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Radiology of the chest in neonates

Susan J. Morris

Consultant Radiologist, University Hospital of Wales, Heath Park, Cardiff, UK

KEYWORDS
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Summary The unique diseases encountered in the neonate, their small size, fragile nature and increased susceptibility to the damaging effects of radiation all combine to make their imaging both challenging and interesting for the radiologist and radiographer. All radiation exposures must be justified. Meticulous attention to detail is a prerequisite to performing a good chest x-ray in young infants, requiring dedicated staff and equipment. Low dose techniques and adequate lead protection must always be employed. A knowledge of the normal radiological anatomy in the neonate are essential for correct interpretation of the films, as well as the appearance and evolution of the disease processes which occur in this age group. A team approach with good communication between obstetricians, paediatric surgeons, neonatologists and radiologists is vital as interpretation of the images is dependent on many clinical and technical factors. Different imaging modalities are available each with specific uses, risks and limitations, knowledge of which is necessary for their appropriate use. An overview of many of the common abnormalities seen in the chest in the newborn period is described and illustrated.

PRACTICE POINTS

- Assess technical factors of the film first (name,side markers, exposure, rotation, inspiratory effort, motion artifact)
- Compare with previous films if available
- Check position of lines, tubes and drains
- Systematic approach in looking at film
- Pertinent clinical history which may be helpful including prenatal history and scan findings
- Discuss with radiologist the need for further imaging (US, CT, MRI)

INTRODUCTION

The unique diseases encountered in the neonate, their small size, fragile nature and increased susceptibility to the damaging effects of radiation combine to make their imaging both challenging and interesting. A team approach with good communication between obstetricians, paediatric surgeons, neonatologists and radiologists is vital as interpretation of the images is dependent on many clinical and technical factors. Different imaging modalities are available, and knowledge of their specific uses, risks and limitations is necessary for their appropriate use. An overview of many of the common abnormalities seen in the chest in the newborn period is described and illustrated in this paper.

Correspondence to: SJM. Tel.: +44 (0) 2920 74 4887; fax: +44 (0) 2920 74 3069

RADIATION RISK

Irradiation of radiosensitive bone marrow, present in most bones at birth, increases the risks of induced leukaemia and genetic effects.[1] Unnecessary or repeated exposures, poor technique and lack of lead protection increase the risk, so meticulous care must be taken to minimize radiation while still providing diagnostic images. Alternative modes of imaging that do not use ionizing radiation should be considered wherever possible. Computed tomography (CT) and magnetic resonance imaging (MRI) pose particular difficulties in the neonate; the latter requiring highly specialized anaesthetists and dedicated equipment. The radiology department is not the ideal environment for sick infants.

TECHNICAL FACTORS IN PERFORMING A CHEST X-RAY

The plain chest x-ray is still the most frequently requested radiological examination on the neonatal unit. It requires a core group of staff responsible for performing the examination and ensuring, through audit, that acceptable image quality is maintained.

The use of a high-speed film/screen system with a mobile x-ray unit with a powerful high-frequency or constant potential generator is recommended.[2] A short exposure time, high KV-low MAS technique is employed and the exposure factors must be recorded on the film. Tight collimation is essential, and lead shielding should be placed on the baby and incubator. Unnecessary lines and tubing should be removed from the chest area, and the infant should be immobilized by holding the arms flexed either side of the head. The mandible must not obscure the upper chest, and the field of view must include the cervical trachea as its upper limit and T12/L1 vertebral bodies as the lower limit.

Errors in interpretation may occur due to poor technique. A film taken in expiration may cause the lungs to appear consolidated. Rotated films produce an increased lucency on one side, and line positions appear misplaced. A lordotic film alters the appearance of the heart contour and the apparent position of the endotracheal tube (ETT).

The recent increased use of digital imaging and storage systems has many benefits on the neonatal unit; previous films are instantly available and the image can be manipulated to optimize viewing even in poorly exposed films, resulting in fewer repeat exposures.

THE NORMAL CHEST

Having first assessed these technical factors, the position of the lines, drains, tubes and catheters should be noted. The ideal location of the ETT tip is 1 cm above the carina. The umbilical arterial catheter (UAC) tip should be above the coeliac axis and superior mesenteric arteries, preferably in the lower thoracic aorta. The umbilical venous catheter (UVC) tip should be just above the liver.

The normal shape of the chest in the newborn is trapezoid with horizontal ribs. The thymus is prominent but rapidly involutes during the first few days of post-natal life, especially in stressed infants. It is a smooth, usually bilateral quadrilateral shape of soft tissue density. Pulmonary vessels can be seen through it. A notch may be seen at its inferior edge adjacent to the heart, and a 'sail'-like projection from the mediastinum and scalloping of the lateral or inferior margins are not uncommon. The assessment of heart size in young infants is difficult; cardiothoracic ratio is not usually helpful although a normal upper limit of approximately 65% can be used. Pulmonary vascular markings taper gradually towards the periphery of the lungs, and air bronchograms are commonly seen in the medial third of the lung bases.[3]

PROBLEMS IN THE PRE-TERM INFANT

Respiratory distress syndrome

Respiratory distress syndrome is a disease of hypoventilation and a manifestation of pulmonary immaturity and surfactant deficiency. Surfactant usually coats the alveolae and prevents atalectasis by lowering surface tension. In respiratory distress syndrome, the lungs are poorly compliant with acinar atelectasis, and there is a gradual development of thickening of the interstitium and dilatation of the terminal airways. Radiological abnormalities correlate well with the clinical severity. There is reticulogranular or ground glass opacification, progressive hypo-aeration and air bronchograms (Fig. 1). Symptoms and radiological signs progress during the first 6 h of life, and in mild to moderate disease, the granular densities persist for 3–5 days, clearing from peripheral to central and upper to lower lungs. Artificial surfactant has contributed to a reduced frequency of pneumothoraces and improved early survival of these infants, although the long-term consequences of bronchopulmonary dysplasia may still occur. Uneven distribution of surfactant can cause patchy areas of increased aeration, radiologically simulating pulmonary interstitial emphysema or meconium aspiration.[4]

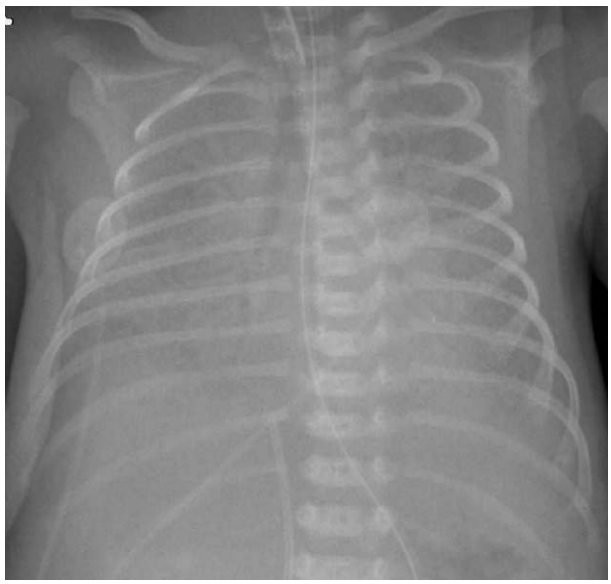

Figure 1 Respiratory distress syndrome.

Air block complications of RDS

Decreased lung compliance and high positive pressure ventilation may lead to air leaks from the pulmonary parenchyma, resulting in pneumothorax, pneumomediastinum and even pneumoperitoneum (Fig. 2). If air leaks into the interstitial space and spreads through the lymphatics along the perivascular sheaths, it leads to pulmonary interstitial emphysema. Radiologically, this produces a pattern of small bubbles radiating from the hilum.[5] If this becomes extensive, it compresses the airways, reducing gas exchange; if severe, it can even result in cardiac compression. Unlike pneumothorax, pulmonary interstitial emphysema cannot be drained and can result in extensive lung damage.

Bronchopulmonary dysplasia (chronic lung disease of prematurity)

A combination of barotrauma, oxygen toxicity, infection, deficiency of anti-oxidant defences and altered inflammatory response in the pre-term lung results in bronchopulmonary dysplasia.[6] The early radiological appearance is of coarse interstitial densities interspersed with small cystic-like areas. This progresses over weeks to months to generalized hyperinflation with larger cystic areas, atelectasis and fibrosis (Fig. 3). The heart is frequently enlarged due to right ventricular strain and cor pulmonale. Wilson Mikity syndrome produces the same radiological picture but in immature infants who have had minimal ventilatory support.[7]

Pulmonary haemorrhage

Bleeding from the airway, often associated with sudden deterioration in respiratory function, suggests acute pulmonary haemorrhage. There is a spectrum from a small amount of fresh blood to catastrophic cardiovascular collapse. The chest x-ray findings are non-specific, ranging from small areas of opacification to a complete whiteout of one or both lungs.

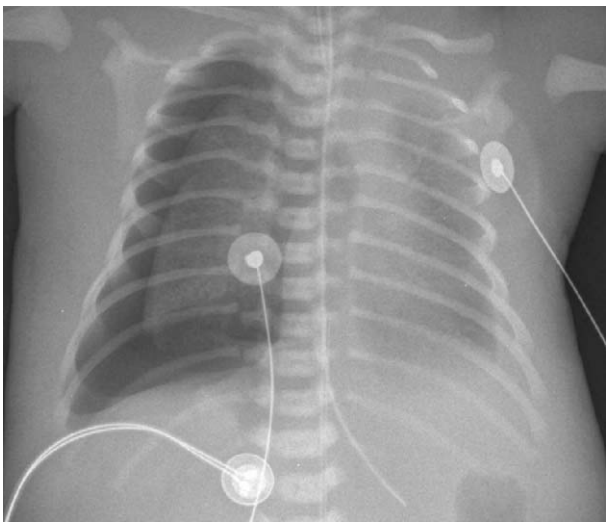

Figure 2 Respiratory distress syndrome with right tension pneumothorax.

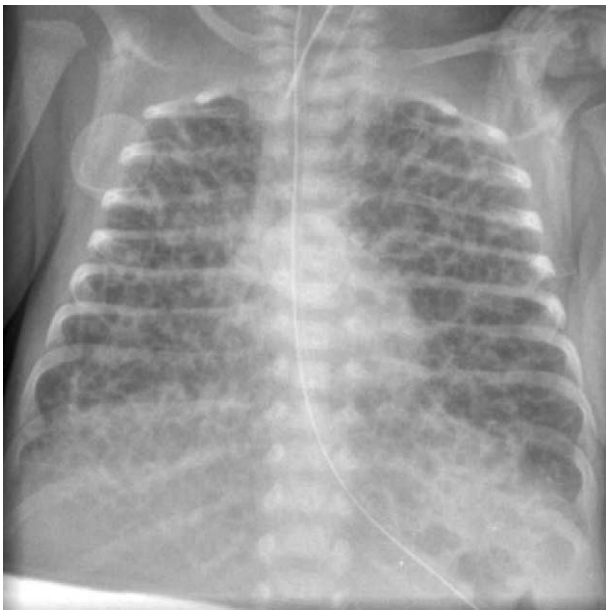

Figure 3 Bronchopulmonary dysplasia.

PROBLEMS IN THE TERM INFANT

Neonatal pneumonia and persistent pulmonary hypertension of the newborn

Pneumonia in the newborn occurs in approximately 0.2% of live births.[8] It may present early with respiratory distress, and clinical features and chest X-ray findings indistinguishable from respiratory distress syndrome due to associated surfactant deficiency. A history of prolonged rupture of membranes may be present. The most common organisms are group B β-haemolytic streptococcus or coliforms. The chest x-ray findings are non-specific. Lobar collapse and consolidation are unusual in neonatal pneumonia, and are more likely to be due to poor ETT positioning.

Persistent pulmonary hypertension of the newborn can occur as a consequence of pneumonia, hypoxia, birth asphyxia and sepsis, and is associated with pulmonary hypoplasia. It may also develop without any obvious predisposing cause.[9] Failure of the right heart pressure to fall, leads to the ductus arteriosus remaining open and a persistent right to left shunt. The chest x-ray may show evidence of pneumonia, but the lung fields may have minimal changes or there may be oligaemia. The echocardiogram is helpful in demonstrating the right to left shunt.

Transient tachypnoea of the newborn

Delayed clearing of the amniotic fluid from the lungs during the first few breaths results in 'wet lungs' or transient tachypnoea of the newborn. The infant presents within the first few hours of life with tachypnoea and mild to moderate hypoxia. Predisposing factors include Caesarean section, precipitous delivery, birth asphyxia and maternal diabetes. There is usually a gradual clinical improvement with minimal respiratory support over the subsequent few days. The chest x-ray typically shows normal volume lungs and streaky perihilar markings representing the distended pulmonary veins and lymphatics. There may be small effusions and fluid in the horizontal fissure. Clearing starts at about 12 h starting peripherally and from the upper lung fields.[10]

Meconium aspiration syndrome

Meconium staining of amniotic fluid is present in approximately 10–15% of live births but only 1–1.5% have MAS. The viscid, hyperosmolar substances are aspirated resulting in widespread, patchy, asymmetrical opacities with areas of collapse and hyperinflation from air trapping due to the ball-valve effect of the meconium plugs (Fig. 4). There is a spectrum of severity depending on the quantity of meconium aspirated, and up to 25% develop air block complications of pneumothorax and pneumomediastinum.[11] A chemical pneumonitis causes slow resolution of the opacification. Secondary surfactant deficiency and superimposed pneumonia are common. Survivors may have long-term respiratory failure due to lung damage.

Pleural effusions and chylothorax

Pleural effusions may be part of generalized hydrops, secondary to perinatal infection, or due to abnormal lymphatic drainage of the pleural spaces.[5] Chylothorax is the most common cause of a large pleural effusion in the newborn, although the pleural fluid does not become chylous until milk has been ingested (Fig. 5). Increasingly, large effusions are detected pre-natally when they are sometimes drained by thoracentesis or intra-uterine thoraco-amniotic shunt insertion in an attempt to prevent pulmonary hypoplasia. The male to female ratio is 2:1 and most affect the right hemithorax. The majority can be treated with single or repeated thoracocentesis and appropriate feeding with medium-chain triglycerides.

Congenital abnormalities of lung development

By the third week of gestation, the primitive larynx, trachea and lung buds develop from a ventral diverticulum of the foregut. The oesophagotracheal ridges split the oesophagus from the trachea proceeding from caudal to cranial. By 6 weeks, primitive lung buds are present representing the early bronchial divisions. Anomalous budding results in bronchopulmonary foregut malformations including congenital lobar emphysema (CLE), congenital cystic adenomatoid malformation (CCAM) and pulmonary sequestration. Incomplete splitting of the oesophagotracheal ridge leads to oesophageal atresia with tracheo-oesophageal fistula (TOF) anomalies.[12] By 6 months' gestation, 17 generations of branching have occurred. Terminal saccules are present and the number

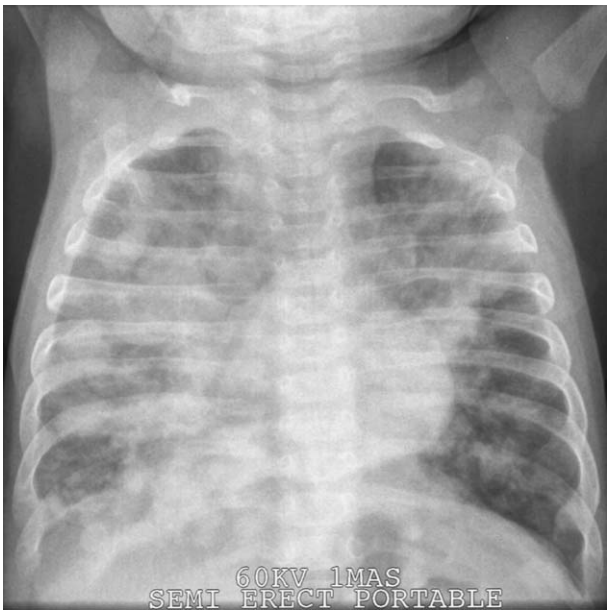

Figure 4 Meconium aspiration syndrome.

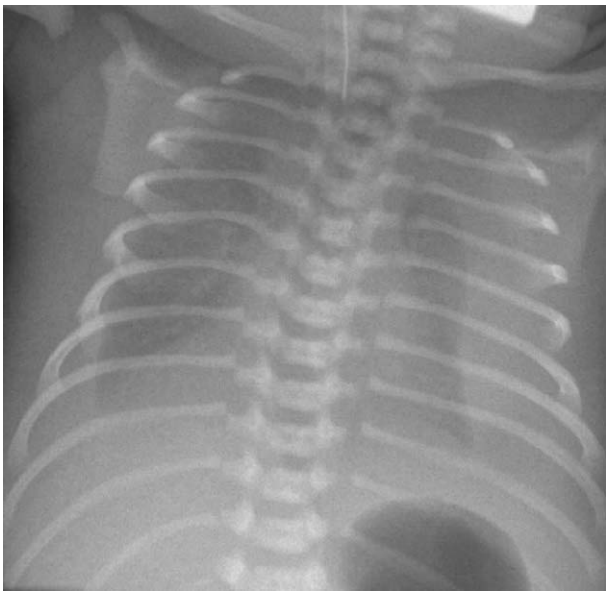

Figure 5 Bilateral chylothorax.

increases until birth and beyond. Surfactant-forming pneumocytes appear at approximately 24 weeks' gestation. True alveolar development occurs after term for up to 3 years.

Pulmonary agenesis and hypoplasia

Pulmonary growth and development can be affected at different stages of fetal life resulting in hypoplasia or agenesis of one or both lungs. Restricted lung growth occurs due to oligohydramnios as in renal agenesis (Potter's syndrome) or infantile polycystic kidneys, or in premature rupture of membranes. The thorax is small and bell-shaped with small-volume lungs. The abdomen appears hugely distended in cystic kidney disease (Fig. 6). Pneumothoraces are common. Unilateral lung agenesis appears as a completely opaque hemithorax with rib crowding and displacement of the mediastinum towards that side (Fig. 7). Associations with lung aplasia are cardiac anomalies, TOF, imperforate anus, renal dysgenesis and vertebral anomalies.[13] Space occupying lesions within the thorax (diaphragmatic hernia, CLE, CCAM, effusions) can compress the ipsilateral lung causing hypoplasia. The contralateral lung may also be affected due to mediastinal compression. Many of these conditions are diagnosed on pre-natal ultrasound allowing for appropriate counselling and planning of delivery of the infant.

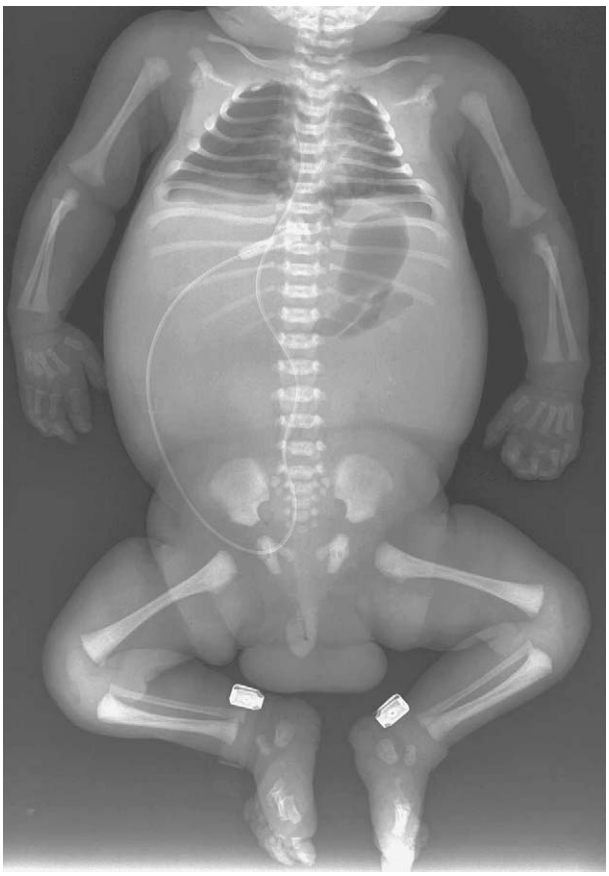

Figure 6 Polycystic kidneys with pulmonary hypoplasia.

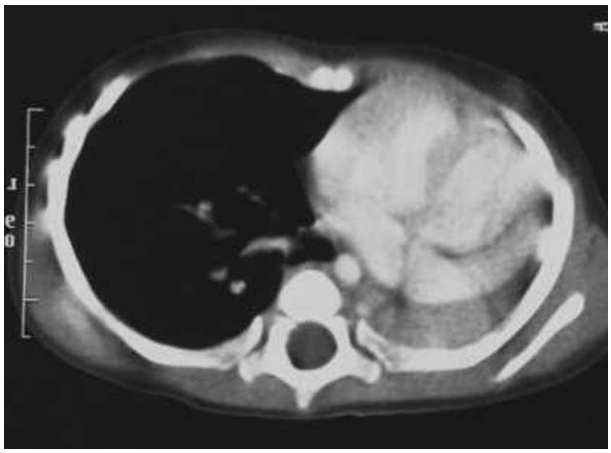

Figure 7 CT scan of left pulmonary hypoplasia.

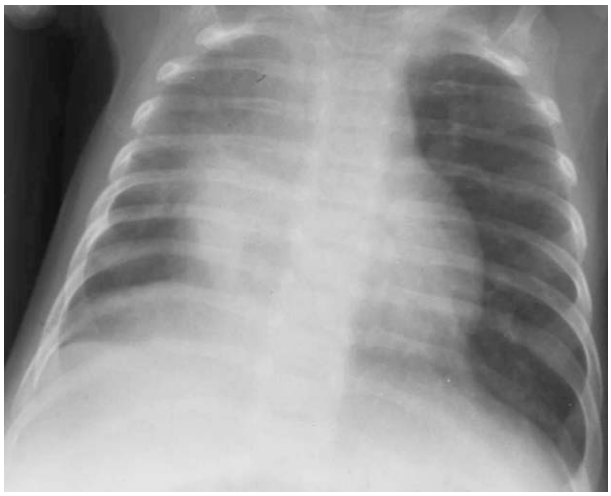

Figure 8 Scimitar syndrome.

Scimitar syndrome (venolobular syndrome) is a form of right lung agenesis or hypoplasia associated with anomalies of pulmonary vessels.[14] The anomalous draining vein is seen as a scimitar-shaped density extending inferiorly towards the diaphragm (Fig. 8). It usually drains into the inferior vena cava but may enter the portal vein, hepatic vein or right atrium. The right pulmonary artery may be partly or completely absent. The condition has been associated with pulmonary sequestration, bronchogenic cyst, atrial septal defect and abnormalities of the diaphragm and bony thorax.

Pulmonary sequestration

This is defined as an aberrant mass of pulmonary tissue, usually at the left base, that has no normal connection with the bronchial tree or pulmonary arterial system. It possesses a systemic blood supply. It is frequently

asymptomatic unless suspected from antenatal scans or recurrent chest infections. It may be associated with foregut malformations and CCAM. Systemic feeding vessels are best demonstrated in the fetus or early neonatal period with Doppler ultrasound, or with MRI angiography.[15] In the newborn, the chest x-ray may be normal, but with repeated infection, a persisting opacity is seen at the lung base. Air fluid levels may also be seen within infected cavities.

Oesophageal atresia with tracheo-oesophageal fistula

This condition may be suspected pre-natally with polyhydramnios and a small fetal stomach, or in the newborn period with choking, cyanosis and coughing during feeds. Failure to pass a nasogastric tube produces the typical chest x-ray with the tube coiled in the upper oesophageal pouch (Fig. 9). Air within distal bowel indicates a distal tracheo-oesophageal fistulous connection. Approximately 50% of babies with tracheo-oesophageal atresia have associated anomalies of the heart, renal system, spine, limbs and anorectal malformations. Further imaging is required to identify these problems. If an H-type fistula is suspected, a contrast-tube oesophagogram is performed to outline the fistulous track. It usually arises anteriorly from the upper trachea near the thoracic inlet and extends cephalad into the oesophagus (Fig. 10).[16]

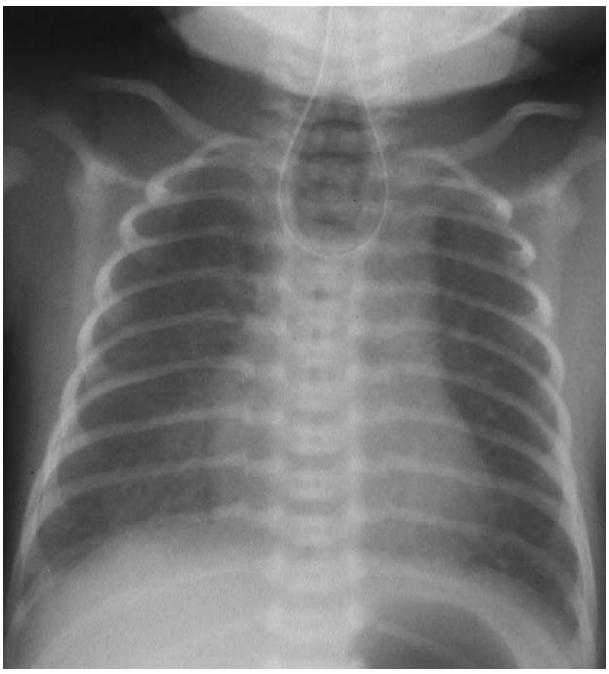

Figure 9 Oesophageal atresia.

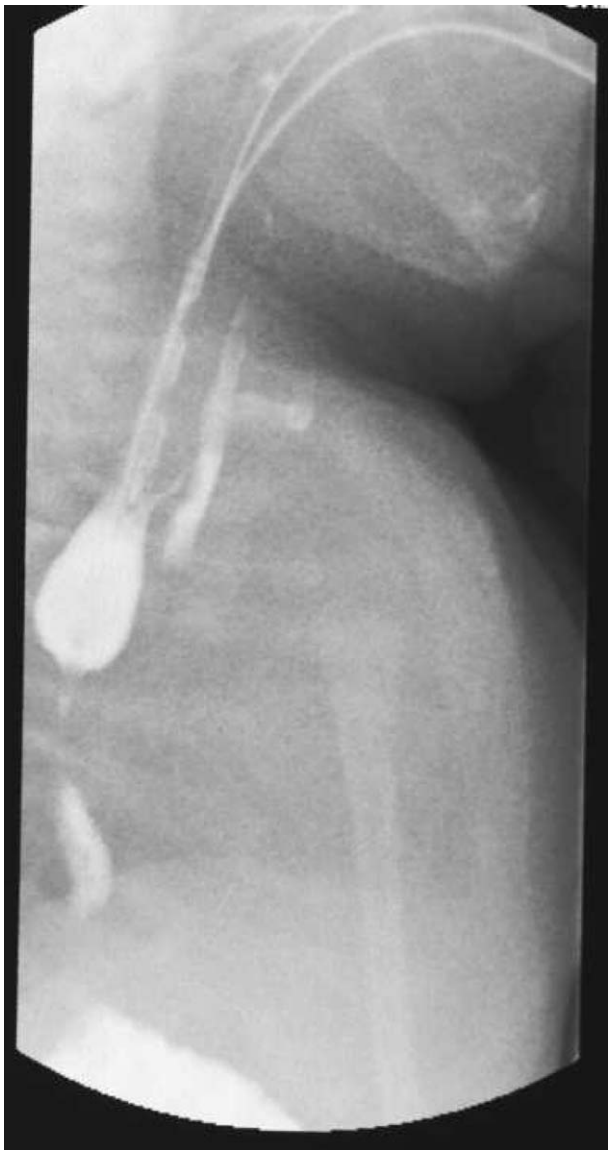

Figure 10 Barium tube oesophagogram showing H-type tracheo-oesophageal fistula.

Congenital lobar emphsema

This condition is characterized by progressive overdistension of (usually) one lobe. The majority are due to some kind of bronchial obstruction causing a ball-valve effect. The male to female ratio is 2:1 and up to 50% have associated anomalies, usually of the cardiovascular system. Upper lobes are most commonly affected, followed by the right middle lobe. On the chest x-ray, there may initially be trapped fluid within the affected lobe, which appears opaque (Fig. 11a). As the fluid re-absorbs, a hyperinflated, hyperlucent lobe produces mediastinal shift and compression of adjacent lung (Fig. 11b). CT is helpful in differentiating this condition from cystic lung lesions (Fig. 11c). The abnormal lobe may require surgical resection if symptomatic; however, many cases resolve spontaneously.[17]

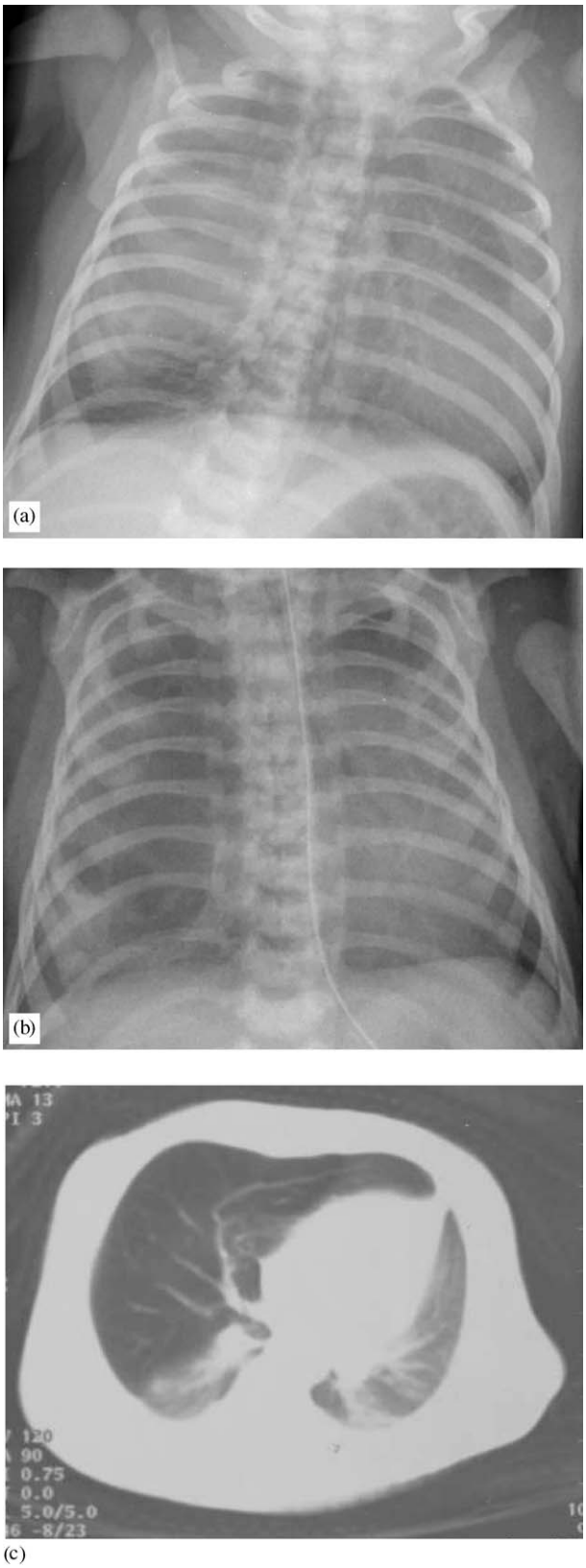

Figure 11 (a) Right congenital lobar emphysema showing mediastinal shift and opacification of the abnormal lung; (b) Right congenital lobar emphysema after clearing of fetal lung fluid; and (c) CT scan of right middle lobe congenital lobar emphysema.

Foregut malformations

Foregut cysts are anomalies of either foregut or tracheobronchial budding. Bronchogenic cysts are commonly mediastinal, either paratracheal or subcarinal, but are occasionally intrapulmonary. They have no communication with the tracheobronchial tree initially.[18] Enteric cysts tend to be in the posterior mediastinum and include oesophageal duplication cysts. They may contain gastric mucosa, and spinal anomalies are associated. Neurenteric cysts have a similar radiological appearance of a posterior mediastinal soft tissue mass; however, vertebral anomalies are more common. MRI is the imaging modality of choice when the spine is involved; otherwise, CT defines the extent and relationship of these masses to other structures.

CCAM is diagnosed with increasing frequency in the pre-natal period. It is caused by a hamartomatous proliferation of terminal bronchioles. The cysts communicate with the bronchial tree. Histologically, type I has cysts of varying sizes, at least one being more than 2 cm (approximately 50%). Type 2 has small uniform cysts of 1–10 mm and is associated with other congenital abnormalities. Type 3, composed of microcysts, has the poorest prognosis and is associated with fetal hydrops. The infant may present early with respiratory distress due to the mass effect and pulmonary hypoplasia. Late presentation due to recurrent infection also occurs. The initial chest x-ray may show mediastinal shift and opacification of the abnormal lung. As fluid is resorbed, the hyperinflated area is hyperlucent with a bubbly appearance of multiple cysts. In many cases, the initial chest x-ray is normal; CT scanning demonstrates the extent of the abnormality and differentiates it from other cystic lung abnormalities (Fig. 12).[5,19]

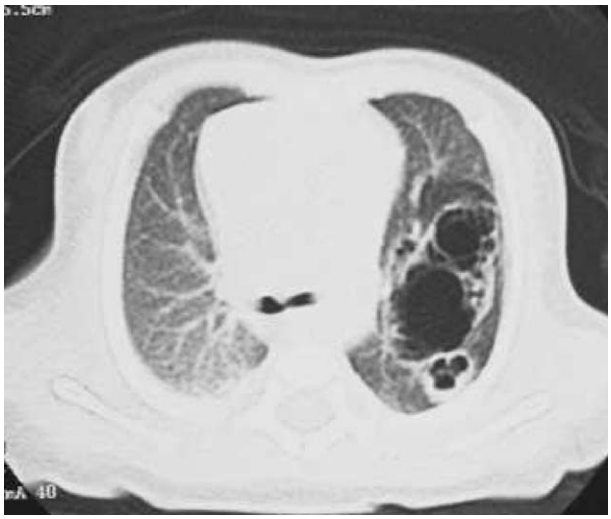

Figure 12 CT scan of left congenital cystic adenomatoid malformation.

Congenital diaphragmatic hernia

Abdominal viscera herniate through the posterior pleuroperitoneal fold into the chest causing compression of the lungs and mediastinal shift; it is most frequent on the left (75%). The diagnosis is often made pre-natally. There is usually severe respiratory distress due to associated pulmonary hypoplasia. The typical chest x-ray shows mediastinal displacement and air-filled loops of bowel occupying one hemithorax (Fig. 13).[5] Liver, spleen and stomach can all be herniated into the chest. The prognosis correlates closely with the degree of underlying lung hypoplasia.

Chest masses

Congenital cysts and tumours of neurogenic origin (neuroblastoma, ganglioneuroma, neurofibroma) make up most of the mediastinal masses encountered in the neonatal period, comprising 50–60% of all masses. Teratomas and dermoid tumours account for 15–20%. The remainder is made up of thymomas, haemangiomas, cystic hygromas and sarcomas.[3] Calcification may be present in neurogenic tumours and teratomas. Compression of local structures with displacement of the trachea and bronchi can cause stridor and lobar collapse. Neuroblastoma can cause rib and vertebral erosion, and intraspinal extension can enlarge intervertebral foramina and cause pedicular flattening and spinal canal widening. Cystic hygromas in the thorax are usually from direct extension from the neck.

CT or MRI with its multiplanar imaging ability are the imaging modalities of choice for thoracic tumours.

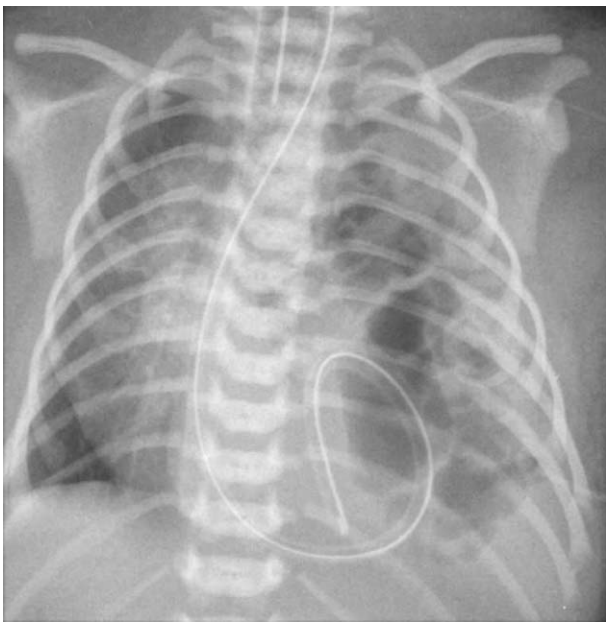

Figure 13 Congenital left diaphragmatic hernia.

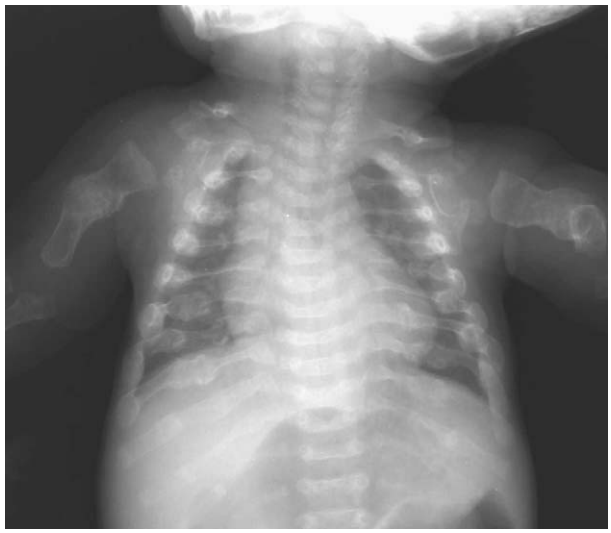

Figure 14 Osteogenesis imperfecta type II. Multiple Rib fractures and short fractures humeri.

Congenital chest deformities

A number of skeletal dysplasias are associated with a very small thorax with concomitant pulmonary hypoplasia.[20] The most severe and more frequently encountered are thanatophoric dysplasia, osteogenesis imperfecta (lethal type) (Fig. 14) and asphyxiating thoracic dystrophy (Jeune's). The radiological features of each syndrome are often diagnostic. Many die in the early neonatal period from lung hypoplasia.

SUMMARY

This review has demonstrated the importance of close liaison of all clinical groups involved in the pre- and postnatal management of these infants who are often critically ill. There may be disorders of many systems and the condition may evolve requiring a frequent review of the imaging and further investigations. A wide variety of radiological tests are available and each must be tailored to the particular clinical circumstance to provide the best information. Highly trained staff and specialized equipment are necessary to minimize the risks while obtaining quality images.

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