International Scholars Journals

African Journal of Internal Medicine ISSN: 2326-7283 Vol. 11 (1), pp. 001-003, January, 2023. Available online at www.internationalscholarsjournals.org © International Scholars Journals

Case Report

Acute Bilateral calf compartment syndrome in young lady following alcoholic binge– An Interesting case report

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Accepted 02 October, 2022

A fit 41 year old female presented with unbearable bilateral calf pains and passing coca-cola coloured urine after an alcohol binge. She subsequently developed rhabdomyolysis causing critical illness which subsequently ended up needing urgent surgical intervention and some damage to the muscles and neurovascular integrity permanently. She was promptly diagnosed as Calf Compartment Syndrome. Although Leg pain is the common presentation to the accident and emergency, bilateral calf compartment syndrome is rare cause of bilateral calf pain. A well documented condition in young athletes, but rarely reported as alcohol induced. We describe the presentation and evaluation of the condition, along with the review of the Literature. Early recognition and prompt treatment is essential to prevent complications.

Keywords. Compartment syndrome, Rhabdomyolysis.

INTRODUCTION

Compartment syndrome describes a vicious cycle occurring in the osseofascial compartments of the extremities. After a period of ischemia with reperfusion, the integrity of the capillaries may be compromised, causing interstitial oedema and increases the compartment pressure. If left untreated, leads to permanent neurovascular damage, myoglobilinuria, contractures, limb amputation, renal failure and death. Though compartment syndrome after trauma is a well recognized, atraumatic compartment syndrome is rare.

First ever case report of compartment syndrome was described in 1881 by Richard Von Volkmann. He described it as neural palsies and muscular contractures which resulted from prolonged ischaemia in paediatric forearm and supracondylar fracture (McKee and Jupitor, 1995; Burns et al).

In the past case reports compartment syndrome following lithotomy position, following anabolic steroid use, prolonged spine surgery in kneeling position, following alcoholic binge and acute exercise induced compartment syndrome. Rhabdomyolysis increases the risk of developing compartment syndrome subsequently. Among the risk factors attributed to non-traumatic rhabdomyolysis, including malignant hyperpyrexia, extreme exertion, repeated seizures, bacterial and viral infections, the use of certain medications or illicit drugs, ethanol ranks highest; up to 67% cases of non-traumatic rhabdomyolysis involved alcohol (Kahan et al., 1994; Matsen et al., 1980; Lea et al., 1997; John et al; Sofat, et al., 1999; Ling et al).

We report a case of alcohol induced acute bilateral idiopathic compartment syndrome in a healthy young lady. The purpose of this case report is to emphasize on the importance of diagnosing compartment syndrome clinically.

Case report

A 41 year old perfectly fit lady from Europe presented to the Accident & Emergency complaining of acute onset of severe bilateral leg swelling with excruciating pain with a score of 10/10 since 5-6 hours. This lonely lady had an

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alcoholic binge at home previous night and gone to bed. She woke up mid day with bilateral leg swelling with excruciating pain. She was unable to stand and had to drag her legs as the pain and the leg swellings were so disabling. She grazed her shins due to dragging. The pain and the swelling were gradually increasing. One of her close friends who accompanied her said that her legs are double to normal.

Other significant history includes her occupation as a cleaner at one of the superstores, so she was exposed to a variety of chemicals at work. Second thing to note was that her legs were clean shaven, so another possibility was exposure to epilating creams. Vital signs were all stable. Another interesting thing of note was her popliteal areas bilaterally had irregular fluid filled vesicles, which again made me to think of the possibility of some hot liquid burns.

Other systems examination was fine. On examination both her lower legs were very tense, swollen. The pedal pulses were present, but feeble and sensation was intact. Capillary refill time was around 3 seconds in both feet and but both calves were very tender on palpation and pain was elicited on minimal dorsiflexion. No calf pressure monitoring was available. Intravenous access was gained and she was given some regular analgesia. Considering in mind the possibility of allergic reaction, she was given intravenous antihistamines, steroids and Intravenous fluids which did not result in any benefit. Bloods were waiting. The pain didn't ease away after simple analgesia. Hence we decided to start her on stronger analgesics from opioid family. So we gave some morphine, but again it didn't touch the pain In the meantime patient expressed desire to pass urine. She passed only around 100 -150 ml of coca-cola (dark) coloured urine. I sought senior advice and he suspected compartment syndrome as the Homan's sign was positive and the acuteness of the presentation.

Meanwhile her blood results were back and all the inflammatory markers were raised. C - reactive protein 68.5mg/l, Haemoglobin 19.3g/dl, White blood cell count 19.9,potassium 7.0, Urea 10.7mmol/l, Creatinine 131umol/l and the last but not the least Creatine kinase 374800 IU/L.

ECG: tall tented T waves, small p waves.

Urine dipstick: blood 4+, others could not be determined.

She received some calcium gluconate 10ml 10% IV with rescue insulin. She was immediately referred to the Orthopaedics. She had urgent Doppler ultrasound of the bilateral calves which showed critical ischaemia, fluid in the posterior compartments of bilateral legs, absent posterior tibial pulse but feeble dorsalis pedis pulse. They transferred her urgently to the theatre, where she underwent surgical decompression. Post operatively she stayed in High dependency unit because of her renal failure where she underwent haemofiltration. She was in the hospital for nearly two weeks where she had aggressive fluid and blood replacement. She subsequently required four additional debridements and wound dressings. At the end when she was near normal she was referred to the plastic surgeons for further management, where she underwent split skin grafting. She subsequently has some physiotherapy and further rehabilitation and was discharged back.

DISCUSSION

Muscle accounts for approximately 40% of total body mass and falls victim to a wide variety of toxic, ischaemic, infectious, inflammatory, and metabolic insults. Acute rhabdomyolysis was originally described as a distinct entity by Bywaters and Beall, who described the "crush syndrome" after the bombing raids of London during second world war. The role of alcohol is of particular importance as it accounts for at least 20% of all cases of acute rhabdomyolysis. (Sofat, et al., 1999)

Rhabdomyolysis is a form of muscle necrosis triggered by derangements in oxidative or glycolytic energy production and the resulting ATP depletion. Pressure-stress myopathy induced in this case in which external pressure/tension on muscle increases the influx of cellular sodium and calcium into the intracellular compartment down their concentration gradients. External pressure induced occlusion of the microcirculation also occurs, rapidly depleting the ambient and myoglobin oxygen content. Cells may remain viable for considerable periods of time, since the pressure induced vascular occlusion limits calcium delivery to ischaemic tissues, which itself delays the onset of necrosis. Mitochondrial oxygen free radical production is markedly reduced during ischaemia and acidosis, both intracellular and extracellular, also occurs. The restoration of blood flow results in an influx of neutrophils, which can then reocclude the microcirculation and release proteases and free radicals into the microenvironment. Therefore,

Although the crush lesion sets the stage for rhabdomyolysis, many of the critical events occur during the reperfusion period. It is during this latter period that myoglobin gains ready access into the circulation. Extracellular fluid depletion also results because of fluid sequestration in muscle and other soft tissues (Sofat, et al., 1999). Myoglobin, the 18.8-kd hemoglobin-like protein, is freely filtered by the glomeruli and reaches the tubules, where it may cause obstruction, renal tubular necrosis, and most seriously acute renal failure (Ling et al). Renal vascular flow is also impaired due to the activation of the renin-angiotensin system with increased sympathetic activity, altered prostaglandin production, high circulating concentrations of antidiuretic hormone, and the deposition of microthrombi (Sofat, et al., 1999).

Compartment syndrome is a pathological condition characterized by vascular insult in a closed soft tissue

compartment resulting from rhabdomyolysis secondary to muscle ischaemia and rising compartment pressures. Most of these cases are traumatic in origin. They may complicate fractures, burns and blunt/crush injuries to the extremities. Sometimes it can be due to certain positions adopted for surgery, notably lithotomy and knee chest position, iatrogenic, traumatic vascular injury snake bite, DVT, revascularization limb ischemia (Kahan et al., 1994). Very few acute, atraumatic, exercise induced compartment syndromes have been reported (Dietrich et al., 1994; Kahan et al., 1994; McKee and Jupitor, 1995) . These atraumatic cases can very easily be overlooked and devastating complications such as severe permanant muscle atrophy, loss of sensation or even amputation may result (Shaw and Spencer, 1995).

Compartment syndrome is a clinical diagnosis. Normal pressure within the compartment is less than 20mm Hg. Pressures around 50-55 mm Hg for 4-8 hours have been cited to result uniformly in compartment syndrome. The patient's baseline arterial pressure in the ankle must be taken into account. For eg if the ankle systolic pressure is 100 mm Hg, then longer exposure a higher pressures is required compared to a peripheral vascular disease patient whose pressure may be 40 mm hg (Dietrich et al., 1994; McKee and Jupitor, 1995) Reneman proposed that there was a 20% increase in muscle volume caused by an increase in transcapillary filtration resulting from an increase in capillary pressure and surface area not compensated for by drainage (Burns et al).

Under short-term alcohol intoxication, immobilization or coma induced by ethanol-related central nervous system sedation plays an important role in developing rhabdomyolysis. It causes muscle compression and muscular ischemia, which will superimpose or accelerate short-term alcohol myotoxicity, resulting in a massive breakdown of skeletal muscle within a short period. Because of the rapid release of osmotically active agents into the interstitial space and rapid increase of compartmental pressure, patients with non-traumatic rhabdomyolysis with short-term alcohol intoxication are likely to present with severe muscle pain and even compartment syndromes (Ling et al; Hewittn et al).

Because of rapid release of muscle cell contents into circulation, these patients are also likely to be complicated with hyperkalemia, metabolic acidosis, acute renal failure, acute tubular necrosis, multiorgan failure, or disseminated intravascular coagulation (Sofat, et al., 1999; Ling et al).

Urine should be checked for presence myoglobin although not very reliable because in 50% cases it is not filtered from the glomeruli and the Serum for Creatine kinase. There should be a high index of suspicion in patients at risk. Because compartment syndrome is a reperfusion injury; signs and symptoms may not be apparent immediately. In suspicious cases intra-compartmental pressure should be measured (Shaw and Spencer, 1995). The differential diagnoses include deep vein thrombosis and peripheral nerve injury.

Early recognition and prompt treatment by decompressive fasciotomy is of vital importance if limb function is to be preserved and complications are to be avoided (Matsen et al., 1980).

CONCLUSION

Compartment is a clinical diagnosis. Earlier the diagnoses, earlier the treatment, better the prognosis. An awareness of this entity, accurate clinical history and careful physical examination will lead to an early recognition and diagnosis of this condition and hence ensure prompt treatment by decompressive fasciotomy is of vital importance if limb function is to be preserved and complications to be avoided.

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