CASE REPORT

Lower limb compartment syndrome due to computer tomography contrast infiltration

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Introduction

The development of compartment syndrome can be a devastating complication of, what may otherwise be, minor injury or routinely performed procedures. Little has changed in the possible treatment options available and therefore much of our emphasis is on prevention and early detection. Compartment syndrome following contrast injection is a rare complication of an investigation often considered to be of low risk and thus far has only been described in the upper limb. We aim to highlight a previously undescribed potential cause of compartment syndrome in the lower leg and comment on current suggested precautions to be aware of to avoid this complication after contrast media extravasation.

Case report

An 86-year-old female was involved in a high-speed road traffic accident. She was admitted via the accident and emergency unit with multiple open injuries to the thorax and upper limbs, as well as a cervical fracture. The initial response to fluid resuscitation was transient and a widened mediastinum was suspected on trauma chest radiograph. According to the hospital protocol, a CT scan of the chest with contrast was arranged to assess the mediastinal structures.

One hundred millilitres of non-ionic, water-soluble contrast medium (Ultravist, Berex Laboratories, Berlin, Germany) was injected through a plastic venous cannula on the dorsum of the foot using a rapid injector system at approximately 5 ml/s. Due to the upper limb injuries this was one of the few convenient peripheral veins accessible. The contrast was presumed to have extravasated, as it was not visible on the chest CT scan. The limb at the time was only mildly swollen around the injection site.

Ten hours following CT, large areas of blistering were evident extending from the foot to the knee on the side of contrast injection (Fig. 1). Further examination revealed a tense lower limb. Intracompartmental pressures of the peroneal, anterior and posterior compartments were 52, 50, and 35 mmHg, respectively. The systemic blood pressure at the time of monitoring was 115/57 mmHg. Compartment syndrome was diagnosed, and the patient was prepared for immediate fasciotomy. A preoperative radiograph of the lower leg and foot to exclude an underlying fracture showed no bony injury but obvious contrast in the soft tissues (Fig. 2).

The anterior and peroneal compartments were fully decompressed through an antero-lateral...
incision. The superficial and deep posterior compartments were decompressed through a medial approach. Significant swelling and oedema were noted throughout all the fatty tissue around the lower leg. The anterior and peroneal compartments particularly were bulging and oedematous. The muscles however, were healthy, reactive with no haematoma or necrotic tissue seen.

Two days after the initial fasciotomy the wound was examined under anaesthesia with closure of the medial wound possible and split skin grafting performed to the lateral side. This healed uneventfully.

Discussion

Compartment syndrome is due to increased pressure in a closed fascial space reducing the capillary blood perfusion below the level necessary for tissue viability. This compromise to the circulation will affect all the structures within the involved compartment. The full syndrome, as described by Richard von Volkmann in 1881, includes paralysis and post ischaemic contractures but these late sequelae are now seen infrequently because fasciotomy is usually performed as soon as the diagnosis is made.12

From descriptions in the literature of compartment syndrome occurring in the upper limbs in similar circumstances, we may consider the following as possible explanations to this condition occurring in the leg. A hypertonic solution, usually in the region of six times that of plasma, causes increased tissue oncotic pressure leading to extravasation of intravascular fluid into the extra-vascular space. This causes a rapid exchange of fluid with oedema and worsening occlusion of blood flow.6,19 A second factor is the cytotoxicity of the contrast medium, with conflicting evidence presented in the literature.9 In a laboratory study, Cohan et al. found that extravasated ionic contrast media produced acute inflammation followed by a chronic inflammatory process with a peak at 24–48 h.5 They found that ionic contrast was more toxic than non-ionic agents although Jacobs et al. found no difference.5,7 A factor of greater significance in the upper limb is the volume of the extravasated contrast medium. The majority of reported cases have occurred with large-volume extravasations,18 which may cause mechanical compression.11,15,20 When evaluating skin reactions that occur with low-osmolar contrast agents, it appears the pathogenesis may be related to T-cell mediated reactions. The skin reactions often show features typical of late hypersensitivity including exanthematous rash, positive skin tests, and lymphocyte rich dermal infiltrate with eosinophils.4

However, in this case the contrast media used was Ultravist, which has an osmolality of approximately 1.1 times that of plasma. It is non-ionic, water soluble and easily excreted by the kidneys and we found only one other described case of this being related to compartment syndrome following extravasation.16 Despite this, our case had a predominantly extra compartmental soft tissue reaction with oedema that increased the extra-compartmental...
tal volume. This would suggest a predominantly cytotoxic or hypersensitivity based response to the contrast agent which resulted in a large fluid shift into the extra-vascular space. This was sufficient to compress the muscle compartments and compromise circulation in a similar way to tight dressings or bandaging.

The lower limb has superficial and deep lymphatic vessels with the superficial vessels accompanying the saphenous veins and their tributaries within the superficial fascia. There are a number of communicating vessels connecting the superficial with the deep venous systems. The vessels following the great saphenous vein end in the superficial inguinal lymph nodes and those vessels accompanying the small saphenous vein enter the popliteal lymph nodes. The deep vessels accompany the deep veins and enter the popliteal lymph nodes. The great saphenous vein is formed by the union of the dorsal vein of the great toe and the dorsal venous arch of the foot, while the small saphenous vein arises from the union of the dorsal vein of the little toe and the dorsal venous arch. It is these superficial veins and associated lymphatics involved in the extravasation and following tissue reactions of our case. As the lymphatic drainage of this area is relatively slow and potentially compromised in polytrauma patients, the cytotoxic effects of the contrast media may be prolonged. This may be further compromised by a hypersensitivity response, which may increase the passage of fluid into the extra-vascular space and thereby overwhelm the normal lymphatic drainage.

Extravasation of contrast does occur in up to 70% of cases when foot veins are used for injection, which may suggest greater fragility of these veins or greater resistance to injection. Although compartment syndrome of the hand and forearm secondary to intravenous fluid extravasation has been reported, to our knowledge this has not been reported in the leg. This may be related to the greater ability of the lower limb to compensate for an expansion in volume or to it being a less favourable choice for injection of contrast.

The injector system used to administer the contrast delivers a specific amount of contrast at a regulated pressure of 80 mmHg. Typically 100–150 ml of fluid is administered at 2.5–5 ml/s. The system does not have a pressure sensor or automatic cut-off if increased resistance is encountered and therefore will continue to administer fluid if vessel rupture and extravasation should occur. This case identifies this potential failure in this type of injector system.

The normal compartment pressure in an uninjured extremity is 0–8 mm Hg. The authors consider the criterion of Mubarak and Hagens, of a compartment pressure of 30 mmHg as a guide to make the diagnosis, as unreliable. McQueen and Court-Brown support this. They correctly draw attention to the importance of limb perfusion in interpretation of the compartment pressure and therefore consider the difference between diastolic and compartment pressure to be of more importance. Decompression of the compartment is indicated should there be a difference be less than 30 mmHg, a figure compatible with capillary perfusion pressure. Provided the difference is greater than 30 mmHg, relatively high compartment pressures may be safely observed. McQueen and others make the argument for continuous compartment pressure monitoring as the onset of a compartment syndrome may be delayed and deteriorating pressures may suggest a developing compartment syndrome before clinical symptoms are conclusive. Ultimately the diagnosis in the majority of cases is clinical and treatment should be instigated as soon as the condition is suspected.

Although there is no consensus as to the best way to manage contrast extravasation, the European Society of Urogenital Radiology produced guidelines into the management of contrast media injection in some patients and then to the management of contrast media reactions. Bellin et al. expand on this to suggest ways of detecting, preventing and treating extravasation injuries and highlight the risks of using large volumes of high osmolar contrast agents particularly in foot veins. They propose a conservative approach to the majority of cases, as serious reactions are rare. When serious injury is suspected they acknowledge surgical drainage and options, such as suction as possibilities in the prevention of severe effects. There are no randomized controlled trials to support this.

This case highlights the risks of using peripheral veins for rapid injection and particularly those of the foot for the injection of contrast agents. If this method of injection were used, a large central line, if available, would prove less of a risk. This may be particularly important in those patients at risk, such as those with multiple injuries, the elderly where vein fragility is common and in those debilitated with co-morbidity. If contrast has extravasated we propose that continuous or at least regular monitoring of the compartment pressure be done when clinical assessment is in any way impaired or when compartment syndrome is being considered. Early suspicion and prompt treatment is the best way of ensuring a favorable outcome and early fasciotomy is essential to prevent long-term sequelae when a compartment syndrome is diagnosed.
References