Tuberculosis on the plain chest film: recognising a disease that can take a lot of forms

Poster No.: C-0668  
Congress: ECR 2015  
Type: Educational Exhibit  
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Keywords: Education and training, Diagnostic procedure, Conventional radiography, Thorax  
DOI: 10.1594/ecr2015/C-0668

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Learning objectives

To review the variety of forms that tuberculosis (TB) presents on a plain chest radiograph.

To know when to suspect TB and how to differentiate it from other diseases.

To become familiar with the unusual presentations that have become very rare in the last 2-3 decades but should be taken into consideration due to the increasing incidence of the drug resistant form.

Background

Initially thought extinct, tuberculosis has seen a comeback in various regions of Europe during the last few years, mainly due to globalization and the lack of patient compliance to the treatment. Multiple drug resistant forms have high clinical and economical impact, making TB a challenge for every clinician and radiologist alike. Young radiologists may have little knowledge of this pathology, however the serious threat of this ailment calls for a fast and accurate diagnosis. The chest radiograph is the first step in diagnosing TB and thus a young radiologist should be prepared to deal with its various forms on a plain film. Therefore, we decided to review the chest radiographs from our archive and use them to show the multiple forms and complications of this TB.

Findings and procedure details

We consider that we should first discuss the main lesions caused by TB, from an anatomical and pathological point of view. These lesions are exudative, caseous, cavitary, proliferative/productive and fibrous.

The exudative lesions affect the alveoli and bronchi and appear on the radiograph as opacities of various shapes and sizes, of low intensity, with blurry contours. The content is initially homogenous and the evolution is towards resorption, fibrosis or necrosis.

The caseous lesions derive from a specific necrosis, caused by the coagulation of the alveolar exudate. Liquefaction and evacuation lead either to the formation of a cavern or calcification.
The cavitary lesions/caverns are areas of missing pulmonary parenchyma that appear after the necrosis of caseous lesion.

The productive lesions represent epithelioid nodules that are formed in the alveolar septa and interstitia. They can sometimes merge and can be surrounded by conjunctive and endothelial tissues. Radiologically, they are miliary or micronodulary opacities, with clear contours and medium intensity.

The fibrous lesions are represented by scarring tissues and are the result of the exudative and productive injuries. These lesions can either be localized or deforming, with potential retractive effect. Radiologically, they are linear, star-shaped or irregular shaped opacities of high intensity.

PRIMARY TB

In 90% of the cases, the first TB infection is airborne and the lungs are the main location of the lesions. The pathological and radiological aspect is that of a primary complex made of primary lesion (of exudative nature), lymphangitis and adenopathy in the hilum and mediastinum. Both infection and Koch bacillus spread throughout the respiratory system. However, the infection disseminates through the lymphatic system, whereas the bacillus spreads haematologically.

1. Primary lesion (Fig. 1,2)

From the pathological point of view, Koch bacillum causes an uncharacteristic alveolitis that evolves to caseous necrosis and tuberculous foliculi form in the periphery of the lesion.

In theory, lesions can be anywhere in the lung, but the most commonly localization is at the bases of the lungs. Often times there is a singular lesion that varies from a few millimeters to gigantic sizes.

From a radiological point of view, lesions present as a round or irregular opacity of variable size (usually 2-4 cm). Difficult or impossible diagnostic occur if the size is very small, if the localization is the costo-diafragmatic sinuses or close to the mediastinum, or if there are preexistent pulmonary or pleural lesions that can cover the TB lesion.
Common evolution patterns are represented by total resorption (with normalization of the radiological aspect), fibrosis (expressed by the reduction of size and intensity of the opacity which becomes heterogeneous) and calcification. Also, caseification may occur, followed by ulceration, which leads to the formation of a cavity: the primary cavernous lesion. Lastly, the caseous mass may become a tuberculoma (Fig. 5).

2. Lymphangitis (Fig. 2)

Represents the specific inflammation of the lymph vessels, followed by a lymphomonocytes infiltrates. Although rarely detectable, radiographically it appears as discrete linear opacities between the primary lesion and the adenopathy.

3. Adenopathy

It is the most frequent visible radiological element of the primary complex. In small children it may be the only sign. On the radiograph, it is displayed as round or oval opacities, of medium intensity, well defined, which can be situated in the hilum, lateral to the trachea, peri- or intrabronchial. It can be confined to the first lymph node or it can extend to the whole chain, even bilaterally.

The adenopathy lateral to the trachea can widen the superior part of the mediastinum, which becomes wavy, with polycyclic contour. Also, the intrabronchial adenopathy may enlarge the tracheal bifurcation angle.

A favorable outcome of the adenopathy leads to the reduction of intensity and size of the lymph node opacity. The healing process is achieved slower than in the case of other lesions. However, the calcareous deposits formed in the caseous areas will persist forever.

The unfavorable outcome associates with massive caseification, followed by perforation and evacuation in a bronchus. Thus, a lymph node cavern is formed favoring bronchial dissemination of the pathogenic element in the lung parenchyma.

4. Complications

I. Local complications

a. Epituberculosis (perifocal congestion)
It is a nonspecific allergic alveolitis around the primary lesion or adjacent to the adenopathy. It is caused by an increased sensitivity to the Koch bacteria. Radiologically, it manifests as variable sized opacities, homogenous, of low intensity, with blurry and imprecise contours, covering the image of the primary lesion or adenopathy. Compression or obstruction of bronchi may determine atelectasis. When localized next to scisures, epituberculosis may give a triangular opacity with the top towards the periphery (the "Sluka triangle"). The outcome is usually favorable, healing occurring spontaneous or under treatment within weeks.

b. Extensive caseous primary infection

It represents the extension of the specific alveolitis, followed by caseification, the most common form being the caseous pneumonia (Fig. 3,4). Radiologically, this is an opacity of medium intensity, with a segmental or lobar topography. Note that the intensity is always higher than the one produced by a common pneumonia. Should it occupy an entire lobe, the exsudate will spread beyond the scisure, giving the opacity an imprecise contour.

In evolution, the opacity becomes heterogeneous due to the appearance of a number of transparencies, which represent ulcerations that have the tendency to merge, thus making it possible for a large cavern to form.

Also, multiple macronodular opacities may appear near the pneumonic opacity or further away, even in the other lung. These lesions are characteristic for bronchoalveolitis and represent the caseous bronchopneumonia produced by the spread of the disease from the ulcerative areas.

c. Primary lesion/lymph node cavern (Fig. 4)

It is formed by a process of necrosis followed by evacuation at the site of alveolitis in the primary lesion. Radiologically, it is a transparent image, sometimes hydroaeric, that partially or totally replaces the previous image. After the cavern is formed, the process may be continued by the bronchogenic spread of the disease, with the appearance of an area of bronchopneumonia.

d. Bronchial complications (mainly adeno-bronchial fistulae)

Are a consequence of the cavern formation and lead to the spreading of the disease and formation of bronchopneumonia areas.

e. Atelectasis (Fig. 6)
Its formation is closely related to that of the fistulae. The walls of the fistula may over thicken and totally obstruct the bronchus, leading to a complete atelectasis in the lung territory corresponding to that bronchus. If the cause of the obstruction is not eliminated, the atelectasis will become over infected, leading to suppuration and severe fibrosis.

f. Pleural complications (mainly pleuritis and pleural fluid: Fig. 7,8)

Might be fibrous pleuritis or scisuritis, or inflammatory pleural reaction, with fluid buildup either contained or free. The radiological aspect is identical to the pleural involvement of other diseases and therefore is not a specific TB sign.

II. General complications

a. Tuberculous bronchopneumopathy (Fig. 9,10)

The origins of this complication are the evacuated caseous areas. The main lesions are the caseous bronchoalveolitis nodules, with a tendency to cluster and become excavated, concerning small functional territories or covering large lung areas. Radiologically, these lesions are depicted as numerous micro and macronodular opacities, homogenous, of medium intensity, with blurry contours, spread in both lungs or in just one lobe. In time, the opacities tend to merge. Nodule ulceration may lead to hyper transparent images.

b. Haematogenous dissemination (Fig. 11,12,13)

It is the result of a rupture of a caseous focus into the blood vessels. The most notable aspect is that of the miliary spread in acute or chronic forms. Haematogenous spread of the disease is followed by the forming of numerous areas of tuberculous foliculi in the interstitial spaces and alveolar septa. On the radiograph, there are numerous micronodular opacities ("miliaries"), spread in both lungs uniformly or just in the top and middle regions, of equal size, with well-defined borders and of medium intensity. Moreover, when analyzing the radiograph, one should consider the remnants of the primary complex and the pleural involvement, the latter being the result of a simultaneous evolution of the pleural spread. Localized complications may appear, such as focal areas of exudative bronchopneumonia that tend to undergo a caseous and ulcerative processes. General complications consist of the massive spread towards other organs.

The chronic form is defined by the step-by-step development of small nodules, randomly spread in the lungs and evolve into fibrosis.
The discrete dissemination is frequent and consists of a small number of nodules in the top areas of the lungs or retroclavicularly. Although they might be fibrous or calcified when diagnosed, note that these nodules retain their full pathologic potency and can be the cause of a secondary tuberculosis. Thickening of the apical pleura may occur.

c. Lymphatic dissemination

Rarely seen on the radiograph. Depicted as linear opacities, it is the result of specific lymphangitis. It may present a spoke-wheel pattern around the hilar lymph nodes or may link these lymph nodes to lesions found in the lung parenchyma.

5. Sequelae

The pulmonary sequelae are represented by processes of fibrosis, that replace various portions of the lung parenchyma. The healing of the caseous zones are followed in many cases by calcifications, and the fibrosis of the productive nodules leads to small sized irregular star-shaped form scars.

The bronchial sequelae comprise various forms of bronchial stenosis and dilations due to the near-by fibrosis.

The lymph nodes sequelae are mainly calcifications while the pleural sequelae are visible on a radiograph as pleural thickening affecting mainly the apical, basal and scisural pleura, with formation of adherences and obstruction of the costo-diaphragmatic sinuses.

SECONDARY TB

It is the consequence of the reactivation of fibro-calcareous lesions determined by primary tuberculosis or the consequence of a secondary infection with the Koch B.

Clinically, it has a cyclic evolution in which the symptomatic phases alternate with those without symptoms.

It is important to note the polymorphic aspect of the disease (Fig. 19), since one can encounter several forms such as exudative-caseous, alveolar and bronchoalveolar processes, with a tendency to excavate, and also productive and fibrous lesions with systematic and/or deforming characteristics.
During each active phase, the disease extends mainly within bronchi, affecting larger areas with each step and progressing from the top to the bottom of the lungs.

1. Secondary TB at its debut

a. Minimum reactive TB

It represents the continuation of the development of nodular foci, located more frequently in the top parts of the lungs and created during the primary phase of the disease.

On the radiograph, the intensity of the fibrous nodules diminishes, along with the loss of their well defined borders. A focus of steady growing alveolitis forms around the scar. It can evolve towards fibrosis or caseification, ulceration and bronchogenic dissemination.

b. Assman infiltration (Fig. 14)

On the radiograph, it appears as a relatively round opacity, 2-4 cm in size, with alveolitis features, placed usually in the subclavicular regions. In the absence of caseification it evolves towards total resorption. Other possible outcomes are fibrosis with partial calcification, caseification followed by the formation of a fibrous shell forming a tuberculoma, or ulceration with the appearance of an early cavern that can lead to bronchogenic spread. Also, it must be differentiated from tumors, metastasis or hydatid cysts.

c. Nebulous infiltration (Raedeker)

It is represented by a diffuse opacity, with an irregular shape, heterogeneous, suggesting alveolitis with fibrous and productive processes. It can evolve in the same way as the Assman infiltration. To differentiate it from tumors, infarction or a nonevacuated abces, one must consider the clinical presentation, the laboratory findings and the evolution of the disease after anti-TB medication.

d. Pseudopneumonic infiltration (Fig. 18)

It appears as an opacity with an alveolitis aspect, confined to a segment or lobe. It becomes more opaque and heterogeneous due to the formation of multiple cavities.

e. Perihilum infiltration
It is an alveolitis process developed in the vicinity of the hilum, as a result of an adenobronchic fistula and the discharge of the caseous content of the lymph node into a nearby bronchi.

2. Ulcerous-fibrous-caseous TB

It is the most common form of secondary TB. Bronchogenic dissemination creates the premises for alveolitis and bronchoalveolitis foci to develop, with an evolution towards ulceration or fibrosis. The simultaneous presence of lesions with an exudative-caseous, productive, ulcerative and fibrous characteristics, as well as pleural implications, confer a great degree of disease polymorphism. The process extents into previously normal parenchyma. Radiologically, there are opacities of low and medium intensity, variable in size, with an imprecise contour, sometimes confluent. Foci of fibrosis or excavation lead to heterogeneous aspects. The most common sign is that of the tuberculous cavern, which can take various forms (Fig. 17).

The recent cavern (Fig. 15) appears as a transparent area situated inside the pneumonic or bronchopneumonic opacity created by an exudative-caseous process.

The elastic cavern is a ring shaped image in the normal lung parenchyma that surrounds a round hipertransparent area. It is the result of the total evacuation of a caseous zone.

The rigid cavern (Fig. 16) has a thick ring shaped wall, of medium intensity, produced by the proliferation of granulary tissue in the periphery of the necrotic area.

The fibrous cavern is surrounded by a thin wall, of high intensity.

If the caseous content is incompletely evacuated, the cavern may present a hydroaeric image. The cavern may frequently be linked to the hilum by linear opacities, which represents the inflammatory or fibrous involvement of the draining bronchus.

The tuberculoma (Fig. 5) ("caseoma") results from the surrounding of a caseous mass by specific granulary tissue at its periphery. Radiologically, it is a round opacity of 1-2 cm, well defined, of medium intensity, homogenous or with multiple calcifications. The image may persist indefinitely, or it can become a cavitary image due to excavation.

The productive lesions are represented by micronodulary opacities, well defined, of medium intensity.
The fibrous lesions are irregular linear opacities, of high intensity, that replace the parenchyma (in the deforming disease) or are placed on the trajectory of the peribronchovascular and perilobular interstitium (in the systemic disease). They may be accompanied by bronchial dilatations and rejections, altering the place of the mediastinal organs, hilum, and diaphragm. The pleural involvement consists of a variable sized pleuritis with calcifications; the extensive forms may retract the thoracic walls.

3. Fibrous TB (Fig. 20, 21, 22)

As the name explains it, it is marked mainly by fibrous lesions. The disease is reactivated at a local level and the initial exudative processes do not evolve into caseification but instead lead to an abundance of conjunctive tissue.

There are various ways of evolution. One possibility is that it can develop peribronchovascular or perilobulary, leading to lineary opacities, that may be accompanied by bronchial dilatations or emphysema. Another possibility is the apical form, with fibro-calcarous nodules of various shapes and high intensity, placed in the apical regions and accompanied by a thickened pleura. The third form comprises fibrous processes that completely occupy a lobe, giving it an intense opacity, heterogeneous, reducing its volume and leading to the traction of the nearby organs and structures. Lastly, the whole lung can be caught in the fibrous processes, leading to fibrothorax. This is usually associated with marked thickening of the pleura. On the radiograph, the lung is intensely opacified, heterogeneous, with forms of retraction concerning the mediastinum, diaphragm and thoracic wall.

4. Evolution towards cancer (Fig. 8)

The scar resulting after the healing process is a modified type of pulmonary tissue that underwent a lot of stress and had its evolution altered. Therefore, this can be the site of primary tumorigenesis and can be very difficult or impossible to diagnose in the early stages due to the fact that it is masked by the very opaque sequelae.

Images for this section:
**Fig. 1:** Primary lesion seen in the right hemithorax with calcification. On the left, a cavern with fluid level can be easily seen.
Fig. 2: Lateral view of primary TB. Note the primary lesion and the lymphangitis linking it to the hilum.
**Fig. 3:** Close up of tuberculous pneumonia in the right lung; in this case, it is associated with fibrous complications.

**Fig. 4:** Pneumonic process partially healed; the rest underwent caseification and begins to show a hidroaeric level.
Fig. 5: Tuberculoma; note that even though initially calcified, it begins to present a slight process of caseification
Fig. 6: Atelectasis: rare complication of a tuberculous process
Fig. 7: Confined pleural effusion: a common complication of tuberculosis
**Fig. 8**: Pleural effusion at the base of the right lung; fibrous and calcarous sequelae within the entire area of the lung. The subclavicular opacity with the spiked inferior border causes suspicion for malignancy. Proved to be an adenocarcinoma formed on a post-tuberculous scar.

**Fig. 9**: Bronchogenic spread of the infection, resulting in a bilateral bronchopneumopathy.
Fig. 10: Complicated primary TB in a post partum patient. Dissemination in both lungs; the radiograph shows pneumonic and bronchopneumonic lesions.
Fig. 11: Milliary TB
Fig. 12: Remember that milliary TB must be differentiated from other diseases. This patient had metastases distributed throughout both lungs.
Fig. 13: Milliary TB (close up)
Fig. 14: Subacivular reactivation of TB. The infiltrate evolves towards heterogeneity; the arrowheads mark the wall of a future cavern.

Fig. 15: Close up of a recent cavern situated in tuberculous process that is undergoing healing.
**Fig. 16:** Old school tomographic section of a rigid cavern; note the thick wall with the draining bronchi.

**Fig. 17:** Secondary TB in the top and middle parts of the right lung. Note the opacified upper portion and the hidroaeric level inside the cavern.
Fig. 18: Secondary TB disseminated in both lungs. The caverns can merge with one another.
**Fig. 19:** Secondary TB with polymorphic aspect: bronchogenic dissemination, caverns, pleural involvement and heterogenous aspect due to caseification.
Fig. 20: Fibrous and calcarous sequelae.
Fig. 21: Fibrothorax
**Fig. 22:** Fibrous lesions that deform the anatomy of the left hemithorax
Conclusion

TB incidence is increasing and drug resistant forms are more frequent. A challenge even to experienced radiologists, TB has more forms that any other disease therefore young residents should take this diagnosis into account when confronted with an atypical chest image.

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