

Severe lipid pneumonia following attempted suicide by mineral oil immersion

I R Hussain, F P Edenborough,
R S E Wilson, D E Stableforth

Abstract

Following an attempted suicide by drowning in a vat of mineral oil, a previously fit man survived the usually fatal lipid pneumonia resulting from total immersion after intensive support and prolonged steroid therapy with recovery of chest radiography and pulmonary function at one year.

(Thorax 1996;51:652-653)

Keywords: lipid pneumonia, mineral oil immersion, suicide attempt.

Lipid pneumonia usually presents after chronic recurrent ingestion of oily substances, commonly laxatives, or exposure at work. Massive exposure by total immersion is usually fatal. We report a unique case of survival after attempted suicide by deliberate immersion in mineral oil.

Case report

A 43 year old non-smoking industrial supervisor with chronic depression was rescued after deliberately jumping into a vat of mineral oil used for machine oil blending. He was admitted as an emergency, coughing and breathless having ingested oil. Clinical examination was normal. Investigations revealed a leucocytosis of $24\,000/\text{mm}^3$ and hypoxia of PaO_2 9.0 kPa, Paco_2 5.7 kPa on 35% inspired oxygen which responded to an increased concentration of 60% inspired oxygen. Initial chest radiography and serum electrolytes were normal.

Despite oral ampicillin 500 mg four times a day he became pyrexial with a productive cough and by the fifth day was agitated and confused with hallucinations which required haloperidol and amitriptyline. Computed tomography (CT) of the thorax showed bilateral basal pneumonic consolidation. Broad spectrum antibiotics were commenced despite negative blood and sputum cultures, and over the next 10 days his condition remained stable. From day 16 he deteriorated further with worsening hypoxia (PaO_2 5.9 kPa, Fio_2 60%), respiratory alkalosis and pyrexia accompanied by increased agitation and confusion. Chest radiography showed right basal consolidation but sputum culture

grew respiratory commensals only. Empirically, 30 mg of oral prednisolone was commenced.

Fibreoptic bronchoscopy with trans-bronchial biopsy specimens on day 22 confirmed lipid pneumonia with an organising pneumonitis and foamy macrophages containing lipid droplets. No infective organisms were isolated. Repeat thoracic CT scanning confirmed increased basal consolidation and a right pleural effusion (fig 1) which, on aspiration, revealed 200 ml of blood stained, sterile exudate with a protein content of 46 g/l. Liver function became mildly deranged with an elevated international normalised ratio of 1.4 and he was commenced on vitamin K. A CT brain scan revealed no focal abnormalities and it was felt that his mental state was due to a post-anoxic confusional state rather than cerebral oil embolism. He developed troublesome ventricular and supraventricular tachycardias which responded to intravenous and oral amiodarone; this was stopped prior to discharge. Electrocardiographic and echocardiographic findings were normal.

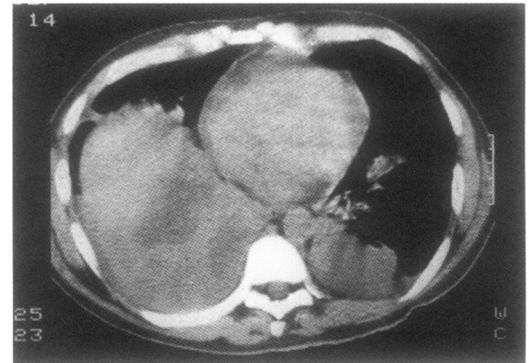


Figure 1 T1 weighted thoracic CT scan (day 23, no contrast) showing bilateral consolidation with effusion more marked on the right.

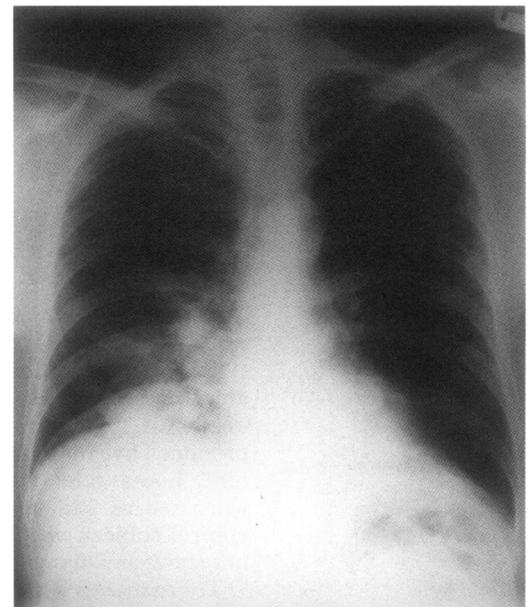


Figure 2 Chest radiograph (AP erect, day 54) showing right basal consolidation that had developed by day 5, intensified by day 23, and persisted for some six months before resolution at one year.

Kingston Hospital,
Gallsworthy Road,
Kingston on Thames,
Surrey KT2 6QH, UK
I R Hussain

Department of
Respiratory Medicine,
Birmingham
Heartlands Hospital,
Bordesley Green East,
Birmingham B9 5SS,
UK
F P Edenborough
D E Stableforth

Royal Shrewsbury
Hospital,
Mytton Oak Road,
Copthorne,
Shrewsbury SY3 8XQ,
UK
R S E Wilson

Correspondence to:
Dr D E Stableforth.

Received 14 February 1994
Returned to authors
14 June 1994
Revised version received
10 January 1995
Accepted for publication
7 March 1995

He was finally discharged after 33 days on 20 mg oral prednisolone per day. Three weeks after discharge (day 54) his cough persisted and his chest radiograph remained abnormal (fig 2) but by eight weeks after discharge he had regained 15 kg in weight and pulmonary function tests revealed only mild abnormalities (forced expiratory volume in one second/forced vital capacity 74%, lung volumes 90%, gas transfer factor 83% predicted). His prednisolone was tailed down and at one year his lung function tests and chest radiograph were normal.

Discussion

Reported causes of lipoid pneumonia include chronic ingestion of small quantities of mineral oil-based laxatives in the old¹ or very young,^{2,3} oily nasal drops, lip gloss or Vaseline, and exposure in the work place such as while siphoning diesel fuel, oil spraying in the engineering industry,^{4,5} or whilst cleaning oil-containing vats.⁶ Total immersion is rare and usually fatal.⁷ No references to deliberate suicide attempts have been found.

Presenting features include cough, fever, and hypoxaemia with radiographic consolidation, leucocytosis, and a negative infection screen. Traditionally, the diagnosis requires recognition of lipid laden macrophages in sputum or open lung biopsy specimens.^{3,5} Fibreoptic bronchoscopy with bronchoalveolar lavage has a high diagnostic yield² and, in some cases, lavage may be therapeutic.⁸ In our case, despite significant aspiration, the bronchoalveolar lavage on day 22 was negative for oil staining.

Magnetic resonance imaging (MRI) and CT scanning have been used as non-invasive methods for aiding the diagnosis of lipoid pneumonia. Lipid deposits show high signal intensity on T1 weighted MRI sequences whilst CT scans may reveal low density (fatty) areas within consolidated lung.^{1,6} Neither technique is in itself diagnostic.

The pathology of lipoid pneumonia due to animal fats is a brisk inflammatory pneumonitis leading to necrosis. Vegetable and mineral oils emulsify and induce a foreign body reaction with foamy lipid laden macrophages, interstitial giant cells, and progressive fibrosis which is often indolent until infection supervenes.⁶ Im-

mediate survival after total immersion usually leads to death within hours with evidence of systemic emboli. Later deaths in the absence of embolisation result from prolonged hypoxia associated with pulmonary fibrosis.⁷

Treatment of lipoid pneumonia involves identification and removal of the initiating agent, oxygen therapy and oral steroids with or without antibiotics.^{7,9,10} Steroids may effect rapid improvement and fever lysis, though premature reduction may lead to relapse and prolonged treatment has been advocated.^{6,10} Although persisting radiographic abnormalities and reduced lung function due to fibrosis have been reported after chronic ingestion,^{4,6} our patient now has normal radiographic and lung function tests at one year.

Lipoid pneumonia due to chronic ingestion often presents diagnostic challenges, though bronchoscopy with transbronchial biopsy now offers a safer route to histological diagnosis than traditional open lung biopsy. Massive exposure usually results in death due to oil emboli. In both situations oxygen and steroid therapy are currently the treatment of choice with prolonged therapy aimed at reducing pulmonary fibrosis. The role of therapeutic bronchoalveolar lavage in acute cases remains to be evaluated.

The authors thank Dr J Henderson, Consultant Radiologist, for reviewing the thoracic CT scans and Miss K Boyce for typing the manuscript.

- 1 Stern EJ. Chronic progressive, bibasilar infiltrates in a woman with constipation. *Chest* 1992;102:263-5.
- 2 Kameswaran M, Annobil SH, Benjamin B, Salim M. Bronchoscopy in lipoid pneumonia. *Arch Dis Child* 1992;67:1376-7.
- 3 Fan LL, Graham LM. Lipoid pneumonia from mineral oil aspiration. *Arch Paediatr Adolesc Med* 1994;148:205-6.
- 4 Glynn KP, Gale NA. Exogenous lipoid pneumonia due to inhalation of spray lubricant. *Chest* 1990;97:1265-6.
- 5 Van den Plas O, Trigaux J-P, Van Beers B, Delaunoy L, Sibille Y. Gravity dependent infiltrates in a patient with lipoid pneumonia. *Chest* 1990;98:1253-4.
- 6 Graham JR. Pneumonitis following aspiration of crude oil and its treatment by steroid hormones. *Trans Am Clin Climatol Assoc* 1955;67:104-12.
- 7 Piperno D, Gaussorgues P, Bachmann, P, Guerin C, Boyer F, Robert D. Acute respiratory distress syndrome after accidental immersion in mineral oil. [French] *Rev Med Interne* 1988;9:414-6.
- 8 Lauque D, Dongay G, Levade T, Caratero C, Carles P. Bronchoalveolar lavage in liquid paraffin pneumonitis. *Chest* 1990;98:1149-55.
- 9 Miller A, Bader RA, Bader ME, Teirstein AS, Selikoff IJ. Mineral oil pneumonia. *Ann Intern Med* 1962;57:627-34.
- 10 Ayvazian LF, Steward DS, Merkel CG, Frederick WW. Diffuse lipoid pneumonitis successfully treated with prednisolone. *Am J Med* 1967;43:930-4.