Chapter

Acute Compartment Syndrome of the Extremities and Paraspinal Muscles

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Abstract

Acute compartment syndrome (ACS) occurs when the pressure within the closed osteo-fascial compartment raises above perfusion pressure leading to irreversible tissue ischemia and necrosis. Any closed compartment in the body can be affected by ACS. The leg is the commonest site. Trauma is the common cause of compartment syndrome in young patients. In older patients, medical causes can cause it. The diagnosis in a conscious patient can be made based on clinical features. Pain out of proportion to the injury is the most important symptom. Exacerbation of pain on stretching the affected muscles and paresthesia are the common signs. Compartment pressure measurement is important for the diagnosis in unconscious and uncooperative patients. The treatment of established ACS is emergency fasciotomy. Untreated compartment syndrome can lead to neurovascular injuries and muscle contractures. In this chapter, we will see the etiologies, clinical features, investigations, and management of acute compartment syndrome of the extremities and the paraspinal region.

Keywords: acute compartment syndrome, compartment syndrome of extremities, compartment syndrome of paraspinal muscles

1. Introduction

Compartment syndrome is a condition where the pressure within the closed osteo-fascial compartment raises above the perfusion pressure leading to irreversible tissue ischemia and necrosis. A decrease in the compartment volume, an increase in the contents of the compartment, or external pressure can cause it. The compartment syndrome can be acute or chronic. Untreated acute compartment syndrome (ACS) can cause cosmetic problems due to muscle contractures, and functional problems due to neurovascular damages. These can be reasons for litigations against treating doctors. If detected early and treated properly most of the sequelae of ACS are preventable [1]. In 1881 Dr.Richard von Volkmann a German doctor first described ACS [2]. Paul Jepson in 1924 demonstrated ischemic contracture of muscles in animals [10]. The incidence of ACS is 0.7 to 7.3 persons per 100,000 people [3]. The leg is the most common site of ACS. About 2–9% of fractures of the tibia are associated with ACS [4]. There is an equal incidence of ACS in closed and open fractures [5]. A higher incidence of ACS is seen in grade 2 compound fractures than in grade 3A or 3B due to the phenomenon of self-decompression seen in higher grade open injuries. Due to the bulk of muscles attached to the diaphysis of long bones, fractures through

diaphysis are prone to develop ACS [6]. The forearm, hands, feet, buttocks, thighs, and paraspinal muscles are other sites. Any closed fascial space can be affected [7]. There can be fracture-related and non- fracture-related ACS. Fracture-related ACS is common in young males and its diagnosis is early. Whereas non- fracture-related ACS is common in the elderly with medical comorbidities. The ACS in the elderly can be traumatic or nontraumatic. The posterior compartment of the leg is affected commonly in non-fracture-related ACS group. In older people with swollen limbs, a compartment pressure measurement is needed to rule out ACS [8].

1.1 Etiologies

Most cases of ACS occur following trauma. Young males are about ten times more affected than females. Other than fractures there are many other etiologies. Arterial injuries, snake bite, burns, gunshot injuries, leakage from arterial and venous access, drug overdose, pulsatile lavage, contusions in hemophilia, infection, and intraosseous fluid replacement in infants are other causes for acute compartment syndrome. Over-exertion can lead to acute or chronic compartment syndrome. Lithotomy positioning during surgery or constricting casts or wraps can cause it. ACS can also occur due to non-traumatic medical conditions like nephrotic syndrome, viral myositis, hypothyroidism, bleeding disorders, malignancies, diabetes mellitus (Diabetes –associated muscle infarction), and in rheumatological conditions like ruptured Baker's cyst [9].

An awareness regarding the etiology, pathophysiology, clinical features, investigations and management is essential for all doctors dealing with patients in the emergency department. In this narrative review, we intend to give a detailed overview of acute compartment syndrome.

2. Anatomy of the compartments

The major groups of muscle and neurovascular structures are separated by a thick layer of fascia. The fascia provides a surface for the attachment of muscles and keeps the contour of the muscles. It improves mechanical advantage during contractions. The fascia helps in coordinated actions of muscle and proprioception. These fasciae divide the extremity into different compartments.

There are 4 compartments in the leg. They are anterior, lateral, superficial, and deep posterior compartments. The anterior intermuscular septum separates the lateral muscles from the anterior muscles, and the posterior intermuscular septum separates the lateral muscles from the posterior muscles. The interosseous membrane spans the gap between the tibia and fibula, separating the anterior and deep posterior compartments. The transverse intermuscular septum separates the musculature of the superficial and deep posterior compartments. The anterior compartment contains the anterior tibial artery and veins, deep peroneal nerve. Tibialis anterior, extensor hallucis longus, and extensor digitorum longus are the muscles in this compartment. Peroneus longus and peroneus brevis muscles with superficial peroneal nerves are in the lateral compartment. The superficial posterior compartment contains gastrocnemius, soleus, and plantaris muscles. The peroneal vessels, posterior tibial vessels, tibial nerve and tibials posterior, flexor digitorum longus, and flexor hallucis longus muscles are the contents in the deep posterior compartment of the leg [10].

There are controversies regarding the actual number of compartments in the foot. Various authors reported three to nine compartments in the foot. The medial, lateral, and superficial compartments run along the entire length of the foot. The

four interossei and an adductor compartment are confined to the forefoot. Manoli and Weber described a calcaneal compartment containing quadratus Plantae muscle, posterior tibial, and lateral plantar vessels and nerves. The medial compartment contains abductor hallucis brevis, and flexor hallucis brevis, and lateral compartment abductor digiti quinti and flexor digiti quinti. Flexor digitorum brevis and lumbrical muscles in the superficial compartment [11].

The forearm is divided into three compartments volar dorsal and lateral. The interosseus membrane separates the volar and dorsal compartments. The lateral compartment containing the mobile wad muscles is separated by the antebrachial septum is lying in the posterior and lateral part. Volar muscles are commonly affected in ACS. There is anatomical communication between various compartments of the forearm so the release of the volar compartment alone can reduce pressure in others [12]. The thenar, hypothenar, adductor, and interosseous compartments are the main osteo-fascial compartments of the hand [13].

The anterior, medial, and posterior are the fascial compartments in the thigh. The lateral intermuscular septum is very tough whereas the medial and posterior septum is thinner. The hamstrings are in the posterior compartment, adductors in the medial, and quadriceps in the anterior compartments. There is a lot of potential space in the thigh compartments before the elevation of intra compartmental pressure [14].

3. Pathophysiology

ACS is due to elevation of interstitial pressure due to any reason. The difference between interstitial pressure and capillary perfusion pressure (CPP) is the determinant of tissue perfusion. When the volume of an osteo-fascial compartment increases as in intra-compartment bleeding due to injury, both tissue and venous pressure increases. Once this pressure exceeds CPP there is a collapse of capillaries resulting in ischemia of muscles and nerves. This can happen due to external compression also. This is the arteriovenous pressure gradient theory for the development of ACS [15]. A vicious cycle follows that (Appendix 1). The decreased capillary pressure leads to decreased tissue perfusion leading to increased capillary permeability and increased extravasation of fluid into the interstitial spaces further increasing the tissue pressure and decrease in tissue perfusion. A decrease in venous return also results in a decrease in tissue perfusion due to an increase in interstitial pressure [16]. When the intra-compartment pressure is more than 10 to 30 mm of Hg above the diastolic pressure tissue perfusion is compromised, and when it exceeds mean arterial pressure muscle ischemia starts. There is a direct relation between systemic blood pressure and intra-compartmental pressure in the development of ACS. Hence a hypotensive patient is more likely to develop ACS compared to normotensive [17].

Most muscle injury occurs not during the phase of ischemia but at the time of reperfusion. Ischemia–reperfusion syndrome is the cellular and systemic effects of ischemia followed by reperfusion. Normally the energy demands are met by oxidation of free fatty acids leads to aerobic conversion of ADP to ATP. During ischemia, cells try to preserve energy. Ischemia induces two anaerobic pathways for energy production. The first is from creatine phosphate stored in the muscles. The creatine kinase in the muscle produces a large amount of ATP by transferring phosphate from creatine phosphate to the ADP molecule. The creatine phosphate stores in the muscles will be depleted within three hours. The glycogen within the muscles is the next source of energy. The glycogen is broken down into pyruvate and lactate. The hydrogen ions released during this process decrease the intracellular pH. This inhibits glycolysis by negative inhibition of the rate-limiting enzyme

phosphofructokinase. This is an inefficient mechanism of ATP production. This will end in six hours. Later dephosphorylation of adenosine nucleotide continues leading to the production of fat-soluble precursors like inosine monophosphate, adenosine, hypoxanthine, and xanthine. These products are washed away during reperfusion and unavailable for adenine nucleotide restoration. The vasodilation during revascularization leads to hyperemia to the extremity. This will wash away the lactate and precursors of adenine nucleotide metabolism. The hyperemia causes increased extravasation fluid through the capillaries leading to a rise in interstitial pressure. The muscles are the only tissue in which xanthene dehydrogenase is converted to xanthine oxidase during reperfusion and not during ischemia. This is due to the increased concentration of cytosolic calcium. The oxygen-free radicles produced by the xanthene oxidase react with proteins and enzymes. The free radicles attack the unsaturated bonds of free fatty acids in the phospholipid bilayer of the cell membrane called lipid peroxidation. Lipid peroxidation causes fragmentation and structural-functional alteration in the membrane leading to increased permeability. This reaction in the capillary leads to increased permeability cell swelling and interstitial edema increasing the vascular resistance. These abnormalities in the cell wall functions allow calcium influx into the cytoplasm. Increased cytoplasmic calcium will completely uncouple the oxidative phosphorylation and production of ATP in the mitochondria. This influx of calcium leads to cell death and necrosis. This reperfusion injury cascade can induce further local and systemic organ failure [18].

The irreversible changes and reduction of aerobic metabolism in the tissue due to ischemia are different for different tissues. It depends on the ischemic time which can vary from minutes to hours. Within 6 hours of acute ischemia irreversible tissue necrosis and inflammatory cascade leading to fibrosis sets in muscles. Ischemia of 1 hour produces reversible neuropraxia in nerves. Irreversible axonotmesis sets in about 4 hours of acute ischemia [19, 20].

4. Clinical features

The signs and symptoms of ACS evolve within few hours. A high index of suspicion is required for the diagnosis of ACS. Griffiths described pain, paresthesia, paresis, and pain with stretch as the main symptoms of ACS ("four Ps") later pulselessness and pink color of skin were added [21, 22]. Pain out of proportion to the known injury is the earliest symptom. Pain not responding to analgesics also make us suspicious. Resting pain and exacerbation of pain on passive stretching of affected muscles are present. Paresthesia due to ischemia of nerves can be an early sign. But an assessment of neurological functions for the diagnosis of ACS can be tricky. The extreme pain, anxiety, and altered mental status due to an injury can make proper neurological examination impossible. The motor nerve fibers can withstand the ischemia to a longer extent than sensory fibers hence motor weakness will be present at a later stage. Swelling and distension of the affected extremity should alert the surgeon about the possibility of an incumbent ACS. Resting pain or pain due to passive stretching of muscles, paresthesia, pallor, pulselessness, and paresis (5Ps) can be seen in ACS. Any one of the above signs may not be indicative of ACS. When three or more of the above signs are present in combination in a patient at risk of developing ACS will increase the sensitivity of these signs for diagnosis. Among these signs, the paresis may take longer to appear. The 5 Ps described above are characteristic features of arterial ischemia. In a conscious patient pain out of proportion to known injuries and paresthesia are the most important signs. Two-point discrimination is a more sensitive test than a light touch. Sometimes a 6th P - Poikilothermia a change in temperature of the extremity or coolness of

the affected limb may be present in ACS [23]. In young children with injury, the above-mentioned features may not be useful for diagnosis. They may not be able to communicate regarding their symptoms and signs. The increasing need for analgesics, features of agitation, and anxiety (3 As) are indicators for the development of ACS. Clinical diagnosis of ACS is challenging in an unconscious patient, in a patient using patient-controlled analgesia, regional anesthesia, and use of epidural pain catheters because of masking of clinical features [24].

The leg is the most common site of acute compartment syndrome. The anterior and lateral compartments of the leg are commonly affected. Fractures of the tibia, tibial plateau fractures, and fracture-dislocations of the knee are the common injuries producing ACS of the leg. According to the compartment involved the clinical features can change. Paresthesia of the first webspace of the foot is an early sign. Later weakness of dorsiflexion of the great toe, inversion of the foot, and dorsiflexion of the ankle are seen in anterior compartment involvement. The lateral compartment syndrome produces a sensory loss in the dorsolateral aspect of the foot with weakness of eversion of the foot. Deep posterior compartment involvement leads to loss of plantar flexion of the toes with loss of sensations in the plantar aspect of the foot. Plantar flexion of the ankle will be weak when the superficial posterior compartment is affected [25].

Dislocations of Chopart and Lisfranc joints are the commonest cause for foot compartment syndrome. Isolated fractures of the mid-foot bones are a very rare cause. The symptoms are similar to leg compartment syndrome. Frequent checking of sensations especially two-point discrimination is a sensitive test. Passive stretching of muscles results in exacerbation of pain [26].

ACS of the thigh is a very rare and potentially devastating condition. Fracture of the shaft of the femur, contusion, coagulopathy, vascular injuries, intramuscular hematoma, arthroplasties of hip and knee, and arthroscopy of the knee are some of the etiologies. The outcome can be an uneventful recovery to severe morbidity and mortality. The diagnosis of ACS in a conscious patient able to cooperate with examination is based on the following criteria. Pain out of proportion to the injury, significant swelling of the thigh, palpable induration of the involved compartment, increase in measured thigh circumference, pain with passive stretching, weakness of the involved muscle, sensory or motor weakness in the nerves in the affected compartment are all seen in varying combinations in acute compartment syndrome of the thigh. An excessively painful, tensely swollen thigh is the most consistent finding of ACS of thigh [27].

The lumbar paraspinal muscle compartment syndrome can be either acute or chronic. Acute cases are due to injuries from downhill skiing, or surfing, direct injury to muscles, or lifting weight. Localized paraspinal muscle tenderness, board-like rigidity of the muscles, deep tenderness on palpation of the abdomen, absent bowel sounds, and loss of sensation over the paraspinal area are the common features. Localized loss of sensations in the paraspinal region is a pathognomonic finding [28].

Supracondylar fracture of the humerus with vascular injury is the commonest cause of ACS in the forearm. The deep volar compartment is commonly involved, flexor pollicis longus and flexor digitorum profundus are commonly involved. Trauma, crush injuries, insect bites are the commonest cause of ACS of hand. Other than the usual symptoms and signs pain on passive motion of the metacarpophalangeal joints of the corresponding intrinsic muscle is a sensitive test [29, 30].

5. Investigations

Despite the awareness among doctors about the possibility of ACS in trauma patients it is one condition frequently missed leading to devastating complications. The clinical suspicion of this condition must lead to immediate decompression

without many investigations. This is especially true in an unconscious patient, intubated patients, and who cannot respond appropriately.

The compartment pressure measurement is the most common method used to diagnose ACS. The measurement of pressure should be done within 5cms from the fracture and not at the site of fracture. Compartment pressure measured over time is more useful than a single measurement. The diagnosis depends on the delta pressure measurement. Delta pressure is the difference between diastolic pressure and compartment pressure. When delta pressure is equal to or less than 30 mg of Hg it indicates ACS. If we use only delta pressure for treatment it has been shown that many asymptomatic patients will undergo fasciotomy. So diagnosis must be confirmed only with clinical findings and hemodynamic and metabolic parameters [31]. In an unconscious patient compartment pressure measurement is the only way to diagnose ACS. Compartment pressure must be checked every 4 hours in the first 24 hours in all high-risk unconscious patients after an injury.

Several techniques can be used for measuring compartment pressure. The needle manometry technique is the simplest and cheapest technique. This is introduced by whitesides et al. (1975). The Wick catheter, the slit catheter, the solid-state transducer intra- compartment catheter, myopress catheter, and transducer-tipped fiber optic catheters are other methods used [32].

The intramuscular partial pressure of oxygen can be measured noninvasively using Near-Infrared spectroscopy (NIRS). There is an increase in perfusion to the injured site. So the partial pressure of oxygen will increase at the site of injury. If there is no increase in the partial pressure of oxygen at the site of injury it can indicate ACS. This is the principle of NIRS. The intramuscular partial pressure of oxygen can vary among individuals and different compartments. A comparison of the NIRS value of the same compartment of the opposite uninjured leg is a useful tool for diagnosis. There is some controversy regarding the use of NIRS for the monitoring of ACS. Different factors like depth of tissues, discoloration of the skin, a hematoma can interfere with the results of NIRS [33].

Many different techniques are used for detecting compartment pressure and perfusion but they are still in the development or experimental stage. Ultrasonography can show increased echogenicity of the compartment when pressure increases. It can also be used to detect changes in elasticity when standardized external pressure is applied. Both techniques are in an experimental stage. Pulse Phased Locked Loop (PPLL) ultrasound is useful in detecting the displacement of fascia with arterial pulsation. This technique was found to be useful to detect raised compartment pressure in cadaveric studies. Photoplethysmography, laser Doppler flowmetry, and scintigraphy, intramuscular glucose monitoring are all used to find out raised compartment pressure. They are not used widely in clinical practice [34].

The serum biomarkers are used for the diagnosis of ACS without many shreds of evidence. Elevated Troponin Levels and myoglobinuria can assist in the diagnosis of ACS. There are also reports suggesting the usefulness of lactate levels from femoral veins in the diagnosis of ACS in patients with femoral artery injury. Serum biomarkers are not useful in the delayed diagnosis of ACS [35]. The creatine kinase level increases during ACS. Creatine kinase level > 1000 U/ml or myoglobinuria are suggestive of ACS. There are abnormalities in renal function tests and hyperkalemia due to rhabdomyolysis [36].

6. Treatment

Acute compartment syndrome is a surgical emergency. If not diagnosed early and treated promptly it can lead to devastating complications. The sequel of untreated

or mismanaged ACS includes unacceptable deformities, neurological injuries, crush syndrome, renal failure, limb amputation, and death. There are certain preventive measures that we should do to prevent the development of acute compartment syndrome in all patients with limb injuries. All circumferential and tight bandages should be removed. Split tight plaster cast. The Limb should be kept at heart level. Avoid patient going into hypotension, and maintain oxygen saturation. These measures reduce the risk of the development of ACS in high-risk patients.

The treatment of ACS is emergency decompression of the compartment. The fasciotomy is used for decompression. Fasciotomy should be done within 6 hours or definitely within 12 hours after diagnosis of ACS. Fasciotomy must be done in all patients with clinical findings of ACS when compartment pressure is more than 30 mm of Hg, and when delta pressure is less than or equal to 30 mm of Hg. The fasciotomy should be liberal decompressing all the compartments of the limb including the epimysium to relieve integumental compartment pressure. In the legs all 4 compartments, and the forearm, both dorsal and volar compartments should be decompressed. The fasciotomy wound can be closed by delayed primary intension, or by a split-thickness skin graft. The use of negative pressure wound therapy for fasciotomy wounds is controversial. It can help to reduce the swelling and early closure of wounds or skin grafting. A fasciotomy is not indicated when there is irreversible intra-compartmental damage like neuromuscular or vascular damage in an adult patient. If fracture fixation is needed in such a patient either external fixation or plaster cast can be used without violating the affected compartment. In a patient with ACS fracture fixation, either internal or external fixation can be done along with fasciotomy [35, 37]. In children, delayed fasciotomies have shown better outcomes than adults. We can do fasciotomies up to 24 hours after injury in children [38].

ACS of the leg is common in the fractures of the diaphysis, proximal fracture of tibia with comminution, long periarticular fragments, fracture-dislocations, and medial tibial condyle fractures. Comminuted fracture of the fibula at the same level of fracture of tibia is also associated with a high incidence of ACS [39].

All four compartments have to be decompressed. The commonly used 2 incision technique involves a longitudinal incision on the posteromedial aspect of the leg extending from the proximal tibia to distally up to the musculotendinous junction of the gastro-soleus muscle. The incision is made 2 cm posterior to the posteromedial corner of the tibia. Care should be taken to avoid injuring the sural nerve proximally and saphenous vein distally. The fascia is incised in line with incision decompressing the superficial compartment. The deep compartment is decompressed by elevating the soleus muscle and cutting the fascia covering it. We can extend the incision proximally and distally as needed for the release. The second incision is made 2 cm anterior to the head of the fibula. The incision extends from the head of the fibula to the distal fibula. The anterior skin flap is raised and the lateral intermuscular septum is identified and the anterior compartment is released by incising the entire length of the facia from proximal to distal. The superficial peroneal nerve is at risk at the distal third. The lateral compartment is released by incising the fascia along the posterior border of the fibula (**Figure 1**) [40].

A single incision technique can be used for the fasciotomy of the leg. A longitudinal incision is made along the entire length of the fibula. A transverse incision is made to identify the lateral intermuscular septum then decompression of the anterior and lateral compartment is done. The posterior compartment is decompressed after elevating the attachment of the soleus from the fibula and then identifing the posterior compartment and decompressing it. Fibulectomy can be used to decompress all 4 compartments of the leg, but it should be avoided especially in patients with complex tibial fractures [41].

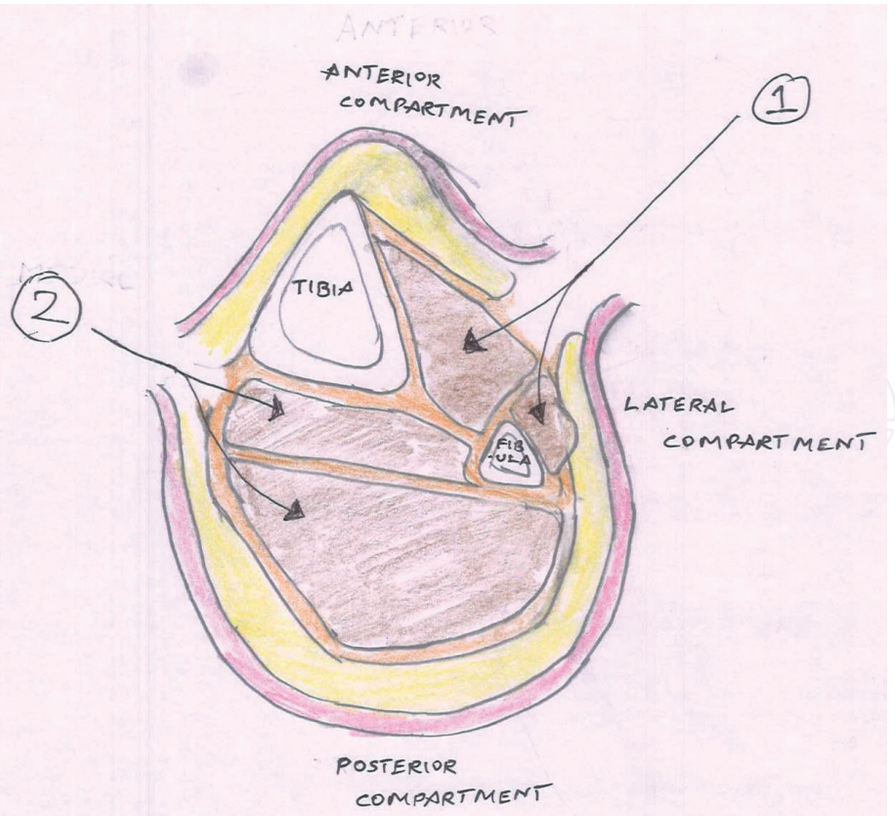


Figure 1.
Diagram showing the compartments of the leg and arrows 1 & 2 represent the approach for the fasciotomy of the leg.

There are different methods for fasciotomy of the foot. The most commonly used method is a combination of medial and dorsal approaches. The dorsal approach involves two longitudinal incisions one medial to the second metatarsal and another lateral to the fourth metatarsal. There should be adequate space between these 2 incisions to prevent necrosis of the skin. The medial incision is about 6 cm long starting from about 4 cm anterior to heel and about 3 cm above the plantar surface of the foot. The dorsal approach helps to decompress the interossei and adductor compartments (**Figure 2**). The medial approach releases the medial, superficial, calcaneal, and lateral compartments [42].

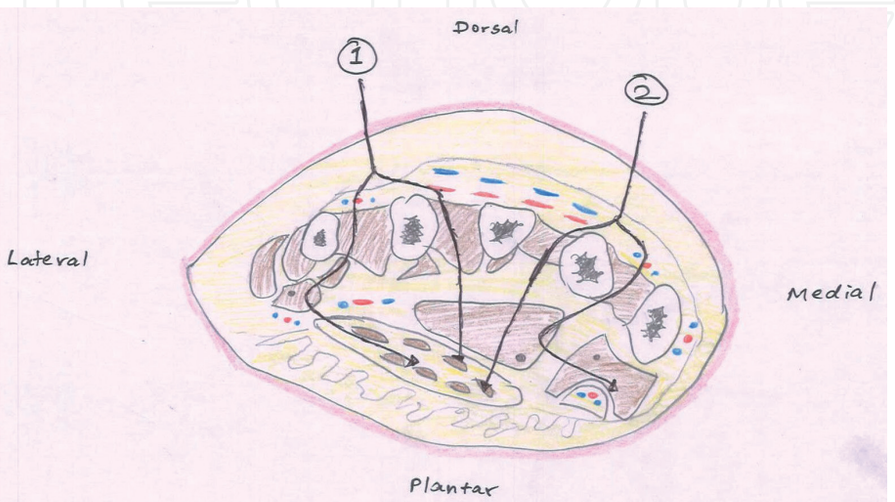


Figure 2.
Dorsal approach for fasciotomy of the foot- a cross-section.

The decompression of all three compartments is indicated in ACS of the thigh. The anterior and posterior compartments are decompressed using a single lateral incision extending from the intertrochanteric line to the lateral epicondyle of the femur. The skin and subcutaneous tissue are opened along the skin incision. The iliotibial band is identified. By dividing the iliotibial band fascia covering the vastus lateralis the anterior compartment is released. The posterior compartment is opened after retracting the vastus lateralis and dividing the intermuscular septum. The medial compartment is decompressed using a separate longitudinal medial incision and dividing the medial intermuscular septum (**Figure 3**) [43].

The paraspinal muscles are enclosed in the thoracolumbar fascia which acts as a closed fascial compartment. It covers the muscles from all sides except medially where it is attached to the spinous process and interspinous ligaments (**Figure 4**). Surgical release of the thoracolumbar fascia gives better results than nonoperative treatment in the compartment syndrome of the paraspinal muscles. There is no consensus regarding the timing of fasciotomy. Most reports agree on fasciotomy within 7 days. The approach is Wiltse paraspinal incision. The thoracolumbar fascia is divided and individual muscle compartments are released [44].

The management of ACS of the forearm involves decompression of the compartment using volar and dorsal approaches. The volar incision is curvilinear. It extends from proximal and medal to cubital fossa then extends distally along the radial side of the forearm till the distal third. Then again it is curved medially to the midline of the forearm over the wrist for release of carpal tunnel (**Figure 5**). This incision helps to decompress the median nerve and helps to cover the median nerve using the radial flap. The lacertus fibrous is released and the superficial volar compartment is released. The identification of the deep fascia and its release is very important. The pronator quadratus should be identified and its fascia should be released separately. The dorsal compartment is decompressed using a single midline incision

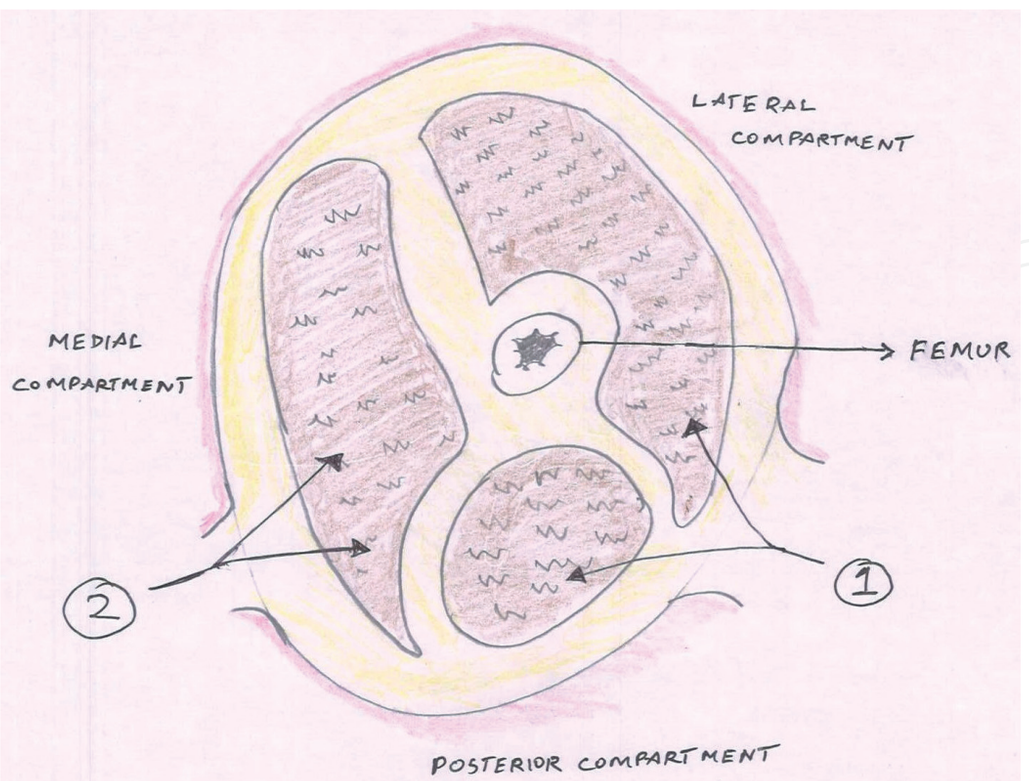


Figure 3.
The anterior, lateral, and posterior compartments of the thigh and arrows representing the lateral and medial fasciotomy approaches.

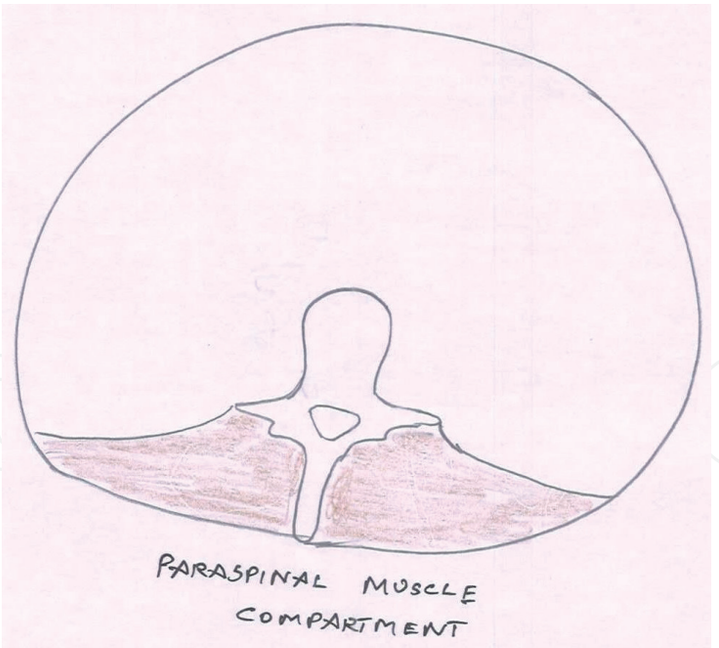


Figure 4.
Diagram showing the paraspinal muscle compartment, the thoracodorsal fascia covering the muscles all around and medially to the spinous process and interspinous ligaments.

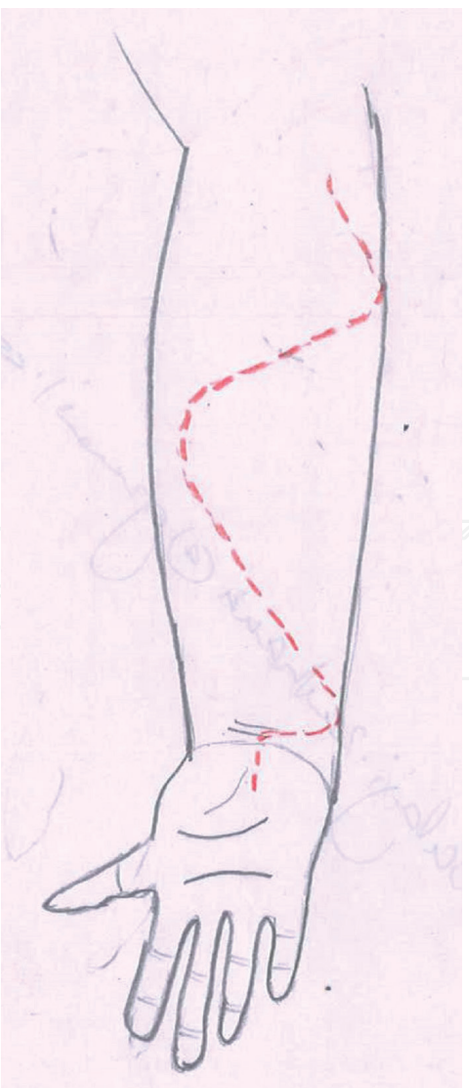


Figure 5.
The volar approach for fasciotomy of the forearm.

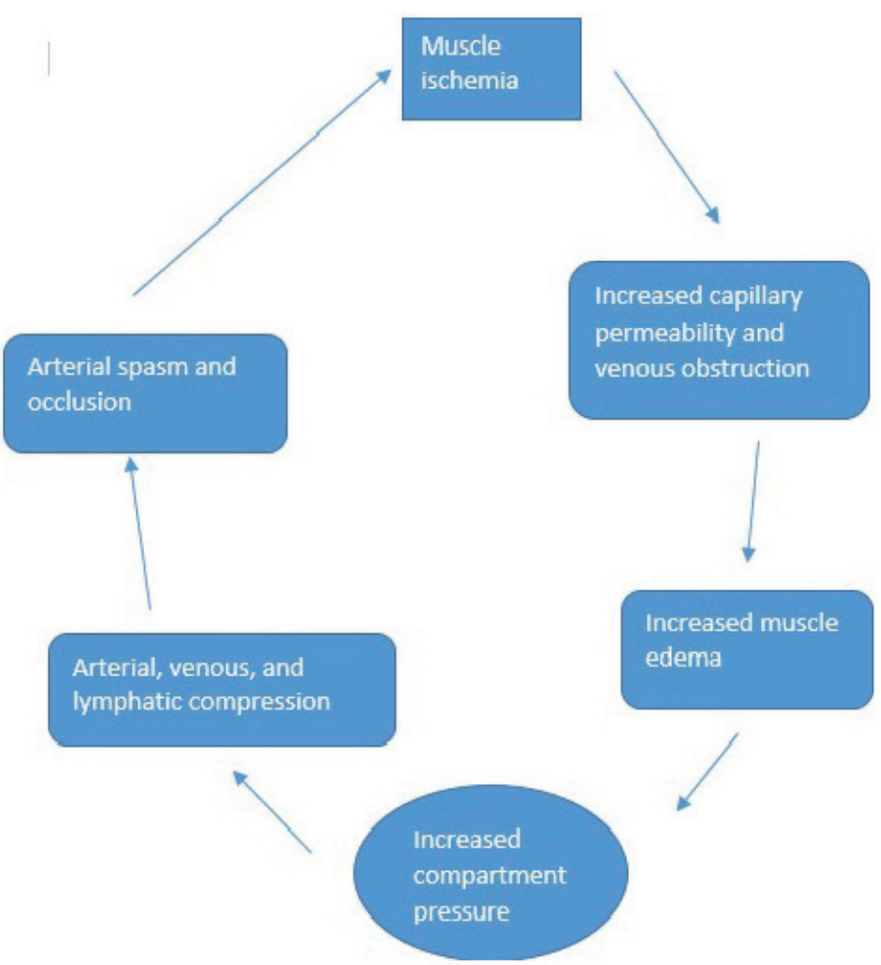
extending from lateral epicondyle to distal radioulnar joint. The individual septum separating the muscles should be released individually [45].

The hand compartment is released by the volar and dorsal approach. Dorsally decompression is done using 2 incisions along the 2nd and 4th metacarpals. The release is done on either side of the metacarpals to decompress the interossei. Deeper dissection along the radial aspect of the 2nd metacarpal is used for decompressing the adductor compartment. The volar incision is used to release the thenar and hypothenar compartments. The carpal tunnel should also be released [46].

7. Conclusion

Acute compartment syndrome is a surgical emergency. The diagnosis is based on clinical findings so careful history and physical examination are required. In obtunded patients, the diagnosis is made when delta pressure ≤ 30mm of Hg and compartment pressure > 30 mm of Hg. Emergency fasciotomy and decompression of the compartment is the treatment of choice. Usually, a repeat inspection of the fasciotomy wound after 24 to 48 hours should be done. Delayed closure of the wound is done.

Appendix 1: The vicious cycle in the pathogenesis of acute compartment syndrome



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