



CASE REPORT

A Surprising Cause of Shock

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Introduction

A mayor challenge in emergency medicine is identifying life threatening conditions and simultaneously starting an adequate therapy. Therefore physicians often have to quickly commit themselves to the most likely diagnosis to avoid any delay of treatment. Nevertheless clinicians have to stay alert, repeatedly reassess differential diagnoses and change the initial treatment when needed.

Case Description

A 59-year-old man was admitted to our emergency department by ambulance in state of shock. He presented with reduced consciousness, hypotension of 42/36 mmHg, a heart frequency of 77 bpm and hypothermia of 34.3 °C. Relevant pre-existing diagnoses were diabetes mellitus type II, protein S deficiency and seronegative oligoarthritis.

Blood tests showed Leukocytosis (22,3 G/l) with left shift, mild CRP-elevation, impaired renal function (creatinine 494 µmol/l, serum urea 29 mmol/l, potassium 7.9 mmol/l) and elevated high sensitive troponin t (122 ng/l). Blood clotting parameters were impaired with anti-factor-Xa activity larger than 2.0 U/ml. Elevated liver enzymes pointed at a hepatic involvement. Blood gas analyses showed a massive lactic acidosis of 17 mmol/l, pH 7.05, base excess -26.4 mmol/l and partial respiratory compensation (pCO₂ 1.9 kPa).

Initially, the patient was diagnosed with sepsis of unknown origin, treated with piperacilline/tazobactam and transferred to the ICU, where he received i.v.-fluids, nor adrenaline and dobutamine. We noticed

a conspicuously poor response of his blood pressure to these treatments. He later was intubated because of respiratory failure. We started continuous venovenous haemofiltration because of a severe accumulation of potassium and serum urea caused by a persisting glomerular filtration rate of less than 20 ml/min.

We further diagnosed upper GI-bleeding, which stopped spontaneously, and a non-ST-Segment Elevation myocardial Infarction, which was caused by severe hypotension.

After further diagnostic procedures including laboratory analyses, X-ray and ultrasonography of the abdomen, the initial diagnosis could not be confirmed and antibiotic treatment was stopped consecutively. The supportive therapy was continued.

The patient regained sufficient diuresis, was extubated on the 5th and admitted to the regular medical ward on the 6th day of hospitalization. An echocardiography showed an impaired biventricular function with a left ventricular ejection fraction of 30 to 35%.

When the patient's family delivered his regular medication, we noticed one completely empty package of enalapril among his full packages of drugs. We analyzed a blood sample for his enalapril serum level, which was identified as more than 20 times above regular serum levels.

Later on, the patient stated that he tried to commit suicide with enalapril because of socioeconomic problems. He was referred to our psychiatrist after leaving the ICU and admitted to a rehabilitation clinic.

Conclusions

Enalapril is an Angiotensin Converting Enzyme In-

hibitor (ACE Inhibitor) used in the treatment of hypertension, congestive heart failure and left ventricular dysfunction as well as diabetic nephropathy. Main side effects include hypotension and myocardial infarction, renal failure and liver failure. Patients with pre-existing renal failure should be treated with lower doses and/or less frequently [1]. There is very poor data on how much the blood pressure is reduced in a dose dependent manner [2]. Maximum blood pressure lowering occurs approximately 5 hours after ingestion while there seems to be no difference between different ACE Inhibitors [3]. Interestingly, ACE Inhibitor induced hypotension does not provoke reflex tachycardia [1].

In this particular case, the patient's overdosing of enalapril lead to severe hypotension with subsequent Lactic acidosis, myocardial infarction and to renal as well as liver failure. This caused further drug accumulation and aggravation of the situation. The consecutive metformin accumulation increased Lactic acidosis and xarelto accumulation lead to an upper GI-bleeding.

There are reports of patients with ACE Inhibitor

overdoses who were treated successfully for hypotension and anuria with intravenous infusion of angiotensin II, although a rapid identification of the overdose is necessary [4,5]. Otherwise, like in our patient, a supportive treatment has to be performed until plasma concentration is reduced to harmless levels.

References

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