PICTORIAL REVIEW

Focal pleural thickening mimicking pleural plaques on chest computed tomography: tips and tricks

1KHALID M ALFUDHILI, MBBS, PhD, 2DAVID A LYNCH, MD, 3,4FRANCOIS LAURENT, MD, 5,6,7GILBERT R FERRETTI, MD, 1VINCENT DUNET, MD and 1CATHERINE BEIGELMAN-AUBRY, MD

1Radiodiagnostic and Interventional Radiology, CHUV-University Hospital, Lausanne, Switzerland
2Department of Radiology, National Jewish Health, Denver, CO, USA
3Department of Radiology, Centre Hospitalier Universitaire de Bordeaux, Pessac, France
4Cardiothoracic Center, Radiology Department, Inserm 1065, Université de Bordeaux, Bordeaux, France
5Radiology Department, Centre Hospitalier Universitaire A Michallon, Grenoble, France
6INSPER IN U 823, Institut A Bonniot, Grenoble, France
7Université Grenoble Alpes, Grenoble, France

Address correspondence to: Dr Khalid M Alfudhili
E-mail: drkmf@hotmail.com

ABSTRACT

Diagnosis of pleural plaques (PPs) is commonly straightforward, especially when a typical appearance is observed in a context of previous asbestos exposure. Nevertheless, numerous causes of focal pleural thickening may be seen in routine practice. Diagnosis of PPs is usually feasible, especially when a typical appearance is associated with a history of previous asbestos exposure. However, the diagnosis of PT may be problematic and medico-legal issues may occur. The analysis of the location and shape of the PT as well as associated findings are determinants to recognize their actual nature, which may be malignant. Reaching a correct diagnosis requires a good knowledge of normal loco-regional anatomy and different features of PPs. The common pitfalls in the diagnosis of PT and practical clues to recognize them must be mastered.

INTRODUCTION

Numerous causes of focal pleural thickening (PT) may be seen in routine chest CT. Diagnosis of pleural plaques (PPs) is usually feasible, especially when a typical appearance is associated with a history of previous asbestos exposure. However, the diagnosis of PT may be problematic and medico-legal issues may occur. The analysis of the location and shape of the PT as well as associated findings are determinants to recognize their actual nature, which may be malignant. Reaching a correct diagnosis requires a good knowledge of normal loco-regional anatomy and different features of PPs. The common pitfalls in the diagnosis of PT and practical clues to recognize them must be mastered.

PLEURA AND ADJACENT CHEST WALL ANATOMY: CT APPEARANCE

The outer surfaces of the lungs are successively covered with the visceral pleura, parietal pleurae, extrapleural fat, endothoracic fascia, muscles and ribs. Both the pleural layers and fluid-containing pleural space, which have an overall thickness <0.5 mm, are invisible on high-resolution CT. Extrapleural fat separating the parietal pleura from the endothoracic fascia can be markedly thickened, especially over the lateral 4–8th ribs. Fibroelastic endothoracic fascia then lines the thoracic cavity by covering the surface of the intercostal muscles and intervening ribs. Next, three layers of intercostal muscles lie between the ribs. The relatively thin innermost muscle is separated from the inner and external intercostal muscles by a layer of fat-containing intercostal vessels and nerves (Figure 1). No anatomical structure is visible internally to ribs, except hypertrophied extrapleural fat (Figure 2). Therefore, it is commonly felt that any visible soft-tissue stripe passing internally to the ribs or the intercostal stripe usually represents PT. Various muscles of the thorax are, nevertheless, frequently
responsible for exceptions to this rule. A typical appearance of normal muscles is based on the observation of a smooth lung–chest wall interface on lung window, conversely to PPs that are more sharply defined. Several muscles can be identified by their shape and topography. The transversus thoracis muscle (Figure 3) is composed of four or five slips arising that are more sharply defined. Several muscles can be identified by their shape and topography. The transversus thoracis muscle (Figure 3) is composed of four or five slips arising

Figure 1. Normal innermost (intimi) intercostal (IC) muscle (thin arrow) separated from the middle (internal) and superficial (external) IC muscles (empty arrow) by a layer of fat, transversus thoracic muscle (thick arrow) and IC vessels (arrowhead) mimicking a pleural thickening. Note that IC veins are easily recognized by their drain into the azygos vein.

Figure 2. Normal extrapleural fat (arrows) lying internal to a rib on mediastinal (a) and lung (b) windows. Note the smooth and regular interface on lung window.

Figure 3. Transverse thoracic muscle appearing as linear densities on axial sections in mediastinal window (a, b) (arrows). Despite a slight asymmetry related to a previous left upper lobectomy, the bilateral location nearby the internal mammary vessels as well as the typical fascicular appearance on coronal reformat with bone window (c) and three-dimensional coronal reconstruction (d) allow the muscular nature of these densities to be recognized.

Figure 4. Normal subcostal muscle (outlined arrows) that could simulate a pleural plaque (PP) on mediastinal windows (a). However, the smooth and regular lung–chest wall interface on lung window (b) differs totally from a real PP that deforms the lung (solid arrows) (c, d).
from the xiphoid process or lower sternum and passing superolaterally from the 2nd to 6th costal cartilages, behind the internal mammary vessels. Posteriorly, the thin and variable subcostal muscles (Figure 4) extend from the inner side of the angle of the lower ribs and cross one or two ribs and intercostal spaces to the inner side of a rib below. In the paravertebral regions, though the innermost muscle is anatomically absent, a thin paravertebral line representing the pleura and endo-thoracic fascia is sometimes visible on CT at the lung–chest wall interface.2

TECHNICAL APPROACH
Thin-section CT acquisition in full inspiration with a volume CT dose index of around 3–7 mGy is recommended for scanning the thorax.5 The presence of posterior PT on supine position requires an additional acquisition in prone position, which will be performed at a dose not exceeding the one used for supine scanning. Such an approach will differentiate a real plaque from reversible PT (Figure 5). Looking at the soft tissue, lung and bone window/level settings are useful to clearly separate the PP from the ribs, to recognize the parallel orientation with the ribs and to identify extrapleural fat. The bone window (L: 300/W: 2000–3000) particularly allows distinction between calcified costal cartilages and PPs and detection of potential erosion of the rib cortex by plaques (Figure 6). Additional coronal or sagittal reformats may be helpful in case of atypical or doubtful features on axial sections, especially near the dome of the diaphragm (Figure 7). In addition, three-dimensional reconstructions by using an air threshold may display impressions on the lung surface in case of actual PPs.

ASBESTOS-RELATED PLEURAL PLAQUES
PPs are indicative of asbestos exposure, most commonly in an occupational setting. Typically, they are seen 20 years or more after asbestos dust inhalation. They consist of discrete well-demarcated areas of hyaline fibrosis predominantly in the parietal pleura.4,5 Although PPs are most commonly asymptomatic, several studies proved that they can potentially be painful.6 Therefore, although chest pain is usually connected to malignant mesothelioma, pain related to PPs still should not be excluded from differential diagnosis list. PPs are commonly multiple, with variable size, thickness and extent. Their shape in profile is usually quadrangular, but early plaques may be minimally elevated or flat.7,8 Plaques may be smooth or may have an irregular or nodular interface with the lung (Figure 6). They are of soft-tissue attenuation, with calcification in 10–15% of cases.4 They most commonly involve the parietal pleura of the lateral thoracic wall between the 6th and 9th ribs, the posterolateral chest wall between the 7th and the 10th ribs (Figure 8), the dome of the diaphragm, the mediastinum and rarely involve the fissures.5,8 They typically spare the costophrenic angles and the apices and are most commonly observed below the level of the aortic arch.

Figure 5. Functional pleural thickening appears as a posterior non-calcified thickening seen on supine position (a) that reverses on prone position (b).

Figure 6. Analysis with three window/level settings permits confident diagnosis of a typical pleural plaque at the anterolateral part of the pleura on the right side. In addition to mediastinal (a) and lung (b) windows, the bone window (c) discriminates well the plaque from the rib, furthermore detecting a slight erosion of the internal cortex of the rib (arrow).
CONDITION MIMICKING PLEURAL PLAQUES

Normal structures

Normal structures such as the transversus thoracis muscle (Figure 9) or the subcostal muscle, especially on expiration (Figure 10), may mimic PPs. The variable contraction of the diaphragmatic fibres, an oblique orientation of innermost intercostal muscle (Figure 11) and a hypertrophied upper intercostal muscle (Figure 12) should not be confused with PT. In all these situations, a sharp and regular lung–chest wall interface on lung window setting will help in avoiding false-positive findings.

Figure 7. Nodular appearance of a partially calcified pleural plaque at the level of the dome of the left hemidiaphragm. Although somewhat atypical on axial section (a) and three-dimensional rendering view (b), the quadrangular shape is obvious on coronal reformat (c).

Figure 8. Multiple bilateral calcified pleural plaques located anterolaterally, laterally and along the paravertebral gutters. Note the typical parallel orientation to the ribs on mediastinal (a) and lung (b) windows.
Pictorial review: Focal pleural thickening mimicking pleural plaques on chest CT

**Figure 9.** Pseudoplaque appearance of the transversus thoracic muscle on axial view on mediastinal window (a) and three-dimensional (3D) reconstruction on a posterior view (b). Performing 3D rendering centred on the elevation (arrows) helps in correctly recognizing the typical fascicular structure of the muscle.

**Figure 10.** Pseudoplaque appearance of the subcostal muscle on expiration. Axial slice with mediastinal (a, c) and lung windows (b, d) on inspiration (a, b) and expiration (c, d). The exclusive presence on expiration (c, d) and the location of the pleural thickening allow its muscular and functional nature to be recognized.

**Functional pleural thickening**

Focal solitary or multiple non-calcified PT uniquely seen in a posterobasal location in supine examination may also mimic real PP. An additional acquisition in prone position will confirm their reversibility in most cases (Figure 5). These transient areas of PT could be related to accumulation of lymphatic fluid, or less probably represent subpleural atelectasis.
Visceral pleural thickening
A focal thickening of the visceral pleura, reflecting sequelae of any cause of previous pleural effusion, should not be confused with a PP, either calcified or not. Indeed, visceral pleural fibrosis may be related to asbestos-associated diffuse PT, coronary bypass graft surgery, pleural infection, mainly tuberculosis, drug-induced pleuritis, rheumatoid pleurisy, uraemic pleurisy and haemothorax. The clue to the diagnosis in such situations is that the PT, either focal or diffuse, is typically associated with crow’s feet, parenchymal bands or rounded atelectasis. Costophrenic blunting is also observed in this setting (Figure 13).5,8

Previous infectious disease
PT is a common finding in a setting of previous infectious disease. Old tuberculosis or fungal infection often causes calcified or non-calcified visceral PT near the lung apices. Upper lobe scarring and volume loss associated with nodules, calcifications or linear densities are clues to this diagnosis (Figure 14).8

Pleural metastasis
Although malignant mesothelioma is the most common primary pleural tumour, pleural metastasis from adenocarcinoma of the lung, breast, stomach and ovary as well as lymphoma and thymoma may occur. Pleural metastasis should be suspected when an atypical shape or location of a PT is observed (Figures 15 and 16) in the absence of other findings suggesting tuberculosis or silicosis. Other features suggestive of malignant disease as well as the history of the patient and/or positron emission tomography results should be carefully looked at in this setting.

Silicosis
Silicosis or coal-worker’s pneumoconiosis may give rise to pseudoPP (Figure 17) formed by coalescent small nodules.7 Like tuberculosis, they also lie above the aortic arch. In this case, however, focal PTs are associated with multiple centrilobular small nodules predominantly located in the upper
lobes that may calcify. Silicosis may also cause true PT.\textsuperscript{10} Other findings related to silicosis such as progressive massive fibrosis, which affects upper and middle zones, are more rarely encountered. Furthermore, a history of exposure to silica dust will help in reaching the final diagnosis.

**Sarcoidosis**

Sarcoidosis may also present with pseudoplaques owing to subpleural clustering of granulomas (Figure 18).\textsuperscript{7} Other findings such as mediastinal lymph node enlargement and perilymphatic distribution of micronodules suggest the diagnosis.

Figure 14. Pseudoplaque appearance of a partially calcified pleural thickening. The predominant upper location of the pleural abnormalities associated with subpleural densities and nodules (arrows) strongly suggest post-tuberculous sequelae. Axial sections on mediastinal (a) and lung (b) windows and sagittal reformat on bone window (c).

Figure 15. Pseudoplaque appearance of a pleural metastasis in a patient with lung and pleural metastasis of an oro-pharyngeal carcinoma. The new appearance of this non-dependant pleural thickening within 3 months (a, before; b, after 3 months), despite its quadrangular borders, is highly suggestive of a metastasis.
Drug-induced pleural focal thickening
Drug-induced pleural disease may occur as an isolated disorder, associated with parenchymal disease or generalized systemic reactions. A number of commonly used drugs, such as ergotamine or cyclophosphamide, can cause pleural abnormality including PT (www.pneumotox.com).

Miscellaneous
Osteophytes (Figure 19), bone tumours (Figure 20) or structures such as opacified vessels should finally not be confused with real PP. PT of fat attenuation is usually due to extrapleural fat or lipoma, although the shape may simulate a PP.

SUMMARY
Knowledge of the typical appearance and location of PPs is crucial for their correct recognition and their differentials. The frequent reversibility of dependent non-calcified PT on prone position will prove their functional nature. The history of the patient should always be kept in mind and the associated findings carefully looked at.

TEACHING POINTS
(1) Any focal thickening should be differentiated from a normal structure by recognizing the regular and sharp lung–chest wall interface with lung windowing.
(2) Any dependent focal non-calcified PT should raise the possibility of its functional nature assessed by its

Figure 16. Pseudoplaque appearance of a pleural metastasis in a 57-year-old female patient with cervical carcinoma. The atypical location of the pleural thickening that was seen on the mediastinal (a) and lung (b) windows at the anterolateral part of the hemidiaphragm but better detected as a slight elevation in (b) raised the possibility of a pleural metastasis. This was confirmed by the high uptake on positron emission tomography-CT (c).

Figure 17. Pseudoplaque in a case of silicosis seen on two axial consecutive sections with lung window (a, b). The bilateral pleural elevations are related with confluence of granulomas. Note the location above the aortic arch in (a) and the associated centrilobular micronodules in this patient with a history of occupational exposure.

Figure 18. Pseudoplaque appearance (arrows) on native axial slice with lung window (a), 6-mm-thick maximum intensity projection reformat (b) and sagittal reformat (c) in a case of sarcoidosis. The numerous granulomas typically predominate in the posterior part of the right upper lobe with a perilymphatic distribution, well seen nearby the great fissure.
reversibility on an additional low-dose acquisition on prone position.

(3) Focal PT located above the aortic arch should raise the possibility of (a) old tuberculosis if there are nodules and linear densities as well as scarring at the apex of the lung and (b) silicosis if there are centrilobular or perilymphatic nodules on maximum intensity projection reformats.

(4) Any atypical shape or location of focal PT should raise the possibility of pleural metastasis and prompt evaluation of other findings suggestive of malignant disease, the history of the patient and/or positron emission tomography results.

(5) Focal visceral PT can be distinguished from PPs by looking at the presence of parenchymal features such as crow’s feet and arched bands or rounded atelectasis.

ACKNOWLEDGMENTS
Steven Hajdu, Radiodiagnostic and Interventional Radiology CHUV-University Hospital, Rue du Bugnon 46, CH-1011 Lausanne, Switzerland.

REFERENCES