P-54
Hemolysis following carbon monoxide poisoning in a patient with G6PD deficiency

Kaiwei Yang
Emergency Department of China Medical University Hospital, Taiwan

Objective: Carbon monoxide poisoning (COP) is a prevalent medical problem that also causes significant socioeconomical impact because of its potential of causing prolonged neuropsychiatric impaired and death. So far, there is scarce reports of hemolytic anemia in patients with COP. Here, we present a case suffering from hemolytic crisis after COP, depicting a rare hematologic complication of COP.

Case report: A 26-year-old male without known medical history presented with consciousness change after burning coal. Physical examination found BP 91/57 mmHg, HR 93 bpm, RR 30 breaths/min, BT 38.4℃ and Glasgow coma scale of seven. Initial laboratory examination revealed serum potassium concentration 7.0 mmol/L, lactic acid concentration 61 mg/dL, Troponin-I concentration 2.15 ng/mL, serum creatine phosphokinase concentration 2264 IU/L, serum creatinine concentration 1.48 mg/dL, white cell count 24700/ul, hemoglobin 16.5 g/dL, and platelet count 224000/ul. Serum COHb concentration was 38.7%. We initiated hyperbaric oxygen therapy in combination with mechanical ventilation support, antibiotics, and intravenous fluid supply under the diagnosis of carbon monoxide poisoning with acute respiratory failure, aspiration pneumonitis, and rhabdomyolysis. Four days after the event, his skin became icteric and conjunctivas became pale. Follow up laboratory examination showed hemoglobin 10.3 g/dL, serum total/direct bilirubin concentration 5.3/0.2 mg/dL, LDH concentration 254 IU/L, G-6-PD quantitative concentration 2.1 U/g Hb. Coomb test was negative, indicating acute hemolytic anemia triggered by oxidative stress in a patient with Glucose-6-phosphatase dehydrogenase (G6PD) deficiency. The only medications prescribed were acetaminophen and Amoxicillin/clavulanic acid, both in therapeutic range. The hemolysis resolved after 2 days observation. The patient was discharged without any neurological sequelae.

Conclusion: Previously, three cases were reported to have hemolysis, rhabdomyolysis, and systemic capillary leak syndrome as a result of COP from coal fire.[1] All three cases did not have G6PDD history. This may be the first report to describe COP inducing acute hemolytic anemia in a G6PDD patient. G6PDD is an inherited disorder caused by a genetic defect in the red blood cell (RBC) enzyme G6PD, which generates NADPH and protects RBCs from oxidative injury. Aside from causing hypoxia, CO also causes oxidative stress by binding to cytochrome oxidase, thus directly causing apoptosis and organ damage. In conclusion, we should be vigilant of acute hemolysis in patients diagnosed with COP when they also have underlying G6PDD.

Reference: [1] Abdul-Ghaffar et al, Acute renal failure, compartment syndrome, and systemic capillary leak syndrome complicating carbon monoxide poisoning. J Toxicol Clin Toxicol. 1996;34(6):713-9.