



e-Poster Presentations

EP – 01

Hemolysis following Carbon monoxide poisoning in a patient with G6PD deficiency

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Objective: Carbon monoxide poisoning (COP) is quite a serious social and medical problem in the modern world, which causes definite mortality. Commonly, COP affects the myocardial, neuropsychiatric systems and moreover, leads to cardiopulmonary collapse. In literature, only one case report discussed about hematologic complication after COP. Here, we present a case who suffered from hemolytic crisis after COP.

Case Report: A 26-year-old male with G6PD deficiency (G6PDD) suffered from consciousness change after burning charcoal. PE revealed GCS 7, BP 91/57 mmHg, HR 93, RR 30. Initial laboratory examination revealed Troponin-I 2.15, CPK 2264, creatinine 1.48, and hemoglobin 16.5. Toxicology examination shows COHb 38.7%. We started hyperbaric oxygen therapy, mechanical ventilation support, antibiotics, and intravenous fluid supply under the diagnosis of COP with acute respiratory failure, aspiration pneumonitis, and rhabdomyolysis. Four days after the incident, his skin became yellow and conjunctivas became pale, and repeated laboratory examinations showed hemoglobin 10.3, total/direct bilirubin 5.3/0.2, LDH 254, and negative Coombs test, indicating acute hemolytic anemia. Tracing back his drug history, the only medications prescribed were acetaminophen and Amoxicillin/clavulanic acid in the therapeutic range and these medications were used continuously, even after hemolysis. The hemolysis relieved after 2 days.

Conclusion: Previously, three cases reported in Kuwait encountered hemolysis and rhabdomyolysis after COP from the same scene. One of these three cases died possibly because of severe hemolysis and none of the victims had G6PDD history. However, the fact that all patients from the same scene suffered from hemolysis possibly indicated that the triggering factor is not only COP, but also other environmental toxins. Our patient is the first and pure case with G6PDD who had acute hemolytic anemia after COP. 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. Acute hemolytic anemia is often incited by drugs, food, or medical illnesses. Carbon monoxide poisoning influences target organs by several mechanisms, including tissue hypoxia, cytotoxic effect by oxidative injury, and inflammation. Therefore,



the possible explanation of hemolysis in our patient might be due to high oxidative injury to cells by encountering severe COP and receiving hyperbaric oxygen therapy. However, further study to prove this mechanism should be conducted.