Managing vascular impairment following orthopaedic injury

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Orthopaedic trauma is associated with vascular impairment in approximately 1% of cases, complicating management of the injury, particularly to the shoulder, elbow, pelvis and knee. Doctors treating trauma would be expected to detect and manage several such patients a year.

Vascular impairment complicates approximately 1% of fractures or dislocations compromising a patient's recovery (Rockwood et al, 1996). Early revascularization, judicious fasciotomies of soft tissue compartments, vein grafting for the arterial injury and repair of at least one major vein have all been shown to improve the outcome and reduce the amputation rate following major vascular injury to the limbs (d'Sa, 1982).

A number of authors have stressed the importance of training orthopaedic and other accident surgeons in techniques to repair vascular injuries associated with long bone fractures (Bonney, 1963; Birnstingl, 1982; Birch et al, 1998). The popularization of the acute response trauma team within hospitals has increased the need for the on-call surgeon to be able to control haemorrhage and repair injured blood vessels. Spasm and intimal flaps develop in a vessel as a result of blunt trauma to the limb (De Mello and Khan, 1990). Wall defects, vessel transection and arteriovenous fistulae follow penetrating trauma.

Injuries to the knee, shoulder and elbow joint are particularly associated with arterial or venous lesions and predispose the patient to the development of compartment syndrome and subsequent vascular impairment.

Figure 1. Proximal humeral fracture to axillary artery injury.

SHOULDER DISLOCATION

The proximity of the axillary artery to the humeral head predisposes this artery to damage during anterior dislocation of the shoulder or fractures of the proximal humerus (*Figure 1*). The majority of injuries occur to the third part of the axillary artery (Byrd et al, 1998) distal to the lower edge of pectoralis minor muscle (*Figure 2*). Milton (1954) proposed that the axillary artery is fixed in that location by the anterior and posterior circumflex arteries as well as by the subscapular artery, exposing it to direct injury by the hyperabducted humeral head. Others feel that the pectoralis minor itself acts as a fulcrum to injure the axillary artery directly (Gibson, 1962; Brown and Navigato, 1968). Patients with this type of injury tend to be over 50 years of age and some authors have suggested that pre-existing atherosclerotic changes in the artery predispose to damage during shoulder dislocation (Hawkins et al, 1986). Late closed reduction of shoulder dislocations results in a high rate of arterial injury.

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THE ELBOW

In children supracondylar fractures account for 65% of fractures around the elbow (*Figure 3*) (Rockwood et al, 1996). Vascular injury is believed to occur in 0.5–1% of cases (Wilkins et al, 1996), but figures of up to 38% have been reported (Campbell et al, 1995). The brachial artery forms the lifeline to the forearm and hand and may be injured by simple compression, spasm, an intimal tear or rarely complete rupture. Arterial spasm is usually self limiting and disappears within a few minutes following relief of the external local stimulus. In the treatment of trauma, any persistent ischaemia should be considered the result of arterial injury and not the result of spasm. If following reduction of the fracture or dislocation at the elbow distal ischaemia persists then it should be assumed that the artery is damaged or thrombosed (Birch et al, 1998).

THE PELVIS

Pelvic fractures occur as the result of high energy trauma and are frequently associated with other major skeletal, thoracic and abdominal injuries. The mortality rate following a pelvic fracture has been recorded as high as 20% and a quarter of victims of fatal road traffic accidents have a pelvic fracture (Tile, 1996). Massive haemorrhage may result from the pelvic fracture where the integrity of the pelvic ring has been disrupted leading to a displaced unstable fracture (*Figure 4*). Blood loss after an unstable fracture can be as high as 15 units (McMurtry et al, 1980). Where a patient remains haemodynamically unstable despite aggressive fluid resuscitation the pelvis needs to be closed as an emergency measure to tamponade the blood loss using an external fixator (*Figure 5*).

Figure 2. Angiogram showing occlusion of the third part of the axillary artery following proximal humeral fracture.

Figure 3. A supracondylar fracture in a child that impinged upon the brachial artery.

Figure 4. An open book fracture requiring surgical stabilization.

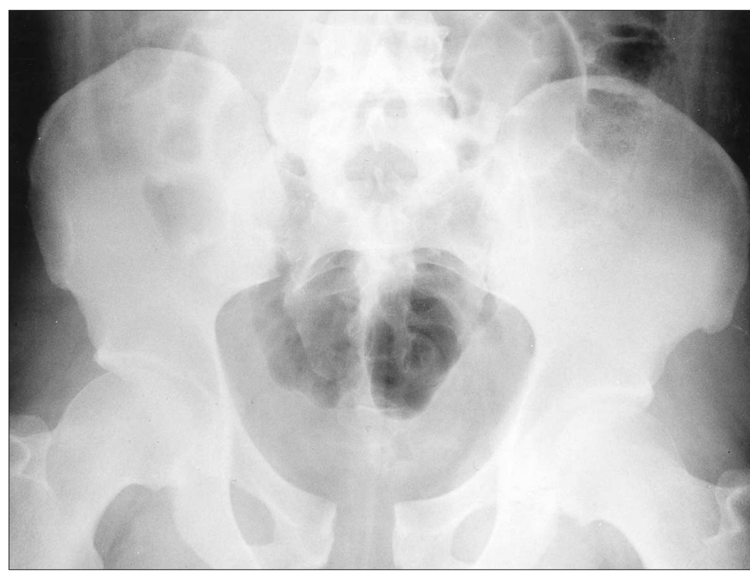
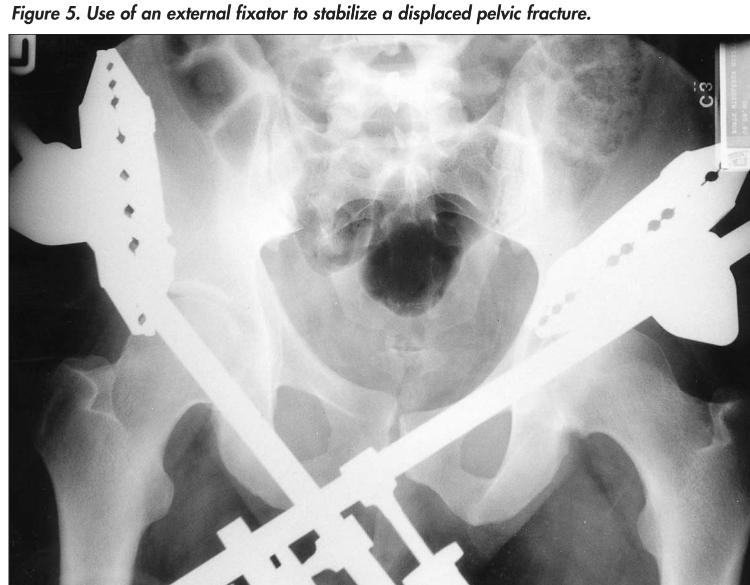


Figure 5. Use of an external fixator to stabilize a displaced pelvic fracture.



If haemorrhage does not abate angiography and embolization may be indicated as bleeding can continue from small-bore arteries such as the gluteal and obturator artery. In a patient with an unstable pelvic fracture laparotomy is ill advised without the application of an external fixator.

THE KNEE

There are four injuries associated with arterial compromise (Birch et al, 1998): arterial rupture occurs in 3% of supracondylar fractures of the femur, in 30% of dislocations of the knee (*Figure 6*), and in 10% of proximal tibial fractures. In addition, fractures of the proximal part of the fibula are associated with damage to the anterior tibial artery.

The arrangement of the popliteal artery, its branches and collateral vessels makes it vulnerable to injury and complicates the repair. Technical difficulties of repair increase with the more distal injuries, particularly around the trifurcation. Proximal tibial fractures are often high energy and associated with gross injury to the skin, muscle and nerve.

ASSESSMENT

Clinical presentation

Arterial insufficiency leads to the development of pallor, pulselessness, paraesthesiae, paralysis and pain in the involved limb. Complete transection from a penetrating injury is often dramatic causing massive haemorrhage and leading to haemorrhagic shock. The presence of an expanding haematoma, thrill or bruit confirms the need for urgent surgical intervention.

Figure 6. Dislocation of the knee.

The location of a wound, a history of penetrating trauma, fracture pattern, nerve injury, non-pulsatile haematoma over an artery, and delayed capillary refill greater than 2 seconds, all increase the suspicion of a vascular injury.

Spasm, hypotension and platelet aggregation occur as a result of arterial injury helping to reduce blood loss. It is dangerous to presume that compromise is a result of post-traumatic arterial spasm. Absent pulses are often associated with intimal damage and intraluminal thrombosis, which may only be excluded by arteriography or exploration. Early involvement of a surgeon trained in vascular injury assessment and treatment is required when symptoms and signs of arterial insufficiency are detected following injury.

Investigations

In the absence of palpable pulses the quickest test of perfusion to the limb is with a Doppler flow probe (Shaw et al, 1990). A value for the ankle/brachial index less than 0.9 is indicative of an arterial injury.

Duplex ultrasound is a more sophisticated technique which is very accurate, but not always readily available. The 'gold standard' for determining the presence and site of a lesion causing arterial insufficiency remains angiography, which can be undertaken preoperatively in the radiology department or in theatre. Digital subtraction images are available in the angiography suite but these may be time consuming and unavailable out of hours.

The pulse oximeter can be misleading and unreliable because collateral circulation can maintain distal skin perfusion (Wilkins et al, 1996).

MANAGEMENT

Initial treatment

Immediate control of acute massive haemorrhage by the application of direct pressure forms part of the primary survey when resuscitating a multiply injured patient. Tourniquet application is rarely required as a life- or limb-saving measure. Definitive control is achieved in theatre following exclusion of other life-threatening injuries. Reduction of fractures and dislocations is required in the presence of deformity and an absent peripheral pulse.

In the case of a supracondylar elbow fracture in a child where there is clinical suspicion of a vascular injury which does not respond to reduction of the fracture, further investigation is required. A Doppler can be used to compare the waveform in each limb. Urgent surgical exploration should be undertaken if the clinical signs are obvious or if no facilities for appropriate investigation are available. Shaker et al (1976) based their decision for vascular exploration on clinical signs only in 75% of patients. There is no role for the pulse oximeter as collateral circulation can maintain skin perfusion while the forearm muscles are deprived of adequate perfusion leading to permanent ischaemic damage and ultimately to Volkmann's contracture.

More distal arterial injuries in the upper limb and below the knee, associated with single vessel occlusion, may be managed non-operatively if there is adequate peripheral circulation. This is usually determined by assessing the capillary return. Regular monitoring is mandatory.

Operative treatment

Skeletal stabilization is usually required before vascular repair to prevent damaging the repair during subsequent treatment of the fracture or dislocation. Immediate revascularization may be required using temporary shunts where surgery is delayed beyond 4–6 hours to prevent permanent ischaemic damage to the inadequately perfused distal musculature (Eastcott, 1992).

Surgical repair follows proximal control with vascular clamps and resection of the damaged vessel. Access to the whole limb may be required in order to obtain adequate control. Approaches to the subclavian vessels may involve division but never removal of the clavicle (Rockwood, 1993). A deltopectoral incision allows access to the axillary and brachial artery lower down, with a lazy 'S' incision being used at the elbow. A transverse incision in the groin enables access to the femoral vessels.

Repair is achieved via a number of techniques (direct repair; end to end anastomosis; or end to side anastomosis) using a 6/0 non-absorbable suture to construct a tension free anastomosis (Eastcott, 1992). A vein graft patch may be required to avoid tension and repair defects. A reversed vein graft is preferable to a prosthetic graft when repairing defects in the vessel, especially in the presence of a contaminated wound. Embolectomy, local heparinization and flushing help to restore flow in the injured vessel. Fasciotomies may be required to prevent the development of compartment syndrome.

COMPARTMENT SYNDROME

Increased soft tissue pressure within an enclosed soft tissue compartment of a limb can lead to devastating muscle necrosis, contracture, functional impairment and nerve damage secondary to compromise of the microcirculation within the compartment. The most common causes include fractures of the tibia and soft tissue crush injuries. Pain is the earliest indication of elevated compartment pressure in the conscious patient and the most reliable clinical sign is increased pain on passive stretch of the involved muscle group.

Suspicion is often enough to warrant surgical exploration, but where findings are equivocal the limb should be elevated and reassessed regularly. In the obtunded patient diagnosis is difficult and compartment pressure monitoring may be required. The normal pressure within a compartment is 0 mmHg. Pressures within 20–30 mmHg of the diastolic pressure or with an absolute value above 40 mmHg require surgical decompression by fasciotomy within 4 hours of the onset of symptoms (Pellegrini et al, 1996).

Poor outcome results from a delay in diagnosis and frequently occurs as a consequence of insufficient awareness of the condition (McQueen et al, 2000). Patients at particular risk include children, hypotensive patients, those with a bleeding diathesis, young men with tibial diaphysis or distal radial fractures, the unconscious, and patients with high energy limb injuries (Matsen et al, 1980; McQueen et al, 2000). Early monitoring of these 'at risk' groups may lead to a reduction in the consequences of this syndrome, which include infection, contracture and amputation.

Initial decompression is achieved by immediate splitting or removal of casts or circular dressings. Failure to respond necessitates surgical decompression by fasciotomy, where the aim is to pre-

KEY POINTS

- Vascular impairment following orthopaedic injuries occurs in 1% of fractures.
- An orthopaedic or accident and emergency specialist would be expected to detect and manage several patients with compromised circulation per year.
- Orthopaedic injuries in certain anatomical regions such as shoulder, elbow, pelvis and knee has a high rate of associated vascular compromise.
- Pain is the earliest indication of compartment syndrome in the conscious patient and the most reliable clinical sign is increased pain on passive stretch of the involved muscle group.

serve viable muscle and neurological function within the limb. The adequacy of the decompression should not be compromised by concerns over cosmesis as the skin may act as a potentially constricting structure. Mubarak and Owen (1977) describe the classic two-incision technique to the lower limb that enables decompression of all four compartments. In the forearm the technique described by Gelberman et al (1978) is used to obtain adequate decompression.

CONCLUSIONS

A high index of suspicion combined with the realization that early treatment improves outcome will enable the trauma surgeon to optimize the care of the vascularly impaired patient with orthopaedic injuries. HM

Conflict of interest: none.

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