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Differentiation of linear and reticular opacities in high resolution computed tomography (HRCT) in interstitial lung diseases

High resolution computed tomography revolutionized imaging diagnosis of interstitial lung disease. HRCT reveals changes invisible on chest radiograms and in conventional CT, enabling their assessment at the level of the smallest structural unit of the lung interstitium, lung lobule. Large groups of morphological changes visible on HRCT in interstitial lung diseases are linear and reticular opacities.

The aim of the study was assessment of the frequency and diagnostic value of linear and reticular opacities in interstitial lung diseases.

MATERIAL AND METHODS

The material comprised a group of 61 patients (12 women and 49 men), aged between 24 and 78 years. The HRCT examination was preceded by chest radiograms, revealing changes in interstitial lung diseases. Scans were obtained with patients in supine and prone positions, in different respiration phases. The scan collimation was 2mm.

RESULTS

In 4 patients the thickening of the bronchial walls formed peribronchial cuffs. In 3 cases linear opacities few millimeters thick, parallel to the pleura, and localized 1 cm from it formed so-called subpleural lines (Fig.1). In 9 cases of sarcoidosis, interlobular septal thickening coexisted with multiple nodules, adjacent to vessels and bronchi, forming reticular pattern (Fig. 2). Irregular, parahilar peribronchovascular interstitial thickening, forming the interface sign was found in 11 cases of pulmonary fibrosis. In 6 patients, parenchymal bands, 2–5 cm long, were localized in the lung periphery and contacted with the surface of the pleura. The parenchymal bands were found in pulmonary fibrosis, coexisting with nodular opacities (Fig. 3). In 9 patients the thickness of bronchial walls exceeded normal relation of the bronchial wall to its diameter, which suggests peribronchovascular interstitial thickening (Fig. 4). In 12 cases the thickened interlobular septa in the lung periphery, outlined the lobules perpendicular to pleural surface. The lobules outlined by thickened interlobular septa showed central nodular or branching small centrilobular artery. In 7 cases the evidence of peribronchial fibrosis coexisted with thickening of interlobular septa (Fig. 5). Irregular linear opacities were related with distortion of surrounding lung parenchyma and bronchovascular structures. The linear opacities of thickened interlobular septa and interstitial fibrosis were the most frequent in pneumoconiosis and sarcoidosis.

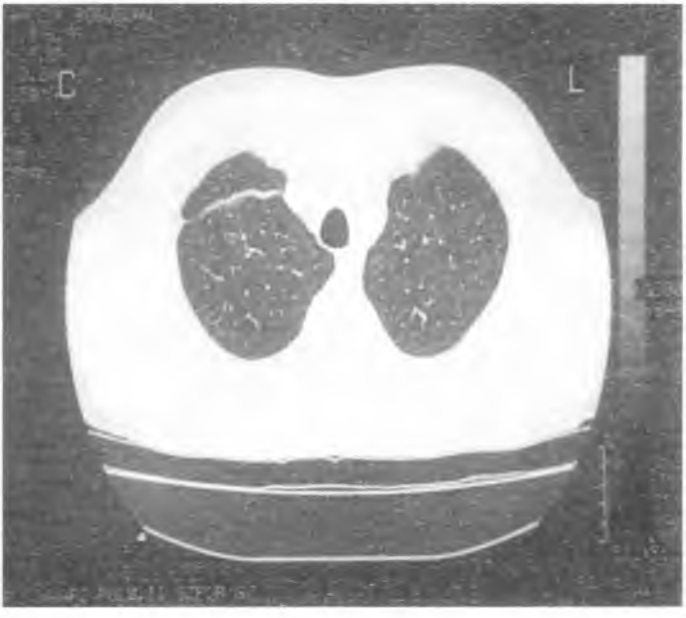

Fig. 1. Linear thickening parallel to the pleura in the right apex

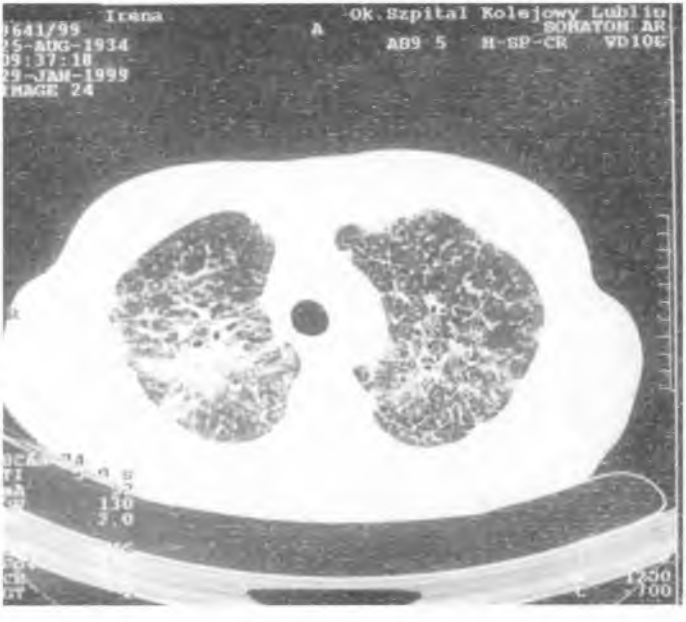

Fig. 2. Diffuse reticulo-nodular opacities confusing into inhomogeneous conglomerates, especially on the right. The areas of centrilobular emphysema associated with bronchiolectases

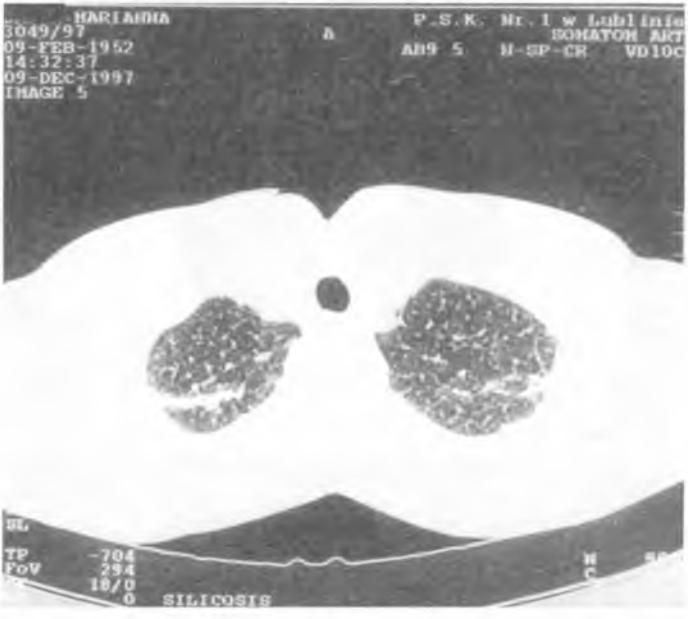

Fig. 3. Parenchymal bands in both apices form linear opacities with diffuse nodules

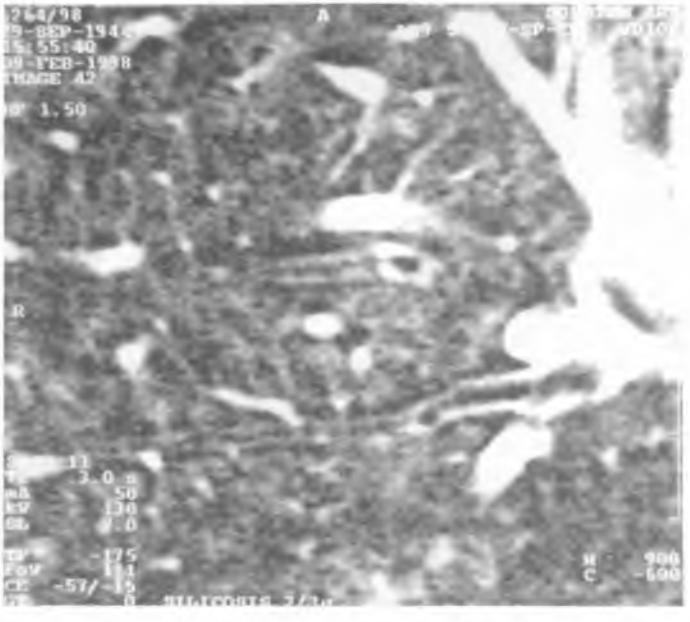

Fig. 4. Thickened walls of parahilar bronchi form the tram-line sign. Multiple nodules and centrilobular emphysema

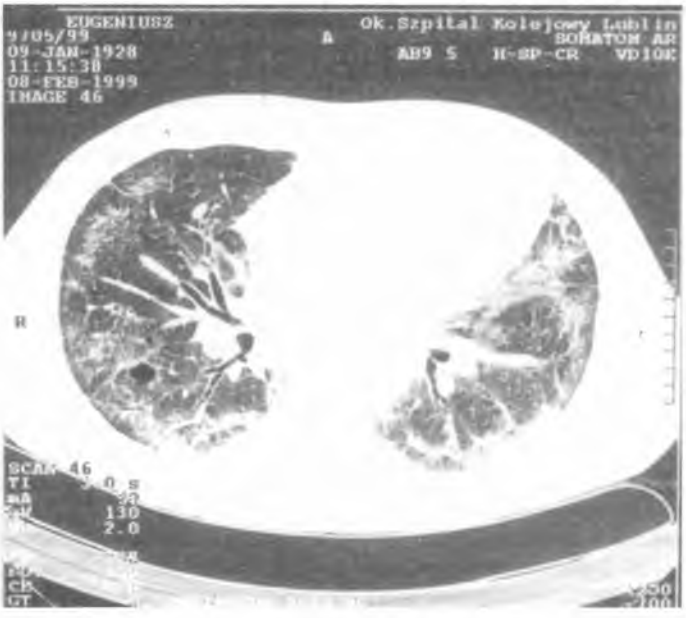

Fig. 5. Peribronchovascular interstitial thickening extending from the hila with the diffuse parenchymal fibrosis and irregular emphysematous areas

DISCUSSION

The thickening of interstitial fiber network of the lung by fluid, fibrous tissue, or because of interstitial infiltration by cells or other material, results in linear and reticular lung opacities seen on HRCT (21). They may be manifested by the interface sign, peribronchovascular interstitial thickening, interlobular septa thickening, intralobular interstitial thickening or subpleural lines. The term interface sign refers to the presence of irregular interface between the aerated lung parenchyma and bronchi, vessels or visceral pleural surface (21). Usually it is associated with the increase in lung reticulation. Because the thin linear opacities contact the vessels, bronchi and the pleural surface, they form the irregular appearance of these structures on HRCT (8,11). The interface sign is most frequent in patients with fibrotic lung diseases, in up to 94–98% of cases (14,21). It may be also visible in sarcoidosis and other interstitial abnormalities (5).

Central bronchi and pulmonary arteries are surrounded by a strong connective tissue sheath, termed the peribronchovascular interstitium, which extends from the level of the pulmonary hila into the peripheral lung. Since the thickened peribronchovascular interstitium cannot be distinguished from the underlying opacity of the bronchial wall or pulmonary artery, this abnormality is usually perceived on HRCT as an increase in bronchial wall thickness or an increase in diameter of pulmonary artery branches (21). Peribronchovascular interstitial thickening can be smooth, nodular or irregular. Smooth peribronchovascular interstitial thickenings are typical in pneumonias, lymphangitic spread of carcinoma, interstitial pulmonary edema, and lung fibrosis (17,21). Nodular thickening of the peribronchovascular interstitium may be found in fibrotic lung diseases, especially in sarcoidosis. In lymphangitic spread of carcinoma both nodular and smooth thickening of peribronchovascular interstitial thickening may be found, associated by septal and pleural thickening (9,12,21). The small subpleural and

large nodules may also be found (21). The peribronchovascular interstitial thickenings were described in 19% of patients with hypersensitivity pneumonia (21).

The thickness of normal bronchial wall should measure from 1/10 to 1/6 of its diameter, but it is not reliable diagnostic criterion (21). The measurement vary depending on the lung window chosen; too low a window mean can make normal bronchi or vessels appear abnormal. In unilateral or patchy changes, normal and abnormal lung regions can be easy contrasted. In lung fibrosis and peribronchovascular interstitial thickening bronchial dilatation is common, due to traction by the fibrous tissue on bronchial wall (traction bronchiectases) (3). They result in varicose bronchial appearance. Traction bronchiectases usually involves segmental and subsegmental bronchi, most common in parahilar lung regions.

The interstitial pathology may be differentiated from airways disease on the basis of symptoms, or pulmonary function abnormalities. In bronchiectases the thickened bronchial wall and dilated lumen are usually larger than the adjacent lung artery branch (7,21). That form large annular opacity with accompanying small nodular density is called signet-ring sign, typical of bronchiectases (3,7,21).

In peribronchovascular interstitial thickening the size relation between the bronchus and adjacent artery is maintained, and they appear approximately equal size (7,17,21). The bronchial wall thickening, due to inflammation, is considered typical in patients with asthma, often associated with the presence of air-trapping (4,10).

Interlobular septal thickening results from the presence of interstitial fluid, cells infiltration or fibrosis (3,15,21). Lobules at the pleura surface may have variety of shapes, are often longer than they are wide, resembling a cone. Within the central lung, thickened septa outline lobules, which are 1 to 2.5 cm in diameter, and polygonal in shape (21). Septal thickening may be smooth, nodular or irregular. The smooth thickening may be seen in lymphangitic spread of carcinoma, lung edema, alveolar proteinosis, pneumonia and in a small percentage of patients with pulmonary fibrosis (8,9,11,12,13,21). In alveolar proteinosis they form crazy pavement appearance (13). Nodular septal thickening may be seen in lymphangitic spread of carcinoma, sarcoidosis, pneumoconiosis (9,11,12,21). In lung fibrosis, septal thickening are often irregular (11,15). The fibrosis and honeycombing make the recognition of septal thickening difficult (21).

Parenchymal bands, primarily described in asbestosis, may be seen in sarcoidosis with lung fibrosis, silicosis and tuberculosis (1,20,21). In asbestosis they are often associated with pleural thickening and dominate in parabasal lung areas (3,20,21).

Interlobular septal thickening in the lung periphery, is called subpleural interstitial thickening. They are difficult to recognize in locations where the lung contacts the chest wall or mediastinum, but are easy to see adjacent to the major fissures. In this location two layers of subpleural interstitium are seen adjacent to each other and any abnormalities appear twice abnormal, resulting in thickening of the fissure. The smooth thickening of fissure may result from the presence of fluid, difficult to differentiate form subpleural interstitial thickening (21). The differential diagnostics of subpleural interstitial thickening and interlobular septal thickening is similar. The subpleural interstitial thickenings are the most common in idiopathic pulmonary fibrosis and pneumonia. Nodular subpleural interstitial thickenings have similar differentiating diagnostics like nodular interlobular interstitial thickening (21). Nodular subpleural interstitial thickenings were described in sarcoidosis, pneumoconiosis, asbestosis and lymphangitic spread of carcinoma (1,9,15).

Intralobular interstitial thickenings form a fine reticular pattern on HRCT, with lines of opacity separated by a few millimeters. Involved lung regions have characteristic net-like appearance. Intralobular bronchioles are often visible, because of coexistence of dilatation of bronchioles (traction bronchiolectases) and thickening of surrounding them peribronchiolar interstitium (21). Interlobular septal thickening, when present, appears irregular, like the surface of pleura. Visibility of intralobular bronchioles with association of fine reticular pattern and increased lung attenuation was described in 96% of interstitial pneumonias, indicating bronchiolar dilatation, fibrosis and microscopic honeycombing (14). Interlobular interstitial

thickening may also be visible in lymphangitic spread of carcinoma, pulmonary edema and alveolar proteinosis (9).

Intense interstitial and alveolar fibrosis, resulting in alveolar disruption and bronchiolectases form characteristic appearance of honeycombing (3,21). They indicate the presence of end-stage lung fibrosis (21). The diameter of cystic spaces in honeycombing usually averages about 1 cm, with distinct, 1–3 mm thick walls. The cysts are air-filed and appear lucent in comparison to normal lung regions or regions of intralobular interstitial thickening. The adjacent honeycomb cysts typically share walls. They often predominate in peripheral and subpleural regions of the lung. Subpleural honeycomb cysts typically occur in several adjacent layers, which allow them to be distinguished from subpleural cysts, usually occurring in single layer (21). Honeycombing is usually associated with other symptoms of fibrosis, including: interlobular interstitial thickening, traction bronchiectases and bronchiolectases, irregular subpleural interstitial thickening. Apart from sarcoidosis, interlobular interstitial thickening may not be seen in association with honeycombing (18). In septal thickening, the presence of honeycombing allows differentiating fibrosis from other reasons of reticular pattern on HRCT, like pulmonary edema or lymphangitic spread of carcinoma.

Linear opacities, several millimeters thick, localized less than 1 cm from the pleural surface and parallel to it, are called subpleural lines, were first described in asbestosis, and then in alveolitis (3,16). It was suggested that subpleural lines reflect the presence of fibrosis associated with the appearance of honeycombing, and confluence of honeycombing cysts may result in appearance of subpleural line. They may also be visible in healthy people as a result of atelectasis of dependant lung areas (19).Patchy, poor defined subpleural opacities, so called dependant densities, can also be seen in normal patients, as a result of volume loss. Such physiological dependant densities are transient and disappear in the prone positions (19,21).

The diagnostic difficulties cause non-transient subpleural lines, associated with areas of lucencies peripheral to it, which can be reversible in period of weeks or months following treatment. They may reflect atelectasis, with small bronchial or bronchiolar obturation and air-trapping within the peripheral lucencies (21).

The centrilobular bronchioles are sometimes visible in interlobular interstitial thickening, because of increased attenuation of surrounding lung, thickening of the peribronchiolar interstitium, and dilatation of the bronchiole, which occurs as a result of fibrosis (2,21) Diseases that involve small airways can also result in prominence of centrilobular branching structures, resulting in increased reticulation in HRCT. The visibility of centrilobular bronchiole without other interstitial thickening, suggest airway disease. The changes suggest the dilatation of the bronchiole and thickening of its wall, peribronchiolar fibrosis and inflammation (2).

Small airways, dilated and filled with pus, mucus or inflammation exudation, appear like small, well defined, centrilobular, nodular, linear or branching structures of interstitial thickening. This appearance on HRCT is called tree-in-bud (2,3). Abnormal bronchioles producing a tree-in-bud appearance can usually be distinguished from normal centrilobular vessels by their more irregular appearance, a lack of tapering of a bulbous appearance at the tops of small branches. That reflects the presence of bronchiolar dilatation and peribronchiolar inflammation. Tree-in-bud patterns are often patchy in distribution in diffuse airway abnormalities. The appearance of the abnormal airways and normal vessels in other lung regions can be easily contrasted (21).

Centrilobular bronchiolar abnormalities characterized by dilatation and a tree-in-bud pattern are seen in patients with endobronchial spread of tuberculosis, cystic fibrosis, bronchopneumonia, bronchiectases of any cause, and other airway diseases that result in accumulation of mucus or pus within small bronchi (2,21).

CONCLUSIONS

HRCT enables evaluation of linear and reticular densities invisible on chest radiograms and even on conventional CT. The linear and reticular opacities occur in different interstitial lung disease. They are typical of both active inflammatory changes, potentially treatable and irreversible pulmonary fibrosis. The linear opacities without evident lung architecture distortion suggest active process, whilst cystic changes, honeycombing and evident lung dissertation suggest presence of irreversible fibrosis. The tree-in-bud pattern is a very specific finding in pathology of airways, and suggests prance of bronchiolitis. The linear and reticular densities separately do not allow reliable differential diagnosis, and must be assessed in association with other HRCT findings.

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SUMMARY

High resolution computed tomography revolutionized imaging diagnosis of interstitial lung disease. HRCT reveals changes invisible on chest radiograms and in conventional CT, enabling their assessment on the level of the smallest structural unit of the lung interstitium, lung lobule. A large group of morphological changes seen on HRCT in interstitial lung disease are linear and reticular opacities. HRCT enables evaluation of linear and reticular densities invisible on chest radiograms and even on conventional CT. The linear and reticular opacities occur in different interstitial lung disease. They are typical of both active inflammatory changes, potentially treatable and irreversible pulmonary fibrosis. The linear opacities without evident lung architecture distortion suggest active process, whilst cystic changes, honeycombing and evident lung distortion suggest presence of irreversible fibrosis. The tree-in-bud pattern is a very specific finding in pathology of airways, and suggests the presence of bronchiolitis. Apart from that, the linear and reticular densities separately do not allow reliable differential diagnosis, and must be assessed in association with other HRCT findings.

Różnicowanie linijnych i siateczkowatych zacienień w tomografii komputerowej wysokiej rozdzielczości (TKWR) wybranych schorzeń śródmiąższowych płuc

Tomografia komputerowa wysokiej rozdzielczości zrewolucjonizowała diagnostykę obrazową chorób śródmiąższowych płuc. TKWR uwidacznia zmiany niewidoczne na radiogramach klatki piersiowej i w konwencjonalnej tomografii komputerowej, umożliwiając ocenę zmian na poziomie najmniejszej jednostki strukturalnej–zrazika płucnego. Dużą grupę zmian morfologicznych widocznych w TKWR w śródmiąższowych schorzeniach płuc stanowią zacienienia linijne i siateczkowate. TKWR umożliwia ocenę linijnych i siateczkowatych zacienień niewidocznych na radiogramach klatki piersiowej i w konwencjonalnej TK. Linijne i siateczkowate zacienienia występują w różnych schorzeniach śródmiąższowych. Są typowe zarówno dla aktywnych zmian zapalnych, potencjalnie wyleczalnych, jak i nieodwracalnego zwłóknienia płuc. Linijne zacienienia bez obecności ewidentnego zniekształcenia architektury miąższu płuc sugerują obecność procesu aktywnego, gdy zmiany torbielowate, obrazy plastra miodu i ewidentne zniekształcenie miąższu świadczą o obecności nieodwracalnego zwłóknienia. Obecność drzew w pąkach jest bardzo specyficznym objawem patologii dróg oddechowych i sugeruje zapalenie oskrzelików płucnych. Poza tym linijne i siateczkowate zacienienia same nie umożliwiają wiarygodnej diagnostyki różnicowej i muszą być oceniane wraz z innymi zmianami widocznymi w HRCT.