

Acute compartment syndrome of the forearm: A case report of radius fracture with concomitant brachial artery transaction

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SUMMARY

Acute compartment syndrome (ACS) is a surgical emergency that requires timely recognition and early management for a good outcome. We report a case of a 71-year-old male seen at the Emergency Department of Universiti Sains Malaysia (USM), Malaysia, on anticoagulant therapy for valvular atrial fibrillation, who had sustained a closed fracture of the left distal end radius following a fall. Examination of the left upper limb showed deformity and tenderness over the left wrist, associated with swelling and a tense anterior compartment with blisters formation and rapid expansion of hematoma at the cubital fossa away from the fracture site. Both radial and brachial pulses were absent and confirmed with the absence of a doppler signal over the brachial and radial artery and CT angiography of the left upper limb showed there was a vascular injury of the brachial artery. Fasciotomy of the left upper limb and revascularization of the left brachial artery was done. Intraoperative findings showed a tight anterior compartment with muscle bulging upon compartment release with a complete cut of the left distal brachial artery. The primary end-to-end vascular anastomosis was done and distal circulation was restored. The distal end radius fracture was treated conservatively. The patient underwent split skin grafting of the left forearm after 6 weeks post-injury and went home well. It is critical to recognize a concomitant vascular injury in fracture-related ACS as the clinical feature may overlap. Failure in detection of concomitant vascular injury may lead to emergency fasciotomy without vascular exploration and repair.

INTRODUCTION

Acute compartment syndrome (ACS) is a surgical emergency. Recognizing ACS in a closed distal end fracture of the upper limb with concomitant proximal acute vascular injury may be challenging and easily overlooked. Overlapping of the clinical features between these two conditions in ACS may lead to emergency fasciotomy without a complete vascular assessment and investigations. We report a case of ACS with acute left brachial artery injury in a patient with a closed fracture of the left distal end radius.

CASE REPORT

A 71 years old gentleman with underlying hypertension and chronic rheumatic heart disease with valvular atrial fibrillation (AF), on warfarin therapy was brought to the Emergency Department (ED) of the Universiti Sains Malaysia (USM), Malaysia after sustaining an injury to his left forearm. He fell onto an outstretched hand while herding his cow with a rope. Initially, he sustained swelling and deformity of his left wrist. As time progressed, his entire forearm becomes progressively swollen and painful.

The injury occurred at around 1400H and he arrived at the ED 4 hours later. He was triaged to the Red Zone and was attended to immediately. He was alert, in pain as shown by a constant facial grimace. He was normotensive with good oxygen saturation under room air. His cardiac monitoring showed AF with a rate of 120-130 beats per minute (bpm). His rate slowly improved to 90-100 bpm after the patient was given adequate analgesia. The primary survey was unremarkable. Examination of the left upper limb showed deformity and tenderness over the left wrist, associated with swelling and tense anterior compartment, extending to the elbow joint. There were formation of blisters and rapid expansion of hematoma at the cubital fossa. Both wrist and elbow joints had a restricted range of movement (ROM). The pain was aggravated by the passive stretching of the fingers. The sensation was reduced over the median nerve distribution. The fingers were in dusky coloured and cold (Figure 1A and 1B). The capillary refill time was prolonged. Radial and brachial pulses were absent. Left radius and ulna radiograph showed distal end radius fracture (Figure 2A and 2B). Blood investigations showed a slightly elevated total white cell count of 13.9×10^9 , haemoglobin of 12.5g/dL, platelet of 253×10^9 , INR of 2.10, a significant rise in CK level of 12,313 and potassium level of 4.1 mmol/l.

He was subsequently reviewed by the Orthopaedics team. He was clinically diagnosed with ACS even though no intracompartment pressure measurement was done due to the unavailability of the equipment. He also was suspected to have vascular injury due to the absence of doppler signal of brachial and radial arteries based on the bedside handheld Doppler device. Computed tomography angiography (CTA)

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Fig. 1: The red arrow shows the left fingers appear swollen and dusky compared to the right fingers. The yellow arrow shows deformity and swelling over the wrist. The black arrow shows blister and hematoma formation over the medial cubital fossa proximal to the distal end radius fracture.

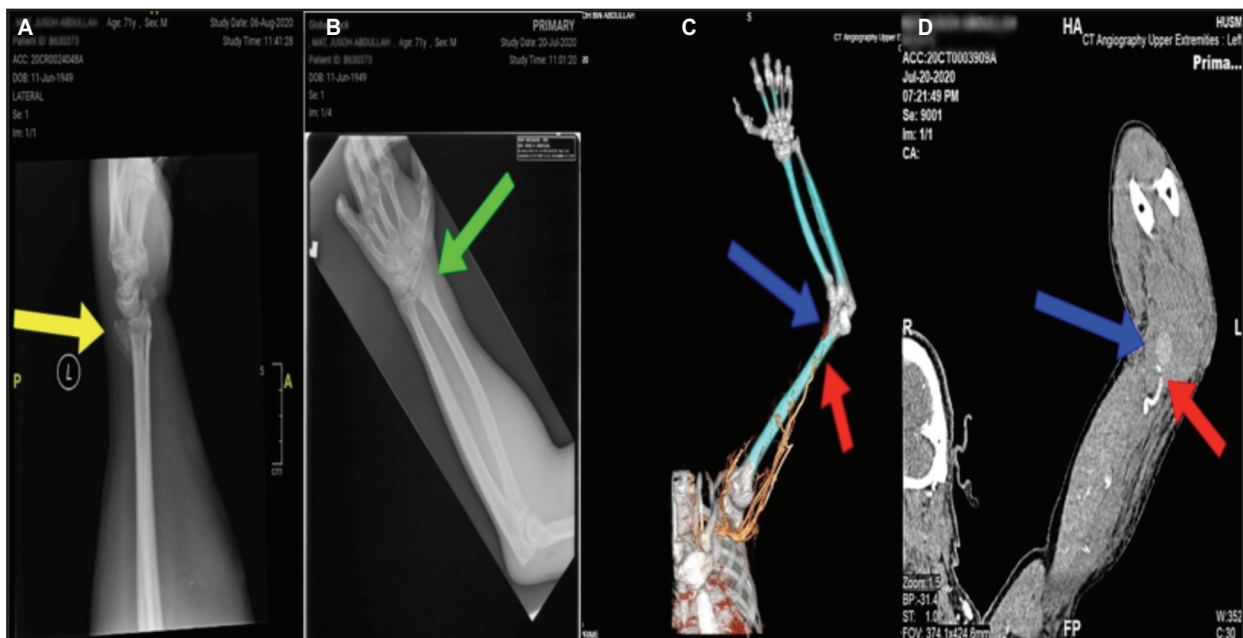


Fig. 2: Yellow arrow shows dorsal angulation of distal end radius on the lateral view of radius-ulna radiograph. The green arrow shows a linear fracture of the distal end radius on the anteroposterior Radius-Ulna radiograph. The red arrow shows abrupt non-opacification to the distal of the left brachial artery, while the blue arrow shows contrast extravasation adjacent to the distal brachial artery which appears to be connected to the injured vessel on the volume rendering technique reconstruction image and coronal reconstruction image of the left upper limb computed tomography angiography.

of the left upper limb was done and the result showed there was an abrupt non-opacification of the distal left brachial artery with contrast extravasation in all phases in keeping with vascular injury (Figure 2C and 2D).

The patient was sent to the Trauma Operation Theatre after 7 hours of the injury for fasciotomy of the left upper limb and

revascularization of the left brachial artery. Intraoperative findings showed a tight anterior compartment with muscle bulging upon compartment release. There was a complete cut of the left distal brachial artery before bifurcation to the radial and ulnar artery with surrounding hematoma over the cubital fossa, extending to the proximal forearm. The primary end-to-end vascular anastomosis was done by the

Reconstructive and Plastic Surgery team. Distal circulation was restored as shown by the post-procedural doppler where a triphasic signal was present. The colour of the fingers became pink and there was normal capillary refill time. The distal end radius fracture was treated conservatively.

The raised CK level of 12,313 indicated as a sign of rhabdomyolysis, as a complication of ACS. The patient was put on adequate hydration with a targeted urine output of 3 to 4 ml/kg/h monitoring. The warfarin was temporarily withheld, and he was started on subcutaneous enoxaparin sodium on day one post-operation. The CK value gradually showed downtrend and normalized on day 18 post-injury and no acute kidney injury ensued. 6 weeks post injury, the patient underwent split skin grafting of the left forearm and was discharged home well.

DISCUSSION

ACS occurs when tissue pressure within a closed muscle compartment exceeds the perfusion pressure and results in muscle and nerve ischemia. It is considered a surgical emergency that requires timely recognition and early management for a good outcome. Unrecognised ACS may lead to a non-viable limb. The flexor compartment of the forearm is the commonest site and fracture-related is the most common cause of ACS in the upper limb extremity.¹ Injury to soft tissues without fracture is the second most common cause of ACS and one-tenth of the patients had a bleeding disorder or were taking anticoagulant drugs.²

The diagnosis of ACS is always controversial and is based on the clinical hallmark of 5 Ps' - pain, paraesthesia, pallor, paralysis, and pulselessness. However, the most sensitive clinical signs is disproportionate pain and pain on passive muscle stretching (sensitivity 98%, negative predictive value 98%).³ The remaining 4 P's of ACS are mostly late signs after prolonged ischemia and subsequent neurovascular injury. Pulselessness and paralysis are rare features and usually occur after an arterial injury or after a substantial amount of time has elapsed, particularly in a patient on anticoagulation therapy.⁴

Our patient had all the classic 5 Ps' for ACS during the presentation. It is critical to consider a vascular injury as a cause of ACS if there is a rapid absence of distal pulses and expanding hematoma. In ACS case without vascular injury, the peripheral pulses will be preserved until long after the intra-compartment pressure exceeds the systemic blood pressure and the large arteries are compressed.⁵ Pulselessness is considered a late sign. The patient was on anticoagulant (warfarin) therapy for his valvular AF and this should raise the suspicion of haemorrhage that lead to ACS, even though his INR was within the therapeutic range.

The decision either to proceed for operation or perform a CTA of the left upper limb first was crucial in this case. CTA is the gold standard for vascular injury as it provides 100% sensitivity and specificity. Bedside ultrasound doppler did show an absence of radial and brachial pulse. However, this finding was inadequate to distinguish ACS as the sole cause or concurrent presence of vessel injury as one of the causes.

Location, nature, and extent of the vascular injury can be objectively identified with CTA and thus guide the approach and type of repair (either end-to-end anastomosis or venous graft). Furthermore, by obtaining CTA, the prognosis and soft tissue viability could be determined via the establishment of collateral circulation.⁶

A CTA of the affected limb must be performed to evaluate the presence, types and level of vascular injury. A volume rendering technique (VRT) and coronal reconstructive CT provide the site and types of vascular injury like spasm, intimal tears, intramural haematoma, pseudoaneurysms, arteriovenous fistula (AVF), vessel transection or avulsion. In this patient, the finding was an abrupt non-opacification to the distal left brachial artery with contrast extravasation in all phases, in keeping with vessel transection. With the imaging, it helps the surgeon to locate the injury and guide the approach and type of vascular repair. Urgent fasciotomy and vascular exploration were required.

CONCLUSIONS

ACS is a clinical diagnosis. ACS caused either by a fracture or vascular injury can manifest as pulselessness on the affected limb. Failure to identify vascular injury may result in only fasciotomy is done without vascular exploration and repair. We recommend that in the setting of minor trauma, past medical history of anticoagulant use with clinical findings of rapid absence of distal pulses and rapid expansion of hematoma away from the fracture site warrant a CTA. Vascular injury must be ruled out prior to fasciotomy and vascular exploration. A VRT reconstruction image and coronal reconstruction image yield an excellent anatomical site, types and the extent of vascular injury.

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