Negative pressure pulmonary edema in the coronary care unit

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A 63-year-old woman with no known cardiac history presented with pulmonary edema accompanied by electrocardiographic evidence of ischemia. Echocardiography demonstrated normal cardiac dimensions, normal wall motion and mild diastolic dysfunction. Despite repeat attempts at extubation following aggressive diuresis, the patient required ongoing ventilatory support. Although cardiac catheterization revealed normal coronary arteries, computed tomography revealed a 4 cm × 9 cm multinodular goiter extending into the mediastinum and compressing the trachea. A diagnosis of negative pressure pulmonary edema should be considered in the differential diagnosis of any patient presenting with acute heart failure.

Key Words: *Computed tomography; ECG; Echocardiography; Heart failure*

Negative pressure pulmonary edema (NPPE) is a rare but life-threatening complication of upper airway obstruction. Although the clinical presentations of NPPE and cardiogenic pulmonary edema are similar, management of these clinical entities differ significantly. We describe the case of a 63-year-old woman with an initial presentation of pulmonary edema, accompanied by electrocardiographic evidence of ischemia, who was subsequently diagnosed with NPPE secondary to a large, multinodular goiter. In addition, the pathophysiology, diagnosis and management of NPPE are reviewed.

CASE PRESENTATION

A 63-year-old woman with no known cardiac history presented with a sudden onset of dyspnea requiring intubation and ventilatory support out of hospital. She denied preceding symptoms of chest discomfort, palpitations, syncope or infection.

The patient was afebrile and normotensive, with a sinus tachycardia of 140 beats/min. The cardiorespiratory examination was remarkable for an elevated jugular venous pressure at the angle of the jaw, a left-sided third heart sound and bibasilar crackles in both lung fields.

The complete blood cell count, electrolytes and cardiac biomarkers were within normal limits. An initial electrocardiogram revealed ST depression in leads V2 to V6, suggestive of anterolateral ischemia (Figure 1). A chest radiograph demonstrated pulmonary vascular congestion consistent with a diagnosis of pulmonary edema (Figure 2). An echocardiographic examination revealed normal cardiac dimensions, normal wall motion and mild diastolic dysfunction with an early diastolic to late diastolic transmitral ratio of less than 1, prolonged deceleration time (250 ms) and a reduced early diastolic annular velocity of the lateral mitral valve annulus.

Un œdème pulmonaire par pression négative à l'unité de soins coronariens

Une femme de 63 ans ayant des antécédents cardiaques connus a consulté en raison d'un œdème pulmonaire accompagné d'observations électrocardiographiques d'ischémie. L'échocardiographie a démontré des dimensions cardiaques normales, un mouvement normal de la paroi et un léger dysfonctionnement diastolique. Malgré des tentatives répétées d'extubation après une diurèse énergique, le patient a eu besoin d'un soutien ventilatoire continu. Le cathétérisme cardiaque a révélé des artères coronaires normales, mais la tomographie a mis au jour un goitre multinodulaire de 4 cm x 9 cm se prolongeant dans le médiastin et comprimant la trachée. Il faut envisager un diagnostic d'œdème pulmonaire par pression négative dans le diagnostic différentiel d'un patient consultant à cause d'une insuffisance cardiaque aiguë.

Figure 1) A baseline electrocardiogram demonstrating ST-T wave changes in the anterolateral leads, suggestive of ischemia

Following aggressive diuresis and requiring minimal ventilatory support, the patient was extubated one day following her initial presentation. Within 1 h of extubation, the patient developed recurrent pulmonary edema clinically and radiographically, requiring mechanical ventilation. Cardiac catheterization was performed and revealed normal coronary arteries. On day 4 of the coronary care unit admission, the patient failed a repeat attempt at extubation.

A computed tomographic scan revealed a 4 cm × 9 cm multinodular goiter extending into the mediastinum and suspected associated tracheal stenosis (Figure 3). With a diagnosis of NPPE secondary to an enlarged goiter, the patient underwent surgical intervention for a thyroidectomy. Intraoperative bronchoscopy revealed tracheal stenosis of greater than 50%. The postoperative course was uncomplicated, and three months following discharge, the patient returned to her previous activity level without subjective dyspnea.

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Figure 2) A chest x-ray demonstrating bilateral pulmonary vascular congestion

DISCUSSION

NPPE is a rare diagnostic entity (1). The clinical suspicion should be considered in any patient presenting with respiratory distress associated with tracheal stridor, hypoxemia, hypercapnea and with radiographic evidence of pulmonary edema. Although the patient's age, symptoms, electrocardiogram and chest x-ray were most suggestive of cardiogenic pulmonary edema, NPPE should be considered as the differential diagnosis.

The pathophysiology of NPPE is incompletely understood and is likely multifactorial. Initially, upper airway obstruction leads to the generation of markedly negative intrathoracic pressures. In the current case, thyroid enlargement resulted in tracheal narrowing, with incomplete airway obstruction. Intrathoracic pressures, which vary during normal inspiration between -2 cm and -12 cm H_2O , can fall to as low as -50 cm H_2O with airway obstruction (1,2). It is believed that the excess pressure generated in response to obstruction produces mechanical stress and direct capillary injury, increasing permeability and leakage into the interstitium (3). In addition, Starling forces balance the movement of fluid across the pulmonary capillary bed, with excess fluid draining through the lymphatic system at a maximum estimated rate of 20 mL/h (4). With falling intrapleural pressures and impaired pulmonary capillary integrity, movement of fluid into the interstitium is favoured, leading to the clinical setting of pulmonary edema.

The onset of symptomatic NPPE following critical upper airway obstruction ranges from 3 min to 150 min (5). Typically, NPPE is self-limited, with clinical and radiographic improvement within 12 h to 48 h. Delayed recognition and treatment is associated with longer recovery times (5). Once identified, the initial treatment of NPPE is the relief of the airway obstruction, usually with placement of an airway catheter via intubation or surgical tracheostomy. In reviews of both children and adults diagnosed with NPPE, 83% required intubation, while tracheostomy was required in 20% of cases (elective or emergent) (4). Adequate oxygenation can be achieved with the supplemental use of oxygen and positive end expiratory pressure. Invasive hemodynamic monitoring, as well as drug therapy, such as diuretics, are unnecessary if the diagnosis of NPPE is clear. However, as illustrated in the present case, it is frequently not. The first step toward the

Figure 3) A A three-dimensional, volume-rendered image of the lungs and airway reconstructed from the chest computed tomography scan dataset demonstrates the endotracheal tube (small arrows) and its balloon (large arrow). Note that although the trachea around the tube is well seen below the balloon (arrowhead), it is not visible above the balloon. B A transverse computed tomographic image through this level shows the endotracheal tube (arrow) surrounded by a grossly enlarged thyroid (arrowheads). The thyroid has compressed the trachea against the endotracheal tube and no air is seen around it

appropriate diagnosis of NPPE, therefore, is its consideration by the clinician in the differential diagnosis.

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