Poster Presentations - Day 3, 18th November 2018

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Severe lactic acidosis and acute toxic myopathy as complications of ethanol intoxication and malnutrition

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Objective: To describe a case of severe lactic acidosis, electrolyte imbalance, seizures, and toxic myopathy caused by ethanol ingestion with malnutrition.

Case report: A 54-year-old female with a history of arterial hypertension, arrhythmia and, insomnia was admitted to the Emergency Department due to altered consciousness and the episode of seizures. The patient ingested strong alcoholic drinks for 4 days and vomited.

At admission, the patient's blood pressure was 140/80 mmHg, heart rate 120 bpm and oxygen saturation 98 %. On electrocardiogram (ECG), atrial fibrillation was registered. The patient was malnourished, confused, agitated and the episode of generalized tonic clonic seizures repeated on admission. Benzodiazepines were administrated as first line treatment. The patient was intubated and mechanically ventilated on the Intensive Care Unit. Cranial computer tomography detected cerebral edema. Chest and pelvis X-ray examinations were without pathological findings. Abdominal ultrasound examination revealed only liver cysts.

Blood tests on admission revealed thrombocytosis (443x10⁹/L), hyponatremia (sodium 114 mmol/L), hypokalemia (potassium 2.9 mmol/L), hypochloremia (chloride 79 mmol/L), hyperglycemia (glucose 9 mmol/L), lactacidemia (lactate 14.9 mmol/L), blood urea nitrogen 0.9 mmol/L, creatinine 52 μmol/L, ALT 0.66 ukat/L, GMT 1.88 μkat/L, AST 0.73 μkat/L, total protein 59.7 g/L, albumin 34 g/L, osmolality 272 mmol/ kg. Arterial blood gas analysis demonstrated pH 6.99, pO₂ 19.8 kPa, pCO₂ 4.57 kPa, HCO₃ 8.1 mmol/L, and base deficit 22.4 mmol/L. The serum concentrations of methanol, formic acid, ethylene glycol and its metabolites were under the level of detection. Serum ethanol concentration was 0.96 per mille. Diabetes mellitus was excluded by repeated measurements.

The patient was administered intravenous sodium bicarbonate (4.2% - 200 mL), intravenous rehydration, electrolytes, nutrition supplementation, thiamine, propafenone and PPI. On day 2 of hospitalization, she spontaneously breathed and was extubated. On day 3 of hospitalization, the alcohol withdrawal syndrome and delirium tremens developed and was treated with benzodiazepines and haloperidol. On day 6 of hospitalization, acute toxic myopathy with pain and weakness of muscles of lower limbs was developed. Elevated muscle enzymes in blood serum revealed rhabdomyolysis (myoglobin 359 μ g/L; creatine kinase 136 μ kat/L; creatinine 61 μ mol/L). On the electromyogram no myogenic lesions were registered and intravenous rehydration therapy continued. In following days the patient's condition stabilized. The patient was discharged on day 12 without neurological symptoms, normal ECG and blood tests.

Conclusion: We present the case of ethanol intoxication and malnutrition with life-threatening complications required complex therapeutic approach. It underlines the importance of supportive therapy.