Effects of postpartum uterine curettage on maternal well-being in severe preeclamptic patients

A. Alkan[1], *M.D.*; **S. Tugrul**[1], *M.D.*; **Ö. Oral**[2], *Assoc. Prof.*; **H. Uslu**[1], *M.D.*; **D. Köse**[1], *M.D.*; **F. T. Çatakli**[1], *M.D.*

Zeynep Kamil Gynecologic and Pediatric Training and Research Hospital, Istanbul (Turkey)

Summary

Purpose: We assessed the effect of postpartum uterine curettage on maternal recovery time in severe preeclamptic patients.

Method: Fifty-six pregnant women with the diagnosis of severe preeclampsia in their third trimester were enrolled in the study. Uterine curettage was performed in the early postpartum period on 31 randomly selected patients and curettage was not performed in the remaining 25 patients. Prepartum mean arterial pressure (MAP) values, quantitative platelet counts, presence of proteinuria tested semiquantatitively, lactic dehydrogenase (LDH), aspartate transferase (AST), alanine transferase (ALT), and uric acid levels were determined.

Findings: In the group that underwent curettage, we observed a faster drop in the mean arterial pressures monitored at two-hour intervals, especially after the sixth postpartum hour ($p < 0.05$). Average urine output recorded at four-hour intervals in the postpartum period was significantly higher in the curettage group compared to the non-curettage group ($p < 0.05$). The difference in the platelet counts of both groups was not significant at the 12th postpartum hour, however, at 24 hours, platelet counts in the curettage group were higher. In the postpartum period at the 12th and 24th hours there was no difference between the two groups with regard to LDH, AST, and ALT values ($p > 0.05$).

Results: In our study we have observed that uterine curettage performed in the postpartum period had favorable effects on blood pressure, platelet count, and urinary output and also helped in faster recovery from severe preeclampsia. We, therefore, consider that postpartum uterine curettage is useful for patients with severe preeclampsia that require faster recovery.

Key words: Preeclampsia; Postpartum curettage.

Introduction

Preeclampsia is defined as development of edema, proteinuria, and hypertension in the second half of pregnancy in an otherwise normal woman, and the overall incidence is approximately 7-10% in all pregnancies.

Preeclampsia is considered as an endothelial cell disorder seen only in pregnant woman [1]. It is a disease that results in increased perinatal and maternal morbidity and mortality rates. The only known treatment mode is removal and/or ceased functioning of pregnancy byproducts, especially after removing the placenta and its remnants, the patient quickly recovers.

Maternal complications of preeclampsia can be severe. Included among some of the complications are abruptio placentae, thrombocytopenia, disseminated intravascular coagulation (DIC), intracerebral hemorrhage, cardiopulmonary insufficiency, and HELLP syndrome. Shortening recovery time in preeclampsia and eclampsia may prevent some of the complications and may decrease the duration of stay in the intensive care unit and in the hospital [2].

In our study, we investigated the effect of early uterine curettage (within 30 minutes after delivery) on maternal recovery time.

Materials and Methods

This study was conducted at the Zeynep Kamil Obstetric, Gynecology, and Pediatric Research and Training Hospital in Istanbul, between December 1996 and October 1997. Fifty-six pregnant women with the diagnosis of severe preeclampsia in their third trimester were enrolled in the study. Uterine curettage was performed in the early postpartum period on 31 randomly selected patients. In this group of 31 patients, 22 had deliveries by cesarean section while nine patients underwent vaginal deliveries. In the remaining 25 patients curettage was not performed (in this group, 20 patients had deliveries by cesarean section and five had vaginal deliveries).

Severe preeclampsia was defined as the presence of the following findings in combination: 1) systolic blood pressure ≥ 160 mmHg or diastolic blood pressure ≥ 110 mmHg at two measurements taken at six-hour intervals, 2) proteinuria of 5 g or + 3 using the semiquantative method in a 24 hour collected urine sample, 3) a urinary output of < 400 ml over 24 hours, 4) persistent cerebral or visual abnormalities or cerebral edema, 5) persistent epigastric pain with nausea or vomiting, and 6) pulmonary edema and cyanosis.

After the patients were admitted to the hospital, mean arterial pressure (MAP), hemoglobin, hematocrit, quantitative platelet count, semi-quantitative proteinuria, lactic dehydrogenase (LDH), aspartate transferase (AST), alanine transferase (ALT) and uric acid levels were measured.

In patients undergoing cesarean section, the decidua basalis layer was curetted using large Bumm curettes (curette size: 4). Curettage for patients with vaginal delivery was performed under ultrasonographic guidance after appropriate sedation was achieved. All curetted material underwent histopathologic evaluation.

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In the postpartum period all patients were monitored in the intensive care unit for 24 hours. Arterial blood pressure and urine output was monitored on an hourly basis and hemoglobin, hematocrit, and platelet counts were taken every six hours. AST, ALT, LDH levels were measured every 12 hours. All patients received magnesium sulphate at a dose of 2 g/hour after a bolus dose of 6 g IV.

For statistical analysis, the chi square, Fisher's exact test, and Student's t-test were used. A value of $p < 0.05$ was considered significant.

Results

The patients in the postpartum uterine curettage and non-curettage groups had comparable maternal and gestational ages. The measured values for MAP, uric acid, AST, ALT, and LDH during admission did not show any significant difference. In both groups MAP was over 110 mmHg (Table 1).

Table 1. — *Initial in-patient admittance values.*

	Patients with curettage n = 31		Patients without curettage n = 25		p
	Avg	± SD	Avg	± SD	
Maternal age (years)	22.8	3.4	24.6	7.5	NS*
Duration of gestation (weeks)	34.6	3.2	33.5	8.7	NS
MAP (mmHg)	120	7.2	118.7	6.8	NS
Uric acid (mg/dl)	5.4	1.2	5.3	1.5	NS
Aspartate transferase (IU/l) AST (IU/l)	74	63.1	80	72	NS
Alanin transferase (IU/l) ALT (IU/l)	47	28	48	24	NS
Lactic dehydrogenase LDH (IU/l)	300	147	302.8	116	NS

Avg = average; MAP = mean arterial pressure; NS = not significant ($p > 0.05$)*.

Mean arterial pressures recorded at 2-hour intervals in the postpartum period revealed a faster decline in the group with uterine curettage, especially after the sixth hour. There was a statistically significant difference between the two groups after the sixth hour ($p < 0.05$) (Table 2).

Table 2. — *Postpartum mean arterial pressure values (mmHg).*

Hour	Patients with curettage n = 31		Patients without curettage n = 25		p
	Avg	± SD	Avg	± SD	
0	120.0	7.2	118.7	6.8	NS
2	118.2	3.6	120.0	8.5	NS
4	116.0	6.6	118.0	1.7	NS
6	116.0	4.0	120.0	7.4	< 0.05
8	110.3	7.0	116.7	3.2	< 0.05
10	110.6	8.3	116.7	0.2	< 0.05
12	106.0	9.7	119.7	7.8	< 0.05
14	107.0	5.0	117.1	0.7	< 0.05
16	107.4	7.0	114.4	0.9	< 0.05
18	106.2	4.0	116.6	1.9	< 0.05
20	104.2	7.8	114.5	3.1	< 0.05
22	104.6	1.7	113.0	2.7	< 0.05
24	103.4	7.8	110.2	4.8	< 0.05

Avg = average; NS = not significant ($p > 0.05$).

Urine output recorded at 4-hour intervals in the postpartum period was significantly higher in the uterine curettage group when compared with the non-curettage group. The difference between the two groups became statistically significant after the fourth hour ($p < 0.05$) (Table 3).

Table 3. — *Postpartum mean urine output (ml/4 hours).*

Hour	Patients with curettage n = 31		Patients without curettage n = 25		p
	Mean	± SD	Mean	± SD	
4	138.3	5.0	118.7	4.7	< 0.05
8	140.7	4.7	109.6	28.3	< 0.05
12	182.0	5.2	128.9	42.5	< 0.05
16	200.4	50.1	150.9	45.7	< 0.05
20	207.4	54.5	164.7	10.2	< 0.05
24	198.7	39.8	158.7	30.4	< 0.05

There was no difference between the two groups with regard to prepartum mean platelet counts. This was also the case in the 12th postpartum hour, however, higher platelet counts were obtained in the uterine curettage group at the 24th hour ($p < 0.05$) (Table 4).

Table 4. — *Mean postpartum platelet count ×1000/μl.*

Hour	Patients with curettage n = 31		Patients without curettage n = 25		p
	Mean	± SD	Mean	± SD	
0	169.4	105.6	172.0	108.1	NS*
12	164.2	114.8	168.7	82.3	NS
24	170.0	113.7	165.3	93.4	< 0.05

*NS: not significant ($p > 0.05$).

In the postpartum period, no differences for the LDH, AST, and ALT values were obtained at the 12th and 24th hours ($p > 0.05$) (Table 5).

Table 5. — *Mean postpartum platelet count ×1000/μl.*

Hour	Patients with curettage n = 31		Patients without curettage n = 25		p
	Mean	± SD	Mean	± SD	
LDH (IU/l)	12	267 111	300	102	NS*
	24	290 13	306	110	NS
AST (IU/l)	12	49 50	64	50	NS
	24	39 23	40	24	NS
ALT (IU/l)	12	39 15	40	25	NS
	24	30 20	30	23	NS

*NS: not significant ($p > 0.05$).

Postpartum histopathologic examination basically revealed minimal placenta and decidua. There were no complications (uterine perforation or infection) related with the curettage.

Discussion

Delivery and removal of the placenta with its remnants has been known for a long time as the only effective method of treating preeclampsia. Today, the only effective means of treating preeclampsia and eclampsia is delivery and removal of functioning trophoblastic tissue.

Presence of trophoblastic tissue and not the fetus is a prerequisite for the development of preeclampsia and this fact has been proven with preeclampsia evolving in molar pregnancies. Rodgers *et al.*, Musci *et al.* and Wilczynski *et al.* have reported that trophoblastic cells produce a factor that has cytotoxic effects on endothelial cells and this factor is also responsible for most of the physiopathology and clinical symptoms seen in preeclampsia [3-5].

It has been known since the research conducted in 1960 by Hunter and Howard that a toxin, for which they have coined the term 'hysteronin', is present in the amniotic fluid and decidua of preeclamptic patients [6]. For the first time in 1961, Hunter *et al.* reported faster recovery in patients with preeclampsia and eclampsia after performing uterine curettage in the early postpartum period [7]. Pritchard *et al.* observed that in order to achieve effective treatment, chorionic villi have to be removed or surgically excised [8].

It is believed that endothelial cell damage is the cause of most of the pathophysiologic changes seen in preeclampsia [1]. Potent vasoconstrictors released from endothelial cells such as thromboxane A and endothelin seem to be responsible for the end-organ damage [3]. It has been suggested that the mechanisms responsible for endothelial cell damage in normal pregnancies might well be due to insufficient development of immune tolerance in the first trimester [4]. This immune tolerance results in biochemical and morphologic changes both in the maternal systemic circulation and uteroplacental flow in normal pregnancies. Included in some of these changes is endovascular invasion of the spiral arteries by trophoblasts and loss of the muscular layer, development of low pressure, low resistance, and high flow circulation and synthesis of some vasoactive agents. It is thought that immune tolerance in preeclampsia limits invasion of spiral arteries by trophoblasts to the decidual layer without being able to reach the myometrium and lack of appropriate vasodilatation in the spiral arteries [9]. According to a different hypothesis, abnormal maternal immune response results in release of free radicals in the decidual lymphoid tissues that result in endothelial damage [10]. Before preeclampsia two clinically evident waves of vascular invasion by the trophoblasts occur in the implantation site. The first wave of invasion takes place between the 10th and 16th weeks of gestation, while the second wave moves inbetween the 16th and 22nd weeks. It is suggested that in preeclamptic women this second wave is unsuccessful [11]. This unsuccessful invasion by the trophoblasts results in poor perfusion of the fetoplacental unit and release of the toxin that causes endothelial damage [12]. All these findings suggest that trophoblastic toxin or toxins are the cause of the pathophysiologic changes in preeclampsia and in order to treat preeclampsia and eclampsia, the fetus and the fetoplacental unit have to be removed.

We have observed a significant decrease in the mean arterial pressure values of the patients in the uterine curettage group measured at two-hour intervals compared to the non-curettage group. Magaan *et al.* have also reported significantly reduced mean arterial pressure values in preeclamptic patients that underwent postpartum uterine curettage [13]. Göçmen *et al.* have reported similar findings [14]. Magann *et al.* studied three groups of preeclamptic patients in 1994, one group undergoing postpartum uterine curettage, another group treated with nifedipine, and a third non-curettage group. They reported a significant reduction in the mean arterial pressure values of the curettage group [15]. Despite these findings, however, Schlenzig *et al.* were not able to show a significant difference in the MAP values of 24 HELLP syndrome cases studied retrospectively [16].

Increased urine output after delivery in preeclampsia and eclampsia patients is an indication for clinical improvement [17]. In our study we monitored urinary output at 4-hour intervals and observed a significant increase in the uterine curettage group. Magann *et al.* in their articles published in 1993 [13] and 1994 [15] reported a significant increase in mean urine outputs of preeclamptic pregnant women who underwent uterine curettage after delivery. Urine output increase in the control group, however, was much smaller. These two parameters show that removal of residual trophoblastic tissues causes a faster recovery in preeclampsia.

Platelet counts have a tendency to fall in the early phase of preeclampsia and is an indication of the severity of the disease [18]. The drop in platelet count in the circulation is due to increased peripheral destruction. Thrombocytopenia may be an indication for pending clinical disease or may reflect ongoing endothelial damage. Rapid and complete removal of decidual placenta tissue soon after delivery theoretically could result in the disappearance of some factors that are thought to be released by preeclamptic tissues causing peripheral platelet destruction. In our study, we observed a significant effect of uterine curettage on platelets, with an increase in the count at the 24th hour. Similar findings in platelet counts have been reported in studies by Magann *et al.* in 1993 and 1994, and Göçmen *et al.* in 1996 [13-15].

A more rapid resolution of preeclampsia-eclampsia has been observed to occur with the surgical removel of any remaining decidual tissue by dilation and curettage. Early in this century in parts of Western Europe, routine early postpartum uterine curettage was used for patients with severe preeclampsia-eclampsia. Recently, this approach has been revived at the University of Mississippi Medical Center with the addition of ultrasound guidance. An accelerated recovery from severe preeclampsia has been observed in women with severe preeclampsia who undergo ultrasound-directed gentle uterine curettage of the placental bed in addition to intensive postpartum surveilance. An accelerated recovery of the platelet count was also observed in women treated with this modality in comparison with patients who received conventional intensive postpartum surveillance [19].

Deviations in liver enzyme functions in preeclamptic patients are quite variable and with the exclusion of HELLP syndrome complications they neither reflect the

severity nor the recovery from the disease accurately [20, 21]. In our study and also in the studies by others [13-15] there were no significant differences between the curettage and non-curettage groups with regard to serum LDH, AST, and ALT values. Also in the study by Schlenzig *et al.* there was no significant difference in the LDH, AST, and ALT values between the uterine curettage and non-curettage groups among HELLP syndrome patients [16]. In light of these findings, we think that postpartum curettage does not affect serum LDH, AST, and ALT values in preeclamptic patients.

There were no complications (uterine perforation or infection) related to the curettage.

Conclusion

In our study we have observed that uterine curettage performed in the postpartum period had favorable effects on the blood pressure, platelet count, and urinary output and also helped in faster recovery from severe preeclampsia. We, therefore, consider that postpartum uterine curettage is useful for patients with severe preeclampsia who require faster recovery.

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Address reprint requests to:
S. TUĞRUL, M.D.
Bağdat Cad. Bağdat cikmazi
Serap apt. 89/3
Kızıltoprak - Kadıköy - Istanbul
(Turkey)