Markedly Elevated Procalcitonin after Cardiac Arrest: A Case Report

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Abstract

Background: Procalcitonin (PCT) is an amino acid prohormone of calcitonin that is released by various tissues in response to infection, systemic inflammation or sepsis[1]. High levels of PCT have been described in circumstances other than sepsis or infection, including major surgery, trauma, acute coronary syndromes, cardiogenic shock and cardiac arrest[2,3].

Objective: To describe a patient with severely elevated procalcitonin levels after cardiac arrest and multiple external defibrillations as another diagnosis other than infection leading to its elevation.

Case Report

A 70-year-old Caucasian male with a past medical history of hypertension and alcohol abuse presented to a local community hospital emergency department (ED) after a syncopal episode earlier that evening. He admitted to heavy drinking daily for several months. Imaging of the head and cervical spine were negative for any trauma. In the ED he became unresponsive and the electrocardiogram showed polymorphic ventricular tachycardia (PMVT). He was defibrillated with return of normal sinus rhythm but had recurrent episodes of ventricular fibrillation (VF) and received 10 consecutive external defibrillations. He was transferred to our hospital and on arrival his temperature was 101.0°F and the remainder of his vital signs were within normal limits. Physical examination was unremarkable except for a right wrist splint and edema of the left ankle. Chest X-ray demonstrated left lower lobar atelectasis. He was hypokalemic (potassium of 2.2 mMol/L) and his cardiac enzymes were mildly elevated, Troponin T was 0.05 ng/mL, CK 210 U/L and CKMB 2.4 ng/mL. His lactate was elevated at 3.0 mMol/L and he had a markedly elevated procalcitonin at 45.98 ug/L. His blood ethanol level was < 0.010 g/dL. Blood and urine cultures demon-
Discussion

Few studies have been conducted on the role of PCT after cardiac arrest and recurrent external defibrillations. Some data suggests that the course of PCT concentrations post-cardiac arrest may reflect the duration and impact of organ and tissue ischemia and thus provide prognostic information\[2,3\]. Although sepsis was a concern from the date of presentation, the degree of PCT elevation in this patient may suggest another etiology. PCT levels reported in the literature of patients with urinary tract infections report maximum PCT levels far lower than those observed in our patient, for example Van Nieuwkoop et al., reported a maximum of 9.07 ug/L\[4\]. In our patient, the PCT was observed to dramatically increase after cardiac arrest and recurrent external defibrillations and thus seemed correlative. The role of procalcitonin in clinical practice is evolving and its applications beyond the diagnosis of sepsis deserve further investigation.

References