Effect of Immediate Postpartum Curettage on the Recovery of Severe Preeclampsia

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Abstract

Objective: To evaluate the effect of immediate postpartum curettage upon maternal recovery from severe Preeclampsia.

Methods: Sixty parturient women with severe Preeclampsia were randomly assigned to either undergo ultrasound directed curettage following delivery (n=30) or to have no curettage (controls=30).

Results: Subjects who underwent immediate postpartum curettage had a significant decrease in mean arterial pressure at 16 hours post delivery (p<0.0002). The mean urine output after uterine curettage was significantly greater after the first 12 hours post delivery compared to controls (p<0.0002). Platelet count increased in the curettage group from 12 to 24 hours following curettage, while controls exhibited a decrease in the platelet count from 12 to 24 hours (p<0.02).

Conclusion: Immediate puerperal uterine curettage of the parturient with severe Preeclampsia appears to accelerate disease recovery with no adverse sequelae.

Introduction

Preeclampsia is considered as an endothelial cell disorder observed only in the pregnant human female(1). Defined classically as the development of hypertension, proteinuria and/or edema in the second half of in a woman who has previously been normotensive and otherwise clinically well (2). It occurs in 7% of all pregnancies and is associated with increased maternal and perinatal morbidity and mortality (3).

There are many maternal complications of Preeclampsia such as abruptio placenta, thrombocytopenia, hepatic hemorrhage and rupture, eclampsia, disseminated intravascular coagulation, stroke, intracerebral hemorrhage, acute renal failure, and the syndrome of hemolysis, elevated liver enzymes and low platelet count. (HELLP).(4)

Accelerated recovery from this disease process following delivery could avert these complications and shorten the time required for intensive care and hospitalization.

Subjects and Methods

The subjects of this study were 60 women who presented to labor and delivery department, Al-Hussein University Hospital, with physical findings consistent with severe Preeclampsia.

Severe Preeclampsia was defined as combinations of the following:
1-Blood pressure at least 160/110mmHg on two occasions at least 6 hours apart.
2-At least 3+ proteinuria on a semi qualitative assay
3-Less than 400 ml of urine output over 24 hours.

Because hypertension, proteinuria and oliguria are not always present consistently and concurrently in patients with severe Preeclampsia, additional criteria involved verifying the diagnosis of severe disease. These include:
1-Cerebral or visual disturbances such as altered consciousness, headaches, scotoma, or blurred vision.
2-Pulmonary edema or cyanosis.
3-Epigastric or right upper quadrant pain
4-Impaired liver function of unclear etiology
5-Thrombocytopenia, or
Subjects signed informed consent and a computer-generated random card was drawn and the patients were assigned to receive immediate postpartum curettage or no curettage. At admission, evaluation included mean arterial pressure (MAP), hemoglobin and hematocrit, urine dipstick for presence of proteinuria, quantitative platelet count, uric acid and serum creatinine, liver function tests, neurological examination and Ophthalmological examination of the fundus. Thirty patients underwent uterine curettage after vaginal or caesarian delivery. At Caesarian, the presumed area of the decidua basalis was curetted with large banjo curette (fig.1). Patients who delivered vaginally were sedated with intravenous butorphanol tartrate (Stadol, Bristol Laboratories, Evansville, IN, USA) and promethazine hydrochloride (Phenergan, Wyeth, Ayerst, Philadelphia, PA) and underwent ultrasound guided curettage using large Banjo curette (fig.1). All curettage specimens were sent for pathological examination.

During the immediate puerperium, all subjects were observed in the obstetric recovery room until disease remission was apparent. Intensive postpartum surveillance maintained for 24 hours, and included hourly measurement of MAP, hourly urine output, hemoglobin and hematocrit with platelet count every 6 hours, and uric acid and creatinine assessment at 12 and 24 hours. All medications given in the obstetric recovery room were recorded. All women received magnesium sulfate by continuous IV infusion at a rate of 1.3g/hour during the study interval. None of the patients received fluid challenge in the obstetric recovery area. Discharge criteria from this area included: 1) adequate diuresis (output > 100ml/hour sustained for at least 2 consecutive hours), 2) blood pressure control, with diastolic values below 100mmHg and systolic below 160mmHg, and 3) rising platelet count above 50,000/ml. The time each patient spent in the obstetric recovery room were recorded. Longitudinal data between groups were analyzed by repeated measure analysis of variance over time. Grouped demographic data were analyzed by X2 or Fisher exact test as appropriate. Group means were analyzed by unpaired t test. Data are expressed as mean ± Standard deviation (SD). P<0.05 was considered statistically significant.

**Results**

The age, number of, and duration of gestation were similar in both groups (table 1). The initial MAP in all patients was more than 110 mmHg. As shown in table 1, laboratory indices of renal function, uric acid and platelet count was similar in both groups.

On pathological examination, the curettage specimens revealed many decidua with minimal placental tissue.(fig.2,3)

The MAP of the curettage group was significantly reduced compared with controls at each 12 hours intervals after delivery (fig.4) with the largest difference at 14 hours postpartum (p<0.0002). Mean urine output for the curettage group was significantly greater than for the non-curettage group at 4-hour interval postpartum (fig.5). The smallest difference between both groups was observed at 4 hours and the largest difference at 20 hours postpartum (p<0.0002).

Platelet counts also demonstrated a significant change over time after delivery (fig.6) with the concentration increasing from 159,000/ml at 12 hours to 162,000/ml at 24 hours postpartum in the curettage group. On contrast, the non-curettage group had a mean platelet count of 148.500/ml at 12 hours postpartum, decreasing to 135,000/ml at 24 hours (p<0.05).

Mean serum levels of uric acid, urea and creatinine were not significantly different between both groups at 12 and 24 hours postpartum (table 2). Mean serum levels of uric acid, urea and creatinine were not significantly different between both groups at 12 and 24 hours postpartum (table 2).

Also, there was no significant difference in the frequency of administered to either group. In the group with curettage 8 patients had eclamptic fits before delivery, none of them had further fits after delivery and curettage, while 2 out of 6 patients with antepartum fits had further fits after delivery in the non-curettage group.

There were no complications from curettage. Twenty-two patients who underwent curettage were eligible for discharge from the obstetric recovery room at 24 hours, compared with nineteen controls, a non significant difference.

Follow-up for all curettage patients revealed that 11 had onset of menstruation within 6 weeks and 19 had lactation amenorrhea, non had infection or hemorrhagic complications after the curettage.

For control group 14 had onset of menstruation within 6 weeks of delivery and 16 had lactation amenorrhea.
# Table 1
**Demographic Characteristics and admission criteria**

<table>
<thead>
<tr>
<th>Group</th>
<th>Non-curettage (n=30)</th>
<th>Curettage (n=30)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal age (years)</td>
<td>25.8 (±6.2)</td>
<td>24.9 (± 5.1)</td>
<td>NS</td>
</tr>
<tr>
<td>Primigravida (n)</td>
<td>24/30</td>
<td>23/30</td>
<td>NS</td>
</tr>
<tr>
<td>Gestational age (weeks)</td>
<td>37.9 (± 2.6)</td>
<td>38.2 (± 2.3)</td>
<td>NS</td>
</tr>
<tr>
<td>Mean arterial pressure (mmHg)</td>
<td>129.9 (± 7.6)</td>
<td>129.5 (± 10.7)</td>
<td>NS</td>
</tr>
<tr>
<td>Uric acid (mg/dl)</td>
<td>7.7 (±2.1)</td>
<td>7.9 (±1.9)</td>
<td>NS</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>1.42 (± 0.5)</td>
<td>1.42 (± 0.3)</td>
<td>NS</td>
</tr>
<tr>
<td>Urea (mg/dl)</td>
<td>51.3 (± 23)</td>
<td>49 (± 22)</td>
<td>NS</td>
</tr>
<tr>
<td>Platelet count (x 100/ul)</td>
<td>175.0 (±59)</td>
<td>188.0 (± 80)</td>
<td>NS</td>
</tr>
</tbody>
</table>

NS=Non-significant

# Table 2
**Postpartum Evaluation**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Non-curettage (n=30)</th>
<th>Curettage (n=30)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uric acid</td>
<td>7.8 (± 2.1)</td>
<td>7.9 (± 2.2)</td>
<td>NS</td>
</tr>
<tr>
<td>12</td>
<td>7.7 (± 2.1)</td>
<td>7.6 (± 2.8)</td>
<td>NS</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>1.4 (± 0.5)</td>
<td>1.3 (± 0.3)</td>
<td>NS</td>
</tr>
<tr>
<td>12</td>
<td>1.4 (± 0.4)</td>
<td>1.3 (± 0.3)</td>
<td>NS</td>
</tr>
<tr>
<td>Urea (mg/dl)</td>
<td>49.4 (± 23)</td>
<td>46.5 (± 24)</td>
<td>NS</td>
</tr>
<tr>
<td>24</td>
<td>48.7 (± 2.1)</td>
<td>43.9 (± 21)</td>
<td>NS</td>
</tr>
</tbody>
</table>

P=significance

NS= non-significant

**Discussion**

Resolution of Preeclampsia occurs only with delivery and subsequent removal of functioning trophoblastic tissue. As postulated by Rodgers et al (50 and Musci et al(6), these trophoblastic cells produce a factor that is cytotoxic to endothelial cells and is responsible for the multiplicity of clinical expression of Preeclampsia pathophysiology.

The presence of a toxin that acts as a pressor substance in the decidua and amniotic fluid of patients with Preeclampsia has been suggested since the work of Hunter and Howard in 1960 (7). The high decidual concentration of this substance, which they called 'hystrotonin' led those investigators to perform postpartum curettage in 70 women with Preeclampsia. After curettage, they observed more rapid resolution of the elevated blood pressure that accompanied Preeclampsia. In one of their...
Eclamptic patients who were treated with morphine sulfate for sedation but who had not undergone curettage after delivery, three additional seizures occurred before a uterine curettage was performed, thereafter, the eclampsia resolved promptly. These findings are consistent with the long studied observation that the only permanent cure for Preeclampsia/eclampsia is delivery of the fetus and removal of fetoplacental unit, this eliminating the trophoblastic toxins believed to be responsible for the pathophysiologic changes of Preeclampsia.

Our study compared two groups of patients with severe Preeclampsia at each time point over the first 24 hours after delivery. One group underwent postpartum curettage and the other had no curettage. Mean arterial pressure was significantly lower at each 2-hour interval postpartum in the group that underwent curettage as compared to the other group. Urine output had a similar pattern, with increased production following curettage at each of the four intervals compared with that observed in the non-curettage group. Both of these indices reflect a more rapid resolution of Preeclampsia, presumably via more complete removal of residual trophoblastic tissues.

Circulating platelets were significantly affected by uterine curettage, the treatment group demonstrated an overall mean increase in platelet count from 156,000/ml at 12 hours to 162,000/ml at 24 hours post delivery. The non-curettage group had a mean drop in platelets from 148,000/ml at 12 hours to 135,000/ml at 24 hours (p<0.05). Platelet counts tend to drop early in patients with Preeclampsia and reflect the severity of this disease (11). The decrease in circulating platelets appears to be due to increased peripheral destruction.

More rapid and complete removal of any residual placental tissue after delivery would theoretically abort much earlier the effect of any factors produced by preeclamptic placental tissue that might directly or indirectly lead to peripheral destruction of circulating platelets. This could be applied to eclamptic patients where none of the 8 patients who had antepartum fits exhibit further fits after delivery.

The findings of this study suggest that curettage at delivery helps to effect a more rapid resolution of severe Preeclampsia as measured by MAP, platelet count and urine output. Uric acid, creatinine, and urea levels were not significantly different in both groups at 24 hours post delivery and we recommend further studies with longer duration of observation to evaluate the effect of uterine curettage on these indices.

Fig 1. Large Banjo Curette 35 x 2.5 cm

Fig.2 pregnancy type decidua of a case of preeclampsia showing absence of intravascular trophoblastic invasion (H/E x 400)
Fig. 3 pregnancy type decidua of a case of preeclampsia showing multiple vascular thrombi (H/Ex100)

Fig. 4 Mean arterial pressure for the first 24-hour postpartum for the curettage group and non-curettage group.
Fig. 5 Mean urine output at 4-hour intervals for the first 24-hour postpartum for the curettage group and non-curettage.

Fig. 6 Mean platelet count at 12-hour intervals following delivery for the curettage group and non-curettage group.
References:

10. Thiagrajah S; Bourgeoisia FJ; Harbert GM; Caudle MR. Thrombocytopenia in preeclampsia: Associated abnormalities and management principles

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