The Point of Crossover of Infectious Pneumonia and Interstitial Pneumonia

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Key words: aseptic pneumonitis, crossover phenomenon, cryptogenic organizing pneumonia

(Inter Med 48: 1125, 2009)
(DOI: 10.2169/internalmedicine.48.2283)

Reading a short article of the last issue of this journal (1), most readers will suppose that the clinical diagnosis of this patient will be one of cryptogenic organizing pneumonia (COP). However, I believe that the author’s concept as described in their Figure 2 is very important. When doctors see infiltrating shadows on the lung as demonstrated in their Figure 1A, they first check for causal bacteria and then start antibiotics. Within a couple of days, a doctor will obtain the data profile about the pathogen including cultivation and sensitivity tests of the bacteria which is thought to be growing in the pneumonia lesion. However, like the first doctor who was puzzled over this aggravating shadow, most doctors will continue to check for other possible responsible microbes like mycoplasma and will change the antibiotics.

Instead, in this short article, the intravenous steroid is expected to pacify the overzealous and expanding reaction of the immune system in the face of inflammation. Although the efficacy of immune globulin is controversial, the author used it to neutralize several kinds of epitopes, including unknown antigen determinants that supposedly triggered the inflammation.

Despite the wide clinical use of antibiotics, many elderly people die from pneumonia. Also, about 30 to 50% of patients have no identifiable pathogen, despite there being a clinical impression of bacterial pneumonia. Furthermore, although the term aseptic meningitis is frequently used in meningitis, most meningitis is caused secondary to respiratory infections through the route of droplet infection, the term “aseptic pneumonitis” has not been described before.

This short paper also describes the “crossover phenomenon” in infectious disease. As the author mentioned, the course of an infectious incidence in a living being could not be as simple as Pattern I, which was first reported and established as a golden rule by Robert Koch. A rule which many doctors have since been ensnared. By contrast, immune malfunction should be a prime culprit; it is not always the microbe itself that triggers inflammation in a deteriorating case of an infectious incident. This is especially true in the elderly, whose immune systems are prone to deviate from normal functioning.

In this case, we should also consider the effects of any drugs during catching a cold in the development of “aseptic pneumonitis” because there is no evidence of pathogens before the occurrence of “aseptic pneumonitis” and the chest X-ray finding in the article seems to be the pattern of COP which is also caused by a reaction to drugs. In addition, I believe that to estimate the timing of the crossover might be difficult in clinical situations.

The concept of “crossover phenomenon” proposed by this article seems reasonable. Without consideration of this crossover phenomenon, the argument concerning infectious diseases, including interstitial pneumonia, cannot be settled.

Reference