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PARAQUAT INDUCED BILATERAL SPONTANEOUS PNEUMOTHORAX-DAISLEY BARTON SYNDROME – A CASE REPORT

Medical Science

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ABSTRACT

Paraquat (1,1-dimethyl-4, 4-bipyridylium dichloride) is an effective herbicide which is widely used all over the world and India is no exception. It is toxic to humans due to its redox activity which produces superoxide anions. Accidental or suicidal poisoning causes ulceration over the mouth and gastrointestinal tract and the majority of patients die of acute renal failure, hepatic failure and acute lung injury causing mainly lung fibrosis and consolidation. Treatment of paraquat poisoning is extremely difficult as it does not have any specific antidote. Treatment is mainly symptomatic. Very few patients may develop spontaneous pneumothorax, known as Daisley Barton syndrome, which further impairs survival of the patient Here we present a patient of paraquat induced spontaneous bilateral pneumothorax who survived with conservative treatment.

KEYWORDS

Paraquat, Spontaneous pneumothorax, Daisley Barton syndrome.

INTRODUCTION:

Paraquat is an organic compound with formulae (C6H7N2) Cl2. It is used as a herbicide and it is very potent and toxic. The first case of mortality due to paraquat was published in the British Medical Journal in 1966[1]. Paraquat ingestion is seen in many parts of Asia, and in the Pacific nations and the Americas [2]. Paraquat ingestion is mostly suicidal. Paraquat ingestion has a mortality of 60 to 80% [3]. Mild poisoning occurs with ingestion of paraquat of less than 20 mg / kg body weight, moderate poisoning with 20-50 mg/kg body weight and severe more than 50mg/kg of body weight[4]. In cases of mild paraquat poisoning symptoms are usually confined to gastrointestinal system. Moderate to severe poisoning presents mostly with multi organ failure, local gastrointestinal system ulceration or perforation, renal and pulmonary complications and manifests within days to weeks [5]. Pulmonary manifestations are mainly extensive bilateral fibrosis, pneumonia or consolidation and a few may present with spontaneous pneumothorax only to add to the mortality. As no specific antidotes are available, patients are treated conservatively with steroids, cyclophosphamide [6], anti-oxidants [7], N-acetylcysteine and early haemodialysis[8] all of which have been shown to decrease mortality in treatment. Serial chest X-rays were done which showed spontaneous some studies. Here we report a case of paraquat poisoning complicated by oral ulceration, renal impairment, bilateral lung fibrosis and consolidation in both upper zones who developed bilateral spontaneous pneumothorax (Daisley Barton syndrome) during the course of the illness. The patient was treated conservatively and discharged to follow up.

CASE REPORT:

An 18-year-old male was admitted in our hospital with suicidal paraquat ingestion of approximately 1.5 grams. On admission he had ulceration over the tongue, buccal mucosa and lips. He was treated with iv fluids, N- acetylcysteine (600mg thrice daily), methylprednisolone (1gm once daily for three days),[9] and electrolyte and nutritional balance was maintained. Alternate day serum electrolytes, complete hemogram, urea, creatine, and liver function were measured. On routine examination his serum urea and creatinine were rising and he also had oliguria. He was put on early haemodialysis and started on total parenteral nutrition. During third week of hospital stay, he developed dry cough and progressive shortness of breath. CXR PA showed bilateral upper lobe heterogenous opacities (Fig:1). HRCT thorax showed irregular fibrotic parenchymal lesions with patchy fibrotic collapse consolidation in bilateral upper lobes. Mantoux test, sputum for Acid Fast Bacilli were done which were found to be negative. He complained of increasing respiratory distress and repeat CXR PA view showed bilateral pneumothorax. (Fig:3). He was treated with low dose moist oxygen inhalation and nebulisation and iv Meropenem thrice daily. The patient responded well with conservative treatment. Serial chest X-ray were done which showed spontaneous resolution of the pneumothorax (Fig,4,5). A subsequent HRCT thorax showed resolution of pneumothorax and improvement in B/L upper zone fibrosis and consolidation. Renal function and liver function became normal. Pre-discharge UGIE was done and was normal. The patient was discharged and is now under follow up.

Table-1

Day	Hb gm/dl	TC /mm^3	PLT lac/mm^3	Urea	Creatinine	Sodium	Potassium	Bilirubin	AST	ALT
1	12.1	8,510	1.62	29.0	0.9	137	3.9	0.87	95	92
3	11.9	10,200	1.51	56.4	1.59	135	4.2	0.82	78	75
10	12.2	9,000	1.97	36.4	1.22	144	3.6	1.12	84	70
20	11.6	9,320	2.20	32.9	1.17	139	3.9	1.0	50	38
30	11.7	7,110	1.72	39.1	1.02	140	3.3	1.11	30	21
50	12.5	6,500	2.20	35.7	1.00	134	3.2	0.9	32	25
60	11.4	7,650	1.99	38.7	1.10	129	4.1	1.5	40	38
80	10.9	5,500	1.80	30.0	0.97	135	4.3	1.7	35	39

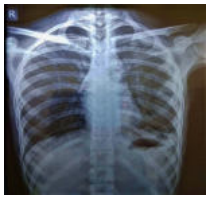

FIG 1: CXR(PA) with B/L upper lobe opacity.

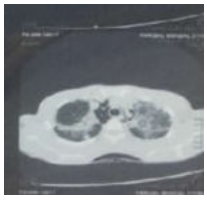

FIG 2: HRCT thorax B/L collapse consolidation and fibrosis

FIG 3: CXR(PA) B/L pneumothorax

FIG 4: CXR(PA) left sided pneumothorax

Fig5: spontaneous resolution of pneumothorax

DISCUSSION-

Paraquat is a herbicide which causes multiorgan failure due to its oxidant properties. The paraquat is chiefly excreted by the kidneys, so it causes renal failure. Paraquat gets accumulated in a highest concentration in the lungs, 6-10 times more than in the blood causing a diffuse consolidation. The peripheral and subpleural alveoli may eventually rupture to cause pneumothorax and pneumomediastinum. In many cases the pneumomediastinum and pneumothorax go unnoticed. Paraquat forms reactive oxygen species, hydrogen peroxide, hydroxyl radical which cause lipid peroxidation, activation of NF-kB, mitochondrial damage destroying the integrity of cell membranes. Acute lung injury is caused by damage of type I and type II pneumocytes by reactive oxygen species.Damage to type I pneumocytes prevents gas exchange and type II pneumocyte damage limits surfactant production [2]. The decrease in surfactant increases surface tension within alveoli which causes atelectasis of lung, emphysematous changes and rupture of alveoli causing pneumothorax and pneumomediastinum. There is also development of pulmonary hypertension [10]. There is destruction in type II pneumocytes and increase in surface tension causing atelectasis, rupture and emphysematous changes and allowing alveoli gas to escape and causing spontaneous pneumothorax. Spontaneous Pneumothorax as a sign of acute paraquat ingestion is uncommon and mostly goes unnoticed and was first observed in 1990 by Daisley and Barton[11].

CONCLUSION:

Acute paraquat poisoning is very common and lethal. Morbidity and mortality are governed by multi-organ dysfunction. Pulmonary complications like diffuse fibrosis and consolidation play the major role. Spontaneous pneumothorax or pneumomediastinum can add to the mortality and morbidity further, which can go unnoticed. Hence knowledge of the condition and early detection of the syndrome can improve outcome and save many lives.

Conflict of interest-None declared.

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