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Acute Septic Shock From Routine Ureteroscopy With Laser Lithotripsy

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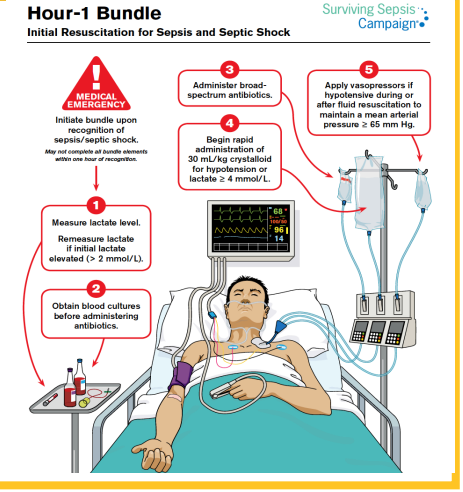
Background

Diagnosis and treatment of sepsis is an area of medicine that is continually evolving and actively studied. One concept that is crucial for successful treatment of sepsis is minimal time from diagnosis to onset of treatment. Research and clinical work done for the "Surviving Sepsis Campaign" have emphasized the importance of timing with the "Hour-1 Bundle." To achieve this clinical goal, communication among healthcare providers and team-based care is paramount for appropriately executing management and ultimately successfully resuscitating the patient. In this case-report, we present a patient who developed acute sepsis in the post-anesthesia care unit (PACU) following ureteroscopy with stent removal.

Figure



Case Presentation

Patient was a 65-year-old male with a history of CAD, CKD III, HTN, DM, HLD, and nephrolithiasis with prior ureteral stent placements presenting for ureteroscopy with lithotripsy and bilateral stent exchange. The procedure was performed under spinal anesthesia without complications and brought to the PACU to recover. Operative note mentioned that the left ureteral stent removal was difficult; the stent was adhered to underlying mucosa. Upon arrival into the PACU, the patient was following commands and spinal anesthetic was regressing appropriately. Patient exhibited shivering a few minutes after arrival with an initial temperature of 36.0 Celsius. Shivering continued and patient was given meperidine. Over the course of the next twenty minutes, patient progressively became hypotensive without symptoms of dizziness, lightheadedness, nausea, or vomiting. Mentation remained intact. Hypotension progressed and MAPs dropped below 65mmHg. Crystalloid boluses were given in addition to boluses of phenylephrine. Patient's MAPs transiently improved. Ten minutes after hemodynamic improvement; patient became tachycardic to the 120s and temperature increased to 40 C. Labs were drawn which revealed a lactate of 14. At this time, patient's presumptive diagnosis was sepsis. Given the hypotension and worsening tachycardia, patient was bolused with vasopressin, arterial line was placed, empiric antibiotics were started, and a central line was placed. Norepinephrine was made available and patient was bolused multiple times with norepinephrine and vasopressin. Patient remained communicative and mental status was intact. Patient quickly decompensated into a wide complex ventricular tachycardia requiring synchronized cardioversion. First cardioversion was unsuccessful and a second attempt was performed. During this time, preparations were made to intubate the patient. After the second attempt, the patient went into asystole, ACLS was performed, patient was intubated, and ROSC was achieved. The patient continued to exhibit arrhythmias on EKG and amiodarone infusion was started. After stabilization in the PACU, patient was transferred to the ICU. The patient improved clinically and was extubated on POD 7; however, the patient developed an acute GI bleed the following day and massive transfusion protocol was initiated; patient was re-intubated. On POD 10, the patient underwent tracheostomy and mechanical ventilation was weaned toward the end of the hospital course. Patient was discharged to LTAC on POD 17.

References

1. Dünser MW, Mayr AJ, Ulmer H, et al. The effects of vasopressin on systemic hemodynamics in catecholamine-resistant septic and postcardiotomy shock: a retrospective analysis. Anesth Analg. 2001;93(1):7-13.
2. Available at: http://www.survivingsepsis.org/SiteCollectionDocuments/Surviving-Sepsis-Campaign-Hour-1-Bundle. Accessed October 23, 2019.

Discussion

The precipitous decline in this patient's clinical status was impressive. The etiology of the patient's sepsis was likely related to the patient's ureteral stents that were placed many months ago. After discussing the case with Urology, the recommended maximum time to removal of ureteral stents is 3 months. Since the patient had these stents much longer, a biofilm of bacteria may have developed. Upon mechanical manipulation of these stents and high irrigation pressure from uteroscopy, bacteria were seeded into the venous system. The response to the acute decline from the PACU team was timely and efficient; however, there remains room for improvement. First, blood cultures were not drawn prior to antibiotic therapy. Second, norepinephrine was not the first line pressor used for initial management of hypotension. The acute boluses of pressors likely reduced splanchnic blood flow further aggravating the patient's baseline gastrointestinal ulcerations and poor mucosal integrity resulting in a massive post-operative GI bleed. Current literature in regards to the use of vasopressin in septic shock has shown a positive correlation between its use and decreased splanchnic blood flow. Overall, the patient survived septic shock due to early recognition and timely treatment.

Conclusions

This case demonstrates the importance of interdisciplinary communication and team-based medical management in order to successfully resuscitate patients from septic shock. Moreover, this case illustrates the leadership role anesthesia providers must take during the post-operative period to not only identify early signs of sepsis but also begin its treatment.