Case report

A 17-year-old, white male with gynecomastia underwent bilateral subcutaneous mastectomy combined with a liposuction of the thoracic wall. A complete medical history and physical examination was done prior to surgery and did not reveal any stigmata of cardiac or pulmonary disease.

Surgery was performed under general endotracheal anesthesia and the patient was extubated in the operating room without complications.

On arrival in the recovery room, the patient remained at normal oxygen saturation levels while receiving 2L of oxygen. Removal of the oxygen mask, some minutes after extubation, induced respiratory distress with vigorous inspiratory efforts. The patient didn’t develop a petechial rash nor did he suffer from an altered mental status.

Imaging findings

A portable chest X-ray was taken. It revealed bilateral hazy lung opacities, more pronounced at the right side. Heart size was within normal limits and pleural effusions were absent.

The differential diagnosis of (asymmetric) pulmonary edema or fat embolism was suggested (Fig. 1).

Subsequently, a CT scan of the chest was performed to exclude pulmonary thromboembolism, pneumothorax and aspiration pneumonia.

The scan showed extensive areas of patchy ground-glass opacities in a lobular distribution and areas of crazy paving in the right lung and in the left upper lobe (Fig. 2A,B), findings compatible with the clinical diagnosis of pulmonary edema. No signs of pulmonary thromboembolism or pneumothorax were detected.

He received 10 mg of Furosemide intravenously and the O2 administration was sustained.

A new chest X-ray (Fig. 3) 2 hours later showed partial resolution of the lung opacities which almost disappeared on the last chest X-ray 13 hours later.

He was kept in observation during 24 hours and was discharged on the next day.

The diagnosis of postextubation pulmonary edema due to laryngospasm was made, based on the clinical and radiological signs and their evolution in time.

Discussion

Pulmonary edema in the postoperative setting can have a number of causes including cardiogenic and renal failure as well as lung injury (incl. aspiration, embolism, upper airway obstruction).

Postextubation pulmonary edema is recognized by development of hypoxia shortly (1-90 min) after a laryngospasm (1). It can be induced by repeated upper airway irritation from an endotracheal tube or by a difficult intubation, from the accumulation of blood or secretions into the posterior pharynx during surgery, from vomiting, from hyperactive laryngeal reaction related to chronic smoking or occult asthma, and from esophageal reflux (2, 3).

The incidence of postoperative laryngospasm ranges from 0.05-1% in patients undergoing general anesthesia (2). These patients typically had uncomplicated anesthetic and operative courses (4). There is a number of risk factors reported in literature including obesity, young male adults, upper aerodigestive tract surgery, smoking, asthma, sleep apnea or esophageal reflux (2, 4-7).

The pathogenesis remains unclear but is probably related to the negative intrathoracic pressure, due to inspiration against a narrow/closed upper airway (8). Transient injury to

Key-word: Lung, fluid.
the pulmonary endothelium may also play a role in the pathogenesis but is still considered controversial (9). Both mechanisms establish an augmented transcapillary fluid efflux, which results in interstitial and alveolar edema.

The X-ray findings are those seen in non-cardiogenic pulmonary edema: perihilar alveolar opacities most often symmetric and bilateral (bat-wing appearance), sometimes unilateral with a wide vascular pedicle. Pleural effusions are rare and the heart size is normal.

CT findings consist of thickening of the septae, peribronchial cuffing and patchy ground-glass opacities around the hilum in the right lung and in the left upper lobe.

The differential diagnosis with fat embolism, based on radiographic images alone, may be difficult and therefore the diagnosis is predominantly made on clinical basis (slow onset of dyspnea, petechial rash and an altered mental status).

A CT-scan can sometimes help in the differential diagnosis when fat containing embolic lesions are present in the pulmonary arteries. When the emboli are smaller, CT findings are more subtle and show ground glass or sometimes nodular opacities in a subpleural and centrilobular distribution, with more pronounced impairment of the peripheral lung regions compared to pulmonary edema (11).

Treatment is focused on the resolution of the upper airway obstruction (O2, bronchodilatator, reintubation) and administration of diuretics (4).

References