AFRICAN JOURNAL OF MEDICINE and medical sciences

VOLUME 23, NUMBER 4, DECEMBER 1994

EDITOR: B.O. ONADEKO
ASSISTANT EDITORS:
B.O. OSOTIMEHIN and A.O. UWAIFO

SPECTRUM BOOKS LIMITED
Ibadan • Owerri • Kaduna • Lagos

ISSN 1116–4077

Afr. J. Med. med. Sci. (1994) 23, 401-403

A fatal case of paraquat poisoning in a Nigerian

E.E.U. AKANG[1,*], F.A. ADEYEMI[2], R.W. BAMIDELE[3], H.A ADEWOYE[4], O. GUREJE[3], G.T.A. IJADUOLA[2], P.U. AGHADIUNO[1] and S.B. LAGUNDOYE[5]

Departments of [1]Pathology, [2]Otorhinolaryngology, [3]Psychiatry, [4]Haematology and [5]Radiology, University College Hospital, Ibadan, Nigeria.

Summary

A fatal case of paraquat poisoning in an adolescent girl is reported. Death resulted from respiratory failure because of extensive interstitial and intra-alveolar fibrosis.

Résumé

Un cas fatal d'empoisonnement dû á paraquat dans une fille adolescent est rapporté. Elle a mouri par suite d'insuffisance pulmonaire resulter de fibrose interstitielle et intra-alvéolaire.

Introduction

Paraquat (1,1 - dimethyl - 4,4 -dipyridylium dichloride) is an extremely toxic herbicide, first introduced for commercial use in 1963[1]. Millions of gallons of this chemical are used annually in agriculture worldwide[2,3].

The predominant toxic effect of paraquat is on the lungs but multiple organ involvement may occur in individual patients depending on the dosage and route of administration of the pioson[1,4]. Because paraquat preferentially accumulates in the lungs, death usually occurs within 2 weeks of administration from respiratory failure[3]. Morphological pulmonary changes in reported fatalities range from intra-alveolar exudation following high dose exposure and early death, to progressive interstitial and intra-alveolar fibrosis with longer term survival[5].

The majority of paraquat-related deaths have been reported from Europe, Japan and America[1], and 80 cases of paraquat poisoning occur annually in the United Kingdom alone[6]. Despite the widespread use of herbicides and pesticides, paraquat poisoning appears to be distinctly uncommon in Africa and Asia[7].

We report here a fatal case of paraquat poisoning in a Nigerian female. As far as we know no previous Nigerian case has been reported.

Case report

A 17 year old girl first presented at the University College Hospital, Ibadan on the 30th September, 1990 with a history of having deliberately ingested a tablespoonful of Gramoxone (20% paraquat[1]) at home the previous evening. She vomited several times. Her parents empirically administered 5 tablespoonsfuls (75ml) of palm oil before bringing her to the hospital.

On initial evaluation she complained of a painful swallowing and retrosternal pain. Otolaryngological examination showed hyperaemia and congestion of the mucosa overlying the soft palate and anterior fauces. There was no oropharyngeal or hypopharyngeal ulceration.

The chest appeared clinically clear. Oesophagoscopy could not be carried out immediately. A nasogastric tube was passed and the patient was given a gastric lavage and placed on intravenous fluids, magnesium trisilicate and parenteral antibiotics. She also had a psychiatric consultation on 3 October, 1990 during the course of which it was learnt that the patient was previously unaware of the lethal effects of paraquat and had only intended to punish her parents. On the seventh day following ingestion of paraquat (6th October, 1990) the patient developed jaundice associated with elevated serum alkaline phosphate (283 IU/L), aspartate aminotransferase (603 IU/L) and alanine aminotransferase (514 IU/L).

A chest X-ray done on 6 October, 1990 showed no abnormality of the heart and lung fields. A second chest X-ray done 8 days later showed minimal distension of the pulmonary vasculature which would be in keeping with engorgement of the interstitial compartment.

* To whom correspondence should be addressed.

The patient continued to vomit repeatedly and on the fifteenth day after admission (14 October, 1990) she developed tachypnoea, central cyanosis, persistent hypothermia (35°C) and subsequently died.

At post mortem examination, the most characteristic morphological changes were in the respiratory system. The right and left lungs were firm and enlarged, weighing 620gm and 585gm respectively. There were bilateral subpleural petechial haemorrhages and accompanying serosanguineous pleural effusion of about 100ml on each side. The cut surfaces of both lungs had a mottled, congested appeareance.

Microscopically, there was extensive interstitial fibrosis both within the alveolar septa and around blood vessels, as well as focal areas of intra-alveolar fibrosis (Fig. 1). The intervening terminal bronchioles were dilated giving a honeycomb appearance at low magnification. In some areas, there was desquamation of the lining bronchiolar epithelium, while in other areas there was epithelial hyperplasia with focal squamous metaplasia. Moderate pulmonary oedema as well as fibrin exudates lining the walls of respiratory bronchioles to form hyaline membranes were also seen (Fig. 2). There were patchy interstitial infiltrates of lymphocytes, plasma cells, macrophaes and occasional inflammatory giant cells.

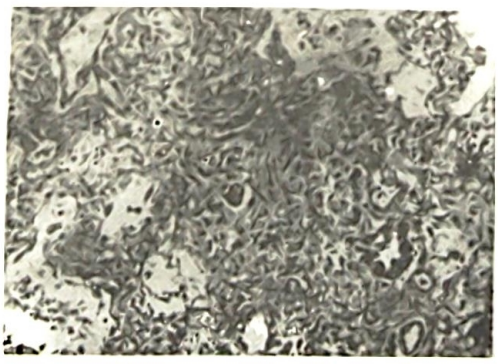

Fig. 1: Histological section of patient's lung showing interstitial fibrosis.

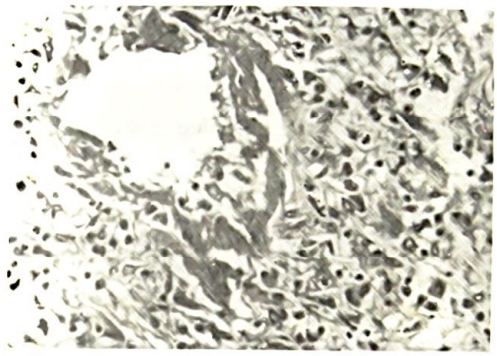

Fig. 2: Section from the lung showing hyaline membrane formation and patchy interstitial infiltrates of chronic inflammatory cells.

Significant structural changes were also present in the liver which weighed 1480gm and had a yellowish colour. There was centrizonal hepatic necrosis accompanied by polymorphonuclear infilatration and prominent Kupffer cells.

There were multiple superficial oesophageal ulcers and gastric erosions. Apart from cerebral oedema, no remarkable lesions were present in the other organs examined.

The anatomic findings were consistent with respiratory failure due to paraquat induced interstitial pulmonary fibrosis.

Discussion

Most of the reported fatalities following paraquat exposure have been due to suicidal, accidental or uncommonly homicidal ingestion[2,4]. Because paraquat is rapidly inactivated by soil, occupational exposure does not usually result in death[1].

Human deaths have occurred after oral ingestion of as little as 4mg/kg weight of paraquat[1]. Our patient ingested a tablespoonful (15ml) of weedkiller probably containing approximately 3gm (50mg/kg) of paraquat.

The basis of toxic lung injury is the generation of oxygen derived free radicals by the bipyridylium cation, resulting in lipid peroxidation of alveolar epithelial membranes and impaired surfactant activity[3,8]. This toxic effect is enhanced by high oxygen tensions, and by the persistence of high

levels of paraquat in the lungs for several days. All other organs rapidly eliminate paraquat within 24 to 36 hours[3]. These factors explain the localization of the major lethal consequences of paraquat poisoning to the lungs.

The radiological changes in the lungs are those of early engorgement of the interstitial compartment which like in our case may be so subtle as to pass for being within normal limits. The fact that the radiological changes in the lung do not reflect the severity of the histological changes has been stressed in the literature[3,4]. In severe pulmonary involvement by paraquat poisoning the radiological changes are not different from those seen in a multiplicity for aetiological factors leading to diffuse alveolar damage with consequent increased pulmonary permeability. Notable causes of diffuse alveolar damage (adult respiratory distress syndrome) include inhalation of toxic fumes or noxious fluids, circulating toxins and poisons, acute radiation injury, transfusion reactions, smoke from major disasters, aspiration of gastric contents (Mendelsohn's syndrome) and recovery from drowing disasters[9].

Our patient developed liver dysfunction due to centrizonal liver necrosis. Hepatotoxicity is dose-dependent, ranging from centrizonal or midzonal necrosis to bridging hepatic necrosis[4].

Corrosive oral and oesophageal mucoal damage is the earliest manifestation of paraquat intoxication as illustrated by our case[17]. Other reported lesions include acute tubular necrosis and cerebral oedema[1,3].

Two important principles guide patient management following paraquat ingestion, namely, limitation of absorption by gastric lavage, administering Fuller's earth or by inducing emesis and secondly, elimination of already absorbed toxin by haemoperfusion, plasmapheresis, haemodialysis or forced diuresis[1,3,7]. Unfortunately, there is no effective antidote to paraquat and pulmonary changes are irreversible and ultimately fatal[1,3]. The use of free radical scavengers[1], lung irradiation[6] and even lung transplantation[1] have only yielded equivocal results.

In most environments, paraquat poisoning is unlikely to constitute a major medical problem. However, accidental or suicidal ingestion of paraquat and other toxic chemicals can be drastically curtailed by restricting their distribution. Commercially available preparations must also be distinctly labelled as poisonous products.

References

1. Fletcher K. Paraquat poisoning. In: Ballantyne B, ed. Forensic Toxicology. John Wright and Sons Ltd., Bristol; 1974: pp 86-98.
2. Teare RD. Poisoning by paraquat. Med. Sci. Law. 1976; 16: 9-12.
3. Conradi SE, Olanoff LS, Dawson WT. Fatality due to paraquat intoxication. Confirmation by post mortem tissue analysis. Am. J. Clin. Pathol. 1983; 80: 771-6.
4. Ramachandran S, Rajapakse CNA, Perera MU. Further observations on paraquat poisoning in man. Forensic Sci. 1974; 4: 257-66.
5. Rebello G, Mason JK. Pulmonary histological appearances in fatal paraquat piosoning. Histopathology. 1978; 2: 53-66.
6. Williams MV, Webb DB, James KW, Davied BH. Lung irradiation for paraquat toxicity. Br. J. Radiol. 1985; 58: 1144.
7. Obel AOK. Practical therapeutics; first aid and therapy of poisoning from agricultural insecticides and herbicides. East Afr. Med. J. 1984; 61: 335-8.
8. Manktelow BW. The loss of pulmonary surfactant in paraquat poisoning — A model for the study of the respiratory distress syndrome. Br. J. Exp. Pathol. 1967; 48: 366-9.
9. Grainger RG, Allison DJ. Diagnostic radiology, An Anglo American textbook of imaging. Vol. 1, Churchill-Livingstone, Edinburgh. 1986; pp 261-272.

(Accepted 31 March, 1992)