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Rare complication of four extremity compartment syndrome requiring fasciotomy from influenza A viral myositis

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Rare complication of four extremity compartment syndrome requiring fasciotomy from influenza A viral myositis

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SUMMARY

Influenza A and B commonly cause benign respiratory disease in humans, but can cause more severe illness in high-risk populations. We report an unusual case of a previously healthy adult patient who presented with myositis and severe rhabdomyolysis secondary to influenza A infection that resulted in atraumatic compartment syndrome of all four extremities, each requiring emergent fasciotomy. The patient was subsequently managed with delayed primary closure and skin grafting in the operating room. Prompt recognition of this rare complication by the team resulted in no limb amputations. On his first follow-up appointment, 1 month after discharge, he had regained full functionality in both his hands and his feet were both close to 50% of baseline and improving with physical therapy.

BACKGROUND

Influenza A and B are RNA viruses, and members of the Orthomyxoviridae family, which typically cause benign respiratory disease in humans, but can be associated with serious complications. These include, but are not limited to, viral or secondary bacterial pneumonia, rhabdomyolysis, myocarditis and encephalitis.[1] Annual influenza epidemics are estimated to result in three to five million cases of severe illness, and 290 000–650 000 respiratory deaths worldwide;[2] however, the frequency of complications, such as rhabdomyolysis and compartment syndrome remains unclear.[3] We report an unusual case of a previously healthy adult man who presented with severe rhabdomyolysis secondary to influenza A viral myositis, resulting in compartment syndrome of all four extremities, each requiring fasciotomy.

CASE PRESENTATION

A previously healthy 45-year-old man with a medical history of antinuclear antibody-positive arthritis (not on medication) and smoking, left work early with viral symptoms on 27 December 2019. Symptoms included cough, fevers, myalgias, arthralgias and chills. The patient did not receive his annual influenza vaccine, had no recent travel and no sick contacts. Over the weekend, he remained in bed due to diffuse weakness. On 30 December 2019, the patient presented to the local community hospital emergency department with worsening symptoms. He was given 2 L of intravenous fluids and was discharged home with the diagnosis of viral syndrome. At this time, influenza testing was not done and he was not treated with an antiviral medication. However, following Centers for Disease Control and Prevention influenza guidelines, the patient likely should have been tested and treated based on worsening symptoms suggestive of musculoskeletal complication (eg, myositis and rhabdomyolysis).[4] The following day, he was unable to stand in the shower and his wife called emergency services. He was transported via ambulance to the local community hospital.

At the hospital, initial diagnostic work-up was notable for positive influenza A/H1, white blood cell count 23.50×10^9/L, lactate 9.3 mmol/L, creatine kinase 6400 U/L, aspartate transaminase 2358 U/L, alanine transaminase 654 U/L and chest CT showed bilateral pulmonary infiltrates.

The patient's wife noted that while waiting in the emergency department for admission, she had already noticed bilateral arm swelling with decreased movement. The patient received a 30 mL/kg sepsis fluid bolus totalling 3 L and was placed on maintenance intravenous fluids. His intravenous access included one peripheral intravenous catheter and a triple lumen central venous catheter. Throughout his stay, the intensive care unit team had difficulty obtaining additional intravenous access. On hospital day 2, the decision was made to transfer the patient to a tertiary care centre for escalation of care. On arrival to the medical intensive care unit, on hospital day 3, he was alert and oriented to person, time, place and situation, afebrile, making appropriate urine and haemodynamically stable on 3 L nasal cannula. However, his respiratory status rapidly deteriorated and he was intubated at 07:00.

According to the medication administration record, on hospital day 3, he received 1 L fluid bolus of normal saline and was on 75 mL/hour of lactated Ringer's. On admission at 12:15, his creatine phosphokinase was up to 110 990 U/L from 6400 U/L 2 days ago. In addition, his creatinine increased rapidly from 0.86 mg/dL on transfer to 2.50 mg/dL by 17:00 with anuria. Shortly thereafter, the plastic surgery team was consulted for possible compartment syndrome as the primary team noted skin mottling and increasing tension in all four extremities. When we first evaluated the patient, at 16:00, his vital signs were blood pressure 101/59 mm Hg (on norepinephrine), heart rate 151, temperature 38.2°C and respiratory rate 42. He was intubated, diaphoretic and breathing over

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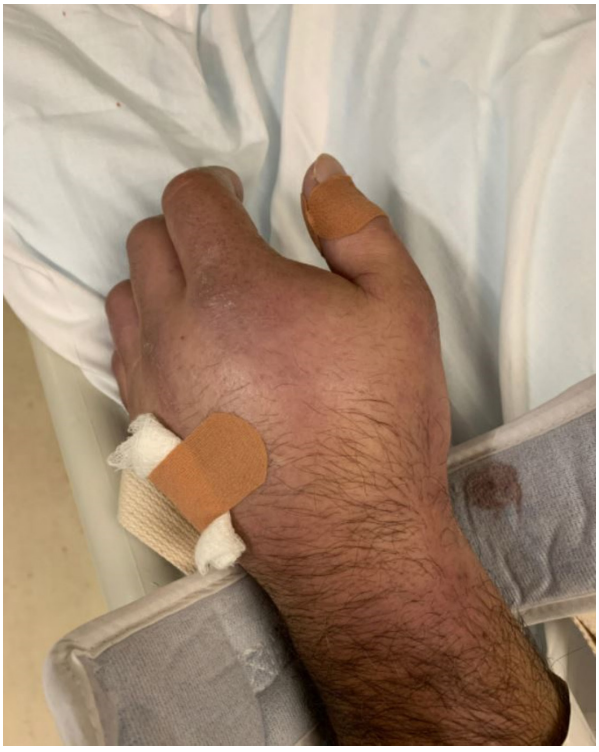

Figure 1 Patient's left dorsal hand with notable tense and swollen appearance.

the ventilator with heavy sedation. On physical examination, the compartments of his hands, forearms and legs were tense bilaterally (figure 1). His hands and legs were cool to touch with prominent mottling (figures 2 and 3). Fingernail capillary refill was 3–5 s. There were dopplerable radial pulses with no palmar or digital pulses bilaterally.

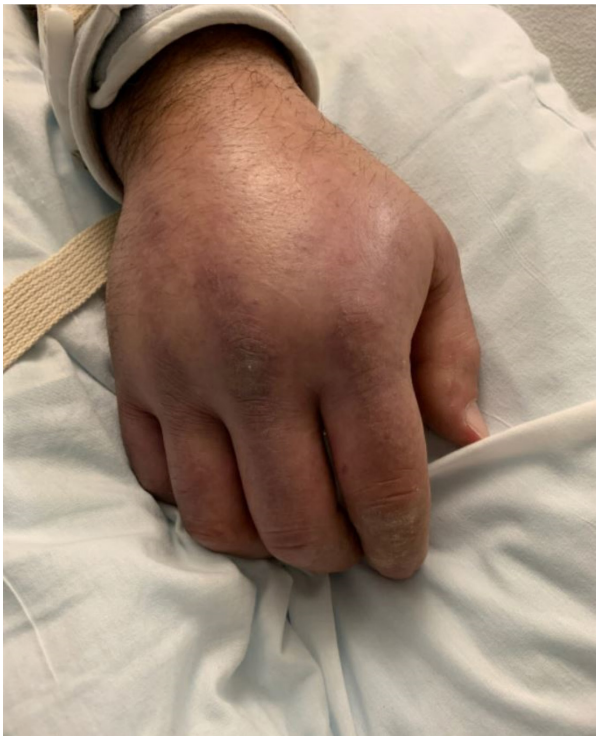

Figure 2 Patient's right dorsal hand with prominent mottling.

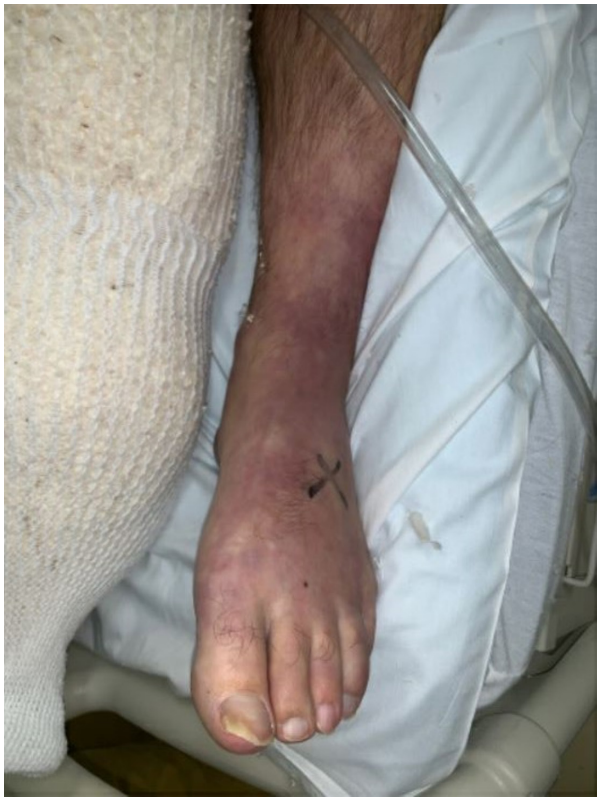

Figure 3 Patient's left anterior leg with prominent mottling.

INVESTIGATIONS

The patient's examination was concerning for compartment syndrome in all four extremities even in the setting of no recent trauma, crush injury, severe thermal burns, penetrating trauma and injury to vascular structures. The diagnosis was confirmed at bedside with the Stryker Intra-Compartmental Pressure Monitor System (Stryker Surgical, Kalamazoo, Michigan): right arm volar 40 mm Hg, right arm dorsal 33 mm Hg, left arm dorsal 44 mm Hg, left arm volar 47 mm Hg, and lower extremity lateral and posterior compartment pressures 25–35 mm Hg.

DIFFERENTIAL DIAGNOSIS

A diagnosis of compartment syndrome was made.

TREATMENT

With a confirmed diagnosis of compartment syndrome, consent was obtained for emergent fasciotomy. A sinusoidal volar incision was made in the forearms, bilaterally (figure 4). The brachioradialis muscle belly was identified and the overlying fascia was incised. The muscles in this region were noted to be swollen with thrombosed veins, which were non-responsive to electrocautery. The pronator teres was identified, and adjacent fascial compartments released. On the dorsal aspect of the hand, incisions were made within the second and fourth metacarpal interspaces. The interosseous muscles were released and also noted to be swollen and non-reactive to electrocautery. An incision was made over the thenar and hypothenar eminence to release the compartments. After upper extremity compartmental release, the right radial and ulnar arteries did not produce an audible Doppler signal. Therefore, Guyon's canal in the right hand was released to further decompress the adjacent neurovasculature. The left brachial, ulnar and radial arteries did produce audible Doppler signals; however, no signal was produced in the palmar region.

Figure 4 Postoperative day 1 right forearm with sinusoidal volar incision.

For both lower extremities, a longitudinal incision was made along the medial surface of the calf. Once adequate decompression was achieved in the superficial and deep medial compartments, the lateral compartment was subsequently released. The medial incisions were meshed with vessel loops (figure 5). The lateral incisions were primarily closed.

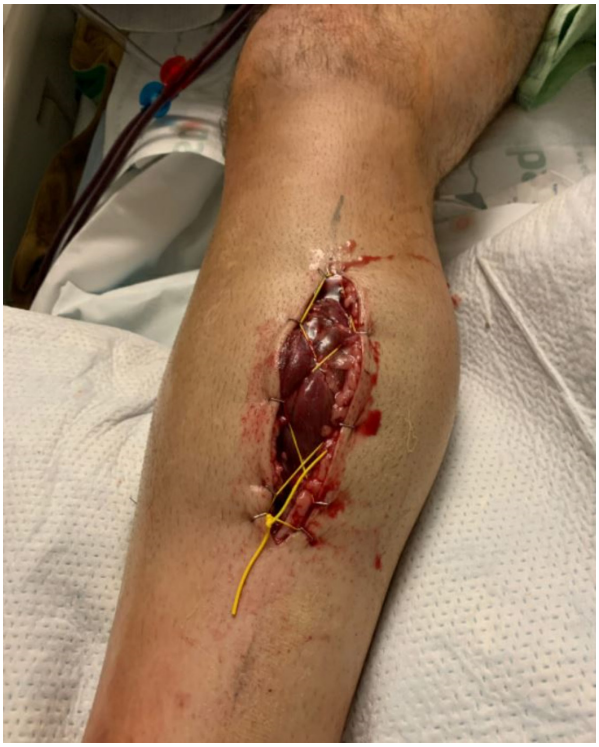

Figure 5 Postoperative day 1 right medial leg with vessel loops.

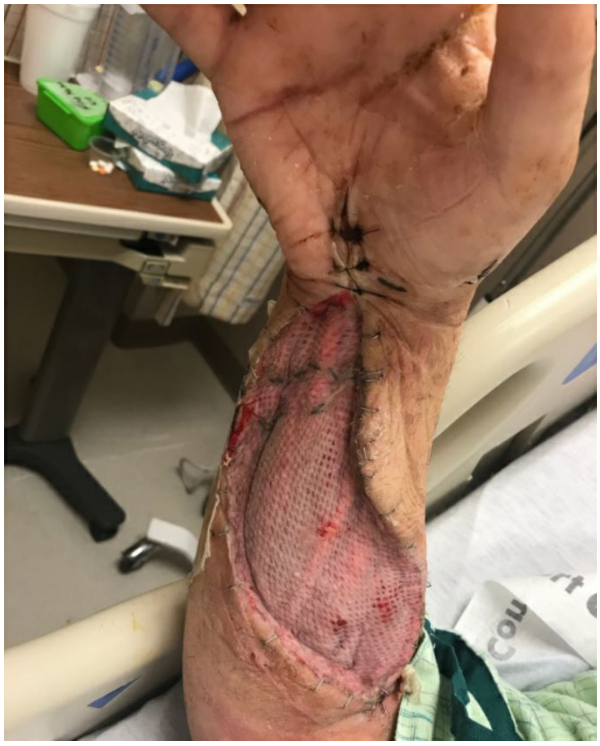

Figure 6 Well-healing right volar fasciotomy site with skin graft.

On postoperative day 1, the compartments were less tense and warm on examination. Audible Doppler signals bilaterally were noted to the palmar area, dorsalis pedis and posterior tibial. On day 4, the patient's creatine phosphokinase peaked at 140 850 U/L and rapidly declined to 54 087 U/L on day 6 and 1448 U/L on day 7. Over the course of the next 2 weeks, daily dressing changes were completed in addition to bedside closure of the hand incisions. Splints were placed on the upper extremities to minimise contracture.

On hospital day 17, the patient was taken to the operating room for delayed primary closure and skin grafting. The upper extremity fasciotomy sites were primarily closed with the exception of the volar forearm incisions, which were covered with split-thickness skin grafts (figure 6). The left lower extremity medial fasciotomy was primarily closed. The right medial incision site was covered with a split-thickness skin graft. Skin grafts were harvested from the thigh. Negative pressure dressings were placed on all graft sites.

The wound vacuum-assisted devices were removed on hospital day 24 and he was discharged home on hospital day 34.

OUTCOME AND FOLLOW-UP

The patient was seen in follow-up plastic surgery clinic 1 month later. Overall, he reported feeling well and was working with physical therapy with major improvement. He did endorse burning sensation in his feet, before which he had no sensation. His estimation was that his right and left feet were 50% and 40% better, respectively. Functionally, he was back to his baseline with his hands. On physical examination, he was vascularly intact with decreased sensation in the saphenous and tibial nerve distributions with well-healing scars at all fasciotomy and skin graft sites. Nine months after the surgery, the patient continues to struggle with nerve damage, particularly to his right foot. However, his scars have healed without any additional complication and he has full functionality (figures 7 and 8).

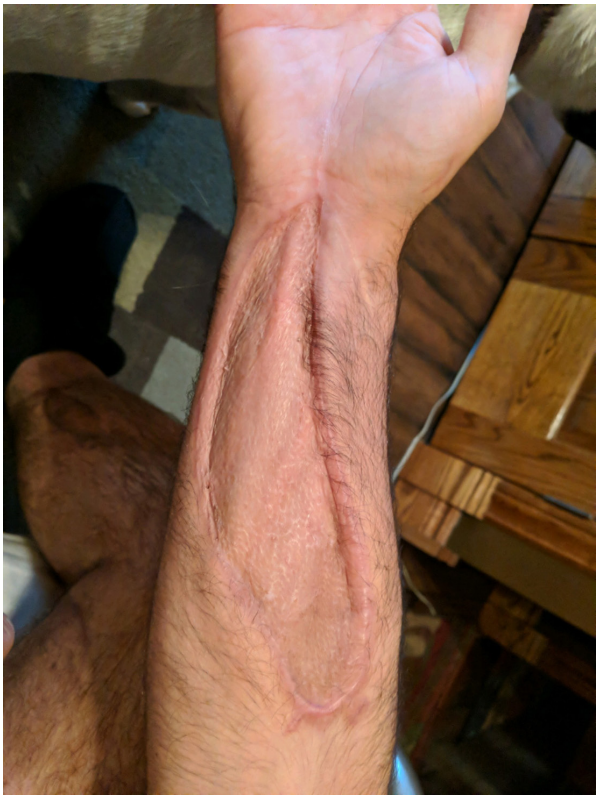

Figure 7 Nine months postoperative right forearm.

DISCUSSION

Further studies are needed to more accurately determine the frequency of influenza causing rhabdomyolysis; however, it appears to be an uncommon occurrence.[1 3 5–10] One study looking at 316 paediatric patients with influenza A or B found rhabdomyolysis occurred in only 3% of patients and led to renal failure and compartment syndrome in 80% and 20% of these patients, respectively.[11] Several studies have concluded that, as with our patient, rhabdomyolysis is seen more often with influenza A rather than B infections.[11 12]

The pathogenesis of influenza causing rhabdomyolysis has not been fully elucidated, but it is postulated that it occurs via three mechanisms. Direct muscle invasion by the virus, immunological reaction resulting in collateral muscle damage, and/or viral toxins causing direct muscle injury.[3 13] The mechanism whereby viruses directly invade cells has been supported by studies in vitro, where influenza A virus was shown to invade human myocytes and cause cell lysis.[14] Regardless of the mechanism, in vivo, the death of muscle cells releases myoglobin and osmotically active cellular elements causing edema of the interstitial space, which can result in compartment syndrome and the myoglobin can cause glomerular damage, leading to acute renal failure.[5]

Our patient's presentation was unique in that his rhabdomyolysis was severe enough to require fasciotomy in all four extremities. From our review of the literature, only three other cases requiring four-limb fasciotomy from influenza infection have been reported.[5 10 15] One of these was a child and the other two were patients aged 35 and 43 years, both of whom had few medical comorbidities similar to our patient.

Our patient presented 3 days before his transfer to our hospital, primarily reporting myalgias and weakness. Perhaps, if myositis and/or rhabdomyolysis was identified earlier, therapeutic interventions could have decreased the need for fasciotomy and haemodialysis. No studies to date have investigated the use of oseltamivir in modifying influenza-induced rhabdomyolysis. However, our case does raise the question on whether the patient's morbidity would have been significantly reduced if he was treated initially with oseltamivir. Influenza vaccination has been shown to modify disease severity, as one study found that vaccinated adults hospitalised for influenza were about 75% less likely to die than unvaccinated patients.[16] This further emphasises the need for healthcare providers to promote the influenza vaccine to all patients, even those not typically viewed as being high risk, such as our patient.

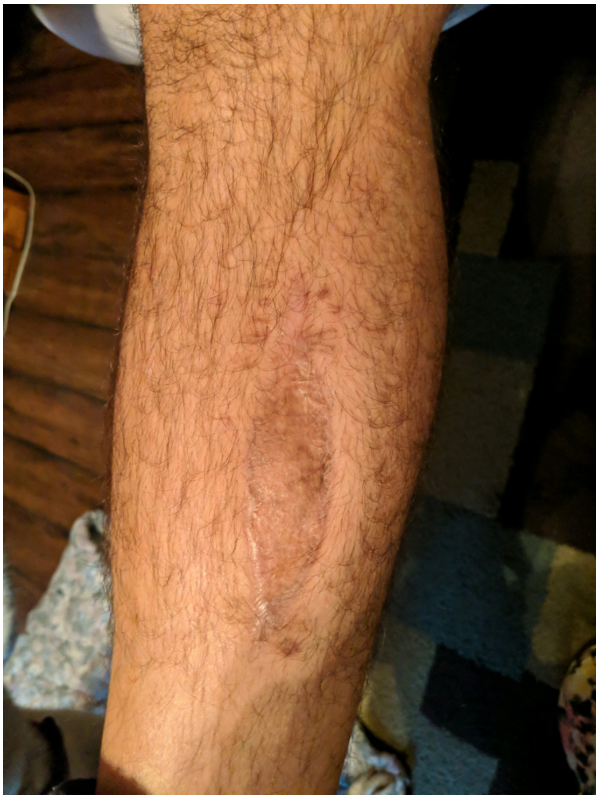

Figure 8 Nine months postoperative right medial leg.

Learning points

- Clinicians caring for hospitalised patients with influenza should be on the lookout for the less common and more severe complications even in young low-risk patients.
- Atraumatic compartment syndrome is a rare, but possible complication from influenza-induced viral myositis.
- The differential diagnosis for worsening renal failure (rising creatinine, decreasing urine output) should prompt consideration of rhabdomyolysis and ordering of a creatine phosphokinase level.
- Prompt recognition and aggressive treatment of rhabdomyolysis (fluids and haemodialysis) in the setting of influenza can lower the risk of developing compartment syndrome.
- Although compartment syndrome can be diagnosed clinically, a multistick needle catheterisation pressure reading should be obtained correctly (eg, avoid injecting local anaesthetic and regional nerve blocks; instead intravenous narcotics or conscious sedation for pain control) prior to open fasciotomy.

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