

Fits After An Epidural Block For Analgesia In Labor

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Abstract

This is a case report of eclampsia in a 20 year-old primigravida appearing soon after the second top-up of epidural analgesia. There was no prior clear indication of hypertensive disease of pregnancy or pre-eclampsia. This case represents the complexity of the situation and the dilemma of diagnosis of eclampsia in a supposedly non-pre-eclamptic patient, versus local anesthetic toxicity, and the steps of proper resuscitation in this special situation. Immediate activation of emergency staff, immediate LSCS (lower segment cesarean section) improved the outcome for mother and child.

INTRODUCTION

Eclampsia is a feared condition of generalized convulsions in pregnant women with pre-eclampsia. Eclampsia fits are a serious threat to life, and demand an immediate response, or the situation may develop into cardio-respiratory arrest₂. The coincident use of an epidural block for pain relief may complicate the matter as far as the etiology of convulsions. Convulsions in late pregnancy and during labor may be due to eclampsia while the use of amide group local anesthetics has been also reported to produce serious convulsions and even cardiac arrest₂.

We describe a case of intrapartum convulsions after epidural analgesia top-up. The patient was a 20 years old primipara, who experienced a gradually increased blood pressure during labor. Since pain was thought to be the cause of the elevated blood pressure, the patient received an epidural. Two hours later, a top-up was given and 15 minutes later the patient suffered convulsions, treated by intravenous diazepam and magnesium sulphate. Initially, the cause of the convulsions was thought to be the epidural top-up, but the gradual increase in blood pressure indicated the possibility of eclampsia. The complexity of the situation and the dilemma of diagnosis of eclampsia versus local anesthetic toxicity are discussed, as well as the proper steps of resuscitation in this particular situation. A short review of the clinical differences between eclamptic fit and local anesthetic toxicity is given, and the anesthetic, obstetric and resuscitative measures are discussed.

CASE REPORT

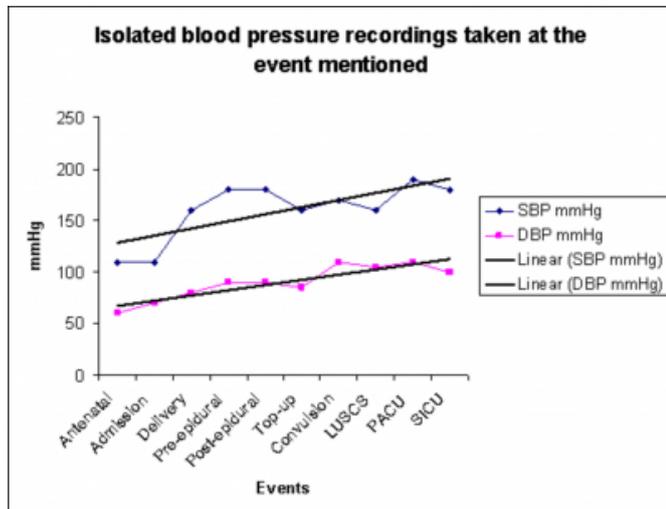
A 20 years old primigravida was admitted to the delivery

ward at King Khalid University Hospital in labor. On admission (at 1300 hrs) she was found to be term pregnant, and the cervix was 2-3 cm dilated. She had had labor pain for 6 hrs prior to admission. She attended the antenatal clinic only once at 20th week of gestation, when all routine investigations were normal. And the blood pressure (BP) measured 140/80 mm Hg.

During the course of labor a BP recording of 170/105 mm Hg was noted and attributed to labor pain and anxiety. An epidural was requested and performed at 1735 hrs, when 8 ml of 0.25% bupivacaine was injected into the epidural space. Despite the epidural, two BP recordings showed values of 160/90 and 180/90 mm Hg respectively (fig 1).

Figure 1

Figure 1: The cardiovascular recordings during the various stages of medical care, antenatal recording and on admission recordings showed normal readings after the epidural block and on two occasions DBP was 90 mm Hg and during convulsions and during operation DBP raised to more than 100 mm Hg.



Two hours after the initial block a top-up was needed and 8 ml of 0.25% bupivacaine was injected over 10 minutes. Fifteen minutes after (at 1930 hrs) the patient developed convulsions, which were controlled by intravenous injection of 10 mg diazepam followed by 4000 mg magnesium sulphate over five minutes and continuous infusion of 1000 mg/h. Immediate emergency LSCS was performed under general anesthesia at 2000 hrs. During general anesthesia the blood pressure was controlled by intravenous infusion of labetalol and inhalation of isoflurane. The intra-operative blood pressure was maintained in the range of 130/60-160/90 mm Hg. By the end of surgery the blood pressure increased again to 165/95 mm Hg. In the immediate postoperative period, the blood pressure increased to 170/110 mm Hg, and the patient suffered another minor fit, treated by 5 mg diazepam iv. Postoperatively, the patient was transferred to the surgical intensive care for close cardiovascular and neurological monitoring and she received anticonvulsants, fluid and analgesic therapy. Further treatment consisted of: 1000 mg/h magnesium sulphate continuous iv infusion, intravenous diazepam 5 mg t.d.s, continuous intravenous hydralazine, oral nifedipine, labetalol p.r.n. and epidural infusion of 0.125% bupivacaine with fentanyl 100ug at rate of 6 ml/hrs in the same original epidural catheter.

The stay in the intensive care unit was uneventful with stable haemodynamic and neurological conditions. The blood

chemistry blood gases and blood picture were normal. After 24hrs she was transferred to the postnatal ward. The patient recovered her cardiovascular and neurological stability and discharged home within four days.

DISCUSSION

The present case demonstrates the classical features of eclampsia. A significant proportion of all eclampsias occur in young (<25 years) primiparas, and death from eclampsia constitutes 12% of the maternal mortality rate worldwide, second to obstetric hemorrhage.

In the report on maternal mortality in Saudi Arabia (1989-1992) Al-Meshari et al., demonstrated that death due to hypertensive disorder in mothers was 12.4 % of the all maternal mortality. These constitute 13 cases due to this complication and they were directly related to eclampsia. In these mothers the eclamptic fits occurred after 32 weeks of gestation, some may happen even in the post partum period. Eclampsia was the fourth most cause of direct maternal death, as compared to reports from USA which showed it to be the second most important cause of death, like in Hong Kong. The cause of death in these women was postulated, (in absence of autopsy), to be intracranial hemorrhage 53.8%, pulmonary complications ARDS 15.4%, renal failure 15.4% and hepatic failure 15.4%. The report went on suggesting that care was substandard in ten out of these thirteen cases 77%. The main factor was the patient herself, since there were seven patients who were un-booked for obstetric services and antenatal clinics and three were partially booked for delivery and one patient delivered at home. The women who had post partum eclampsia were not properly monitored after her delivery. All thirteen women had convulsions before the diagnosis and treatment of preeclampsia. Peri-natal outcome was considered reasonable with four deaths out of fourteen babies 28%. The recommendations of the study were to ensure antenatal care, proper time in admission to hospital and the presence of intensive care facilities to care for this group of women.

Hibbard B.M. in England and Wales ranked hypertensive diseases as number one cause of maternal death during the period 1952-45 at a rate of 119.8/million maternities, including toxemic accidental haemorrhage. And hypertensive disease was still ranked as number one cause, with rate of 19.5 / million maternities during the period 1979-81.⁴

In this case report it appeared that the lady did not attend regular antenatal clinics and was not booked for delivery and

was admitted as an emergency. The initial delivery course was unremarkable except for moderate elevation of blood pressure which was attributed to the stress and pain of labor. The interesting finding (in retrospective) was the continuation of blood pressure level (relating to the pre-epidural block) appeared to be within the norms in this situation.

In this case, the timing of the initial convulsions, which occurred 15 minutes after an epidural top-up complicated the picture. The question on whether the convulsions were provoked by an intravenous local anesthetic effect (migrating epidural catheter), or were eclamptic in origin (only moderately increased blood pressure and no proteinuria), or were due to the rapid elevation of local anesthetics serum level, (there were no evidence of cardiovascular collapse and persistent hypertension in spite of labetalol and general anesthesia), or was it an eclamptic fit (the blood pressure was moderately elevated, but there is no proteinuria). The decision to terminate the pregnancy is understood in this context. The recurrent postoperative convulsions tipped the balance in favor of eclampsia.

Table 1: Different forms of hypertension encountered in pregnancy 9:

- Chronic hypertension
- Pregnancy induced hypertension without proteinuria
- Pregnancy induced proteinuria without hypertension
- Preeclampsia
- Eclampsia

Table 2: Risk factors for developing preeclampsia - eclampsia 9.

- Family history of preclampsia-eclampsia
- Primiparity
- Age <20 or >35 years
- Patient history of preeclampsia
- Chronic hypertension or renal diseases
- Vascular diseases

- Diabetes Mellitus
- Obesity
- Multiple gestation
- Hydatidiform Mole

Table 3: General policy of treatment of hypertension in pregnancy, including preeclampsia, HELLP syndrome and eclampsia 8.

- Conservative management
- Immediate delivery
- Anti-hypertensive treatment
 - Adrenergic inhibitors
 - Vasodilators
 - Beta-blocking agents
 - Calcium-antagonists
 - Magnesium sulphate
- Preventive treatment
 - Acetylsalicylates (ASA)
 - Indomethacin

ANESTHETIC PROSPECTIVE

Epidural block may offer a reliable method for pain relief and may be extended for deep anesthesia in case of instrumental and operative deliveries provided no convulsions or hypertension are present. In the latter cases, epidural may be contraindicated as anesthetic method in Caesarean sections (LSCS) since an uncontrolled situation may occur. The reduction of blood pressure caused by an epidural block may produce a decline in uterine perfusion leading to fetal hypoxia, acidosis and fetal death.

General anesthesia in this group of patients carries special risks due to edema of the mouth and larynx which make intubation difficult. The cardiovascular response to laryngoscopy and intubation may be another risk if not attended to. Esmolol infusion has been recommended in this situation supplemented by labetalol.

Convulsions are severe complications which may endanger the life of both the mother and fetus. Emergency control and

protection of the airway from acid aspiration and to supply the necessary oxygen preventing hypoxia is a challenging task. The convulsions may necessitate the prophylactic administration of anticonvulsant depressant drugs like diazepam. Other anticonvulsants like magnesium sulphate may be used and this recently been considered as the anticonvulsant drug of choice. The regimen must extend to the post delivery period.

OBSTETRIC PROSPECTIVE

Fits of pregnant women were recognized as early as the 4th century BC by Hippocrates. The condition was termed eclampsia - a Greek word which translates as "shine forth" - implying a sudden development. In the year 1843, Lever found that many of the women who had fits also had albumin in their urine. Later using sphygmomanometer it was demonstrated that hypertension may precede the fits. So the term preeclampsia was coined although small proportion of these mothers would develop fits ⁵.

Pre-eclamptic toxemia (PET) is a complication peculiar to pregnancy, characterized by the triad of hypertension, proteinuria and edema. The etiology is unknown, but the pathophysiological changes that accompany the syndrome are due to vasculitis, resulting in endothelial damage affects the microcirculation of all vital parenchymatous organs. In the cardiovascular system, this lead to generalized vasoconstriction, hypertension, increased vascular permeability with shift of fluids to the interstitial space and reduced intravascular volume and eventually causing non-cardiac edema ^{6,7}. In the liver, the edema causes increased pressure, hemolysis, elevated liver enzymes and low platelets (HELLP - syndrome), as well as periportal hepatic necrosis. This can presented as follows: Renal vasoconstriction, increased sensitivity to circulating angiotensin, decreased renal perfusion and glomerular filtration manifested as edema, progressive oliguria and acute tubular and cortical necrosis with anuria. Unregulated release of thrombin and generation of free plasmin lead together with the thrombocytopenia to increase consumption of coagulation factors, disseminated intravascular coagulopathy (DIC) and uncontrolled bleeding. Cerebral vasospasm, ischaemia and edema cause convulsions, hypertensive encephalopathy and cerebral hemorrhage.

Uteroplacental vasoconstriction causes decreased placental perfusion leading to intrauterine fetal growth, retardation, intrauterine fetal death, premature labor, and increased risk of abruptio placentae.

The only treatment of PET is termination of the pregnancy. Care should start early (if mother are compliant and attending the clinics) in the antenatal period and it includes frequent clinical examination. Laboratory investigations for systemic organs affection, proper treatment for hypertension by sympatholytic drugs like alpha-methyldopa, labetalol, vasodilators as hydralazine, sodium nitroprusside and magnesium sulphates in severe cases. Aspirin in minimal doses as antiprostaglandin may reduce the process of pre-eclampsia as a whole. In hospital more vigorous treatment may change the course of the disease. During labor, proper systemic sedation, or properly applied epidural may address the anxiety and stress of pain, provided that there are no coagulation defects. Epidural anesthesia also provides adequate analgesia for instrumental deliveries and may be extended for deep analgesia after LSCS.

RESUSCITATION PROSPECTIVE

Cardiopulmonary resuscitation (CPR) in obstetrics is a special resuscitation situation according to American Heart Association (AHA) & Saudi Heart Association (SHA) ². The chain of survival should be activated and the usual algorithm should be followed. Some modifications may be performed to adjust for the physiological adaptation taking place in the pregnant women, such as low oxygen reserve, high metabolic demand from mother and fetus, the gravid uterus compression's on major vessels, and tendency for gastric regurgitation and aspiration. Thus, early CPR and defibrillation in the presence of fibrillation are recommended. To insure good oxygenation and protecting the airway from aspiration of gastric contents, early intubation is recommended. In cases of impaired respiration or respiratory arrest, immediate termination of the pregnancy would improve the outcome of mother and child. In this particular case although there were no documented cardiac arrest, still the line of management is holding since the convulsions limit the ventilation and endanger the process of oxygenation. Compression of the gravid uterus on major vessels in supine position will reduce cardiac output and the extra-risk of increased intra-abdominal pressure may increase the likelihood of acid aspiration syndrome. The combination of these factors will lead to hypoxemia and cardiovascular instability may lead to cardiac standstill.

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