6 Infectious Pneumonia of the Newborn

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6.1 Infectious Pneumonia

Infectious pneumonia is the most common disease in newborns and is also an important cause of neonatal death. According to statistics, infectious pneumonia accounts for more than 1/3 of the total number of newborn hospitalizations, and the number of deaths due to infectious pneumonia accounts for more than 1/4 of the total number of deaths in newborns and for more than 1/5 of neonate autopsy cases [1, 2]. Infection can occur inside the uterus, during delivery or after birth, and prenatal pathogens can infect fetuses through the blood circulating through the placenta and amniotic membrane, or pathogenic bacteria can ascend to infect fetuses due to the premature rupture of membranes. In intrapartum cases, infection is induced because fetuses are contaminated with amniotic fluid or maternal cervical secretions during delivery [2, 3]. Postpartum infection primarily occurs through the respiratory tract, through the blood or through an iatrogenic route. Common pathogens include *Escherichia coli*, *Staphylococcus aureus*, viruses (such as cytomegalovirus, herpes simplex virus, rubella virus, *Coxsackie virus*, and chicken pox virus), *Klebsiella*, *Listeria*, *Mycoplasma*, and *Chlamydia* [2, 3].

6.1.1 Etiologic Factors

1. Intrauterine infection: Primary intrauterine infection pathogens include group B β-hemolytic streptococcus, pneumoniae bacteria, *Escherichia coli*, *Klebsiella pneumoniae*, *Bacillus proteus*, cytomegalovirus, rubella virus, and herpes simplex virus. (1) Maternal infection: when the mother suffers from sepsis, viremia, or viremia and chorioamnionitis, pathogens can enter the fetal circulatory system from the mother, through placental barriers, and reach the fetal lungs to induce fetal infectious pneumonia. (2) Premature rupture of membrane (PROM): the PROM and infections in fetuses and neonates are closely related. It was previously thought that the when PROM was longer, the fetal and neonatal infection rates would be increased and the degree of infection would be more serious. However, our recent survey shows that the PROM duration and incidence of neonatal infections have no correlation. For neonates with a PROM time ≤24 h, the positive blood culture rate was 7.7%; with a PROM time of

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24–72 h, the rate was 7.2%; and with a PROM time >72 h, the rate was 9.6%. The differences in the positive blood culture rate among different PROM durations were not statistically significant ($\chi^2 = 2.70, p = 0.259$). When examining the pathogenic bacteria involved in neonatal infections caused by PROM, the proportion of G+ bacteria was significantly greater than that of G− bacilli. Bacteria such as *Staphylococcus epidermidis*, human *Staphylococcus*, *Klebsiella pneumoniae*, *Escherichia coli*, and *Staphylococcus haemolyticus* were among the most common strains that induced a neonate infection after PROM and together accounted for more than 75.0% of the positive blood culture results. Fungi are common pathogenic microorganisms that cause premature infant infection after PROM. (3) Excessive obstetric operations: excessive obstetric operations are prone to cause neonatal infections, and bacteria can infect the fetus either directly or through blood circulation.

2. Intrapartum delivery: Intrapartum infection pathogens primarily include G-bacilli (*Escherichia coli*, *Bacillus proteus*, and *Bacillus gasoformans*), *Salmonella*, and group B β-hemolytic streptococcus. (1) Prolonged second stage of labor: a premature rupture of membranes has not yet occurred, but the patients are in a state of extreme tension; the fetal membrane permeability increases, creating favorable conditions for the invasion of pathogens. (2) Precipitate labor: in precipitate labor, it is often difficult to completely disinfect the sites, thus increasing the chances of infection.
3. Postpartum infection: Postpartum infection is the primary cause of late advanced neonatal pneumonia, and the involved pathogens include *Pseudomonas aeruginosa*, *Klebsiella pneumoniae*, and some conditional pathogenic bacteria with low virulence, such as *Staphylococcus*. (1) Close contact with a respiratory tract infection: if infants inhale air that contains pathogenic microorganisms, various viruses and bacteria can lead to infection after birth. (2) Spread through blood: in cases of damage, infection, and sepsis of the skin and mucous membranes in newborns, pathogens can spread to the lungs through blood circulation. (3) Iatrogenic infection: pathogen accumulation is induced by excessive invasive operations, careless device disinfection, non-strict implementation of sterile operations for working personnel, damage to the integrity of infantile skin during puncture and care, or a drafty atmosphere in the nursery or hospital ward.

6.1.2 Pathophysiology

When lesions are primarily located in the alveoli, alveolar wall congestion, edema, inflammatory cell infiltration, and exudate filling, the alveoli are pathologically visible, causing a reduction in the alveolar diffusion area, an increase in the thickness of the blood gas barrier film, an extension of the diffusion time, and a reduction of the diffusing capacity. During the early stage, alveolar lesions are primarily influenced by the diffusion of oxygen, while during the late stage, they are caused by CO_2 retention. Because the oxygen diffusion capacity decreases, some of the venous blood is transported to the pulmonary veins and the arterial system without being oxygenated in the lungs, leading to a reduction of the arterial partial pressure of oxygen and an increase in the alveolar-arterial oxygen tension difference ($A\text{-}aDO_2$). During severe pneumonia, due to severe hypoxia, pulmonary vascular endothelial cells become swollen, pulmonary vessels become spasmodic and paralytic, pulmonary artery pressure increases, pulmonary capillary permeability increases, blood oozing and flow are slowed, and the lung ventilation/perfusion ratio imbalance worsens, leading to progressive hypoxia and, in severe cases, pulmonary bleeding.

When the lesions are primarily located in the bronchioles and capillary bronchi, the respiratory tract wall is edematous, with inflammatory exudation and increased secretions, causing bronchiolar spasms and ventilation disorders. When the airway is not completely blocked, the inspiration of air is greater than the expiration of air, causing emphysema, decreased alveolar ventilation, and a decreased ventilation/perfusion ratio.

When the airway is completely blocked, some alveoli collapse, atelectasis occurs, the alveolar ventilation/perfusion ratio decreases and becomes further aggravated, and hypoxia is more obvious.

Hypoxia can indirectly stimulate intra-alveolar stretch receptors, which can partially compensate for hypoxia through reflections, tachypnea, and increasing the ventilatory capacity increase, but in cases of serious lung disease or obviously obstructed airways, the compensatory effect is poor and compensation fails. The oxygen consumption of respiratory muscles accounts for 3–5% of the systemic muscle consumption under normal circumstances; in cases where hypoxia cannot be relieved, the infants experience labored respiration, and accessory respiratory muscles are used, causing the oxygen consumption to increase to 5–10 times the normal level, and respiratory failure can be induced due to respiratory muscle fatigue.

6.1.3 Pathology

In cases of intrauterine infectious pneumonia, no special mechanisms are observed. When examined under a microscope, alveoli are involved and full of polynuclear neutrophils and monocytes, erythrocytes are occasionally observed, and the rare exudation of fibrin occurs. Some alveoli have ectasia and contain contents, such as amniotic fluid. In cases of pneumonia caused by the infection of the intrauterine blood circulatory system, alveoli do not contain amniotic fluid contents, but there is a relatively large amount of fibrin exudation.

6.1.4 Clinical Manifestations

1. Intrauterine infectious pneumonia: The onset of intrauterine infectious pneumonia is often early and primarily occurs within 3 days following birth; the patients often have a medical history of asphyxia, and severe intrauterine infections can cause intrauterine fetal death. The manifestations include no crying at birth, difficulty breathing after resuscitation, and the presence of three depression signs, including moaning, cyanosis, and foaming at the mouth. Cough is rare and apnea can be observed. Temperature does not rise or is normal, lung auscultation may indicate that no obvious abnormality is observed, and, sometimes, the symptoms and signs are absent. The primary manifestations of ascending infection are respiratory system symptoms, such as faster breathing, moaning, and abnormal body temperature; in severe cases, the patient can experience respiratory failure, heart failure, convulsions, coma, DIC, shock, and persistent pulmonary hypertension, and lung auscultation indicates that rales and rhonchi can be heard. Hematogenous infections are primarily manifested as jaundice, hepatosplenomegaly, retinal choroiditis, meningoencephalitis, and other multi-system syndromes (fetal lungs are in a compressed state, with the majority of the pulmonary blood flow entering the aorta through the artery catheter and only a small part being diverted into the lungs), and the primary manifestation in the lungs is interstitial pneumonia, usually without rales. Cord blood IgM measurements are >200–300 mg/L, and the specific IgM increase indicates the diagnostic value. The manifestation observed in a chest X-ray examination of those with bacterial infection is usually bronchial pneumonia, while, in those with viral infections, the primary manifestation is interstitial pneumonia.
2. Infectious pneumonia during delivery: Infection is caused by common pathogens (the most common is *E. coli*, followed by *Streptococcus pneumoniae*, *Klebsiella*, *Listeria*, and group B hemolytic streptococci), *Chlamydia trachomatis*, herpes simplex virus, and mycoplasma. The time of onset and the type of infection pathogen are related, and the onset is generally late, appearing after a certain incubation stage. *Chlamydia trachomatis* infection often occurs after 5–14 days, with manifestations of purulent conjunctivitis and the presence of inclusion bodies in conjunctival epithelial cells, and *Chlamydia trachomatis* can be isolated from the nasopharynx. A

cough, which is paroxysmal, without heat or fever, occurs only after 2–12 weeks, and fine crackles can be observed in the lungs; the manifestation observed in the chest X-ray is local interstitial pneumonia. During herpes simplex virus infection, herpes can be observed on the head skin at birth, appearing 5–10 days after birth, and the symptom of meningitis is often more prominent. Bacteria (such as *E. coli* and other intestinal bacteria)-induced pneumonia usually occurs 3–10 days after birth; in addition to respiratory symptoms, it often leads to sepsis, and the onset occurs as an even outbreak with a high mortality; chest X-ray examinations present the manifestation of bronchopneumonia.

3. Infectious pneumonia after birth: The routes of pathogen invasion include descending infection (pathogens spread through droplets from the respiratory tract to the lungs), hematogenous infection, and iatrogenic infections (such as ventilator-associated pneumonia, the use of broad-spectrum antibiotics). A variety of viruses, bacteria, and other microorganisms can cause pneumonia infection after birth, and the common pathogens of ventilator-associated pneumonia are *Klebsiella pneumoniae*, *Pseudomonas aeruginosa*, and *Escherichia coli*. Those who undergo the long-term use of broad-spectrum antibiotics are prone to experience *Candida albicans* pneumonia, and those with immune deficiencies are susceptible to *Pneumocystis carinii* pneumonitis. The respiratory symptoms include difficulty breathing, flaring of nares, spittle, cyanosis, nodding breathing, and restractions, but pulmonary symptoms are not typical. Respiratory syncytial virus pneumonia may be manifested as wheezing, and lung auscultation indicates that wheeze can be heard. Pneumonia caused by different pathogens may have different results from chest X-ray examinations, with the primary manifestation of viral infection being interstitial pneumonia and the primary manifestation of bacterial infection being bronchial pneumonia. *Staphylococcus aureus* infections are apt to be complicated by empyema and pyopneumothorax. Bacterial culture of nasopharyngeal secretions, virus isolation, fluorescent antibody tests, and serum-specific antibody tests is used to aid in the etiological diagnosis.
4. Common Complications
   (a) Congestive heart failure: In cases of pneumonia, heart failure often occurs due to the following reasons:
      - Hypoxic acidosis induces pulmonary vascular spasms and increased pulmonary artery pressure.
      - Inflammatory exudation causes pulmonary edema, increasing the before and afterload and causing right heart failure.
      - Myocardial hypoxia-ischemia, energy metabolism disorder, bacterial toxins, acidosis, and electrolyte imbalance induce the cardiac muscle cell to experience ultrastructural damage, excitation-contraction coupling disorder, and decreased myocardial contractility.
      - Lower diastolic blood pressure and decreased coronary blood flow may further weaken myocardial contractility.
      - In cases of severe pneumonia, under the influence of bacterial toxins, the peripheral α-receptor is excited, vasoconstriction occurs, and the left heart afterload increases, causing a decrease in cardiac output; simultaneously, the sympathetic nerve excitation causes the redistribution of systemic blood flow, decreases the renal blood flow, activates the renin-angiotensin-aldosterone system, causes an increase in water and sodium retention and the returned blood volume, and increases cardiac preload, causing congestive heart failure.

   (b) Acid-base balance disorder: In cases of severe pneumonia, due to an increase in anaerobic metabolism, a large amount of lactic acid is generated, causing lactic acidosis and leading to metabolic acidosis. During the early stages, hypoxia may be complicated by mild respiratory alkalosis, due to ventilatory compensation; the

seriousness and progression of patient conditions cause CO_2 retention and induce mixed acidosis. However, metabolic alkalosis rarely occurs except in the case of excessive alkali supplementation or improper mechanical ventilation during the treatment. When the pH <7.2, kidney function becomes involved, urination is stopped, liver function is impaired, and the activity of enzymes involved in the metabolism of bilirubin is inhibited, which can induce bilirubin encephalopathy; during acidosis, the response of the body to catecholamines weakens and dysemia occurs. When the pH <7.0, myocardial glucose metabolism is fully terminated, followed by circulatory failure and cerebral hypoxic injuries.

(c) Water and electrolyte balance disorder: In cases of pneumonia, fever and tachypnea increase the loss of water and electrolytes from the respiratory tract and the skin, and hypertonic dehydration may occur; if accompanied by vomiting and diarrhea, which can further exacerbate the loss of water from the body, dehydration can become further aggravated.

Potassium metabolism disorders: The following factors often cause elevated serum potassium levels in infants: (1) in cases of acidosis, the H^+ concentration in plasma increases, and H^+ enters into cells, through the H^+/K^+ exchange, causing K^+ to be released from cells and inducing intercellular fluid; and (2) in cases of infection, cell injury, glycogen, and protein decomposition induce K^+ release, leading to the elevation of serum potassium levels. However, the total in vivo potassium decreases in infants for the following reasons: (a) renal compensation causes an increase in K^+ excretion; in cases of dehydration, reflexivity increases the secretion of aldosterone, causing an increase in the renal excretion of K^+; (b) inadequate K^+ intake; (c) loss due to vomiting and diarrhea; and (d) the neonatal ability to secrete antidiuretic hormones is immature and renal K^+ retention is relatively weak.

In cases of metabolic acidosis, the plasma HCO_3^- content decreases; in addition to lactic acidosis, the plasma Cl^- level also increases with the occurrence of hyperchloremic acidosis. In cases of respiratory acidosis, the blood $H_2CO_3^-$ concentration significantly increases and the renal compensatory reabsorption of $H_2CO_3^-$ and the discharge of Cl^- increase, causing hypochloremic acidosis.

(d) Cerebral edema and toxic encephalopathy: Cerebral edema is relatively common in patients with ventilatory disorders. Because CO_2 retention and acidosis induce cephalemia and telangiectasia, blood capillaries become dilated, the vascular permeability increases with exoserosis, and local microcirculatory disturbances occur in the brain to produce intercellular edema (angioedema). Hypoxia, acidosis, and low blood sugar induce energy metabolism disorders, decreasing the activity of the Na^+/K^+/ATPase, causing Na^+ and H_2O to enter the cells but making them difficult to be discharged from the cells, and resulting in edema. In addition, electrolyte imbalances and sodium retention further exacerbate cerebral edema. Severe cerebral edema can induce an increase in intracranial pressure, often accompanied by convulsions and even leading to brain herniation.

(e) Toxic enteroparalysis: Severe pneumonia increases catecholamine secretion; redistributes blood flow; induces gastrointestinal tract microcirculation disorders, intestinal ischemia, passive hyperemia of the lungs, hypoxia, edema, and exudation; reduces intestinal motility and smooth muscle relaxation; and induces intestinal paralysis and aeration. Hypokalemia can cause enteroparalysis. Serious intestinal gas causes the diaphragmatic muscles to increase, limiting lung ventilation and further aggravating infant dyspnea.

(f) Renal impairment: Due to the total redistribution of blood flow, renal blood flow is also reduced; a reduced glomerular filtration rate may cause sodium retention. Long-term ischemia and hypoxia can lead to tubular necrosis and renal failure.

(g) Pulmonary hemorrhage: In cases of acidosis, blood viscosity increases, causing slowed blood flow, and late infectious pneumonia is often complicated by polycythemia and a reduced tendency toward deformation, which causes embolization; shock also can cause blood stasis and thrombosis. The deposition of IgG, C3, and antigen-antibody complexes on capillary basement membranes can induce pulmonary vascular injury and blood leakage; congestive cardiac failure increases pulmonary vascular pressure and resistance, inducing pulmonary edema, and the occurrence of pulmonary hemorrhage can be promoted.

(h) Hypoglycemia: In cases of serious infections, glucose consumption triples, and the activity of the enzyme that promotes gluconeogenesis in the neonate liver is relatively low, so that it is not difficult to transform amino acids into glucose. In cases of aggravated infections, the brown fat becomes exhausted, and gluconeogenesis is reduced so that hypoglycemia is apt to occur. In addition, fever and increased respiratory movement both increase sugar consumption, and low food intake, vomiting, and gastrointestinal dysfunction decrease the intake of exogenous sources of energy, which can cause or aggravate the occurrence of hypoglycemia.

(i) Circulatory disorder and shock: In cases of severe pneumonia, anoxia, toxins, acidosis, heart failure, stress, and other reasons can cause microcirculation disorders, resulting in small blood vascular spasms and reduced blood supply, and the opening of capillaries causes microcirculatory blood flow stasis, inducing shock, disseminated intravascular coagulation, and organ necrosis.

(j) Disseminated intravascular coagulation (DIC): The effects of hypoxia and endotoxins induce vascular endothelial injuries, acidosis induces vasodilation and slow blood flow, and shock induces microcirculation stasis and plasma exosmosis, causing and aggravating ischemic hypoxia, anoxia, and acidosis and initiating the endogenous coagulation pathway that leads to DIC. In addition, during the neonatal period, the plasma clotting factor content is low, the content of hemoglobin is high, and blood viscosity is high, and the ability of the liver to synthesize clotting factors is low, making DIC apt to occur to neonates. The combined effects of the above factors increase the likelihood of neonates to experience DIC in cases of serious pneumonia.

6.1.5 Auxiliary Examination

1. Conventional examinations: Routine blood test, CRP, and blood culture.
2. Chest X-ray examination: (1) For pneumonia induced by intrauterine and delivery processes, no abnormal findings can be found during X-ray examination on the first day after birth, and the following changes can occur 24 h thereafter: changes of interstitial pneumonia; the lungs become full of small, patchy or linear, blurred shadows, which become a diffuse fan shape around the pulmonary portae; the bronchial walls become thickened; and particle-shaped shadows are observed, accompanied by air bronchogram and emphysema. (2) Pneumonia after birth often has the following manifestations: a wide range of spot-shaped and patch infiltration shadows in the lung field, likely accompanied by emphysema and atelectasis; occasional large lobar consolidation with empyema, pyopneumothorax, lung abscess, and bunamiodyl; diffuse blurred shadows in both lungs, varying in shadow density and depth, with bacterial pneumonia being common; and cord-shaped shadows in two pulmonary por-

tae and interstitia of lung fields, likely accompanied by scattered pulmonary infiltration and obvious emphysema, with virus pneumonia being common.

6.1.6 Treatment

1. Strengthen nursing, maintain body temperature and maintain a neutral ambient temperature, and prevent disease progression and deterioration.
2. Strengthen respiratory tract management: Receive physical therapies, such as aerosol inhalation, postural drainage, and regular turning-over and back-tapping, and maintain smooth airway.
3. Oxygen supply: Maintain the arterial blood PaO_2 concentration at 50–80 mmHg. Hoods can be used for oxygen supply; in severe cases, CPAP and even breathing machines may be used for treatment. Attention should be paid to the following problems when using oxygen therapy: (1) If the diseases are accompanied by severe hypoxemia induced by CO_2 retention, high concentrations of oxygen should be given; because CO_2 paralysis cannot excite the respiratory center, suppression of peripheral chemoreceptors cannot stimulate the respiratory center and can inhibit spontaneous respiration. (2) Measures should be adopted to gradually reduce oxygen concentration after hypoxemia symptoms have improved following oxygen therapy; otherwise, the oxygen concentration will plunge, inducing sudden pulmonary spasms and leading to pulmonary hypertension and right to left shunt, which is difficult to reverse.
4. Pathogenetic therapy: Bacterial pneumonia should be treated intravenously with antibiotics; however, it is often difficult to quickly determine the responsible pathogen. Cephalosporins can be administered, and the categories of antibiotics used can be adjusted according to the patient's conditions. Penicillin can be given to treat group B β-hemolytic streptococcus, ampicillin can be given to treat *Listeria* pneumonia, erythromycin can be given to treat chlamydia and mycoplasma, acyclovir can be used to treat herpes simplex virus, ganciclovir can be used to treat cytomegalovirus pneumonia, and 200,000–1,000,000 U/day of intramuscularly injected α1-interference is effective against viral pneumonia, with the treatment courses ranging from 5 to 7 days.
5. Support therapy: Circulation disorder and water-electrolyte disorder should be corrected, with a daily total liquid volume of 60–80 ml/kg and a slow infusion speed to avoid pulmonary edema and heart failure and ensure adequate calories. Albumin and immunoglobulin infusion can be given as needed to improve the body immune function.

6.2 Ultrasound Diagnosis of Infectious Pneumonia

Lung ultrasound has been increasingly used for the diagnosis and differential diagnosis of severe lung disease in the field of critical care medicine, and it is rarely used in the diagnosis of neonatal pneumonia, which is not domestically reported. Based on the experience of using ultrasound for the diagnosis of infectious pneumonia in infants and adults, we investigated the value of applying ultrasound to the diagnosis of infectious pneumonia in neonatal lungs (the study protocol was approved by the institutional review board of the Beijing Military General Hospital and Beijing Chaoyang District Maternal and Child Health Care Hospital), and the results confirmed that using real-time ultrasound for the diagnosis of neonatal infectious pneumonia was accurate and reliable, with a relatively high sensitivity and specificity. The primary ultrasound imaging features of neonate infectious pneumonia include a massive area of pulmonary consolidation with air bronchogram, abnormalities of pleural lines, AIS or pulmonary edema, and the disappearance of A-lines [4–7].

Lung consolidation with air bronchogram is the primary basis for using ultrasound to diagnose pneumonia, which is characterized by [5–8]:

1. Hypoechoic areas with varying size and shape (irregular consolidation area), and usually a

larger consolidation range, with irregular borders and a jagged shape.
2. Consolidation with irregular jagged edges with air bronchogram. The consolidation can be located in any part of the lung field.
3. In cases of serious and large-scale consolidation, using real-time ultrasound, the air movement in the bronchi can be observed, which is known as dynamic air bronchogram.
4. Color Doppler ultrasound can indicate the pulmonary blood flow signals in the consolidation area. Color or energy Doppler can indicate the pulmonary blood flow signals in the consolidation area, showing that blood supply is present in the consolidated lung tissues, which is necessary for pulmonary lesions to recover or heal.
5. Pleural effusion may be present to varying degrees. It can be differentiated with RDS and MAS, according to the above features.

Neonatal infectious pneumonia is common, and nonspecific ultrasound changes include abnormalities in pleural line ultrasound abnormalities, the disappearance of the A-line and AIS. Abnormal pleural lines and AIS are related to inflammatory exudation, and, in severe cases, a small amount of pleural effusion can be noted. Because A-line is the reflection line of the pleural line, abnormalities of pleural line must be accompanied by abnormal A-lines, which is primarily manifested as the disappearance of the A-line. The above symptoms are also observed in other lung diseases, such as RDS, MAS, and TTN, and are not specific to IPN [8].

The disappearance of lung sliding and lung pulse under real-time ultrasound are important imaging features of neonatal infectious pneumonia, and we observed that 84.4% of newborns with infectious pneumonia showed varying degrees of lung pulse and the disappearance of lung side when examined using real-time ultrasound. Lung pulse is also found in severe atelectasis, suggesting that the existence and extent of lung pulse are associated with the formation and extent of pulmonary consolidation and/or atelectasis, and it is considered to be one of the characteristic symptoms of atelectasis.

Lung consolidation is the necessary ultrasonographic observation for the diagnosis of pulmonary atelectasis and RDS. However, in the case of atelectasis, the edges of pulmonary consolidation areas are relatively clear and regular, and usually one site of consolidation is observed in one lateral field and often in an air bronchogram that is arranged in parallel; in pneumonia, multiple consolidations can be observed, and a dynamic air bronchogram is common. The consolidation of RDS begins from the subpleural regions and is associated with the range and extent of the lesion, and the air bronchogram in consolidation areas presents a relatively fine, short, linear shape. Of course, a truly accurate identification not only requires adequate knowledge and experience using a lung ultrasound but also the combination of the medical history and clinical manifestations, which is the advantage of a neonatal physician performing the ultrasound examination.

Please see the related pictures and movie in Figs. 6.1–6.16 and Video 6.1.

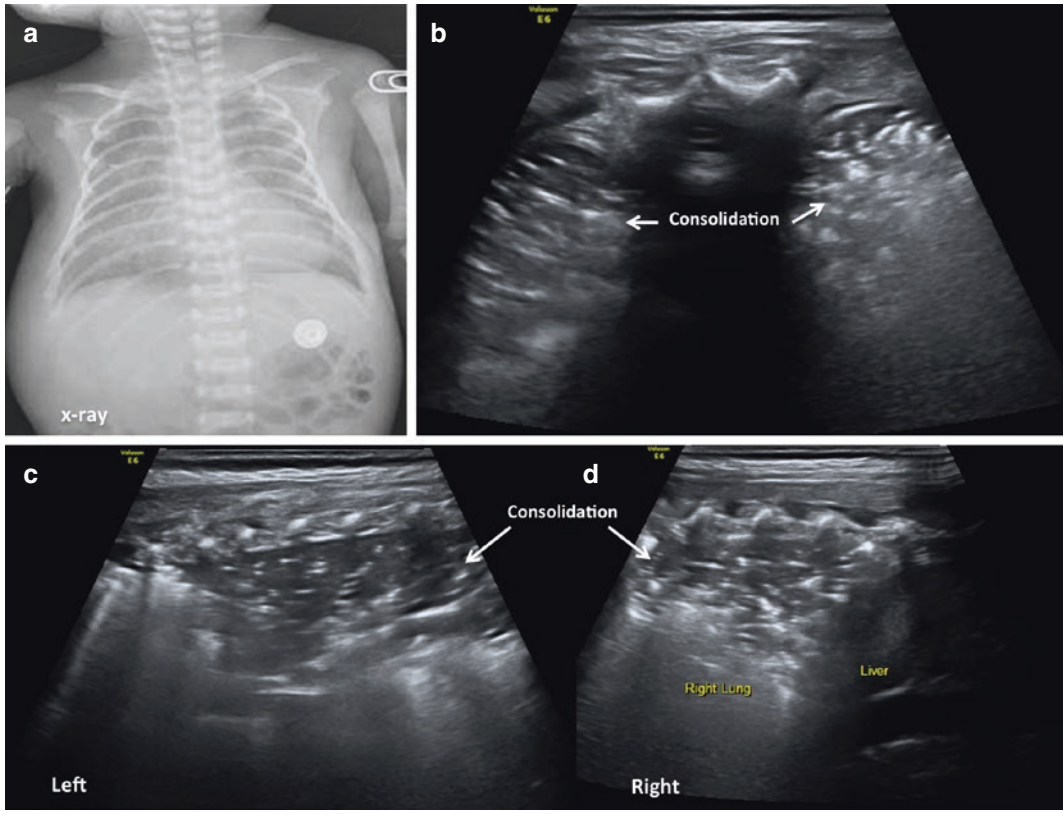


Fig. 6.1 Ultrasound manifestations of infectious pneumonia. Gestational age of 38^{+2} weeks, delivered by cesarean section, with a birth weight of 3420 g. The patient was admitted due to dyspnea 3 days after birth. Temperature was 38 °C, and dense moist rales were heard in both lungs. Routine blood test results: WBC 22 × 10^9/L, N% 78%, MONO% 12.2%, and CRP 66.8 mg/L. Chest X-ray (**a**) shows significant pneumonia in bilateral lungs. Lung ultrasound shows large-scale consolidation areas with irregular margins and air bronchogram; part of the pleural line disappears, and the remaining parts present changes consistent with alveolar interstitial syndrome, with the disappearance of A-lines (**c**, **d**). The infant is lying in the prone position and is scanned through the transverse section of the spinal column, and the results further display the presence of consolidation in both lungs (**b**)

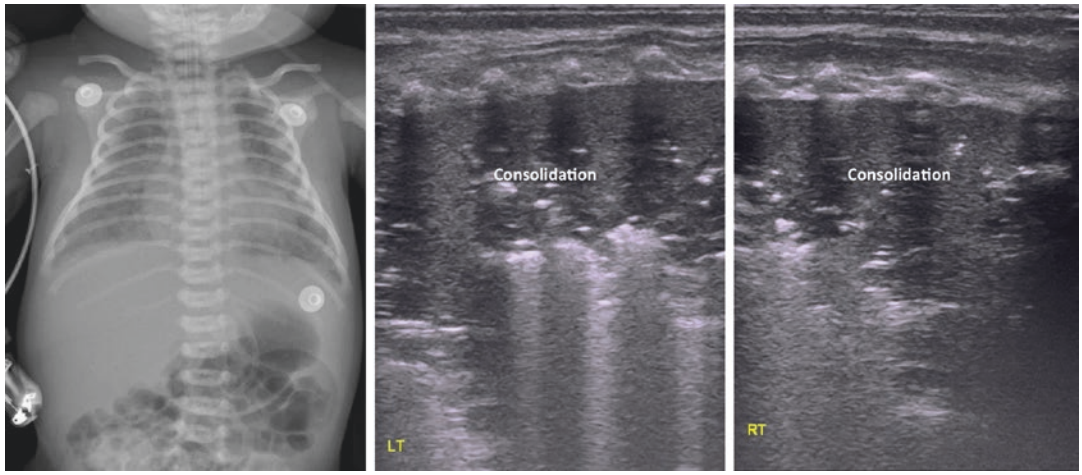


Fig. 6.2 Ultrasound manifestations of infectious pneumonia. Gestational age of 37 weeks, delivered by cesarean section, with a birth weight of 3700 g. No asphyxia, history of PROM, dyspnea after birth, or fever. Routine blood test results: WBC 30 × 10^9/L, N% 81% mononuclear PLT 67 × 10^9/L, CRP 39 mg/L. Lung ultrasound reveals a large area of consolidation with irregular edges, accompanied by air bronchogram in bilateral lung fields; the pleural line is present; the A-line disappears. The chest X-ray indicates neonatal pneumonia with obvious manifestations in both sides of the lungs

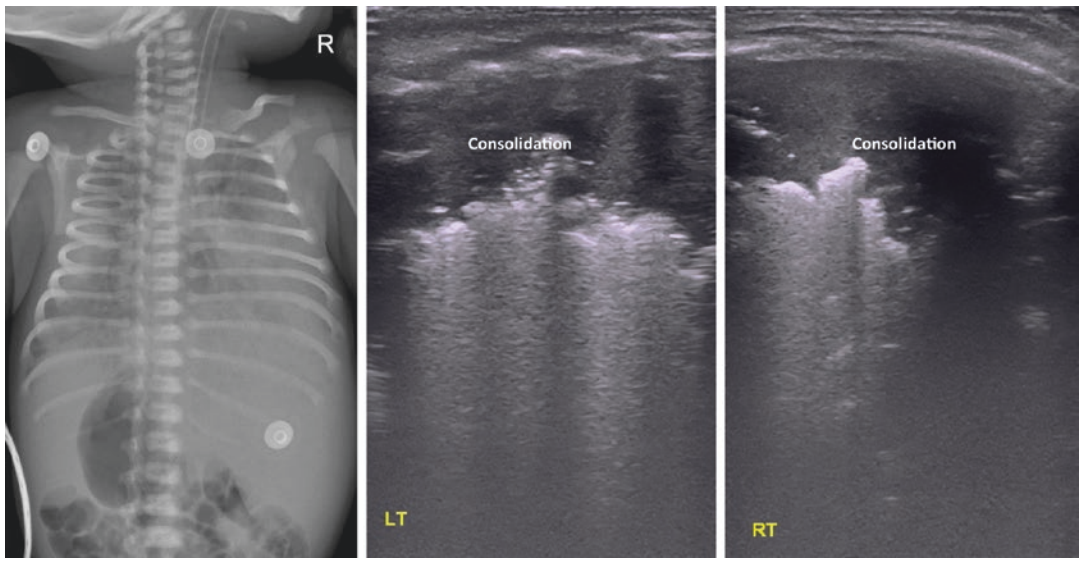


Fig. 6.3 Ultrasound manifestations of infectious pneumonia. Gestational age of 38 weeks and a birth weight of 4200 g. The patient was admitted for 3 days due to dyspnea with fever, and dense moist rales were heard in the lungs. In combination with a routine blood test and chest X-ray examinations, the diagnosis was severe pneumonia in both sides of the lungs. Lung ultrasound reveals a large area of consolidation with irregular edges and uneven internal echoes in the bilateral lung fields, accompanied by air bronchogram (left lung, the probe is perpendicular to the ribs; right lung, the probe is parallel to the ribs)

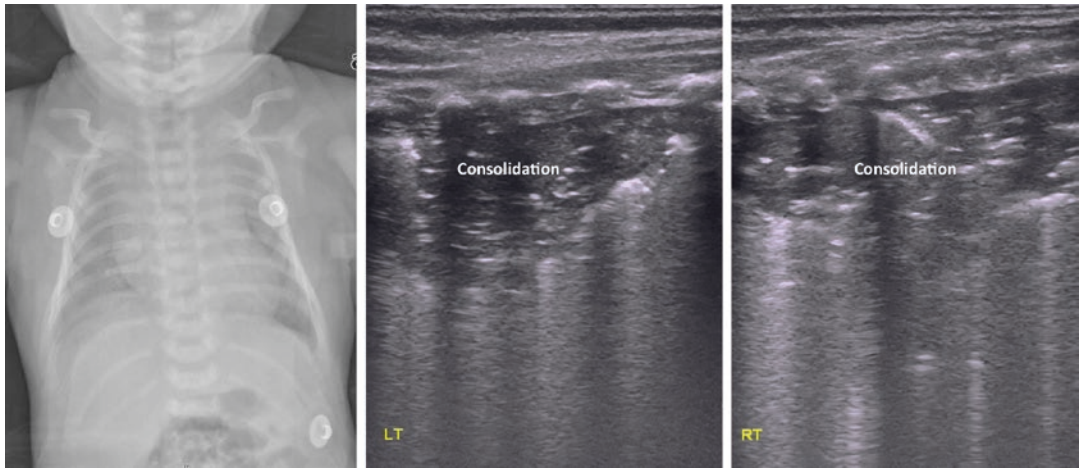


Fig. 6.4 Ultrasound manifestations of infectious pneumonia. Gestational age of 35^{+1} weeks, spontaneous labor, and a birth weight of 1850 g. PROM occurred 49 h prior to the examination, and the amniotic fluid was turbid. Not long after birth, the patient experienced dyspnea with a low temperature and received respiratory treatment. Routine blood test results: WBC 2980 × 10^9/L, N87%, PLT 87 × 10^9/L, and blood culture indicated the growth of *Klebsiella pneumoniae*. Chest X-ray shows a shadow with increased density in the right lung, with obvious manifestations at the right lower lung. Lung ultrasound reveals large areas of consolidation in the bilateral lung fields, accompanied by significant air bronchogram; the pleural line partially disappears, and the A-line disappears

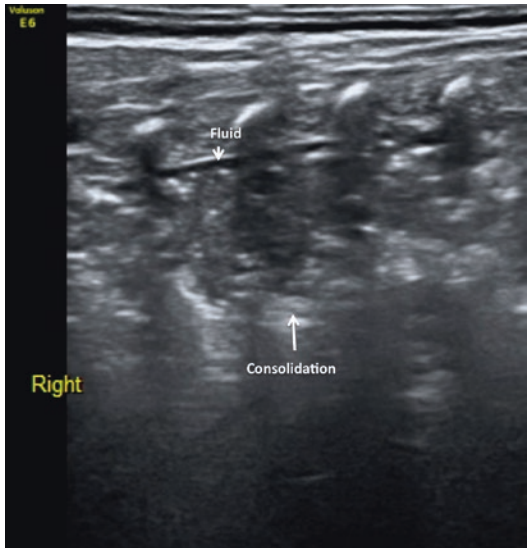


Fig. 6.5 Ultrasound manifestations of infectious pneumonia. Gestational age of 37 weeks and a birth weight of 2150 g. The patient was admitted due to fever and breathing difficulties 2 days after birth, and lung auscultation indicated dense rales. A significant elevation was observed in the WBC, N%, and CRP, as indicated in the routine blood test results, and the chest X-ray is consistent with the changes observed in pneumonia. Lung ultrasound shows large consolidation shadows with irregular, jagged edges, accompanied by air bronchograms; the A-line disappears, some of the pleural line disappears, and a very small amount of pleural effusion was observed

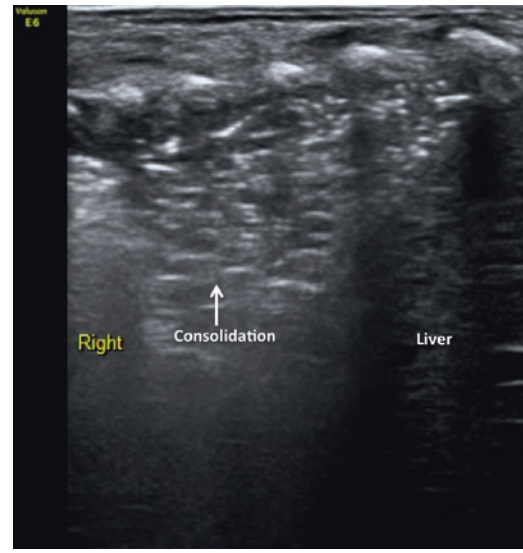


Fig. 6.6 Ultrasound manifestations of infectious pneumonia. This was a premature newborn infant with a gestational age of 30^{+1} weeks, and chest X-ray shows right lower lung pneumonia. The ultrasound shows a large area of consolidation, with significant air bronchograms, in the right lower lung field

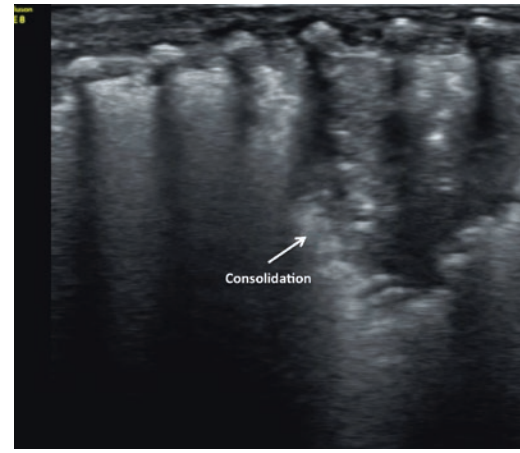
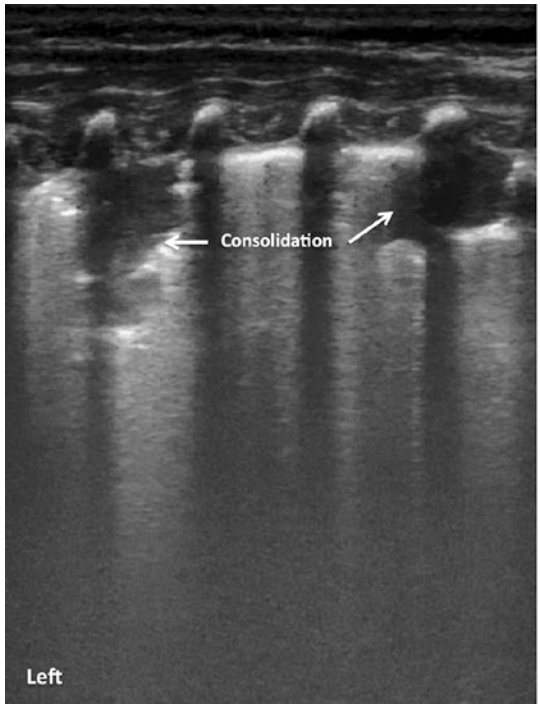


Fig. 6.7 Ultrasound manifestations of infectious pneumonia. Gestational age of 33^{+4} weeks and a birth weight of 1650 g. The patient was admitted for 3 days due to dyspnea with fever and received ventilator treatment. Chest X-ray examination shows double markings that have become thickened, blurred, and disorderly, and visible chunks of dense shadow are observed in right lower lung. In combination with the clinical manifestations, the diagnosis was pneumonia. Lung ultrasound shows three intercostal consolidation shadows in the right lung field, and the margins of the consolidation areas in the deep area of the lung fields present jagged edges, accompanied by air bronchogram; the remaining parts present significant alveolar-interstitial syndrome, with disappearing pleural and A-lines



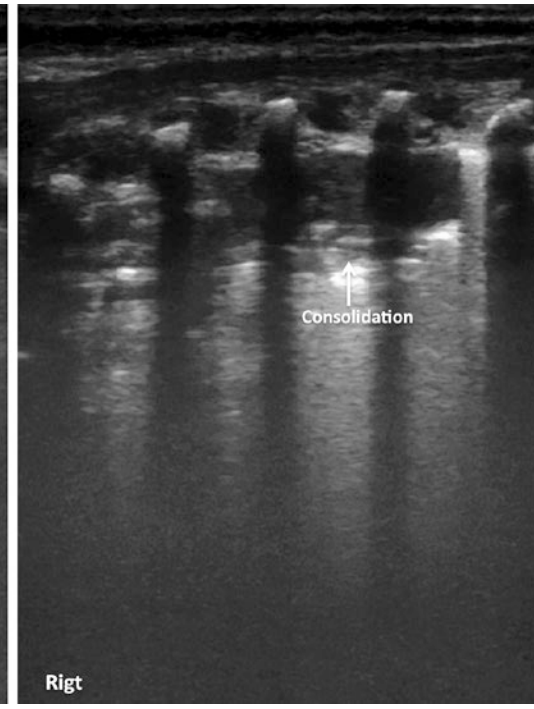


Fig. 6.8 Different areas of lung consolidation in pneumonia. Ultrasound examination shows the presence of different areas of lung consolidation in different regions of lung fields accompanied by an air bronchogram. In combination with clinical manifestations, laboratory tests, and a chest X-ray, the diagnosis is infectious pneumonia. The presence of different degrees and different ranges of consolidation in the lung fields is one of the important ultrasound manifestations of neonate infectious pneumonia, and the heterogeneity of intrapulmonary lesions of pneumonia is reflected

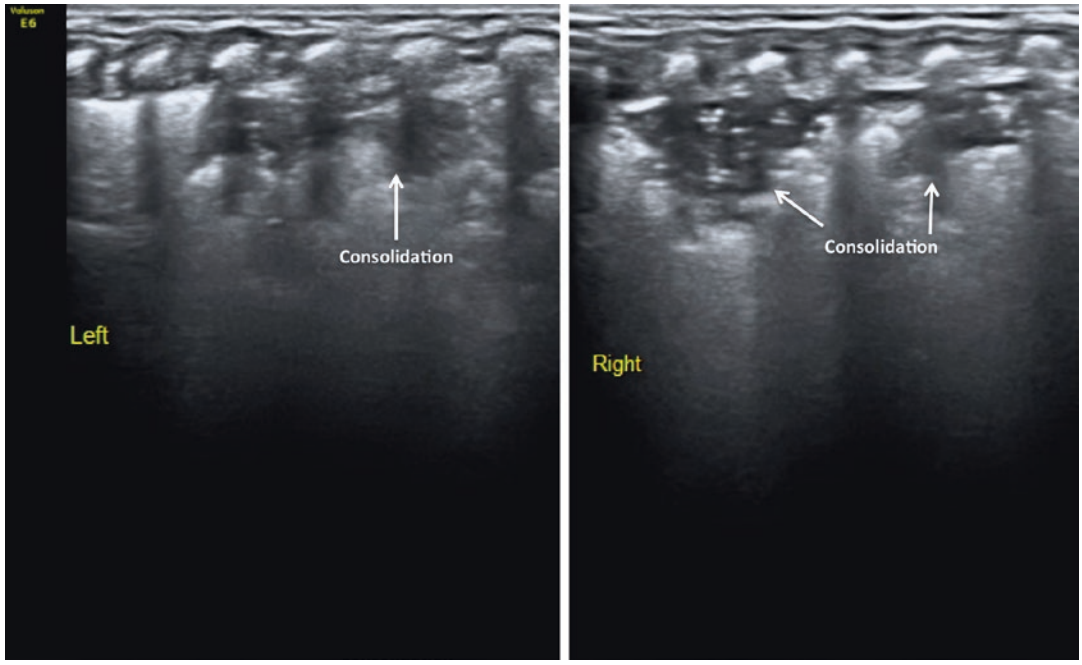


Fig. 6.9 Different areas of lung consolidation in pneumonia. Gestational age of 38^{+5} weeks, delivered by cesarean section, with no asphyxia and a birth weight of 3935 g. The patient experienced dyspnea soon after birth, the body temperature was 39 °C, and a large quantity of moist rales could be heard in the lungs. Routine blood test results: WBC 22×10^9/L, N% 89.1%, CRP 31 mg/L. Lung ultrasound reveals a large area of consolidation with irregular edges in the left lung, and different consolidation areas with different sizes and shapes are observed, all accompanied by air bronchogram

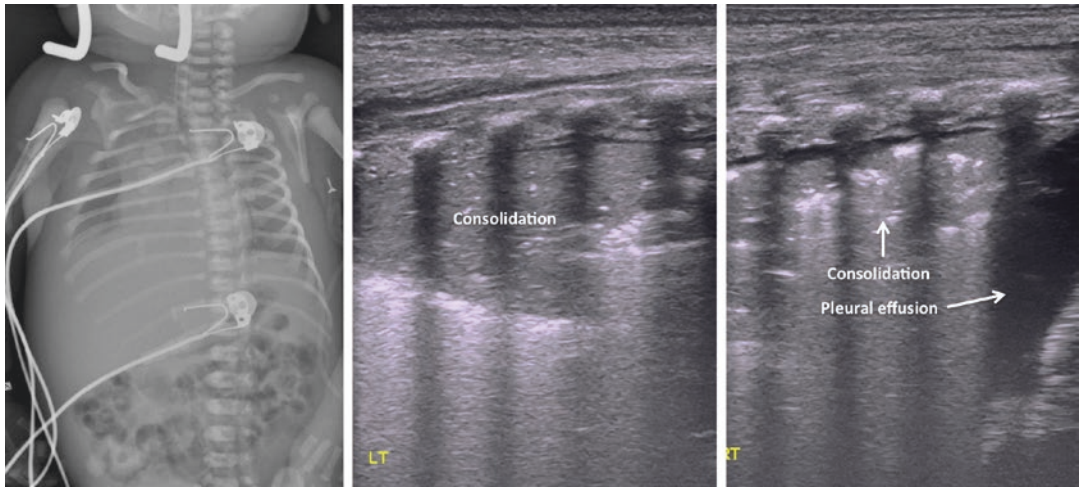


Fig. 6.10 Pleural effusion in pneumonia patient. Gestational age of 38^{+3} weeks and a birth weight of 3270 g. The patient was admitted to the hospital 5 h after birth because of pneumonia, which was diagnosed according to clinical manifestation and chest X-ray findings. Lung ultrasound showed large areas of consolidation with significant air bronchograms in bilateral lung fields, as well as pleural effusion, which is more significant in the right lung

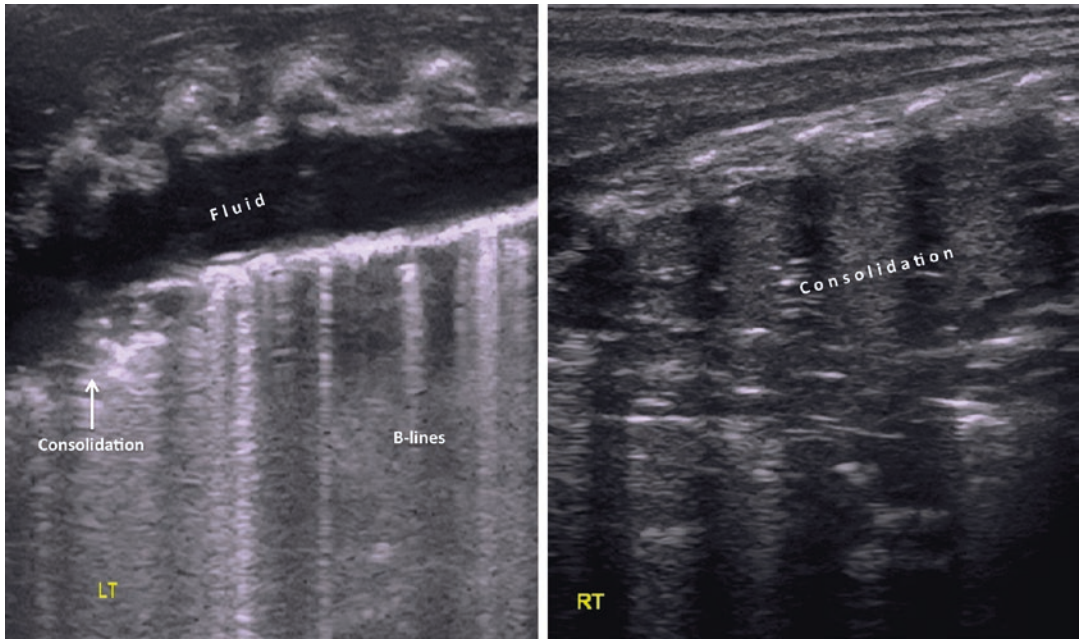


Fig. 6.11 Different ultrasonic findings for different sides of the lungs in a pneumonia patient. Gestational age of 33^{+4} weeks and a birth weight of 1650 g. The patient was admitted for 3 days due to dyspnea with fever and received ventilator treatment. In combination with the clinical manifestations, the diagnosis was pneumonia. Lung ultrasound shows different findings for different sides of the lungs; the left lung primarily manifested as a pleural effusion with a small area of consolidation, with B-lines and disappearing A-lines, and the right lung primarily presented with a large area of consolidation with air bronchograms

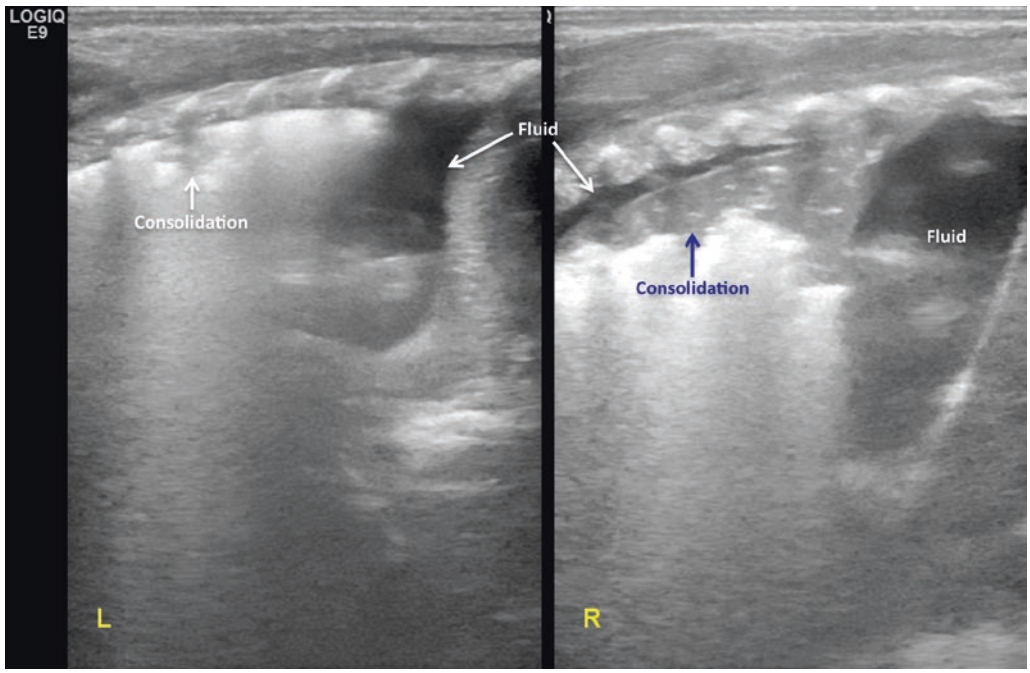


Fig. 6.12 Ultrasound manifestations of infectious pneumonia. This baby was admitted to the NICU because of severe anemia with significant dyspnea. The ultrasound shows significant levels of fluid in the bilateral chest, a large area of consolidation in the right lung and a small consolidation in the left lung, abnormal pleural lines and disappearing A-lines

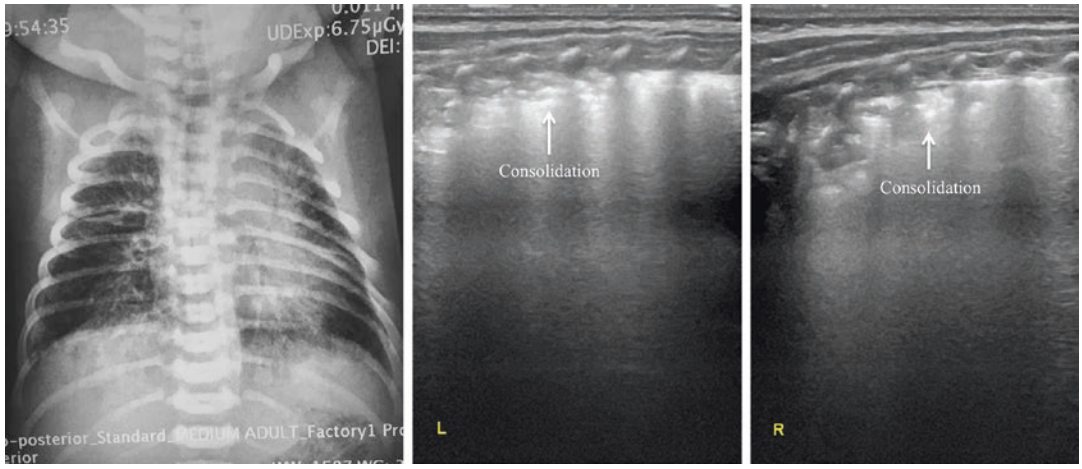


Fig. 6.13 Subpleural consolidation in mild pneumonia. The baby was diagnosed with mild pneumonia, according to the clinical information and chest X-ray manifestation. Ultrasound shows a mild subpleural consolidation in the left lung field and a relatively significant consolidation in the right lung field

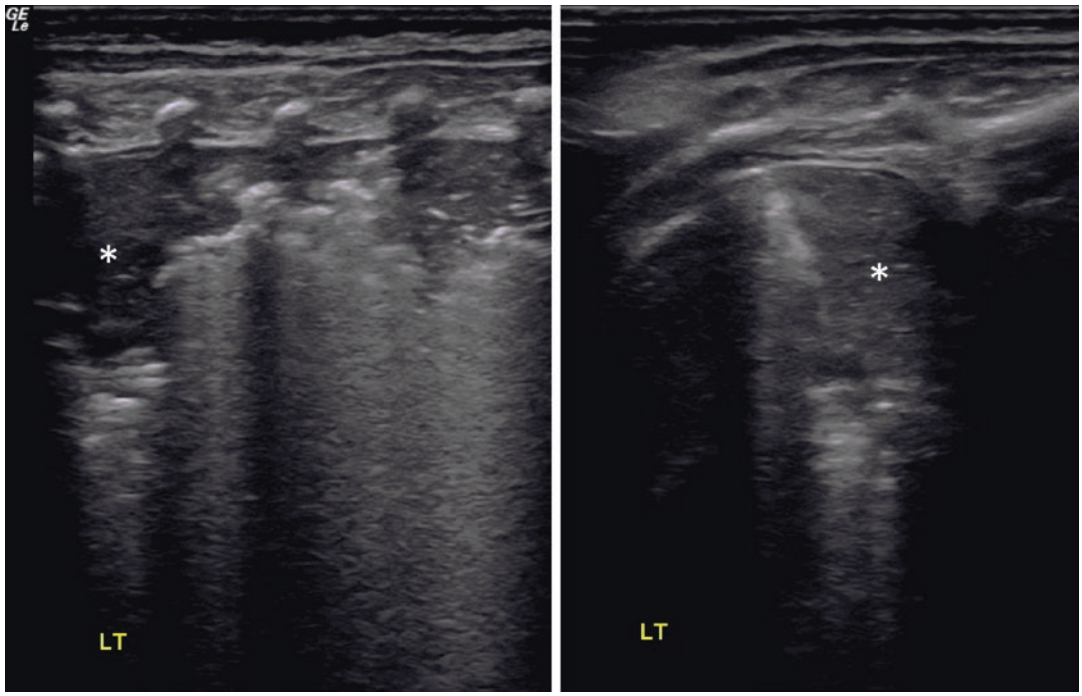


Fig. 6.14 Ventilator-associated pneumonia. A neonatal patient with ventilator-associated pneumonia. Lung ultrasound shows a very large area of lung consolidation, with significant air bronchograms and irregular margins in the left lung (left picture, the probe is perpendicular to the ribs; right picture, the probe is parallel to the ribs)

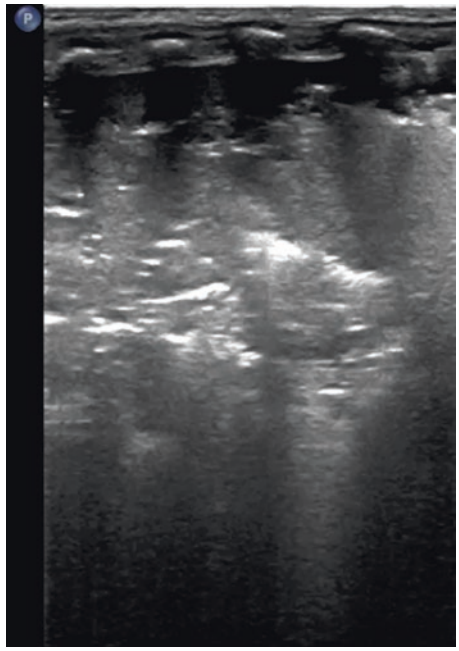


Fig. 6.15 Dynamic air bronchograms in severe pneumonia patients. The ultrasound shows a very large area of consolidation, with significant air bronchograms, in a severe pneumonia patient, and the significant, dynamic air bronchograms can be observed using real-time ultrasound (Video 6.1)

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Fig. 6.16 Severe pneumonia (*volume panorama*). A severe pneumonia patient. Lung ultrasound (*volume panorama mode*) showed large area of consolidation involved in the whole left lung, in fact that, atelectasis has already formed in the area of severe consolidated lung tissues.