Bronchiolitis obliterans Organizing Pneumonia

Trying to Answer an Intriguing Question

Carlos Agustí Antoni Xaubet

Servei de Pneumologia i Alergia Respiratòria, Institut Clínic de Pneumologia i Cirurgia Toràctica, Hospital Clínic, Barcelona, Spain

Organizing pneumonia is a well-defined pathological entity characterized by the formation of organized granulation tissue involving alveolar spaces and ducts [1]. As a pathological process, organizing pneumonia has long been recognized to be associated with various common pathologies in 'post-mortem' lung examination, such as lung carcinoma or slow resolving pneumonia. In these cases, organizing pneumonia seems to represent an indolent inflammatory extension of the primary pathological process. Grinblat et al. [2] and Davidson et al. [3] were the first researchers to notice that organizing pneumonia is also the pathological hallmark of a distinctive clinical syndrome, currently called bronchiolitis obliterans organizing pneumonia (BOOP) since the process may also extend into the bronchiolar lumen [4]. The clinical syndrome of BOOP is characterized by fleeting and patchy radiological infiltrates associated to a myriad of symptoms, such as cough, malaise, fever and dyspnea. Although many cases of BOOP are cryptogenic, this syndrome can also be associated with drug toxicity, respiratory infections or connective tissue disorders. Most patients with BOOP show an impressive response to corticosteroids with complete resolution after several weeks of treatment [5].

Respiratory clinicians must be aware of the clinical syndrome of BOOP and its potential associations since

early diagnosis and treatment prevent any progression of the disease. However, we also have to keep in mind that 'clinical' diagnosis can be risky since corticosteroids are contraindicated in some of the diseases included in the differential diagnosis of BOOP. It is then imperative to confirm this condition histologically [5]. The gold standard for the diagnosis of BOOP is video-assisted thoracoscopic lung biopsy, but in some instances transbronchial biopsies may be sufficient if the samples are conclusive and the clinical picture is typical. A compatible differential cell count of the BAL population showing increased percentage of lymphocytes, a decreased ratio of CD4 to CD8 T cells, as well as moderate neutrophilia and eosinophilia may help to establish the diagnosis [5]. In the present number of *Respiration*, Watanabe et al. [6] present 4 cases of BOOP presumably related to thyroid diseases. Although this association can be questioned in some of the cases presented and other causative links can be inferred (multiple myeloma, treatment with thiamazole, slow resolving pneumonia), the authors judiciously speculate on the potential mechanisms that can facilitate the apparition of BOOP in patients with thyroid disorders. Undoubtedly, the awareness of potential triggers of BOOP can help to answer an intriguing question: Why does organizing pneumonia, which is usually a 'wound-

healing' reaction associated to chronic pathologies of the lung, sometimes turn into a serious disease with distinctive symptoms, a characteristic radiographic pattern, progressive evolution and specific treatment? The creation of an experimental model of BOOP, suggesting genetic pre-

disposing host factors, and the development of active biopathological investigations highlighting the importance of different inflammatory cytokines in the genesis of this condition will undoubtedly help to answer this question [5].

References

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