Bilateral extensive skin necrosis of the lower limbs following prolonged epidural blockade

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Skin necrosis has not been described as a complication following epidural sympathetic blockade. We report a case of bilateral extensive skin necrosis of the lower limbs after a 48-hour lumbar epidural blockade in a 71-year-old patient with right hemiplegia and mitral valve regurgitation, without any preoperative clinical evidence of peripheral vascular disease or diabetes, who underwent transurethral prostatectomy.

Patients with occlusive vascular disease often undergo chemical or surgical sympathectomy in those cases in which it is not appropriate to perform vascular reconstruction in order to promote healing of ischaemic cutaneous ulcers and to relieve rest pain.¹ However, the clinical results of both surgical and neurolytic sympathectomy are uncertain.^{2,3} Indeed these procedures lead to a redistribution of the blood flow in the lower limbs from the muscle to the skin, with a concomitant fall of the regional resistance, mainly in undamaged vessels. The blood flow will be diverted into this part of the vascular tree, so that a "stealing" of the blood flow may occur.

The same events could occur following lumbar epidural anaesthesia in patients with lower limbs vascular disease and undergoing elective surgery. Hypotension, the position of the patient during and after surgery, local compression for the use of straps on restrained parts could precipitate ischaemic consequences.

We observed, and here describe, a bilateral extensive skin necrosis of the heels following prolonged epidural

blockade in a patient with diffuse vascular disease undergoing transurethral prostatectomy.

Case report

A 71-year-old man with a right hemiplegia which occurred three years after a cerebral stroke, with paranoid dementia and mitral valve regurgitation, was scheduled for transurethral prostatectomy. The patient had no clinical evidence of peripheral vascular disease or diabetes. Chest x-ray showed left atrial enlargement. The ECG showed sinus rhythm and left ventricular hypertrophy. Medication at the time of surgery included thioridazine 50 mg and digoxin 0.25 mg daily.

Thirty minutes before surgery atropine ($0.015 \text{ mg} \cdot \text{kg}^{-1}$) and morphine ($0.07 \text{ mg} \cdot \text{kg}^{-1}$) were given intramuscularly as premedication. Upon arrival in the operating room, the patient was calm and cooperative, his blood pressure was 150/90 mmHg, pulse rate was 100 beats $\cdot \text{min}^{-1}$ and regular.

Continuous epidural blockade was established, using an Epidural Mini Pack® (Portex, Hythie, England), with a 1.3 mm Tuohy needle inserted at the second lumbar vertebral interspace. Ten minutes after a 15 mg test dose of 0.50 per cent bupivacaine without epinephrine, a 60 mg bolus dose of same local anaesthetic solution was injected; then a 0.9 mm catheter was inserted through the needle and advanced 2 cm. The other 25 mg of 0.50 per cent bupivacaine without epinephrine were then administered. The total dose of bupivacaine was sufficient to produce anaesthesia to pinprick from the T₁₂ to the S₁ dermatomes. Lactated Ringer's solution (1,000 ml) was given intravenously. Monitoring included blood pressure (automatic blood pressure monitor, EME/LOT, Brighton, England) and ECG. The patient was placed in the lithotomy position, without other form of retention, and surgery started. He remained in this position during the 90-minute uneventful procedure. No sign of ECG changes or hypotension was noted before, during and after surgery. The blood loss was about 300 ml.

The patient was taken to Recovery Room in good

Key words

COMPLICATIONS: skin necrosis; CARDIOVASCULAR SYSTEM: peripheral vascular disease; ANAESTHETICS, REGIONAL: epidural.

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condition. During the first 48 hours postoperatively he received 3,000 ml·day⁻¹ isotonic intravenous solutions. To assure analgesia, 50 mg of 0.25 per cent bupivacaine without epinephrine were injected through the epidural catheter every six hours. The patient was placed on an anti-decubitus air fluidized system and his position changed every two hours, to avoid positional pressure effects. After 48 hours the epidural catheter was removed.

The day after the surgery, bilateral ulcers appeared on the calcaneal and malleolar surface of both legs progressively developing into a clinical status of necrosis (Figure). Digital angiography was performed, showing a hypoperfusion state in the right and left posterior tibial arteries without other abnormalities of both right and left iliac and femoral tree. Seven months later only dystrophic lesions of the skin were present.

Discussion

The loss of sympathetic tone induced by subarachnoid or epidural neural blockade causes profound vasodilation and redistribution of blood flow in the legs.⁴⁻⁷ The skin blood flow, regulated by vasoconstrictive sympathetic fibres, increases; the muscle blood flow, being automatically regulated according to muscle metabolism, should not be affected by sympathetic blockade at rest, when metabolic requirements are low. Experimentally, a significant reduction of blood flow in this area has been found, probably caused by contemporary skin vasodilation.^{8,9} In the patient with vascular disease the consequence of this redistribution is a dramatic fall in muscular blood flow, down to 30 per cent than that before sympathetic blockade.^{4,5} For these reasons it is difficult to predict the effect of a sympathetic blockade in patients with diseased vascular system: indeed only 42 per cent of the patients with ischaemic ulcers of the lower limbs had some improved healing effect after sympathetic blockade.¹⁰

Another reason of these unpredictable results could be the presence of arterial obstructions: a widespread sympathetic blockade will diminish the regional resistance mainly in the branches of vascular tree with undamaged vessels, so that the blood flow will be diverted from the restricted branches into the undamaged parts with a "stealing" mechanism.⁵

Another possible pathogenetic factor of both calcaneal and malleolar skin necrosis in our patient with mitral valve disease could be a thromboembolic event. However, no arrhythmic episodes were detected during the 48 hours epidural blockade; digital angiography did not show any evidence of hypoperfusion or occlusions in the vascular tree of lower limbs besides those previously described in both left and right posterior tibial artery.

We may consider another possible aetiological factor: the lithotomy position during surgery. A case of bilateral

FIGURE Ischaemic ulcers on malleolar and calcaneal surface of both legs.

gangrene following a very long surgical procedure in this position has been previously described.¹¹ On the other hand in patients with advanced arterial disease even a shorter period of extrinsic compression of the popliteal space could have precipitated a previously borderline state of arterial blood flow.

In summary lower limbs haemodynamic changes of lumbar epidural sympathetic blockade should be carefully evaluated in patients with suspected peripheral vascular disease scheduled for surgical procedures needing lithotomy position.

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Résumé

La nécrose cutanée n'a pas été encore décrite comme complication suite à un blocage sympathique épidural. On rapporte ici le cas d'une nécrose cutanée importante 48 heures après un blocage épidural chez un patient de 71 ans atteint d'hémiplégie droite, et de régurgitation valvulaire mitrale sans évidence de diabète ou d'atteinte vasculaire périphérique.