Short report

Acute bilateral anterior tibial compartment syndrome after Caesarian section in a diabetic

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SUMMARY An acute bilateral anterior tibial compartment syndrome is described in a young diabetic after Caesarian section with subsequent recovery. A vascular aetiology appears most likely.

Acute inflammation of the muscles of the anterior compartments of the legs of ischaemic origin is a well-recognised syndrome usually following trauma or prolonged unaccustomed strenuous use of these muscles, nearly always in males. A variety of direct and indirect vascular causes have also been described.¹ I report here the syndrome occurring in an otherwise healthy diabetic after an uncomplicated Caesarian section.

Case report

A 29 year old white primigravida with an 11 year history of uncomplicated well-controlled insulin-dependent diabetes mellitus was admitted for induction of labour at 38 weeks of pregnancy. Prostaglandin E₂ gel 375 µg in tylose was inserted extra-amniotically and the next day artificial rupture of membranes was performed together with the addition of an intravenous oxytocin drip (5 units in 500 ml 5% dextrose). She received epidural anaesthesia (total dose 15 ml 1% lignocaine) under which lower segment Caesarian section was performed after five hours because of failure to advance in the first stage of labour. She was delivered of a healthy male infant of weight 3.44 kg. She then received the following drugs: ergometrine 0.5 mg, droperidol 10 mg, fentanyl 0.1 mg, perphenazine 5 mg, and papaveretum 10 mg, the last mentioned being repeated

postoperatively. There were no immediate pre- or postoperative complications.

The next day the patient had a transient fever of 38° C, and she noted tenderness, pain, and swelling of the anterior compartments of both legs, greater on the left, and numbness of the dorsal surfaces of the feet. She was unable over the next few days to walk because of the pain caused by weightbearing. There were signs of superficial inflammation in the affected areas, and a provisional diagnosis of streptococcal cellulitis was made for which she received benzyl penicillin. Neurological examination was not performed at this stage. Nine days later, after much of the pain and inflammation had subsided, it was realised that she had bilateral foot drop.

Examination at this time showed mild redness, tenderness, and oedema of the lower half of the left anterior compartment with mild tenderness only on the right. The foot pulses were easily palpable and, as throughout her admission, she was normotensive. Neurological abnormalities were confined to the legs. There was bilateral wasting of extensor digitorum brevis. Power was normal proximally. On the right there was mild weakness of dorsiflexion of the foot and moderate weakness of extension of the great toe, other movements being normal. On the left there was moderate weakness of dorsiflexion, mild weakness of inversion and of toe extension, and marked weakness of extension of the great toe. Dorsiflexion, inversion, and eversion were all painful but the power of eversion was considered to be normal. Tendon reflexes were symmetrical but the ankle jerks were less brisk than the knee

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jerks. There was anaesthesia over the dorsum of the great and second toes extending to the medial aspect of the dorsum of the foot on the right, with a similar abnormality extending proximally to just above the ankle on the left. There was no other sensory deficit.

INVESTIGATIONS

Routine antenatal haematological, serological, and biochemical tests were normal. Postoperative skin swabs were sterile but culture of midstream urine showed a growth of Coliforms. Three days postoperatively, haemoglobin was 13.4 g/dl with WBC $11.2 \times 10^9/l$.

Further postoperative investigations at nine days, some of uncertain significance because of the recent surgery, showed normal haemoglobin, WBC, and platelet count with ESR 110 mm/hr. Serum CPK was 215 IU/l (upper limit 140) and serum hydroxybutyrate dehydrogenase 321 IU/l (upper limit 300). Myoglobin was not detected in urine or plasma. Serum urea, electrolytes, cholesterol, proteins and thyroxine, and chest radiography were normal. Nine days later, ESR had fallen to 21 mm/hr and the serum enzymes were normal.

Electromyography (10 days after operation) disclosed a distal latency to the right extensor digitorum brevis (surface electrode) of 5 ms. Right lateral popliteal motor conduction velocity was 42 m/s. No muscle action potential was recordable with surface electrode over the left extensor digitorum brevis. Distal latency to the tibialis anterior (90 mm below the stimulation site) was 3.5 ms on the left, and 3.2 ms on the right. Sampling with concentric needle electrode showed a normal pattern in the right tibialis anterior. The right extensor hallucis longus showed no spontaneous activity but brief motor unit potentials of reduced amplitude (up to 1 mV) in a reduced interference pattern. The left tibialis anterior showed no spontaneous activity but prolonged insertion activity in several areas, and brief and polyphasic units of reduced amplitude (up to 0.5 mV) in a reduced interference pattern. The left extensor hallucis longus was electrically silent. Amplitudes of sural nerve sensory action potentials were 19 μV (right) and 18 μV (left).

CLINICAL PROGRESS

Improvement occurred without specific treatment. Three weeks after operation there was mild residual tenderness and induration on the

left with only mild to moderate muscle weakness, signs on the right being very mild. After six weeks there was minimal weakness of great toe extension on the right and of dorsiflexion on the left, on which side great toe extension remained moderately weak.

The patient was reviewed by another physician after a further month and was reported to be normal.

Discussion

This patient had no evidence of diabetic vascular or neurological complications. The symptoms and signs are attributable to acute inflammation of the anterior compartment muscles and nerve lesions are confined to these regions. Both compartments were more severely affected distally. Both anterior tibial nerves were affected, and the more extensive sensory loss on the left suggests that the medial cutaneous branch of the superficial peroneal nerve was involved. Although EMG studies were confined to the lower limbs, there was no evidence of a generalised neuropathy and the changes in the affected muscles were those of a myopathy.

Although unlikely, local infective inflammation cannot be excluded as a cause. Local trauma or muscular exertion do not appear to be relevant especially as the labour was not allowed to progress. These two factors were presumably responsible for the lesions described after prolonged tetany² and post-partum eclampsia.³ The latter case, the sole report I have been able to find of the syndrome complicating pregnancy, was delivered vaginally, received 0.5 mg ergometrine, suffered four fits, and then developed a unilateral lesion. Likewise, a focal drug-induced myopathy seems unlikely although it is noted that ergotamine has been reported to cause an ischaemic lateral popliteal nerve lesion.⁴

Anterior compartment syndromes may arise from a variety of vascular causes. Commonly, these are either embolic or arteriosclerotic⁵ but vascular trauma or arteritis may also be responsible.^{6,7} Interestingly, the site of the causative lesion may be considerably proximal to the origin of the anterior tibial artery, at either knee or pelvis.^{7,8}

This patient underwent uncomplicated pelvic surgery. There was no record of hypotension, although there were several predisposing causes (epidural anaesthesia, blood loss, and general anaesthetic drugs). She received the vasoconstrictive drug ergometrine. The cause of the

muscle damage is unclear but an ischaemic aetiology is most likely and to this several factors may have contributed. She made an unusually complete recovery.

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