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Critical Care Medicine

Marked serum procalcitonin level in response to isolated anaphylactic shock

Abstract

The objective of this study was to present a case report that highlights the limitation of serum procalcitonin levels greater than 10 ng/mL as being almost exclusively secondary to septic shock. Data source was a medical intensive care unit patient at the University of Louisville. Anaphylactic shock may cause elevations of serum procalcitonin to levels greater than 10 ng/mL.

Case report

A 52-year-old woman with hypertension and seborrheic dermatitis presented to the emergency department with nausea, vomiting, hives, dyspnea, and a feeling of pharyngeal fullness that began suddenly 30 minutes after taking a double-strength trimethoprim-sulfamethoxazole tablet. She was in a good state of health before onset of symptoms and was recently prescribed a second course of trimethoprim-sulfamethoxazole by her primary care physician for possible forehead folliculitis. She stated that she last finished a short course of this antibiotic more than 2 weeks ago; however, her skin lesions did not improve.

On presentation, the patient had a temperature of 102.2°F, heart rate of 120 beats per minute, blood pressure of 87/55 mm Hg, oxygen saturation of 95% on room air, and nonlabored breathing with a respiratory rate of 20 breaths per minute. She displayed urticarial changes on her trunk and extremities and appeared to be in slight distress; however, she did not exhibit stridor, drooling, oropharyngeal swelling, or other signs of airway compromise. A slight wheeze was noted on end expiration, and breath sounds were clearly auscultated throughout the lungs bilaterally. Her abdomen was soft and nontender. Her extremities were warm with adequate peripheral pulses. We did not detect any signs of cellulitis; however, her forehead did reveal findings consistent with extensive seborrheic dermatitis, which involved her entire scalp and the areas behind her ears.

A portable chest radiograph was obtained and was unremarkable, and an ECG revealed sinus tachycardia. Pertinent laboratory findings included a white blood cell count of 9000/mm³ with a left shift of 72% granulocytes and 21% bands. An erythrocyte sedimentation rate was obtained, which was within normal limits (25 mm per hour). Her urinalysis revealed only trace bacteria without white blood cells, leukocyte esterase, blood, ketones, or nitrites. Her chemistry revealed an anion gap acidosis of 18 with an elevated lactic acid level of 6.2 mmol/L. An arterial blood gas obtained on 32% of Fio₂ showed a pH of 7.44, PaCO₂ of 17 mm Hg, and a PaO₂ of 123 mm Hg. Her serum tryptase level was elevated at 33 ng/mL as well

as her C-reactive protein at 0.83 mg/dL (reference range 0.00–0.49 mg/dL). A serum procalcitonin (PCT) was also obtained and was markedly elevated at 29 ng/mL (our laboratory reference states levels <0.5 makes sepsis unlikely, whereas levels >10 are almost exclusively secondary to sepsis/septic shock). Blood and sputum cultures were procured as well as a serum cortisol; however, the patient had already been given methylprednisolone, and the resultant cortisol level was elevated at 46 µg/dL. In addition to initiating resuscitation for possible anaphylaxis we administered 1 dose of vancomycin and piperacillin/tazobactam for probable sepsis based on systemic inflammatory response criteria, PCT elevation, bacteremia, and possible cellulitis.

The patient was admitted to the medical intensive care unit (ICU) with the diagnosis of shock secondary to anaphylaxis. Initial therapy included crystalloid resuscitation, epinephrine infusion at 2 µg/min, methylprednisolone, albuterol nebulizers, famotidine, and diphenhydramine. Broad spectrum antibiotics were discontinued. She received 7 L of normal saline, and the epinephrine infusion was titrated off within several hours. The patient did have several episodes of fever during the first 12 hours of admission with temperatures around 101°F. Her lactic acid level peaked 6 hours into admission at 8.7 mmol/L and rapidly trended downward while her anion gap closed. A transthoracic echocardiogram obtained after resuscitation revealed an ejection fraction of 60% with diastolic dysfunction and a right atrial pressure of 10 to 15 mm Hg. She remained coherent throughout her hospitalization and did not require ventilatory support. The blood, urine, and sputum cultures were negative. Her symptoms resolved without recrudescence while on prednisone, diphenhydramine, and famotidine. The patient was discharged home after 72 hours with a topical steroid cream for dermatitis, an epinephrine pen, and a follow-up appointment with an allergist.

Although this patient had a classic presentation of anaphylactic shock, the concerning PCT elevation (29 ng/mL) presented us with a clinical dilemma. Serum procalcitonin levels greater than 10 ng/mL are reported to be more than 90% sensitive and 60% specific for septic shock and are "almost exclusively" elevated to this degree from sepsis [1–4]. However, a review of the literature provides us with a different view for those patients bound for the ICU. A meta-analysis by Tang et al [5] found that sensitivity and specificity for elevated PCT levels in the critically ill is only 71%. They also concluded that PCT levels cannot reliably differentiate systemic inflammatory response syndrome (SIRS) from sepsis. This severely limits the use of this laboratory test in triaging patients with undifferentiated early shock or SIRS. In the setting of critical illness and shock, PCT may be better interpreted as a marker that is reflective of overall inflammation. This is

supported by Becker et al [6], who performed an exhaustive literature search of PCT values in the clinical setting. They state that multiple organ dysfunction syndrome, trauma, severe pancreatitis, rhabdomyolysis, hypovolemic and cardiogenic shock, and burns can dramatically elevate PCT and that a more sensitive assay is needed. Their findings also reveal that peak PCT levels lack the specificity to differentiate infection from noninfection in the critically ill. However, they suggest that the rate of PCT decline may aid in this differentiation. A rapid decline within 24 hours is more supportive of a noninfectious etiology of SIRS or shock. These views are also supported by a multicenter observational study by Reynolds et al [7]. Thus, PCT may be an appropriate marker to follow on ICU admissions for shock differentiation and antibiotic de-escalation.

In summary, when physicians encounter elevated PCT levels, they often feel obligated to administer broad spectrum antibiotics and order a battery of radiologic and laboratory investigations to "work up" this laboratory finding. Based on our literature search, caution should be used when interpreting these results in the setting of shock. This case is important as it is only the second in the literature to document an association between anaphylactic shock and PCT [8]. Despite the elevation of this serum marker, we elected to treat this patient solely for anaphylactic shock. Although we did not repeat a PCT level, a rapid rate of decline may have further supported our clinical decision to stop antibiotics. This case clearly highlights the limitation of PCT elevations of greater than 10 ng/mL as an "exclusive" marker for sepsis in critically ill patients. Further understanding regarding the mechanism of PCT elevation as it relates to anaphylactic shock is needed as well as more accurate assays.

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