Pulmonary hypertension and acute pulmonary edema in a 23-year-old male with a history of an upper respiratory tract infection

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ÖZET

Yirmiüç yaşında bir erkekte üst solunum yolu infeksiyonu sonrası gelişen pulmoner hipertansiyon ve akut akciğer ödemi


SUMMARY

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The pathophysiology of upper-airway obstruction (UAO) is complex. Possible causes of UAO that may lead to acute respiratory failure, are as follows: infections like acute epiglottitis and croup, obstructing tumors in the base of the tongue, larynx or hypopharynx, aspirated food or liquid contents, obesity and anatomical variations. Management changes according to the pathogenesis of the disorder. In patients with severe carbon dioxide retention or apnea, emergency endotracheal intubation must be carried out. Hereby, we describe a 23-year-old patient with susceptible upper-airway anatomy and UAO occurred following an upper respiratory infection and complicated with pulmonary hypertension and pulmonary edema. Our patient seems to be one of the complicated UAO cases, with an unusual but critical clinical presentation, evaluated in a wide spectrum and nicely returned to life. Key Words: Upper-airway obstruction, pulmonary edema, pulmonary hypertension.

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The pathophysiology of upper-airway obstruction (UAO) is complex and still under active investigation. Possible causes of UAO that may lead to acute respiratory failure, are as follows: infections like acute epiglottitis and croup, obstructing tumors in the base of the tongue, larynx or hypopharynx, aspirated food or liquid contents, obesity and anatomical variations. Management changes according to the pathogenesis of the disorder. In patients with severe carbon dioxide retention or apnea, emergency endotracheal intubation must be carried out (1).

With sleep onset, the wakefulness stimulus is lost and the negative pressure reflex is diminished. Thus, within a short time after sleep onset, upper airway muscle activity generally decreases and upper-airway resistance increases, even in normal subjects. With sustained sleep, the activity of the genioglossus may return to waking levels, but the activity of the palate muscles often remains below that during wakefulness. Thus, susceptible upper-airway anatomy and a loss of upper-airway muscle activity are the key elements UAO (2).

The major site of upper-airway narrowing in normal subjects appears to be in the retropalatal area (3). Especially in predisposed, anatomically variant individuals, sleep disorders should gain importance when complicated with other circumstances.

Hereby, we describe a patient with susceptible upper-airway anatomy and UAO occurred following an upper respiratory infection and complicated with pulmonary hypertension and pulmonary edema.

**CASE REPORT**

A 23-year-old male presented with slowly progressive dyspnea following an upper respiratory tract infection, cyanosis, and swelling of face and legs for last two months, on December 2002. His abdominal ultrasonographic examination, on November 2002, has revealed dilatation of inferior vena cava and hepatic veins with right pleural effusion and invasive hemodynamic study has shown pulmonary arterial pressure (PAP) measured as 65 mmHg. His clinical course quickly worsened within hours and he was entubated and his follow-up was performed in intensive care unit.

His past medical history was perfect except his mental retardation. Physical examination revealed bilateral decreased breath sounds at both bases, fine crackles up to both apices on auscultation, S3 gallop rhythm, hepatomegaly, pretibial edema and jugular venous distension. His blood gas measurements necessitating intubation revealed the following: pH: 7.14, PaCO2: 158.1 mmHg, PaO2: 7.4 mmHg, and oxygen saturation: 2.9%. Complete blood count, blood chemistry and urin analysis were normal. The chest radiography revealed changes that were consistent with cardiomegaly with enlarged pulmonary conus, bilateral pleural effusion and pulmonary edema (Figure 1). Electrocardiographic examination showed incomplete right bundle branch block. Congestive heart failure was diagnosed and diuretics with ACE inhibitor therapy was begun. Clinical response were quiet perfect and he was extubated on the second day; and his high resolution computed tomography (CT) of thorax on day four was compatible with cardiogenic pulmonary edema. On echocardiography, systolic PAP was 85 mmHg.

Nocturnal desaturations and apnea following sleep were observed during follow-up and bile-
vel positive airway pressure (BiPAP) during sleep time was commenced. His blood gas measurements on the seventh day of BiPAP usage revealed the following: pH: 7.39, PaCO2: 51.1 mmHg, PaO2: 78 mmHg, and oxygen saturation: 92%. Clinical picture was under control; but aetiological investigations for rapidly progressive pulmonary hypertension and sleep disorder were performed.

Cranial MRI and neck CT were normal. No neurological abnormality causing sleep disorder was found. Psychometric evaluation was performed and high cortical functional evaluation report informed that his IQ was 35-40. Ear-nose-throat examination revealed narrowed left nasal cavity with septum deviation. Distance between nasopharynx and soft palate was narrow and occluding during snoring. Epiglottis was omega shaped and collapsing with deep inspiration. Surgical indication for septum deviation and volume reduction of soft palate was present; but his mental retardation was a contraindication.

Polisomnographic study showed nocturnal desaturation (mean saturation: 70%), five obstructive and one central apnea and 19 hypopnea. Mean apnea duration was 53 seconds (84 sec, max). Apnea-hypopnea index was 3.6 and BiPAP was suggested during sleep.

On January 2003, his systolic PAP was 40 mmHg with minimally enlarged right heart. He was discharged to be on follow-up. Echocardiography on March 2003 was a victory: Normal cardiac dimensions with systolic PAP measured as 20 mmHg. He is still well with normal blood gas analysis and normal radiology (Figure 2).

**DISCUSSION**

The patency of the upper-airway depends on extraluminal tissue/gravitation factors, anatomical factors, the amount of negative intraluminal pressure, as well as amount of upper-airway muscle activity (4).

In polysomnographic study, mixed apneas and hypopneas were determined in our patient. Mixed apneas are those in which the initial portion of the apnea is central and remaining portion is obstructive. The postapnea ventilatory period may have consequences if the patient returns to sleep quickly. If the PaCO₂ falls below a point called the “apneic threshold”, then respiratory drive ceases, resulting in central apnea. This is thought to be the origin of the central portion of mixed apnea. Periods of reduced tidal volume or airflow during hypopneas may also be associated with desaturations or sleep disturbance (2).

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Although those could be due to a fall in central drive or partial airway obstruction, a separation is not always possible. Stabilization of the upper airway by nasal continuous positive airway pressure (CPAP) or BiPAP usually abolishes both components of mixed apnea. BiPAP quickly improved the clinical picture and stabilized the laboratory parameters in our young patient.

Like in patients with bony abnormality, obstructive sleep apnea, long soft palate, a large posteriorly placed tongue, increased fat deposition, or tissue edema; a possible obstruction to airflow occurred in our patient during sleep at one or several locations in upper-airway. To our suggestion, his story began with an upper airway infection with tissue edema; a possible obstruction to airflow occurred in our patient during sleep at one or several locations in upper-airway. To our suggestion, his story began with an upper airway infection with tissue edema. In addition, he had an anatomical variation with a narrow distance between nasopharynx and soft palate occluding during snoring and an omega-shaped epiglottis collapsing with deep inspiration. So, he had had an increased supraglottic resistance and, unlike normal subjects, required a positive intraluminal pressure to prevent airway closure during sleep.
His mental retardation which was a contraindication for septum deviation and soft palate volume reduction surgery necessitated BiPAP administration.

Pulmonary arterial blood pressure rises during apneas and then falls when the oxygen saturation returns to normal after apnea. The main cause is hypoxic vasoconstriction. In addition, an increase in systemic blood pressure is also associated with each obstructive apnea/hypopnea (2). Congestive heart failure and increased PAP was present in our case, which was thought to occur due to his sleep disorder caused by his anatomical variation.

One of the probable explanations of our patient’s clinical picture was pulmonary edema which may complicate UAO in both children and adults. The mechanism for the formation of pulmonary edema was elucidated in analysis of three cases, showing a hydrostatic mechanism for edema fluid formation (5). In another study, the cause was attributed to pulmonary and hemodynamic changes resulting from high negative intrathoracic pressures during obstructed respiration (6). The incidence and the reasons of pulmonary edema development in only certain patients are unclear (5,6).

In conclusion, our patient seems to be one of the complicated upper-airway obstruction cases, with an unusual but critical clinical presentation, evaluated in a wide spectrum and nicely returned to life.

REFERENCES