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Exogenous lipoid pneumonia: HRCT, MR, and pathologic findings

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Abstract. The objective of this study was to describe high-resolution CT (HRCT) and MR findings of exogenous lipoid pneumonia and to correlate them with pathologic findings. A retrospective review of the medical records of our institution revealed seven patients with a diagnosis of lipoid pneumonia based on clinical data, chest films, bronchoalveolar lavage, and follow-up. Both HRCT and MR imaging were reviewed by two readers. Pathologic examination of the resected specimen or surgical biopsies were also reviewed in the four available cases. The HRCT findings were pulmonary consolidations ($n = 6$) with fatty ($n = 3$) or unspecific but low attenuation values ($n = 3$), areas of ground-glass opacities ($n = 5$), septal lines, and centrilobular interstitial thickening ($n = 5$). In five of the seven cases, a crazy-paving pattern of various spread was also present, either isolated ($n = 1$) or surrounding a pulmonary consolidation. In two cases traction bronchiectasis and cystic changes consistent with fibrosis were seen. At MR imaging ($n = 2$) a pulmonary consolidation of high signal intensity on T1-weighted image consistent with lipid content was present in one case. Pathologic examination ($n = 4$) showed the coexistence of lobules with lesions of various ages, sometimes in contiguous lobules, within the same patient. Recent lesions were those with alveolar fill-in by spumous macrophages and almost normal alveolar walls and septae. In more advanced lesions, lobules were filled in with larger vacuoles often surrounded by inflammatory infiltrates of alveolar walls, bronchiolar walls, and septa. The oldest lesions were characterized by fibrosis and parenchymal distortion around large lipid-containing vacuoles. The HRCT findings reflect pathologic findings in exogenous lipoid pneumonia. Although non-specific, consolidation areas of low attenuation values and crazy-paving pattern are frequently associated in exogenous lipoid pneumonia and are indicative of the diagnosis.

Key words: Lipoid pneumonia – Diagnosis – Lung – Disease – CT – MRI

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Introduction

Exogenous lipoid pneumonia refers to an uncommon condition resulting from aspiration or inhalation of mineral, animal, or vegetable oils into the lung over an extensive period of time [1, 2, 3]. Chronic aspiration of oil is often undetected during lifetime. Few autopsy cases documenting exogenous lipoid pneumonia at autopsy have reported significant clinical symptoms [4]. Computed tomography and MR imaging findings have been reported in small series. The most often reported and evocative appearance at CT is one or multiple areas of consolidation of low attenuation, with sometimes specific fatty attenuation [5, 6, 7, 8, 9, 10]. The fatty component may also be detectable at MRI [11, 12]. Recently, a distinctive pattern consisting of crazy-paving appearance, similar to the pattern described in alveolar proteinosis, was reported with high-resolution CT (HRCT) [13]. Therefore, it has been suggested that HRCT and MR play a significant role in the diagnosis of lipoid pneumonia. Nevertheless, correlations between imaging and pathologic findings have seldom been reported. The purpose of our study was to review cases of exogenous lipoid pneumonia explored and treated at our institution in recent years in an attempt to correlate HRCT findings with pathologic features.

Materials and methods

Between April 1986 to January 1998, we retrospectively reviewed the clinical and radiographic records of seven patients diagnosed with exogenous lipoid pneumonia. The inclusion criteria were: (a) presence of abnormal imaging features compatible with the diagnosis of lipoid pneumonia; (b) presence of intrapulmonary lipids at pathologic examination; (c) exogenous origin assessed by clinical history; and (d) availability of HRCT, pathologic examination, and follow-up. We identified 7 patients corresponding to these criteria. Chest radiograph and HRCT were available in all cases, whereas MR was only available in two. In four cases exogenous lipoid pneumonia was proven at biopsy by surgical specimen after lobectomy ($n = 2$) and open lung biopsies ($n = 2$). In the remaining patients the diagnosis was made on the basis of clinical, bronchoalveolar lavage results, radiographic findings, and follow-up. Criteria to assess diagnosis were: a compatible clinical history with exogenous origin of the lipid, radiographic findings showing pulmonary consolidations in dependent areas, bronchoalveolar lavage (BAL) showing predominance of lipid-laden macrophages, and stability of symptoms and chest radiograph findings for at least 1 year.

The HRCT technique was performed using a Somatom Plus 4 scanner (Siemens, Erlangen, Germany) in three cases, a DRH scanner (Siemens, Erlangen, Germany) in three others, and a GE 8800 (General Electric, Buck, France) in one case. Scans were obtained with 1-mm slices at 10-mm intervals from the apex to the lung base, or by 1-mm slices performed at selected levels after whole-lung scanning using 7- to 10-mm contiguous slices. Scans were photographed in windows appropriate for pulmonary parenchyma (level –660 to –650 HU, width 1500–1800 HU) and mediastinum (level 50 HU, width 350–400 HU). Measurement of attenuation in areas of consolidation was noted when available.

The MR technique was performed in two cases with a Magnetom at 1 T (Siemens, Erlangen, Germany) with conventional T1-weighted spin-echo and T2-weighted turbo spin-echo sequences. On MR scans signal intensity of pulmonary consolidation was subjectively compared with the normal component, fat, and muscles of the thoracic wall and mediastinum.

Chest radiographs, HRCT scans, and MR examination were reviewed by two readers independently and final evaluation was reached by consensus. The HRCT findings were noted and classified according to the glossary of terms used in HRCT [14].

In the four patients in whom a resected specimen ($n = 2$) was obtained or surgical biopsies ($n = 2$) were performed, the pathologist reviewed the available material in order to describe and date the lesions.

Results

Clinical, HRCT findings, BAL results, treatment chosen, and follow-up of the seven patients are reported in Table 1. The seven patients were three women and four men aged from 40 to 72 (mean age 53 years). All patients had a characteristic history of oil aspiration. For three of them the leading cause was chronic and prolonged use of paraffin in nose drops and for a fourth patient, a singer, it was drops for vocal cords. Exogenous lipoid pneumonia was related to aspiration of liquid paraffin used for the treatment of constipation in the three other patients. Clinical symptoms were absent in five subjects. One patient suffered from an irritative cough for 3 months and another suffered from known rheumatoid arthritis without pulmonary symptoms clinically related to this disease. In all cases chest films showed one or multiple areas of consolidation, with ill-defined irregular boundaries. In patient 6 the suggested diagnosis was lung cancer, despite the low attenuation of the consolidation. He was referred to surgery.

The HRCT findings consisted individually of one or several areas of pulmonary consolidation ($n = 6$), areas of ground-glass opacities ($n = 6$), thickened interlobular septa ($n = 6$), centrilobular interstitial thickening ($n = 5$) and small cystic areas ($n = 1$). Areas of consolidation were isolated in two cases and multiple in five cases. Measurement of attenuation in the most hypodense areas showed typical fatty attenuation in three cases. In three other cases, areas of attenuation were greater than fat and lesser than soft tissue (Fig. 1 a). A CT angiogram sign was detectable within the pulmonary consolidation in six of six cases. In five of six cases the areas of consolidation were centrally located, leaving a peripheral band of normally aerated pulmonary parenchyma (Fig. 1 b). They were surrounded by areas of ground-glass opacities superimposed on thickened septal lines and centrilobular interstitial thickening, with the characteristic crazy-paving pattern in five cases (Fig. 1 b). The segments involved were the posterior segment of the upper lobe, the segments of the lower lobes, the middle lobe, and the lingula.

One patient showed dilated and distorted bronchi within areas predominantly showing consolidation and a crazy-paving pattern (Fig. 2 a). Another patient showed areas of ground-glass opacities with centrilobular interstitial thickening, crazy-paving pattern, and some areas of cystic changes without any consolidation area (Fig. 3).

Two patients underwent MR examination in addition to HRCT since the diagnosis had been suggested on CT. Abnormal areas of consolidation with irregular limits were found, with a fairly high signal intensity on T1-weighted images. Nevertheless, this signal was lower than in the chest wall and not considered to be characteristic of fat. No areas of ground-glass attenuation surrounding areas of consolidation as well as septal lines were visible (Fig. 1 c, d).

Bronchoalveolar lavage findings were available in five cases and showed an increased cellularity with predominance of lipid-laden macrophages. One case had neutrophilic alveolitis, and mixed lymphocytic, and neutrophilic alveolitis was detected in three other cases.

Pathologic examination of surgical specimen ($n = 2$) and biopsies ($n = 2$) showed diffuse lesions. The most striking feature in the four cases reviewed was the coex-

Table 1. Clinical, HRCT, BAL Results and follow-up

	Clinical findings	HRCT	BAL results/diagnosis	Treatment/follow-up
1	F/55 yo – cough for 3 months – an[al] chest film – paraffin oil for constipation	– middle lobe consolidation of low density (– 49 HU) surrounded by ill defined areas of ground glass opacities and a crazy paving pattern	BAL: increased cellularity. lymphocytes and lipid-laden macrophages	Lobectomy no symptoms
2	M/72 yo – rhumatoïd arthritis – an[al] chest film – paraffin in drops for vocal cords	– areas of consolidation (– 60 HU) surrounded by groundglass opacities, segments S6R, S10R, S6L – one enlarged right hilar node	BAL: increased cellularity. lipid-laden macrophages	Stable over 8 years no symptoms
3	F/46 yo – an[al] chest film – paraffin oil for constipation	– larges areas of groundglass opacities with crazy-paving pattern and small consolidation of low density attenuation – S3R, S10R and L, S4R, S5R	BAL: increased cellularity lipid-laden macrophages	Stable over 1 year No symptoms
4	M/65 yo – paraffin in nose drops for chronic rhinitis – an[al] chest film	– areas of consolidation of low density surrouneded by ground glass opacities and crazy-paving pattern – S6R and L/S3R/S4 and 5R and L and S10R and L	BAL: increased cellularity lipid-laden macrophages	Stable over 2 years No symptoms
5	F/40 yo – an[al] chest film – paraffin in nose drops	– large areas of ground glass opacities with crazy paving pattern; cystic changes in some areas – S4 and 5R and L, S9 and 10R and L	BAL: 300 000 elements with 50 % of polynuclear surgical biopsy	Stable over 1 year No symptoms
6	M/53 yo – an[al] chest film – paraffin in nose drops	– areas of consolidation of low attenuation (– 80 HU)		Lobectomy
7	M/42 yo – an[al] chest film – paraffin oil for constipation	– areas of ground glass opacities without consolidation with small cystic areas and a crazy-paving pattern – S4 and 5R and L, S6R and L, S8, 9, 10R and L	surgical biopsy: macrophages containing vacuoles with some areas of inflammatory reaction and fibrosis	Stable over 1 year

Pulmonary segments involved are named according to Boyden classification. R is right and L is left. BAL is bronchialveolar lavage

istence of lobules occupied by lesions of different ages in the specimen of a patient. Recent and old lesions were sometimes seen in contiguous secondary pulmonary lobules (Fig. 1 e). Recent lesions were those with alveolar fill-in by spumous macrophages containing cytoplasmic microvacuoles and an abundant lipoproteinaceous material with normal or almost normal (few inflammatory infiltrates) alveolar walls and septae. In more advanced lesions, larger vacuoles were present, often surrounded by macrophages and inflammatory infiltrates of the alveolar walls and interlobular septae (Fig. 1 f). The oldest lesions were those with interstitial fibrosis and parenchymal distortion (Fig. 2 b). In a single patient (patient 5), a typical appearance of follicular bronchiolitis was present in addition to the other changes described previously (Fig. 2 c). The thickening of interlobular septae was due to edema, large vacuoles, inflammatory infiltrates, or fibrosis, depending on the lesions visible within the adjacent lobules.

Considering follow-up, we had chest films available and at least one CT examination performed after the diagnosis (between 7 months and 8 years) in the seven cases. In every patient who was not operated on, stability of pulmonary lesions was noted. No regression, even partial, was observed despite the recommendations given to the patients not to use their drops.

Discussion

Lipoid pneumonia is caused by aspiration oils. Predisposing factors, such as neuromuscular disorders and esophageal abnormalities, have been reported [1, 2, 3, 4]. In many cases excessive use of an oily substance is the presumed cause. Unlike animal oils, which elicit a very active inflammatory response [15], mineral oils are fairly inert because they have no fatty acids and are rapidly emulsified and consumed by pulmonary macrophages. Vegetable oils are emulsified and not hydrolyzed by the lung lipase and result in a foreign-body reaction. The macrophages containing phagocytosed oil fill in the alveoli and distend the alveolar walls and interlobular septae. Fibrosis is present occasionally and is of variable extent [1, 2, 3].

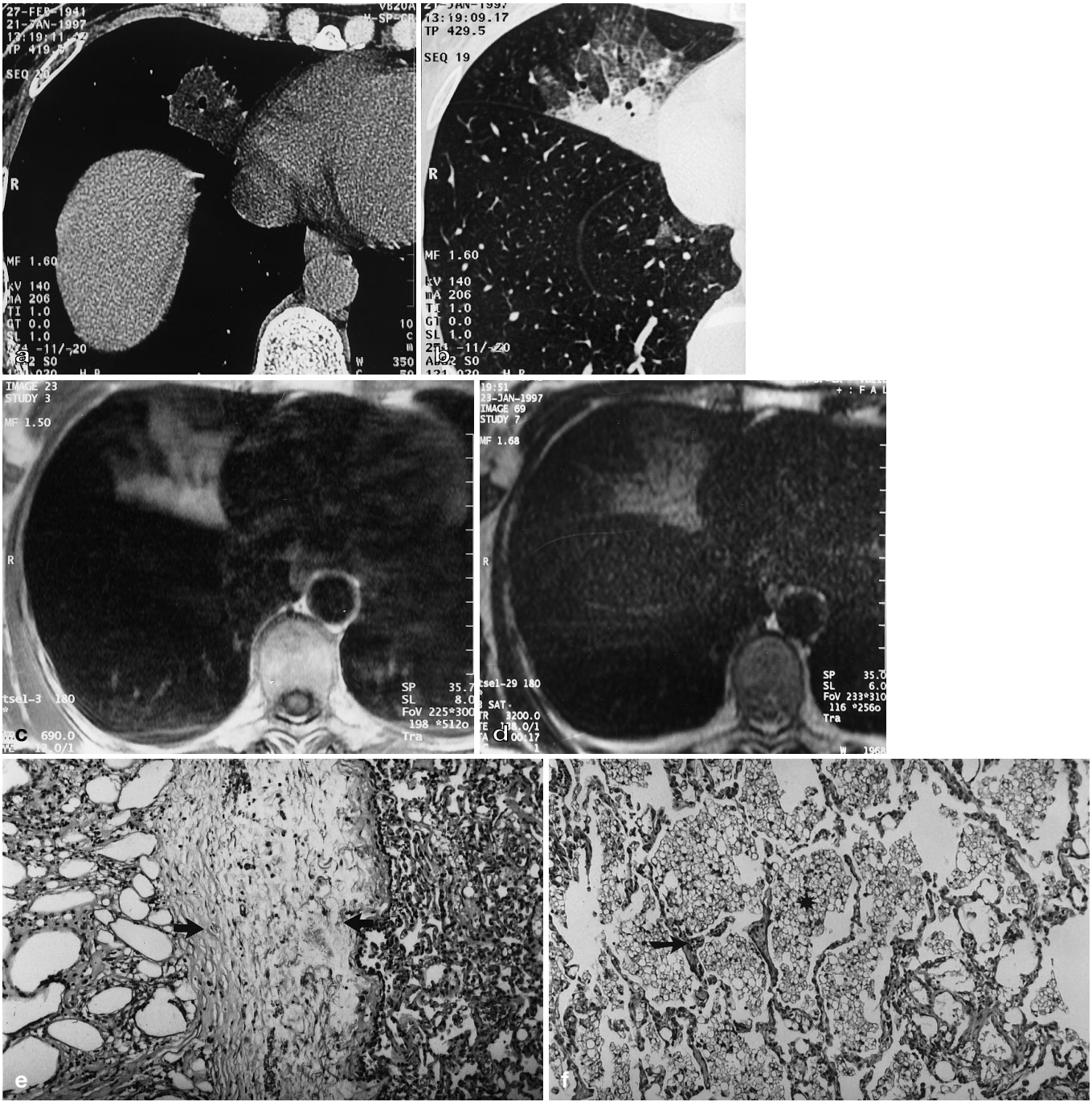

Fig. 1a–f. Patient 1. **a** A CT scan. Mediastinal windows. Area of consolidation of the middle lobe of uniform attenuation value, lower than soft tissue but not characteristic of fat. **b** Lung window at a slightly higher level: consolidation area abutting the main fissure surrounded by a crazy-paving pattern (ground-glass opacity with superimposed septal thickening and centrilobular interstitial thickening). **c** T1-weighted MR image: area of high signal intensity corresponding to the main area of consolidation. **d** T2-weighted MR image: low signal intensity of the consolidation area. **e** Microscopic view of a septum (between *arrows*) thickened by edema and separating two lobules with lesions of different ages. On the right-hand side, characteristic young lesions are visible: sparse spumous cells within alveolar lumina and normal alveolar walls. On the left-hand side, large vacuoles of lipoid inclusion are separated by interstitial fibrosis. **f** Microscopic higher-power view of young lesions: alveoli are filled in by macrophages with intracytoplasmic vacuoles *(star)* and separated by normal alveolar wall *(arrow)*

Aspiration of oil commonly occurs in elderly subjects who may take their oily nose drops or cathartics at bedtime. In our series the mechanism was always a chronic aspiration and symptoms were light. Suspicion of pulmonary cancer on the chest film was the main reason for chest CT. Fat may be trapped in the lymph nodes [4]. One patient in our series had an enlarged hilar node.

Pathologic microscopic features of exogenous lipoid pneumonia include intra-alveolar spumous macrophages associated with interstitial pneumonia. Sometimes fat is entrapped in the interstitium [1, 2, 4, 15]. In our series lesions of various ages were identified in the specimens of the four patients whose pathological features could

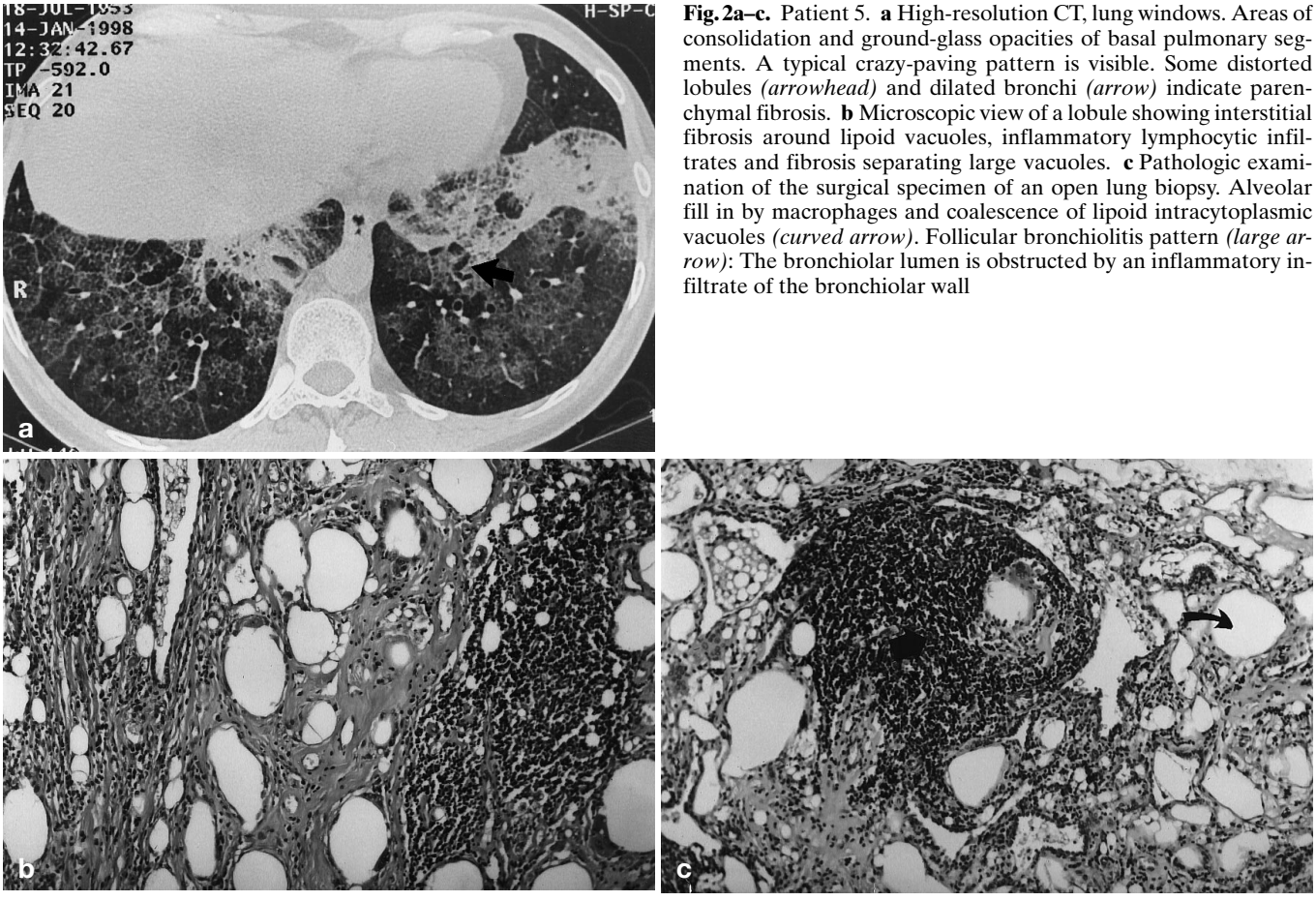

Fig. 2a–c. Patient 5. **a** High-resolution CT, lung windows. Areas of consolidation and ground-glass opacities of basal pulmonary segments. A typical crazy-paving pattern is visible. Some distorted lobules *(arrowhead)* and dilated bronchi *(arrow)* indicate parenchymal fibrosis. **b** Microscopic view of a lobule showing interstitial fibrosis around lipoid vacuoles, inflammatory lymphocytic infiltrates and fibrosis separating large vacuoles. **c** Pathologic examination of the surgical specimen of an open lung biopsy. Alveolar fill in by macrophages and coalescence of lipoid intracytoplasmic vacuoles *(curved arrow)*. Follicular bronchiolitis pattern *(large arrow)*: The bronchiolar lumen is obstructed by an inflammatory infiltrate of the bronchiolar wall

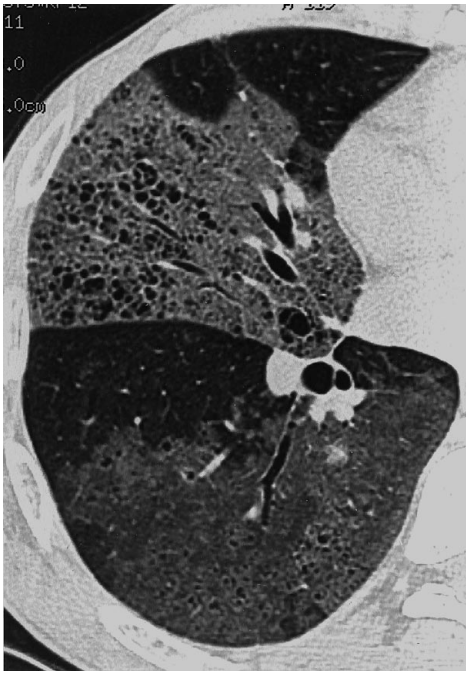

Fig. 3. Patient 7. High-resolution CT at lung windows. Large areas of ground-glass opacities of the middle lobe and right upper segment of the lower lobe. A superimposed centrilobular interstitial thickening and cystic changes are prominent in the middle lobe. Pathologic examination (not shown) showed coexistence of lesions of different ages

be reviewed. The youngest lesions consisted of alveolar fill-in by macrophages containing microvacuoles with normal alveolar walls and interstitial septae. Inflammatory infiltrates of alveolar walls and thickened interlobular septae by edema or macrophagic infiltration were likely to represent chronic inflammatory changes. It is noteworthy that all these changes were observed in different areas in the four patients. The distribution of lesions corresponded to the secondary pulmonary lobule anatomy with lesions of different ages in contiguous lobules. Fibrosis and destruction of the background lung architecture was the latest stage and was observed in two patients. It corresponded to those with HRCT findings of fibrous changes. Follicular bronchiolitis was a pattern observed in a single specimen. This entity is defined as the abnormal presence of lymphoid follicles along small airways and has been reported with many chronic inflammatory conditions, especially rheumatoid arthritis [16]. To the best of our knowledge, the association with lipoid pneumonia has not been previously reported.

The simple presence of lipid-containing macrophages from bronchoalveolar lavage aspirates is not sufficient to make the diagnosis, but the amount of lipid-rich macrophages is noteworthy. For the three patients without pathologic proof, the diagnosis was based on compatible clinical and radiological features, BAL results, long-term stability over time, and absence of any other possible cause of pulmonary diffuse lung disease.

Chest-film patterns have been reported to be airspace consolidation, irregular mass-like lesion. or reticulonodular pattern [8]. These findings are non-specific. Nevertheless, discrepancies between the extent of pulmonary abnormalities and the paucity of symptoms is an appealing feature for the referring clinician.

The most characteristic CT finding of lipoid pneumonia is lung consolidation of fat attenuation [5, 6]. This probably occurs when the vacuoles are sufficiently clustered and fused to lower CT density. The HRCT technique is likely to be more sensitive than conventional CT because of the lesser partial-volume effect and higher spatial resolution obtained with thin slices. Nevertheless, CT attenuation measurements are not always characteristic due to the averaging with attenuation values from surrounding inflammatory infiltrates. In these cases extensive consolidation is of lower attenuation value than expected but is higher than subcutaneous fat [8]. In our series, when measurement was performed in the most hypodense part of the consolidation area of our patients, three cases showed specific fatty values of attenuation.

Recently, an evocative although unspecific appearance at HRCT was reported in three patients with lipoid pneumonia: the so-called crazy-paving pattern [13]. This pattern consists of interlobular septal thickening and superimposed ground-glass opacities and/or centrilobular interstitial thickening [17]. We found this pattern to a variable extent in five of our seven patients, either isolated in wide areas or located at the periphery of the consolidation, much less predominant than the latter and therefore easily ignored by the previous descriptions of lipoid pneumonia. The pattern is non-specific since it has also been described in alveolar proteinosis [18] and bronchoalveolar carcinoma [19]. It reflects gross and microscopic features of pathologic findings. In the particular case of lipoid pneumonia, ground-glass opacities are very likely to reflect the fill-in of alveoli with macrophages and the variable amount of inflammation. Thickened septae and centrilobular thickening are due to inflammatory infiltrates of interstitial tissue. Cystic changes and traction bronchiectases are known HRCT findings indicating parenchymal distortion; however, pathologic parenchymal distortion in areas of consolidation is not detectable at HRCT.

In this retrospective review of seven cases, low attenuation areas of consolidation and crazy-paving pattern were present together in six of seven cases. These findings, considered together in an appropriate clinical context, are very evocative of the diagnosis of lipoid pneumonia. The particular location of these findings, consolidations centrally located and the more peripheral crazy-paving pattern, are particularly of value in suggesting the diagnosis of lipoid pneumonia. All these findings were remarkably stable at follow-up, and patients remained asymptomatic.

Magnetic resonance imaging did not prove to be as efficient as HRCT for the diagnosis of lipoid pneumonia. Lack of specific signal intensity may be due to the relative inability of conventional sequences to demonstrate a fatty component compared with more sophisticated sequences such as "in-and-out-of-phase" techniques [11, 12]. Spatial resolution of MR images is also much lower than that of HRCT. This explains why MR imaging was unable to show the thickened septae and ground-glass opacities at the periphery of pulmonary consolidation areas.

Our data confirm that consolidation of low-attenuation, sometimes specifically fatty and crazy-paving patterns, are the most common and evocative findings of lipoid pneumonia. These findings are frequently associated. They reflect pathologic findings before the advent of extensive fibrosis: fill-in of alveoli by macrophages, inflammatory reaction and thickened septa by inflammatory infiltrates, macrophages, or vacuoles. We believed that HRCT is the imaging method of choice for the diagnosis of lipoid pneumonia. Although this is an uncommon diagnosis, coexistence of centrally located low-attenuation-value consolidation areas surrounded by a crazy-paving pattern are very evocative and may help to suggest this diagnosis.

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Book review

European Radiology

Freedom R. M., Mawson J. B., Yoo S. J., Benson L. N.: Congenital heart disease: textbook of angiocardiography (2 volumes). Armonk, NY: Futura Publishing, 1997, 1432 pages, 2703 illustrations, $ 275.00, ISBN 0-87993-656-8

Professor Robert Freedom, head of the Department of Pediatric Cardiology in The Hospital for Sick Children in Toronto, is known as an exceptional clinician. He possesses an amazing skill in the cardiac catheterisation laboratory. He has an imposing memory and an impressive knowledge of the worldwide literature of congenital cardiology. Above all, he has an exceptional teaching ability and is proud of sharing his knowledge freely with his fellows and colleagues.

Congenital Heart Disease: Textbook of Angiocardiography consists of two beautifully bound volumes comprising 1432 pages and 2703 illustrations. The book reflects the extensive clinical and catheterisation experience of the authors. The 51 chapters cover morphological descriptions of the whole range of congenital heart malformations, including common as well as very rare cardiac abnormalities. For each malformation the different anatomical subtypes and frequently associated cardiac anomalies which may complicate the lesion are described. The numerous high-quality digitally constructed angiogaphic images are the highlight of each chapter. Particular attention is paid to angiographic technique and the various projections required for a complete anatomical evaluation of each malformation. Furthermore special techniques such as transhepatic catheterisation, coronary angiography using balloon occlusion of the aorta and retrograde pulmonary venous wedge injections to visualise 'absent' pulmonary arteries are reported. The angiographic images are accompanied by a clear descriptive comment.

Each chapter is similarly structured and well organised in a logical sequence, starting with an initial section on embryology, morphogenesis and prevalence. This is followed by an extensive description of the anatomy, beautifully illustrated by schematic drawings, pathological specimens and numerous angiographic images. The final section of each chapter discusses the post-surgical status and possible complications. The authors underline the importance of a segmental and sequential analysis, which was introduced by Van Praagh and Anderson. There is a clear explanation of the concepts of atrial arrangements, atrioventricular and ventriculoarterial connections, pulmonary and systemic venous connections and the spatial relationships of the different cardiac chambers. In addition the authors discuss techniques and results of transcatheter interventions: for example occlusion of a patent arterial duct, balloon valvuloplasty of stenotic valves, angioplasty and stent implantation of pulmonary arteries, and fulguration of atretic pulmonary valves. In contrast to the first edition this second edition is much more extensive and adapted to current interventional and surgical techniques. The book provides a wonderful source of references which are up-to-date and extensive.

The first volume covers general principles of angiography followed by seven chapters on malformations characterised by left-to-right shunts and 12 chapters on right heart malformations. The reader will find extensive information on all the possible anomalies and variations of pulmonary blood supply, including pulmonary arteriovenous fistulae, vascular slings and scimitar syndrome. As remarkable advances in surgery have been made to treat patients with pulmonary atresia and ventricular septal defect it has become essential to define precisely the complex pulmonary circulation of the different bronchopulmonary segments. Chapter 23 is exceptional in its description of the pathology of pulmonary atresia and intact septum. This is beautifully illustrated by outstanding angiographic images and pathological specimens of the right ventricle, the atretic pulmonary valve and the ventriculocoronary communications which may complicate this lesion.

The second volume describes malformations of the left heart and malformations of atrioventricular and ventriculoarterial connections. The final chapters discuss important acquired and miscellaneous disorders. The controversial and difficult subject of double outlet right ventricle is very clearly described and beautifully illustrated with picture specimens, drawings and angiograms (Chapter 27). The complexity of univentricular hearts, atrial isomerism or heterotaxia and corrected transposition are elucidated in an exceptional and unique manner.

The book is very comprehensive and will provide the reader with many hours of reading pleasure. It is also an excellent reference book for both invasive and non-invasive paediatric cardiologists, as well as radiologists involved in cardiovascular assessments. The book is a unique and authoritative source of information. The beautiful angiographic images are a work of art. Although echocardiography and nuclear magnetic resonance imaging have assumed an important place in the assessment of congenital heart malformations, cardiac catheterisation and angiography will remain a very important diagnostic and therapeutic modality.

Unfortunately the two volumes are large, heavy and difficult to transport. The price of $ 275.00 is expensive but worth the investment. The library of any paediatric cardiologist would be incomplete without this reference book. B. Eyskens, Leuven