Compartment syndrome

Essay

Submitted for Partial Fulfillment of
Master Degree in orthopedic surgery

By

Mostafa Kamal kotb
M.B., B.Ch

Supervisors

Prof.Dr. Hany Mamdouh Hefny
Professor of orthopedic surgery
Faculty of Medicine
Ain Shams University

Dr. Mahmoud Mohamed Fayed
Assist. Professor of orthopedic surgery
Faculty of Medicine
Ain Shams University

2009

بسم الله الرحمن الرحيم

الحمد لله الذي هدانا لهذا وما كنا لنهتدي لولا أن هدانا الله .

صدق الله العظيم

الأعراف (43)

Acknowledgment

First and foremost I feel always deeply indebted to **ALLAH,** the Most Gracious and the Most Merciful.

I wish to express my deepest gratitude and respect to Prof. **Dr. Hany Mamdouh Hefny**, Professor of orthopedic surgery, Faculty of medicine, Ain Shams University, for his constructive and instructive comments and valuable suggestions.

I would also like to thank **Dr. Mahmoud Mohamed Fayed,** Assist. Professor of orthopedic surgery, Faculty of medicine, Ain Shams University, for his precious efforts and his helpful advices. He gave me a lot of his precious time following every step in the work.

Lastly and not least, I send my deepest love and gratitude to my parents and my family, for their care and ever- lasting support.

Contents

Page

List of figures

Page

List of tables

Page

- 1-
Introduction and historical background

Introduction and historical background

A compartment syndrome is defined as a condition in which high pressure within a closed fascial space causes reduced blood flow with nerve and muscle ischaemia. Untreated compartment syndrome leads to ischaemia with necrosis, contractures, myofibrosis and irreversible functional impairment in the involved part of the limb [(1)].

In 1881, Richard Volkmann described a contracture involving the injuried muscles and nerves of the forearm. He felt the contracture was due to tight application of bandages to the injured limb, thereby causing massive venous stasis and simultaneous arterial occlusion [(2)].

In 1888, Petersen had reported a case of contracted upper extremity that had some return of distal function following release of an entrapped median nerve in the forearm. It was his feeling that the return of function following release of the scar tissue demonstrated that scarring and inflammation were major factors in the pathology of Volkmann's ischemic contractures [(3)].

In 1906, Hildebrand who was a student of Volkmann theorized that the damage to the muscle was the prime cause of the contracture, but unlike Volkmann he also felt that nerve contusion and scarring were factors leading to the poor prognosis. Of significance is the fact that Hildebrand noted that an elevated pressure in the tissue may be significant for the etiology of the ischemic contractures. It was his feeling that increased venous effusion resulted in an increased pressure in the muscle, which subsequently compromised the small arteries and

retarded the development of the collateral circulation. It is also important to note that Hildebrand was the first to describe the term "Volkmann's ischemic contracture" [4].

In 1909, Thomas reviewed the medical literature and noted the association of compartment syndrome with number of medical and surgical conditions including fractures, arterial injuries, embolus and external compression [5].

In 1914, Murphy emphasized that following a fracture, blood and serum effusion results, and the tension in the sub fascial zone in the forearm may be as great as to cause cyanosis of the entire forearm and hand. He related that a blood clot forms in the tissue and then inflammation results in a deposit of inflammatory products in the tissue, and it is an increase in pressure that results in the cell destruction. Murphy's treatment for the problem was to perform a fasciotomy prior to the development of a contracture state. He had described releasing forearm fascia from the anterolateral aspect of the arm when symptoms persisted. His paper was one of the earliest to advocate prevention of a contracture state, rather than a delayed reconstructive treatment program [6].

In 1922 Brooks thought the precipitating event to be acute venous obstruction resulting in swelling and diminished tissue perfusion. During and shortly after World War II, a number of high velocity gunshots wounds with associated long bone fracture and arterial injury were noted to have developed acute compartment syndrome. The cause of compartment syndrome was thought to be acute arterial spasm, and

attention was drowning toward relieving the arterial spasm rather than fasciotomy itself [7].

In 1958 Ellis reported a 2% incidence of ischemic contracture in the lower extremity following tibial fractures. Before that most of attention had focused on upper extremity and specifically supracondylar fractures of humerus and fractures of the radius and ulna as precipitating events leading to compartment syndrome and subsequent contractures[8].

In 1967 Kelly and Whitesides demonstrated the existence of four osseofascial compartment in the lower extremity and thus the potential need to decompress all four compartments [9].

In 1972 Spinner et al, had good results up to three weeks after onset of compartment syndrome, especially in children. This last group also reports progressive deterioration of histological picture with increasing periods of unrelieved compression, and concluded that spontaneous recovery without fasciotomy is unlikely [6].

In 1973 Dolich and Aiache reported that fasciotomy even in late stage worthwhile. They found complete recovery in one patient who had fasciotomy one month after the onset of compartment syndrome [10].

In 1975 Eaton and Green found that fasciotomy was effective after 16 weeks. It is now well recognized that good distal pulses and capillary return do not exclude the diagnosis of compartment syndrome [11].

In 1979 Holden, reiterated the two possible causes of compartment syndrome (Volkmann's ischemic contracture) type I is a

proximal arterial injury resulting in ischemia distally, whereas type II is a direct injury giving rise to severe swelling and eventual ischemia [12].

The early diagnosis and management of acute compartment syndrome are critical for the prevention of long term disability. Diagnosis may be delayed when the condition is unexpected and when symptoms are masked by the use of analgesia [13].

Early diagnosis of an acute compartment syndrome (ACS) is important. Despite its drawbacks, clinical assessment is still the diagnostic corner stone of acute compartment syndrome. Intracompartmental pressure measurement can confirm the diagnosis in suspected patients and may have a role in the diagnosis of this condition in unconscious patients or those unable to cooperate. Whitesides suggests that the perfusion of the compartment depends on the difference between the diastolic blood pressure and the intracompartmental pressure. They recommend fasciotomy when this pressure difference, known as the Delta p, is less than 30 mm Hg. Access to a precise, reliable, and noninvasive method for early diagnosis of ACS would be a landmark achievement in orthopedic and emergency medicine [14].

-2-

Anatomy

Anatomical locations

Certain factors may favour the development of a compartmental syndrome in a specific location. Examples include exposure to trauma or ischemia, and vigorous use of the compartmental musculature. The frequency with which the different compartments are involved may vary from one geographical area to another [15].

Anatomical locations of compartmental syndromes

A compartmental syndrome may potentially occur wherever a limiting envelope surrounds neuromuscular tissue. Certain anatomical locations are particularly predisposed to the development of compartmental syndrome. This predisposition may result from the limited compliance of the compartment. The human anterior compartment of the leg less compliant than the superficial or deep posterior compartments of the leg. A high susceptibility to trauma may be another predisposing factor. The four compartments of the leg are often affected by ischemic conditions of the lower extremity, a situation that places them at risk for compartmental syndromes resulting from post ischemic swelling. The muscles of the leg and forearm are often exercised vigorously; thus, their compartments are potential sites of compartmental syndromes from intensive use of muscles. Additionally, other factors predispose the compartments of the upper and lower extremities to the development of compartmental syndromes, including their accessibility for drug injection and their vulnerability to burns [16].

Applicable anatomy of the arm:

The anatomy of the arm is consisting of two major muscle compartments: flexor and extensor that share responsibility for three major nerves and arteries:

- The anterior flexor compartment contains three muscles: The coracobrachialis, biceps brachaii and brachialis.
- The posterior extensor compartment contains one muscle: triceps brachii in two thirds of the arm the muscle compartment are separated by lateral and medial inter muscular septa.
- Medial intramuscular septum: this septum extends along the medial supracondylar line behind the coracobrachialis insertion and fades out above, between that muscle and the long head of triceps it is pierced by: ulnar nerve and ulnar collateral artery.
- Lateral inter muscular: extends along the lateral supracondylar line and fades out behind the insertion of deltoid it is pierced by: radial nerve and profunda brachii artery.
- Three nerves:
    - The radial nerve pierces the lateral intramuscular septum to enter the anterior compartment.
    - The median nerve remains in the anterior compartment.
    - The ulnar nerve pierces the medial intramuscular septum to enter the posterior compartment.