XXV Congreso SEAP-IAP · Zaragoza 2011

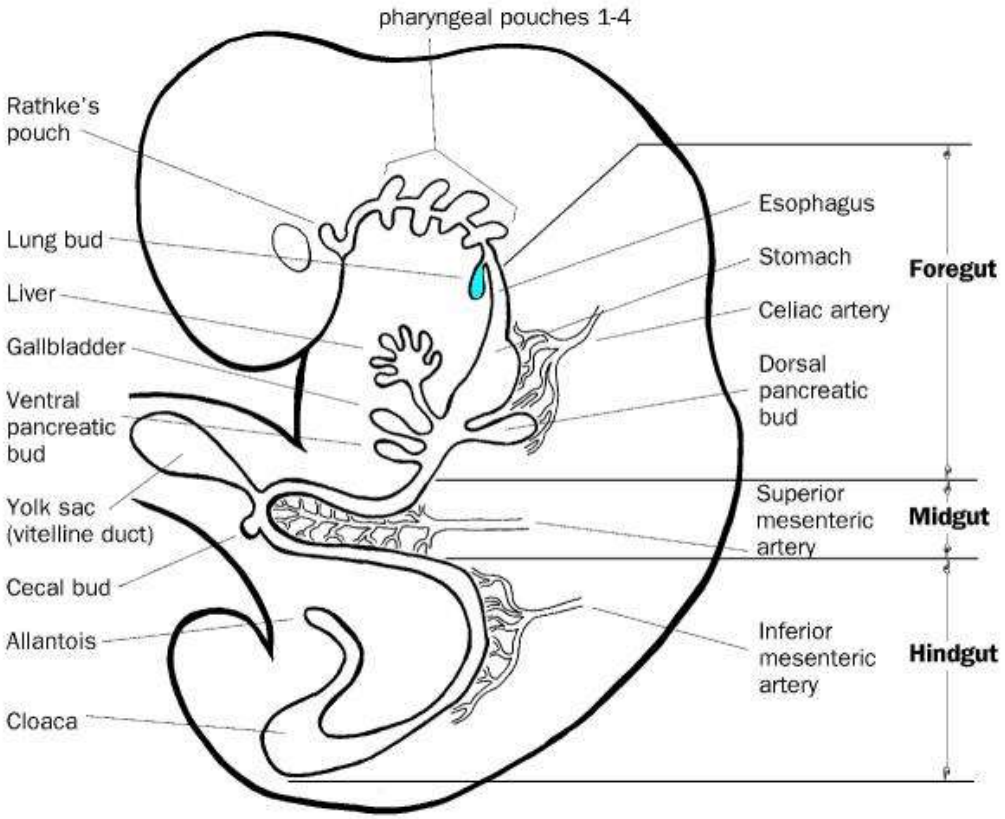
Avances en patología pulmonar pediátrica

MALFORMACIONES DE LA VIA AÉREA Y DEL PULMÓN: MORFOLOGÍA Y PATOGENIA

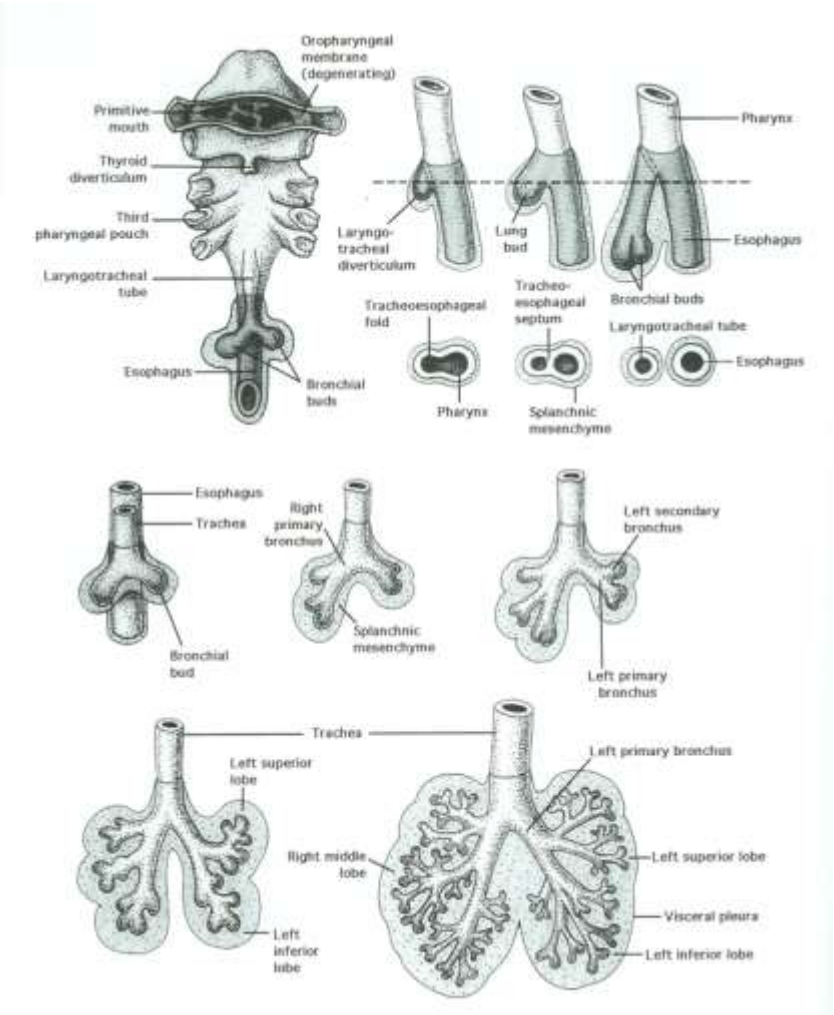
Dra.Carlota Rovira · Hospital Sant Joan de Déu

Barcelona

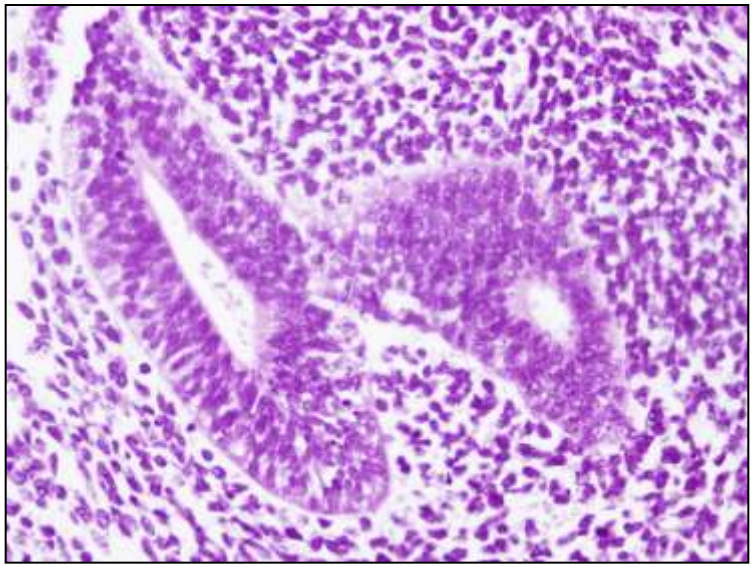
DESARROLLO PULMONAR



PERIODO EMBRIONARIO



- Inicio a 4ª semana.
- 26 días a 6 semanas.
- Desarrollo de la vía aérea principal.



PERIODO PSEUDOGLANDULAR

6-16 SEMANAS

PERIODO ACINAR/CANALICULAR

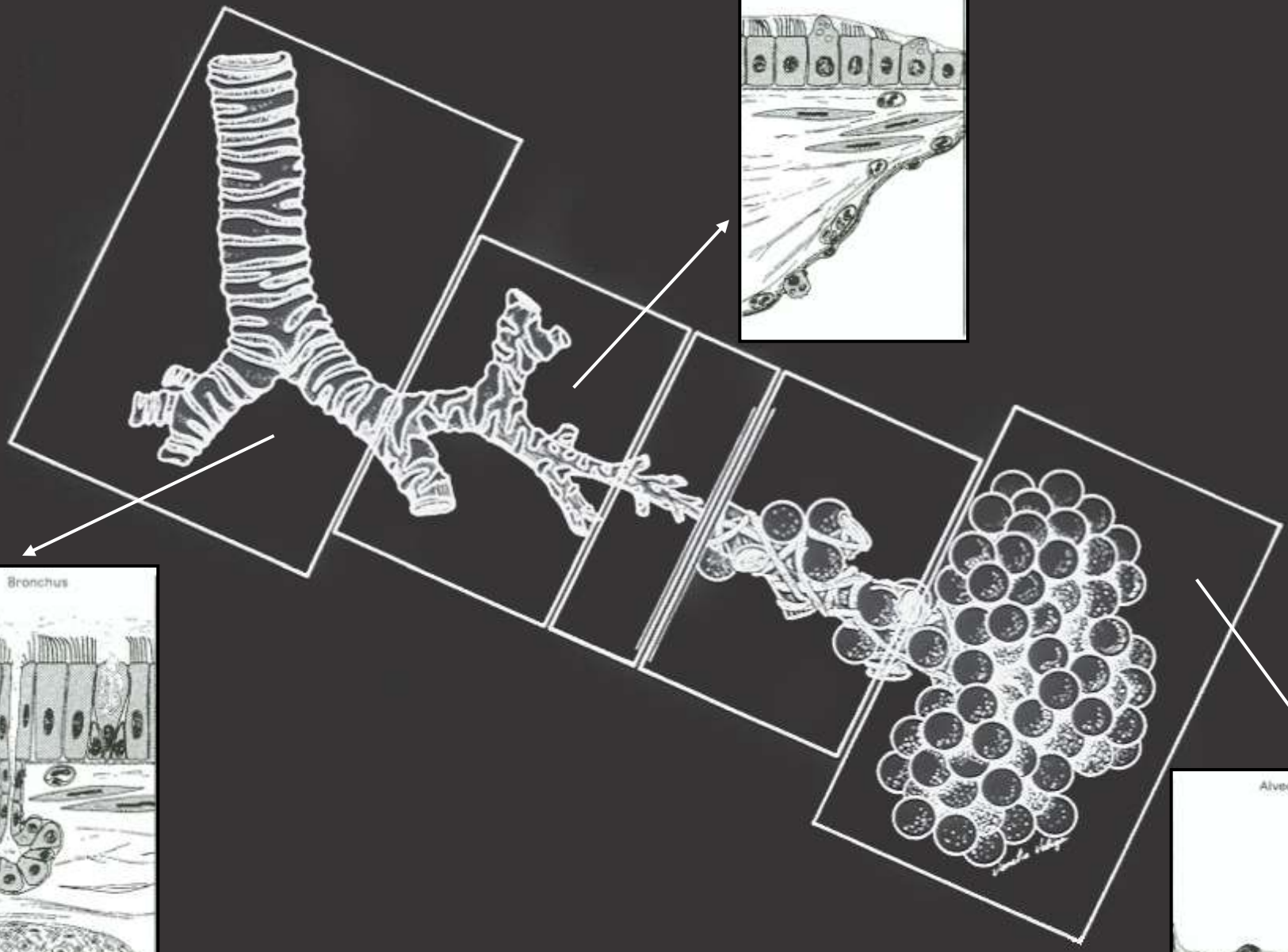
16-28 SEMANAS

PERIODO SACULAR

28-34 SEMANAS

PERIODO ALVEOLAR

DESDE SEM 34 EN ADELANTE


Bronchiolus
Bronchus
Alveolus

MALFORMACIONES

- Definición y clasificación problemática y compleja.
- Lesiones morfológicamente similares reciben distinta nomenclatura.
- Aplicación de la misma nomenclatura a distintas lesiones.
- Diferencias nomenclatura entre especialidades (patólogo, radiólogo, cirujano)
- Patrones histológicos que se solapan.

CLASIFICACIÓN

Malformaciones difusas, base genética: deficit surfactante, displasia alveolar capilar, displasia acinar.

Malformaciones congénitas locales:

- Malformación congénita vía aérea pulmonar (MCVAP).
- Secuestro pulmonar: intralobar (SIL), extralobar (ELS).
- Enfisema lobar (EL).
- Quiste broncogénico (QB).

Anomalías del crecimiento pulmonar: hipoplasia, hiperplasia pulmonar.

PATOGENIA

- Estudio prenatal US rutinaria: origen prenatal y permite seguimiento.
- Factor común: obstrucción de la VA intraútero

"secuencia de atresia bronquial"

» *Grado*

» *Nivel*

» *Tiempo*

PATOGENIA

- Atresia bronquial en gestación temprana (6-16 sem): MCVAP.
- Atresia bronquial posterior (16-18 sem): EL.

Table 3. Classification of Congenital Lung Malformations

- Bronchopulmonary malformation
  - Bronchogenic cyst (noncommunicating bronchopulmonary foregut malformation)
  - Bronchial atresia
    - Isolated
    - With systemic arterial/venous connection (intralobar sequestration)
    - With connection to gastrointestinal tract (intralobar sequestration/complex or communicating bronchopulmonary foregut malformation)
    - Systemic arterial connection to normal lung
  - Cystic adenomatoid malformation, large cyst type (Stocker type 1)
    - Isolated
    - With systemic arterial/venous connection (hybrid lesion/intralobar sequestration)
  - Cystic adenomatoid malformation, small cyst type (Stocker type 2)
    - Isolated
    - With systemic arterial/venous connection (hybrid lesion/intralobar sequestration)
  - Extralobar sequestration
    - Without connection to gastrointestinal tract (with/without CAM, small cyst type)
    - With connection to gastrointestinal tract (complex/communicating bronchopulmonary foregut malformation)
- Pulmonary hyperplasia and related lesions
  - Laryngeal atresia
  - Solid or adenomatoid cystic adenomatoid malformation (Stocker type 3)
  - Polyalveolar lobe
- Congenital lobar overinflation
- Other cystic lesions
  - Lymphatic/lymphangiomatous cysts
  - Enteric cysts
  - Mesothelial cysts
  - Simple parenchymal cysts
  - Low-grade cystic pleuropulmonary blastoma

Langston C. New concepts in the pathology of congenital lung malformations. Semin Pediatr Surg 2003;12:17-37.

Seminars in Pediatric Surgery (2010) 19, 171-179

SEMINARS IN PEDIATRIC SURGERY

Congenital lung lesions—underlying molecular mechanisms

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Genes influyentes en el desarrollo pulmonar:

Hoxb-5

KGF

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MALFORMACIONES DE LA VIA AEREA PULMONAR (MCVAP)

- Lesiones que representan una aberración en el proceso de ramificación en distintos estadios del desarrollo pulmonar fetal

- Asincronía entre la interacción mesénquima-epitelio debido a alteraciones de diferentes factores de crecimiento y proliferación.

MALFORMACIONES DE LA VIA AEREA PULMONAR (MCVAP)

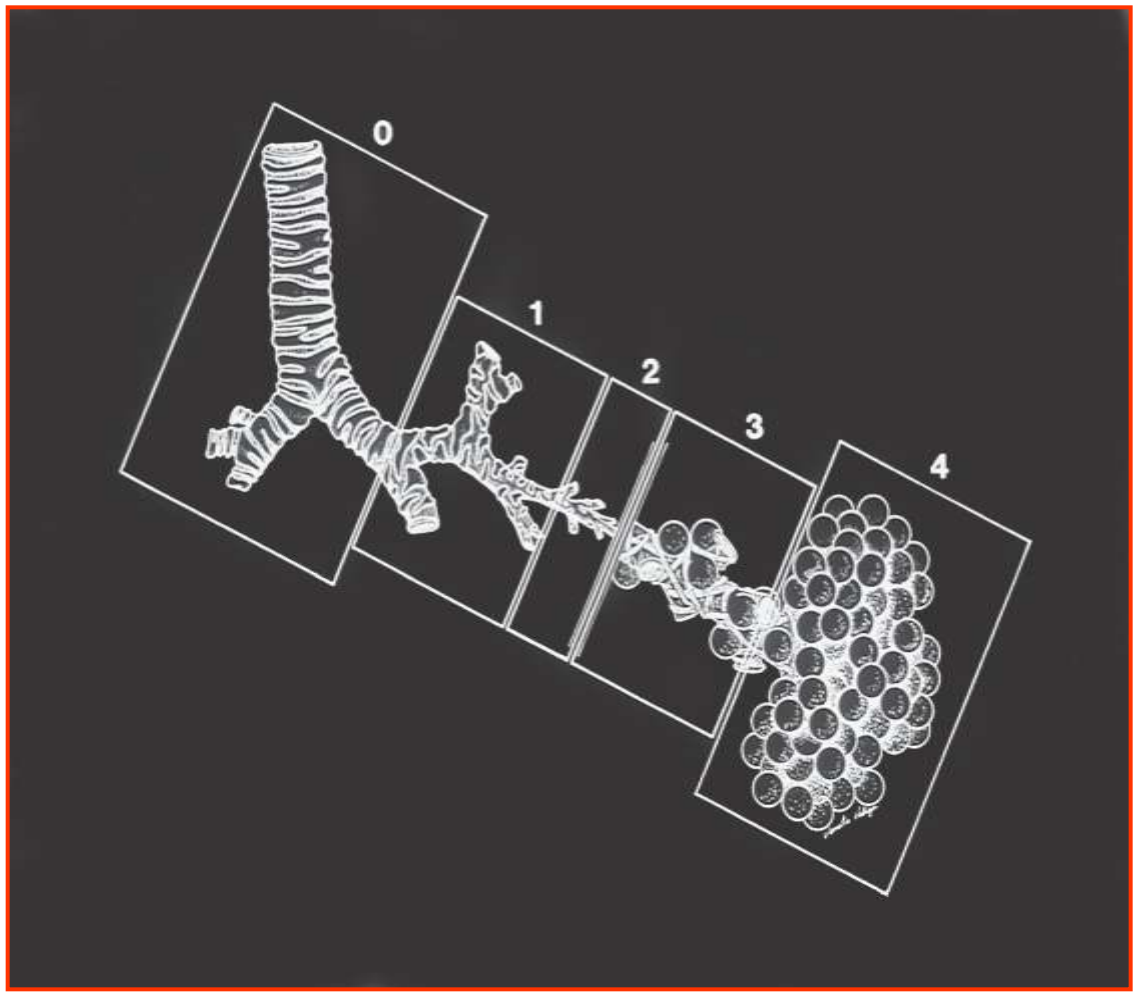
- Chin and Tang.1949: malformación congénita adenomatoidea.
- Stocker 1977: malformaciones congénitas quísticas adenomatoideas (MACC)
  - I: quistes grandes con epitelio respiratorio con células mucogénicas.
  - II: estructuras bronquiolares. Asociación otras anomalias.
  - III: forma adenomatoidea.

MALFORMACIONES DE LA VIA AEREA PULMONAR (MCVAP)

Stocker 2002 (MCVAP/CPAM): 5 tipos basados en la similitud de la lesión hamartomatosa con la porción del árbol traqueobronquial donde presuntamente se origina:

- Tipo 0: disgenesia acinar/displasia
- Tipo 1: quiste grande
- Tipo 2: quiste pequeño
- Tipo 3: adenomatoidea
- Tipo 4: periférica

MCVAP



0: traqueobronquial

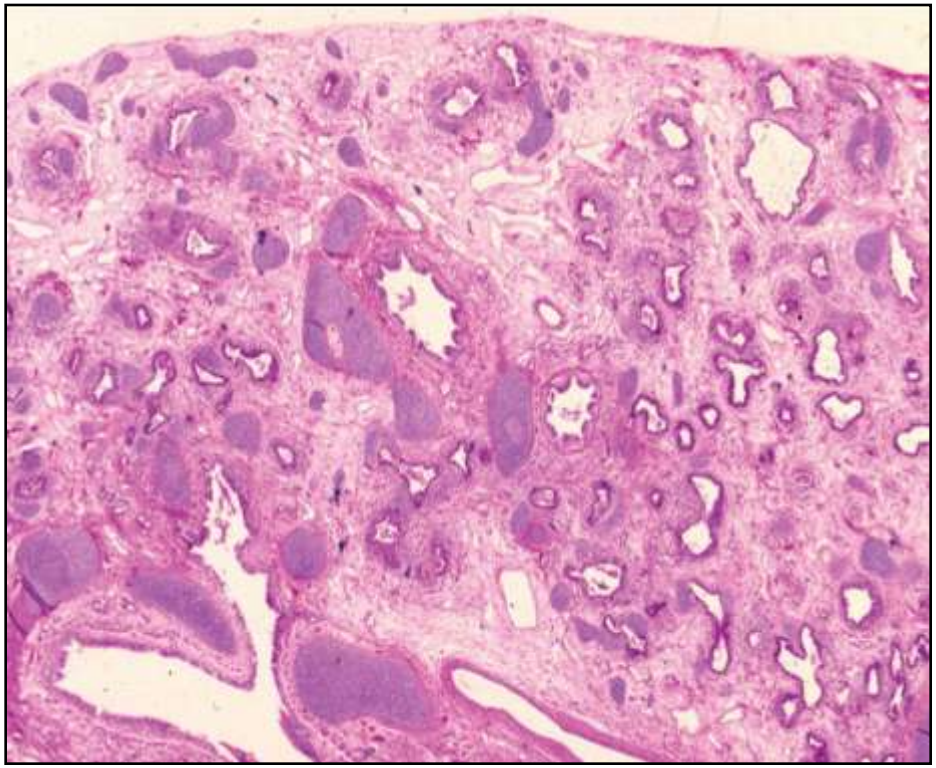
1: bronquial/bronquiolar

2: bronquiolar

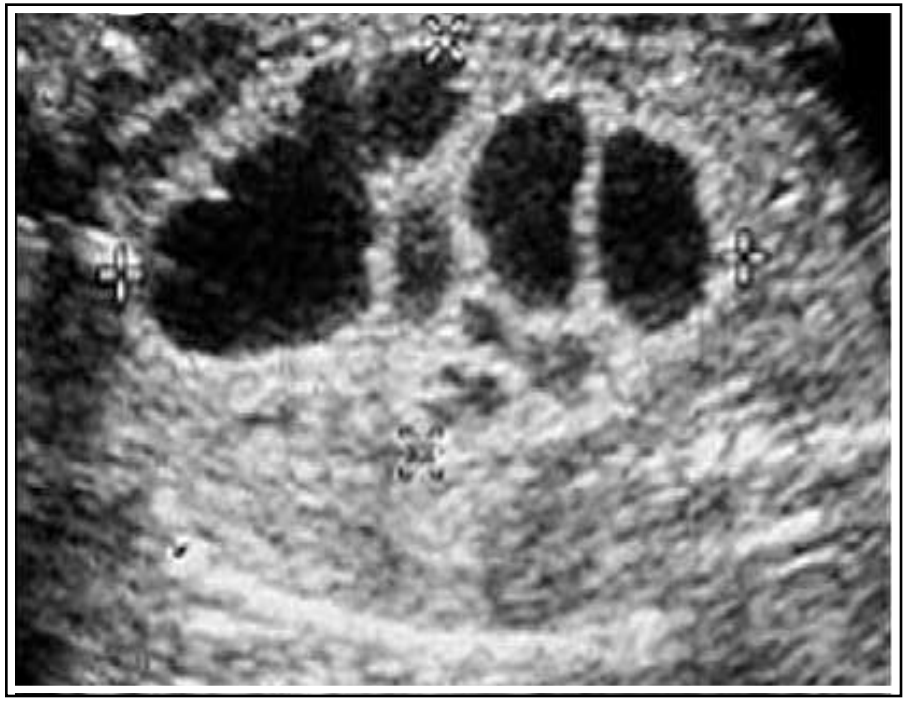
3: bronquiolar/ducto alveolar

4: acinar Distal

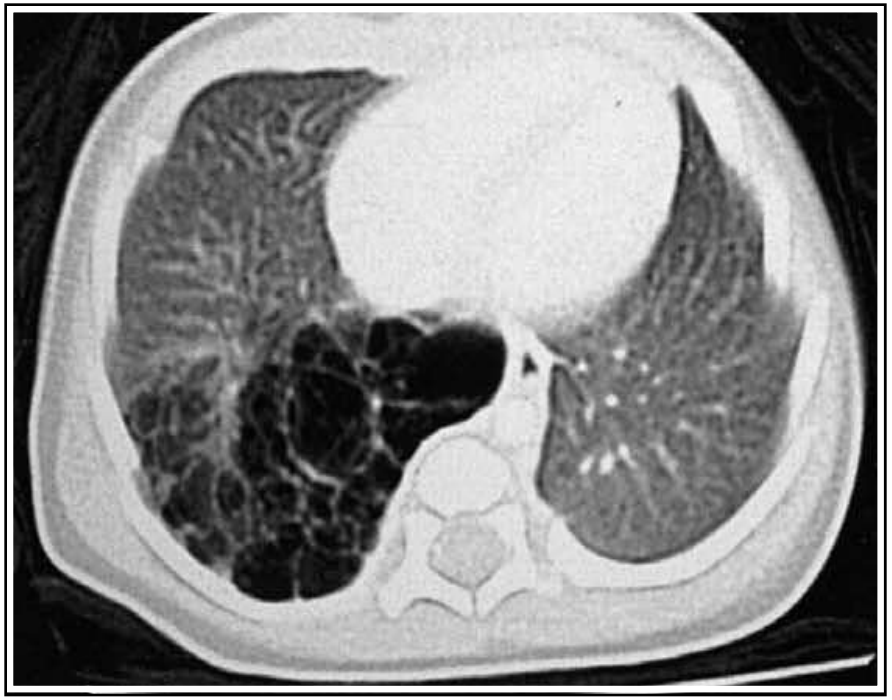
MCVAP 0 - DISPLASIA ACINAR



MCVAP 1

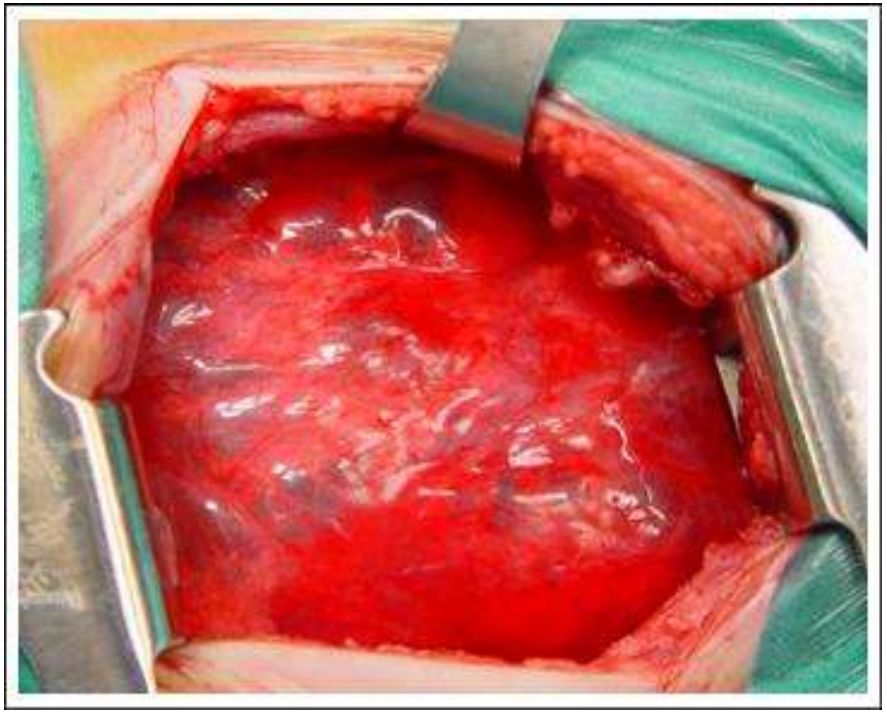


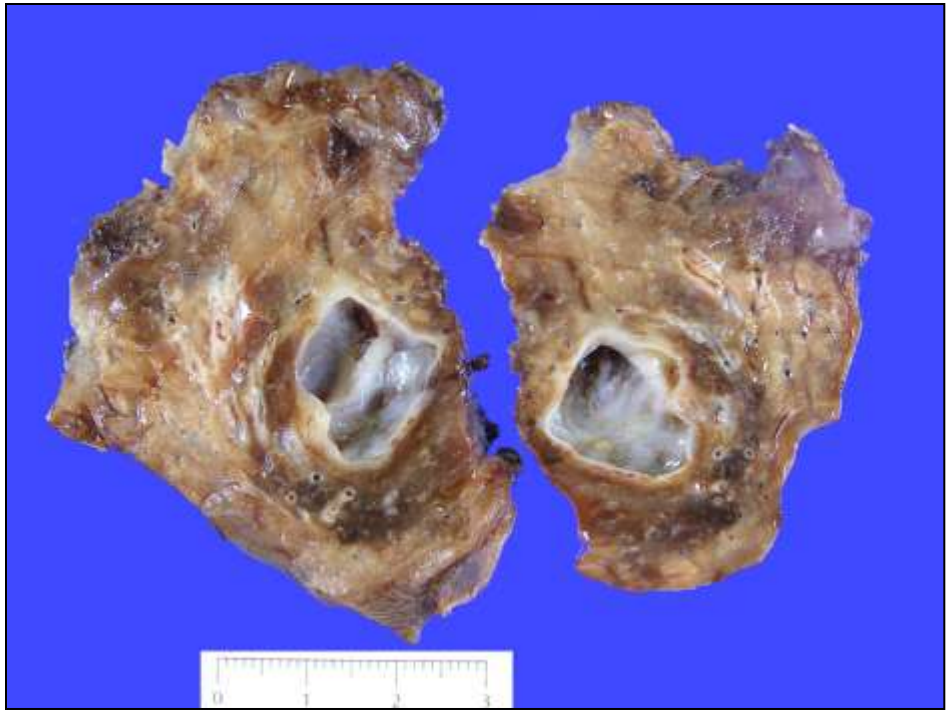
ECO PRENATAL

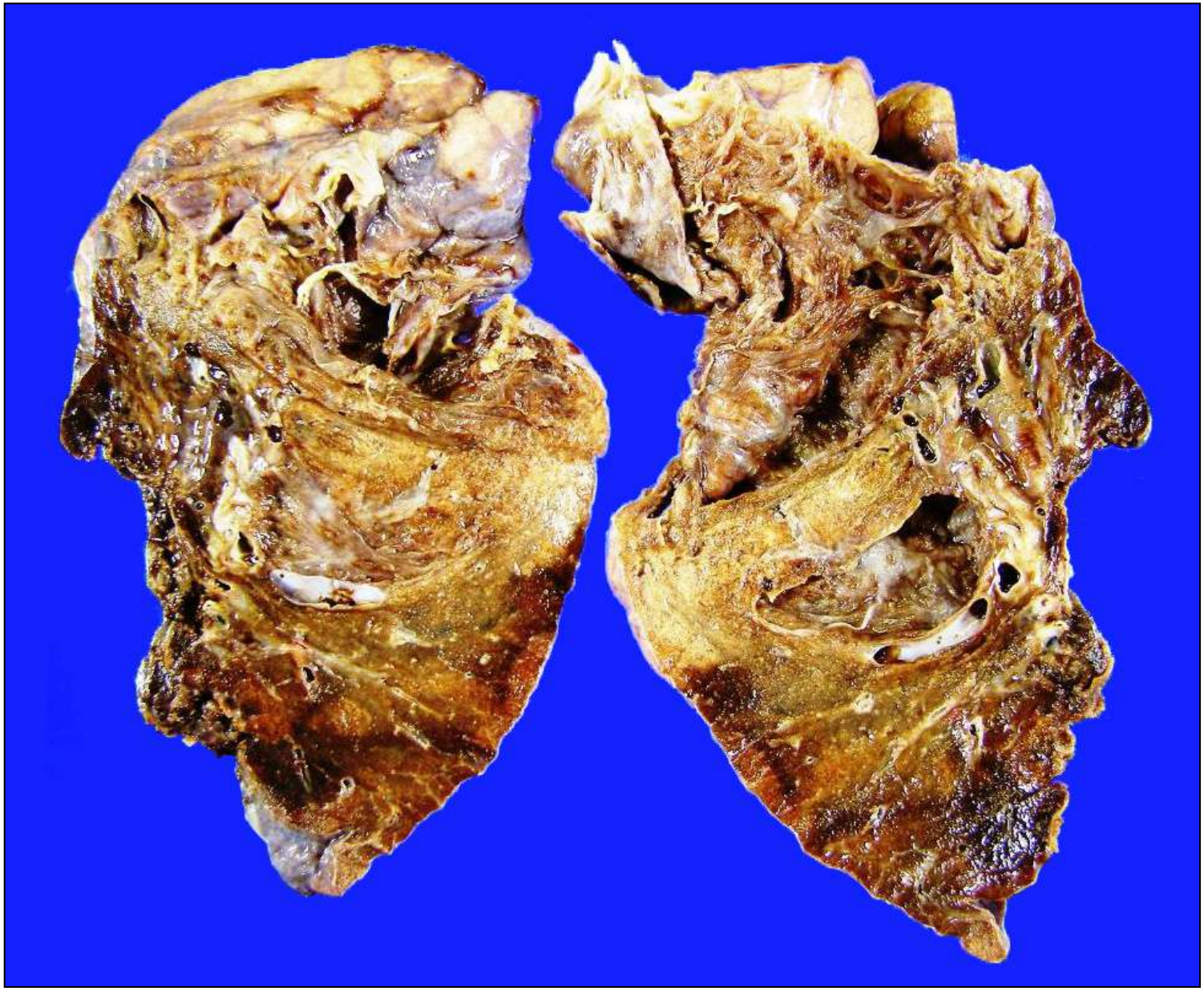


TAC POSTNATAL

MCVAP 1







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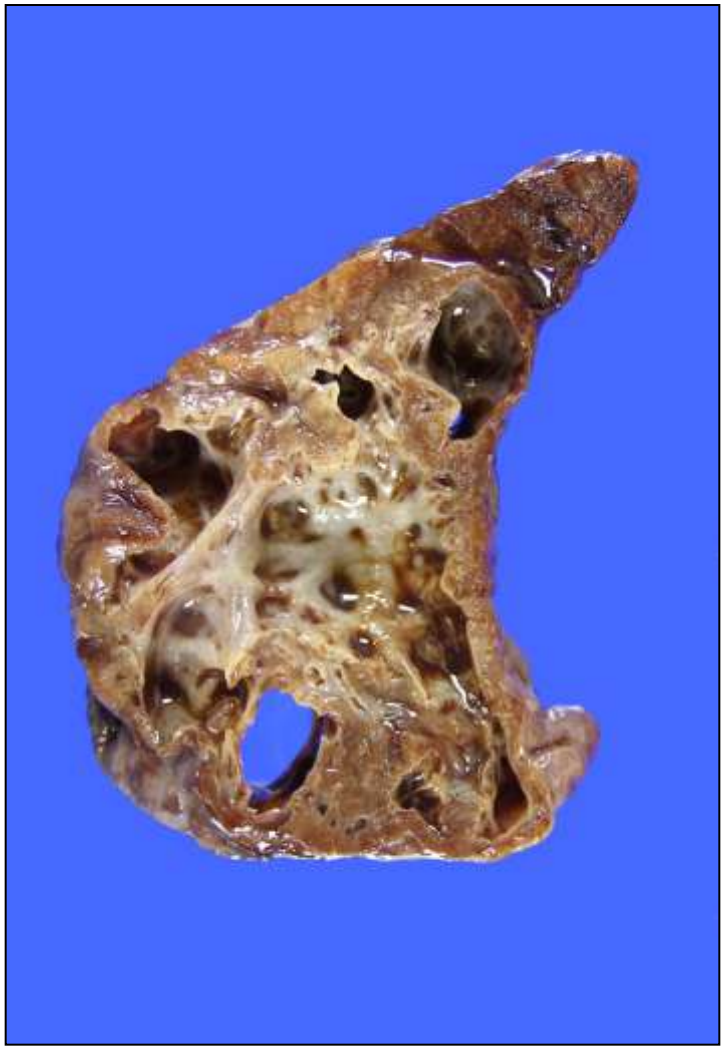

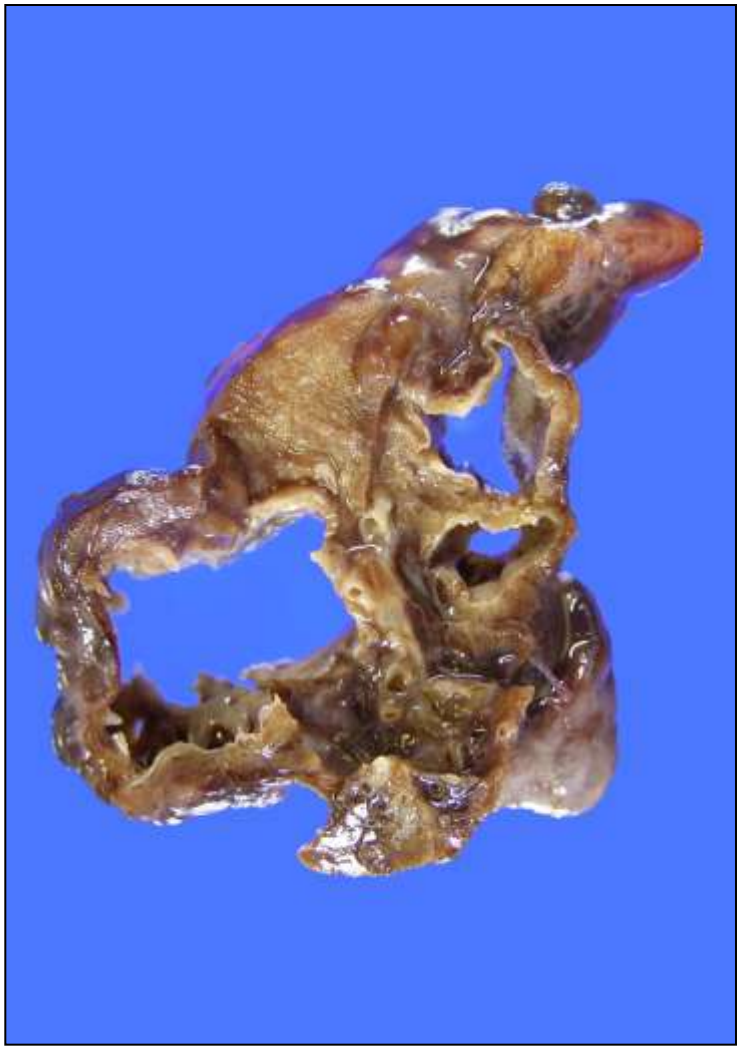

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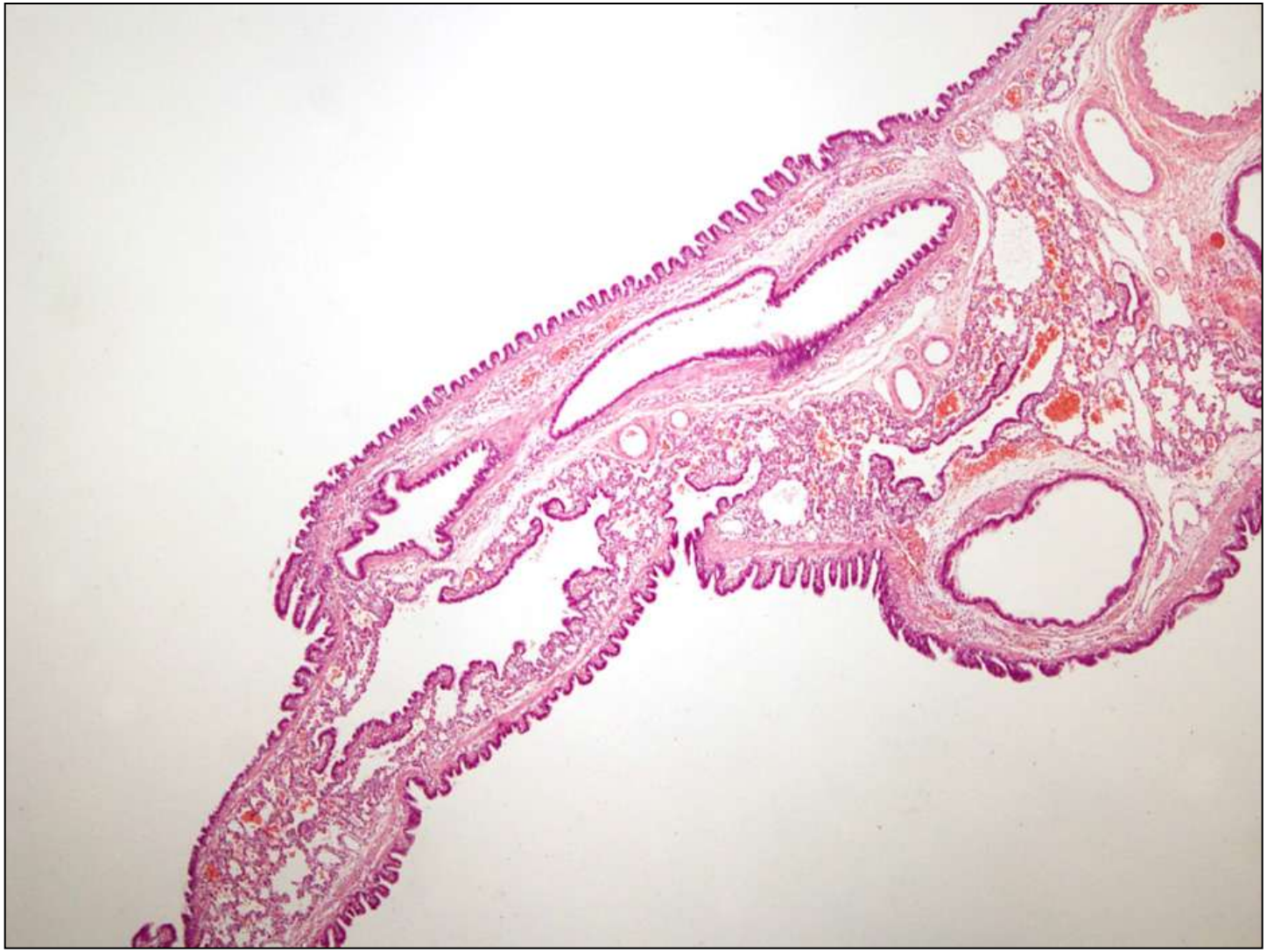

MCVAP 1

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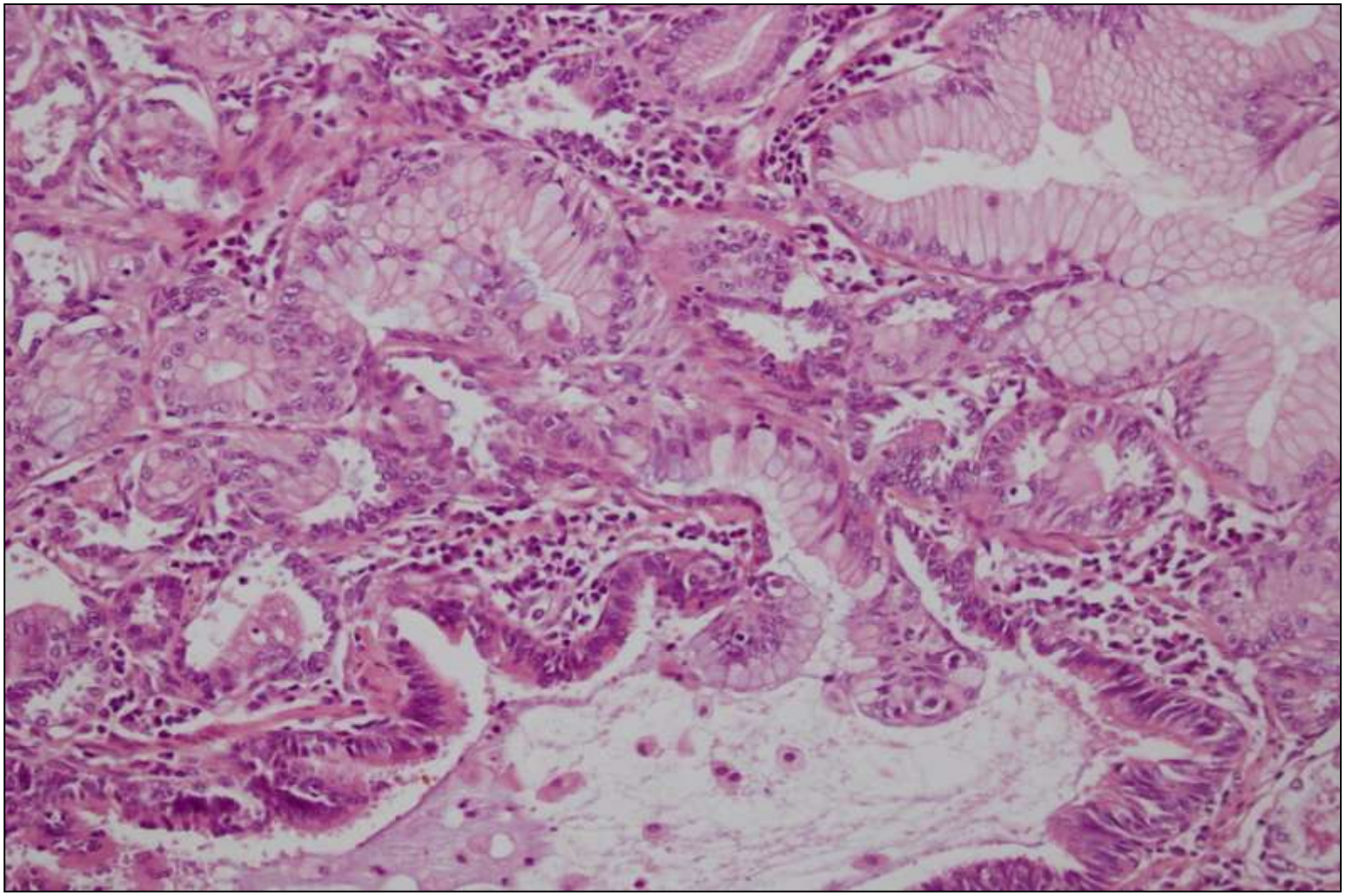

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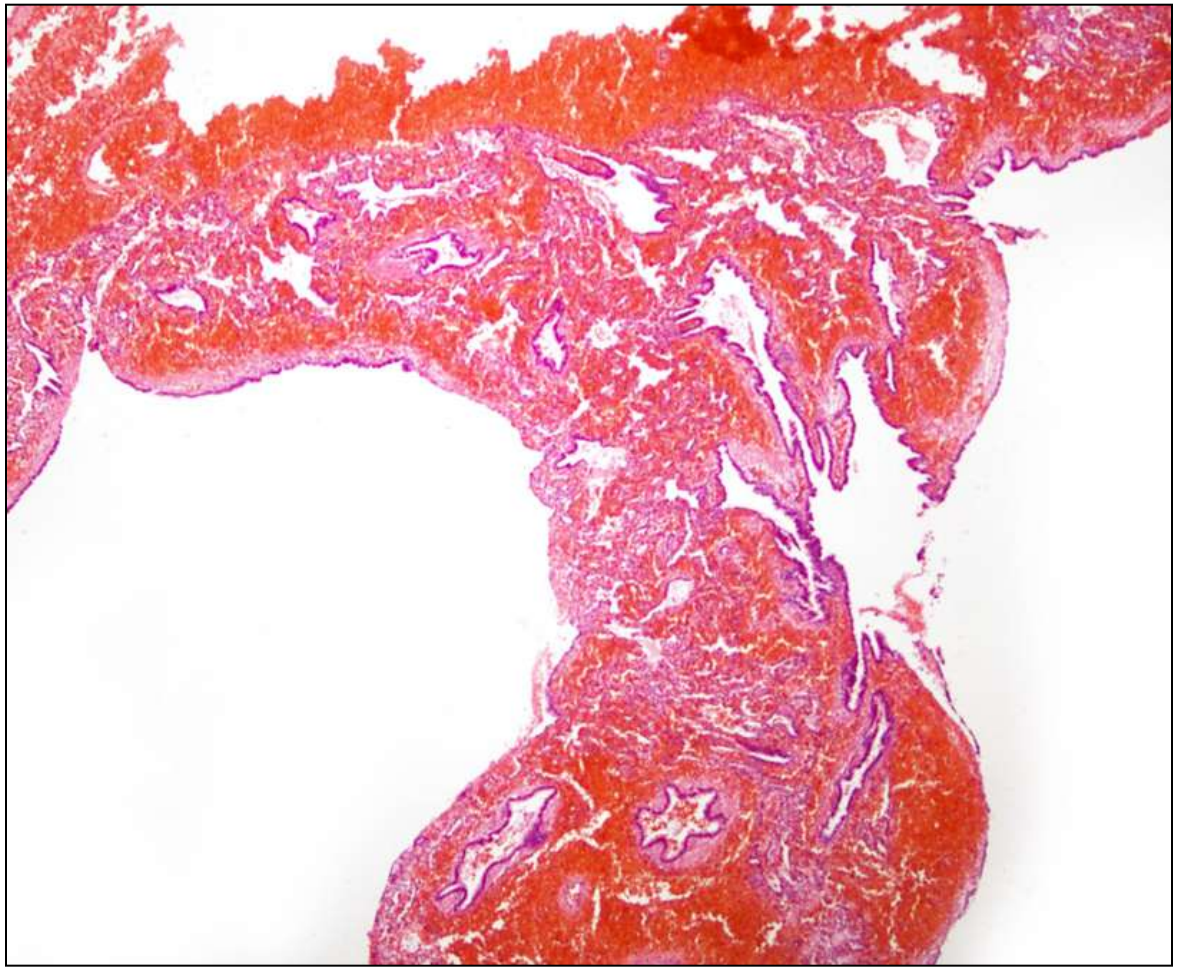

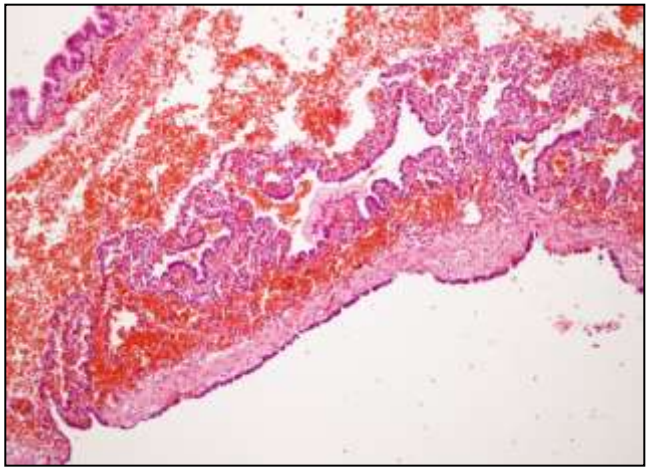

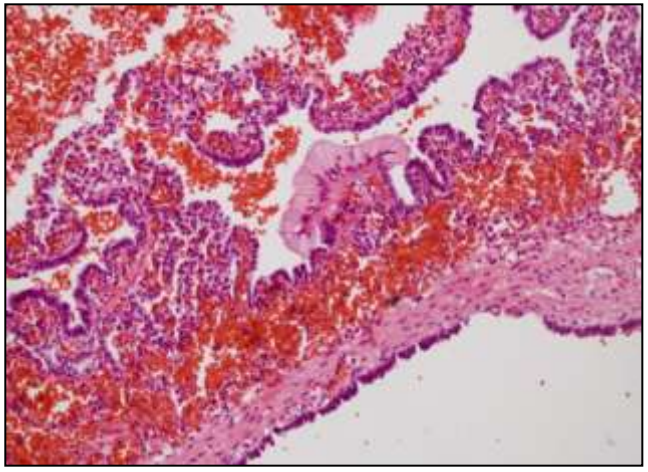

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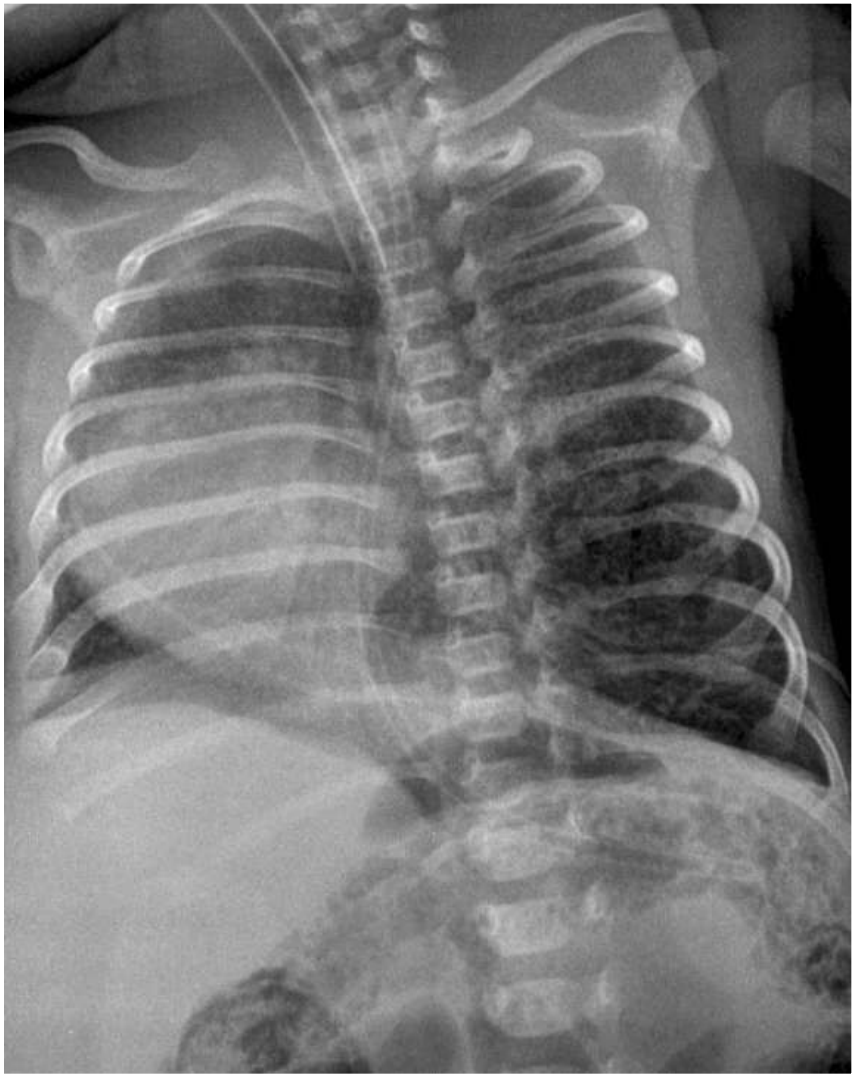

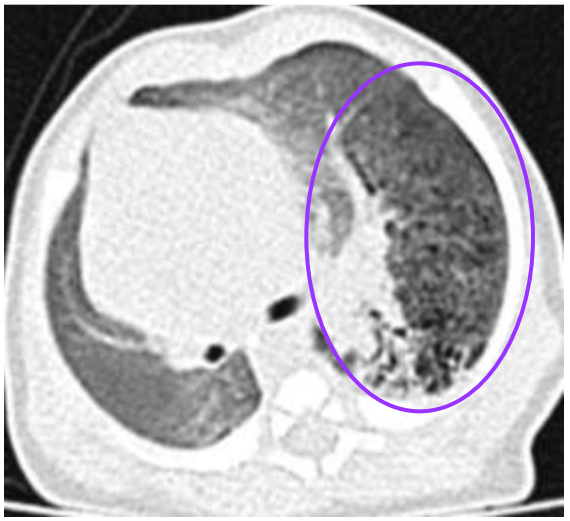

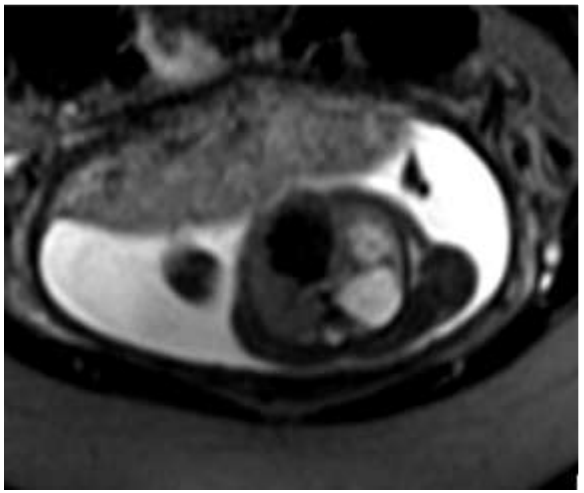

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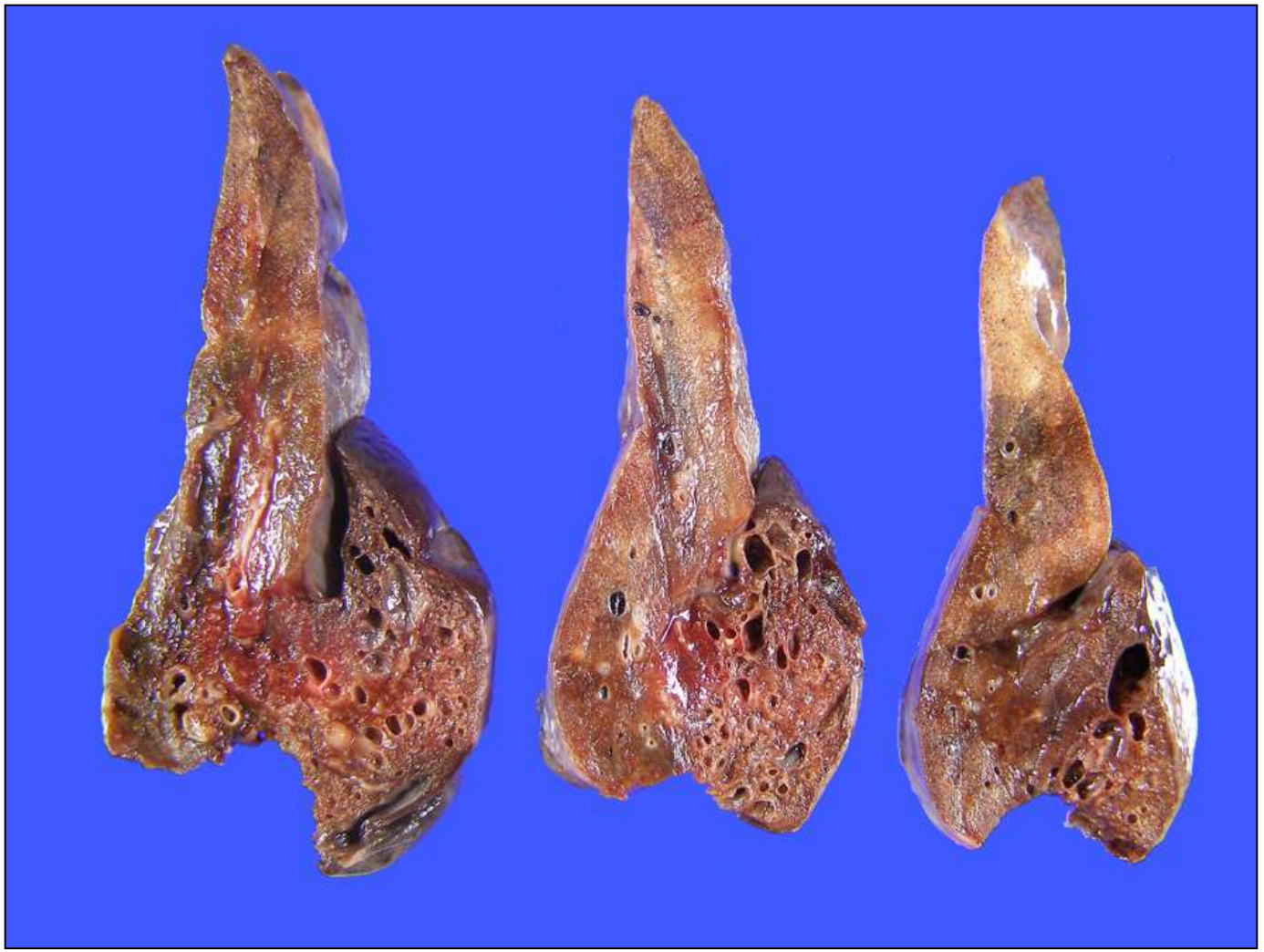

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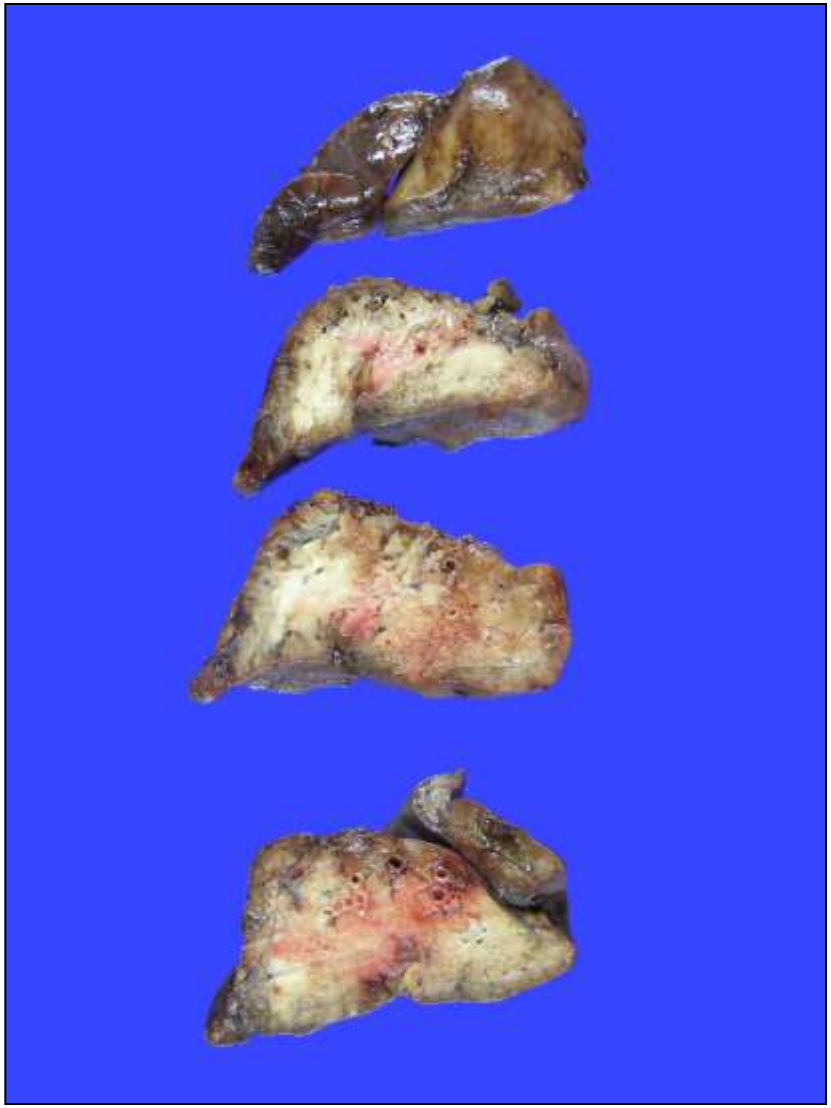

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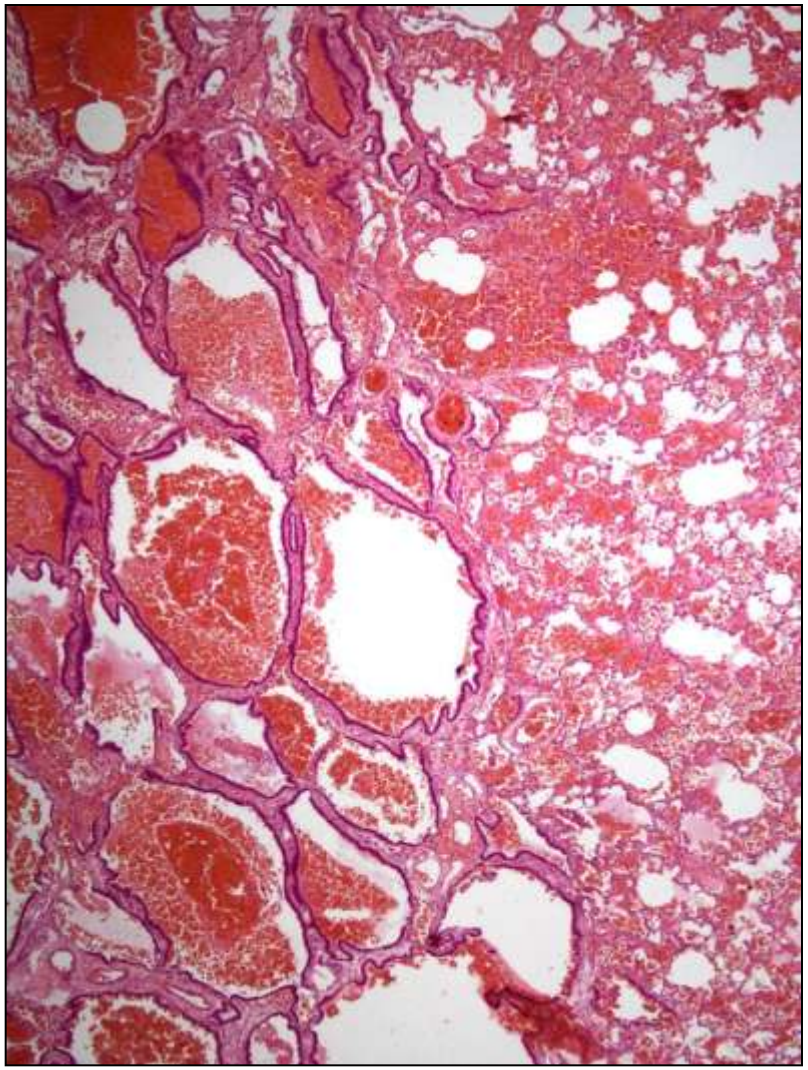

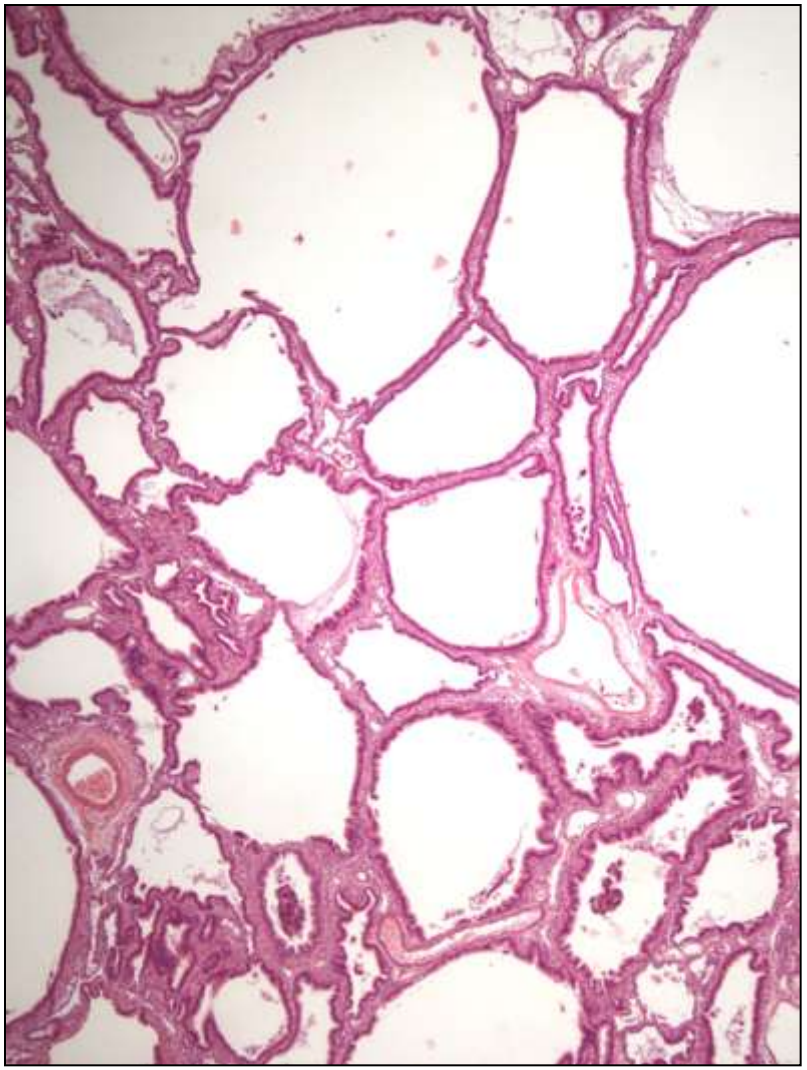

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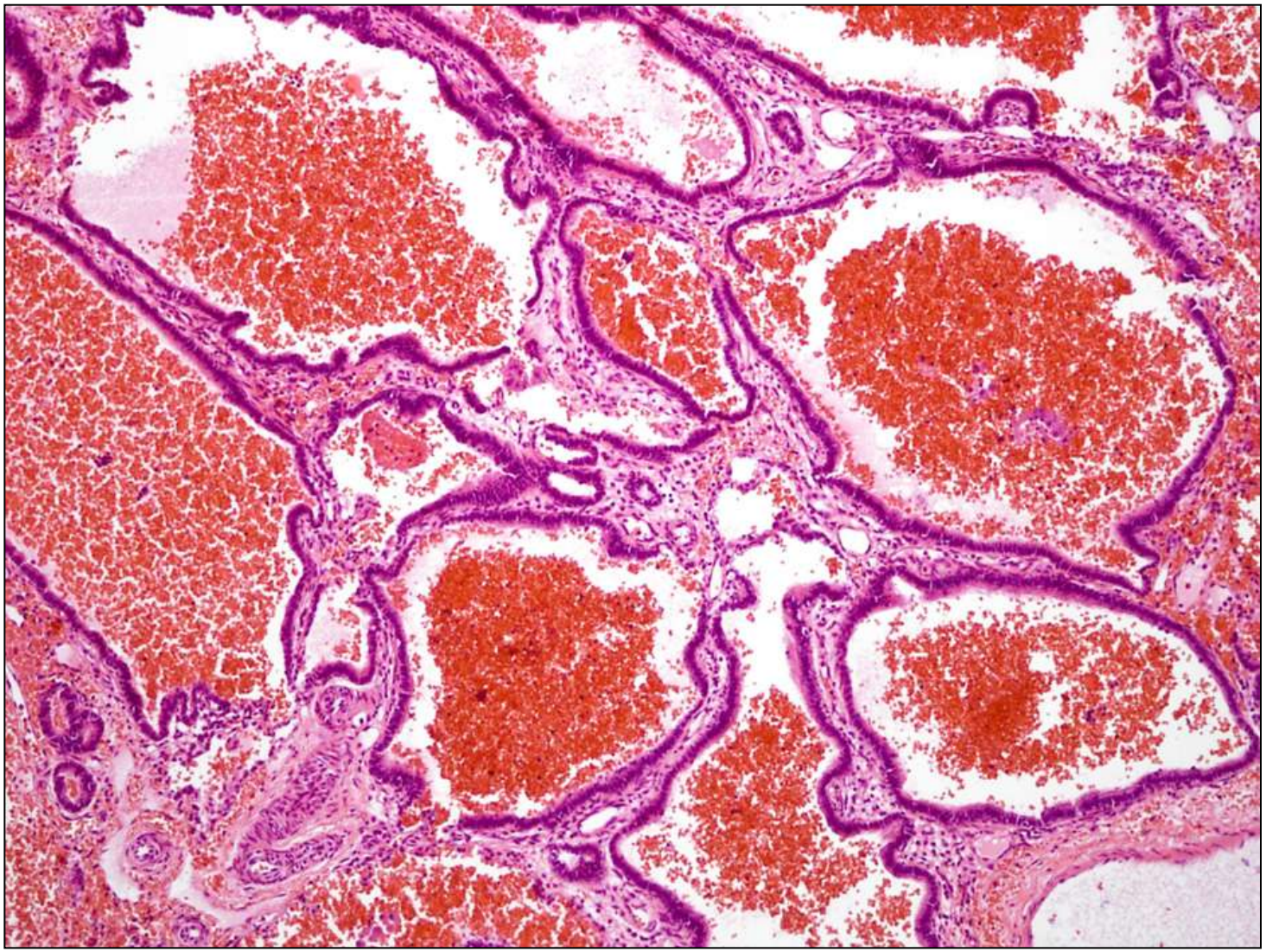

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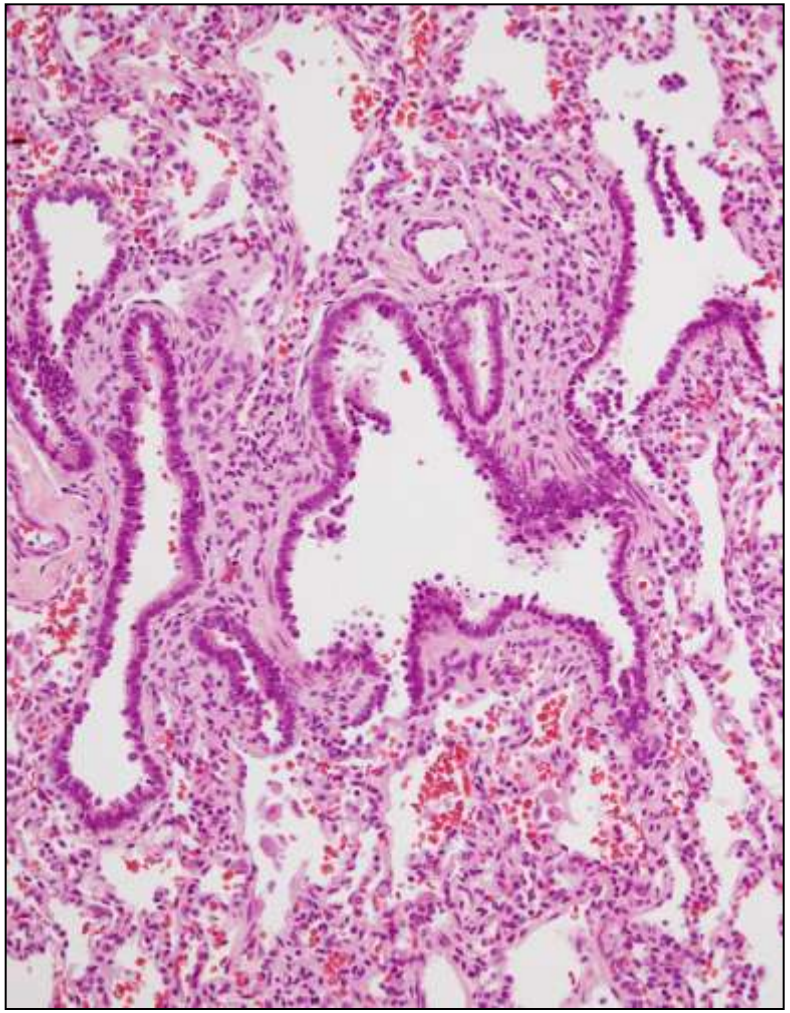
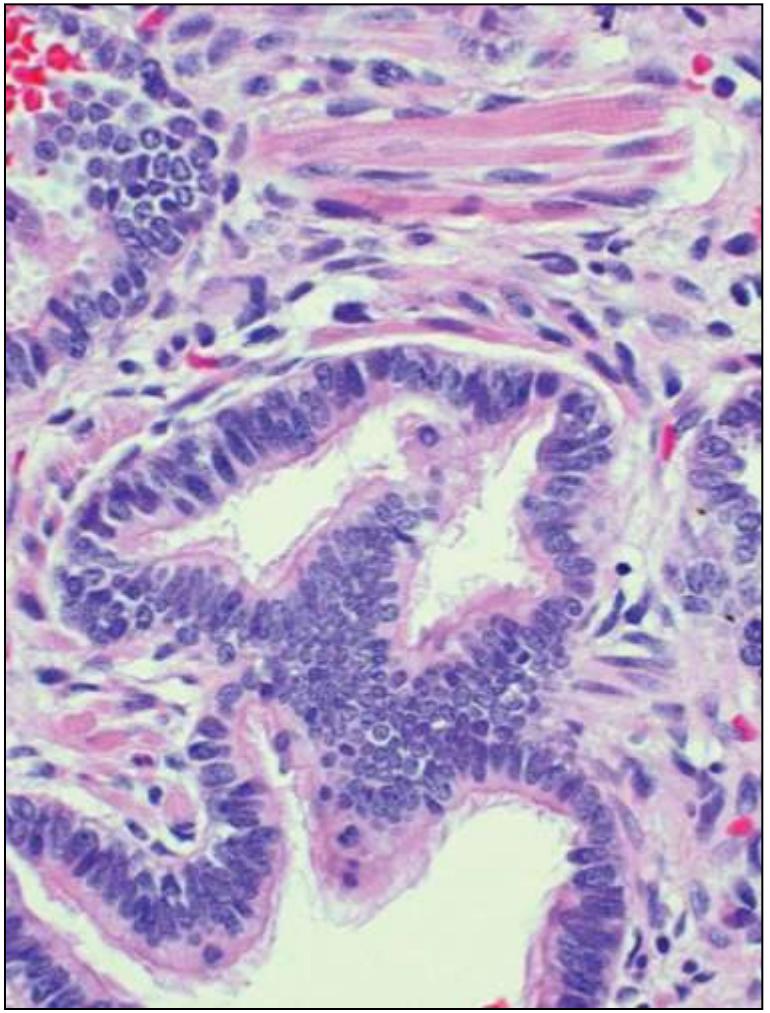
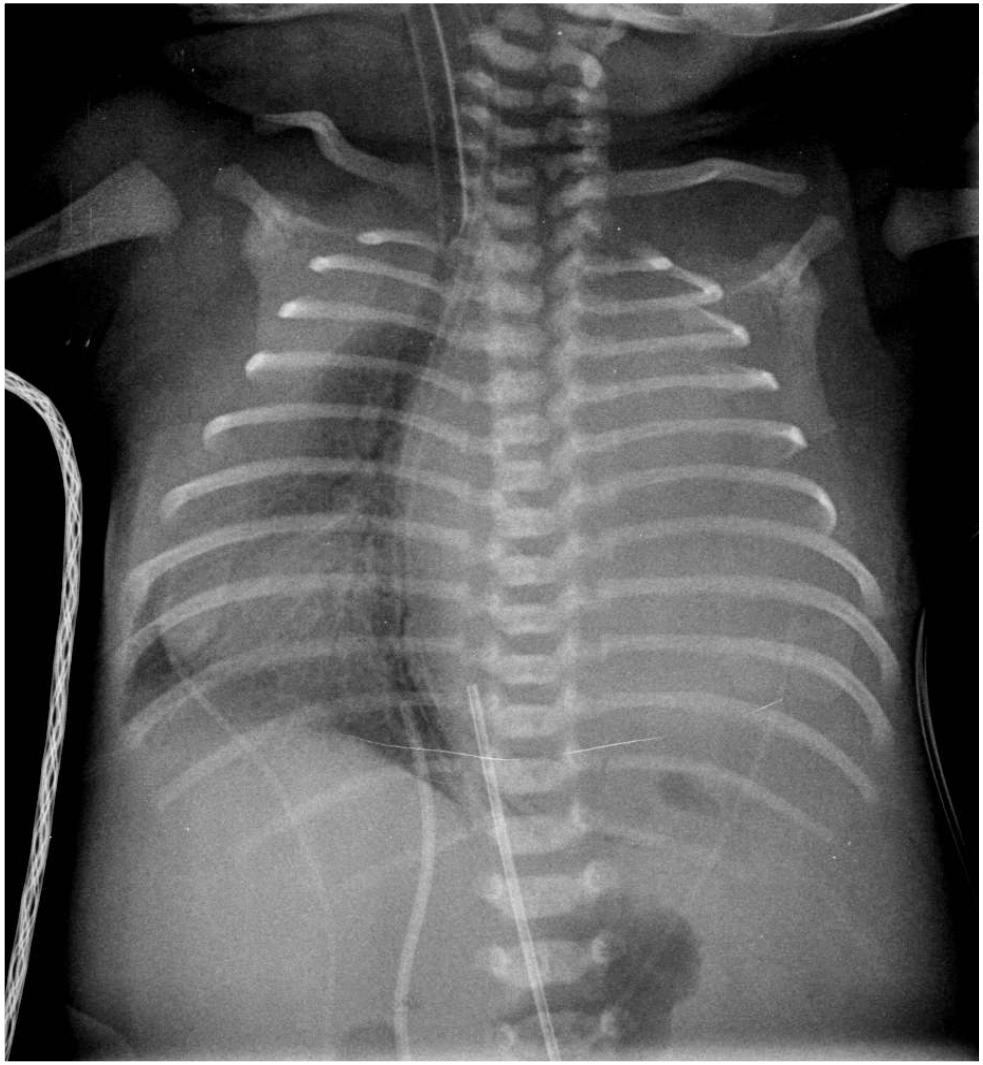
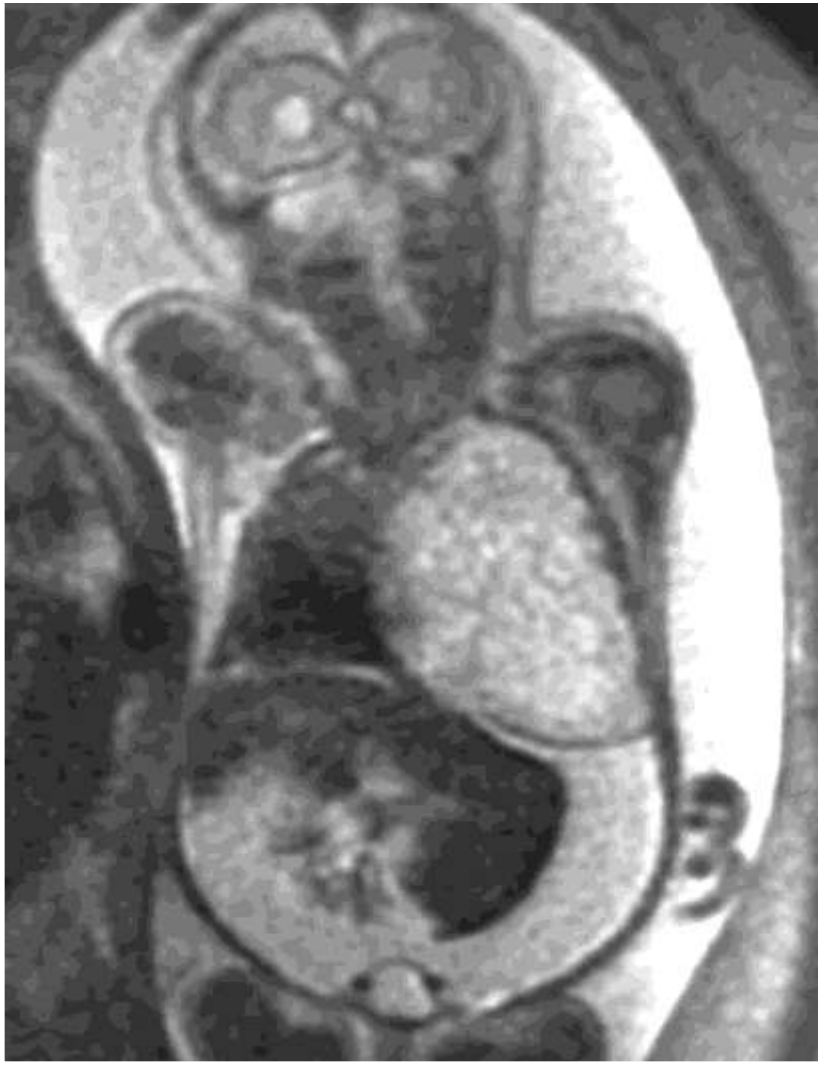




MCVAP 3





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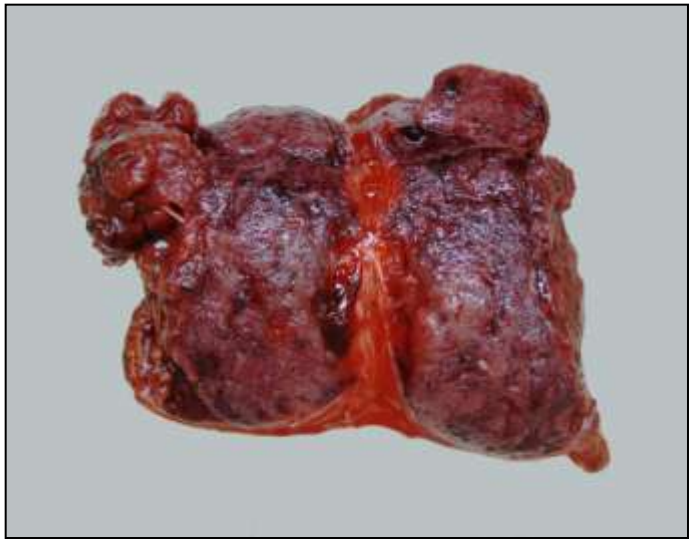

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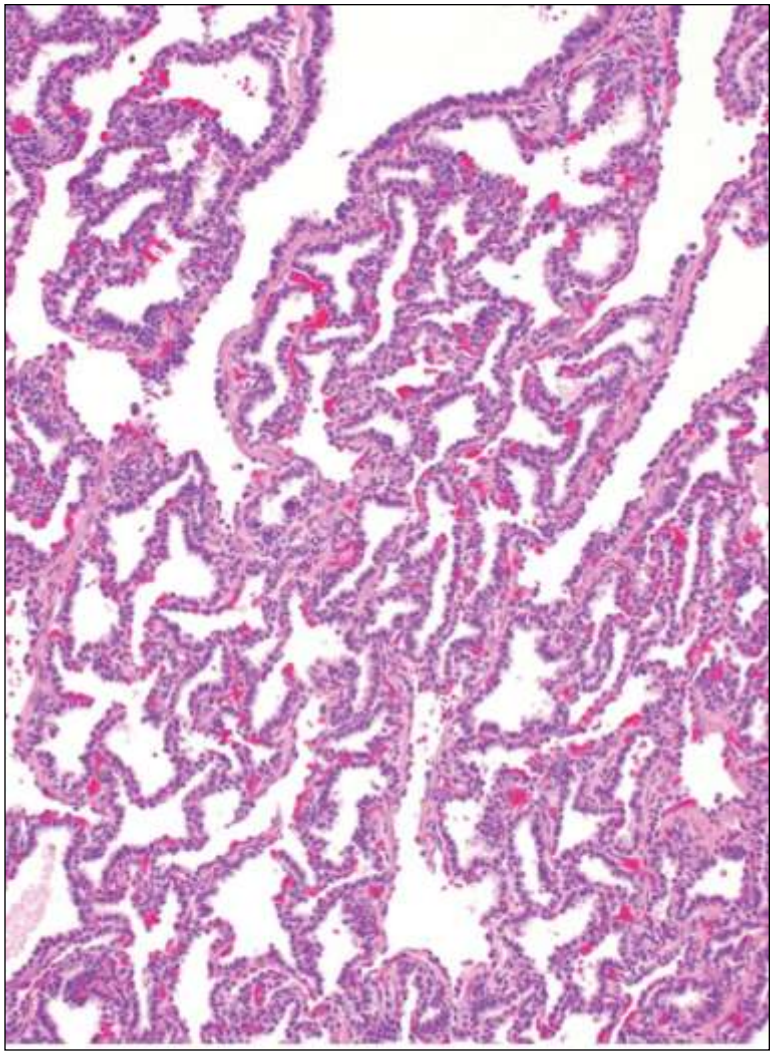

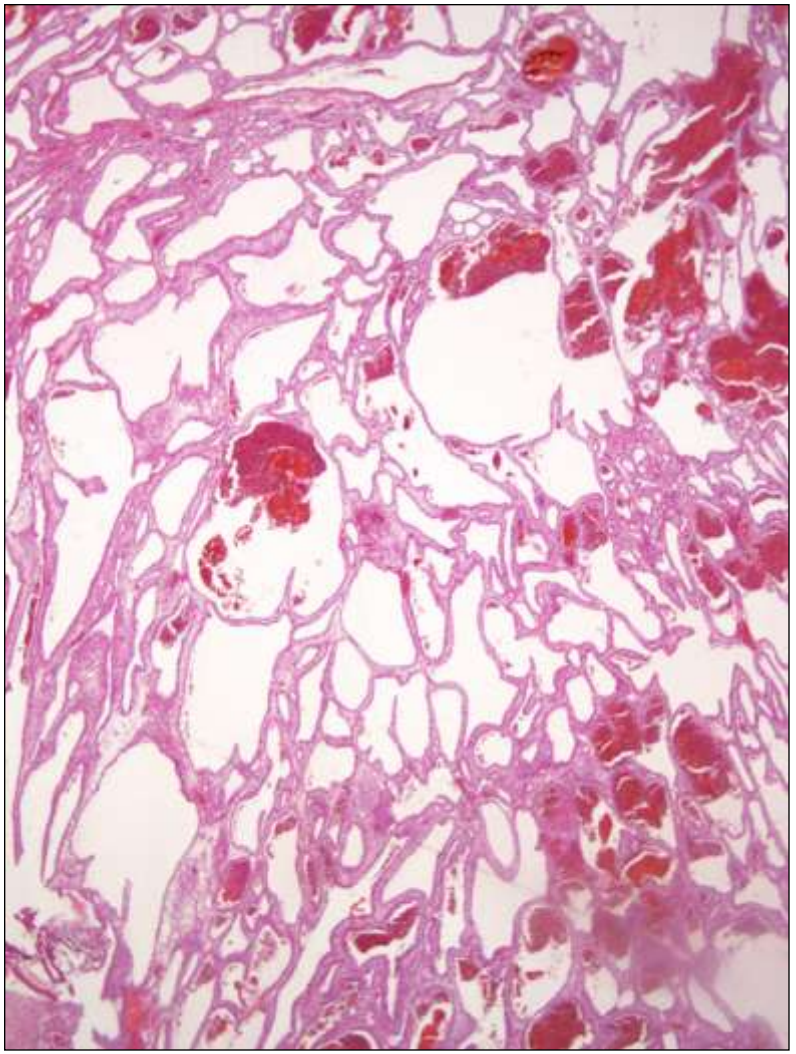

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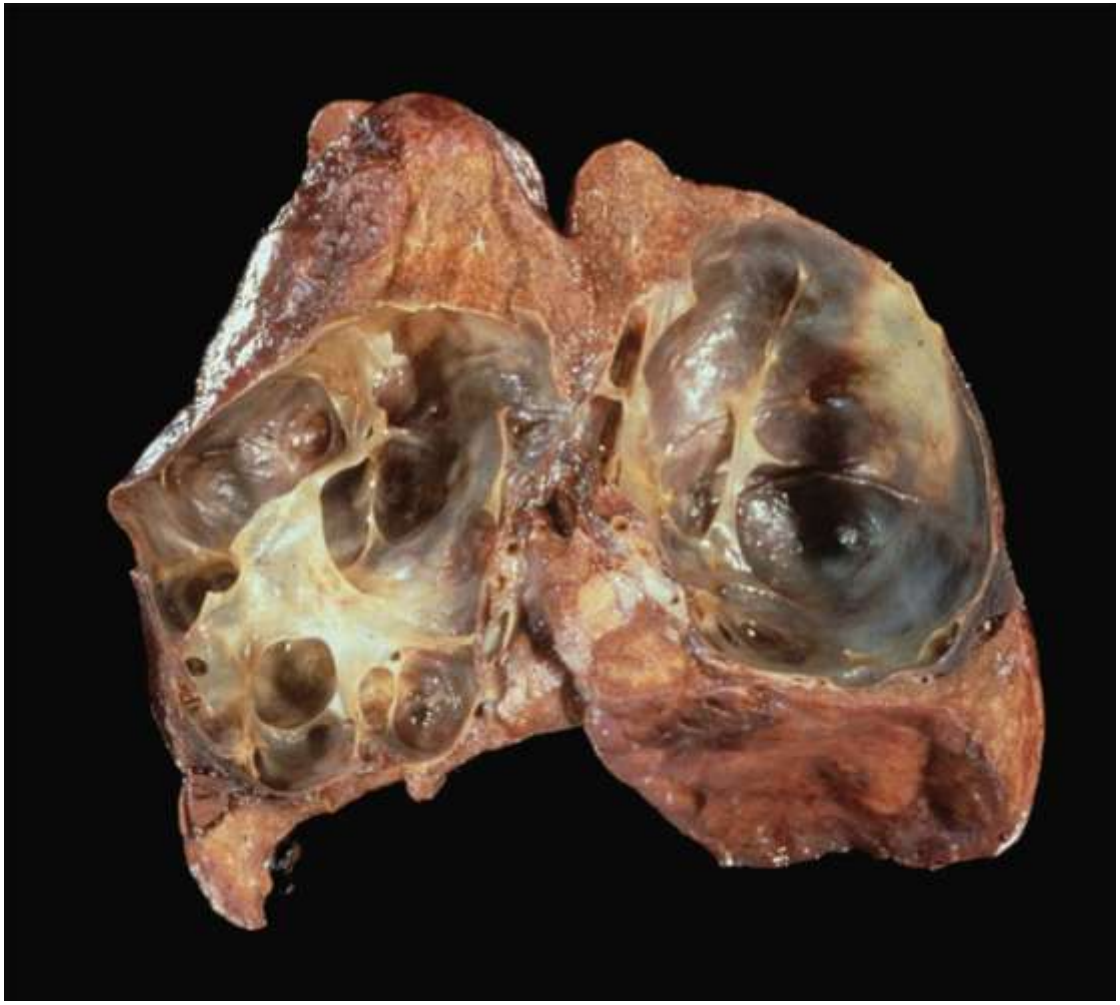
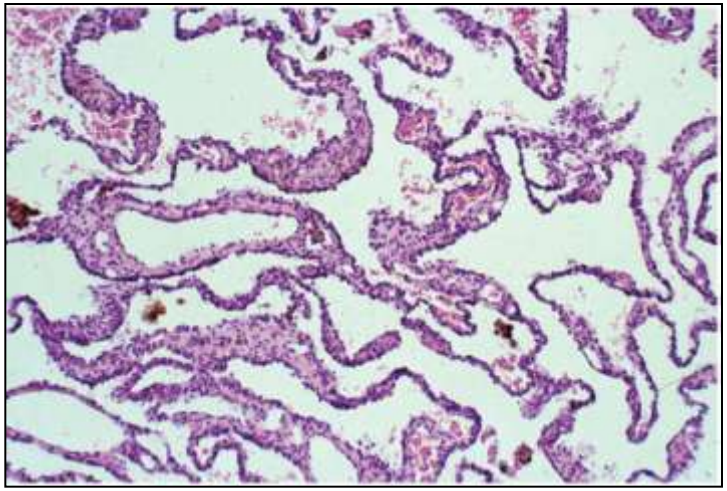
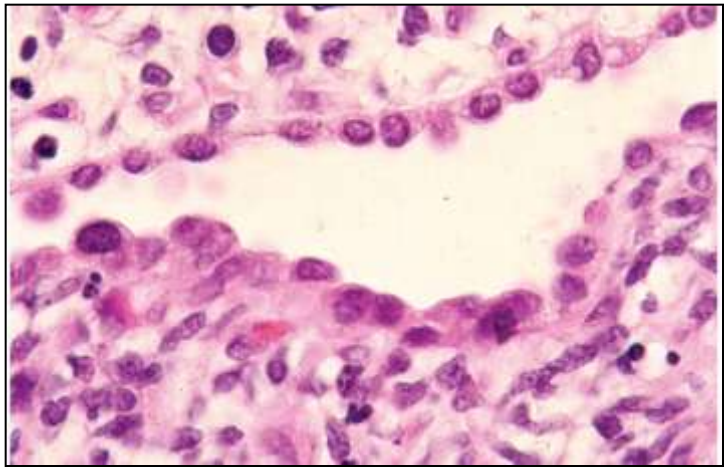
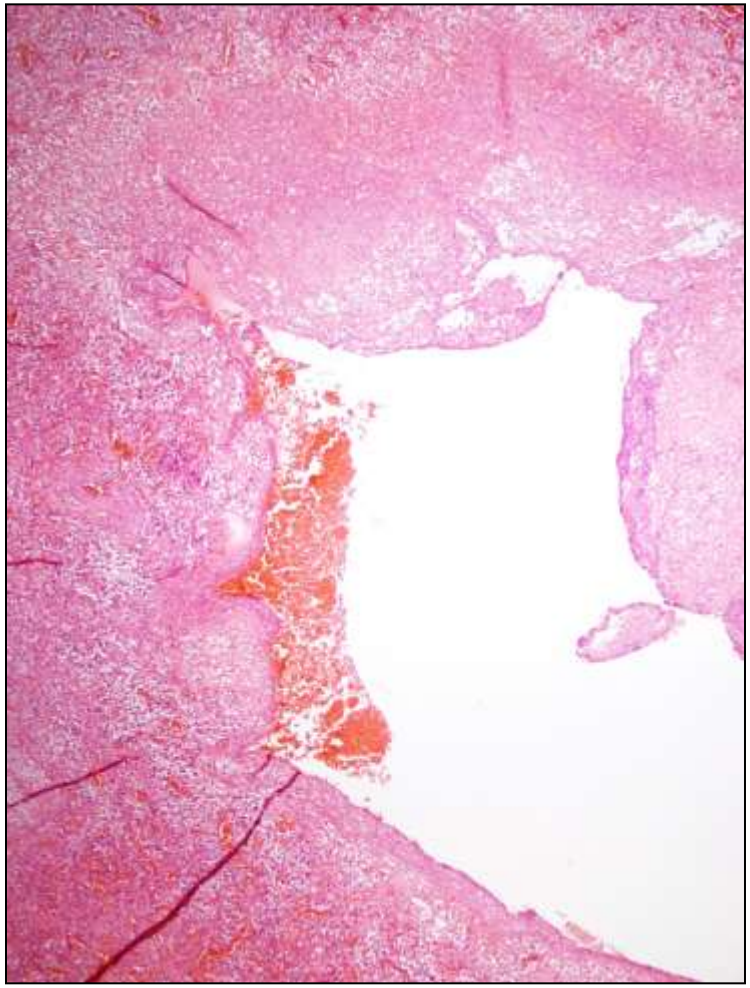
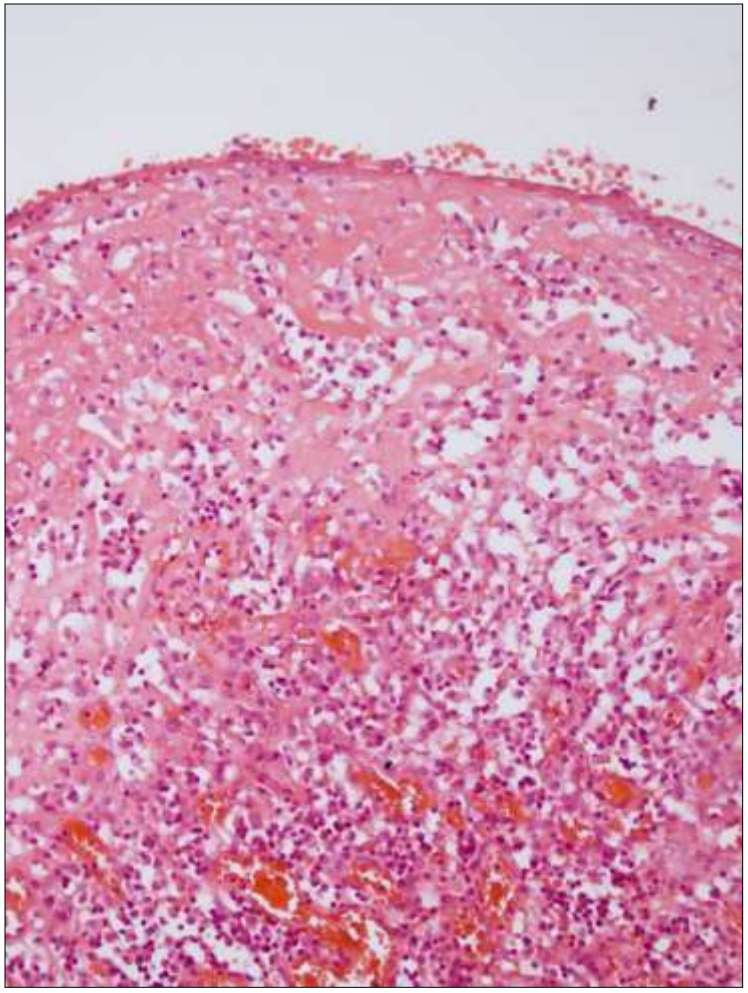






MCVAP





MCVAP · CLINICA

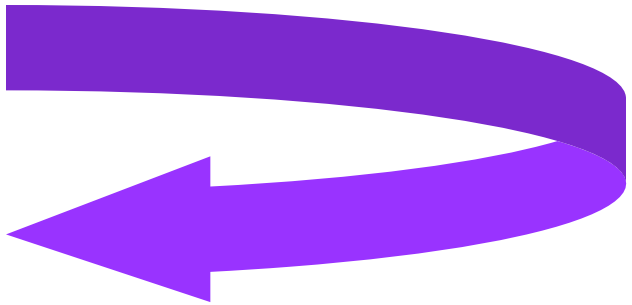
	Frecuencia Edad	Localización	Clínica	Pronóstico
Tipo 0	<2% Prematuros a término	Difusa	Cianosis Anom CDV, hipoplasia dérmica focal	Letal
Tipo 1	60-70% Intraútero colapsa RN al mes vida Niños y adultos	1 Lóbulo (95 %)	Distress respiratorio Infección Asintomáticos	Bueno con IQ, sin hipoplasia pulmonar
Tipo 2	15-20% Primer mes de vida	1 lóbulo	Asociada a otras malformaciones (ag renal, HD, SEL, CDV)	Resecables Depende de malformación
Tipo 3	5-10% (varones) Lesión sólida intraútero que no colapsa Primeros días-mes	1 lóbulo o todo el pulmón. Produce hipoplasia pulmonar y deformidad del mediastino.	Polihidramnios. Distress resp severo RN muerto	Malo Supervivencia si IQ y 35% del peso pulmonar.
Tipo 4	10% RN a 4 años	1 lóbulo. Afectación periférica	Distress leve Neumotórax Neumonía Silente	Bueno con IQ

MCVAP · HISTOLOGIA

	Quistes	Epitelio	Mesénquima	C.mucina	Cartílago	M.Esquelético
Tipo 0	Sólida	Pseudoestrat. ciliado	Abundante laxo. HEM.Vasos finos.A y V no des.	SI	SI	NO
Tipo 1	1-10cm 2arios	Pseudoestrat ciliado Cuboidal, Columnar bajo		SI (45%)	SI	NO
Tipo 2	O,5-2cm	Cuboidal, columnar bajo		NO	NO	SI (displ.rabdomiomatosa)
Tipo 3	Sólida 0,5cm	Cuboidal	Pocos vasos	NO	NO	NO
Tipo 4	Grandes	Plano alveolar	Laxo con vasos prominentes	NO	Raro <10%	NO

MCVAP . MALIGNIDAD

- Rabdomiosarcoma



- Blastoma pleuropulmonar
  - Estroma celular, rabdomioblastos,cartílago inmaduro.
  - MCVAP 4 son BPP no reconocidos?

- Carcinoma bronquioloalveolar → Células mucinosas

Human Pathology (2007) 38, 1772–1778

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Original contribution

Expression of epidermal growth factor receptor, but not *K-RAS* mutations, is present in congenital cystic airway malformation/congenital pulmonary airway malformation

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[d]*Department of Pathology, Hospital Pereira Rossell, Montevideo 11.200, Uruguay*
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EVOLUCION

- *Fc. Pronósticos:*
  Gestación, tipo, tamaño, deformidad mediastino, polihidramnios, hydrops fetal, malformaciones asociadas

Prenatal

- Regresión espontánea (15% -20%)
- 90% parto a término
- Hydrops 10%

Postnatal

- Distress respiratorio
- Infección
- Degeneració maligna

SEGUIMIENTO

Control Prenatal

ECO,RNM: 3 grupos de riesgo:

- Alto riesgo fetal: Hydrops: Terapia fetal vs finalizar gestación.
- Alto riesgo neonatal: EXIT vs cesárea electiva i tratamiento neonatal urgente.
- Bajo riesgo (90%): Parto a término. Seguimiento postnatal. Cirurgia electiva antes que sea sintomático.

.Control Postnatal

RX tórax, TAC contraste, angioRNM

CLASIFICACIÓN

Malformaciones difusas, base genética: deficit surfactante, displasia alveolar capilar, displasia acinar.

Malformaciones congénitas locales:

- Malformación congénita vía aérea pulmonar (MCVAP).
- Secuestro pulmonar: intralobar (SIL), extralobar (ELS).
- Enfisema lobar (EL).
- Quiste broncogénico (QB).

Anomalías del crecimiento pulmonar: hipoplasia, hiperplasia pulmonar.

SECUESTRO

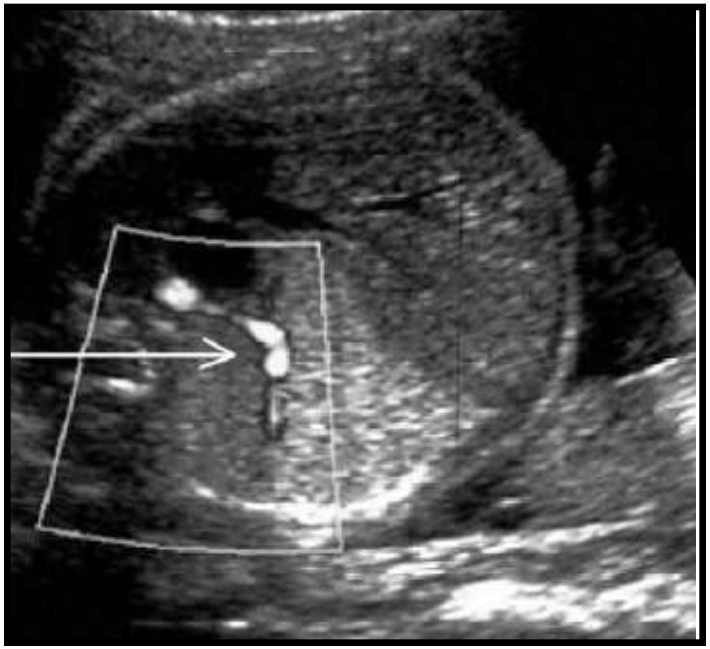
= Masa no funcionante de tejido pulmonar, sin comunicación traqueobronquial y aporte vascular sistémico.

Intralobar (SIL): no pleura propia.

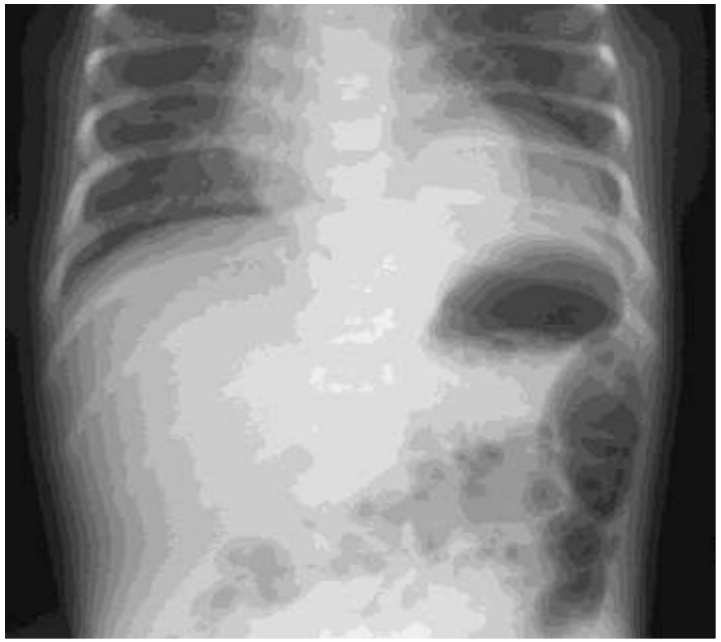
Extralobar (SEL): pleura propia.

SIL

- Lóbulo inferior
- Asintomáticos, distress o neumonias repetición.

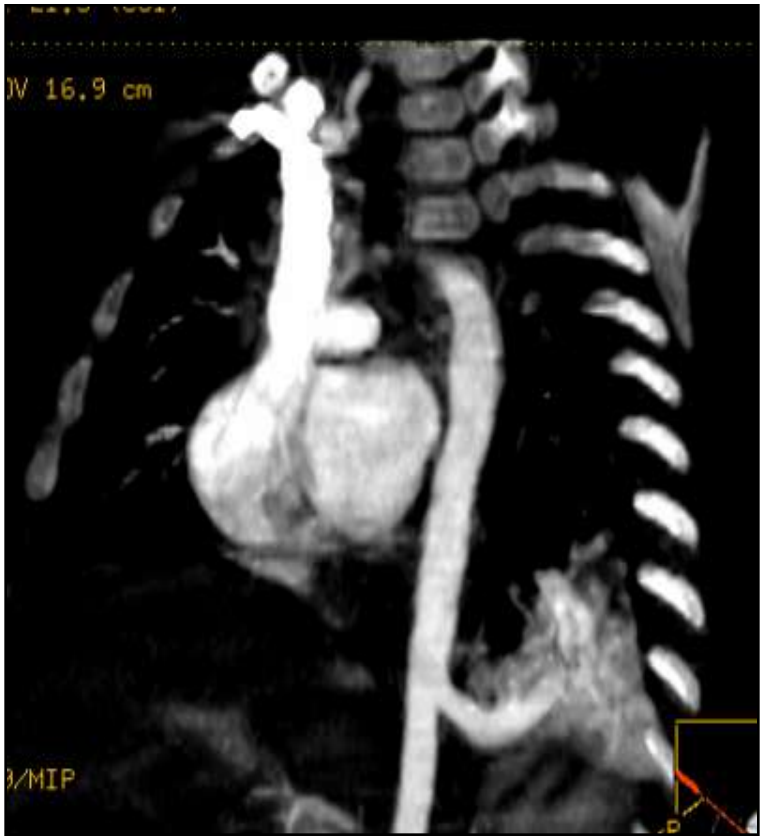


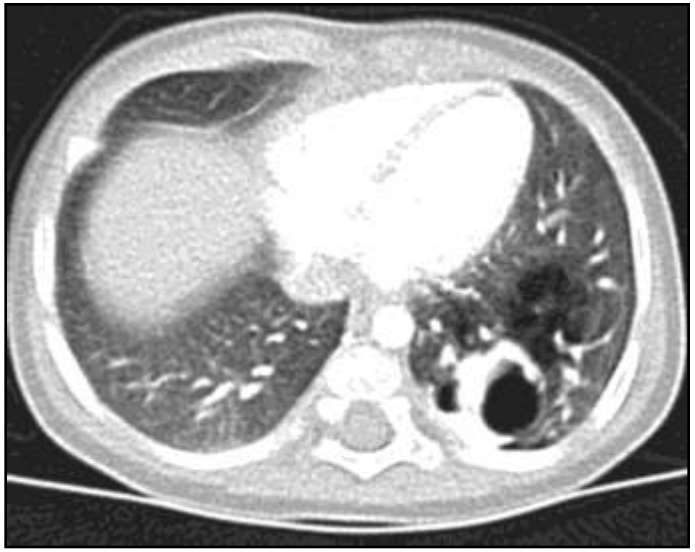
prenatal

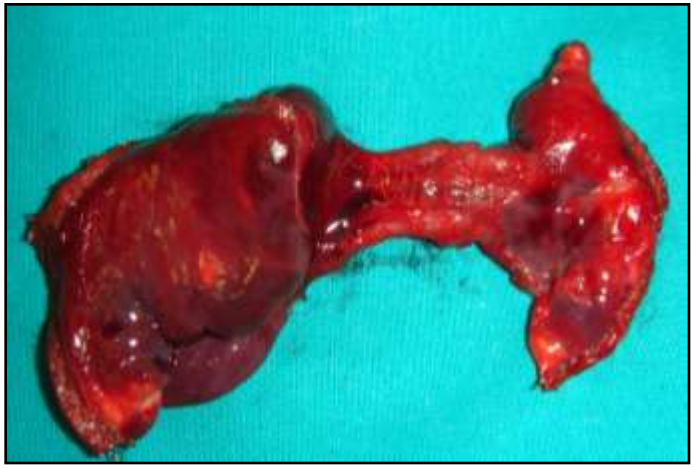


postnatal

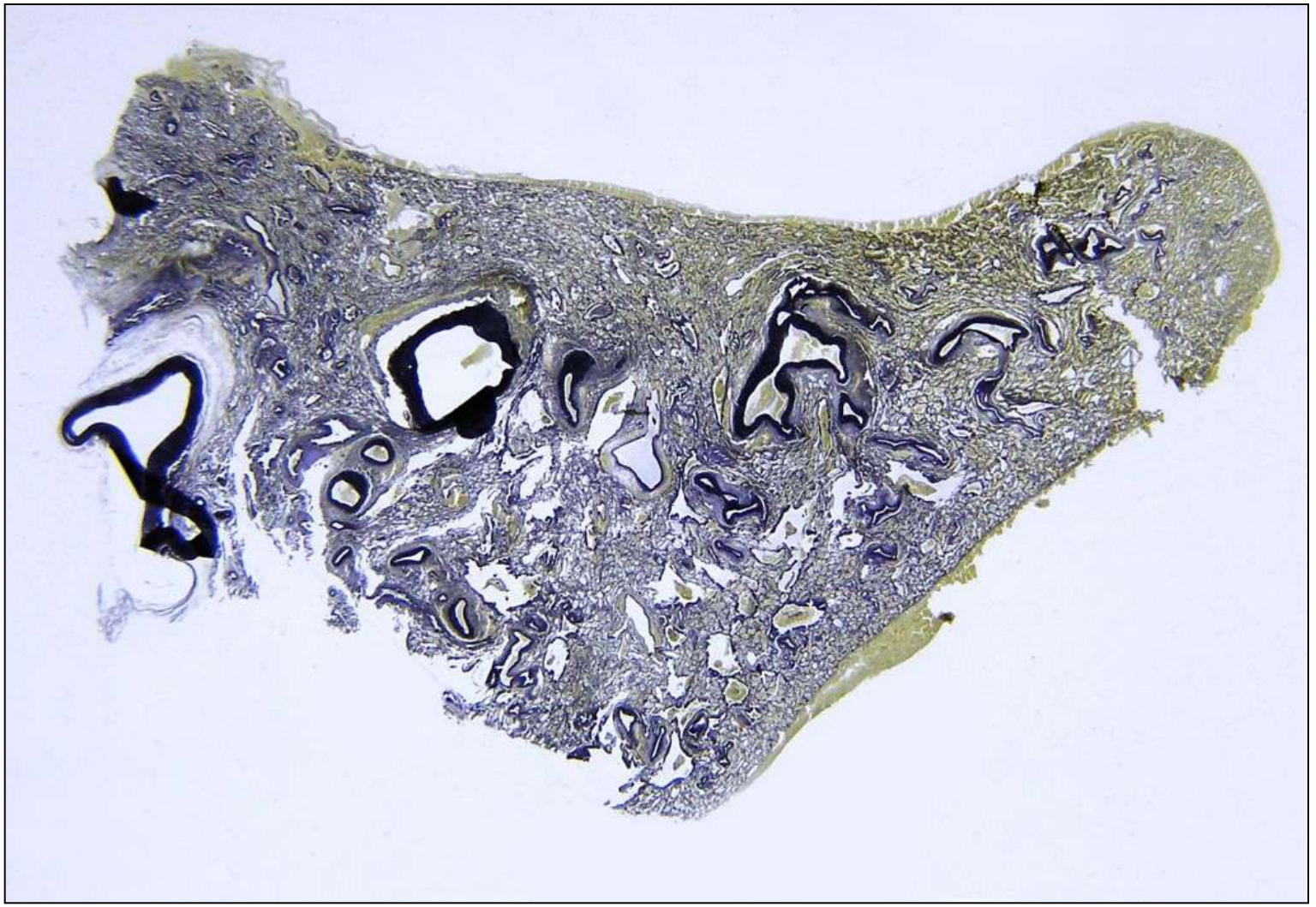
SIL







SIL



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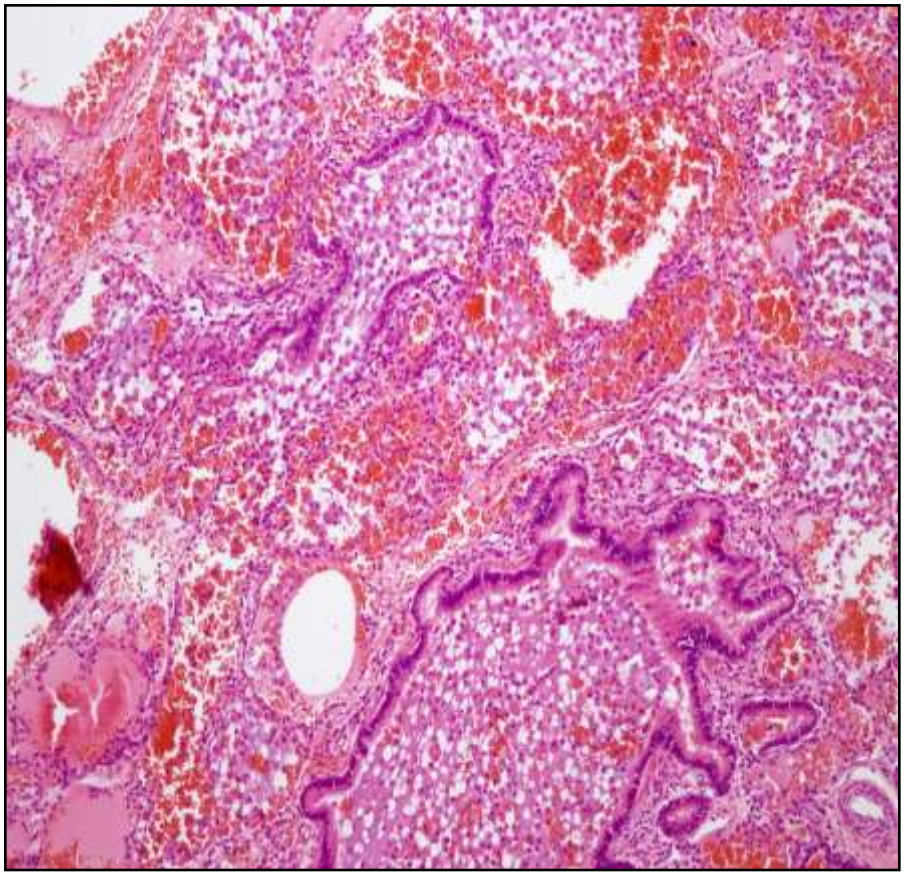
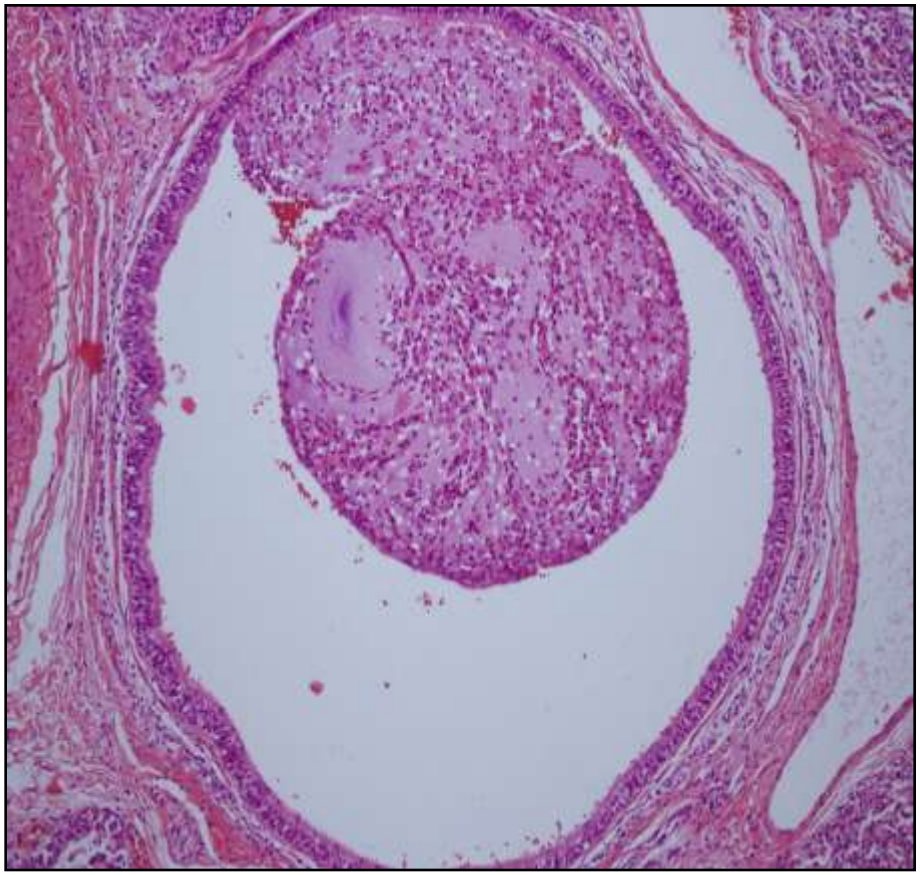
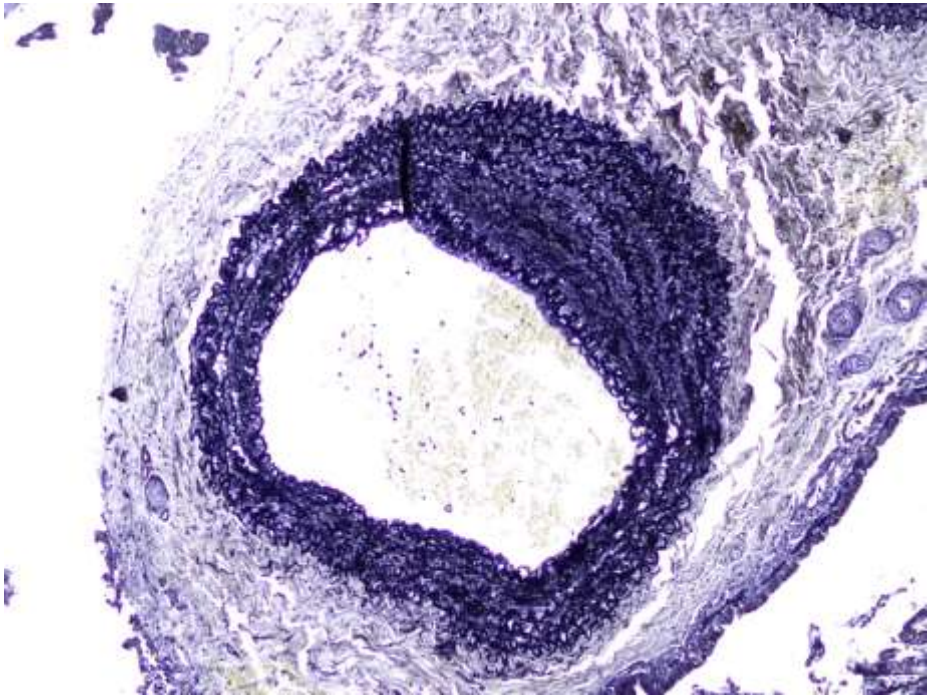
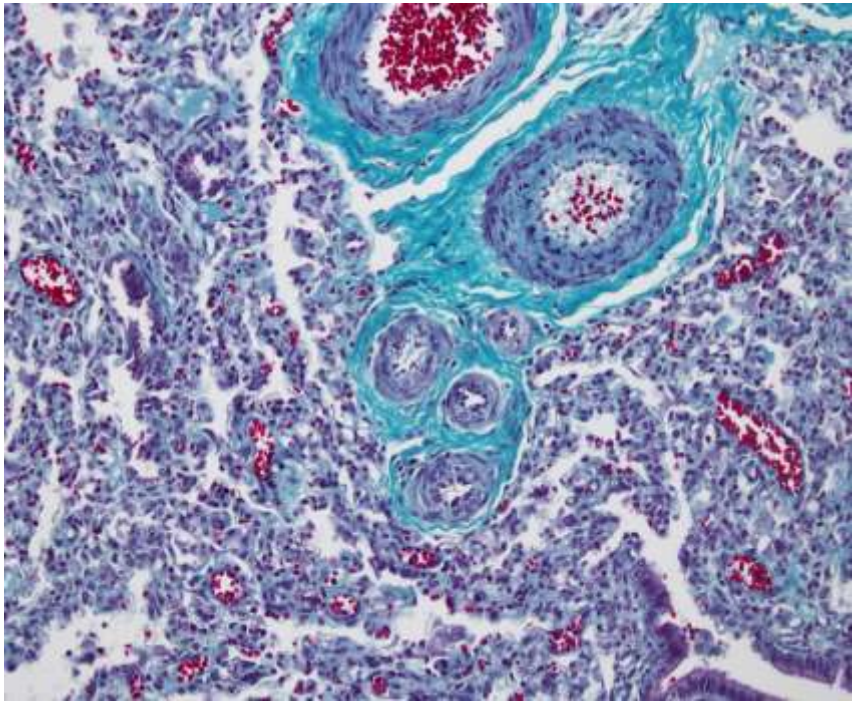




SIL

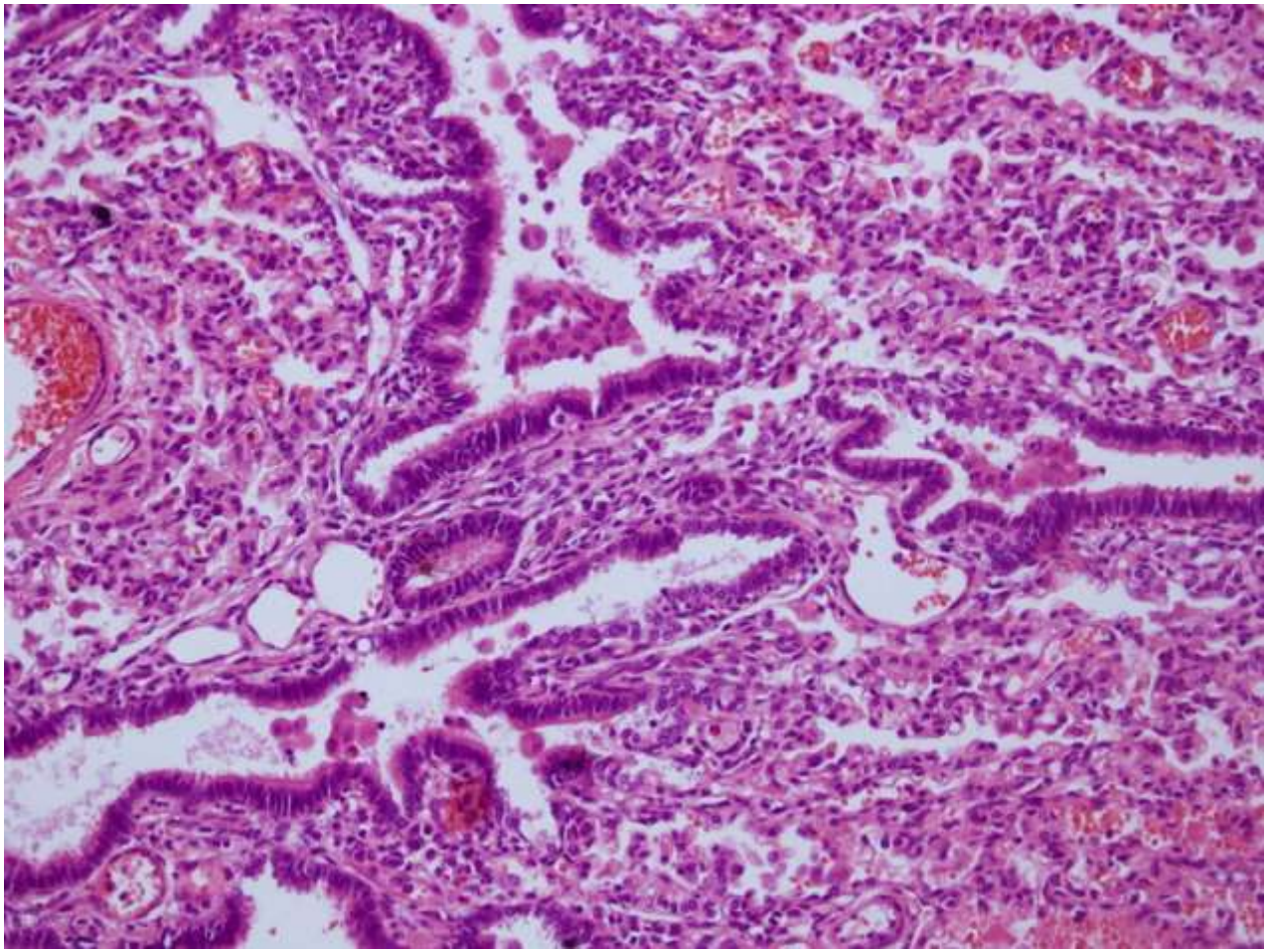


Fibras elásticas



tricrómico

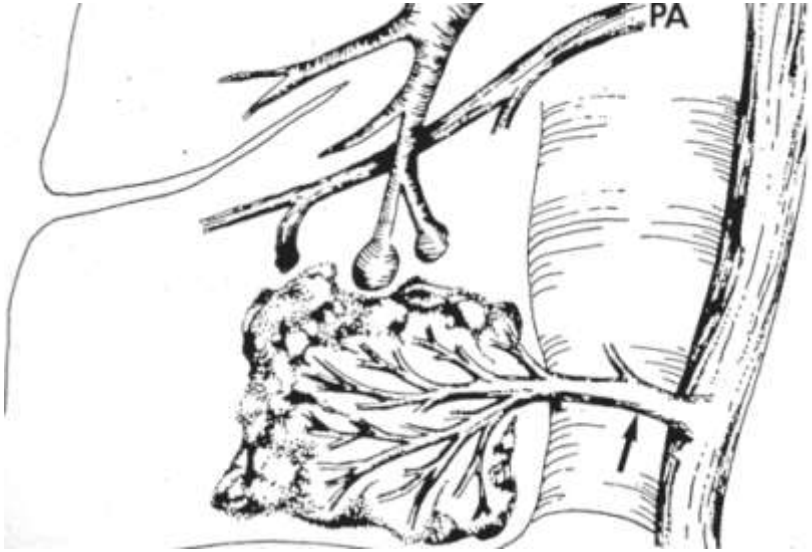
SIL



25 %

SIL - Patogenia

- Etiologia adquirida (Stocker):
  - Rareza en el RN.
  - Lóbulos inferiores (98%).
  - Infecciones repetidas.
  - Poca asociación a otras malformaciones.



Ar. Lig pulm

SIL Patogenia

- Origen congénito:
  - Detección prenatal por ECO.
  - Asociación con anomalias congénitas.
  - Asociación SEL-SIL.

  Arterias (?)

  - Ligamento pulmonar

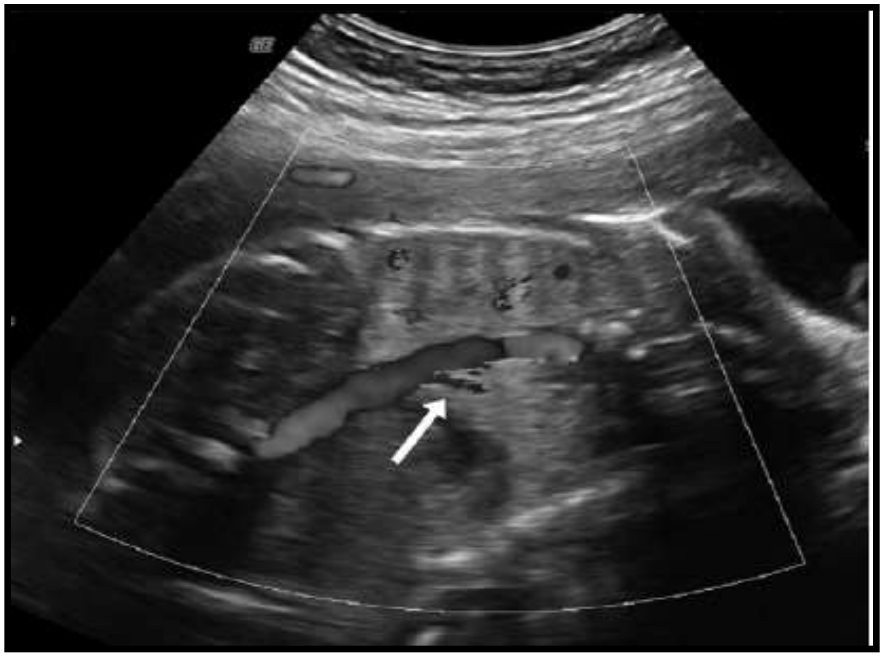
  - MAV

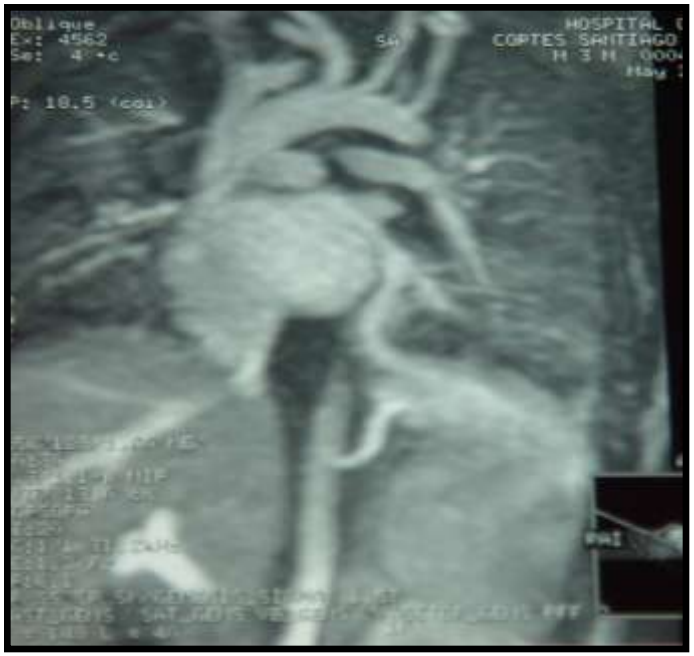
  - Plexo esplácnico conecta Ao dorsal-pulmón embrionario (eje celíaco)

    (6 sem regresa)

SEL

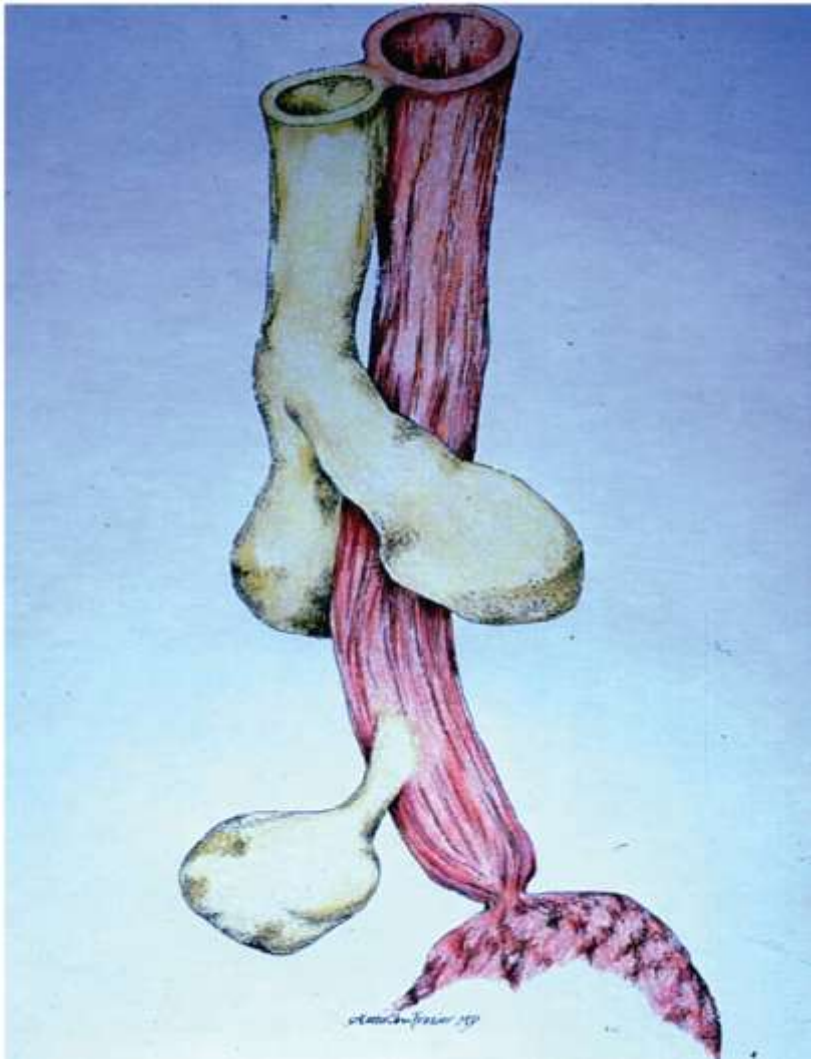
- Primer año de vida.
- Cianosis, disnea, dificultad deglución. Asintomáticos (10%).





Prenatal 25%

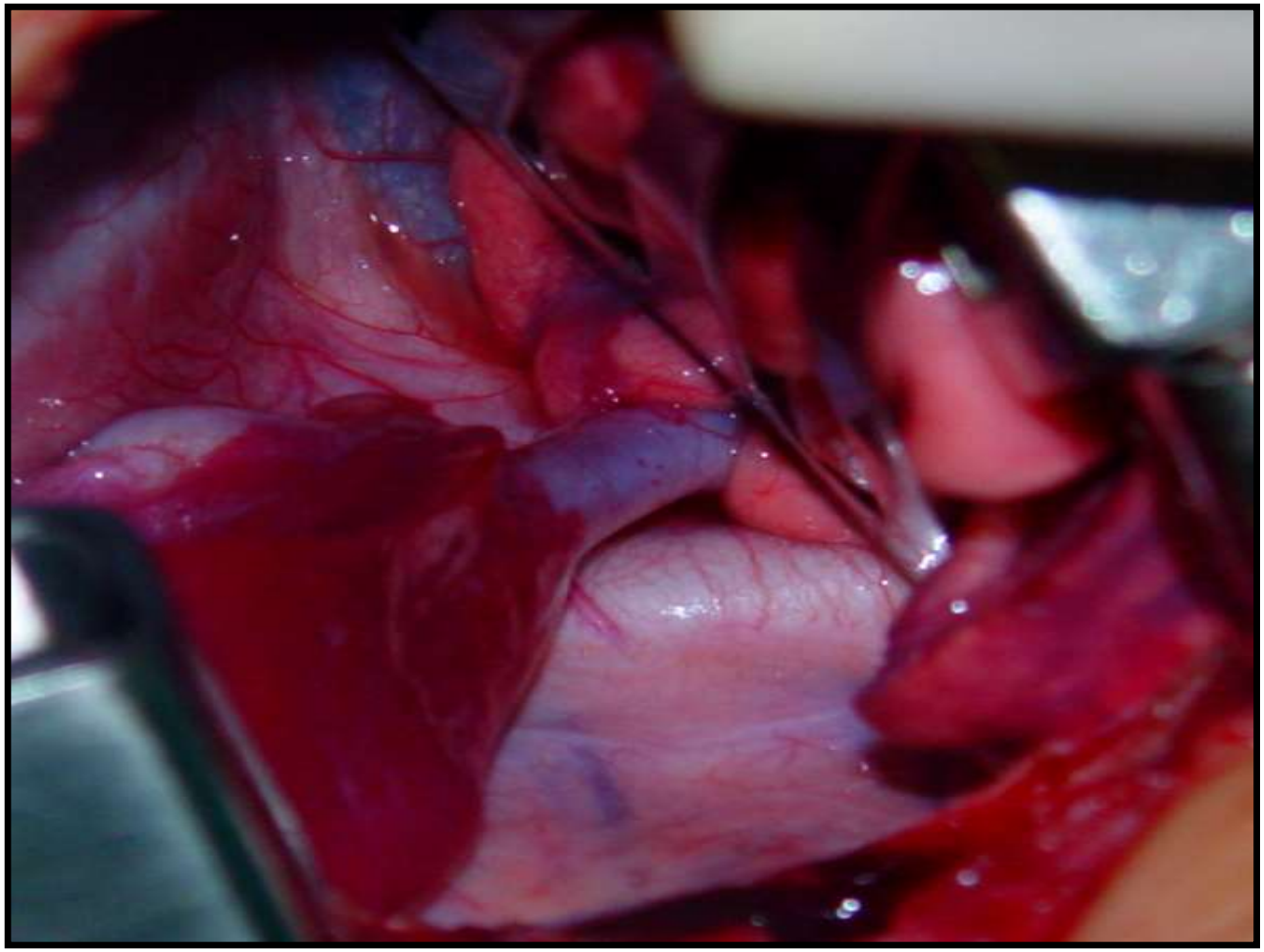
SEL- Patogenia



- Divertículo separado del origen pulmonar.

- Asociado a anomalías congénitas (CPAM 2, CDV, HD).

SEL



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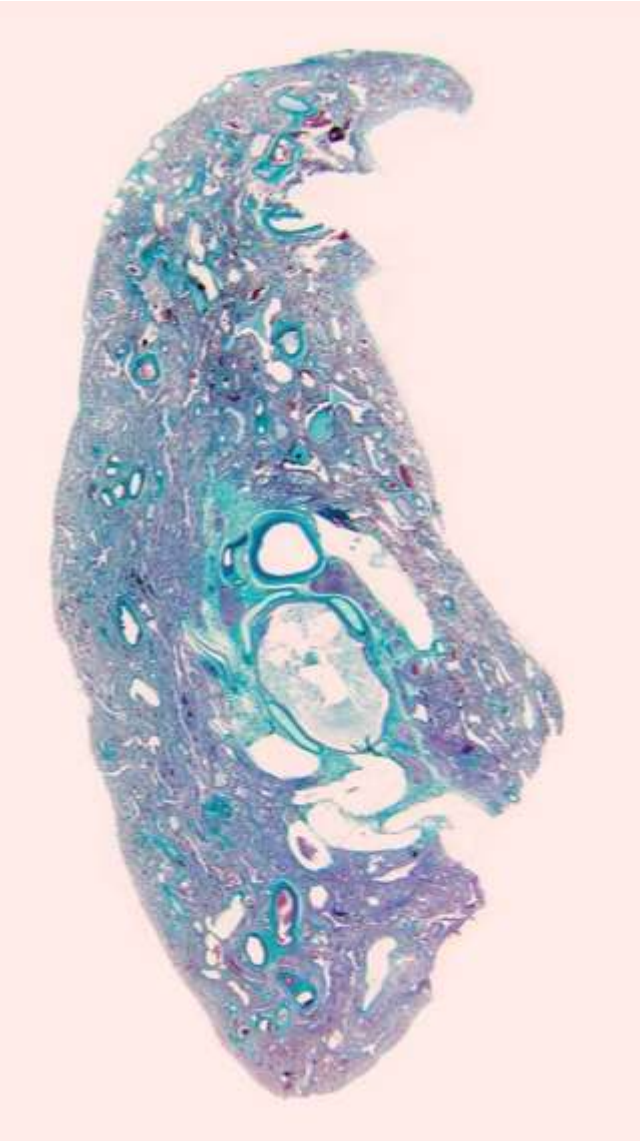

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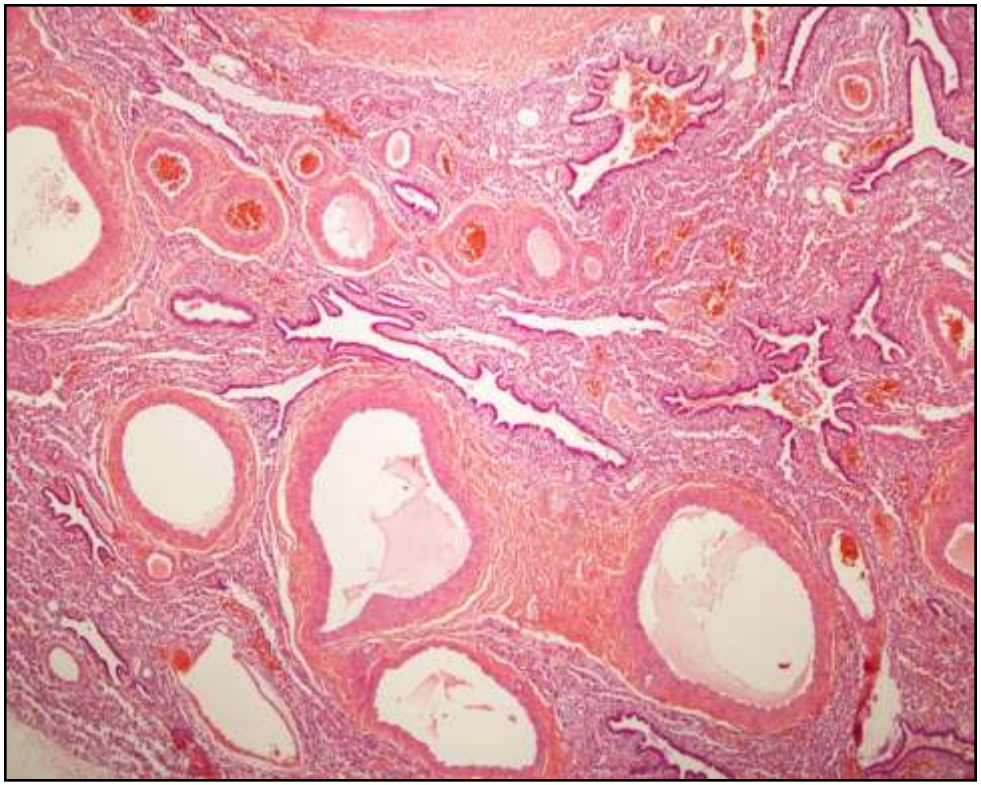

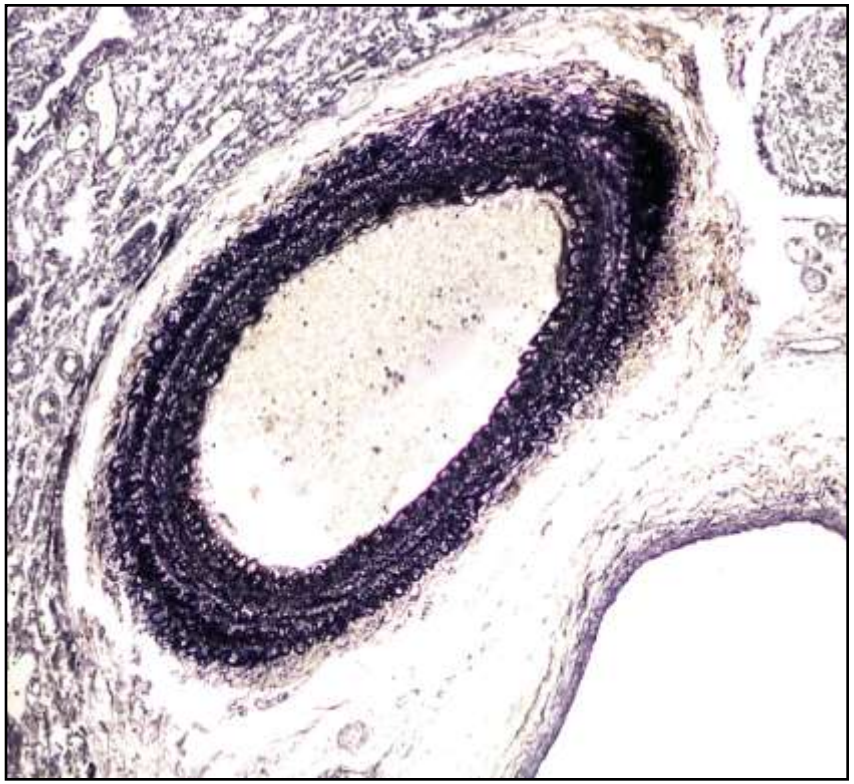

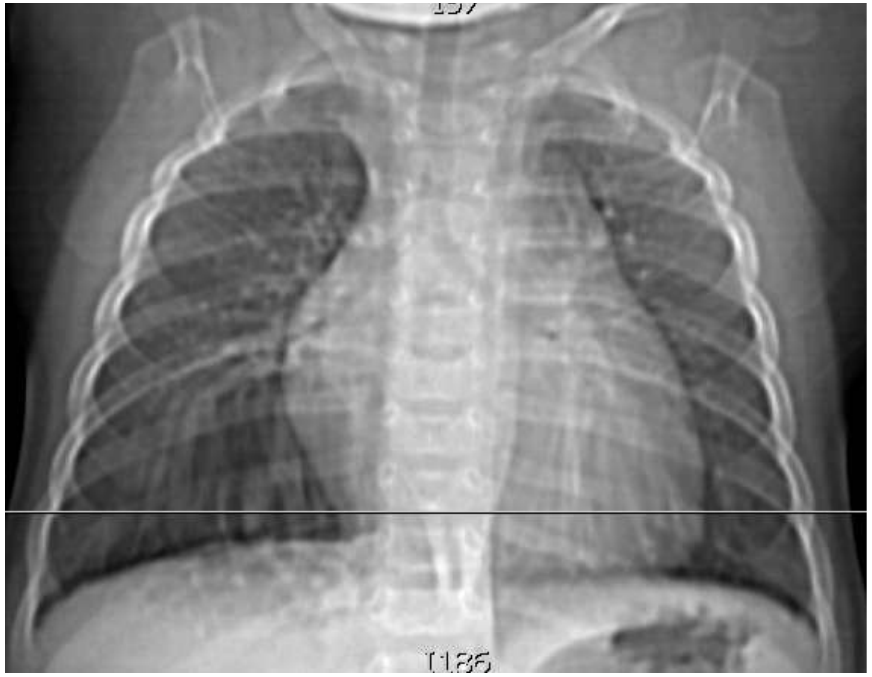

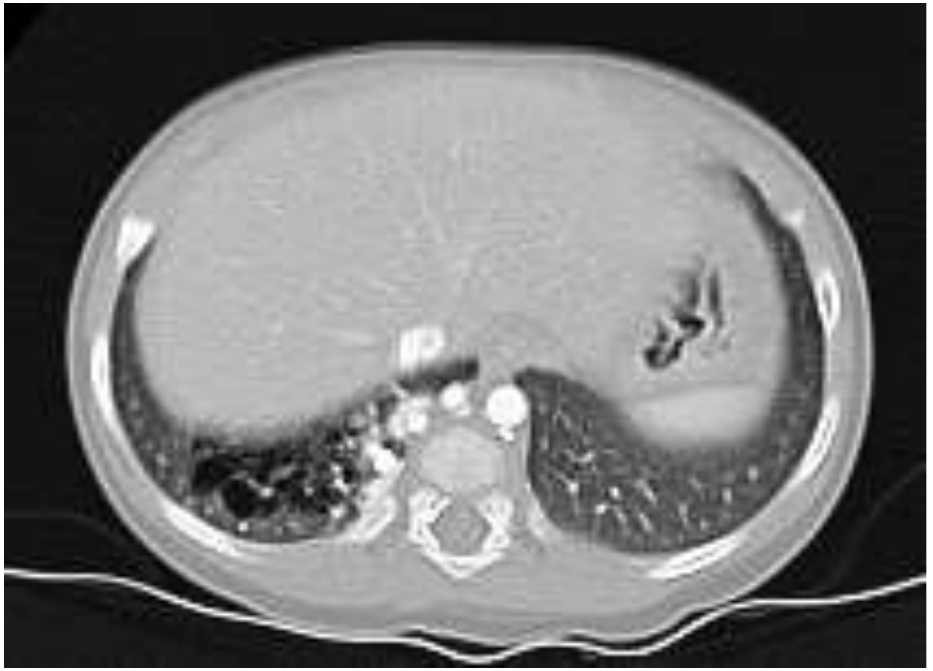

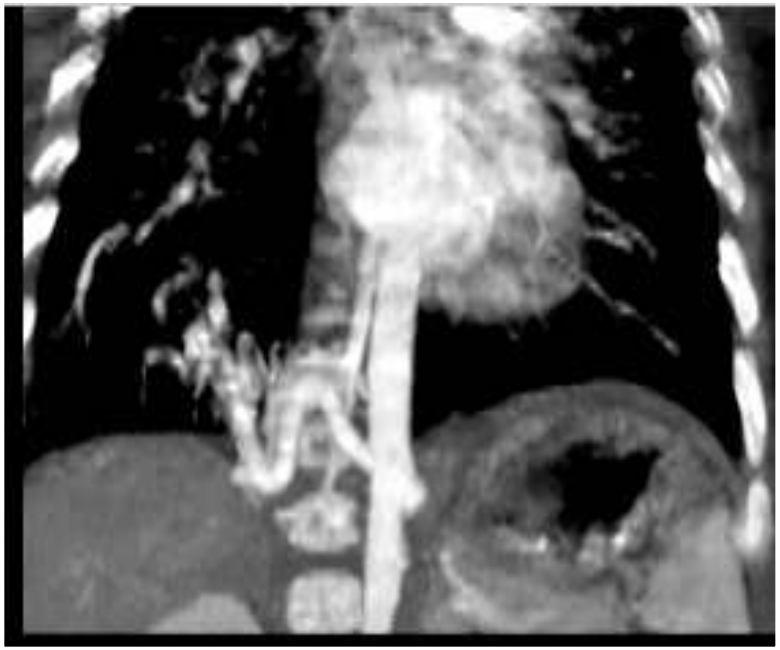

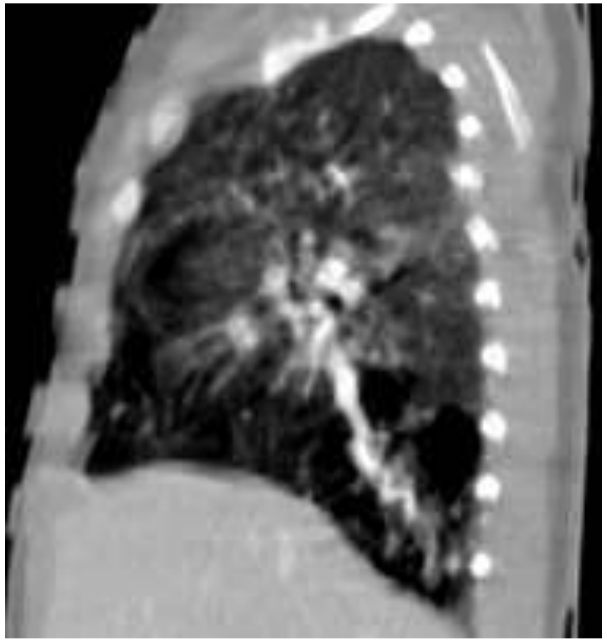

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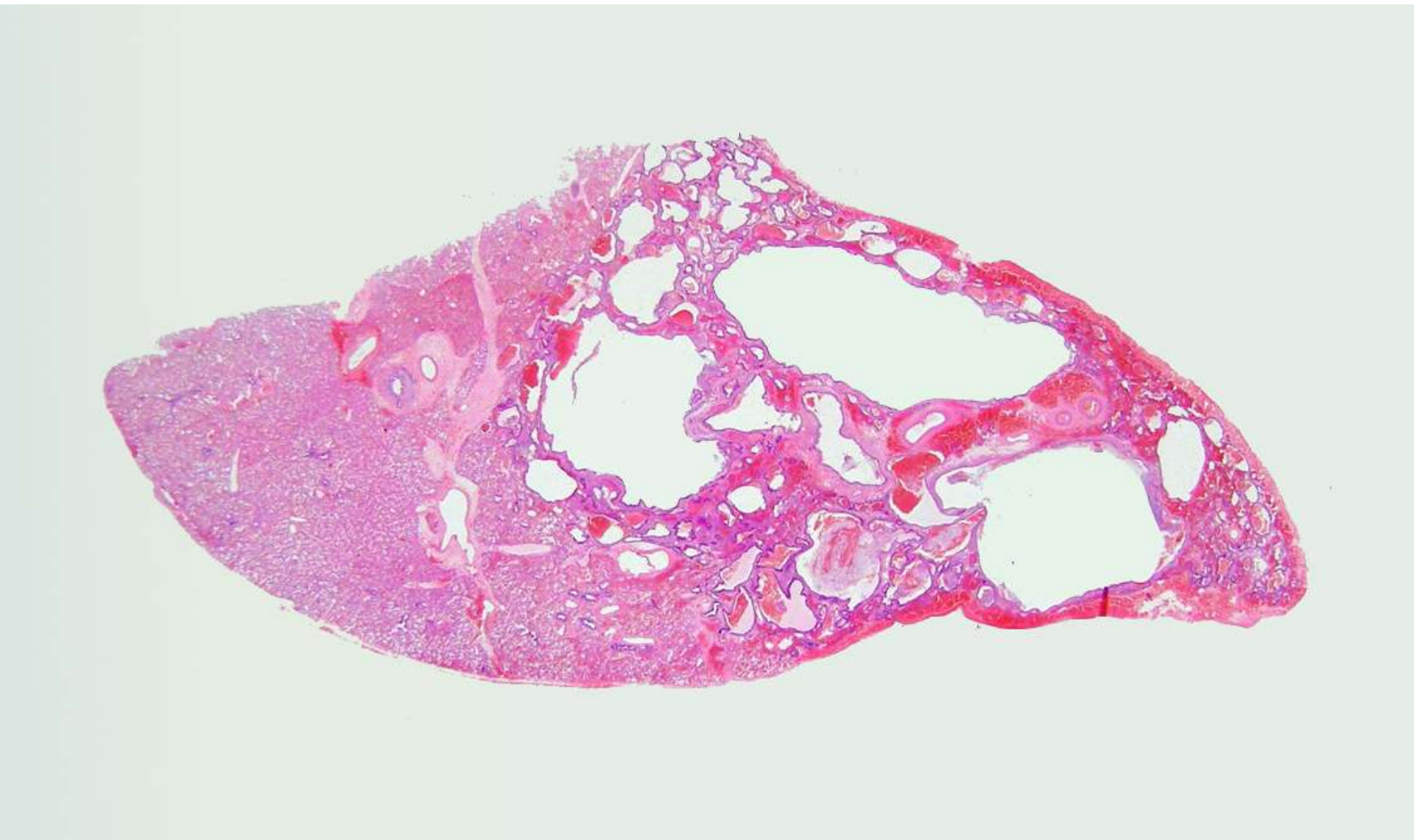

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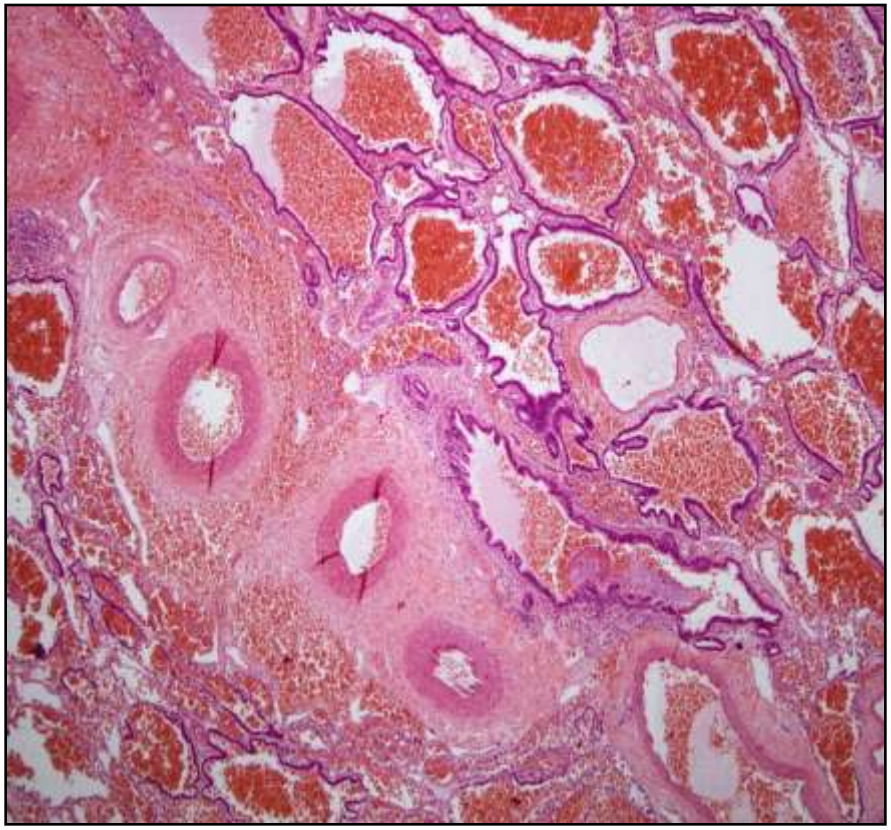

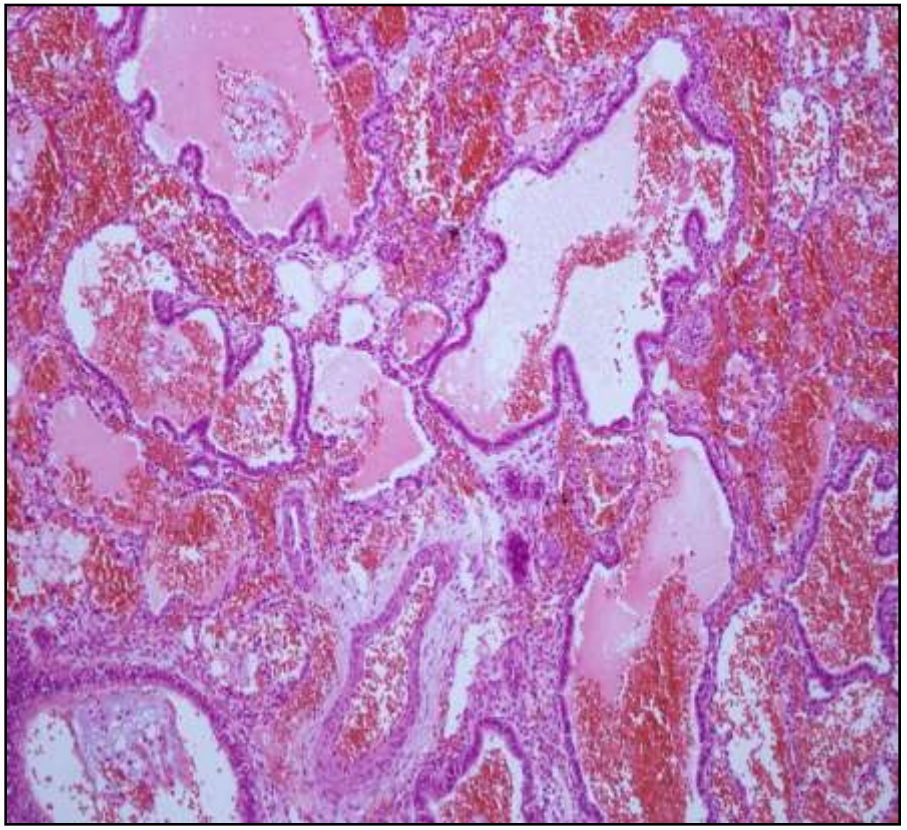

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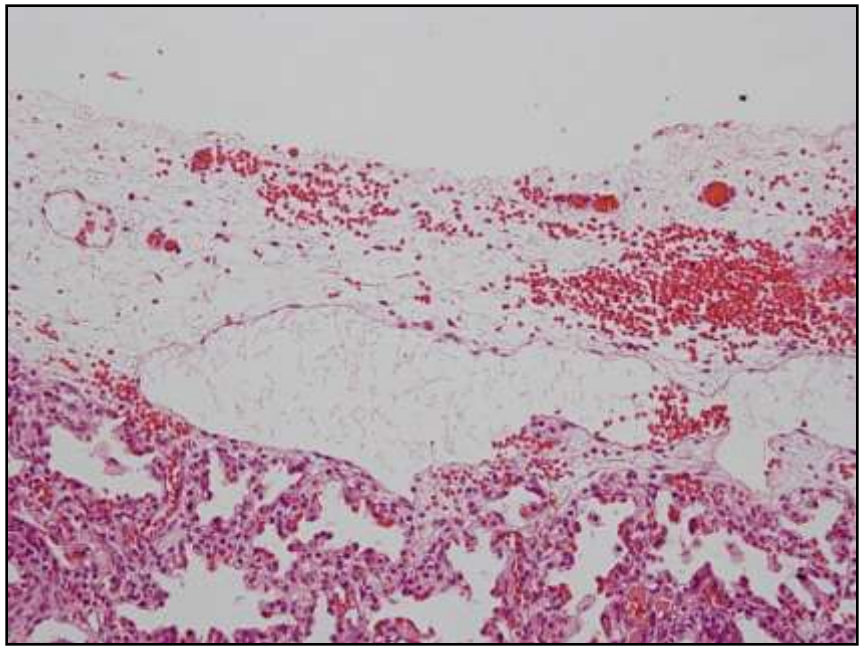
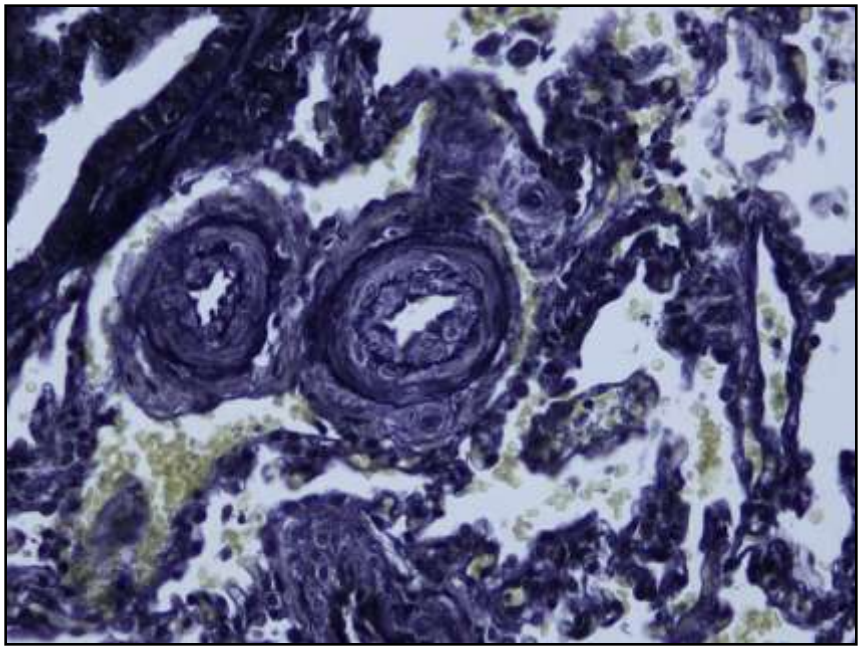




CAMBIOS VASCULARES

Histopathology 2010, **57**, 121–127. DOI: 10.1111/j.1365-2559.2010.03586.x

Secondary vascular changes in pulmonary sequestrations

Saral Desai, Michael Dusmet,[1] George Ladas,[1] Sabine Pomplun,[2] Simon P G Padley,[3] Nyree Griffin,[3] Jamal Badreddine,[4] Peter Goldstraw[1] & Andrew G Nicholson

Department of Histopathology, Royal Brompton and Harefield NHS Trust, [1]Department of Surgery, Royal Brompton and Harefield NHS Trust, [2]Department of Histopathology, Kings College Hospital, [3]Department of Radiology, Royal Brompton and Harefield NHS Trust, London, UK, and [4]Cabinet d'anatomie-pathologique Richier, Rennes, France

CLASIFICACIÓN

Malformaciones difusas, base genética: deficit surfactante, displasia alveolar capilar, displasia acinar.

Malformaciones congénitas locales:

- Malformación congénita vía aérea pulmonar (MCVAP).
- Secuestro pulmonar: intralobar (SIL), extralobar (ELS).
- Enfisema lobar (EL).
- Quiste broncogénico (QB).

Anomalías del crecimiento pulmonar: hipoplasia, hiperplasia pulmonar.

ENFISEMA LOBAR (EL)

= Hiperinsuflación (clasico) o hiperplasia (polialveolar) de un lóbulo pulmonar, resultado de obstrucción p/c bronquial.

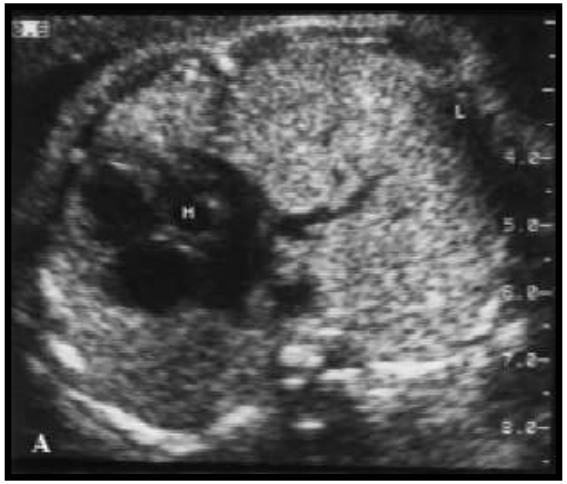
Lóbulo superior (Izq)

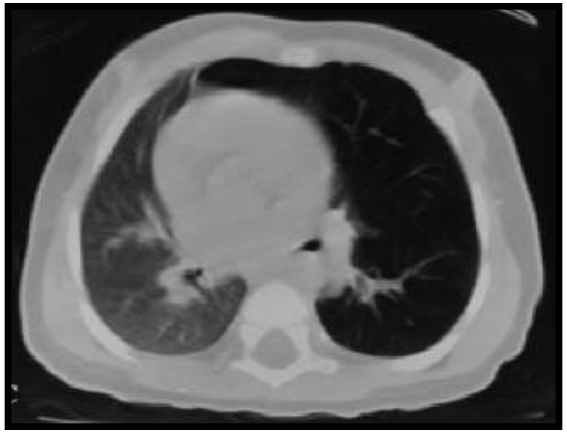
6 meses de vida. Distress. Cardiopatia.

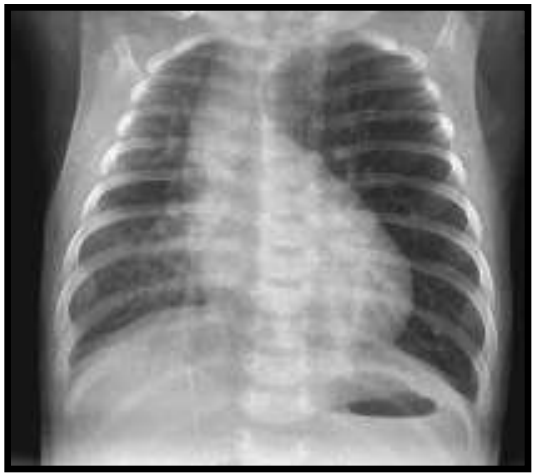
Causa intrínseca: anomalias bronquiales, aspiración, moco, c.extraño…

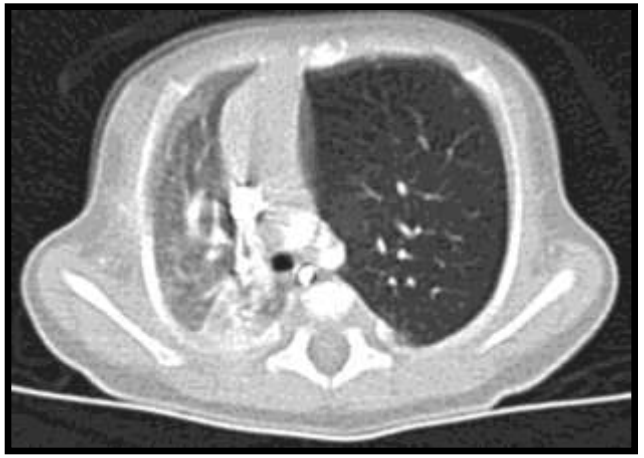
Causa extrínseca: Anomalias vasculares, quiste broncogénico.

EL





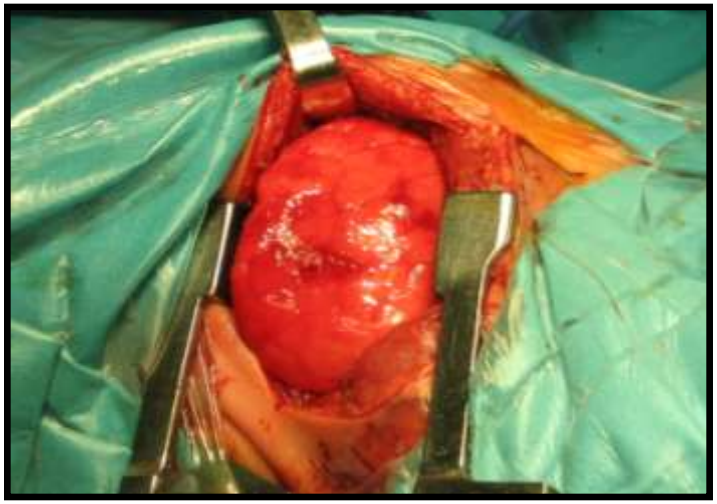


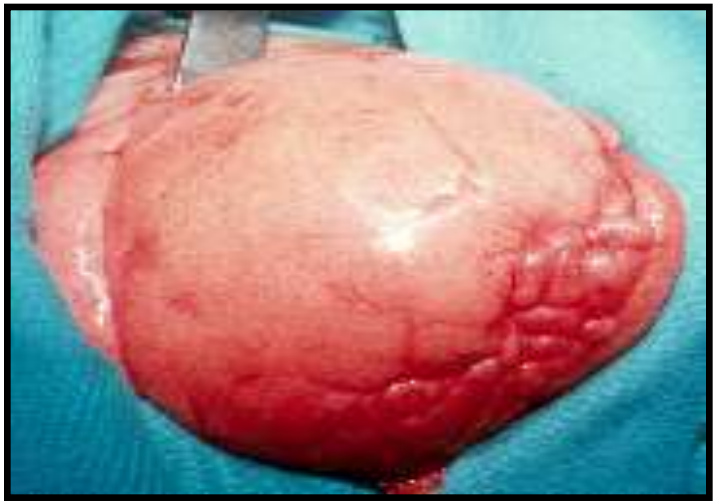


PRENATAL

POSTNATAL

EL





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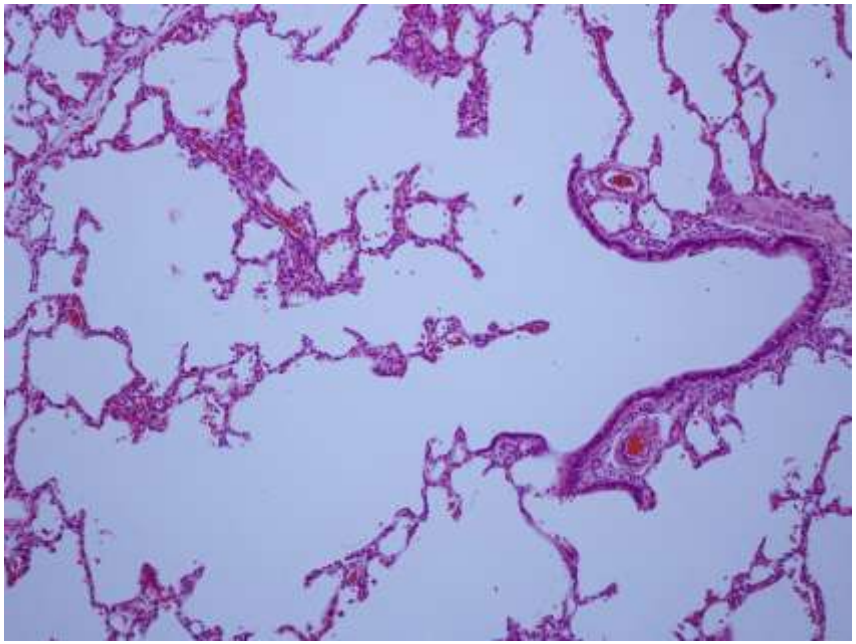

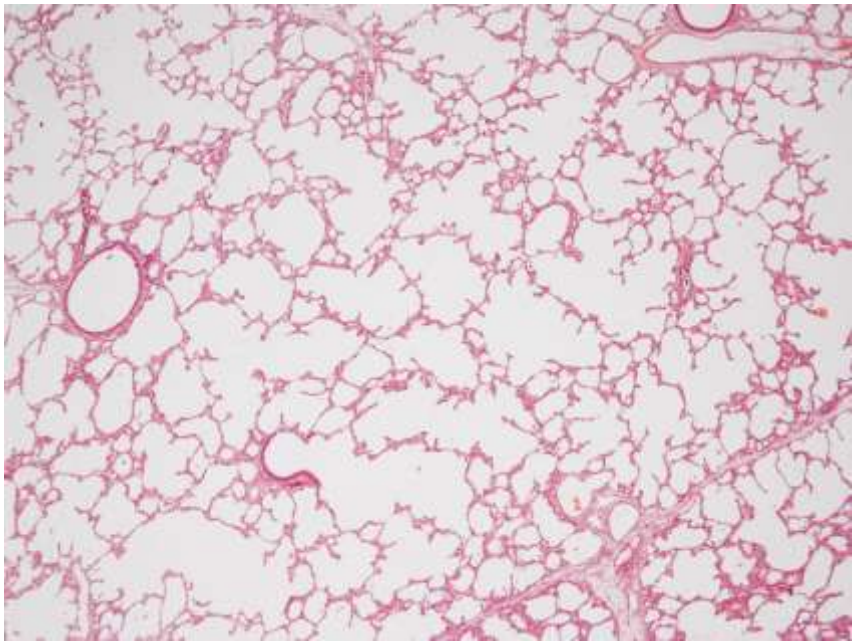

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- Quiste broncogénico (QB).

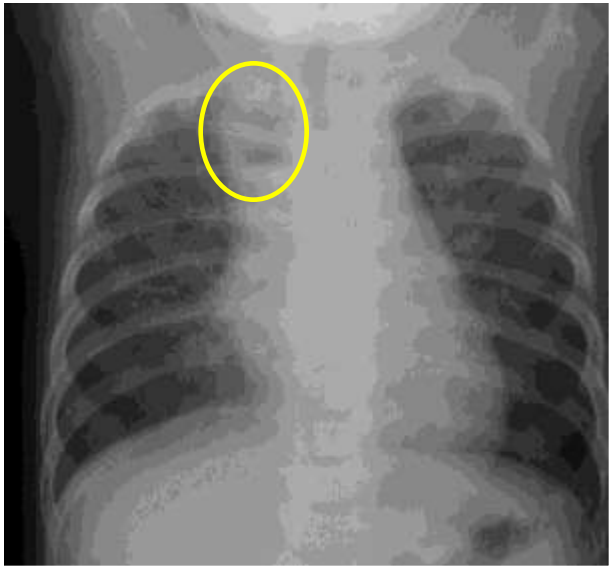
Anomalías del crecimiento pulmonar: hipoplasia, hiperplasia pulmonar.

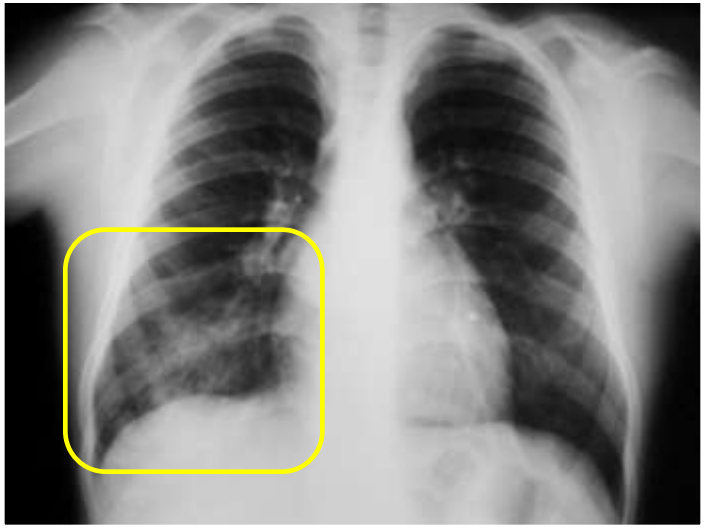
QUISTE BRONCOGÉNICO(QB)

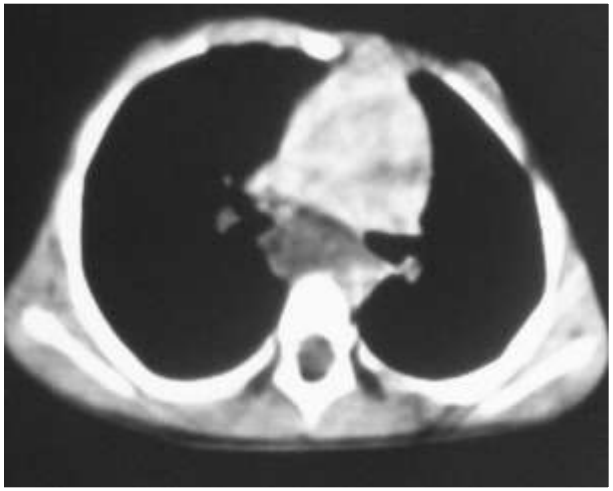
- = Quiste unilocular, única repeta de moco o líquido.No conexión bronquial.

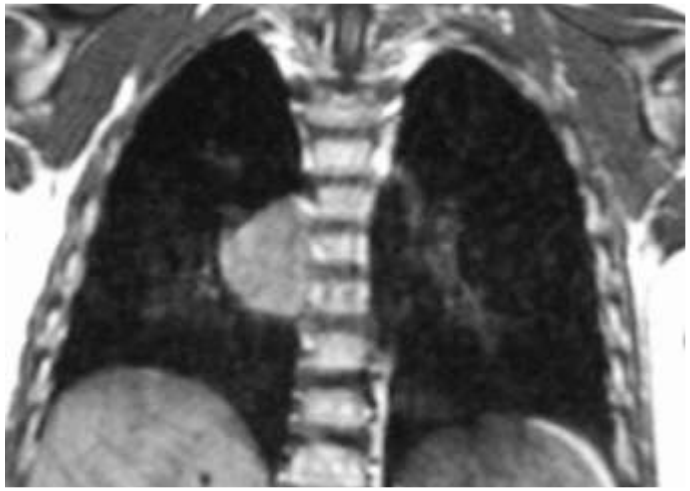
- Mediastino
- Pulmonar (hiliar,parénquima)
- Extratorácica.
- Asintomáticos, compresión VA, infección, hemoptisis, neumotórax.

QB

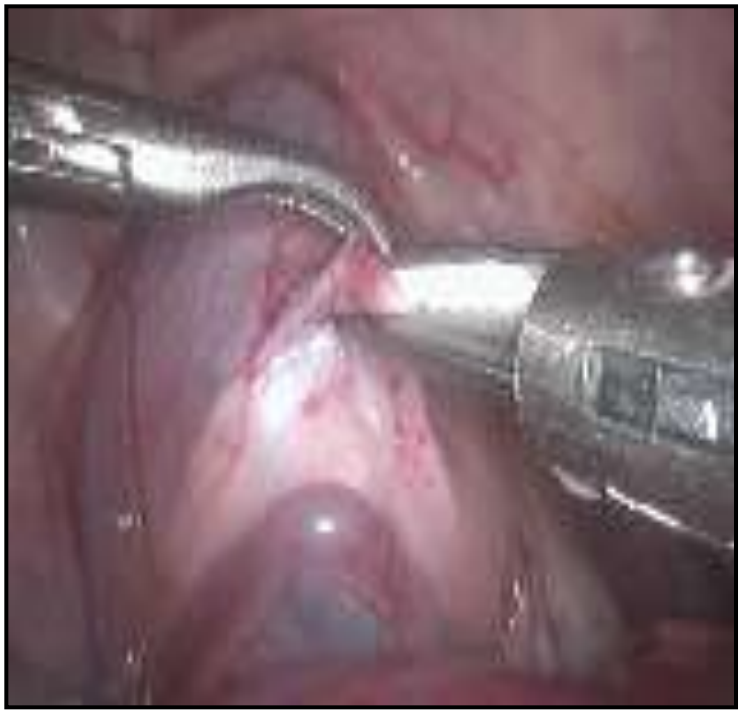


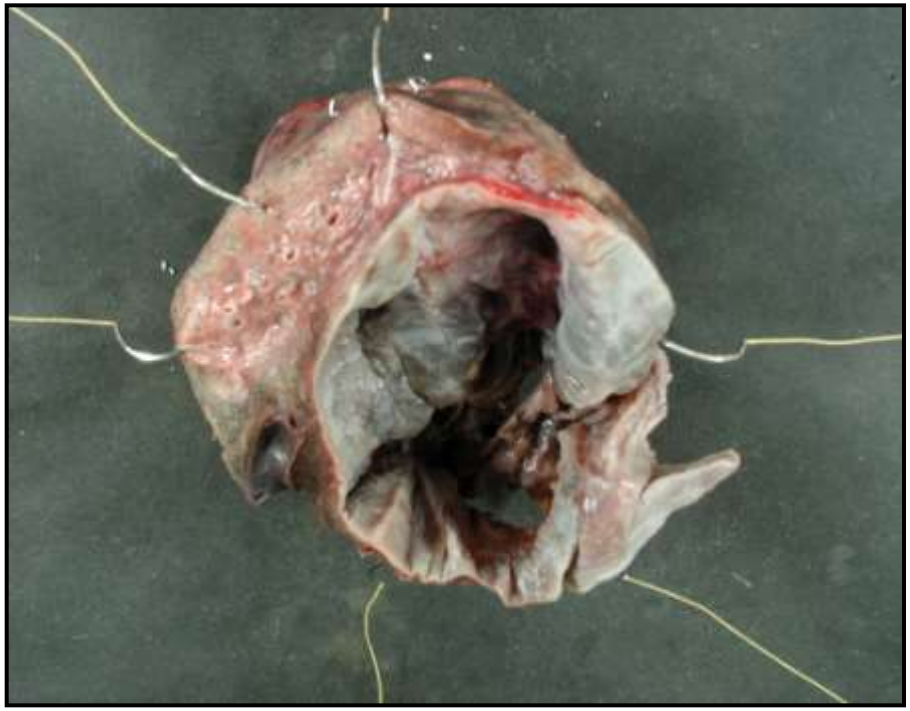




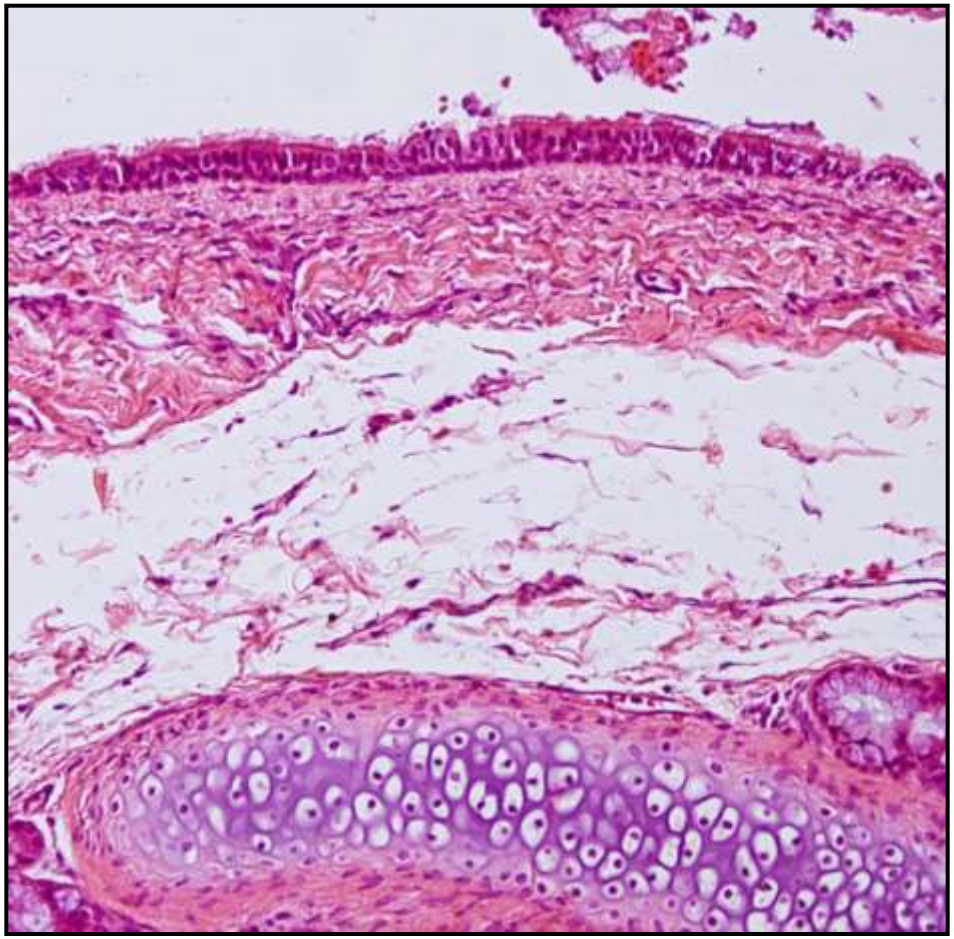


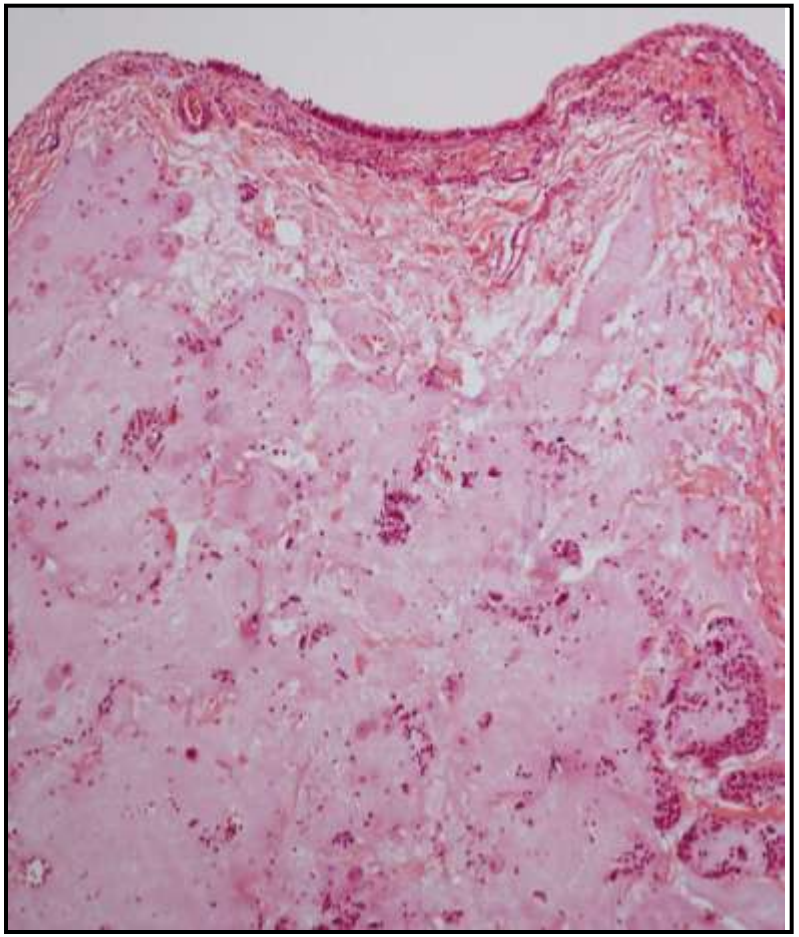
QB





QB





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Anomalías del crecimiento pulmonar: hipoplasia, hiperplasia pulmonar.

HIPOPLASIA PULMONAR

- = Defecto del desarrollo con tamaño pulmonar disminuido.

- *Peso pulmonar.*
- *Ratio peso pulmonar/peso corporal (0.222+/-0.002).*
- *Recuento alveolar radial (RAC) (4.4+/-0.9)*

HIPOPLASIA PULMONAR

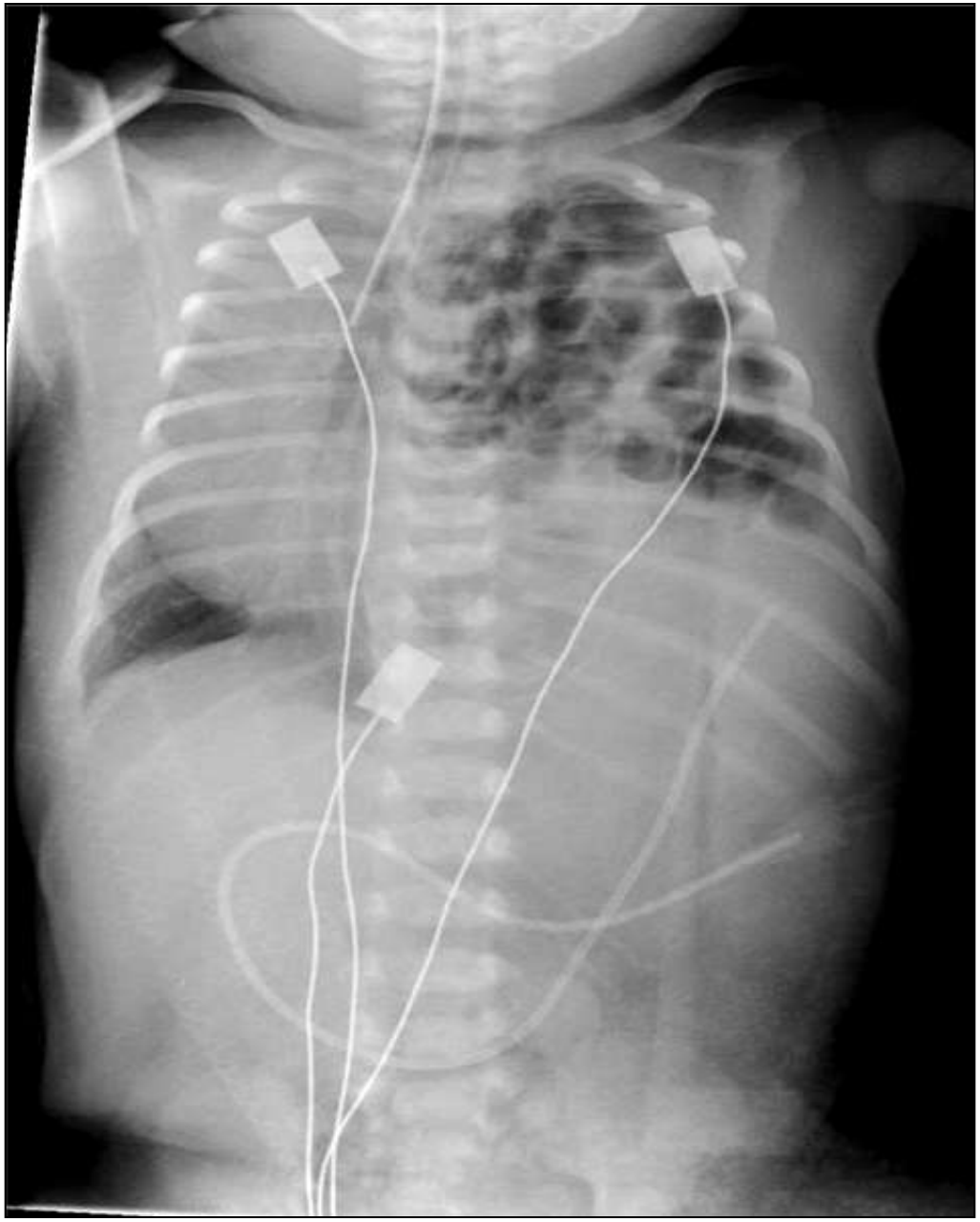
- 10% autopsias neonatales. 85% asociada a otras malformaciones.
- Reducción del espacio torácico extrínseco o intrínseco
- Formas primarias o RPPM.

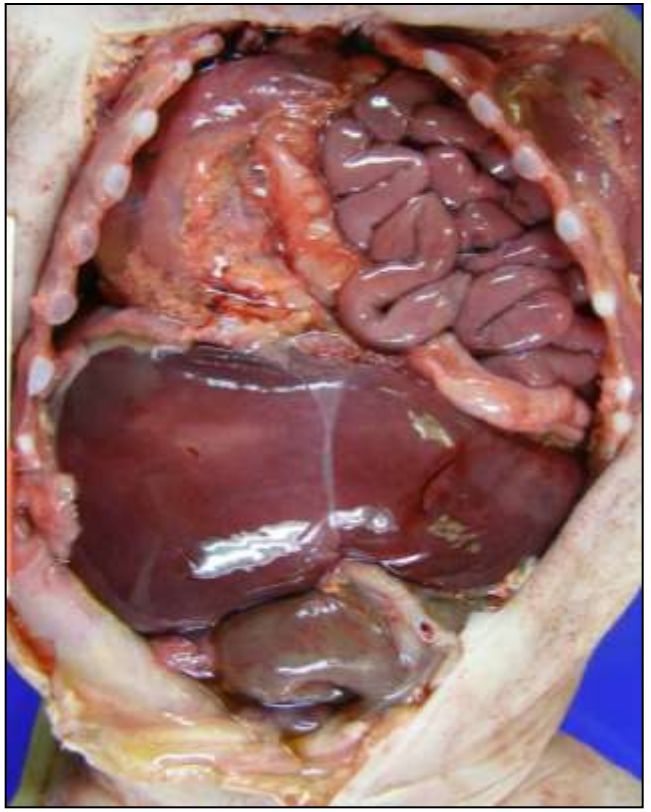
Anomalies Associated with Pulmonary Hypoplasia

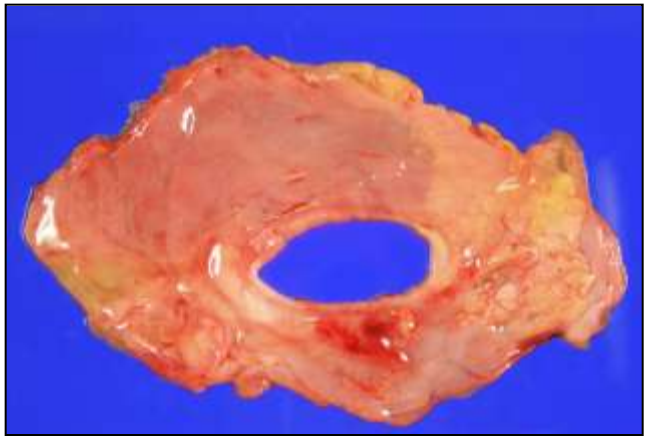
Common
- Diaphragmatic hernia
- Renal agenesis, bilateral
- Renal dysgenesis, bilateral
- Obstructive uropathy
- Polycystic renal disease (Potter, type I)
- Large abdominal wall defects

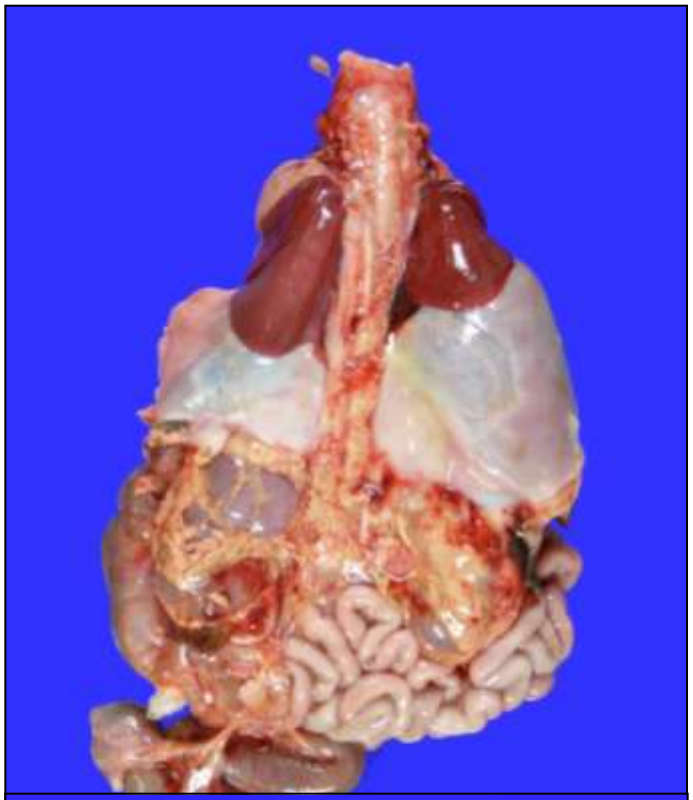
Infrequent
- Diaphragmatic hypoplasia or eventration
- Hemolytic disease of the newborn
- Pleural effusion, as with nonimmune fetal hydrops
- Musculoskeletal abnormalities such as thoracic dystrophies
- Anencephaly
- Scimitar syndrome
- Chromosomal anomalies, including trisomy 13, 18, and 21

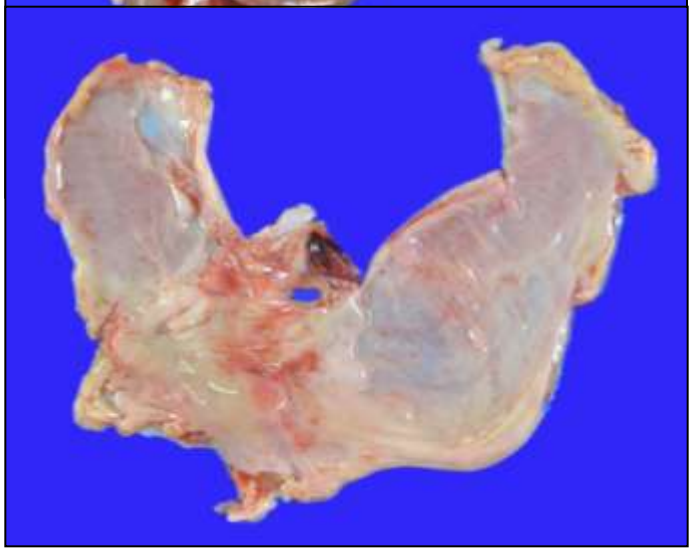
Rare
- Abdominal pregnancy
- Ascites secondary to congenital cytomegalovirus infection
- Cloacal dysgenesis
- Congenital hydropericardium
- Down's syndrome
- Glutaric acidemia, type II
- Laryngotracheoesophageal cleft
- Neonatal hypophosphatasia
- Pena-Shokeir I syndrome
- Phrenic nerve agenesis
- Right-sided cardiovascular malformation as with hypoplastic right heart
- Thoracic neuroblastoma
- Upper cervical spinal cord
- Extralobar sequestration

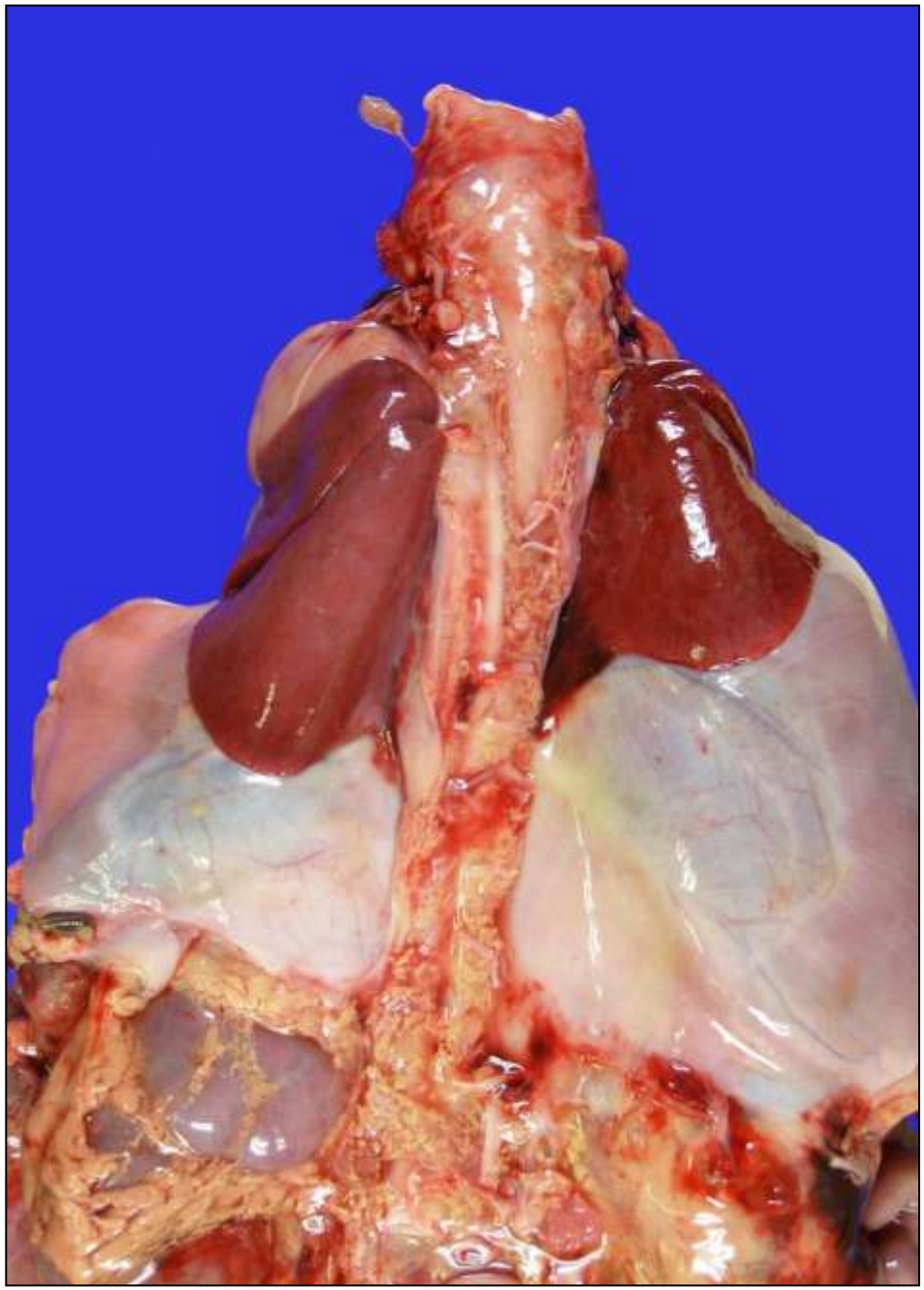


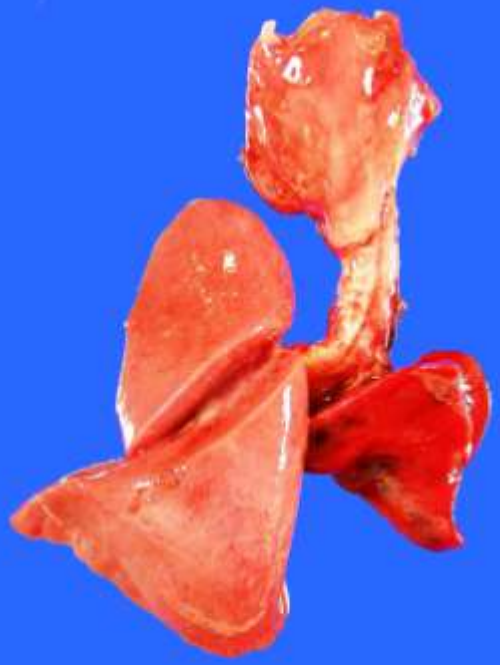












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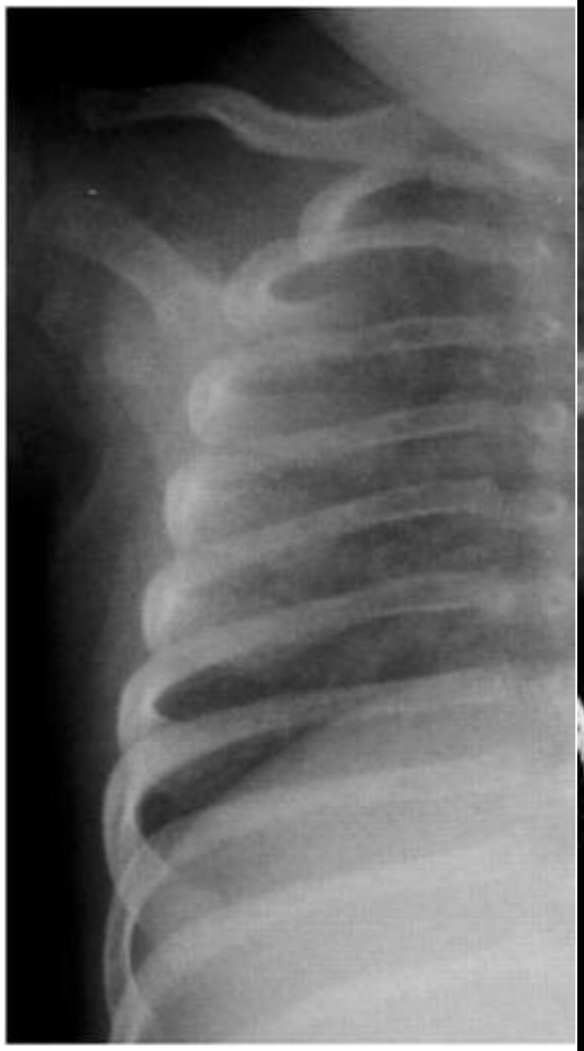
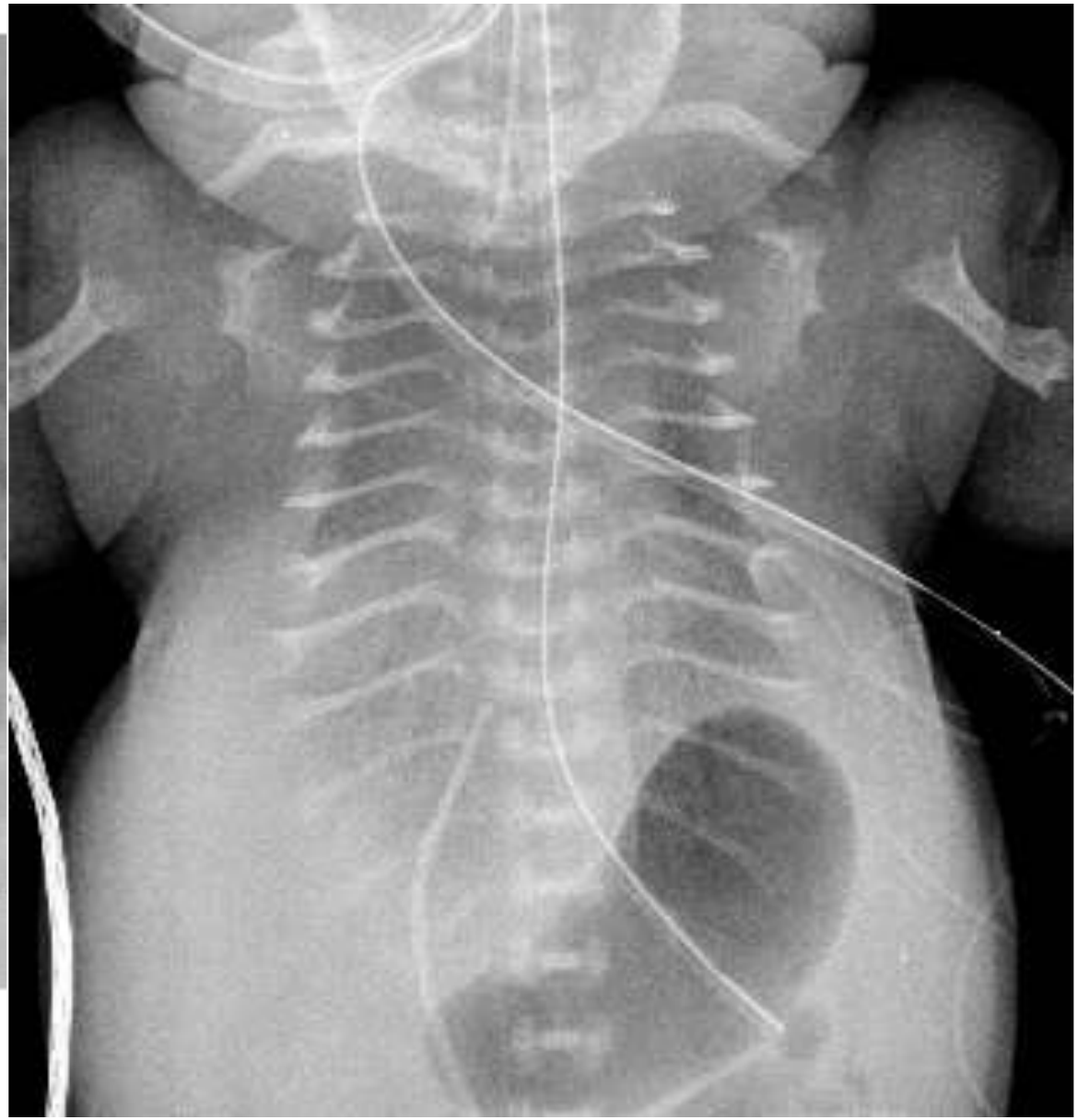
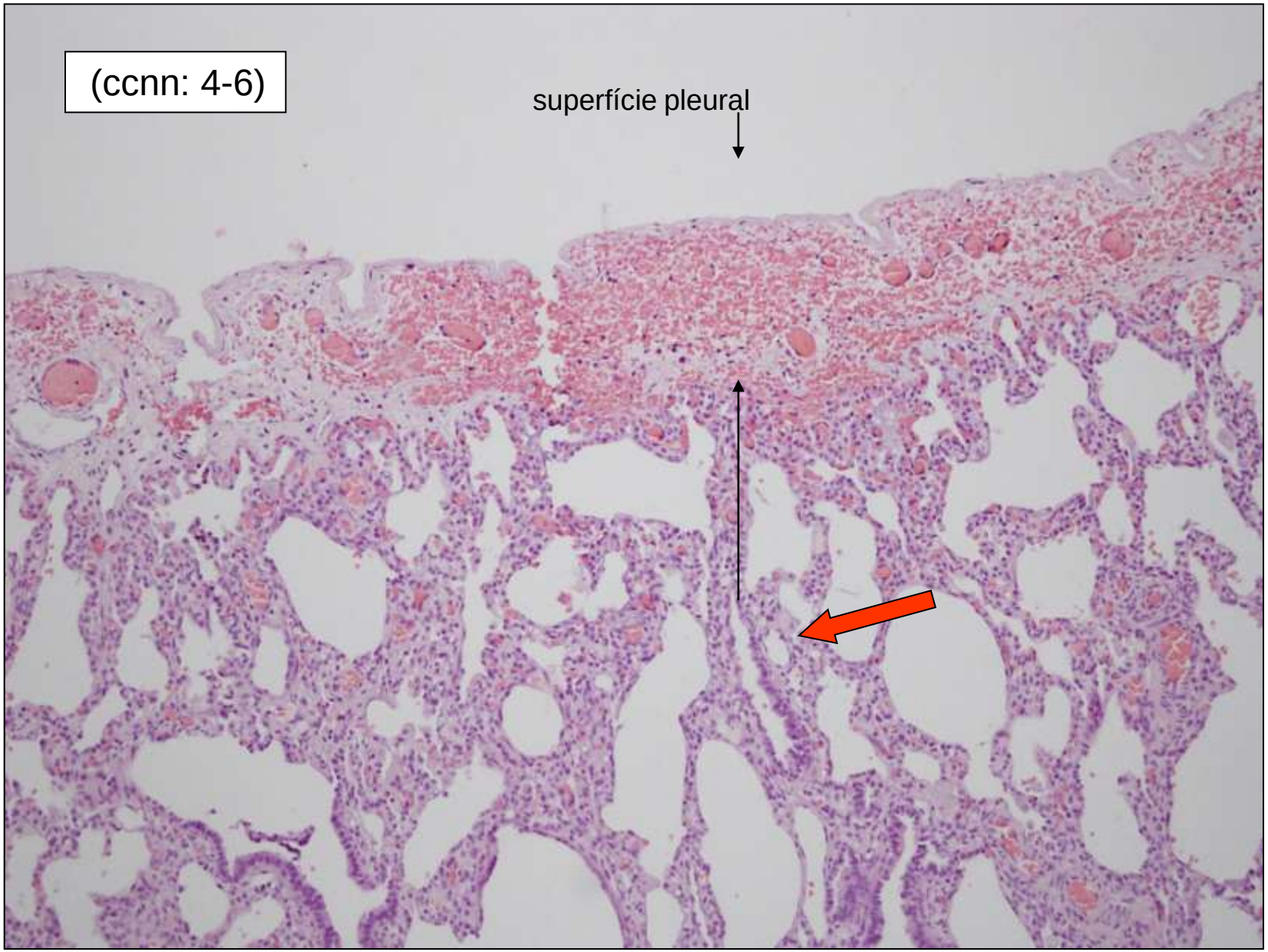


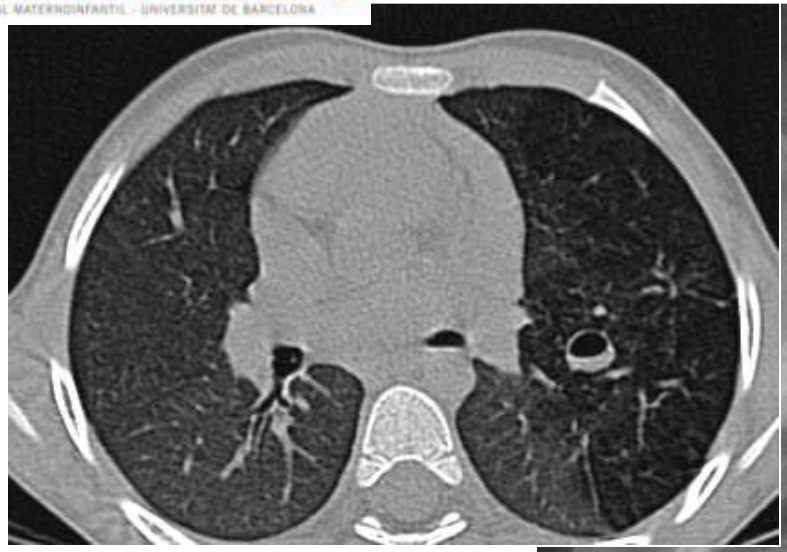


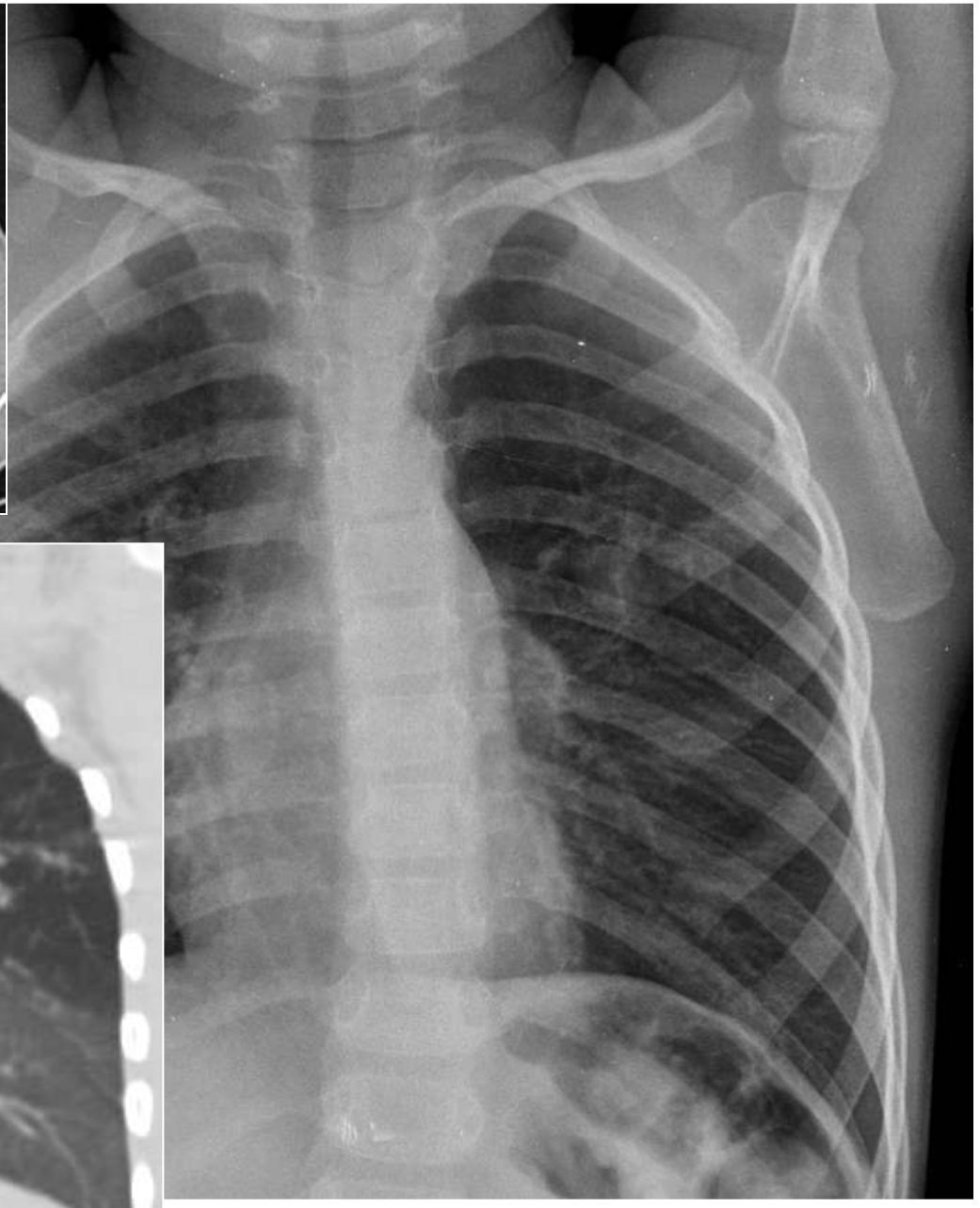

(ccnn: 4-6)
superfície pleural

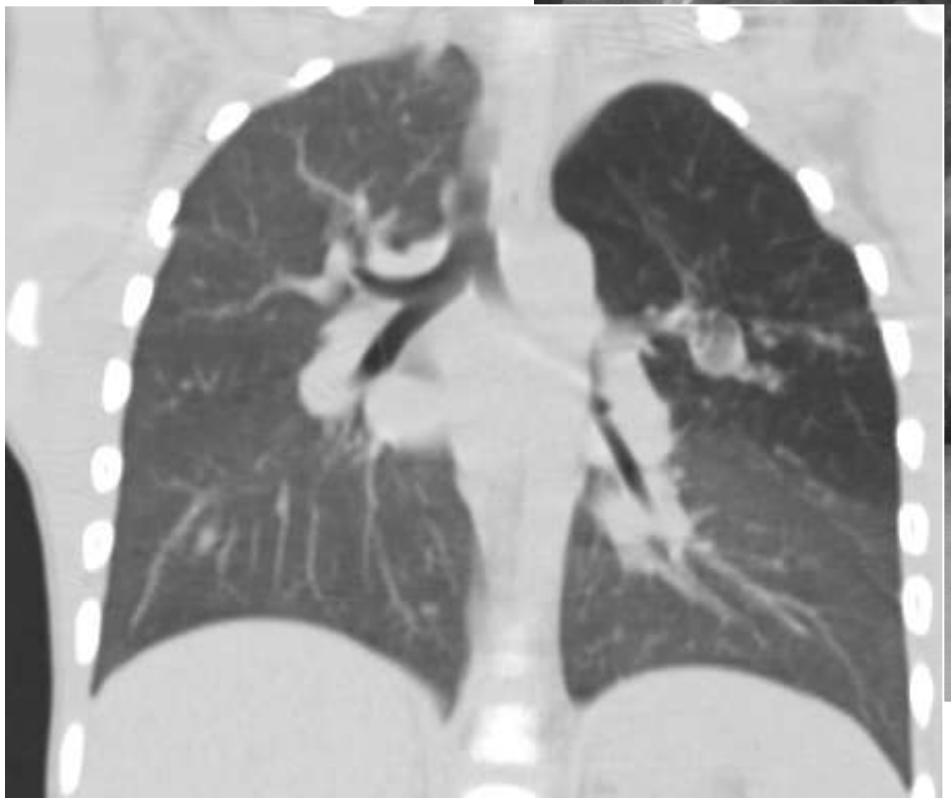
ATRESIA BRONQUIAL

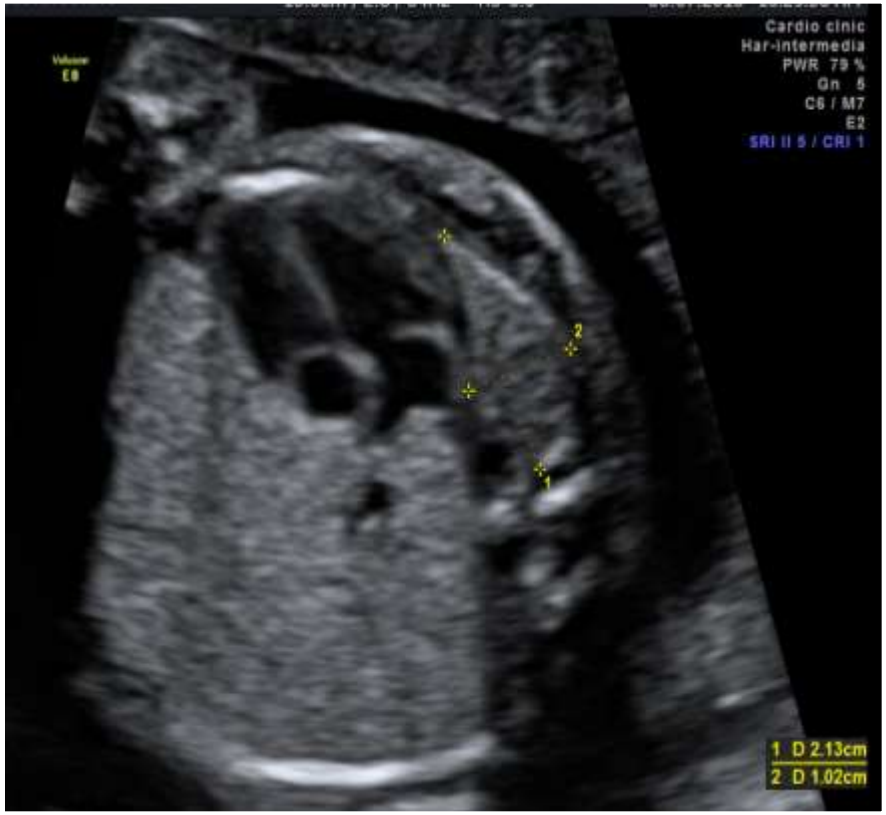
- Niños, adolescentes,adultos.LIS, bronquio segmentario.
- US: diagnóstico prenatal-neonatal.

- *Mucocele*
- *Cambios obstructivos*
- *Hiperplasia pulmonar*
- *Cambios microquísticos*

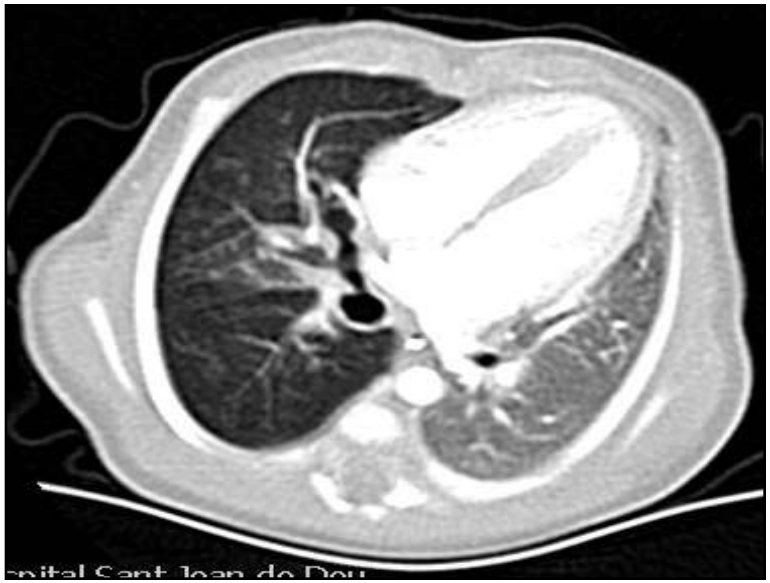


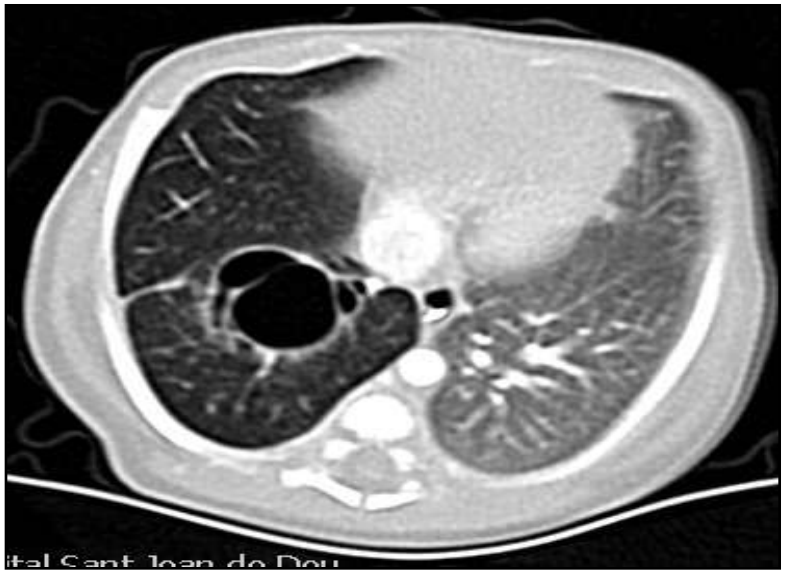






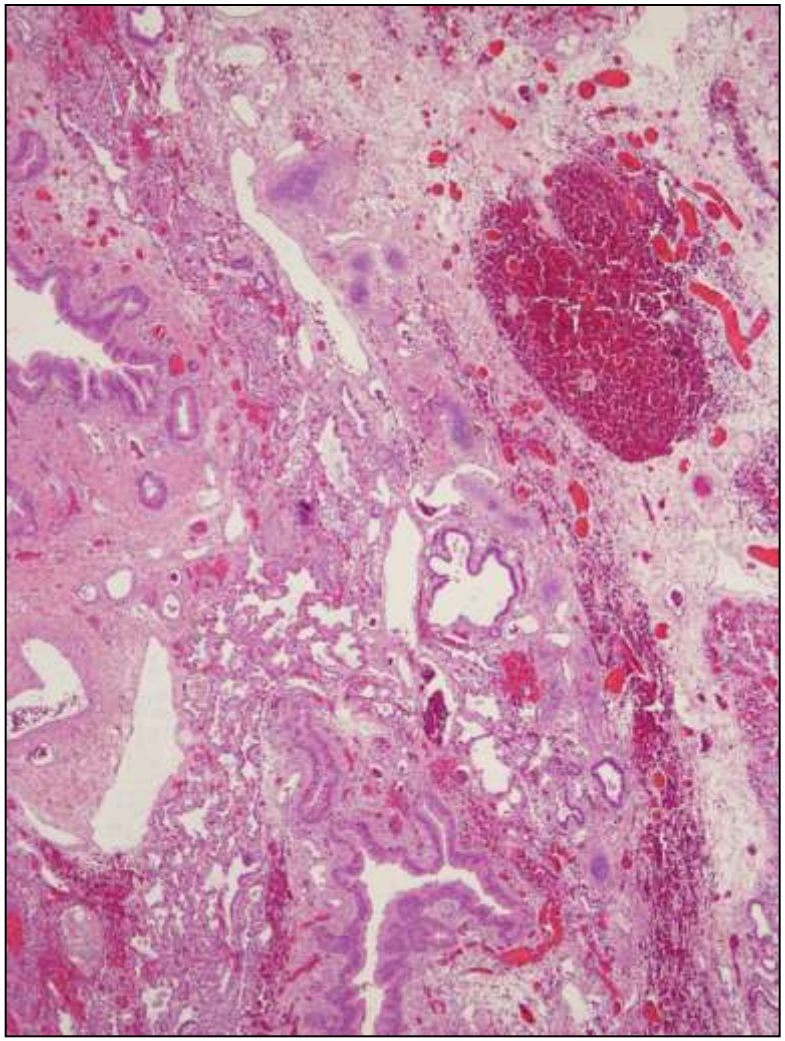
Eco prenatal

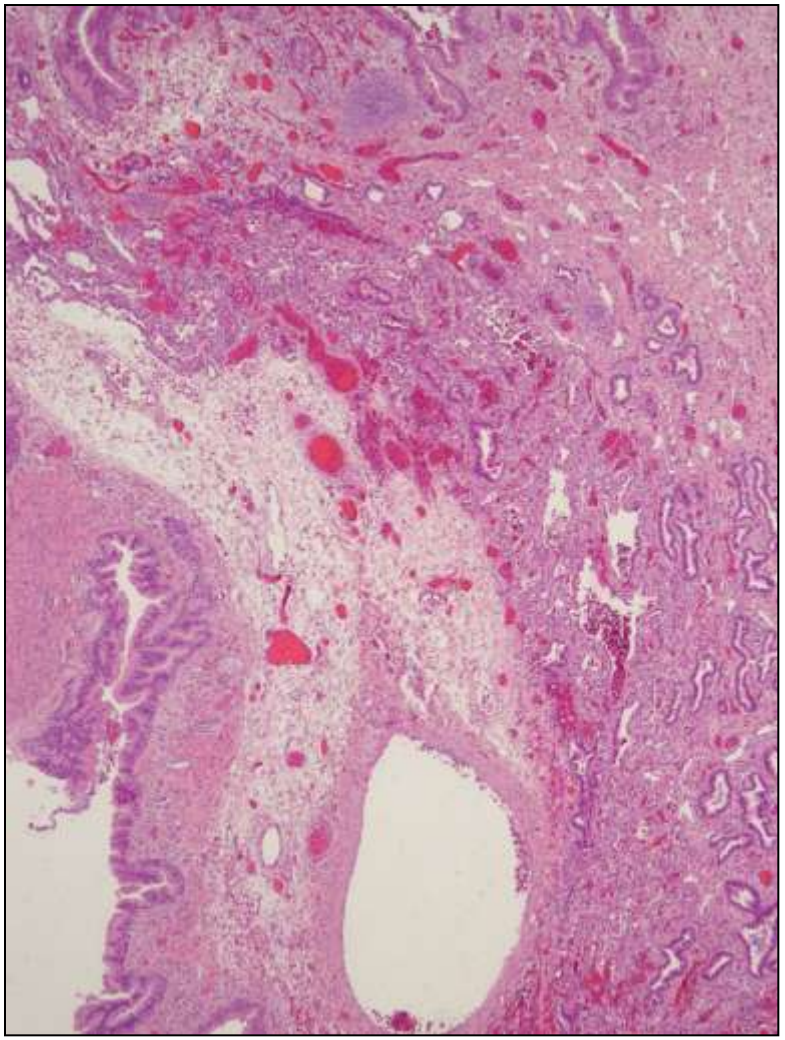




Status post tto laser

ATRESIA BRONQUIAL





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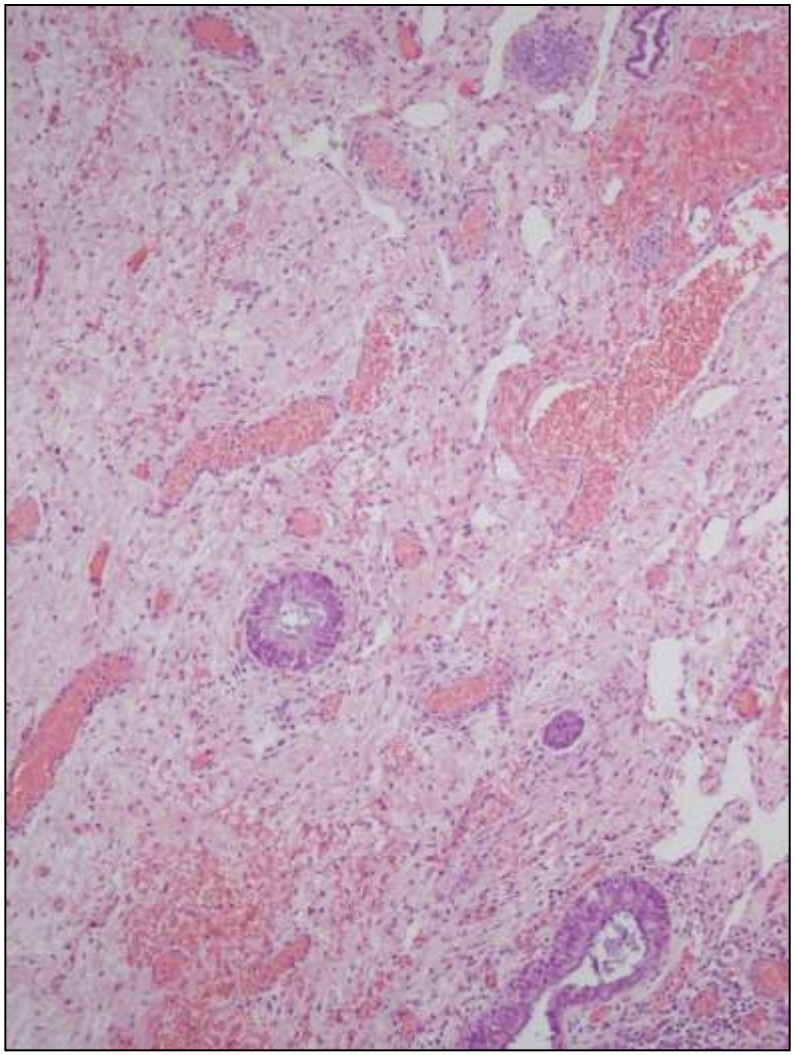

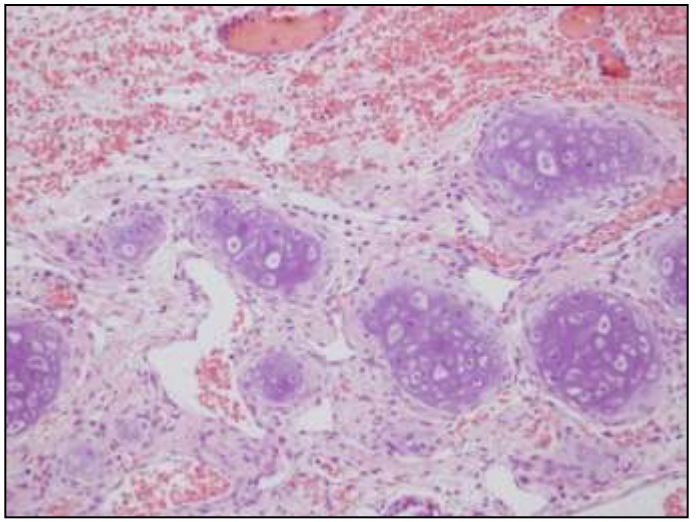

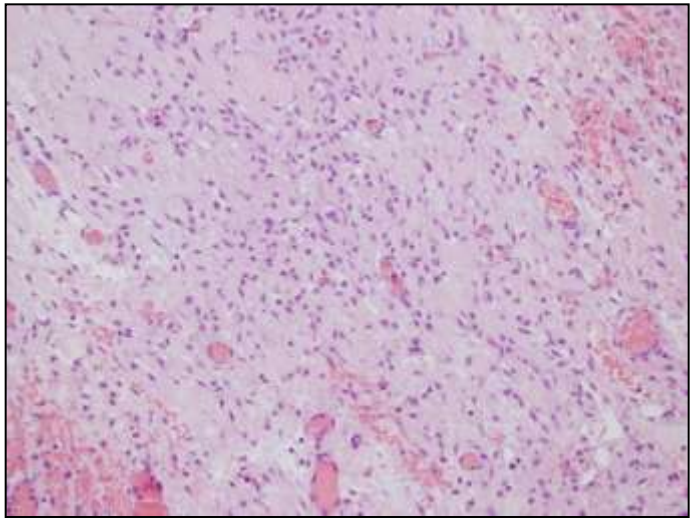

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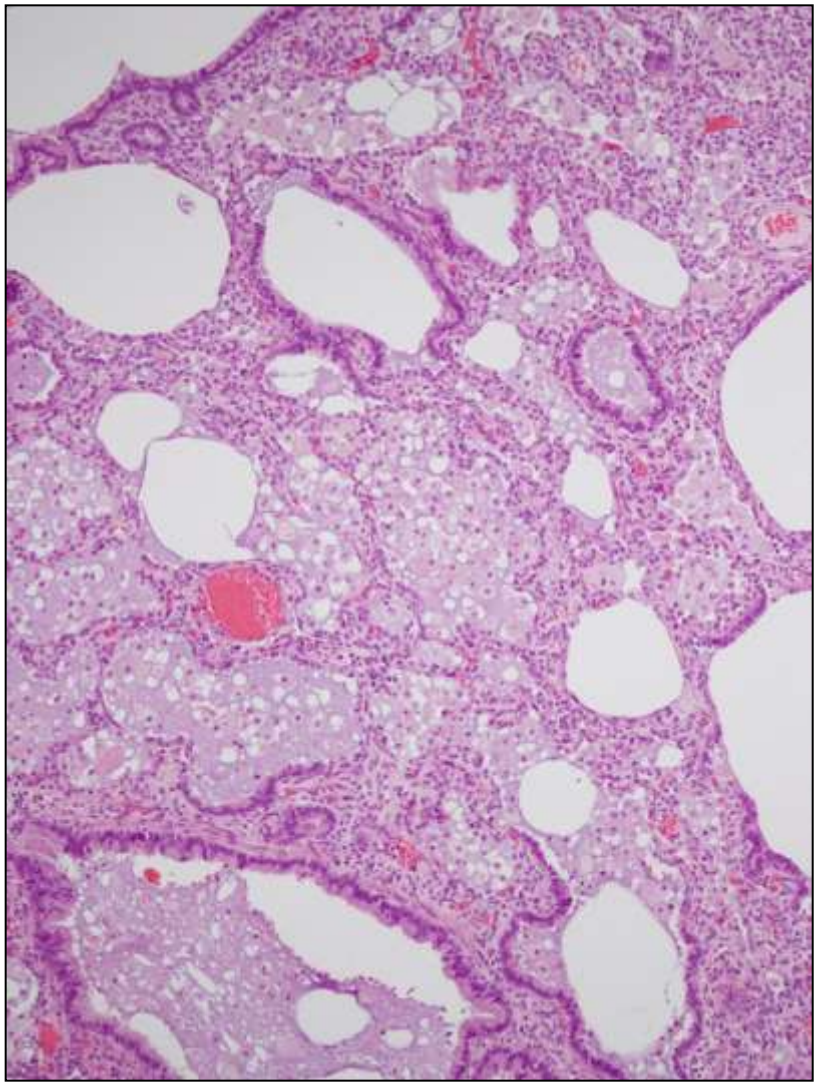

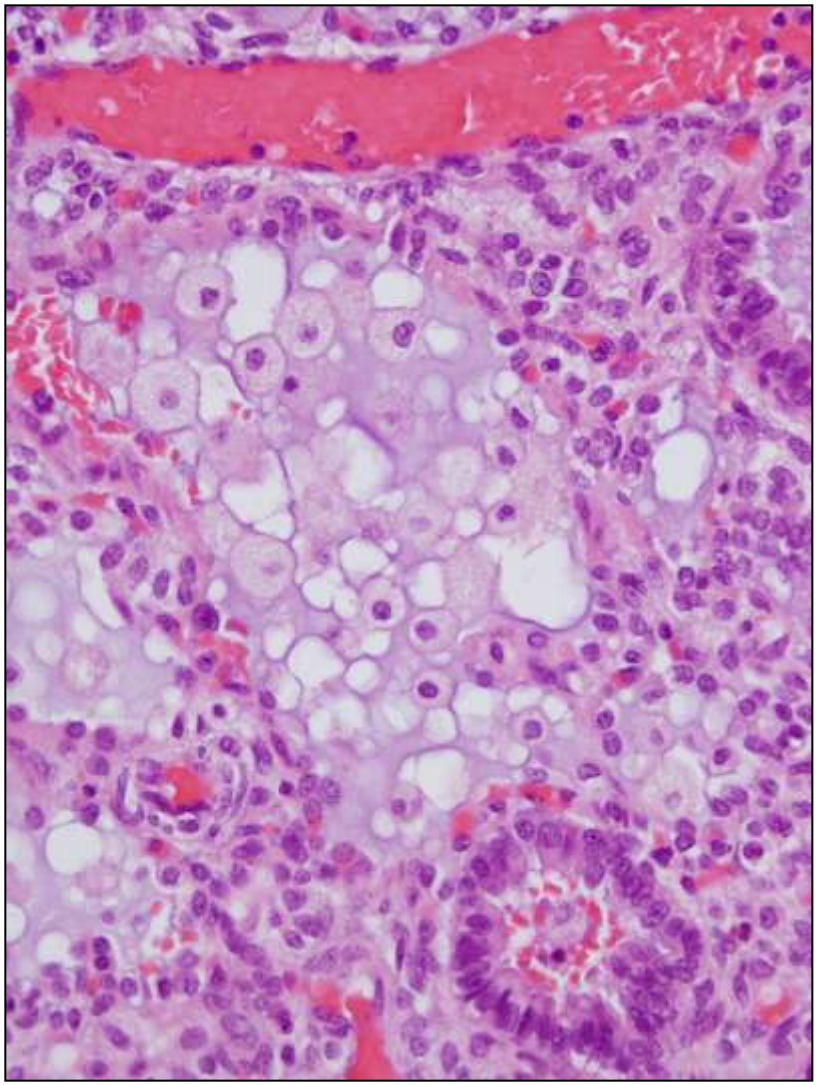

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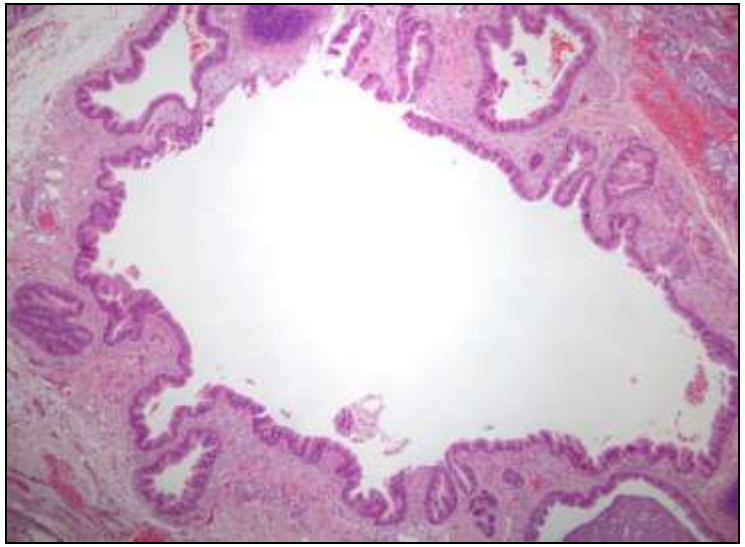

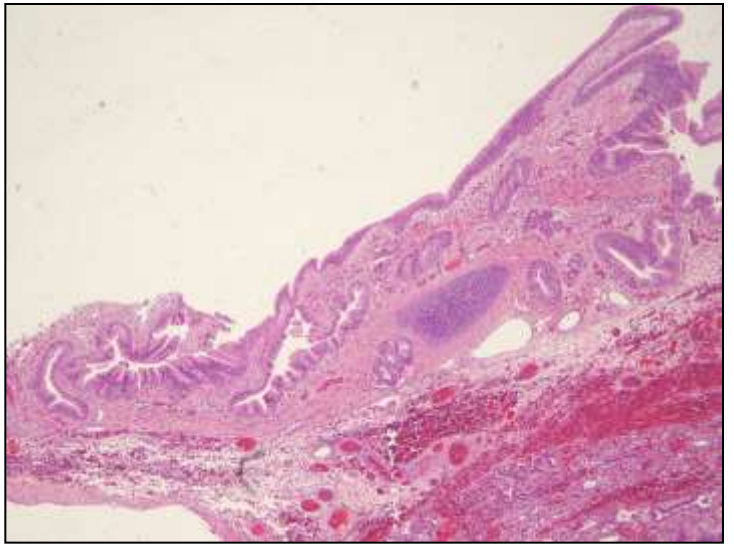

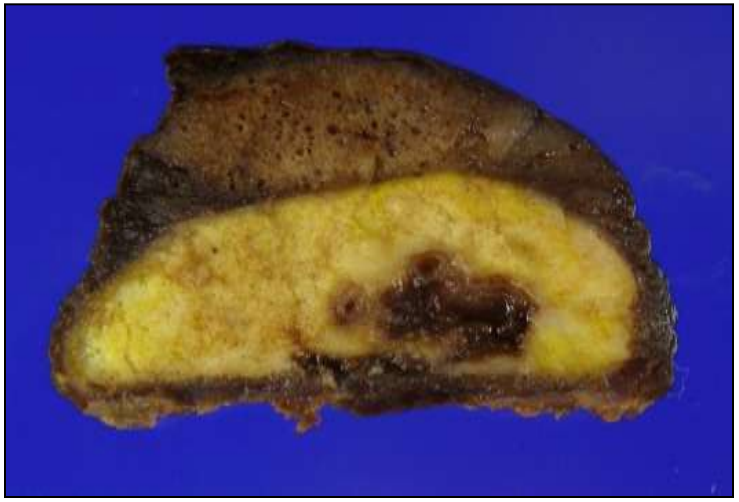

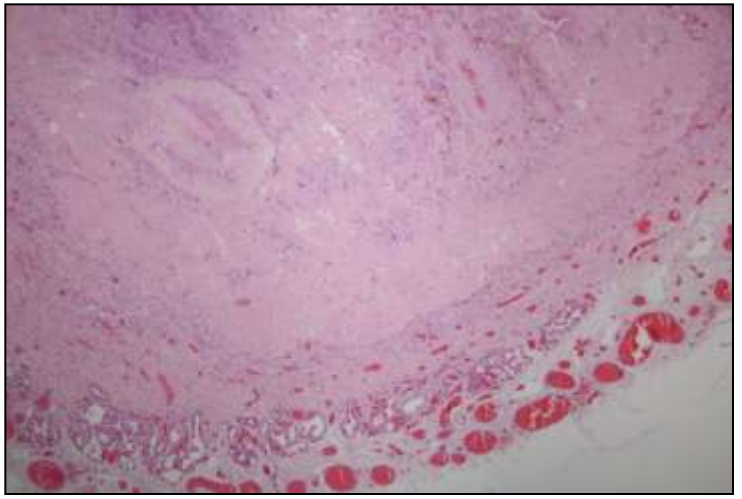

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DOI: 10.2350/06-01-0023.1

Bronchial Atresia Is Common to Extralobar Sequestration, Intralobar Sequestration, Congenital Cystic Adenomatoid Malformation, and Lobar Emphysema

Wolfram F.J. Riedlinger,[1] Sara O. Vargas,[1] Russell W. Jennings,[2,3] Judy A. Estroff,[3,4] Carol E. Barnewolt,[3,4] Craig W. Lillehei,[2,3] Jay M. Wilson,[2,3] Andrew A. Colin,[5] Lynne M. Reid,[1] and Harry P.W. Kozakewich[1*]

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Journal of Pediatric Surgery (2006) 41, 61–65

Bronchial atresia: the hidden pathology within a spectrum of prenatally diagnosed lung masses

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[b]*Department of Radiology, Children's Hospital Boston and Harvard Medical School, Boston, MA 02115, USA*
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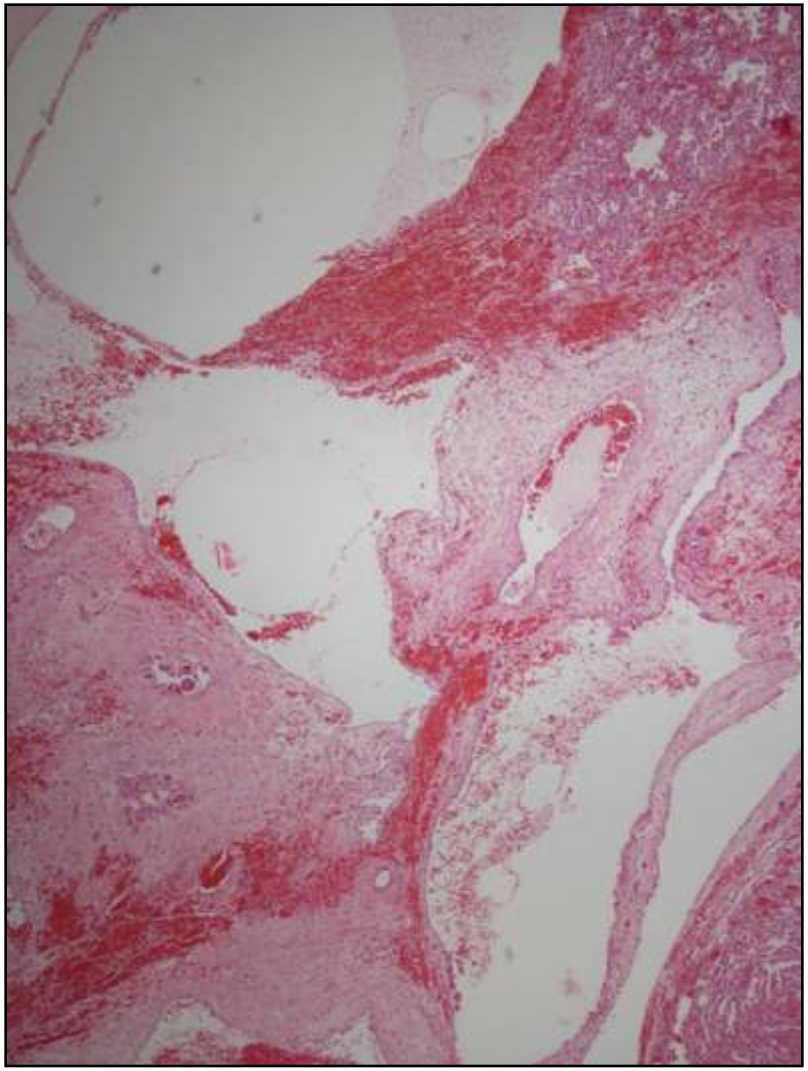
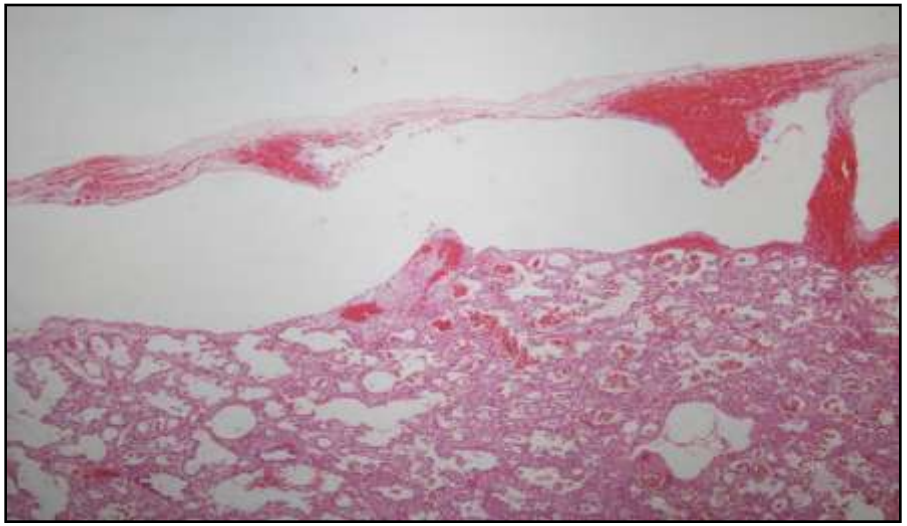
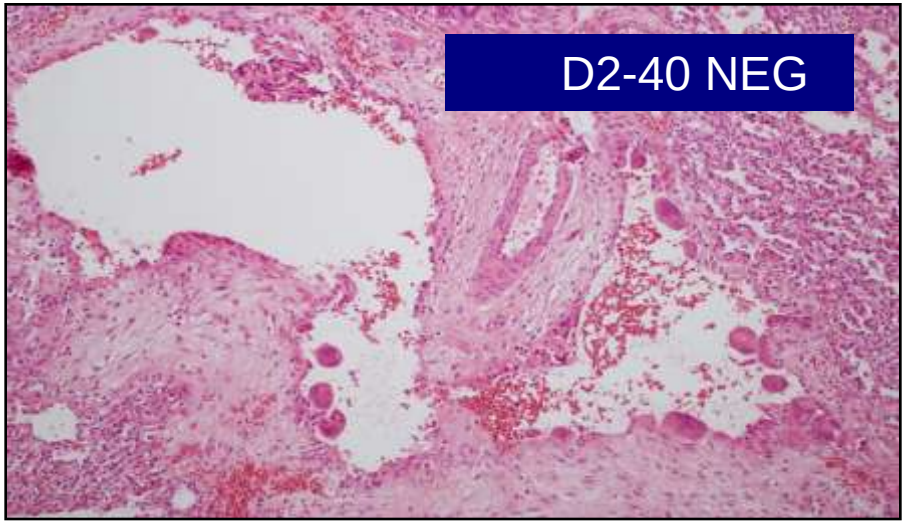
unexplored. More recently, pathologists have shown that the diagnosis of bronchial atresia often requires special diagnostic techniques, including specimen bronchography and microdissection [5]. Bronchography typically reveals lack of filling of the bronchus to the affected portion of the lung. The hallmarks of bronchial atresia on microdissection include a cystically dilated bronchus filled with mucus just distal to the point of atresia [7].

Imai and Mark [8] have made the observation that bronchial atresia is common in cystic lung disease.

ENFISEMA INTERSTICIAL







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Sant Joan de Déu