



Early detection of acute compartment syndrome

Quick action prevents serious harm.

By Aaron M. Sebach, PhD, DNP, AGACNP-BC, FNP-BC, CEN, CPEN, CNE, CNECl, SFHM

BRIAN WICK*, age 33, is admitted to the orthopedic medical-surgical unit for observation and pain management after an ATV accident. His right forearm was pinned under the vehicle, and he required assistance from a passerby to free it. An X-ray of the forearm in the emergency department shows a distal radius fracture, which is splinted by an orthopedic surgical resident. Outpatient open reduction and internal fixation of the fracture is recommended, but Mr. Wick is admitted due to uncontrolled pain.

History and assessment

Throughout the day, Mr. Wick receives oxycodone 5 mg and acetaminophen 325 mg every 4 hours as needed for pain as well as I.V. morphine sulfate 1 mg every 2 hours as needed for breakthrough pain. Neurovascular assessments of Mr. Wick's right upper extremity every 4 hours remain unchanged with a radial pulse rated 3/4, capillary refill time of 3 seconds, full sensation and finger range of motion, and warm, pink skin.

At shift change, you hear Mr. Wick calling out for pain medication. You determine that he's due for I.V. morphine sulfate 1 mg. Mr. Wick reports 10/10 burning pain and pressure in the right forearm. He also reports a pins-and-needles sensation and difficulty moving his fingers. You're unable to palpate a radial pulse and note that his skin is pale. His vital signs are temperature 98.4° F (36.9° C), heart rate 124 beats per minute, respiratory rate 22 breaths per minute, blood pressure 154/86 mmHg, and oxygen saturation 95% on room air.

Taking action

You immediately identify that Mr. Wick is likely experiencing acute compartment syndrome (ACS), an orthopedic emergency. You remove the circumferential wrap holding the splint and page the orthopedic surgeon. You do not elevate the extremity. While waiting for her ar-

rival, you administer the I.V. morphine sulfate and ask the charge nurse to bring a Doppler ultrasound to the bedside. The orthopedic surgeon confirms your assessment and hears a faint, weak radial pulse via Doppler ultrasound. She calls the operating room and plans for an emergency fasciotomy.

Outcome

The fasciotomy restores arterial flow. The wounds are left open for 4 days to allow the swelling to resolve. Mr. Wick is then taken back to the operating room for wound irrigation and closure, as well as open reduction and internal fixation of the radius fracture. He's discharged the next day and will require outpatient physical and occupational therapy with close orthopedic surgery follow-up.

Education and follow up

ACS develops as a result of increased pressure within an anatomic compartment, which can lead to decreased or absent blood flow to muscle and nerve cells. It's most common after traumatic injury to an extremity, although it also can occur after surgery. Chronic compartment syndrome, which occurs with exercise and resolves with rest, is not a surgical emergency.

ACS is a clinical diagnosis. When assessing for it, use the 5 Ps: disproportionate pain, paresthesia, paralysis, pallor, and pulselessness. Paralysis frequently is a late finding. Early identification, rapid response team activation, and surgical intervention are critical to prevent permanent disability of the affected extremity.

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*Name is fictitious.

Access references at myamericannurse.com/?p=75950.

Aaron M. Sebach is associate professor and chair of the doctor of nursing practice program at Wilmington University in New Castle, Delaware, and a hospitalist nurse practitioner at TidalHealth Peninsula Regional in Salisbury, Maryland.

About myxedema coma

Myxedema coma is the most severe, potentially life-threatening form of hypothyroidism. It can result in the slowing of multiple organs. Hallmarks of myxedema coma include altered level of consciousness (ranging from lethargy to psychosis) and hypothermia. It also can cause hypotension, hypoventilation, bradycardia, hyponatremia, and hypoglycemia.

Myxedema coma can occur in acutely ill patients with pre-existing primary or central hypothyroidism that is untreated or undertreated. Predisposing factors include infection, cold exposure, myocardial infarction, surgery, and certain drugs. Patients with undiagnosed or poorly managed hypothyroidism and older adult women are at highest risk.

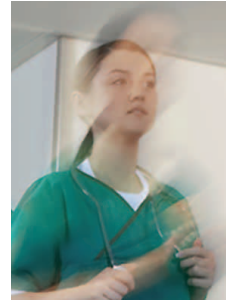
Myxedema coma diagnosis is based on history, physical exam, and exclusion of other diagnoses; it's confirmed with thyroid function studies. TSH levels are highly elevated in patients with primary hypothyroidism but may be closer to normal or only slightly elevated in those with central dysfunction. Free T4 levels usually are very low.

Myxedema coma treatment should be initiated quickly if the condition is suspected and may include both T3 and T4 I.V., although optimal treatment is still being investigated. Patients with central hypothyroidism are at risk of hypopituitarism and adrenal insufficiency, so they may be treated with stress doses of glucocorticoids until those conditions are ruled out. Hypotension and bradycardia are corrected by thyroid hormone replacement therapy, although vasopressors may be required in the short-term. Passive warming helps correct hypothermia. Treatment advances have reduced mortality rates to between 20% and 25%; however, emergent and aggressive treatment are key.

Bradykinin-induced angioedema

Quick treatment results in a good outcome.

By Veronica Y. Amos, PhD, CRNA, PHCNS-BC



MARCUS GREEN* is a 45-year-old Black man with a 5-year history of hypertension who takes his blood pressure medication on the way to work. About 20 minutes after taking it this morning, his tongue begins to feel heavy and his lips and tongue begin to swell. He drives himself to the emergency department (ED).

History and assessment

Jane Jackson, Mr. Green's nurse, conducts a quick assessment, noticing the swelling. When she learns that Mr. Green is taking lisinopril for his hypertension, she realizes he's likely experiencing angioedema. She obtains vital signs and, per protocol, starts oxygen at 2 liters/minute via nasal cannula. Mr. Green's vital signs are BP 98/60 mmHg, heart rate 128 bpm, respiration rate 24 breaths/minute, oxygen saturation 98%, and axillary temperature 98.1°F (36.7°C).

Mr. Green is alert and oriented, but he's becoming increasingly anxious and his lips and tongue continue to swell. Jane starts ECG monitoring; inserts an 18-gauge peripheral I.V. in his right arm; and administers epinephrine, methylprednisolone, and diphenhydramine, as ordered, but without effect.

Clinical management

Concerned that Mr. Green may rapidly deteriorate, the ED provider asks an anesthesia provider to perform a fiberoptic nasal intubation to protect the patient's airway. The anesthesia provider doesn't give Mr. Green a benzodiazepine or medication for pain because it may cause sedation and impede spontaneous breathing. However, the anesthesia provider does order 2 units of fresh frozen plasma (FFP) I.V.

After two unsuccessful intubation attempts, the anesthesia provider administers glycopyrrolate 0.2 mg I.V. to decrease Mr. Green's excessive bronchial secretions and prevent bronchospasm. Mr. Green remains alert and cooperative and the airway is secured nasally with an endotracheal tube. He's immediately

given propofol 200 mg I.V. as a general anesthetic, and the respiratory therapist places him on a ventilator. Mr. Green is taken to the ICU where his angioedema subsides in 4 hours and he's extubated 24 hours later. He's discharged home 3 days later, and his provider prescribes losartan, an angiotensin-receptor blocker.

Education

Angiotensin-converting enzyme (ACE) inhibitors such as lisinopril can cause bradykinin-induced angioedema (increased release of the enzyme bradykinin, which can cause increased tissue permeability with dilatation of the vessels and swelling). In the United States, 30% to 40% of angioedema diagnoses in the ED are related to ACE inhibitors, with 0.1% to 2.2% being life-threatening.

Typical signs of angioedema related to ACE inhibitors include lip, face, and tongue swelling without rash or itching. However, the larynx and pharynx also can be involved. In most cases, angioedema resolves without complications in a few days, but Mr. Green's respiratory manifestations made prompt treatment essential.

No standardized treatment for bradykinin-induced angioedema exists, but the off-label use of FFP has been shown to be an effective treatment for severe episodes. Bradykinin responds to the kininase II in FFP, which is identical to ACE.

Unfortunately, in one reported case, despite a patient receiving epinephrine, methylprednisolone, and diphenhydramine, followed by I.V. FFP, their symptoms progressed and 2 hours later intubation was required, as was the case with Mr. Green. Therefore, nurses should follow the research in this area.

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*Name is fictitious. To view a list of references, visit myamericannurse.com/?p=71927.

Veronica Y. Amos is an assistant director of the nurse anesthesia program and assistant professor at the University of Maryland School of Nursing in Baltimore.