Phentolamine for Neurogenic Pulmonary Edema

Bench to Bedside Progress

To the Editor:

We read with interest the article by Davison et al in CHEST (March 2012). The authors reported rapid resolution of refractory ARDS/neurogenic pulmonary edema after IV phentolamine was administered as a last resort. We believe that, although merely a singlet, when seen within the larger context of the literature on the pulmonary effects of catechol excess, the authors’ description points to a novel treatment modality for this poorly understood disease.

The association of pulmonary edema with catecholamine excess was recognized more than a century ago by investigators who found that high-dose epinephrine reliably induced cardiopulmonary failure in various animal models of shock. Although Berk et al showed detrimental effects on pulmonary gas exchange after infusion of epinephrine in anesthetized dogs. We reported the acute deterioration of arterial oxygen tension and pulmonary gas exchange in rats within a minute of bolus epinephrine administration. Interestingly, this effect was attenuated or completely prevented by pretreatment with phentolamine. Although the mechanism continues to remain speculative, preliminary studies in our isolated murine lung model suggest that catecholamine-induced pulmonary edema and secondary hypoxemia result from the combination of increases in pulmonary capillary pressure and shear injury secondary to elevated cardiac output. The use of α blockade may help correct one factor of this pathophysiologic equation and, thereby, improve gas exchange. This putative explanation of our experimental observations should also apply to the clinical success of phentolamine in repairing severe neurogenic pulmonary edema.

The authors are to be commended for their excellent clinical care and particularly for their “thinking outside the box.” We add this case to the growing body of data confirming a potent, pathologic effect of catecholamine excess on pulmonary circulation. Both laboratory and clinical experience now suggest a potential clinical benefit to giving α-blockers in such situations. Clinical trials are the next step.

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REFERENCES


Response

To the Editor:

We thank Drs Krishnamoorthy and Weinberg for their comments on our article in CHEST. We agree that clinical trials are the next step. Our group has proposed a pragmatic clinical definition of neurogenic pulmonary edema (NPE) that may assist investigators in developing inclusion/exclusion for such a clinical trial: (1) bilateral pulmonary infiltrates, (2) PaO2/FIO2 ratio<200, (3) no evidence of left atrial hypertension, (4) presence of CNS injury (severe enough to have significantly increased intracranial pressure), and (5) absence of other common causes of ARDS (eg, aspiration, massive blood transfusion, sepsis). Given the rare nature of NPE, we also suggest that an online registry using this definition would be worthwhile to assess the incidence, morbidity, and mortality of NPE.

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