## A nap on his arm

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A 24 years old man was found collapsed at home with an empty bottle of hypnotics next to him. He was last seen well 10 hours ago. He was unconscious on arrival.

His vital signs on arrival were
- BP: 88/62mmHg, Pulse: 156bpm
- RR: 24 breaths per minute
- GCS: E1V1M4, Pupils: equal, reactive
- SpO2: 99% on 100% oxygen, H'stix: 8.9

**What is the initial management?**

We should assess the C-A-B status of the patient with simultaneous resuscitation following the ACLS protocol.[1] Fluid resuscitation should be initiated with cardiac monitor to check the rhythm. The patient had a reduced conscious level. He could not protect his airway and was at risk of aspiration. Intubation with mechanical ventilation was indicated. Once the endotracheal tube was in place, we may consider decontamination with gastric lavage or activated charcoal if indicated.

**Progress of patient**

The patient was intubated and he was a rapid responder to fluid resuscitation. After initial stabilization, a head-to-toe examination was performed. His right forearm was swollen with mixed patchy erythema and pallor. It was very firm on palpation. Capillary refill was prolonged and distal pulse was barely palpable. It was believed that the patient lied on his right forearm for a long time after he collapsed. (Figure 1)

Figure 1: Imagine picture: head on his right arm

**What is the likely diagnosis for his right forearm condition?**

The likely diagnosis is acute compartment syndrome (ACS). ACS occurs when the tissue pressure within a closed muscle compartment exceeds the perfusion pressure resulting in muscle and nerve ischaemia.

ACS most often develops after significant trauma especially with long bone fracture (75%) e.g. tibia or distal radius.[2] Other trauma without fracture that can predispose to ACS include crush injury, severe thermal burn, overlying constrictive bandages, vigorous exercise, penetrating trauma, injury to vascular structures in extremity and envenomation.

Lying on a limb in an unconscious or obtunded patient due to drug overdose can cause ACS. A study was published in 1979, which measured intra-compartmental pressures in various positions common in drug overdoses (Table 1).[3]

| Position | Average pressure |
|---|---|
| Head resting on forearm | 48mmHg |
| Forearm under rib cage | 178mmHg |
| One leg was folded under the other | 72mmHg |
| Normal compartment | 0-4mmHg |

Table 1. Average intra-compartmental pressures in various positions in drug overdose

**What is the pathophysiology of ACS?**

Any condition that results in an increase in compartment contents or reduction in a compartment's volume can lead to the development of an acute compartment syndrome. The most widely believed hypothesis for ACS is the arteriovenous pressure gradient theory.[4]

When the compartment pressure is elevated, venous pressure increases and capillary blood flow is compromised. Oedema of the soft tissue within the compartment further raises the intra-compartment pressure, which compromises venous and lymphatic drainage of the injured

area. Pressure, if further increases in a reinforcing vicious circle, can compromise arteriole perfusion, leading to further tissue ischaemia (Figure 2).

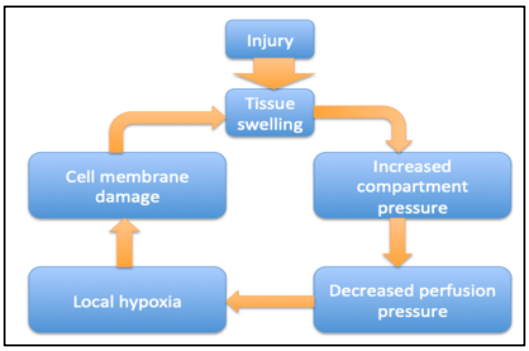


Figure 2: Pathophysiology of ACS

Compartment perfusion is compromised when the compartment pressure rises to within 30 mmHg of diastolic pressure and ischaemia begins.[5] If left untreated, ACS can lead to more severe conditions including rhabdomyolysis and renal failure, which is potentially lethal.

**If the patient is conscious, what are the symptoms of ACS?**

Symptoms of ACS include

- Pain out of proportion to apparent injury
- Persistent deep ache or burning pain
- Paraesthesia (nerve dysfunction)

Physical signs of ACS include

- Pain with passive stretch of muscles in the affected compartment
- Tense compartment with a firm “wood-like” feeling
- Pallor from vascular insufficiency
- Diminished sensation
- Muscle weakness (late)
- Paralysis (late)

However, data supporting the accuracy of the above findings is limited.[5] A systematic review of four studies of ACS associated with tibial fracture found common clinical findings e.g. pain, paresthesia and pain with passive movement, to have a poor sensitivity and specificity.[6]

Pain "out of proportion to injury" is often described as an early and sensitive sign of ACS. Nevertheless, pain can be nonspecific.[4] Most patients at risk of ACS have sustained trauma, and a fracture or an injury to a nerve or soft tissue may be the source of pain. Furthermore, associated injuries with severe pain can also distract patients from the pain that stems from an ACS.

Pain in response to passive stretching of muscles within the affected compartment is another widely described sensitive early sign of ACS, but it too may be unreliable in some patients.[7]

Paraesthesia or numbness is an unreliable early complaint because peripheral nerve injury may result directly from trauma or from an ACS.[5,8] Sensory deficits typically precede motor deficits and manifest distal to the involved compartment. Decreased 2-point discrimination is a more reliable early test and can be helpful in diagnosis.

The traditional 5P’s of acute limb ischaemia (pain, paresthesia, pallor, pulseless and poikilothemia) are not clinically reliable and only manifest in the late stage of compartment syndrome.

Therefore, in the presence of a painful tense muscle compartment, obtaining immediate surgical consultation and compartment pressure measurements are important.

The forearm contains four interconnected compartments (Figure 3)

- Superficial volar (flexor)
- Deep volar
- Dorsal compartment (extensor)
- Mobile wad compartment

Elevated pressures most commonly affect the volar compartments but the dorsal and mobile wad compartment may also be involved, alone or in addition to the volar compartment.

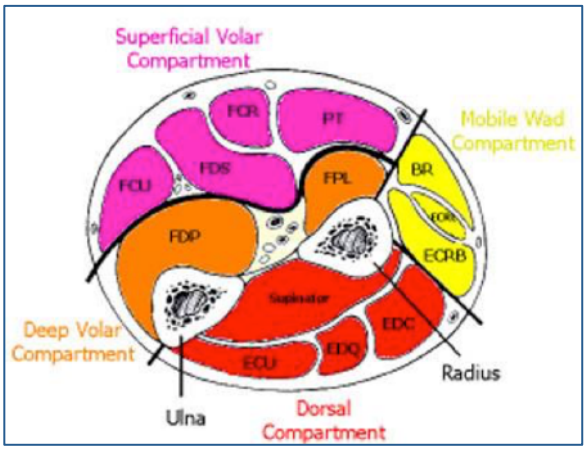


Figure 3: Compartments in forearm

**How to confirm the diagnosis of ACS?**
Measurement of intra-compartmental pressure remains the standard for diagnosis of compartment syndrome.

The Stryker intra-compartmental pressure monitor system (STIC) (Figure4) is a handheld portable device that is easy to use without the need for complex equipment.

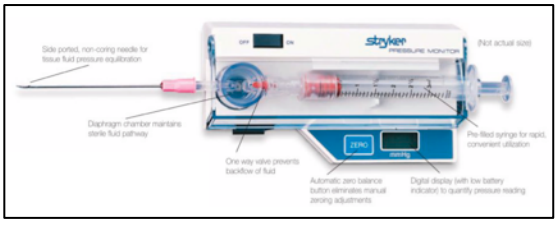


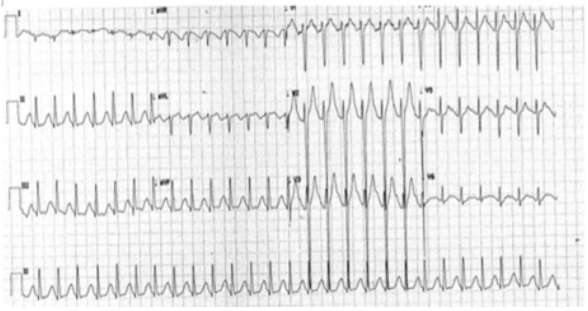
Figure 4: Stryker intra-compartmental pressure monitor system (STIC)

A STIC is introduced into the compartment (Figure 5). Measurements must be made in all compartments.


Figure 5: STIC is introduced into the compartment

The normal compartment pressure is 0-4mmHg. The exact pressure threshold for diagnosing ACS is controversial but typical ranges are from 30-45mmHg at rest. Some sources state that it is better to associate this pressure to diastolic pressure (i.e. within 10-30 mm Hg of diastolic pressure).

**Progress of the patient**
ECG and point of care test for blood were performed.



POCT: Rapid point
- pH: 6.903, BE: -8.7
- K=6.4mEq/L, Na = 147.7mEq/L
- PCO2: 19.55kPa

Serum creatine kinase: 32014IU/L
ECG:
- Sinus tachycardia (167beats per minutes)
- Peaked T wave in V2-3

Blood tests showed severe mixed respiratory (elevated pCO2) and metabolic (negative BE) acidosis (pH < 7.3) with hyperkalaemia.

**Please explain the above investigation result**
The patient was last seen well 10 hours ago and he was under the effect of hypnotics. The ACS of his right upper limb had probably developed for several hours leading to muscle ischaemia and rhabdomyolysis.

Rhabdomyolysis means disintegration of striated muscles that results in release of muscle cells contents into the extracellular fluid.[9] Among these substances, myoglobin, potassium and lactic acids are the most important ones.

Release of lactic acids into the systemic circulation will cause high anion gap metabolic acidosis. Myoglobin is filtered out in the glomerulus, but once the renal threshold is exceeded, it precipitates in the distal convoluted tubules causing obstruction, myoglobinuria and renal failure.

The release of potassium in the circulation causes wide QRS complex and arrhythmias (Figure 7).

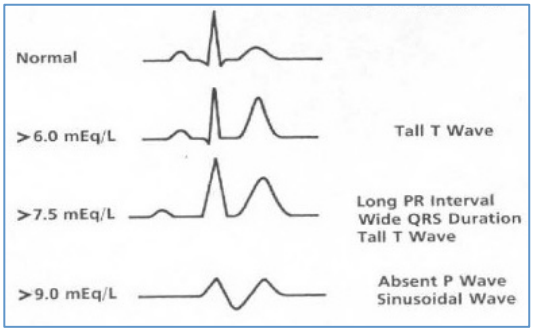


Figure 7: ECG changes with hyperkalaemia

Finally, the respiratory acidosis was related to hypnotics overdose leading to respiratory depression.

**What is the treatment for rhabdomyolysis?**
In rhabdomyolysis, the combination of hypovolaemia, acidaemia, and myoglobinaemia may cause acute renal failure.

Aggressive and early hydration with isotonic sodium chloride solution is important for the prevention of renal failure. Because injured myocytes can sequester large volumes of extracellular fluid, crystalloid requirements may be surprisingly large.

The composition of repletion fluid is controversial and may also include sodium bicarbonate. Urinary alkalization with sodium bicarbonate or sodium acetate is unproven, as is the use of mannitol to promote diuresis.

**Progress of patient**
Intensivists and orthopaedic surgeons were consulted. Fluid resuscitation, mechanical ventilation and treatment for hyperkalaemia were commenced. He was directed to the operating theatre for emergency operation.

**What is the treatment for ACS?**
Immediate management includes relieving all external pressure on the compartment e.g. dressing, splint or cast.

The limb should neither be elevated nor placed in a dependent position. Placing the limb level with the heart helps to avoid reduction in arterial inflow and increase in compartment pressures from dependent swelling, both of which can exacerbate limb ischaemia.[10, 11]

The definitive therapy for compartment syndrome is emergency fasciotomy to release the involved compartment, with subsequent fracture reduction or stabilization and vascular repair, if needed.

Principle of fasciotomy
- Adequate and extensive incision
- Complete release of all involved compartment
- Preservation of vital structure
- Thorough debridement
- Skin coverage at a later date (7-10days)

There is no consensus regarding the exact pressure at which fasciotomy should be performed. A measured compartment pressure of >30mmHg or whenever diastolic pressure minus compartment pressure <30 mm Hg were cited as an indication for fasciotomy.

Whichever the indication, prompt treatment is necessary. Studies showed that nerve tissue shows abnormal function after 30 minutes of ischaemia while muscles have functional impairment after 2-4 hours of ischaemia and irreversible functional loss after 4-12 hours.[12]

Delays in performing fasciotomy may result in[13,14]

- Muscle contracture
- Sensory deficits
- Paralysis
- Infection
- Fracture nonunion
- Limb amputation

Therefore, when in doubt, the compartment should be released.

**Progress of patient**

The compartment pressure was measured before the operation

- Volar: 60mmHg
- Dorsal: 140mmHg
- Mobile wad: 30mmHg

A volar S-shaped incision was made from the thenar crease to elbow (Figure 8). The volar, dorsal and mobile wad compartment were released. After the operation, the patient was transferred to ICU. A staged operation was planned followed by rehabilitation and psychiatric consultation.

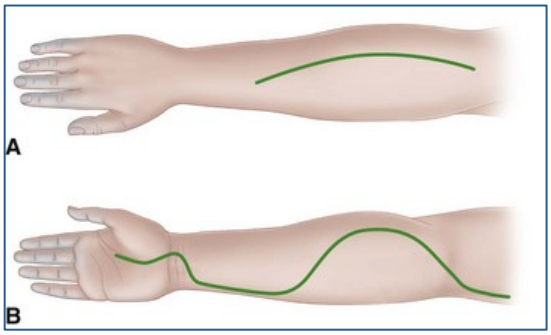


Figure 8: S-shape incision fasciotomy

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