Recurrent Chest Infections

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Rama hazzaa awad

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Definition

Infections that are too great in number , too severe , too long lasting or unusual.

- Two or more severe infections in one year.
- Three or more respiratory infections in one year.
- The need for antibiotics for two months/year.
- The need for IV antibiotics or hospitalization.

Definition

Infections that are too great in number , too severe , too long lasting or unusual.

- Infections with unusual pathogen.
- Infections with unusual complication.
- Persistent lab or imaging abnormalities.
- With confinement to bed for a week or more.

Causes

- Normal child
  - Average child
  - Risk factors
- Child with atopy
  - Asthma
- Child with chronic disease
  - Cystic Fibrosis
  - TB
  - Chronic aspiration
  - GERD
  - Congenital disease
- immunodeficiency
  - primary
  - secondary

Normal Child

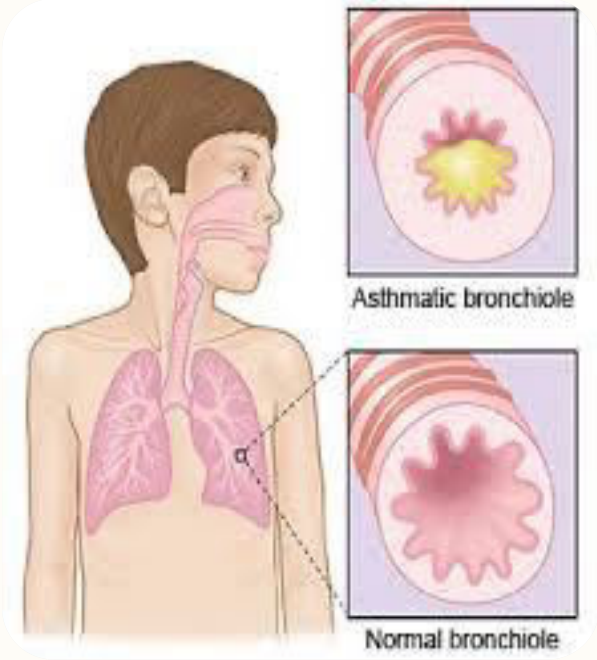
- Approximately 50 percent of children with recurrent infections referred for evaluation have no known significant cause for these infection .
- Average child has 4-8 respiratory infections per year up to 12 can be normal.
- Average duration of symptoms is 8 days up to 14 can be normal.
- Normal growth and development.
- Respond quickly to treatment, with complete recovery.
- Appear healthy between infections.
- Physical examination and lab tests are normal

Normal Child

Risk Factors

- Prematurity.
- Low birth weight.
- Reduction of breast feeding.
- Malnutrition.
- Missed vaccination.
- Overcrowding.
- Smoking.
- Pets.
- Climate and environments.


Asthma


Asthmatic bronchiole
Normal bronchiole

- Approximately 30 percent of children with recurrent infection have atopic disease.
- Usually develop coughing and wheezing following respiratory infections (reactive airway disease).
- Respond well to allergy or asthma medication.
- Characteristic physical finding.
- Elevated serum IgE.

Asthma

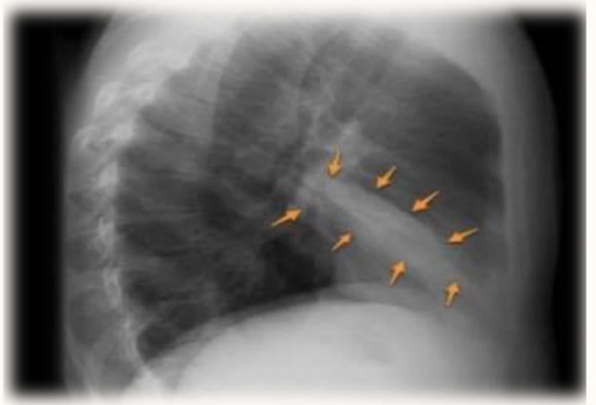
- Children with atopic disease are more likely to develop recurrent and persistent upper respiratory infections due to :

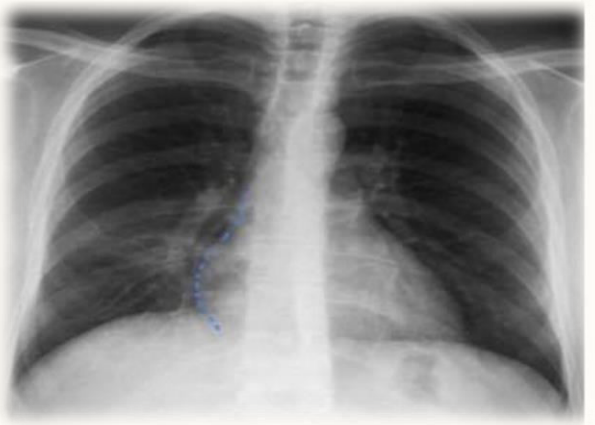
- Enhanced adherence of pathogens to inflamed respiratory epithelium.
- Increased mucosal permeability.
- Altered immune response to certain viral and bacterial pathogens.
- Obstruction leads to stasis.
- It may associate with primary immunodeficiency.
- Complication of asthma

Right Middle Lobe Syndrome

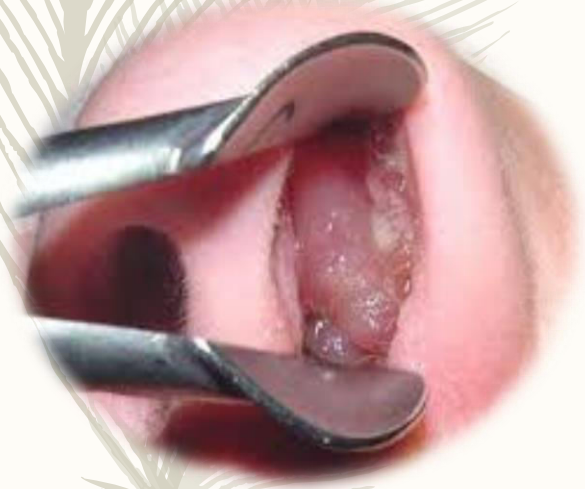
❖Right middle lobe is anatomically susceptible to atelectasis because of :

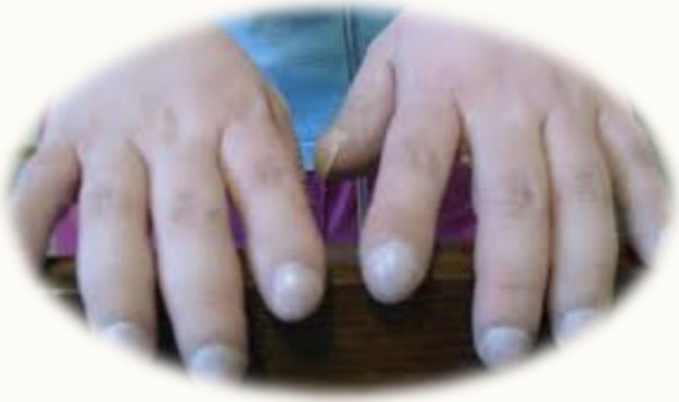
- Narrow diameter of lobar bronchus.
- Acute takeoff angle of lobar bronchus.

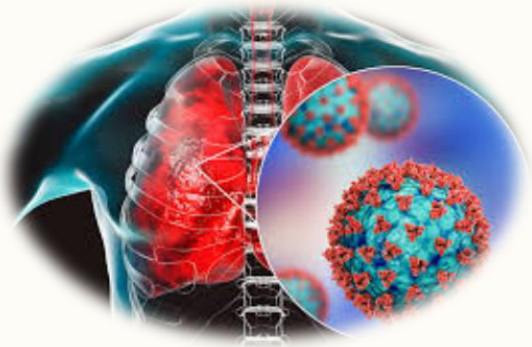




Cystic Fibrosis

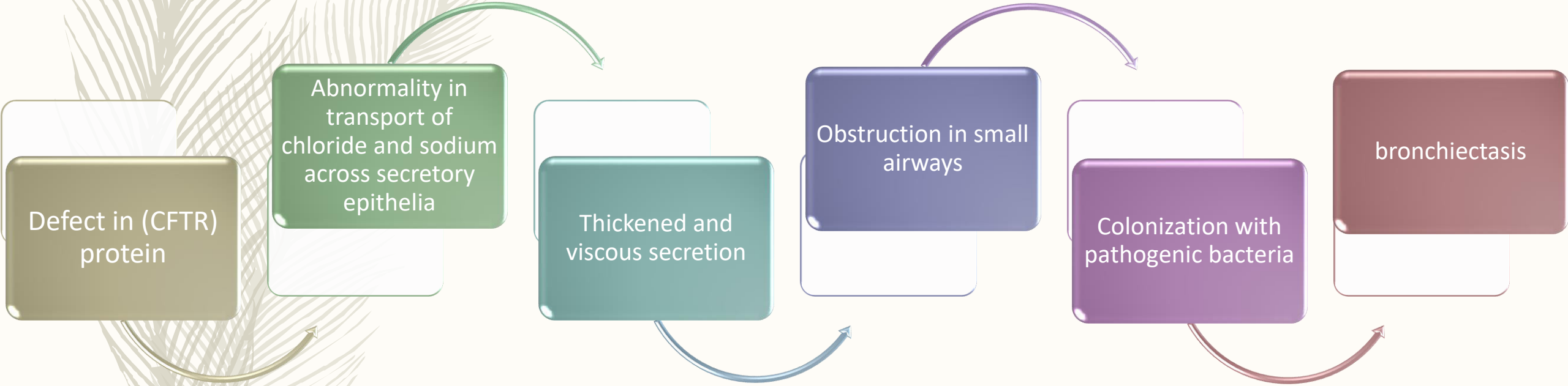






Cystic Fibrosis

- Pulmonary involvement due to cystic fibrosis is the most common cause of mortality and morbidity in cystic fibrosis.



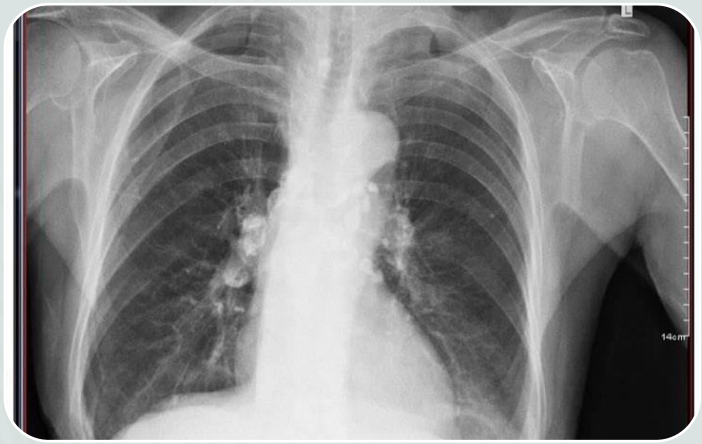
Cystic Fibrosis

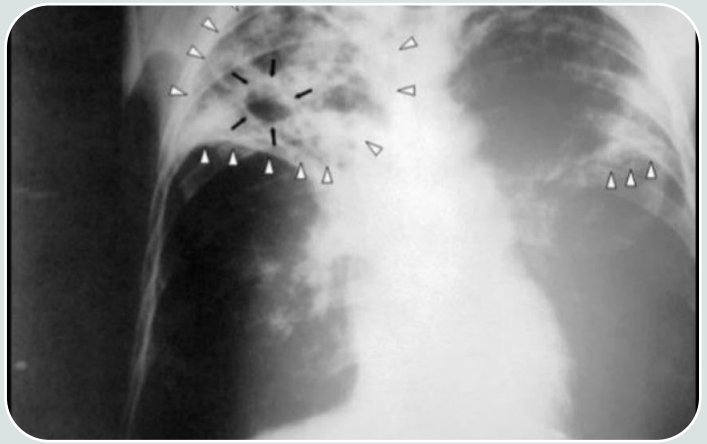
❖Treatment:

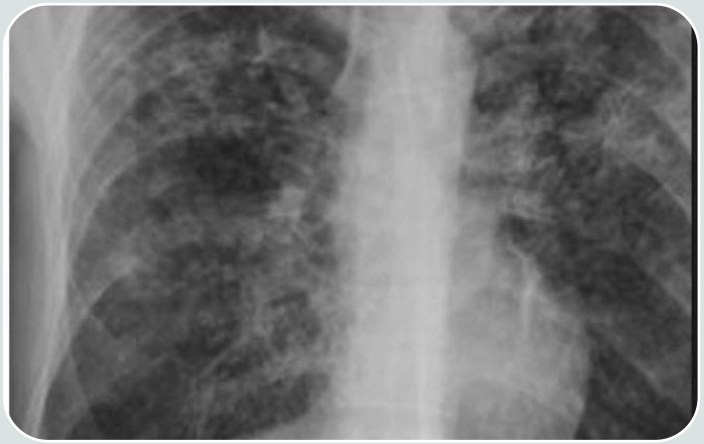
- CFTR modulator
- Airway clearance therapies
  - Inhaled DNase
  - Inhaled hypertonic saline
  - Chest physiotherapy
- Prevention of infection
  - Vaccination
  - Infection control measures
- Bronchodilators
  - Inhaled beta-2 adrenergic receptor agonist
- antibiotics

TUBERCULOSIS

TB







Primary TB (latent)

-Non contagious.

-no s+s

-normal chest Xray.

-positive PPD.

-negative sputum culture.

- Treatment.

Secondary TB (reactivation)

-contagious.

-S+S.

-Abnormal chest Xray.

-positive PPD.

-positive sputum culture.

-Treatment.

Miliary TB

-Pulmonary.

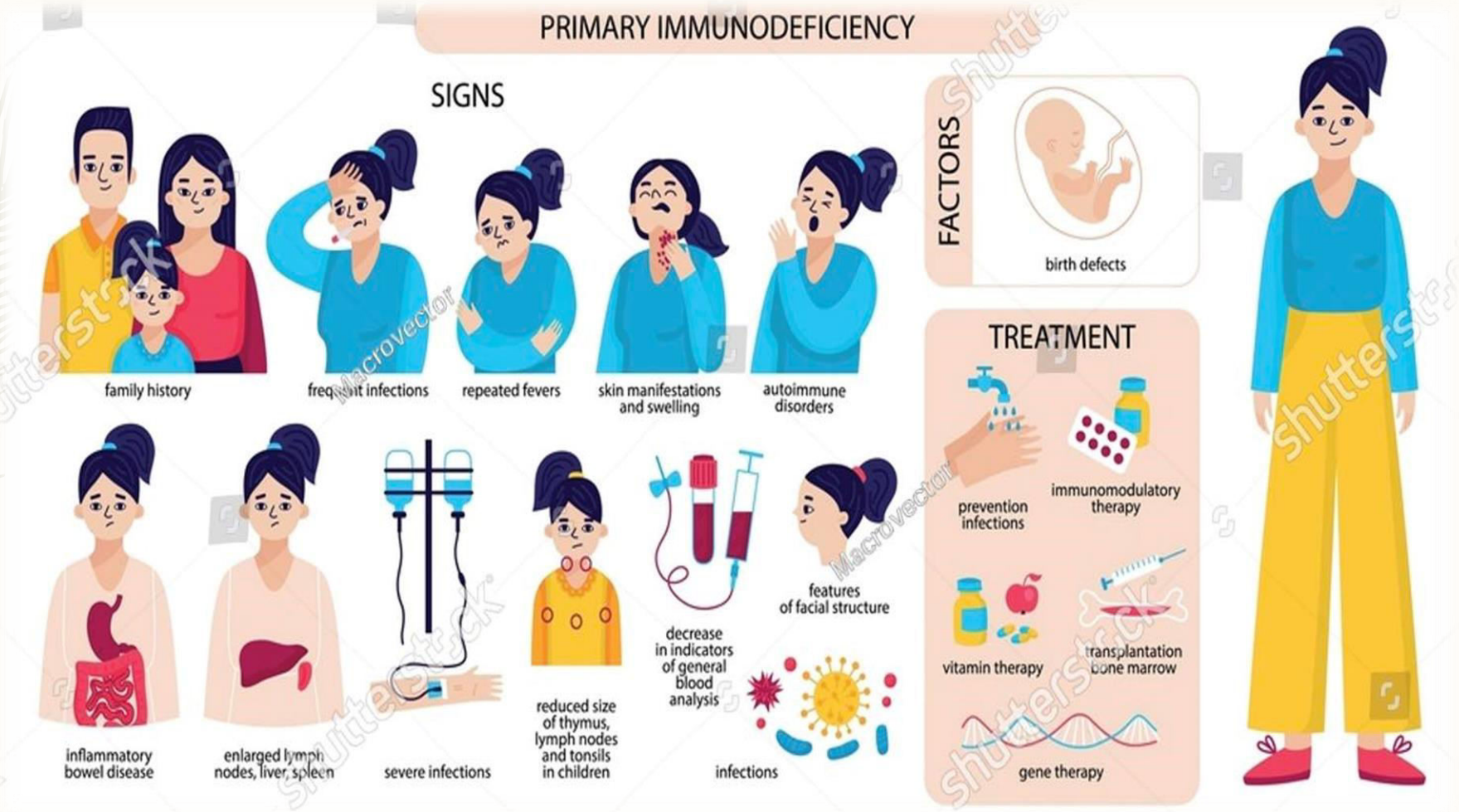
-extrapulmonary.

-infant are more predisposed.

-S+S.

-Treatment.

Immunodeficiency



10 Warning Signs of Primary Immunodeficiency

Primary Immunodeficiency (PI) causes children and adults to have infections that come back frequently or are unusually hard to cure. 1:500 persons are affected by one of the known Primary Immunodeficiencies. If you or someone you know is affected by two or more of the following Warning Signs, speak to a physician about the possible presence of an underlying Primary Immunodeficiency.

1. Four or more new ear infections within 1 year.
2. Two or more serious sinus infections within 1 year.
3. Two or more months on antibiotics with little effect.
4. Two or more pneumonias within 1 year.
5. Failure of an infant to gain weight or grow normally.
6. Recurrent, deep skin or organ abscesses.
7. Persistent thrush in mouth or fungal infection on skin.
8. Need for intravenous antibiotics to clear infections.
9. Two or more deep-seated infections including septicemia.
10. A family history of PI.

Immunodeficiency

Initial work-up

- Complete blood count with differential.
- cultures/imaging.
- IgG, IgA, IgM, IgE level.
- antibody titers to tetanus, diphtheria, haemophilus influenza and pneumococcus

TABLE 73.1 Antibody Deficiency Diseases

DISORDER	GENETICS	ONSET	MANIFESTATIONS	PATHOGENESIS	ASSOCIATED FEATURES
Agammaglobulinemia	X-linked, AR	Infancy (6-9 mo)	Recurrent infections, sinusitis, pneumonia, meningitis (encapsulated bacteria, enteroviruses)	Arrest in B-cell differentiation (pre-B level); mutations in: *Btk* gene (X-linked); μ chain, BLNK, Igα, Igβ, γ5, BLNK (AR)	Lymphoid hypoplasia
Common variable immunodeficiency	AR; AD; sporadic	2nd to 3rd decade	Sinusitis, bronchitis, pneumonia, chronic diarrhea	Arrest in plasma cell differentiation, mutations in ICOS, TACI, CD19, CD81, CD20, CD21	Autoimmune disease, RA, SLE, Graves disease, ITP, malignancy, granulomatous disease
Transient hypogammaglobulinemia of infancy		Infancy (3-7 mo)	Recurrent viral and pyogenic infections	Unknown; delayed plasma cell maturation	Frequently in families with other immunodeficiencies
IgA deficiency	Variable	Variable	Sinopulmonary infections; can be normal	Failure of IgA expression	IgG subclass deficiency common, autoimmune diseases
IgG subclass deficiency	Variable	Variable	Sinopulmonary infections; can be normal	Defect in IgG isotype production	IgA deficiency, ataxia telangiectasia, polysaccharide antibody deficiency
Specific antibody deficiency	Variable	After 2 years of age	Sinopulmonary infections	Unknown	IgG subclass deficiency
Hyper-IgM syndrome	AR	Variable	Sinopulmonary infections	Defect in AID, UNG	Autoimmunity

Foreign body

Epidemiology

- Aspiration of foreign bodies into the trachea and bronchi is **relatively common**
- the majority of children who aspirate foreign bodies are **under 3 years of age.**
- Patients with **developmental delay** or **with older siblings** are at increased risk.
- the most common foreign bodies aspirated are:
- In young children : **food** (**especially nuts**) and **small toys**
- In **older** children : **rubber balloons**

Clinical Manifestations

- **Many children** who aspirate foreign bodies have **clear histories** of choking, witnessed aspiration, or physical or radiographic evidence of foreign body aspiration.
- Some patients have a **negative history** because the aspiration went **unrecognized**.
- Foreign body aspiration should be in the deferential diagnosis of patients with recurrent or persistent pneumonia, persistent wheezing unresponsive to bronchodilator therapy, persistent atelectasis, , or chronic cough without another explanation

Diagnosis

- **History**
- **Physical exam:**

On auscultation, these patients have wheezing, decreased breath sounds, or both on the side of the foreign body.

- **Radiographic studies:**

- x-ray

- Posteroanterior inspiratory and expiratory views or decubitus films

they will reveal the presence of **radiopaque objects** and can also identify focal air trapping on expiration and atelactasis on inspiration

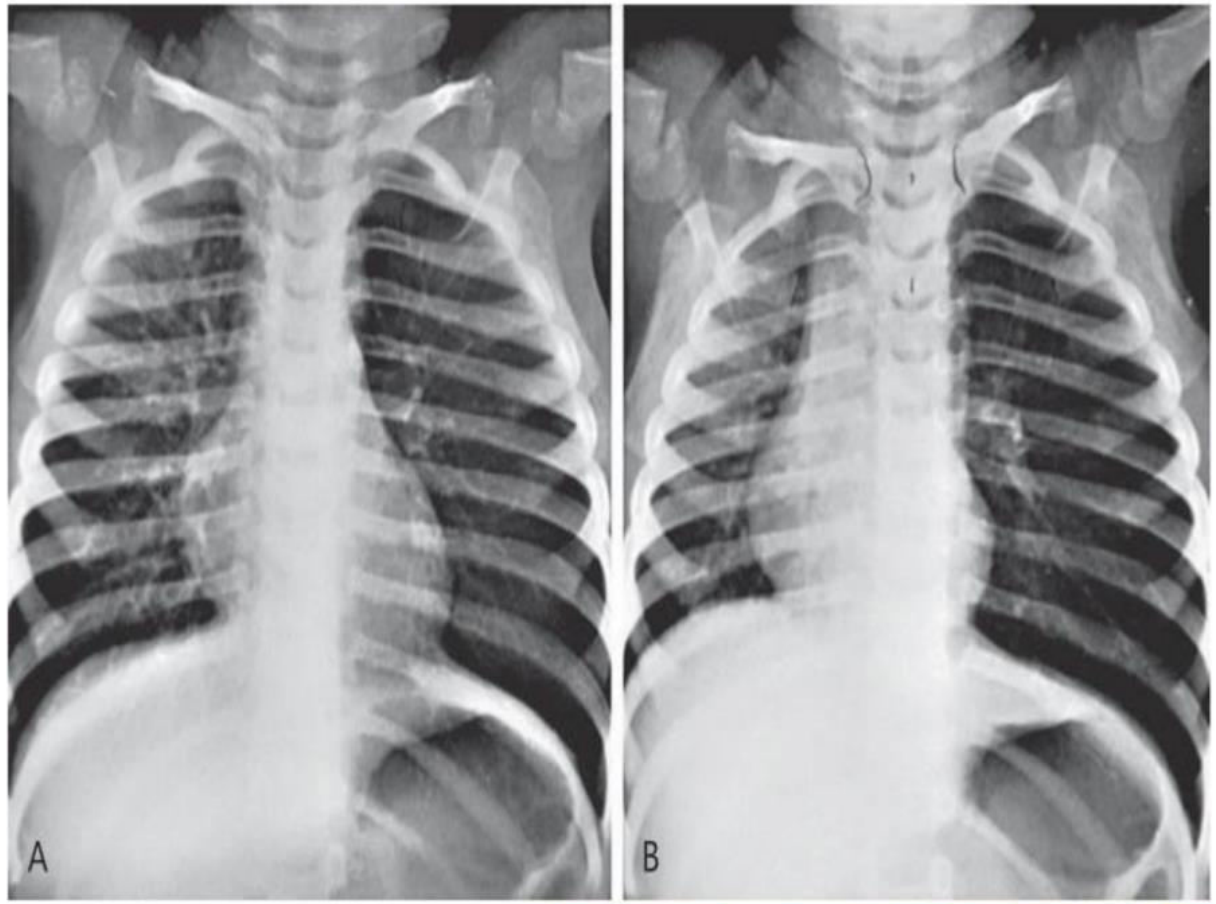


Figure 387-2 **A,** Normal inspiratory chest radiograph in a toddler with a peanut fragment in the left main bronchus. **B,** Expiratory radiograph of the same child showing the classic obstructive emphysema (air trapping) on the involved (left) side. Air leaves the normal right side allowing the lung to deflate. The medium shifts toward the unobstructed side.

- CT
- Bronchoscopy:

- Flexible

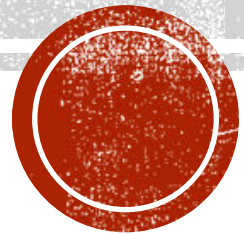
- Rigid ; If history or exam is suggestive of foreign body aspiration, the patient should undergo rigid bronchoscopy.

Management and Prevention

- foreign body removal is best performed via rigid bronchoscopy.

- Older siblings should be counseled to keep small toys separate and not present younger children with small parts.

CHRONIC RECURRENT ASPIRATION



BACKGROUND

- Aspiration syndromes include all conditions in which foreign substances are inhaled into the lungs. Most commonly, aspiration syndromes involve oral or gastric contents associated with gastroesophageal reflux (GER), swallowing dysfunction, neurological disorders, and structural abnormalities.
- According to the volume aspirated symptoms can be acute(if large amount) or episodic and intermittent(if small amount).
- Therefore the extent of injury is related to:
  1. The volume of the aspirate
  2. Frequency of aspiration
  3. Efficacy of lung clearance mechanisms

HISTORY

- Acute aspiration may manifest as coughing, wheezing, fever, and chest discomfort.
- In the setting of massive aspiration, the patient may present with cyanosis and/or pulmonary edema.
- Occasionally, aspiration may be silent with no overt signs or symptoms.
- The 4 syndromes that may be associated with chronic lung aspiration are:
  1. recurrent wheezing (can be related to aspiration of gastric contents)
  2. apnea (obstructive or central)
  3. chronic cough(>3 weeks)
  4. recurrent pneumonia.
  5. Noisy breathing, Recurrent vomiting, Choking, gagging, coughing, and/or spitting during feeds

PHYSICAL EXAMINATION

1. Increased work of breathing; grunting, flaring, retractions
2. Crackles or stridor
3. Fever
4. Tachypnea

LABORATORY STUDIES

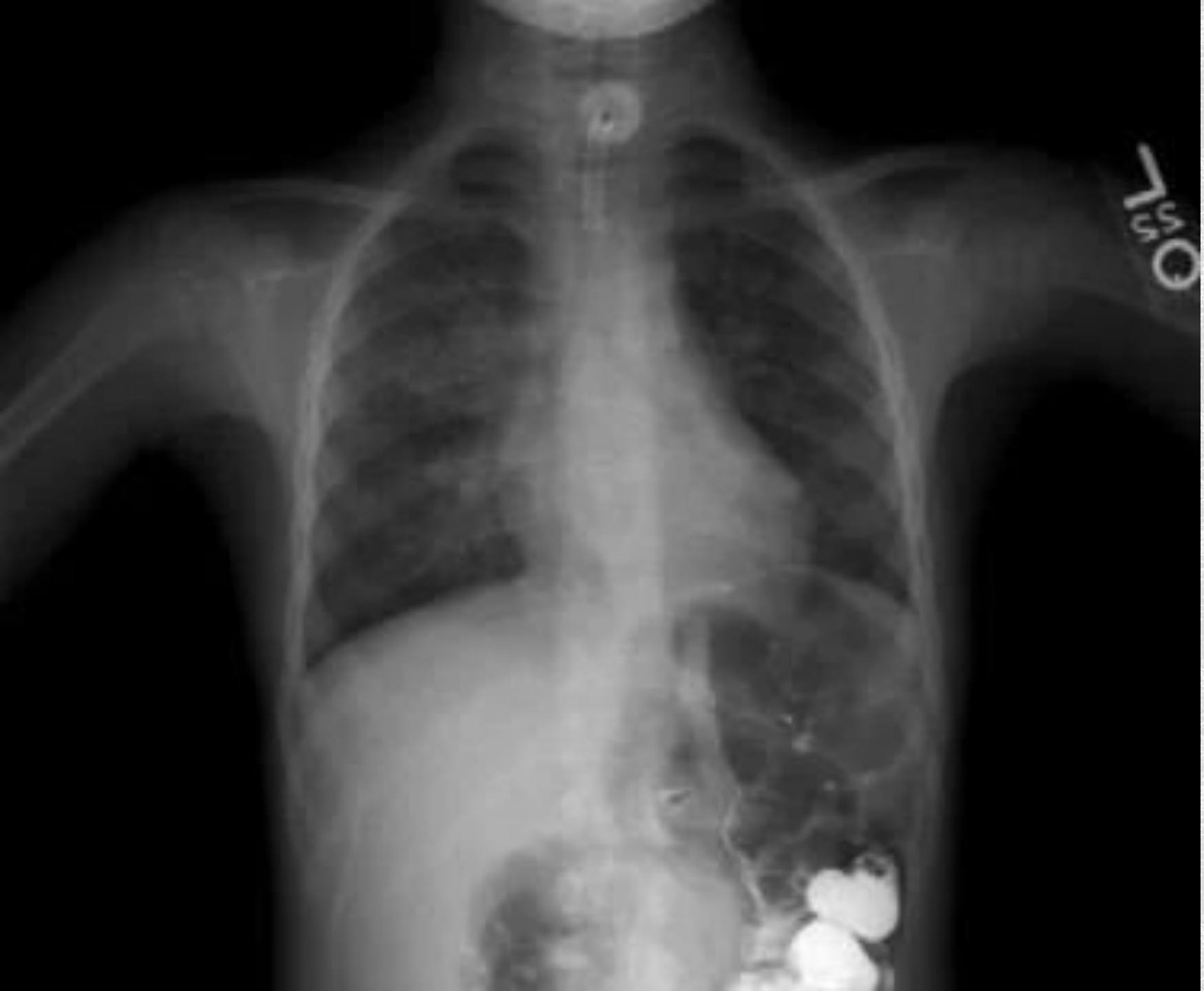
1. CBC count with differential
2. ABG or pulse oximetry
3. Sweat chloride
4. Pulmonary function test

IMAGING STUDIES

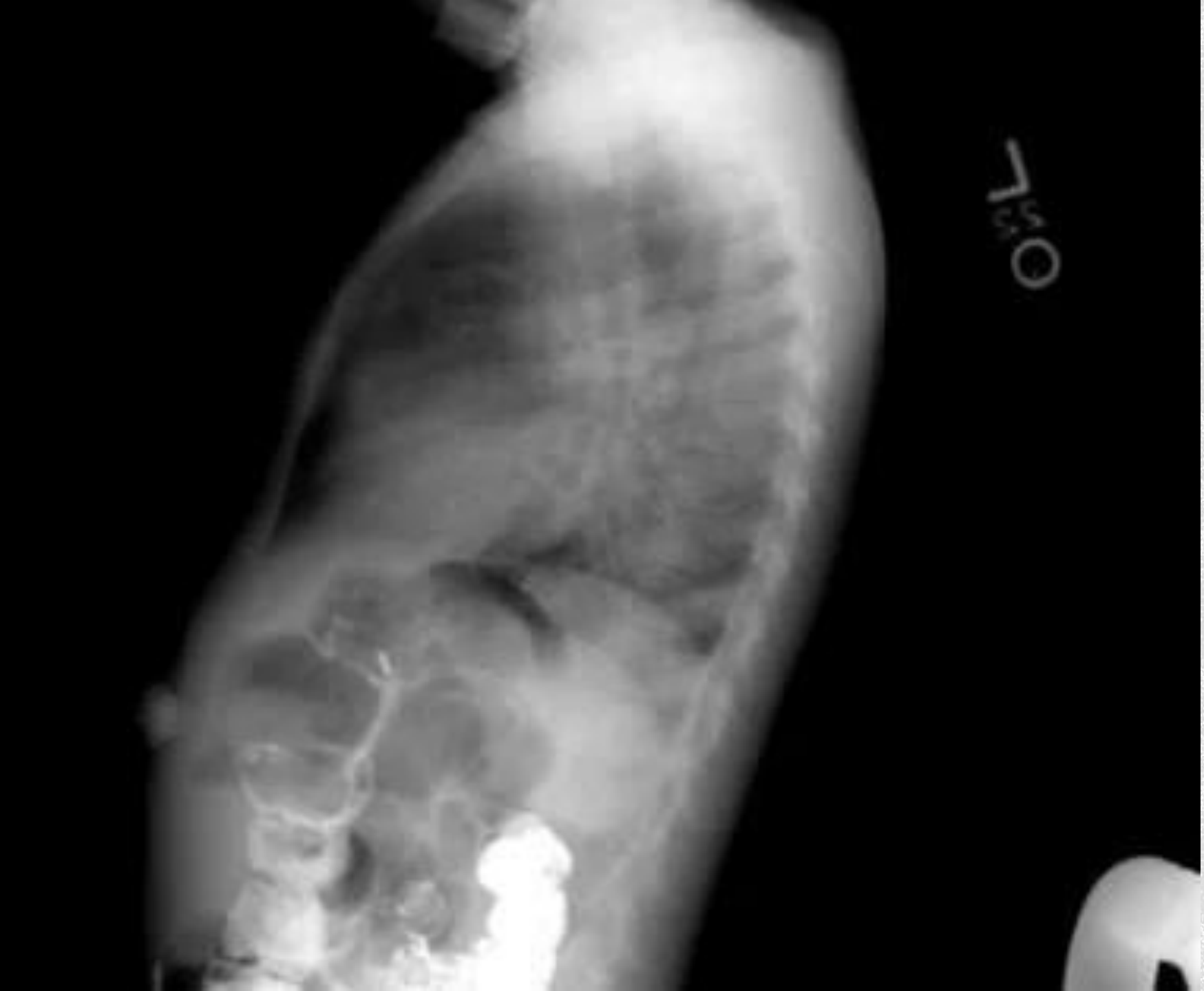
- CXR:

1. hyperinflation
2. marked diffuse interstitial or perihilar infiltrates
3. unilateral or bilateral; peribronchial thickening
4. pleural effusion
5. lobar or segmental consolidation
6. Bronchiectasis
7. or atelectasis

- BARIUM SWALLOW



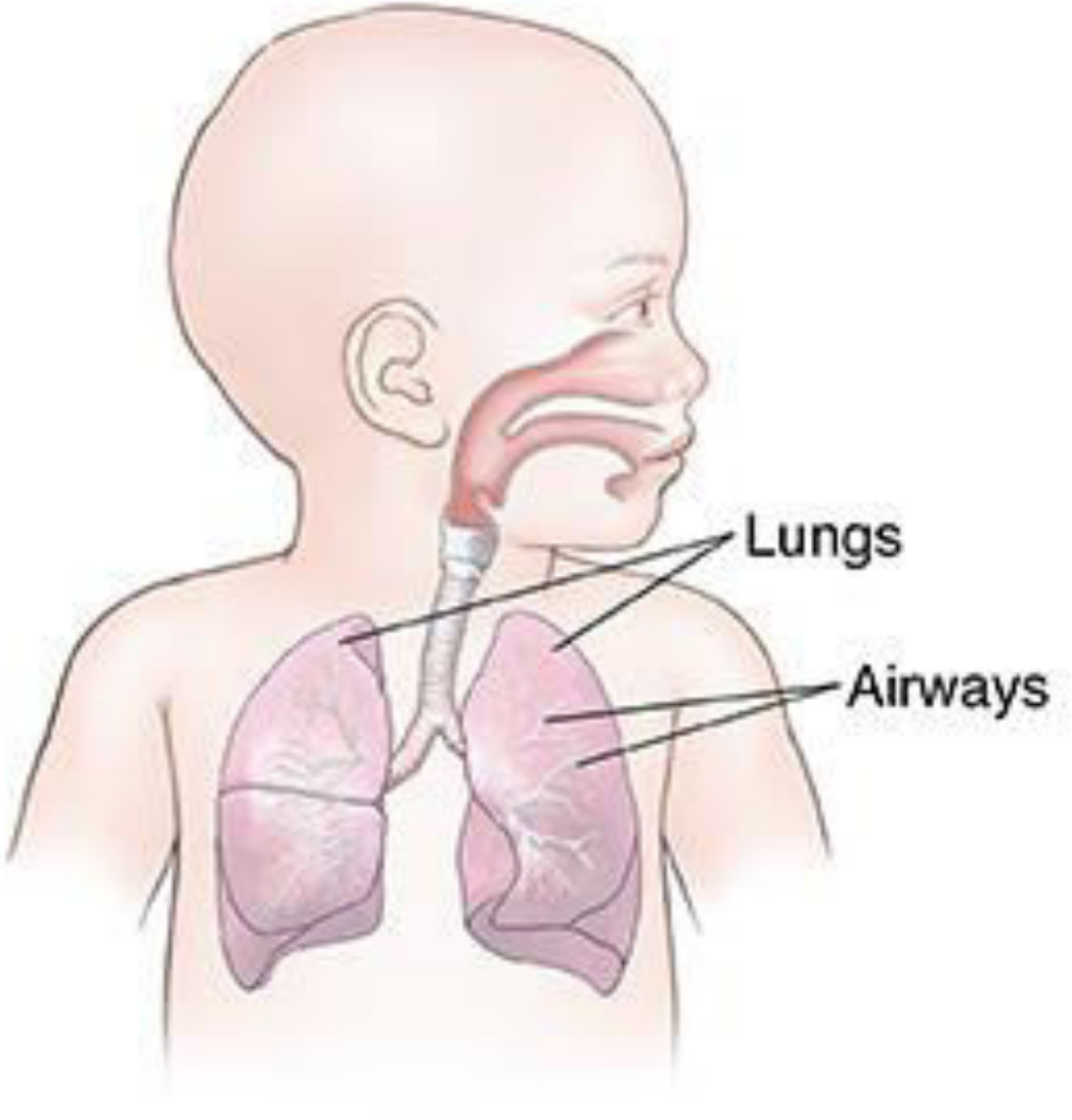
Chest radiograph of a child with a tracheostomy and recurrent aspiration reveals patchy infiltrates and increased interstitial markings.



Lateral chest radiograph of the same child as in the previous image reveals increased interstitial markings and patchy and perihilar infiltrates.

MANAGEMENT

- Conservative therapy is the initial treatment of choice to prevent aspiration syndromes
  1. Position infants in the prone or upright position
  2. Dietary modifications (formulas, breastfeeding, decreasing volume of feeds and feeding small, frequent meals.
- Initially, empiric antibiotic therapy is not recommended
- Medications to enhance gastric emptying (prokinetic agents) / H2 receptor
- antagonists/ PPIs.
- Surgical intervention in complicated unresponsive cases



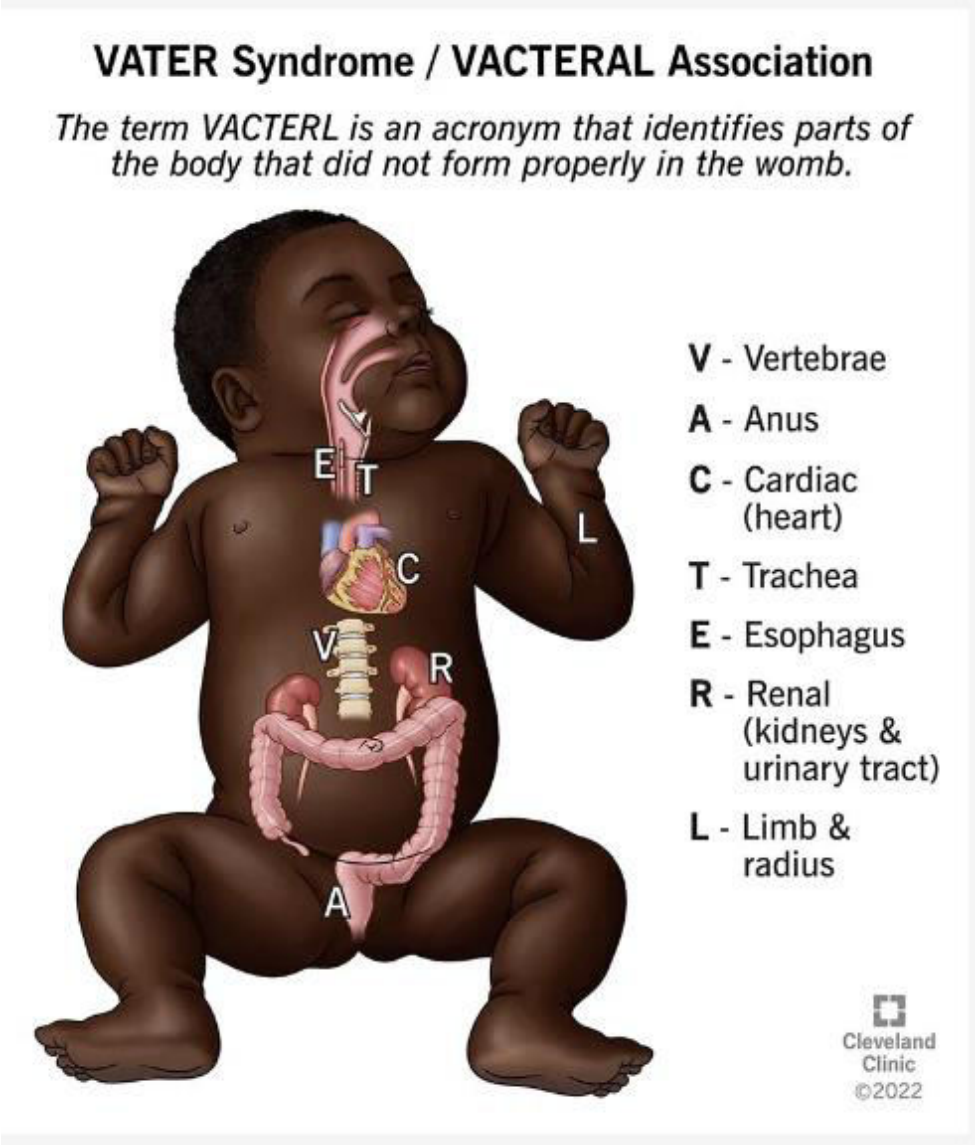
CONGENITAL ANOMALIES OF THE RESPIRATORY TRACT

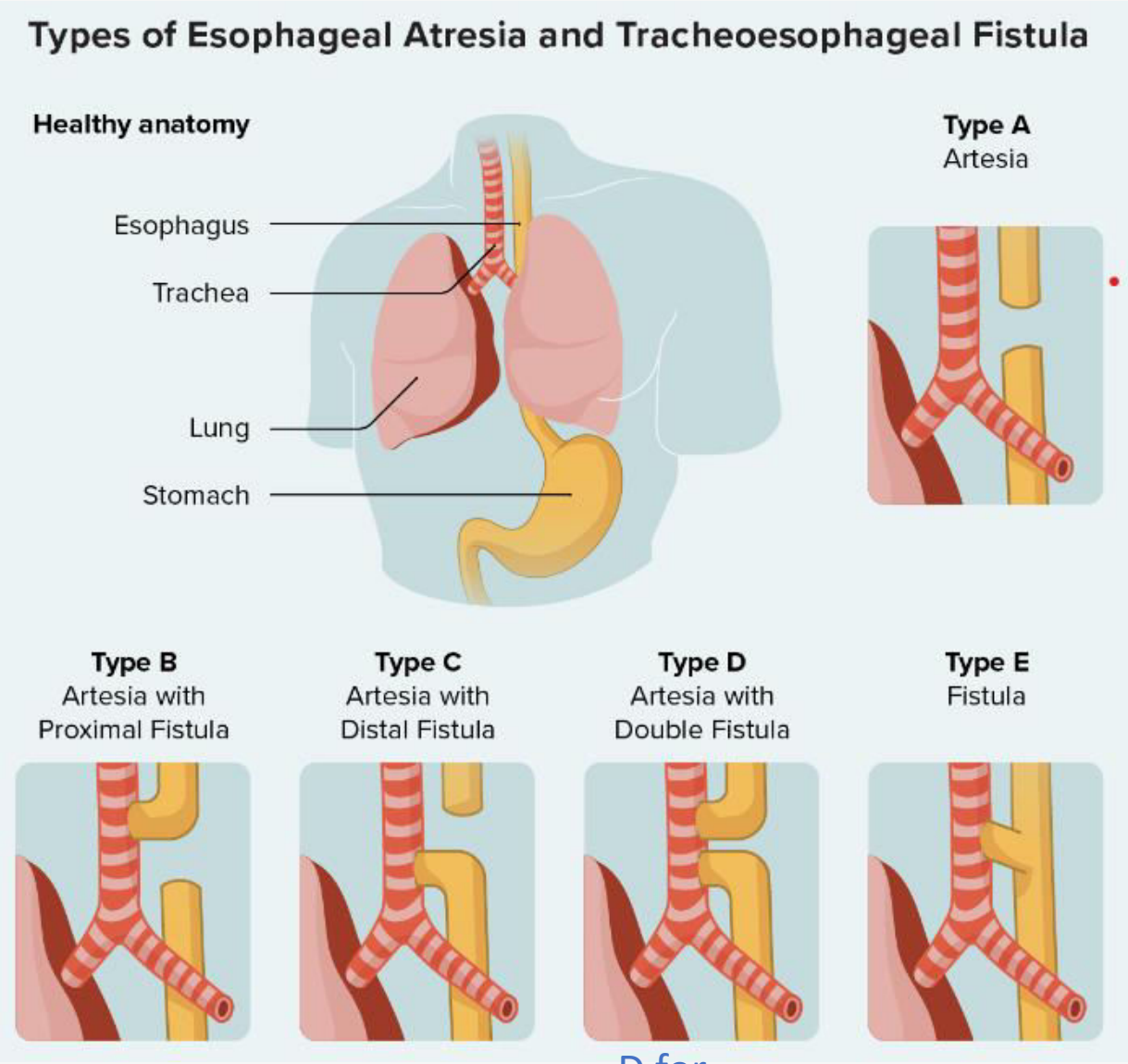
TRACHEOESOPHAGEAL FISTULA

- Its an abnormal communication between the **trachea** and **esophagus.**
- Typically (most types) , occur with (esophageal atresia) which is congenital discontinuity of esophageal lumen from pharynx to stomach during embryonic development .
- It is associated with multiple **complications**, including recurrent pneumonia, acute lung injury, acute respiratory distress syndrome, lung abscess, poor nutrition, bronchiectasis from recurrent aspiration, respiratory failure, and death.

- Etiological factors may include:Maternal alcohol and smoking , Exogenous sex hormones ,Exposure to methimazole ,Prolonged mechanical ventilation via Endotracheal or tracheotomy tube ,maternal DM in first trimester

- 17-70% of children with TEFs have associated development anomalies like **Down syndrome**, **duodenal Artesia** and **VACTERL association**





D for
DOUBLE

- **Type C (85%)**is the most common type .
- **Types B (1%), C , D (1%) , E (4%)** can present with recurrent chest infections
- **in Type e (H type)** , the most common mode of presentation is recurrent chest infections, and it usually presents later on .

PRESENTAION OF TEF :

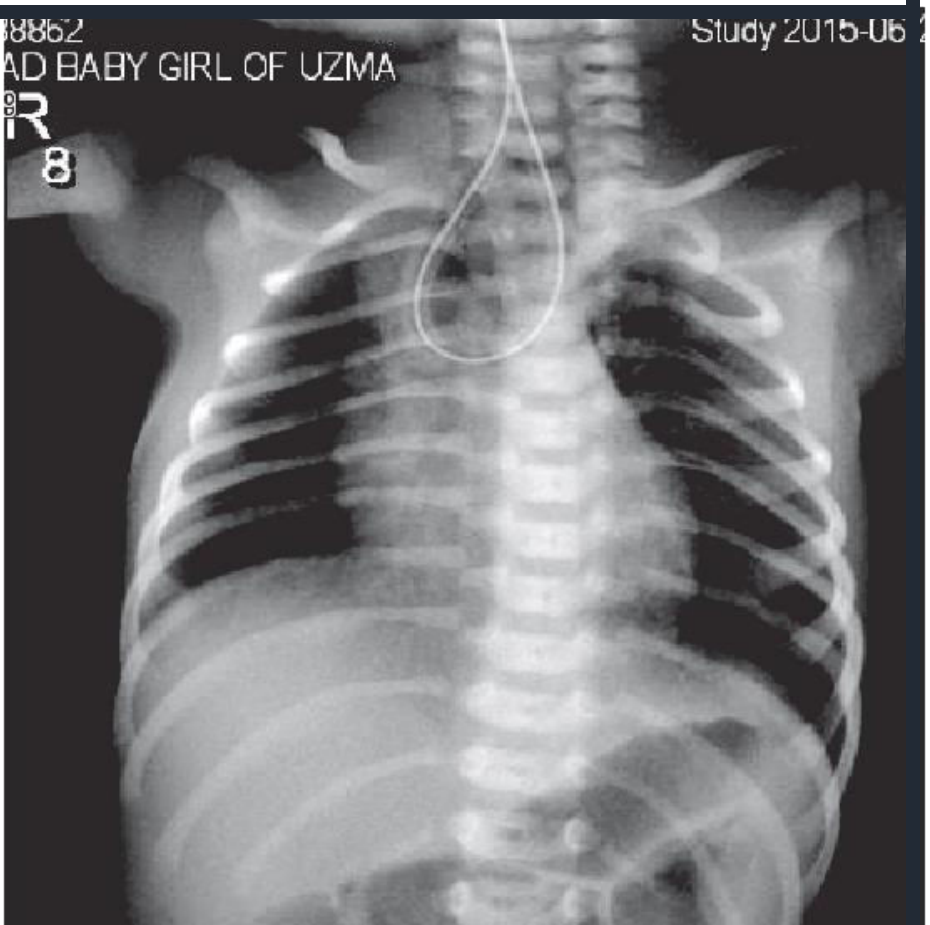
1 -History of possible **polyhydramnios** as the affected fetus cannot swallow amniotic fluid.

2-present within the first few hours of life by: “vomiting with first feeding” and Excessive salivation

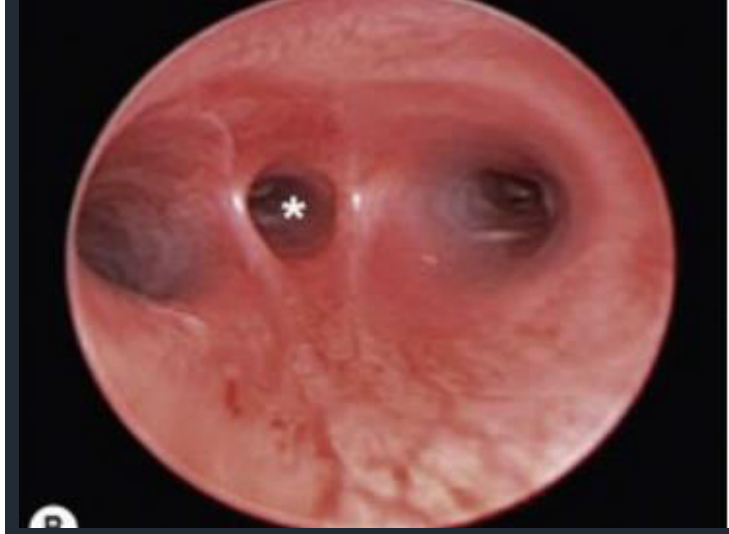
3-Respiratory distress , Cyanosis , Choking , Coughing and Recurrent aspiration pneumonia

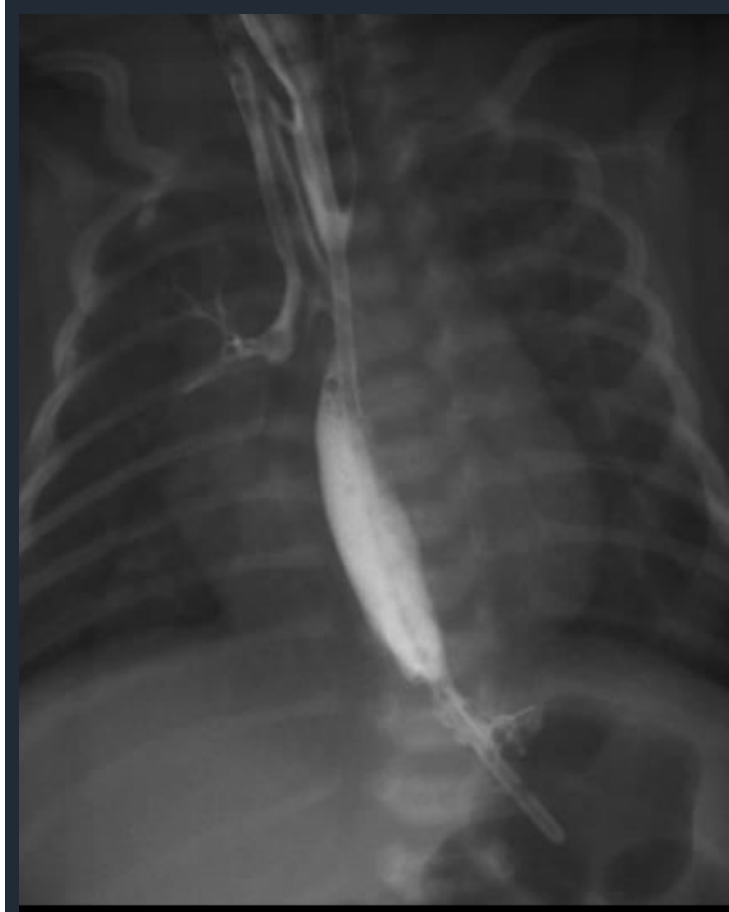
4- Inability to pass a nasogastric tube is pathognomonic for TEF and useful for diagnosis **“ Coiling sign** “ , also A gastric air bubble and esophageal air bubble can be seen on chest X-ray (CXR)

Coiling sign :



- 4- Inability to pass a nasogastric tube is pathognomonic for TEF and useful for diagnosis **“ Coiling sign “** , also A gastric air bubble and esophageal air bubble can be seen on chest X-ray (CXR)
- Note:H- type : Esophagogram with <u>contrast medium</u> and we can visualize the level of TEF by using <u>Bronchoscopy .</u>





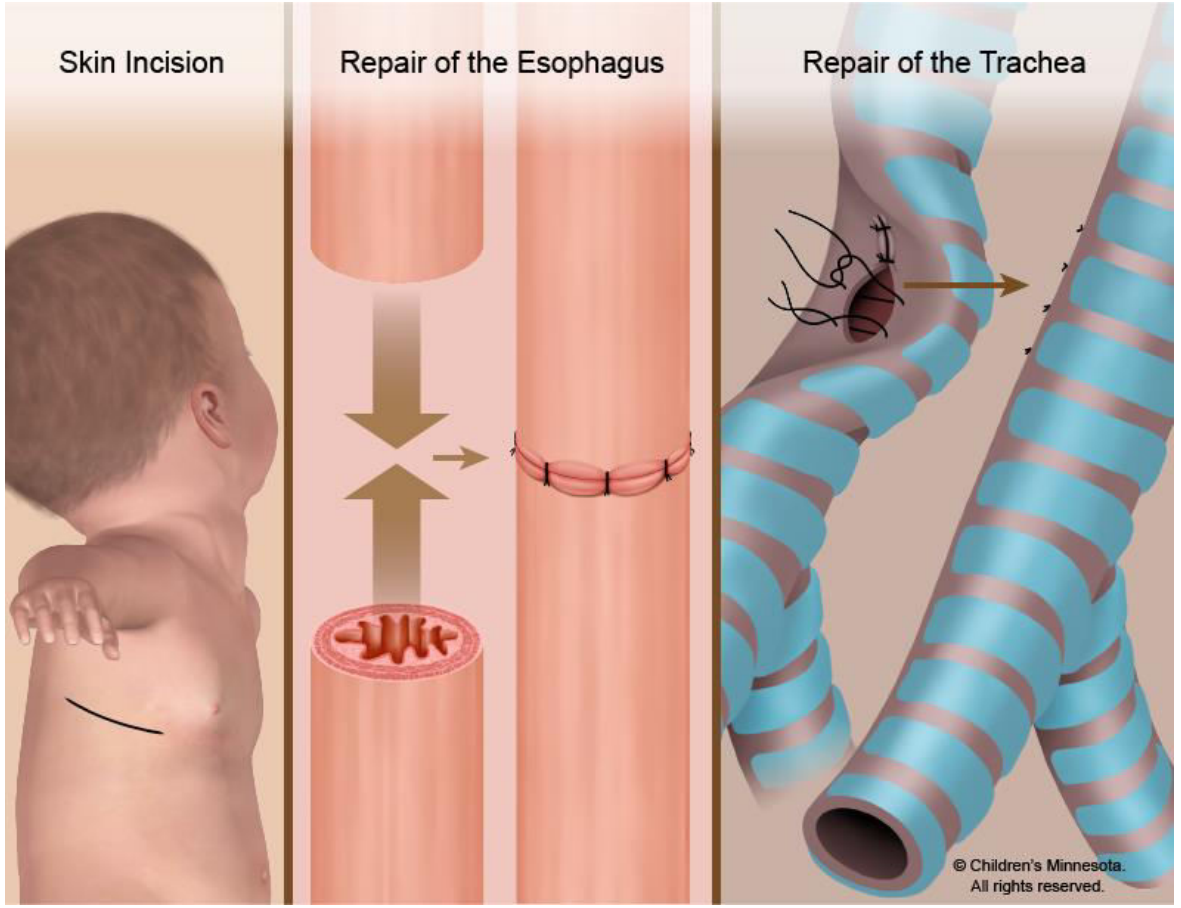
Management and Treatment

- Mainly surgical by division and closure of the fistula and anastomosis of the two esophageal segments
- Preoperative medical management includes:

1-Propping infant at 30-degree angle and Nasogastric tube remains in the esophagus, and it is aspirated frequently.

2-NPO and meeting nutritional requirements IV fluid, antibiotics.

3-respiratory support.

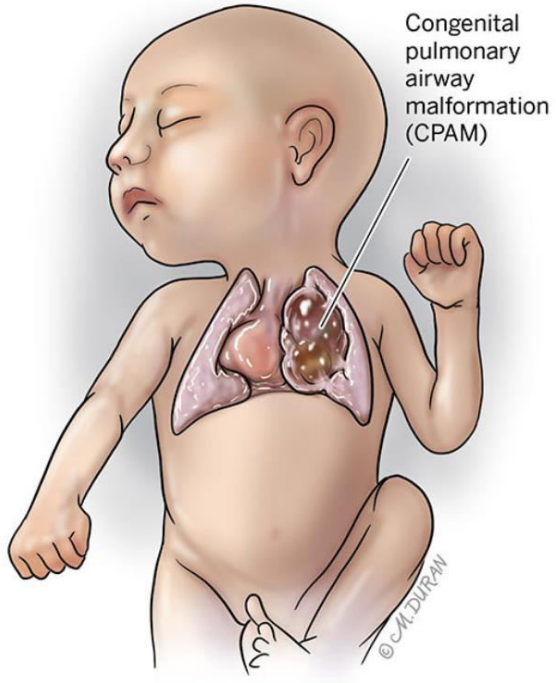


Congenital Pulmonary Airway Malformation:

Congenital cystic adenomatoid malformation (CCAM)

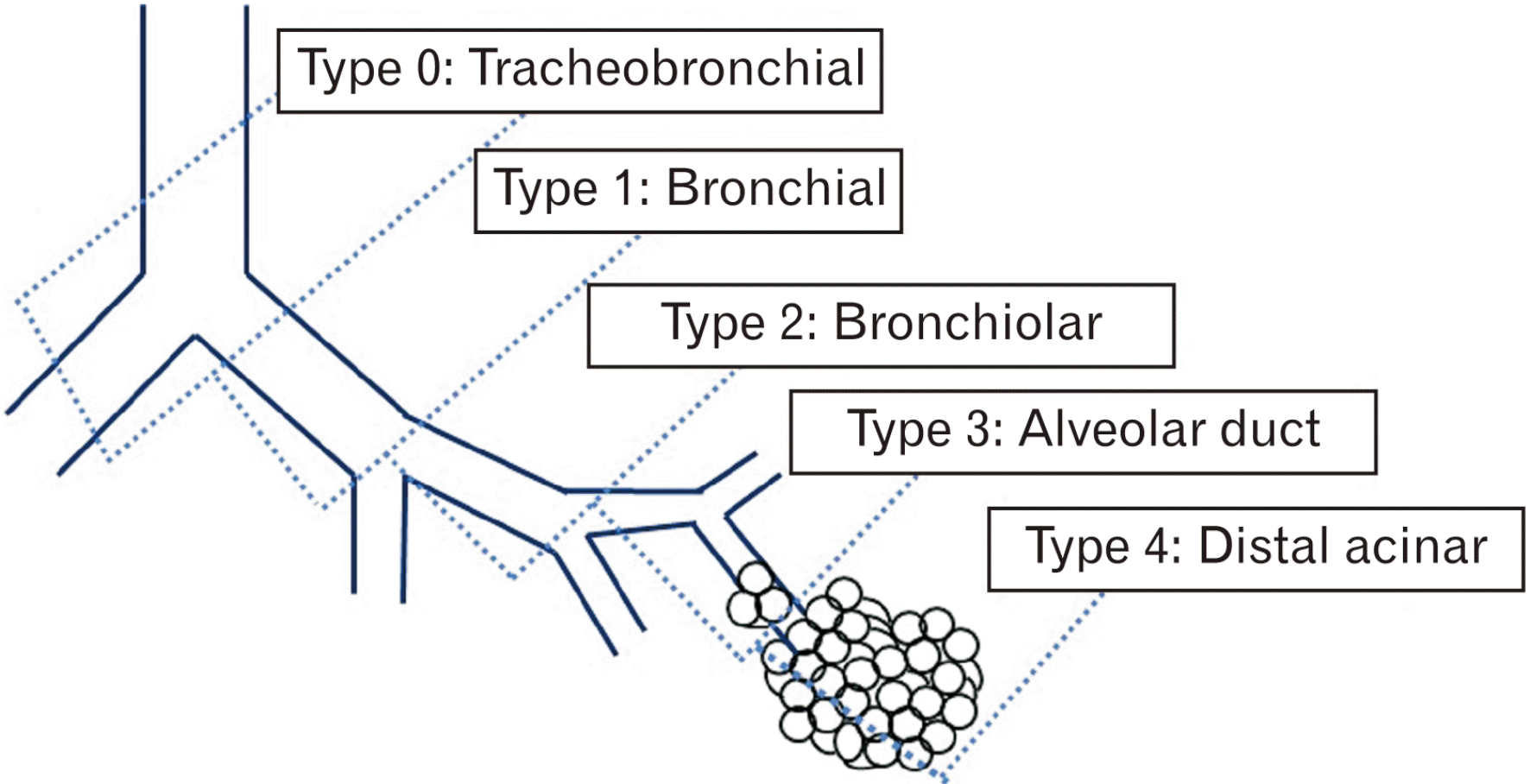
- They account for 25% of congenital lung lesions.
- With prevalence 1 : 3800 live births
- M > F
- Mostly Sporadically

- Lesions are hamartomata's(formed from cells that are normally found in the area)
- Contain **cystic** and **adenomatous** elements
- Connected to tracheobronchial tree (vs bronchogenic cysts)
- Have blood supply from pulmonary circulation
- Usually unilateral and limited to one lobe



Stoker classification for CPAM

- 5 types (0-4)
- Classified according to the **level of insult** and the **different stages of lung development**



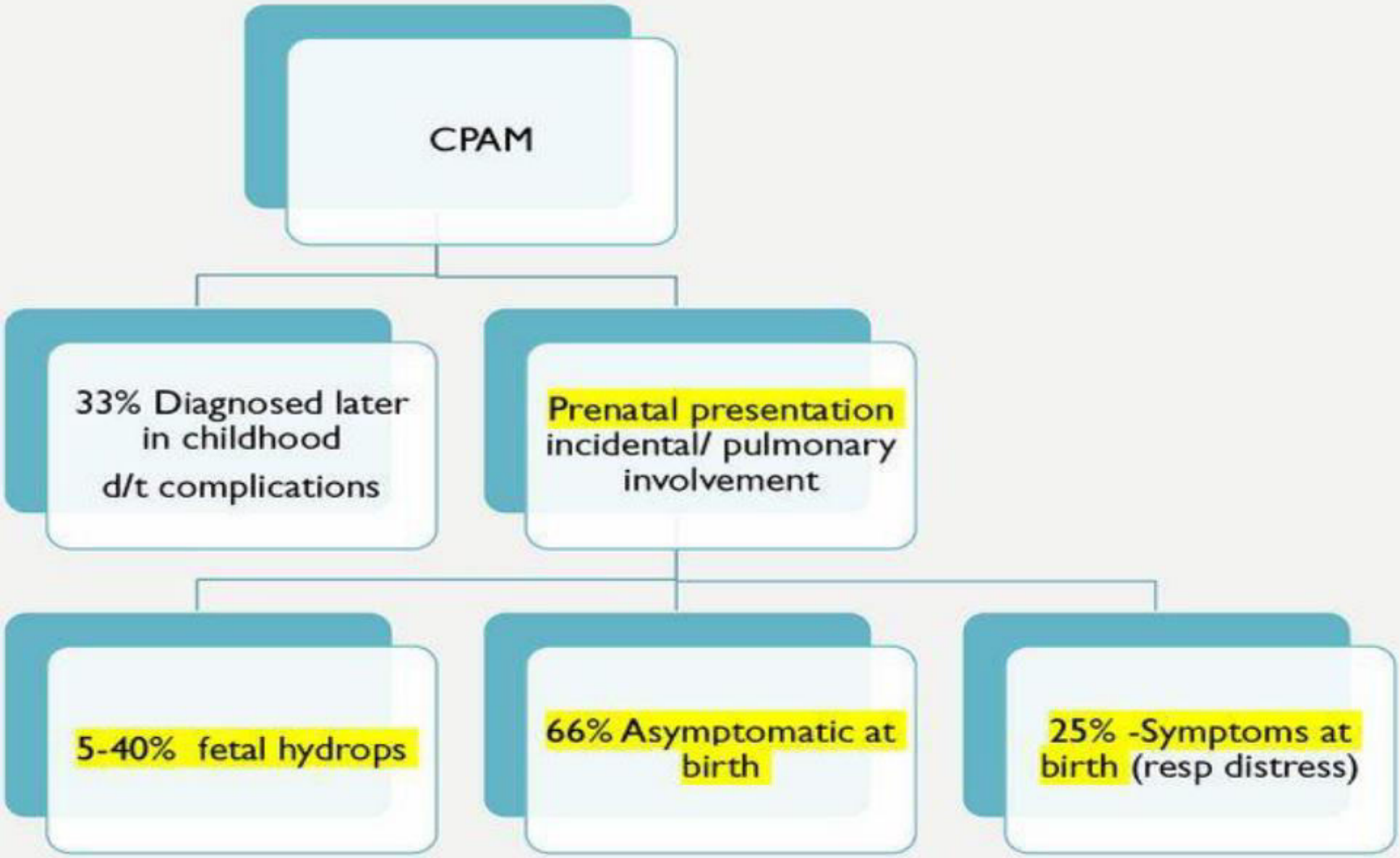
Presentation of CPAM:

1- **Asymptomatic**
2-In utero compression of normal fetal lung can cause Pulmonary hypoplasia → **respiratory distress**
3-**Recurrent URTI**
4-**Pneumothorax**
5-**Malignancy** Sx.(type 2)

CPAM- 5 TYPES PATHOLOGICALLY

Types	Percentage	Location in the airways	Lesion description	Prognosis	Comments
Type 0	1-3%	From the trachea/bronchi and **involve the whole lung**	Solid, lungs are small and firm	**Fatal-** incompatible with life.	
Type 1	**60-70%**	From distal bronchi or proximal bronchioles	**One Large cyst** 2-10cm, presentation may be late	Most common type –**good prognosis**	**Associated with malignancy BAC (rare)**
Type 2	**15-20%**	Bronchiolar origin	**Multiple small cysts** 0.5-2cm Neonatal period	**Poor prognosis**	**Associated with other anomalies**
Type 3	5-10%	Alveolar origin	Small cystic area with solid tissue. **Solid appearance**	Respiratory distress in neonatal period/death	**Severe**
Type 4	10-15%	Acinar origin	Thin walled Large fluid filled/ air filled cysts up to 10cm.	**Good prognosis**	**Associated with pneumothorax and PPB**

CPAM- CLINICAL PRESENTATION



Diagnosis: -

1-**Prenatal ultrasound** (appear as solid or cystic intrathoracic mass, also there can be a mass effect where the heart may appear displaced to the opposite side) → follow up echocardiogram + fetal MRI

2-**CT** allows <u>accurate diagnosis</u> and sizing of the lesion and is indicated even in asymptomatic neonates

3-Definitive diagnosis by Histological examination - It may grow, stay, regress …

FOLLOW UP

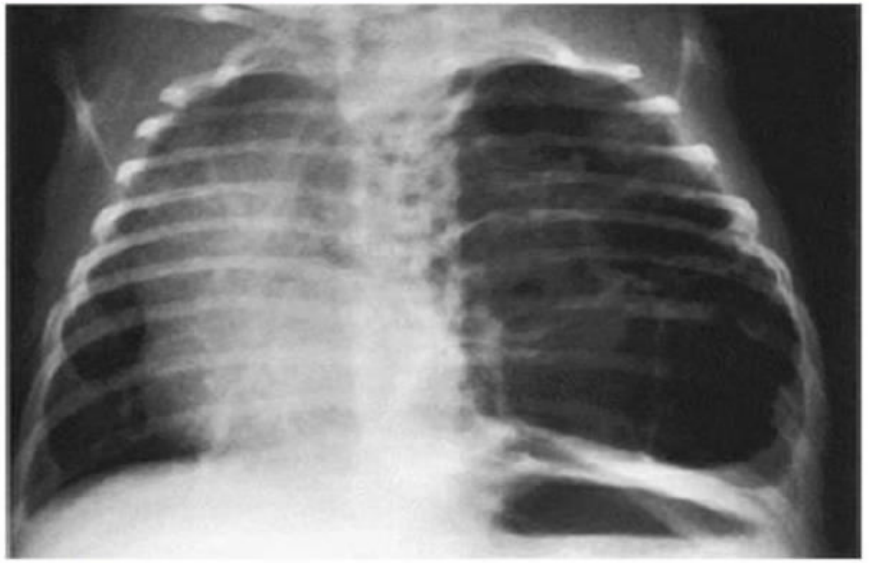
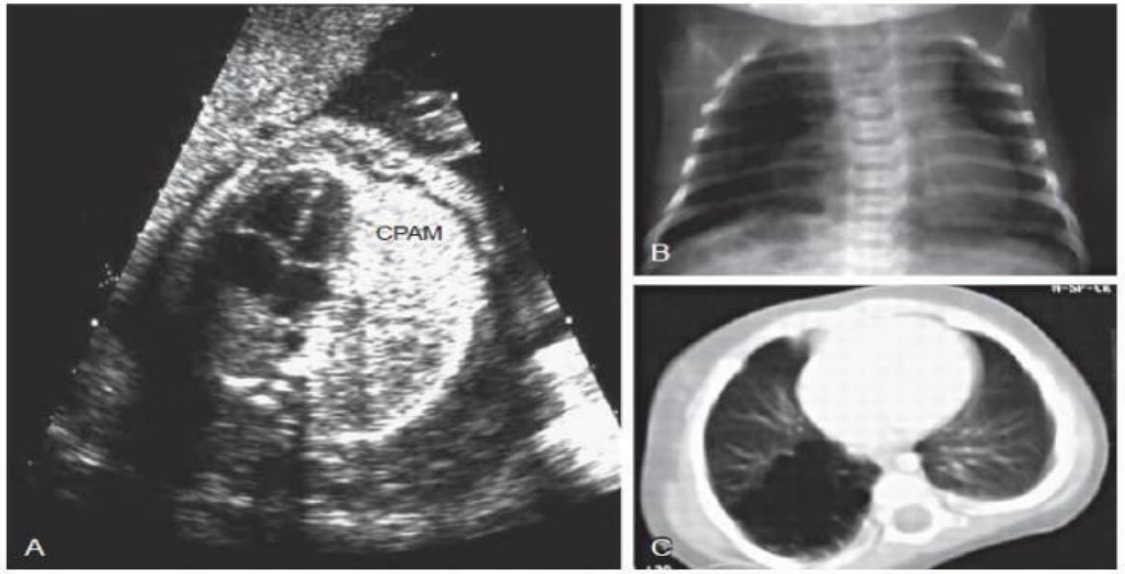
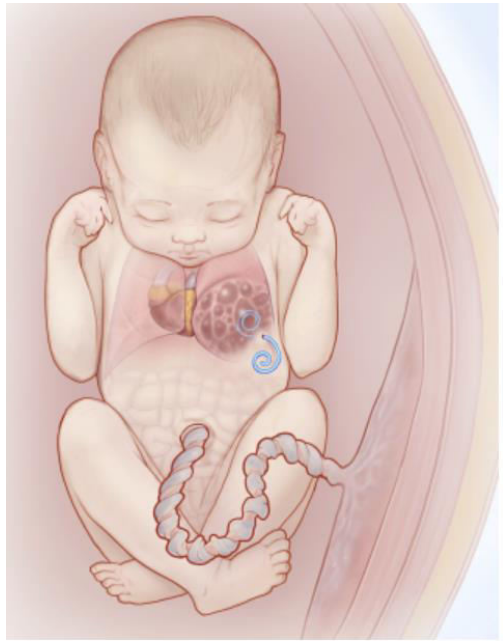


Figure 395-2 Neonatal chest x-ray showing large multicystic mass in the left hemithorax with mediastinal shift as a result of congenital pulmonary airway malformation (CPAM). *(From Williams HJ, Johnson KJ: Imaging of congenital cystic lung lesions,* Paediatr Respir Rev *3:120–7, 2002.)*



re 395-1 Imaging of congenital pulmonary airway malformation of the lung (CPAM) on the same patient with prenatal u hest radiograph **(B)**, and CT scan **(C)**. Note that the lesion is not visible on the chest radiograph. *(From Lakhoo K: Manageme adenomatous malformations of the lung,* Arch Dis Child Fetal Neonatal Ed *94:F73–F76, 2009.)*

Management of CPAM:



- **Antenatal intervention**
  in severely affected infants is controversial but can include:

1. excision of the affected lobe for microcystic lesions

2. aspiration of macrocystic lesions, and

3. rarely, open fetal surgery

- **postnatal period**→ surgery for symptomatic patients.

1-In asymptomatic surgery is delayed in the hopes of resolution (but true complete resolution is rare)

2--surgical resection by 1 year of age is recommended to limit malignant potential. The mortality rate is <10%.

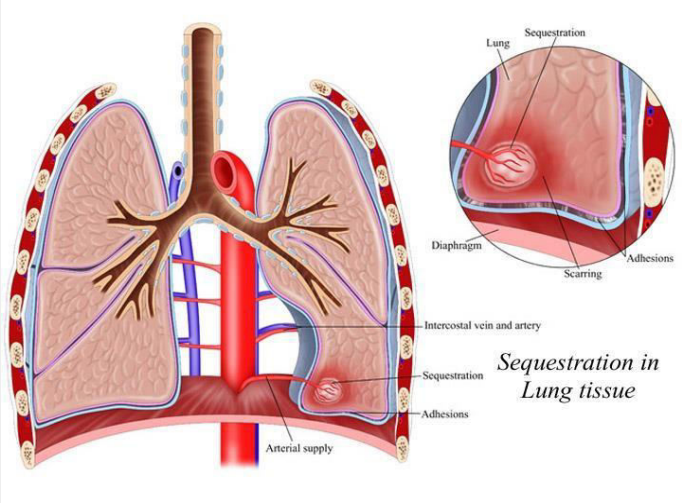
3-Another indication for surgery is to rule out pleuropulmonary blastoma, a malignancy that can appear radiographically similar to type I CPAM.

Pulmonary Sequestration (accessory lung)

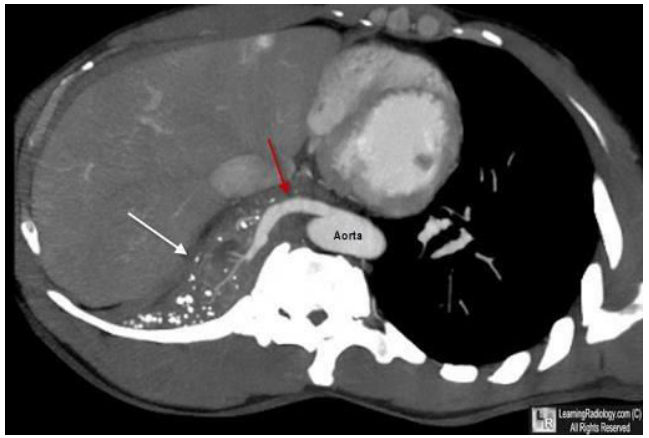
- Cystic-solid mass of nonfunctioning lung tissue
- Is a space-occupying lesion
- completely separated from airways and receives its arterial supply from the systemic arteries.
- Usually in the left lower chest
- Gastric or pancreatic tissue may be found.
- **Complications:**

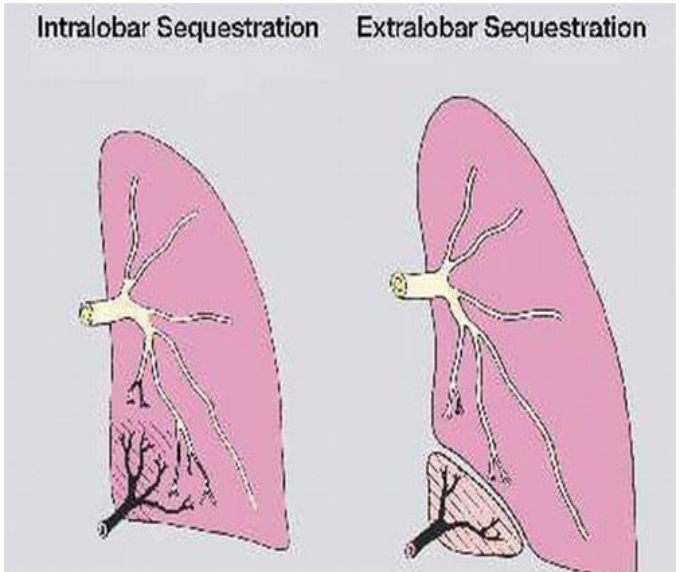
-Hemorrhage

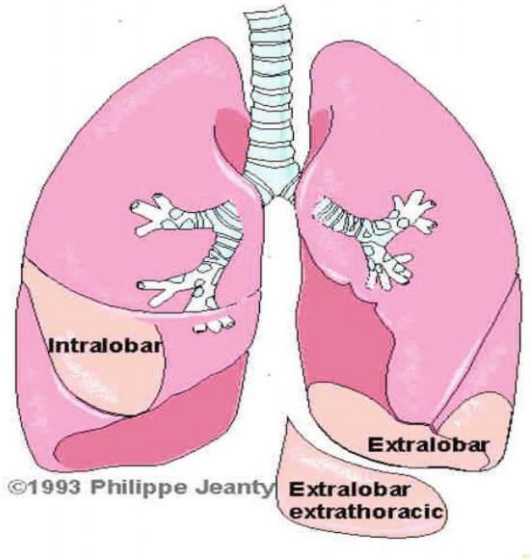
-Chronic infection



CLINICAL MANIFESTATIONS AND DIAGNOSIS







Physical findings in patients with sequestration include:

- an area of dullness to percussion
- decreased breath sounds over the lesion.
- If infection, crackles may be present.
- A continuous or purely systolic murmur may be heard over the back.
- If findings on routine chest radiographs are consistent with the diagnosis **CT with contrast or MR angiography + U\S**

Intrapulmonary vs Extrapulmonary

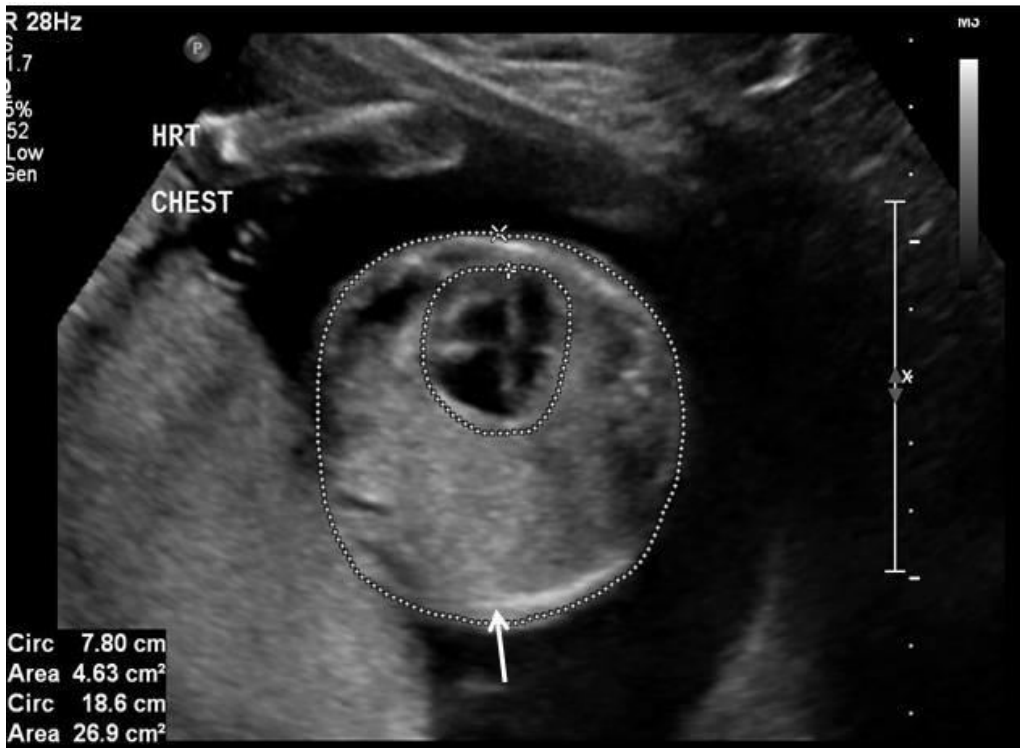
Intrapulmonary

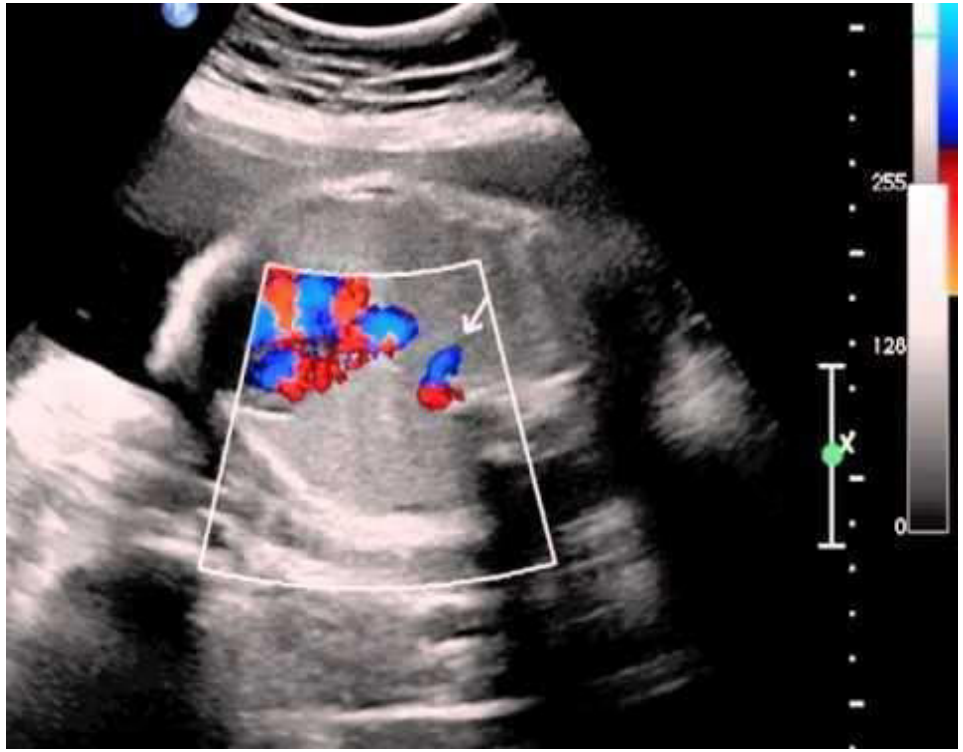
- Found in a lower lobe
- does not have its own pleura.
- Patients usually present with infection.
- In elderly patients, hemoptysis is common.
- There is no difference in the incidence of this lesion in each lung.

Extrapulmonary

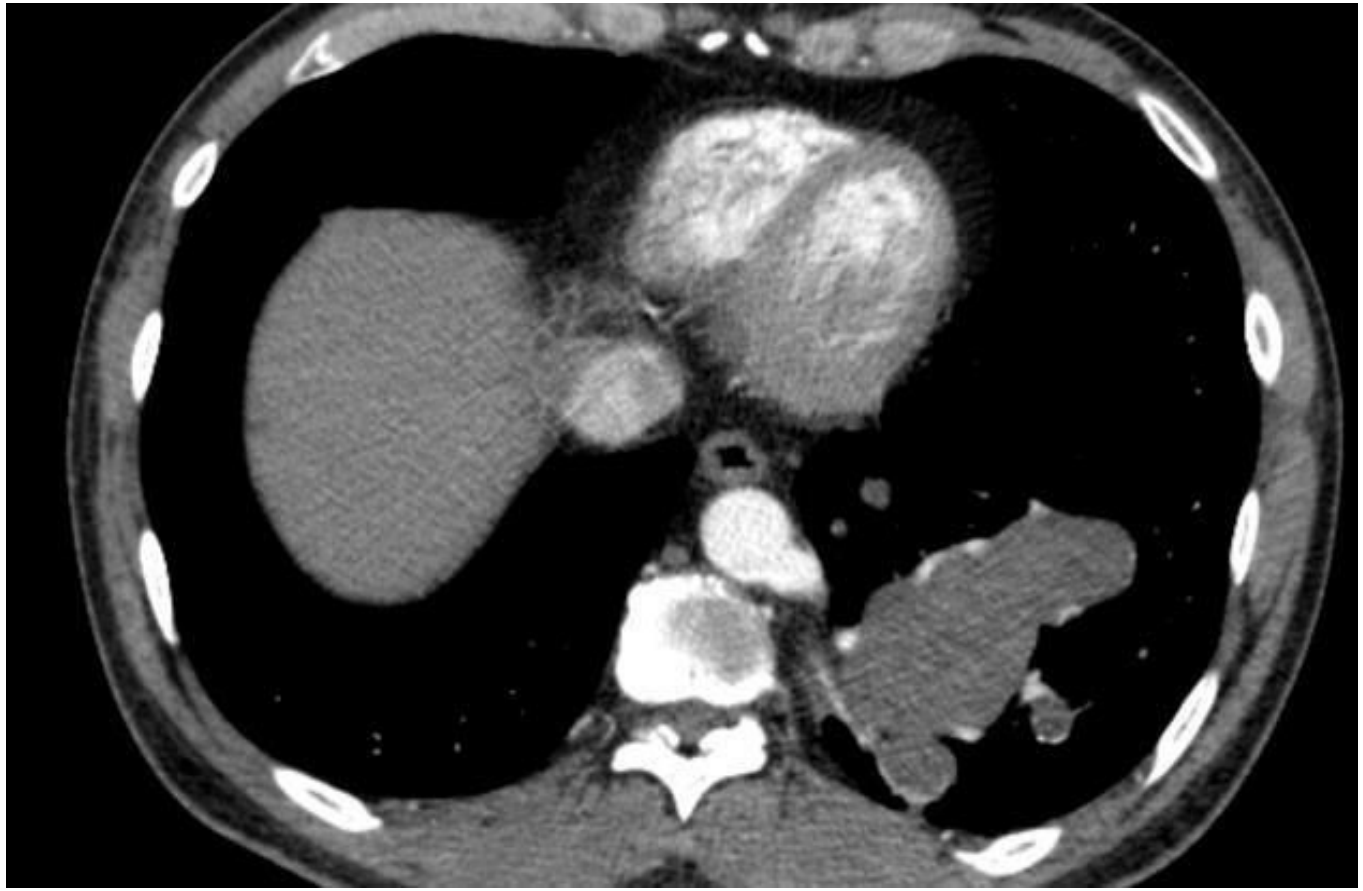
- More common in males
- Left lung always involved
- This lesion is enveloped by a pleural covering and is associated with diaphragmatic hernia and other abnormalities such as **colonic duplication, vertebral abnormalities, and pulmonary hypoplasia.**
- Many of these patients are asymptomatic when the mass is discovered incidentally on a CXR.
- Other patients present with respiratory symptoms or heart failure.
- Subdiaphragmatic extrapulmonary sequestration can manifest as an abdominal mass on prenatal ultrasonography.

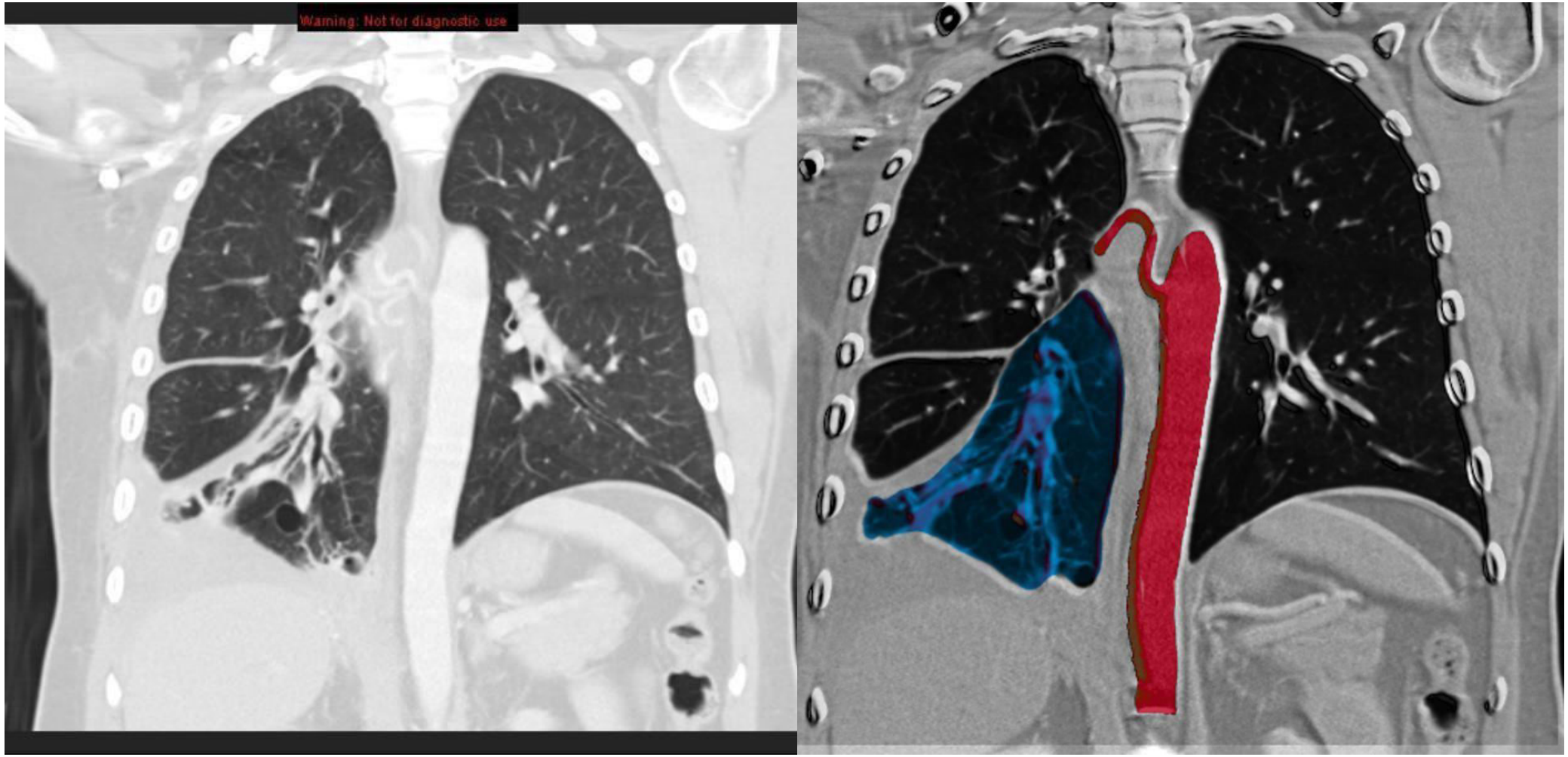
- Diagnosis is usually made prenatally and confirmed on postnatal CT scan





- **Postnatal CT scan**



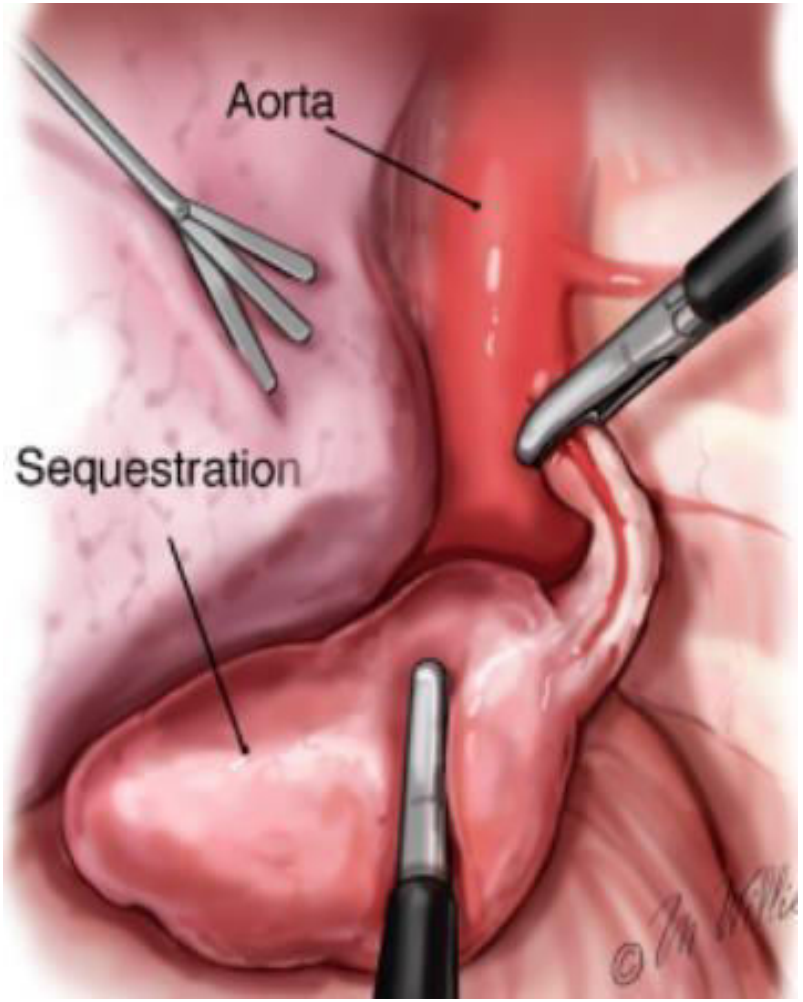

Warning: Not for diagnostic use

Managment

• intrapulmonary is surgical removal of the lesion, a procedure that usually requires excision of the entire involved lobe, but Segmental resection occasionally suffices.

- extrapulmonary sequestration Surgical resection of the involved area is recommended.

- Coil embolization of the feeding artery has also been successful



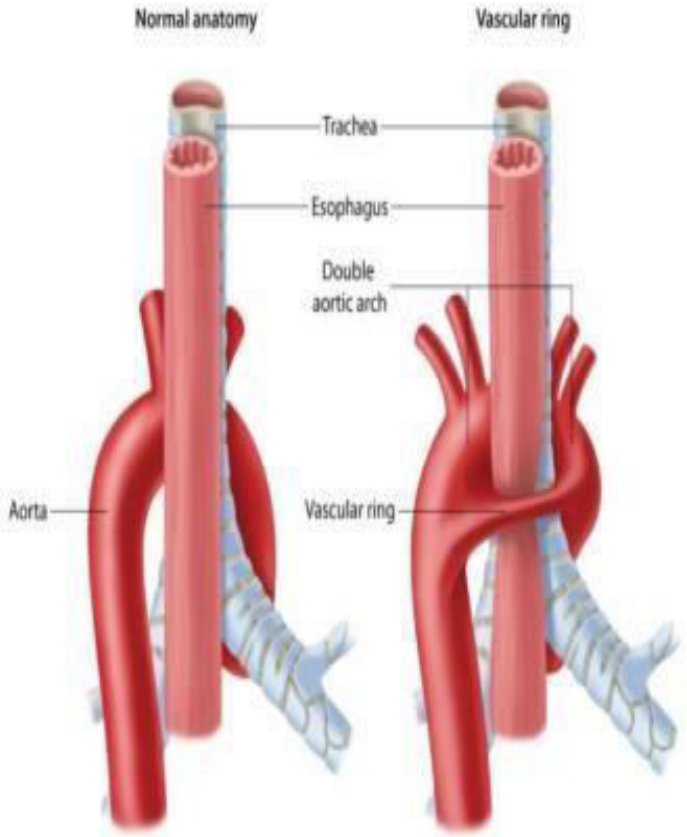
Vascular ring

- Vascular rings, or slings, result from abnormal development of the aortic arch, causing tracheal, bronchial, and/or esophageal compression.
  - It is congenital

- This happens when certain parts of the aorta that normally disappear during fetal development persist abnormally.

- They can be either complete (circumferential around the trachea and/or esophagus), such as a double aortic arch, or incomplete (pulmonary artery sling).

- Vascular rings present in patients age <1 with respiratory (stridor, wheezing, coughing) and/or
esophageal (dysphagia, vomiting, difficulty feeding) symptoms.



Clinical Presentation

STRIDOR	**18 (90%)**
NOISY BREATHING	10 (50%)
BRASSY COUGH	12 (60%)
CHOKING EPISODES	10 (50%)
WHEEZES (misdiagnosed asthma)	15 (75%)
INTERRUPTED FEEDING	13 (65 %)
RECURRENT CHEST INFECTION	8 (40 %)
ATTACKS OF CYANOSIS	9 (45%)
RECURRENT VOMITTING	4 (20%)
APNEA	3 (15 %)

Diagnosis and treatment

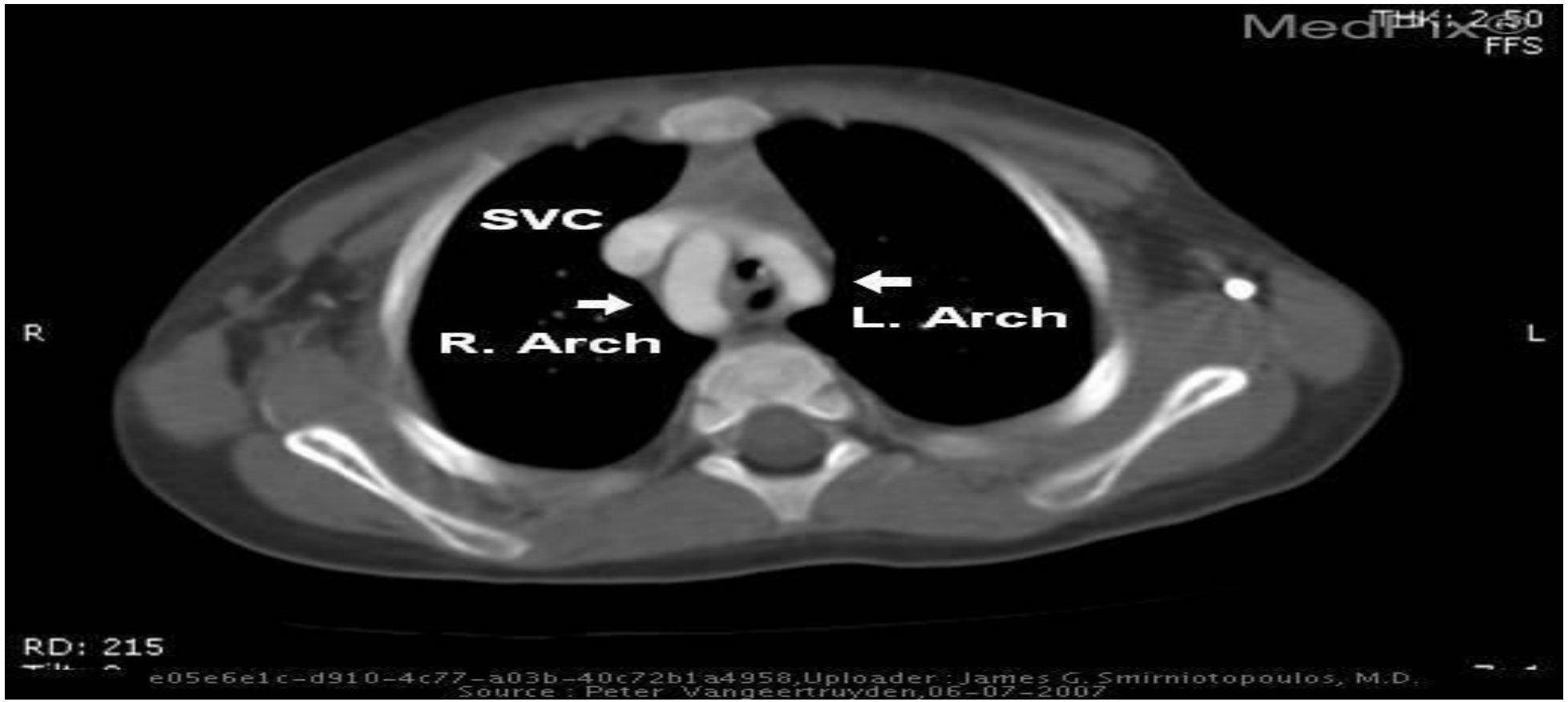
- Diagnosis can be made with a CT scan to delineate the precise anatomy forming the vascular ring and evaluate any associated tracheal abnormalities.

Up to 50% of patients also have a cardiac anomaly (ventricular septal

defect, tetralogy of Fallot)

- Due to possible concurrent cardiac and airway abnormalities, all patients require direct laryngoscopy, bronchoscopy, and echocardiogram.

- Treatment is surgical division of the structures creating the ring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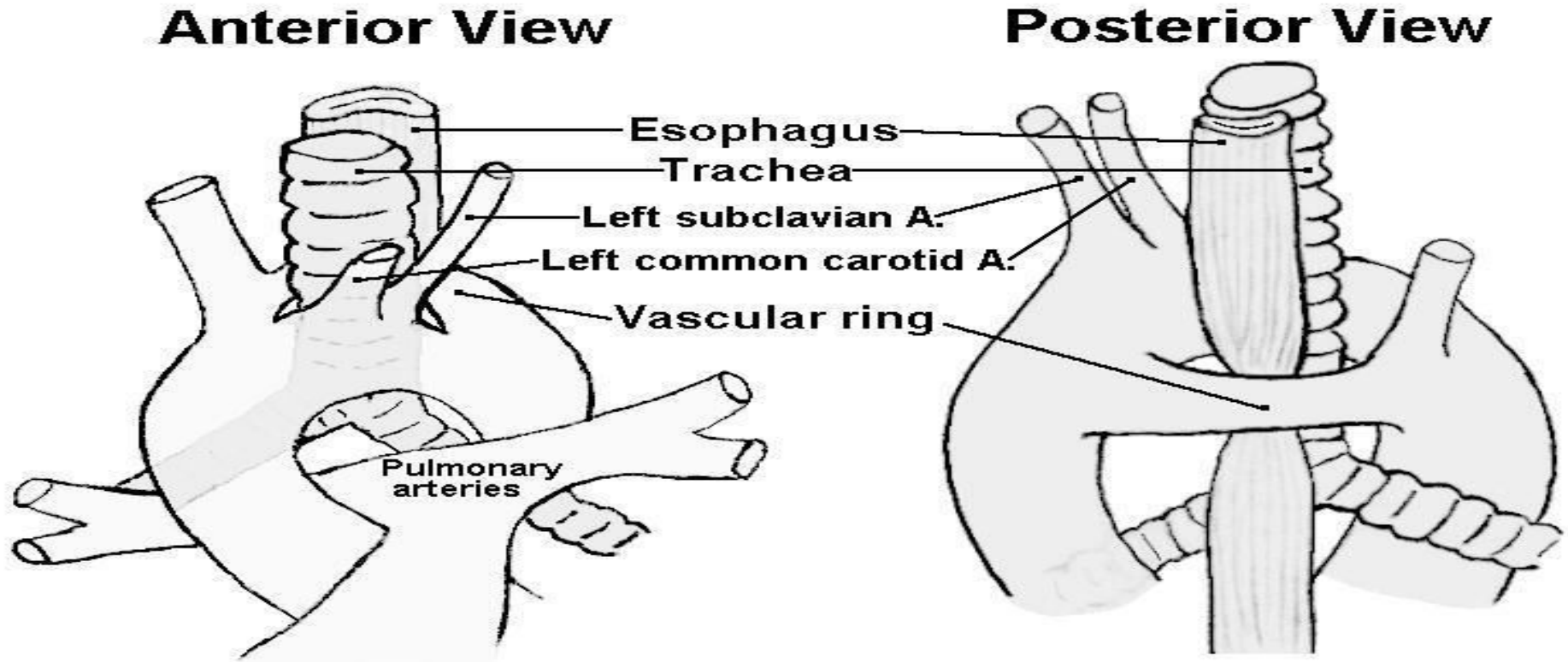
Double Aortic Arch



Primary Ciliary Dyskinesia (Immotile Cilia Syndrome, Kartagener Syndrome)

- Primary ciliary dyskinesia (PCD) is an autosomal recessive genetic condition in which cilia do not function normally. This prevents the clearance of mucous from the lungs, paranasal sinuses and ears. Bacteria and irritants in the mucous lead to frequent respiratory infections .
- Kartagener syndrome is a type of Primary ciliary dyskinesia associated with a mirror-image orientation of the heart and other internal organs.

Motile cilia, the type of cilia impaired in PCD

- Vigorously beating motile cilia, working together with airway mucus, provide a critical, first-line defense against unwanted particles (debris, pathogens, etc.) in the airways.
- In PCD, inherited genetic mutations alter the structure or function of motile cilia, impairing normal ciliary clearance.
- its prevalence in children with repeated respiratory infections has been estimated to be as high as 5%.

- The functional impairment caused by PCD results in:

- Chronic lung disease leading to respiratory failure (chronic sinopulmonary disease) chronic coughing /need for transplant
- Chronic middle ear and sinus infection
- Hearing loss
- Infertility/subfertility (ectopic pregnancy)
- Increased risk of hydrocephalus and retinitis pigmentosa(inherited blindness)

- Motile cilia activity is also required for organ placement during embryonic development.
- About 50% of people with PCD have organ ‘laterality defects,’ meaning their organs are not in the ‘normal’ place.
- Sometimes the organs are in a complete mirror image arrangement called **‘situs inversus.**
- As unusual as it is to have backwards organs, **situs inversus is not generally dangerous.**
- Sometimes in PCD the organs are neither completely reversed nor completely where they should be. This condition, **called heterotaxy or ‘situs ambiguous’ is potentially life threatening.**

DIAGNOSIS

- diagnosed definitively through examination of lung or sinus tissue obtained from a biopsy. (electron microscopy visualization of ciliary abnormalities)
- Screening for levels of nasal nitric oxide is helpful to identify individuals who may have Primary ciliary dyskinesia and should proceed with a biopsy .
- Early diagnosis is important in order to provide prophylactic treatment to prevent or decrease damage to the respiratory system from recurrent infections.

MANAGEMENT

- **AIRWAY CLEARENCE THERAPY** is used to keep the lung tissue healthy for as long as possible. This therapy may include **routine washing and suctioning of the sinus cavities and ear canals. Antibiotics, bronchodilators, steroids and mucus thinners** are also used to treat Primary ciliary dyskinesia .
- **hearing evaluation** is important for young children and speech therapy and hearing aids may appropriate for children with hearing loss and speech problems.
- **Lung transplantation** is an option for severe, advanced lung disease.

Surgery may be indicated if heart defects are present.

THANK YOU

References

- Nelson textbook
- Nelson essentials
- Kaplan pediatrics
- Medscape