

Paraffinoma of lung: lipoid pneumonia

Report of two cases

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Paraffinoma of lung—localized lipoid pneumonia due to exogenous oil in the alveoli—must be remembered when considering unusual lung disorders, for this benign lesion can mimic lung cancer clinically, radiologically, during operation, and even on frozen section. Though usually caused by mineral oils taken as nose drops, sprays or laxatives, it can be caused by animal or vegetable oils. The lesion is essentially a macrophage response with phagocytosis of oil droplets. Symptoms can be minimal, simulating 'virus pneumonia', local, with shortness of breath, cough, sputum, haemoptysis, and chest pain, or general. The history of oil ingestion, the finding of lipophages or free lipoid in the sputum, may suggest the diagnosis. Two cases treated 10 years earlier by lobectomy, one as lung cancer and the other as tuberculosis, and proven histologically to be paraffinoma of lung are described. Both patients had used oily nasal drops for chronic nasal sinusitis. Today they remain well.

Paraffinoma of lung, which is localized lipoid pneumonia due to mineral oil in the alveoli, is important to bear in mind when considering unusual disorders of the lung for this benign lesion can mimic lung cancer clinically, radiologically, during operation, and even on frozen section biopsy (Berg and Burford, 1950).

Lipoid pneumonia was first described in 1925 by Laughlen in one adult and three infants. Ikeda (1937) showed that infantile lipoid pneumonia differed pathologically from that in fit adults where there develops a tumour-like lesion he called 'paraffinoma'. Paterson (1938), in an extensive experimental survey of 18 types of oil instilled into lungs, confirmed that the lesion was essentially a macrophage response with phagocytosis of the oil droplets. He found the more unsaturated the oily substance, the more finely is it emulsified within the lung, and the more marked is the resulting phagocytosis.

Reviews covering numbers of paraffinomas have appeared in the American literature (Berg and Burford, 1950; Davis, Hampton, Bickham, and Winship, 1954; Buechner and Strug, 1956; Steinberg and Finby, 1956). In the British literature there are fewer reports, cited by Siddons (1958) when describing his own three cases.

Lesions due to exogenous oil and fat granulomas must be distinguished from those containing

endogenous fat that occur with obstructed bronchi and disorders of fat metabolism.

Though usually caused by mineral oils, taken as nose drops, sprays, or laxatives, these lesions can be caused by animal and vegetable oils, even from those formed by a dermoid cyst leaking into a bronchus (Brown, 1950). The lesion in its tumour-like state occurs in adult life and usually in fit people. The patient may have associated neurological disorders, especially Parkinsonism, rheumatoid arthritis, or motor disorders of the oesophagus, especially cardiospasm.

DIAGNOSIS

Though symptoms can be minimal, paraffinomas often simulate virus pneumonia with general symptoms such as lassitude, malaise, chills, and fever and local symptoms of shortness of breath, cough, sputum, haemoptysis, and chest pain. Occasionally the lesion has been detected by routine chest films.

Routine chest investigations are not usually helpful or conclusive in making the diagnosis. A history of taking oil in some form can be helpful. If the diagnosis is made, an operation may be avoided, or a lesser resection be undertaken. Included in methods of investigation are the following:

1. sputum—for lipophages or free lipoid;
2. bronchoscopic aspiration from the lung—lipoid-containing macrophages;

3. swallowing of radiographic oil (Lipiodol) to determine if this enters the lung.

The purpose of this paper is to describe two such patients, each treated by lung resection, and both thereafter observed over a 10-year period, and it also serves to remind those treating lung disorders that such lesions can still occur.

CASE REPORTS

CASE 1 H.A. (No. 40622), aged 55, a law clerk, was admitted on 29 April 1961. He complained of five years' lassitude, worse for the past year, cough for a year, and three months' wheeze and yellow sputum. He had aching left chest pain unrelated to breathing or coughing. He had no shortness of breath nor blood staining of the sputum. He had lost one stone (6.3 kg) in weight to 7 st 12 lb (49.9 kg).

He had had rheumatoid arthritis for five years, and twice suffered from nasal sinusitis 15 and 4 years previously, receiving treatment from his doctor. He had smoked 3 oz tobacco a week since the age of 18 years.

Physically there was reduced air entry at the base of the left lung and a few scattered rhonchi.

Investigations Simple biplane chest films in March 1961 revealed two rounded lesions in the lower lobe of the left lung (Fig. 1). Bronchography showed that the basal segments were displaced by these rounded opacities. Tomography revealed that there were at least five such lesions in the left lower lobe. Intravenous pyelography and barium examination of the alimentary canal revealed no primary focus for a possible undetected neoplasm. A mass miniature chest film four years earlier (1957) was normal.

Blood count, sedimentation rate, blood urea, acid phosphatase, alkaline phosphatase, Wasserman re-

action, Kahn and hydatid complement fixation tests were normal. The sputum grew Gram-positive diplococci. Cytological examination of the sputum showed no abnormality.

Bronchoscopy revealed a normal bronchial tree. Sigmoidoscopy showed no rectal lesion as a possible primary focus.

Diagnosis At that time it was considered that the patient had secondary deposits in the left lung without in any way proving this or locating the 'primary focus'. He was discharged for observation on 12 May 1961. One lesion later cavitated (Fig. 2).

He was readmitted on 27 September 1961. Ten days previously he had had severe left-sided chest pain and his practitioner noted signs of bronchopneumonia localized to the lower lobe of the left lung (Fig. 3). At the time of readmission his pneumonic flare had resolved, but the five shadows isolated in the left lower lobe remained. He had general emphysema. Another bronchoscopy showed no evidence of neoplastic obstruction to the left lower lobe bronchus. However, 'atypical cells' were now recovered from the sputum.

Lung function tests The following results were obtained:

Predicted vital capacity 3,950 ml

	Before isoprenaline	After isoprenaline
Vital capacity (VC)	3,300 ml	3,350 ml
Forced expired volume (1 sec)	1,860	2,000
Forced vital capacity	2,800	2,880
% VC exp. (1 sec)	57	60
Insp. vol. (1 sec)	2,910	3,340
% VC insp. (1 sec)	89	99
Maximum breathing capacity	63 l.	68 l.

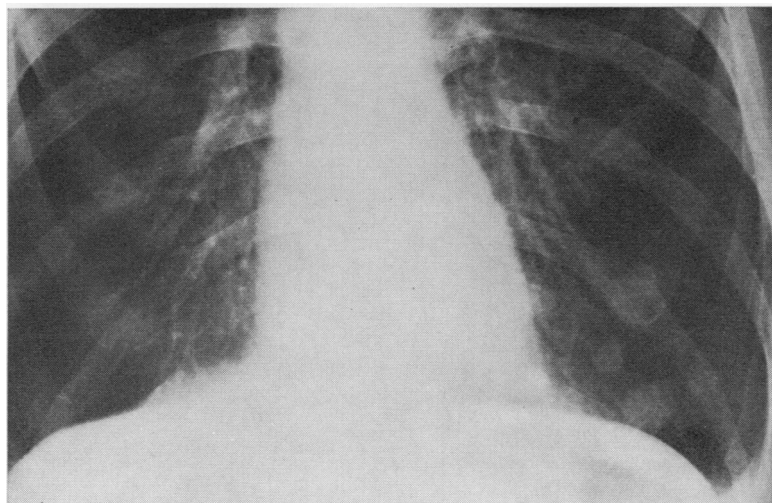


FIG. 1. Case 1. Postero-anterior chest film in March 1961 shows multiple round lesions in the lower lobe of the left lung.

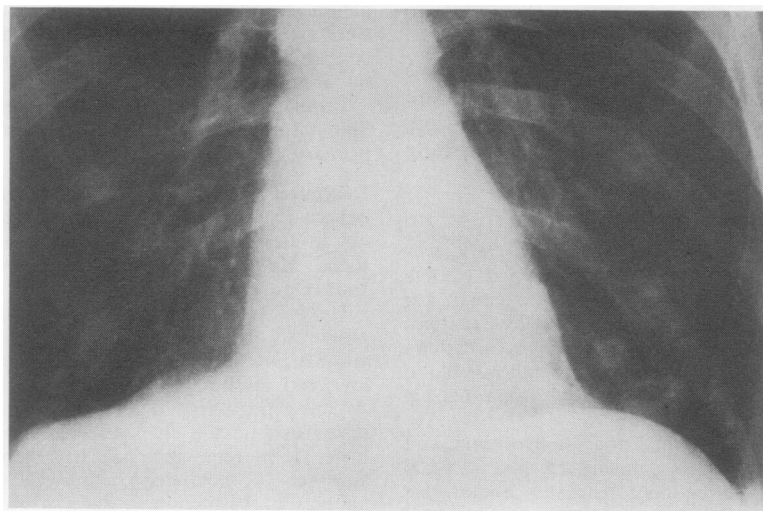


FIG. 2. *Case 1. One lesion cavitated three months later (June 1961).*

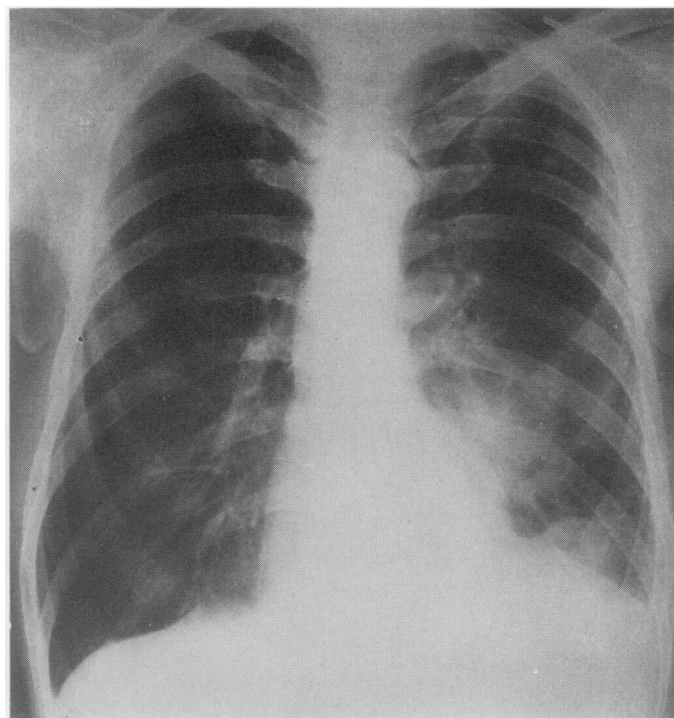


FIG. 3. *Case 1. Postero-anterior chest film in Sept. 1961 shows lesions in the left lower lobe and also towards the left lung hilum, simulating lung cancer.*

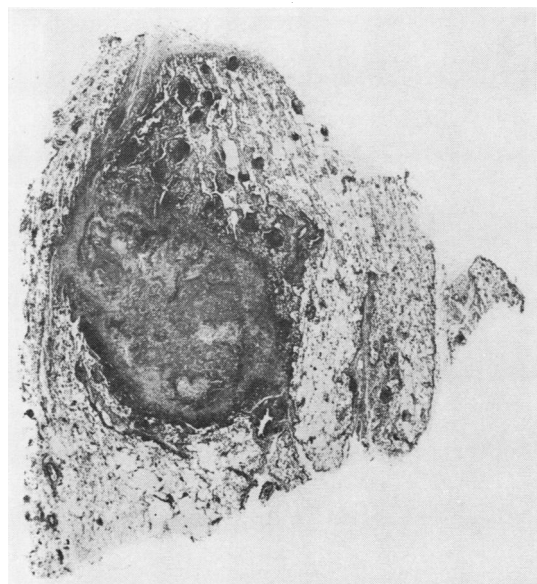


FIG. 4. Case 1. Slice through one of the resected nodules showing it completely replacing normal lung tissue ($\times 4.5$).

Opinion In the absence of any previous primary neoplasm and, in order to confirm the diagnosis and remove a localized focus that had caused him severe chest pain and continued symptoms, left lower lobectomy was finally advised.

Operation A left fifth intercostal thoracotomy was performed on 9 October 1961. Adhesions from a pleurisy affecting both lobes were divided. Superficially both lobes contained small bullous emphysematous cysts. Five discrete nodules were found in the substance of the posterior segment of the left lower lobe, extending into the medial basal segments. No other nodules were palpated in the lung parenchyma. Because of the localized nature of the lesion standard dissection lobectomy was performed. The patient made an uncomplicated recovery and was discharged fit on 23 October 1961.

Pathology report (No. 61B2458) 'The specimen consists of the pulmonary left lower lobe, in the posterior basal segment of which are five discrete nodules measuring up to 1 cm in diameter. The nodules are firm and uniformly whitish in colour (Fig. 4).

'Histologically (Figs 5 and 6) the nodules comprise irregular, loosely arranged zones of fibrous connective tissue enclosing partly necrotic material and small strands of collagen fibres. Outside the fibrous zone

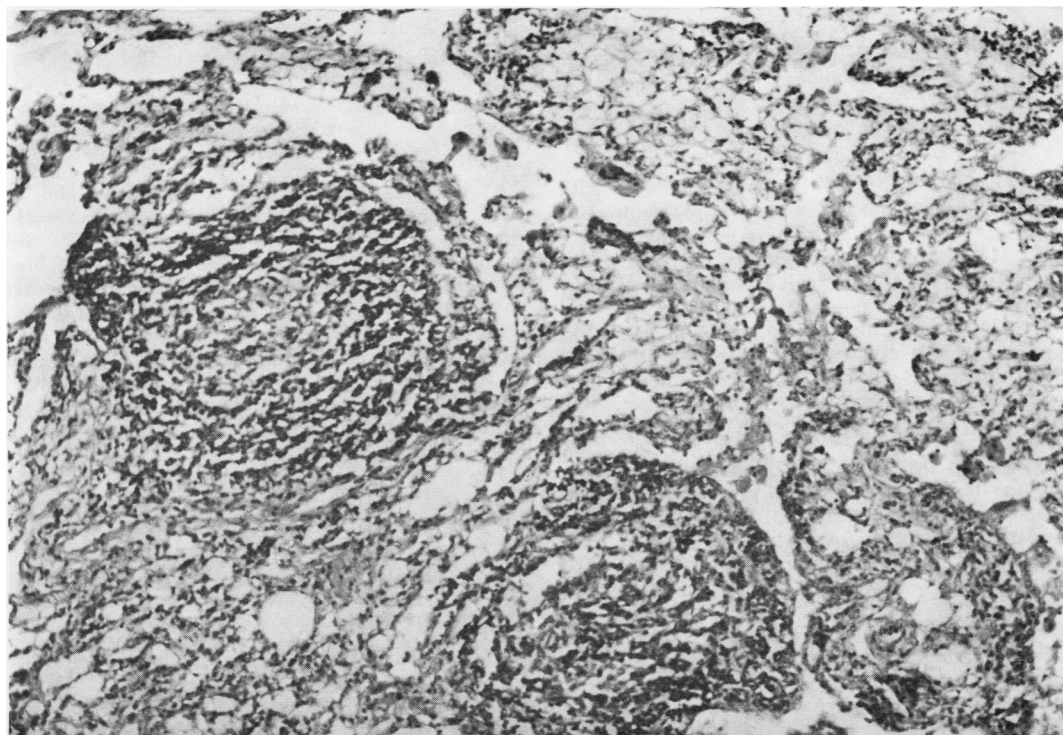


FIG. 5. Case 1. Photomicrograph showing disorganized lung parenchyma with abundant vacuolated lipid material in association with chronic inflammation and lymphoid hyperplasia (H. and E. $\times 90$).

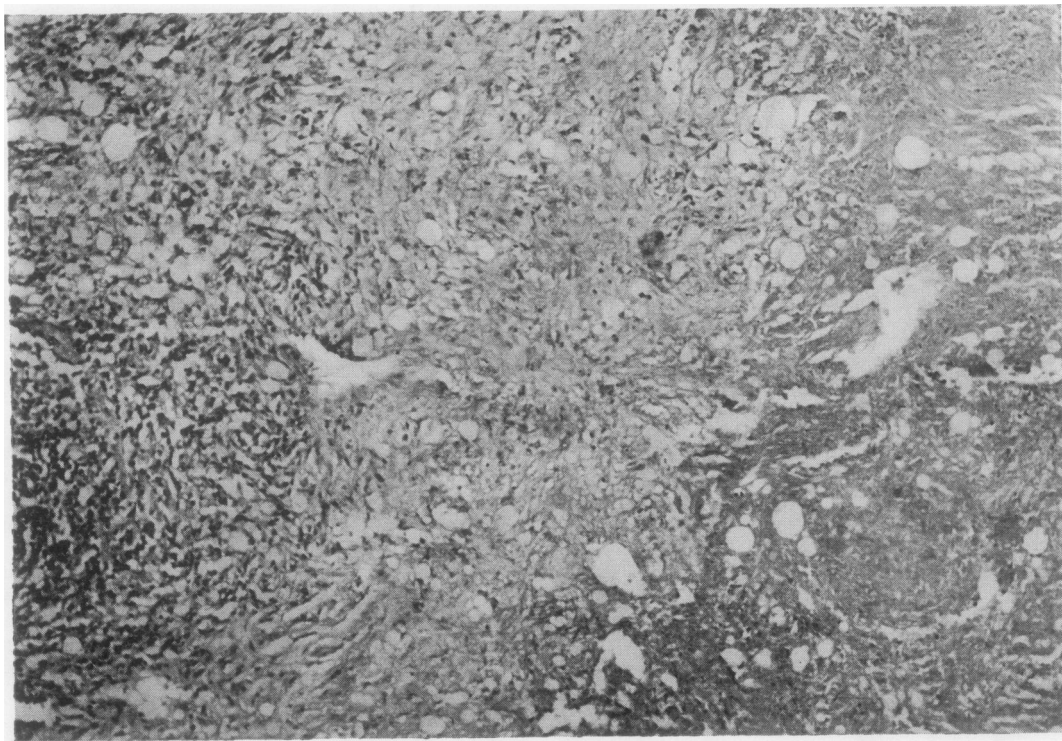


FIG. 6. Case 1. Photomicrograph showing edge of lesion with necrotic vacuolated material adjacent to dense fibrous tissue and zone of chronic inflammation (H. and E. $\times 90$).

there are many scattered lymphoid follicles with germinal centres, and a compressed pulmonary parenchyma and alveoli containing numerous macrophages. Most of the macrophages contain clear globular material. Small bronchi at the periphery show a normal epithelium and masses of surrounding lymphocytes. Endarteritis obliterans is present. Fat staining shows abundant lipid material within the nodules and in the surrounding macrophages. There is no evidence of tuberculosis. The appearances are those of chronic lipid granulomata (paraffinoma).'

Comment In the light of these findings, close questioning revealed that over the past few years the patient had on several occasions used oily nasal drops, which were presumed to be the basis of his oil granuloma.

The patient has now been followed annually for 10 years. He has no further episodes of lung symptoms. His chest films have remained clear. His rheumatoid arthritis required surgical relief in 1970, and at this time there were no associated anaesthetic difficulties nor evidence of inadequate lung function.

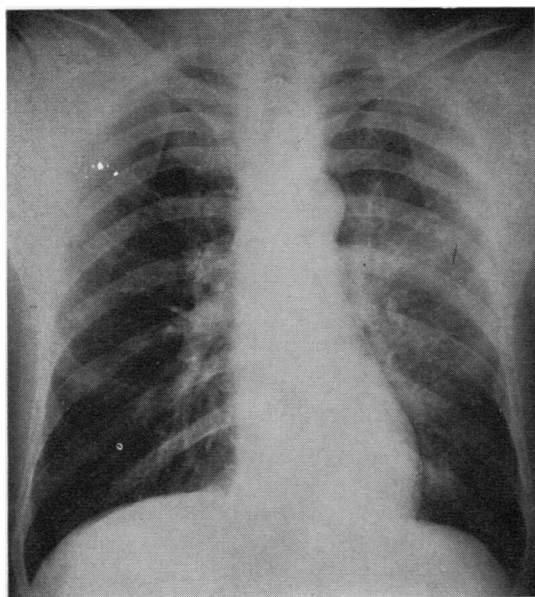
CASE 2 G.F.S. (No. 45934) aged 53 years, a joiner, was admitted on 5 December 1961 for further investigation of an 11-week pain in the left side of

the chest and nine weeks' haemoptysis. The pain followed an attack of 'influenza'; within a week it was aggravated to 'virus pneumonia' and a week later he began coughing up small amounts of blood. This pneumonia lasted seven weeks with increasing haemoptysis. On 29 and 30 November the haemoptyses were more severe, especially on rising in the morning when he coughed up blood clots. He also noticed wheeze and some shortness of breath on effort. Over the preceding three months he had been lethargic, but his weight had remained steady at 10½ st (66.8 kg).

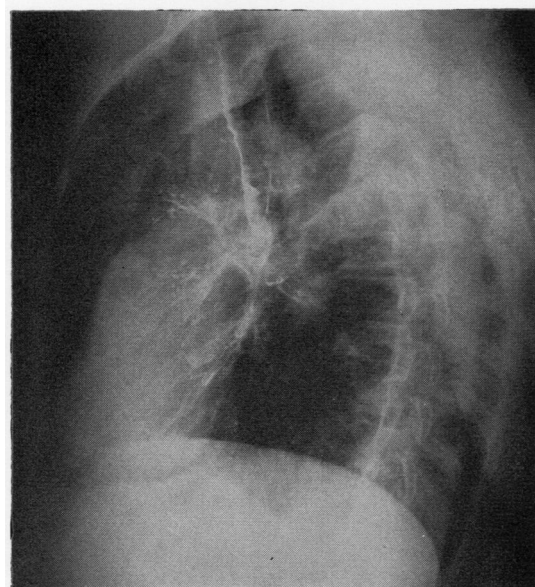
He gave a long history of sinusitis. In 1936 he was in a sanatorium for several months with suspected tuberculosis (one sister died from tuberculosis). He had been smoking up to 50 cigarettes a day since the age of 16 years.

Physically, apart from some vesicular breathing over the apical segment of the left lower lobe, there was no significant finding.

Investigations Chest films showed an ill-defined mass lying in the apical segment of the left lower lobe, the appearances being suspicious of neoplasm (Fig. 7). Cytological examination of the sputum showed no atypical cells. Culture revealed no pathogens. His blood count was normal. Bronchoscopy



(a)



(b)

FIG. 7. Case 2. Postero-anterior (a) and left lateral (b) chest films showing shadowing in the apical segment of the left lower lobe simulating neoplasm.

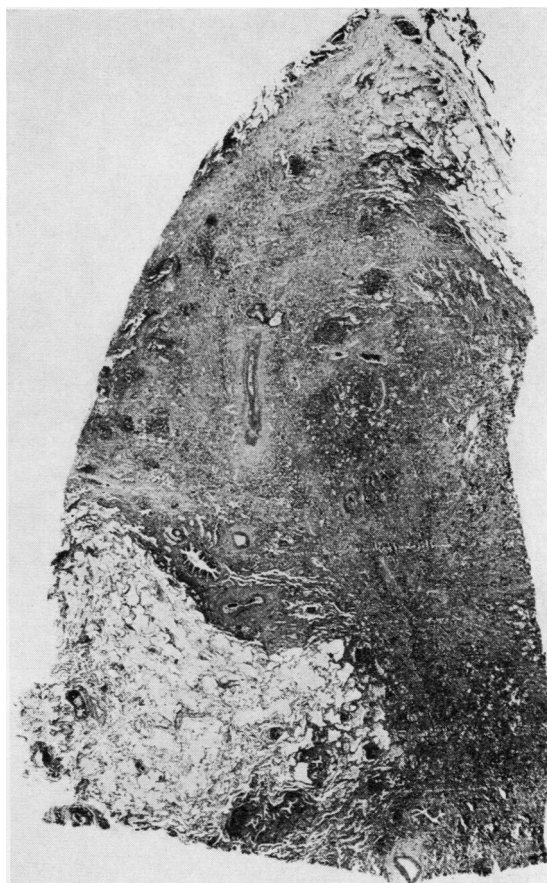


FIG. 8. Case 2. Slice through the resected apical segment of the left lower lobe shows the solid material interspersed with fibrous tissue and replacing lung tissue ($\times 8$).

revealed narrowing of the apical bronchus to the left lower lobe, but no neoplasm was found on sectioning a biopsy. He was given a further trial of chemotherapy for 14 days without any change in his symptoms or radiographic shadowing.

Operation In the belief that the lesion was a neoplasm, a left thoracotomy was performed on 28 December 1961. Adhesions over the apical segment were divided in the extrapleural plane. By palpation and general examination the disease appeared to be 'tuberculous' in nature, and therefore local resection of the apical segment was performed by dissection technique. The patient made an excellent recovery from his operation and was discharged on 20 January 1962.

Pathology report (No. 61B3200) 'The specimen consists of a portion of lung $5 \times 3 \times 3$ cm. On slicing, solid yellow material interspersed with fibrous tissue is seen replacing the lung substance (Fig. 8).

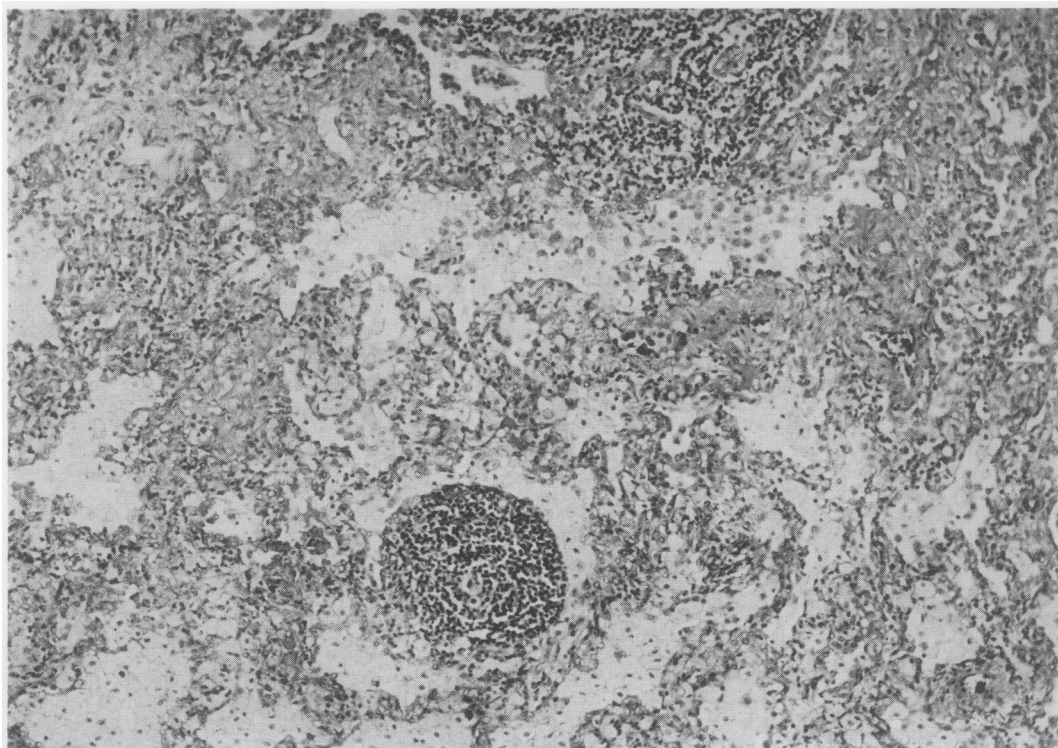


FIG. 9. *Case 2. Photomicrograph showing thickened alveolar walls, chronic inflammation, and prominent lymphoid aggregation. The alveolar spaces contain numerous foamy macrophages (H. and E. $\times 90$).*

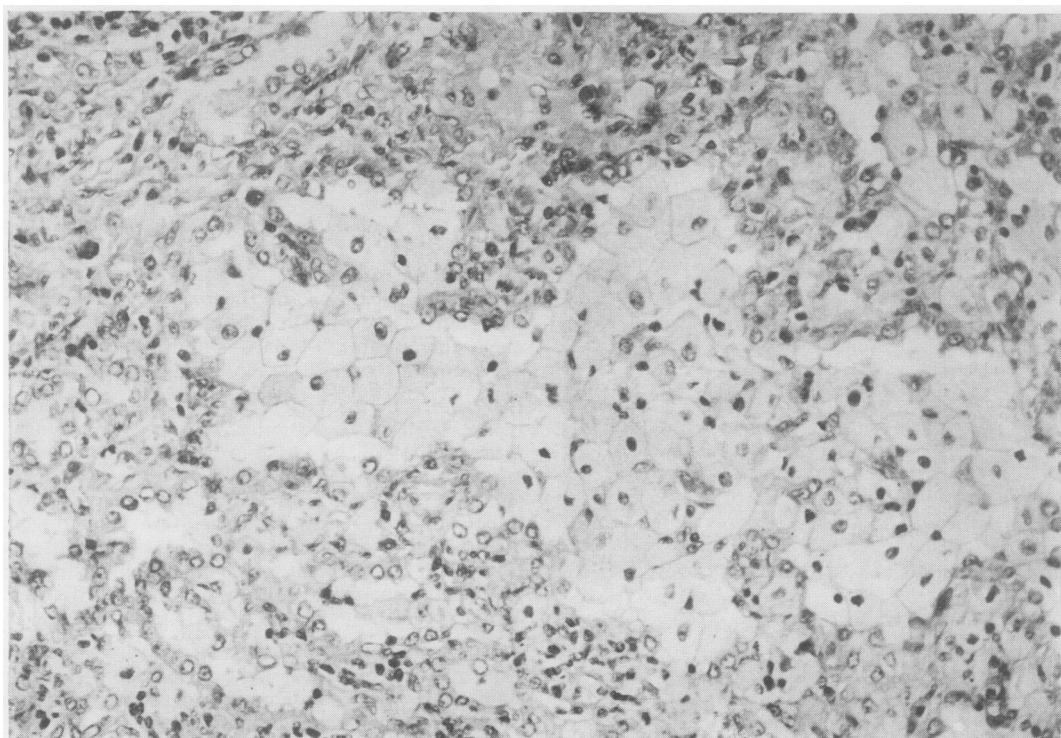


FIG. 10. *Case 2. Photomicrograph showing detail of vacuolated macrophages filling air spaces. Adjacent alveolar walls are thickened and hypercellular (H. and E. $\times 255$).*

'Histologically (Figs 9 and 10) the parenchyma of the lung has almost completely lost its normal architecture, the alveolar walls being markedly thickened by a chronic inflammatory tissue comprising macrophages, fibroblasts, lymphocytes, and plasma cells. Irregular bands of collagen run through this tissue and are also seen within some of the remaining alveolar spaces. There are many foci of lymphocytes forming well-marked follicles and in some places there are several small foci of acute inflammatory exudate. Multinucleated giant cells of foreign body type are common, lying singly and in groups. Almost all the alveoli and remaining alveolar spaces are packed by large foamy macrophages.

'Fat staining of frozen sections shows that these cells contain lipid material, which also lies throughout the tissue in large quantities. No acid-fast organisms are demonstrated. The vessels show marked changes of obliterating endarteritis and the degenerative changes spread through the muscular layers of the walls. Many of the alveoli and terminal bronchioles have become epithelialized with "tumourlet" formation. The appearances are consistent with those of a *lipoid pneumonia*.'

Careful questioning revealed that he had treated his nasal sinusitis with 'Rawleigh's nose drops', which then contained an oily base.

Follow-up The patient has been seen annually since then, and has from a chest viewpoint remained fit, well, and radiologically clear.

CONCLUSION

It is important to remember that lipoid pneumonia occurs in both young and old ; when localized it

produces tumour-like lesions (paraffinoma). It is possible to detect the nature of such a lesion by sputum examination, searching for lipophages or lipid droplets with fat staining of the sputum ; when localized the lesion simulates a neoplasm and is amenable to local resection.

In the patients described the onset of paraffinomas appeared to be related to prolonged treatment of nasal sinusitis with oily nose drops.

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