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The ups and downs of peripartum hypertensive disorders

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Hypertensive disorders during the peripartum period are common and associated with significant materno-fetal morbidity and mortality. Although the prevalence of peripartum hypertensive disorders has shown a decline in the last triennium, the confidential enquiries into maternal deaths have shown that hypertensive disorders remain the second most common cause of maternal mortality in South Africa.[1,2] This article provides an update on the assessment and management of peripartum hypertensive disorders relevant to the anaesthetist.

Keywords: peripartum hypertensive disorders, materno-fetal morbidity, mortality

Definitions and diagnosis

According to the International Society for the Study of Hypertension in Pregnancy (ISSHP), peripartum hypertensive disorders may be chronic (predating pregnancy or diagnosed before 20 weeks of pregnancy) or de novo (gestational hypertension or preeclampsia).[3]

Hypertension in pregnancy is diagnosed if the systolic blood pressure (SBP) is 140 mmHg, and/or the diastolic blood pressure (DBP) is 90 mmHg, ideally confirmed on two occasions or at least 4 hours apart. Gestational hypertension is defined as new-onset hypertension that develops after 20 weeks gestation without any features of preeclampsia. Twenty-five per cent of women with gestational hypertension or chronic hypertension will develop preeclampsia.[3,4]

Preeclampsia

Preeclampsia is a multi-systemic disorder. Current theory suggests that preeclampsia occurs as a result of a dysfunction in vascular remodelling of the spiral arteries and the invasion of cytotrophoblasts resulting in the activation of coagulation pathways, release of cytokines and antiangiogenic proteins, vasoconstriction and endothelial dysfunction, and reduced organ perfusion.[5]

However, the classical triad of features (inadequate placentation, placental insufficiency, and vascular reactivity) are not often found clinically. It has also been postulated that maternal systemic and uterine vascular impairment may predate placental development. At-risk patients may have a predisposition towards the development of cardiovascular disease and preeclampsia, which results in a maladaptive response to the physiological changes of pregnancy, exaggerated concentric myocardial hypertrophy, increased left ventricular mass, and diastolic dysfunction.[5]

Evidence of elevated systemic vascular resistance and lower cardiac output prior to conception has been found in patients at risk, and early-onset disease has been found to be associated with greater systolic and diastolic dysfunction. There is also evidence to suggest that there is a shared genetic mutation between peripartum cardiomyopathy and preeclampsia.[5]

The ISSHP defines preeclampsia as new-onset hypertension (SBP 140 mmHg, DBP 90 mmHg, or both) accompanied by one or more of the following features at, or after, 20 weeks gestation:[3]

- Proteinuria (urine protein:creatinine ratio [PCR] 30 mg/mmol, albumin:creatinine ratio [ACR] 8 mg/ mmol, or both).
- Maternal organ dysfunction including acute kidney injury (increase in serum creatinine of 90 mmol/l), liver dysfunction (elevated transaminases, increase in alanine transaminase 70 IU/L, or twice upper limit of normal range), haematological (thrombocytopaenia), or neurological complications (eclamptic seizures, severe headaches, persistent visual scotomata, clonus, blindness, altered mental status, or stroke).
- Uteroplacental dysfunction (abnormal umbilical artery Doppler waveform analysis, restricted fetal growth, or stillbirth).

Preeclampsia can present without proteinuria, and represents a potentially progressive clinical condition; therefore, the sub-categories' mild', 'moderate' and 'severe' should no longer be used.[1,3,6]

The American College of Obstetricians and Gynecologists (ACOG) describes preeclampsia as being with or without severe features, which include:[4]

- SBP 160 mmHg or DBP 110 mmHg.
- Thrombocytopaenia (platelet count < 100 000 ml/L).
- Impaired liver function (aspartate transaminase or alanine transaminase levels elevated to twice the upper limit of normal

and severe persistent right upper quadrant or epigastric pain not accounted for by alternative diagnoses).

- Renal insufficiency (doubling of the serum creatinine concentration in the absence of other renal disease).
- Pulmonary oedema.
- New onset headache, unresponsive to medication and not accounted for by alternative diagnoses.
- Visual disturbance.

Eclampsia

Eclampsia remains a challenge in resource-constrained environments. Young maternal age and low body mass index have been shown to be significant predictors of eclampsia. Eclamptic seizures may be associated with cytotoxic and/or vasogenic oedema, cerebral infarction, loss of cerebral autoregulation, and increased capillary permeability. Eclamptic seizures are usually self-limiting but may cause maternal hypoxia and a risk of pulmonary aspiration requiring airway protection. The risk of stroke in women with eclampsia is approximately 10 times higher than in women with preeclampsia.[2,5]

Magnesium sulphate is the first-line treatment for the prevention of eclamptic seizures. It is usually prescribed when a woman with preeclampsia presents with persistent neurological symptoms, or signs (severe intractable headache, signs of cerebral irritability, clonus, or visual disturbance).[1,6] It is given as an initial loading dose of 4–6 g intravenously over 20–30 minutes, followed by a continuous infusion of 1–2 g/hour until delivery for 24 hours. Further 2–4 g boluses can be given for recurrent seizures. Magnesium sulphate can also be given intramuscularly into the gluteal muscle with a loading dose of 10 g given as 5 g into each buttock, followed by further doses of 5 g every four hours.[6]

Deep tendon reflexes should be monitored throughout treatment as they are diminished with magnesium toxicity. Risk of magnesium toxicity is increased in compromised renal function and can lead to reduced ventilatory frequency, low oxygen saturations, and progressive muscle paralysis. The targeted serum magnesium therapeutic range is 2–4 mmol/L. Magnesium toxicity is treated with calcium gluconate (10 ml of 10% concentration given over 10 min intravenously). The National Institute for Health and Care Excellence (NICE) does not recommend the use of benzodiazepines or other standard anticonvulsants as an alternative to magnesium sulphate in women with eclamptic seizures.[1,3,6-8]

Patients with eclampsia who have a Glasgow Coma Score (GCS) < 14 are considered to be at high risk of raised intracranial pressure (ICP). Normoxia, normocarbia, adequate analgesia and sedation, good BP control, and maintenance of cerebral perfusion pressure are therefore imperative.[5]

HELLP syndrome (haemolysis, elevated liver enzymes and low platelets)

This is potentially life-threatening to both mother and baby, and represents a severe form of preeclampsia. Women may be critically unwell at presentation with placental abruption or disseminated intravascular coagulation (DIC).[1]

Risk prediction

Risk prediction, including an assessment of maternal risk factors, placental biomarkers, and uterine artery doppler measurements, might aid earlier diagnosis, assist with the decision to initiate preventative measures, and improve outcomes.[1]

Maternal risk factors

The maternal risk factors for the development of preeclampsia are presented in Table I.

Table I: Maternal risk factors for the development of preeclampsia[1]

Strong risk factors	Moderate risk factors
Previous preeclampsia	Primiparity
Chronic hypertension	Primipaternity, i.e. changed paternity and interpregnancy interval > 5 years
Maternal BMI > 30	Advanced maternal age ≥ 40 years
Pregestational diabetes	Family history of preeclampsia
Antiphospholipid syndrome	Multiple gestation
Systemic lupus erythematosus (SLE)	Chronic kidney disease
Assisted reproductive therapies	

Biomarkers

Angiogenic biomarkers can help to rule out preeclampsia and expedite diagnosis. NICE recommends testing the placental growth factor (PGF), and calculating a soluble fms-like tyrosine kinase 1 (sFlt-1):PGF ratio, in combination with standard clinical assessment, to help exclude women with preeclampsia between 20 weeks and before 35 weeks of gestation. PGF is a placental biomarker that peaks between 26 and 30 weeks gestation and reduces towards term. PGF is decreased in preeclampsia, particularly in severe disease.[8]

Studies have shown that PGF levels may be superior to clinical markers in the prediction of adverse outcomes in women with suspected preeclampsia. PGF levels are considered to be either normal (> 100 pg/ml), low (12–100 pg/ml), or very low (< 12 pg/ml).[9]

Soluble sFlt-1 is an antagonist of PGF that is increased in preeclampsia. sFlt-1 binds to vascular endothelial growth factor, is antiangiogenic, and results in severe systolic hypofunction. There is an increased risk of preeclampsia in women with a high sFlt-1 to PGF ratio.[5]

Risk prediction

Although a plethora of preeclampsia prediction models have been developed in recent years, individualised prediction of preeclampsia is rarely used in clinical practice. The most frequently used predictors usually include medical history, body mass index, BP, parity, uterine artery pulsatility index, and maternal age. There is some evidence to support the use of new risk prediction models for identifying risks of maternal adverse

outcomes from preeclampsia. Both the PREP-S (Prediction model for Risks of complications in Early-onset Pre-eclampsia) and fullPIERS (Pre-eclampsia Integrated Estimate of Risk) are validated risk prediction models based on gestational age, vital signs and biochemical observations. PREP-S can be used up to 34 weeks gestation, whereas fullPIERS can be used at any time during pregnancy.[7]

Although neither of the models can predict fetal outcomes, they are recommended by NICE and can be used to help guide decision making, particularly regarding the decision to admit a woman to hospital.[1,8,10]

The use of ultrasound techniques is increasingly being utilised to predict severity and outcome of disease. Recently, studies have shown that lung ultrasound may be useful in predicting the development of interstitial and alveolar pulmonary oedema. Optic nerve sheath diameter (ONSD) determination has also been found to be a predictor of raised intracranial pressure and disease severity.[5]

Pharmacological agents for prevention and management

Aspirin, supplemental calcium, and supplemental folic acid (4 mg/day) in the first trimester of pregnancy may be beneficial. Aspirin (75–150 mg daily from 12 weeks until delivery) is recommended for women with two or more risk factors, and those considered to be at high risk of developing preeclampsia. Where dietary calcium intake is low, supplementation (> 1 g/day) may lower the chances of developing preeclampsia.[11] Statins and biguanides are sometimes also recommended as part of preventative and management strategies.[5]

The main aim of controlling maternal BP is the prevention of intracerebral haemorrhage and stroke. Therapy should target a BP ≤ 135/85 mmHg. NICE recommends offering oral labetalol as initial therapy, followed by nifedipine and then methyldopa as alternatives. Second- and third-line agents include hydralazine and prazosin. Care should be taken in women with asthma when using beta blockers. Immediate-release oral nifedipine has also been shown to cause profound hypotension, and its use should be avoided with concomitant magnesium sulphate. Thiazide diuretics, angiotensin-converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARBs) are not recommended as they may result in congenital abnormalities.[1,6,8]

Labour analgesia

Women with preeclampsia with severe features benefit from neuraxial analgesia during labour as this can help reduce the sympathetic response to pain, facilitate cardiovascular stability, and offers easy conversion of the epidural for operative delivery.[1]

When regional analgesia is contraindicated, inhalation, intramuscular, parenteral, and remifentanil patient-controlled (PCA) analgesia techniques are considered good alternatives.[1]

Anaesthetic concerns and considerations

Pre-anaesthetic evaluation

Severity assessment

Pre-anaesthetic evaluation should ideally take place early in labouring patients, or prior to booking in elective cases, with the expectation that an emergency delivery may be required at any time.[1,6]

As patients are at increased risk for life-threatening events, including placental abruption, cerebral haemorrhage, pulmonary oedema, acute kidney injury, hepatic failure or rupture, DIC, and progression to eclampsia, the evaluation of these patients should focus on severity of disease, the airway examination, haemodynamic status, and coagulation parameters, all of which may change over time. Anaesthetists should also ascertain whether magnesium has been prescribed and administered to patients, as well as the names, dosages, and timing of any other pharmacological agents that have been taken/administered.[1,5,6]

Airway assessment and preparation

Airway management may be particularly difficult as patients are prone to oedema and bleeding with airway instrumentation. Airway oedema may worsen over the course of labour, and may be present even with a reassuring airway examination. Equipment necessary for difficult and emergency airway management should be available.[1,5,6]

Haemodynamic status

Patients may have received oral and/or intravenous antihypertensive agents/vasodilators (e.g. nifedipine, labetalol, hydralazine, and magnesium), which may affect the choice of vasoactive drugs administered during analgesia and anaesthesia. With severe preeclampsia, patients should be assessed for cardiac dysfunction, myocardial damage, and pulmonary oedema, which may affect the choice and dose of anaesthetic medications, the need for invasive monitoring, and the decision with regard to the level of postoperative care required. The need for additional investigations (e.g. electrocardiogram, focused assessment of transthoracic echocardiography, and formal echocardiography) should be made on a case-to-case basis keeping in mind the ethical principles of beneficence, non-maleficence, and distributive justice. The risk:benefit ratio of performing such procedures, urgency of surgery and risk of delay, and availability of resources and skills should be taken into account.[1,5,6]

Coagulation and bleeding

Both the absolute platelet count, and the trend in the count over time, are important considerations for the timing and advisability of neuraxial procedures.[1]

The platelet count necessary to safely perform neuraxial anaesthesia is still under debate, and practice varies. In the absence of other coagulation abnormalities, it is acceptable

to perform a neuraxial anaesthetic if the platelet count is ≥ 75 000/microL, with close follow-up for signs of spinal epidural haematoma. Neuraxial procedures should not be performed if the platelet count is ≤ 50 000/microL. Individualised decisions for patients with a platelet count between 50 000 and 75 000/microL should be made taking into account the risk:benefit ratio. The use of thromboelastometry (TEG/ROTEM) should be considered. The prescription of low-dose aspirin should not affect the decision to use neuraxial techniques. However, coagulopathies may develop (e.g. DIC and liver function abnormalities) that may preclude the use of neuraxial techniques.[5,12]

Coagulation testing, other than a platelet count, should be individualised based on patient factors (e.g. liver function test abnormalities, history of abruption, and intrauterine fetal death).[5,12]

Changes in prothrombin time (PT), partial thromboplastin time (PTT), and fibrinogen do not occur in most pre-eclamptic patients with a normal platelet count. There is no evidence to recommend the infusion of platelets solely for the purpose of allowing neuraxial anaesthesia. However, if the obstetrician administers platelet transfusion prior to Caesarean delivery, there may be an opportunity for spinal anaesthesia.[1,5,6,12]

Intraoperative concerns and considerations

Choice of anaesthetic technique

Airway status, availability of equipment, and materno-fetal risk:benefit ratio (including the risk of bleeding and opportunity for correction, the use of anticoagulants, and anticipated difficulty of the procedure) should be taken into account when choosing an anaesthetic technique. When operative delivery is required, central neuraxial anaesthesia is preferred over general anaesthesia for most women with preeclampsia. Spinal, epidural, or a combined spinal/epidural anaesthesia can all be used with good effect.[1,5]

Neuraxial techniques are contraindicated in the presence of coagulopathy and thrombocytopaenia. Although the risk of epidural haematoma has been shown to be extremely low (< 0.2%) in the presence of a platelet count > 70×10^9/L, most clinicians would still recommended a platelet count > 75×10^9/L.[1,5,6,12]

It is recommended that the platelet count should be obtained within six hours of performing the neuraxial procedure, or even more recently in those with HELLP syndrome or DIC. Spinal anaesthesia is not recommended in patients with pulmonary oedema, severe systolic dysfunction, pericardial effusions, hypertrophic obstructive cardiomyopathy, complicated congenital heart disease, and significant valvular heart disease especially mitral valve stenosis.[5,6]

General anaesthesia is associated with risks of airway problems and increased systemic and cerebral BP during laryngoscopy leading to cerebrovascular haemorrhage. The hypertensive response to laryngoscopy must be actively managed. Although controversial, intravenous opioids (e.g. alfentanil 25 µg/kg or remifentanil 1 µg/kg) or other antihypertensive drugs (e.g. labetalol 0.25 mg/kg or esmolol 500 µg/kg), or nitroglycerine should be used. The goal is to maintain BP at pre-induction values and prevent the increase of mean arterial pressures above 110 mmHg. Any anaesthesia-related hypotension can be treated with intravenous boluses or infusions of an alpha agonist such as phenylephrine, titrated to effect. The routine use of dexmedetomidine is not recommended as maternal recovery may be prolonged.[5,6]

It is important to continue preoperative magnesium sulphate infusions to reduce the risk of seizures. The rate may be increased temporarily to reduce the hypertensive response to laryngoscopy.[6] Alternatively, an intravenous bolus of magnesium sulphate (30–45 mg/kg) after administration of the induction agent may be used with minimal risk to the fetus.[5] Magnesium sulphate potentiates the action of all non-depolarising neuromuscular blocking agents, so smaller doses are required. An intubating dose of rocuronium 1.2 mg/kg may be used and residual block reversed using sugammadex. There is no clear evidence regarding the benefit of preferential use of suxamethonium over rocuronium. Patient factors, anticipated duration of surgery, and availability of resources should be taken into account when making this decision.[1,5,6]

Other risks associated with general anaesthesia include failed intubation secondary to generalised airway and subglottic oedema, mucosal bleeding, rapid desaturation and hypoxaemia, and pulmonary aspiration.[1,6]

There is an increased risk of failed intubation in these patient and Mallampati scores can deteriorate as labour progresses The use of videolarygoscopy is recommended. A smaller-than-expected tracheal tube diameter (e.g. 5.5–6.5 mm ID) may also be required if there is subglottic oedema.[1,5,6]

Haemodynamic goals and monitoring

Women with preeclampsia should have regular monitoring of oxygen saturations, ventilatory frequency, heart rate, and non-invasive arterial pressure. Invasive arterial monitoring forms part of accurate fluid balance assessment and is used to reduce the risk of pulmonary oedema. Urine output should be continuously monitored as acute deterioration in renal function can occur.[5,6]

It is recommended that the patient's BP be kept close to the patient's baseline (20% of mean arterial pressure) but always less than systolic 160 mmHg and diastolic 110 mmHg, to preserve uteroplacental perfusion. BP > 160/110 mmHg should be lowered aggressively, while monitoring the fetus to ensure that uteroplacental perfusion is maintained and late decelerations do not develop. Severe systolic hypertension is associated with peripartum haemorrhagic stroke, and permanent disability.[1,5,6]

Although BP may be labile, invasive haemodynamic monitoring (arterial catheterisation, and central venous catheter placement) is not recommended routinely for these patients. If arterial

cannulation should take place prior to induction of general anaesthesia to provide continuous BP monitoring and rapid response to adverse changes, especially during rapid sequence induction and during emergence. The decision to use invasive monitoring is individualised. Radial artery catheterisation is, however, considered a low-risk procedure that may be beneficial for continuous BP monitoring, may facilitate blood sampling, and should be considered in the following circumstances:[1,5,6]

- Persistent, severe hypertension (e.g. SBP > 160 or DBP > 110) refractory to treatment.
- Use of vasoactive infusions to control BP.
- Need for frequent blood sampling (e.g. patients with coagulopathy, haemorrhage, severe renal, or hepatic dysfunction), particularly for patients with difficult peripheral venous access.
- Need for frequent arterial blood gas monitoring (e.g. patients with pulmonary oedema and hypoxia).
- For use of a minimally invasive cardiac output monitor to guide haemodynamic management.

Central venous catheters and pulmonary artery catheters are rarely used in parturients with preeclampsia. Complication rates for central line placement are relatively high in patients with severe preeclampsia, and there are no randomised trials, or systematic reviews to support their use. Placement may also delay delivery for patients with severe preeclampsia. Indications for placement are similar to those for patients without preeclampsia, including difficult peripheral venous access, central administration of vasoactive infusions, and measurement of cardiac function and/or preload. Central venous pressure correlates poorly with pulmonary capillary wedge pressure in patients with preeclampsia.[1,5,6]

Transthoracic echocardiography (TTE) is safe in pregnancy and may be useful in assessing cardiac function in the setting of hypertension, haemodynamic instability, or respiratory failure. TTE may also be used to assess volume status and guide therapeutic management However, TTE requires specialised training and frequent use to maintain competency.[5,6]

Intravenous fluid management

Peripartum fluid administration should be monitored closely. Patients with severe preeclampsia are at risk for pulmonary oedema. The aetiology of pulmonary oedema in these patients may be multifactorial, including myocardial dysfunction as a result of acutely increased systemic vascular resistance, low colloid oncotic pressure with capillary leak, and iatrogenic fluid administration.[1,6]

Current recommendations are to restrict intravenous fluids to < 80 ml/hour. Preloading of intravenous fluids, to prevent a hypotensive response after administration of a neuraxial procedure, is not recommended. There is also insufficient evidence to support the preferential use of either colloids or crystalloids.[5,6,8]

Vasopressor use

There is no evidence to support materno-fetal benefit due to preferential use of either phenylephrine or ephedrine. Vasopressor use should be based on maternal haemodynamic status.[5]

Phenylephrine is considered to be the first-line vasopressor if systolic function is preserved. Noradrenaline and adrenaline are not recommended due to their potent vasoconstrictor effects.[1,5,6]

Uterotonic agents

Although haemodynamic response to bolus oxytocin may be variable in severe preeclampsia, the slow administration of a bolus dose of syntocinon is currently recommended. There is growing evidence to suggest that the use of carbetocin is safe and efficacious despite preeclampsia being listed as a contraindication by the manufacturers. Misoprostol remains the second-line uterotonic agent of choice as the administration of ergot alkaloids may result in profound hypertensive responses.[1,5,6]

Postoperative care

Patients with preeclampsia may require invasive monitoring and intensive care during the peripartum period. The decision to admit patients to high care or intensive care (ICU) units should be institution-specific, requires a multidisciplinary collaborative team approach, and should be based on available resources, clinical expertise, and the ethical principle of distributive justice.[1,6]

Transfer to the ICU should be considered under the following circumstances:[1,5,6]

- Need for respiratory support, including possible intubation.
- Tachypnoea > 35 breaths per minute.
- Heart rate < 40 or > 150 beats per minute.
- Need for additional vasopressor or other cardiovascular support.
- Need for more invasive monitoring.
- Abnormal ECG requiring further intervention (e.g. cardioversion).
- Need for additional intravenous antihypertensive medication.
- Acid-base or severe electrolyte abnormalities.

Postoperative analgesia

A multimodal analgesic approach is recommended. Regional techniques may be considered in cases where there are no specific contraindications to use.[5] Dennis et al.[13] have shown that postoperative analgesic requirements may be lower in pre-eclamptic patients as a result of the peripartum infusion of magnesium, and the higher neuraxial dose of local anaesthestic administered during delivery of growth-restricted and preterm fetuses.[5]

The use of nonsteroidal anti-inflammatory drugs (NSAIDs) is controversial due to the perceived risks of renal and platelet function impairment. A clear association between the use of NSAIDs for postoperative analgesia and persistent postpartum hypertension has not been demonstrated.[5]

Post-discharge follow-up

Life-long post-discharge follow-up is recommended for these patients. Postpartum hypertension can persist for up to 6–8 weeks, and in some cases, even longer. Preeclampsia is a recognised risk factor for long-term cardiovascular disease. Regardless of the type of hypertensive disorder during pregnancy, these patients have an increased risk of stroke, diabetes, chronic kidney disease, and venous thromboembolism, compared with women who have had normotensive pregnancies.[1,5,6]

Eclamptic patients may also be at risk of developing white-matter lesions. Posterior reversible encephalopathy syndrome (PRES) commonly manifests as an encephalopathy (delayed awakening, and/or acute confusional state), seizures, or acute blindness. Magnetic resonance imaging (MRI) usually shows typical bilateral white matter changes. Usually, these clinical and radiological changes are reversible in two to three weeks. However, a history of peripartum hypertension has been found to be associated with impaired memory and verbal learning in the long term.[14,15]

Conclusion

Hypertensive disorders during pregnancy continue to be a considerable cause of morbidity and mortality to both mother and fetus, especially in resource-constrained environments. Anaesthetists play an important role in the care of these women, providing analgesia, anaesthesia, and postoperative care. Judicious management of these patients is required to ensure optimal patient outcomes.

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