

PARAQUAT POISONING – A CASE REPORT

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ABSTRACT

Paraquat is one of the most dangerous poisons in the medical field as Ingestion of small quantities (>10mL) may damage lung irreversibly and also lead to renal failure causing to death. The paraquat is sold in more than 130 countries around the world to be used in small as well as large farms. The easy availability of this poison makes it a commonly employed agent for suicide. More than 93% of deaths due to paraquat poisoning are suicidal in nature and most of them are occurring in developing countries. The clinical features vary from case to case based on the amount and type of compound consumed. As it is a fatal condition, early diagnosis and proper management is warranted which can reduce the morbidity and mortalities. But sometimes early diagnosis may not save the victim as in our case. We present a case wherein the victim succumbed to acute respiratory distress following consumption of paraquat.

Key words: Paraquat, lung damage, renal failure, respiratory distress.

INTRODUCTION

Poisoning from pesticides and other agricultural chemicals is a major public health problem worldwide, especially in the developing countries. The most commonly employed herbicides are paraquat and glyphosate. Paraquat is a green colored, strongly aromatic herbicide commonly available and widely used in the agricultural community. It is a very dangerous pollutant of the environment, as it readily binds to both clay and organic matter in soil and is very slowly biodegraded. Its chemical composition is 1,1'- dimethyl-4,4'- bipyridinium. It belongs to the group of dipyridyl herbicides (1). It is present in varying concentrations ranging from 10%-40% and requires to be diluted prior to use. Its cost effectiveness and ready availability is a major factor for its continual use. Unfortunately, this also makes it a highly used agent in suicides and homicides.

Paraquat is highly toxic chemical to human beings. Even a teaspoonful of the active ingredient is fatal. It has both acute and delayed effects. It causes serious and irreversible delayed effects if absorbed.

CASE REPORT

The present case had a relatively short and vague history as taken by the police and subsequently by the clinicians. The deceased was a 29 year old female, engaged in farming with her family. There were certain losses in the household due to inadequate crop yield and it was stated that she took the step of consuming poison as she was troubled with all the problems she faced. There was no evidence or history of any prior psychological disorders or any medications. Her medical history prior to the terminal event had been unremarkable. The deceased was admitted to Kasturba Hospital, Manipal after ingestion of the poison. The quantity consumed was unknown. However, the identity of the poison was confirmed as the family had brought an empty pack of "Gramaxone" with them. She was treated for acute respiratory distress and renal failure. However, she did not recover and passed away.

On Autopsy: The external examination revealed the body to be that of an adult female, moderately built and nourished and fair in complexion. There were no obvious external injuries on the body. A few therapeutic artefacts were

noted, consistent with hospital treatment records. Conjunctiva of both the eyes was pale. She had yellowish discolouration of the palms and feet. The body was cold and stiff due to storage in the mortuary cold chamber. Oral cavity had a few ulcerations. All other orifices were intact and unremarkable. Her hospital case records showed an elevation in creatinine, urea and liver enzyme levels along with deranged arterial blood gases.

On internal examination, the dura was found to be intact and unremarkable. However upon removal of the dura, a diffuse subarachnoid haemorrhage was found over the fronto-temporo-parietal region on both sides. The brain was removed and no evidence of any fractures was found over the base of the skull. The cut section of brain showed multiple, petechial haemorrhages in the white matter on both sides. The oesophagus showed mucoid blood stained fluid and the mucosa was pale and unremarkable. The lungs were found to be firm, congested and showed a blood stained-purulent discharge on cut section. The stomach was found to have a greyish black coloured fluid without any obvious odour or identifiable food particles. The mucosa of the stomach was haemorrhagic at places. Liver was yellowish on cut section and all other organs were unremarkable. Viscera were sent for further forensic analysis to the Regional Forensic Science Laboratory which was positive for the compound.

Cause of Death: As per the hospital investigations cause of death in all probability is due to complications arising secondary to consumption of paraquat poisoning.

DISCUSSION

Paraquat poisoning is a relatively understudied and under-researched condition as evident in the search of literature. This is saddening in view of the knowledge that paraquat toxicity causes fatality in a large number of cases despite treatment in a tertiary care setting(2). With an expected mortality of 55 % in clinical settings being reported in healthy males without significant co-morbidities, it remains a serious health issue throughout the world(3). The concentrations of Paraquat available in the market vary from 10-40%. However, the lethal dose is calculated as being 35mg/kg which translates to as low as 10-15 ml of a 20% solution of paraquat available commercially(4).

The symptoms of paraquat poisoning may be classified as mild, moderate or fulminant depending on the route of intake, dose and the time of presentation to the hospital. Being a caustic agent, the local signs of inflammation will be seen even with mild doses. Taken orally, it shows oral and oesophageal erosions along with gastric mucosal damage. The main activity of paraquat occurs in the lungs. Upon reaching the lungs via the blood, it induces lung injury which is morphologically characterised by an early destructive phase, in which the alveolar type I and type II epithelial cells are damaged; and a second proliferative phase defined by alveolitis, pulmonary oedema and infiltration of inflammatory cells. The entire activity occurs as a result of superoxide radicals and other free radicals created due to the accumulation of paraquat in the lungs(5). Other theories put forward also hypothesise that inflammatory mediators and immune activation also play a major role in the damage caused due to the toxin(6,7). The biotransformation of the toxin occurs primarily in the kidney and hence urine concentrations are often high enough, however it also has a detrimental effect in the form of rapidly inducing acute kidney injury, thus forming a vicious circle in which the only way to remove the toxin is effectively shut down(8).

Though the intact human skin is relatively impermeable to paraquat, the fatalities have been documented from dermal exposure (9). Such dermal exposure to paraquat can lead to skin injuries which include rashes, severe dermatitis of second degree burns and even death (10). A UK agricultural worker died due to paraquat poisoning when container with the liquid slipped from his hand and the chemical splashed on his face (11). A farmworker in UK in 1994 suffered from a severe groin infection after applying paraquat with a knapsack sprayer (12). A study conducted in Malaysia reported health problems among plantation sprayers after exposure to paraquat (13).

The present treatment regimes advocate the use of gastric lavage with activated charcoal as an effective first aid measure. Apart from this the initiation of haemodialysis as soon as possible is proven effective in removal of the toxin and reducing mortality. Certain authors have also advocated the use of immunosuppressant agents and antioxidants for the treatment as an adjunct, but never as a substitute to haemodialysis (14).

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CONCLUSION

The presence of paraquat poisoning is a real and present danger in a society as agriculturally prosperous and profound as ours and must be always suspected in all cases with a history of accidental, intentional or homicidal poisoning. As there is no specific antidote for the toxin, the mainstay of treatment remains supportive in nature, thus causing a high mortality. Hence, as a specialist in forensic medicine, there must always remain a suspicion of paraquat toxicity.

There must be further research to develop adequate tests to determine the presence of paraquat toxicity in live or deceased patients as the post mortem features are grossly vague and resemble multi system failure. (15) Hence more and more researches in regards to management of paraquat toxicity are warranted to prevent the fatalities due to this life threatening condition. The paraquat toxicity should be considered in case of unexplained combination of respiratory and gastrointestinal symptoms and acute renal damage, even in the absence of a proper history of consumption.

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