Clinical management of the neonatal pneumomediastinum

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Abstract. Pneumomediastinum (PM) occurs in approximately 0.1% of newborns but its incidence is underestimated because it is often asymptomatic. PM generally has a benign course. Our knowledge of PM is insufficient, and its management is mainly based on the best practice and experience of each hospital rather than on evidence-based data. (www.actabiomedica.it)

Key words: mediastinal emphysema, infant newborn, disease management

Introduction

Pneumomediastinum (PM), also known as mediastinal emphysema, is a condition in which air is present in the mediastinum. PM was first described by Laennec in 1819 as a consequence of traumatic injury (1). In the 1939 Hamman described the first case of spontaneous PM hence the name "Hamman sign" characterized by a typical crunching, rasping sound, synchronous with the heartbeat.

Mediastinal anatomy

The mediastinum has been defined as the mass of tissues and organs separating the two pleural sacs, located between the sternum in front and the vertebral column behind and extending from the thoracic inlet above to the diaphragm below. In the Zylak method, the mediastinum is divided into three longitudinal compartments extending uninterruptedly from the level of the thoracic inlet to the level of the diaphragm. The anterior mediastinal compartment (prevascular space), the middle mediastinal compartment (vascular space) and the posterior mediastinal compartment (postvascular space) (2).

Neonatal pneumomediastinum

Neonatal PM is a condition resulting from a mediastinal air leak and occurs in about 0.1-0,2% of newborns (3). The pathophysiology of PM has been studied in experimental models and seems to be due to alveolar breakage caused by the difference in pressure between alveoli and surrounding tissues. The consequent air leakage into interstitial tissues diffuses throughout the peribronchial and perivascular tissues, reaching the mediastinum (4, 5).

The most common reported causes of PM in the neonates are exposure to positive pressure ventilation, meconium aspiration syndrome (MAS), pneumonia or other infectious diseases (3, 6). However, PM may also be idiopathic and occur without any underlying risk factors (3).

Clinical diagnosis

In the neonate the clinical presentation of PM is extremely insidious. In many patients PM could be completely asymptomatic, in someother cases, is associated with symptoms of respiratory distress (i.e.: grunting, tachypnea, nasal flaring, need of oxygen sup-

plementation). The clinical diagnosis is often based on a cardioauscultatory exam, characterized by paraphonic tones, rarely in the newborn is possible to recognize the Hamman sign. This explains why the incidence of PM is often underestimated. The experience of the neonatologist can significantly affect the recognition of PM in asymptomatic patients.

Instrumental diagnosis

PM is diagnosed by frontal or lateral chest x-ray demonstrating its typical features, such as the continuous diaphragm sign (interposition of air between the pericardium and the diaphragm, which becomes visible in the central mediastinal part), linear bands of mediastinal air paralleling the left side of the heart, and the spinnaker-sail sign, consisting of a large, wedge-shaped opacity extending from the right hemidiaphragm to the superior mediastinum, representing thymic tissue displaced from its usual location by a collection of gas under pressure (7).

Recently one paper has evaluated the feasibility of ultrasound in detecting PM in the neonate (8). This small study reports that the most common sonographic finding in PM was a thick echogenic line along the anterior margin of the thymus but sometimes on the lateral or posterior margin of the thymus. Jung et al concluded that ultrasound cannot replace chest radiography, but maybe this could be a first step towards a diagnostic rx-free as more and more is going on for lung diseases.

Clinical management

In the case of symptomatic PM clinical management is clearly based on symptoms and therefore mainly aimed at respiratory support pending the spontaneous resolution of PM. If an infectious etiology was suspected the respiratory support will be associated with antibiotic therapy.

Much more difficult is to determine which is the most appropriate and safe clinical management for infants with asymptomatic PM. This is an important question because the unexpected diagnosis of PM, although asymptomatic, is usually followed by admission of the infant to the neonatal intensive care unit for monitoring and surveillance of the course of the pathology, which causes a painful separation between mother and infant which can negatively affect breastfeeding and the process of bonding.

So, if PM is suspected is essential to perform a chest X-ray in two projections for the definitive diagnosis of the disease even in asymptomatic newborns.

Questionable is the need to hospitalize these infants in neonatal wards (in case they are completely asymptomatic and without radiological signs of other intrathoracic airleak). Probably these infants could continue a more physiological hospital stay close to their mothers, making a simple clinical monitoring.

These infants are closely monitored for the possible evolution of the asymptomatic PM in pneumothorax although this evolution is not described with certainty in this population, while it is well-known the risk in symptomatic patients (3, 9).

Unfortunately, the literature does not explore what is currently the best choice and therefore the most common approach is conservative, resulting in hospitalization of the newborn.

Conclusions

Neonatal PM is a well-known pathology since a long time but the study of the real risks related to it, especially in a population of asymptomatic newborns, it is very lacking in the literature.

In order to ensure a physiological course of the first days of life, without neglecting safety, future studies will be needed to assess how effectively helpful or harmful is medical intervention in this disease in asymptomatic infants.

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