continued to deteriorate and was eventually intubated. She required a prolonged course of high-dose IV corticosteroids and inhaled bronchodilators before she was extubated. Toxicology workup was positive for cocaine. When confronted with this data, the patient admitted to using large quantities of free-base cocaine for many months. After extubation, she was transferred to a medical bed where she once again used free-base cocaine. This resulted in severe stridor and necessitated reintubation and eventual tracheostomy.

We believe that upper airway edema can develop after direct airway irritation from the drug itself or from a variety of chemicals used in their preparation. The edema may be aggravated by the deep inspiratory maneuvers, which free-base cocaine users commonly perform to prolong the effects of the drug.

In summary, we believe this case report is the first to describe recurrent upper airway edema from the repeated use of inhaled free-base cocaine. Since the popularity and availability of this drug is rising, it is likely that similar cases will be increasingly encountered.1

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REFERENCE


Other Local Effects of Nasal-Continuous Positive Airway Pressure

To the Editor:

We have read with interest the recent article by Pépin and colleagues, (CHEST 1995; 107:375-81) in which side effects of nasal-continuous positive airway pressure (N-CPAP) are described. Between the side effects commented, pain or abrasion on the bridge of the nose, dryness of the mouth or nose, sneezing and nasal drip, nasal congestion, sinusitis, nosebleeding, and swallowing were noted. They concluded that the side effects of N-CPAP are mainly local to the nose.

In this sense, although we think that this article is very interesting, we would like to emphasize the ocular side effects of N-CPAP. Several years ago, we reported1 in CHEST about a patient with glaucoma and obstructive sleep apnea syndrome in whom the intraocular pressure increased during N-CPAP treatment. Recently, we evaluated 18 patients previously diagnosed as having glaucoma and 22 normal subjects. N-CPAP was used during the wakefulness at 12 cm H2O for 15 min. The results showed that N-CPAP significantly increases intraocular pressure in patients with glaucoma (20.3±6.3 vs 22.3±5.7 mm Hg, p<0.01); whereas no significant difference in normal subjects (15.0±2.2 vs 15.0±2.7 mm Hg) was found.2 Although this intraocular pressure increase did not produce any ocular discomfort in our patients, we believe that ocular side effects of N-CPAP might be always considered. For this reason, we think that the intraocular pressure measurement should be performed in those patients who will use N-CPAP for a long time.

Another beneficial effect of N-CPAP may be the efficient remedy as the only treatment of patients suffering from obstructive sleep apnea syndrome and gastroesophageal reflux.3

We would like to enlarge the exposition of the local effects of N-CPAP described by Pépin et al. Even though the nasal effects of N-CPAP are the most important, other local manifestations, like ocular and esophageal, should be considered.

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REFERENCES


Differential Diagnosis of Postpartum Pulmonary Edema

To the Editor:

I have read with great interest in the December 1994 issue in the section Clinical Problems in Cardiopulmonary Disease the report on the problem of pulmonary edema developing postpartum as reported by Morton E. Tavel (CHEST 1994; 106:1883-84). The differential diagnostic considerations list strikingly avoided the mention of pulmonary edema associated with tocolytic therapy. In this clinical situation terbutaline is the most common drug involved. I have recently cared for a patient who was beset with this complication during pregnancy in 1992 and again in 1993. This diagnostic possibility becomes obvious with good historic data. In 42% of patients reported in the literature, the symptoms have occurred after the tocolytic therapy has been discontinued.1 Symptoms of dyspnea and chest pain are nonspecific. It occurs in women who have received sympathomimetic agents to arrest uterine contractions occurring during premature labor. The incidence is higher in women in twin gestations as seen also in postpartum cardiomyopathy. This generally occurs within 12 h of delivery and the chest x-ray usually shows bilateral pulmonary infiltrates with a normal cardiac size. The major criteria for diagnosis of pulmonary edema associated with tocolytic therapy are (1) recent (less than 24 h) or current use of sympathomimetic drugs; (2) dyspnea occurring before delivery or less than 12 h postpartum in women who deliver despite tocolytic use; (3) a chest roentgenogram showing unilateral or bilateral alveolar infiltrates; (4) evidence of hemodilution (decreased hematocrit or hypokalemia); and (5) rapid clinical response (less than 24 h) to treatment with diuretics and oxygen. Generally, the prognosis is good with a mortality rate of less than 3%. Patients respond dramatically to discontinuation of tocolytic therapy, the use of diuretics, and oxygen. The underlying mechanism appears not to be increased permeability or drug toxicity but one related to an increase in hydrostatic pressure as seen in high altitude pulmonary edema. Also mentioned is β-agonist receptor down regulation and desensitization thus mimicking the β-adrenergic receptor alterations seen in idiopathic dilated cardiomyopathy. This is an important entity to consider in postpartum pulmonary edema.

Communications to the Editor