**Introduction:** It is well known that the risk of brain injury, shown by neuroimaging and/or by neurodevelopmental tests, is increased in children with isolated CHD. Despite being classically attributed to extracorporeal surgery, recent studies support a prenatal onset. Structural fetal brain lesions, reduced brain volume and changes in fetal cerebral circulation have been described in the third, and even in the second, trimester neuroimaging.

The aim of the study was to evaluate the presence of abnormal cerebral findings in fetuses affected by isolated CHD.

**Material and Methods:** Prospective cohort study including 40 consecutive fetuses with isolated CHD (without associated malformations and/or genetic abnormalities).

Fetal biometry (BPD and HC), middle cerebral artery pulsatility index (MCA-PI) and cerebroplacental ratio (CPR) were assessed at the time of diagnosis and then every 4 weeks. Neurosonography was performed at 28, 32 and 36w and fetal MRI at 36w.

**Results:** CHD were classified according to the expected main pattern of placental versus systemic mix of blood supply to the brain:
1. subgroup 1 (low), n=17; 2. subgroup 2 (intermediate), n=20; 3. subgroup 3 (high), n=3

Regarding cefalic biometry, HC was below 10th centile in 2/34 (5.9%) and in 5/38 (13.1%) at 28 and 36w, respectively. When MCA was analyzed, 9/33 (27.3%) and 8/37 (21.6%) showed a PI < 10th centile at 28 and 36w, respectively, and a CPR < 10th centile in 6/33 (18.2%) and 15/36 (41%) at 28 and 36w, respectively (table 1).

<table>
<thead>
<tr>
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<th>CC &lt; p10 n (%)</th>
<th>MCA-PI &lt; p10 n (%)</th>
<th>CPR &lt; p10 n (%)</th>
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</thead>
<tbody>
<tr>
<td>28w</td>
<td>2/34 (5.9)</td>
<td>9/33 (27.3)</td>
<td>6/33 (18.2)</td>
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<tr>
<td>36w</td>
<td>5/38 (13.1)</td>
<td>8/37 (21.6)</td>
<td>15/36 (41.7)</td>
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</table>

Table 1: CC, MCA-PI and CPR < 10 centile at 28w and 38w.

Neurosonography at 28 weeks was abnormal in 1 case (truncus arteriosus; frontal fibrin strands + bilateral VMG) (figure 1). Fetal MRI at 36 weeks showed abnormal findings in 6 cases (4 mild VMG, 1 subependymal cysts + partial ACC, 1 white matter lesion).

**Conclusion**

Fetuses with CHD show signs of brain sparing. Moreover, neurosonography and fetal MRI abnormalities are detected in prenatal life. These findings suggest that brain lesions associated with CHD might have an early onset. However, long-term follow-up studies correlating prenatal findings with postnatal neurodevelopment are limited.