We also appreciate the comment that the inflammatory status in COPD might be of multifactorial origin. It is our study that demonstrates the independent association of the metabolic syndrome, physical inactivity, and COPD with systemic inflammation. Therefore, we identified at least three conditions that might contribute to systemic inflammation in a population that was primarily classified according to severity of COPD.

Henrik Watz, MD
Benjamin Waschkì, MD
Anne Kirsten, MD
Kai-Christian Müller, PhD
Gunther Kretschmar, MD
Grosshansdorf, Germany
Thorsten Meyer, PhD
Lübeck, Germany
Olaf Holz, PhD
Helgo Magnusen, MD
Grosshansdorf, Germany

Affiliations: From the Pulmonary Research Institute (Drs Watz, Waschkì, Kirsten, Kretschmar, and Magnusen); Hospital Grosshansdorf (Drs Müller, Holz, and Magnusen); and University Lübeck (Dr Meyer).

Financial/nonfinancial disclosures: The authors have reported to CHEST that no potential conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

Correspondence to: Henrik Watz, MD, Pulmonary Research Institute, Center for Pneumology and Thoracic Surgery, Hospital Grosshansdorf, Wochrendamin 80, D-22927 Grosshansdorf, Germany; e-mail: h.watz@pulmoresearch.de

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DOI: 10.1378/chest.09-2684

REFERENCES


Postobstructive Pulmonary Edema

A Positive End-Expiratory Pressure Effect

To the Editor:

I refer to the article published in CHEST (June 2007) by Fremont et al1 describing the hydrostatic mechanism for postobstructive pulmonary edema. This theory only explains the situation in which acute obstruction occurs after extubation (eg, acute laryngospasm), but does not explain conditions with chronic upper airway obstruction. We came across a case of postextubation pulmonary edema that could not be explained solely by hydrostatic mechanism. We report on an elderly man with carcinoma of the larynx, with tumor growth over the vocal cords for 2 years. In preoperative evaluation his cardiovascular system was unremarkable with normal ejection fraction and no diastolic dysfunction. He underwent total laryngectomy with permanent tracheostomy in situ; the surgery was uneventful. Postoperatively he remained hemodynamically stable and was ventilated with positive pressure ventilation in the recovery room. Once he was conscious and obeying instructions, he was shifted to a T-piece as a weaning protocol. On the T-piece, he developed respiratory distress and went into pulmonary edema, requiring furosemide, nitroglycerin drip, and positive pressure ventilation with a positive end-expiratory pressure (PEEP) of 10. He was symptomatically relieved within a few hours. Repeat echocardiogram did not reveal any cardiac dysfunction, and ECG was also normal. He was gradually tapered off PEEP for the next 24 to 48 h; his intake and output were balanced to the negative side and weaned off by the end of 48 h.

If we consider the hydrostatic mechanism described by Fremont et al1 as the only cause, then the edema should occur when there was an obstruction, but here edema occurred after the relief of obstruction. We hypothesize the following mechanism, in addition to the hydrostatic mechanism. In chronic obstruction of the upper airway, expiration occurs against a resistance, causing a PEEP effect in the bronchial tree. This PEEP is transmitted to alveoli, opposing the perialveolar hydrostatic pressure of heart physiology.2 While the obstruction is there, there is no influx of fluid into the alveoli and fluid remains in the interstitium and drains into the peribronchial lymphatics.3 Surgical relief of this obstruction causes a sudden release of the PEEP effect and as the hydrostatic forces do not come back to normal immediately,4 it causes alveolar flooding and pulmonary edema. The usual measures of decreasing preload and using positive pressure ventilation help in rapid recovery. This hypothesis may help the anaesthetist to enable a slow and gradual weaning of patients with chronic upper airway obstruction postoperatively, with a gradual decreasing of the PEEP in the recovery room.

Anand Joshi, Diplomate in NB
Mahim, Mumbai, India

Affiliations: From the Department of Critical Care Medicine, PD Hinduja National Hospital and Medical Research Center.

Correspondence to: Anand Joshi, Department of Critical Care Medicine (ICU), PD Hinduja National Hospital and MRC, Veer Savarkar Marg, Mahim, Mumbai 400016 India; e-mail: drjoshianand@gmail.com

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DOI: 10.1378/chest.09-1596

REFERENCES


Vitamin D and Mortality From Pulmonary Fibrosis

To the Editor:

In a recent article in CHEST (July 2009) Olson and colleagues1 found that mortality rates from pulmonary fibrosis exhibited significant seasonal variation with the highest rates occurring in the winter; seasonal variation in pulmonary fibrosis mirrored COPD exacerbation and mortality. We suggest that some of the mechanisms involved in this detrimental association might also be due to seasonal variations in blood vitamin D levels.

Anand Joshi, Diplomate in NB
Mahim, Mumbai, India

Affiliations: From the Department of Critical Care Medicine, PD Hinduja National Hospital and Medical Research Center.

Correspondence to: Anand Joshi, Department of Critical Care Medicine (ICU), PD Hinduja National Hospital and MRC, Veer Savarkar Marg, Mahim, Mumbai 400016 India; e-mail: drjoshianand@gmail.com

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DOI: 10.1378/chest.09-1596

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