Journal of Health Study and Medicine
2016, nr 1
ss. 33-40

Compartment syndrome – pathophysiology and clinical presentation. Part 1

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Introduction

Compartment syndrome (CS) is manifested with symptoms resulting from increased pressure within the closed anatomical compartment developing over an extended time and involving also compartments in lower extremities. Increased intrafascial pressure influencing the network of capillary vessels leads to reduction of the capillary flow, and when a threshold value is reached, to a complete closure of vessels and arrest of the micro-circulation. As a result function of muscles and nerve fibres within the given compartment or compartments becomes impaired [1]. A non-managed acute CS may lead to development of deformations associated with fibrosis and contracture of muscles and degeneration of injured nerves. Considering commonly coexisting shin disorders, the diagnosis of CS is not easy, and is based on the assessment of dominating symptoms, as well as results of laboratory and functional tests and imaging diagnostics. Treatment always involves reduction of pressure within the affected compartment, and is often reduced to a fasciotomy procedure involving extensive incision of fascia within all affected compartments [2,3].

Anatomy of shin compartments

Shin compartments constitute the most common localisation of compartment syndromes in extremities. There is an interosseous membrane extending between the tibia and fibula, forming a middle ligament, and muscles are covered with a continuous and not highly flexible superficial fascia. In its middle part the superficial fascia adheres directly to the surface of the tibia, and in the lateral part with the fibula via two

intermuscular septa. As a result, four compartments are formed, limited with fascial lamina and closing individual muscles along with their nerves and blood vessels [4]. The anterior compartment is spoon-shaped and contains the tibialis anterior muscle, the extensor hallucis longus and extensor digitorum longus muscles, and the deep fibular nerve. Anterior and lateral to the fibula there is a lateral shin compartment surrounded by two intermuscular septa. Peroneus longus and brevis muscles contained therein are combined within the compartment, filling it up and extending to the lateral ankle. They are accompanied by the superficial fibular nerve and the fibular artery along with veins. The other two compartments are: deep posterior compartment and the superficial compartment. Three muscles combine within the first one: the tibialis posterior muscle, the flexor hallucis muscle and the flexor digitorum longus muscle, filling the space between shin bones behind the interosseous membrane. Also the tibial nerve extends within the deep compartment. The deep posterior shin compartment, beginning from the tarsal joint is completely covered by the superficial posterior compartment containing the soleus muscle and the plantaris muscle (deep layer) and the gastrocnemius muscle (superficial layer), and the sural nerve [5,6]. 25% of vascularisation is contained within the anterior compartment (the anterior tibial artery and vein), and the remaining 75% is contained within the deep posterior compartment (the posterior tibial artery and vein and the fibular artery and vein) [7].

Forms and causes of CS

Depending on causative factors and the course of compartment syndrome, it is classified as acute or chronic. The acute form of compartment syndrome (ACS) may be a result of numerous factors, including ischaemic-reperfusion ones being consequence of a thrombotic or embolic occlusion, fractures or injuries of soft tissues of the shin [8,9,10]. Other causes are: too tight circumferential dressings, burns (especially circumferential), bites, ballistic trauma, prolonged lithotomy position, shock and transfusion of a large volumes of fluids, intoxications [2,7,11,12]. Nearly half of ACS cases is a result of fractures within the lower extremity, including displaced and open fractures of the tibia and fibula, and fractures within the tarsal joint [10,13]. According to Matsen the etiology of ACS may be divided into three groups. The first one covers reduced volume

of a compartment as a result of excessively tight suture of fascia, too tight dressing or local external compression. The second group of factors is associated with increased content of the compartment in consequence of bleeding (injury of blood vessels, clotting disorder) and increased permeability of capillaries (post-ischaemic oedema, excessive exercise, trauma, burn, intraarterial or intravenous medication administration, surgery). The third group involves reasons associated with increased pressure in blood vessels (venous outflow disorders, muscular hypertrophy, infusion, nephrotic syndrome) [14].

The chronic form of CS is usually a result of excessive, long-term muscular exercise within the given compartment – most commonly the anterior or the lateral one. That results in increased volume of a muscle, as a sum of micro-trauma, and consequential rise of pressure in the affected compartment, along with local tissue perfusion disorders. That leads to a disproportionate blood supply in relation to the muscular demand during an intensive exercise, which manifests with a strong pain. A differential diagnosis of vascular disorders with CS is important. CS is not associated with neurological disorders or pulse changes in the dorsalis pedis artery, even at the time of the most intensive pain [15].

Pathophysiology of ACS

The principal role in tissue perfusion is played by the arteriovenous gradient associated with a normal pressure in capillaries and initial segments of veins. The capillary pressure is within the range of 25 – 30 mmHg, and the pressure in initial segments of veins localised after the capillary network is lower by nearly 10 mmHg [16,17]. If an increased pressure develops inside a compartment, small veins of the post-capillary system become compressed, leading to increased pressure in their lumen, and then in the lumen of capillary vessels. The back-transmitted increased pressure reduces flow, which leads to under-supply of structures contained in the affected shin compartment. Compression or a complete closure of thin-walled and low-walled lymphatic and venous vessels results in venous stasis and a passive congestion of the shin [18]. The persistent high arterial pressure increases transudation and oedema, and excessing the critical threshold value leads to the extreme stenosis of arteries, arrest of the micro-circulation and ischaemia of muscles, nerves and other tissue structures [19].

The ischaemic-reperfusion mechanism of injury is associated with release of substances damaging cells of tissues, of vascular epithelium, and increasing permeability of capillary vessels. Active oxygen forms, including superoxide and hydroxyl radicals, are produced by xanthine oxidase in course of transformation of purine metabolites, including those originating from decomposition of ATP, during the progressing tissue ischaemia – xanthine and hypoxanthine. The reaction occurring presumably in endothelial cells may damage all cellular components, and resulting free radicals may activate the cascade of arachidonic acid, leading to the production of leucotrienes and prostaglandins, including prostacyclin [7]. That may lead to oxidation of cellular membrane fats and production of more reactive oxygen forms. Toxic oxygen metabolites activate neutrophils that damage the endothelial cells and result in a final damage of tissues of the affected compartment. That is probably a result of over-expression of adhesion molecules of β-integrin character on the surface of neutrophils, as well as of intracellular adhesion molecules on endothelial cells. That inhibits the movement of granulocytes along the vascular endothelium resulting in their adhesion to its walls, and enzymes produced in granulocytes (myeloperoxidase, proteases) and toxic free radicals damage the endothelium. The next step is migration of neutrophils through the wall of venous vessels and into the extravascular space [20]. As those changes develop, damage of the cellular membrane leads to extracellular and cellular oedema, and in consequence to increase in tissue pressure and development of CS. Injured tissues release inorganic phosphates, amino acids, purines and proteolytic enzymes, that activate the clotting system, along the arachidonic acid metabolites. Thrombosis is intensified in the micro-circulation and pro-inflammatory cytokines are released, along with vasoconstrictive substances [21].

Tissues contained in the affected compartment have a variable resistance to ischaemia. The resistance of the striated muscle tissue to ischaemia is approx. 4 hours, with a permanent damage occurring after 8 hours of ischaemia. In case of nerves the ischaemia tolerance time is approx. 8 hours, and irreversible changes develop within 12-24 hours. For peripheral nerves the critical pressure value is the value over 35 mmHg maintained for over 6 hours. The resistance of the adipose tissue extends for approx. 12 hours, of the skin to approx. 24 hours, and of the osseous tissue in normal thermal conditions to 3-4 days [22, 23].

The clinical presentation of CS

Most commonly signs and symptoms are manifested within the anterior and deep posterior compartments (25% of cases), and simultaneous involvement of them both is observed in approx. 8% of cases [24]. The main symptom of ACS of an increasing stretching pain, often not proportional to the injury and resistant to the pharmacotherapy, sensory disorders within the area supplied by nerves passing through the affected compartment, and paresthesias, muscular weakness, as well as hard and painful to palpation oedema of the affected compartment. It should be noted that the peripheral circulation may be normal in the developing compartment syndrome. Pulse is palpable in the physical examination, and colour of the skin remains normal [9]. The „6 P" rule has been long mentioned as a diagnostic criterium of CS. The acronym stands for principal symptoms of acute compartment syndrome: pain, pressure, pallor, paresthesia, paralysis, pulselessness. In the diagnostic process the key role is played by sequence of developing symptoms, especially those associated with peripheral nerves of the affected extremity. First symptoms developing with pain are paresthesias, as a consequence of sensory disorders of skin nerves. When motor nerve disorders appear, or a complete paralysis occurs, the syndrome is fully developed, which may suggest irreversible character of pathological changes. Starting a therapy on that stage may prevent from further damage, which does not mean that the full efficiency of the extremity could be restored [25, 26]. Presence all characteristic symptoms of the syndrome, and especially the absence of pulse suggesting injury of the artery, indicates that the process had entered its final stage, associated with irreversible damage of structures within the affected compartment. In case of the syndrome with no injury of a large vessel, changes are usually delayed, and the pulse is palpable for a long time. Therefore, it should be underlined that peripheral pulse palpability does not exclude CS. However, the absence of pulse should not be the crucial symptom in the diagnosis of CS, because the symptom may appear at the point of time when pathological changes are already irreversible [23].

Summary

The phenomenon of structural damage as a consequence of shin compartment syndrome is well known. That damage results from primary and secondary changes of perfusion within the affected compartment, often

leading to irreversible changes resulting in functional disability of the affected lower extremity. The awareness of pathophysiology and of the clinical presentation often allows early diagnosis and introduction of an effective therapy, stopping the progress of the pathology and minimisation of its consequences.

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