Introduction

Indoxacarb poisoning is a well-known cause for methaemoglobinemia. It’s an insecticide that is considered a safe substitute for organophosphate insecticides [1]. It’s main action is to block sodium channels in the nervous system of insects causing neurological manifestations [1,2].

This case illustrates the occurrence of methaemoglobinemia following ingestion of indoxacarb in an attempt of deliberate self-harm. Indoxacarb is an oxidative insecticide and human toxicity include blurred vision, skin sensitization and alteration in blood counts [5]. This case highlights the early recognition of methaemoglobinemia in cases of indoxacarb poisoning.

Case Report

A 70 year old male with a background of type II Diabetes mellitus, hypertension, dyslipidemia and bronchial asthma presented to the emergency unit with a history of ingestion of an unknown poison 12 hours before, with drowsiness and vomiting.

On examination, he was afebrile, PR- 92 bpm, BP 160/90, GCS 12/15, pupils were mildly dilated, reactive to light and SPO$_2$ was 82% on 5 l of oxygen. Organophosphate poisoning was suspected and he was treated with atropine 0.2 mg/ h and pralidoxime 200 mg/h infusion were initiated. He was mechanically ventilated due to persistent desaturation while being treated for possible aspiration. He was treated with IV Co-Amoxiclav, IV metronidazole and nebulized salbutamol.

Peripheral cyanosis was noticed with the arterial PaO$_2$-154mm/Hg on FiO$_2$-100%, while SPO$_2$ reading was on the pulse oxymeter was 80%. Blood during venipuncture was brownish color. Aetiologically indoxacarb poisoning was suspected as the cause for methemoglobinemia at a level of 8.55g/dl with the control being 0.05g/dl. He was treated with Methylene blue 60mg in 100ml normal saline over 15 min, repeated in 12 hours and a dramatic improvement was noted.

This patient was also managed for ventilator associated pneumonia and had an uneventful recovery.

Discussion

Methaemoglobinemia is a common presentation of indoxacarb poisoning. Methaemoglobin is generated by oxidation of haem iron to the ferric state, causing a characteristic bluish-brown colour resembling cyanosis [4]. Methaemoglobin has a high affinity to oxygen, which is not delivered to tissues and the oxygen dissociation curve is shifted towards the left [1,4]. Methaemoglobinemia should be suspected in patients with hypoxic symptoms and low SPO$_2$, though PaO$_2$ levels on ABG analysis are sufficiently high to fully saturate the haemoglobin. Bluish-brown appearance of freshly drawn blood may be a critical clue [1]. The methaemoglobin levels are usually less than 0.5%. Cyanosis usually manifests at a level of 15% and treatment is warranted at levels above 30%. Levels more than 60% is considered lethal [1].

Treatment includes ceasing exposure to the offending agent, correcting metabolic abnormalities, administrating methylene blue at a dose of 1-2mg/kg loading dose over 30 to 60 minutes and then twice daily [4].

Methylene blue gets reduced to leucomethylene blue which in turn reduces the methaemoglobin by NADPH reductase. Methylene blue also reduces the half-life of methaemoglobin. The main side effect of methylene blue is haemolytic anemia and a mild haemolysis was noted in this case.

References


