

Chapter 1 : Orthopaedic Diagnosis And Management - Boyd Goldie - Google Books

*Orthopaedic Diagnosis And Management [Boyd Goldie] on www.nxgvision.com *FREE* shipping on qualifying offers. Answers specific and frequently asked questions about orthopaedic diagnosis and management Designed to fit into the pocket of a white coat for a junior doctor to carry as reference.*

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Abstract Almost all orthopaedic surgeons come across acute compartment syndrome ACS in their clinical practice. Diagnosis of ACS mostly relies on clinical findings. If the diagnosis is missed and left untreated, it can lead to serious consequences which can endanger limb and life of the patient and also risk the clinician to face lawsuits. This review article highlights the characteristic features of ACS which will help an orthopaedic surgeon to understand the pathophysiology, natural history, high risk patients, diagnosis, and surgical management of the condition.

Introduction Almost all orthopaedic surgeons come across acute compartment syndrome ACS in their clinical practice. Volkmann, a German doctor in , described ACS by reporting the hand contracture which was a consequence of this particular condition [1]. In , Petersen for the first time reported the management of ACS [2]. The compartment syndrome is mostly diagnosed on variation in clinical symptoms and signs in sequential examinations. If the diagnosis is missed and left untreated, it can lead to serious damage to the soft tissues of the limb including muscles, nerves, and vessels. It can sometimes result in limb loss or even Loss of Life. An orthopaedic surgeon must have an understanding of this condition, including specific injuries and specific group of patients which are more vulnerable in getting ACS. A surgeon should understand the basics of compartment syndrome including pathophysiology, epidemiology, diagnosis, and management [3]. Two factors are responsible for this condition, either a decrease in a compartment volume or an increase in the contents of a compartment, or sometimes both of these factors act at the same time. Elevated ICP results in raised pressure at the venous capillary end and increases hydrostatic pressure, leading to arteriolar compression [5]. The microcirculation compromised due to arteriolar compression, hence reducing or diminishing perfusion of the tissues. Inadequate perfusion and oxygenation result in soft tissue ischemia and anoxia and death of the cells. The most ischemic vulnerable tissue in a compartment is skeletal muscle [6]. Extent of muscle death is dependent on the duration of ischemia, temperature of the tissues, and the available residual microcirculation. Sufficient collateral blood supply and lower local temperature slow down the ischemic process [7]. Rorabeck and Clarke showed that the duration of increased pressure is significant in the return of neurological function. Pressures 40 to 80 mm Hg sustained for 4 hours do not cause permanent nerve dysfunction, but, when applied for 12 hours or more, permanent neurological changes occurred [8]. In conclusion, the amount of skeletal muscle necrosis is directly proportional to duration of ischaemia and inversely proportional to temperature.

Epidemiology Acute compartment syndrome usually occurs in traumatized patients who have such injuries which distract the clinician from diagnosing ACS. In management of these patients, the clinician should have a high degree of suspicion. The most common site of ACS is leg which is followed by forearm, arm, thigh, foot, gluteal region, hand, and abdomen. Various risk factors are related to compartment syndrome and age is one of the important factors. Younger patients are more prone to get ACS as compared to elderly patients with the same nature of trauma [9]. Another risk factor is the type and site of injury. Closed tibial shaft fracture is the most common cause of compartment syndrome and is comprised of one-third of all cases of ACS. One-fourth of the cases result from blunt and crushed soft tissue limb trauma while radius ulna shaft fractures are responsible for 20 percent of the cases. Revascularization after acute arterial injury or obstruction can also result in ACS; hence in most of cases patients need fasciotomy after revascularization [12]. Males are more prone to develop ACS which is ten times higher than females. Incidence of ACS in open and closed fractures is equal. Other less common causes of traumatic ACS include burns and blunt or crushing trauma to the limb. ACS can develop by poor positioning of legs in prolonged surgical procedures, particularly lithotomy position [13]. Excessive exercise by athletes or nonroutine physical activity or overuse in nonathletes can also lead to acute compartment

syndrome ACS of the leg which needs urgent medical attention [14]. Acute compartment syndrome can also result from nonaccidental causes like medical conditions which include nephrotic syndrome, viral myositis, hypothyroidism, bleeding disorders, malignancies, and diabetes mellitus [15]. Diabetes-associated muscle infarction DMI is a condition in diabetics which results from compartment syndrome [16]. Clinical Diagnosis Compartment syndrome is mostly diagnosed clinically. Lack of knowledge and inadequate practical exposure lead to delayed or missed diagnosis. Examination should be done serially more at various times than at any one specific point of time for making any definitive diagnosis. It is preferred to have one surgeon who should perform serial assessment and make the diagnosis. If the sign and symptoms are equivocal, then it is preferred to take a second opinion from the senior colleague. One of the most important prognostic factors for outcome is the time of development of ACS to the time of diagnosis and the time of surgical treatment. Though all of the mentioned clinical signs and symptoms are important clinical findings, mostly all are not present in every case, and in fact presence of pulselessness indicates that it is already too late to get good outcome. But if the ACS is already established and ends up in late stage, pain may not be the clinical finding as the pain receptors and nerve fibers face ischemic necrosis and death. Moreover, pain can be absent in regional anaesthetised patients and sedated and relaxed patients in ICU. The first sign of nerve ischemia is paraesthesia which is followed by hypoaesthesia, anaesthesia, paresis, and paralysis. Sensory assessment should be done by pinprick testing, light touch, and two-point discrimination in awakened patients. Pulselessness in ACS is also a late finding. In ACS, pressure in the compartments is not usually high enough to compress arteries. Loss of pulse and presence of Pallor limb could be an indication of direct arterial injury. Capillary refill is mostly present even in well-developed ACS if there is no direct arterial injury. The only clinical sign in impending ACS could be massive swelling of the limb with firm compartments. In unconscious patients most of the clinical findings cannot be elicited; hence it is necessary to check compartment pressure by devices. Intracompartment Pressure Monitoring Various techniques and devices for intracompartment pressure measurement ICP are mentioned in the literature. ICP is nearly 8 mm Hg in resting adults and almost double in paediatrics [19]. Various techniques for measuring ICP include hand-held monitor for single pressure readings, Stryker needle with side portal, and regular needle with arterial line setup. If more sophisticated equipment is unavailable, compartment pressure can be measured using intravenous tubing, a three-way stopcock, a syringe, and a mercury manometer, as described by Whitesides et al. Boody and Wongworawat compared three commonly used devices for measuring compartment pressures which included Stryker Intracompartmental Pressure Monitor System Figure 2 , arterial line manometer, and Whitesides apparatus Figure 3. Manual setup for intracompartmental pressure measurement Campbell Operative Orthopaedics, 11th Edition. Synthes West Chester, PA hand-held compartment pressure monitor. Compartment pressures were found different at various locations within compartments in relation to injury site; hence there is a relationship between ICP and distance from the fracture site. Various authors mentioned different values of compartment pressure considered to be the threshold for surgical decompressive fasciotomy. McQueen and Court-Brown suggested that if the difference between diastolic blood pressure and ICP was less than 30 mm Hg, it was highly suspicious of ACS and needed to be decompressed [24]. Other authors have recommended fasciotomy for pressures greater than 40 mm Hg or delta values of 40 mm Hg the difference between mean arterial pressure and the compartment pressure [26]. Establishing diagnosis on these measurements alone in a conscious patient may lead to unnecessary surgery [27]. While Whitney et al. Patients who are not awake and alert or who have been given regional block for anaesthesia or postoperative analgesia must be observed more carefully as clinical signs and symptoms cannot be picked up [30]. The clinician should have the high index of suspicion for ACS diagnosis in such patients and should not make delay in monitoring and measuring ICP with the available devices. Infrared Spectroscopy A new technique which is called near infrared spectroscopy NIRS is a noninvasive and continuous technique. It is based on absorption of light in near infrared spectrum which corresponds to oxygenated and deoxygenated hemoglobin. Assessment of tissue oxygenation was done by comparing with the oxyhaemoglobin and deoxyhaemoglobin concentration in venous blood. He recommended that the patients with ACS can be early and accurately identified using IM pH monitoring and subsequently reduce the morbidity associated with ACS [33]. Fasciotomy Once the diagnosis of ACS is established, then the surgical

decompressive fasciotomy should be performed urgently but a good surgical technique is mandatory. Once the decision for fasciotomy is made, the theatre arrangements should be expedited. In the meanwhile, keep leg elevated in order to increase venous return and decrease swelling. All dressings should be loosened or removed if possible. Send blood samples for baseline investigations and group and screen for possible transfusion in postoperative period. There are various techniques of fasciotomy of leg in the literature, which include single incision fasciotomy with fibulectomy, single incision fasciotomy without fibulectomy, and the most common surgical approach two-incision fasciotomy with anterolateral and posteromedial incisions. In two-incision technique, the anterolateral incision is made to approach the anterior and lateral compartments. It is midway between the tibial crest and the fibular head Figure 4. Incision starts 5 cm distal to fibular head and extends up to 5 cm proximal to lateral malleolus. Fascia of the anterior and lateral compartments should be released through this incision. Surgeons should be careful about superficial peroneal nerve which comes across around 10–12 cm proximal to the lateral malleolus while exiting from the fascia. This approach could expose periosteum of the lateral malleolus and the peroneal tendons. The viability of the muscles should be assessed after fasciotomy. All nonviable muscles should be excised. The exposed tendons, periosteum, and the muscles should be kept moist to avoid desiccation of the tissues and prevent infection [31]. The second incision is posteromedial incision which is made 2 cm posterior to the medial border of the tibia. This incision is utilised to release the superficial and deep posterior compartments and approach the muscles in these compartments for assessment of viability. Soleus insertion should be released to adequately decompress the posterior compartment. Surgeons should try to avoid sacrificing the saphenous nerve and vein while doing the procedure. Cross section through leg showing site of fasciotomy incisions to decompress all four compartments [31]. The single incision technique is successful in experienced hands but it is less popular Figure 5. A longitudinal incision is made over the fibula extending 5 cm distal to fibular head and 5 cm proximal to lateral malleolus. Through this approach, the anterior, lateral and superficial posterior compartments are released first and then followed by release of the deep posterior compartment at the posterolateral fibular insertion site of lateral intermuscular septum. This approach risks the peroneal nerves and vessels when entering into deep posterior compartment.

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