Corpus callosum (CC) keeps the two hemispheres of brain connected to each other. It links the wide areas of cerebral cortex of two hemispheres, except the fibers from the primary visual area and hand & foot somesthetic area. It consists of about 300 million myelinated fibers, approximately 2-3% of all cortical fibers. Glial cell populations mainly “Midline Zipper Glial” & “Glial Wedge” are identified and found to have a significant role in the development of corpus callosum. Any disturbances in one or more of these events and absence or damage to cross the midline can be seen growing medially within the hemisphere. The fibers cross the midline through mass commissurals at 11-12 weeks of gestation to form corpus callosum. Generally, axon tracts form according to a conserved ontogenic sequence. They develop under the guidance of non-neural substrate cells like glia. Glial cell populations mainly “Midline Zipper Glial” & “Glial Wedge” are identified and found to have a significant role in the development of corpus callosum. Hence the development of corpus callosum depends on series of highly regulated complex development events which begins at time of gestation & continues to adulthood. Any disturbances in one or more of these events and absence or damage to cross the midline can be seen growing medially within the hemisphere.
to glial cells, midline sling can result in agenesis of corpus callosum [12]. Among the congenital brain defects, agenesis of corpus callosum is the most common with occurrence of 1.4 - 1.8 per 10,000 live births [13, 14]. Morphologically two types of agenesis of CC have been identified. In type 1- corpus callosal axons are formed, but do not cross the midline, they form aberrant fiber bundles of Probst and in type 2- corpus callosal axons are not formed [15]. Based on Mid sagittal MR imaging three morphological variants have been described like complete agenesis, partial agenesis and hypoplastic corpus callosum. Complete agenesis – in this type all components of corpus callosum are lacking. Partial agenesis – in this type absence of some components of corpus callosum is seen. Hypoplastic corpus callosum – in this type it is thin but structurally intact [15].

Disorders of corpus callosum can occur as an isolated case or may be associated with cranial (brain) and extracranial malformations with context of a congenital syndrome. About 10% of cases cause of Ag CC is associated with genetic disorder and 20-35% have specific monogenic or polygenic disorders related to teratogenic infectious, toxic or metabolic exposures [16]. Despite gross anatomical consequences the spectrum of neurological manifestations varies from severe intellectual disability to normal intelligence [12]. In individuals with isolated Ag CC subtle neuropsychological alterations happen when the cognitive demand increases as the age advances [18, 19]. Hence requiring long term follow up with essential neuropsychological screening. The present study is done to find out association between the corpus callosum abnormalities with other brain anomalies and enlist the different morphological variations of corpus callosum.

**MATERIALS AND METHODS**

The present cross-sectional study was conducted by collecting and observing brain MRI images from medical records of hospital. Institutional Ethical Committee approval was taken to conduct the study. MRI images of patients visited between 2013 to 2021 were collected from medical records. All images of patients were stored in PAX database, which can be accessed for conduction of any clinical research study.

**Inclusion criteria**

All patients with age ranged from 0-78 years with recorded diagnosis of AgCC were included in the study.

**Exclusion criteria**

Patients with secondary causes of callosal anomalies like congenital hydrocephalus, stroke & metabolic disorders were excluded.

**RESULTS**

Suitable descriptive and inferential statistics were used. Data is represented in the form of frequencies and percentages. 31 cases with labelled diagnosis of Ag CC were obtained from records. Out of which, 8 were excluded; 5 cases excluded due to non-availability of MRI images, one case was excluded because AgCC was secondary to ventriculomegaly, in two cases holoprosencephaly was the cause for AgCC. Remaining 23 cases were included in the study. In 23 cases images were reviewed. Partial agenesis was observed in 16 cases & complete agenesis in 7 cases. Ag CC associated with suboccipital meningoencephalocele (N=1, 4.6%), lipoma (N=2, 8.6%), cyst (N=3, 13%), Dandy-Walker malformation (N=1, 4.3%), hippocampal malformation (N=1, 4.3%), colpocephaly (N=2, 8.6%) & white matter abnormalities (N=1, 4.3%).

<table>
<thead>
<tr>
<th>Table 1: shows pie frequency of results</th>
<th>Frequency</th>
<th>percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meningoencephalocele</td>
<td>1</td>
<td>9.1</td>
</tr>
<tr>
<td>Lipoma</td>
<td>2</td>
<td>18.2</td>
</tr>
<tr>
<td>Cyst</td>
<td>3</td>
<td>27.3</td>
</tr>
<tr>
<td>Dandy walker malformation</td>
<td>1</td>
<td>9.1</td>
</tr>
<tr>
<td>Hippocampal malformation</td>
<td>1</td>
<td>9.1</td>
</tr>
<tr>
<td>Colpocephaly</td>
<td>2</td>
<td>18.2</td>
</tr>
<tr>
<td>White matter malformation</td>
<td>1</td>
<td>9.1</td>
</tr>
<tr>
<td></td>
<td>11</td>
<td>100.0</td>
</tr>
</tbody>
</table>
DISCUSSION

The most common congenital malformations observed in humans is agenesis of corpus collosum.\textsuperscript{[12]} The estimated prevalence is 3-7 per 1000 birth in general population, & 2-3 per 100 in children with development disabilities.\textsuperscript{[20]} Most of the cases of AgCC were discovered by chance while investigating some other related disorders.\textsuperscript{[21]} The preponderance of male: female ratio in adults is 2:1, in children it is 3:2.\textsuperscript{[21]} In about 70% of cases of complete isolated agenesis of corpus collosum, the causative condition remains unknown. In 10% of cases of Ag CC have chromosomal anomalies mainly trisomies 8, 13, & 18, while in 7% of Ag CC are due to foetal alcohol syndrome. About 20-35\% have specific monogenic or polygenic disorders.\textsuperscript{[16]} Congenital CNS anomalies commonly associated with Ag CC are holoprosencephaly, grey matter heterotopia, Arnold Chiari II malformation & Dandy-walker syndrome.\textsuperscript{[22]} Other development disorders found associated with Ag CC are interhemispheric cysts, cleft palate, Andermann syndrome & Apert syndrome.\textsuperscript{[24,25]} Tang et al described apparent delayed sulcation in review of 29
cases of foetal Ag CC. They also described sulcal delay in fetuses before 30 weeks of gestation & no such detectable sulcal delay in more than 30 weeks of gestation. They presented a case of sulcal delay at 26.4 weeks which eventually turn to be normal for age. According to clinical & neurocognitive characteristics, patients with Ag CC can be classified into three groups. In first group patients have severe neurocognitive symptoms & very low IQ. In second group Ag CC play a major role as a causative factor for neurodevelopmental diseases. In third group patients are neurologically asymptomatic with normal IQ. Diagnostic approach to Ag CC during prenatal period by performing USG between 18 & 22 weeks. Ideally this can be supplemented with structural USG & prenatal MRI of brain. In addition to this repeat Brain MRI & amniocentesis for screening chromosomal disorder & congenital infections. During post-natal period approach by multidisciplinary team with complete medical history, physical examination & imaging of all systems like cardiovascular, respiratory, gastrointestinal & urinary system. Asymptomatic cases of Ag CC require follow up as they are bond to develop mild behavioral & cognitive deficits during early adulthood. Symptomatic cases require rehabilitation. According to recent studies on sigmoid bundle found out that interhemispheric connections can be rerouted.

In a recent study it is been confirmed that Ag CC has better outcome if it is not associated with other congenital brain malformations. Some studies have reported worse outcome in cases of Ag CC where as multiple studies reported no difference in behavioral & medical outcomes on comparing partial & complete Ag CC. Advantage of rehabilitative treatment is on long run it may modify neurological pathways through CNS plasticity. Rehabilitative treatment can give a specific exercise & a suitable environment to improve structural & functional brain adaptation. Parents & teachers of affected children should be counselled to help them overcome neuropsychological deficits with adequate strategies.

**CONCLUSION**

Even though most of the cases of Ag CC are asymptomatic but requires regular follow up to track the late manifestations. As Ag CC is not always associated with other brain anomalies early recognition of problems and timely intervention can be done with appropriate rehabilitative measures.

**REFERENCES**


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