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# A Review on Paraquat Poisoning

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**ABSTRACT:**
Paraquat is a bipyridyl compound also known as methyl viologen dichloride hydrate. Paraquat poisoning has been the most increasing type of suicidal poisoning as it has high rate of mortality. Consumption of more than 30ml leads to multiple organ failure and cardiogenic shock which is fatal. While little amounts consumed (like 10ml) can result in ulcers, edema, necrosis, inflammation, pulmonary congestion etc. Paraquat doesn't affect the brain as it cannot cross the blood-brain barrier.
There is no proper antidote for this poisoning hence, supportive treatment is given. Even under proper medical assistance the chances of mortality is very high. Because of severe toxicity and lethal effects it is banned in many countries.
**KEYWORDS:** Paraquat, Viologen, Agrochemicals, Superoxide.

## I. INTRODUCTION:

Agrochemicals like herbicide, fungicide, pesticide, weedicide etc are widely used over the world to protect the yield. India being rich in agriculture consumes a large scale of these chemicals to produce healthy crop. The average growth of these chemicals increased to 13.07% between 2015-2019. With the growing population the need for more quality and quantity of harvest has increased, indirectly increasing the use of these agrochemicals. Suicide using the agrochemicals has always been the regrettable social problem because of their lethal activity.

Paraquat being highly toxic herbicide has been classified into Restricted Use Pesticide by the U.S. Environmental Protection Agency (EPA) [1].As this agrochemical doesn't have proper antidote it becomes more difficult to control its lethal activity. The major cause of death is due to respiratory failure.

**MECHANISM OF PARAQUAT TOXICITY:**

Paraquat dichloride is a yellow solid organic compound having chemical formula [(C6H7N)2]Cl2 [2]. The production of superoxide anions gives it a toxic quality. Paraquat is an active ingredient of Grancoxone, which is used as defoliant on crops like cotton and to control weeds and grasses[3].

Paraquat undergoes redox activity and generates reactive oxygen species. Metabolism of paraquat generates paraquat mono-cation radical which gets re-oxidised to paraquat di-cation radical generating superoxide [4-7]. Hydroxyl free radical are formed in presence of iron.

Peroxinitrite, a very strong oxidant is generated as nitric oxide combines with superoxide[8]. The toxicity of organs is due to highly reactive nitrite species and oxygen. Paraquat is taken against the concentration gradient into the lung due to which lungs gets severly toxicated[9].

**EXPOSURE TO PARAQUAT:**

1. Skin exposed to concentrated amounts of paraquat for a long period.
2. Skin having sores, rashes, cuts.
3. Paraquat mixed in food, water, other beverages could poision people.
4. Most common route is ingestion of paraquat.
5. Licensed applicators of paraquat are the ones who are at high risk of exposure.

**TOXICOKINETICS OF PARAQUAT:**

Paraquat is incompletely absorbed and rapidly distributed to liver, lung, kidney and muscle. Within 24hrs of ingestion 90% of absorbed paraquat is excreted unchanged in urine. Kinetic parameters are non-linear and bioavailability increases substantially with increasing doses. After few hours, renal clearance declines rapidly in severe poisoning. Small portions deposited into deeper layers are slowly eliminated by kidneys over many days to weeks [10]. The initial elimination half life is around 6hrs. Levels of Paraquat following ingestion peak in plasma around 1–2 hours, in the lung cells by 4–5 hours and 90% is cleared from plasma by 5–6 hours[11,12].

**SIGNS AND SYMPTOMS:**

Ingestion of small quantities will show effect within several days to weeks. These effects are as follows :

- Nausea
- Vomiting

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- Abdominal pain
- Diarrhea
- Liver failure
- Kidney failure

Ingestion of large quantities will show effect within few hours to few days. These effects are as follows:

- Low blood pressure
- Coma
- Acute kidney failure
- Lung scarring
- Injury to heart
- Fast heart rate
- Confusion
- Fluid in lungs
- Dehydration
- Electrolyte imbalance
- Sore throat
- Seizures
- Respiratory failure
- Muscle weakness

**APOPTOSIS:**

Production of reactive oxygen species and activation NF-kB lead to DNA fragmentation and nuclear condensation.[13-15]. Apoptosis and unbalanced homeostasis are caused by DNA altering cellular enzymatic and signaling pathway [16]. Peroxinitrite also reacts with proteins and lipids.

**PARKINSON'S DISEASE:**

Exposure to paraquat shows a link to acquire parkinson's disease. Production of oxygen derivatives increases which harm the cellular structures and may help in developing Parkinson disease[17].

**DIAGNOSIS:**

Amount of paraquat present in body fluids like serum, urine, plasma are determined to confirm intoxication. Dithionite urine test is performed to know the paraquat level in plasma, which gives information about the severity of paraquat intoxication. Principle involved in dithionite test is the paraquat absorbance due to which blue colour urine produced on reacting with dithionite.

After a week of paraquat ingestion patient is advised to go through high-resolution computed tomography (HRCT)[18].

**TREATMENT:**

Early treatment is the most important factor. Decontamination is initial step that should be taken to remove paraquat from the body. Nasogastric suction can be used when the ingestion time is within 1hour. Renal protection is a vital job in early treatment.

Haemodialysis and haemoperfusion are most standard treatment. The reduction rate of plasma paraquat levels is more in haemoperfusion than in haemodialysis[19].

Antioxidants like GSH and NAC should be administered intravenously as a large amounts of relative oxygen species are produced immediatelyafter ingestion[20,21]. Patients with acute paraquat intoxication are given prednisone,a synthetic corticosteroid that is given as immunosuppressant drug.

**EPA ACTIONS TO PREVENT INGESTION AND REDUCE EXPOSURE OF PARAQUAT:**

New closed-system packaging designed to prevent transfer or removal of the pesticide except directly into proper application equipment. This will prevent spills, mixing or pouring the pesticide into other containers.

Changes to the pesticide label and distribution of supplemental warning materials to highlight the toxicity and risks associated with paraquat products.

Restricting the use of paraquat to certified pesticide applicators only. Individuals working under the supervision of a certified applicator are prohibited from using paraquat.

**PRECAUTIONS:**

- Never be used around home gardens, schools, recreational parks, golf courses or playgrounds.
- Never be transferred to a food, drink or any other container
- .Never be stored in or around residential dwellings.
- Always be kept secured to prevent access by children and/or other unauthorized persons.
- Be used only by a certified applicator.
- Follow label instructions.
- Use the required personal protective equipment specified on the product label.

## II. CONCLUSION:

As paraquat doesn't have proper antidode and treatment it is advised to follow all the precautions and rules while using it.

## REFRENCES:

[1]. https://www.ohsu.edu/sites/default/files/2019-11/paraquat/20poster/2011.11.19.pdf
[2]. Michaelis, L.; Hill, Edgar S. (1933). "The viologen indicators"

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[3]. Paraquat herbicide information,UT institute of agriculture. https://psep.tennessee.edu/paraquat/
[4]. Yang W, Tiffany-Castiglioni E. The bipyridyl herbicide paraquat induces proteasome dysfunction in human neuroblastoma SH-SY5Y cells.2007
[5]. Castello PR, Drechsel DA, Patel M. Mitochondria are a major source of paraquat-induced reactive oxygen species production in the brain.2007
[6]. Bonneh-Barkay D, Reaney SH, Langston WJ, Di Monte DA. Redox cycling of the herbicide paraquat in microglial cultures.2005
[7]. Adam A, Smith LL, Cohen GM. An assessment of the role of redox cycling in mediating the toxicity of paraquat nitrofurantoin.1990
[8]. Ahmad I, Shukhla S, Singh C. The involvement of nitric oxide in maneb and paraquat-induced oxidative stress in rat polymorphonuclear leukocytes.2008
[9]. Rannels DE, Kameji R. Spermidine uptake by type II pneumocytes:interactions of amine uptake pathways.1989
[10]. Mouy R, Bismuth C, Bourdon R, Baud FT. Toxicokinetics of paraquat in humans.1990
[11]. Hong SY, Yang JO, Kim SH. Effect of haemoperfusion on plasma paraquat concentration in vitro and in vivo.2003
[12]. Bismuth C, Garnier R, Baud FJ. Elimination of paraquat.1987
[13]. Velez-Pardo C, Rio MJ. Paraquat induces apoptosis in human lymphocytes: protective and rescue effects of glucose, cannabinoids and insulin like growth factor-1.2008
[14]. Yang W, Tiffany-Castiglioni E. Paraquat-induced apoptosis in human neuroblastoma SH-SY5Y cells involvement of p53 and mitochondria.2008
[15]. Fordel E, Thijs L, Van Bockstacle D, Moens L, Dewilde S. Neuroglobin and cytoglobin overexpression protects human.
[16]. Denicola A, Radi R. Peroxynitrite and drug-dependent toxicity.2005
[17]. "Two pesticide-retenone and paraquat-linked to parkinson disease" Science daily 2011
[18]. Lee SH, Lee KS,Kim SH, Hong SY. Paraquat poisoning of lung thin section CT findings radiology.1995
[19]. Kim SH, Hong SY, Yang JO. Effect of haemoperfusion on plasma paraquat concentration in vitro and in vivo.2003
[20]. Hong SY, Gil HW, Yang JO, Chung YH, Lee EM. Effect of high dose intravenous N-acetylcystein on the concentration of plasma sulfur- containing amino acids.2005
[21]. Hong SY, Yang JO, Lee EY, Lee ZW. Effects of N-acetyl-L-cysteine and glutathione on antioxidant status of human serum and 3T3 fibroblasts 2003