Obstetrical anaesthesia for a parturient with preeclampsia, HELLP syndrome and acute cortical blindness

Obstetric Forum

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Purpose: To report the management of a multigravida presenting with preeclampsia, HELLP syndrome and acute cortical blindness for Caesarean section.

Clinical features: A 39-yr-old woman, with three past uncomplicated pregnancies presented at 33 wk with acute cortical blindness. Based on clinical and laboratory assessment, a diagnosis of preeclampsia with HELLP syndrome was made. A CT scan of her head demonstrated ischaemic lesions of her basal ganglia, extending superiorly to involve both posterior parietal and occipital regions. Infusions of magnesium sulphate and hydralazine were started and an urgent Caesarean section was performed under subarachnoid anaesthesia after insertion of an arterial line and intravenous hydration. The course of anaesthesia and surgery was uneventful and she delivered a live 1540 g female infant.

By the following morning, she had recovered some vision and visual recovery was complete by 72 hr postpartum. She underwent an MRI with angiography on the first postpartum day. Ischaemic lesions were confirmed in the same sites identified on CT scan but all major cerebral vessels were patent and no significant vascular abnormality was noted. Her postoperative course was uneventful and she was discharged home seven days postpartum after being prescribed labetalol for continued hypertension.

Conclusion: The anaesthetic management of a parturient with acute cortical blindness and HELLP syndrome is modeled on the underlying preeclamptic condition. Invasive monitoring is not routinely indicated but is specifically indicated in some cases. Provided that it is not contraindicated because of prohibitive risk to the mother, regional anaesthesia has particular advantage in these patients. In particular, the use of spinal anaesthesia, which has been discouraged by some for this patient population, should be re-evaluated.

Objectif : Rapporter la prise en charge d'une multigeste présentant une toxémie gravidique, un syndrome HELLP et une cécité corticale aiguë et devant subir une césarienne.

Éléments cliniques : Une patiente de 39 ans, avec histoire de trois grossesses sans complication, s'est présentée à 33 semaines de sa 4^e grossesse avec une cécité corticale aiguë. Sur la base des données cliniques et du laboratoire, un diagnostic de prééclampsie avec syndrome HELLP a été posé. Une tomodensitométrie cérébrale a démontré des lésions ischémiques des ganglions de la base, avec extension vers le haut pour impliquer les deux régions pariétales et occipitales. Des perfusions de sulfate de magnésium et d'hydralazine ont été débutées et une césarienne urgente réalisée sous rachianesthésie après hydratation intraveineuse et installation d'une canule artérielle. L'anesthésie et la chirurgie se sont déroulées sans problème et une fille vivante de 1540 g est née.

Le lendemain matin, la patiente avait récupéré une partie de sa vision, cette récupération étant complète à 72 heures. Le premier jour post partum, elle a subi un résonance magnétique avec angiographie; cet examen a confirmé les lésions ischémiques aux sites déjà identifiés par la tomodensitométrie mais tous les vaisseaux majeurs étaient fonctionnels et aucune anomalie vasculaire n'était notée. Les suites postopératoires ont été sans histoire et elle a reçu son congé à la maison au bout de sept jours avec une prescription de labétalol en raison d'une hypertension persistante.

Conclusion : La prise en charge anesthésique d'une parturiente présentant un syndrome HELLP et une cécité corticale doit tenir compte de la condition prééclamptique sous-jacente. Le monitoring invasif n'est pas indiqué de routine mais il est spécifiquement indiqué dans certains cas. À condition de ne pas être contre indiquée à cause de risques majeurs pour la mère, l'anesthésie régionale est particulièrement avantageuse pour ces patientes. En particulier, l'usage de la rachianesthésie, déconseillé par certains chez ce type de patiente, devrait être réévalué.

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Case report

A 39-yr-old woman, with three past uncomplicated pregnancies, experienced a right temporal headache at 33 wk gestation in her fourth pregnancy. She took oral analgesics, lay down for a nap and when she awoke she was blind. She presented to the emergency room the following morning.

At the time of presentation there was no complaint of abdominal pain or symptoms referable to the abdomen although, briefly, she had been nauseated while at home. She had been previously healthy and there was no history of hypertension or migraine. On examination, her initial blood pressure was 160/90 mmHg and there was pitting oedema to the lower thoracic spine. She was able to distinguish between light and dark but unable to differentiate objects. Although gaze was deviated to the left, she was able to direct gaze to all quadrants. She had normal reflexes and no clonus. The assessment of the fetus was favourable and there was a vertex presentation. Her laboratory results included an elevated haemoglobin concentration ($170 \text{ g}\cdot\text{L}^{-1}$), a low platelet count ($99 \times 10^9\cdot\text{L}^{-1}$), normal INR, PTT, fibrinogen levels, and absent fibrin split products. Her liver enzymes and serum urate levels were elevated. There was haemolysis on the blood smear with schistocytes and red cell fragments.

A diagnosis of preeclampsia with HELLP syndrome was made; she received a bolus of magnesium sulphate *iv* and an infusion was started. A neurological consultation was obtained and an urgent CT scan of her head demonstrated bilateral symmetrical hypodense lesions involving the basal ganglia, extending superiorly to involve both posterior parietal and occipital regions. The opinion was that these lesions were ischaemic and probably represented a more extensive expression of preeclamptic disease than is typically described.

An urgent Caesarean section was scheduled and Anaesthesia was asked to assess her. At this time, she was alert and oriented. Her blood pressure had been consistently measured at about 150/90 mmHg while she was receiving infusions of magnesium sulphate and hydralazine *iv*. She was volume depleted, evidenced by increased haemoglobin and uric acid concentrations, but there was no evidence of coagulopathy, either on clinical or laboratory evaluation. A decision was made to provide surgical anaesthesia with a subarachnoid block after insertion of an arterial line and intravenous hydration. She received a bolus of $30 \text{ ml}\cdot\text{kg}^{-1}$ warmed normal saline and 10 mg metoclopramide *iv*. The hydralazine infusion was discontinued and a subarachnoid block was performed with 1.5 ml (11.25 mg) hyperbaric bupivacaine 0.75%, 10 μg fentanyl and 200

μg preservative-free morphine. Anaesthesia and surgery were uneventful and, in particular, the maternal blood pressure remained within 10% of the pre-block reading, without intervention. She delivered a live 1540 g female infant. About four hours postoperatively, she developed a small wound haematoma, attributed to inadequate surgical haemostasis and had a limited exploration of her wound under local anaesthesia.

She had an MRI with angiography on the first postpartum day. There was abnormal hyperintensity involving the basal ganglia bilaterally, both occipital lobes as well as a small area over the left frontal convexity. (Figure) The findings were consistent with ischaemic infarction, although no specific vascular territory was identifiable and all major cerebral vessels were patent. Visual recovery was complete by 72 hr postpartum. Laboratory tests returned to near normal levels by the fourth postpartum day.

The postoperative course was otherwise uneventful and she was discharged home seven days postpartum. At the time of discharge, she was prescribed labetalol for persistent hypertension.

FIGURE Magnetic resonance image of the brain.

There are abnormal, hyperintense lesions evident in both the basal ganglia (upper arrows) and the occipital cortex (lower arrows) bilaterally. These are consistent with oedema and ischaemia.

Discussion

The combination of HELLP syndrome and acute cortical blindness complicating preeclampsia is uncommon, having been reported by two other authors.^{1,2}

HELLP syndrome

The acronym HELLP, coined by Weinstein, describes a syndrome of (H) haemolysis, (EL) elevated liver enzymes and (LP) low platelets, which is a variant presentation of severe preeclampsia/eclampsia.³ The common clinical features of HELLP syndrome are shown in the Table. A microangiopathic haemolytic anaemia is present to a variable degree in all patients with HELLP syndrome. The decrease in circulating platelets is secondary to an increased rate of consumption. Intravascular deposition of fibrin in the liver sinusoids results in obstruction of blood flow, liver distention, right upper quadrant or epigastric pain and elevation of liver enzymes.

The incidence of HELLP syndrome in pregnancies complicated by preeclampsia varies from 4 to 12% and, in our institution, is 15%.⁴ Our centre is a regional obstetric referral centre and 50% of our HELLP patients are referred from other centres, possibly accounting for our higher incidence. Maternal mortality rates as high as 66% were recorded in early reports and Sibai, reporting on 112 patients with HELLP syndrome, noted two deaths (1.8 %).⁵ Perinatal mortality rates are also high with Sibai reporting a rate of 367 per 1000 deliveries.⁵ We have had no maternal mortality and the perinatal mortality rate in our series was 29 per 1000 deliveries.⁴

The most common presenting complaints in HELLP syndrome are right upper quadrant abdominal pain and nausea and vomiting.^{4,5} Visual disturbances are common and blood pressure elevations are consistent with severe preeclampsia. Delivery by

Caesarean section is usually indicated because of a deteriorating maternal condition.^{4,5} Abnormal bleeding is commonly seen and there is a higher than expected incidence of perioperative bleeding complications including increased surgical blood loss, an increased incidence of wound haematoma and a higher incidence of postpartum transfusion.^{4,5} Blood pressure, platelet count and serum levels of liver enzymes typically return to normal 48–96 hr postpartum although 18% of our patients have been discharged on oral anti-hypertensive therapy.⁴

Cortical blindness in preeclampsia

The neurological manifestations of preeclampsia-eclampsia include headache, visual disturbances, seizures, depressed levels of consciousness and stroke. Although visual disturbances develop in 25% of women with severe preeclampsia, blindness is rare with a cited incidence of 1% to 3%.⁶ Cunningham described blindness occurring much more commonly, developing in 15 of 70 women (21%) with eclampsia.⁷ There have been a number of case reports of women with acute cortical blindness complicating preeclampsia-eclampsia and many have included neuroradiological assessment. Neuroimaging findings have ranged from normal to widespread low-density areas, attributed to localized areas of decreased perfusion associated with evidence of arterial spasm, infarction, or cerebral oedema.^{7–15} Lesions of the occipital lobes are typical and the basal ganglia, brain stem and cerebellum are also commonly affected. Deep white matter lesions are less common and are more likely associated with changes in mental status. Follow-up scans demonstrate partial resolution at three to five days and complete resolution by 14 days in most patients. Clinical recovery typically precedes normalization of neuroimaging findings.

There is evidence that these brain lesions are induced by vascular changes. Doppler velocimetry, and cerebral angiography in parturients with preeclampsia demonstrate both an increased resistance to flow in the cerebral circulation and diffuse spasm of the large and medium calibre cerebral vessels.^{16–8} Schwartz, in a report on hypertensive encephalopathy, suggested two possible mechanisms by which normal cerebral autoregulation may be overcome by severe hypertension.¹⁹ The first, *overregulation*, proposes that severe cerebral vasoconstriction results in the lesions seen. The second, *breakthrough*, proposes that the loss of cerebral vascular regulation with resultant vasodilatation leads to focal vasogenic oedema. Sympathetic innervation of the cerebral vasculature is variable and the posterior circulation is less well innervated than the anterior circulation. This

TABLE Essential and associated features of HELLP syndrome

Essential:	thrombocytopenia (platelet count < 100 × 10 ⁹ .L ⁻¹) elevated AST, ALT abnormal blood smear: haemolysis
Associated:	abdominal pain nausea and vomiting headache and neurological dysfunction renal insufficiency intrauterine growth retardation intrauterine fetal death placental abruption disseminated intravascular coagulation premature delivery postpartum haemorrhage

may explain the preferential predilection of symptoms and neuroimaging findings to the posterior circulation.

Cerebral vascular reactivity may be heightened in pregnancy.²⁰ Shearer reported vasospasm predominantly affecting the posterior cerebral circulation in eight women with postdural puncture headache. These women initially presented with severe headache which progressed to visual symptoms and new-onset generalized seizures two to seven days postpartum.²¹ Three of the seven had transient cortical blindness. None of these women had preeclampsia. Magnetic resonance imaging studies were obtained for five women and in two, lesions were evident in the occipital lobes; both studies were normal when repeated after headache resolution.

Hinchey suggested that the clinical findings in these patients, and others with acute hypertensive encephalopathy of varying aetiology, make up a recognizable syndrome characterized by headache, altered levels of consciousness and mental functioning, seizures, and visual loss, including cortical blindness.²² Hinchey labelled this syndrome "Reversible Posterior Leukoencephalopathy."

Anaesthetic management

The anaesthetic management of the parturient with preeclampsia begins with an assessment of intravascular volume and adequacy of blood pressure control.²³ Preeclamptic women have smaller plasma volumes than their normal pregnant counterparts and the reduction is greater in women with severe disease and those with a growth retarded fetus (IUGR). The intravascular volume depletion typically correlates with the severity of hypertension. A measure of the volume deficit in our patient is provided by her haemoglobin concentration of 170 g·L⁻¹ and haematocrit of 0.51, reflecting haemoconcentration.

We chose to insert an arterial line in our patient. An arterial line is useful in the parturient in whom repeated blood sampling is required, who is receiving potent vasodilators for control of blood pressure, in whom the blood pressure is poorly controlled, and to provide immediate assessment of the impact of anaesthetic interventions in those with severe disease. Central haemodynamic monitoring is rarely necessary in women with severe preeclampsia unless it is complicated by oliguria, pulmonary oedema or underlying cardiac pathology. Although controlled clinical outcome studies have not been done, use of the pulmonary artery catheter has been recommended in women with: 1) refractory hypertension; 2) oliguria resistant to fluid therapy; or 3) signs and symptoms of pulmonary oedema.^{24,25}

An assessment of haemostatic function is recommended before regional anaesthesia in parturients with pre eclampsia. In the absence of clinical signs of disordered coagulation, the risk of increased bleeding is often determined by laboratory assessments, principally the bleeding time. However, there is no evidence that the bleeding time is a predictor of the risk of haemorrhage, nor that it is a useful indicator of efficacy of therapy.²⁶ Thromboelastography, though perhaps useful in differentiating the aetiology of bleeding in patients with HELLP syndrome, has not been shown to predict bleeding in patients at risk.²⁷ Thus, we concentrate our evaluation of haemostatic function on a clinical assessment of the patient's condition, relying not ONLY on her current condition and laboratory assessments but also on the stability of the findings. We rarely make the decision to withhold neuraxial block on the basis of a laboratory result alone and, specifically, do not use the bleeding time in our assessment. Although it is occasionally clear that the risk of bleeding is so high as to make neuraxial block prohibitively risky, in most cases this is not so; the risks and benefits must be considered in each case. We agree with Writer that, in patients at risk for disordered haemostasis, subarachnoid block may be preferable to epidural block as there is reduced potential for trauma without the requirement for catheter insertion.²⁸

Epidural anaesthesia to a T₄ sensory level effectively blunts the maternal haemodynamic and neuroendocrine stress responses during Caesarean section in women with severe preeclampsia. More controversial is the use of subarachnoid block for Caesarean anaesthesia in this patient population. The major concern cited is the potential for exaggerated hypotension with onset of the block, although there is little evidence that this is a common problem.²⁹ In our experience, subarachnoid anaesthesia is well tolerated and clinically important decreases in blood pressure with onset of the block are not common.

General anaesthesia may be occasionally indicated in the setting of severe preeclampsia, primarily for poor maternal condition with cardiopulmonary decompensation or coagulopathy. Induction of general anaesthesia should follow intravascular volume replacement with attention paid to the need for airway protection with cricoid pressure as well as the marked lability of blood pressure and exaggerated haemodynamic response to intubation.

Our use of subarachnoid anaesthesia was predicated on a number of factors. Although she was at risk of developing coagulopathy, at the time of intervention she had no evidence of abnormal haemostatic func-

tion. The administration of single shot subarachnoid anaesthesia arguably involves less potential for epidural space vascular trauma than does siting an epidural catheter. Additionally, in a patient with an acute neurological event, we were of the opinion that there would be merit in having our patient conscious so as to monitor changes in her neurological condition. Based on the CT scan, there was no intracranial hypertension and, in particular, no space occupying lesion. Thus, we considered that there was no imbalance of forces that we were likely to further derange with a neuraxial block administered with a small gauge, pencil-point needle.

We have reported a very unusual presentation of preeclampsia. After consideration of the underlying condition and clinical and laboratory evaluation, we constructed a plan for anaesthetic management that resulted in a stable perioperative course and an uneventful recovery. Central to our plan was the use of subarachnoid block in a patient that past teaching would not have favoured. In view of the increased experience and very positive results experienced in many centres regarding the use of subarachnoid block in obstetrical anaesthesia, it is time to reevaluate this thinking.

COMMENTARY

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This case report by Crosby *et al.* documents the significant complication of acute cortical blindness secondary to severe preeclampsia and suggests a method of management. From the case report, it is difficult to ascertain the severity of the patient's HELLP syndrome. The incidence of HELLP syndrome at BC Women's Hospital is between 10 and 15%, similar to that reported by the authors. Certainly at our institution a platelet count $< 100 \times 10^9 \cdot L^{-1}$ and evidence of liver dysfunction begins to define the HELLP syndrome. It would have been appropriate for the authors to document the level of liver function abnormality (i.e., AST, ALT, LDH), which should be at least two standard deviations above normal to meet the criteria of HELLP.⁵

This patient is interesting because she presented with acute cortical blindness with ONLY a modest elevation of her blood pressure to 160/90 mmHg. As appropriately stated, the mechanism of development of cortical blindness and eclampsia is uncertain with ischaemic hypoperfusion or hyperperfusion syndrome

as possible aetiologies.³⁰ In approximately 45% of eclampsics, there are no recognizable CT or MRI changes even when the assessment is done in close proximity to the incident.³¹

We have presented data on maternal cerebral blood flow velocity changes in preeclampsia as measured with the transcranial Doppler.¹⁶ In three cases of eclampsia there was a significant elevation of cerebral blood flow velocity which we feel was indicative of cerebral vasospasm.³² In addition, cerebral vascular changes did not return to normal values until more than 48 hours after the seizure.³³ Thus, these patients continue to be at risk for recurrence of seizure-like activity and possibly, cortical blindness. As noted by Crosby *et al.* there is partial resolution of acute cortical blindness at three to five days and complete resolution by 14 days in most patients.

The authors have discussed the patient's presentation and the lack of reliability of most of the tests used to predict which patient should receive an epidural or spinal anaesthetic. However, the authors fail to make specific recommendations about the risks of giving a spinal or epidural in the patient with HELLP syndrome and have left it to the clinical judgement of the anaesthetist. It would have been useful if they had been able to define more clearly those patients in whom they would choose one type of anaesthetic over another.

COMMENTARY

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Drs. Crosby and Preston should be congratulated for this case report which both describes an unusual complication of pregnancy-induced hypertension (PIH) and addresses the controversial use of spinal anaesthesia in this group of parturients.

This woman's primary difficulty was the development of an acute neurological complication of pregnancy-induced hypertension, that of reversible posterior leukoencephalopathy. Had this complication developed without the associated HELLP syndrome the case discussion would have been simpler. Management of these patients will always be dictated by the urgency for delivery. If urgency prevents the evaluation of acute neurological deficits to rule out intracranial hypertension, or identify the aetiology, administration of central axial blocks is relatively contraindicated. General anaesthesia, however, is not ideal because the usual goals of neuroanaesthesia (smooth induction, use of non-depolarizing muscle relaxants,

intubation when appropriate depth of anaesthesia achieved to reduced haemodynamic responses to intubation, skin incision) contradict the goals of anaesthesia for a "full-stomach" patient with a fetus. As well, the surveillance of any neurological deterioration under general anaesthesia is limited by the paucity of available intraoperative CNS monitors. The choice of spinal anaesthesia is not commonly reported in patients with central neurological disease (more commonly, epidurals have been used), but this technique did allow monitoring of the patient's neurological status and avoided the potentially deleterious hypertensive events associated with a general anaesthetic or the increased potential of a difficult intubation.³⁴

Important in this case's anaesthetic management was the time which permitted an extensive neurological evaluation (e.g., neurological consultation and access to diagnostic modalities such as a CT scan and/or MRI scan). The use of MRI was not available for this patient, although radiological evidence suggests that this is the most discriminatory tool in identifying deficits resulting from vasospasm-induced ischaemia or loss of cerebral autoregulation with vasogenic oedema.³⁵ Depending upon the aetiology, vasospasm *vs* vasogenic oedema, contrasting therapy is required and may alter anaesthetic plans. (Cerebral vasospasm is currently treated by ensuring intravascular hydration and maintaining adequate to elevated blood pressure to ensure perfusion, while vasogenic oedema responds to reductions in mean arterial pressure). It appears that most of the neurological complications of PIH are improved with lowering mean arterial pressures, and most do not evolve into permanent deficits. However, if radiological evidence demonstrates vasospasm perhaps we should reconsider our usual management of parturients with neurological deficits due to PIH.

The controversy in this case centred on the use of spinal anaesthesia for Caesarean section delivery of a parturient with PIH and HELLP syndrome. Use of spinal anaesthesia in parturients with PIH has generally been discouraged due to the rapid sympathectomy and potential for decreased placental perfusion as well as maternal morbidity associated with significant hypotension. However, despite the theoretical concerns of spinal anaesthesia, obstetric anaesthetists are re-examining this dogma as evidenced by this year's programme of the Society of Obstetric Anesthesia and Perinatology (SOAP). The appeal of spinal anaesthesia for the parturient with PIH is that this population benefits most from the limited surgical sensation, (minimising exaggerated activation of the sympathoadrenal axis), as well as from the reduced

risk of intravascular or CNS toxicity, compared with that of epidural anaesthesia. Several reports have evaluated the use of spinal anaesthesia in parturients with PIH, with these patients showing mean decreases in systolic arterial pressure of 19–30% from baseline. ONLY one of the two evaluative studies^{29,36} demonstrated significantly lower mean arterial blood pressures in those patients receiving spinal anaesthesia than those receiving epidural anaesthesia.²⁹ Vasopressor use was the same for both epidural and spinal groups in the two studies, and fetal indices of well-being (mean maternal pulsatility index) did not change with spinal anaesthesia.³⁶

Would you place a central axial block in a parturient with PIH and platelets <100,000? Unfortunately, the evidence is currently unavailable to quantify the risk of a specific platelet count's contribution to the development of an epidural or subdural haematoma. Evaluation of risk requires scrutiny of an extremely rare event in a medical system in which information (numerator and denominator) is not reliably collected nationally or internationally. However, in this particular patient there appeared to be an isolated abnormality with the low platelet count, and I would have administered either spinal or epidural anaesthesia. Challenges to Bromage's traditional teaching³⁷ are being discussed in respected obstetric anaesthesia texts³⁸ and reported in retrospective evaluations of clinical practice.³⁹ Also, evidence is growing which supports many anaesthetists' feelings that the risk of spinal haematoma is reduced using a single shot intrathecal needle compared with an indwelling epidural catheter.⁴⁰

My preference for patients with PIH requiring Caesarean section is to utilize either spinal or combined spinal-epidural anaesthesia (the CSE technique ensures excellent sacral anaesthesia with an initial small intrathecal dose of local anaesthetic plus opioid, and raise the level of the block gradually with epidural doses of local anaesthetic). The decision is made evaluating the severity of the hypertension, necessity for rapid anaesthesia, and status of coagulation parameters. The anaesthetist must be ever vigilant for the rapid arterial pressure changes when using spinal anaesthesia in these patients and have appropriate intravenous fluids and vasopressor agents immediately available.

This case has stimulated discussion on the controversial use of regional anaesthesia in a parturient with both PIH and its complications. Questions for future research in this high-risk population include identifying the optimal laboratory technique for assessing coagulopathy risk and a rigorous reassessment of the relationship between anaesthetic techniques and outcomes.

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