UIP, NSIP and OP patterns. Benefit of ancillary findings

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Learning objectives

1. To describe the features and diagnostic criteria of each pattern according to the 2013 ATS/ERS guidelines.

2. To show some ancillary findings that may help to differentiate an idiopathic interstitial lung disease from the other one with a known cause.
Background

The appearance of interstitial lung disease varies greatly in the thoracic computed tomography studies. UIP, NSIP and OP patterns are three of the most common forms of presentation. Despite many of the cases are idiopathic, the exposure to organic or inorganic dust or lung involvement in the setting of collagen vascular disease may produce identical parenchymal manifestations. The existence of some ancillary findings mixed with the main pattern of disease may help to differentiate the idiopathic causes from the ones with underlying aetiology.

Firstly, we describe the features and discuss the diagnostic criteria of UIP, NSIP and OP patterns, according to the 2013 ATS/ERS guidelines. Proper pattern recognition is the basis for further investigations. Whilst UIP pattern is more often associated with IPF, lung reaction following the inhalation of organic (i.e. chronic hypersensitivity pneumonitis) and inorganic dust (asbestosis) may be indistinguishable. NSIP pattern usually is related to collagen vascular diseases or pulmonary drug reactions. A subacute hypersensitivity pneumonitis may also be difficult to differentiate from an idiopathic NSIP. OP is frequently seen as a reaction to a previous infection, fume inhalation or, as in case of NSIP, related with collagen vascular diseases and drug reactions. A single disease may manifest with different patterns, so the presence of a specific subtle finding may be the key to work out the diagnostic. The ancillary findings that we discuss in this poster are:

- Calcified pleural plaques
- Peripheral small centrilobular nodules.
- Spared pulmonary lobules
- Oesophageal dilatation
- Hyperdense consolidation
- Bronchiectasis in areas of little or absent fibrosis
The different idiopathic interstitial pneumonias (IIP) are classified accordingly their histologic features, being each histologic pattern associated with a characteristic CT pattern (1). Therefore, CT pattern recognition is of special relevance in the assessment of interstitial lung disease. Table 1 shows the revised American Thoracic Society/European Respiratory Society classification of idiopathic interstitial pneumonias. (2)

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It is important to note that, although these pathologic and radiologic criteria are used to define the IIP, identical patterns of disease may be seen as a manifestation of a known underlying cause. Regarding the diagnostic approach to an interstitial lung disease there are two steps to consider: the first one is to recognize the radiologic pattern of disease, the second one is to assess which known etiologies may relate with that pattern. Only when no specific cause may be found an IIP should be diagnosed (2).
Although most of the times it is not possible to differentiate an idiopathic pattern from other identical with a known cause, in some cases there are ancillary findings that can be useful on this purpose. In this poster we focus on usual interstitial pneumonia, nonspecific interstitial pneumonia and organized pneumonia patterns. First, we describe the features and diagnostic criteria, according to the 2013 ATS/ERS guidelines. Thereafter we comment the different entities that may lead to that pattern and show some radiological aspects that, when present, may help to suggest a discrete etiology.

**UIP:**

UIP is the radiological pattern associated with idiopathic pulmonary fibrosis (IPF). IPF is a specific form of chronic fibrosing interstitial pneumonia of unknown etiology, limited to the lung and associated with the histologic appearance of usual interstitial pneumonia on surgical lung biopsy (3). It occurs most frequently in patients older than 50 and the symptoms (progressive dyspnea, cough, weight loss, finger clubbing) may precede the diagnosis by 6 months. Pulmonary function tests show restriction with a reduced diffusing capacity. The prognosis is poor (5-year survival rate of 20% with mean survival of 2 to 4 years from the onset of the symptoms) and there is no effective treatment (4).

The histological hallmark is characterized by the presence of temporal and spatial heterogeneity. Temporal heterogeneity manifests as the presence of fibrotic lesions of different stages: fibroblastic foci, mature fibrosis and honeycombing. Spatial heterogeneity denotes a patchy lung involvement with normal lung adjacent to severe fibrotic lung (5).

The typical manifestations of UIP/IPF pattern at High Resolution CT consist of peripheral reticulation and honeycombing with subpleural and basal predominance (apicobasal gradient). Traction bronchiectasis and architectural distortion, which reflect fibrosis, are often associated and prominent. Volume loss is seen in cases of more advanced fibrosis. Ground glass opacities are frequent but less extensive than the reticular pattern and not seen out of the areas of fibrotic changes. Honeycombing is defined as clustered cystic airspaces, typically of 3-10 mm and comparable diameters, but occasionally as large as 2.5 cm.
Fig. 1: UIP. CT scan of a patient with UIP. Transverse plane in lung window shows peripheral reticulation in right middle lobe, lingula and lower lobes. Honeycombing is seen in both lower lobes. Note traction bronchiectasis in right lower lobe (blue arrows).


According to the 2013 ATS/ERS guidelines, in the absence of other findings inconsistent with UIP, the diagnostic of IPF may be made when reticulation and honeycombing with peripheral and basal distribution are seen.

The findings that should make to consider an alternative diagnosis are:

• Micronodules,
• Air trapping,
• Non-honeycomb cysts,
• Extensive ground glass opacities,
• Consolidation,
• Peribronchovascular predominant distribution.

Although UIP pattern is most often associated with IPF it may be result of other etiologies, such as: asbestosis, end-stage hypersensitivity pneumonitis, drug toxicities or collagen vascular disease.
**Fig. 2:** Typical UIP pattern distribution. CT scan. Transverse plane shows the characteristic apicobasal gradient commonly seen in UIP. There is a relatively small amount of honeycombing in the anterior region of both apex (1a). In the mid parts of the lungs (1b) more extensive fibrotic changes are seen (also of anterior distribution), with honeycombing and traction bronchiectasis. The most severe extent is present in lung bases with large areas of honeycombing extending from the subpleural regions of the lung.

**References:** Hammersmith Hospital. Imperial College Healthcare. NHS trust. London. 2014

**NSIP:**

NSIP was definitely accepted as distinct entity among the idiopathic interstitial pneumonias in 2008 (2).

This entity is known to clinically manifest in younger patients than IPF (around 40-50 years old) and has no clear gender predilection. The symptoms are similar to those in IPF (dry cough, worsening dyspnoea over moths, fatigue) and, as in IPF, most patients show a restrictive ventilatory defect on lung function tests. In contrast to IPF cigarette smoking is not a risk factor in the development of NSIP (1,2).

The prognosis is better than that of UIP/IPF (6,7) and the treatment is based in the use of corticosteroids in combination with cytotoxic drugs (cyclophosphamide, cyclosporin). This leads to a stabilisation or improvement in the majority of cases.

Histologically NSIP is recognized by its spatial and temporal homogeneity which varying amounts of fibrosis and inflammation. Depending of which component predominates a "cellular" and "fibrotic" form of NSIP are distinguished. Patients with predominant fibrosis have poorer prognosis than patients with predominant inflammatory findings (7). The fibrosis expressed in NSIP, due to its temporal homogeneity it is always of the same age.

On HRCT NSIP shows lower lobe predominance with central, diffuse or sub-pleural distribution. Ground glass is seen in approximately half of the patients and reticulation and traction bronchiectasis are common. In cases with predominant reticulation and peripheral distribution the distinction between UIP and NSIP may be challenging. In those cases a relative sparing of the immediate subpleural zone of lung is a useful sign, seen in approximately 20% of NSIP cases (6).
**Fig. 3:** CT scan of a patient with NSIP. Transverse plane of lower lobes in lung window shows a widespread combination of peripheral reticulation and ground-glass opacity. Tractional dilatation of the small airways (asterisks) is seen, consistent with an established interstitial fibrosis. Note relative sparing of the subpleural regions (arrows). **References:** Hammersmith Hospital. Imperial College Healthcare. NHS trust. London. 2014
Fig. 4: CT scan of a patient with fibrotic NSIP. Transverse plane of lower lobes shows a major degree of fibrotic changes than the figure 3. Extensive tractional bronchiectasis and pulmonary distortion. Diffuse ground-glass opacity and a relative sparing of the subpleural regions are also seen.


Importantly, NSIP pattern not only occurs as an idiopathic entity but also in a diverse number of conditions such as collagen vascular disease, hypersensivity pneumonitis and drug toxicity.

OP:

Organizing pneumonia is characterized by its histological substrate: intra-alveolar fibrosis, resulting from organization of inflammatory exudates. An initial epithelial alveolar injury leads to migration of fibroblasts inside the alveoli and production of connective matrix (8).
In the first stages intra-alveolar inflammatory cells and fibrin deposits are found. The inflammatory component progressively disappears as the fibroblasts proliferate and the bundles of connective tissue are produced. Often, mild interstitial inflammation is also present.

OP is a lung parenchyma response that might be related to an extensive list of causes. One of the most common is a previous respiratory infection. Other frequent entities that may be associated to a pattern of organizing pneumonia are: connective tissue disorders, drugs reactions, organ transplantation or immunological disorders.

As many cases are secondary, the use of the generic term "OP" is preferred, with qualifiers when the possible etiology is known (for example, OP associated with rheumatoid arthritis) (6). The term Cryptogenetic Organized Pneumonia (COP) is reserved to those cases considered idiopathic.

This entity clinically manifests as subacute mild flu-like illness with of relatively short duration (typically, less than 3 months). The typical symptoms are fever, cough, malaise, progressively mild dyspnoea and weight loss. A mild or moderate restrictive ventilatory defect is the most common abnormality at spirometry. When OP is suspected the bronchoalveolar lavage (BAL) is a very useful technique. It helps in excluding other diagnostics with may have similar imaging findings (lymphoma, broncholoalveolar carcinoma) or determining a cause of organising pneumonia.

Radiologically, OP may manifest in many different ways. The most typical consist in multiple and bilateral areas of air space consolidation with peripheral distribution and often migratory. Their size varies from a few centimetres to a whole lobe, and an air bronchogram is often present in consolidated opacities (8).
Fig. 5: Organising pneumonia. CT scan showing a typical organizing pneumonia pattern with multiple and bilateral areas of peripheral consolidation. Tractional dilatation of the airways is seen, indicating associated fibrosis (blue arrows). Some of the consolidative areas show a perilobular distribution (red arrows), a sign characteristic of OP.

References: Radiology, Hospital de la Santa Creu i Sant Pau, Hospital de la Santa Creu i Sant Pau - Barcelona/ES

Other presentations of OP in HRCT include a solitary focal mass (usually undistinguishable from lung cancer only by imaging), a nodular pattern in form of well defined "acinar" nodules (approximately 8mm) or ill-defined micronodules (<4mm), a bronchocentric pattern in form of consolidation of peribronchovascular distribution, and a perilobular pattern with opacification around the periphery of individual secondary lobules (9).
Fig. 6: Peribroncovascular pattern of organizing pneumonia. Transverse CT scan showing multiple areas of consolidation of peribroncovascular distribution. Air bronchogram is seen within the areas of consolidation.

References: Radiology, Hospital de la Santa Creu i Sant Pau, Hospital de la Santa Creu i Sant Pau - Barcelona/ES

Corticosteroid treatment in COP results in rapid clinical improvement and clearing of the opacities on chest imaging without significant sequelae. However, relapses are common upon stopping or reduction of corticosteroids, thus often leading to prolonged treatment (8).

ANCILLARY FINDINGS

Pleural abnormalities:
Pleural plaques consist of discrete areas of hyaline or calcified fibrosis localized on the parietal pleura. They are considered highly specific for asbestos exposure. In the setting of a fibrotic lung disease the diagnosis of asbestosis should be raised when pleural plaques are present (10).

Fig. 7: Calcified pleural plaques. CT scan. Transverse plane in bone window at the level of pulmonary artery bifurcation shows multiple and bilateral calcified pleural plaques. Note the enlarged pulmonary artery, a sign consistent with pulmonary hypertension.

Fig. 8: Calcified pleural plaques. Reformatted coronal CT scan in bone window shows multiple and bilateral calcified pleural plaques. Note the calcified plaques in diaphragmatic pleural surfaces (blue arrows), typical of asbestos exposure.


When focal fibrosis involves the parietal and visceral pleura parenchymal bands are formed. They consist in peripheral linear opacities extending from the lung parenchyma to the visceral pleura, usually being 1-3mm thick and up to 5cm long. The crow's feet sign consist in the presence of various parenchymal bands irradiating to a common point in the pleural surface. If the fibrosis of the visceral pleural advances it may cause incomplete inflation or shrinking of the underlying lung, leading to the formation of a rounded atelectasis.
Fig. 9: Parenchymal bands related to a calcified pleural plaque. CT scan. Transverse plane at the level of right middle lobar bronchus and bifurcation of left main bronchus. The lung window (6a) shows three parenchymal bands arising from a calcified pleural plaque and extending perpendicular to the pleural surface. This finding is typical of asbestos exposure. Mediastinal (6b) window shows better the large calcified pleural plaque from which the parenchymal bands arise.


Besides focal pleural thickening, asbestos exposure may produce diffuse pleural thickening. This is no so specific as may be seen as a result of collagen vascular disease or drug exposure.

Small centrilobular nodules

Akira et al. (11) described that, apart from pleural changes, there were other combinations of signs of such utility in discerning between asbestosis and IPF. Thus, the presence of centrilobular opacities in the subpleural region was found to be a highly reliable sign, present in patients with asbestosis and absent in patients with IPF. Instead, dilated bronchioles in subpleural regions (bronchiolectasis) were much more frequent in IPF.
Fig. 10: UIP pattern in asbestosis. CT scan. Lung window of transversal plane at the same level as Figure 4. There are signs of interstitial disease in keeping with UIP, such as peripheral reticulation and small amount of honeycombing (asterisk). Subtle small subpleural nodules (blue circles) are present in some areas. These nodules are not typical in IPF and are more consistent with asbestosis.

Fig. 11: Small subpleural nodules. CT scan. Transversal plane of same patient as in figure 8 with small subpleural nodules adjacent to the major fissure of the right lung (blue circle). Note the posterior calcified pleural plaques (blue arrows).


**Spared secondary pulmonary lobes / air trapping**

Even when large fibrotic changes (peripheral reticulation, traction bronchiectasis, architectural distortion and honeycombing) are present, the existence of areas of decreased attenuation and vascularity or air trapping on expiratory CT are not typical from IPF and should lead to an alternative diagnosis.
These areas of low attenuation or air trapping represent indirect signs of bronchiolar obstruction, which is one of the histologic features of hypersensitivity pneumonitis (extrinsic allergic alveolitis) (12). Often, these areas adopt a lobular distribution and, mixed among multiple lobules affected by fibrotic changes lead to the term **spared pulmonary lobules**.

![Subacute hypersensitivity pneumonitis. Transverse CT scan that shows bilateral patchy areas of ground glass opacity interpersed with pulmonary lobes of normal appearance (spared pulmonary lobules).](image)

**Fig. 12:** Subacute hypersensitivity pneumonitis. Transverse CT scan that shows bilateral patchy areas of ground glass opacity interpersed with pulmonary lobes of normal appearance (spared pulmonary lobules).

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Hypersensitivity pneumonitis stands for an inflammatory pulmonary reaction caused by the inhalation of an allergen (organic or inorganic: microbes, animal or plant proteins, and certain chemicals) by sensitized individuals. Depending on the allergenic burden inhaled and the duration of the exposure an acute, subacute and chronic forms are distinguished. In chronic HP after weeks or months of continuous exposure, lung fibrosis develops and the CT appearance may be practically indistinguishable from a fibrotic form of NSIP or even a UIP/IPF. The presence of spared pulmonary lobules or air trapping in expiratory CT should make us think of end-stage HP.
Esophageal dysmotility is common in patients with scleroderma and dermatopolymyositis (14). Asymptomatic esophageal dilatation may be identified in 40% to 80% of patients with scleroderma, with an esophageal luminal diameter ranging from 12mm to 40mm in the coronal plane (15). Esophageal dysmotility may lead to aspiration pneumonia.

**Fig. 13**: NSIP pattern associated with esophageal dilatation. Transverse CT scan showing extensive, diffuse and bilateral ground glass opacities with sub pleural sparing and tractional airways dilatation. Findings consistent with NSIP. Note the important esophageal dilatation (blue arrow). This finding suggest the diagnosis of systemic sclerosis (scleroderma).

Hyperdense consolidation

Multiple areas of consolidation are a common and nonspecific finding in thoracic radiology with many different causes, acute and chronic (4). The existence of high attenuation inside the area of consolidation at a noncontrast enhanced CT is a useful sign that allows narrowing considerably the diagnostic alternatives (16). In this setting, the presence of hyperdense peripheral consolidations, signs of interstitial fibrosis and high density of the liver or spleen is characteristic of amiodarone exposure.

Fig. 14: Amiodarone related interstitial lung disease. Figure 13a. CT scan in lung window that shows a notably enlarged heart with wires of a implantable cardioverter defibrillator. In the lung parenchyma there is interstitial thickening, patchy areas of ground glass opacity and associated traction bronchiectasis. Figure 13b. CT scan in mediastinal window that shows an overall increased attenuation of the liver consistent with amiodarone impregnation.


Amiodarone is a triiodinated drug used in the treatment of tachyarrhythmias (37% iodine by weight). Typically the drug accumulates inside macrophages (the liver, spleen and lung are rich in these cells) and pneumocytes type II of the lung. The most characteristic histological feature is the presence of foamy macrophages with lamellar inclusion bodies (17).

The overall pulmonary toxicity occurs in approximately 5%-10% of patients, being the most important risk factors an age >60 years, a treatment duration longer than 2 moths and a daily dose >400mg (18,19). NSIP is the most common manifestation of
amiodarone-induced lung disease. OP is less common and typically occurs in association with NSIP. Pleural effusion may be present as result of associated pleural inflammation (18).

**Bronchiectasis in areas of little or absent fibrosis**

Traction bronchiectasis is a common finding in fibrosing interstitial lung disease. As described previously, both UIP and NSIP patterns may show architectural parenchymal distortion and bronchiectasis as signs of fibrosis. In both cases the bronchial dilatation is seen in areas of affected lung parenchyma (there are other signs of disease such as peripheral reticulation or ground glass opacity). Nonetheless bronchial dilatation can be an isolated sign of disease.

Bronchiectasis are common in patients with rheumatoid arthritis (up to 30%) and, with less frequency, Sjögren's syndrome and systemic lupus erythematosus (14). They are typically cylindrical and located in the lower lobes. Interstitial lung disease is rare in systemic lupus erythematosus (its primary thoracic manifestations are serosal involvement and infection) and Sjögren's syndrome typically relates with lymphocytic interstitial pneumonia (20). Hence, among these three entities, rheumatoid arthritis is the most likely to show a UIP or NSIP patterns along with "non tractional" bronchiectasis. In this setting, the presence of cylindrical bronchiectasis in areas of otherwise normal parenchyma should raise the rheumatoid arthritis.
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Conclusion

Identifying the specific pattern is of paramount importance in the diagnostic approach to diffuse interstitial lung disease. Despite most of the cases are idiopathic the identification of ancillary findings apart from the main pattern of disease may help to distinguish that cases with an underlying cause.
References


