

CASE REPORT

POSTPARTUM CURETTAGE IN PRE-ECLAMPSIA AND ECLAMPSIA

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ABSTRACT: OBJECTIVE: To access the ability of immediate postpartum curettage to accelerate maternal recovery from severe pre eclampsia and eclampsia. **MATERIAL AND METHODS:** 100 patients with severe pre-eclampsia and eclampsia where assigned to postpartum curettage following delivery or to have no curettage. **RESULTS:** subjects who underwent immediate curettage had a decreased MAP at each fourth hourly for the first 24 hrs compared to those who were not curetted. The urine output after uterine curettage was significantly greater in the first four hours of 24hrs of postpartum period compared with controls. Platelet counts increased in 24hrs following a curettage where as controls exhibited at unchanged platelet count for 12 to 24hrs. **CONCLUSION:** Immediate postpartum curettage of the parturient in severe pre eclampsia and eclampsia appears to accelerate the recovery of the disease with no apparent adverse sequel.

KEYWORDS: pre-eclampsia, eclampsia , pregnancy, proteinuria.

INTRODUCTION: Pre eclampsia is considered as an endothelial cell disorder observed during pregnancy, mainly in nulliparous women. In preeclampsia, the physiological changes in the spiral arteries are confined to the decidual portion of the arteries. The myometrical segments remain anatomically intact and do not dilate. It was found that neutrophil activation occurs in preeclampsia , localized in part to the placental bed. Neutrophils have been implicated in the pathogenesis of vascular damage in those who were not pregnant. Activated neutrophils release various substances which are capable of mediating vascular damage. In addition, the release of toxic oxygen free radicals can cause membrane lipid peroxidation, lysis of endothelial cells, disruption of endothelial and increased vascular permeability and reactivity. The local increase in tissue leukotriene B4 levels might contribute to necrotising arteriopathy of the disease.

Resolution from this disease process occurs following delivery and subsequent removal of functioning trophoblastic tissue.

MATERIALS AND METHODS: The present study was carried out in pregnant women with severe pre-eclampsia/eclampsia in McGann hospital for the period of 2yr (Jan 2011- Dec 2012)

In this 100 study groups included 50 cases of eclampsia and 50 cases of preeclampsia. The control group constituted 50 cases of preeclampsia and eclampsia each. Besides detailed history and routine antenatal examination, other evaluation included MAP, Hb%, blood group and Rh typing, urine examination, RFT, LFT, serum platelets and fundoscopy. The study group underwent uterine curettage after vaginal/caesarean delivery. At caesarean, the area of decidua basalis was curetted with uterine curette. In patients who delivered vaginally, they underwent curettage at the site of placental location, if USG was done prior to vaginal delivery and whole uterine cavity was curetted when no USG was done. During immediate puerperium, fourth hourly MAP, urine output, daily urine routine test for albumin and platelets were done. Criteria for discharge from the recovery room

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included-adequate diuresis(>100ml per hour) , BP controlled with diastolic values below 100 mm Hg and systolic below 160. The time spent by each patient in the recovery room was recorded.

RESULTS: In this study, severe cases of pre-eclampsia and eclampsia was found more common between ages 20-30yrs and in primigravida. Majority of the them were admitted as emergency case. Most of the booked cases were not a known case of PIH and many belonged to low socio economic group. MAP in severe preeclampsia was more, between 118-128 mm Hg of mercury and in eclampsia MAP was >128 mm Hg. Most of the cases of severe preeclampsia and eclampsia was associated with proteinuria of 3+. Severe proteinuria which persisted was one of the indication for termination of pregnancy. In study group 87% had vaginal delivery and 13% underwent LSCS. In control group 74% had vaginal delivery and 26% underwent LSCS.

	Curative	Non-curative
Within 24 hrs	101.9+/-10.6	111.4+/-11.1
24-48 hrs	100.5+/-6.7	112.9+/-5.5
48-36 hrs	94.4+/-2.9	104.7+/-5.9
36-72 hrs	93.0+/-5.5	116.1+/-56.7
72-96 hrs	96.1+/-3.1	108.7+/-5.9
MAP AFTER DELIVERY		

In study group, difference in MAP in 24hrs after delivery was 10mmHg as compared to control. After 48 hrs of delivery it was 12mmHg, after 72 hrs it was 23mmHg and after 92 hrs MAP was 12mmHg.

In the study group, 7 patients of severe preeclampsia, postpartum check curettage was done for 3 patients on third day for uncontrolled blood pressure under I.V Sedation and antibiotic. Of which two patients had fall in MAP within 24hrs after curettage and one patient had raised MAP for 5 days after curettage. The other 4 cases had uncontrolled BP for 6 days and had to stay in the hospital for its control.

In two of our eclamptic patients who had not undergone curettage after delivery had convulsions within 24hrs. On the second day uterine curettage was performed under I.V sedation and thereafter eclampsia was resolved promptly. In 6 patients of eclampsia BP was uncontrolled for 4 days even with anti hypertensive drugs but none of them had postpartum eclampsia. This failure may be due to improper uterine curettage which failed to remove trophoblastic tissues. In control group one patient had postpartum eclampsia for two days after delivery and two patients had BP which was elevated for eight days even after being treated with antihypertensive drugs.

Proteinuria after delivery: In study group 88% of them had proteinuria nil after 72hrs and 2% of them had proteinuria 3+ after 72hrs, where 79% of them had proteinuria nil after 72hrs and 3% of them had proteinuria 3+ after 72hrs in control group.

DURATION OF STAY IN HOSPITAL: In study group 87% delivered vaginally and 74% in control group. Duration of stay in hospital was comparatively less in study group compared to controls. Only 11% of study group had hospital stay for more than a week as compared to control group. In study

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group 13 patients underwent LSCS , among them 9 patients had BP controlled in 24 hrs and 3 patients had BP controlled in 78 hrs and in 1 patient it was persistently elevated for 5 days and none of the patient had postpartum eclampsia and patients were discharged after removal of sutures on 7th day. In control group 26 patients underwent LSCS . Among them 14 patients had BP within 24hrs and 6 patients BP was under controlled after 48 hrs and two patients within 72 hrs and in rest of them BP was persistently elevated and had to stay in hospital for three days after removal of sutures for control of BP. One patient had postpartum eclampsia and was put under magnesium sulphate.

DISCUSSION: Preeclampsia is a clinical syndrome defined as the new onset of hypertension and proteinuria during the second half of pregnancy.¹ It afflicts 3% to 5% of pregnancies and is a leading cause of maternal mortality, especially in developing countries.^{2,3} Because the only known remedy is delivery of the placenta. In developed countries preeclampsia is an important cause of premature delivery, usually medically indicated for the benefit of the mother.

Both hypertension and proteinuria implicate the endothelium as the target of the disease. Hypertension in preeclampsia is characterized by peripheral vasoconstriction and decreased arterial compliance.^{5,6} The proteinuria of preeclampsia is associated with a pathognomonic renal lesion known as glomerular endotheliosis, in which the endothelial cells of the glomerulus swell and endothelial fenestrations are lost.^{7,8} The glomerular filtration rate is decreased compared with normotensive pregnant women. In rare cases, acute renal failure may develop.

Preeclampsia is a systemic vascular disorder that may also affect the liver and the brain in the mothers. When the liver is involved, women may present with abdominal pain, nausea, vomiting, and elevated liver enzymes .The severe preeclampsia variant HELLP syndrome (hemolysis, elevated liver enzymes, low platelets) occurs in $\approx 20\%$ of women with severe preeclampsia,⁹ and is named not only for the liver involvement, but also for the disorder of the coagulation system that develops. Approximately 20% of women with HELLP syndrome develop disseminated intravascular coagulation, which carries a poor prognosis for both mother and fetus. Placental abruption, ascites , hepatic infarction, hepatic rupture, intra-abdominal bleeding, pulmonary edema and acute renal failure are all severe clinical manifestations associated with preeclampsia that can result in maternal death. Perhaps the most feared complication of preeclampsia is eclampsia itself, defined by the presence of seizures, for which women with severe preeclampsia are often treated with magnesium sulphate prophylaxis. Cerebrovascular complications, including stroke and cerebral hemorrhage, are responsible for the majority of eclampsia-related deaths. Complications affecting the developing fetus include indicated prematurity, intrauterine fetal growth restriction, oligohydramnios, bronchopulmonary dysplasia, and increased risk of perinatal death.

The diagnosis of preeclampsia is clinical. As defined by the American College of Obstetrics and Gynecology, the diagnosis requires blood pressures $>140/90$ mm Hg on 2 occasions combined with urinary protein excretion >300 mg/d. Edema, a classic feature of the disease, is no longer considered a diagnostic feature given its lack of sensitivity or specificity.¹ Importantly, in 20% of cases, eclampsia may present without preceding hypertension or proteinuria, suggesting that the currently employed diagnostic criteria are imperfect. Laboratory tests, such as liver function tests, quantification of urinary protein, and serum creatinine may be helpful in characterizing the degree of end-organ damage, but none is specific for preeclampsia.¹ Hyperuricemia, which is more likely to be present in women with preeclampsia than in normotensive pregnant women, has been used as a

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diagnostic aid and to predict adverse outcomes in preeclampsia, but its predictive value is generally modest.

The Preeclamptic Placenta: The placenta is the central organ in the pathogenesis of preeclampsia. Removal of the placenta abolishes the disease; moreover, only the placenta, and not the fetus, is required for its development. This is best demonstrated by the case of molar pregnancy, which carries an elevated risk for preeclampsia. During normal placentation, the embryo-derived cytotrophoblast cells invade the maternal uterine wall. After invasion, cytotrophoblasts are found in the smooth muscle and endothelial layers of the maternal decidual arteries. This interaction acts to induce the remodeling of these maternal vessels into high-capacitance and low-resistance vessels that provide access to maternal oxygen and nutrients for the placenta and developing fetus. As part of this process, the cytotrophoblasts adopt an endothelial phenotype, expressing adhesion molecules classically found on the surface of endothelial cells. In preeclampsia, this process is aberrant. The first wave occurs between 10-16 wks and second wave occurs at 16-22 wks. In the patient with preeclampsia and eclampsia this second wave of trophoblastic invasion is believed to fail which leads to poor perfusion of feto-placental unit with resultant elaboration of toxin that causes endothelial damage.

The invasion of the cytotrophoblasts is incomplete, with cytotrophoblast cells present only in the superficial layers of the decidua. The spiral arteries fail to be invaded or remodeled, resulting in constricted, high-resistance vessels, visible on pathological examination of preeclamptic placentas. This shallow invasion has been shown to be related to a failure of the cytotrophoblasts to adopt an endothelial adhesion phenotype 44 (Figure 1).

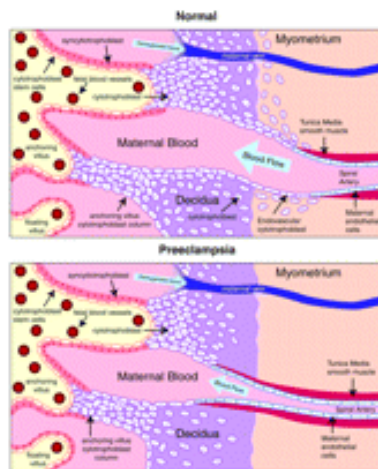


Fig 1: Abnormal placentation in preeclampsia. In normal placental development, invasive cytotrophoblasts of fetal origin invade the maternal spiral arteries, transforming them from small-caliber resistance vessels to high-caliber capacitance vessels capable of providing placental perfusion adequate to sustain the growing fetus. During the process of vascular invasion, the cytotrophoblasts differentiate from an epithelial phenotype to an endothelial phenotype, a process referred to as pseudovasculogenesis, or vascular mimicry (**top**). In preeclampsia, cytotrophoblasts fail to adopt an invasive endothelial phenotype. Instead, invasion of the spiral arteries is shallow, and they remain small-caliber resistance vessels (**bottom**).

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CONCLUSION: Resolution of the preeclampsia occurs only with delivery and subsequent removal of functional trophoblastic tissue. To accelerate disease recovery chorionic villi must be expelled or surgically removed by puerperal curettage. The trophoblastic cells produce a factor that is cytotoxic to endothelial cells and is responsible for the multiplicity of clinical expression of preeclampsia and eclampsia. These findings are consistent with long standing observation that the only permanent cure for preeclampsia/eclampsia is delivery of the fetus and removal of the feto-placental unit thus eliminating the trophoblastic toxins believed to be responsible for pathophysiological changes of pre-eclampsia and eclampsia. So uterine curettage after delivery eliminates the trophoblastic toxin which causes rapid resolution of elevated BP and disappearance of proteinuria. It reduces hospital stay for the patients without any maternal side effects.

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