

Recovery from severe pulmonary damage due to paraquat administered intravenously and orally

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The most impressive changes occurring in paraquat toxicity are to be found in the lung, and when pulmonary lesions develop radiographically after a latent period death is almost invariable. There are only two published reports of patients surviving despite radiographic changes. One patient developed appearances thought to be due to paraquat induced pulmonary oedema¹ and the other developed atelectasis and pleural effusions.² We describe a patient who recovered from diffuse lung damage after self administration of paraquat by intravenous and oral routes.

Case report

A 42 year old man had suffered recurrent bouts of endogenous depression for at least 20 years. He was admitted to hospital after intentional intravenous and oral self poisoning using a solution of Weedol (a granular preparation of 2.5% paraquat with 2.5% diquat). Two hours before admission he had dissolved the contents of about half a packet of Weedol in 500 ml of cold tap water and injected, using a sterile needle and syringe, two 10 ml aliquots of this solution intravenously. He had then drunk the remainder of the solution. One hour later he dissolved the contents of one full packet of Weedol in about 1000 ml of cold tap water and injected 3 ml of this solution intravenously. He then drank about 500 ml of the remaining solution. He thus took about 2500 mg of both paraquat and diquat, of which about 60 mg of both paraquat and diquat were administered intravenously.

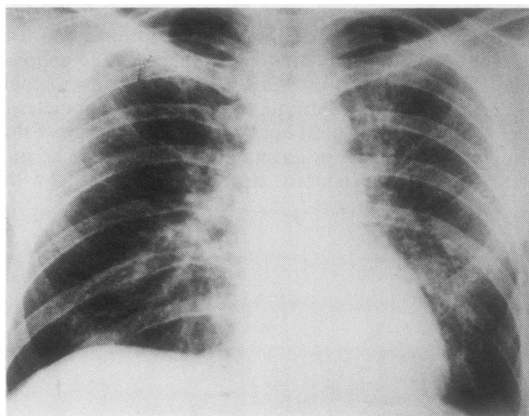
On admission he was not distressed and examination revealed nothing remarkable apart from venepuncture marks in both antecubital fossae. A chest radiograph showed a normal sized heart and clear lung fields.

Initial management consisted of gastric lavage followed by oral administration of Fuller's Earth and magnesium sulphate. A simple qualitative urine test (dithionite) confirmed the presence of paraquat. The four hour blood paraquat concentration was 2.3 µg/ml. Forced diuresis using ethacrynic acid was started six hours after admission and continued for 18 hours. The 10 hour blood paraquat concentration 0.28 µg/ml. On the third day he developed a severe pharyngitis, for which regular Difflam mouth washes (0.15% benzydamine hydrochloride) were prescribed. On the fourth day he became dyspnoeic and cen-

trally cyanosed but had no cough or expectoration of sputum. A chest radiograph showed widespread interstitial and intra-alveolar shadowing compatible with paraquat toxicity (fig). The arterial oxygen tension was 5.1 kPa (38 mm Hg), carbon dioxide tension 3.3 kPa (25 mm Hg) and pH 7.28. Because of considerable dyspnoea oxygen was reluctantly allowed as required. As a qualitative urine test for paraquat on day 5 was still positive and mild renal failure had developed (serum creatinine concentration 0.23 mmol/l (mg/100 ml)) two six hour haemoperfusions through an activated charcoal column (Gambro Adsorba 300C) were performed on days 6 and 9. Subsequently no paraquat could be detected in urine or blood. Pathogens were not isolated from repeated blood cultures. The serum creatinine concentration reached a maximum of 0.26 mmol/l (mg/100 ml) on the seventh day, but mild renal impairment continued for a further 12 days. At no time was the patient oliguric.

Two weeks after admission he no longer required oxygen. The chest radiograph showed right upper lobe collapse with left mid and upper zone confluent shadowing. Both lower zones showed clearing. Results of pulmonary function tests are shown in the table.

Two months later he was well and symptom free.



Chest radiograph revealing widespread interstitial and intra-alveolar shadowing on the fourth day of hospital admission.

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Table Results of lung function studies*

	Predicted value	Days after ingestion							
		4	6		9		14	22	62
			Before	After	Before	After			
			haemoperfusion		haemoperfusion				
FEV ₁ (l)	4.10	2.86		2.84			3.61	3.71	4.42
FVC (l)	5.05	3.36		3.38			4.30	4.60	5.47
TLC (l)	6.9	4.3		4.5			5.6	6.3	6.3
TLCO	10.6	3.5		3.6			5.1	6.3	7.5
KCO	1.6	0.8		0.8			0.9	1.1	1.1
PaO ₂ (kPa)	11.3–12.7	5.1	5.2	7.3	7.4	9.2	9.3	10.1	10.7
PaCO ₂ (kPa)	4.7–6.0	3.3	3.2	3.3	3.8	4.2	5.6	5.6	4.5
Arterial pH	7.35	7.28	7.40	7.39	7.40	7.41	7.44	7.41	7.45

*All arterial samples were taken without supplementary oxygen for at least one hour.

TLC—total lung capacity; TLCO—transfer factor (units mmol min⁻¹ kPa⁻¹); KCO—transfer coefficient (units mmol min⁻¹ kPa⁻¹) (Conversion to traditional units $\times 0.33$).

Discussion

This report describes the recovery of a patient with paraquat poisoning severe enough to cause widespread pulmonary damage. There is only one other report of intravenous paraquat administration—the patient succumbed on the twentieth day after poisoning with 2 ml of a commercial preparation of paraquat (500–600 mg).³

Although Fuller's Earth may have reduced absorption of the orally administered poison that taken parenterally would have reached the pulmonary circulation in a high bolus concentration. Paraquat is preferentially concentrated in the lung.^{4,5} Although the total dose of paraquat taken was low the parenteral administration and resulting high serum concentration may have determined the lung damage in our case. Furthermore, the injected granular particles could have contributed to the pulmonary damage, especially as they are likely to have paraquat attached to them, which would ensure a high local concentration.

It is now recognised that paraquat when taken orally may have an early toxic effect on the lung.⁶ Von der Hardt and Cardesa⁷ describe a case of death in two days after Gramoxone ingestion (20% solution of paraquat) in a patient with lung haemorrhage and almost complete loss of alveolar and bronchiolar epithelium was present; and Yoneyama *et al*⁸ found lung haemorrhage in a patient dying one day after swallowing Gramoxone. Our patient, although he did not take a concentrated preparation of paraquat, developed diffuse chest radiographic changes on the fourth day associated with severe respiratory distress. We initially considered his prognosis to be poor as the blood paraquat concentration at four hours was in the lethal range.⁹ Oxygen was allowed to alleviate his hypoxic symptoms despite the fact that there is evidence that oxygen increases paraquat lung toxicity.¹⁰ He did not have the features of pulmonary oedema described in the case reported by Gardiner,¹ neither did he develop oliguria or weight gain or have a positive fluid balance during the period of forced diuresis (which had been completed 48 hours before the onset of respiratory symptoms). He thus appears to be unique in surviving widespread pulmonary damage.

The role of haemoperfusion in this case is unclear. The amount of paraquat removed was small (about 0.72 mg) and we do not think that this affected the outcome despite an improvement in arterial oxygen tension of about 2 kPa (15 mm Hg) noted after haemoperfusion.

The case demonstrates that when diffuse interstitial pulmonary damage is found in paraquat poisoning progression is not invariable.

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