

Exaggeration of hypoxic lung injury in a patient with glucose-6-phosphate dehydrogenase deficiency

Sir,

A 56-year-old female with glucose-6-phosphate dehydrogenase (G-6-PD) deficiency and von Willebrand disease was admitted in the Intensive Care Unit (ICU) with delirium and severe hyponatremia (119 meq/L). She was on treatment for hypothyroidism, and her chronic kidney disease was being managed by biweekly hemodialysis prior to hospitalization and by sustained low-efficiency dialysis after admission in the ICU. During her stay of 48 days, she developed nosocomial pneumonia with

acinetobacter and pseudomonas which was treated with imipenem and vancomycin and blood stream infection with candida albicans which was treated with caspofungin. She was successfully weaned off the ventilator and was maintaining a SpO₂ of 90–94% on T-piece. Her serum creatinine and blood urea had stabilized to 2.8 mg/dL and 150 mg/dL, and she was now on weekly hemodialysis. Her bicarbonate levels were 20 mmol/L, sodium was 135 meq/L, and potassium were 3.8 meq/L. Twelve hours after discontinuation of mechanical ventilation, she developed a sudden drop in saturation (SpO₂ = 50%) followed by bradycardia and hypotension. Atropine and adrenaline were administered, and mechanical ventilation was commenced. She gained consciousness within 5 min but developed pulmonary edema and subsequently severe acute respiratory distress syndrome and eventually died after 2 days.

We could not find a definite cause of hypoxia, and it was difficult to explain the rapid deterioration following the hypoxic event. The patient was conscious with good respiratory efforts, and regular suctioning was being done to prevent mucous plugging from tracheal secretions. Cardiac and respiratory events were ruled out by bedside echocardiography, chest X-ray, electrocardiogram, and compression ultrasound. Volume overload, aspiration, cardiogenic shock, upper airway obstruction, and negative pressure pulmonary edema were not responsible for this episode.

Glucose-6-phosphate dehydrogenase deficiency has been considered as a benign condition and the only emphasis till date has been on the prevention of oxidative injury and acute hemolytic crisis.^[1] There is no literature on the mortality and morbidity of patients admitted in the ICU with this enzyme deficiency. In our patient, an episode of transient hypoxia during weaning from mechanical ventilation could have led to the production of oxidants by injured lung parenchymal cells which further promoted neutrophil infiltration and development of lung edema and inflammation.^[2]

Glucose-6-phosphate dehydrogenase enzyme generates reduced form of nicotinamide-adenine dinucleotide phosphate. This is essential for keeping the glutathione in a reduced state, which is an antioxidant and thus, prevents lipid peroxidation at the cellular level. In a patient with G-6-PD deficiency, the level of reduced glutathione is inadequate, and the deficient erythrocytes are unable to handle the oxidized form. Bilateral pulmonary edema immediately after recruitment of collapsed lung has been reported in a patient with G-6-PD deficiency who received bilateral endoscopic sympathectomy under alternate

one-lung anesthesia. Hypoxemia, G-6-PD deficiency, and sympathectomy were all implicated in the development of acute pulmonary edema.^[3] Further research is thus needed to establish a pattern of lung injury in patients with this enzyme deficiency exposed to oxidative stress in ICU and perioperative period.

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