PROCOAGULANT ACTIVITY OF ALVEOLAR MACROPHAGES IN INTERSTITIAL LUNG DISEASES

Nobuyuki Kobayashi, Haruhito Sugiyama, Makoto Dohi, Matsunobu Suko, Hirokazu Okudaira, Terumasa Miyamoto
Department of Medicine and Physical Therapy, University of Tokyo School of Medicine, Tokyo, Japan

It is suggested that coagulation abnormalities may be implicated in the pathogenesis of fibrotic processes in the lung (1). In this study, we examined the procoagulant activity (PCA) of alveolar macrophages in patients with interstitial lung disease (ILD) associated with collagen vascular diseases (CVD).

MATERIALS AND METHODS

Ten patients with progressive ILD (group A), 12 patients with non progressive ILD (group B) associated with CVD and 8 healthy subjects (group C) were studied. They were all females and non-smokers. Alveolar macrophages recovered by bronchoalveolar lavage (BAL) were separated into 5 density fractions on discontinuous Percoll gradients. The PCA of alveolar macrophages was measured with a one-stage coagulation assay (2). The PCA was expressed as human placental thromboplastin equivalents for normal human plasma.

RESULTS AND DISCUSSION

The PCA of unfractionated alveolar macrophages in group A was significantly greater than that in groups B and C (Fig.1). In contrast to the predominance of high-density macrophages found in groups B and C, the majority of macrophages in group A consisted of low-density subpopulations Procoagulant activity in ILD (Fig.2). The PCA of low-density macrophages in group A was significantly greater than those in groups B and C (p<0.01). In this study, we observed that the alveolar macrophages in patients with progressive ILD had increased PCA and this was due in part to the increase in the low-density macrophage subpopulation, which had greater PCA. Although the role of fibrin deposition in the development lung fibrosis remained to be defined, the activation of the coagulation pathway may contribute to the progression of interstitial fibrotic lung diseases.
Fig. 1. PCA of unfractionated alveolar macrophages.

Fig. 2. Density distribution of alveolar macrophages.

REFERENCES