We provided care for a 37-year-old African-American man who had experienced recent infection with hepatitis B. He presented with mononeuritis multiplex, significant weight loss, and fever. A sural nerve biopsy showed necrotizing vasculitis involving medium-sized arteries. He was diagnosed with PAN and was treated with high-dose corticosteroids, laninudine and plasmapheresis. In the course of treatment, the patient developed respiratory distress requiring mechanical ventilation. Chest radiographs showed bilateral patchy alveolar infiltrates, and his hemoglobin and hematocrit levels had dropped from 13.9 g/dL and 43% to 11 g/dL and 32%, respectively. There was no evidence of hemolysis or GI bleeding. Over the next few days, there was progression of alveolar infiltrates associated with a further drop in hemoglobin and hematocrit levels to 6.5 g/dL and 19%, respectively, which is suggestive of alveolar hemorrhage. After about 3 weeks of continued treatment with corticosteroids and plasmapheresis, the patient’s condition clinically improved, with clearing of the infiltrates on the chest radiographs.

Pulmonary involvement, although rare in cases of PAN, can occur in the form of alveolar hemorrhage or diffuse alveolar damage and can often be fatal.2,3 Hepatitis B surface antigen, the trigger for the immune complex disease, results in the activation of the complement cascade and, in turn, neutrophil activation, is thought to play an important pathogenic role. Anti-endothelial cell antibodies also have been shown to play a role in endothelial damage and can often be fatal.2,3 Immunohistochemical studies in PAN patients have shown inflammatory infiltrates with macrophages and T lymphocytes.4 Some patients respond to aggressive therapy with corticosteroids and plasmapheresis, supporting the notion that immune complex-induced endothelial cell damage may indeed play a role in its pathogenesis.

Yamini Menon, MD
Ranju Singh, MD
Raquel Cuchacovich, MD
Louis R. Espinoza, MD
Louisiana State University Health Sciences Center
New Orleans, LA

Correspondence to: Luis R. Espinoza, MD, Professor and Chief, Section of Rheumatology, LSU Health Sciences Center, 1542 Tulane Ave, New Orleans, LA 70112-2822.

REFERENCES

Obesity Hypoventilation Syndrome
What’s in a Name?
To the Editor:

It is with great interest that I read the article by Kessler et al in CHEST (August 2001) regarding obesity hypoventilation syndrome (OHS). I think that there are yet important aspects to clarify in regard to the definition of this entity, because there is some confusion in the literature about this subject.

The development of hypoventilation in obese patients is composed of multiple and complex mechanisms that comprise at least the following three entities: obstructive sleep apnea (OSA), COPD, and OHS. Kessler et al assumed that obese patients with hypercapnia may be understood to have OHS without regard for whether they had experienced sleep apnea. In fact, in the article, the authors include obese hypercapnic OSA patients as “OHS patients.”

Nevertheless, in this regard, the author’s experience (which has been confirmed by others2,3) shows that some nonobese OSA patients may develop hypercapnia. Then it seems that hypercapnia in patients with sleep apnea may occur independently from obesity, denoting a complex autonomous mechanism.

For this reason, it seems more logical not to include sleep apnea patients into the entity OHS and to restrict the term OHS to patients in whom hypercapnia persists after eliminating sleep apnea as the mechanism of hypercapnia (ie, after a trial of continuous positive airway pressure [CPAP] at an effective pressure).4

For this reason, I propose to restrict the term OHS to patients in whom the only mechanism responsible for alveolar hypoventilation is obesity itself (independent of apneas). In this field, it is preferable to call this condition obesity-linked hypoventilation (OLH).3 This entity could be defined in the following two situations: (1) hypercapnia in obese patients without OSA or COPD (ie, pure OLH); and (2) persistence of hypercapnia in OSA patients who are receiving CPAP (ie, OLH plus OSA).

This approach also has therapeutic implications. We will restrict long-term therapy with bilevel pressure ventilation (or any kind of positive-pressure ventilation) only to patients who experience persistent hypercapnia while receiving CPAP.

Consequently, this new approach can enlarge and clarify the field of respiratory syndromes in obese patients and the approaches to therapy.

Claudio A. Rabec, MD, FCCP
Torner Hospital
Buenos Aires, Argentina

Correspondence to: Claudio A Rabec, MD, FCCP, Alemania 2739, C1419FJA Buenos Aires, Argentina; e-mail: cirhtaruc@yahoo.com.ar

REFERENCES

Efficacy of Microdrainage in Severe Subcutaneous Emphysema
To the Editor:

Severe subcutaneous emphysema may complicate the postoperative period of patients who undergo thoracic surgery. Although the condition rarely is life-threatening, discomfort from cutaneous tension and palpebral occlusion, as well as an increase