A case of symmetrical peripheral gangrene associated with transvenous lead extraction

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Introduction

Central venous vascular tears constitute the most lethal complication encountered during transvenous lead extraction. Although they are infrequent, the case fatality rate for these events has been reported as high as 63% owing to rapid exsanguination.[1] Of those who survive these encounters, significant morbidity often follows. In this report, we describe a case of symmetrical peripheral gangrene (SPG) following endovascular laceration during laser lead extraction. SPG is a rare but well-documented clinical syndrome characterized by ischemic necrosis simultaneously involving the distal portions of 2 or more extremities without any proximal arterial obstruction or vasculitis.[2,3]

Case report

At an outside facility, a 57-year-old Hispanic man with a history of coronary artery disease, myocardial infarction, and ischemic dilated cardiomyopathy presented to the emergency department with a pocket infection of his cardiac resynchronization therapy defibrillator. His initial device and leads were implanted 9 years ago and his system underwent a generator change 6 months ago. He had a 2-month history of erythema and discomfort around his left upper chest implant site that had progressively worsened to include edema and serosanguinous drainage, despite oral amoxicillin treatment. He was nonbacteremic and afebrile. Laboratory investigations were within normal limits. Echocardiography revealed severely reduced left ventricular systolic function with an ejection fraction of 20%–25% and global hypokinesis of the left ventricle. No evidence of vegetations was seen. Preoperative computed tomography revealed that his leads were scarred to the lateral wall of the superior vena cava.

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The patient was taken to the operating room for transvenous lead extraction. The cardiac resynchronization therapy defibrillator pocket capsule was dissected out and the device removed. The coronary sinus and right atrial leads were extracted with gentle traction and laser assistance, respectively. When the 16F laser sheath passed the first coil of the right ventricular lead, however, the patient suddenly became hypotensive, and transesophageal echocardiography revealed a large pericardial effusion. An emergency midsternotomy was performed and upon opening of the pericardium, a significant amount of blood was seen obscuring the operative field. Bleeding was manually controlled with pressure until cardiopulmonary bypass was instituted. The surgeon located a 5-mm tear in the superior cavoatrial junction, a perforation in the right atrium, and an oozing hematoma at the level of the innominate vein. All 3 lesions were repaired with multiple 4-0 polypropylene sutures with pledgets. The right ventricular lead was capped and abandoned. The patient was taken off cardiopulmonary bypass and an intra-aortic balloon pump was placed through the left femoral artery owing to the patient's growing hemodynamic instability. Following multiple blood transfusions, the patient developed a coagulopathy, which improved after transfusions of cryoprecipitate, platelets, fresh frozen plasma, and factor VII. The chest was closed in standard fashion once his bleeding was under control. In total, 26 units of blood products were given intraoperatively, 10 of which were packed red blood cells.

Postoperatively, he was transferred to the intensive care unit in critical condition, where he developed severe cardiogenic shock and multiorgan failure. The patient was hypotensive (79/48 mm Hg, pulse 99 beats/min) and required large doses of vasopressin, epinephrine, and norepinephrine (50 mcg/h) over several days. Suffering from hypoxic respiratory failure, he remained dependent on mechanical ventilation. His liver failure was managed with albumin and multiple blood products were given in response to persistent coagulopathy. Broad-spectrum antibiotics were adjusted to treat his underlying infection. The patient was oliguric and on postoperative day (POD) 1 he was placed on continuous venovenous hemodialysis for acute renal failure.

In the subsequent days, the patient began exhibiting bilateral, symmetrical cyanotic changes to all 5 digits of his upper and lower extremities. Vasopressor administration was stopped

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KEY TEACHING POINTS

- The lethality of central venous lacerations during transvenous lead extraction remains high. Associated morbidities can follow patients long after successful resuscitation.
- Symmetrical peripheral gangrene is a rare complication following hemodynamic collapse, with sinister prognostic implications in terms of loss of life or limbs.
- Rescue protocols should encompass strategies that delay hemodynamic collapse and decompensation in the event of vascular injury during lead extraction.

upon recognition of marked surface pallor and coldness in the affected areas. By POD 9, however, upper- and lower-digit ischemia had progressed to dry gangrene. Upon evaluation, the patient complained of dull pain and had no ability to move his fingers and toes, with bilateral stiffness, 2+ pitting edema, and nonexistent capillary refill time. Palpable 2+ peripheral pulses were noted and Doppler study showed flat waveforms on all digits and toes bilaterally without proximal occlusion or stenosis.

The patient's condition improved over the following month. His intra-aortic balloon pump and endotracheal tube were removed by POD 7. Albumin was discontinued and liver enzymes returned to normal limits by POD 11. Kidney function gradually improved and hemodialysis was stopped on POD 41. Over this period, the patient's mental status improved as he was weaned off sedation. His necrotic lesions were treated conservatively with povidone-iodine dressings. Despite debridement on POD 27 and negative-pressure wound therapy, however, he continued to drain purulent, foul-smelling material from his left infraclavicular operative site.

For that reason, the patient was stabilized and transferred to our facility on POD 54 for further management of his pocket infection. Preoperative transesophageal echocardiography revealed a diminished ejection fraction of 10%–15%. He subsequently underwent laser extraction of his retained lead on POD 58 without complications (Figure 1). Of note, 60 mL of pus was drained from the subfascial area within the infraclavicular space and the patient was started on the appropriate antibiotic regimen after microbial cultures grew *Enterobacter cloacae* and *Staphylococcus epidermidis*. The patient failed screening for subcutaneous implantable cardioverter-defibrillator and thus, a transvenous implantable cardioverter-defibrillator system was implanted on POD 64 for secondary prevention. Evaluation of his hands and feet revealed no signs of local infection or wet gangrene. Black skin changes and demarcation lines were clearly defined, compatible with frank mummification of his digits and toes (Figures 2 and 3). The patient was discharged on POD 77 to home health services and subsequently underwent amputation and debridement of his necrotic feet on POD 106. Amputation of his fingers is scheduled for a later date.

Discussion

Major vascular injuries from transvenous lead extraction are uncommon but carry significant mortality. When they occur, surgeons face the daunting task of performing a hasty thoracotomy and repair on a rapidly exsanguinating patient. To make matters worse, patients often present with comorbidities such as a low ejection fraction or septicemia that may exacerbate their hemodynamic instability and complicate the resuscitation process. Historically, many of these rescue attempts have been unsuccessful. In a single-center study, Brunner and colleagues[4] report a mortality rate of 31.2% for superior vena cava lacerations during lead extractions at the Cleveland Clinic, a high-volume extraction center with significant resources and comprehensive rescue protocols in place. When looking at national data in 2010, Hauser and colleagues[1] found the complication mortality rate to be

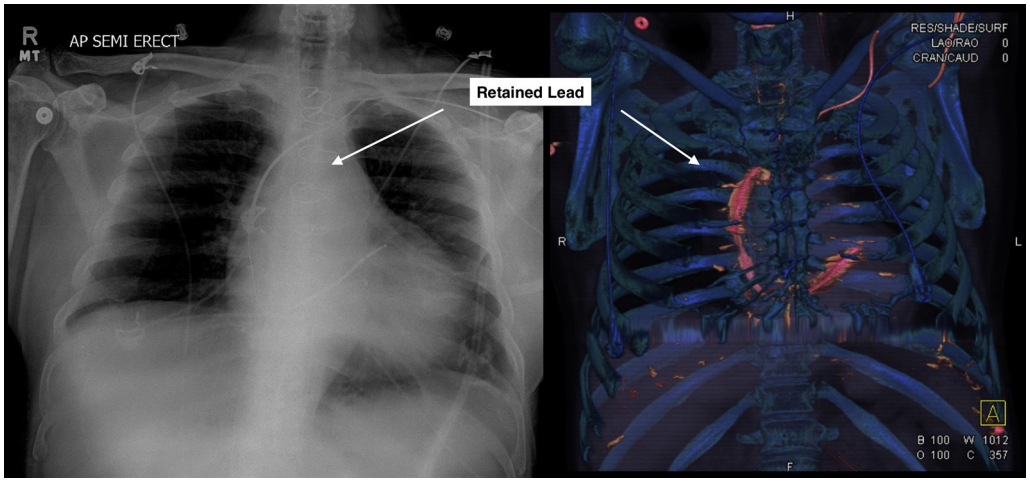

Figure 1 Chest radiograph and 3-dimensional computed tomography of abandoned lead.

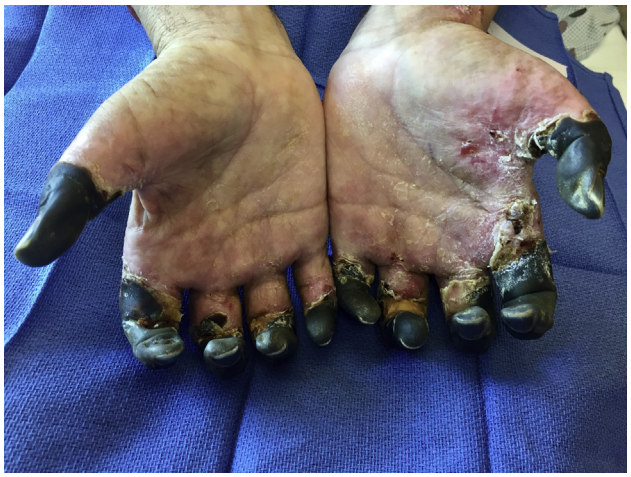

Figure 2 Symmetrical peripheral gangrene of digits of both hands.

significantly higher, with a 63.0% case fatality rate associated with superior vena cava tears during sheath-assisted lead extraction. It cannot be understated, however, that patients can be left with significant long-term morbidity and diminished quality of life even in instances of successful resuscitation.

First described by Hutchinson in 1891,[2] SPG is characterized by the sudden onset of symmetrical ischemic damage of 2 or more extremities without any signs of large vessel obstruction or vasculitis. Ischemic changes begin distally and may advance proximally to involve a whole extremity. Although the pathogenesis is not well understood, the prototypical clinical presentation of SPG is suggestive of disseminated intravascular coagulation in the presence of a low-flow state.[3] Pathologic examination of amputated specimens often reveal thrombi concentrated in the small vessels. SPG is most commonly reported in bacterial infections like pneumococcus, staphylococcus, meningococcus, and streptococcus. Additionally, SPG has been associated with a wide variety of disorders, including falciparum malaria, viral gastroenteritis, shock, trauma, surgery, myocardial infarction, congestive heart failure, paroxysmal ventricular tachycardia, vasopressor use, peripartum cardiomyopathy, paraneoplastic syndromes, malignancies, ergotism, dog bite, and so forth.[5–9] Our literature search yielded 141 results when using the terms "symmetrical peripheral gangrene" and "symmetric peripheral gangrene." To our knowledge, this is the first reported case of SPG complicating transvenous lead extraction.

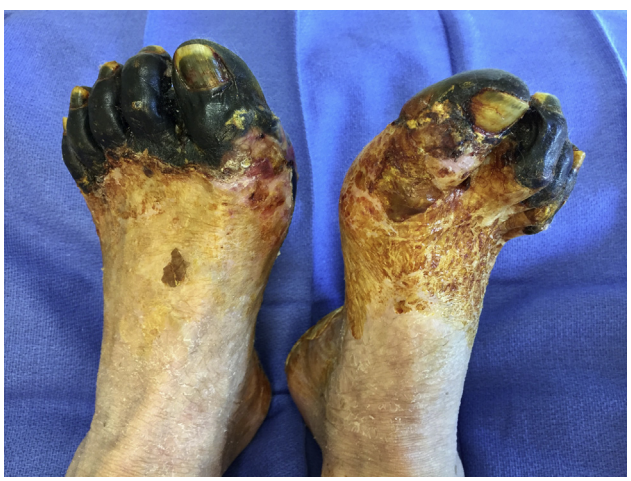

Figure 3 Symmetrical peripheral gangrene of both feet.

As this applies to our case, we suspect that sustained vasopressor use exacerbated the significant reflex vasoconstriction that took place in our patient's extremities during acute hemorrhage and cardiogenic shock.[7] The basic structural instability of small blood vessels allows intense vasoconstriction and nonthrombotic occlusion of microvasculature to occur when intraluminal hydrostatic pressure falls precipitately below a critical threshold.[10] Moreover, the necrotic changes seen in SPG can result from low cardiac output following hemodynamic collapse. In cases of septicemia, the activation of neutrophils and release of vasoactive substances may also play a contributing role in reducing peripheral blood flow and inducing ischemia. Coupled with our patient's systolic dysfunction and underlying infection, emergent resuscitative measures likely led to the cyanotic changes seen in our patient's distal extremities and the subsequent onset of SPG. Early recognition and aggressive circulatory support may prevent further progression of limb ischemia and potentially salvage injured tissues. Vasoactive drug therapy should be reduced or discontinued at the earliest possible chance and treatment of sepsis and disseminated intravascular coagulation should be promptly initiated. No treatment has been found to be completely or universally effective; prevention remains the cornerstone of management.[6] Unfortunately, once the clinical signs of SPG are recognized, amputation and debridement are often inevitable.[3]

This case underscores the important role of comprehensive rescue strategies in reducing both the morbidity and mortality of vascular tears during lead extraction. The strong consensus is that open surgical access and repair must occur within 10 minutes of the onset of injury to avoid fatal outcomes.[11] This entails the availability of surgical back-up, anesthesia support, cardiopulmonary bypass equipment, and cell saver primed on standby. If left hypoperfused, patients are susceptible to hemodynamic shock and ischemic injury, both of which carry unfavorable long-term sequelae. Emerging technology such as the endovascular occlusion balloon may hold promise in delaying hemodynamic collapse by providing effective tamponade of the superior vena cava, thereby preserving blood volume and systemic perfusion throughout resuscitation.[12] Early evidence suggests that this rescue tool can assist lead extractors in the event of catastrophic complications and improve the likelihood of survival.[13,14] Furthermore, the deployment of an endovascular cover stent has also been reported in isolated cases as a viable repair strategy for superior vena cava injuries, sparing select patients the additional trauma of open surgical repair.[15] To effectively employ these rescue strategies, lead extraction teams can better prepare to manage

these complications by planning for the possibility of hemodynamic collapse and ensuring familiarity with rescue protocols. Altogether, lead extractors can reduce both the morbidity and mortality of this catastrophic complication by ensuring the availability and rapid deployment of comprehensive rescue strategies that delay hemodynamic collapse.

Conclusion

SPG is a rare clinical syndrome that can ensue following vascular injury during transvenous lead extraction, with sinister prognostic implications in terms of loss of life or limbs.

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