Bushoi et al., J Trauma Treat 2014, S2
DOI: 10.4172/2167-1222.S2-008

Journal of Trauma & Treatment

Review Article Open Access

Compartmental Syndrome: A Case Report and Literature Review

Alexander Bushoi, Luis Schnapp, Gabriel Agar and Amir Oron*

Kaplan Medical Center, Rehovot, Israel

Abstract

Acute compartment syndrome of the extremities is a serious and potentially limb and, life endangering condition. Compartment syndrome of the extremities is considered a medical emergency. Despite widely described classic clinical signs and profound physiologic understanding of its mechanism, most cases show only partial, often insidious clinical expression. In conjunction with lack of uniform, reliable and easily applicable diagnostic tools, the diagnosis of compartment syndrome is a challenge, as is the decision making tree of conservative versus surgical treatment. Unconscious patients present an especially difficult subgroup of patients. In this article we will try to compile and simplify the up-to-date knowledge about the etiology, pathophysiology, diagnosis and treatment of compartment syndrome of the extremities. We will present the rigid box model of acute compartment syndrome which can aid in medical personnel education as well as assist in diagnosis and treatment decision making. Early recognition, competency in practical application of intra compartmental pressure measurement techniques and a timely performed surgical decompression are the primary requisitions of successful treatment. A keen awareness by all medical personnel and a high grade of suspicion to this elusive but devastating entity is in place.

Keywords: Acute compartment syndrome; Keen awareness; Surgical treatment; Pathophysiology

Definition

Compartment syndrome was first described by Malgaigne, and the first medical publication was that of Volkmann [1] in 1881 which used the term "ischemic contracture".

Acute Compartment Syndrome (ACS) is defined as "a condition in which increased pressure within a limited space compromises the circulation and function of the tissues in that space" [2]. Under this definition one can include also such conditions as expanding subdural hematoma, tension pneumothorax, some cases of intestinal obstructions and closed angle glaucoma; although in this article we will only discuss acute compartment syndrome of the extremities.

Etiology

ACS of the extremities is most commonly seen following injuries to the leg and forearm (40% caused by tibial shaft fractures and 18% by forearm fractures), but can also occur in the arm, foot, thigh, buttock, hand and lumbar paraspinous muscles.

Acute compartment syndrome can be classified according to its etiology as shown in Table 1, but this would be a simplification for instructional purposes, since most acute compartment syndromes are caused by a combination of factors. A good example may be a use of a surgical tourniquet which can cause acute compartment syndrome by each of the following mechanisms: muscle crush, reperfusion damage, mechanical injury to blood vessels, venous thrombosis and relative venous insufficiency or any combination thereof if the tourniquet is inflated or placed improperly [3-7] (Table 1).

Pathophysiology

Normal intracompartmental pressure is below 10-12 mmHg.

Compartmental Perfusion Pressure (CPP) = Mean Arterial Pressure (MAP) – Compartmental Pressure (CP)

Normal values of compartmental perfusion pressure are above 70-80 mmHg. Acute compartment syndrome develops when tissues within a compartment do not receive an adequate blood supply. Factors that increase CP or decrease MAP will cause decreased compartmental perfusion and ischemia. Most of the etiologic factors listed in Table 1 influence CP. Decreased MAP itself rarely causes compartment syndrome, but can happen when a limb is elevated for a prolonged period, e.g. in patient undergoing surgery in lithotomy position [8]. Even then, it is assumed to be at least partially due to reperfusion injury and an elevation in compartmental pressure.

The Rigid Box Model

The Rigid Box Model can help in understanding the interaction of factors influencing the magnitude of the intracompartmental pressure. A compartment can be assumed to be a rigid, non-expansible box (fascial envelopes, interosseous membranes and bone walls) that is filled with a mass (muscle, fat, neural and vascular structures), with very limited free space (intra- and extravascular) constantly filled by afferent

Bone related	Fractures Iatrogenic - surgery
Vascular	Arterial injury (bleeding or occlusion) Venous injury (bleeding or occlusion) Reperfusion injury Decreased oncotic pressure
Iatrogenic	Plaster of Paris/circular dressing application Invasive procedure in anticoagulated patient Excessive Pulsatile irrigation
Soft tissue damage	Crush injury Muscle overuse or tear Reperfusion damage Burns Animal bite (venomous and non-venomous) Pressurized fluid injury (by machinery)

Table 1: Causes of acute compartment syndrome of extremities.

***Corresponding author:** Amir Oron, Kaplan Medical Center, Rehovot, Israel, Tel: 972542427072; E-mail: amironmd@gmail.com

Received January 06, 2014; **Accepted** May 29, 2014; **Published** May 31, 2014

Citation: Bushoi A, Schnapp L, Agar G, Oron A (2014) Compartmental Syndrome: A Case Report and Literature Review. J Trauma Treat S2: 008. doi:10.4172/2167-1222.S2-008

vessels and evacuated by efferent vessels. A bone can be considered as a non-compressible traversing vessel that without fracture does not contribute to compartmental pressure. Increase in inflow (rare), in mass volume from an internal (edema, bleeding, abscess) or external source (injections) or a decrease in outflow (venous compression or insufficiency) will elevate the pressure within the compartment. Although oversimplified, this model can be useful for educational and rapid diagnostic purposes, and can aid in treatment decision making (Figure 1).

Natural History

Acute compartment syndrome can ensue minutes after the insult or develop insidiously over hours. If left untreated, it can resolve spontaneously in mild cases or evolve into a full blown fulminant course of progression of signs and symptoms with rhabdomyolysis, vascular occlusion, permanent injury to peripheral nerves and resulting palsy, electrolyte abnormalities, acute renal failure due to myoglobinopathy and possible death due to the above mentioned systemic complications. Patients that survive the systemic disease are left with a miserable result of local muscle necrosis and neural and vascular deficit, described as Volkmann's ischemic contracture. Infection and delayed fracture healing are other possible late sequelae of acute compartment syndrome [9-11].

Since acute compartment syndrome occurs most often in young patients [9], the overall impact in terms of daily function, ability to work and wellbeing of patients is tremendous.

Clinical Presentations

The initial clinical presentation of compartment syndrome can be very mild. Mubarak and Rorabeck [6] listed six classical signs of compartment syndrome, described as "the six P's": pain, pallor, paresthesias, pulselessness, paralysis, poikilothermia.

Pain

It can be the first and only sign of compartment syndrome, often initially misrelated to the trauma itself. A typical sign is pain out of proportion to the magnitude of injury or a constant increase in pain over time. Still, pain may just be mild. Pain irresponsive to analgesics (even opiates) is more suggestive of compartment syndrome. Pain on passive stretching of muscles contained in a suspected compartment

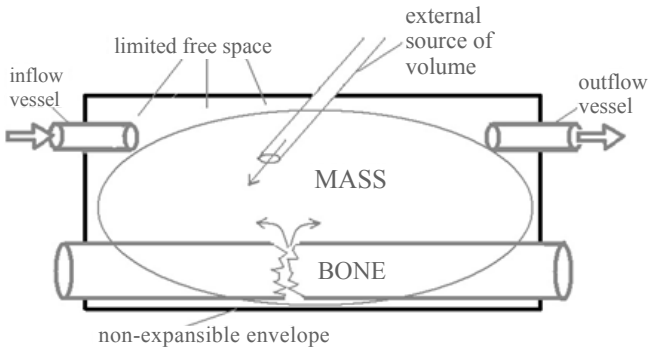

Figure 1: The Rigid Box Model: A compartment can be assumed to be a rigid, non-expansible box filled with a soft tissue mass, with a very limited free space (intra- and extravascular) constantly filled by afferent vessels and evacuated by efferent vessels. A bone can be considered as a non-compressible traversing vessel. External source can be traumatic or iatrogenic.

may raise suspicion of the presence of an acute compartment syndrome and aid in pointing to a specific compartment within a limb. Another important physical sign is a tender or rigid area upon palpation of a limb, which anatomically correlates with a specific compartment. Every suspected compartment should be evaluated and re-evaluated, preferably by the same physician, while establishing the diagnosis.

Paresthesia

Related to nerves passing through a specific compartment. It is also a helpful sign for localization. The first sensory modality to be impaired is static two-point discrimination [7].

Paralysis

Rendered as an inability to activate muscles within an involved compartment, may be a sign of pain avoidance or of functional nerve impairment. Functional impairment will usually appear late in the course of acute compartment syndrome evolvement. Paralysis of a muscle group that is located distal to an involved compartment but innervated by a nerve passing via this compartment may be more specific to acute compartment syndrome.

Pallor

It is a result of diminished blood outflow via the exiting vessel of the compartment. One should seek for pallor in areas that are supplied by a relevant artery distal to its exit level from the compartment.

Pulselessness

Even in full blown acute compartment syndrome distal pulses may still be preserved. Although a compartmental pressure equal to or higher than systolic blood pressure is needed to abolish a peripheral sensation, this may be decreased at lower compartmental pressures. It is prudent for a physician to examine and compare pulses of both extremities, whenever possible.

The major drawback of the majority of the listed symptoms and signs is their late appearance. Patients presenting with signs other than increasing pain, pain on passive muscle stretching or paresthesias (early symptoms) are usually at an irreversible stage of tissue injury [6]. In addition, most of these symptoms and signs are inapplicable in a patient with altered conscious, patients at age extremes or in drug abusers.

The need of early diagnosis of acute compartment syndrome cannot be overstressed in this dramatic condition. The key to a timely successful treatment is a high level of suspicion, prompt and correct implementation of anatomic knowledge of limb compartments and their content, with a stress on distal sensory and motor nerve distribution

Objective Diagnostic Methods

Compartmental pressure measurement

Several methods are described to measure compartmental pressure. A needle manometer was the first device used for this purpose. White sides [12] described a device composed of a needle connected to a manometer by a fluid filled infusion system. An interpretation of the intracompartmental pressure was done by observing the shape of a fluid-air interface ("meniscus") within the system. When the meniscus becomes flat during elevation of fluid pressure, the pressure within the system is considered to be equal to that within the tested compartment. This system had the disadvantage of the need to inject

additional, although small, amounts of fluids into a compartment, and the needle became quickly obstructed and did not allow continuous ICP monitoring. In addition, the readings had many inaccuracies [13]. Several types of catheters were proposed and tested in trying to overcome the above disadvantages, including side ported needles, the wick catheter [13], the slit catheter [14], and the solid-state transducer intracompartmental catheter [15]. Although the catheter measurement techniques allow continuous pressure monitoring for up to 24 hours, they still require a heparinized saline system to maintain patency of the probe. None of the systems was uniformly agreed upon to be superior [16,17].

Laboratory

No specific laboratory markers for compartment syndrome are used today. A very high plasma CPK level measurement is not specific and may be a result of trauma and ischemia. Although without evidence of trauma it may suggest acute compartment syndrome, it is not helpful in early diagnosis.

Magnetic resonance imaging

MR imaging can clearly identify edema of soft tissues within a compartment. The edema appears as a high signal on T2-weighted images [18]. Nevertheless, the swelling is present in any case of acute trauma to some extent, and cannot be distinguished from ACS on MRI, especially in the early stages of ACS. Thus, MRI is useful only for chronic cases of CS [19].

Near infrared spectroscopy

Is based on the fact that light in the near infrared spectrum passes through skin and bone and is absorbed differentially by hemoglobin and oxyhemoglobin [20]. It was designed and tested for cases of chronic exertion compartment syndrome with good success in detecting changes in relative oxygenation as compared to normal individuals [21,22]. Near infrared spectroscopy is still inapplicable in ACS for several reasons. First, in ACS relative oxygenation may be impaired prior to measurements being performed. Second, near infrared spectroscopy has a limited depth of measurement (3-40 mm) and cannot detect changes in the deep muscle compartments of the limb. Third, no normal limits of venous oxyhemoglobin were defined so as to be used in clinical practice. Although promising, near infrared spectroscopy still remains clinically inapplicable.

Scintigraphy

This method uses Tcm99-methoxyisobutyl isonitril (MIBI) to evaluate regional perfusion in muscle tissue and is widely used in evaluating coronary perfusion. Scintigraphy was shown to be of value in chronic exertional compartment syndrome with positive predictive value of 89% [23], but its role in acute compartment syndrome is not known.

Laser doppler flowmetry

This invasive modality is still under investigation.

Treatment

Initial care of a suspected acute compartment syndrome includes immobilization of the injured limb, removal of any circular dressing and positioning of the limb at heart level. Excessive elevation of the limb should be avoided to allow adequate perfusion. In case of fracture of the long bones- stabilization of the limb in a grossly normal alignment is preferable to diminish further soft tissue damage and avoid torque of blood vessels. Monitoring blood pressure, preventing hypotension and oxygen supplementation are essential.

When measured intracompartmental pressure is above 30 mmHg and clinical signs have appeared or are worsening, decompression fasciotomy should be performed as a treatment of choice. Preventive fasciotomy is done in cases of a limb that has suffered warm ischemia for more than 4-6 hours, in patients with ligation of major veins in the lower limb and with crush injury. In case of forearm or leg ACS, all compartments must be released by a one- or two-incision technique. Necrotic tissue must be debrided, and skin coverage of swollen and expanded areas of the limb is performed by an elastic closure technique that utilizes rubber tension sutures and an artificial tissue cover. An additional surgical procedure to complete skin closure may be needed after tissue swelling subsides (Figure 2).

The fasciotomy technique can utilize multiple longitudinal incisions to approach each compartment individually, or a single incision technique that allows approach to all compartments. In any case, it is widely accepted that all the compartments of the involved limb segment must be released in acute compartment syndrome cases.

Recently, Bedside fasciotomy under local anesthesia was found to be a feasible and reliable procedure in selected cases [24].

The occurrence of compartment syndrome affects the time to fracture healing and rate of fracture delayed union and non-union in the tibia [25] a pattern which may very well be the case in other sites.

Summary

Acute compartment syndrome is a serious and potentially limb and life threatening condition, uniformly considered as a medical emergency. The classic clinical signs are often not distinguishable from the signs of a “simple” uncomplicated trauma, but the toll of missed acute compartment syndrome is tremendous in terms of loss of limb function, lifetime disability and mortality of often an otherwise healthy young trauma patient. Unconscious patients present an especially diagnostically difficult subgroup of patients. Of many proposed diagnostic tools, intracompartmental pressure measurement remains the only widely accepted method and even it is rendered problematic in terms of reliability and utility.

A good clinical education, a high index of suspicion, an early recognition of the condition, competency in practical application of

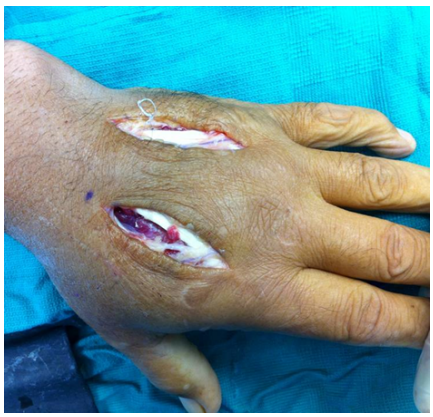

Figure 2: Fasciotomy of the Hand: A hand following an isolated crush injury and decompression of all compartments is shown. Both preoperative and postoperative assessment of neurovascular structures is imperative.

intracompartmental pressure measurement and the confidence to take the daring decision to proceed to surgical decompression is the keys to successful treatment.

References

1. von Volkmann R (1881) Die ischa¨mischen Kontrakturen. Zentralbl Chir 8: 801.
2. Matsen FA 3rd, Winquist RA, Krugmire RB Jr (1980) Diagnosis and management of compartmental syndromes. J Bone Joint Surg Am 62: 286-291.
3. McQueen MM, Gaston P, Court-Brown CM (1980). Acute compartment syndrome: who's at risk? J Bone Joint Surg [B] 82-B: 200-203.
4. Rorabeck CH (1984) The treatment of compartment syndromes of the leg. J Bone Joint Surg Br 66: 93-97.
5. McQueen MM, Gaston P, Court-Brown CM (2000) Acute compartment syndrome. Who is at risk? J Bone Joint Surg Br 82: 200-203.
6. Mubarak SJ, Hargens AR (1983) Acute compartment syndromes. Surg Clin North Am 63: 539-565.
7. Zweifach SS, Hargens AR, Evans KL, Smith RK, Mubarak SJ, et al. (1980) Skeletal muscle necrosis in pressurized compartments associated with hemorrhagic hypotension. J Trauma 20: 941-947.
8. Beraldo S, Dodds SR (2006) Lower limb acute compartment syndrome after colorectal surgery in prolonged lithotomy position. Dis Colon Rectum 49: 1772-1780.
9. McQueen MM1, Christie J, Court-Brown CM (1996) Acute compartment syndrome in tibial diaphyseal fractures. J Bone Joint Surg Br 78: 95-98.
10. Sheridan GW, Matsen FA 3rd (1976) Fasciotomy in the treatment of the acute compartment syndrome. J Bone Joint Surg Am 58: 112-115.
11. Court-Brown C, McQueen M (1987) Compartment syndrome delays tibial union. Acta Orthop Scand 58: 249-252.
12. Whitesides TE, Haney TC, Morimoto K, Harada H (1975) Tissue pressure measurements as a determinant for the need of fasciotomy. Clin Orthop Relat Res : 43-51.
13. Mubarak SJ, Hargens AR, Owen CA, Garetto LP, Akeson WH (1976) The wick catheter technique for measurement of intramuscular pressure. A new research and clinical tool. J Bone Joint Surg Am 58: 1016-1020.
14. Rorabeck CH, Castle GS, Hardie R, Logan J (1981) Compartmental pressure measurements: an experimental investigation using the slit catheter. J Trauma 21: 446-449.
15. McDermott AG, Marble AE, Yabsley RH (1984) Monitoring acute compartment pressures with the S.T.I.C. catheter. Clin Orthop Relat Res : 192-198.
16. Moed BR, Thorderson PK (1993) Measurement of intracompartmental pressure: a comparison of the slit catheter, side-ported needle, and simple needle. J Bone Joint Surg Am 75: 231-235.
17. Uliasz A, Ishida JT, Fleming JK, Yamamoto LG (2003) Comparing the methods of measuring compartment pressures in acute compartment syndrome. Am J Emerg Med 21: 143-145.
18. Rominger MG, Lukosch CJ, Bachmann GF, Langer C, Schnettler R (1995). Compartment syndrome: value of MR imaging. Radiology 197: 296.
19. Eskelin MK, Lotjonen JM, Mäntysaari MJ (1998) Chronic exertional compartment syndrome: MR imaging at 0.1 T compared with tissue pressure measurement. Radiology 206: 333-337.
20. Arbabi S, Brundage SI, Gentilello LM (1999) Near-infrared spectroscopy: a potential method for continuous, transcutaneous monitoring for compartmental syndrome in critically injured patients. J Trauma 47: 829-833.
21. Breit GA, Gross JH, Watenpaugh DE, Chance B, Hargens AR (1997) Near-infrared spectroscopy for monitoring of tissue oxygenation of exercising skeletal muscle in a chronic compartment syndrome model. J Bone Joint Surg Am 79: 838-843.
22. Mohler LR, Styf JR, Pedowitz RA, Hargens AR, Gershuni DH (1997) Intramuscular deoxygenation during exercise in patients who have chronic anterior compartment syndrome of the leg. J Bone Joint Surg Am 79: 844-849.
23. Edwards PD, Miles KA, Owens SJ, Kemp PM, Jenner JR (1999) A new non-invasive test for the detection of compartment syndromes. Nucl Med Commun 20: 215-218.
24. Ebraheim NA, Abdelgawad AA, Ebraheim MA, Alla SR (2012) Bedside fasciotomy under local anesthesia for acute compartment syndrome: a feasible and reliable procedure in selected cases. J Orthop Traumatol 13: 153-157.
25. Reverte MM, Dimitriou R, Kanakaris NK, Giannoudis PV (2011) What is the effect of compartment syndrome and fasciotomies on fracture healing in tibial fractures? Injury 42: 1402-1407.

This article was originally published in a special issue, **Trauma Injury and Orthopaedic Surgery** handled by Editor(s). Dr. Robinson Esteves Santos Pires, Federal University of Minas Gerais, Brazil