A MORPHOLOGICAL STUDY OF PLACENTA IN CHILDREN WITH AND WITHOUT HYPOSPADIAS

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Abstract:

Introduction: Hypospadias can be defined as an abnormal urethral orifice under surface of the penis with or without chordee and with or without dorsal hood. At a critical time in sexual differentiation of the male fetus, HCG enters fetal plasma from syncytiotrophoblast; acts as an LH surrogate and stimulate replication of testicular Leydig cells and testosterone synthesis to promote male sexual differentiation. The placental insufficiency may disrupt the supply of nutrients and HCG to the fetus leading to growth retardation and hypospadias.

Aim: The aim of this study was to observe and document morphological changes of placenta in children with hypospadias and compare with controls.

Materials & Methods: The present study was a case control study from July 2008 to July 2011. The data base of the labor registries of the hospital indicated that there were total 3243 male births during this period. All examined for presence/absence of hypospadias by attending pediatrician. Hypospadias was detected in 17 male newborns. Control cases comprised of 68 male newborns without hypospadias of similar gestational age and birth weight collected by cluster sampling.

Result: Total number of male birth during the study period was 3243, in that 17 children born with hypospadias. The incidence of hypospadias in our hospital was 0.52%. Gestational age, Birth weight, Placental weight, Placental thickness, Placental volume, volume of infarcts, F.P Ratio, Cord length, were similar in children with hypospadias when compared with controls. CONCLUSION: Fetal factors like gestational age, birth weight, placental weight, Feto-placental ratio were not significantly associated with hypospadias. This study shows no role of placenta in the etiology of hypospadias in children with normal birth weight.

Keywords: Hypospadias, Feto-placental ratio, Placenta.
differentiation of the male fetus, HCG enters fetal plasma from syncytiotrophoblast; acts as an LH surrogate and stimulate replication of testicular Leydig cells and testosterone synthesis to promote male sexual differentiation.

The placental insufficiency may disrupt the supply of nutrients and hCG to the fetus leading to growth retardation and hypospadias. To validate this hypothesis, we analyzed all the male infants born at our hospital with hypospadias for fetal growth parameters, and collected placenta for detailed evaluation. And assessed maternal risk factors associated with hypospadias by questionnaire proforma.

Materials & methods:
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Once hypospadias was identified, the neonate was examined in detail to identify other anomalies, weight at birth, and gestational age. The placenta was collected and examined for placental weight, thickness, placental volume and cord information. Feto-placental weight ratio was measured as a reference for placental function and intrauterine fetal growth. The placenta of these controls was also subjected to detailed evaluation and examination.

Data was compiled and analyzed by descriptive analysis; comparison of risk factors was done using student t test. P value <0.05 was considered as significant.

Result:
Total number of male birth during the study period was 3243, in that 17 children born with hypospadias. The incidence of hypospadias in our hospital was 0.52%. The characteristics of child at birth are considered as fetal demographic factors associated with hypospadias (Table 1).

Characteristics of child at birth—Gestational age, Birth weight, Placental weight, Placental thickness, Placental volume, volume of infarcts, F.P Ratio, Cord length, were similar in children with hypospadias when compared with controls (Table 1). Gestational age was similar in hypospadias (38.64±0.99weeks) when compared with controls (38.37±1.14weeks). Birth weight in children with hypospadias was (2.96±0.19kg), when compared with controls (3.01±0.17kg). There was no significant difference in placental weight in children with hypospadias (462.31±8.56gm) when compared with controls (461.92±8.04gm). Placental thickness was similar in children with hypospadias (2.08±0.27cm) when compared with controls (2.00±0.00cm). There was no significant difference in placental volume in children with hypospadias (362.65±14.14cc) when compared with controls (364.22±17.17cc). Feto-placental ratio was not higher in children with hypospadias (6.53±0.40) when compared to controls (6.74±0.42). There was no significant difference in length of umbilical cord in children with hypospadias vs controls (58.31±2.52 vs 56.85±2.91, P=0.18). Number of blood vessels in the umbilical cord were normal in children with hypospadias.

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**Table 1:** Comparison of fetal demographic factors associated with hypospadias

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Hypospadias (n=17)</th>
<th>Controls (n=68)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gestational age (week)</td>
<td>38.64±0.99</td>
<td>38.37±1.14</td>
<td>0.96</td>
</tr>
<tr>
<td>Birth weight (kg)</td>
<td>2.96±0.19</td>
<td>3.01±0.17</td>
<td>0.11</td>
</tr>
<tr>
<td>Placental weight (gm)</td>
<td>462.31±8.56</td>
<td>461.92±8.04</td>
<td>0.90</td>
</tr>
<tr>
<td>Placental thickness (cm)</td>
<td>2.08±0.27</td>
<td>2.00±0.00</td>
<td>0.32</td>
</tr>
<tr>
<td>Placental volume (cc)</td>
<td>362.65±14.14</td>
<td>364.22±17.77</td>
<td>0.78</td>
</tr>
<tr>
<td>Feto-placental ratio</td>
<td>6.53±0.40</td>
<td>6.74±0.42</td>
<td>0.16</td>
</tr>
<tr>
<td>Cord length (cm)</td>
<td>58.31±2.52</td>
<td>56.85±2.91</td>
<td>0.18</td>
</tr>
</tbody>
</table>

P-value <0.05 is considered as significant

**Discussion:**

Many authors have suggested that disturbance of placental function early in pregnancy is the key mechanism underlying both preterm birth/low birth weight and the improper closure of the urethra, because the placenta is the main producer of pregnancy hormones in early pregnancy and is thus instrumental in the differentiation and development of the fetal organs\(^6,7,8\). This study could not find an association between hypospadias risk and preterm birth (< 37 weeks gestation) and/or being small for gestational age (< 10\(^{th}\) percentile) because in this study all the children born with hypospadias were normal birth weight (>2.8 kg) and all of them were term birth (>39 weeks). It is well known that in normal, preterm and term infants there is a direct relation between birth weight and weight of placenta\(^9\). In this study, all the placentae were of normal thickness because none of the children in the study group were low birth weight. Ultra sonographic study of placental volume found that, placental volume was directly proportional to the birth weight of the babies\(^10\). However, there is limited information on the relationship between intrauterine placental volume and birth weight\(^11\). In this study all the children born with normal birth weight and placenta were in normal weight hence volume of placenta in children with hypospadias was similar with the control on comparison.

The ratio of placental weight to birth weight is described as a marker of fetal growth. The correlation of birth weight and placental size is to be expected as both placental weight and size are known to increase as birth weight increases\(^12\). In this study the feto-placental ratio in hypospadias children was not found to be increased in comparison with the controls.

**Conclusion:**

Several studies have found reduced placental function as underlying etiology for low birth weight and hypospadias. In the present study all the children born with hypospadias were of normal birth weight. Fetal factors like gestational age, birth weight, placental weight, Feto-placental ratio were not significantly associated with hypospadias. This study shows no role of placenta in the etiology of hypospadias in normal birth weight children.

**Acknowledgement:**

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**References:**

8. Aschim EL, Haugen TB, Tretli S, Daltveit AK, Grotmol T. Risk factors for...