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Anesthesia in severe preeclampsia

Abstract

Preeclampsia is a common cause of maternal mortality and morbidity. The etiology is unknown though a lot is known about its pathophysiology. A pregnant women with preeclampsia presenting with an indication of caesarean section, is an anesthetic challenge. There is an increase in blood volume by 40% in pregnancy after 20 weeks of pregnancy. This can result in severe hypertension in a non-pregnant individual but still blood pressure decreases in second trimester of pregnancy. This happens because of decreased peripheral vascular resistance and increased venous capacitance. If the vascular system is nonresilient and the vessel walls still maintain their stiffness and elastic recoil pregnancy induced hypertension can result. There is multiple organ hypo perfusion in severe preeclampsia.

The cardiovascular, pulmonary and cerebral changes of severe preeclampsia have been described. The principles of choice of hypertensive drugs and anesthetic monitoring in severe preeclampsia are explained.

Keywords: Anesthesia, Caesarean Section, Hypertension, Preeclampsia, Pregnancy

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Introduction

The word "Preeclampsia" is derived from the Greek word Eklampsis meaning "lightening" or "convulsions". A pregnancy with preeclampsia whose hypertension has been treated ante partum generally present for delivery with contracted plasma volume, normal or increased cardiac output, vasoconstriction, and hyper dynamic left ventricular function. There may also be a coexisting left ventricular systolic and diastolic dysfunction. Other problems are increased airway edema, decreased glomerular filtration, platelet dysfunction, or a spectrum of hemostatic derangements (the exaggerated hypercoagulable state).[1] In severe preeclampsia there is chronic placental hypo perfusion.[2] The uteroplacental circulation is not auto regulated and the fetus may poorly tolerate any further decline in perfusion. Thus the primary peripartum goals in the severely preeclampsia parturient are the optimization of maternal blood pressure, cardiac output, and uteroplacental perfusion and the prevention of seizures and stroke. The main concerns to the anaesthetist are an edematous airway, dysfunction of the cardio respiratory system, dysfunction of cerebro-vascular system and the dysfunction of coagulation system.[3]

Pathophysiology of Preeclampsia

Symptomatic preeclampsia reflects widespread endothelial dysfunction, in which placenta-derived mediators cause multisystem organ dysfunction. The endothelium is an important biologically active biophysical barrier that controls the movement between the intra-vascular and extra-cellular compartments. It is also a highly active metabolic organ. The synthesis of many substances including nitric oxide (NO) and prostacyclin (PGI2) may be decreased in preeclampsia. This leads to a major effect on vascular smooth muscle reactivity and platelet adhesiveness.

Anti-hypertensives

Vasoactive drugs during pregnancy affect the utero-placental flow and the fetus. The aim of anti-hypertensive treatment is to prevent maternal and fetal morbidity from labile hypertension. ACE inhibitors and angiotensin 2 antagonists are contraindicated before delivery because of their effects on the fetus.

Magnesium Sulphate

MgSO4 is a safe drug to use in the pregnant patient. It acts on the neuromuscular functional transmission of impulses and also lowers the vascular smooth muscle tone. But, MgSO4 may increase the likelihood of hypotension during regional anesthesia and will tend to blunt the response to vasoconstrictors.[4] Treatment of overdose is intravenous calcium following the rule of 10 (e.g. Calcium gluconate 10 ml of 10% over 10 minutes). Magnesium therapy should be continued for at least 24 hours post partum or last convulsion whichever occurs later. Monitoring of magnesium toxicity is done by checking tendon reflexes and respiratory rate. If the pregnant woman is on continuous ECG monitoring, ECG changes can also be recorded. There is prolongation of P–Q interval and widening of QRS complex, which may progress to conduction defects and cardiac arrest. The risks of toxicity increase in the presence of oliguria since magnesium depends the kidneys for excretion so urine output should be maintained above 30ml/hr. by giving intravenous fluids (normal saline or ringer lactate 500ml over 8hrs).

Choice of Anesthesia

Regional anesthesia in caesarean section has several advantages. Hypertensive response to laryngoscopy (which is pronounced in pre-eclamptic women) can be avoided. There is blunting of the neuro-endocrine response to surgery and prevention of the transient neonatal depression associated with general anesthesia. A spinal is quicker and more reliable in onset and involves less potential trauma in the epidural space. However, the disadvantages include the theoretical risk of a more abrupt hypotension in a patient who may be relatively hypovolemic and with a fetus that may be compromised by placental insufficiency. Recent studies have demonstrated the safety of spinal anesthesia in preeclampsia.[5–7] Alternatively, a combined spinal epidural approach can be used with a limited dose of local anesthetic in the sub arachnoid space and the option of utilizing the epidural as necessary. General anesthesia is necessary in a comatose eclamptic patient and so preparations must be made for difficult intubation. The main concerns are mucosal edema of the upper airway and the severe hypertensive responses to laryngoscopy and surgery. Cerebral hemorrhage is a potential complication.[8] It is important that the obstetric intubation trolley has a range of endotracheal tubes down to

size 5.5 internal diameters and a variety of laryngoscopes and other aids to securing the airway. Drugs used to attenuate the hypertensive response to laryngoscopy include the pre-induction use of magnesium, labetalol, nitrates, nitroprusside, lidocaine and/or potent parenteral opioids. If general anesthesia is necessary, equipment should be immediately available to manage a difficult airway, and every effort should be made to blunt the hemodynamic response to laryngoscopy (e.g., via a bolus of an antihypertensive drug or remifentanil).[9] Patients on magnesium may be very sensitive to the effects of non-depolarizing neuromuscular blocking drugs.

Cerebral Hemorrhage

Cerebral hemorrhage is the single most common cause of maternal death in preeclampsia and currently far outnumbers pulmonary edema. Although there was a focus on diastolic pressure in the past, the present recommendations of the National Enquiries into Maternal Death advocate treatment of systolic blood pressures above 160 mmHg in order to avoid intracranial bleeding.[10–12] Noninvasive monitoring techniques such as pulse wave analysis of middle cerebral artery and T2 weighted FLAIR sequence MRI have provided new insights into the vasogenic and cytotoxic cerebral edema[13](Figure 1 PRES syndrome in T2 weighted FLAIR sequence of MRI).

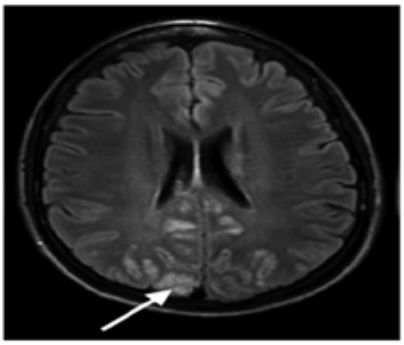

Figure 1 Magnetic resonance image of axial section brain showing foci of FLAIR increased intensity in bilateral parietal and occipital regions.

Pulmonary Edema

Placing a pulmonary arterial catheter can facilitate detection and treatment of blood pressure changes, especially in patients with severe or volatile hypertension. Echocardiography can provide information about volume status and cardiac function. However in preeclampsia, central venous pressure often does not correlate with pulmonary capillary wedge pressure, which in turn may not reflect left ventricular stroke work.[13] Also, pulmonary artery and central venous catheter placement confer a reported 4% risk of complications among hypertensive parturients. Noninvasive measures that can be used to estimate stroke volume are arterial waveform analysis and impedance cardiography. There is a favorable risk–benefit ratio of noninvasive measure (in the early postpartum period among severely preeclampsia patients) with thermo dilution-derived measurements. Figure 2 outlines the pathophysiology behind the development of pulmonary edema in preeclampsia.

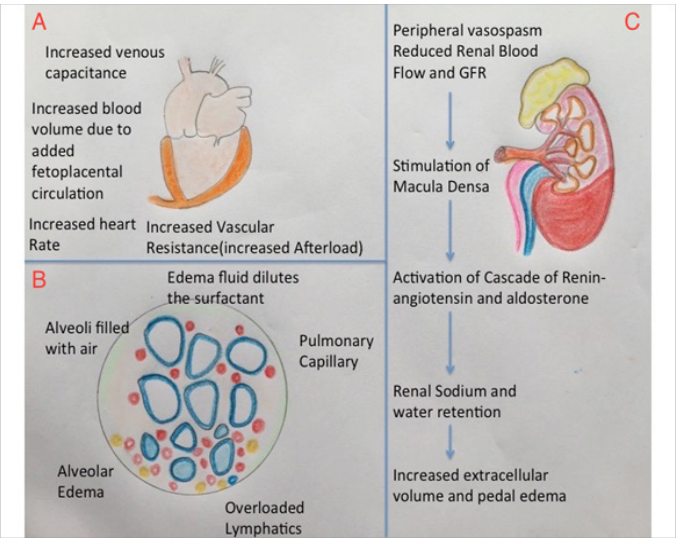

Figure 2 (A) Cardiovascular changes in heart in preeclampsia. (B) Mechanism of pulmonary edema in preeclampsia; (C) Renal hypo perfusion and edema.

Conclusion

In early-onset preeclampsia, the common hemodynamic findings are vasoconstriction, low cardiac output and low filling pressures. Traditionally it was believed that spinal anesthesia in pregnancy with severe preeclampsia can lead to severe hypotension and thereby compromise the uteroplacental perfusion. Thus spinal anesthesia was not used in these patients. However, recent clinical studies in women severe preeclampsia have revealed that they have less frequent, less severe hypotension than healthy parturient because of the increased vascular wall tone in these women. Furthermore, this hypotension is transient and easily treated as evidenced in recent studies.[14–16] No clinical trials have demonstrated significant differences in outcomes when spinal anesthesia is compared with epidural or general anesthesia.

Risk–benefit analysis strongly favors neuraxial techniques like spinal over general anesthesia for cesarean delivery in the setting of severe preeclampsia when the contraindications to neuraxial anesthesia are ruled out.[17] To conclude, spinal anesthesia is a reasonable anesthetic option in severe preeclampsia when cesarean delivery is indicated, when there is no contraindication to spinal anesthesia.

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None.

Conflict of interest

The author declares that there is no conflict of interest.

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