Selected afterbirth parameters among growth restricted mature newborns

PAWEŁ KRAJEWSKI, MARIA KWIATKOWSKA, MAŁGORZATA POKRZYWNICKA

Abstract
The placenta as a fully-developed and efficient organ is formed at the beginning of the third trimester of pregnancy. It is a multifunctional system that for a fetus plays the role of a respiratory, nutritious and endocrine system. The placenta is essential in proper fetal development and its failure or dysfunction influence the condition of a baby and may also lead to hypotrophy (IUGR). Material and methods: The aim of the research was to compare selected placental parameters in cases of mature intrauterine growth restricted newborns and in eutrophic newborns. The research group included 124 mature IUGR neonates and the placentas from those pregnancies. The newborns were delivered at the Department of Clinical Sciences of Perinatology, Medical University of Łódź during a period from 1.01.2007 to 31.12.2008. The control group comprised 91 mature eutrophic neonates and their placentas. A gross examination was carried out in relation to the placentas of the examined population of neonates, taking into account their body weight, surface of the placenta, position of insertion of the umbilical cord and its length, as well as the presence of deposits, infarctions or hematomas in the placenta. The material was elaborated statistically with the use of t-Student’s test, Mann-Whitney test, chi-square test and exact Fisher’s test. Results: Statistically significant differences were observed between IUGR neonates and eutrophic neonates as for their birth body weight and length as well as the head circumference (p < 0.05). In respect of all examined afterbirth parameters, i.e. the placental weight, its surface, the weight ratio, the length of the umbilical cord and the type of its insertion as well as the occurrence of deposits, hematomas and infarctions – the differences between the groups examined were also statistically significant (p < 0.05). Conclusions: 1) Pathological changes in placentas of the births of intrauterine growth restricted neonates were observed significantly more often than in placentas of the births of eutrophic neonates. 2) Placentas of mothers who delivered babies with IUGR were characterized by significantly lower weight and surface, significantly higher weight ratio and more frequent anatomical changes.

Key words: intrauterine growth restriction, birth at term, newborn, placenta

Introduction
The placenta as a fully-developed and efficient organ is formed at the beginning of the third trimester of pregnancy. It is a multifunctional system that for a fetus plays the role of a respiratory, nutritious and endocrine system as well as that of kidneys and liver [1].

A fetus, a placenta and a mother form a composite system of dynamic equilibrium [2]. During the intrauterine life the fetus is well-protected by the placenta, fetal membranes and amniotic fluid. The placenta is essential in proper fetal development and its failure or dysfunction influence the condition of a child and may also lead to hypotrophy (IUGR – intrauterine growth restriction) [3].

IUGR is diagnosed in about 3-10% of all neonates born alive and in about 20% of those who were delivered dead [4]. It seems that the main reason leading to IUGR is uterine-placental failure, in 10% of cases IUGR originates due to secondary infections, and in 5-15% due to chromosomal disorders or other genetic defects [4].

Material and methods
The aim of the research was to compare selected placental parameters in cases of mature intrauterine growth restricted newborns and in eutrophic neonates.

The research group included 124 mature IUGR neonates and the placentas from those pregnancies. The newborns were delivered at the Department of Perinatology, Medical University of Łódź during the period from 1.01.2007 to 31.12.2008. The control group comprised 91 mature eutrophic neonates and their placentas.

The parameters checked included – the body weight (with the use of centile charts according to professor Chlebna-Sokół), the total length and the head circumference (verified by means of the Ballard’s scale). Intra-
uterine growth restriction is a state when the birth body weight of a neonate and/or its length are below the 10th centile for their gestational age [5]. In our research hypothesis was assessed according to the body weight.

A gross examination was carried out in relation to the placentas of the examined population of neonates, taking into account their body weight, surface of the placentas, position of insertion of the umbilical cord and its length as well, as the presence of deposits, infarctions or hematomas in the placenta.

The material was elaborated statistically with the use of t-Student’s test, Mann-Whitney test, chi-square test and exact Fisher’s test.

Results

The demographical data of the examined population of IUGR neonates and eutrophic neonates is presented in table 1.

Table 1. Demographical data of the examined population of mature IUGR neonates and eutrophic neonates

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Mature neonates</th>
<th>Statistically significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>With IUGR n %</td>
<td>Eutrophic n %</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>– male</td>
<td>67 (54.03%)</td>
<td>41 (45.05%)</td>
</tr>
<tr>
<td>– female</td>
<td>57 (45.97%)</td>
<td>50 (54.95%)</td>
</tr>
<tr>
<td>Birth body weight (g)</td>
<td>2145 (± 437.47)</td>
<td>3368.35 (± 432.48)</td>
</tr>
<tr>
<td>Me = 2250 800-2700</td>
<td>Me = 3340 2430-4300</td>
<td></td>
</tr>
<tr>
<td>Length (cm)</td>
<td>49.04 (± 4.15)</td>
<td>55.22 (± 2.78)</td>
</tr>
<tr>
<td>Me = 50 32-59</td>
<td>Me = 55 49-63</td>
<td></td>
</tr>
<tr>
<td>Head circumference (cm)</td>
<td>31.31 (± 2.08)</td>
<td>32.71 (± 2.10)</td>
</tr>
<tr>
<td>Me = 32 25-36</td>
<td>Me = 32 28-38</td>
<td></td>
</tr>
</tbody>
</table>

Me – mean value (median)

Table 2. Characteristics of selected parameters of afterbirth in births hypotrophic neonates and in births of eutrophic newborns

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Placentas</th>
<th>Statistically significance</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>In births of hypotrophic neonates</td>
<td>In births of eutrophic neonates</td>
</tr>
<tr>
<td>1. Placental weight (g)</td>
<td>504.96 (± 121.37)</td>
<td>672.31 (± 99.13)</td>
</tr>
<tr>
<td>Me = 500 200-800</td>
<td>Me = 660 500-920</td>
<td></td>
</tr>
<tr>
<td>2. Size (cm²)</td>
<td>326.98 (± 125.51)</td>
<td>433.45 (± 97.24)</td>
</tr>
<tr>
<td>Me = 310.50 80-650</td>
<td>Me = 440 170-675</td>
<td></td>
</tr>
<tr>
<td>3. Weight ratio = (Placental weight)/(Weight of a neonate)</td>
<td>0.24 (± 0.06)</td>
<td>0.20 (± 0.03)</td>
</tr>
<tr>
<td>Me = 0.24 0.14-0.65</td>
<td>Me = 0.20 0.15-0.31</td>
<td></td>
</tr>
<tr>
<td>4. Length of the umbilical cord (cm)</td>
<td>53.98 (± 11.02)</td>
<td>58.89 (± 6.95)</td>
</tr>
<tr>
<td>Me = 55 15-87</td>
<td>Me = 58 40-80</td>
<td></td>
</tr>
<tr>
<td>5. Cord insertion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>– central</td>
<td>99 (79.84%)</td>
<td>61 (67.03%)</td>
</tr>
<tr>
<td>– centrifugal</td>
<td>17 (13.71%)</td>
<td>1 (1.10%)</td>
</tr>
<tr>
<td>– marginal</td>
<td>5 (4.03%)</td>
<td>25 (27.47%)</td>
</tr>
<tr>
<td>– velamentous</td>
<td>3 (2.42%)</td>
<td>4 (4.40%)</td>
</tr>
<tr>
<td>6. Cord artery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>– double</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>– single</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>7. Deposits, infarctions, premature placenta ablation, placental hematoma, umbilical vein thrombosis</td>
<td>27 (21.77%)</td>
<td>2 (2.20%)</td>
</tr>
</tbody>
</table>

Me – mean value (median)
The statistically significant differences observed referred to the birth body weight and the head circumference in both groups of newborns ($p < 0.05$).

There were no differences found in the percentage of primigravida mothers and multipara mothers ($p > 0.05$).

Table 2 contains data concerning selected parameters in the afterbirth of the births of hypotrophic neonates and of the births of the newborns from the control group. In case of all examined parameters – i.e. the placental weight, its surface, the weight ratio, the length of the umbilical cord, the kind of its insertion or the presence of deposits, hematomas and infarctions – the differences between the examined groups were statistically significant ($p < 0.05$) (Fig. 1 and 2).

**Discussion**

The afterbirth (the placenta, the umbilical cord, the extraplacental fetal membranes) is an organ that in many ways is a unique one.

The growth of a fetus in the first two trimesters mainly depends on the inborn potential, but in the third trimester the most important role is played by placentas and appropriate transport of nutritive substances. Diminishing the nutritive efficiency of the placenta results in intrauterine growth restriction [4-6].

A placenta, in 95% of cases, is round or oval and its average measurements equal respectively 22.5 × 21 × 2.2 cm (without the extraplacental fetal membranes and the umbilical cord). The weight of a placenta is contained in the range 300-1600 g, and the fetal/placental weight ratio equals from 0.12 to 0.14 [4].

A placenta without focal lesions has homogenous compactness and spongiform structure all over its surface. The umbilical cord may be inserted at various positions on a placenta (centrally, marginally and eccentrically). The occurrence of calcium deposits is related to the preterm pregnancy, the excessive amount of calcium and estrogens in a mother, necrosis and decay of placental tissues. The length of the umbilical cord varies from 30 to 70 cm [1, 4].

The placental anatomical problems include: ablation of the placenta, hematoma, single umbilical artery, infarctions, abnormal position of the umbilical cord, thrombosis of the umbilical vein and placental failure.

The birth body weight of a neonate is fully dependent on efficient transport, from a mother to a fetus of nutritive substances, water, electrolytes, vitamins, hormones and gas exchange [1, 3, 4, 7]. The measurement of the placental weight during the second trimester of pregnancy gives a good idea about its weight before delivery and enables early detection of pathology [3, 7].

Gross examination of placentas in IUGR neonates reveals changes responsible for the restriction of placen-
tal functions. They can be (and should be) diagnosed to a great extent in utero. Suska et al. [8] enumerated to serious changes in a placenta, the reduction of its weight or surface and the occurrence of infarctions. Other gross changes were not causatively related to IUGR and were equally often observed in case of eutrophic neonates’ placentas [8].

In case of IUGR neonates, a substantial drop in the level of the placental growth hormone was discovered, i.e. the hormone that is responsible for the appropriate size of a placenta [9].

Amato et al. [10] who examined placentas of primigravida and multipara mothers did not find in them gross changes, but in 37 out of 45 and in 12 out of 15 of these women the weight of placentas was lower than 400 g.

The size of the changes observed macroscopically (infarctions, thromboses) was not statistically different between the placentas of the IUGR neonates and the control group in the research conducted by Deregowski et al. However, they observed a significant difference in the placental size in both groups [11].

Bjoro [12] showed that deposits, infarctions of the placenta and a single umbilical artery occurred more often in the group of newborns suffering from IUGR than in healthy neonates and the same pathological changes were confirmed in our study in IUGR births.

Biswas et al. stated that placentas of mature neonates were bigger and in most cases there was a central insertion of the umbilical cord. On the other hand, 11% of IUGR neonates had abnormal position of the umbilical cord (marginal or velamentous insertion) and the weight of their placentas as well as their volume and surface were significantly lower [6]. These placentas were functional and compensated abnormal morphology. Therefore, the authors did not decide to put forward the thesis that big morphological abnormalities in the structure of a placenta induce IUGR [6].

Salafia et al. [13] examined the reasons for idiopathic fetal hypotrophy and proved that placentas of hypotrophic neonates more often were subject to various kinds of damage (55% of placentas with IUGR, 32% of those without IUGR), including blood vessels thrombosis (in 71% of cases with IUGR, and in 9% without IUGR).

It is considered that the most important factor having significant influence on the transport of substances from a mother to a fetus is proper maternal-fetal circulation in a placenta. Another emphasized factor is the size and functional volume of the exchange area [3].

Pardi et al. [14] discovered that the relation between the maternal and fetal circulation in a placenta has a key meaning for the exchange of oxygen and nutrients. Retrogressive changes in a placenta significantly reduce the fetal/placental ratio because they reduce placental transport and disturb metabolism of a fetus.

The placentas from pregnancies complicated by IUGR may show gross changes, and vascular damage can lead to pregnancy induced hypertension [3, 7].

According to Jain et al. [2] PIH (pregnancy induced hypertension) is common in primigravida mothers and most probably is the main reason for the IUGR onset. In the placentas of mothers whose babies suffered both from IUGR and PIH, but also in case of hypotrophic neonates without PIH the researchers observed focal lesions, infarctions, ischemia and intervillous hemorrhages.

In case of women who were diagnosed with deep vein thrombosis or apoplectic incidents and who had infarctions in their placentas and delivered hypotrophic babies it is necessary to take into account the existence of the factor V Leiden mutation that predisposes those patients to embolia, fetal-placental circulation disorders (recurrent placental ischemia, infarctions and failure) [15, 16].

Karowicz-Bilińska et al. [17] drew a conclusion that, apart from the decrease in the blood exchange between a fetus and a mother in case of pregnancies complicated by fetal IUGR, the reason for such state is on the one hand lower hampering of the apoptosis process in the trophoblast (than the one in a normal pregnancy), and on the other higher expression of proapoptotic proteins.

Thorough evaluation of a placenta (morphological examination, assessment of the fetal-placental circulation and metabolism) indicates fetuses who are endangered with IUGR and who should be subject to much more detailed perinatal care. It seems that a routine examination of a placenta after birth may help to explain the pathogenesis of intrauterine growth restriction in fetuses in case of a pregnant woman with normal blood pressure.

Prevention and treatment of hypotrophy occurring in fetuses and neonates has much sense, since those children are in their later life more prone to development of metabolic disorders such as obesity, hypertension, hypercholesterolemia, circulation system diseases, diabetes type 2 and hiperandrogenism in girls [5].

Conclusions

1) Pathological changes in placentas of the births of intrauterine growth restricted neonates were ob-
served significantly more often than in placentas of the births of eutrophic neonates.

2) Placentas of mothers who delivered IUGR neonates were characterized by significantly lower weight and surface, significantly higher weight ratio and greater occurrence of anatomical changes.

References


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