

Short Communication

Compartment syndrome

Arcot Rekha

Sri Ramachandra Medical College and Research Institute, Porur, Chennai, India. Tel: 91-044-22520256.
E-mail: rekha_a@yahoo.com.

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Compartment syndrome is a condition that trauma surgeons are familiar with. A high index of suspicion is needed among clinicians for early recognition and timely referral of this limb and life threatening condition.

Key words: Compartment syndrome, Fasciotomy, Hyperbaric oxygen.

INTRODUCTION

Compartment syndrome (CS) is defined as a condition occurring in a closed anatomical space (commonly an osseo-fascial compartment) where the perfusion pressure falls below the tissue pressure -Rorabeck (1992). The earliest description of increased intracompartmental pressure was by Volkmann (1872) who described contracture of the forearm muscles following a supracondylar fracture (Volkmann, 1872). In 1909, Thomas documented paralysis after muscle contusion and in 1914 Murphy discovered that hemorrhage into the muscles caused pressure to rise, impeding venous flow. He was also the first to suggest that fasciotomy might be effective, although, the first decompression for CS was done by Jepson in 1926 (Jepson, 1926). In 1941, Bywaters and Beall revealed the mechanisms and consequences of CS based on their experience with crush injuries (Bywaters and Beall, 1998). In 1968 the wick catheter technique for pressure measurement was popularized by Owen et al. (1975) and Whitesides and Haney developed an infusion technique using a slit catheter (Whitesides and Haney, 1975). Matsen is credited with the development of guidelines for fasciotomy (Matsen, 1975).

Pathophysiology of compartment syndrome

Any closed anatomic space has a finite volume and can withstand a finite pressure. When there is fluid introduction or any extraneous compression, the intracompartmental pressure rises and this increases the tissue pressure and decreases the intravascular pressure. Tissue perfusion is calculated by subtracting interstitial fluid pressure (IFP) from capillary perfusion pressure (CPP). The rising intracompartmental pressure

causes the venous pressure to rise and the capillaries collapse. This activates histamine like substances that increase vascular permeability and also increase the sludging in the capillaries. The arteriolar spasm is also compounded by the nitric oxide pathway. Muscle ischaemia and infarction ensues and the released myofibrillar proteins are osmotically active, drawing more fluid into the compartment. The tissue pressure rises further, starting a vicious cycle (Mubarak and Hargens, 1983; McQueen and Gaston, 2000). This is outlined in Figure 1. Reperfusion activates neutrophils and the production of free oxygen radicals, producing a simultaneous double hit injury that causes damage and dysfunction of cellular membranes and further acceleration of intracellular and extra cellular edema. Nerve conduction decreases and the switch to anaerobic metabolism occur. Myoglobin release occurs and this progresses to renal failure, contributing to loss of limb and life.

Normal cellular metabolism is maintained with a tissue perfusion of 20 mmHg (CPP 25 mmHg - IFP 5 mmHg). When intracompartmental pressure rises above 30 mmHg, fasciotomy is advocated.

Delta p is a measure of perfusion pressure (diastolic pressure-intracompartmental pressure). When the level is < 30 mmHg there is an urgent need for fasciotomy.

Etiology of compartment syndrome

CS occurs due to increased fluid content or decreased compartment size. The important causes for the former is fracture of the bones, burns, hemorrhage, intra-arterial injection, vascular disruptions, heroin, carbon monoxide intoxication, snake venom, infiltrated infusion, decreased

Pathophysiology of CS

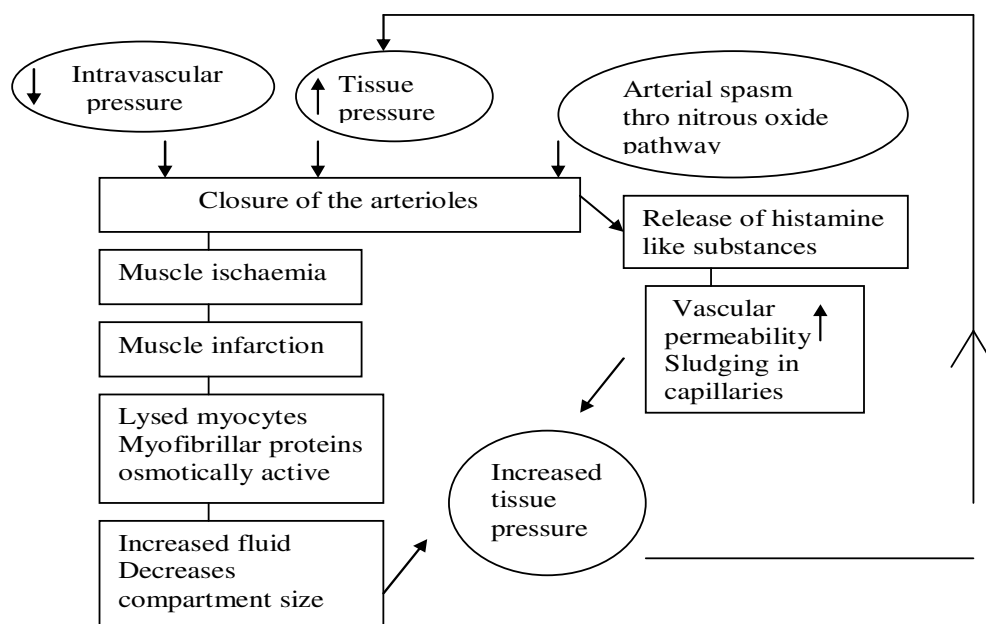


Figure 1. Vicious cycle.

osmolarity, muscle over-use, crush injury, rhabdomyolysis, nephritic syndrome (Tornetta and Templeman, 1997).

Causes for decreased compartment size are burns, casts and military anti shock trousers (MAST). CS can be classified as acute and chronic. Acute CS occurs after surgery, crush injury or steroid abuse and chronic CS (CCS) is caused by repetitive heavy use of muscles as in a runner or athlete (Pedowitz and Hargens, 1990; Hach and Prave, 2000). CCS is also called exertional CS, recurrent CS or sub-acute CS. Pedowitz and Hargens, proposed criteria for diagnosis of CCS requiring one or more of the following:

A resting compartment pressure of 15 mmHg or higher
 A 1 min post exercise compartment pressure of 30 mmHg or higher.
 A 5 min post exercise compartment pressure of 20 mm Hg or higher
 Table 1 delineates the difference between acute and chronic CS.

Clinical features

The patient suffering from CS has severe pain out of proportion to the signs. There is tense, woody feeling in the extremity. The paraesthesia, pallor, poikilothermia, pulselessness described, are often late. The earliest reliable sign is the presence of pain on passive stretching of the muscles. Severe pain and rest pain are also early indicators of CS. Neural compromise then follows with sensory nerves being affected before motor nerves. Decreased two-point discrimination is also an early sign.

MATERIALS AND METHODS

A high index of suspicion is needed for diagnosis. Laboratory studies like blood count, coagulation studies, creatine phosphokinase and urine myoglobin are done.

An X-Ray of the extremity is taken and an ultra sound is useful in evaluating arterial patency, flow and also to exclude deep vein thrombosis. When in doubt we need to measure the compartmental pressure. Several commercial tonometers are available but it is possible to construct a simple pressure transducer using a 4-way stopcock, two intravenous tubings, a Luer Lok tip syringe and an IV catheter (Matsen and Mayo, 1976; Styf and Wiger, 1998). There are several opinions as to what constitutes normal intra compartmental pressures, but every one agrees that when the pressure rises above 30 mmHg, is the time to intervene with a fasciotomy.

Other investigations like xenon scanning, methoxy isobutyl isonitrile enhanced magnetic resonance imaging, phosphate nuclear magnetic resonance spectroscopy, thallous chloride 201 and technetium sestamibi scanning are available in specialist centers.

RESULT

Medical management

When there is suspicion of an evolving CS, the limb is placed at the level of the heart (do not elevate the limb as it compromises arterial flow further). A cast if present is released, bivalve or removed altogether. To avoid rhabdomyolysis mannitol and intravenous fluid administration, alkalinisation of urine and diuresis are done (Daniels and Reichman, 1998). Acute renal failure results from hypovolemia, myoglobinaemia and academia. Hyperbaric oxygen (HBO) is useful in improving local

Table 1. Difference between acute and chronic CS.

Acute CS	Chronic CS
Direct blow	No trauma
Hours to days	Weeks to months
Any muscle group	Anterior or lateral compartment leg
Clinical symptoms	Compartmental pressures Stress thallium testing
Emergent fasciotomy	RICE (rest, ice, compression, elevation)

blood flow which reduces edema and swelling. It reduces edema through oxygen-induced vasoconstriction while maintaining oxygen perfusion and supports tissue healing by allowing oxygen delivery when perfusion is low. It also protects against reperfusion injury (Wattel and Mathieu, 1998).

Surgical treatment

Fasciotomy is the gold standard in treatment and is the emergent treatment (Field and Senkowsky, 1994). Adequate decompression should be given priority over cosmesis. All tight compartments need to be thoroughly decompressed and liberal incisions are needed to release the limiting skin.

Sheridan and Matsen reported that good limb function returned if fasciotomy was done within 12 h (in over 68% patients) and when it was delayed over 12 h only 8% of patients had normal function (Sheridan and Matsen, 1976). Permanent nerve damage, loss of limb and death are seen when CS progresses.

Conclusions

Not only trauma surgeons, but also general and family physicians need to be aware of this potentially deadly compartment syndrome, as cause prompt correction saves limb and life.

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