NEONATAL ASPECTS REGARDING NEWBORNS RESULTED FROM MOTHERS WITH PREGNANCY-INDUCED HYPERTENSION

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Abstract

Introduction The paper addresses from the neonatologist clinician's perspective to a category of high risk newborns, affected by pregnancy-induced hypertension, a pathological entity particular only in gestational period. It is different from pre-existing hypertension before pregnancy and disappears after childbirth and delivery of the placenta.

Material and method Based on rigorous inclusion and exclusion criteria, were established two equal groups of newborns (n=116): a study group including newborns of mothers with pregnancy-induced hypertension and a study group including newborns whose mothers have not pregnancy-induced hypertension, randomized case-control pattern. There were analyzed for comparison a number of 18 parameters, focused on their postnatal development issues. For their registration it was used the database from Neonatology Clinic "Bega" Timisoara. For statistical data processing we used SPSS 17.0.

Results Significant results were considered those who presented major differences in adaptation and neonatal pathology, the group study being affected. For a confidence interval of 95%, we recorded $p < 0,05$ (significant differences), for next parameters: low birth weight, the degree of immaturity, early neonatal adapting difficulties, the need of neonatal reanimation and mechanical ventilation in the first 24 hours of life and the incidence of neonatal hypoxic pathology.

Conclusions By reducing placental blood flow, pregnancy-induced hypertension significantly affect fetal nutrition and oxygenation with whole metabolic consequences. This is more important when the onset is in early pregnancy and the duration of fetal suffering is more prolonged. This category is represented by neonates of mothers who developed pregnancy-induced hypertension.

Keywords: newborn, pregnancy-induced hypertension

Introduction

Pregnancy-induced hypertension, with all its forms and complications represents *a crossroad* where there are meeting concerns of the obstetrician, cardiologist, neonatologist, and not least the pathologist because the disease's morphological substrate is given by the placental vascular lesions, or more precisely by *I.M.F lesion with damage of the placental homeostatic unity.* The strongest argument of the morphological substrate is that *the disease occurs only in pregnant women, so in the presence of the placenta and disappears after childbirth and delivery of the placenta.* We are talking about pregnancy-induced hypertension only in pregnant women who did not have hypertension before pregnancy [1, 2].

As a matter of the human species pathology, *the disease does not benefit of models, research and experimental results.*

Major fetal consequences of the disease are dependent on the age of onset, intensity and duration of aggression and therapeutic control of the disease. There are two major fetal consequences [3, 4, 5]:

- *Chronic fetal hypoxia*, resulting in deterioration of gas exchange in the maternal-fetal interface;
- *Fetal malnutrition* as a result of maternal-fetal alteration of food intake at the same level with the exchange surface.

Pregnancy-induced hypertension has a significantly influence on the fetal prognosis and can lead to various degrees of fetal distress or even death of the fetus in the womb. Equally signifiant affects the disease the prognosis of premature newborns, intrauterine growth restriction (limitation), early neonatal adapting difficulties or severe neonatal pathology with sustainable neurological sequelae (cerebral palsy) [6, 7, 8].

Materials and methods

The study was conducted at the Clinic of Obstetrics-Gynecology and Neonatology of the Emergency County Hospital Timisoara. To achieve the study group there have been used criteria to answer the study objectives in the clinical-research stage and criteria to allow the selection of the casework required for pathological research of the placenta.

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Inclusion criteria: **g**estational age ≥ 28 weeks, birth weight ≥ 1000 g, mother with pregnancy-induced hypertension without complications, single pregnancy, neonatal favorable evolution, with discharge to home.

Exclusion criteria: GA <28 weeks, BW<1000 g, twins, death in the neonatal period, serious associated pathology (infection, congenital malformations, perinatal asphyxia), mother with complications of pregnancy-induced hypertension.

The study group included 116 infants of mothers with pregnancy-induced hypertension over a period of 4 years (2008-2011).

Compared to the total number of births during the study (n = 9664) the incidence of pregnancy-induced hypertension was 1.26%.

For comparison was formed a group of newborns whose mothers have not pregnancy-induced hypertension *(randomized case - control pattern).*

There were analyzed for comparison a number of 18 items,which focused on physical and maturity parameters of the newborn as well as adaptation issues and their neonatal hypoxic pathology.

Equal groups were formed, the database is built in an Excel file by sequential entering of the following documents data (observation sheet) of each patient.

The statistical study of the data was performed using SPSS 17.0.

The parameters taken in the study were:

- Sex of newborns
- Gestational age at birth (weeks of amenorrhea)
- Birth weight (g)
- The size at birth (cm)
- Head circumference at birth (cm)
- Pathological labor
- Pathological presentation
- The way of birth (naturally or by caesarean section)
- 1-minute Apgar score
- The need for reanimation and intensive care at birth
- Mechanical ventilation in the first 24 hours of life
- Duration of hospitalization (days)
- Initial Nutrition
- Hypoxic neonatal pathology (Sarnat classification).

Results and discussion

The parameters considered in the study were analyzed comparatively parameter by parameter between the study group and the control group. Significant differences were registered in terms of physical parameters at birth between the study group and control group.

For the birth weight (Fig.1) there has been a highly significant difference between the cases of the study group and the cases of the control group.Regarding the degree of maturity there has been a significant difference between the study group and the control group in the incidence of prematurity (Fig. 2). Most cases presented, along with the immaturities caused by prematurity and the fetal hypoxic suffering more or less prolonged, also intrauterine growth restriction from moderate to severe.

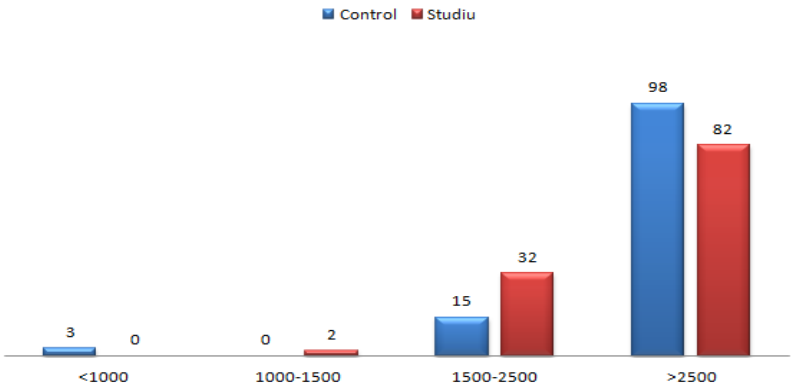

Fig.1. Distribution of cases according to birth weight (g).

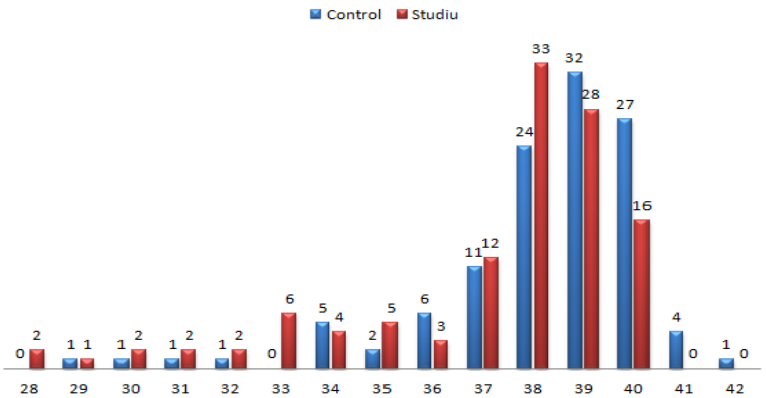

Figure 2. Distribution of cases according to gestational age (weeks of amenorrhea).

The data presented and illustrated record a major impact of hypertension induced by pregnancy on the fetal growth and development, with considerable damage in fetal weight with a mean difference of this parameter above 300 grams between the two groups. Also, the cases with early onset disease and poor outcome of the disease imposed challenge of premature birth in order to remove the fetus from adverse intrauterine environment and prevent fetal death in utero.

The degree of chronic suffering with intrauterine growth restriction and preterm birth significantly more frequent in the study group influenced the other parameters such as neonatal adapting (Apgar score), the need to reanimate at birth and the need for mechanical ventilation in the first 24 hours of life.

The clinical data have been focused on neonatal hypoxic pathologies, whose incidence was significantly higher in the cases of the study group vs the control group (Fig. 3).

The diagnosis of neonatal hypoxic suffering was based on clinical data included in the *quoted Sarnat* (Table 1), on the neurological exam of the newborn and its future evolution. On this basis they could classify the neonates with hypoxic events in mild, moderate and severe, observing *the tenth revision of the International Classification of Diseases WHO.*

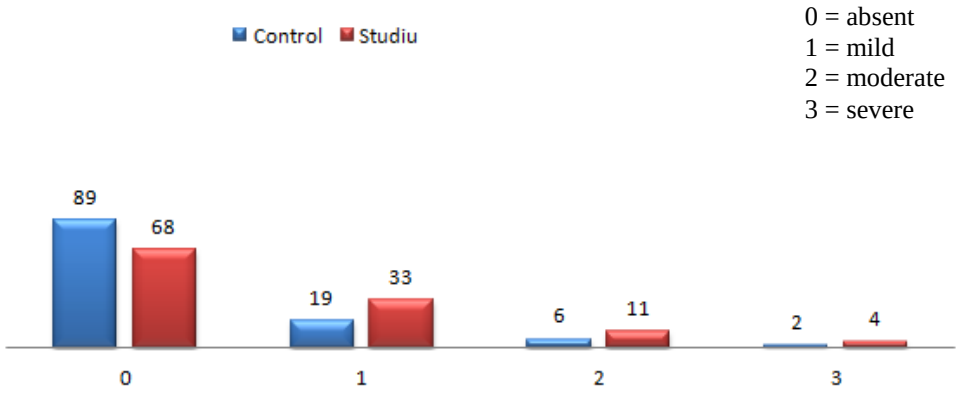

Fig.3. Distribution of cases according to the incidence of hypoxic pathology in the neonatal period.

Table 1. Sarnat Score Sheet.

Variable	**Stage I**	**Stage II**	**Stage III**
Level of Consciousness	Alert	Lethargic	Coma
Muscle Tonus	Normal/hipertonic	Hipotonic	Flaccid
Reflexes	Increase	Increase	Descreased/absent
Myoclonus	Present	Present	Absent
Seizures	Absent	Frequent	Frequent
Complex Reflexes			
• Suck	Activ	Weak	Absent
• Moro (startle)	Exaggerated	Incomplete	Absent
• Grab	Normal/Exaggerated	Exaggerated	Absent
• Tonic Neck	Normal	Overactiv	Reduced/absent
Automatic Function			
• Pupils	Mydriasis, reactive	Miosis, reactive	Variable/fixed
• Respirations	Regular	Variable in number and depth, periodic	Ataxic, apneic
• Heart Rate	Normal/Tahicardic	Bradicardic	Bradicardic

The other parameters studied were not significantly different between the study group vs the cases in the control group.

In Tables 2 and 3 are shown in summary parameters significantly different from the study group and control group, that can be correlated with the impact of pregnancy-induced hypertension on the placental and fetal complex in the prenatal period.

Pregnancy-induced hypertension is defined as an increase of blood pressure over the values of 140/90 mmHg, or more precisely an increase of 30 mmHg systolic or 15 mmHg diastolic blood pressure over the core values of the

pregnant woman. Correct definition is necessary because the disease is often called preeclampsia [9,10]. Preeclampsia is a pregnancy-induced hypertension very early onset (after 20 weeks of gestation) with proteinuria or edema. It is widely accepted that early onset pregnancy-induced hypertension has a major impact on maternal-fetal circulation unknown or uncontrolled can develop into complications. The disease can develop into two major complications [10, 11, 12]:

- Eclampsia – which involves seizures that are not related to a neurological condition, to a pregnant woman which meets the criteria of preeclampsia.
- HELLP syndrome associating three biological disorder: Hemolysis, Elevated Liver enzymes, and Low Platelets.

Reduced placental circulation and blood flow is the main cause maternal-fetal nutrient supply reduction. If onset occurs early in the fetal period (after 20-22 weeks of gestation) and duration (with or without treatment) is prolonged, fetal harm will be severe and often requires its removal by caesarean section, even if it is extreme immaturity. Along with severe malnutrition, in this case the fetus is affected by hypoxemia / severe hypoxia with risk of fetal death. If onset is late (after 28-30 weeks of gestation), fetal maturity is better and hence the tolerance to hypoxia and malnutrition [13, 14, 15].

When the fetus stops growing, in general, it also starts to unfold the impact of fetal hypoxia, but this time is difficult to diagnose. For these reasons a pregnancy complicated with pregnancy-induced hypertension requires frequent monitoring to capture the moment [14,15].

Table 2 Distribution of the average and standard deviations for the parameters studied in the study group vs the control group.

PIH

	Parameters	S. G.		C. G.		Comments
		M	DS	M	DS	
1	GA (weeks)	37,22	2,77	38,20	2,18	P = 0,0032
2	BW (g)	2829,57	571,57	3102,72	700,46	P = 0,0011
3	Length at birth(cm)	49,03	3,39	50,41	3,43	P = 0,0024
4	CP at birth (cm)	32,73	1,91	33,42	2,10	P = 0,0095
5	APGAR score at 1 minute	8,49	1,31	9,00	1,27	P = 0,0030
6	Length of hospital stay(days)	5,68	3,73	5,17	3,10	P = 0,260

Table 3. The numerical and the percentage distribution of the parameters studied in the study group vs in the control group.

PIH

	Parameters	S. G.		C. G.		Comments
		Nr	%	Nr	%	
1	Resuscitation at birth and TIN	21	18,10	13	11,20	P = 0,194
2	Mechanical ventilation in the first 24 hours	15	12,93	3	2,58	P = 0,0069
3	Hypoxic neonatal pathology (Sarnat criteria)	48	41,37	19	16,37	P = 0,041

Conclusions

1. Pregnancy-induced hypertension is a pathological condition affecting the morbidity, mortality and the newborn outcome. In relation to the total number of births during the study (n = 7654), the incidence of the disease was 1.76%.
2. The study conducted on a group of infants of mothers with pregnancy-induced hypertension (n = 116 cases) compared with a equal group of newborns coming from mothers without pregnancy-induced hypertension has recorded the following significant issues:
   - a significant difference in growth parameters (W, L, CP) and the degree of immaturity among the study group vs the control group;
   -an average weight difference of over 300 g between the 2 groups were significantly due to intrauterine growth restriction in the study group, regardless of gestational age;
3. a significantly higher incidence of the need for neonatal intensive care measures and their continuing in the neonatal intensive care unit in the cases of the study group; the hypoxic neonatal pathology and the the respiratory pathology was determined, and the incidence of cases of the study group was highly significant compared to the control group.The degree of fetal immaturity and / or intrauterine growth restriction vs. "the necessity" of pregnancy ending remains one of the most controversial issues of

perinatal medicine: a good obstetrician-neonatologist cooperation is the key to choosing the best solution for "fetal welfare."

4. Improving the fetal prognosis can be achieved only through a careful maternal and fetal monitoring individualized treatment in hospital conditions and allows the optimal timing of birth and birth path.
5. In addition to these research methods the substrate generating perinatal fetal distress, I believe that taking and researching the placenta can provide the key to elucidate this problem and plausible explanations for a possible adverse outcome of these cases.

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