Fertility in Women in End-Stage Renal Disease

**DIALYSIS AND PREGNANCY**

Successful outcome, 20-30%
High incidence of prematurity
Outcome related to residual maternal renal function
Management:
- Increased hours on dialysis
- Erythropoietin therapy
- Blood pressure control
- Therapy with low doses of heparin
- Continuous ambulatory peritoneal dialysis versus hemodialysis?

**FIGURE 10-18**

Because fertility is decreased in end-stage renal disease, pregnancy is uncommon in women on chronic dialysis. When pregnancies occur, however, only about 20% to 30% are successful, with the chances of success increasing when residual renal function exists [20]. The overall strategy should be to maintain blood chemistry levels as close as possible to normal by increasing the number of hours of dialysis to 20 or more. Erythropoietin may be used in pregnancy. Blood pressure control is important, and low doses of heparin should be used to prevent bleeding. There are no apparent advantages of chronic ambulatory peritoneal dialysis compared with hemodialysis. The incidence of worsening maternal hypertension and subsequent premature delivery is high.

Fertility and Renal Transplantation

**RENAL TRANSPLANTATION AND PREGNANCY**

Prognosis depends on blood pressure and baseline renal function
- (<1.5–2 mg/dL; normal blood pressure)
- Controversy over whether pregnancy accelerates graft loss
- Patients are advised to wait 2 y after transplantation before pregnancy

**FIGURE 10-19**

Fertility is restored after successful renal transplantation. Pregnancy outcome is improved if renal function is normal and hypertension is absent. It is advisable to wait 2 years after transplantation before pregnancy so that renal function is stable and doses of immunosuppressants are lowest [21]. Cyclosporine, prednisone, and azathioprine are safe during pregnancy and are not associated with fetal abnormalities. Limited experience exists with mycophenolate mofetil during pregnancy.

Hypertensive Disorders in Pregnancy

**FIGURE 10-20**

Mortality and hypertension. Worldwide, hypertensive disorders are a major cause of maternal mortality, accounting for almost 20% of maternal deaths. Most deaths occur in women with eclampsia and severe hypertension (HTN) and are due to intracerebral hemorrhage [22].

Developing nations
- Hemorrhage 20%
- Septis 8%
- Other 25%

HTN 15%

100,000 (deaths, births)

Developed nations
- Septis 17%
- Embolism 17%
- Other 25%

Hemorrhage 13%

12,100,000 (deaths, births)
### Fetal Consequences of Maternal Hypertension During Pregnancy

<table>
<thead>
<tr>
<th>Condition</th>
<th>Incidence</th>
</tr>
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<tbody>
<tr>
<td>3- to 6-fold increase in stillbirths</td>
<td></td>
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<tr>
<td>5- to 15-fold increase in intrauterine growth restriction</td>
<td></td>
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<tr>
<td>Premature delivery</td>
<td></td>
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<tr>
<td>Long-term developmental and neurologic problems</td>
<td></td>
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</tbody>
</table>

### Classification of Hypertensive Disorders in Pregnancy

- Preeclampsia, eclampsia
- Chronic hypertension
- Chronic hypertension with superimposed preeclampsia
- Transient hypertension

### Clinical Features of Preeclampsia

**Historical:**
- Nulliparity
- Multiple gestations
- Family history
- Preexisting renal or vascular disease

**Hypertension:**
- 140/90 mm Hg after 20 wk or
- 30 mm Hg increase in systolic pressure or
- 15 mm Hg increase in diastolic pressure

**Sudden appearance of edema,**
- especially in hands and face

**Rapid weight gain**

**Headache, visual disturbances,**
- abdominal or chest pain

### Clinical Features of Chronic Hypertension in Pregnancy

- Women are older, more likely to be multiparous
- Hypertension: present before 20 wk, or documented previous pregnancy
- Blood pressure may be significantly lower or normal in mid pregnancy
- Risk of superimposed preeclampsia of 15-30%

### Clinical Features of Chronic Hypertension in Pregnancy

- Women with chronic hypertension are usually older and may be multiparous. Although hypertension is detectable before 20 weeks, in some women the pregnancy-mediated vasodilation is sufficient to normalize blood pressure so that women with stage 1 or 2 hypertension may have normal blood pressures by the time of their first antepartum visit. The risk of preeclampsia is substantially increased in women with chronic hypertension.
**LABORATORY ABNORMALITIES IN PREECLAMPSIA AND CHRONIC HYPERTENSION**

<table>
<thead>
<tr>
<th></th>
<th>Chronic hypertension</th>
<th>Preeclampsia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Renal:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Creatinine</td>
<td>Normal</td>
<td>Increased; increased blood urea nitrogen, creatinine</td>
</tr>
<tr>
<td>Uric acid</td>
<td>Normal</td>
<td>Increased (&gt;55 mg/dL)</td>
</tr>
<tr>
<td>Urinary protein</td>
<td>&lt;300 mg/d</td>
<td>&gt;300 mg/d</td>
</tr>
<tr>
<td>Urinary calcium</td>
<td>&gt;200 mg/d</td>
<td>&lt;150 mg/d</td>
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<tr>
<td>Heme:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hematocrit</td>
<td>Normal</td>
<td>Increased (&gt;38%)</td>
</tr>
<tr>
<td>Platelets</td>
<td>Normal</td>
<td>Decreased</td>
</tr>
<tr>
<td>Liver function tests:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspartate aminotransferase</td>
<td>Normal</td>
<td>Increased</td>
</tr>
<tr>
<td>Alanine aminotransferase</td>
<td>Normal</td>
<td>Increased</td>
</tr>
<tr>
<td>Albumin</td>
<td>Normal</td>
<td>Decreased</td>
</tr>
</tbody>
</table>

**FIGURE 10-25**

Laboratory tests are helpful in making the diagnosis of preeclampsia. In addition to proteinuria, which may occur late in the course of the disease, hyperuricemia, mild azotemia, hemoconcentration, and hypocalciuria are observed commonly. Some women with preeclampsia may develop a microangiopathic syndrome with hemolysis, elevated liver enzymes, and low platelet counts (HELLP). The presence of the HELLP syndrome usually reflects severe disease and is considered an indication for delivery. Women with uncomplicated chronic hypertension have normal laboratory test results unless superimposed preeclampsia or underlying renal disease exists.

**FIGURE 10-26**

Preeclampsia is a syndrome with both maternal and fetal manifestations. Current evidence suggests that an underlying genetic predisposition leads to abnormalities in placental adaptation to the maternal spiral arteries that supply blood to the developing fetoplacental unit. These abnormalities in the maternal spiral arteries lead to inadequate perfusion of the placenta and may be the earliest changes responsible for the maternal disease. The maternal disease is characterized by widespread vascular endothelial cell dysfunction, resulting in vasospasm and intravascular coagulation and, ultimately, in hypertension (HTN), renal, hepatic, and central nervous system (CNS) abnormalities. The fetal syndrome is a consequence of inadequate placental circulation and is characterized by growth restriction and, rarely, demise. Premature delivery may occur in an attempt to ameliorate the maternal condition. IUD — intrauterine death; IUGR — intrauterine growth retardation.