

Exogenous lipid pneumonia: a retrospective multicentre study of 44 cases in France

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ABSTRACT: A nationwide retrospective study of exogenous lipid pneumonia (ELP) was carried out to update the data on this disease, with emphasis on thoracic computed tomography (CT) scan and bronchoalveolar lavage (BAL) findings.

The inclusion criteria were: 1) presence of abnormal imaging features compatible with the diagnosis of ELP; 2) presence of intrapulmonary lipids; and 3) exogenous origin of the lipid pneumonia.

Forty four cases were included (20 males and 24 females; mean age 62±11 yrs), of which four were occupational (chronic inhalation of cutting mist or oily vapour in an industrial environment). Thirty of the 40 nonoccupational cases were related to aspiration of liquid paraffin used for the treatment of constipation. A condition possibly favouring oil aspiration or inhalation was present in 34 patients (77%), most commonly gastro-oesophageal reflux (n=20) and neurological or psychiatric illness (n=14). Fever (39%), weight loss (34%), cough (64%), dyspnoea (50%) and crepitations (45%) were the most frequent symptoms. BAL was performed in 39 cases: 23% had a lymphocytic alveolitis; 14% neutrophilic alveolitis; and 31% a mixed alveolitis (lymphocytic and neutrophilic). Alveolar consolidations (57%), ground glass opacities (39%), and alveolar nodules (23%) were the most common radiological abnormalities. The changes were bilateral (79%), predominant in the posterior and lower zones of the lobes concerned (74%), hypodense (71%), and spared the subpleural zones (52%). In 13 cases, hypodensity was retrospectively established on CT scan by the presence of a "positive angiogram". This sign may be of diagnostic value when the density measurement is either not possible or not reliable.

In conclusion, this study provides an update of the clinical, biological and radiological profile of exogenous lipid pneumonia and, in particular, confirms the diagnostic benefit of computed tomography scan, which revealed bilateral and hypodense changes in a large majority of cases.

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Exogenous lipid pneumonia (ELP) is an uncommon condition resulting from the aspiration or inhalation of fatlike material of animal, vegetable or mineral origin. Most cases of ELP occur from mineral oil, a mixture of inert, long-chain, saturated hydrocarbons obtained from petroleum. Mineral oils can inhibit the cough reflex and ciliary motility, thus facilitating inhalation. Their presence in the pulmonary parenchyma is responsible for a foreign body type of inflammatory reaction [1–3]. The aspiration of milk, olive oil, cod liver oil, and especially paraffin oil, used as a laxative or less frequently in nasal drops, has long been known to be a cause of ELP. Occasionally, the disease has been caused by using poppy seed oil as a bronchography medium. Numerous other conditions, recently reviewed [4] are also possible. Mineral oils, used in industry as lubricants and cutting fluids in either fluid or spray form, for turning, milling and grinding operations [5], are another major cause of ELP.

ELP must be distinguished from "endogenous" lipid pneumonia, sometimes called "cholesterol" pneumonia, which usually develops when lipids that normally reside

in the lung tissue escape from destroyed alveolar cell walls (e.g. obstructive airway lesion, suppurative process) [5]. Endogenous lipids can sometimes appear in the lung with fat emboli, pulmonary alveolar proteinosis, and lipid storage disorders [6]. Medical history, microscopic findings and fat stains can distinguish ELP from endogenous lipid pneumonia [4].

The exact frequency of ELP is difficult to establish. In autopsy series, it varies from 1 to 2.5% [7]. In 1976, ROUFFY *et al.* [7] analysed 81 cases published from 1941 to 1975. We decided to conduct a national retrospective study in order to update the data concerning this disease, including thoracic computed tomography (CT) scan and bronchoalveolar lavage (BAL).

Methods

Cases were collected in a national retrospective survey conducted between August 1992 and June 1993. A questionnaire was sent to French respiratory medicine departments (210 letters). Participants were to return the

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questionnaire along with histological and radiological documents (chest radiograph, anteroposterior (AP) and lateral, thoracic CT scan).

The inclusion criteria for cases of ELP were as follows, according to SPICKARD and HIRSCHMANN [4]: 1) presence of an abnormal pulmonary image, compatible with the diagnosis of ELP according to data in the literature; 2) presence of intrapulmonary lipids, proven by histological examination or BAL (presence of optically empty vacuoles, either free or inside macrophages, suggestive of lipid vacuoles, possibly confirmed by a specific fat stain); and 3) evidence of the exogenous origin of the lipid pneumonia, provided either by clinical interview or by identification of oil by biochemical analysis (thin layer silica gel chromatography or infrared spectrophotometry). If evidence of exposure was not found, the diagnosis was retained in the absence of any clinical, laboratory or radiological features of endogenous lipid pneumonia.

Radiological documents were interpreted independently by two readers: a respiratory physician (JCD) and a radiologist (PhM).

Radiological images were described using the terminology recommended by the "Société Française d'Imagerie Thoracique" and the "Club Thorax" [8], and by the nomenclature committee of the Fleischner Society [9].

Results

Population

Forty four cases in which diagnosis of ELP was established between 1981 and 1993 were included: 24 females and 20 males, mean age 62 ± 11 yrs (range 36–87 yrs). The diagnosis of lipid pneumonia was based on BAL in 18 cases, pulmonary histology or cytology in 16 cases, and a combination of the two methods in 10 cases. Proof of the exogenous origin was obtained by clinical interview in 36 cases, and by formal identification (chromatography or spectrophotometry) in seven cases. In one case, the origin of the oil was not elucidated, but there were no arguments in favour of endogenous lipid pneumonia.

Aetiological circumstances

The majority of cases were not related to occupational exposure (table 1). The commonest aetiology was the administration of liquid paraffin for the treatment of chronic constipation. Liquid paraffin was aspirated either spontaneously or was facilitated by the presence of gastro-oesophageal reflux, a psychiatric illness or a dose at bedtime. The duration of exposure was generally long (mean 9.5 yrs), but sometimes brief (one case after 1 month of treatment with liquid paraffin). There were four occupational cases due to chronic inhalation of cutting mist or oily vapour either in a metallurgic plant (three cases) or a cable factory (one case).

Clinical presentation and concomitant diseases

The commonest symptoms were fever (39%), weight loss (34%), cough (64%), dyspnoea (50%), and crackles

Table 1. – Aetiological circumstances

	n	%
Occupational	4	9
Nonoccupational	40	91
Conditions responsible for oil consumption		
Constipation	32	80
Rhinitis (nasal instillation)	7	18
Amateur singer (buccal instillation)	1	3
Factors predisposing to oil consumption		
Gastro-oesophageal reflux	20	50
Psychiatric illness	14	38
Administration at bedtime	13	33
Neurological disease	3	8
ENT disease	2	5
Other*	2	5
None	10	25
Nature of oil		
Liquid paraffin	30	75
Vaseline	5	13
Other	5	13

*: achalasia (one case) and oesophageal diverticulum (one case) without proven reflux. ENT: ear, nose and throat.

(45%) (table 2). Three cases were associated with primary lung cancers (two squamous carcinomas and one adenocarcinoma) arising in smokers and located away from the site of lipid pneumonia. Six patients had superinfections, due to common organisms in four cases and to mycobacteria in two cases.

Laboratory tests

The erythrocyte sedimentation rate (measured in 36 cases) was greater than $20 \text{ mm} \cdot \text{h}^{-1}$ in 61% of cases. Leucocytosis ($>10 \times 10^9 \text{ cells} \cdot \text{L}^{-1}$) was present in 22% of cases, with a predominance of neutrophil polymorphonuclear cells in all cases, and it was associated with bronchial infections in three cases.

Lung function tests

Twenty four patients underwent lung function tests. A restrictive ventilatory syndrome (total lung capacity (TLC) $<80\%$ predicted) was observed in 39% of cases, an obstructive ventilatory syndrome (forced expiratory

Table 2. – Clinical presentation

	n	%
Circumstances of discovery		
Incidental	18	41
Symptoms	26	59
General signs	26	59
Fever	17	39
Weight loss	15	34
Pulmonary symptoms	33	75
Cough	28	64
Dyspnoea	22	50
Sputum	12	28
Chest pain	9	21
Haemoptysis	6	13
Physical signs	27	62
Crackles	20	45
Bronchial rales	3	7
Finger clubbing	1	3

Table 3. – Imaging features

	n	%
Type of images (n=44)		
Alveolar consolidations	25	57
Ground glass opacities	17	39
Alveolar nodules	10	23
Pseudotumoral consolidations	6	14
Linear opacities	6	14
Mediastinal adenopathies	4	9
Cavitary images	2	5
Pleural effusions	2	5
Sites of opacities (n=38)*		
Bilateral involvement	30	79
Right lung	34	89
Upper lobe	15	39
Middle lobe	31	89
Lower lobe	31	82
Left lung	36	95
Superior segment of upper lobe	8	21
Lingula	18	47
Lower lobe	32	85
CT scan appearances (n=31)**		
Declivity	23	74
Low density (CT angiogram sign)	22(13)	71
Sparing of the periphery	16	52
Retractile nature	7	23

*: six cases only had an anteroposterior (AP) chest radiograph;
 **: the nature of images was evaluated on 31 CT scans. CT: computed tomography; declivity: predominant in the posterior and lower zones of the lobes concerned.

volume in one second/vital capacity (FEV₁/VC) <75% in 28% of cases, and a mixed syndrome in 8% of cases. The transfer factor for carbon monoxide, determined in 16 cases, was less than 80% of predicted in 63% of cases.

Imaging features

Forty four standard chest radiographs and 31 CT scans considered to be of satisfactory quality were analysed (table 3). The site of the images was assessed in 38 cases with an AP and lateral chest radiograph. Representative examples of the predominant features are shown in figures 1–6. Alveolar consolidations (figs. 1, 3 and 6) and

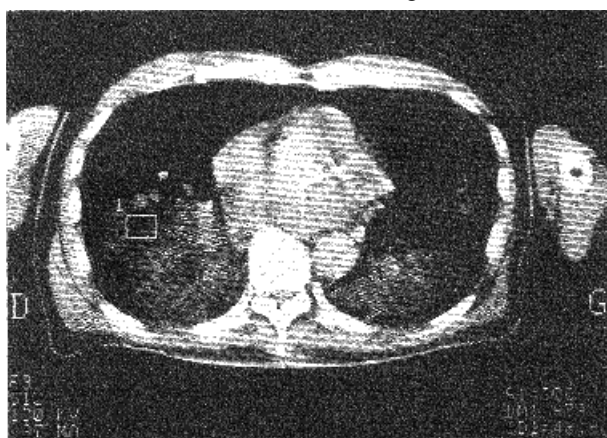


Fig. 1. – Declivitous (*i.e.* predominant in the posterior and lower zones of the lobes concerned), bilateral alveolar consolidations. Hypodensity measured at -79 Hounsfield units and observed by comparison with subcutaneous adipose tissue. Sparing of the periphery.

ground glass opacities (fig. 2) were the most frequent images. In 30 out of 38 cases, the images were bilateral, with a marked predominance for the lower lobes. Declivity (*i.e.* predominance in posterior and lower zones of the lobes concerned) (fig. 4) and hypodensity were the usual features. Hypodense images were assessed by density measurements (in Hounsfield units (HU)), or by visual comparison with subcutaneous adipose tissue (fig. 1), or by the "angiogram sign" (figs. 3, 5 and 6), on parenchymal window setting. The latter sign consists of visualization of normally branching pulmonary vessels



Fig. 2. – Bilateral ground glass opacities. Sparing of the periphery.

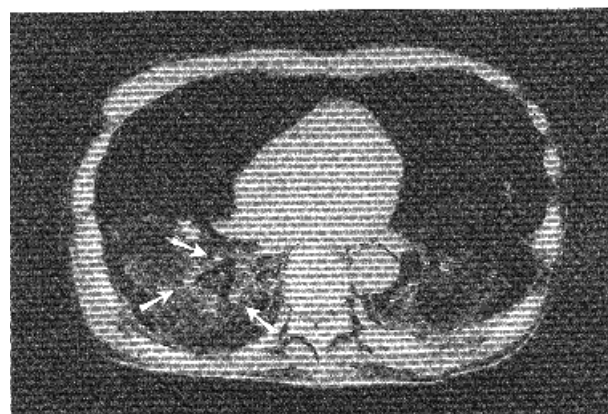


Fig. 3. – Bilateral alveolar consolidations. On the right, the air bronchogram is surrounded by three cross-sectioned angiograms (arrows).

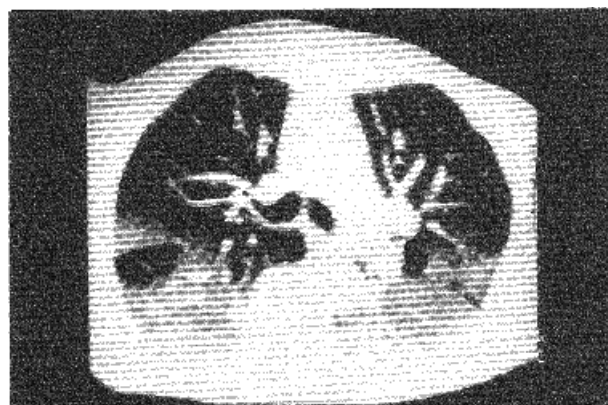


Fig. 4. – Declivitous (*i.e.* predominant in the posterior and lower zones of the lobes concerned), bilateral images. Zone of "spared healthy lung" located under the right major fissure ("sandwich aspect").

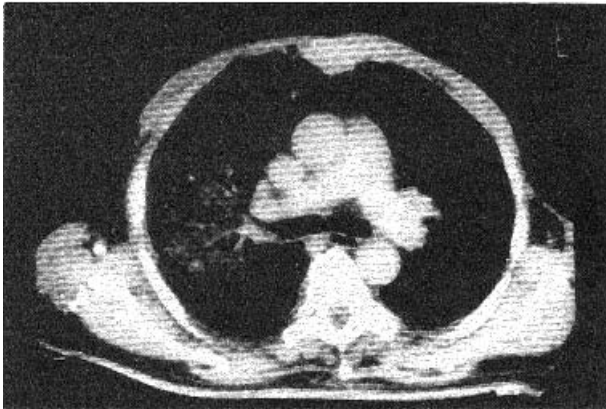


Fig. 5. – Longitudinal sectioned angiogram inside a conglomerate of alveolar nodules.

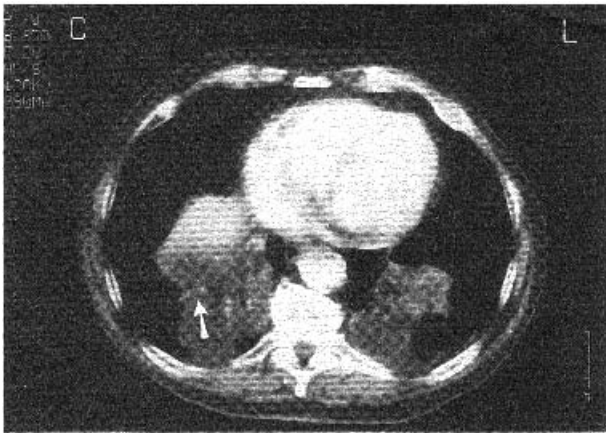


Fig. 6. – Bilateral alveolar consolidations. Normally branching angiogram (arrow). Several bilateral cross-sectioned angiograms.

within the consolidated lung on postcontrast CT. It was present in 13 cases, among which eight were non-contrast enhanced. In seven cases, the declivity of the images gave a "sandwich" appearance with a subfissural zone of healthy lung between two pathological zones (fig. 4). One of the two cavitory images was probably due to an atypical mycobacterial infection. In one case, a mediastinal adenopathy presented a very low density and was therefore, probably related to lipid pneumonia.

The four occupational cases consisted of diffuse interstitial forms, which were not predominant in the lower lobes: reticulonodular pattern (one case); ground glass appearance (one case); nodular pattern (one case); nodular and ground glass (one case).

Fibreoptic bronchoscopy and BAL

The BAL fluid appearance was abnormal (milky, oily or haemorrhagic) in 44% of cases (table 4). A lymphocytic alveolitis (defined by more than 20% lymphocytes at differential cell count) was present in 23% of cases. Neutrophilic alveolitis (defined by more than 5% neutrophils at differential cell count) was observed in 14% of cases. The alveolitis was mixed, both lymphocytic and neutrophilic, in 31% of cases. Finally, in two cases, BAL eosinophilia was present (with 10 and 36% of eosinophils, respectively).

Table 4. – Endobronchial aspect and principal characteristics of the BAL

	Number	%
Endobronchial aspect		
Number of endoscopic examinations performed	43	
Diffuse inflammatory aspect	8	19
Localized inflammatory aspect	3	3
Presence of secretions	5	12
Diffuse telangiectasias	1	2
Normal	24	56
Not specified	2	
Characteristics of BAL		
Number of BAL performed	39	
Aspect		
Oily or milky	11	28
Haemorrhagic	6	15
Normal or not specified	22	57
Cell count (n=35)		
Number of cells·mm ⁻³ *		324±312
Percentage of lymphocytes*		22±16
Lymphocytes >20%		54
Percentage of neutrophils*		14±19
Neutrophils >5%		45

*: mean±SD. BAL: bronchoalveolar lavage.

Thirty nine of the 44 patients underwent a BAL: lipophages with a characteristic appearance made diagnosis possible in 30 cases. Diagnosis was completed in 15 of these cases by specific fat stains, which were positive in all cases (either sudan black, sudan red or oil red O), and in seven cases by identifying the oil by spectrophotometry or chromatography, which in each case made it possible to show that the oil inhaled was the same as the intrapulmonary oil (paraffin in four cases, vaseline in one case, and cutting mist in two cases). This diagnosis was supported in 15 of the 30 cases with characteristic appearance on BAL by a histological examination. Of the nine other cases, whose BAL was not diagnostic, transbronchial biopsies were performed in eight patients and were positive in seven cases. In one case, a negative biopsy led to thoracotomy; a thoracotomy was the initial diagnostic procedure in one case. Five patients did not undergo BAL. In these five patients, diagnosis was reached by thoracotomy in four cases and transparietal puncture in one, but transbronchial biopsies had not been performed beforehand.

Histological features

Twenty four cases were submitted to histological examination: transbronchial biopsy in 14 cases, open lung biopsy in eight cases, thoracoscopic lung biopsy in one case, and bronchial biopsy in one case. The predominant lesion was fibrosis (14 cases), which was either exclusively interstitial (10 cases) or "mutilating" with a disorganization of the pulmonary architecture (four cases). The fibrotic tissue was infiltrated by optically empty vacuoles corresponding to lipids, which were both alveolar and interstitial, free and within macrophages. An inflammatory reaction was detected in 16 cases, with the appearance of a giant cell granuloma in 13 cases.

Treatment and clinical course

Oil exposure was stopped in 37 cases. The therapeutic approaches included treatment of the predisposing factor leading to aspiration (usually gastro-oesophageal reflux) in 13 cases, corticosteroid therapy in six cases, therapeutic lung lavage in six cases, and in the other cases, treatments such as long-term oxygen therapy in a patient who had progressed to the stage of chronic respiratory failure, immediate resection of the lesion, secondary resection as a result of recurrent superinfection, and immunosuppressants following a course of corticosteroids.

The clinical and functional course was specified in 28 cases. The following outcomes were observed: 13 improvements (46%); seven stable courses (25%); six deteriorations or complications (21%) (fibrosis, repeated episodes of infection in the territory of the lipid pneumonia, *Aspergillus* infestation with parenchymal excavation).

There were two deaths unrelated to the lipid pneumonia. Imaging follow-up was available for 21 cases and showed: three complete cures (14%); six improvements (29%); 10 stable courses (48%); and two deteriorations (10%). In the 32 cases in which the intoxication was discontinued and the course known, five patients deteriorated (despite corticosteroid therapy in one case), whilst the other 27 patients remained stable, improved, or were even cured, regardless of concomitant treatment (corticosteroids, therapeutic BAL or treatment of predisposing factors).

Discussion

The commonest aetiological circumstance was administration of liquid paraffin for the treatment of refractory constipation. The predominance of this aetiology is frequently reported in the literature but used to be less marked, and, in some cases, was of similar importance to nasal instillation of oily products for chronic rhinopharyngeal diseases [2, 7, 10, 11]. The oil responsible was almost always a mineral oil (paraffin or vaseline). Two of the present cases were related to vegetable oils (peanut oil and olive oil included in nose drop formulae) which are reputed to be minimally toxic for the lung; however, one case developed a "paraffinoma", *i.e.* a dense lesion with a pseudotumoral appearance, associated with fibrosis of the pulmonary parenchyma. The long duration of exposure (mean of 9.5 yrs) concurs with other results in the literature [2, 7, 10]. One case occurred after administration of an oily substance for only 1 month. Several similar examples, in which the duration of the exposure ranged 1–3 months, were reported in the analysis of ROUFFY *et al.* [7]. The predisposing factors identified have been reported in several papers [2, 10, 12, 13, 15, 16], but with no indication of their frequency. These predisposing factors are not obligatory for oil to penetrate into the airways, as they were absent in 25% of the present cases.

This series is the first to include an extensive study of CT scans, as most of the other publications reporting large series predate the development of the technology. The most frequent aspects were extensive and bilateral alveolar consolidations and ground glass opacities. It is difficult to compare the present results with those of older

series, in which the terms used to describe the radiological images are sometimes no longer used ("plaques", "marbling"). However, in 1970, GENEUREUX [6] reported a radiological description, which, although not providing precise data concerning the frequency of the various images, corresponded to the impression given by the present series. The author did not mention the ground glass opacities, but this aspect was reported by VOLK *et al.* [10]. The density of parenchymal lesions on CT scan can be measured during the examination, but the lesion would have to be homogeneous, as the presence of air inside the lesions would give falsely negative measurements. A negative density (between -150 and -30 HU) is highly suggestive of the presence of intrapulmonary fat [17–19]. In this series, a low-density lesion was detected in only five cases; in contrast, in 13 cases, the low density was established retrospectively by the presence of a positive angiogram within alveolar consolidations (12 cases) or conglomerates of nodular images (one case) (fig. 5). This sign seems to have an interesting diagnostic value as a complement to density measurement, when this measurement cannot be reliably performed. For IM *et al.* [20] the presence of a CT angiogram sign requires two factors. One is that the consolidated lung should present a considerably lower attenuation than the enhancing vessels, and the other is that the arrangement of vascular trees should not be significantly distorted by the pathological process (fig. 6). ELP fulfils these conditions. This sign has previously been described in lobar bronchioloalveolar carcinoma and may also be present in pulmonary oedema, caused by increased hydrostatic pressure or acute pulmonary infarction [20].

The present results concerning the site of the lesions differ from those reported previously. The radiological images were much more frequently bilateral (79%) and affected left and right lungs almost equally (95 and 89%). They were often multilobar and, in particular, quadri-lobar. In contrast, in the studies by GENEVRIER [14], EVEN *et al.* [11], ROUFFY *et al.* [7], and WRIGHT and JEFFREY [13], lesions were described as being more often unilateral and predominantly affecting the right side, as in classical inhalation pneumonia. This difference may be explained by the fact that CT examinations demonstrated images not visible on chest radiographs. We feel that CT scan, therefore, makes a major contribution to the diagnosis of ELP. It provides an exact assessment of the lesion and, in particular, demonstrates the low density of the images and their declivity. The four occupational cases consisted of diffuse interstitial forms. The sizes of inhaled particulates in these forms are smaller than in the other forms; this might explain the radiological aspect of the pneumopathy.

The appearance of BAL was highly suggestive (oily) in almost 20% of cases and this may immediately suggest the diagnosis. Cytological analysis showed a high mean cellularity in the present series (324 cells·mm⁻³). CORWIN and IRWIN [21] reported a similar finding on BAL of nine subjects, but LAUQUE *et al.* [22] reported normal cellularity. Various cytological profiles have been described in the literature for isolated cases: macrophage alveolitis [23–25]; lymphocytic alveolitis [26–28], and neutrophilic alveolitis [22]. Study of the 35 cases submitted to BAL with cytological analysis provides an idea of the frequency of these cytological profiles: normal (31%

cases); isolated lymphocytic (23%); isolated neutrophilic (14%); and mixed alveolitis (31%). No cytological profile is, therefore, more particularly suggestive of this disease.

Both BAL and transbronchial biopsy contributed significantly to diagnosis. Diagnosis was reached in 30 of the 39 BAL performed (77%). In the nine remaining cases, transbronchial biopsy made diagnosis possible in eight cases, and in one case, negative transbronchial biopsies led to thoracotomy. In the five cases where diagnosis was obtained by thoracotomy or transthoracic needle biopsy (one case), had BAL or transbronchial biopsy been performed beforehand, these invasive techniques could probably have been avoided, notably in view of the above results.

Histological examination revealed two important points: the frequency of the granulomatous form of the inflammatory cell reaction (13 cases) suggesting a foreign body disease; and the frequency of fibrotic lesions (14 cases), which explains the potential severity of the disease with a possibility of progression to fibrosis with disorganization of the pulmonary architecture (four cases) and chronic respiratory failure.

The clinical presentation was not very different from that reported in the literature. The latent nature of the disease is classic: 41% of cases in this series were incidental findings, among which 25% of cases remained effectively latent. Fever (39%), weight loss (34%), cough (64%) and dyspnoea (50%) were the most frequent findings.

The respiratory function characteristics of ELP have seldom been reported. The present series confirms the functional profile reported elsewhere in isolated cases, *i.e.* normal respiratory function or restrictive ventilatory syndrome. Transfer factor for carbon monoxide was measured in 16 cases and was frequently abnormal (63% of cases). Obstructive ventilatory syndrome was also detected (seven cases, including five in smoking patients).

The therapeutic approach most frequently adopted was discontinuation of the oil exposure (37 cases). Both in this series and in the literature, corticosteroids and therapeutic BAL have not been demonstrated to be really effective and cannot be proposed as the treatment for all cases. Surgery is seldom required in the treatment of ELP. One patient underwent a lobectomy to treat recurrent infection in the area of the lipid pneumonia. Finally, the predisposing factors are not always treated (13 cases in this series; whilst gastro-oesophageal reflux, which is amenable to treatment, was present in 20 cases). However, such treatment would appear to be justified, as the advice to stop using the oily substance may not always be followed by patients who are very often dependent on their treatment.

In conclusion, the frequently misleading appearance of exogenous lipid pneumonia with clinical signs (cough, dyspnoea, fever, diaphoresis) and radiological signs (alveolar consolidations, ground glass opacities) can suggest an infectious, neoplastic or immunoallergic disease. It is, therefore, important to consider the possibility of lipid pneumonia, which can be very strongly suspected at computed tomography scan by the presence of bilateral, declivitous, hypodense images, which may be notably detected by the angiogram sign. When a lipid pneumonia is suspected, bronchoalveolar lavage completed by transbronchial

biopsies, leads to diagnosis in the vast majority of cases. Open lung biopsy and even thoracoscopy should therefore become the exception in this disease.

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References

1. Pinkerton H. The reaction to oils and fats in the lung. *Arch Pathol* 1928; 5: 380–401.
2. Ikeda K. Lipoid pneumonia of the adult type. *Arch Pathol* 1937; 23: 470–492.
3. Kaplan L. Combined cod liver oil and liquid petrolatum pneumonia in a child. *Am J Dis Child* 1941; 62: 1217–1223.
4. Spickard A, Hirschmann JV. Exogenous lipid pneumonia. *Arch Intern Med* 1994; 154: 686–692.
5. Woodhead M, Parkes WR. Disorders caused by other organic agents: oil granuloma (lipoid pneumonia). In: Parkes WR, ed. *Occupational Lung Disorders*. Oxford, Butterworth-Heinemann Ltd, 1994; pp. 778–782.
6. Genereux GP. Lipids in the lung: radiologic-pathologic correlation. *J Can Assoc Radiol* 1970; 21: 2–15.
7. Rouffy J, Almosni M, Chelloul N, *et al.* Aspects actuels des lipoidoses pulmonaires exogènes de l'adulte. *Ann Med Interne* 1976; 127: 637–643.
8. Frija J. Semantique des pneumopathies infiltrantes diffuses étudiées par la tomodynamométrie. *Rev Im Med* 1991; 3: 355–361.
9. Tuddenham WJ. Glossary of term for thoracic radiology: recommendations of the nomenclature committee of the Fleischner Society. *AJR* 1984; 143: 509–517.
10. Volk BW, Nathanson L, Losner S, Slade WR, Jacobi M. Incidence of lipid pneumonia in a survey of 389 chronically ill patients. *Am J Med* 1951; 10: 316–324.
11. Even R, Lecoeur J, Sors C. Les pneumopathies huileuses de l'adulte. *Sem Hôp Paris* 1948; 24: 1387–1394.
12. Losner S, Volk BW, Slade WR, Nathanson L, Jacobi M. Diagnosis of lipid pneumonia by examination of sputum. *Am J Clin Pathol* 1950; 20: 539–545.
13. Wright BA, Jeffrey PH. Lipoid pneumonia. *Semin Respir Infect* 1990; 5: 314–321.
14. Genevrièr R, Daussy M, Baveria E, Gacouin JC. Les pneumopathies huileuses de l'adulte: à propos d'une observation à forme pseudo-tumorale. *Sem Hôp Paris* 1972; 48: 2607–2614.
15. Sodeman WA, Stuart BM. Lipoid pneumonia in adults. *Ann Intern Med* 1946; 24: 241–253.
16. Weiler J, Roussel G. Les pneumopathies lipidiques. *France Med* 1963; 26: 5–27.
17. Wheeler PS, Stitik FP, Hutchins GM, Klinefelter HF, Siegelman SS. Diagnosis of lipid pneumonia by computed tomography. *JAMA* 1981; 245: 65–66.
18. Joshi RR, Cholankeril JV. Computed tomography in lipid pneumonia. *J Comput Assist Tomogr* 1985; 9: 211–213.
19. Brechot JM, Buy JN, Laaban JP, Rochemaure J. Computed

- tomography and magnetic resonance findings in lipoid pneumonia. *Thorax* 1991; 46: 738–739.
20. Im JGG, Han MC, Yu EJ, *et al.* Lobar bronchioloalveolar carcinoma: angiogram sign on CT scans. *Radiology* 1990; 176: 749–753.
 21. Corwin RW, Irwin RS. The lipids laden alveolar macrophages as a marker of aspiration in parenchymal lung disease. *Am Rev Respir Dis* 1985; 132: 576–581.
 22. Lauque D, Dongay G, Levadet T, Caratero C, Carles P. Bronchoalveolar lavage in liquid paraffin pneumonitis. *Chest* 1990; 98: 1149–1155.
 23. Breton JL, Ranfaing E, Clement F, Faure E. Pneumopathie lipidique exogène d'origine professionnelle. *Presse Méd* 1990; 19: 1724.
 24. Le Bon B, Praet JP, Mostin M, Chami Y, Sergysels R. Pneumopathie secondaire à l'utilisation abusive de spray dépolluant. *Rev Mal Respir* 1992; 9: 213–215.
 25. Perol M, Vallon C, Vallon JJ, Guerin JC. Pneumopathie lipidique par exposition professionnelle à l'huile de coupe. *Rev Mal Respir* 1989; 6: 271–274.
 26. Ameille J, Le Tinier JY, Reigneau O, Rochemaure J, Proteau J. Exposition professionnelle à l'inhalation de paraffine et pathologie respiratoire. *Arch Mal Prof Med Trav Secur Soc* 1984; 45: 264–265.
 27. Fournier P, Aerts J, Damade R, Pasquier P. Une cause rare de syndrome inflammatoire: la paraffinose pulmonaire. *Presse Méd* 1990; 19: 1768–1769.
 28. Pujol JL, Barneon G, Bousquet J, Michel FB, Godard P. Interstitial pulmonary disease induced by occupational exposure to paraffin. *Chest* 1990; 97: 234–236.