Impact of Maternal Disease States on Blood Pressure Changes in Early Neonatal Period

Thesis
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Abstract

Maternal- or pregnancy-associated disease states appear to influence BP in the early neonatal period. Diabetes and altered placental perfusion were associated with higher BP readings**. (Kent Al et al,2009)**

Placental vascular changes associated with maternal disease states may affect fetal vascular development. There is evidence suggesting that being born prematurely is associated with a higher blood pressure (BP) in later life. **(Kent AL and Meskell S et al,2009)**

A study of one hundred and fourty-five placentae was done to find out the morbid and histological changes of placentae of hypertensive mothers and diabetic mothers in comparison to those of mothers with uncomplicated pregnancies. As placenta is the mirror of maternal and foetal status, it reflects the changes due to maternal hypertension or diabetes.

This study was carried out on fifty mothers with uncomplicated pregnancy and fourty-five mothers with pregnancy induced hypertension (PIH) and fourty-two mothers with gestational diabetes and eight mothers with combined hypertension and diabetes.

It was found that mothers with moderate to severe PIH had smaller, irregular placentae with marginal insertion of umbilical cord with deviation in respect of foci of calcification, infarction and histological features of vascular insufficiency like thrombosis, infarction etc. Histological findings like cytotrophoblastic cellular proliferation, syncytial knot formation, fibrin plaque formation etc. were present in greater amount in hypertensive placentae. The changes in the placentae may be the cause / effect or both of hypertension in pregnancy of mothers who were normotensive.

Our study is a prospective cohort review. To observe vascular changes, placentae were collected just after delivery. Macroscopic findings of the study was that there was a trend of increase weight, volume and number of cotyledons in overt diabetes group, where as the in the hypertensive diabetes group was the opposite. The placental diameter was non-significantly larger in the overt diabetes group than in the control group, but a smaller than control value was found in the hypertensive diabetes group. Although neonatal weight did not show any significant change in either group, but in overt diabetes group, it showed a significant positive correlation with placental weight.

Conclusion: Maternal- or pregnancy-associated disease states appear to influence BP in the early neonatal period. Diabetes and altered placental perfusion were associated with higher BP readings.

Keywords: maternal disease states, neonatal hypertension, placenta.

بسم الله الرحمن الرحيم

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عنوان الرسالة :

تأثيرالحالة المرضية للأم أثناء الحمل على ضغط الدم للأطفال حديثى الولاده

الملخص :

يعتمد النمو و التطور الطبيعي للجنين علي الوظائف الحيـوية للمشيمة؛

الحــالـة المـرضية للأم اثناء الحمل وجد أنها تؤثر علي ضغط الدم للطفل حديث الولادة.

ارتفاع نسبة السكر في دم الأم الحامل و اصابتها بأمراض الضغط المرتفع تؤدي الي تغيير امداد الجنين بالدم في المشيمة مما يؤدي الي ارتفاع ضغط دم الطفل حديث الولادة.

يتعرض الطفل حديث الولادة الي تغييرات كثيرة و جزرية في الدورة الدموية عقب الولادة.

فحص المشيمة يمكن ان تعطي فكرة عن الأمراض التي تصيب الأم اثناء الحمل مثل ارتفاع ضغط الدم للأم الحامل و السكر .

وترى اللجنه قبول البحث.

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List of Abbreviations

RH	Retroplacental Hematoma
BP	Blood Pressure
HIF	Hypoxia Inducible Factor
BPD	Broncho Pulmonary Dysplasia
NK	Natural Killer cells
CoA	Coarctation Of the thoracic Aorta
CT	Computed Tomography
CAH	Congenital Adrenal Hyperplasia
CHF	Congestive Heart Failure
PIH	Pregnancy-Induced Hypertension
VUE	Villitis of Unknown Etiology
IUGR	Intra-Uterine Growth Retardation
FMD	Fibromuscular Dysplasia
TNF	Tumor Necrosis Factor
GDM	Gestational Diabetes Mellitus
GLUT	Glucose Transporter
CNS	Central Nervous System
ICU	Intensive Care Unit
Na	sodium
LVH	Left Ventricular Hypertrophy
MRA	Magnetic Resonance Angiography
MRI	Magnetic Resonance Imaging
MAP	Mean Arterial blood Pressure
NICU	Neonatal Intensive Care Unit
PDA	Patent Ductus Arteriosus

PRA	Plasma Renin Activity
PKD	Poly cystic Kidney Disease
PMN	Polymorphonuclear
RVT	Renal Vein Thrombosis
RAS	Renin-Angiotensin System
VSM	Vasculosyncytial Membrane
IL	Interleukin
CO	Cardiac Output
TPN	Total Parenteral Nutrition
US	Ultrasonography
VSD	Ventricular Septal Defect
LBW	Low Birth Weight
PR	Peripheral Resistance
TORCH	Toxoplasma, Rubella,C ytomegalovirus, Herpes simplex

Introduction

Advances in the ability to identify, evaluate, and care for hypertensive infants, coupled with advances in the practice of neonatology in general, have led to an increase awareness of hypertension in modern neonatal intensive care units (NICUs**) *(Flynn, 2006)*.**

Hypertension is not commonly diagnosed in newborn infants. The incidence in infants discharged from neonatal units ranges from 0.7% to 2%.Hypertension is defined by a systolic blood pressure in a neonate which is >95 percentile for age and sex in 3 separate occasions for infants of similar gestational or post conceptual age and size***(Watkinson, 2002)*.**

Hypertension in newborn infants primarily is of renal origin, although cardiac, endocrine, and pulmonary causes have been described as well**.** The gold standard for blood pressure measurement is an appropriately calibrated intra-arterial catheter. However, for babies who do not have or require invasive monitoring the most frequently used technique is via an oscillometric manometer (e.g. Dinamap). Blood pressure should be taken when babies are quiet and not feeding (systolic BP is 5 mmHg lower in sleeping babies) with appropriate sized cuff ***(Flynn, 2006).***

Placenta is the most accurate record of the infants' prenatal experience.After delivery if the placenta is examined minutely it provides much insight into the prenatal health of the baby and the mother. Pregnancy complications like hypertension or gestational diabetes are reflected in the placenta in a significant way (both macroscopically and microscopically). It has been recorded that the maternal utero-placental blood flow is decreased in pre-eclampsia, ***Bowen et al, (2002)*** because there is maternal vasospasm. Reduced maternal utero-placental blood flow leading indirectly to constriction of fetal stem arteries ***Stock et al,***

(1999) has been associated with the changes seen in the placentae of pre-eclamptic women. Maternal vasospasm leads to fetal hypoxia. Fetal hypoxia is not uncommon near term and accordingly it may lead to fetal distress and fetal death. In recent years, it has been revealed that there is a clear relationship between confined placental mosaicism and fetal growth retardation. 70% of the excess fetal deaths in women with hypertension are due to large placental infarcts, markedly small placental size and that histopathological changes related to confined placental mosaicism may be associated with inadequate placentation and hence with retroplacental ischaemia ***Fox, (2000).***

Aim of Work

The aim of this study is to determine the effect of maternal disease states on BP in the early neonatal period and to correlate its effect on placental pathology and neonatal blood pressure.

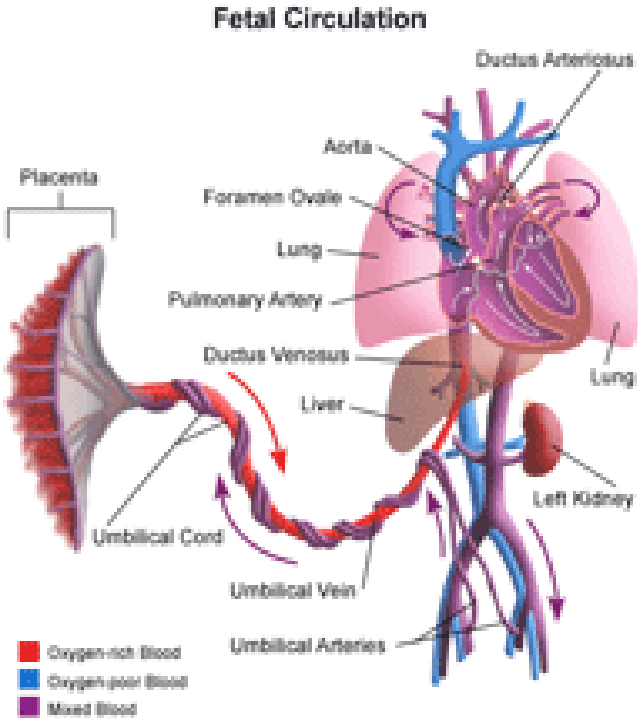
REVIEW OF LITERATURE

CHAPTER 1

PHYSIOLOGY OF NEONATAL CIRCULATION

Physiology of neonatal circulation

1. The fetal Circulation:



In the adult circulation the right heart, the lungs, left heart and systemic circulation are arranged in series. Blood flow through each of these elements is identical, and equals cardiac output. In the fetal circulation, the right and left ventricles each pump blood into the arterial circulation in parallel. In the fetus, the placenta provides for gas and metabolic exchange. The lungs do not provide gas exchange, and vessels in the pulmonary circulation are vasoconstricted. Three cardiovascular structures unique to the fetus are important for maintaining this parallel circulation: the ductus venosus, foramen ovale,and ductus arteriosus. ***(Sousa, 2008)***

The total fetal cardiac output (the combined ventricular output of both the left and right ventricles) amounts to about 450mL/kg/min. Approximately 65% of

descending aortic blood flow returns to the placenta; the remaining 35% perfuse the fetal organs and tissues in the human fetus, which has a larger percentage of blood flow going to the brain. Right ventricular output is probably closer to 1.3 times left ventricular flow. Thus, during fetal life the right ventricle is not only pumping against systemic blood pressure but is also performing a greater volume of work than the left ventricle is ***(Judy, 2001).***

In preterm, fetal combined ventricular output is greater (Approximately 550 ml/kg/min) ***(Hegyi et al., 2004).***

2. The Transitional Circulation:

At birth, mechanical expansion of the lungs and an increase in arterial Po2 result in a rapid decrease in pulmonary vascular resistance. Concomitantly, removal of the low-resistance placental circulation leads to an increase in systemic vascular resistance. The output from the right ventricle now flows entirely into the pulmonary circulation, and because pulmonary vascular resistance becomes lower than systemic vascular resistance, the shunt through the ductus arteriosus reverses and becomes left to right. Over the course of several days, the high arterial Po2 constricts the ductus arteriosus and it closes, eventually becoming the ligamentum arteriosum. The increased volume of pulmonary blood flow returning to the left atrium increases left arterial volume and pressure sufficiently to close the foramen ovale functionally, although the foraman may remain probe patent. Removal of the placenta from the circulation also results in closure of the ductus venosus. The left ventricle is now coupled to the high-resistance systemic circulation, and its wall thickness and mass decrease slightly. The left ventricle, which in the fetus pumped blood only to the upper part of the body and brain, must now deliver the entire systemic cardiac output(approximately 350mL/kg/min),an almost 200% increase in