmed the diagnosis of hypoplasia of corpus callosum in 6.7% of cases.[19,49]

Prognosis prediction remains challenging due to the limited number of cases in prenatal diagnosis. D'Ambrosio *et al*. presented three cases of corpus callosum hypoplasia and two of them have normal neurological development at 1 year and 6 months follow-up. The only development delay child has an intellectual disability due to other intra-CNS anomalies. D'Ambrosio *et al*. also review that isolated hypoplasia is noted in 31% of all 48 hypoplasia cases in literature but scarce information about the neurodevelopmental outcomes. Syndromic hypoplasia is often considered to have a poorer prognosis.[40]

Hyperplasia or thick corpus callosum

Abnormal thickening of the corpus callosum is named hyperplasia. This abnormality is comprised of 5% of corpus callosum pathologies.[50] Similar to the hypoplasia definition, it is also recognized that more than 2SD of the mean values are in any or all of the parts of the corpus callosum.[42] The association fetal anomalies include ventriculomegaly, cortical malformation, and macrocephaly.[19] The difference between hyperplasia and other corpus callosum anomalies is the relevance of neurogenetic etiology, such as Cohen syndrome (OMIM#216550), macrocephaly-capillary malformation (OMIM#602501) or neurofibromatosis type 1 (OMIM#162200).[19,51] For instance, the increasing amount of white matter tracts crossing through the corpus callosum in neurofibromatosis type 1 leads to the hyperplasia of the corpus callosum.[52]

A peculiar sonographic finding in the literature review is cases that could present with hyperechogenicity which may relate to an increased number of aberrant fibers or associated edema rather than the commonly seen pericallosal lipoma.[50,53] An autopsy report for two fetuses after their termination disclosed that an abnormal isolated thick corpus callosum could be associated with increased representation of the key midline glial structures. These conditions lead to possible disturbance of the axon guidance mechanism of callosal formation.[54]

Although the prognosis for hyperplasia combined with other fetal anomalies or abnormal head circumference is a poor indicator, isolated hyperplasia may be associated with a more optimistic outcome.[50,55] Shinar *et al*. also presented nine fetuses with an isolated thick corpus callosum at a mean gestation age of 23 + 5 weeks and six of them were evaluated as having normal neurological development at a median age of 9 months (range, 7–18 months). The data supported that isolated hyperplasia during the second trimester is not a warrant of poor prognosis.[53] Nonetheless, studies also emphasize the thorough checkup and further follow-up to rule out other intracranial or extracranial abnormalities while counseling such cases.[55]

Conclusion

Anomalies of the corpus callosum are challenging both in diagnosis and counseling. Incidentally found an absence of cavum septi pellucidi or dysmorphic septi pellucidi are hints for suspicion of such diseases and a referral to the maternal-fetal medical center is recommended. A detailed neurosonogram after 20 weeks should be performed, including three different planes: axial, coronal, and sagittal planes. The five different categories of the corpus callosum, including cACC, pACC, dysgenesis, hypoplasia, and hyperplasia are usually related to diverse etiology which disrupt the embryogenesis at different stages. Genetic studies are also important because 30%–45% of agenesis of the corpus callosum is related to a chromosome or genetic disease. Whole-exome sequencing is possibly the next feasible tool if negative findings are demonstrated in both karyotype and microarray testing. Fetal MRI may help clinicians to give a more solid conclusion for diagnosis and survey other intracranial or extracranial anomalies. Distinguishing the complex or isolated types of corpus callosum anomalies is essential because the prognosis is usually poor for complex types, whereas the neurological outcome for isolated corpus callosum disorders may be more favorable. However, further long-term follow-ups are still needed for children who were diagnosed with anomalies of the corpus callosum.

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