Repeated thoracenteses affect proinflammatory cytokine, vascular endothelial growth factor and fibrinolytic activity in pleural transudates. 鍾啓禮

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摘要

Abstract

Background: Repeated thoracenteses is indicated in patients with refractory, symptomatic transudative effusions. However, their effect on cytokines and fibrinolytic activity in pleural transudates remains unclear.

Methods: Twenty-one patients with symptomatic, large amount of free-flowing transudative effusions caused by heart failure were studied. Thoracentesis with drainage of 500 mL of pleural fluid per day was done for 3 consecutive days (days 1 to 3). Pleural fluid characteristics, tumor necrosis factor (TNF)- α , interleukin (IL)-1 β , IL-8, vascular endothelial growth factor (VEGF), tissue-type plasminogen activator (tPA), and plasminogen activator inhibitor type 1 (PAI-1) were measured during each tap. Chest ultrasonography was done on day 6 to detect the fibrin strands in pleural effusion and the outcome of effusion was evaluated within 7 days after repeated thoracenteses.

Results: Effusion levels of lactate dehydrogenase, neutrophils, TNF- α , IL-1 β , IL-8, VEGF, and PAI-1 increased significantly during repeated thoracenteses. Furthermore, the values of PAI-1 and PAI-1/tPA obtained on days 2 and 3 were highly correlated with those of TNF- α , IL-1 β , IL-8, and VEGF. On day 6, pleural fibrins were observed on chest ultrasonography in 6 patients (29%, fibrinous group) but were absent in the remaining 15 patients (nonfibrinous group). Compared with the nonfibrinous group, the effusion levels of TNF- α , IL-1 β , VEGF, and PAI-1 on day 2 and day 3, and recurrence of symptomatic effusion after repeated thoracenteses were significantly higher in fibrinous group.

Conclusions: Repeated thoracenteses may induce local release of proinflammatory cytokines, VEGF and PAI-1, which may result in fibrin deposition and impair resolution of pleural transudates.