

Notes and Miscellanea

Acute cadmium pneumonitis: a 17-year follow-up

The follow-up of a case of a welder who developed acute cadmium pneumonitis after a single day's exposure to fumes of the metal is given. When the case was reported four years after the incident it was thought that there had been no permanent pulmonary damage.¹ The man has now developed evidence of progressive pulmonary fibrosis, which is presumed to be a late result of the acute poisoning.

Case history

On the day of the incident in September 1963 the welder, then aged 51, on his first day with a new firm, was instructed to weld plates of cadmium silver alloy. He developed a severe illness the same evening with a rigor, chest pain, and dyspnoea. Investigation of the patient and of the welding process left no doubt that he had suffered from acute cadmium pneumonitis as a result of the single day's exposure to the fumes. The patient later returned to work with the same firm but with no further exposure to cadmium. He continued to work regularly at a light labouring job with only a few periods off work with colds or minor chest infections until he stopped work in 1976 because of the illness of his wife.

The patient now complains only of some shortness of breath on exertion. He has to stop when walking up hills but his walking capacity is also reduced by intermittent claudication. He has almost no cough or sputum, and smokes three to four cigarettes a day.

EXAMINATION IN OCTOBER 1980

Finger clubbing first noticed in 1972 was more pronounced. The patient's weight had remained steady. He had a slight cough with some clear sputum, breath sounds were poorly heard throughout both lungs. Fine crepitations at both lung bases first heard in 1974 were prominent and persisted after coughing. There was no evidence of heart failure.

Haematological and biochemical tests gave normal results except for some changes in plasma proteins. Electrophoresis of the plasma was noted as

normal in 1970 but in 1973 an increase in gamma-globulin was recorded. IgA levels remained high in 1977 and 1979 at 12.6 and 12.7 g/l.

Urine examination had shown a trace of albumin several times during the years but the 1980 specimen was completely normal.

Chest radiography The original radiograph taken three weeks after the incident showed widespread shadowing, particularly at the lung bases. This gradually cleared and within six months the films were almost normal. A slight increase in shadowing at the lung bases remained, however, and this had become more prominent, particularly since 1972. The most recent film showed patchy shadowing in all areas of both lungs with widespread nodulation (nodules 1-2 mm in diameter and most profuse at the right base).

Respiratory function tests have been difficult to compare over the years partly because the earlier tests of the transfer factor were done by the steady state method and partly because the patient now found it difficult to perform the tests. Reliable results are shown in the table.

Results of respiratory function tests

	Sept 75	Oct 77	Nov 78	Feb 79	Oct 80
FEV ₁	1.34	1.85	1.75	1.65	1.42
Predicted FEV ₁	2.45	2.31	2.26	2.23	2.20
% of predicted	55%	80%	77%	74%	64%
FVC	1.69	2.30	2.10	2.0	1.81
Predicted FVC	3.15	2.97	2.96	2.93	2.91
% of predicted	54%	77%	71%	68%	62%
TLC	4.37				4.03
Predicted	8.30				7.43
% of predicted	53%				54%

This man now presents a picture indistinguishable from cryptogenic fibrosing alveolitis. He has progressive dyspnoea, finger clubbing, basal crepitations, and typical radiological appearances. There is a largely restrictive ventilatory defect with reduced gas diffusion.

It is possible that the patient has spontaneously developed cryptogenic fibrosing alveolitis but it seems far more probable that the progressive fibrosis has resulted from the single day's cadmium inhalation 17 years ago. There is no evidence of renal damage.

It was suggested in the previous report that no permanent damage had followed the episode of acute cadmium pneumonitis, and this opinion has been quoted in a DHSS booklet.² There is little doubt that this conclusion was wrong, and that pro-

gressive pulmonary fibrosis developed as a late result of the acute cadmium inhalation.

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References

- ¹ Townshend RH. A case of acute cadmium pneumonitis: lung function tests during a four-year follow-up. *Br J Ind Med* 1968;25:68-71.
- ² Department of Health and Social Security. *Pneumoconiosis and related occupational diseases*. London: HMSO, 1979:26. (DHSS NI 226.)