Anesthetic Management Of A Patient With Streptococcal Necrotizing Fasciitis And Toxic Shock Syndrome.

J Takahashi, C Kroll, C Soria

Citation

J Takahashi, C Kroll, C Soria. *Anesthetic Management Of A Patient With Streptococcal Necrotizing Fasciitis And Toxic Shock Syndrome.*. The Internet Journal of Anesthesiology. 2022 Volume 41 Number 1.

DOI: 10.5580/IJA.56474

Abstract

Necrotizing fasciitis due to group A streptococci (GAS) is a serious soft tissue infection that has a rate of mortality as high as 30% [1]. In even more severe cases, the concomitant presence of streptococcal toxic shock syndrome (STSS) in the setting of necrotizing fasciitis dramatically increases the rate of mortality to as high as 80% [2]. Here, we present the clinical course of a healthy individual who underwent an emergent surgical incision and drainage that was complicated by rapid cardiovascular collapse requiring cardiopulmonary resuscitation shortly after induction of general anesthesia. The patient was later diagnosed with GAS necrotizing fasciitis likely in the setting of STSS. The non-specific clinical features of the underlying disease process and non-toxic appearance of the patient lead to multiple providers underestimating the severity of illness and an overall delay in diagnosis. Anesthesiologists may find themselves managing this rare disease before a definitive diagnosis has been made. Therefore, it is important to remain vigilant to the possibility of this disease even in healthy adults and treat promptly.

BACKGROUND:

Invasive group A streptococci (GAS) infections can range from mild to severe life-threatening complications such as necrotizing fasciitis and streptococcal toxic shock syndrome (STSS). Streptococcal necrotizing fasciitis is a serious soft tissue infection that causes local tissue destruction and is associated with a rate of mortality as high as 30% [1]. In even more severe cases, the systemic host response to GAS causes a toxic shock syndrome and increases mortality to as high as 80% [2].

The following report describes the clinical course of a healthy individual who underwent general anesthesia for an emergent surgical incision and drainage that was complicated by rapid cardiovascular collapse requiring cardiopulmonary resuscitation shortly after anesthesia induction. This patient was later diagnosed with GAS necrotizing fasciitis likely in the setting of STSS.

CASE PRESENTATION:

A 22-year old female with no co-morbidities (weight 54 kg, height 162 cm) presented to the emergency department (ED) after four days of right groin pain, redness, and swelling. In the ED, her temperature was 38°C, blood pressure (BP)105/77 mmHg, heart rate (HR) 105 bpm, and oxygen saturation 100%. On physical examination, she was generally well appearing but had a notable abscess in the right groin with associated cellulitis. An incision and drainage (I&D) was performed with copious purulent fluid expressed. Subsequently, she became hypotensive (BP 78/33 mmHg) and tachycardic (HR 140 bpm) despite multiple fluid boluses. Vasopressor support was then initiated with a norepinephrine infusion and antibiotics were broadened to include vancomycin, piperacillin-tazobactam, and clindamycin. General Surgery was consulted to evaluate for any undrained fluid collection and an emergent surgical debridement was scheduled under general anesthesia.

On arrival to the operating room (OR), the patient's skin appeared flushed but she was otherwise calm and conversant. She had a norepinephrine infusion running at 30 mcg/min and her HR was 110 bpm, BP 79/50 mmHg, and SpO2 99%. After a discussion with the general surgery team, the plan was to proceed with a rapid sequence intubation followed by the placement of a central line. The norepinephrine infusion was continued and a fluid bolus of under one liter was administered pre-induction. Anesthesia was then induced with 100 mg of propofol and 100 mg of rocuronium.

A few minutes after intubation, vital signs rapidly

deteriorated and the patient became visibly cyanotic. At this time, BP was 49/24 mmHg and HR 90 bpm. The patient quickly converted from sinus tachycardia to ventricular fibrillation with no palpable pulse. Chest compressions were immediately started and 1 mg of epinephrine was given. At the two minutes pulse check, the patient remained pulseless and in asystole. Chest compressions were resumed for 1 minute before return of spontaneous circulation (ROSC) was achieved. A femoral triple lumen central line and brachial arterial line was then placed. Initial arterial blood gas (ABG) analysis revealed both respiratory and metabolic acidosis (pH = 7.25, PaCO2 = 46 mmHg, PaO2= 66, BE = -6.8 mmol/L).

Intraoperatively, the patient continued to require escalating vasopressors to maintain a goal mean arterial blood pressure (MAP) above 65 mmHg. Only after multiple fluid boluses, an epinephrine infusion at 0.1 mcg/kg/min, a phenylephrine infusion at 100 mcg/min, and a norepinephrine infusion at 50 mcg/min were we able to maintain a MAP above 65 mmHg. Additionally, 125 mg of IV methylprednisolone was administered. A urine toxicology sample was also sent at this time to further investigate if any recreational drug use could help explain the severe degree of shock.

The surgical incision and drainage was notable for purulent drainage and necrotic soft tissue requiring a debridement area measuring to be approximately 20 x 30 x 2 cm. The phenylephrine infusion was weaned off, the epinephrine infusion was weaned down to 0.06 mcg/kg/min, and the norepinephrine infusion was continued at 50 mcg/min.

Postoperatively, the patient remained intubated and was transported to the surgical intensive care unit (SICU). In the SICU, the patient continued to have high vasopressor requirements (norepinephrine at 30 mcg/min and vasopressin at 0.03 units/min) and was subsequently taken back to the OR the same day for repeat debridement. Initial exam showed no new purulent drainage or dead tissue. Given the lack of obvious signs of continued infection in the wound, the obstetrics and gynecology team was consulted to help assess for any additional source of infection. An intraoperative pelvic ultrasound was performed with no findings to suggest sepsis of a gynecologic nature. Following multiple OR takebacks for debridement, the patient was able to be weaned off of pressors and successfully extubated on postoperative day three. At this point in time, the patient's wound cultures had come back positive for group A streptococci. The patient was taken to the OR a total of 15 times for serial debridement, wound washout, and wound vacuum changes during her hospitalization. She remained hospitalized for a total of 43 days before being discharged home.

DISCUSSION:

Necrotizing fasciitis is a severe soft tissue infection that can rapidly progress and lead to fatal outcomes even in immunocompetent hosts. It typically presents after a minor skin lesion, a traumatic injury, or a nonpenetrating injury and causes rapid tissue destruction [3]. The infection usually spreads along the muscle fascia due to its relatively poor blood supply, leaving the overlying tissue unaffected and can delay diagnosis [3-4].

The most serious complication of streptococcal necrotizing fasciitis is streptococcal toxic shock syndrome (STSS) which is characterized by the sudden onset of fever, rash, hypotension, and end-organ failure. The exact mechanism is not fully understood but is thought to be the result of superantigen proteins that bypass the normal immune pathway and are able to generate a massive secretion of pro-inflammatory cytokines. The exotoxins that these strains produce cause microvascular damage or thrombosis, leading to tissue ischemia with subsequent necrosis [5]. The diagnostic criteria of STSS includes isolation of GAS from a sterile site, the presence of hypotension and two or more signs of renal impairment, coagulopathy, liver involvement, acute respiratory distress syndrome, generalized erythematous macular rash, and soft tissue necrosis [5-6]. In this case, GAS was isolated from tissue cultures on post operative day three. Our patient presented in severe shock (BP 70/30 mmHg) and was found to have soft tissue necrosis. Liver impairment was present (AST 2,974) but was found after cardiopulmonary resuscitation. In retrospect, our patient had GAS necrotizing fasciitis likely in the setting of STSS. However, a definitive diagnosis was not possible at the time of treatment initiation. In addition, the ED physicians and surgery team did not suspect a GAS infection.

Early recognition of necrotizing fasciitis is critical for successful management, but can be difficult to distinguish from common soft tissue infections. As seen in our case, the non-specific clinical features of the underlying disease process and non-toxic appearance of our patient lead to multiple providers underestimating the severity of illness. By the time our patient presented to the operating room for emergent surgical debridement, she was severely under-

resuscitated despite our efforts to provide a fluid bolus prior to induction of general anesthesia. It is regrettable that we did not have an arterial line or central line prior to induction of anesthesia to guide fluid management and vasopressor treatment.

CONCLUSION:

One of the most serious complications of streptococcal necrotizing fasciitis is streptococcal toxic shock syndrome. As demonstrated in our case, the non-specific clinical features of this disease and the non-toxic appearance of the patient lead to a delay in diagnosis. Anesthesiologists may find themselves managing this rare disease before a definitive diagnosis has been made. Therefore, it is important to remain vigilant to the possibility of this disease even in healthy adults and treat promptly.

References

1. Stevens DL, Tanner MH, Winship J. Severe group A streptococcal infections associated with a toxic shock-like syndrome and scarlet fever toxin A. N Engl J Med. 1989;321:1-7.
2. Davies HD, McGeer A, Schwartz B. Invasive group A streptococcal infections in Ontario, Canada. N Engl J Med. 1996;335:547-554.
3. Stevens DL, Bryant AE. Necrotizing Soft-Tissue Infections. N Engl J Med. 2017; 377: 2253–2265.
4. Stevens DL, Bryant AE, Goldstein EJ. Necrotizing Soft Tissue Infections. Infect Dis Clin North Am. 2021;35:135-155.
5. Schmitz M, Roux X, Huttner B. Streptococcal toxic shock syndrome in the intensive care unit. Ann Intensive Care. 2018;8:88.
6. Breiman RF, Davis JP, Facklam RR. Defining the Group A Streptococcal Toxic Shock Syndrome: Rationale and Consensus Definition. JAMA. 1993;269:390–391.

Author Information

Joelle Takahashi
Department of Anesthesiology, University of California San Diego
San Diego, California

Caitlin Kroll
Department of Anesthesiology, University of California San Diego
San Diego, California

Claire Soria
Department of Anesthesiology, University of California San Diego
San Diego, California