

Case report

Electrocardiographic alterations and raised procalcitonin levels during anaphylactic shock

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SUMMARY

Anaphylaxis represents a systemic disease occurring after the exposure to an allergen. Drugs for the treatment of anaphylactic reactions (ie, corticosteroids and adrenalin) could induce autonomic alterations, such as tachycardia, hyperthermia, tachypnoea and leucocytosis. We describe the case of a 52-year-old woman presenting with a severe allergic reaction after the ingestion of amoxicillin-clavulanate. The occurrence of ECG alterations, laboratory abnormalities and procalcitonin (PCT) elevation will be discussed with particular emphasis on the possible misleading role of PCT during anaphylactic shock.

BACKGROUND

Anaphylactic shock represents a systemic disease involving several organs and apparatus. Despite the pathophysiology of allergy, even the drugs used for the treatment of anaphylaxis (ie, epinephrine and corticosteroids) could induce autonomic alterations (eg, tachycardia, hyperthermia, tachypnoea and leucocytosis), similarly to severe infections.

In the last years, procalcitonin (PCT) use in clinical practice to rule-out severe infections has increased. We would like to underline the potentially misleading role of PCT during anaphylactic shock.

CASE PRESENTATION

A 52-year-old woman, without medical history nor known allergies, started treatment with amoxicillin-clavulanate (875/125 mg) because of the persistence, for about 3 days, of sore throat and cough. She was taking this antibiotic for the first time. Few minutes after the first pill, the patient experienced dyspnoea, wheezing, skin rash, dizziness and loss of consciousness. Her husband called emergency medical service who made diagnosis of anaphylactic shock and administered chlorphenamine 10 mg, hydrocortisone 1000 mg and epinephrine 2 mg over 100 mL saline infusion and transferred the patient to emergency department (ED). At ED the patient was severely nervous and dyspnoeic. Blood pressure was 90/50 mm Hg, heart rate (HR) 125 bpm, oxygen saturation 90% in 2 L/min nasal cannula, respiratory rate 28 breathes/min.

INVESTIGATIONS

Lab tests showed leucocytosis (figure 1). Chest X-ray showed a thickening of broncho-vascular bundles in the left lung, behind the heart. Methylprednisolone 1000 mg intravenous and high

flow oxygen were administered. The patient was admitted to internal medicine unit with diagnosis of anaphylactic shock. At admission the patient showed hypotension (blood pressure 70/40 mm Hg, HR 102 bpm), tachypnoea (Respiratory Rate 24/min), normal oxygenation of 97% in FiO₂ 0.5. An ECG showed ST-segment depression in inferior-lateral leads. Troponin was high, in absence of chest pain.

TREATMENT

Treatment with intravenous fluids (ringer lactate 2000 mL/day), hydrocortisone 100 mg three times per day and clarithromycin 500 mg two times per day was started.

OUTCOME AND FOLLOW-UP

During the next days, patient's general conditions progressively improved with normalisation of blood pressure, HR and oxygen saturation about 30 hours after admission. Lab tests showed the persistence of leucocytosis (figure 1). Tryptase levels were not assessed. On the second day, a significant elevation of acute phase indices including PCT was found (figure 1). Blood cultures were collected and resulted negative. An echocardiogram was normal. The patient was discharged on fifth day in good general conditions, after normalisation of ECG and lab tests.

This represents an anecdotal case of anaphylactic shock with ECG alterations and raised PCT levels. Some aspects need to be highlighted.

First, ECG alterations and troponin elevation in the setting of anaphylaxis should be carefully evaluated: possible differential diagnoses are represented by Kounis syndrome¹—also called allergic angina—and ATAK complex (adrenaline, takotsubo, anaphylaxis, Kounis syndrome).² However, in the present case, given the absence of chest pain and of echocardiographic abnormalities, ECG modifications have been attributed to the effect of high dose intravenous epinephrine administration.³ Although in the absence of risk factors for coronary artery disease and chest pain, these alterations led to several troponin determinations until normalisation, and to echocardiography (which resulted normal) (figure 1).

Another—and probably the most important—reflection is needed about PCT elevation. First of all, given the parallel trend of PCT with white cell count and C-reactive protein, it is conceivable that PCT levels were higher on the day of admission.



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Findings that shed new light on the possible pathogenesis of a disease or an adverse effect

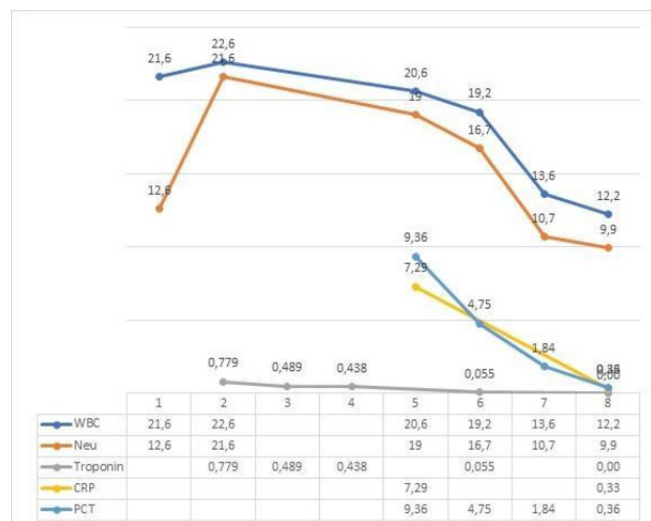


Figure 1 Line graph and table showing the trend of laboratory tests at different times (1: 1 May, 01,11; 2: 1 May, 09,44; 3: 1 May, 14,20; 4: 1 May, 19,30; 5: 2 May; 6: 3 May; 7: 4 May; 8: 5 May). CRP, C-reactive protein (mg/dL; normal value <0.30); neu, neutrophils ($N \times 10^3/\mu L$; normal values 2.2–8.6); PCT, procalcitonin (ug/L; normal values: low-risk <0.5; intermediate risk 0.5–2.0; high-risk >2.0); troponin (ng/mL; normal values <0.045); WBC, white blood cell count ($N \times 10^3/\mu L$; normal values 4.3–10.8).

With this regard, PCT is generally considered a biomarker of bacterial infection used for differentiating infectious from non-infectious clinical syndromes.⁴ In this context, our patient had sore throat, cough, leucocytosis, hypotension and a chest X-ray showing a possible pneumonia. All these elements, associated with significant PCT elevation, could be consistent with a suspicion of sepsis/septic shock. Moreover, in case of sepsis/septic shock, clarithromycin alone does not represent an appropriate antibiotic treatment. With this regard, patient did not show fever nor organ dysfunction (ie, altered mental status, renal failure, coagulopathy, etc) and blood cultures were negative. Although our patient was showing an improvement of haemodynamic and general status, the evidence of high PCT levels produced several effects: first, the test was repeated to obtain a trend over time; second, chest X-ray was repeated; third, the appropriateness of antibiotic treatment was discussed; finally, the patient was discharged only after PCT normalisation. It should be noted that PCT can be elevated in several conditions such as trauma, pancreatitis, autoimmune diseases and shock, without infection.⁵ More specifically, there are three reports of elevated PCT in patients with anaphylactic shock.^{6–8} Physicians should be aware that high PCT levels lack in specificity,^{5,7} and could lead to misdiagnosis of sepsis in patients with other causes of shock (ie, anaphylaxis). On the contrary, the negative predictive

value of low PCT levels (<0.5 ng/mL) is very high (95%–98%) in excluding bacteraemia.⁹

In conclusion PCT should always be interpreted evaluating its trend over time instead of a single measurement, considering the overall assessment of each patient.⁴

Learning points

- ▶ Procalcitonin (PCT) represents a biomarker to identify severe infections and sepsis.
- ▶ Anaphylactic shock, as well shock in general, can induce a raising of PCT levels, probably due to haemodynamic changes or to intestinal bacterial translocation.
- ▶ High PCT levels in the context of anaphylactic shock could be potentially misleading, orienting towards infection.
- ▶ PCT levels must be interpreted in the clinical context of each patient

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