Compartment Syndrome due to Reperfusion Injury

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ABSTRACT

Theoretically, tissue oedema due to reperfusion injury may be severe enough to cause compartment syndrome. One such rare case is presented.

INTRODUCTION

In 1963, Fogarty and co-workers introduced the inflatable balloon embolectomy catheter which made thrombus extraction possible under local anaesthesia via a single incision in a large vessel. The procedure, however, is not without complications such as rethrombosis, myonephropathic metabolic syndrome (Haimovici et al., 1975) and arterial injury secondary to the balloon catheter (Dobrin, 1989).

Arterio-venous fistula and the development of compartment syndrome secondary to vessel wall perforation represent 13.6% of clinical complications of balloon embolectomy (Schwertzer et al., 1976). However, compartment syndrome due to reperfusion without clinical evidence of vessel wall perforation, a very rare complication, is reported here.

CASE REPORT

In January 1996, a 51-year-old diabetic female, with ischaemic heart disease, presented with a 21-hour history of sudden onset pain in the left hip radiating down the leg. The pain was constant with increasing severity and associated with numbness and weakness. Her left lower limb was pale, cyanosed and cold to palpation from the knee downwards. The femoral pulse on the left was palpable and regular but the popliteal, posterior tibial and dorsalis pedis pulses were absent.

A diagnosis of acute arterial occlusion secondary to embolism was made. Four hours later, a left femoral embolectomy was performed. A Fogarty catheter, passed to the knee level, removed a large embolus from the superficial femoral artery. Immediately, the popliteal, posterior tibial and dorsalis pedis pulses on the left leg returned and were easily palpable. There was no swelling of the left leg or foot at this time.

13 hours later, the patient complained of severe left leg pain. Examination revealed a very tense left leg with pain on dorsiflexion of the left foot. All compartments of the leg were uniformly tense. The peripheral pulses remained palpable but were now weaker than the right. The patient was noted to be passing dark-coloured urine which tested positive for myoglobin. The diagnosis of compartment syndrome was made. Fasciotomy was performed in all compartments via medial and lateral incisions in the left leg. No evidence of haemorrhage was

found in any compartment to suggest vascular injury. Postoperatively, the pain relief was dramatic, the swelling subsided and the pulses remained normal. The incisions started healing by secondary intention and they were closed 14 days later. The patient was discharged 17 days post-embolectomy with complete limb salvage. She has no residual disability.

DISCUSSION

Vessel wall injury is best demonstrated by intraoperative arteriography at the completion of embolectomy (Dobrin, 1989). This was not available for our case.

The presence of good popliteal, posterior tibial and dorsalis pedis pulses post-embolectomy suggests that vessel perforation was unlikely. In addition, the presence of significant myoglobinuria, the absence of blood clot or haematoma on fasciotomy, and the uniformly increased tension in all compartments of the leg suggest that single vessel injury did not occur. Further, one passage of a Fogarty catheter to knee level was sufficient to remove a large embolus and restore pulsation, thus minimizing the risk of vessel damage. Therefore, the likely cause of the compartment syndrome was tissue swelling and increased interstitial fluid volume following reperfusion.

Reperfusion injury can occur when ischaemia persists for three hours or more. There are two major factors responsible for reperfusion injury. The first is the generation of oxygen-free radicals by the introduction of oxygen to ischaemic cells. The second appears to be the "no reflow phenomenon" (Ames III et al., 1968) caused by narrowing of the vascular channels secondary to compression by swollen cells.

The resultant increased capillary permeability, swelling and skeletal cell damage can possibly lead to compartment syndrome with interstitial tissue pressures of 60 mm Hg or greater. The use of hypertonic mannitol (Buchbinder et al., 1981) offers some hope in the prevention of reperfusion injury and hence the reduced requirement for fasciotomy.

Compartment syndrome is easily recognised. Deep throbbing pain exacerbated by passive stretching of muscles, hyperaesthesia in the distribution of nerves in the compartment and tenseness of fascial boundaries of the compartment are all classical features (Matsen et al., 1980). The diagnosis is considerably strengthened by the presence of myoglobinuria.

Careful clinical assessment and early fasciotomy of all compartments can lead to salvage of an affected limb. The prognosis is better if compartment syndrome is recognised early and treated promptly before all peripheral pulses are lost.

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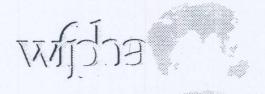
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