ORIGINAL RESEARCH PAPER
Volume - 13 | Issue - 02 | February - 2024 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

INTERPRETING THE NEONATAL CHEST RADIOGRAPH: A GLIMPSE INTO THE RESPIRATORY DISTRESS OF NEWBORNS

Respiratory Medicine

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ABSTRACT

Respiratory distress is one of the leading causes of neonatal morbidity and mortality. Factors such as gestational age at birth, pulmonary maturity, and congenital factors are peculiar to this demographic. Clinical evaluation accompanied by chest radiography is the standard protocol for evaluating the underlying causative factors. Knowledge of the radiographic appearances of various pathologies and associations with certain congenital factors is quintessential for radiologists and primary neonatal care providers to steer the management in the right direction.

KEYWORDS

INTRODUCTION

Distress related to breathing is a sign, not a diagnosis. Breathing becomes more difficult, resulting in nasal flaring, tachypnea, grunting, and chest retractions, among other symptoms. Not all newborns exhibiting respiratory symptoms also experience respiratory distress. A somewhat delayed adaptation to the extrauterine environment may be the cause of several symptoms. In newborn care units, however, respiratory distress is the most frequent reason for admission. Compared to term neonates, preterm neonates experience respiratory problems more frequently. Respiratory distress can be caused by a number of things, such as intrauterine infections, congenital diseases, perinatal stress, newborn lung development, and multisystem illnesses. As the most accessible imaging modality with the highest level of sensitivity and the least amount of radiation exposure, chest radiography is frequently used to Evaluate the newborn's heart and pulmonary systems. The main causes of infant respiratory distress are categorized in the following article along with information on how they look on radiographs.

Pediatric Airway Anatomy

Pediatric airway and respiratory system anatomy is different from its adult counterpart, which makes it prone to various pathologies and susceptible to infections. The differences are more pronounced at the time of birth and infancy. The neonatal head is disproportionately larger than the neck and the mandible. Newborn tongue is large and posteriorly placed and there is physiological enlargement of tonsils and adenoids. Pediatric trachea is shorter than that in adults and glottic opening is higher than that of adults. The narrowest part of the pediatric airway is at the level of cricoid cartilage, whereas in adults the narrowest part of upper airway is at the glottic opening. Normal pediatric subglottic airway has rounded shoulders (convex outwards). Airways tend to be smaller, with poorly developed pores of Kohn and canals of Lambert and thus reduced collateral pathway of aeration. Pulmonary alveoli are thick-walled at birth and fewer in number as compared to adults

Rules While Performing a Pediatric Chest Radiograph

It is essential to observe imaging guidelines while performing a pediatric chest radiograph to get diagnostic quality images. Most of these are also applicable to neonatal radiography. Rotation should be eliminated with an upright image as far as possible. Appropriate exposure factors should be used, keeping in mind the as low as reasonably achievable (ALARA) principle. Increasing kVp and decreasing mAs within the optimal range to allow adequate exposure to the image receptor is advisable for lowering patient dose. Exposure should be timed with full inspiration and the image should be centered between C3 vertebra and lower costal margin with proper film placement at the level of the upper lip to include upper airway. Care should be taken to avoid over-collimation, or else conditions such as diaphragmatic hernia may be missed. Appropriate lead side marker should be placed. Exposure precaution in the form of lead shielding for gonads is very important. Grid is not necessary for younger children as the soft tissue thickness is not adequate to warrant its use. Also, grids tend to increase the radiation dose to patients

The Fundamentals of Reading a Neonatal Chest Radiograph

Standard chest radiograph is an anteroposterior projection. Additionally, lateral view may be occasionally taken to assess pleural effusion and pneumothorax or an oblique view for localizing a pathology in relation to adjacent structures.[1] The cardiac silhouette may appear enlarged during the first few hours of birth due to the inflow of additional placental blood and bidirectional blood flow through arterial duct and foramen ovale prior to closure.[2] In newborns, the thymus is big and can resemble a mediastinal mass. The unusual sail sign, which is more common on the right, and the notch sign, which appears where the inferior border meets the cardiac profile, can be used to identify it [Figure 2]. One relative measure of neonatal maturity is the existence of humeral head ossification. Air is first observed in the stomach shortly after delivery and then moves up to the rectum in the first few hours of life. Before searching for other illnesses, it is important to determine the proper positioning of the tubes and lines [Figure 3]. For example, an improperly positioned nasogastric tube in the left hemithorax may indicate a diaphragmatic hernia, while coiling in the upper esophagus may indicate esophageal atresia.

Respiratory Distress in Neonates

The causes of respiratory distress can be classified into four main categories namely medical causes, surgical causes, congenital anomalies and diseases and lastly systemic causes.

A. Health-related reasons Diffuse lung parenchymal alterations result from these. According to radiography, pneumonia brought on by beta hemolytic streptococci or respiratory distress syndrome caused by surfactant deficit are the main culprits for low lung volumes and coarse reticular opacities. Considerations include meconium aspiration syndrome, neonatal pneumonia, or transitory tachypnea in a newborn if the radiograph shows large lung volumes together with streaky perihilar opacities. The age of the neonate, or whether the child is born at or after a typical gestational age, is a crucial factor in determining the underlying reason, though.

I] Preterm Infant

There are five stages of pulmonary maturation: acinar, saccular, alveolar, pseudoglandular, and embryonic.[3] In the acinar phase, alveolar ducts form. involves the emergence of type II pneumocytes that produce surfactants. The majority of this surfactant-producing cell growth takes place between weeks 24 and 34 of pregnancy. Alveolarization, which is defined as an increase in alveolar mass and a decrease in air space diameter, starts from 36 weeks of pregnancy and lasts for three years after birth.[4] Surfactant diffuses the alveolus's high surface tension. Numerous variables influence the development of the structure and function of the lungs, and as a result, they may be to blame for conditions that cause respiratory distress.

1. Immature Lung

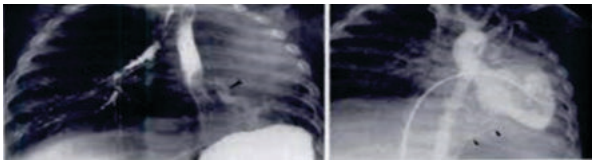
It is seen in neonates of 23–26 weeks' gestational age with birth weight below 1500 g or severe low birth weight below 1000 g. Their initial radiographs may appear unremarkable; however, towards the end of the first week, lungs become hazy and opacified on radiographs, not hypoaerated or hyperaerated and without air bronchograms [Figure 4]. They may develop congestive cardiac failure due to a patent ductus arteriosus and many proceed to develop bronchopulmonary dysplasi

2. Respiratory Distress Syndrome

Respiratory distress syndrome (RDS) is an acute injury to immature

lungs, affecting neonates typically in the 26– 33 weeks' gestational age group. However, it can also occur in term infants after cesarean sections, perinatal asphyxia, or in newborns of diabetic mothers. One aspect of this immaturity is surfactant deficiency, which leads to alveolar collapse, further reducing functional residual capacity and increasing dead space. This can lead to elevated pulmonary vascular resistance and thus right to left shunting, which in turn compound the hypoxia and hypoxemia. [6] Another aspect is defective surfactant quality.

By the end of the first week, the untreated variety of respiratory distress syndrome starts to show symptoms of recovery. Following early recovery, respiratory decompensation may happen as a result of insufficiency of secondary surfactants [Figure 9]. Cells that produce surfactants are harmed by inflammatory mediators released during conditions including pneumonia, pulmonary edema, or bleeding. [8] Surfactant administration is one way to treat secondary surfactant deficit.



Immature lung. (A) Initial radiograph of a premature neonate born at 24 weeks of gestation, weighing 540 grams shows mild coarsening of interstitial markings (arrowheads). (B) Repeat radiograph after 3 weeks reveals diffuse haziness in bilateral lung fields

Chronic Neonatal Lung Disease/ Bronchopulmonary Dysplasia

Bronchopulmonary dysplasia (BPD) is a chronic lung ailment of infancy caused by high inspired oxygen levels and prolonged high pressure mechanical ventilation. [9] It is uncommon in children who are older than 32 weeks' gestation. A large percentage of preterm infants born weighing less than 1000 grams are likely to need ventilator support. Lung edema and infection are common in these extremely preterm neonates as part of a particular form of bronchopulmonary dysplasia. One way to conceptualize respiratory issues connected to preterm delivery is as a Catch-22 situation, wherein any event that causes lung damage necessitates mechanical breathing, which aggravates the damage to the lungs.

I] Full-Term Infant

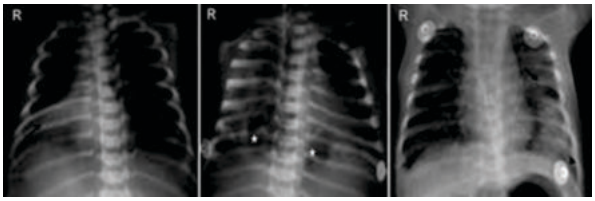
Pneumocytes that produce type II surfactants and definitive alveoli are present in newborns born at full term or after term. However, a number of events, including cesarean birth, amniotic fluid stained with meconium, gestational diabetes, maternal chorioamnionitis, and structural lung abnormalities, among surgical and medical causes, might result in respiratory difficulty in these infants.

Overview of Medical Causes of Respiratory Distress [10,11]

B. Surgical Causes Respiratory distress may be secondary to certain focal pulmonary disorders which have more defined radiographic appearances [14] such as congenital diaphragmatic hernia, bronchopulmonary foregut malformation, congenital cystic adenomatoid malformation (now known as congenital pulmonary airway malformation), congenital lobar overinflation, pulmonary sequestration, and esophageal atresia

1. Congenital Diaphragmatic Hernia (CDH)

Congenital diaphragmatic hernia is considered to be a syndrome rather than an individual entity, consistent of hernia, pulmonary hypoplasia and immaturity, left heart hypoplasia and persistent pulmonary hypertension. The most common site of defect is on the posterolateral aspect of the left hemidiaphragm, known as Bochdalek hernia. Other diaphragmatic hernias include- anterior(typically on the right) known as Morgagni hernia, and central tendon defect (septum transversum defect). Severe diaphragmatic eventration should be treated as CDH.



Pulmonary interstitial emphysema. (A) Frontal radiograph of the chest in a 30-week-old premature infant post surfactant therapy and positive pressure ventilation reveals bilateral cystic lucencies (arrows) and granular opacities (curved arrows) suggesting evolving pulmonary interstitial emphysema. (B) Another preterm infant with development of tubular bubbly lucencies in right lower zone with atelectasis (asterisk) in right upper zone. Note the malpositioned endotracheal tube in right main bronchus (dotted arrow). (C) A localized form of PIE with thin walled pseudocyst (asterisk), commonly seen in right parahilar region

2. Congenital Pulmonary Airway Malformation (CPAM)

A hamartomatous lesion caused by adenomatoid proliferation of bronchial structures that produce cysts and lack alveoli, formerly known as congenital cystic adenomatoid malformation (CCAM). It has to do with providing airways that lack cartilaginous tissue. One lobe is typically the only one affected by CPAM.[15] Five categories exist for CPAM based on the new Stocker classification:[16]

Type 0: lethal; suggests acinar tissue dysgenesis or dysplasia.

Type 1: About half of the patients have one or more large cysts, with each cyst ranging in size from two to 10 centimeters.

Type 2: Many little homogenous cysts less than 2 cm are present in about 40% of patients.

Type 3, which makes up more than 10% of cases, appears solid but is in line with microscopic cysts.

Type 4: many large peripheral cysts that resemble type 1 when observed through imaging.Characterizing the heterogeneously hyperechoic regions observed on antenatal ultrasonography may be more effectively accomplished with prenatal MRI.

3. Congenital Lobar Overinflation (CLO)

Congenital lobar overinflation, also called congenital lobar emphysema, is not a specific condition; rather, it is a group of clinical symptoms associated with specific imaging findings. This is a malformation of the developing brain that results from lobar hyperinflation brought on by bronchial obstruction. The left upper brain is involved in forty to fifty percent of cases, the right middle lobe in twenty to thirty percent, and the right upper lobe in twenty percent of cases. Most of the time, the condition's cause is unknown. There are a few curable causes: (a) parenchymal disorders like polyalveolar lobe or pulmonary alveolar glycogenesis; (b) extrinsic factors; and (c) structural abnormalities of the bronchi, such as esophageal duplication cysts, pulmonary artery slings, mediastinal masses, bronchogenic cysts, and deficient bronchial cartilage.

4. Bronchopulmonary Sequestration

This is a developmental abnormality where focal lung tissue does not demonstrate communication with bronchial tree. It is classified into intrapulmonary and extrapulmonary type, with extrapulmonary (extralobar) type presenting in neonatal period. Sequestration appears as a lower lobe opacity and is indistinguishable from congenital pulmonary airway malformation or neonatal pneumonia on radiographs

C. Thoracic Anomalies and Diseases

That Are Congenital Respiratory discomfort may be the first sign of some congenital abnormalities to appear after birth. Both extrapulmonary and intrapulmonary forms are possible. The previous section covered the main intrapulmonary causes. Extrapulmonary causes include: (a) deformities of the bronchopulmonary foregut; (b) congenital cardiac reasons; and (c) abnormalities of the chest wall. Central airways include tracheomalacia, trachea esophageal fistula, and bronchomalacia.

Congenital cardiac causes Congenital cardiac diseases lead to severe metabolic acidosis and pulmonary edema that are responsible for respiratory distress in newborns. While metabolic acidosis occurs due to hypoxia which is seen in multiple congenital cardiac diseases, pulmonary edema is seen in association with left-sided heart failure or obstructed pulmonary venous return. [19] The presence of cyanosis and cardiac murmurs with less severe respiratory distress points towards an underlying heart disease. Pulmonary vascular congestion secondary to cardiac causes presents with increased vascular markings, cardiomegaly, interstitial and alveolar edema. Patent ductus arteriosus is a common cause of respiratory distress in preterm newborns, presenting with prominent interstitial markings and cardiomegaly due to left to right shunting [Figure 19A]. Atrioventricular canal defect, ventricular septal defect causes respiratory distress in term- and post-term newborns

Chest wall abnormalities Chest wall hypoplasia, either idiopathic or secondary to various skeletal dysplasias may present as respiratory distress in newborns [Figure 20]. Other congenital chest walls disorders such as pectus excavatum, pectus carinatum and Poland syndrome, while clinically appreciable in neonates, may become symptomatic only in late childhood or adulthood.

D. Systemic Causes Respiratory Distress In Newborns Is Not Solely Due To Pulmonary Causes. There Are Many Systemic Causes Which May Lead To Respiratory Distress Such As:

1. Metabolic causes—acidosis, hypoglycemia
2. Infections—sepsis, neonatal TORCH infections
3. Anemia
4. Central nervous system disorders—hypoxic ischemic encephalopathy, vein of Galen malformation
5. Neuromuscular disorders—neonatal myasthenia gravis

Cardiac reasons include newborn myocarditis and cardiac tamponade. A thorough clinical history, physical examination, and laboratory assessment are necessary for an accurate diagnosis. While some cases may not show radiographic abnormalities, few circumstances, such as newborn sepsis and heart disorders, necessitate radiographic assessment. Focal lung infiltrates or consolidation are possible signs of neonatal sepsis

CONCLUSION

A thorough evaluation of neonatal chest radiographs is important for disease classification and management. Despite similar appearances in various pathologies, clinico-radiological correlation can help in accurate diagnosis and timely management. Due to limitations of utilization of other imaging modalities in this age group, sound knowledge of radiographic appearances forms the backbone of neonatal respiratory distress management.

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