HYALINE MEMBRANE DISEASE
RESPIRATORY DISTRESS SYNDROME

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Objectives

1. To understand the risk factors, pathogenesis , pathology, and clinical features of respiratory distress syndrome
2. To list the differential diagnosis of respiratory distress in newborn baby
3. To recognize how to investigate and manage a newborn baby with respiratory distress syndrome
4. To understand the features and management of patent ducutus arteriosus
5. To recognize the pathogenesis and clinical features of bronchopulmonary dysplasia .

Respiratory distress in the newborn is defined by the presence of one or more of the following:

tachypnea, retractions, nasal flaring, grunting, and cyanosis .

INCIDENCE

HMD occurs primarily in premature infants, and its incidence inversely proportional to the gestational age and birth weight.
60-80% of infants less than 28wk of gestation
15-30% of infants between 32&36 wk

in about 5% beyond 37 wk, and rarely at term

The risk of developing RDS increases with

1. Prematurity
2. maternal diabetes
3. Multifetal pregnancy
4. cesarean section delivery
5. precipitous delivery
6. asphyxia
7. cold stress
8. history of previously affected infants.

The incidence is highest in preterm male or white infants .

The risk of RDS is reduced in

1. pregnancies with chronic or pregnancy-associated hypertension
2. maternal heroin use
3. prolonged rupture of membranes
4. antenatal corticosteroid prophylaxis

ETIOLOGY &PATHOPHISOLOGY

Surfactant deficiency [decreased production&secretion] is the primary cause of HMD.

Surfactant Is phospholipid protein ,its major constituents:

1-Dipalmitoyl phostidylcholine [Lecithine]

2-Phosphatidyle glycerol

3-Apoproteins

4-Cholesterol

Surfactants are synthesized and stored in type2 alveolar cells.

Deficiency of surfactant leads to:

-alveolar collapse
-decreased lung volume &compliance
-ventilation-perfusion abnormalities
-right to left shunt
-persistent hypoxemia[<30mm Hg]causes metabolic acidosis
-respiratory acidosis also present because alveolar hypoventilation

Decreased myocardial contractility, decreased cardiac out put&arterial blood pressure
-Perfusion of kidneys,GIT,muscle,&skin is reduced leading to edema & electrolytes disorders.

PATHOLOGY

The lungs appear deep purplish red,&liver like in consistency.

Microscopically:

A number of alveolar ducts, alveoli,& resp. bronchiole are lined with acidophilic homogenous, or granular membrane.

Clinical manifestations

Signs of HMD usually appear within minutes of birth, but may be delayed for several hours in large premature infants.

Early clinical signs of HMD:

1-Tachypnea[>60/min]

2-Expiratory grunting

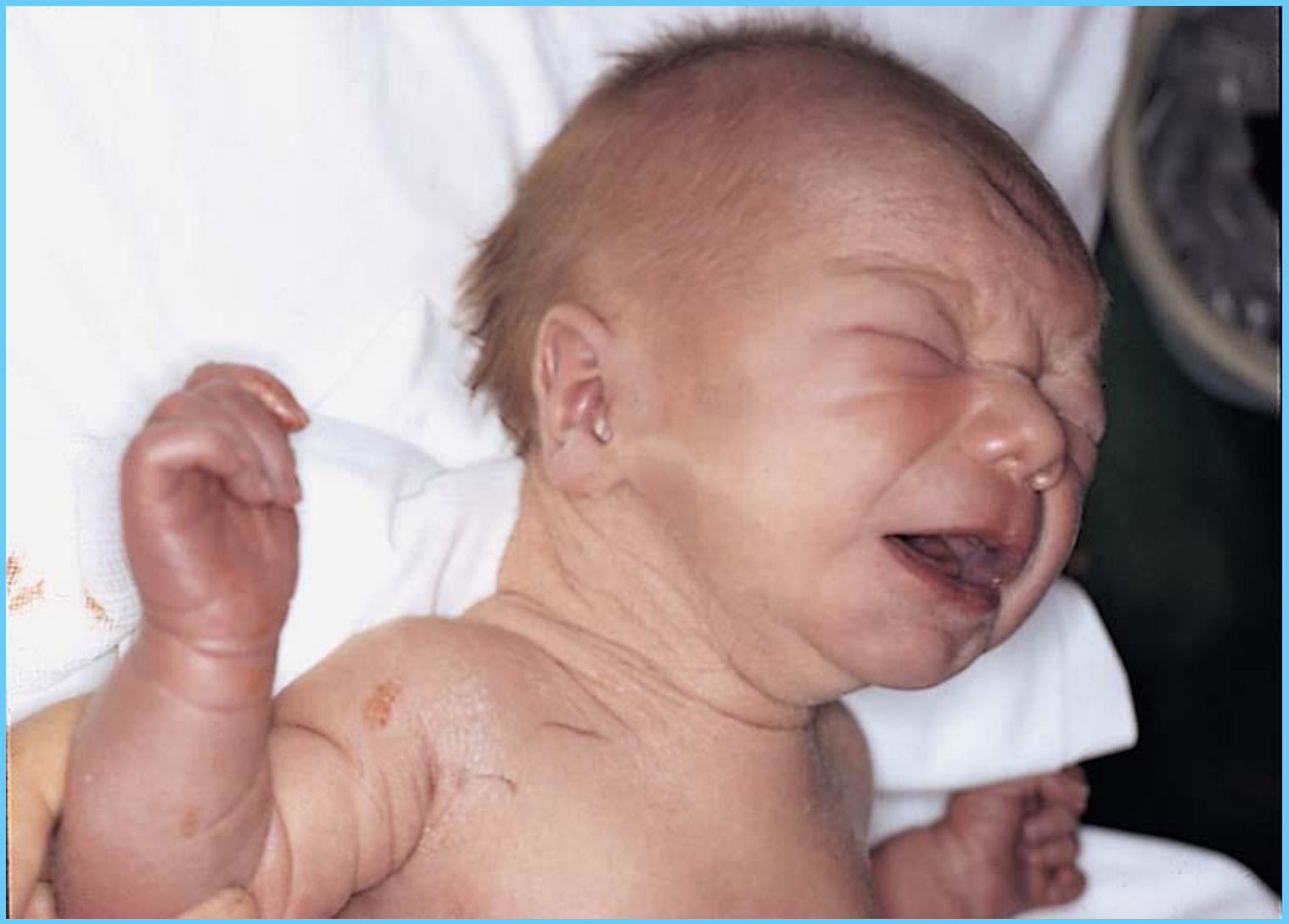
3-Sternal&intercostal recession

4-Cyanosis in room air

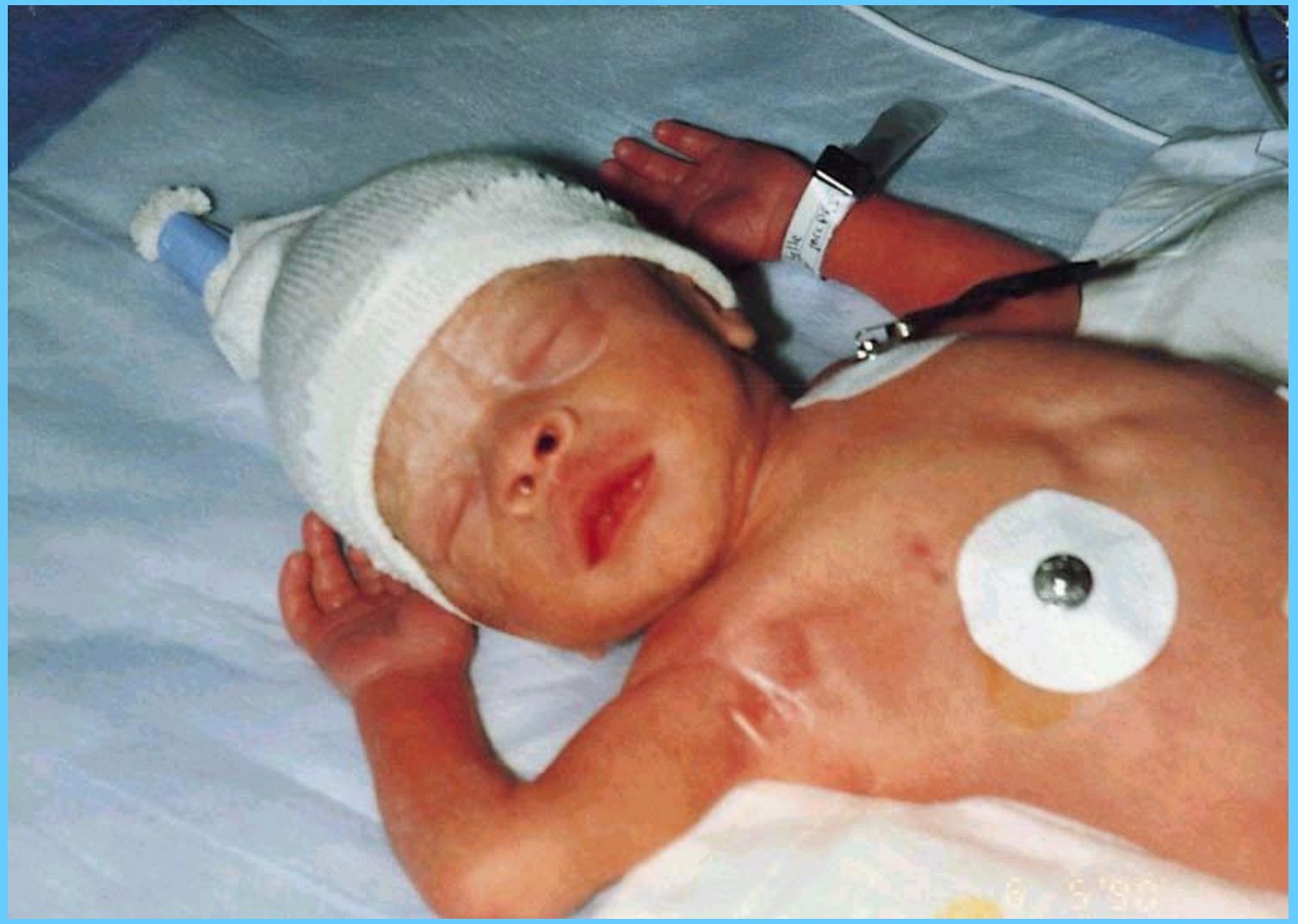
5-Delayed onset of respiration in very immature babies

Late clinical signs in severe HMD

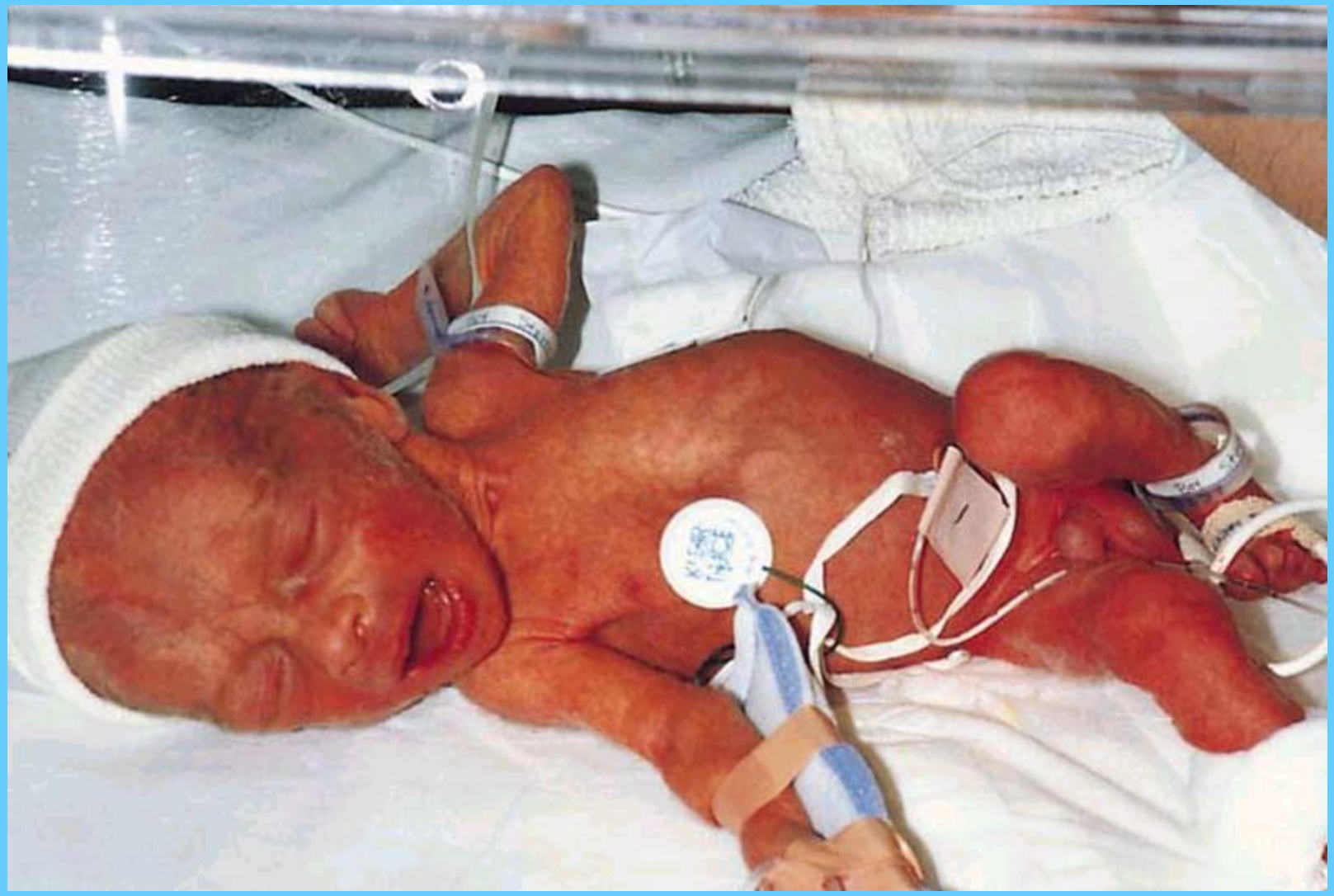
1-Decrease blood pressure
2-Fatigue
3-Cyanosis
4-Pallor increase
5-Grunting decrease or disappears
6-Apnea& irregular respiration[ominous sign]
Other signs:
-Mixed resp.& metabolic acidosis
-Edema,ileus,oliguria
In most cases symptoms&signs reach peak within 3 days after which improvement is gradual.



Cyanosis. This critically ill infant exhibits cyanosis and poor skin perfusio



Flaring. Reflexive widening of the nares may be seen in infants with respiratory distress.



Retractions. The inward collapse of the lower anterior chest wall can be seen in this premature infant with RDS.

INVESTIGATIONS

Chest x.ray

-Grade-1-fine reticular granular mottling, good lung expansion

-Grade-2-mottling with air bronchogram

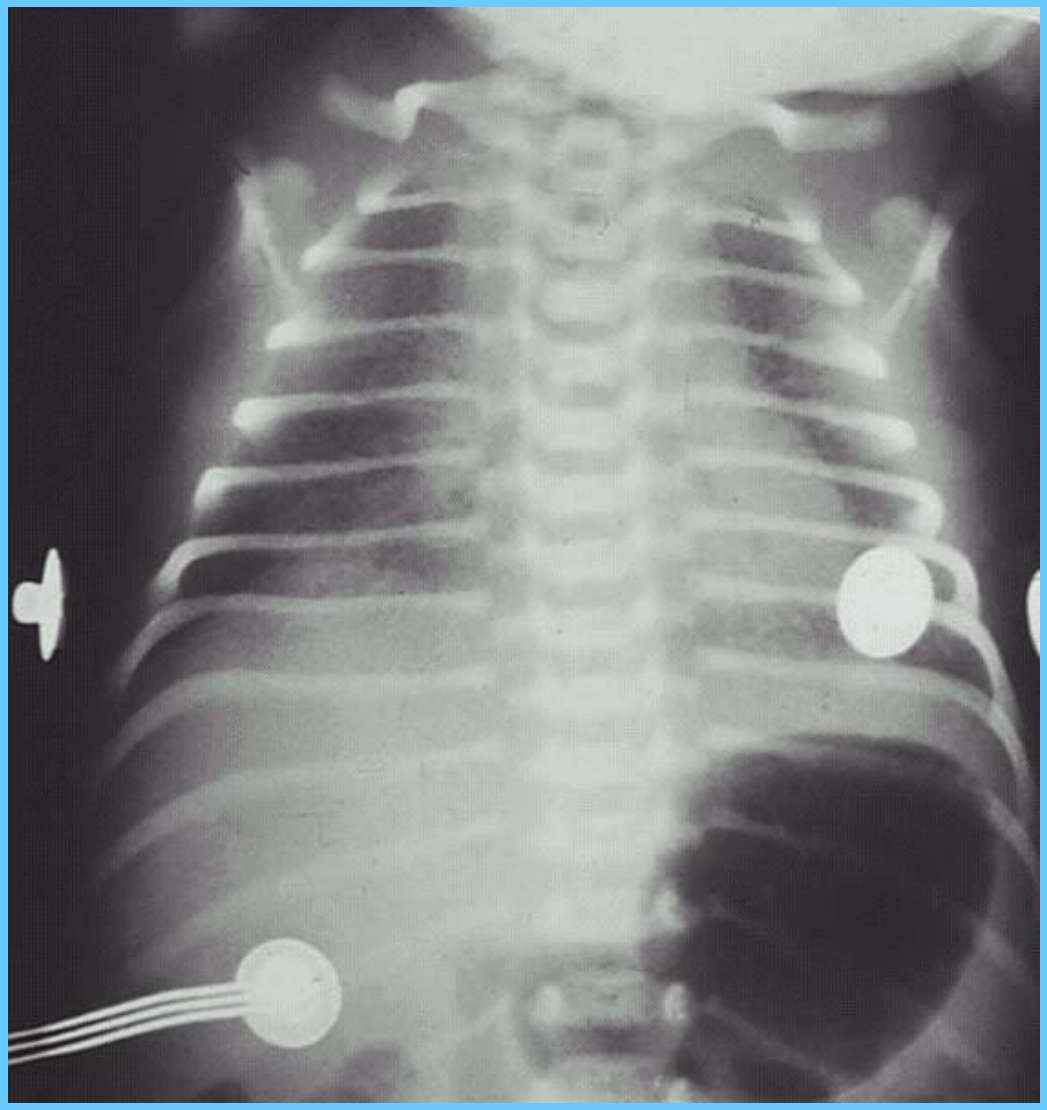
-Grade-3-diffuse mottling,heart border just discernable,prominent air bronchogram

-Grade-4-bilateral confluent opacification of lungs[white out]

BD gas analysis

1-Initially hypoxemia

2-Later progressive hypoxemia ,hypercapnia, &metabolic acidosis



RDS. Note the ground-glass appearance and the presence of air bronchograms

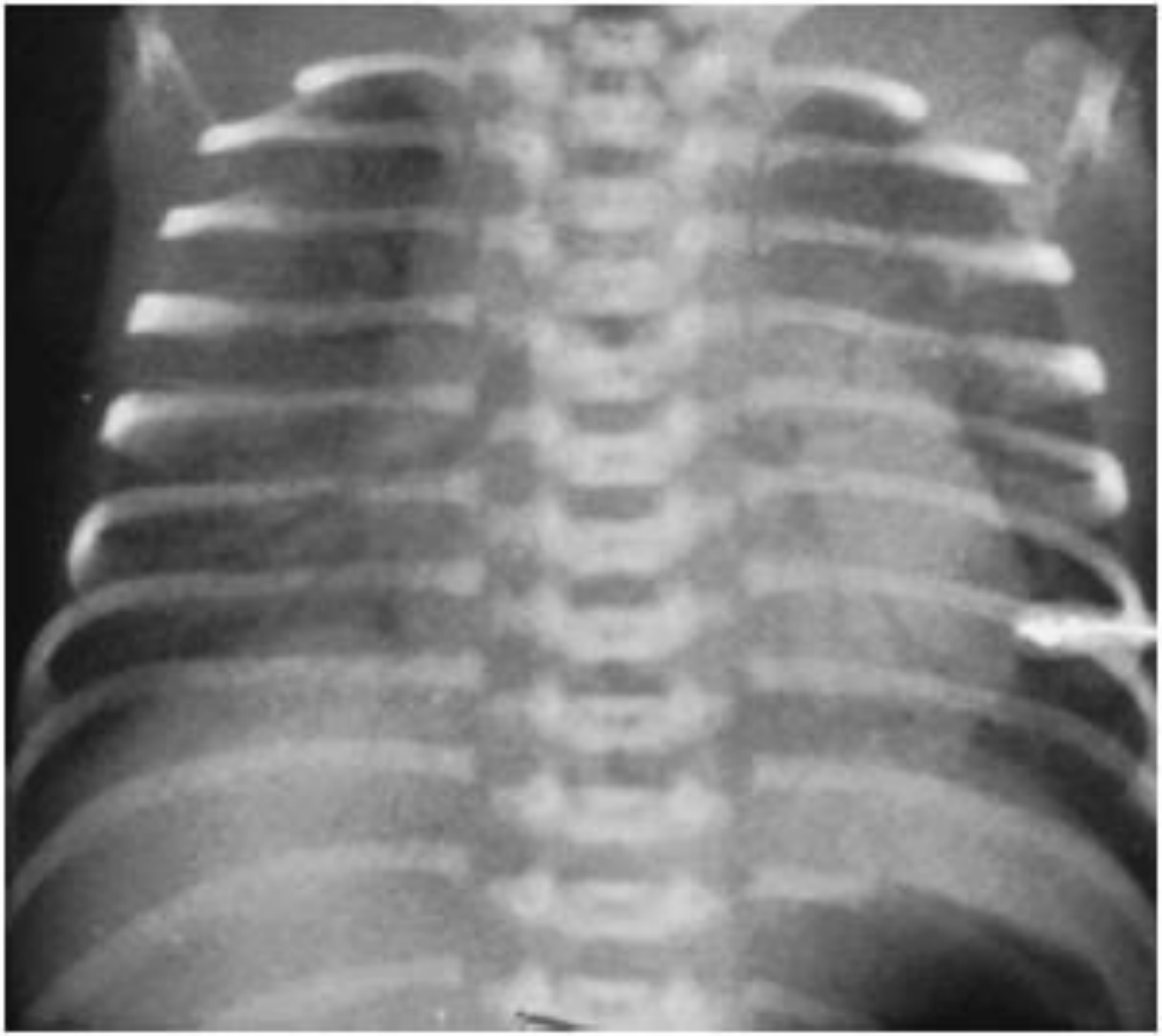


FIGURE 47–6. Typical chest radiograph of an infant with hyaline membrane disease.

Differential diagnosis of RDS

1-congenital pneumonia
2-aspiration pneumonia
3-meconium aspiration syndrome
4-air leak [pneumothorax, pulmonary interstitial emphesema, pneumomediastinum]
5-transient tachypnea of newborn
6-lobar emphesema
7-pulmonary hypoplasia

8-diaphragmatic hernia
9-heart failure
10-persistent pulmonary hypertension
11-asphyxia&increased intracranial pressure
12-metabolic acidosis
13-congenital neuromuscular disorder
14-anemia&hypovolemia

Initial Laboratory Evaluation of Respiratory Distress

1. Chest radiograph
2. Arterial blood gas
3. Complete blood count
4. Blood culture
5. Blood glucose
6. Echocardiogram, ECG

Prevention

1-Prevention of prematurity, including :

-avoidance of unnecessary or poorly timed c.s

-appropriate management of high risk pregnancy& labour.

-Estimation of fetal head circumferance by ultrasound& determination of lecithin concentration in the amniotic fluid by [L/S]ratio decrease likehood of delivering premature infants

2-Adminstration of betamethasone to women 48hr before delivery of fetuses between 24-34wk of gestation significantly reduce the incidence&mortality&morbidity of HMD.One course of corticosteroid required.
3-Adminstration of first dose of surfactant in to the trachea of symptomatic premature infants immediately after birth[prophylactic] reduce air leak&mortality from HMD

Treatment

Maintenance of temperature:

Preterm infants should be nursed in incubator or under radiant heat warmer [maintain core temp 36.5-37° c].

Calories &fluid:

Provided by intravenous fluid.

Excessive fluid contribute to development of PDA,NEC&BPD.

Maintenance of normoxemia:

The aim is to keep arterial oxygen tension in range of[55-75mm Hg].

For babies with spontaneous respiration humidified oxygen should be given.

Too little oxygen will cause hypoxemia, metabolic acidosis,&tissue damage.

Too much oxygen associated with development of retinopathy of prematurity.

Assisted ventilation

1-CPAP: is distending pressure which prevent alveolar collapse during expiration& thus improving oxygenation.

2-Mechanical ventilation:

indication for I.P.P.V

1-failure to establish respiration at birth

2-intractable apneic attacks

3-respiratory failure [ph<7.2,paco2>66mm Hg ,pao2<53mm Hg in 90%O2

3-High frequency ventilation

Surfactant therapy:

Synthetic &natural surfactants[from calf,pig,&cow lungs].Multidose endotracheal instillation of surfactant

Metabolic acidosis:

in RDS may be a result from perinatal asphyxia &hypotension.

The aim to keep pH above 7.25.

It is treated by sodium bicarbonate 1-2meq/kg administered over 15-20min through peripheral or umbilical vein.

Complications of HMD

1-Patent ductus arteroisus

2-Interventricular hemorrhage

3-pulmonary:

A-air leak:

pneumothorax,pneumomediastinum, P.I.E,pneumopericardium,pneumoperitonium,air embolism, subcutanous emphesema.

B-bronchopulmonary dysplasia.

C-pneumonia: aspiration,bacterial.

4-Complication of mechanical ventilation.

5-Long term neurological sequele.

Patent Ductus Arteriosus

The ductus arteriosus constrict after birth in normal term infants in response to elevated PaO2 level.

The ductus in preterm infant is less responsive to vasoconstrictive stimuli due to persistant vasodilator effect of PGE2 in addition to hypoxemia during RDS leads to persistent PDA that creat shunt between the pulmonary&systemic circulation.

Clinical features:

When RDS improves&pulmonary vascular resistance declines &flow through ductus increases in a left to right direction.

It may produce no symptoms or it may cause apnoea and bradycardia, increased oxygen requirement and difficulty in weaning the infant from artificial ventilation.

Pulse pressure widens,active precordial impulse. Active&bounding peripheral pulse. The murmur of PDA may be continous or usually systolic. Heart failure&pulmonary edema result in rales & hepatomegally.

Chest x-ray: cardiomegally &pulmonary edema.

Treatment: during RDS involves an initial period of fluid restriction& diuretics.If no improvement after 24-48 hr indomethacin {prostaglandin synthetase inhibitor} 0.2mg/kg I.V every 12 hr, 3 doses. If the patient not respond to repeated courses of indomethacine & in heart failure surgical ligation is required.

BRNCHOPULMONARY DYSPLASIA

Oxygen concentration above 40% are toxic to the neonatal lung.

Oxygen mediated lung injury results from generation of super oxides, hydrogen peroxides[H2O2],&Oxygen free radicals which disrupt membrane lipids. Mechanical ventilation with high peak pressure produces barotroma.

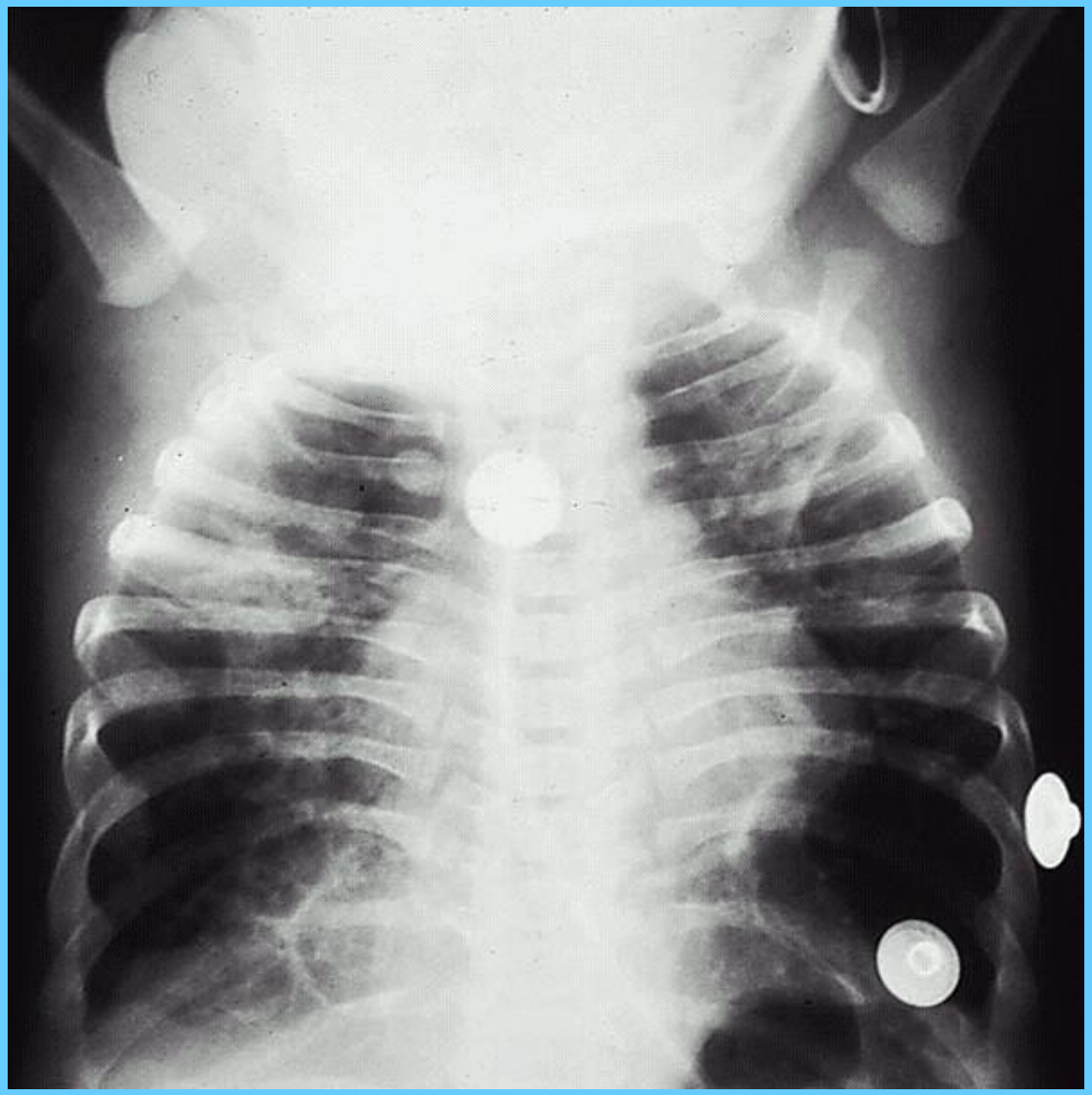
Definition: Failure of RDS to improve after 2 weeks& need for prolonged mechanical ventilation,&oxygen therapy required at 36 weeks post conception age.

Clinical feature:

Oxygen dependence, hypercapnia , compensatory metabolic alkalosis,pulmonary hypertension, poor growth,& development of right sided heart failure.Increase air way resistance with reactive air way constriction.

Treatment:

1-Bronchodilator
2-Fluid restriction& diuretics
3-Mechanical ventilation
4-Dexamethazone



Bronchopulmonary dysplasia. Note the alternating areas of hyperinflation and atelectasis.