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Vascular endothelial growth factor in patients with acute asthma

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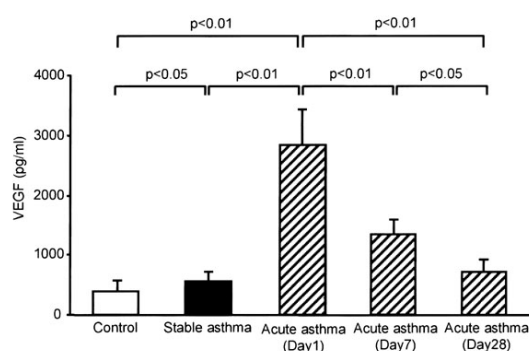
Bronchial asthma is a chronic inflammatory disease of the airways that is characterized by airway remodeling. The histologic characteristics of chronic inflammation include angiogenesis, increased connective tissue deposition, and cellular proliferation of myofibroblasts.¹ There is an increase in vessel area in bronchial asthma.² Vascular endothelial growth factor (VEGF; vascular permeability factor) is one of the most potent proangiogenic cytokines, and it plays a central role in mediating the process of angiogenesis. Inflammation of the airway mucosa of asthmatic patients is usually accompanied by increased vascular permeability and plasma exudation. VEGF apparently increases microvascular permeability by enhancing the functional activity of vesicular-vacuolar organelles so that plasma proteins can leak into the extravascular space.³ In the airways, the plasma exudate can readily pass the inflamed mucosa and thus reach the airway lumen through the leaky epithelium. Plasma protein leakage induces a thickened, engorged, and edematous airway wall, resulting in the narrowing of the airway lumen observed in asthma exacerbations. In addition, plasma can also traverse the epithelium and collect in the airway lumen. Plasma exudation can compromise epithelial integrity, and its presence in the lumen can slow ciliary function and thus reduce mucus clearance.^{4 5}

Plasma proteins can also promote the formation of viscid luminal plugs of exudate mixed with mucus and with inflammatory and epithelial cells. However, no data are available on the possible role of VEGF in acute asthma. We evaluated whether VEGF participates in airway inflammation during acute asthma.

Ten asymptomatic patients with stable asthma, 10 acute asthmatic patients and ten normal healthy subjects were selected for this study. Sputum samples in patients with acute asthma were collected before (day 1) and after (days 7 and 28) treatment. We checked levels of VEGF by means of enzyme immunoassay in the sputum of patients with stable asthma or with acute asthma. VEGF levels were also evaluated during a spontaneous attack of asthma.

The levels of VEGF were significantly increased in stable asthmatic patients and were even higher in acute asthmatic patients in comparison with controls ($P < .05$ and $P < .01$, respectively). Levels of VEGF were significantly higher ($P < .01$) in acute asthmatic patients than in stable asthmatic patients. The elevated VEGF levels on day 1 had significantly decreased by day 7 and had decreased even further by day 28 ($P < .01$ and $P < .01$, respectively; Fig 1).

Fig. 1. Concentrations of VEGF in sputum obtained from control, stable asthmatic, and acute asthmatic subjects and time course of VEGF levels in sputum obtained from acute asthmatic subjects. Samples from acute asthmatic subjects were obtained at day 1 of exacerbation and at days 7 and 28. Data are reported as means $\pm$ SD.



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In acute asthmatic patients, the levels of VEGF in sputum were significantly correlated with the number of neutrophils and eosinophils ($r = 0.797$, $P < .01$ and $r = 0.817$, $P < .01$, respectively).

In conclusion, our data suggest that the overproduction of VEGF is associated with airway inflammation during acute asthma.

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