Delayed retroperitoneal haematoma after failed lumbar plexus block

C. Aveline¹* and F. Bonnet²

¹Département d’Anesthésie-Réanimation, Polyclinique Sévigné, 3 rue du Chêne Germain, F-35510 CESSON-SEVIGNE, France. ²Département d’Anesthésie-Réanimation, Hôpital Tenon, 4 rue de la Chine, F-75020 Paris, France

*Corresponding author. E-mail: caveline@club-internet.fr

A 72-yr-old patient was to undergo a left lumbar plexus block by the posterior approach to achieve postoperative analgesia after hip replacement. The block failed after three unsuccessful attempts to identify nerve structures and a fascia iliaca compartment block was performed. Postoperatively the patient received enoxaparin and then phenylindandione for thromboprophylaxis. She was re-admitted 2 weeks after surgery because of a lower limb motor deficit and a left retroperitoneal haematoma requiring blood transfusion. Clinicians need to be aware of this potential complication of lumbar plexus block in patients receiving thromboprophylaxis.

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Lumbar plexus block is used to provide anaesthesia and postoperative analgesia after total hip replacement (THR).¹ As a result of immediate and delayed postoperative risk of venous thrombosis, THR justifies prolonged thromboprophylactic treatment. Anticoagulant administration is certainly a risk factor for haematoma complicating regional anaesthesia, especially after spinal or epidural anaesthesia.² Such a complication may also occur after performing a deep peripheral nerve block such as lumbar plexus block.

Case report

A 72-yr-old woman (89 kg, 167 cm) was to undergo THR and has given permission for this case report. She had a history of several gynaecological and abdominal surgical procedures complicated by popliteal venous thrombosis and pulmonary embolism. Heterozygous Leyden mutation was diagnosed and the patient was treated with phenylindandione. This treatment was stopped 5 days before surgery and enoxaparin 60 mg twice daily was substituted, and withheld 24 h before surgery. Preoperative laboratory data included haemoglobin of 14.9 g dl⁻¹, platelet count 197·10³, prothrombin time (PT) of 95% with an international normalized ratio (INR) of 1.2, activated partial thromboplastin time of 35 s (normal 35–40 s) and a normal renal function.

Lumbar plexus block was performed via the posterior approach before the induction of anaesthesia, with a 100 mm, 21-gauge short bevel needle connected to a nerve stimulator (Stimuplex®, B.Braun Melsugen AG, Germany). The patient was placed in the right lateral decubitus Sims’ position. The needle was inserted 4.5 cm lateral to the midline at the level of the iliac crest. The neurostimulator was set at a 2 mA current intensity and 1 Hz frequency. The needle was advanced over 8–9 cm along a line parallel to the spinous processes midline. Because of the lack of contact with the

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transverse process of L4, the needle was walked on twice in a more cephalad direction but because of persisting failure to detect the lumbar plexus nerve structure with the 2 mA stimulation current and the lack of bony contact, the technique was abandoned. As the point of local anaesthetic solution injection was not reached, aspiration test through the needle was not performed.

The patient was turned supine and a fascia iliaca compartment block was performed at the first attempt with an 18-gauge Tuhoy needle. Thirty millilitres of ropivacaine 0.75% with 150 μg of clonidine, were injected slowly. Anaesthesia was then induced with propofol 1.5 mg kg\(^{-1}\), sufentanil 0.3 μg kg\(^{-1}\) and cisatracurium 0.1 mg kg\(^{-1}\), and maintained with sevoflurane and nitrous oxide 50% in oxygen. Surgery lasted 1 h and the patient was extubated 10 min after further skin closure.

She received i.v. morphine 6 mg in the recovery room and received morphine by patient-controlled analgesia. Anticoagulation was initiated 14 h later with enoxaparin 40 mg and then 60 mg once daily on the second postoperative day (POD 2) when the patient was allowed to mobilize. Haemoglobin was 11.4 g dl\(^{-1}\) on POD 2 with a normal platelet count (170x10\(^3\)). She received phenylindanedione on POD 3 and enoxaparin was stopped on POD 7 (INR 2.7). She was discharged on POD 10 after a normal Doppler examination of lower limb veins.

On POD 17, she complained of progressive left leg motor deficit and left lumbar back pain. Physical examination disclosed extensive ecchymosis on the left side on her back with sensory and motor deficit in the distribution of the femoral nerve and the lateral cutaneous nerve of the thigh. Haemoglobin was 7.9 g dl\(^{-1}\), platelet count 282x10\(^3\) and INR of 3.5. A CT-scan demonstrated a large left retroperitoneal haematoma with anterior displacement of the left kidney and diffusion of the haematoma into the left psoas and iliac muscles (Fig. 1). Three units of packed red cells and vitamin K 5 mg were administered. The next day, INR was 1.3 and haemoglobin had increased to 11 g dl\(^{-1}\). Motor function started progressively to recover on POD 19 and recovery was complete on POD 45.

**Discussion**

Complications of lumbar plexus block have been related to epidural or spinal extension of the block or to i.v. injection of local anaesthetic.\(^1\)\(^2\) Weller and colleagues\(^5\) have recently described two cases of delayed retroperitoneal haematoma after lumbar plexus block. In the first case, the haematoma was diagnosed on POD 4 but vessel trauma was suggested by blood aspiration after catheter placement. Enoxaparin 30 mg twice daily had been started 40 h after insertion of the catheter in this case. In the second case, no apparent vessel trauma was noted during needle stimulation and heparin was started 8 h later, clinical symptoms of retroperitoneal haematoma occurred on POD 3.

A case of retroperitoneal haematoma has also been reported previously in a patient who received enoxaparin at the time of the anaesthetic procedure and had the block performed after several attempts.\(^6\) In that case the diagnosis of retroperitoneal haematoma was performed after a delay of 9 days, despite the occurrence of lumbar pain on POD 1. In the current case, the patient was not anticoagulated when the block was performed but, as a result of difficulties in nerve structures identification, the needle was inserted several times and this likely resulted in an undiagnosed vessel trauma. As a result of the prolonged delay, the anticoagulant may have been the sole cause of the problem, but the site of bleeding corresponded to the site of needle placement and the retroperitoneal haematoma was maximum at the level of the fourth lumbar vertebrae. Thus, both anticoagulant and lumbar plexus block probably contributed to the occurrence of the haematoma.

![Fig 1](image-url) Left retroperitoneal haematoma and psoas–iliac muscles distension associated with left kidney displacement.
References
5 Weller RS, Gerancher JC, Crews JC, Wade KL. Extensive retroperitoneal hematoma without neurologic deficit in two patients who underwent lumbar plexus block and were later anticoagulated. Anesthesiology 2003; 98: 581–5

Management of severe postpartum haemorrhage by uterine artery embolization

L. Wee1*, J. Barron2 and R. Toye3

1Middlesex Hospital, Mortimer Street, London W1T 3AA, UK. 2Kings College Hospital, Denmark Hill, London SE5 9RS, UK. 3Medway Maritime Hospital, Windmill Road, Gillingham, Kent ME7 5NY, UK

*Corresponding author. E-mail: weeliangh@yahoo.co.uk

We report a case of postpartum haemorrhage which was successfully treated by embolization of the uterine artery. This technique is not well known and is thought to be underused in this condition. We wish to alert medical personnel to its role in this life-threatening situation.

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Obstetric haemorrhage is a significant cause of maternal morbidity and mortality throughout the world.1 Causes of haemorrhage include uterine atony, retained products of conception, and lacerations to the birth canal. If local measures to control bleeding are unsuccessful, bilateral uterine or hypogastric artery ligation may be attempted. However, because of the extensive collateral circulation in the pelvis, the success rate of this approach may be as low as 42%.2 Hysterectomy often follows, but there are reports of persistent bleeding after both arterial ligation and hysterectomy.3 Arterial embolization may be performed as alternative management. We present this case report to highlight its use in cases of severe postpartum haemorrhage.

Case report
A 33-yr-old primigravida ASA I woman was admitted in early labour at 39 weeks gestation. After 16 h, an epidural provided good pain relief and a Syntocinon infusion was commenced to augment labour. Four hours later, delivery by Caesarean section was planned for failure to progress. A live male infant was delivered and Syntocinon 10 U was given as a bolus followed by an infusion of 40 U in 500 ml 0.9% saline over 4 h. The operation was completed uneventfully and blood loss was estimated at 500 ml.

In the recovery room, the patient was initially stable with a blood pressure of 110/50 mm Hg, pulse 90 per min, $\text{SpO}_2$ 99% and respiratory rate 22 b.p.m. Later she complained of feeling light-headed and observations showed a blood pressure of 80/45 mm Hg, pulse 125 per min, $\text{SpO}_2$ 94% and respiratory rate 30 b.p.m. She became very pale and sweaty, so Gelofusine 1000 ml and blood 2 U were given, to good effect. However, 1 h later, the patient again experienced the same symptoms with accompanying hypotension. A further 2 U of blood were given and examination of her abdomen revealed some distension, but it was soft and not tender.

Based on her clinical symptoms, she was assumed to have bled intra-abdominally and was taken back to the operating theatre for a laparotomy. With appropriate monitoring, general anaesthesia was induced using a rapid sequence

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