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A patient with severe lactic acidosis and rapidly evolving multiple organ failure: a case of shoshin beri-beri

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Sir: A 50-year-old manle presented to the emergency department complaining of progressive dyspnea and muscular weakness both lasting for several days. At admission his temperature was 37.5°C, blood pressure 90/50 mmHg, heart rate 135/min, and respiratory rate 25–30/min. The laboratory investigation was inconclusive except for pronounced lactic acidosis with pH 7.08, pCO_2 24 mmHg, lactate 186.5 mg/dl (normal 5.7–22.0), base excess –-20 mmol/l and anion gap of 36 mmol/l. The hemostatic parameters, and liver and kidney function tests were normal. Electrocardiographyhe ECG showed no features suggestive of myocardial ischemia. A diagnostic computed tomography did not revealed no significant causative pathology except for small bilateral pleural effusions. Since the patient hemodynamic status was rapidly deteriorating, he was transferred to the intensive care unit. Upon arrival, the patient became even more unstable. A pulmonary artery catheter was inserted which showed cardiac index of 4.8 l /min^{-1} /m^{-2}, pulmonary arterial occlusion pressure 22 mmHg, and systemic vascular resistance index 950 dynes. sec.$^{-1}$ m^{-2}/ cm$^{-5,}$ mixed venous oxygen saturation of 78%, and central venous pressure of 14 mmHg. The patient was intubated and mechanically ventilated. Vasopressor requirements increased dramatically, reaching norepinephrine doses of more than 200 µg/min. Even after addition of hydrocotisone in stress dose (200 mg/day) and vasopressin in the highest approved concentration (0,04 IUE/kg per /hour), the patient's hemodynamic status failed to improve. Bedside echocardiography demonstrated neither regional wall motion abnormalities nor valvular pathology. In the meantime lactate rose further, reaching 204 mg/dl. Repeated laboratory investigation showed normal acute-phase parameters; repeated blood cultures remained sterile. In the following hours the urine output declined, and the patient developed acute renal failure requiring continuous renal replacement therapy. At this point we started searching for a more specific cause for the lactic acidosis combined with hyperdynamic heart failure. The patient's partner reported long-standing alcohol abuse escalating in the last months (approaching one bottle of vodka daily) in conjunction with progressive muscular weakness over recentthe past weeks. The medical history of long-standing alcohol abuse in conjunction with myopathy/neuropathy along with the present hemodynamic profile [1] was highly suggestive of thiamine deficiency, or beri-beri in its fulminant form, shoshin. After a single dose of intravenous thiamine (100 mg) the patient's hemodynamics improved dramatically within minutes, and we were able to rapidly reduce and finally stop vasopressors in the following hours as the hemodynamic indices returned to normal. Serum lactate returned to normal values within 15 h. We continued the high-dose thiamine substitution (100 mg twice daily) for 2 weeks. In the next days the patient stabilized completely, but unfortunately developed ventilator-associated pneumonia in the weaning phase, which was further complicated by pronounced ICU delirium and critically ill polyneuropathy. After a total of 67 days the patient was discharged from hospital in excellent general condition.

Shoshin Beri-Beri, appropriately designated as "a rapidly curable hemodynamic disaster" [2], is an uncommonly encountered clinical entity, which, if not timely recognized and promptly treated, can result in rapid hemodynamic collapse and death. When lactic acidosis [3] in conjunction with hyperdynamic circulation is diagnosed and no other causeapparent etiology is probable, the diagnosis of Beri-Beri must be considered, and thiamine should be administered.

References

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