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BRADYCARDIA IN PARAQUAT POISONING: A SURPRISING CARDIAC MANIFESTATION IN A YOUNG PATIENT

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SUMMARY

A young male patient was admitted to JSS Hospital after consuming 10-15ml of 24% Paraquat dichloride. Initially treated with induced vomiting and intravenous medication at a local facility. He had no previous medical history. Two days after admission, he developed sinus bradycardia. Despite this, he showed no symptoms like giddiness or syncope, didn't need oxygen support and no lung or kidney injury. Treatment included N-acetyl cysteine infusion, dexamethasone, cyclophosphamide and other symptomatic management. At subsequent follow-ups, patient was hemodynamically stable with no further episodes of bradycardia.

KEY WORDS

Paraquat dichloride, Sinus bradycardia, N-acetyl cysteine

BACKGROUND

Paraquat is a highly toxic chemical widely known for its quick action in killing plants and its effectiveness in weed control. Unfortunately, it is also highly toxic to humans if ingested, inhaled or absorbed through the skin. In fact, it is one of the most toxic herbicides in use. Paraquat acts as a contact poison. In the human body, paraquat causes cellular damage by generating highly reactive oxygen species, which can lead to oxidative stress and tissue damage. Typical symptoms include nausea, vomiting, abdominal pain, difficulty breathing, confusion, muscle weakness, organ failure (especially lung and kidney failure), seizures and coma. The prognosis for paraquat poisoning is often poor, especially in intentional ingestion or high-level exposure cases. Many individuals who ingest paraquat die from multi-organ failure, primarily due to lung and kidney damage. Survival is more likely with prompt and aggressive medical treatment.

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CASE PRESENTATION

A young male patient was admitted to the Department of General Medicine JSS Hospital on September 27th 2023, after consuming 180ml of alcohol at 7:30 pm and later, at around 10:30 pm, 10 - 15ml of 24% paraquat dichloride at his residence. He was a chronic alcoholic, consuming nearly two quarters of alcohol per day. He informed his attendants, who induced vomiting and took him to a local hospital. He was subsequently referred to a district hospital, where he received one dose of 1gm intravenous methylprednisolone, 500mg cyclophosphamide and other supportive treatments. The patient was then brought to JSSHospital for further management. He had no other medical or surgical history. Upon examination, the patient was conscious, oriented to time, place and person, with a pulse rate of 76 bpm regular, Blood pressure of 120/80 mmHg, Respiratory rate of 14 breaths/min and oxygen saturation of 99% on room air. He exhibited multiple hesitation cuts on his left forearm and systemic examination were unremarkable.He developed bradycardia with a heart rate of 52 to 56 bpm after two days. The patient was closely monitored in the ICU for potential complications of paraquat poisoning and bradycardia. He did not require oxygen support and his urine output remained satisfactory.

	28/9/2023	**29/9/2023**	**1/10/2023**
Haemoglobin (g/dl)	14.7	14.4	16.0
Total counts (cmm)	8670	8520	7390
Platelet count (mcL)	2.42	1.8	2.36
Serum urea (mmol/L)	28	32	47
Serum creatinine (mg/dl)	0.63	0.66	0.66
Electrolytes (mEq/L)			
Sodium	136	136	141
Potassium	4.3	4.2	4.4
Total bilirubin (mg/dl)	0.63	0.80	0.68
Serum albumin (g/dL)	4.4	3.9	3.9
AST (IU/L)	62	39	15
ALT (IU/L)	62	49	30
ALP (IU/L)	138	112	102
PT-INR		13.1/0.97	
Urine routine		6-8 RBC/HPF 4-6 pus cells/HPF	

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Trop T (ng/ml)		0.008	
CK-MB (ng/ml)		0.78	
BNP (pg/ml)		31	
S. calcium (mg/dl)		8.6	
S. magnesium (mg/dl)		2.0	
T3 (ng/dL)		0.46	
T4 (ng/dL)		5.7	
TSH (mIU/L)		0.166	
S. cortisol (mcg/dL)		0.40	
HIV **HbsAg** **HCV**		Positive Positive Negative	
Fasting lipid profile (mg/dL) **Cholesterol** **Triglycerides** **HDL** **LDL** **VLDL**		 175 106 32 137 21	
AFP (ng/mL)		5.5	

TABLE – 1 (BLOOD INVESTIGATION)

ECG on admission showed normal sinus rhythm, Chest X-ray showed no radiological abnormalities, ultrasound (USG) of the abdomen and pelvis revealed a normal study. Echocardiogram demonstrated mild tricuspid regurgitation, normal valves and chambers with an ejection fraction of 60%. ECG after two days of admission showed sinus bradycardia. 24hr Holter monitoring was done, due to sinus bradycardia showed no evidence of arrhythmias with sinus bradycardia predominantly at 54 bpm. Incidentally serology for HIV and HbsAg was positive. Due to HbsAg positivity, HbeAg was tested and found to be positive with negative anti-HbeAg antibodies. HBV DNA quantitative analysis indicated a viral load of >97,00,00,000 copies. CD4 count was 86. Fundoscopy showed normal study.Other potential causes of bradycardia, such as hypothyroidism, were ruled out as the patient's thyroid profile was within the normal range. It was noted that the patient had low serum cortisol levels, which were likely a result of the steroid injections he had been receiving. Toxic myocarditis was also considered as a potential diagnosis, but it was ruled out due to the patient's normal levels of trop-T and CK-MB as well as a normal Echocardiogram.The patient was initiated on a 24-hour N-acetyl cysteine infusion. Additionally, he received intravenous dexamethasone at a dosage of 8mg thrice daily for the subsequent 72 hours, coupled with cyclophosphamide administered at 500mg twice daily

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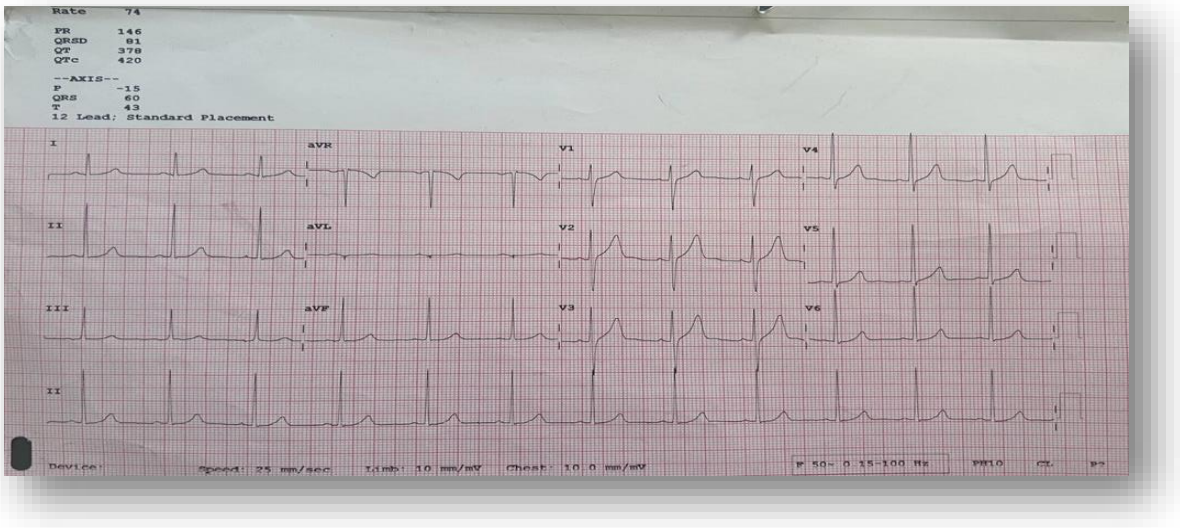
for 48 hours. During this period, the patient was maintained on nil per oral status for 48 hours, provided with appropriate hydration and other supportive medications.

FIG – 1 (ON ADMISSION DAY 1 ECG – NORMAL SINUS RHYTHM WITH HEART RATE 74 BPM)



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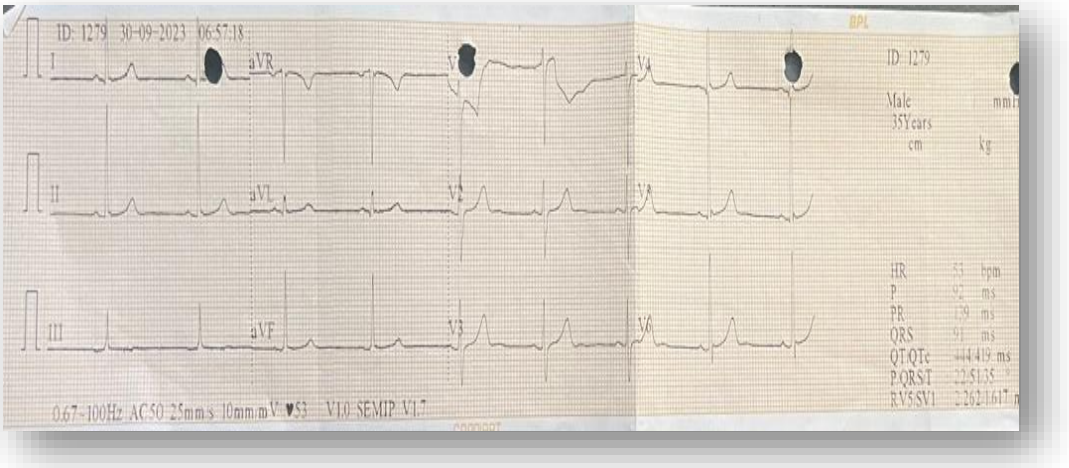

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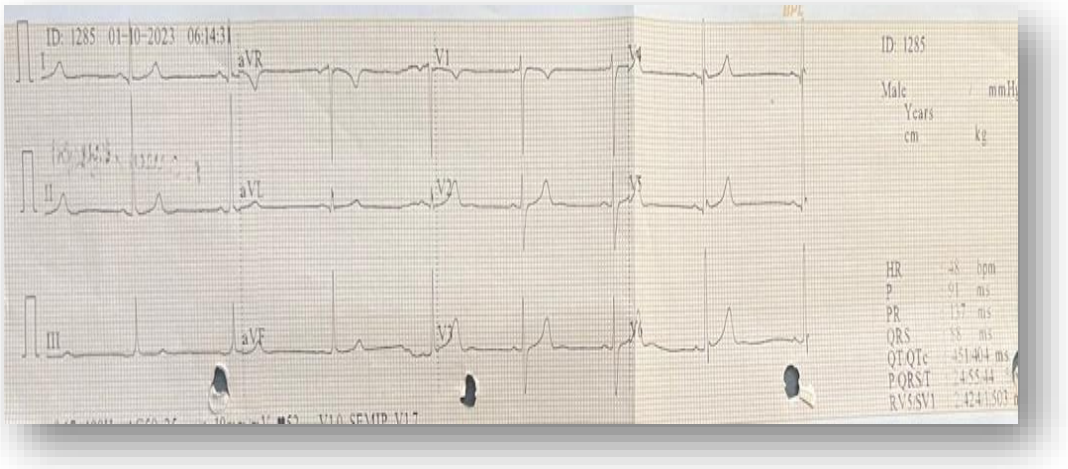

FIG 2 AND FIG 3 – (ECG AFTER 3RD AND 4TH DAY OF ADMISSION SHOWINGSINUS BRADYCARDIA WITH HEART RATE OF 53 BPM AND 48 BPM RESPECTIVELY)

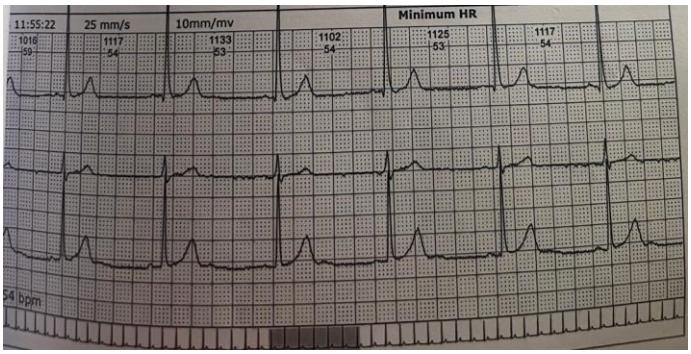

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FIG 4 – (24 HR HOLTER MONITORING – SINUS BRADYCARDIA)

OUTCOME AND FOLLOW UP

Despite the early initiation of treatment, the patient experienced transient sinus bradycardia, which is a very rare complication of paraquat poisoning. Fortunately, the patient did not develop any signs of lung or kidney dysfunction. He was commenced on an appropriate treatment regimen for his HIV and HbsAg infections. During follow-up after 7 days of discharge, patient was reassured and he remained compliant with his medication and repeat ECG showed normal sinus rhythm with heart rate of 78bpm regular.

DISCUSSION

Paraquat is a highly toxic herbicide that can cause a range of severe and potentially fatal toxic effects when ingested, inhaled or absorbed through the skin. Some of the major toxic effects of paraquat poisoning include:

1. **Gastrointestinal Symptoms:** Nausea, vomiting, abdominal pain, and diarrhea are common early symptoms of paraquat poisoning, often occurring within hours of exposure.
2. **Respiratory Distress:** Paraquat is particularly toxic to the lungs. It can cause progressive lung damage, leading to difficulty breathing, coughing, and severe respiratory distress. Lung fibrosis can develop, which is often irreversible and can be fatal.
3. **Cardiovascular symptoms:** Paraquat consumption can lead to tachycardia and shock.
4. **Kidney Damage:** Paraquat can lead to kidney damage, resulting in impaired kidney function and potential kidney failure.
5. **Liver Toxicity:** Elevated liver enzymes (such as AST and ALT) can occur indicating liver damage.
6. **Central Nervous System Effects:** Paraquat poisoning can affect the central nervous system, causing confusion, seizures, and even coma in severe cases.
7. **Multi-Organ Failure:** In severe cases, paraquat poisoning can lead to multi-organ failure, with the lungs and kidneys being particularly vulnerable.

While the most common complications of paraquat poisoning involve the lungs, kidneys, and other major organs, there are some rare but severe complications that can occur, including Cardiac Arrhythmias, Gastrointestinal Bleeding, Bone Marrow Suppression causing anemia, bleeding, and increased susceptibility to infections. Even in cases where patients survive acute paraquat poisoning, they may suffer from long-term lung damage and pulmonary fibrosis, which can significantly impact their quality of life.

Treatment of Paraquat Poisoning:

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The treatment of paraquat poisoning is challenging and often requires immediate and aggressive medical intervention. Although there is no specific antidote for paraquat, the following treatment strategies are commonly employed:

1. **Decontamination:** If the patient presents soon after exposure, decontamination measures like gastric lavage (stomach pumping) and activated charcoal may be used to limit further absorption of paraquat.
2. **Supportive Care:** Patients may require respiratory support, such as mechanical ventilation, due to lung damage. Supportive care also includes fluids and electrolyte balance to maintain organ function.
3. **Specific Therapies:** Some specific therapies have been used, including the administration of antioxidants like N-acetylcysteine (NAC) to reduce oxidative stress and counter the toxic effects of paraquat.
4. **Hemodialysis:** In cases of severe kidney damage, hemodialysis may be necessary to remove paraquat from the bloodstream.

LEARNING POINTS

1) Paraquat is a highly toxic herbicide with severe health risks for humans affecting the lungs, kidneys and other systems.
2) Recognizable symptoms of paraquat poisoning include gastrointestinal distress, respiratory problems, confusion, and organ failure.
3) The prognosis for paraquat poisoning is often poor, especially with intentional exposure and leading to multi-organ failure.
4) Treatment is challenging; it involves supportive care and in some cases, therapies like N-acetylcysteine.
5) Rare complications can include cardiac issues, gastrointestinal bleeding, bone marrow suppression, and long-term lung damage, emphasizing the need for comprehensive care and awareness of potential complications.

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