ORIGINAL PROF-697

PRE ECLAMPSIA & THE ANAESTHETIST

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Hypertensive disorders of pregnancy are leading cause of maternal death .Pre eclampsia is a condition unique to pregnancy and complicates about 15% of primigravid pregnancies Eclampsia complicates pre eclampsia in 0.04 % of deliveries.

DEFINITIONS

Definition and terminology used in pre eclampsia are still not universally agreed.

Followings definitions are generally used in clinical practice;

"Pre eclampsia: defined as onset of hypertension with proteinuria or,oedema or both at more than 20 week of gestation"

"Eclampsia; defined as occurrence of seizures in a patient with pre eclampsia"

Severe eclampsia is diagnosed in presence of one or more of the following;

- BP>160mmHgsystolicor110mmHgdiastolicon two occasions more than 6 hours apart.
- Proteinuria greater than 5g/24 hours.
- Oliguria of less than 400ml /24 hours;
- Cerebral or visual disturbance;
- Pulmonary oedema or cyanosis.

Chronic hypertension; is diagnosed when the pre-pregnant BP is known to be higher than 140/90mmHg or when hypertension is detected before 20 weeks gestation.

Gestational hypertension during pregnancy may occur without other signs of pre eclampsia.

Pre eclampsia is said to be superimposed on chronic hypertension when there is exacerbation of hypertension of more than 30 mmHg systolic or 15 mmHg of diastolic combined with the appearance of significant proteinuria or generalized oedema.

PATHOPHYSIOLOGY

Pre eclampsia is a multiple systemic disease causing not only high blood pressure ,but also affecting the kidneys .hematological integrity, the gastrointestinal tract and the central nervous system. The pathophysiological changes suggest that organ dysfunction is largely related to reduced perfusion Therefore abnormal placentation is regarded as key pathological change in preeclampsia.

There is generalized vasospasm producing changes throughout the body .Total peripheral resistant is increased, and vasoconstriction can be

demonstrated in the kidneys and retinal vessels. Endothelial cell damage may lead to release of vasoactive substances which promote vasospasm and these patients demonstrate an increased sensitivity to circulating catecholamines. In normal pregnancy there is blunted response to catecholamines.

Endothelial cell damage occurs in glomeruiar capillary cells of kidneys, causing increased permeability and consequent proteinuria. Urate clearance decreases early in pre eclampsia and thus raised plasma urate is an early indicator of disease. Cerebral and pulmonary hypertension may lead to cerebral haemorrhage and pulmonary oedema. Both of which are associated with high rate of maternal morbidity and mortality.

MONITORING

Elevation of maternal blood pressure is usually the first indicator of pre eclampsia. Significant proteinuria is present when more than 0.5g protein is excreted in 24 h or more than QJg/l. Hov ever non-proteinuric PET can occur. Absence of heavy proteinuria cannot be used as a reassuring sign.

Oedema is not a reliable indicator of PET .Monitoring maternal weight is useful as a measure of accelerated fluid retention.

Patient with PET should have regular hematoiogy and blood biochemistry performed. Haemoglobin and platelet count and blood urea, urate, creatinine should be assessed daily in patients with moderate and severe PET and liver function tests should be performed in severe pre-eclamptic. There is no longer any justification for performing full coagulation studies on preeclamptic patients The platelets count is an accurate predictor of other abnormalities of coagulation. It is only necessary to proceed to further coagulation studies if the platelet count is less than $100,000/cc^3$. Regular fetal monitoring is essential for patients with PET. Vasoconstriction at the deciauomyometrian junction of the utero-placental bed leads to intrauterire growth retardation (IUGR) and placenta! insufficiency .Placental abruption may also occur.

TREATMENT

The only definite treatment for preeclampsia is delivery of the fetus. Treatment of hypertension cannot halt progress of the underlying disease. The purpose of treating maternal blood pressure is two fold, firstly to protect her from the deleterious effects of uncontrolled hypertension, in particular the cerebral effect, and secondly, in preterm pregnancy to prolong the pregnancy for as long as possible to improve the chances of fetal survival. The risk-benefit ratio for mother and fetus in cases of extreme prematurity and severe PET is not easy to assess because poor placental function may negate any benefit fron significant prolongation of pregnancy .In many cases the aim is to prolong the pregnancy by 2-3 days to enable administration of maternal steroids for the purpose of improving fetal lung maturity.

ANTI-HYPERTENSIVES

The first line drug for treatment of hypertension is still Methyldopa. Hydraiazine is commonly used in the acute situation, but should be used with caution. It acts primarily by causing vasodilatation, and may provoke acute fetal distress from sudden hypotension. Administration of small repeated bolus doses are preferable to intravenous infusion.

Both labetalol and nifedipine have been extensively used as second line drugs for management of hypertension, and some units would use triple therapy (Methyidopa, nifedipine and labetalol) in situations of extreme prematurity (such as in women at 22 - 24 weeks gestation). Subiingual nifedipine can be used to rapidly and reliably drop blood pressure in the emergency situation, such as immediately before delivery by caesarean section.

Nitroglycerin can be used safely if required, as hydraiazine is not available in Pakistan. ACE inhibitors, which may seem a logical choice in treatment of hypertension in PET, are absolutely contraindicated. Their use is associated with a high incidence of fetal death.

ANTICONVULSANTS

The occurrence of actual fits in association with PET - that is, the development of eclampsia - is an important cause of maternal mortality, it is therefore desirable to prevent women from fits. Unfortunately there are no reliable predictors of eclampsia: it is equally possible to fit with a blood pressure of 140/90 as with a BP of 190/120. There are as yet no good randomised controlled studies investigating the use of prophylactic anticonvulsants. Magnesium sulphate is widely used for this purpose in the west, but it is not clear whether this practice is beneficial, nor indeed whether it may actually be harmful.

There is now clear evidence that once *a* woman has had a fit, the use of magnesium sulphate is effective in reducing the likelihood of fit recurrence. The eclampsia trial collaborative group have recently reported a large randomized controlled study comparing magnesium with phenytoin and magnesium with diazepam in women who had fits already, and demonstrated a significant reduction in risk of recurrent convulsions in the magnesium groups compared against either phenytion or diazepam.

Magnesium is usually given as a loading dose of 4g intravenously followed by an intravenous infusion of 1g/h for 24 hours.

ASPIRIN

The role of aspirin in the management of PET is unclear. A huge international trial of low dose aspirin in pregnancy has failed to demonstrate that it is of benefit in preventing PET, although there may be a case for its use in selected patients.

DELIVERY

The mode and timing of delivery are obviously obstetric decisions. Ideally such decisions should be made in discussion with the obstetric anaesthetist and child specialist.

It is imports nt to obtain recent (same day) haematological and biochemical results before planning analgesia and anaesthesia. If a woman has severe PET and there is a downward trend in platelet count the most recent platelet count should ideally have been obtained within four hours of proposed anaesthetic intervention..

VAGINAL DELIVERY

Epidural analgesia should be used whenever possible. There is good evidence that such analgesia will reduce circulating levels of catecholamines, and improve uteroplacental blood flow.

Effective epidural analgesia will prevent acute increase in blood pressure due to pain. It is important to ensure that adequate intravenous fluids are given before establishing epidural analgesia; otherwise vasodilatation and the ensuing acute hyptoension may provoke fetal distress.

If epidural analgesia is not used it is important to provide the best alternative analgesia possible, to try and prevent acute increases in blood pressure occurring in association with labour pain. Invasive blood pressure monitoring can be used so that any hypertensive episode can be treated promptly.

Patient controlled analgesia using any suitable opioid is an appropriate alternative to epidural analgesia. Regular H2 blockers should be given in conjunction with opioid - either ranitidine or cimetidine can be used - and the pediatricians must be warned that the baby may require a naloxone infusion.

OPERATIVE DELIVERY

The mother must be carefully assessed before proceeding with anaesthesia forcaesarean section. Particular attention should be paid to the history and examination, especially with regard to the airway, and also to any epigastric pain, which is often suggestive of stretching of the liver capsule. Recent (same day) biochemistry must be available, and records of blood pressure and urine output should be noted. The in-utero condition of the fetus should be ascertained, and continuous fetal monitoring should be performed whilst preparing the patient for theatre.

REGIONAL ANAESTHESIA

This is the method of choice for the preeclamptic mother. The risks of GA are avoided, and maternal morbidity and mortality are reduced. There is also evidence that in preeclampsia the fetal condition may be improved from the increased uteroplacental blood flow which results from well conducted epidural anaesthesia. Traditionally epidural anaesthesia has been recommended rather than spinal, on the grounds that acute hypotension is less likely to occur, and that epidural is a more controlled technique. Certainly if the mother has an epidural catheter in place (for trial of vaginal delivery) this should be topped up for operative delivery.

There are good theoretical grounds for using a single shot spinal if the platelet count is low, because of the reduced chance of bleeding with a smaller needle.

GENERAL ANAESTHESIA

The indications for GA are:

i. Sever coagulopathy

ii. Severe epigastric pain (because this is not abolished by a high regional block, and in the absence of other sensation this pain can become intolerable).

iii. Insufficient time to establish regional anaesthesia. We suggest that a request for delivery within 30 minutes is an indication for GA unless the anaesthetist is an extremely confident and competent spinalist. If spinal anaesthesia is attempted in these circumstances the anaesthetist must be prepared to abandon attempts and give a GA if successful intrathecal anaesthesia has not been achieved within 30 minutes.

We do not regard extreme prematurity and the possible need for classical Caesarean section as an indication for a GA.

The disadvantages of GA are well known: increased risk of failed intubation, hypoxia and acid aspiration. These apply to all pregnant women. The preeclamptic has additional risks:

i. Hypertensive response to intubation; In severe PET autoregulation of cerebral blood flow may be impaired, and a marked hypertensive response to intubation may provoke an intracranial catastrophe.

ii. Laryngeal and neck oedema may make intubation extremely difficult or impossible.

Blood pressure must be controlled before inducing anaesthesia, and the induction sequence should then include drugs intended to attenuate the hypertensive response to intubation. We personally use lignocaine (usually about 100 mg iv) for intracranial protection (although this is disputed) We would control BP with sublingual nifedipine, or/and nitroglycerine, although many different drugs have been used.

If the patient is receiving magnesium care must be taken with muscle relaxants, since these are potentiated by magnesium.

MONITORING FOR DELIVERY

Direct blood pressure monitoring using a radial artery line is desirable but for most cases non-

invasive blood pressure monitoring is sufficient. Use of pulse oximeter and ECG are mandatory.

The use of central venous and pulmonary artery lines are controversial.

We use CVP lines on most of our severe preeclamptics. The insertion of CVP line has low morbidity in the hands of an experienced anesthetist, and as long as one is aware of the limitations of the information obtained we feel that a CVP measurement is helpful in managing these patients.

FLUID MANAGEMENTS

PET is classically considered a volume-contracted state, with raised peripheral vascular resistance, decreased filling pressure and low cardiac output. In practice the situation is less simple, and there is a spectrum from low output/high resistance to high output/low resistance states. It is this knowledge that prompts the use invasive monitoring. However an accurate hourly fluid balance is mandatory. There are two different schools of thought on fluid management in PET, one who restrict fluids, and other who expands volume.

The rationale for fluid restriction is that in severe PET, fluid shifts occur so that the extra vascular space is overloaded, and that significant morbidity and mortality occur from pulmonary oedema. However, it is well recognized that the preeclamptic woman does not undergo the normal volume expansion of pregnancy, and that she therefore has a deficit of 30-40% in intra vascular volume, even though she is edematous.

Vasodilatation (with nitroglycerine or a regional anaesthetic) in the presence of intra vascular hypovolaemia can cause precipitous drops in blood pressure, and may provoke acute I fetal distress. Recognition of this has led to the more modern \ policy of volume expansion. This usually occurs prior to vasodilatation, and our normal practice would be to infuse aliquots of colloid (human albumin or a synthetic substitute). Up to 500 ml colloid can be given in this way, and we often find that blood pressure settles even before use of vasodilators,

If oliguria is present we would attempt volume expansion before any other measures. Our next step would be to use renal dose dopamine. This does not significantly affect the blood pressure, but improves renal blood flow and promotes urine output. We would use frusemide with caution, as there are anxieties about the use of frusemide in a situation where renal function is impaired, because it is toxic to the renal tubule.

MANAGEMENT OF ECLAMPSIA

The priority is to terminate the fit. Our first line drug is diazemuls, and if necessary general anaesthesia is administered. The airway must be protected. After controlling the fit, the next priority is to control blood pressure, and to institute invasive monitoring, including external fetal monitoring if the mother is undelivered. A short period of fetal bradycardia is common after a maternal fit. It is important not to be rushed into delivery- we suggests a two hour period of stabilization, although in our experience it is extremely difficult to persuade obstetricians to wait this long.

Delivery by caesarean section is not always indicated after an eclamptic fit, and neither is a GA. Regional anaesthesia avoids uncontrolled hypertension, and also allows accurate assessment of the central nervous system. It must be remembered that not all fits in pregnancy are due to eclampsia or epilepsy, and maternal CT scan may be indicated.

As we have already discussed, patients who have had an eclamptic fit should be treated with

magnesium to prevent recurrent fits. Fluid balance, blood pressure control and analgesia must all be managed in a high dependency unit for at least 48 hours, after an eclamptic fit has occurred.

THE PUERPERIUM

All severe pre-eclamptics should be nursed in high dependency unit for at least 48 hours after delivery. Provision of good analgesia is important, because pain causes increased catecholmine
levels, with resulting vasoconstriction, poor blood pressure control, reduced mobility, and thus an increased risk of thromboembolism.

Use of epidural opioids is ideal: if these are unavailable or contraindicated we should use PCA or I/V opioids. Non-steroidal anti-inflammatory analgesics are relatively contraindicated in PET because of impaired platelet function.

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Trust in God Almighty; You can't loose

Shuja Tahir