Obstetric Forum

Spinal subdural haematoma in a parturient after attempted epidural anaesthesia

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We report a case of spinal subdural haematoma with neurological deficit in a 36-yr-old woman following Caesarean section for severe preeclampsia and placental abruption. She had been taking chronic trifluoperazine treatment for depression. Her activated partial thromboplastin time (aPTT) was 49 sec (normal = 26–36) but all other tests of coagulation were normal. Epidural anaesthesia was attempted but, despite a negative test dose, injection of local anaesthetic resulted in a generalized seizure and general anaesthesia was induced. Seventy-two hours after delivery, she was found to have bilateral leg weakness, urinary incontinence, absent rectal sphincter tone and asymmetrical leg reflexes. The diagnosis of spinal haematoma was confirmed by magnetic resonance imaging. She underwent emergency laminectomy and made a full neurological recovery.

Nous rapportons un cas d'hématome sous-dural rachidien avec déficit neurologique chez une femme de 36 ans, après une césarienne pour éclampsie sévère et décollement prématuré d'un placenta normalement inséré. La patiente suivait un traitement de longue durée à la trifluopérazine pour dépression. Son temps de céphaline activé (aPTT) est de 49 sec (N = 26–36) mais tous les autres tests de coagulation sont normaux. Une anesthésie épidurale mise en marche mais, malgré une dose-test négative, l'injection de l'anesthésique local provoque une crise convulsive. Une anesthésie générale est réalisée. Soixante-douze heures après la naissance on constate une faiblesse bilatérale des jambes, une incontinence urinaire, une absence du tonus du sphincter anal et des réflexes asymétriques aux membres inférieurs. Le diagnostic d'hématome sous-dural rachidien est confirmé par le cliché de résonnance magnétique nucléaire. Elle subit une laminectomie en urgence, suivie d'une récupération neurologique complète.

Key words
ANAESTHESIA: obstetrical, epidural;
COMPLICATIONS: seizure, subdural haematoma;
ANTICOAGULANT: lupus coagulation inhibitor;
SURGERY: laminectomy, Caesarean section;
PREGNANCY: preeclampsia.

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Spinal haematoma, occurring either spontaneously or after regional block can result in severe, permanent neurological deficit. However, this complication is exceedingly rare. There are no case reports of spinal haematoma associated with epidural anaesthesia in a parturient without antecedent lumbar pathology.[1] This report describes the occurrence of a spinal subdural haematoma after attempted epidural anaesthesia in a parturient with severe preeclampsia.

Case report

A 36-yr-old, 60 kg, gravida 3, para 2 woman presented at 30.5 wk gestation with severe preeclampsia. Her medical history before pregnancy was unremarkable except for an unspecified psychiatric illness for which she was treated with trifluoperazine. There was no history of a bleeding disorder. Examination of the medical records from the transferring hospital revealed that the patient had complained of severe back pain radiating down both legs which was not associated with any neurological deficit. This lasted about six hours and had resolved before she was transferred to our hospital for management of severe preeclampsia. Upon arrival, her blood pressure was 160/100 mmHg and four plus proteinuria was found on dipstick examination. Serum electrolytes, uric acid and

liver enzymes were normal except for elevated alkaline phosphatase of 880 IU (normal range, 38–110). Her haemoglobin concentration was 121 $g \cdot L^{-1}$. She had a normal prothrombin time (PT, 10.5 sec), bleeding time (three minutes), and fibrinogen (7.66 $g \cdot L^{-1}$). The fibrin split products were marginally elevated (20 $mg \cdot L^{-1}$, normal $= < 10$). The platelet count was 425,000. mm^{-3}, activated partial thromboplastin time (aPTT) was prolonged to 49 sec (n = 24–36). The patient's blood pressure remained elevated in spite of continued treatment with magnesium sulphate and parenteral hydralazine. Ten hours later she had a small vaginal bleed, which suggested the diagnosis of placental abruption. She was prepared for an urgent Caesarean section under epidural anaesthesia.

On arrival in the operating room her blood pressure was 150/100 mmHg. While monitoring the patient with a pulse oximeter, ECG, and automatic blood pressure cuff, a 20-gauge epidural catheter was placed atraumatically through a 17-ga Tuohy needle at the L_2–L_3 interspace. Following a negative aspiration test, a three ml test dose of lidocaine hydrocarbonate 2% with five $\mu g \cdot ml^{-1}$ epinephrine was given without change in blood pressure or heart rate. A further 5 ml of the same solution was then given one minute later and the patient had a grand mal seizure 30 sec later. The epidural anaesthetic was abandoned. After resuscitation with oxygen by mask, general anaesthesia was induced after preoxygenation with thiopentone, succinylcholine and cricoid pressure. Anaesthesia was maintained with additional succinylcholine, enflurane, and nitrous oxide, together with fentanyl after delivery. The estimated blood loss was 500 ml with no excessive capillary bleeding. The postoperative haemoglobin concentration was 109 $g \cdot L^{-1}$ without transfusion. The infant weighed 895 g, with Apgar scores of 6 and 8 at one and five minutes respectively. The epidural catheter was removed at the end of the operation and no blood was seen within the lumen.

The next day the patient complained of trouble walking that was presumed to be due to incisional pain, but 72 hr after operation she had leg weakness and urinary retention with overflow incontinence. Neurological examination revealed absent rectal tone, absent right patellar and bilateral ankle reflexes, and generalized symmetrical leg weakness. Vibration and position senses were reduced in the great toes. There was no back pain or tenderness. A lumbar myelogram was done, but the contrast dye failed to enter the subarachnoid space. Magnetic resonance imaging (MRI) showed a space occupying lesion at L_3 to L_4. A laminectomy was performed 86 hr *post partum*, within six hours of diagnosis. At operation the epidural space was normal, but a blood clot extending from L_3 to S_1 was removed from the subdural space. Over the next six weeks the patient regained normal lower extremity strength and near normal control of both sphincters.

The abnormal aPTT was investigated. When the patient's plasma was mixed with normal plasma, there was only partial correction of the aPTT indicating the presence of factor inhibition. Normal factor assays (with a slight reduction in factor XII) ruled out a specific anti-factor antibody. A lupus anticoagulant (LA) was suspected because of the history of chronic trifluoperazine ingestion, lack of a bleeding history or of surgical bleeding during Caesarean section, and the laboratory findings. However, phospholipid dependence, which is necessary to make the definitive diagnosis of LA, was not demonstrated because of the relatively small elevation of aPTT. Other confirmatory tests were not performed as the patient was lost to follow-up.

Discussion

Spinal haematoma can complicate diagnostic lumbar puncture[2] or may occur spontaneously.[3] It is a recognized complication in patients given spinal or epidural anaesthesia, especially in those who receive perioperative anticoagulants.[4–6] Nevertheless, this must be a rare complication since none was found in a series of 1000 cases of epidural anaesthesia in patients receiving anticoagulation with warfarin.[7]

Spinal haematomas are very uncommon in pregnant patients but have been described in two parturients, in the absence of predisposing factors.[8,9] Although a suspected case of spinal haematoma has been reported in a pregnant patient receiving epidural anaesthesia,[10] none, in the absence of a spinal tumour[11] or arteriovenous malformation,[12] have been proved by myelogram or laminectomy. One case of spinal haematoma was reported in a recent large survey of obstetrical anaesthesia practices in Great Britain. This patient made a full recovery after laminectomy.[13] This voluntary, confidential survey estimated the incidence of spinal haematoma in the obstetrical population at one in 500,000.

The cause of the spinal haematoma in the patient of this report is not apparent. Back pain on the day before admission may have been caused by a spontaneous bleed, although no neurological deficit resulted at that time. The seizure after injection of local anaesthetic was presumed to be due to an intravenous injection although it could have been a coincident eclamptic seizure. Since the incidence of blood vessel trauma in the epidural space may be as high as 10%,[14] and spinal haematoma is rare, blood vessel trauma alone is unlikely to be the cause. However, it is possible that the raised venous pressure caused by the seizure in combination with blood vessel trauma was responsible for the haematoma. The delay in clinical manifestations may have been due to the location of the haem-

atoma since spinal subdural haematomas tend to be less fulminant and more chronic than epidural haematomas.[2]

At surgery, there was no evidence that the patient had a bleeding tendency. The prolonged aPTT was most likely due to the LA.[15] Although LA can be found in normal, asymptomatic individuals, we suspect that the LA in our patient was induced by trifluoperazine, a phenothiazine derivative. Drug-induced LA has been reported with the chronic use of chlorpromazine and its incidence is between 38% and 81%.[16-18] Most patients have a positive antinuclear factor assay but this is not a constant feature. Usually there are no other signs of systemic lupus erythematosis and no abnormal bleeding at surgery. The slight reduction in Factor XII is consistent with this diagnosis.[17]

Although the initial reports of patients with LA described clinical bleeding, subsequent studies have shown that these patients do not bleed. In fact, some are at risk of thrombosis.[16] At least one author[19] is in favour of offering regional anaesthesia to patients with LA, although the patient in that report received a general anaesthetic. Our patient had a nonspecific anticoagulant with most of the features of drug-induced LA, but had no evidence of abnormal bleeding such as ecchymosis or excessive surgical bleeding. The contribution of a slightly elevated aPTT in producing a spinal haematoma in a patient that had no other bleeding problem was probably negligible.

The signs and symptoms of spinal haematoma are variable. Symptoms leading to investigation often include sphincter dysfunction, paralysis of the lower extremities, and back pain. Our patient reported back pain on the day before delivery but had no neurological symptoms at that time. When she developed weakness, there was no back pain or tenderness.

The definitive diagnosis of spinal haematoma is usually made by myelogram or contrast CT scan. The use of MRI assisted in making the diagnosis before laminectomy in this patient. Laminectomy should be performed as soon as possible after the diagnosis is made in order to reduce the amount of neurological damage.[5] Most patients who have made a full recovery had been operated upon within 36 hr of the onset of symptoms,[3,20,21] although total recovery has occurred after a delay of seven days.[5]

In summary, we report an unusual case of a preeclamptic patient who had a seizure while receiving epidural anaesthesia for Caesarean section and who was subsequently found to have a spinal subdural haematoma requiring laminectomy. The aetiology of the haematoma is unknown but may have been spontaneous, caused by the epidural puncture or by the seizure in combination with blood vessel trauma. The marginally elevated aPTT is unlikely to have been an important aetiological factor in the absence of other signs of abnormal bleeding.

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COMMENTARY

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The authors have presented a medical mystery with a variety of clues which are uncovered as the story unfolds. The first clue was the six-hour episode of severe back pain with radiation down both legs which was not associated with neurological deficit which occurred on the day before admission. The authors suggest that the bleed occurred spontaneously at this time. However, it must be remembered that back pain is very common during pregnancy, the reported incidence being almost 50% with one-third of the women having severe pain, which is frequently associated with pain radiating down one or both legs.[22] The cause of the back pain is usually sacro-iliac in origin and rarely due to lumbar disc herniation. The incidence of back pain secondary to spontaneous spinal haematoma must be exceedingly rare (<1 in 500,000). However, as the authors point out the signs and symptoms of a subdural haematoma are variable and although radicular pain is common, it may be painless and neurological deficits may develop quite slowly.

The second clue was the prolonged activated partial thromboplastin time (aPTT) which was found in the admission laboratory work-up. The profile described does not fit the usual pattern of preeclampsia-associated coagulation abnormalities and is definitely not HELLP syndrome. The platelet count and bleeding time were normal. An elevated alkaline phosphatase in association with other normal liver enzymes is an enigma which the authors do not comment upon but biliary stasis can be related to phenothiazine usage. The association of phenothiazine usage and the presence of a lupus anticoagulant was new information for me although drug-induced lupus erythematosis is well-described with drugs such as hydralazine, procainamide, isoniazid, and some nonbarbiturate anticonvulsants. As the authors mention, the action of the lupus anticoagulant is often paradoxical, causing thrombosis. It has been speculated that the anticoagulant either binds with phospholipids in platelet membranes[23]

or inhibits prostacyclin release,[24] causing platelet aggregation and increasing the risk of spontaneous arterial and venous thrombosis. Only rarely is a bleeding diathesis described. I think the authors are quite correct in assuming that the contribution of a slightly elevated aPTT in inducing a spinal haematoma was negligible.

The third puzzling clue was the occurrence of the grand mal seizure after the test dose. Most obstetrical anaesthetists have experienced negative aspiration tests of catheters which are lying intravascularly. I have found it helpful to lower the end of the catheter so that it lies below the point of insertion in the back, usually below the level of the bed surface. When the catheter is open to air in this position, venous blood is able to drain slowly out of a punctured vein through the catheter and becomes visible. Presumably, aspiration causes the holes of the catheter to become occluded by the walls of the vein in which the catheter lies. The patient described may have received, therefore, a total of 160 mg lidocaine *iv* within 90 sec. The threshold for CNS symptoms in man is reported to be a lidocaine dose of 4–6 $mg \cdot kg^{-1}$; while doses causing convulsions usually amount to more than three times as much,[25] although the speed of injection can cause toxic effects at lower concentrations. The authors noted the absence of heart rate or blood pressure changes secondary to epinephrine in the test dose (although the use of epinephrine is controversial in a preeclamptic patient), but they do not mention the presence or absence of symptoms or signs after the test dose which would suggest increasing blood levels of lidocaine. The one-minute interval between the test dose and reinjection is brief. Local anaesthetics should be injected slowly and patients should be asked about symptoms and signs of systemic toxicity rather than waiting for them to volunteer the information. Should symptoms appear after negative aspiration of the catheter, aspiration is often positive when tried again.

Was this seizure related to the rapid intravenous injection of lidocaine in a patient with a decreased seizure threshold despite the presence of magnesium sulfate or was this a conincidental seizure secondary to preeclampsia? The authors favour the *iv* injection theory secondary to accidental venous cannulation during epidural catheter insertion. We shall never know. I think the coincident seizure theory is just as viable.

The final clue was the location of the haematoma: subdural and not epidural. The subdural space is a potential gap between the arachnoid and the dura mater and contains a thin film of lymph. If one is unfortunate enough to nick just the dura and not penetrate through the arachnoid into the subarachnoid space with its cerebrospinal fluid, it is possible to place the catheter in this subdural space. An injection of local anaesthetic into this space can spread a considerable distance and the ensuing block will be very similar to a high or total spinal block because only the arachnoid separates the local anaesthetic from the spinal nerves. It is not surprising that a subdural block needs 20–30 min to develop fully[26] and that even a careful fractional dose technique may not prevent complications. The perplexing question is how does a haematoma occur in the subdural space when it contains few blood vessels? The arteries and veins that serve the spinal cord run longitudinally on the surface of the pia mater and enter the subarachnoid space from the sides along with the nerve roots.[27] If the needle has strayed from the midline, then trauma to a vessel is possible. Despite these observations about the subdural space, subdural haematomas have occurred both spontaneously and in association with epidural anaesthesia. One hypothesis is that the epidural needle may pass through the dura and nick the arachnoid sufficiently to cause a CSF leak into the subdural space; the subdural space becomes distended with CSF and, in the process, small bridging vessels are disrupted, resulting in haematoma formation. Bridging vessels do exist in the meninges covering the brain and are probably the cause of subdural haematomas. However, is it valid to postulate the same mechanism in the spinal cord?

COMMENTARY

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This is an interesting report and important. Epidural and subdural haematomas following epidural anaesthesia in parturients have been reported rarely. This may be because: (1) they occur rarely, (2) patients who are predisposed to develop bleeding complications are considered unsuitable for regional anaesthesia (preselected), and (3) they are under-reported because of the fear of medicolegal ramifications.

Crawford reported one case of an epidural abscess arising in a haematoma in a retrospective series of 26,490 consecutive epidurals for labour.[28] Scott and Hibbard[13] uncovered one case in 505,000. Other cases have been reported associated with other pathology, as noted by the authors. There is one reference to bleeding in the epidural space in a parturient with the HELLP (haemolysis, elevated liver enzymes, low platelets) syndrome.[29] The literature thus tends to confirm their rarity. Certainly, there is an element of preselection. Until recently, patients

with a known or a potential bleeding defect have been denied regional blockade. Anaesthetists agonize over inserting an epidural in parturients receiving heparin, both therapeutic and prophylactic, and in those who have been taking aspirin or who have a low platelet count.

This report points out the dilemma of making a clinical diagnosis of spinal (epidural or subdural) haematoma. Many of us focus on severe back pain with tenderness as the hallmark of that diagnosis. The authors rightly suggest that the symptoms are variable and that the definitive diagnosis is made radiologically. A spinal haematoma must be evacuated as early as possible but this report also demonstrates that improvement may occur even after a considerable time has passed.

An interesting question that arises in this report is "Where was the epidural catheter?" Was it intravascular? The test dose, using currently accepted criteria, was negative. The seizure, immediately after injection of additional local anaesthetic, may have been coincidental, secondary to preeclampsia, or it may have indicated that the catheter was intravascular. Much has been written on the validity of the test dose.[30] While not discounting the value of performing a test dose, I would suggest that one wait a minimum of three minutes and actively solicit evidence of symptoms, prior to further incremental injection of local anaesthetic.

The question remains - "Why did this parturient develop a subdural haematoma?" I suspect that the actual bleed occurred spontaneously at the time that she complained of back pain. The delay in diagnosis may have been related to the use of narcotics and possibly sedatives immediately postpartum. The failed epidural is a "red-herring."

While hesitating over performing regional anaesthesia in parturients who have possible risk factors for spinal haematoma, one has to keep in mind the risks of general anaesthesia. There are serious consequences in the parturient of failure to secure an airway, as rapid desaturation and the risk of pulmonary aspiration of gastric contents present a potentially lethal combination. The incidence of difficult or failed intubation has been estimated at 1:500.[31]

The authors are to be congratulated on the outcome of this particular case. In order to overcome the dilemma of determining the incidence of problems such as this, the Society for Obstetric Anesthesia and Perinatology (SOAP) is working to establish a registry for obstetric anaesthesia complications. Hopefully, not only would this establish the incidence of these rare events, it would also allow reporting of these cases in a neutral environment without the fear of legal consequences.

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