PATOLOGIA PULMONAR

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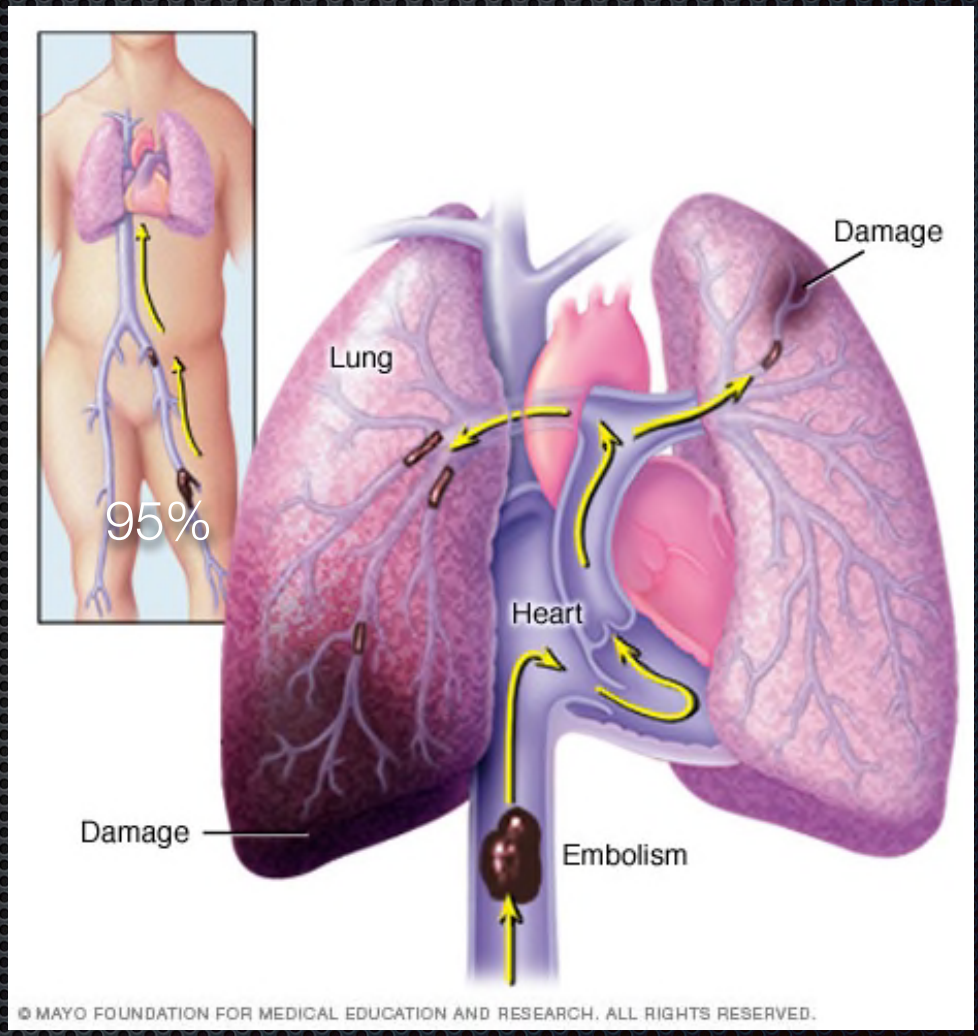
TROMBOEMBOLIA E INFARTO PULMONAR

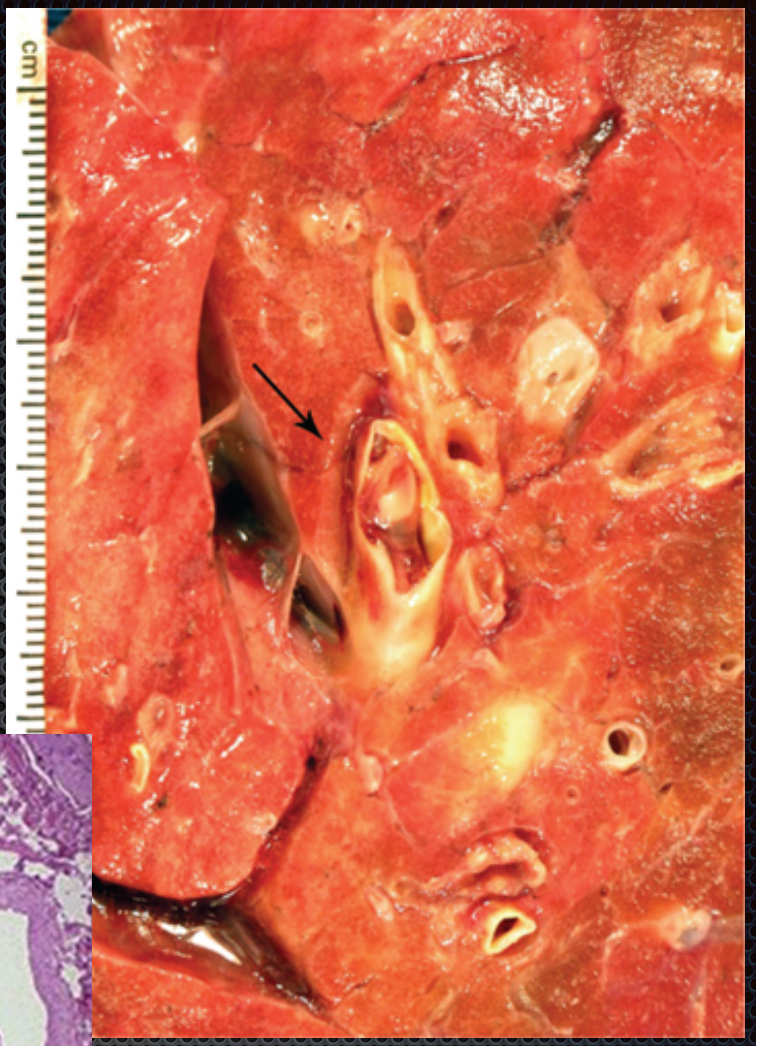
Oclusión arterial pulmonar debido a:

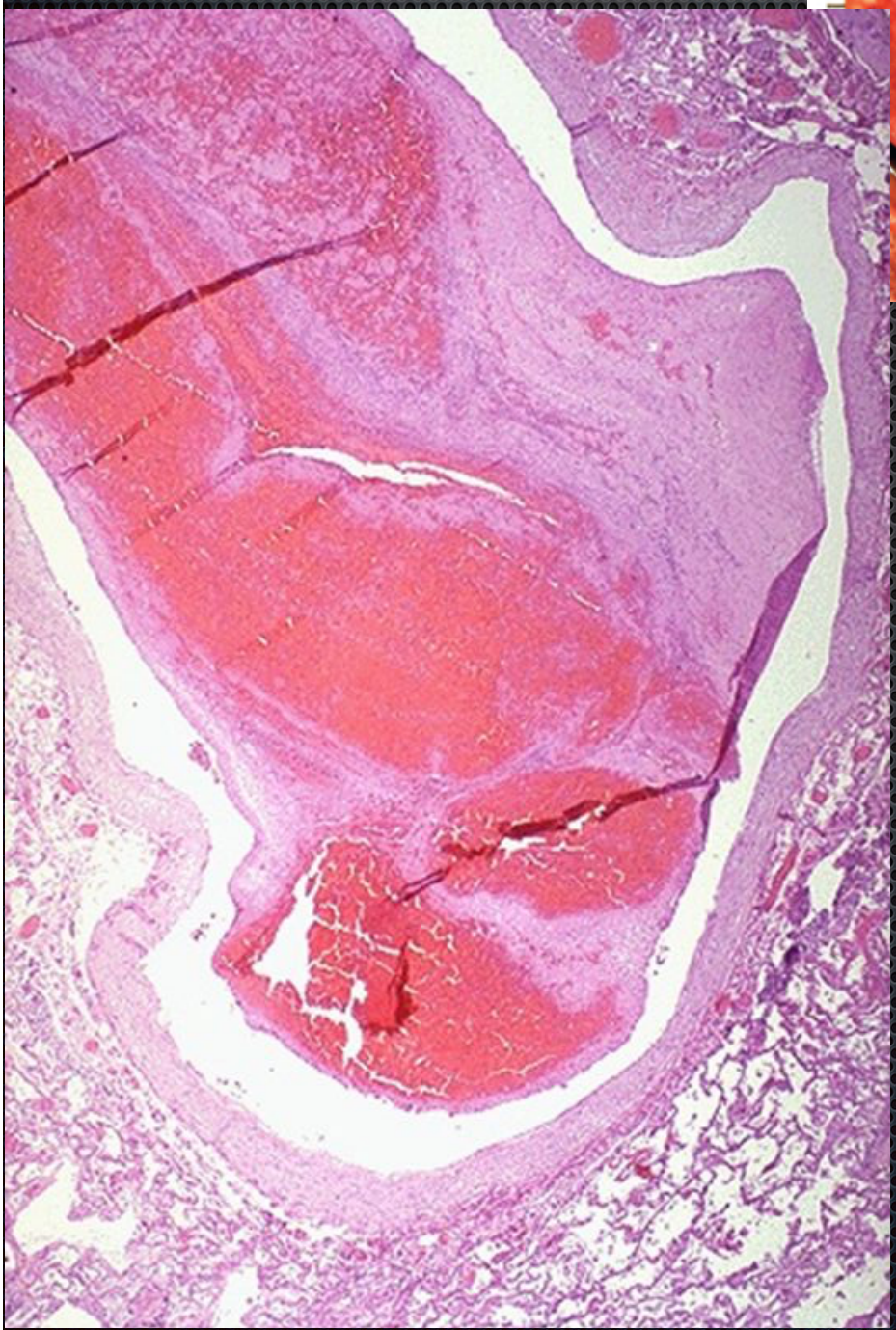
- Trombo (in situ)
- Émbolo (proveniente de la circulación venosa sistémica).

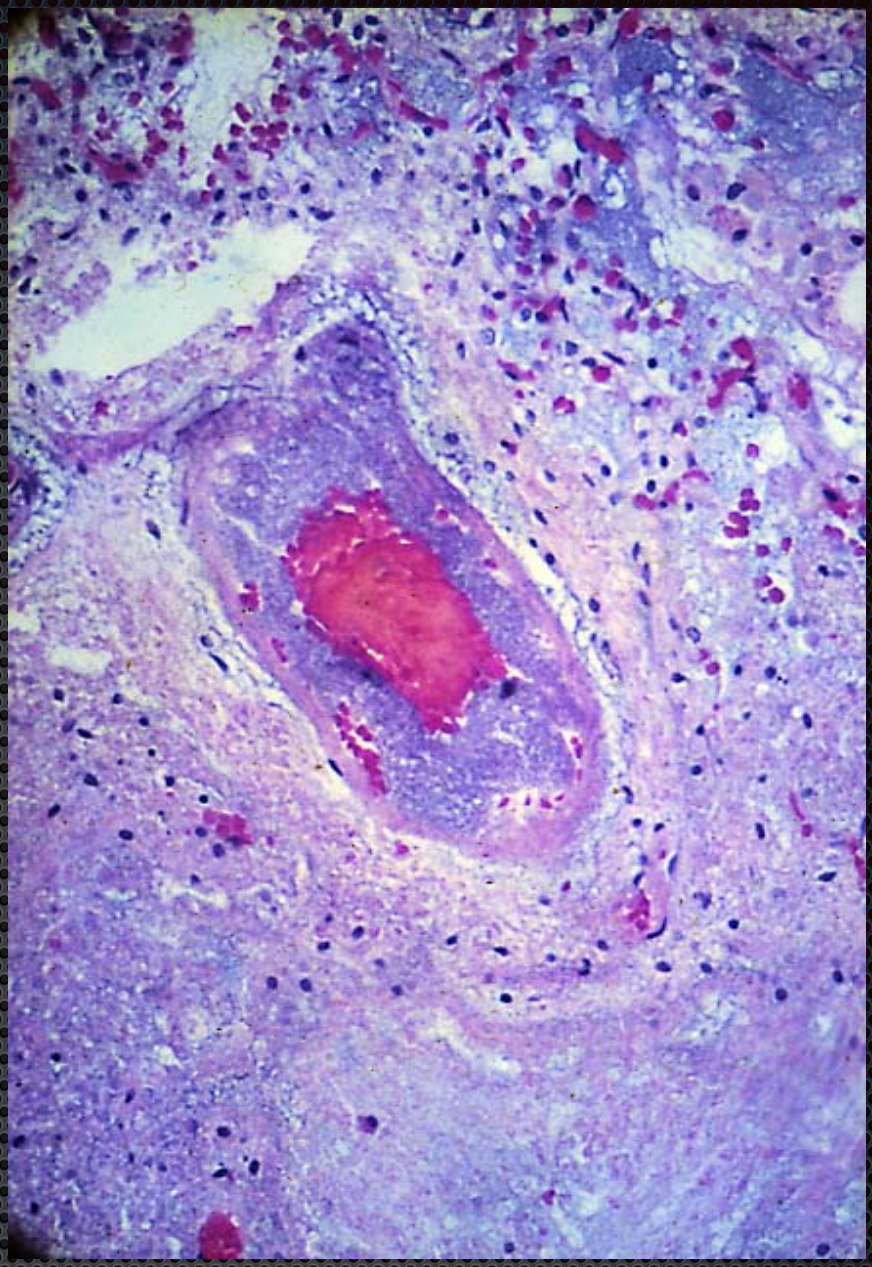
Muerte súbita

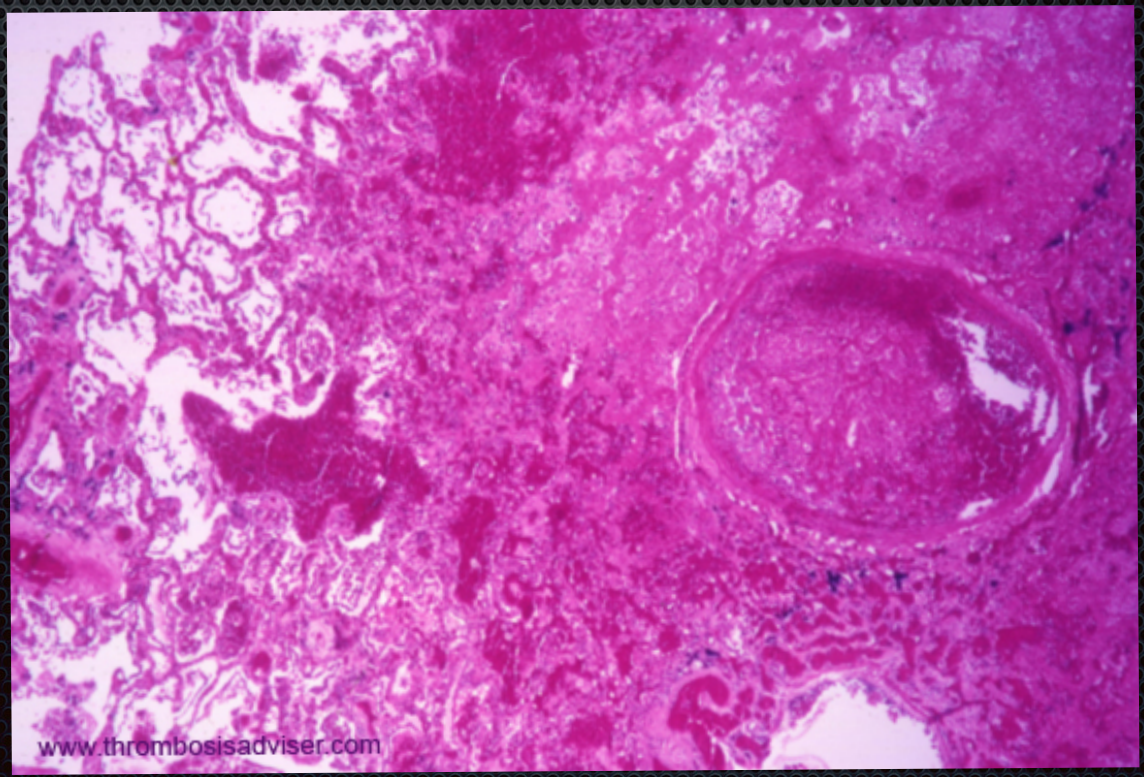
Desbalance de triada de Virchow. (H, DE, EV)




cm



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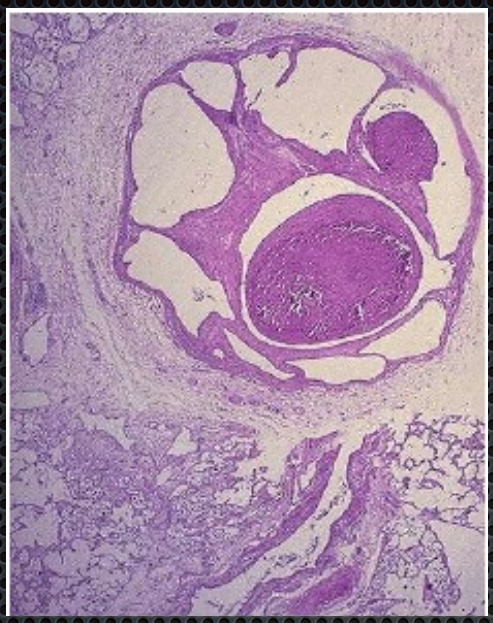
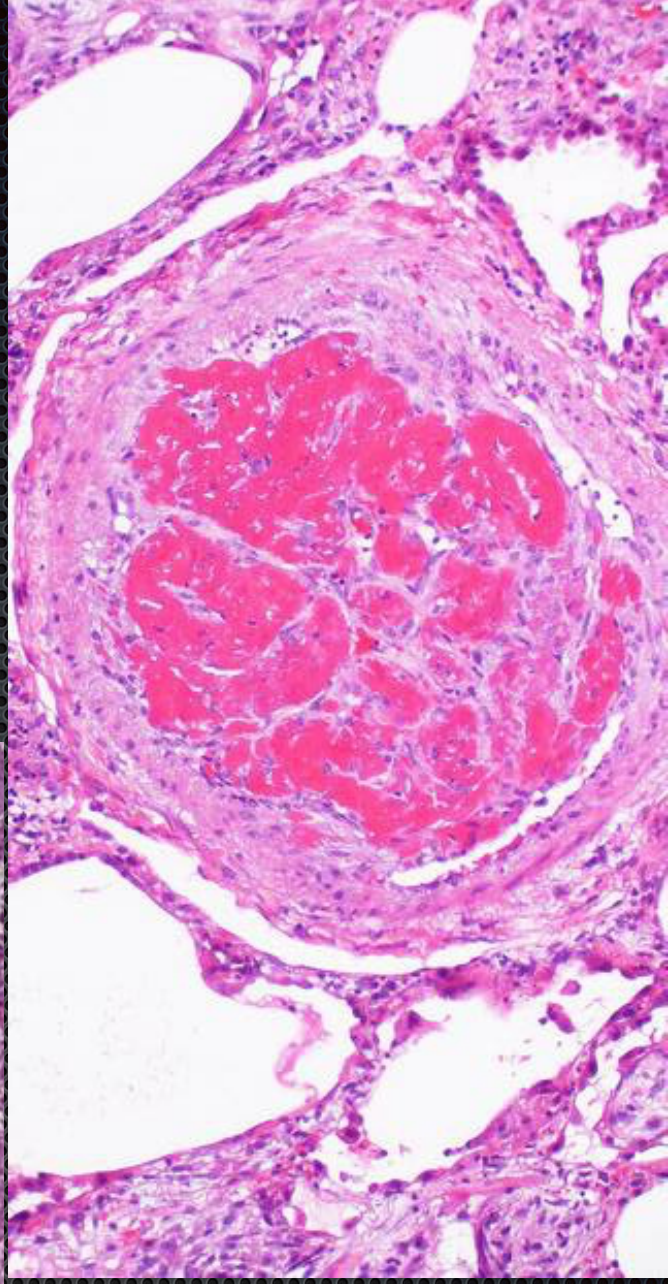
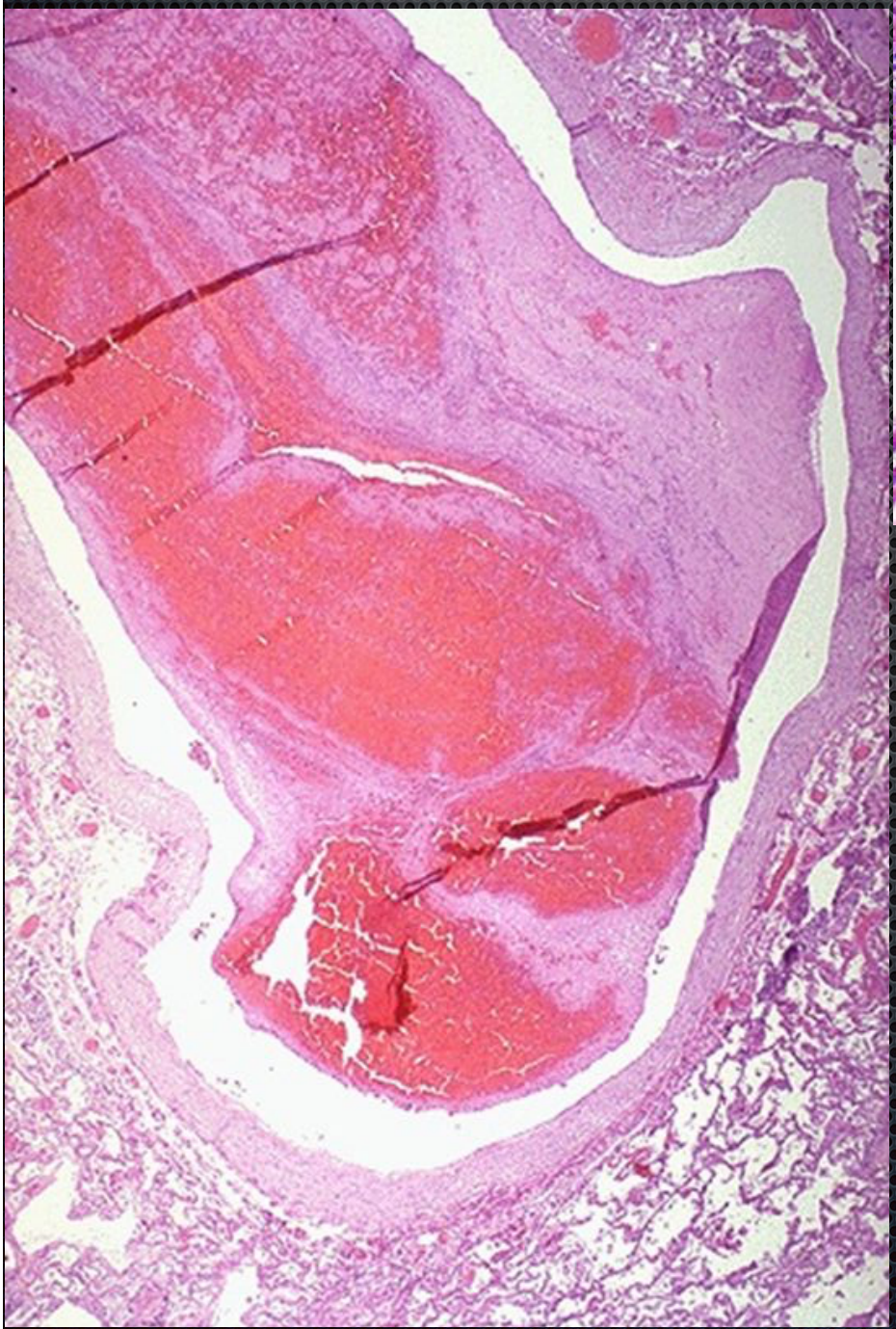
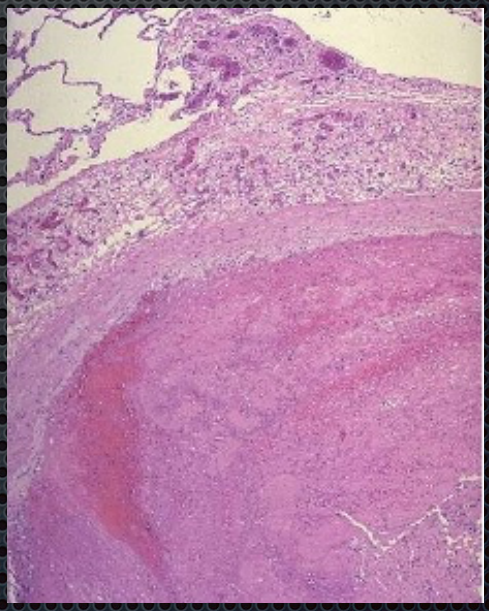


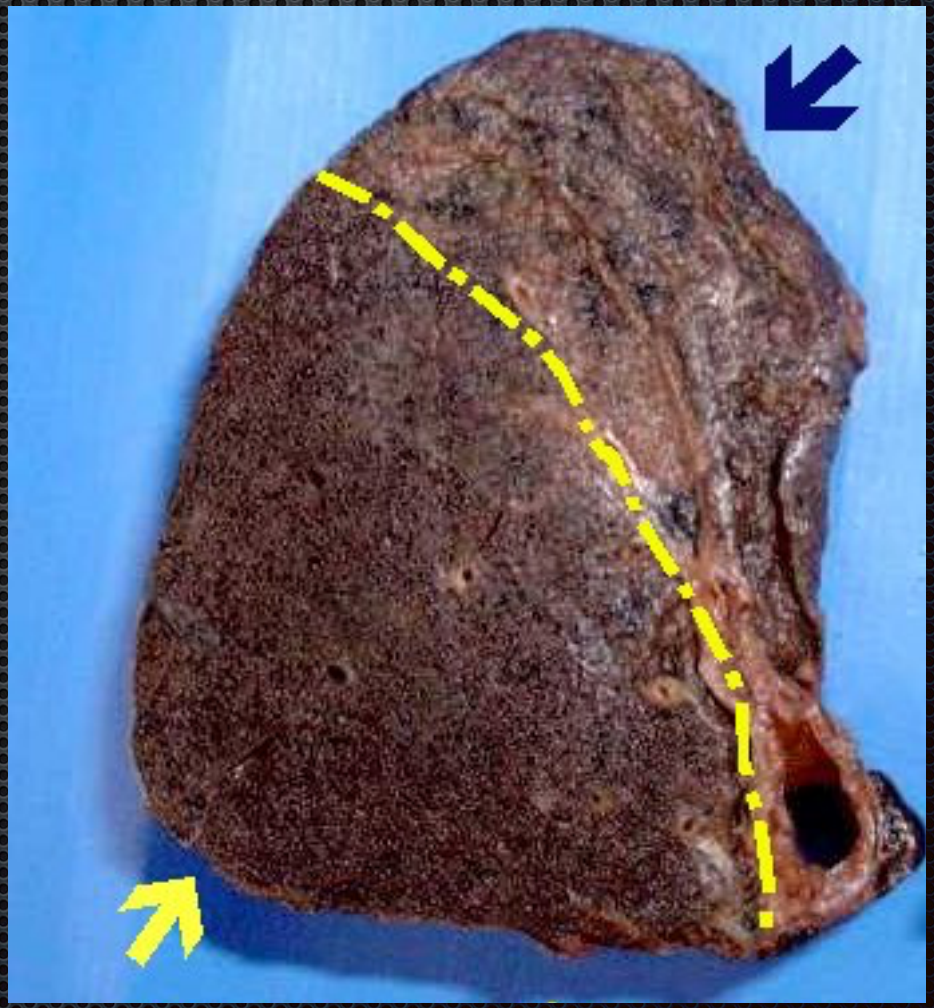




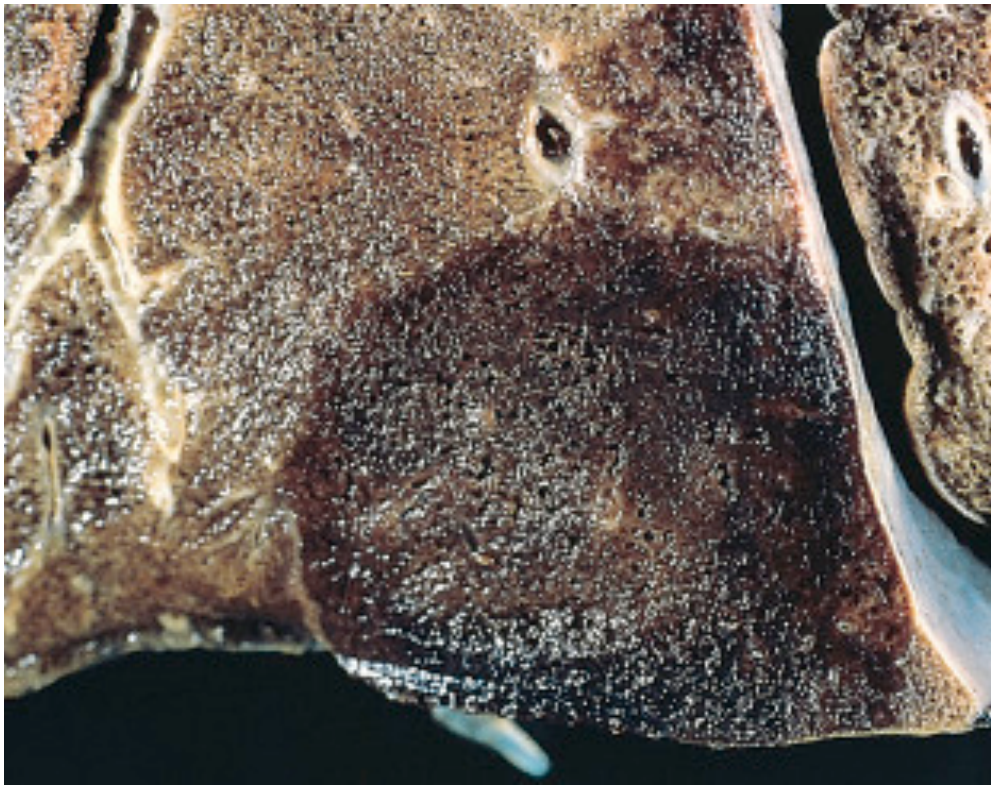


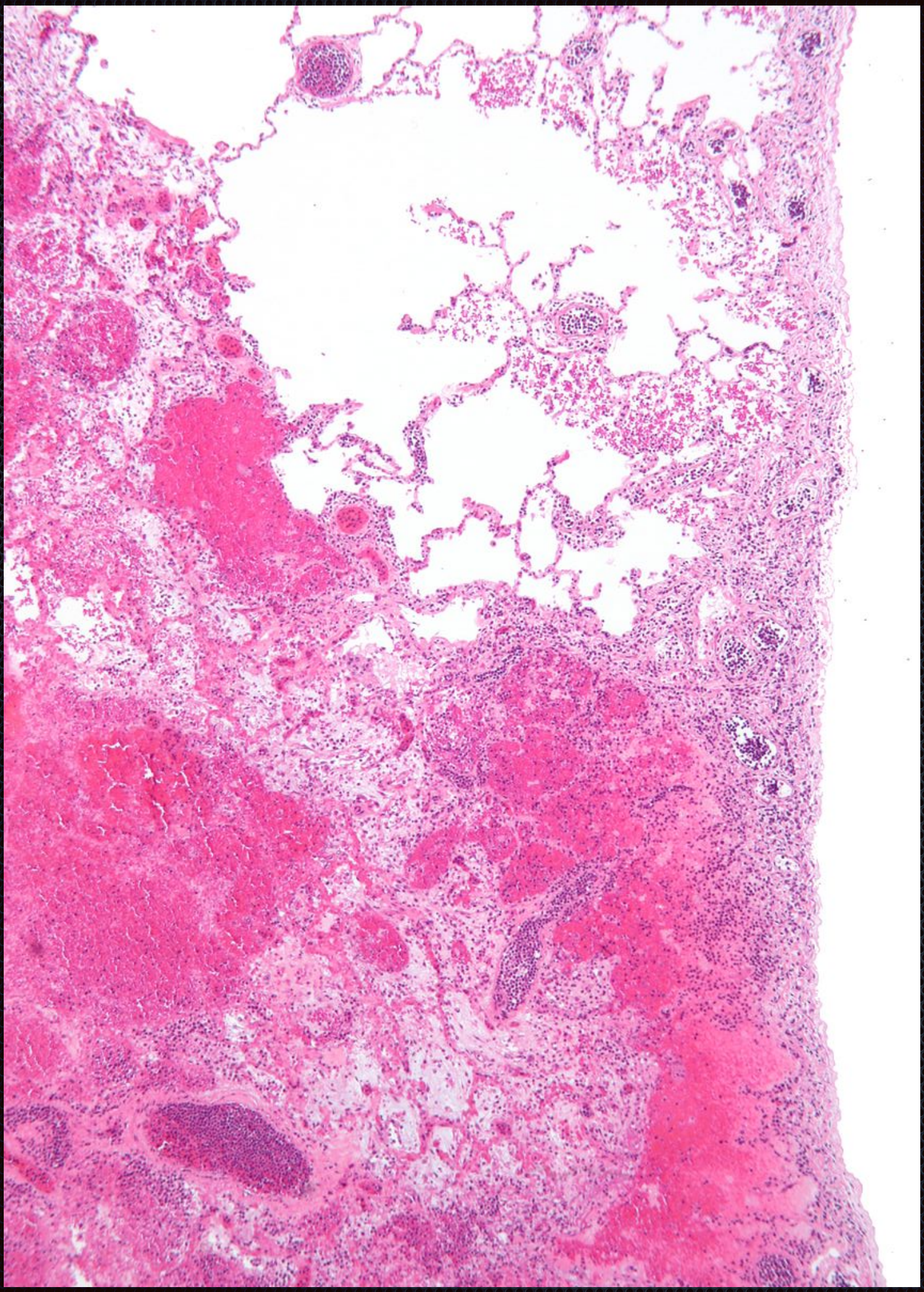
INFARTO PULMONAR

- Suelen ser hemorrágicos
- bien delimitados, rojo firmes, triangulares de base subpleural
- Necrosis del parénquima pulmonar
- Pleura exudado de fibrina
- 48 hrs después palidez de la zona infartada hasta café por hemosiderina
- Posteriormente fibrosis con cicatriz grisácea



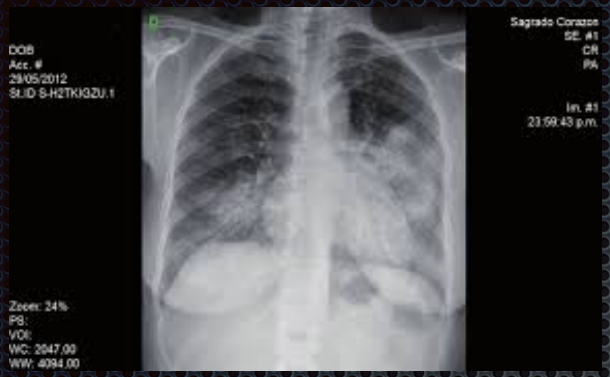
INFARTO PULMONAR





HEMORRAGIA PULMONAR

- FONDO NMUNOLÓGICO:
  - Enf. Goodpasture.- afección P-R Ac MB (alveolo, glomérulo) Cuadro gripal inicial
  - Hemosiderosis pulmonar idiopática.- Hemosiderina (Algunos IgA elevada)
  - Hemorragia pulmonar por complejos inmunes.- Purpura, Beceht, LES (capilariris)
- VASCULITIS PULMONARES (Sistemica o pulmonar)
  - Granulomatosis de Wegener.- Triada (Granulomatosis, vasculitis y GMN) c-ANCA
  - Sd. de Churg y Strauss.- Triada (asma, , eosinofilia, vasculitis sistemica rica en eos)
  - Arteritis de Takayasu.- Inflamación de grandes arterias de tipo elástico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29/05/2012
St.ID S-H2TKI3ZU.1
Sagrado Corazon
SE. #1
CR
PA
Im. #1
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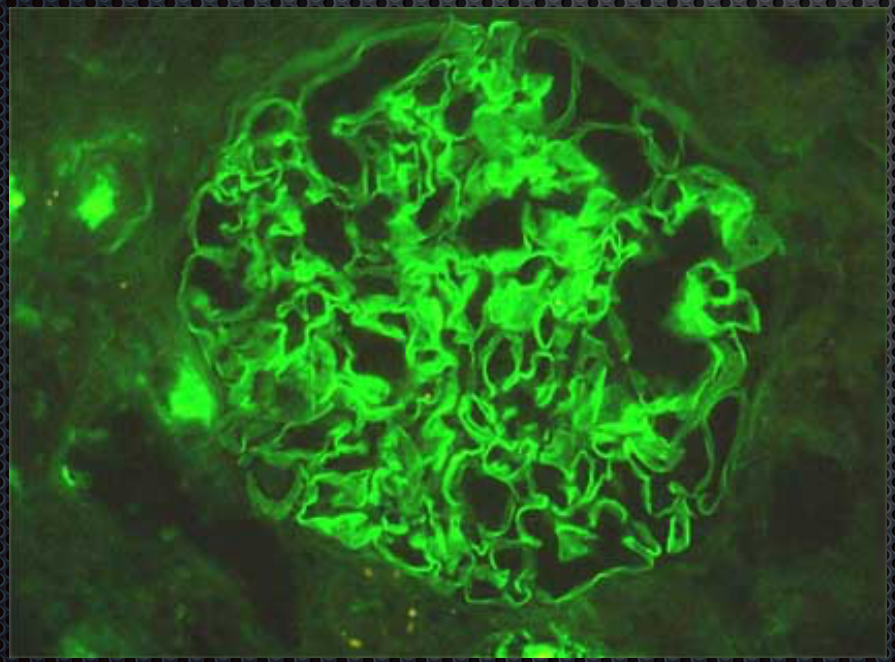
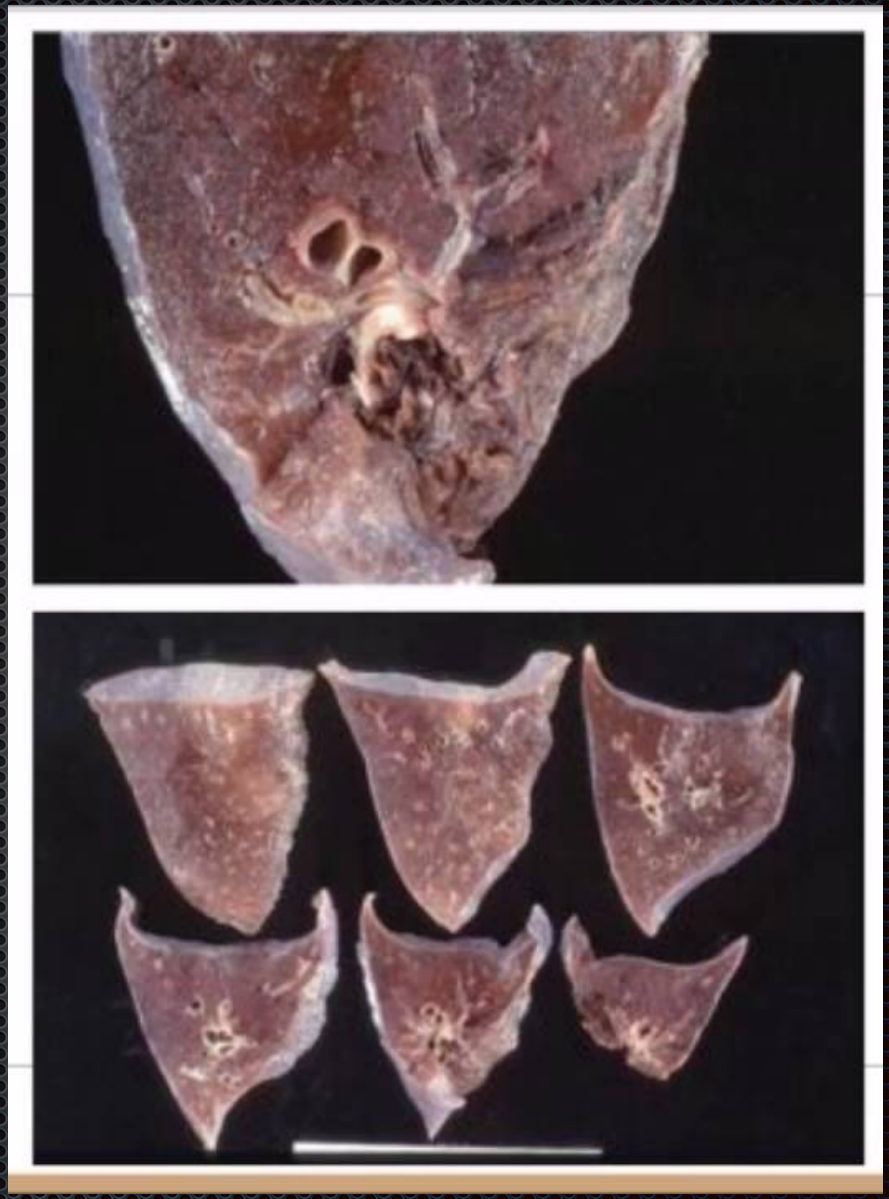
Zoom: 24%
PS:
VOI:
WC: 2047.00
WW: 4094.00

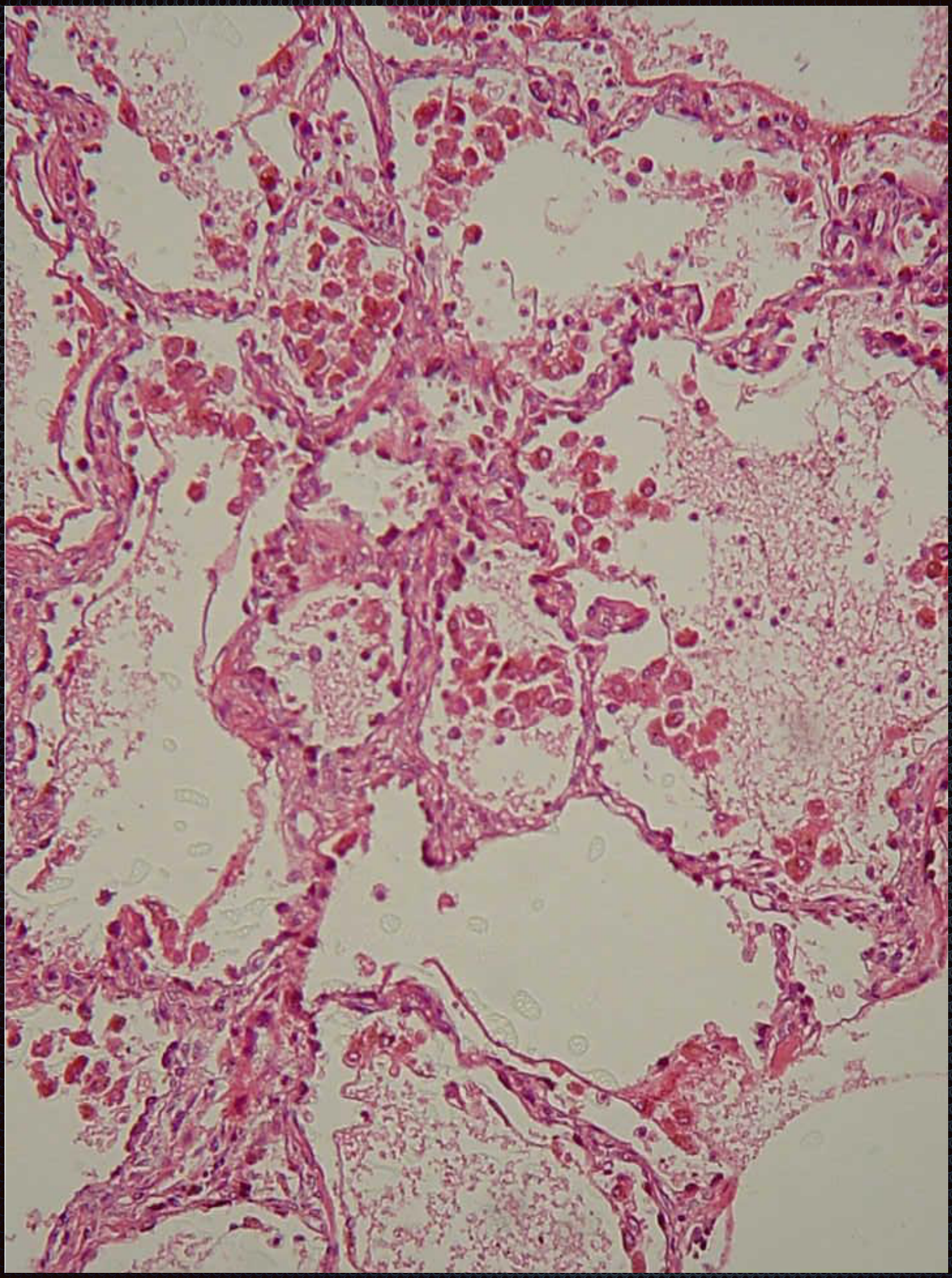


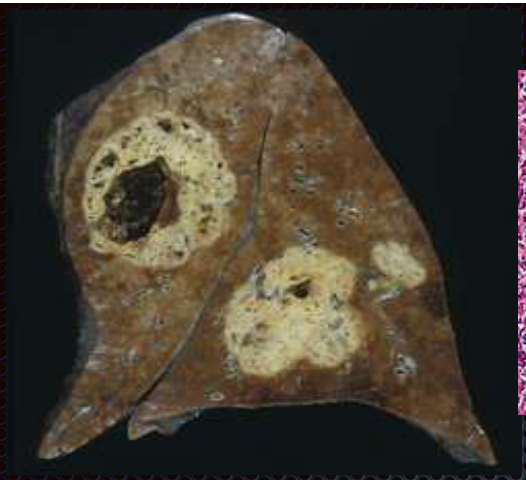


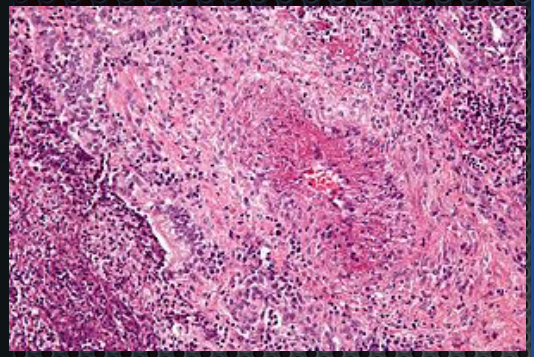
a
b

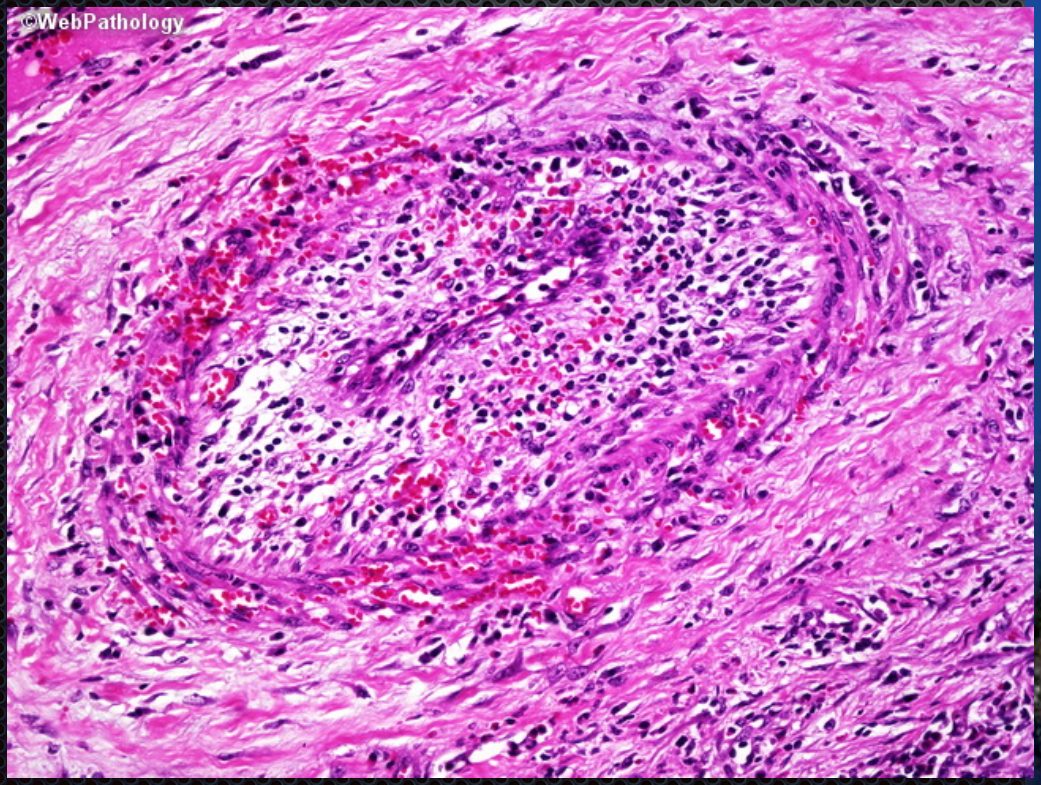
7

6





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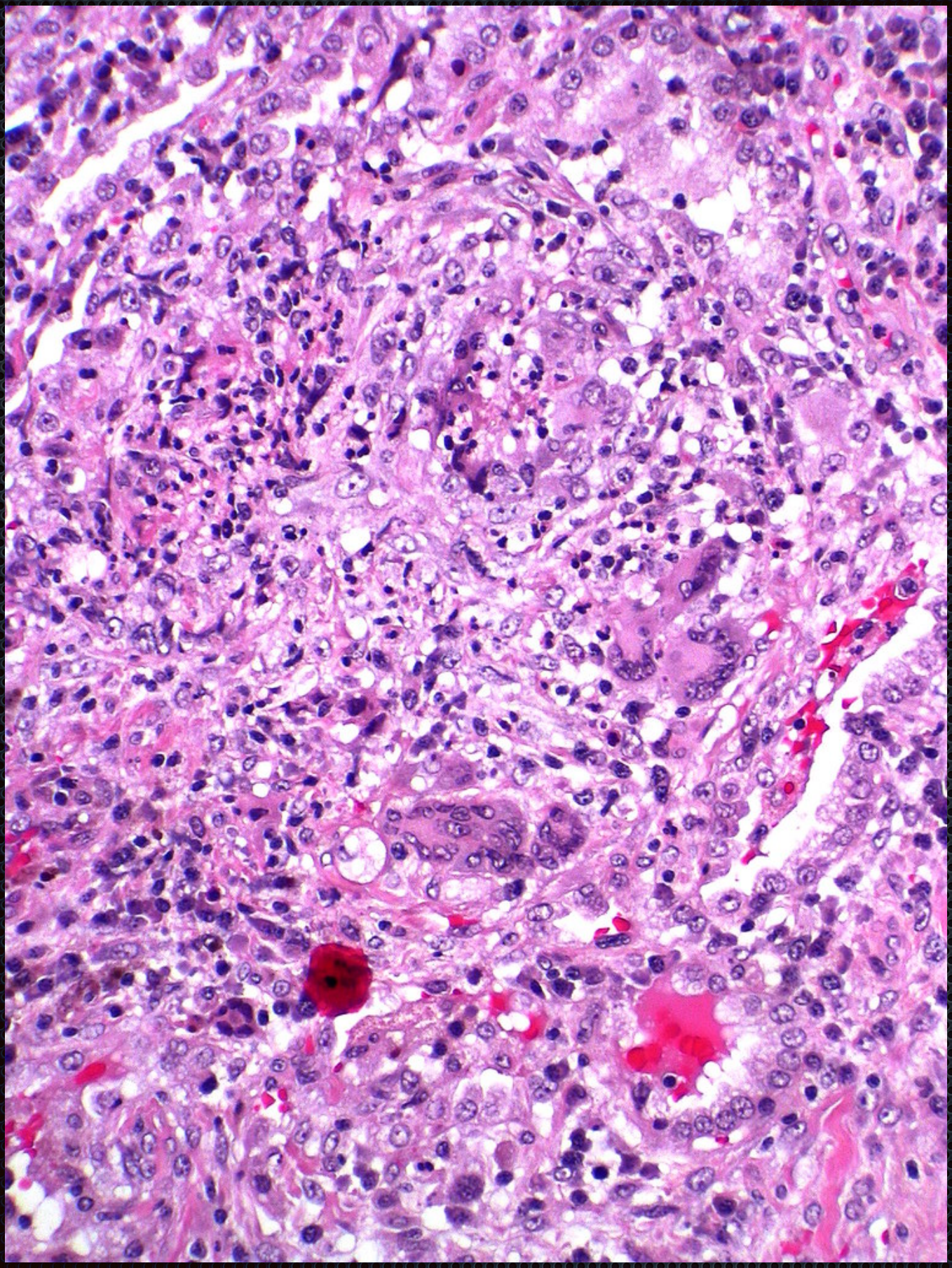
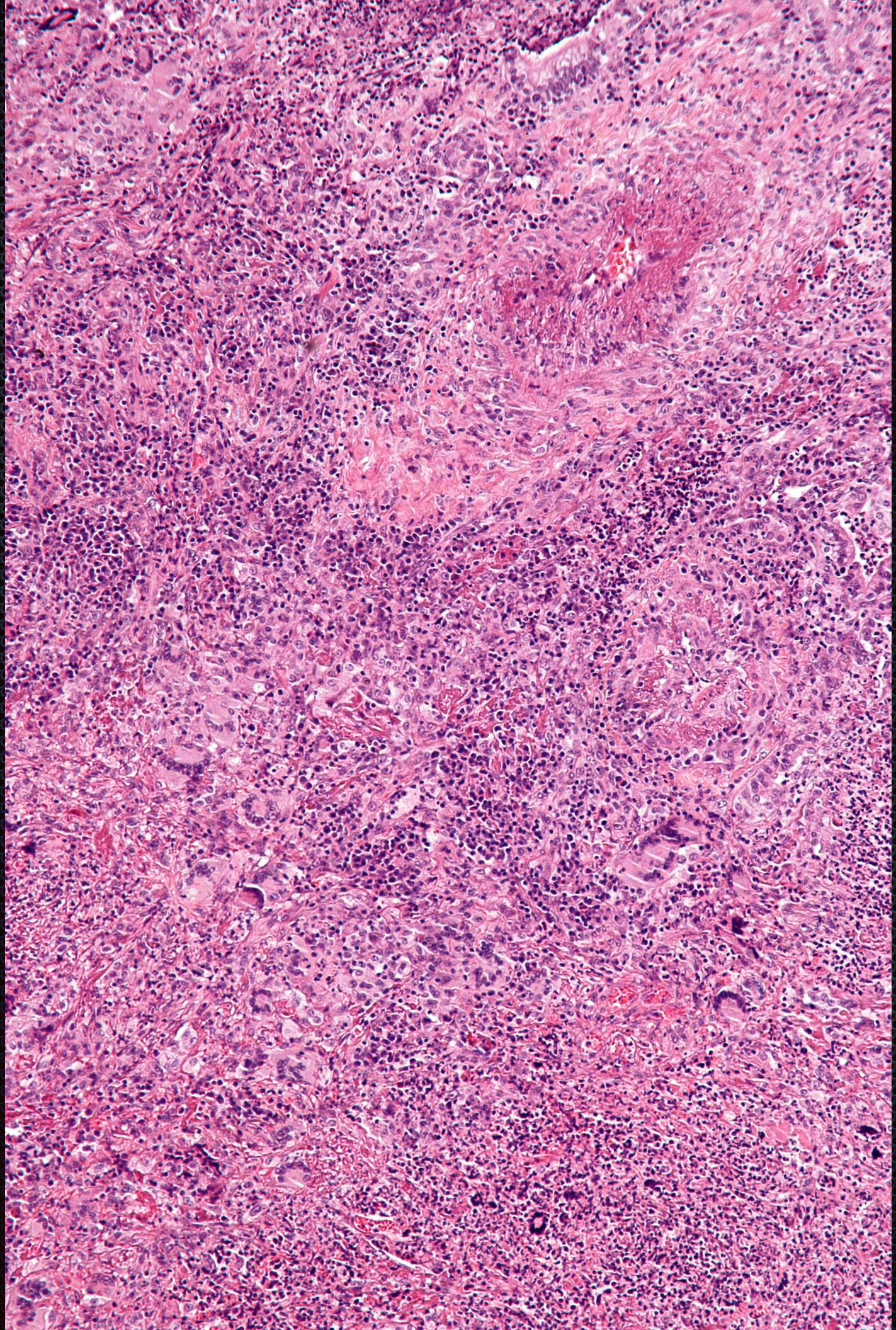
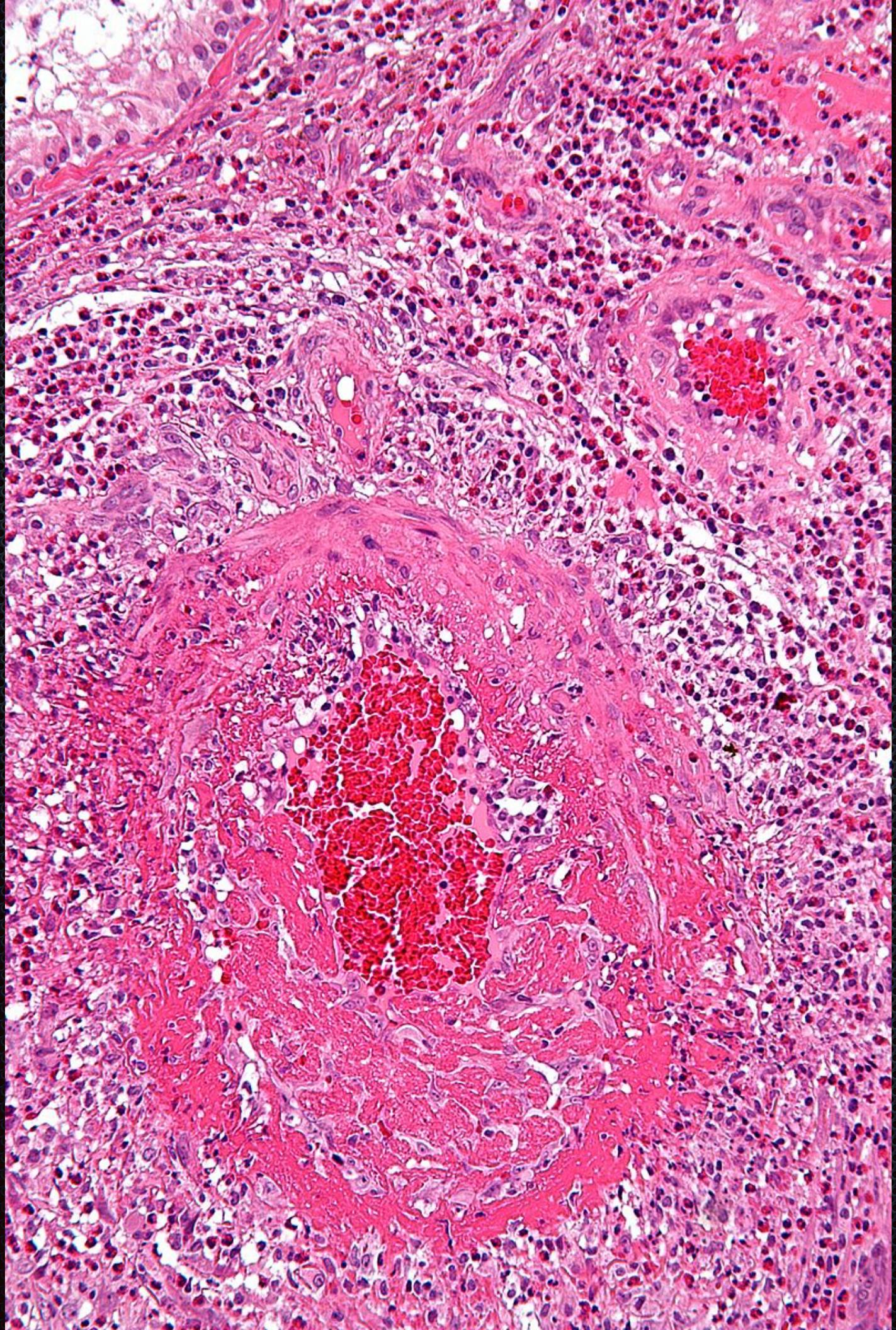
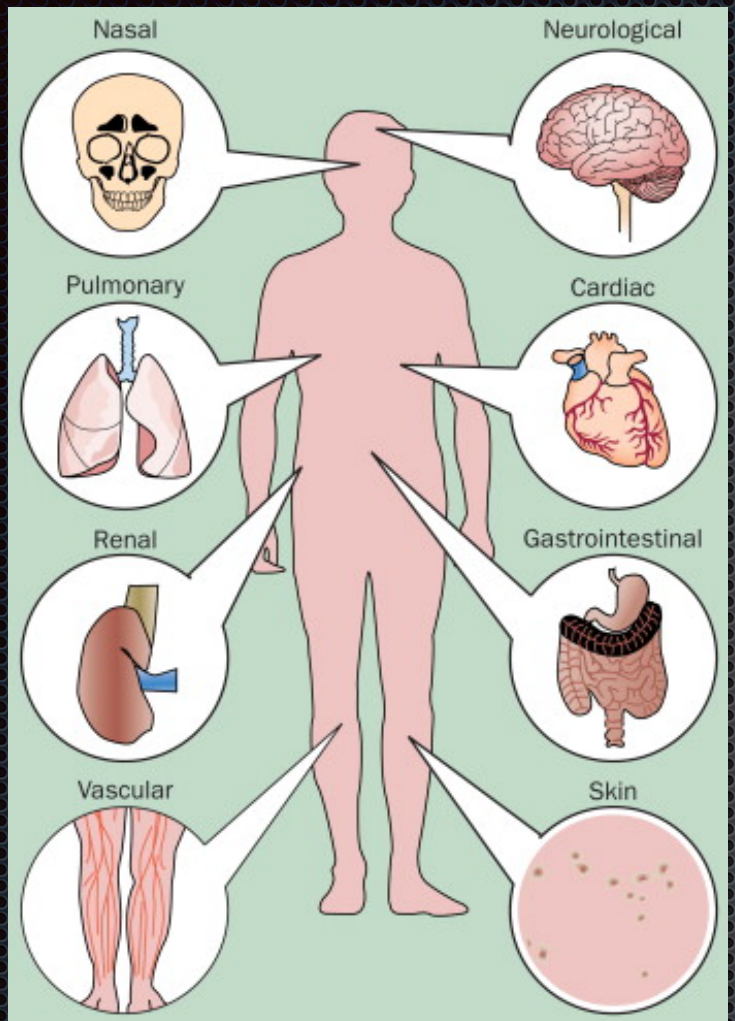
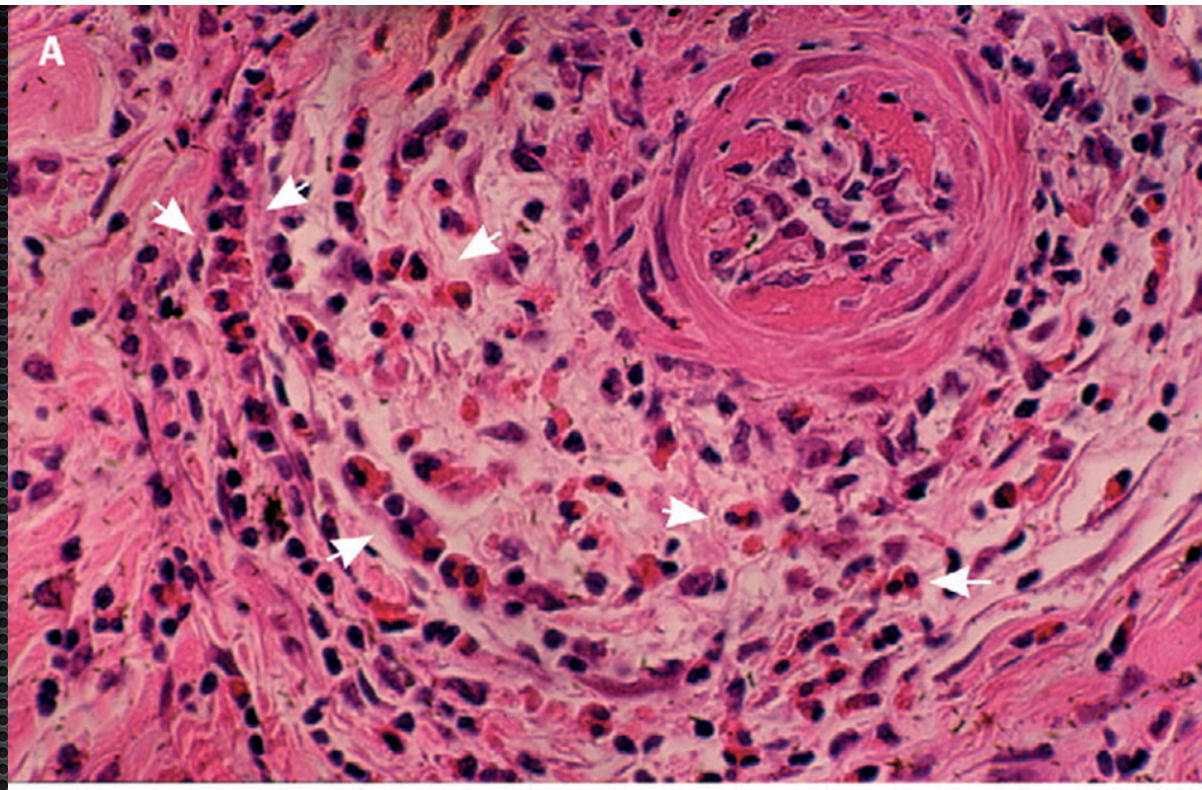
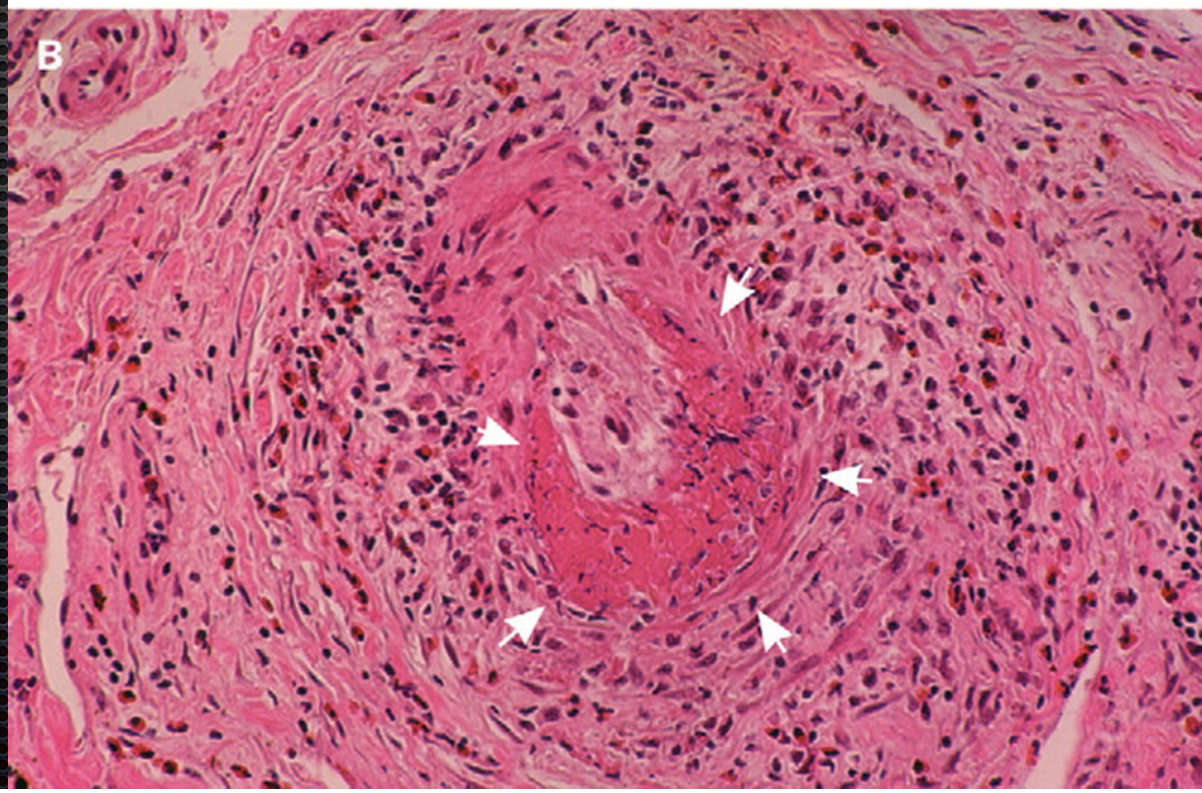




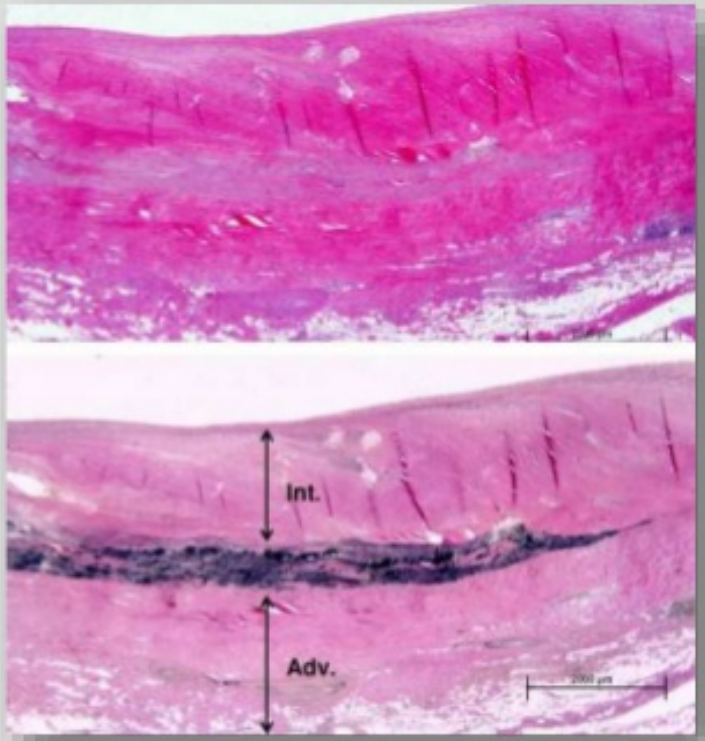



Nasal
Neurological
Pulmonary
Cardiac
Renal
Gastrointestinal
Vascular
Skin

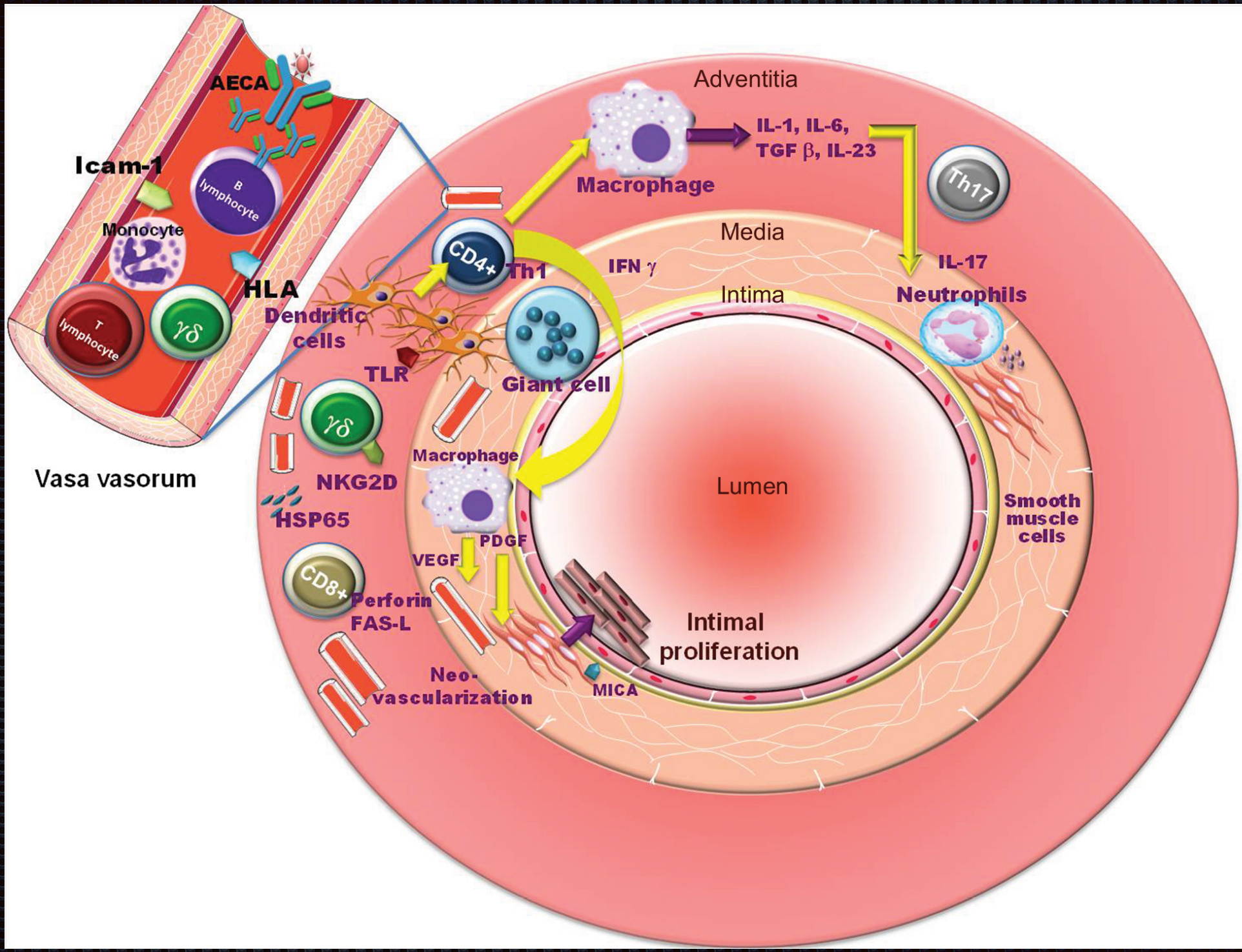

A


B

PATOGÉNESIS

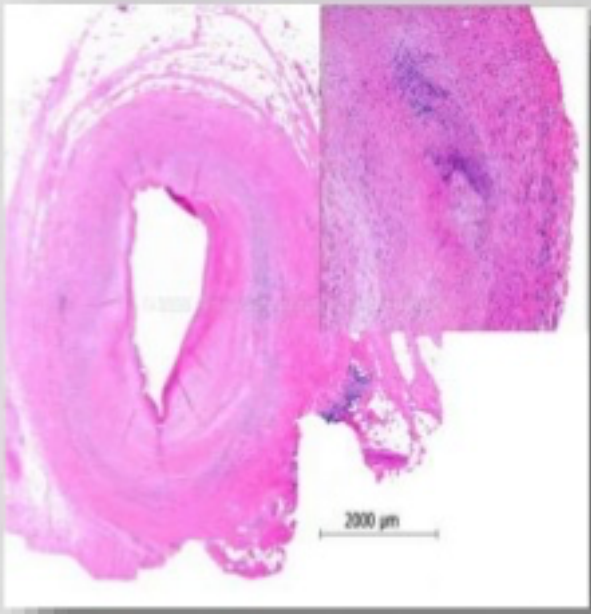


- Etiología permanece desconocida.
- ¿Infecciones? → no relación con algún organismo.
- **Linfocitos T CD8**
  - principal componente del infiltrado vascular.
- Relación con HLA clase I
  - Especialmente HLA-B52.


AECA
Icam-1
B lymphocyte
Monocyte
HLA
T lymphocyte
γδ
Vasa vasorum
Adventitia
Macrophage
IL-1, IL-6,
TGF β, IL-23
Th17
Media
CD4+
Th1
IFN γ
IL-17
Intima
Neutrophils
Dendritic cells
TLR
Giant cell
γδ
NKG2D
HSP65
Macrophage
Lumen
Smooth muscle cells
PDGF
VEGF
CD8+
Perforin
FAS-L
Intimal proliferation
Neo-vascularization
MICA

PATOGÉNESIS

- **Poliarteritis granulomatosa**
  - Con engrosamiento de la adventicia + intenso **infiltrado perivascular** alrededor de los **vasa vasorum.**
- Formación de granuloma y células gigantes
  - Encontrados predominantemente en la media de las grandes arterias elásticas.
- Músculo liso de la media
  - **Destruido** de manera centrípeta y reemplazado por tejido fibroso→
    - Dilatación aórtica + **formación de aneurisma**.
- **Proliferación de la íntima** + trombosis ocasional →
  - Estrechamiento u oclusión total de la luz vascular.

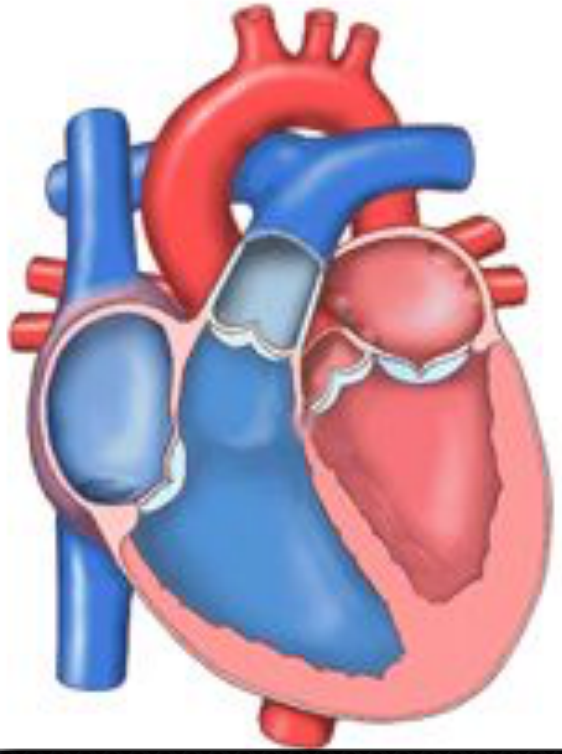


HIPERTENSIÓN PULM ONAR

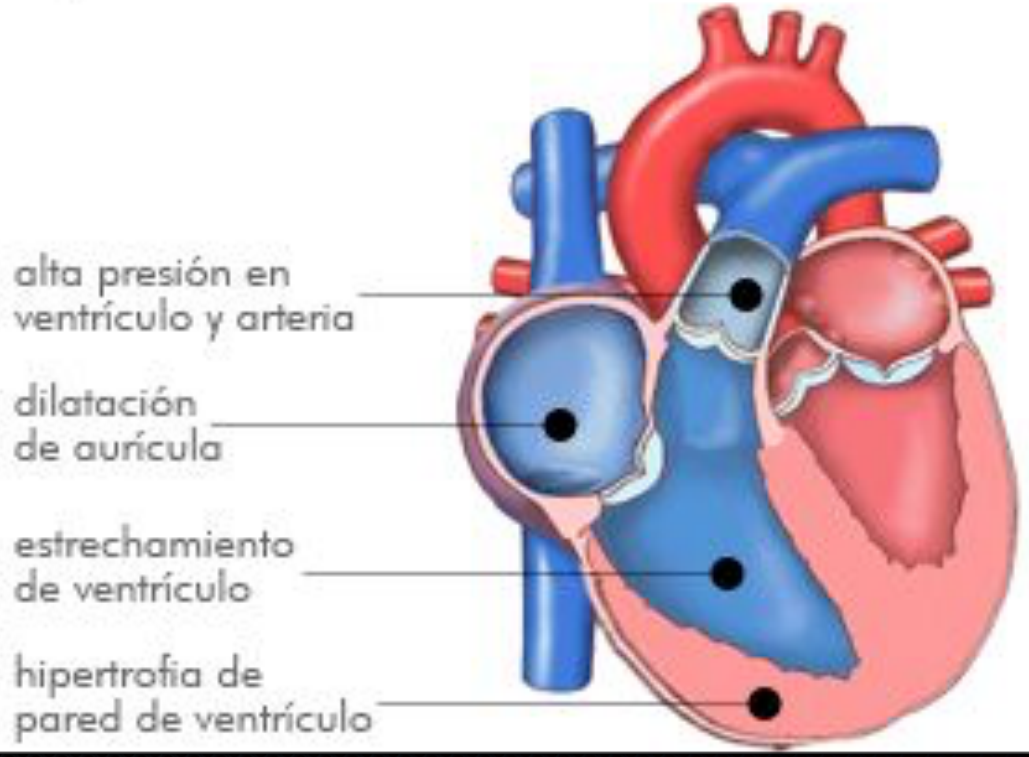
- ADULTOS 16/7 mmHg media 12 mmHg
  - 25mmHg reposo
  - 30 mmHg ejercicio
- Secundaria
- Idiopática (HTP Primaria)

HIPERTENSIÓN PULMONAR

Corazón normal

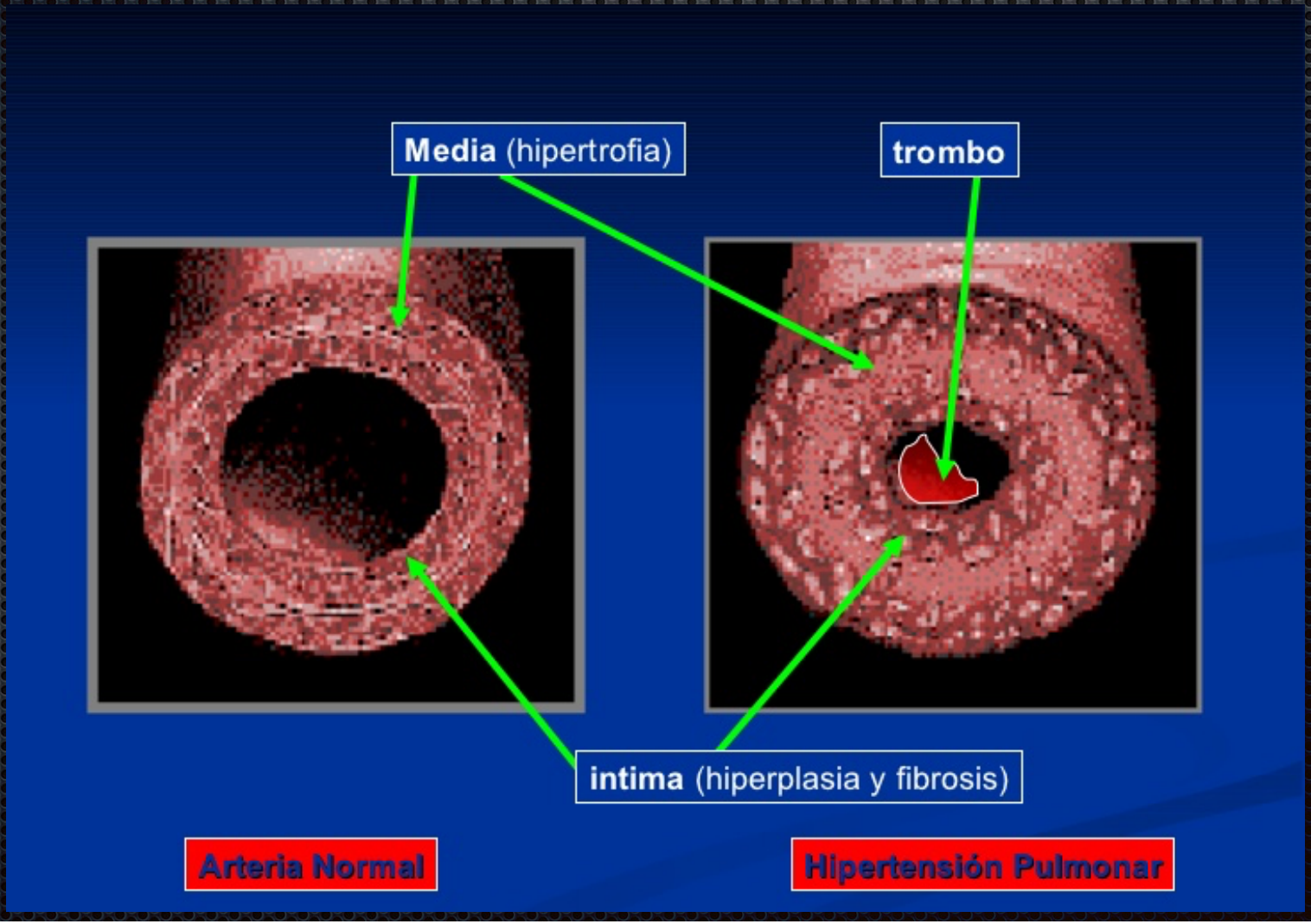


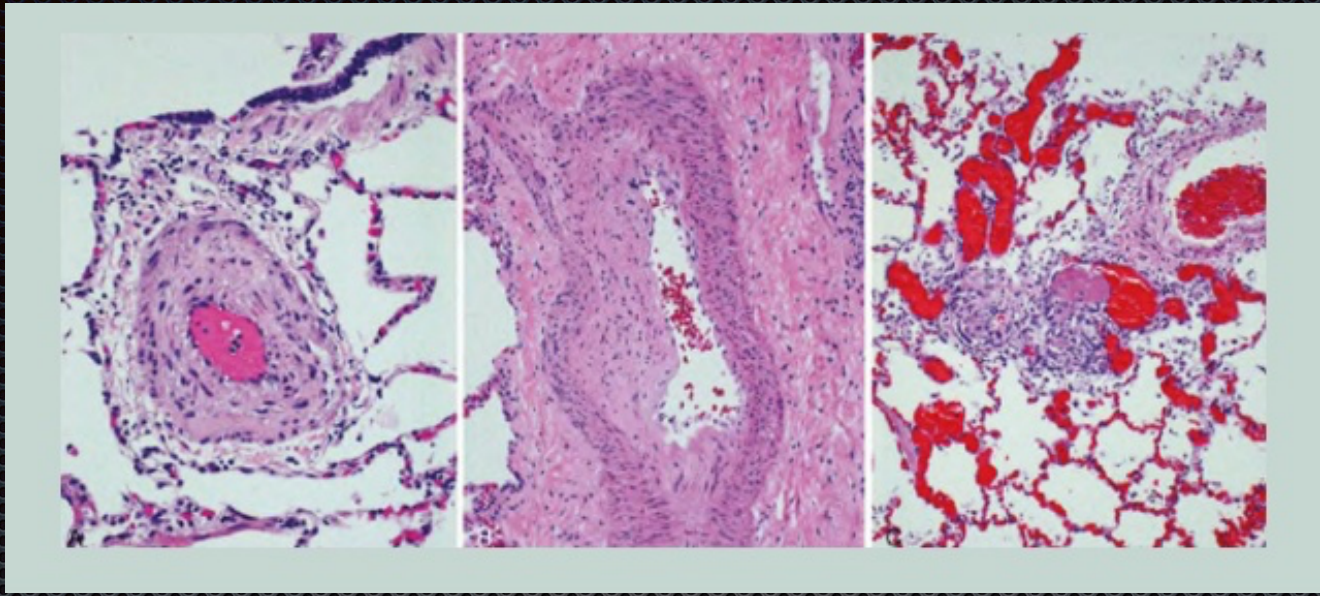
Corazón afectado por
Hipertensión Pulmonar



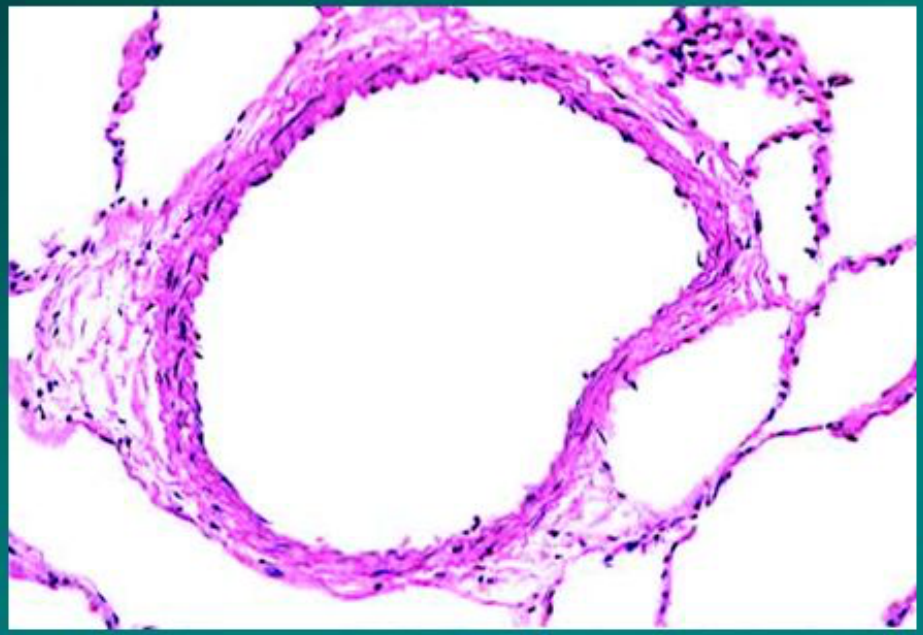
ENGROSAMIENTO DE:

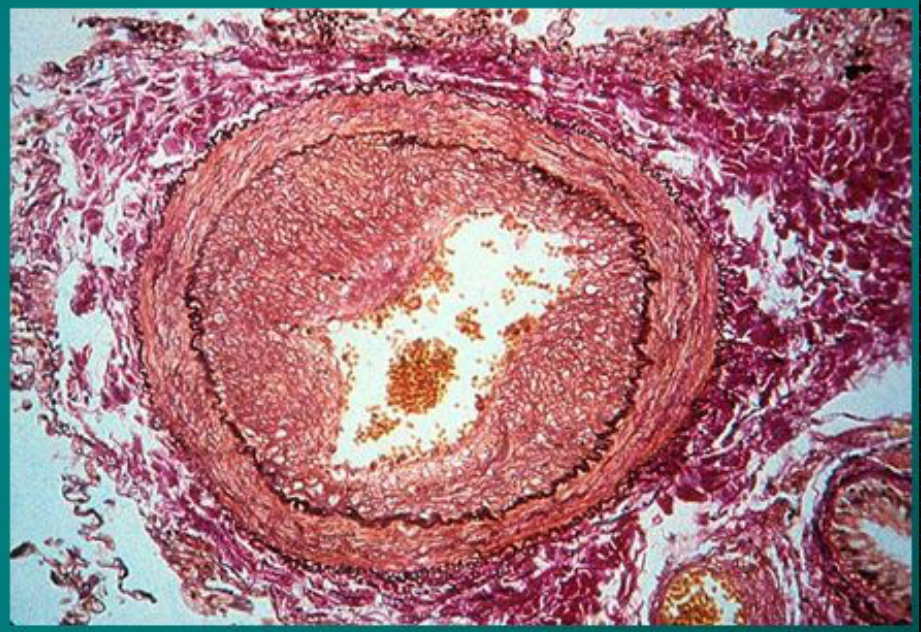
- Capa media arteriolar
- Íntima arterial y arteriolar
- Necrosis fibrinoide y hemosiderosis
- Lesiones plexiformes

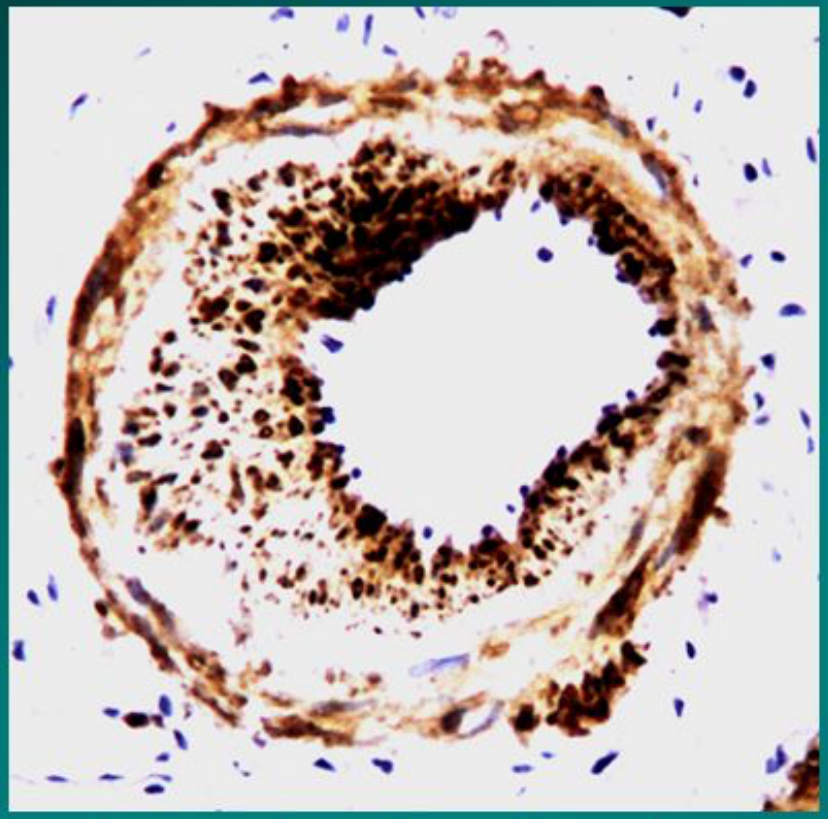




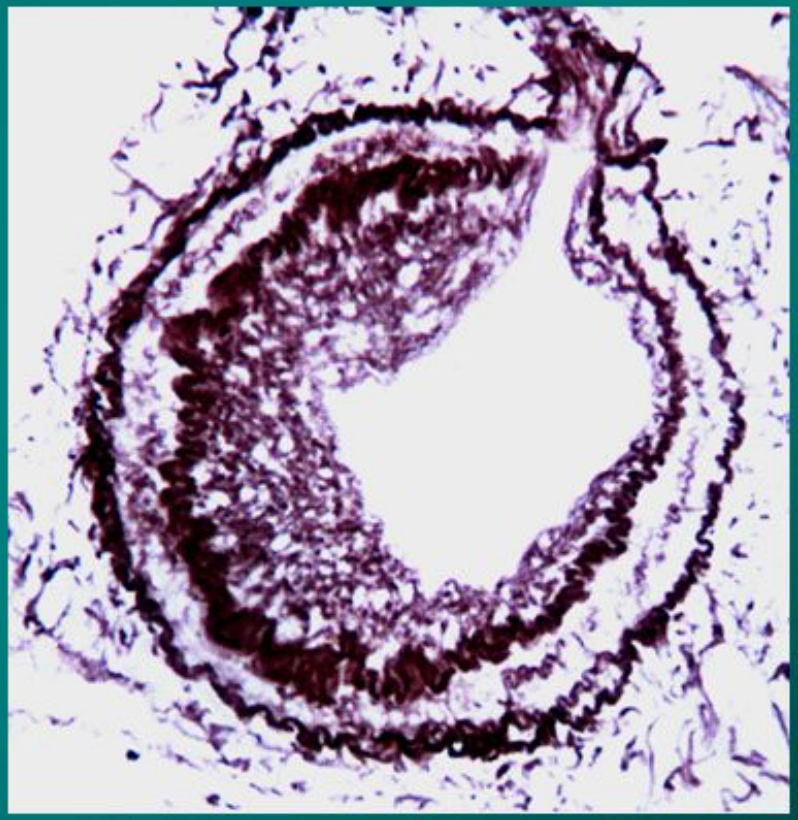
HISTOLOGÍA







Ac monoclonales anti α actina
músculo liso



Tinción de orseína para fibras
elásticas