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Case Report

A case of anaphylactic shock complicated by high C-reactive protein and procalcitonin levels

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Introduction

Anaphylaxis is an acute and potentially fatal allergic reaction with multi organ involvement where prompt recognition and management are lifesaving [1].

Procalcitonin (PCT) is used as a biomarker to aid in the diagnosis of bacterial infection or sepsis and is considered superior to C-reactive protein (CRP), which is an acute inflammatory protein which rises in response to infection [2]. PCT levels are elevated in other conditions as well, including multiple organ dysfunction syndrome, trauma, severe pancreatitis, rhabdomyolysis, hypovolemic and cardiogenic shock and burns with cases of anaphylaxis also being reported [4].

Here, we report a patient presenting with anaphylactic shock following co-amoxiclav who was found to have high CRP and PCT levels which led to a diagnostic dilemma of septic shock vs. anaphylaxis.

Case presentation

A 56-year-old female with a history of allergy to multiple food items was admitted with facial swelling, generalized pruritus and urticarial rash since the morning of the day of admission. This was following the first dose of oral co-amoxiclav which she had been prescribed for abdominal pain.

On examination, her respiratory rate was 26 breaths/ minute with saturation of 93% on air. The pulse rate was 120bpm. The blood pressure was 80/60mmHg. Auscultation revealed bilateral rhonchi in lung fields. Intra-muscular 1:1000 adrenaline 0.5mg was given immediately. She required a second dose of adrenaline to which she responded.

Her full blood count revealed a very high white blood cell (WBC) count of $35\times10^{9/}$/L with neutrophils of 80%. The CRP was 285mg/dl. PCT was 10.46ng/ml.

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A detailed history and examination did not reveal a focus of infection. Septic screening was performed. Chest X-ray and ultrasound scan of the abdomen were normal. Pending the blood and the urine cultures, she was given a dose of oral clindamycin 300mg. But since she developed generalized itching again with clindamycin it was withheld. The cultures were negative.

The patient improved clinically with steroids and antihistamines without antibiotics and was kept under observation. The FBC, CRP and PCT were repeated on the 3rd day. The WBC count was 9×10^9/L. CRP had reduced to 78mg/dl and the PCT was 2.3ng/ml. Hence the abnormal increase in WBC, CRP and PCT was attributed to anaphylactic shock.

Discussion

Many hypotheses have been proposed to explain the release of PCT in anaphylaxis. A promising theory is that the cytokine surge seen in anaphylactic shock with pro-inflammatory cytokines including tumour necrosis factor-α, interleukin-6 and interleukin-1 mediate PCT elevation [3].

An increased WBC count with neutrophil predominance has also been reported in anaphylaxis and is attributed to the result of stress and elevated blood catecholamine levels [5].

In our patient, even though the initial diagnosis was anaphylactic shock, the presence of high CRP, PCT and neutrophils triggered the uncertainty of a concurrent underlying sepsis. But the marked clinical improvement on resolution of the anaphylaxis and reduction of inflammatory markers without antibiotics support the association of elevated PCT, CRP and neutrophils with anaphylactic shock.

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