Effects of preeclampsia on the mother, fetus and child

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High blood pressure complicates almost 10 percent of all pregnancies, and the incidence is higher if the women are nulliparous or carrying multiple fetuses.

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High blood pressure complicates almost 10 percent of all pregnancies, and the incidence is higher if the women are nulliparous or carrying multiple fetuses.

Preeclampsia is a major cause of maternal mortality in developed and developing countries. It is also a major cause of perinatal morbidity and mortality, and it is very strongly associated with fetal growth retardation.

Maternal impact

Women who have or develop high blood pressure during pregnancy are all at increased risk of complications antenatally, intrapartum and in the puerperium. The increased risk applies to the mother as well to the fetus. Pregnant women with hypertension can be divided into two groups: normotensive women who develop the preeclamptic syndrome, which is characterised by hypertension, proteinuria, and edema; and women with chronic hypertension who become pregnant and are at a higher risk of developing superimposed preeclampsia. The impact of preeclampsia affects both mother and fetus, but it is important to differentiate between the complications of the disease from those inevitably associated to the drugs used for its treatment.

Preeclampsia is the most serious form of hypertensive pregnancy complications, but it is not primarily a hypertensive disease; it is a disorder induced by factors based on the presence of placenta. Preeclampsia is initiated by abnormal placentation and, therefore, a low perfunded placenta, release of cytokines and other toxins, and vasoconstriction and platelet activation; so it is a syndrome of generalized endothelial dysfunction, and the complications are associated with the vascular system. Fundamentally, these complications are 1- intravascular coagulation, bleeding and 2- organ failure (hepatic and renal) following poor perfusion.

1- There is a direct relationship between the decreased antithrombin III (ATIII) levels and the severity of the patient's clinical condition, especially after gestational weeks 30-32.

Blood volume, by measurement of red blood cell and plasma volumes, is reduced and has altered distribution in preeclampsia.

2- In hypertensive pregnancy, there is still controversy over the levels of proteinuria that should be considered pathological.

As we have observed, preeclampsia has quite an impact on renal function (Table 1). Preeclampsia may be complicated by seizures: eclampsia. The greatest compromise occurs with the development of the HELLP syndrome (hemolysis, elevated liver enzymes and low platelet count). The HELLP syndrome, alongside preeclampsia, accounts for most maternal deaths associated with hypertension (Table 2).

The process is completely reversed by the delivery of the fetus and placenta, but intrauterine growth retardation and premature delivery pose major threats to the fetus and may require care in a tertiary care center. Treatment of preexisting or pregnancy-induced hypertension does not prevent or reverse the process, but is justified to prevent maternal cardiovascular complications, especially during labor and delivery. The fetus is at increased risk due to growth retardation and hypoxia following placental damage.

The majority of patients with mild chronic hypertension have successful pregnancy outcomes. Most perinatal morbidity is secondary to superimposed preeclampsia. Antihypertensive therapy does not appear to significantly affect pregnancy outcome, or the incidence of superimposed preeclampsia in mild chronic hypertensives. Maternal and fetal risks are considerably higher in severe chronic hypertension and in those patients with target organ disease. These patients should ideally be counseled regarding their risks prior to pregnancy.

Fetal impact
Perinatal outcome is strongly influenced by gestational age and the severity of hypertension as expressed by the need for antihypertensive treatment, irrespective of the underlying syndrome. Severe preeclampsia is associated with different degrees of fetal injury. The main impact on the fetus is undernutrition as a result of utero-placental vascular insufficiency, which leads to growth retardation.

There are short and long-term effects. The immediate impact observed is altered fetal growth resulting in greater fetal liability. Fetal health as well as its weight are highly compromised, leading to various degrees of fetal morbidity, and fetal damage may be such as to cause fetal death. Long-term follow up studies have demonstrated that babies who suffered intrauterine growth retardation are more likely to develop hypertension, coronary artery disease, and diabetes in adult life. There is growing evidence to suggest that patterns of early growth and other life course factors play an important role in the origins and development of cardiovascular disease (CVD), but understanding the processes which mediate these effects is limited.

Unusual perinatal complications involving anoxia or catecholamine release in the mother, fetus, or newborn may predispose the baby to the development of precocious coronary atherosclerosis later in life.

Many fetuses have to adapt to a limited supply of nutrients. In doing so, they permanently change their structure and metabolism. These 'programmed' changes may be the origin of a number of diseases in later life, including coronary heart disease and related disorders: stroke, diabetes and hypertension.

Babies who are small or disproportionate at birth, or who have altered placental growth are now known to have increased rates of coronary heart disease, hypertension and non-insulin-dependent diabetes in adult life. These associations are thought to result from fetal 'programming', whereby a stimulus or insult at a critical, sensitive period of early life has permanent effects on the body's structure, physiology and metabolism. Small size at birth and disproportion in head size, length and weight appear to be surrogate markers for the actual influences that programme the fetus. These observations have prompted a re-evaluation of the maternal regulation of human fetal development. Recent studies suggest that the fetus may be considerably more sensitive to the materno-placental supply of nutrients than previously imagined. Adult cardiovascular disease may be a consequence of fetal adaptations invoked when the materno-placental nutrient supply fails to match the fetal nutrient demand.

It has been demonstrated that intrauterine growth retardation, defined as birth weight below the 10th percentile, gives rise to a reduction in nephron number. Oligonephropathy has been suggested to increase the risk for systemic and glomerular hypertension in adult life as well as enhance risk for expression of renal disease after exposure to potentially injurious renal stimuli.

With the present available evidence there is a need to address the key issues of possible confounding factors of perinatal and early life and those in later life in relation to CVD risk. It is also necessary to replicate studies and establish new ones by assembling cohorts where indicators of prenatal and postnatal growth have been previously recorded from different populations living under different conditions.

**Table Effect of hypertension in pregnancy on uric acid**

<table>
<thead>
<tr>
<th>Classification</th>
<th>&lt; 28 wks. Uric acid</th>
<th>&gt; 28 . Uric acid</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mg% x ± sd</td>
<td>mg% x ± sd</td>
</tr>
<tr>
<td>Gestational Hypertension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A1</td>
<td>3.63 ± 0.93</td>
<td>4.52 ± 1.41</td>
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<tr>
<td>Preeclampsia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A3</td>
<td>4.44 ± 1.55</td>
<td>5.45 ± 1.70 *</td>
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<tr>
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</tr>
<tr>
<td>B1</td>
<td>3.48 ± 1.10</td>
<td>4.21 ± 1.29</td>
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<tr>
<td>Preexisting Hypertension + Superimposed</td>
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<tr>
<td>Preeclampsia</td>
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<td>5.68 ± 1.85 *</td>
</tr>
<tr>
<td>B3</td>
<td></td>
<td></td>
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<tr>
<td>Non Hypertensives</td>
<td>3.61 ± 1.19</td>
<td>4.33 ± 1.35</td>
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</table>
*A3 and B3 vs NH: p < 0.05

References:

REFERENCES

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