Artemether Induced Methemoglobinemia: A Case Report

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Abstract

Artemisinin derivatives are drug of choice in complicated malaria. Here we describe a case of parenteral Artemether induced methemoglobinemia with acute kidney injury in a child which we have treated with intravenous ascorbic acid and conservative management this case emphasizes for need of awareness this potential adverse effect i.e., Artemether induced Methemoglobinemia, its prompt recognition and management.

Case Summary

An eleven years old boy presented to pediatric emergency services with bluish discoloration of lips and nails for four days. He also had hematuria for two days. There were no complaints of cough, breathing trouble or palpitation, urinary frequency, urgency or burning sensation. Past history was unremarkable except a recent episode of febrile illness one week back for which he received intramuscular Artemether and oral paracetamol. His clinical examination revealed perioral and nail bed cyanosis with no other systemic abnormalities. His vitals were stable with respiratory rate of 32/min and heart rate of 96/min on admission. Pulse oximeter detected SpO2 of 65% at room air which persisted despite oxygen supplementation [10L/min]. He was shifted to pediatric intensive care unit and evaluated further. Complete hemograme showed microcytic anemia with neutrophilic leucocytosis. Liver and kidney function showed mild derangement. His arterial blood gas analysis showed hyperoxia despite low SpO2 on pulse oximeter. (Table 1)

On the basis of above findings, he was suspected to have methemoglobinemia along with acute kidney. Anemia was probably due to hemolysis as supported by anisopoikilocytosis on peripheral smear and high LDH. He was started on intravenous vitamin C infusion empirically for methemoglobinemia after drawing blood sample for same. A dose of 500mg ascorbic acid was given intravenously over 6 hours. He received total 1500mg of intravenous ascorbic acid over 36 hour. His SpO2 started improving at 24 hours and normalized at 36 hours from onset of infusion and subsequent doses were not required. On day 2 of admission child developed hypertension [140/100mm of Hg], nausea, abdominal pain along with raising serum creatinine for which symptomatic treatment was provided. However, he remained non-oliguric throughout admission. His hematuria resolved over next 5 days. Maximum serum creatinine noted was 4.4mg/dl and dialysis was not required. Serum methemoglobin level came out 27.5% confirming the diagnosis. He was discharged on day 9 of admission and further follow up of was done on outpatient basis, hypertension resolved in 2 weeks and tab Enalapril was stopped and serum creatinine normalized in 4 weeks (Figure 1).
Ascorbic acid has been found to be equally efficacious for this reaction to occur in vivo [8]. Index case received ascorbic acid infusion and showed good recovery. Methemoglobin is toxic to nephrons as well. Reports of acute renal toxicity and occasional requirement of prolonged dialysis are present [9,10]. Even an infusion of ascorbic acid infusion can cause nephrotoxicity [11-13]. Index case had transient non-oliguric acute kidney injury, probably due to methemoglobinemia. A close monitoring of renal function was done during ascorbic acid infusion and later on which showed gradual improvement and normalization in next 4 weeks. This case report highlights the rare occurrence of significant methemoglobinemia with the use of Artemether. An early suspicion and treatment will lead to complete recovery in such cases.

References