

An Unusual Case of Reversible Cardiomyopathy Secondary to Paraquat Poisoning

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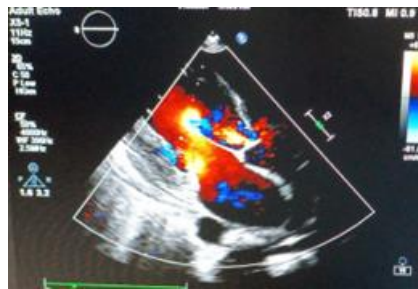
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Abstract: *Paraquat is a commonly used herbicide worldwide and is usually one of the main causes of fatal and lethal poisoning worldwide in the developing nations. The most effected organs are lungs and kidneys. However, experimental researches have shown that the heart can also be affected indirectly through increased vascular permeability. Various animal studies have shown that paraquat poisoning causes myocardial contractile dysfunction by decreased fractional shortening and cardiac remodeling.1 We report a first case in published literature of a 30yr old Dravidian women with reversible cardiomyopathy strongly associated with paraquat exposure. It is essential to obtain a detailed medical history and a proper diagnostic workup including work, social and family history, and echocardiography, baseline EKG, lab work and ischemia cardiac testing as it can lead to improved diagnostic evaluation of possible etiologies of commonly seen dilated cardiomyopathies and help identify less well - known etiologies as seen in our patient.*

Keywords: Cardiomyopathy, Paraquat, Heart failure

1. Introduction

Dilated cardiomyopathy is a subset among cardiomyopathies defined by reduction of ejection fraction to less than 40% and a dilated left ventricle. DCM is currently the most common subtype of cardiomyopathies, affecting 1 in every 2500 adults of 20 - 50 years age.² Prevalence rates are comparatively higher among men. Some commonly seen causes of DCM are genetics, diabetes, obesity, hypertension, and substance abuse, arrhythmias, chemotherapy drugs, coronary ischemia valvular abnormalities and exposure to toxins.³ Despite this, the etiology of DCM is not clearly understood and further evaluation through thorough history, clinical examination, imaging, and genetic studies are needed. Environmental factors can also have a contributing role in the development of DCM. We present a case of DCM which reverted to normal in a home maker in rural India



Echocardiography

Case report: A 30 - year - old Dravidian woman with no past medical history presented to the emergency department with complaints of throat pain, nausea and 1 episode of non - bloody, non - bilious, vomiting, and chronic cough following consumption of small amount of paraquat poison ~3ml. On presentation she was diaphoretic, but had no complaints of chest pain or shortness of breath. EKG showed Q waves in anterior and inferior leads along with T inversions. High sensitive troponins were elevated to

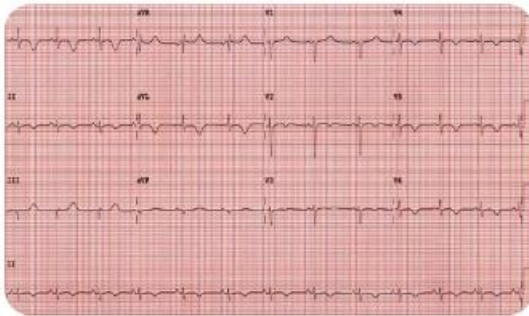
3.9pg/ml. On day 1, Hemodiafiltration with ultrafiltration was done initially with stable vitals for 3 hours. On day 2, 2nd session of Hemodiafiltration with no ultrafiltration was done as her output was adequate and no signs of volume overload.⁵ Despite this, patient gradually transitioned from non - oliguric renal failure to oliguric renal failure with serial rise in the levels of serum creatinine. The patient was subsequently treated with antibiotics, antioxidants, and fluids. Subsequently the patient developed acute pulmonary edema and hypotension for which she was treated with diuretics and inotropic support. Gastric opinion was taken for burning epigastric sensation, and the patient underwent upper gastrointestinal endoscopy which revealed erosive gastritis and was started on antacids for the same. On day3 cardiology consultation was done for high serum troponin and CK - MB.2d - echo demonstrated global LV dysfunction with LVEF 30%. On day 5, loop diuretics were stepped up and nebulization was given for respiratory discomfort. On started. On day 8, beta blockers were started for increased heart rate. On day 9, pt developed carpopedal spasms. Bone profile was done and showed low serum calcium levels and was treated with calcium gluconate. Potassium supplements were given for low serum potassium levels. Patient had initially high levels of nt - pro bnp 884pg/ml, high sensitive trop - I 3.9pg/ml and required non - invasive ventilation for 3days for metabolic acidosis. Following which the patient gradually improved with maintaining good urine output and good renal function parameters. Over the course of hospital admission, the patients presenting symptoms resolved. The patient was ambulatory and had no signs of respiratory distress. She was discharged with home medications and a follow up appointment with cardiology after 2wks of discharge A review 2d echo scan after 4 wks showed improvement of cardiac function with resolving LV dysfunction (LVEF 40%) and after 8 wks showed complete recovery of LV dysfunction (LVEF 50%)



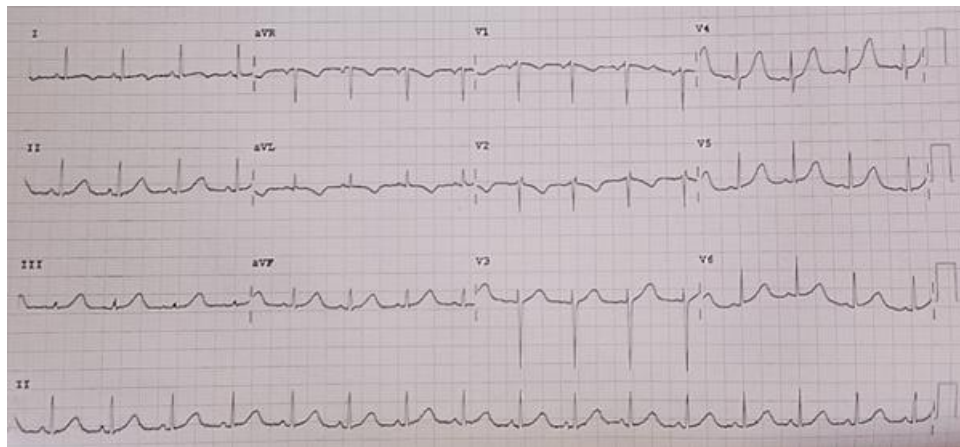
UGI Endoscopy Showing Erosive Gastritis



Chest X-Ray at Admission



EKG at presentation



EKG at discharge

2. Discussion

Paraquat is a toxic herbicide commonly used in developing nations to curb weed growth. It is a fast-acting pro-oxidant compound that induces damage due to reactive oxygen species development and subsequent stress injury via lipid peroxidation. Paraquat has been implicated to cause wide-spread multi-organ failure, most commonly affecting the renal, pulmonary, neurological, cardiovascular, and gastrointestinal through accumulation of ROS. As the primary mode of elimination for paraquat is renal, damage to them induces buildup of toxins in tissues.

Several cases have demonstrated that lungs are the most vulnerable to the acute effects of paraquat toxicity and being least treatable. Paraquat toxicity has several direct and indirect effects on the cardiovascular system.⁶ Histopathological evidence comprises of edema, congestion, and hemorrhage in the myocardium. Indirect effects demonstrated ischemic changes to the heart mediated by ROS.

The patient's diagnosis was reached after thorough investigation excluded other potential causes of cardiomyopathy, including a detailed assessment of medical history and imaging results. There was no indication of heavy alcohol or drug use, ruling out substance abuse. Additionally, there was no history of conditions like hypertension, diabetes mellitus, or exposure to cardiotoxic drugs. Viral or parasitic infections such as Chagas disease were also considered but deemed unlikely due to the absence of relevant symptoms in the patient's history or current presentation.

Sarcoidosis was eliminated as a possibility as the patient's clinical profile did not align with this condition. Despite experiencing dyspnoea on exertion, other typical symptoms of sarcoidosis such as malaise, fatigue, fevers, wheezing, abnormal weight loss, cutaneous findings, or ocular abnormalities were not observed. Chest radiography showed no signs of hilar adenopathy, further supporting the diagnosis of dilated cardiomyopathy (DCM).

To confirm a diagnosis of dilated cardiomyopathy (DCM) secondary to paraquat exposure rather than idiopathic DCM, it would be necessary to conduct an invasive biopsy of the cardiac tissue and genetic testing to exclude other potential causes of DCM. Although a cardiac biopsy could have provided valuable diagnostic insights in our case, it was not pursued due to its invasive nature and the patient's reluctance to undergo the procedure, especially considering her improvement with medical treatment.

The evidence obtained from laboratory tests, imaging studies, and the patient's medical history strongly suggests a link between paraquat exposure and the development of DCM in this patient. After eliminating other primary causes of DCM, such as coronary artery disease, substance abuse (including alcohol), diabetes, hypertension, and exposure to certain chemotherapy agents, it appears likely that the patient's DCM is not just coincidental but rather directly related to his past exposure to paraquat, indicating a causal relationship.

Conclusion: Our study presents the initial documented instance of reversible dilated cardiomyopathy strongly linked to paraquat herbicide exposure. Dilated cardiomyopathy is a prevalent condition globally, highlighting the importance for healthcare providers to gain a deeper comprehension of its diverse causes, including emerging cases like ours involving the toxin paraquat. Through this case, we aim to underscore the importance of understanding the adverse effects of paraquat to enable clinicians in recognizing its symptoms promptly. This heightened awareness can facilitate early detection and the delivery of effective treatment to affected patients.

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