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Manifestation and Management of Paraquat Intoxiction. A deadly poison?

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Abstrac*t: Paraquat is a bipyridilium herbicide, is a bright green corrosive liquid with pungent smell that needs to be diluted before agricultural use. Globally it is the second highest-selling weed killer used and is available in a 20% solution form.[1] Its herbicidal properties were discovered in 1950s and first marketed in 1962. Most cases of intoxication especially in third-world countries are due to suicidal attempts instead of homicidal or accidental exposure, because of its widespread availability and relative low cost. The lethal dose /toxic dose is very low 35mg/kg that is (10 mL or 2 teaspoons is enough to kill) . It is a highly toxic compound and has multisystemic effects which include are pulmonary edema, convulsions, cardiac, renal, and hepatic failure[2] .*
Key words*; Paraquat, Herbicide, Acute respiratory distress syndrome andmulti organ failure.*

I. Introduction

Paraquat is used as a quaternary ammonium herbicide, one of the most widely used herbicides in the world. It is quick-acting, non-selective, and kills green plant tissue on contact and becomes biologically inactive upon contact with soil [3]

The herbicide properties of paraquat were identified in 1955 and it was introduced commercially in 1962. The active ingredient in paraquat is 1,1'-dimethyl-4,4'-bipyridinium, a non-volatile, white, crystalline solid, which decomposes at 300˚C. Pure paraquat, when ingested, is highly toxic to mammals, including humans; potentially leading to acute respiratory distress syndrome (ARDS). (4)

Route of intoxication

Paraquat is highly toxic (Category II) by the oral route, very low dose 35mg/kg just 10-15 ml of 20% of paraquat may be lethal).Intoxiction may occur via inhalation route are also higly toxic but particles used in agricultural practices (400 to 800 μm) are well beyond the respirable range . Case reports of minimal to moderate intoxication by the dermal route are also in litetratue . A large majority (93%) of fatalities from paraquat poisoning are cases of intentional self-administration, i.e., suicides. In third world countries, paraquat is a major suicide agent. [5]

Symptoms

Symptoms depend upon rote of intoxication and are usually dose-dependent. On ingestion of paraqauat posion patient may immediately start with typical symptoms of burns in throat,Sore throat, Stomach pain and Vomiting .Since bipyridyl salts are caustic, the gastrointestinal tract can be severely injured after ingestion of a concentrated solution and there may intense pain abdomen and weakness in muscle(6).Symptoms of parquat poisioning are usually dose-dependent, and intoxication can be categorized to mild, moderate, and fulminant. Doses ≤20 mg/kg produce mild intoxication, with minor gastrointestinal problems like transient vomiting, diarrhea, and oropharyngeal burns, but usually complete recovery is possible. Moderate intoxication can occur with doses between >20 mg/kg and <50 mg/kg of the poison. Patient may suffer lung injury, pulmonary fibrosis, acute renal failure, and in majority of cases, death occurs within 2-3 weeks. Doses of ≥50 mg/kg cause fulminant intoxication, may lead to death within 3 days because of multiple organ failure.(7)

The hallmark target of paraquat toxicity is the lung, where available oxygen reacts with paraquat to produce free radicals. Lung injury undergoes a biphasic response: (1) pneumocytes concentrate the compound through active transport, which leads to direct destruction of alveolar epithelium; and (2) progressive inflammation and proliferation of fibrous tissue produce widespread pulmonary fibrosis.(8) Pneumothorax and pneumomediastinum may result from corrosive injury to the esophagus.(9) Necrosis of proximal tubule cells causes kidney injury, but renal failure is sometimes reversible with aggressive hydration.(8) Direct contact with the eyes, skin, or mucous membranes can cause irritation and ulceration.Once large concentration of this poison accumulates in lungs or renal cells, it leads to generation of toxic reactive oxygen species , which devastate cellular defensive system.

Pulmonary fibrosis due to lipid peroxidation is a major symptom of paraquat intoxication . in moderate to severe cases the cause of death is normally hypoxemia, secondary to lung fibrosis (7). In patients who survive longer, fibrotic changes in the alveoli result in gas exchange interference in the lungs and may progress to ARDS.[6,7]

Renal failure can result due to the direct toxicity and hemodynamic changes .Baseline serum uric acid level might be a good clinical marker for patients at risk of mortality and AKI after acute PQ intoxication. Conservation of renal function is vital to reduce plasma paraquat levels and thereby reduce accumulation in lung cells.(6)

On contact skin patients may develop erythema followed by blistering and hemorrhaging hemorrhagic diabrosis. However, paraquat may be absorbed through skin injuries, and since 1978 there have been several reported cases of severe paraquat poisoning by this pathway (10). It can cause moderate to severe eye irritation and minimal dermal irritation.

In 1978, a middle-aged female was reported to have died from respiratory failure caused by percutaneous paraquat absorption .This showed the extreme toxicity of paraquat and demonstrated that lethal quantities may be absorbed from apparently trivial skin wounds. In 1983, it was reported that a patient whose scrotal skin had been exposed to a concentrated paraquat solution suffered from renal and respiratory failure and hepatic damage, although the patient eventually recovered. This demonstrated that dermal exposure to paraquat, particularly via the scrotum, may produce serious systemic toxicity (10).Chronic exposure can lead to lung damage, kidney failure, heart failure, and oesophageal strictures.

Complication

1Acute respiratory distress syndrome and pulmonary fibrosis ,
2.oesophageal perforation, Mediastinitis,
3.Kidney failure and
4.liver failure, Cardaic failure
5 Seizures, Coma ,and parkansonian like diseases
6.Nose bleeds

Diagnosis

Diagnosis of paraquat poisoning is usually made based on circumstantial evidences. Urine or serum paraquat levels can confirm diagnosis and predict outcomes. Urinary paraquat concentrations of <1 mg/L within 24 hours of toxicity have a high probability of survival.(11) If renal function remains normal, >90% of absorbed paraquat is excreted unchanged within 12 to 24 hours of ingestion. Serum paraquat levels can predict mortality as far as 48 hours after ingestion through the use of various nomograms.(12)]

Other test to be done; Arterial blood gases , BUN and creatinine (kidney function tests)
Chest x-ray , Lung function tests, Urinalysis and Urine toxicology screen.

Treatment

Treatment for paraquat poisoning consists primarily of supportive care. Conventional treatment includes nasogastric tube fixation, gastric lavage with normal saline, charcoal-sorbitol lavage, forced alkalinized diuresis and hemodialysis or hemoperfusion . Early efforts at gastrointestinal decontamination with activated charcoal to minimize absorption .Fluid administration to maintain urinary elimination of paraquat is important.(8)

Hemodialysis may support acute renal failure, but it does not increase clearance of the substance because it is quickly distributed to the lungs and other organs. Hemoperfusion with activated charcoal is effective if initiated within 4 hours of paraquat intoxication (13). Antioxidants such as vitamins C and E have been proposed. Immunosuppressive therapy with steroids and cyclophosphamide was shown to improve outcomes.(14) N-acetylcysteine reduces human lung epithelial cell destruction in vitro, perhaps by increasing glutathione stores and reducing superoxide anion production.(15)Ironically, oxygen supplementation may have a deleterious effect because it increases the number of toxic radicals. Oxygen should, therefore, be given only to correct hypoxemia.[8] Subsequent management includes antibiotics for supervening infection, supporting renal function with hemodialysis or filtration. Potent analgesics such as opiates may be required to alleviate intense pain from gastrointestinal tract injury, ulceration, and inflammation.Lung transplantation after paraquat ingestion has been described in case reports.(16)

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Prognosis ; The mortality rate is very high around 60% to 80%, majority of moderate to severe cases the cause of death is normally hypoxemia, secondary to lung fibrosis (6).Death may occur up to 30 days after ingestion.

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