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Compartment Syndrome – Presentations and Principles

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Abstract

Background: Compartment syndrome (CS) remains an elusive diagnosis with limited awareness despite progress of glorious investigative, diagnostic and imaging modalities. This review aims to reiterate the surgical fraternity to the fragility one faces with the concept of CS.

Methods: A retrospective desk review of published literature has been undertaken through the review of PubMed, Cochrane Library, Medline, EMBASE and Web of Science databases to portray the spectrum of presentation with involvement of rare sites in CS that have the capacity to stupefy the surgical fraternity.

Results: 18 articles and book chapters were reviewed from the search chain of over 200 articles. Through literature review it is appreciated that the sites of involvement with CS are varied and can encompass regions that are once thought unlikely. There is great degree of variance in the incidence of this syndrome. Though the implications are grave, the diagnosis of the syndrome is complex given the presentation spectrum.

Conclusion: Despite its immemorial descriptions and characterization as a surgical emergency, compartment syndrome remains an elusive diagnosis given its lack of awareness and wide presentation profile.

Keywords: Compartment syndrome; Osteo-fascial compartment; Fractures and trauma

Introduction

The seminal paper of Richard von Volkmann in 1872 inspired and moulded the early knowledge, understanding and awareness of the global orthopaedic fraternity to the gravity compartment syndrome (CS) posed as a result of grievous orthopaedic insult [1-3]. However, despite the invent and progress of glorious investigative, diagnostic and imaging modalities through the turn of the century, CS is still regarded a shadow in the darkness given its lack of awareness. The gravity of dealing with such an elusive diagnosis culminates in high rates of litigation.

Methods

A desk review of over 200 articles was undertaken with 18 eventually selected. Journal articles in PubMed, Cochrane Library, Medline, EMBASE and Web of Science databases were searched for CS. The focus of the review was to elucidate the history, signs and symptoms, pathophysiology, sites of involvement, prognosis, complications, treatment and essentially the irregularities with presentations in CS so as to improve the appreciation of the disease and thus improved clinical care. Apart from journal articles; book chapters were a valuable reference resource in undertaking the review.

Only articles chapters published in English were included. The resources were reviewed for the indicated impact and where warranted, articles within the pre-selected articles were reviewed.

Results

CS is defined as perfusion compromise to muscular-nervous contents within an osteo-fascial compartment as a result of increased pressure within a non-compliant intra-compartmental space that exceeds vascular perfusion pressure [3-5]. Groups of muscles and their associated neurovascular bundle comprise a muscle compartment. The latter is encapsulated by an unyielding facials surround. The clinical manifestations of CS stems from the reduction of tissue perfusion. As a fundamental of perfusion physiology, tissue perfusion is proportional to the difference between the capillary perfusion pressure and the interstitial fluid pressure [3]. A narrowed arterio-venous perfusion gradient results from an increase in intra-compartmental pressure from various mechanisms. As a consequence, capillary perfusion pressure is overcome and manifestations of CS results. The formal transmural pressure that results in complete vascular impedance is debatable. In addition to standard clinical acumen, intra-compartmental pressures greater than 30 mm Hg coupled to or in isolation of a raised intra-compartmental pressure as compared to diastolic blood pressure is an accepted requirement to necessitate a fasciotomy [3-6]. Emergent fasciotomy is known to normalize compartmental pressures with muscle cell regeneration considered the most desirable therapeutic outcome [3].

CS is known to exist in 2 forms – acute and chronic. Traumatic fractures are the most commonly implicated etiology in acute CS with the anterior compartment of leg the most commonly studied site [1,3,7]. In an analysis of 164 patients, McQueen and Court-Brown reported a higher propensity of CS occurrence in males with 69% of the sample population succumbing to a fracture - 50% were associated with tibial fractures [1]. Chronic compartment syndrome (CCS), originally described by Maver in 1956 is typically related to exertion and exercise and is primarily characterized by chronic reproducible pain and disability that is alleviated on removal of the precipitant coupled to rest and 5 minutes post-exertional intra-compartmental pressures greater

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than 15mmHg and 20mmHg respectively [8]. CCS has not been known to be an implicated in traumatic events [3].

Despite the degree of variance that exists in the incidence of the abovementioned syndrome, it is well known that the prevalence of CS is directly proportional to the nature and severity of injury sustained [9]. Children are noted to be significantly affected given a 20% incidence in CS with open forearm fractures [3,10]. With a sample of 104 patients, DeLee and Stiehl noted a 6% and 1.2% incidence of CS in open and closed tibial fractures respectively [1,3]. The incidence described previously is in isolation of associated underlying vascular insult. As noted by Blick, with the assimilation of the latter, 9.1% of the sample developed CS in the setting of an open tibial fracture [3,7]. To further complement Blick's results, Feliciano et al. noted 19% of patients who sustained a vascular insult required a fasciotomy [1,3,11].

The prognosis of CS is dependent on two interrelated entities – diagnosis and duration from injury to instituting intervention. The threshold of muscle viability was clinically validated by Rorabeck and Macnab following a finding of near normalization of limb function and return to premorbid level of function with institution of fasciotomy within 6 hours of diagnosis and injury [12]. As per Sheridan and Matsen, 68% and 8% of sampled patients had normal limb function when fasciotomy was performed within and beyond 12 hours of acute CS onset respectively [13]. Majority of fatalities are secondary to uphill battles with multisystem organ failure and prolonged intensive care admissions.

Fasciotomies of the lower limb posterior compartment have shown to have poorer outcomes than those in an anterior CS secondary to the morbidity associated with a complete posterior decompression. One risk's peroneal nerve palsy when diagnosis and or intervention in lower limb CS is delayed [1].

In the event of failure to institute any form of intervention, Volkmann's contractures have been approximated to occur in up to 10% of patients within a short duration of weeks to months [14]. Calcific myonecrosis is an uncommonly encountered manifestation of post-traumatic lower limb musculo-nervous perfusion compromise. Delayed compartmental decompression has shown to result in formidable infections that might cause one to succumb to an amputation. A retrospective analysis conducted by Matsen et al. revealed that 46% of limbs developed an infection following delayed surgical intervention. 45% of those with infections succumbed to an amputation [12].

A multitude of causative factors have been documented. Given the wide causative profile, it is essential to simplify the factors into basic mechanisms as so as to aid understanding and application of management – Increased interstitial pressure within the enclosed compartment, extrinsic compression imposed on the compartment and constriction of intra-compartmental space [15]. A non-exhaustive list of aetiological factors is provided by Hoover and associate [3].

Limb fractures – forearm, tibial and fibula – form the commonest cause of CS [3]. Reperfusion of limb after prolonged ischemia (> 6hours) is a common cause of acute CS as suggested by Rowlands in the beginning of the twentieth century (1910) [9,15]. High risk individuals include those with type 3 open fractures with significant comminution, pedestrians of motor vehicle accidents, poly-trauma, risk of delayed or missed diagnosis owing to significant or distracting injuries, prolonged anaesthesia, altered sensorium secondary to drugs and cognitive impairment, non-communicative, spinal cord injury and or transection [9].

The locations to which CS occur has expanded exponentially since its initial conception in the nineteenth century. Initial reports and understanding of CS were limited to the upper extremity. It was the report of ischemic contractures secondary to open tibial fractures in the mid-20th century that redefined CS localization [9]. Volar compartments of the forearm, anterior and deep posterior compartments of the leg are considered the most commonly involved sites given their unyielding structure [3,4,9]. Whitesides et al. in the 1960s revolutionized the concept of decompressing more than one compartment given their understanding of the existence of 4 compartments in the distal limb.[9,16] Rare and uncommon sites are continually being defined and include but not limited to the shoulder, back, foot - Manoli proposed the existence of nine pedal compartments, thigh - posterior compartment and tensor fascia lata compartment, gluteal, hand, arm, abdomen and paraspinous muscles, leg – Davey demonstrated the existence of a septated tibial posterior compartment [3,15-18].

Despite its immemorial descriptions and characterization as a surgical emergency, CS remains an elusive diagnosis given its lack of awareness and great variance in disease process and presentation. The difficulty in assessing the incidence of the syndrome is an indication that CS has the capacity to stupefy the surgical fraternity.

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