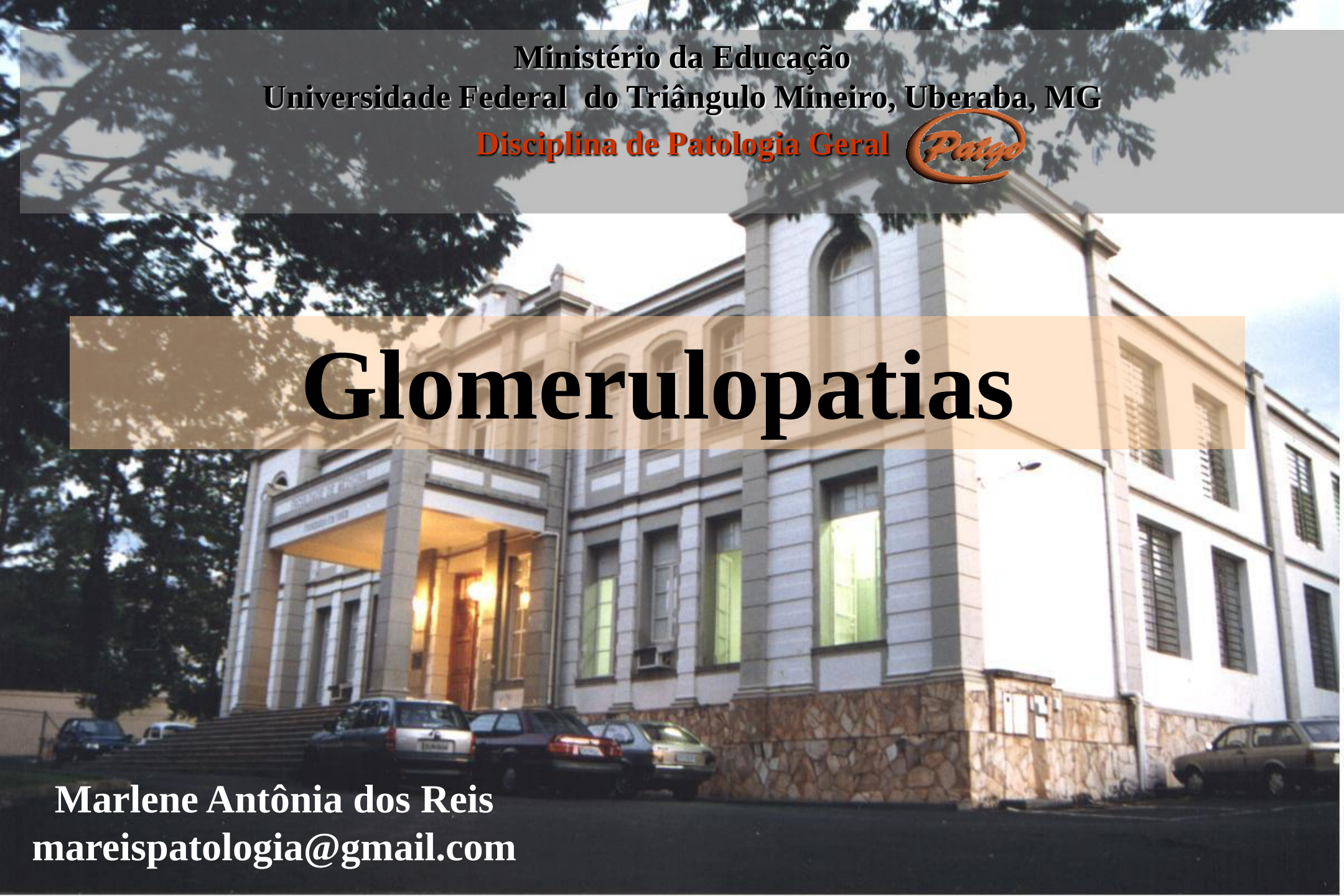
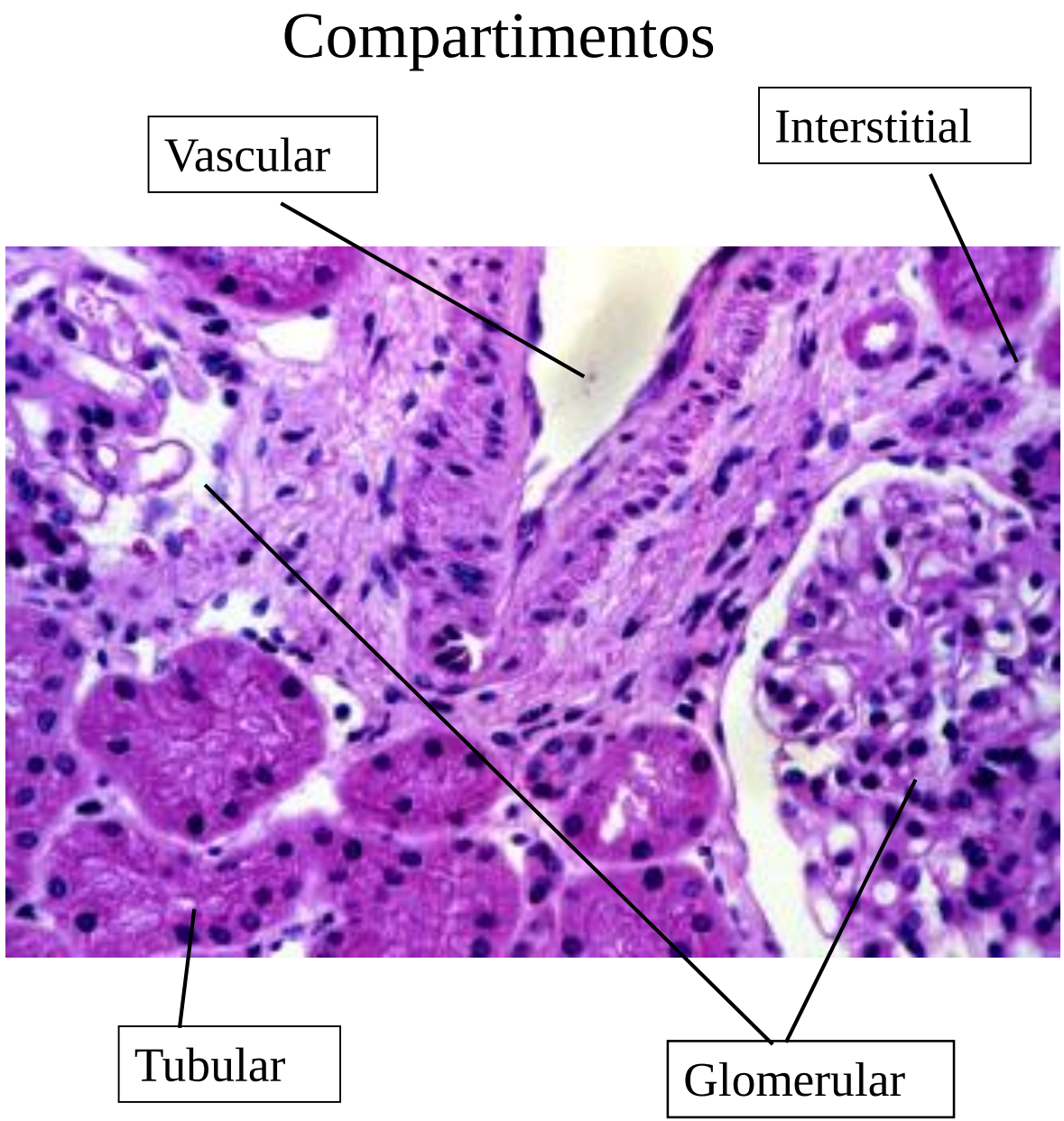
Ministério da Educação
Universidade Federal do Triângulo Mineiro, Uberaba, MG
Disciplina de Patologia Geral

Glomerulopatias



Marlene Antônia dos Reis
mareispatologia@gmail.com



Glomerulopatias

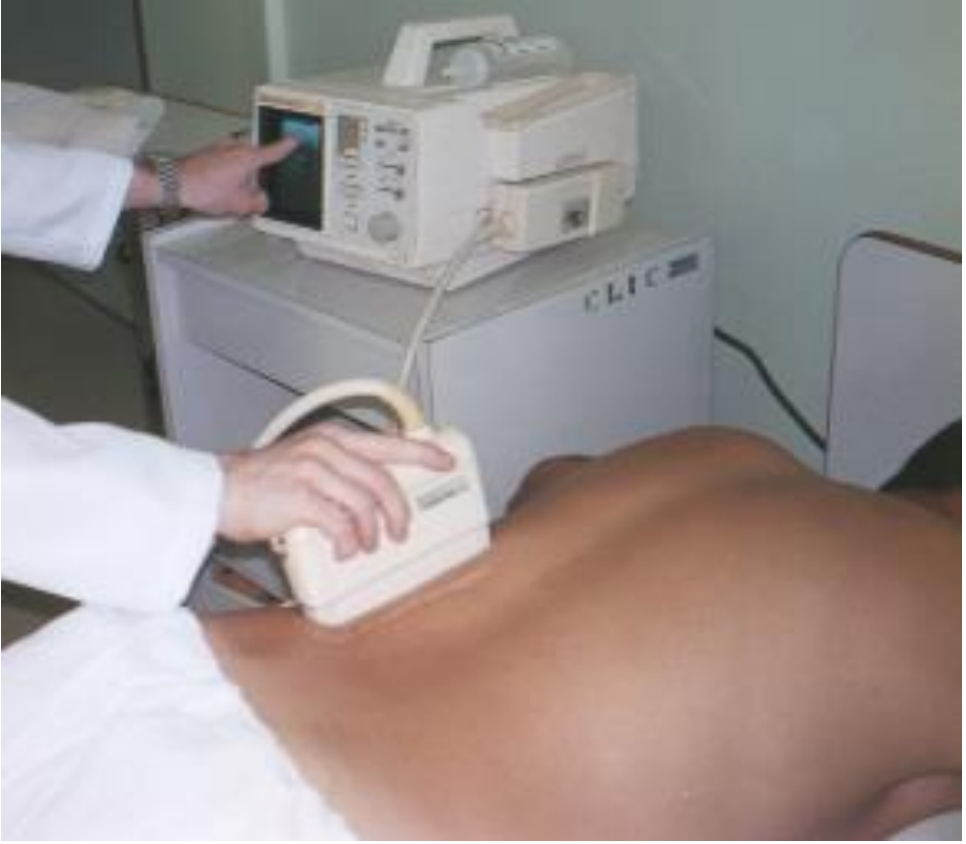
Agressão inicia - glomerular

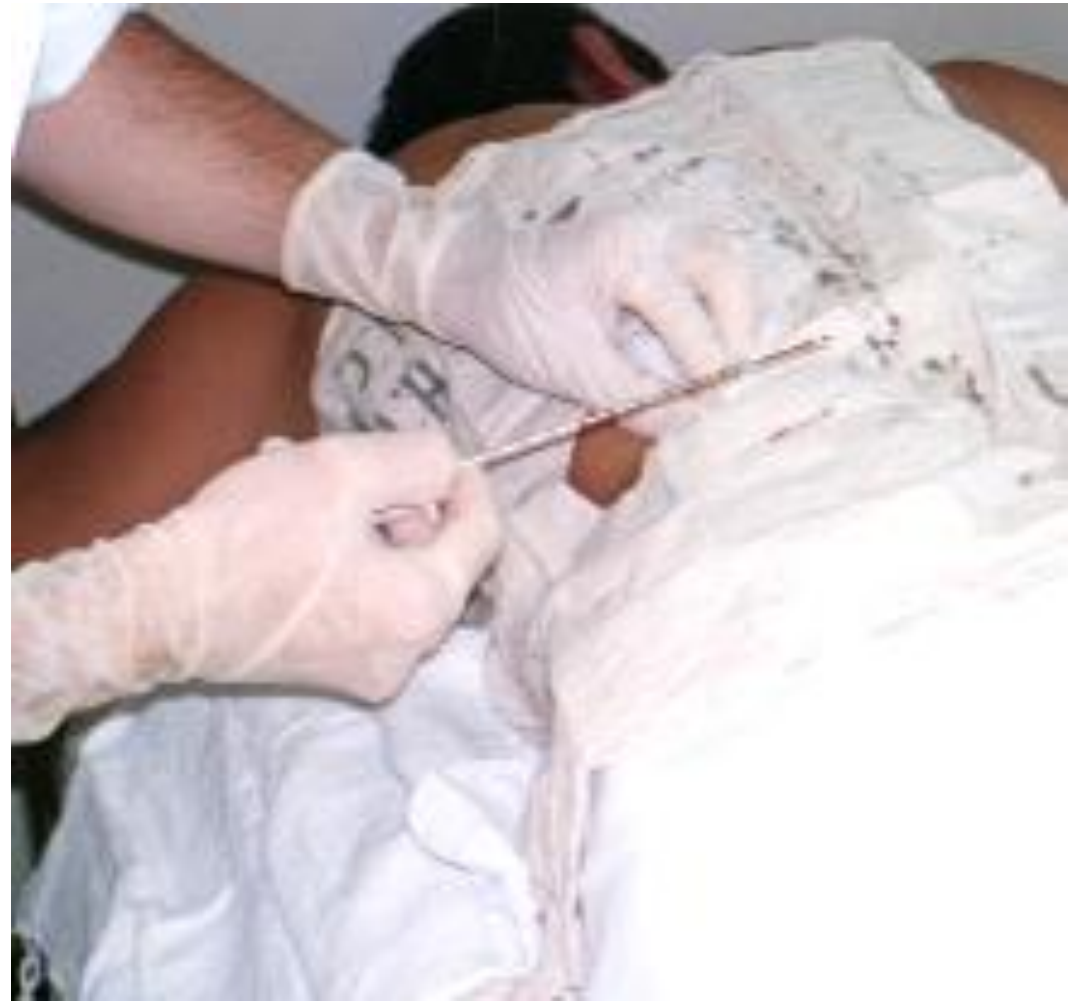
- Secundária – doença sistêmica

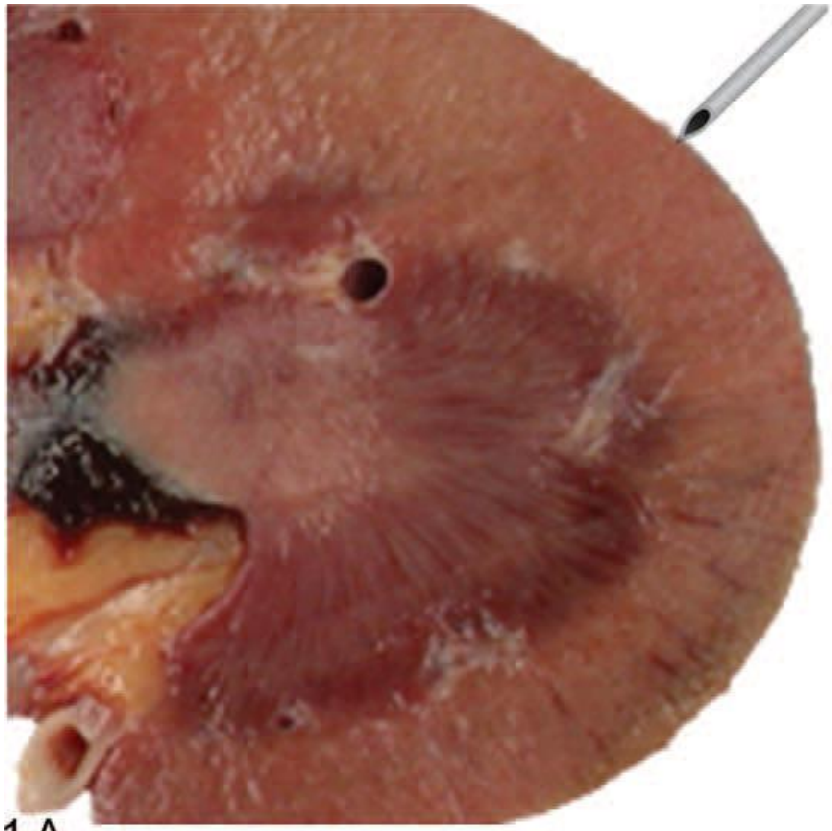
- Primária

Manifestações Clínicas - Glomerulopatias

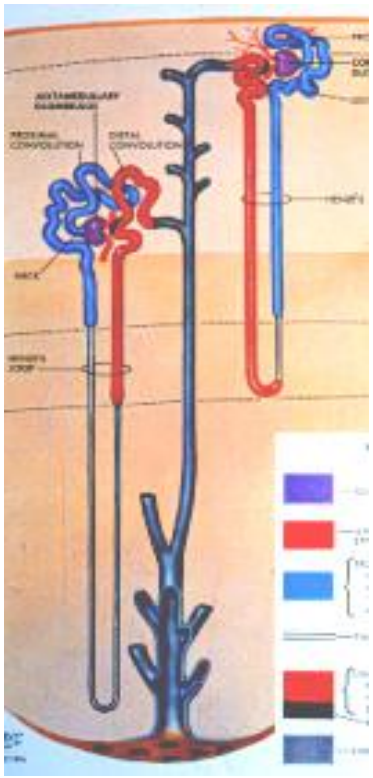
1. Alt urinárias assintomáticas
   a) hematúria glomerular isolada
   b) Proteinúria isolada
2. síndrome nefrótica
3. síndrome nefrítica
4. IRA (insuficiência aguda – rapidamente progressiva)
5. Doença Renal Crônica

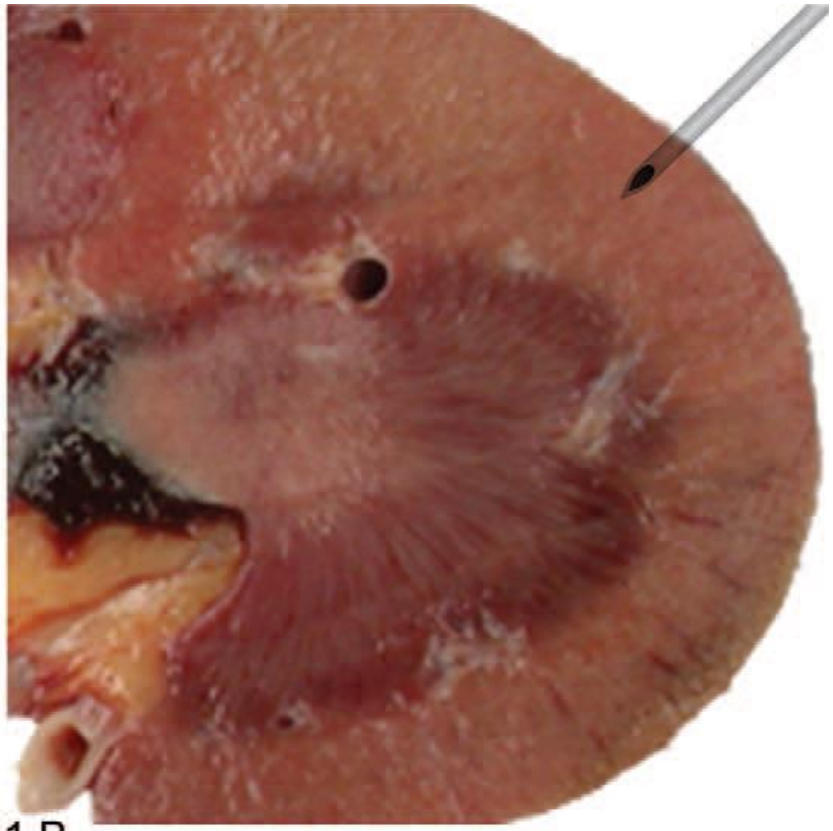






1 A



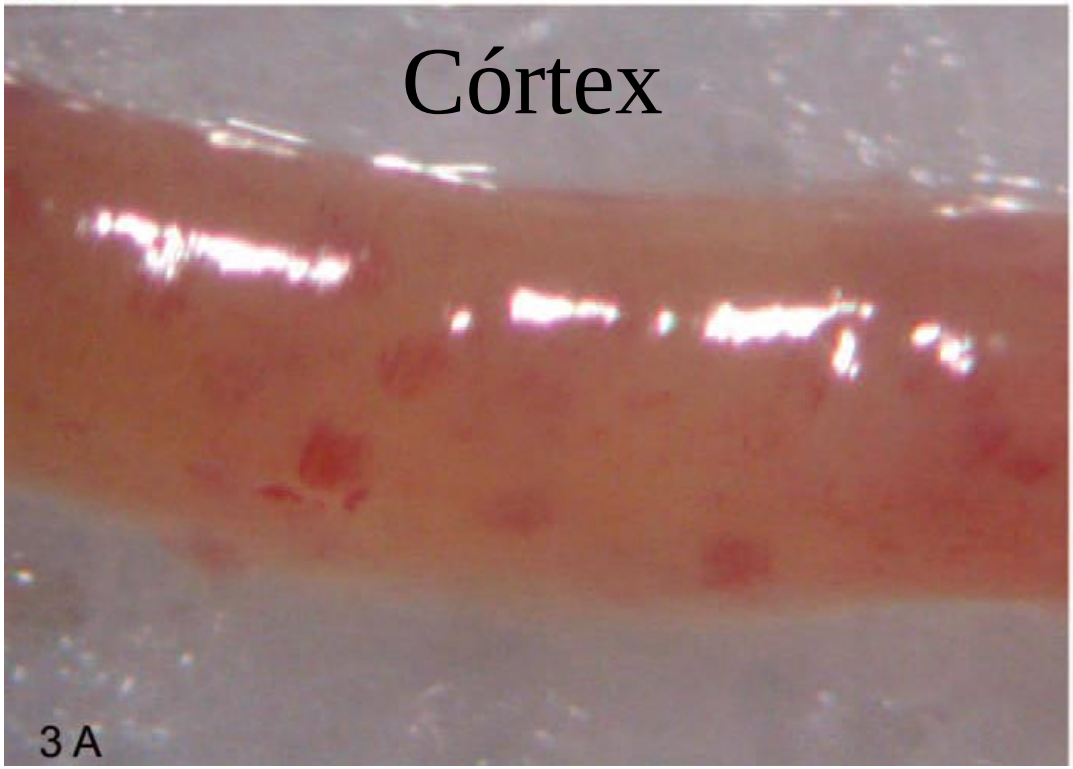


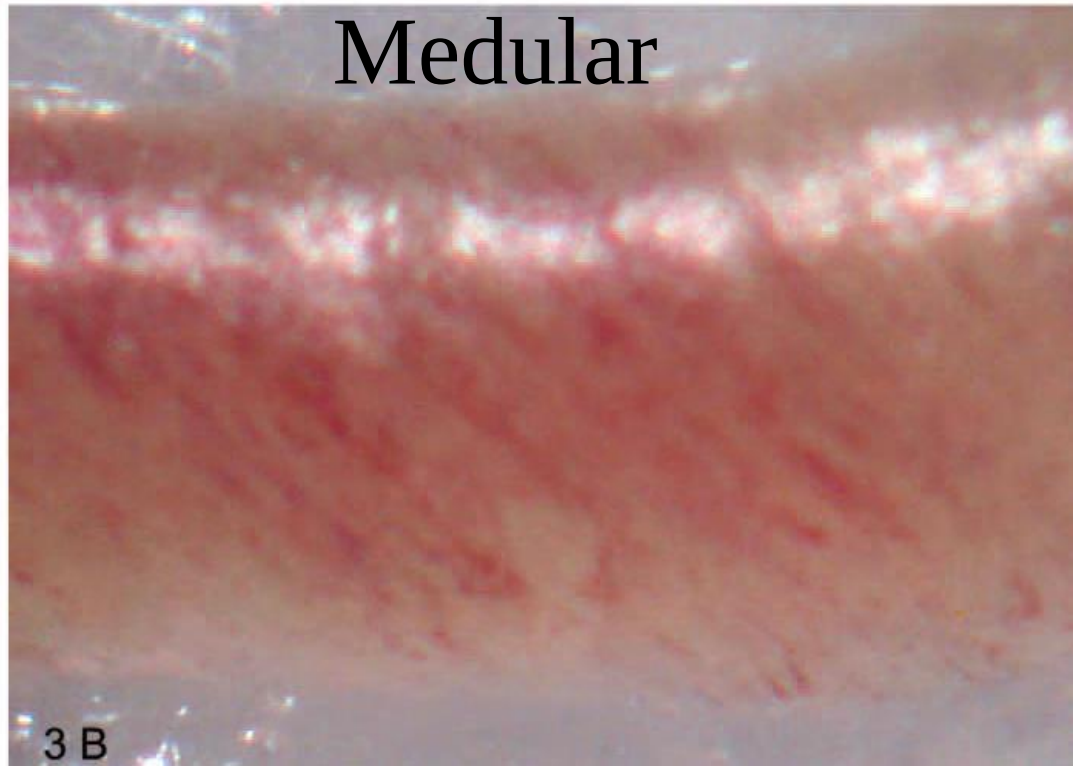
1 B

The Renal Biopsy

Patrick D. Walker, MD

***Arch Pathol Lab Med.* 2009;133:181–188**

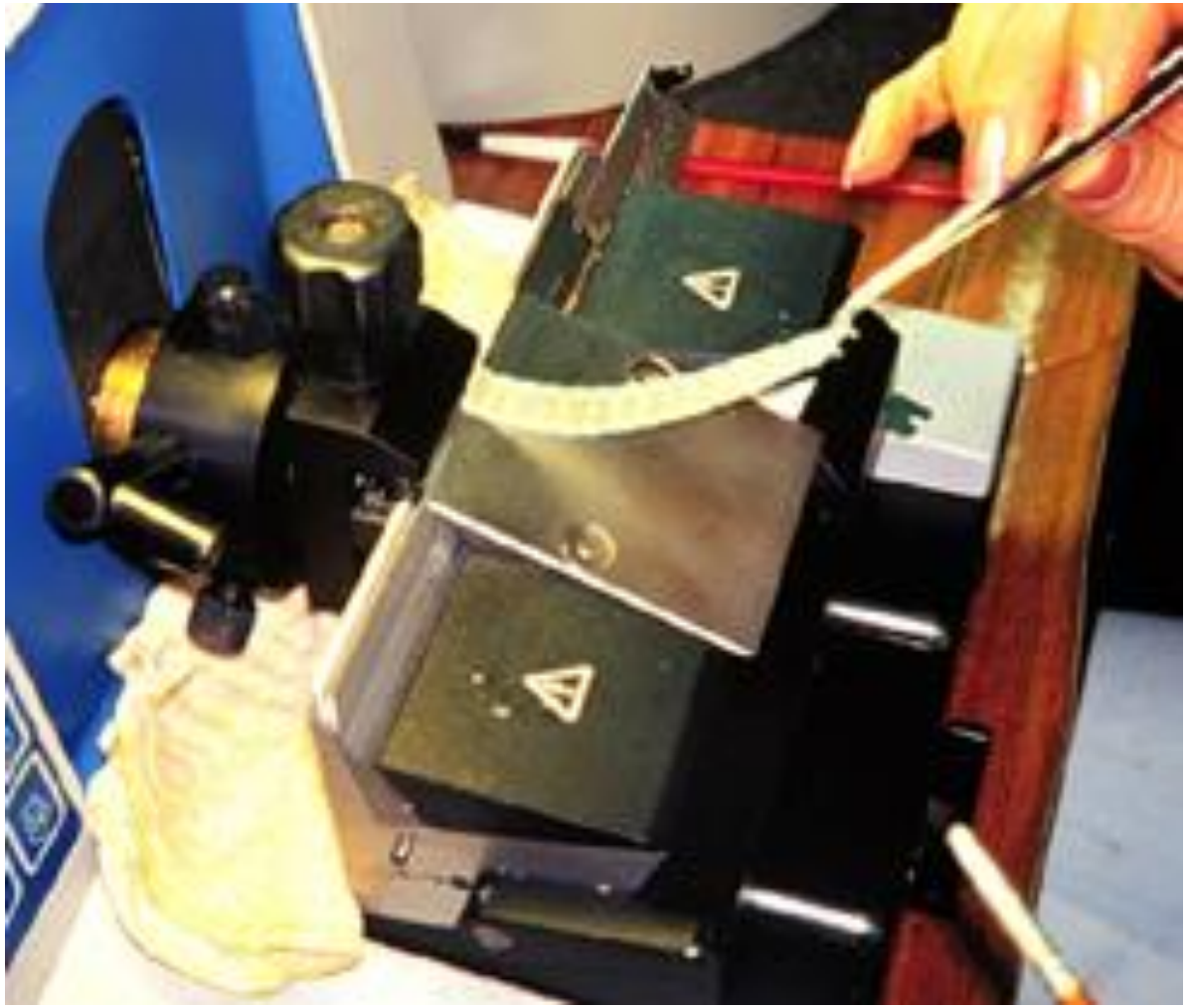




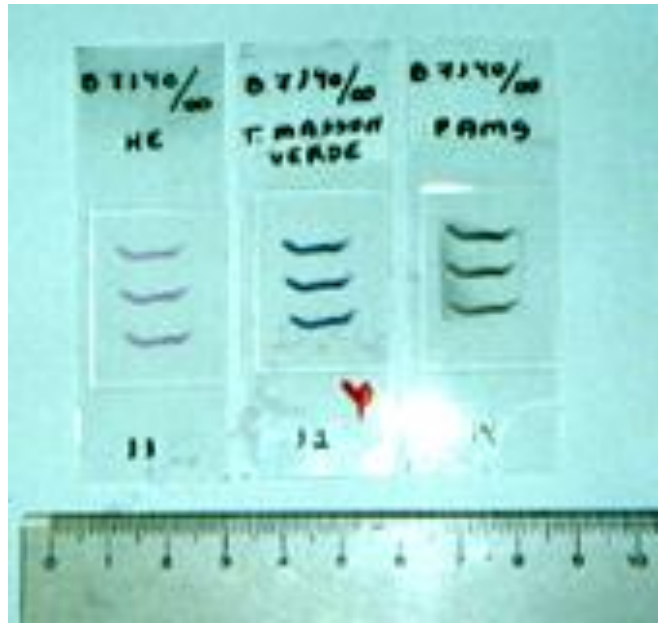
Arch Pathol Lab Med. **2009;133:181–188**

Biópsia Renal

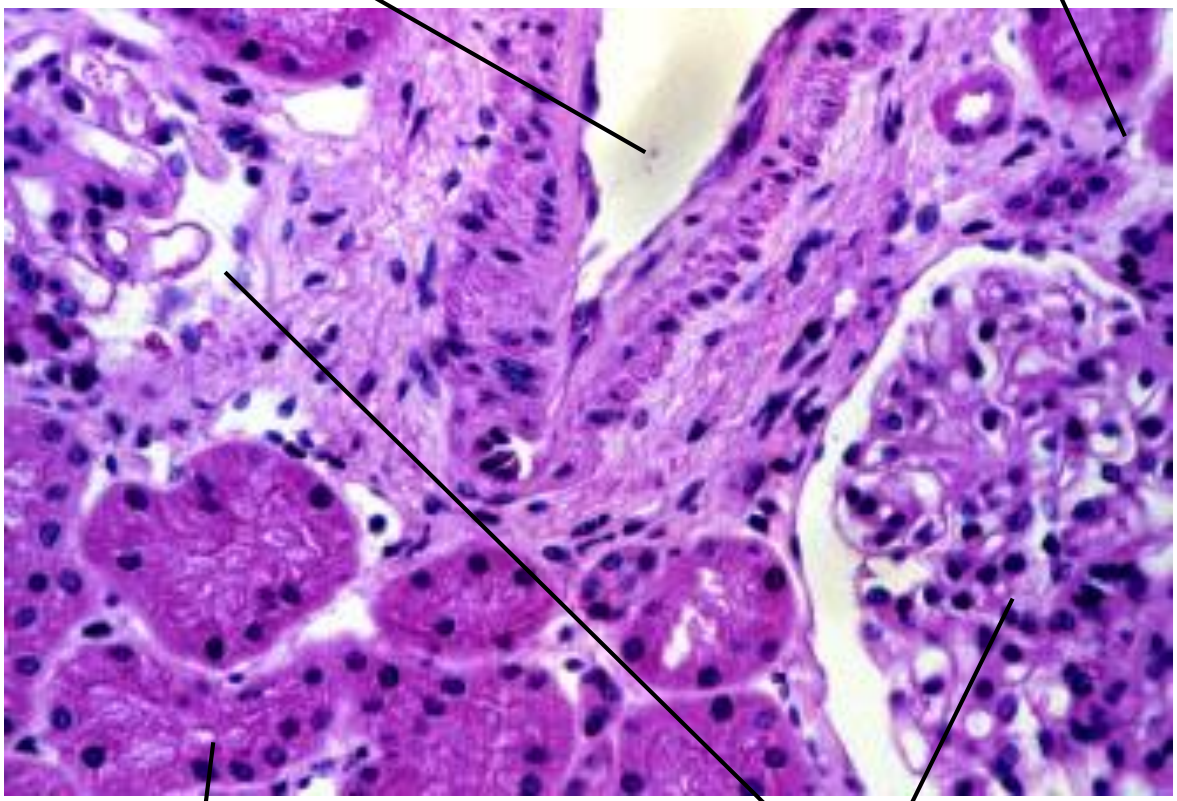
ML-10 gl – paraformaldeido
Formaldeído / Bouin / Metacarn



2 µm

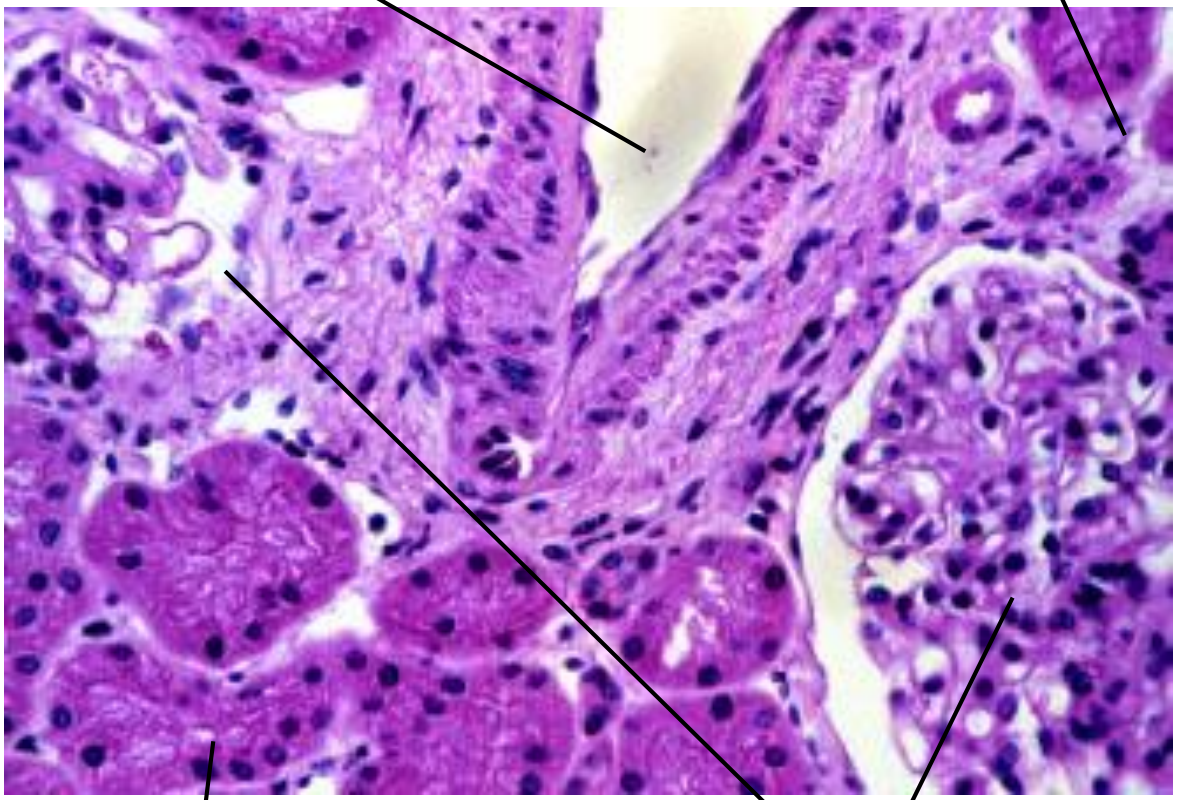


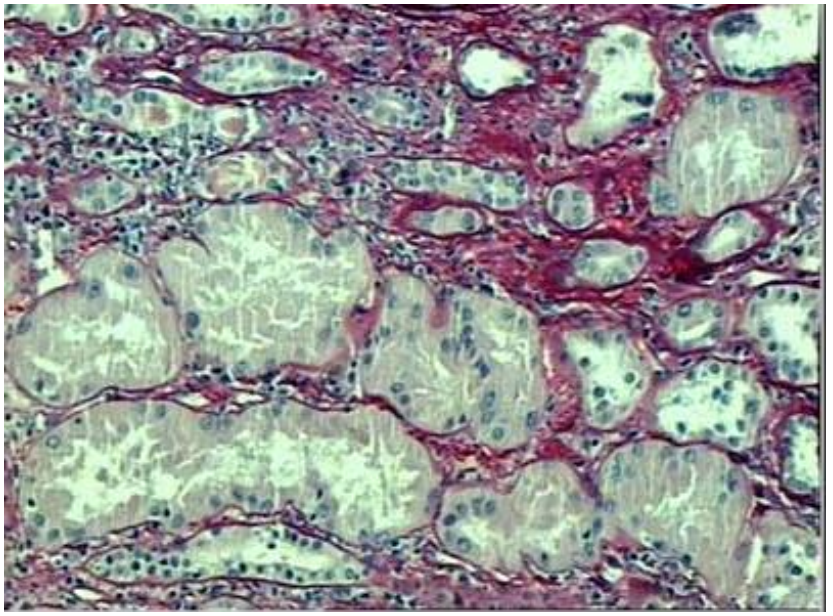
Compartimentos

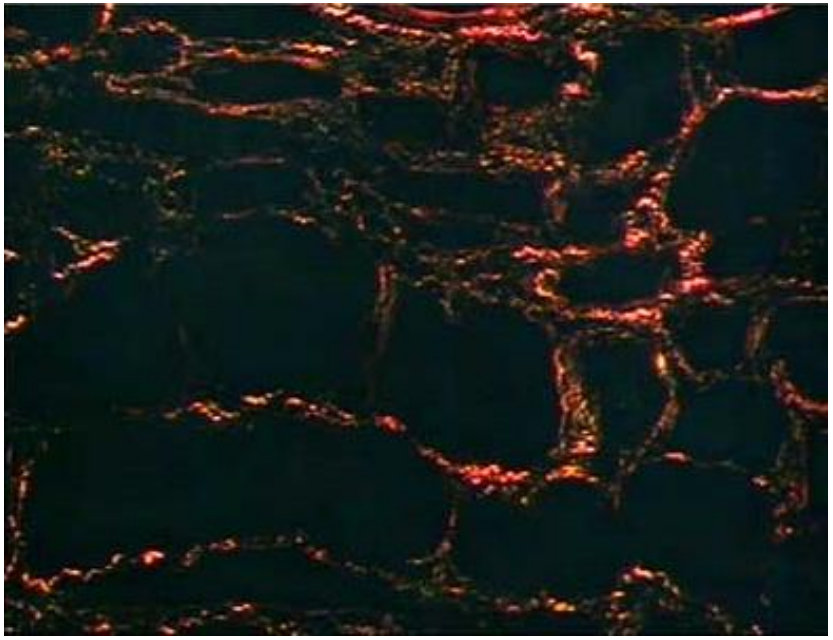


	Aguda (Inflamação / Necrose)	Crônica
Interstício	Inflamação	Fibrose
Tubular	Necrose	Atrofia
	Tubulite	
Vascular	Vasculite	espes. intimal
	Necrose	
Glomerular	**Glomerulite**	**esclerose (↑matriz)**
	Necrose	

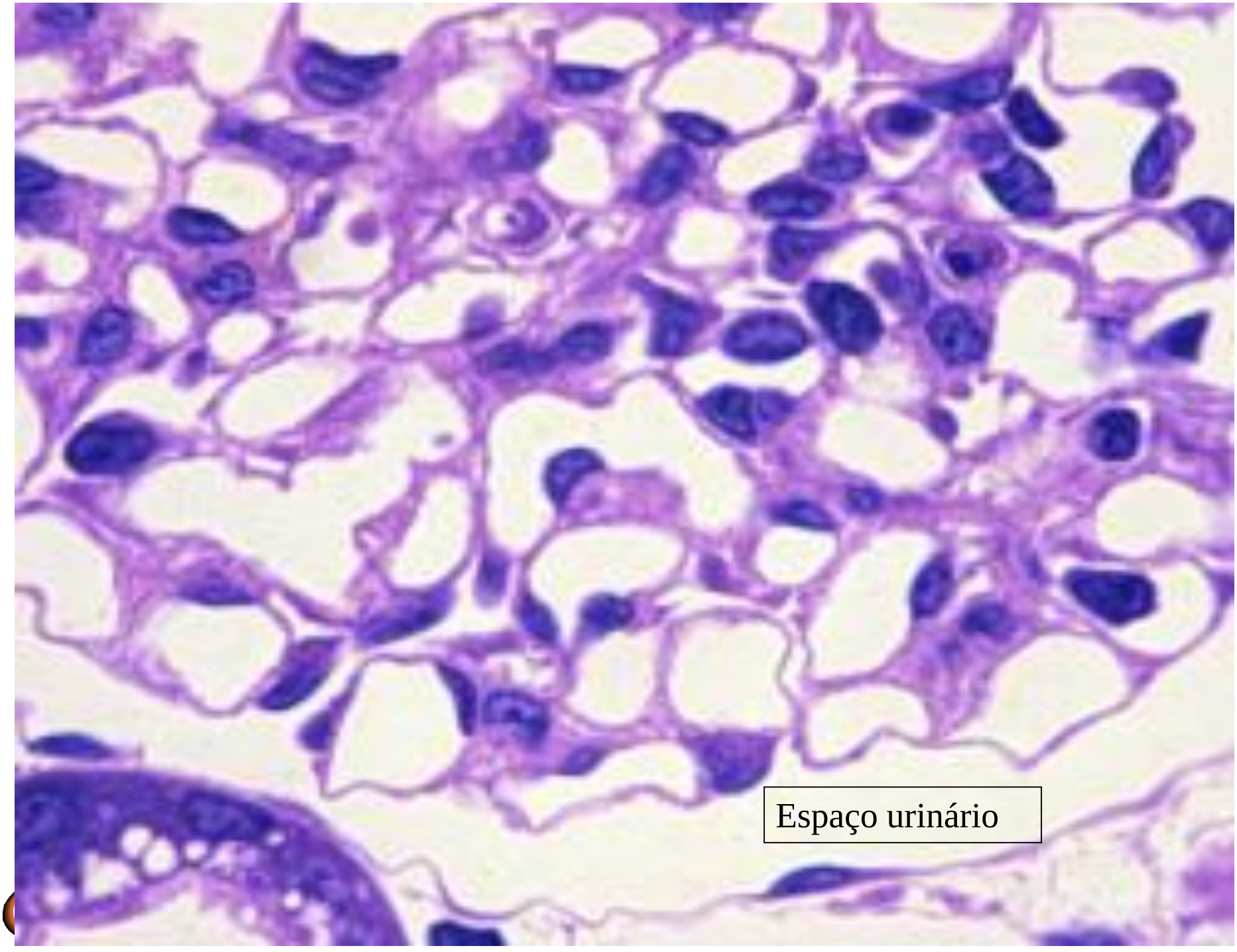
Compartimentos

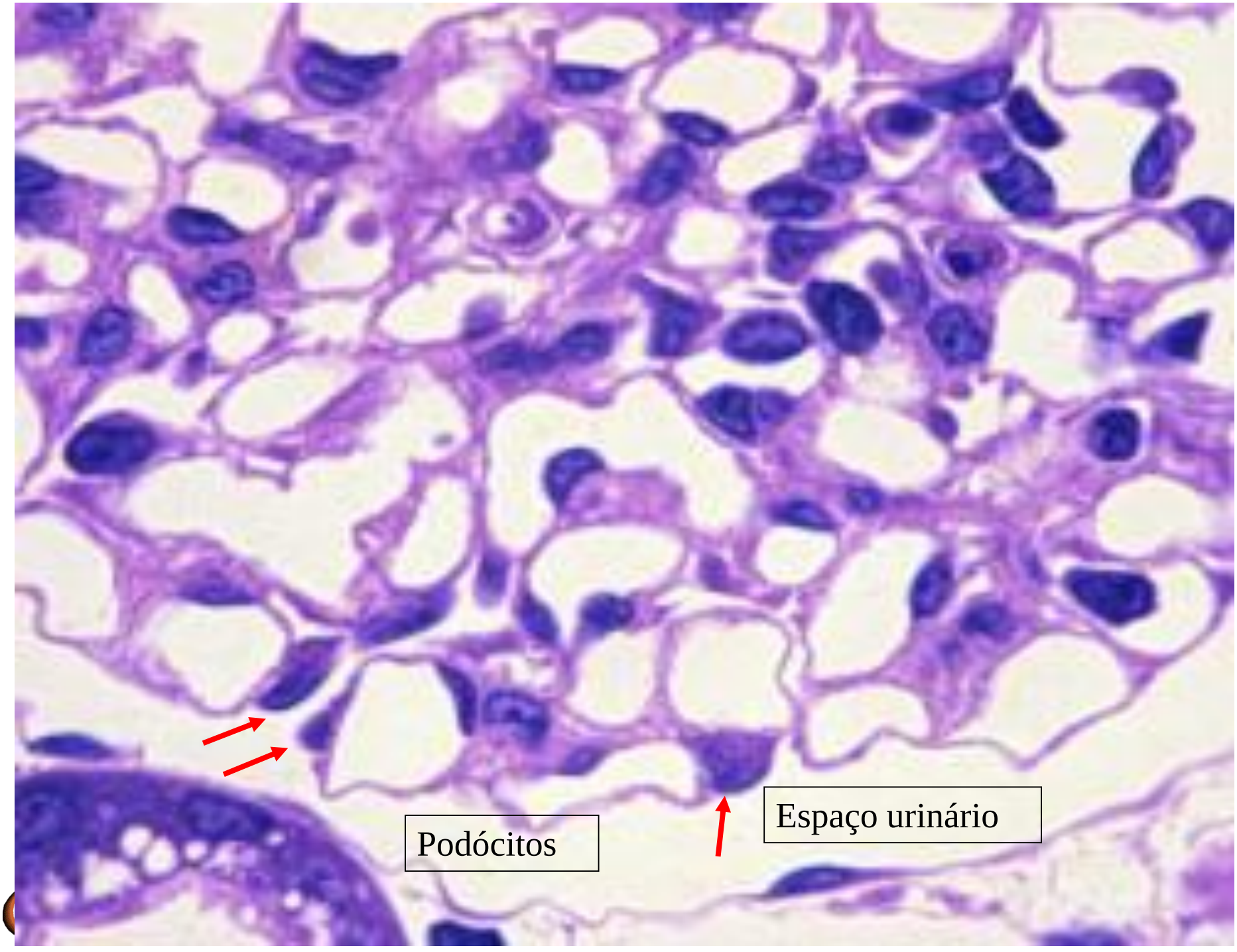


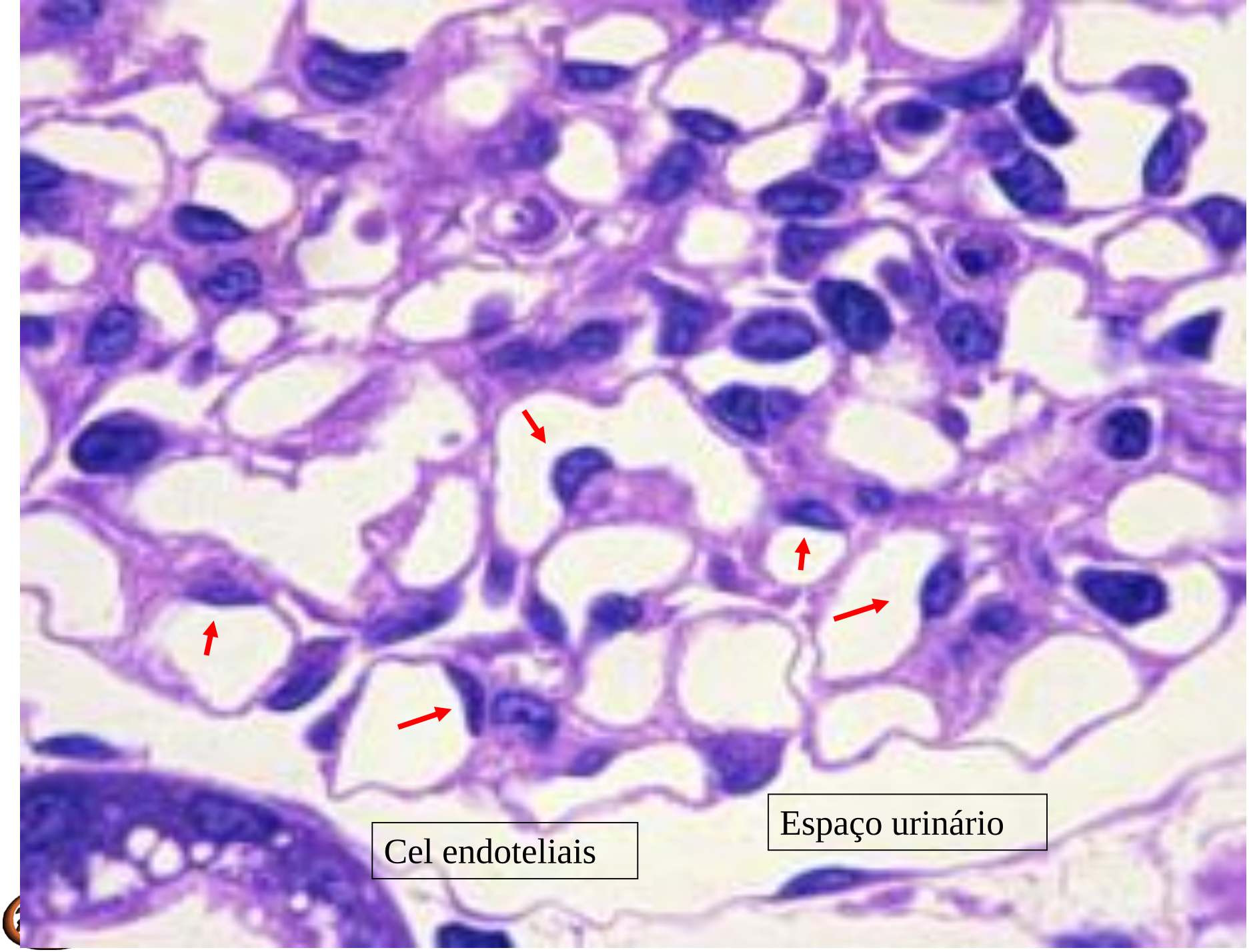


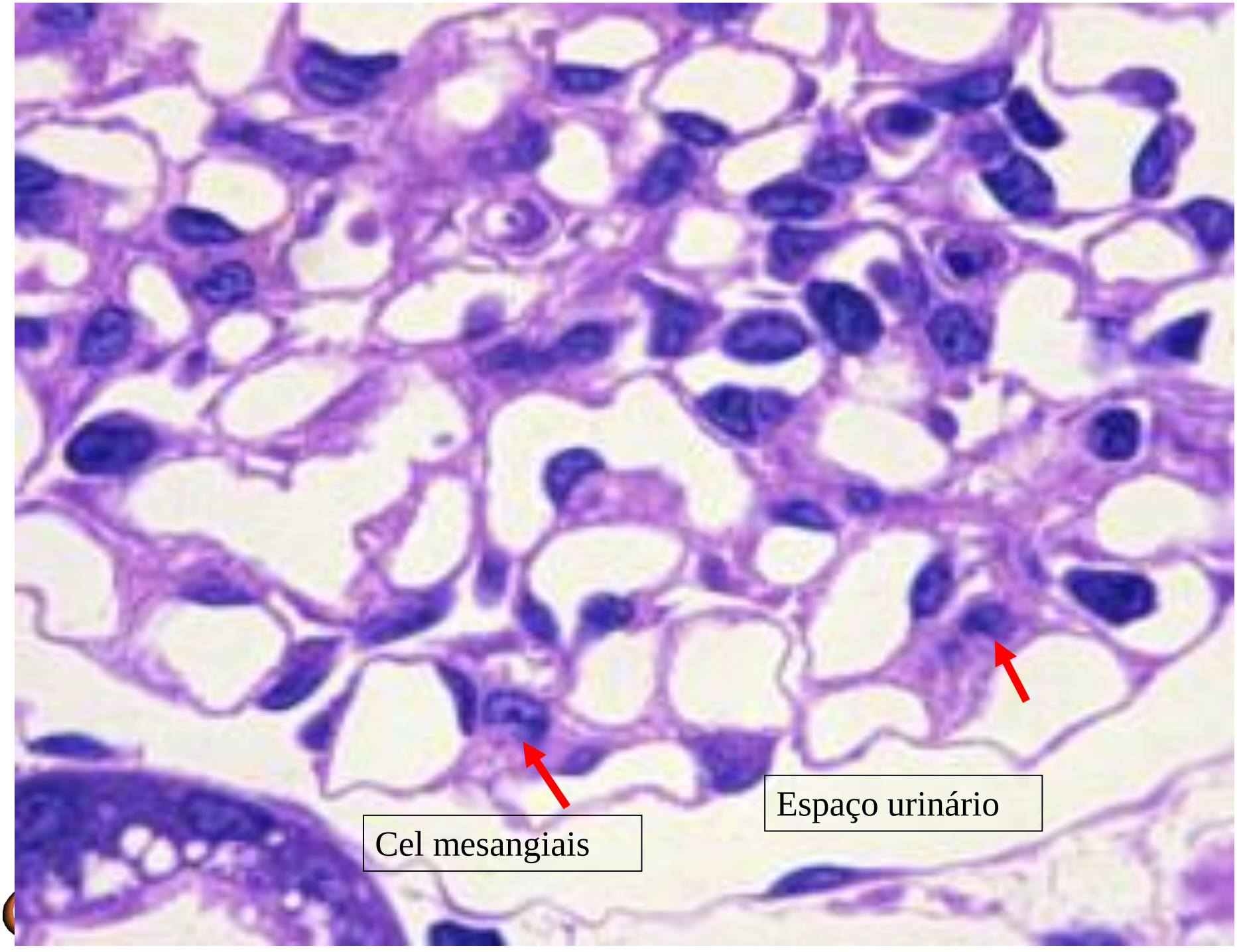


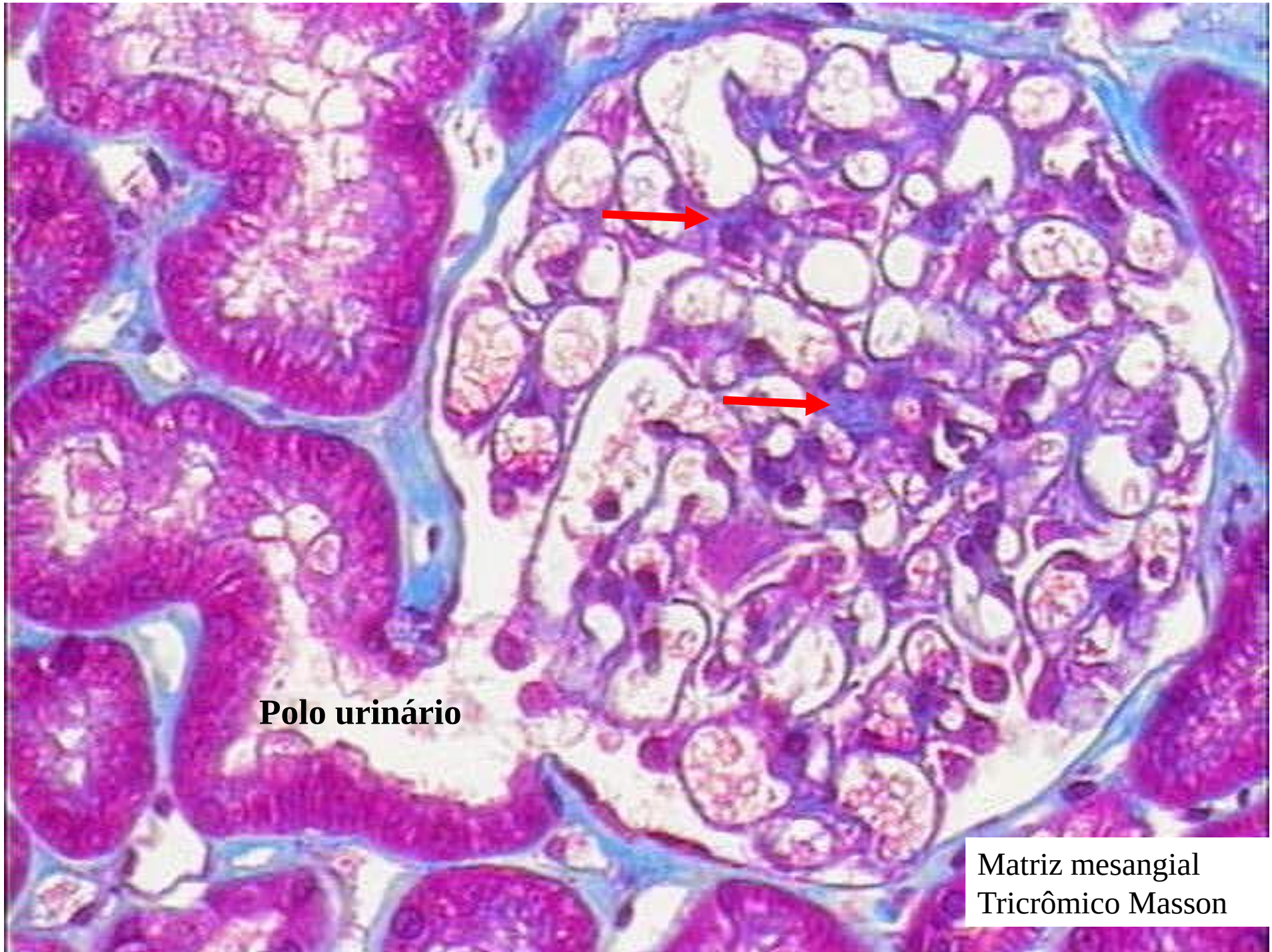
Repercussões túbulo intersticiais
Fibrose intersticial / atrofia tubular


Espaço urinário

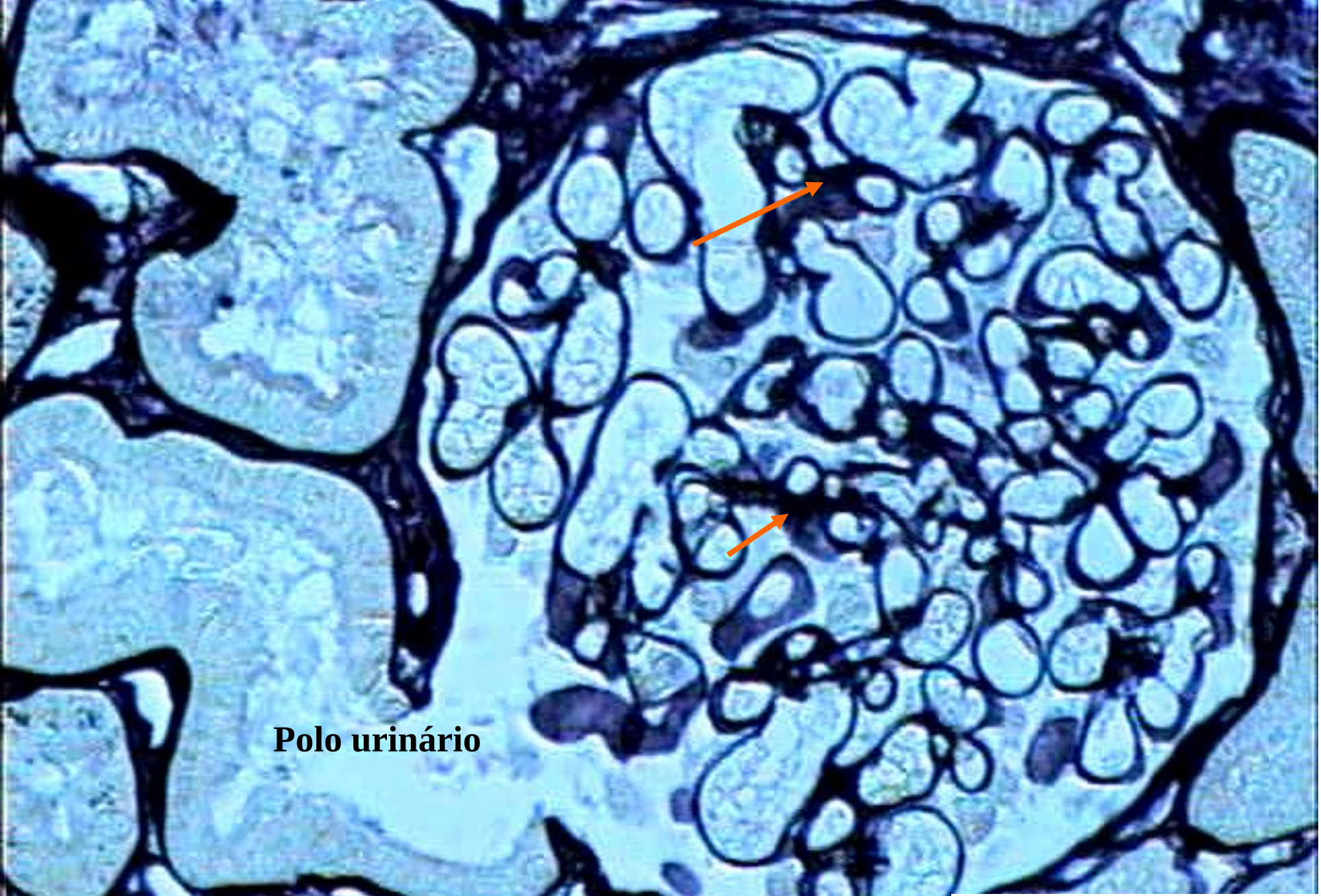

Podócitos
Espaço urinário


Cel endoteliais
Espaço urinário

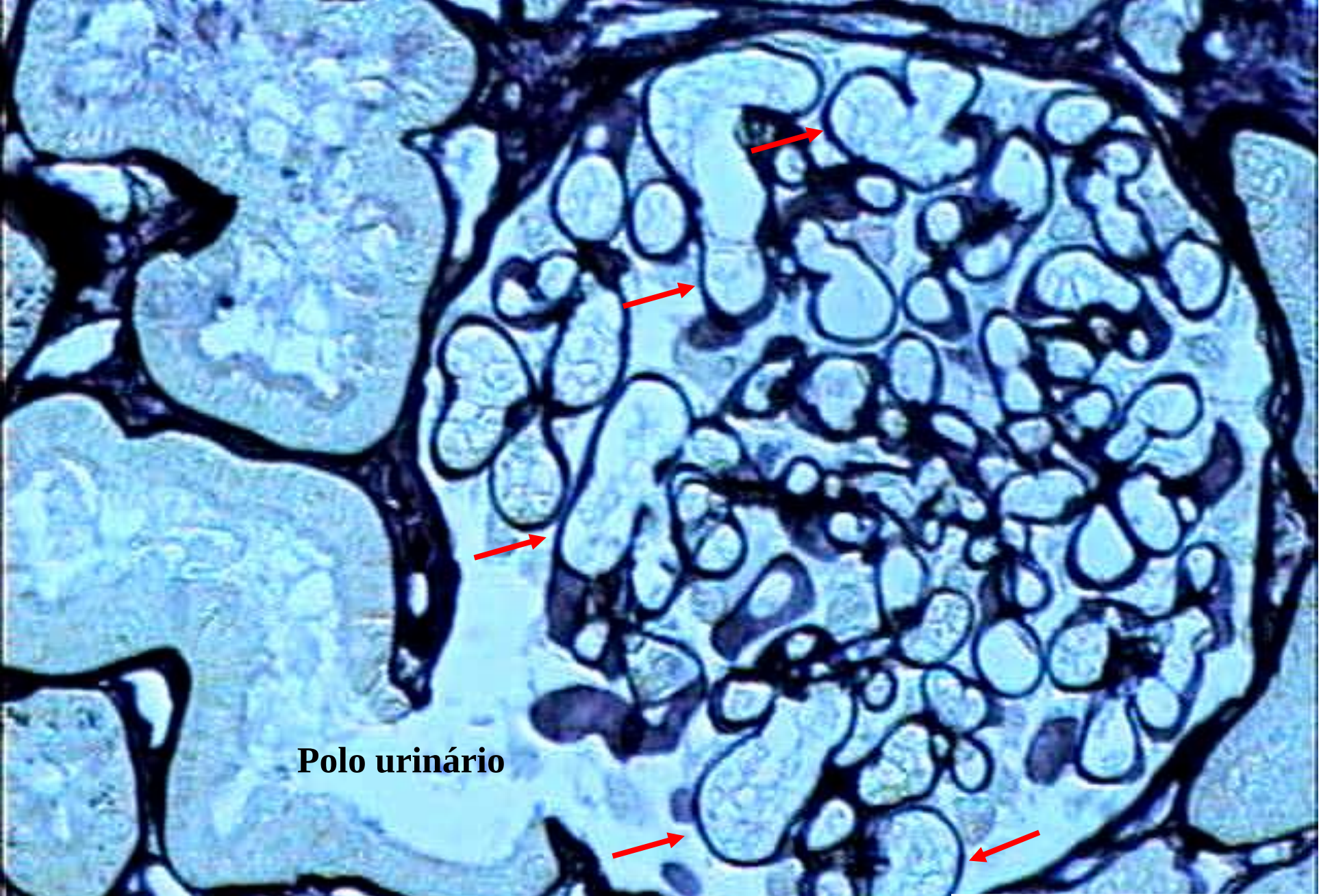

Cel mesangiais
Espaço urinário



Matriz mesangial
Tricrômico Masson



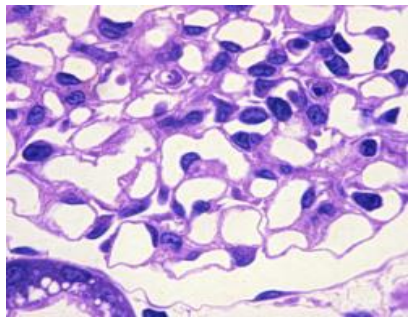
Matriz Mesangial
PAMS – Methenamine Silver

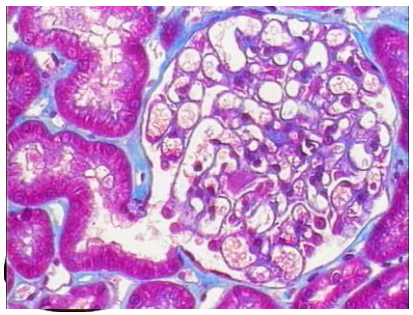


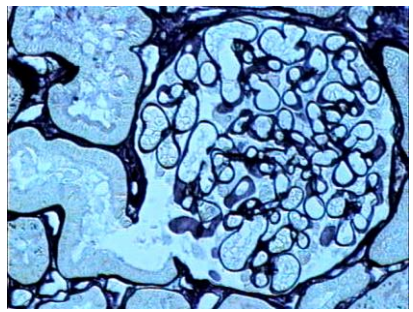
Membrana Basal
PAMS – Methenamine Silver

Biópsia Renal

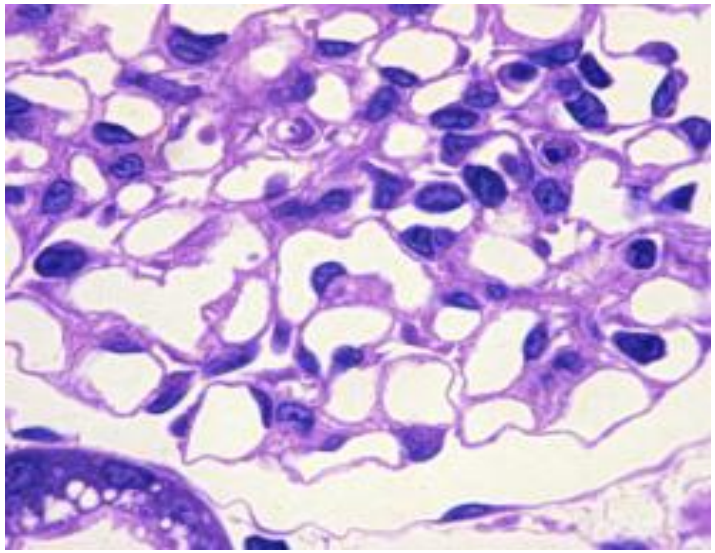
ML-10 gl – paraformaldeido
Formaldeído / Bouin / Metacarn

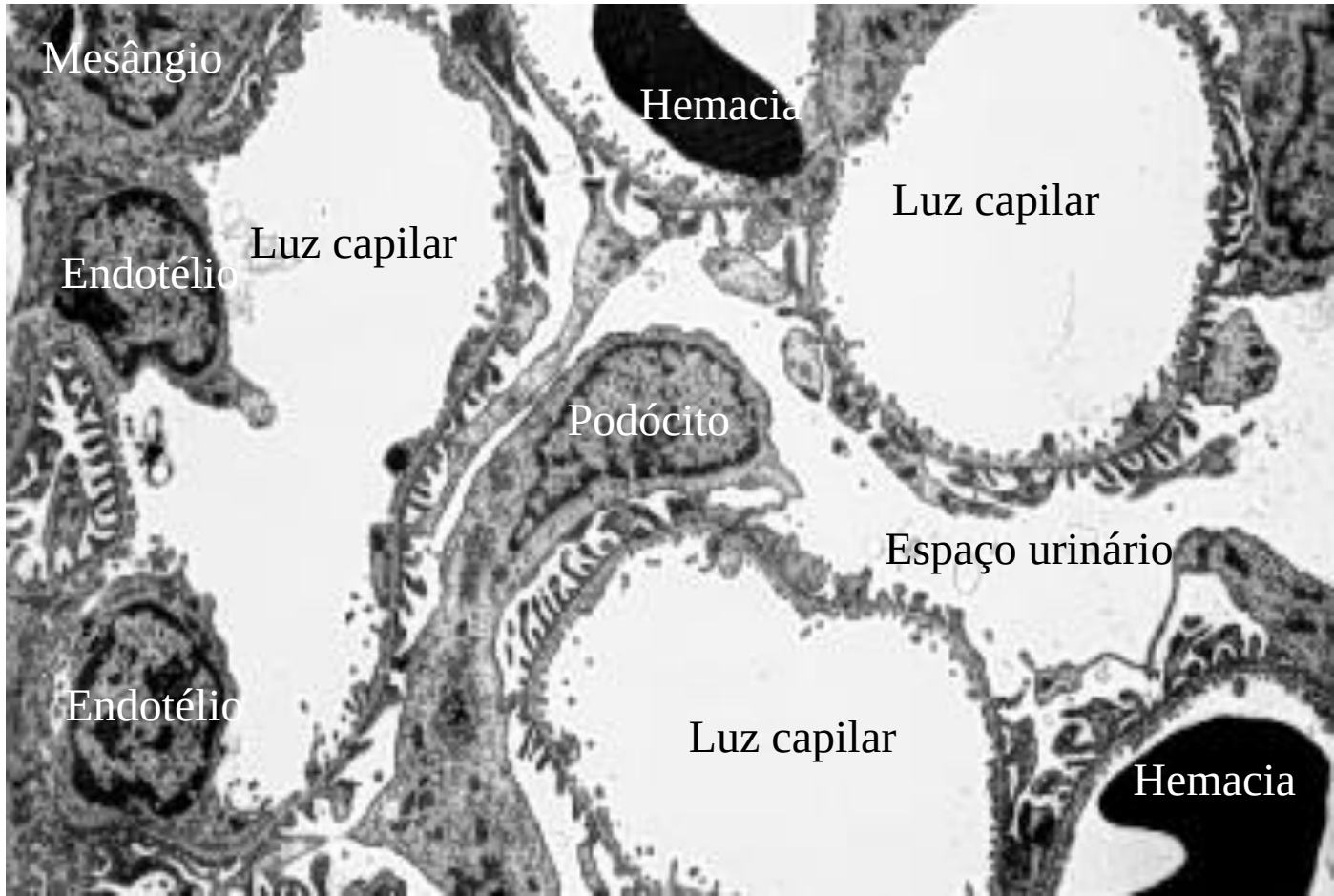






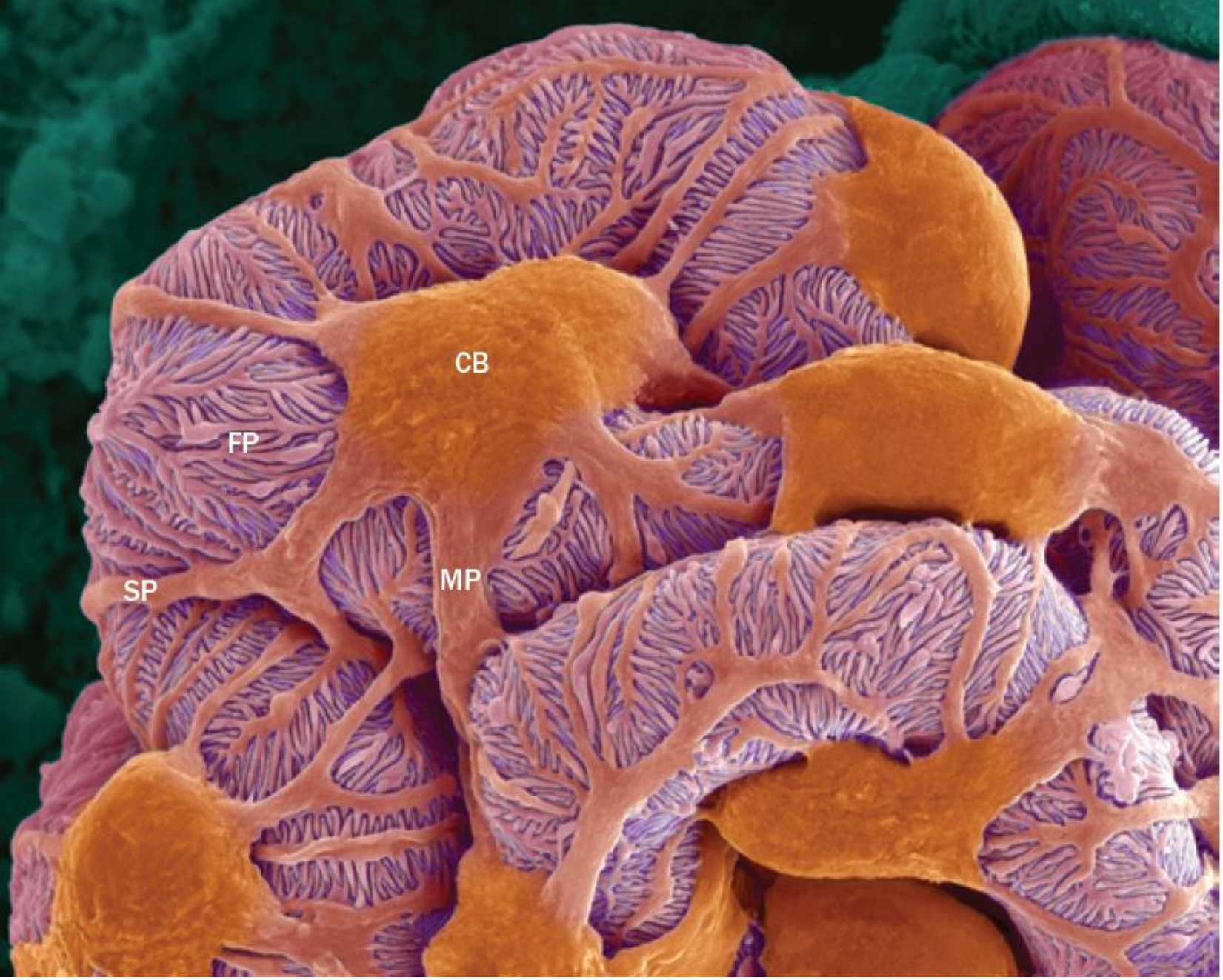
ME-2gl – Glutaraldeído
/ Karnovsky + Vermelho rutênio





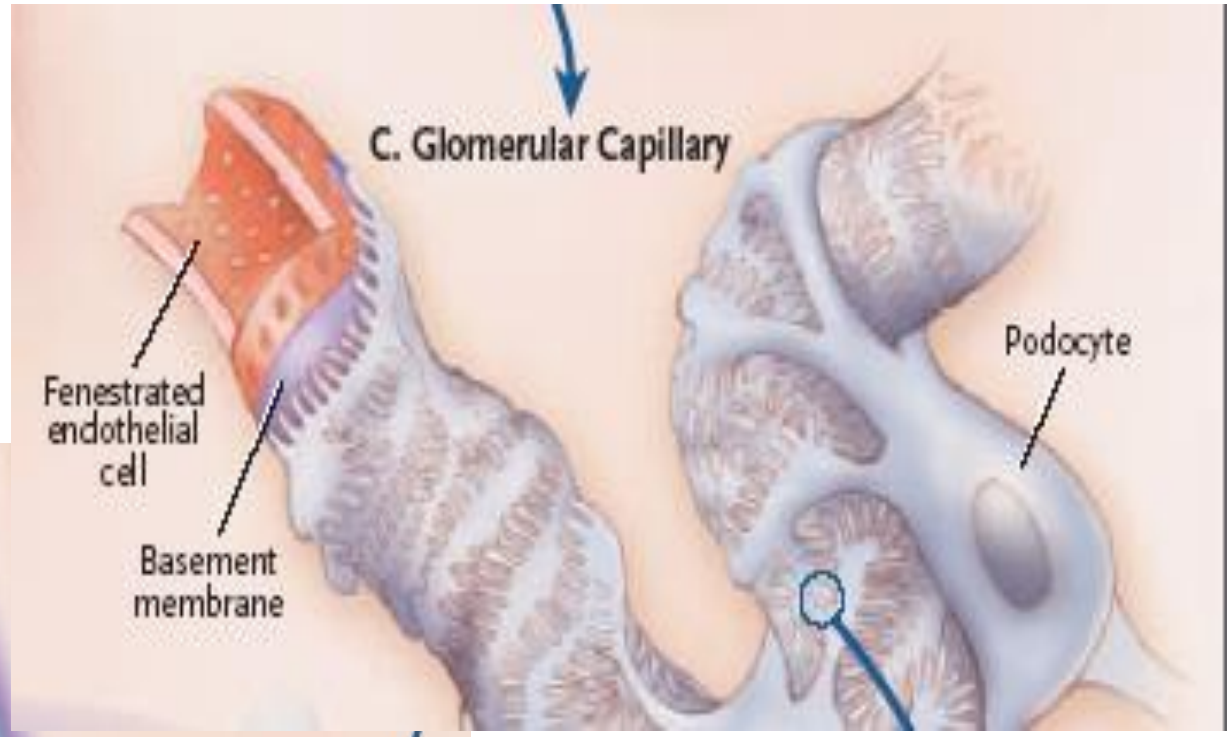
- Diagnósticos:
  - Alt célula
  - Memb basal
  - Matriz mesangial
  - Depósitos estruturados

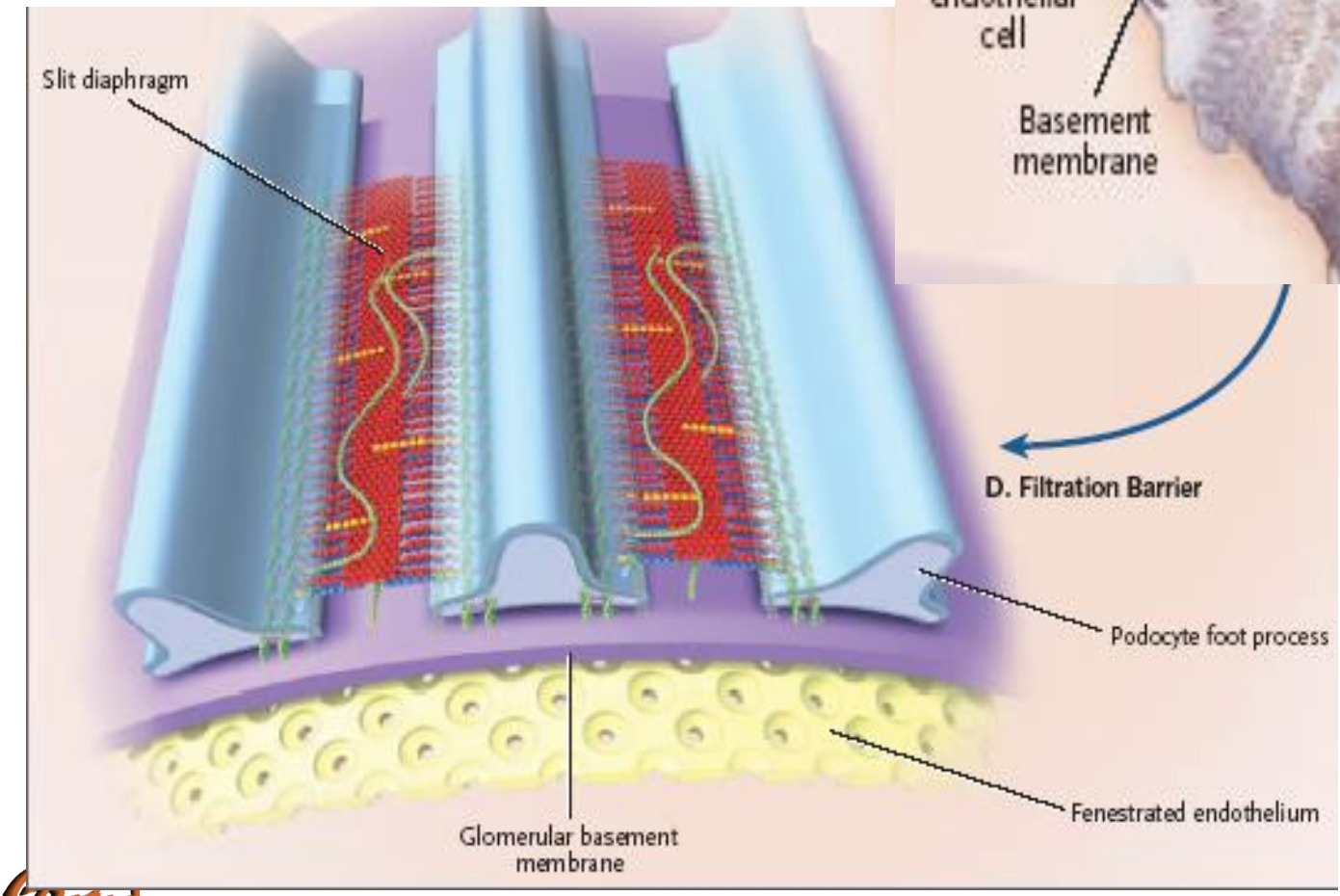
- Entidades sobrepostas
- Afastar entidades



Welsh, G. I. & Saleem, M. A. *Nat. Rev. Nephrol.* **8**, 14–21 (2012)

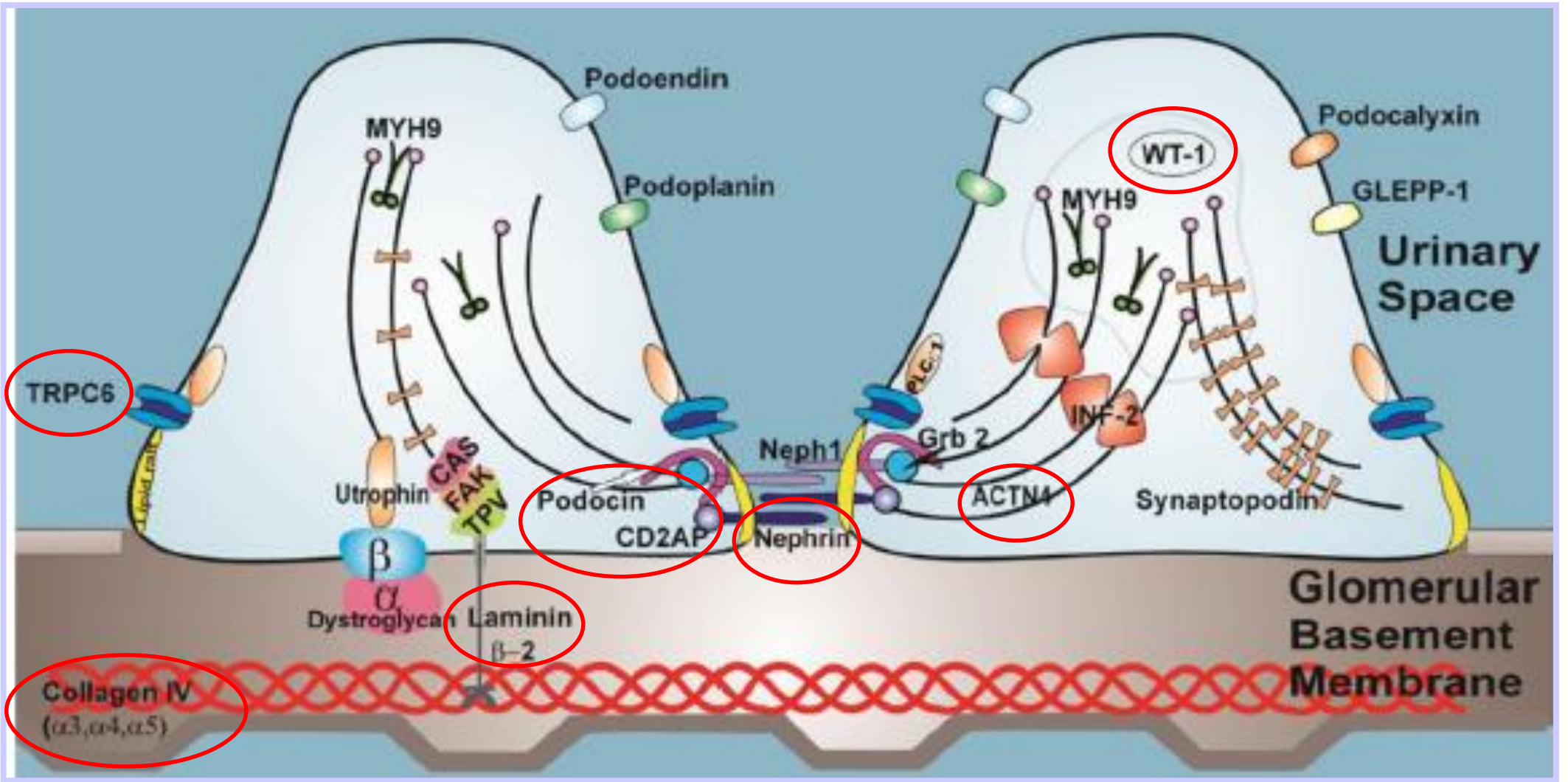
Barreira de Filtração Glomerular

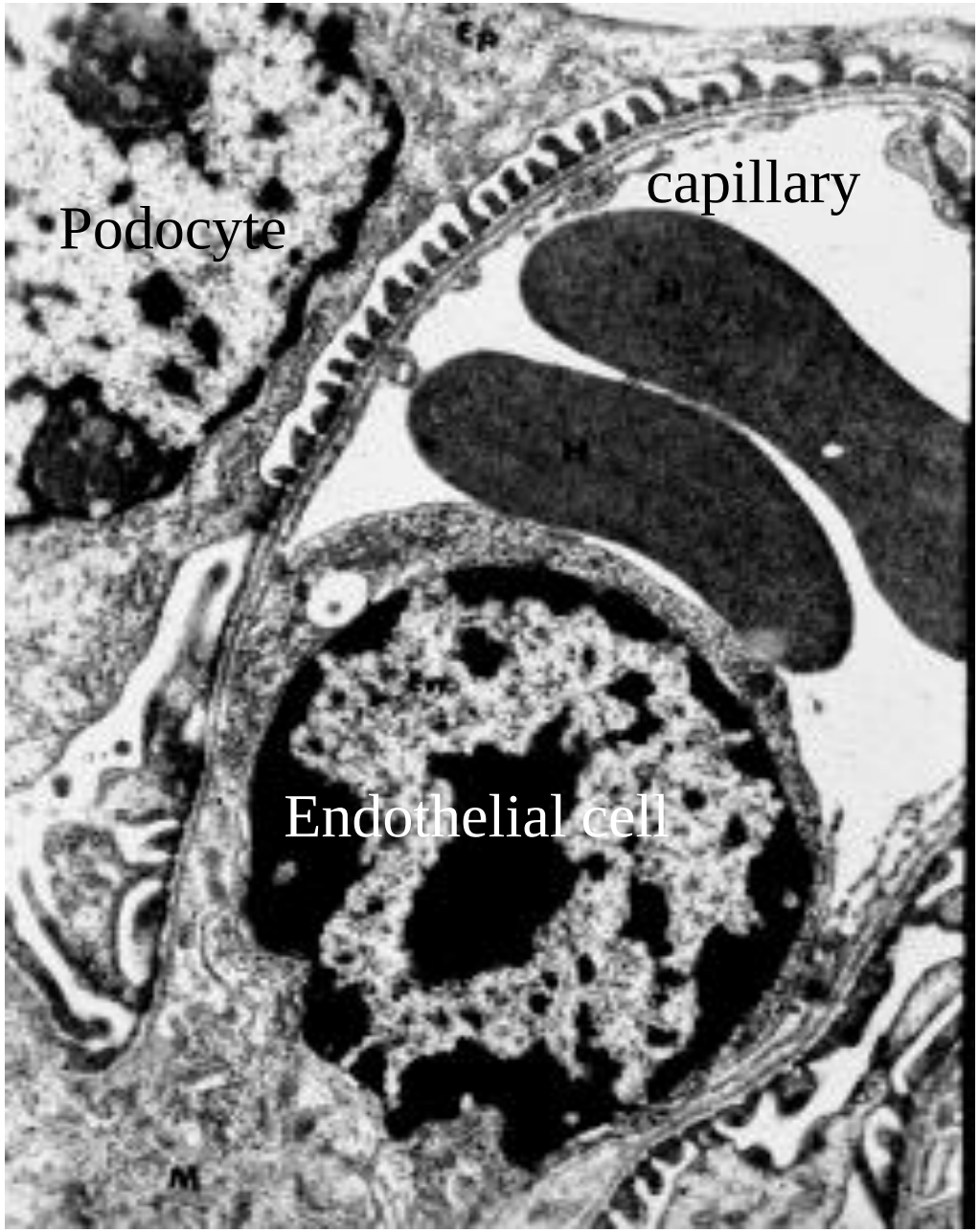


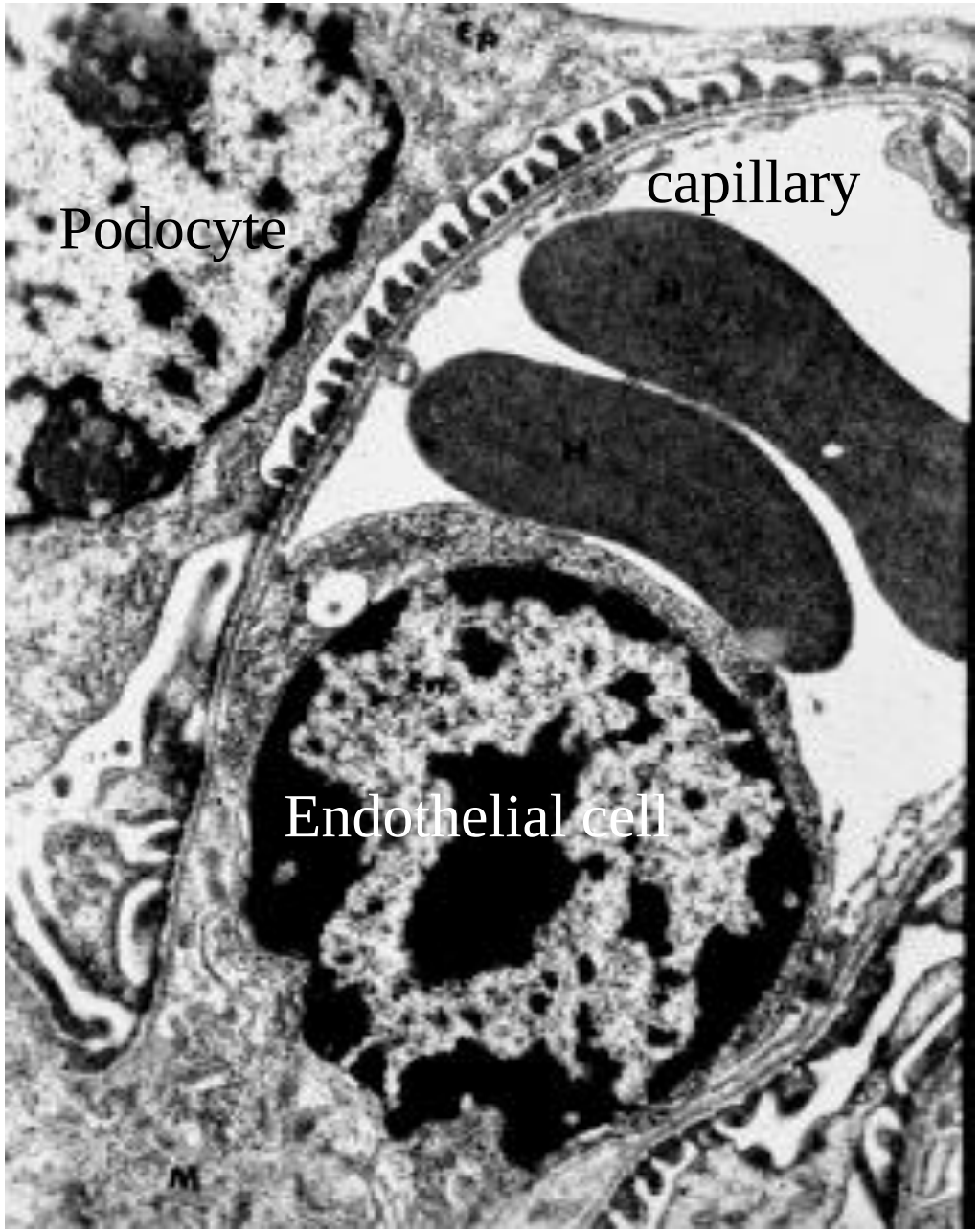


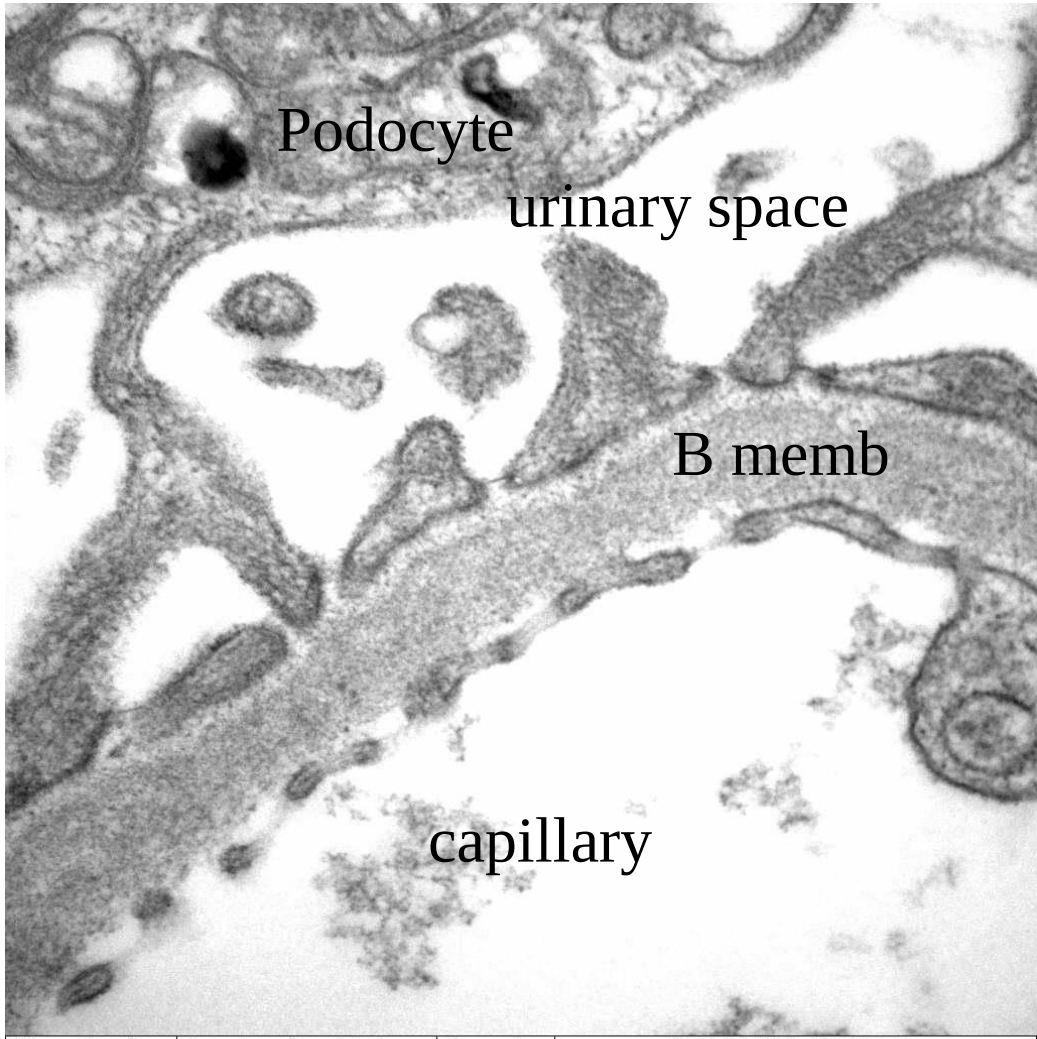
Tryggvason et al., 2006

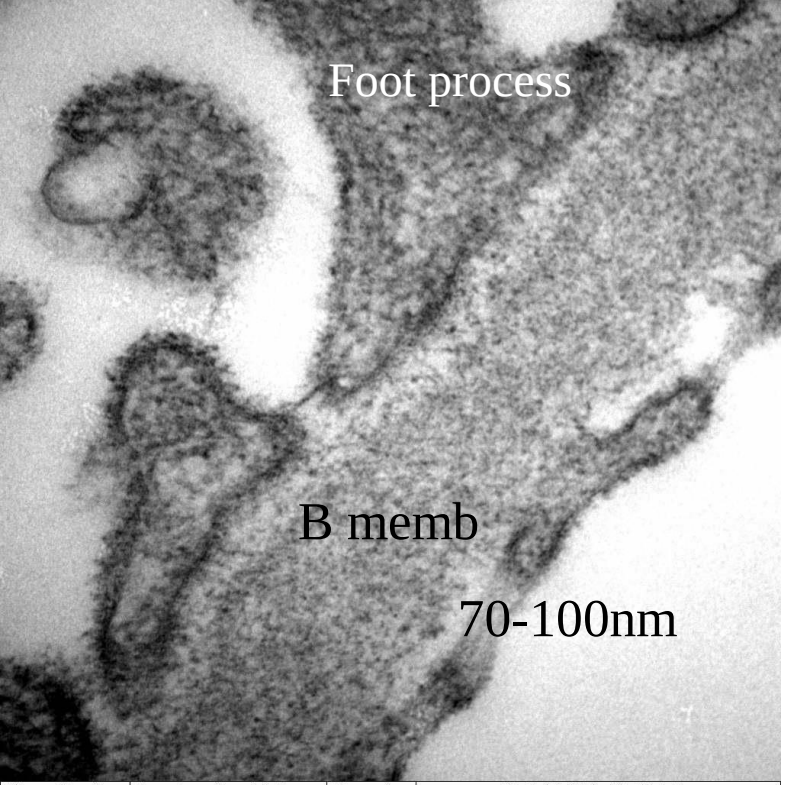
Componentes da barreira de filtração glomerular



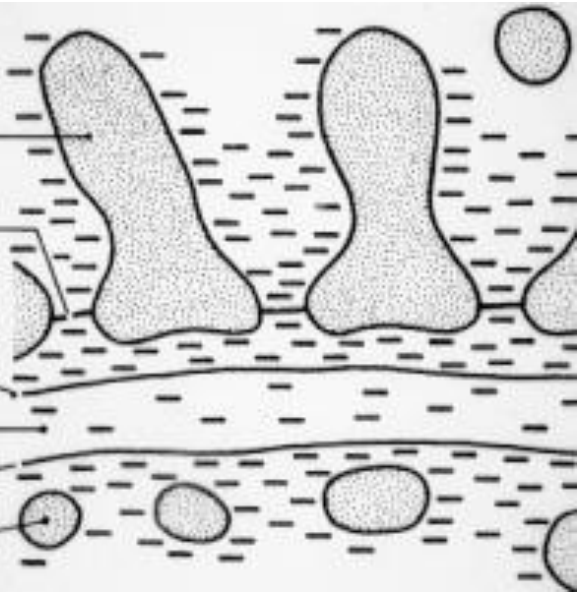

Podocyte
capillary
Endothelial cell

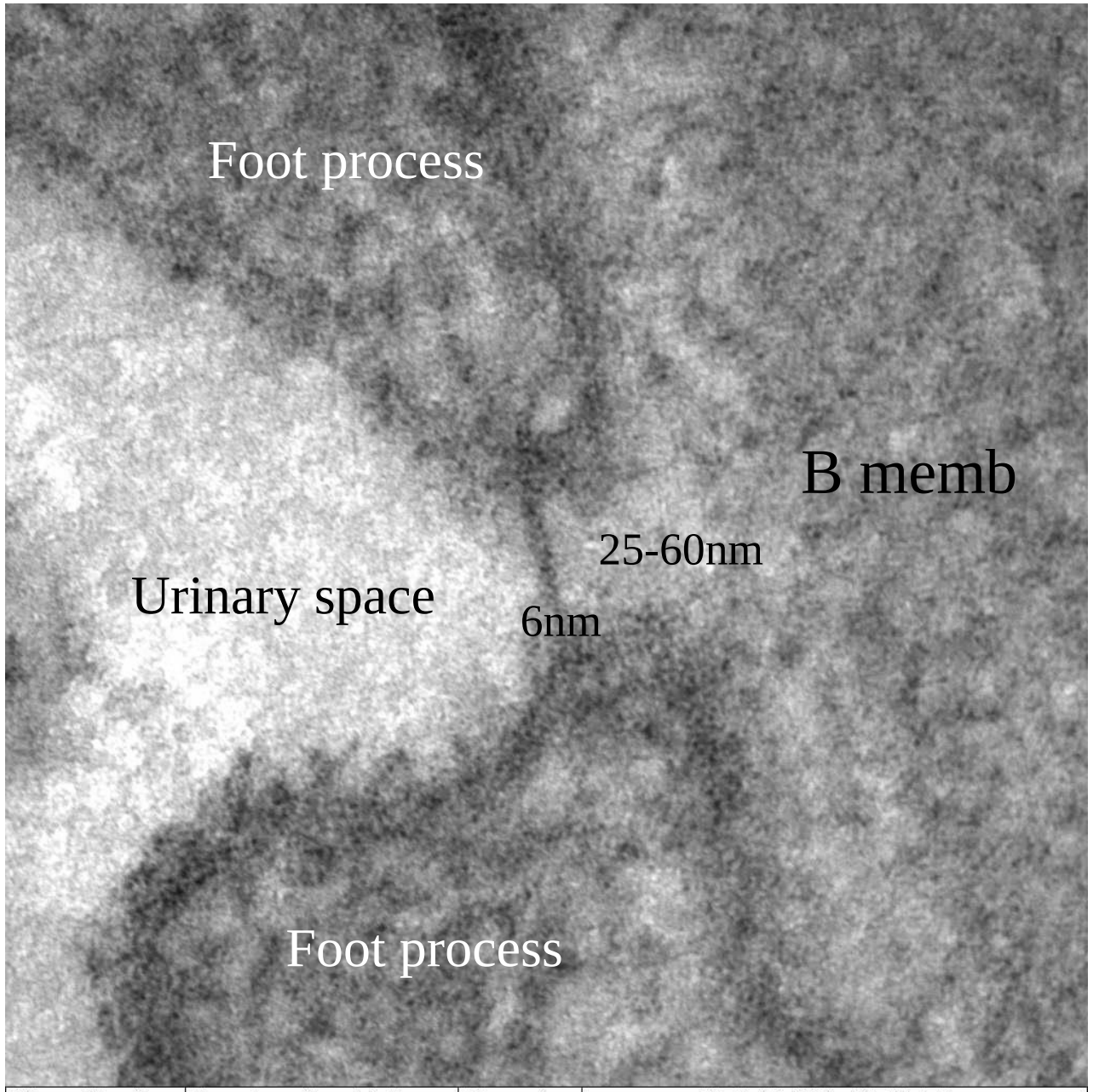

Podocyte
capillary
Endothelial cell


Podocyte
urinary space
B memb
capillary
Magnification
30000 x
Accelerating Voltage
80 kV
Operator
Mar
BR140439_ME2771e
500 nm



Magnification	Accelerating Voltage	Operator	BR140439_ME2771e
85000 x	80 kV	Mar	200 nm

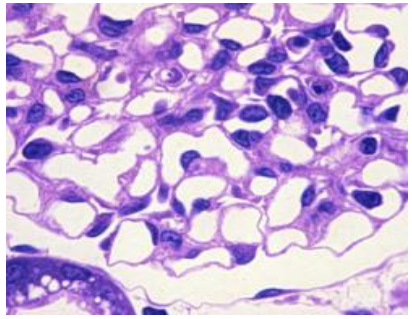


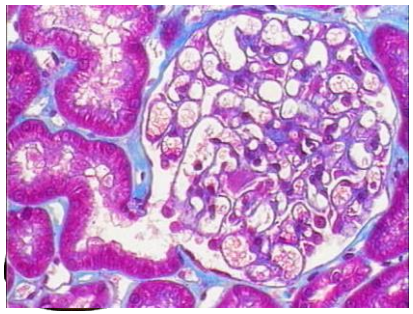


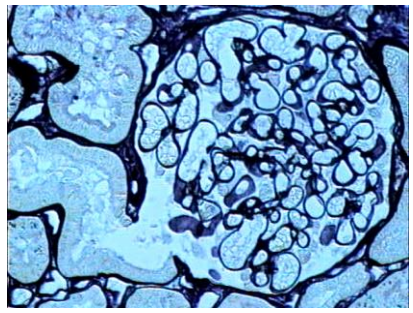
Magnification	Accelerating Voltage	Operator	BR140439_ME2771e
250000 x	80 kV	Mar	50 nm

Biópsia Renal

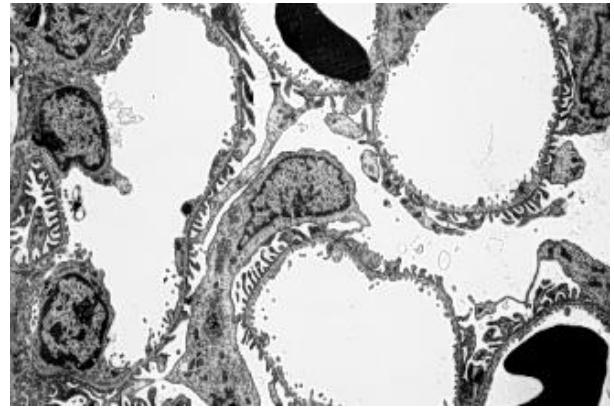
ML-10 gl – paraformaldeido
Formaldeído / Bouin / Metacarn

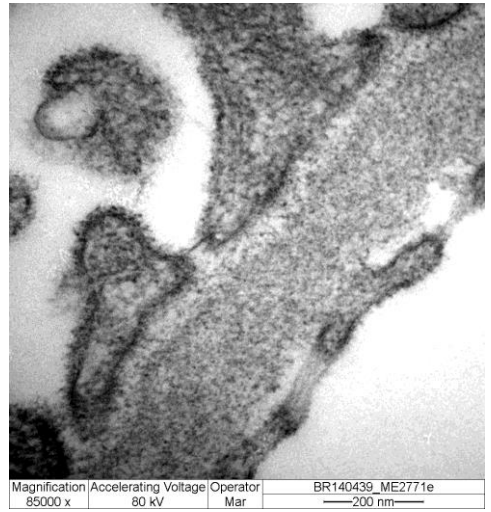


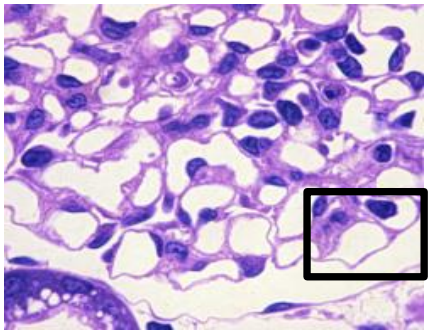


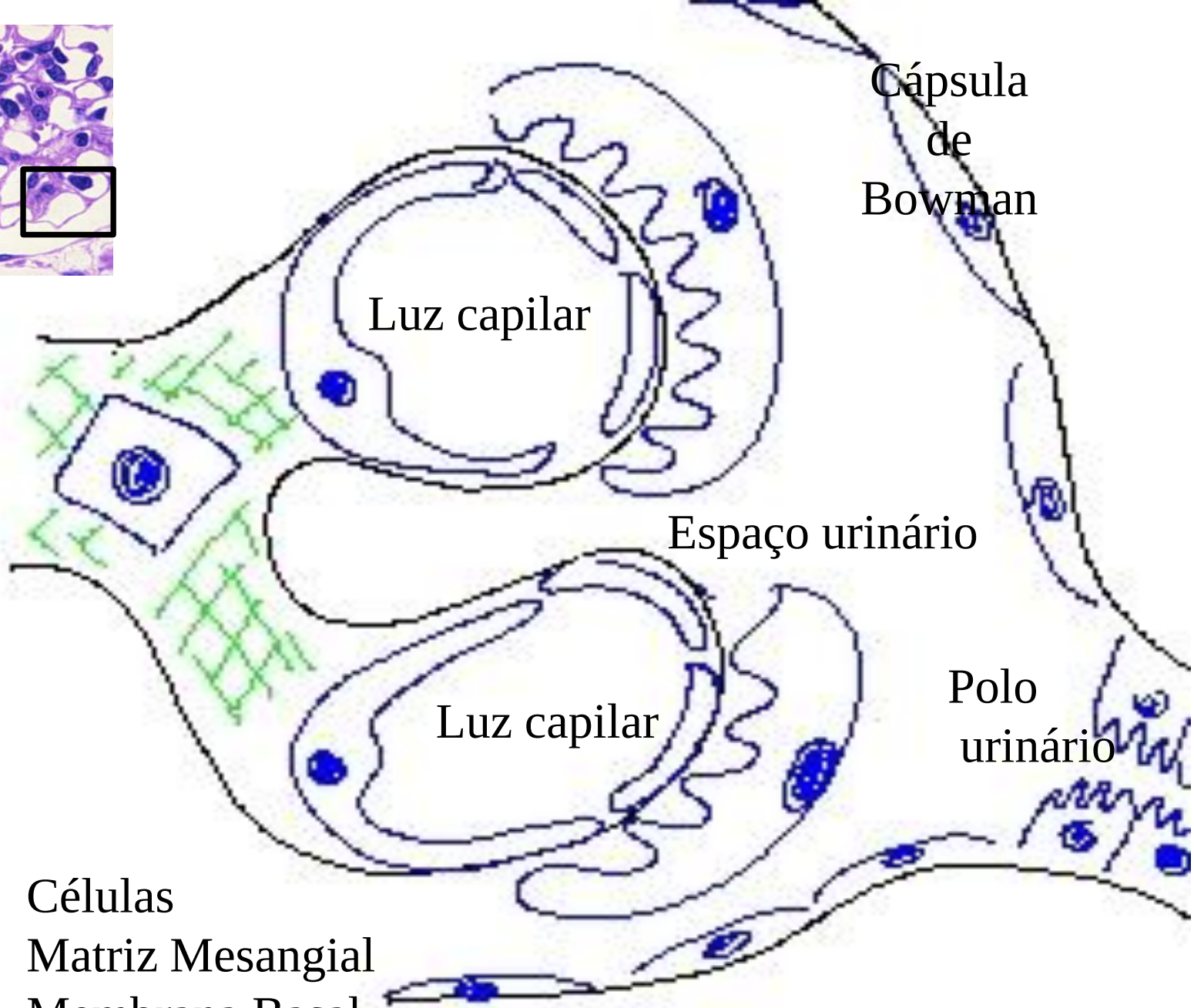


ME-2gl – Glutaraldeído
/ Karnovsky + Vermelho rutênio



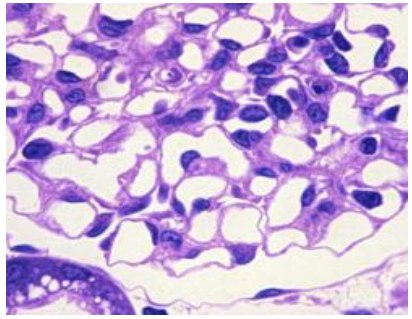





Cápsula
de
Bowman
Luz capilar
Espaço urinário
Luz capilar
Polo
urinário
Células
Matriz Mesangial
Membrana Basal

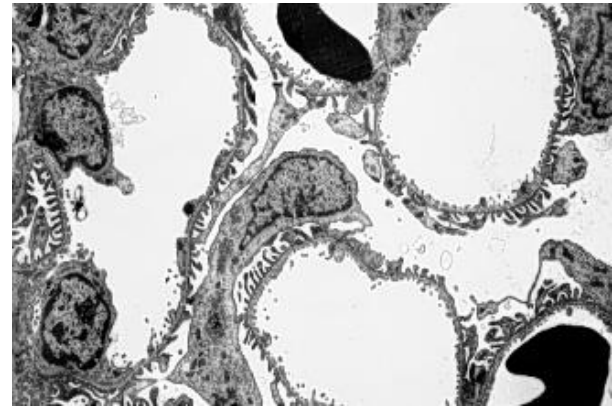
Biópsia Renal

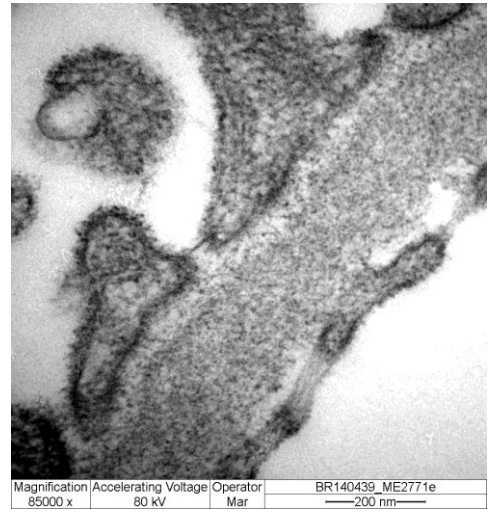
ML-10 gl – paraformaldeido
Formaldeído / Bouin / Metacarn

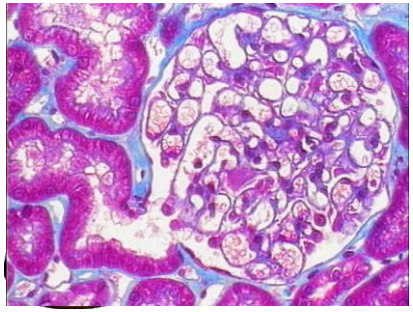


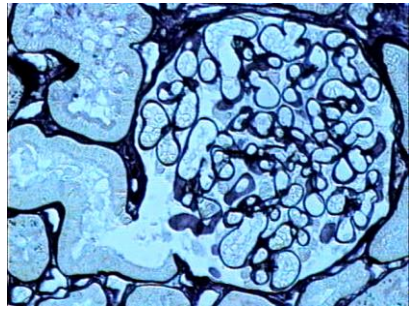
IF-10gl – IC/compl
A fresco / Meio de transporte

ME-2gl – Glutaraldeído
/ Karnovsky + Vermelho rutênio





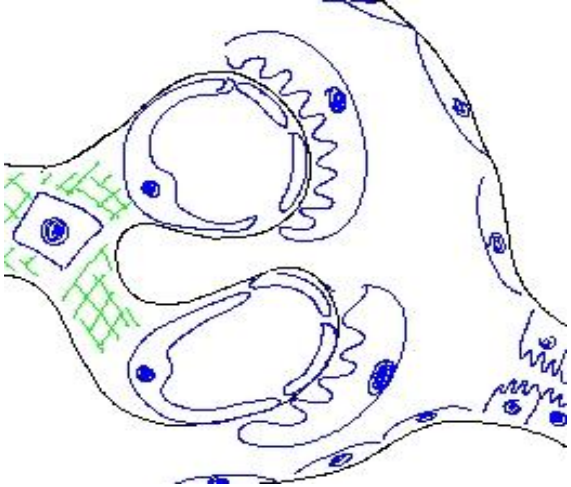




Pesquisa de Imunocomplexos
Imunofluorescência Direta

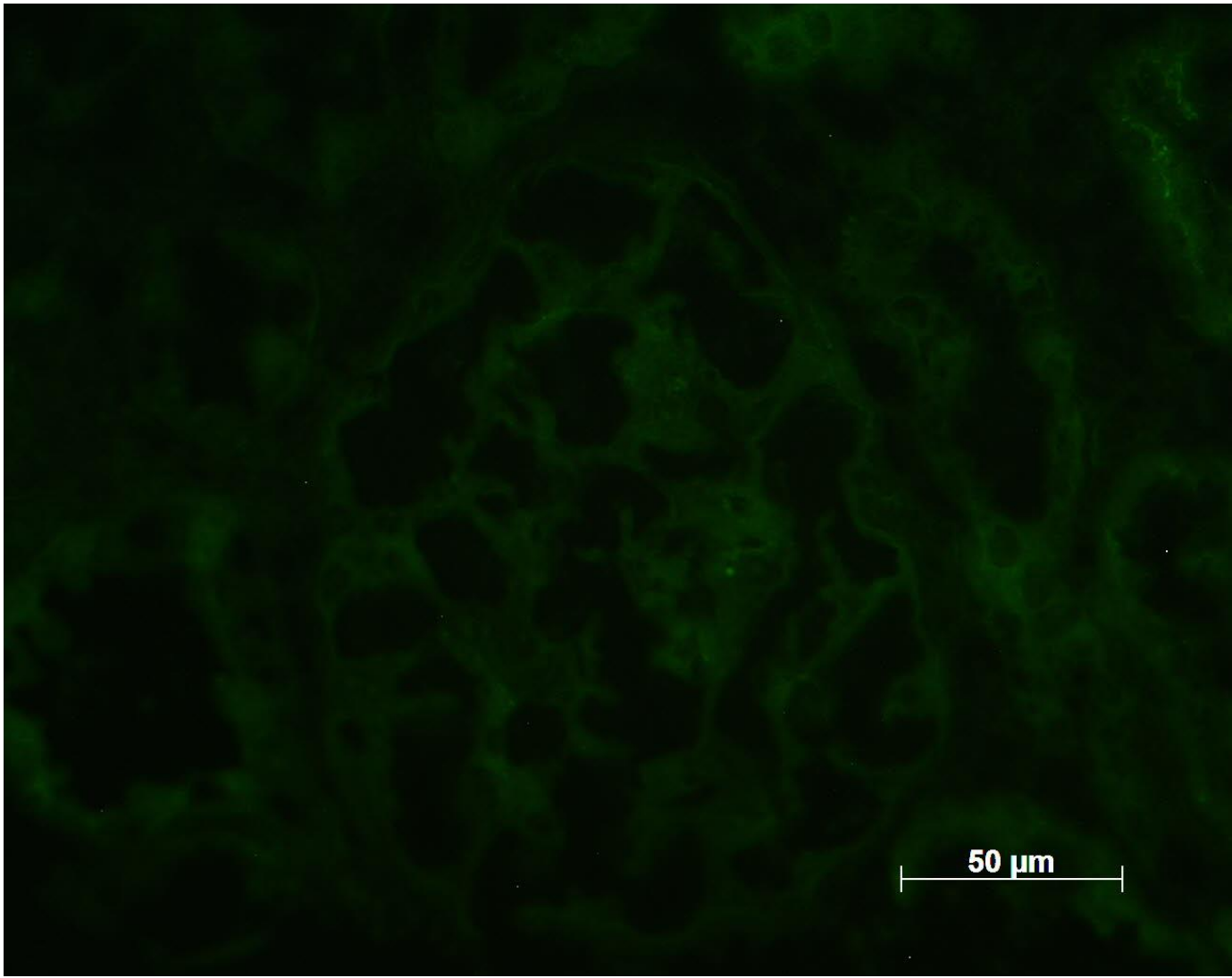
IF: 10 glom

- IgA
- IgG
- IgM
- *Kappa*
- *Lambda*
- Fibrinogênio
- C1q
- C3
- **Controle negativo**



Normal

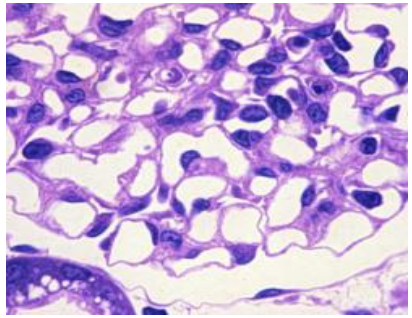
- IgA
- IgG
- IgM
- *Kappa*
- *Lambda*
- Fibrinogênio
- C1q
- C3



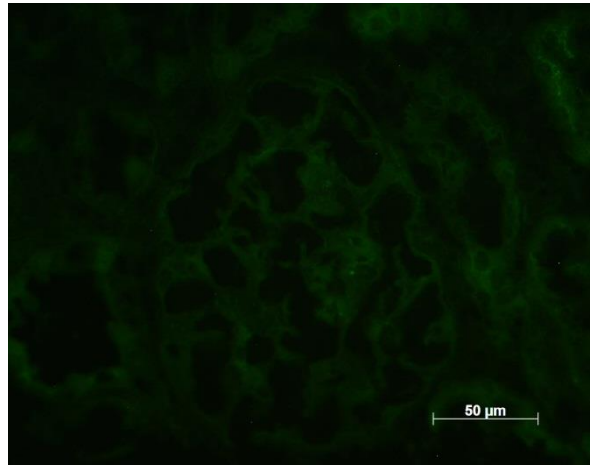
Biópsia Renal

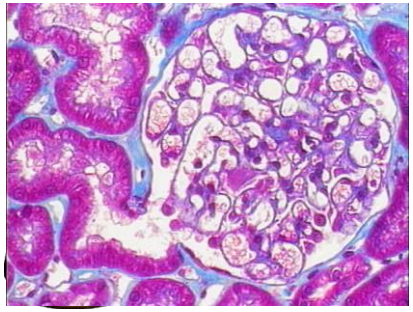
ML-10 gl – paraformaldeido
Formaldeído / Bouin / Metacarn

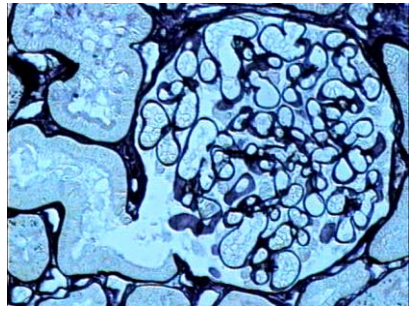
IF-10gl – IC/compl
A fresco / Meio de transporte



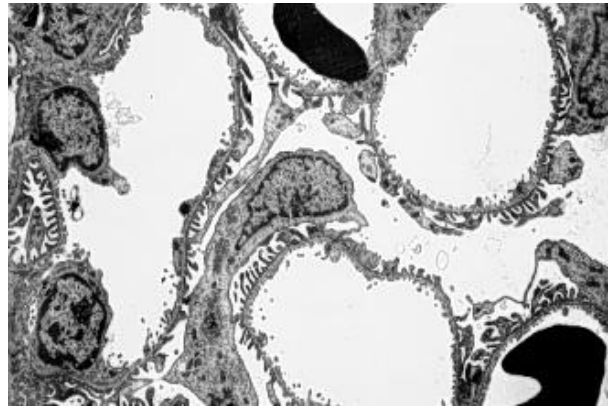
- IgA
- IgG
- IgM
- *Kappa*
- *Lambda*
- Fibrinogênio
- C1q
- C3

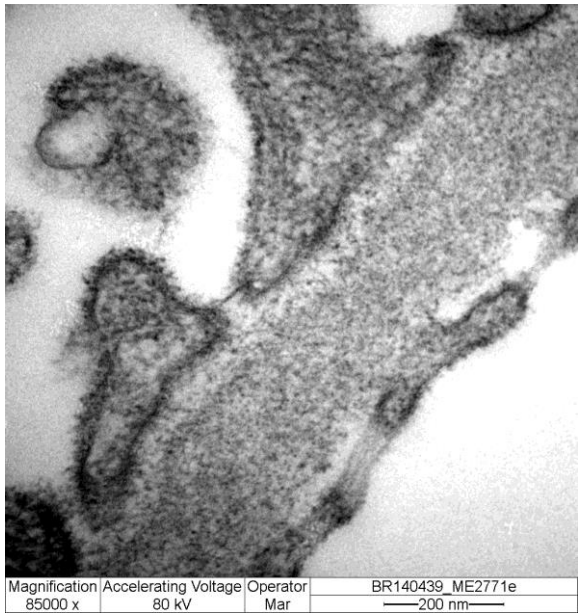




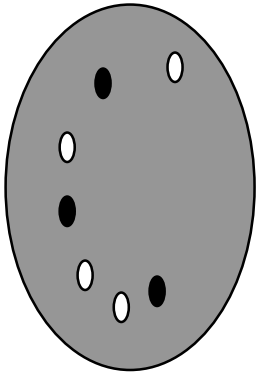


ME-2gl – Glutaraldeído
/ Karnovsky + Vermelho rutênio

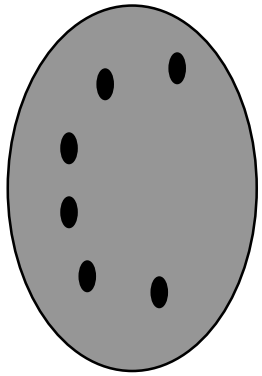




Número glomérulos

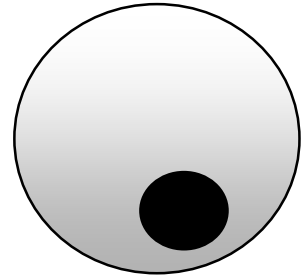


Focal < 50%

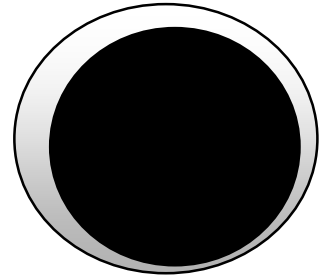


Difuso ≥ 50%

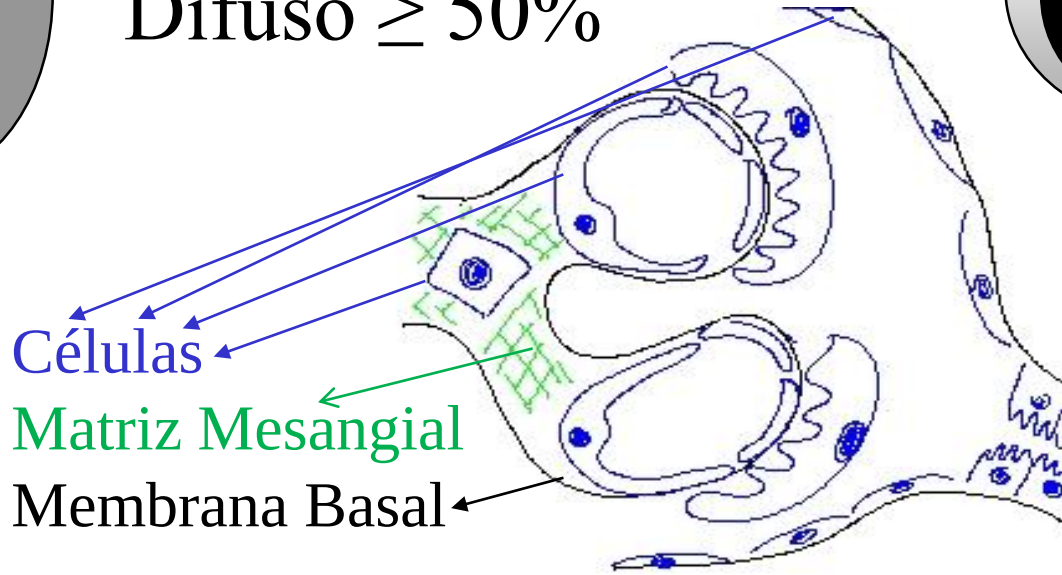
No glomérulo

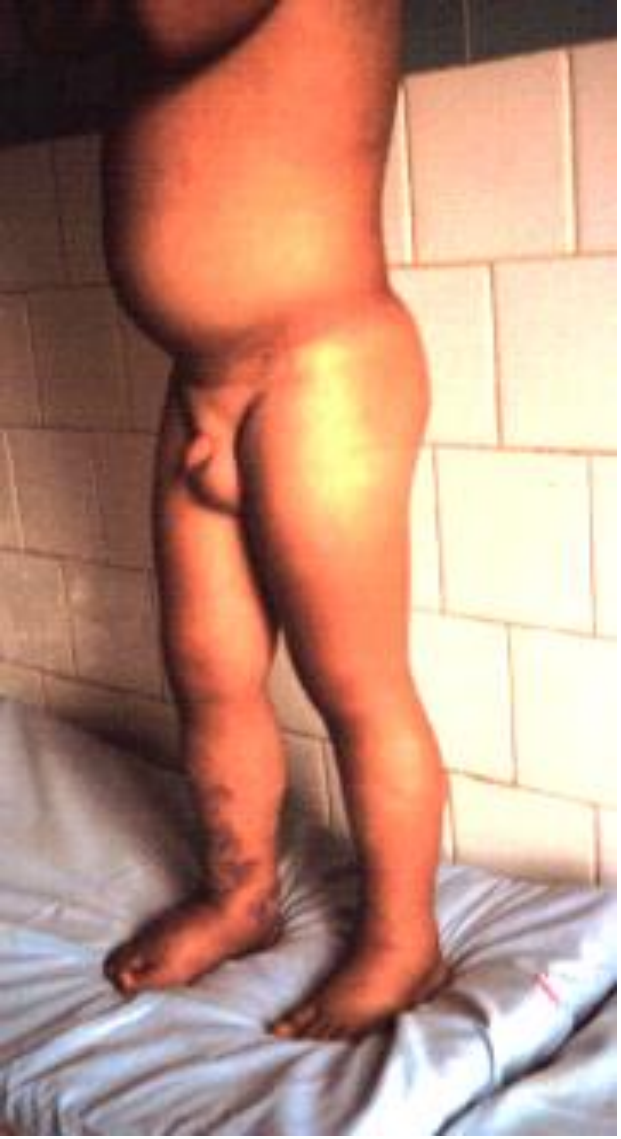


Segmentar



Global





- 5 anos de idade
- sexo masculino
- edema generalizado há 3 meses
- sem HAS ou hematúria

Exames:

- proteinúria = 5g/24h;
- Prot. totais = 3,5g%;
- Albumina sérica = 2,3g%;
- Colesterol=501mg%;
- Uréia=64mg%;
- Creatinina = 0,8mg%

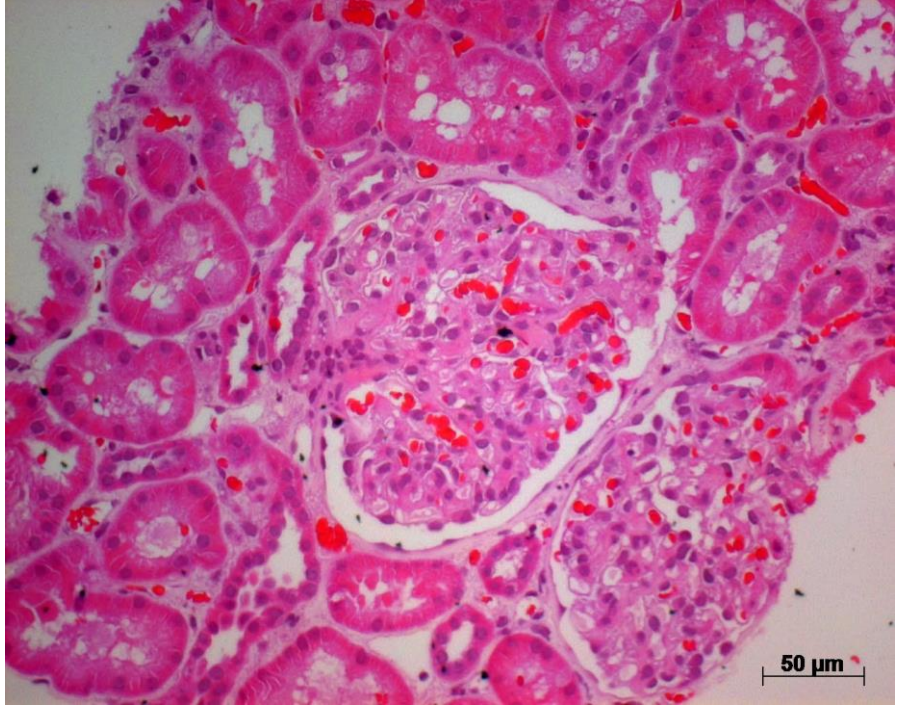
Manifestações Clínicas - Glomerulopatias

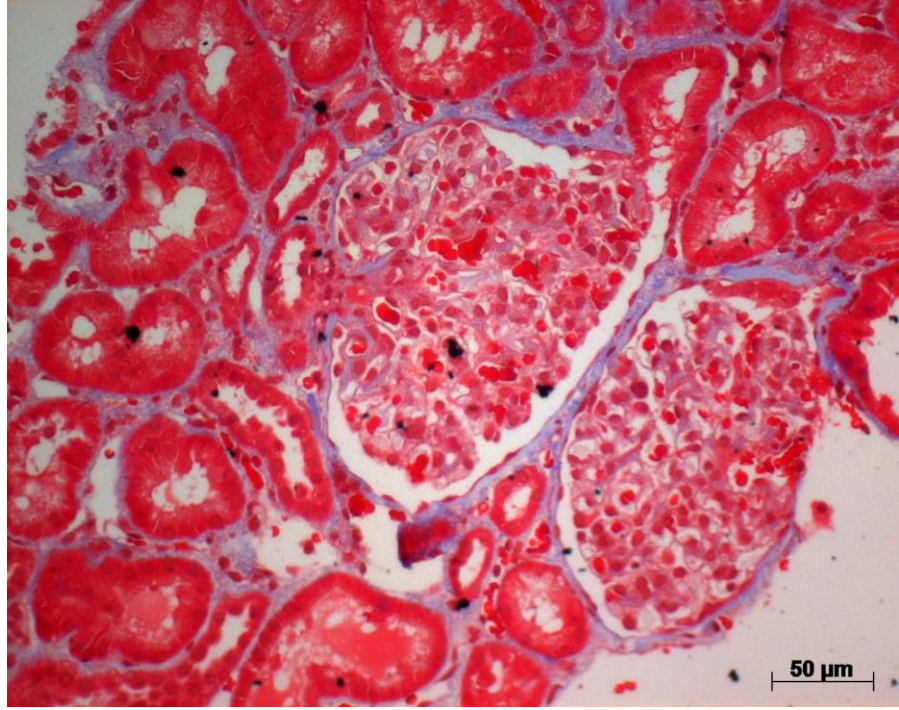
1. **Alt urinárias assintomáticas**
   a) **hematúria glomerular isolada**
   b) **Proteinúria isolada**
2. **síndrome nefrótica**
3. **síndrome nefrítica**
4. **IRA (insuficiência aguda – rapidamente progressiva)**
5. **Doença Renal Crônica**

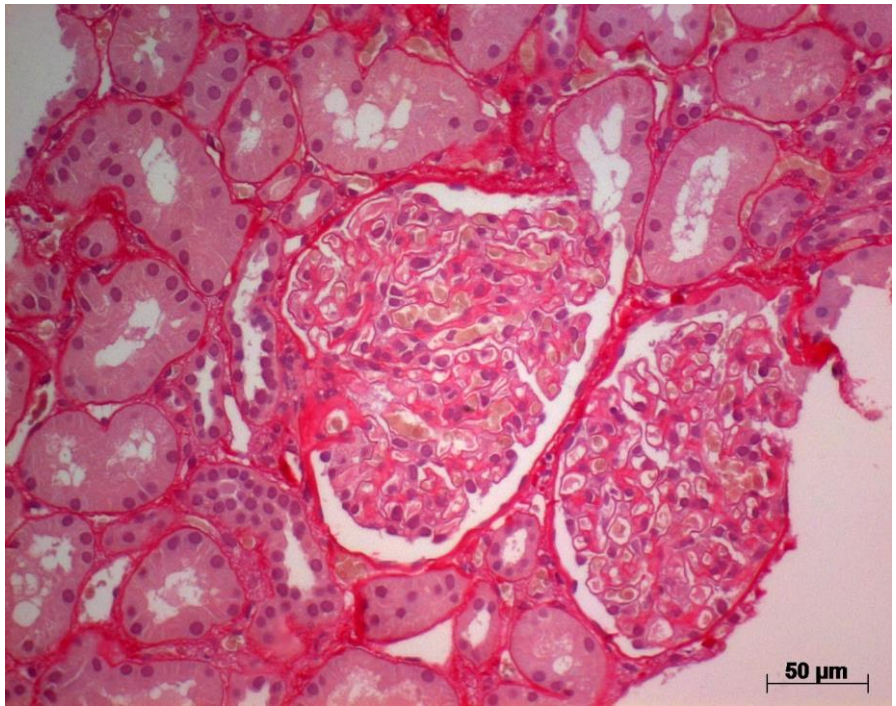
Síndrome Nefrótica

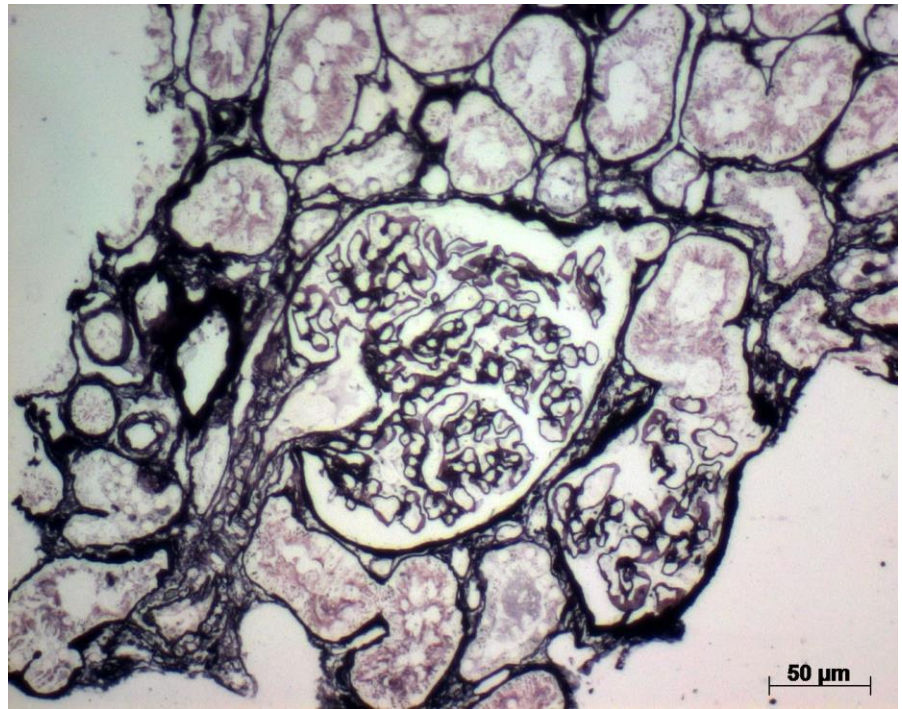
- Proteinúria
  - ≥ 3,5g/dia
  - 50mg/kg/dia (criança)
- hipoproteinemia - albumina (< 3g/dl)
- edema

Síndrome Nefrótica



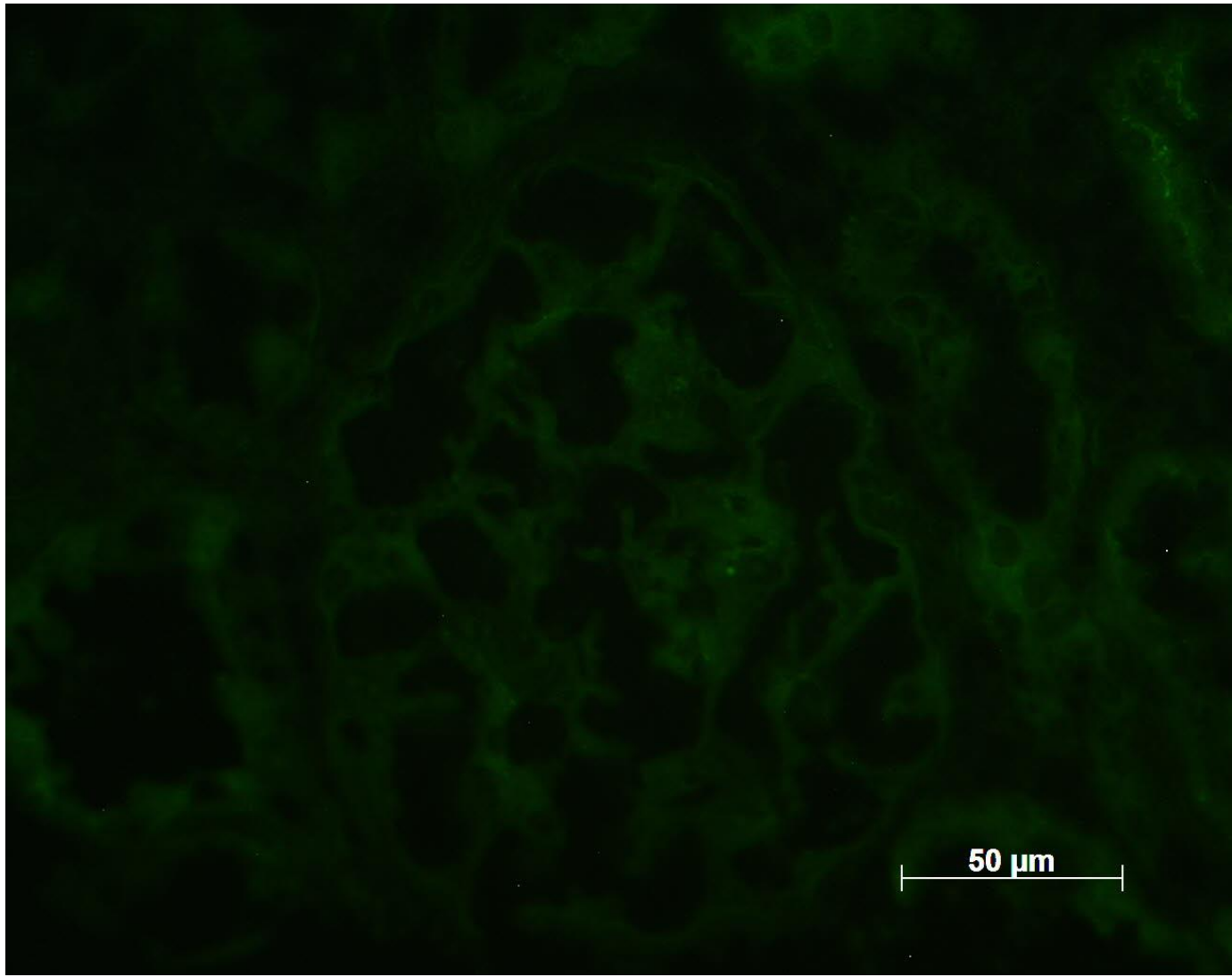


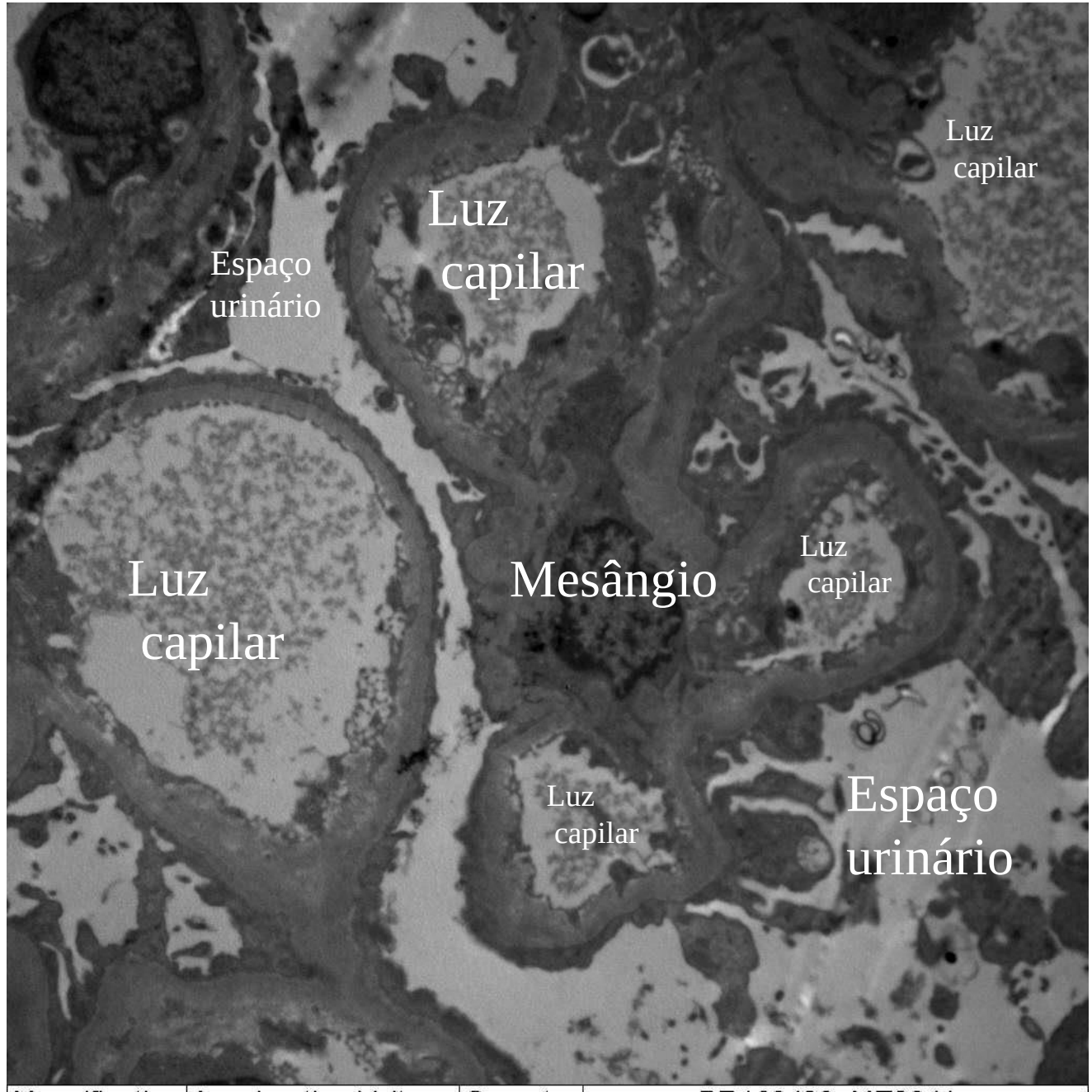


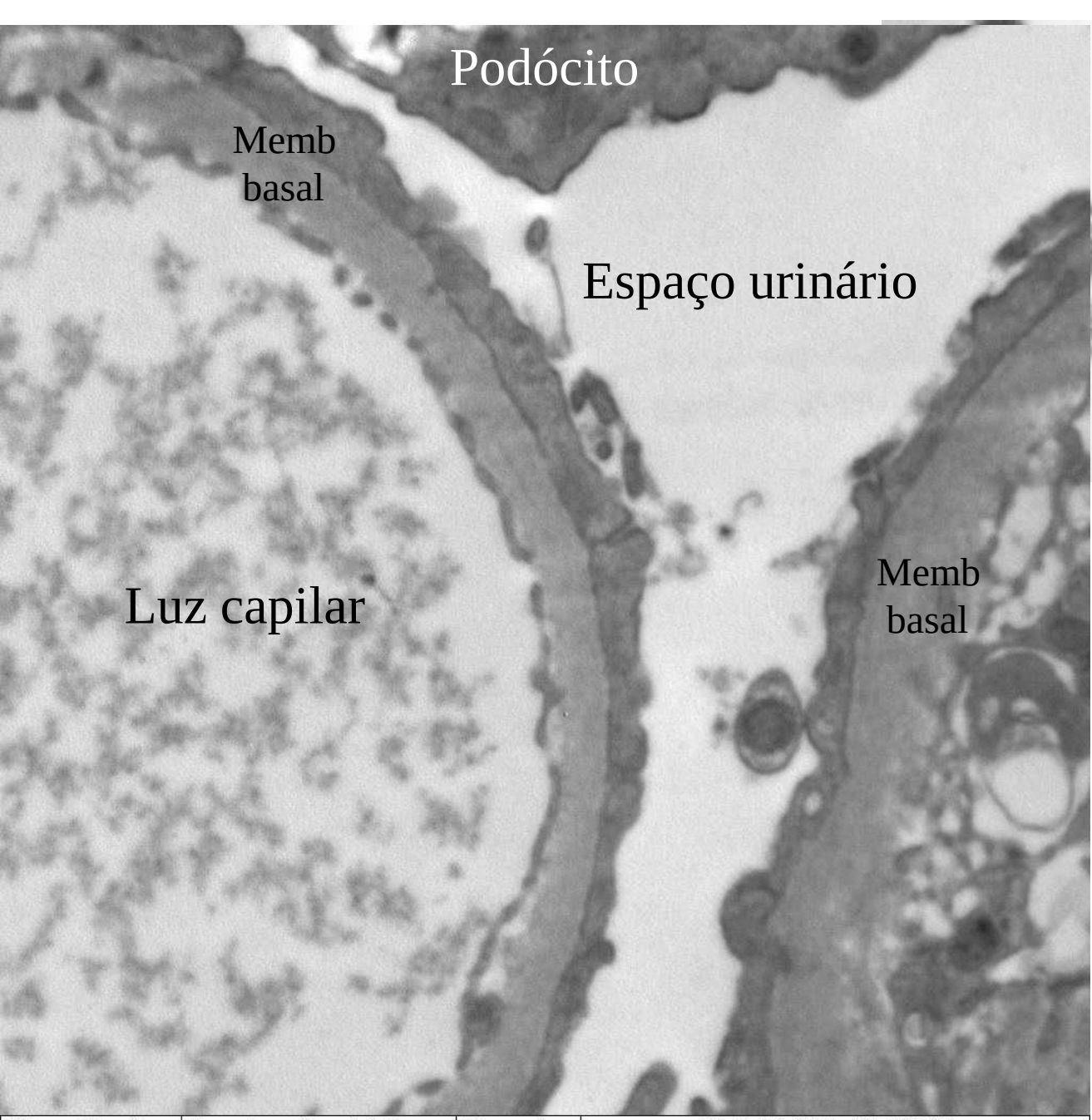


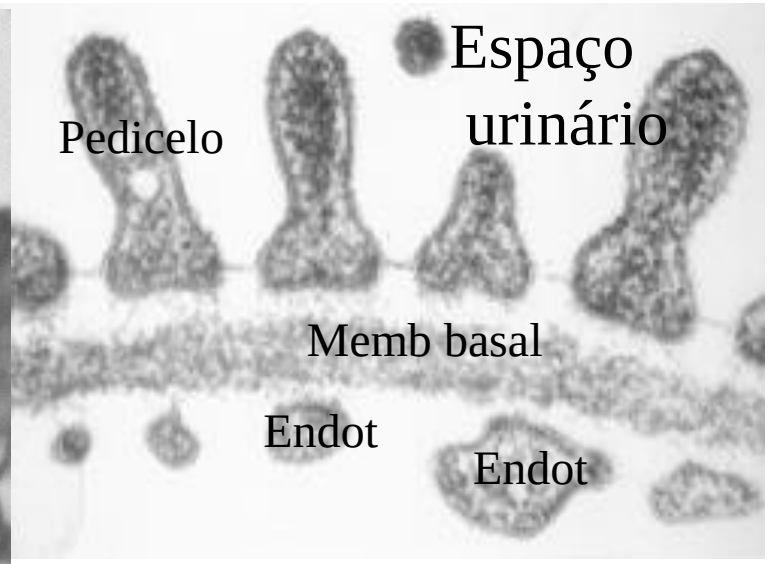
Todos negativos

- IgA
- IgG
- IgM
- *Kappa*
- *Lambda*
- Fibrinogênio
- C1q
- C3




Luz capilar
Luz capilar
Espaço urinário
Luz capilar
Mesângio
Luz capilar
Luz capilar
Espaço urinário
Magnification 3000 x
Accelerating Voltage 80 kV
Operator Mar-ML
BR160428_ME3041a
5 µm





Normal

Glomerulopatia de Lesões Mínimas

Clínica:
Sindrome Nefrótica

Identificação

masculino,

cor branca,

37 anos

Edema generalizado há 9 meses

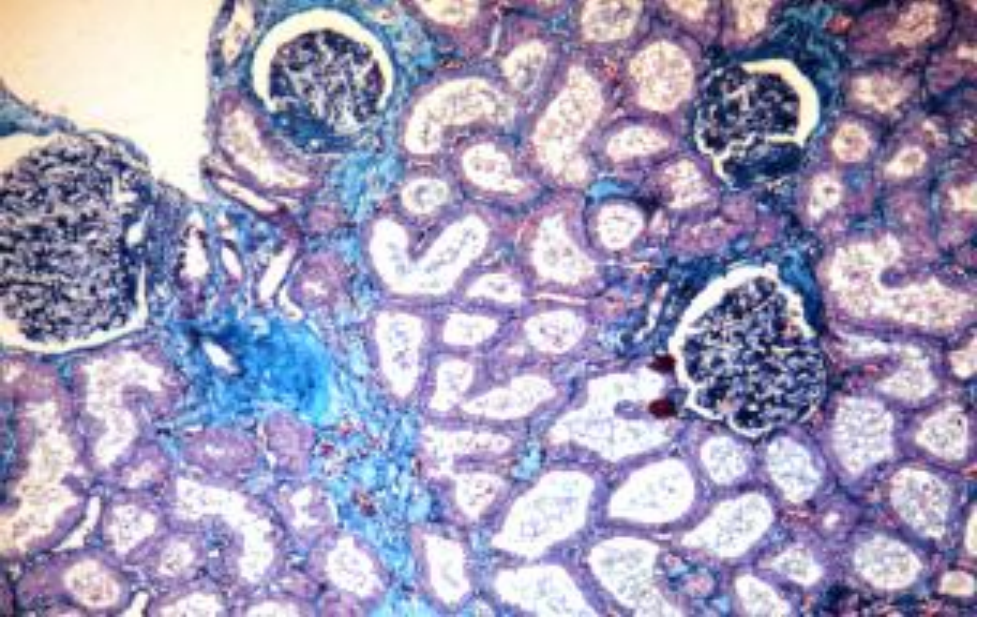
Ex complementares:

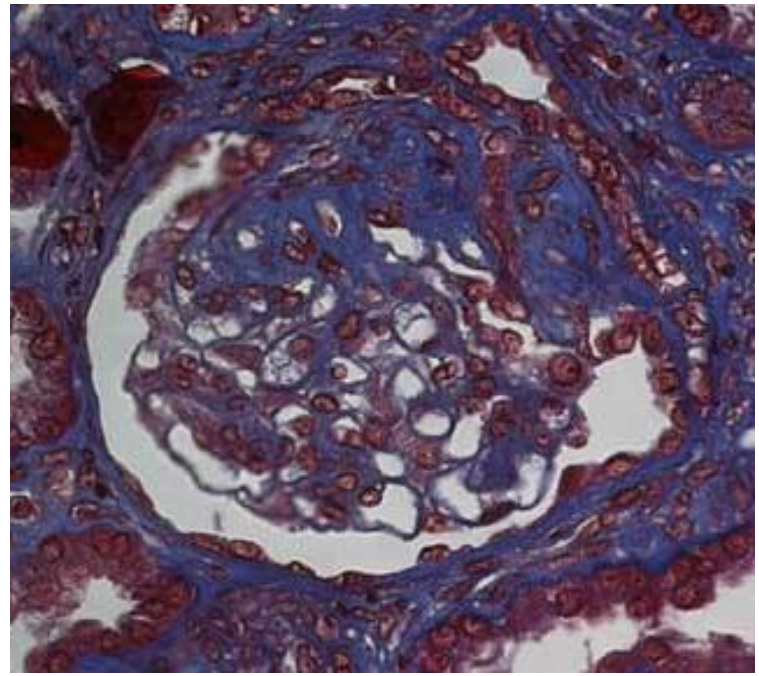
Uprot - 15 a 20g/24h

hipoalbuminemia

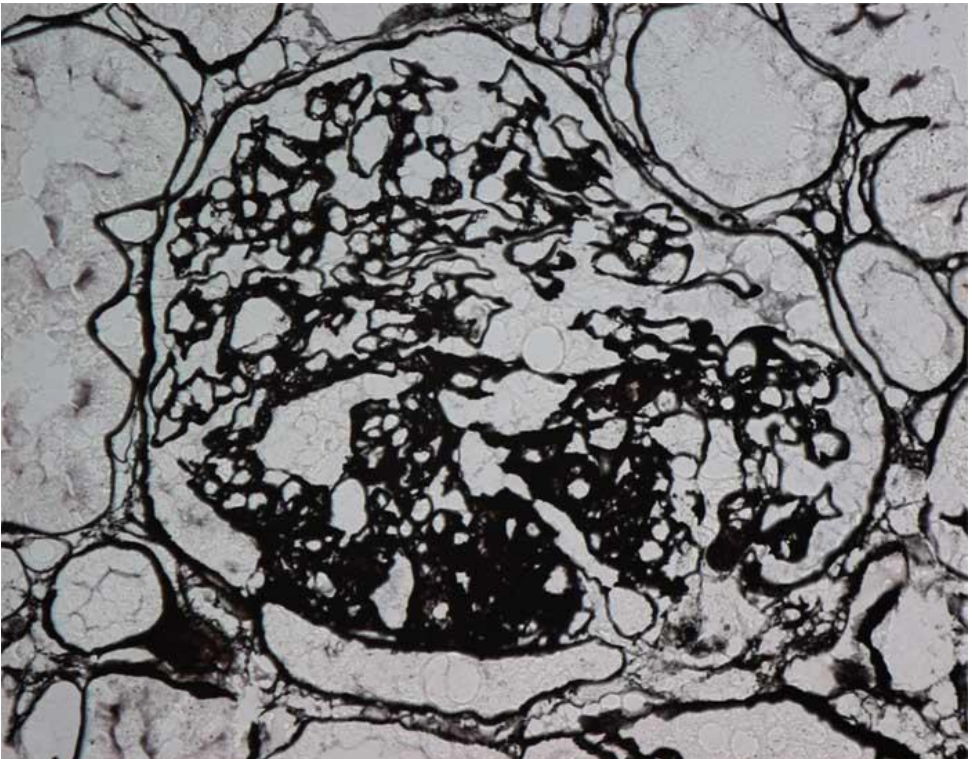
hipercolesterolemia

Síndrome Nefrótica

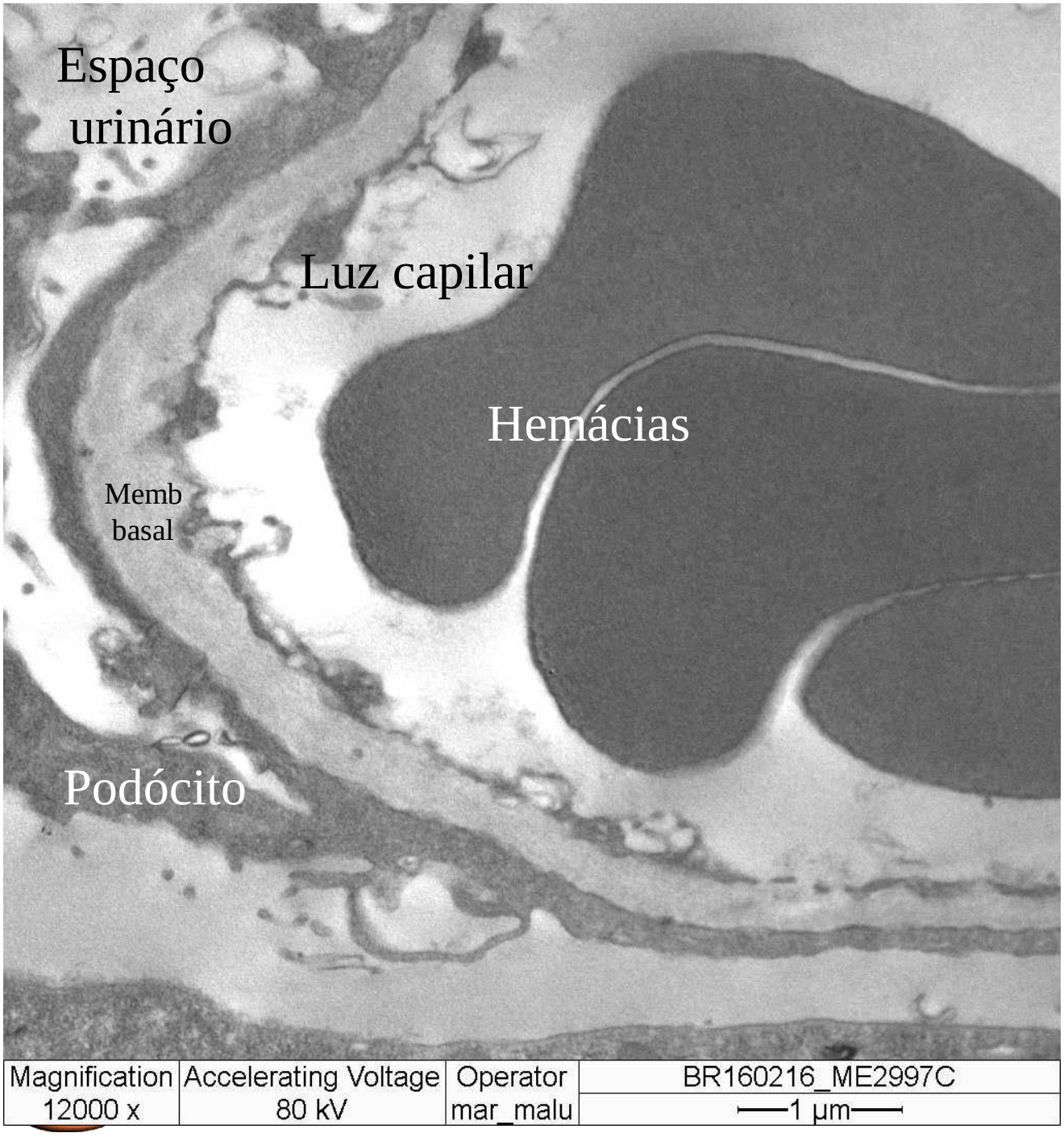


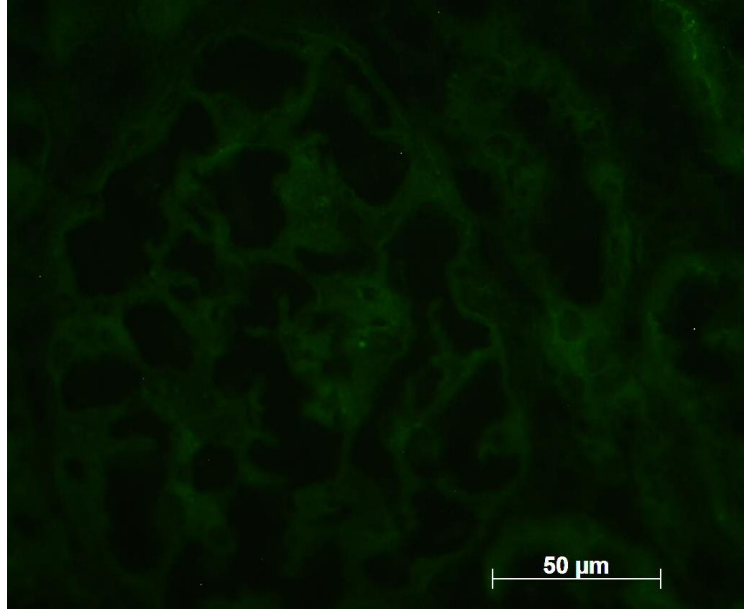


esclerose
Segmentar

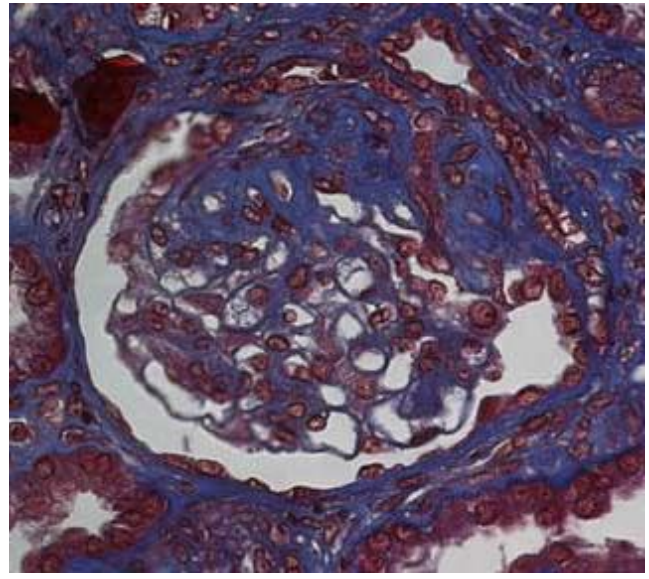


Exclui Lesões Mínimas





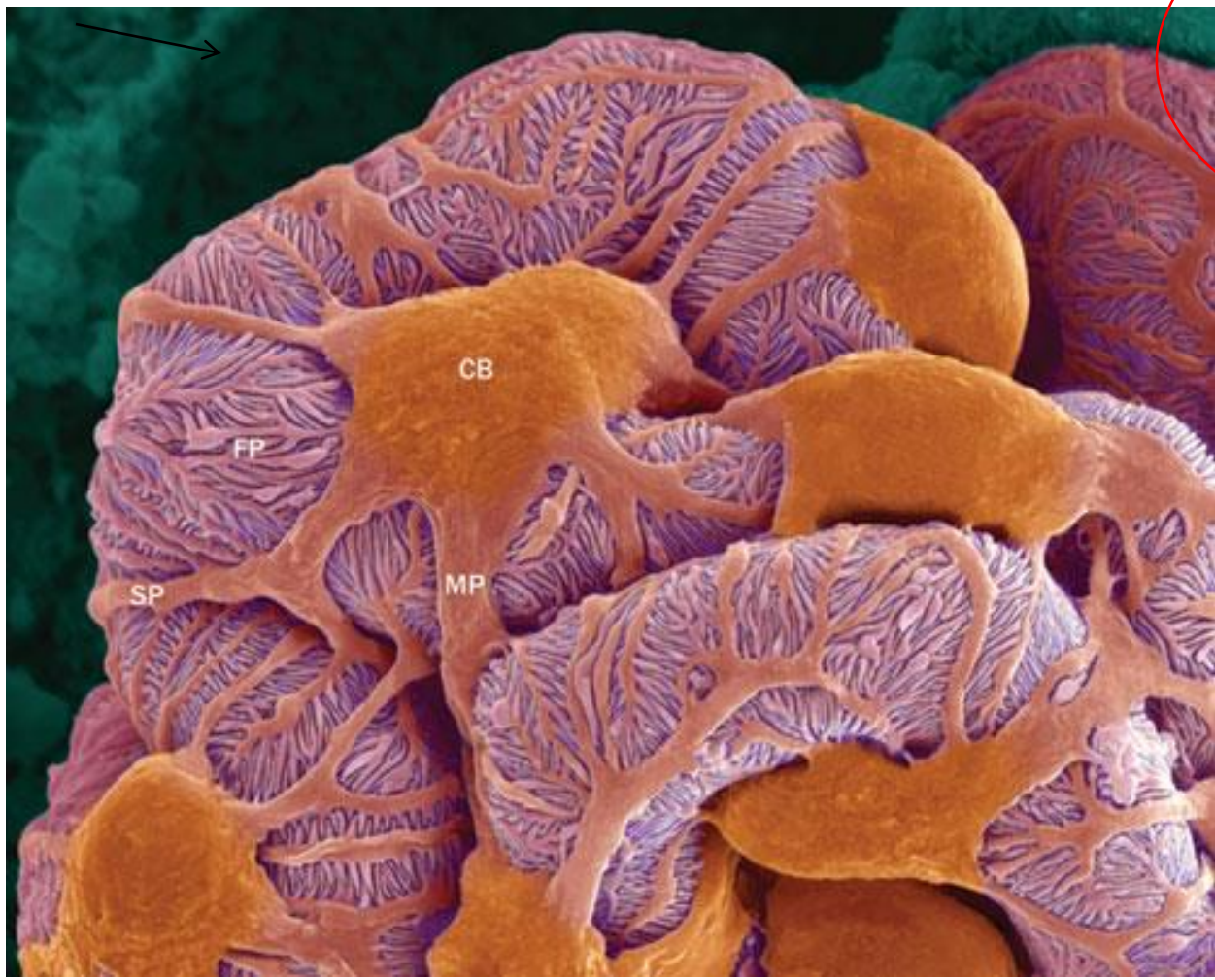
GESF
Glomeruloesclerose Segmentar e Focal



Podocitopatias

Genéticos

Nefrina,
Podocina,
A-actinina 4
WT1,
miR193a,
etc.

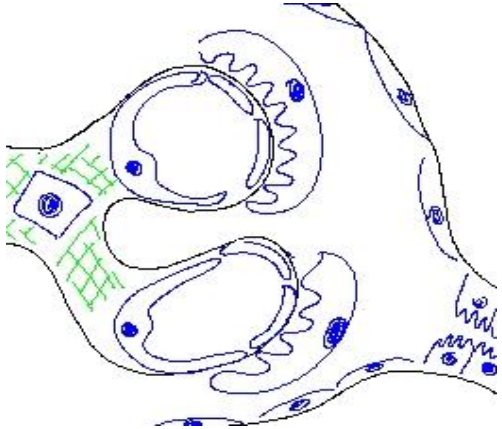


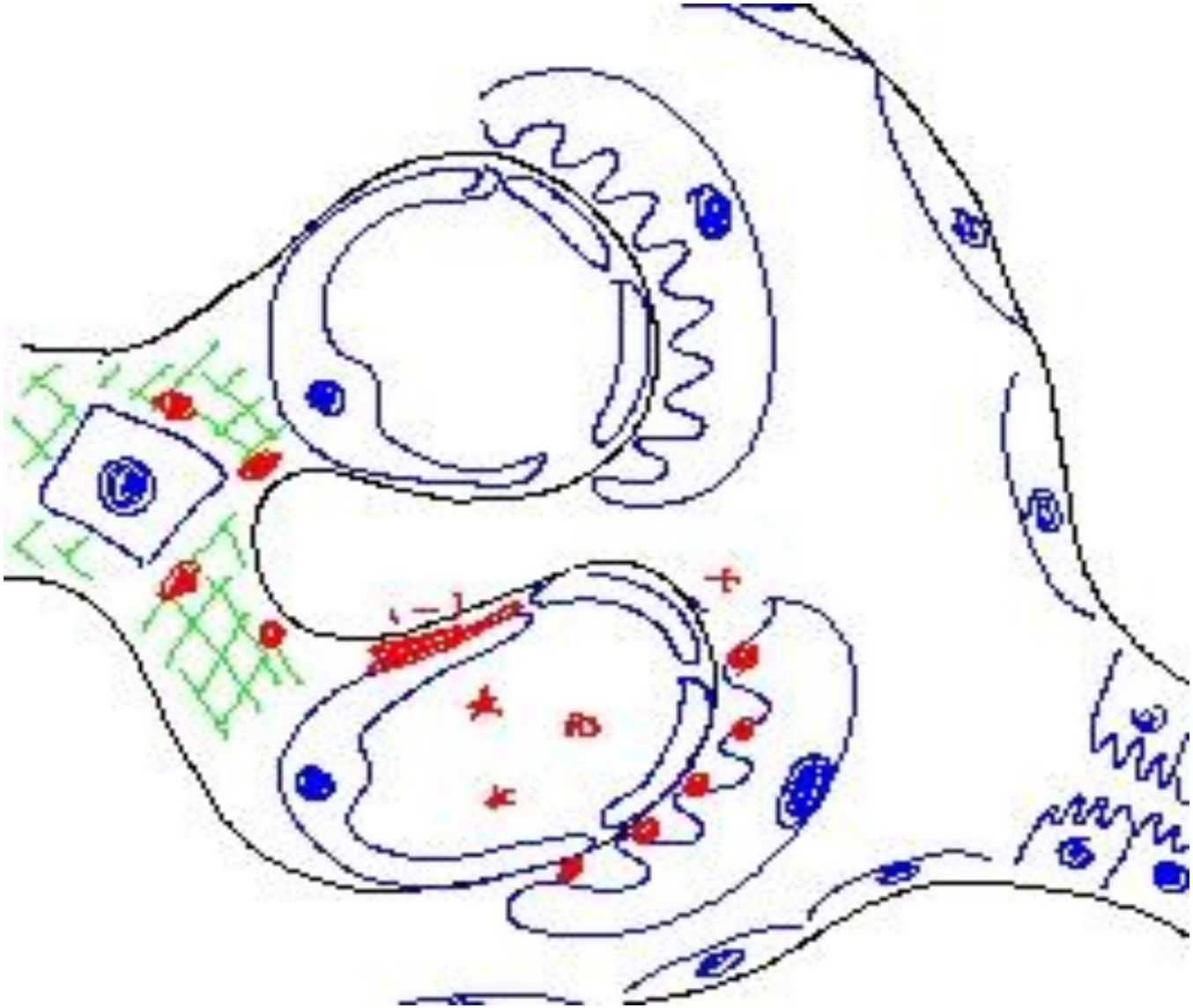
Reativas: mediadores no microambiente:

- Vírus
- Tóxico
- Mecânicas
- Metabólico
- Imune – med
- outros

May et al., 2014; Saleeem, 2015

Entidades com Imunocomplexos / Complementos





Imunocomplexos:

- Circulantes
- Formação "*in situ*"

36 anos de idade, feminino, cor branca

Há mais ou menos um ano:

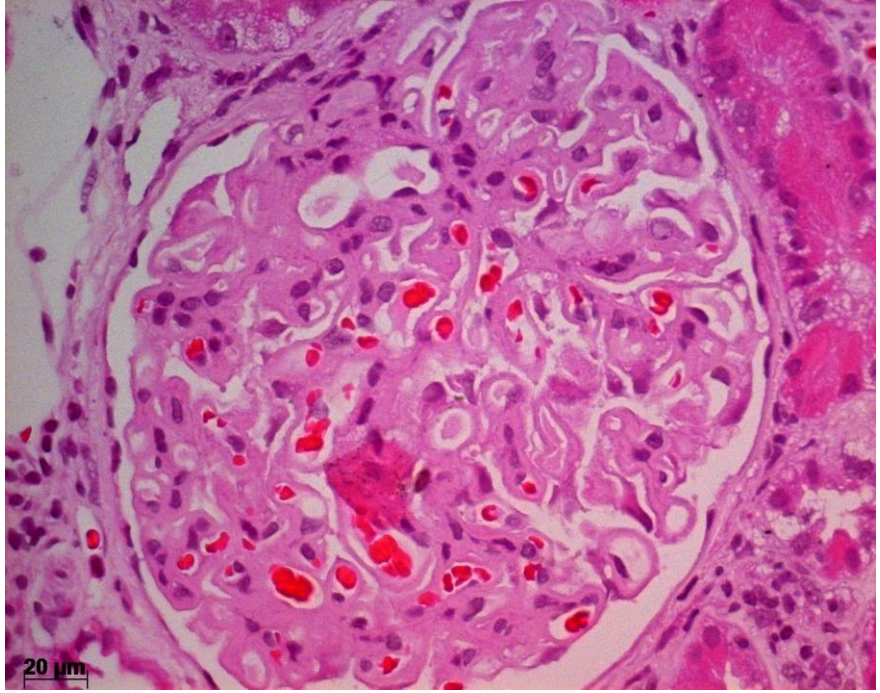
- edema vespertino peri-orbitário

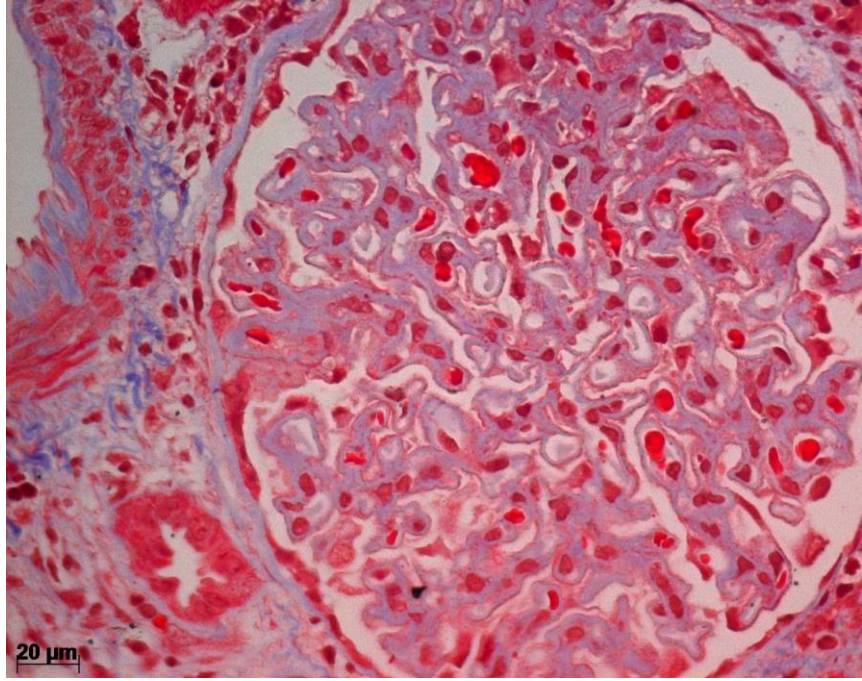
Exame físico: edema de mmii ++/4

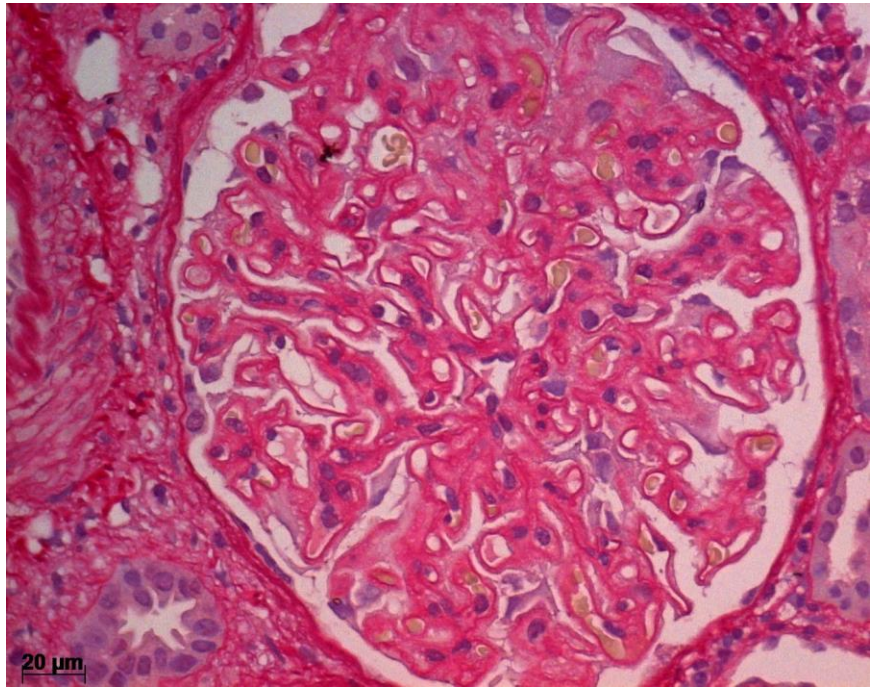
Exames laboratoriais:

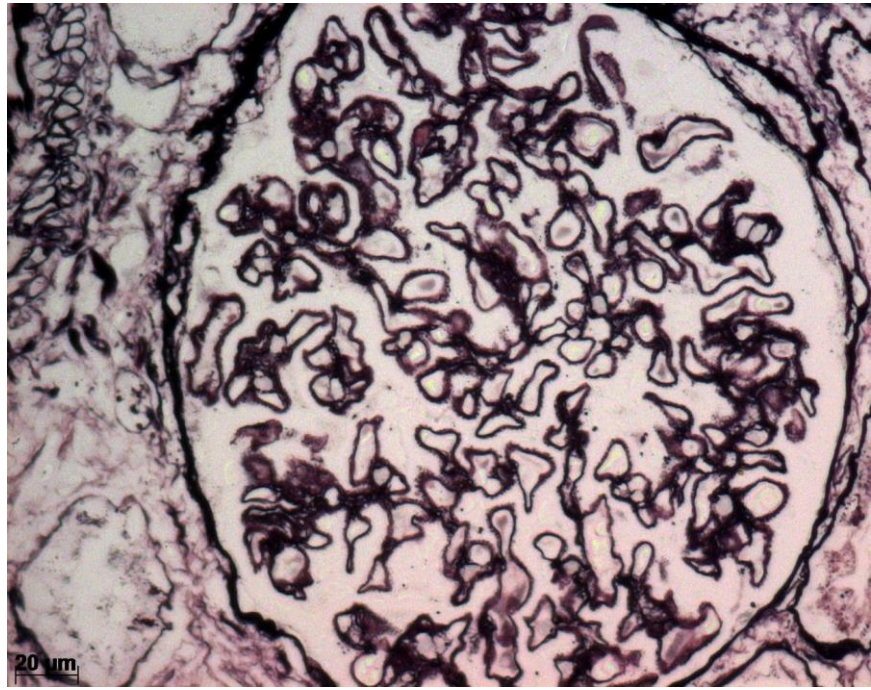
- uréia = 30
- creatinina = 0,8 mg/dl
- **Proteinúria de 24h = 8.424mg**
- Prot totais = 3,9
- **Alb = 1,6**; Glob=2,3; A/G = 0,69
- Colesterol - 432mg/dl

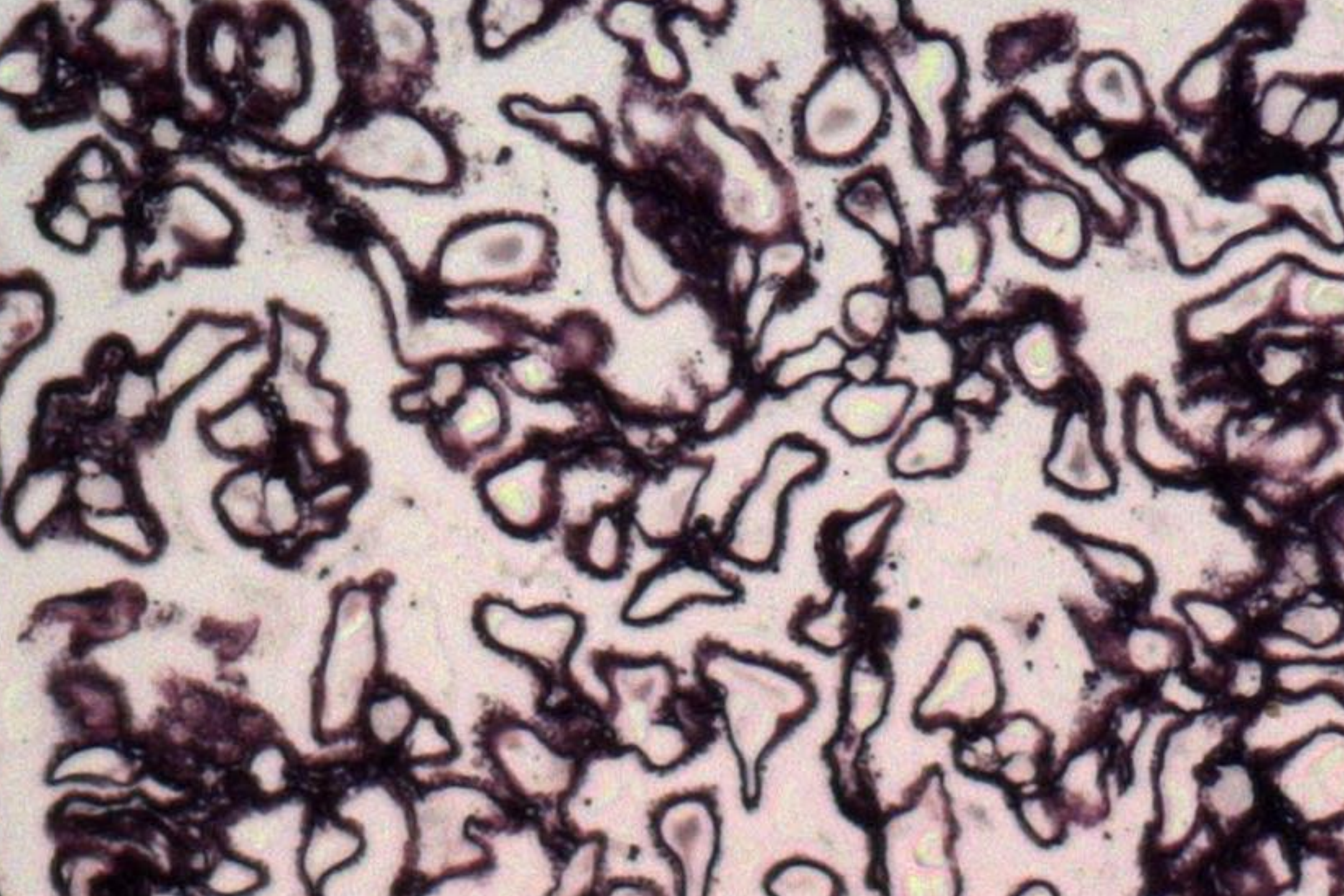
Síndrome Nefrótica

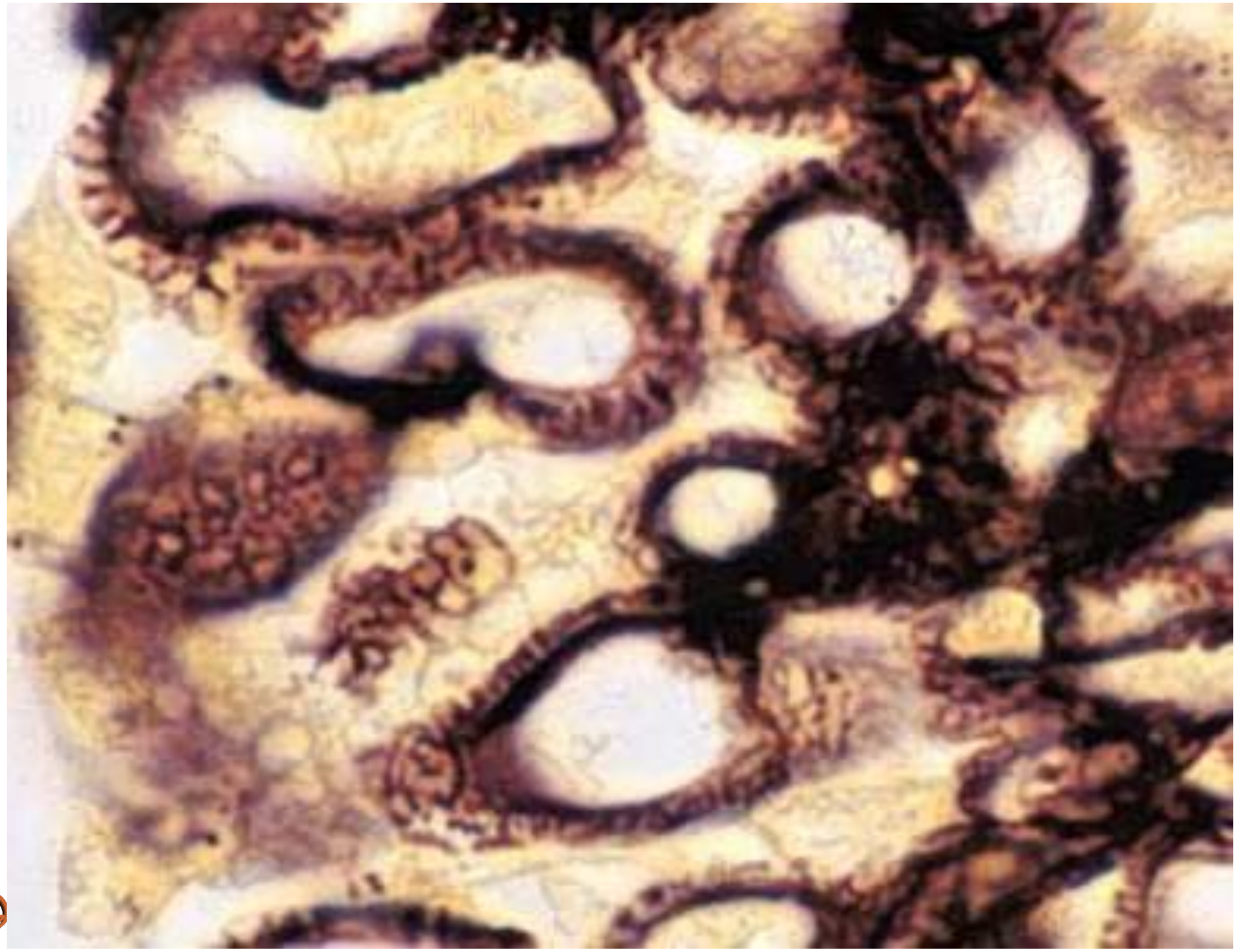


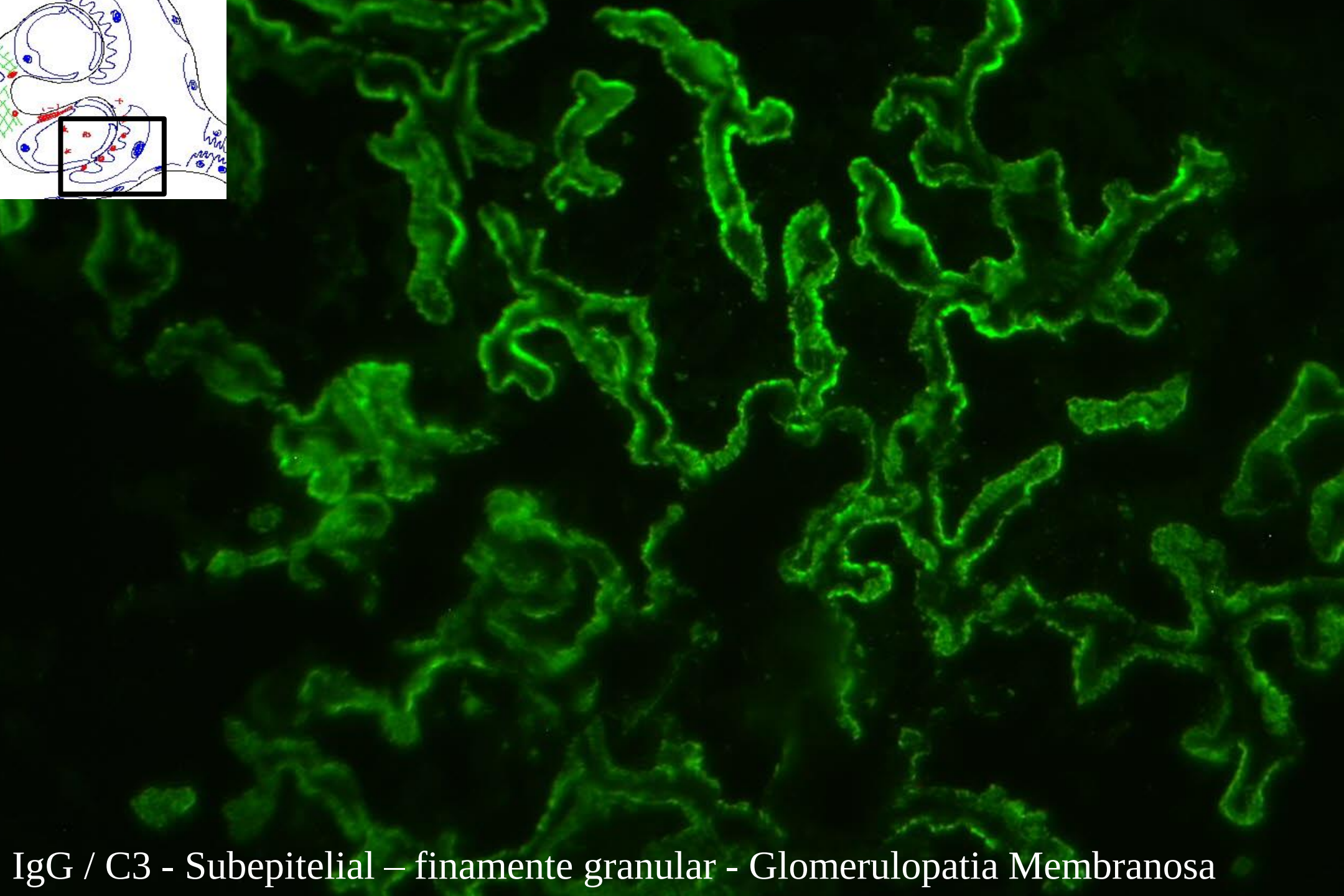




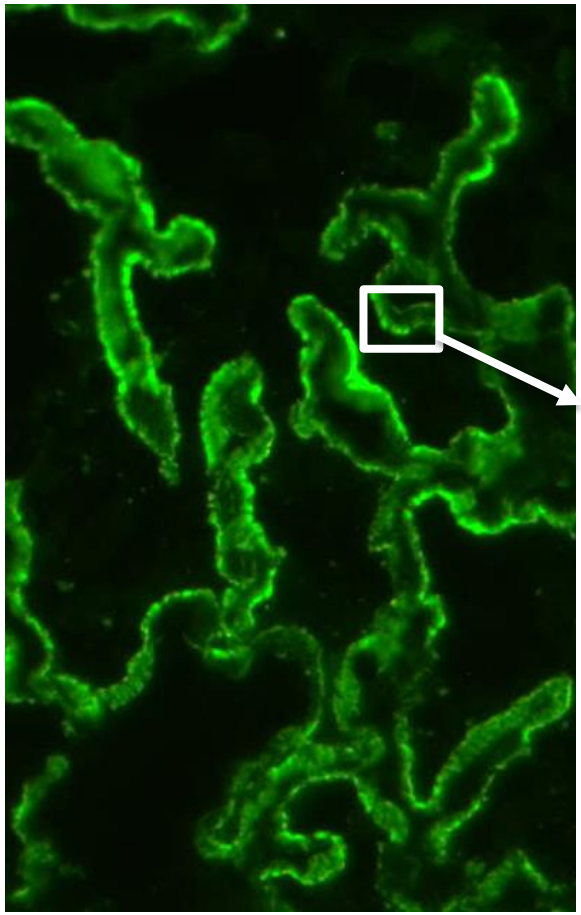


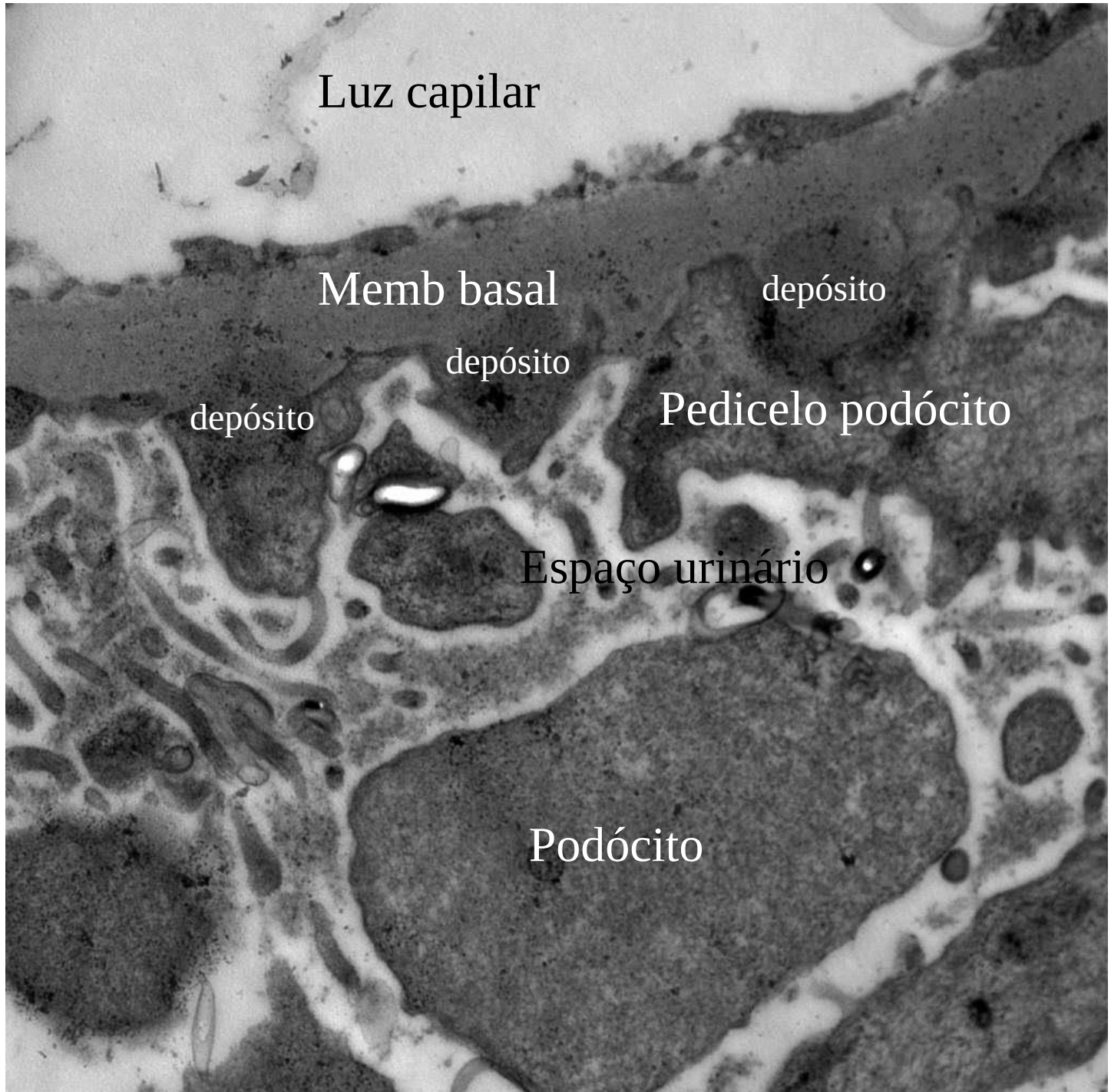


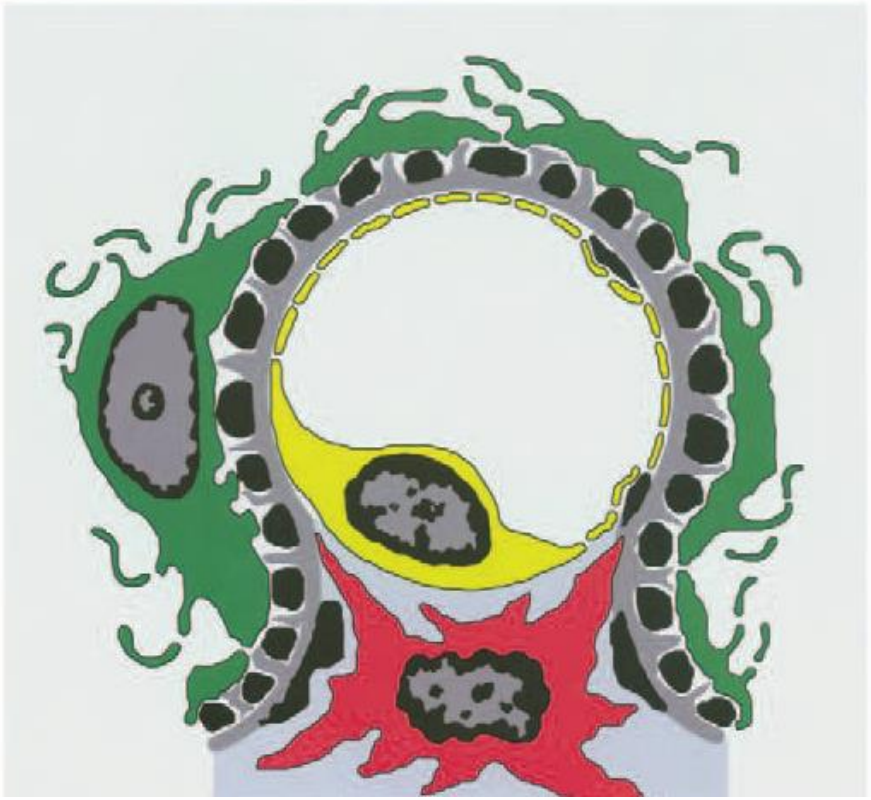


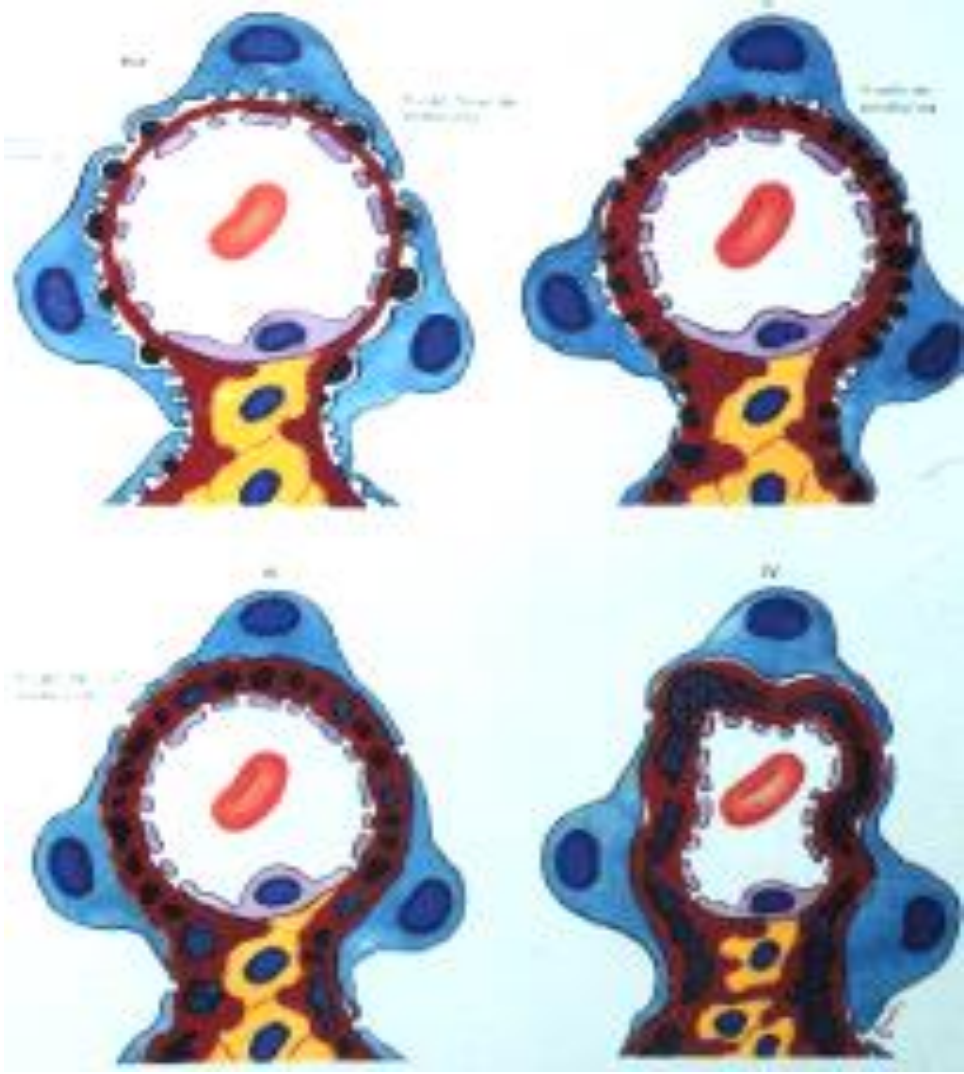


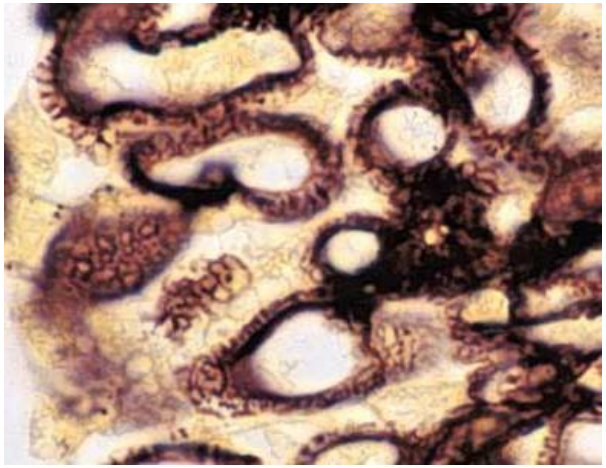
IgG / C3 - Subepitelial – finamente granular - Glomerulopatia Membranosa




Luz capilar
Memb basal
depósito
depósito
depósito
Pedicelo podócito
Espaço urinário
Podócito







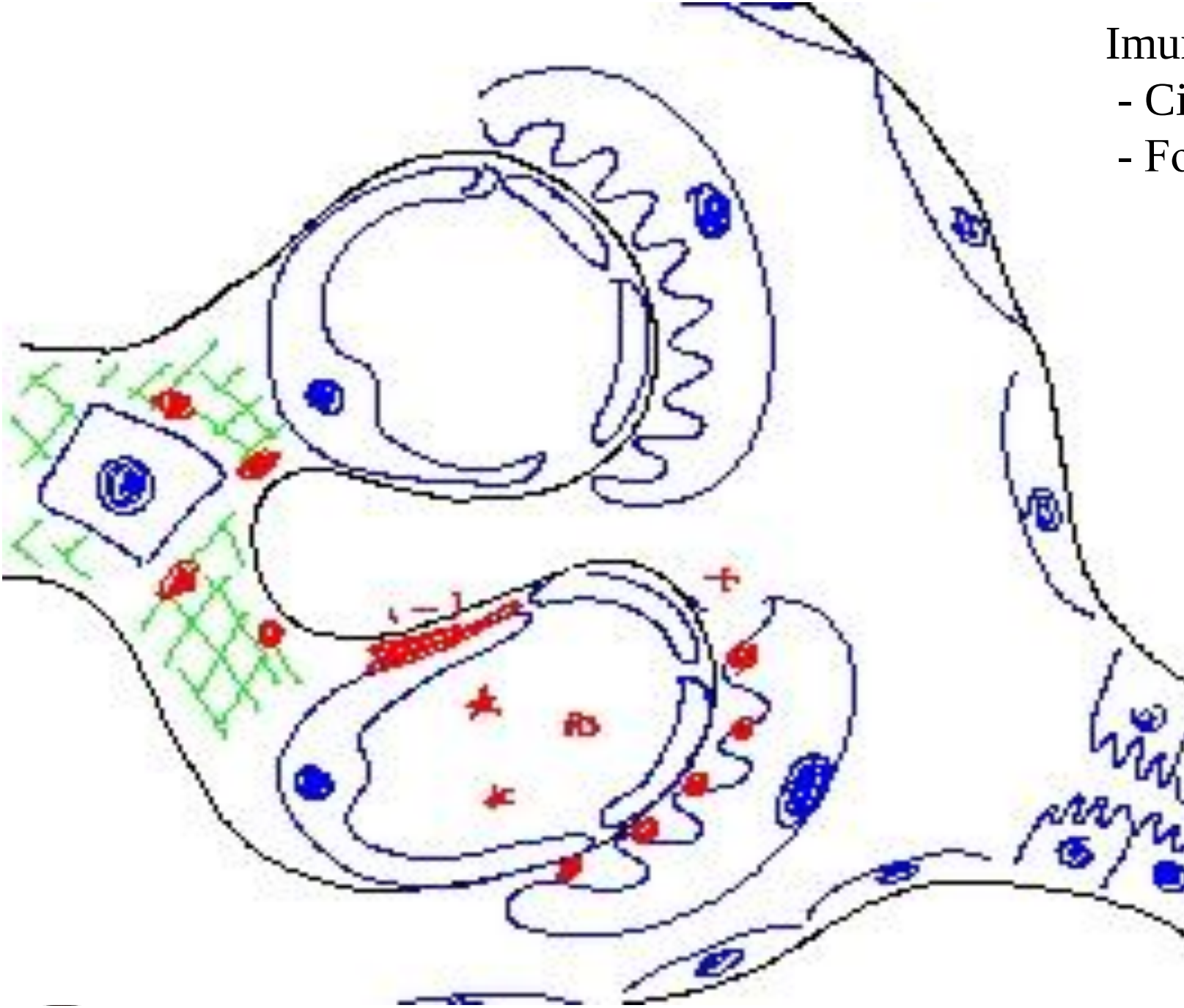
Glomerulopatia Membranosa Estadio II

Glomerulopatia Membranosa

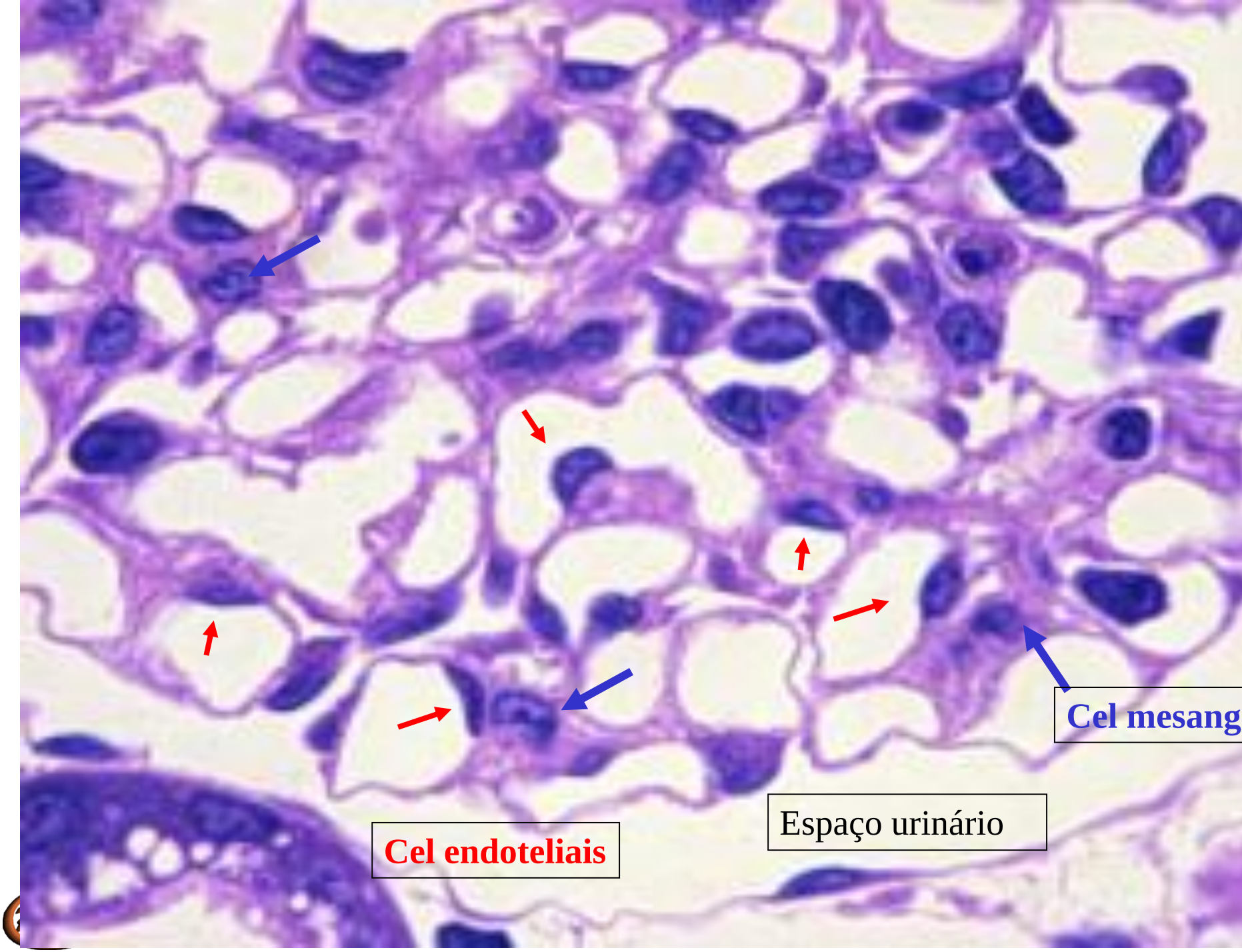
Primária – Ac anti-receptor Fosfolipase A2

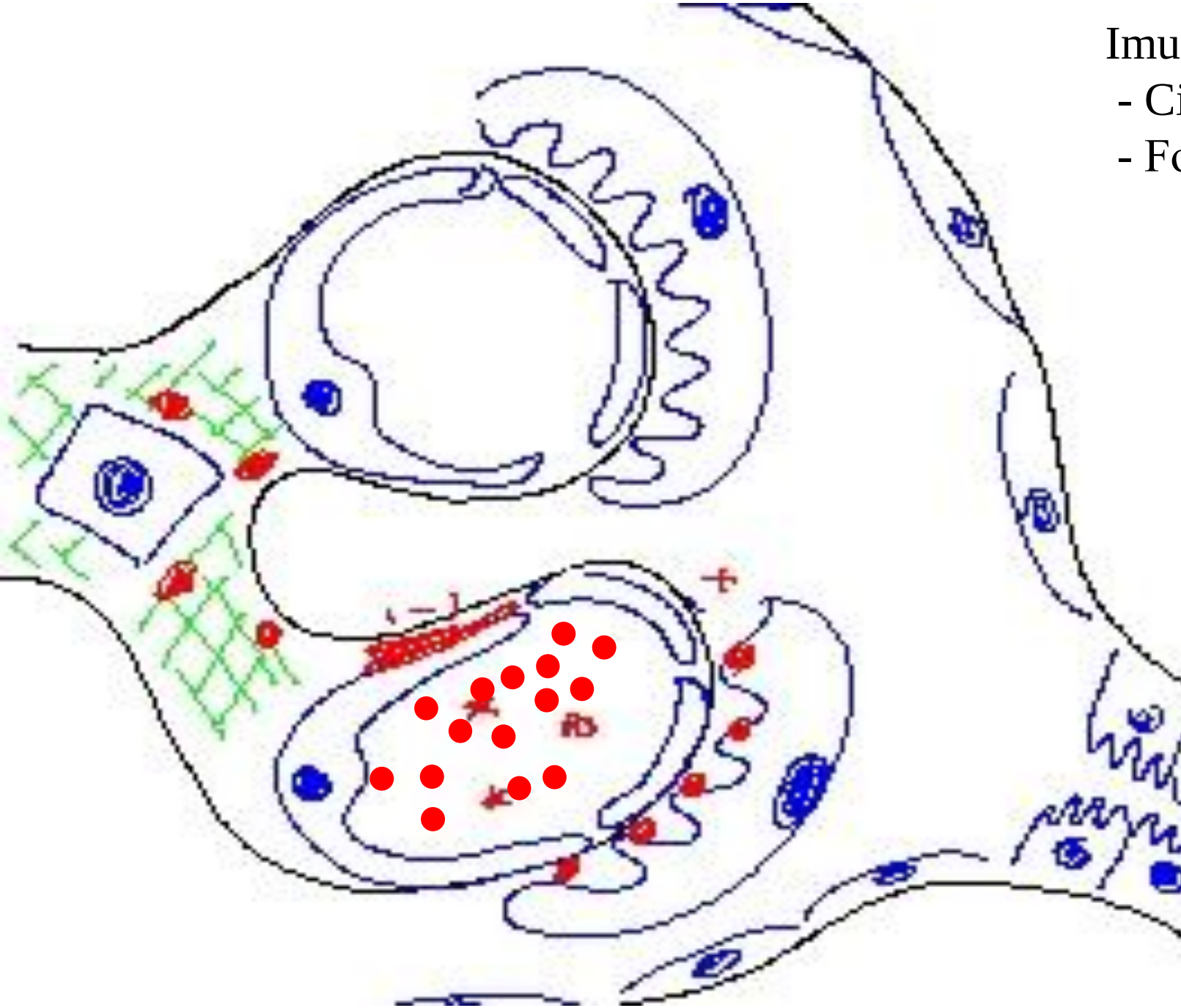
Secundária

- Lupus eritematoso sistêmico
- Sífilis
- Hepatite B
- Neoplasias
- Terapêutica:
    - Ouro
    - Penicilamina
    - Mercúrio
    - Captopril

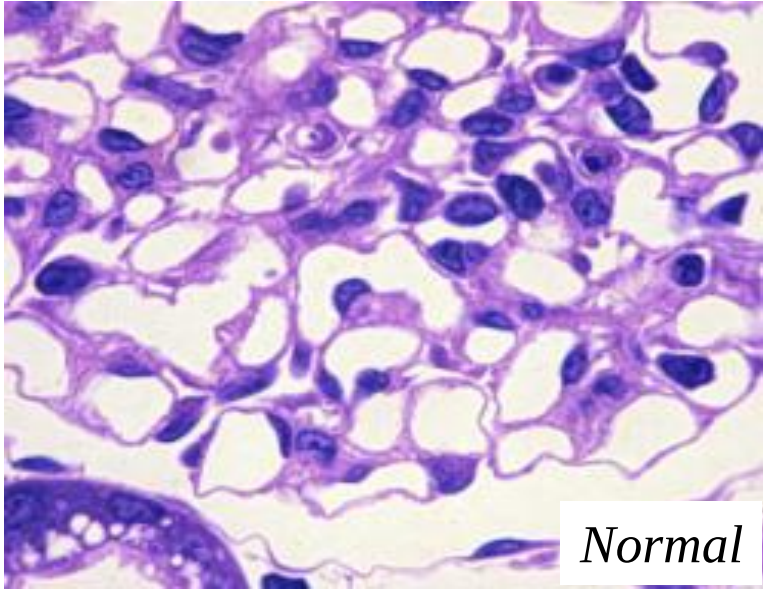


Imunocomplexos:
- Circulantes
- Formação "*in situ*"


Cel mesangiais
Espaço urinário
Cel endoteliais

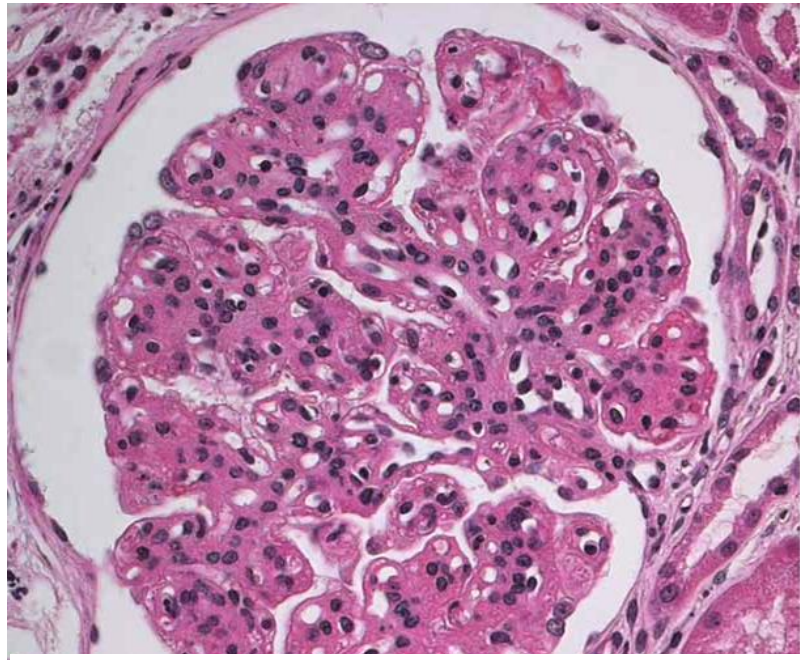


Imunocomplexos:
- Circulantes
- Formação "*in situ*"



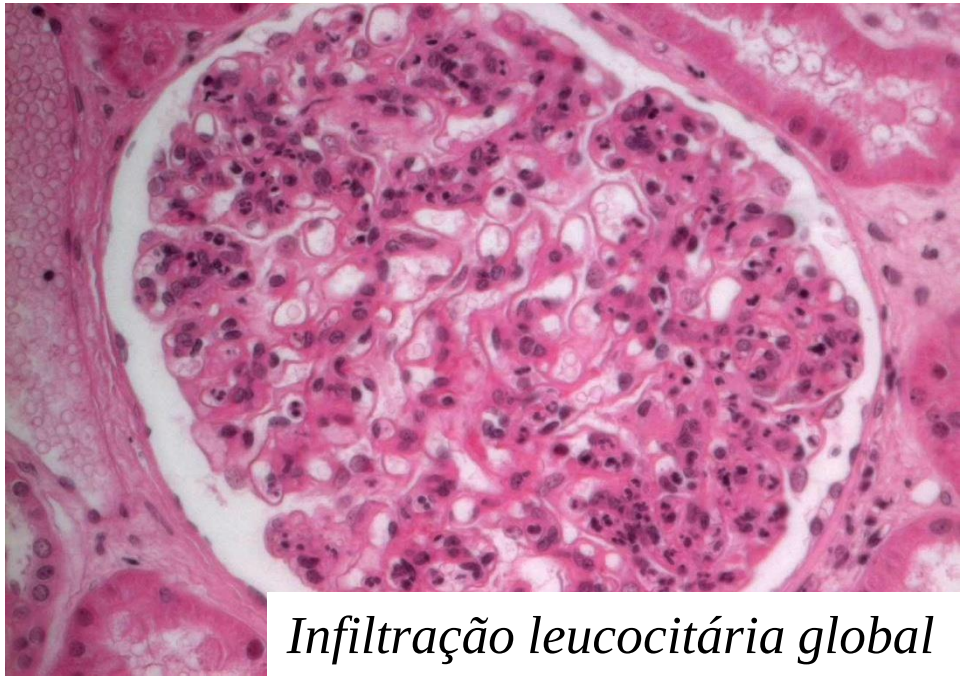
Normal

Células

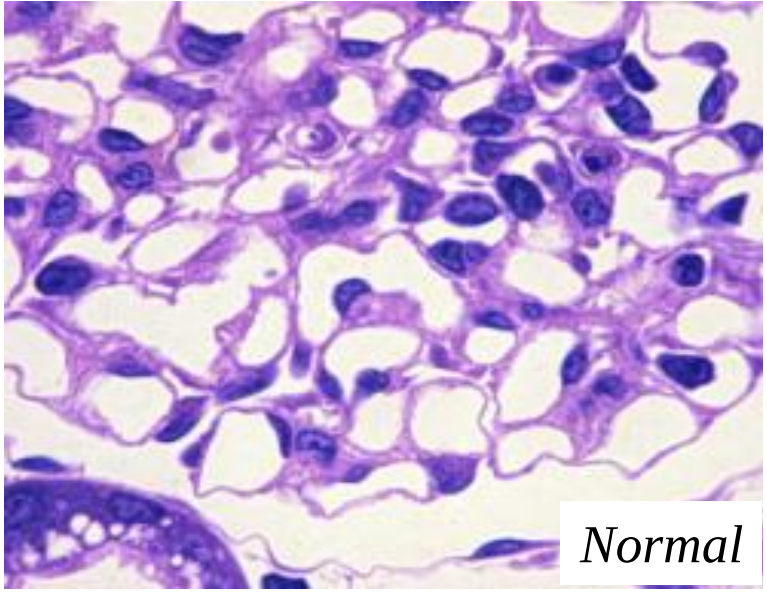


Proliferação endocapilar global

Hipercelularidade



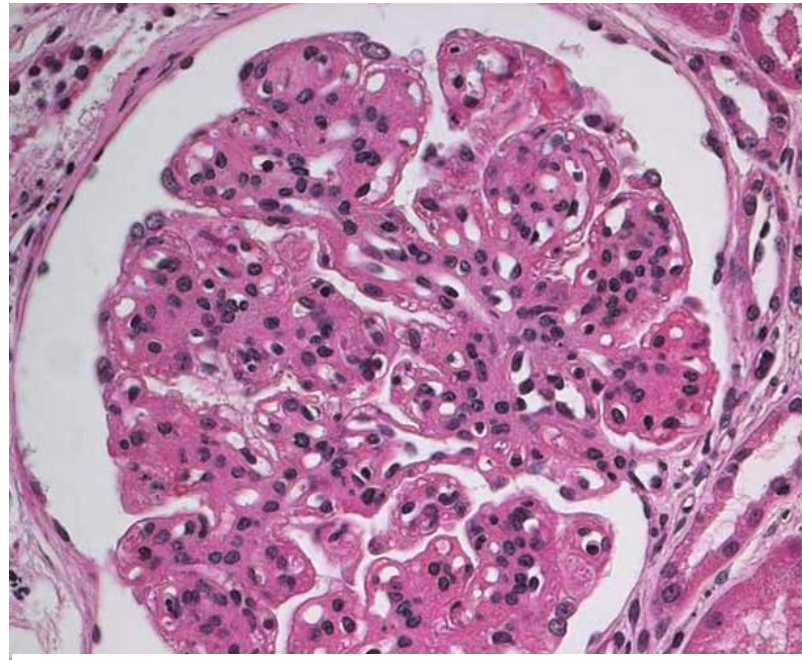
Infiltração leucocitária global



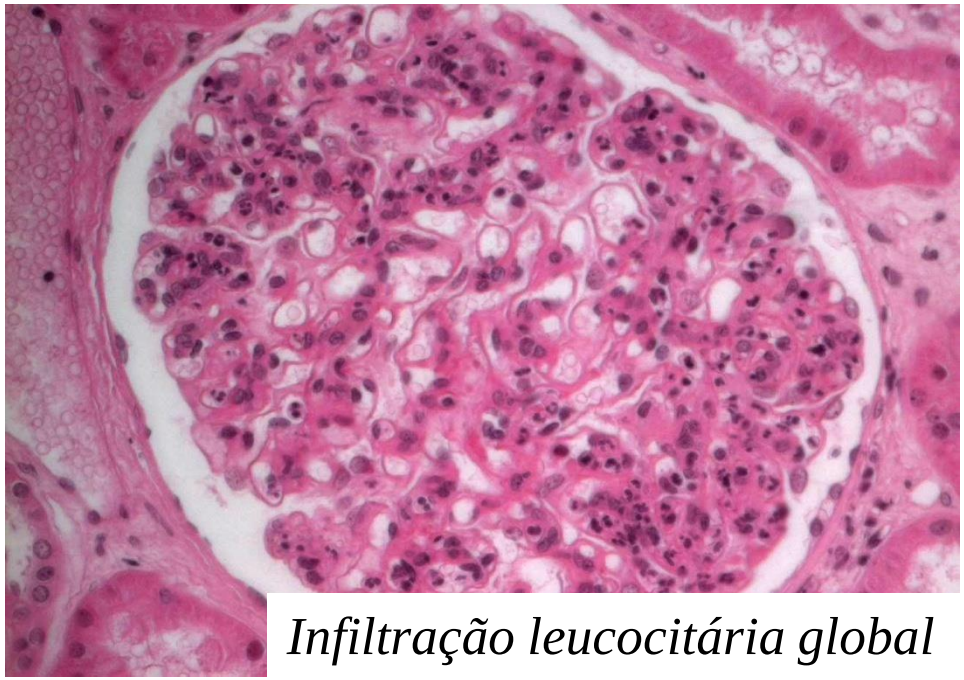
Normal

Células

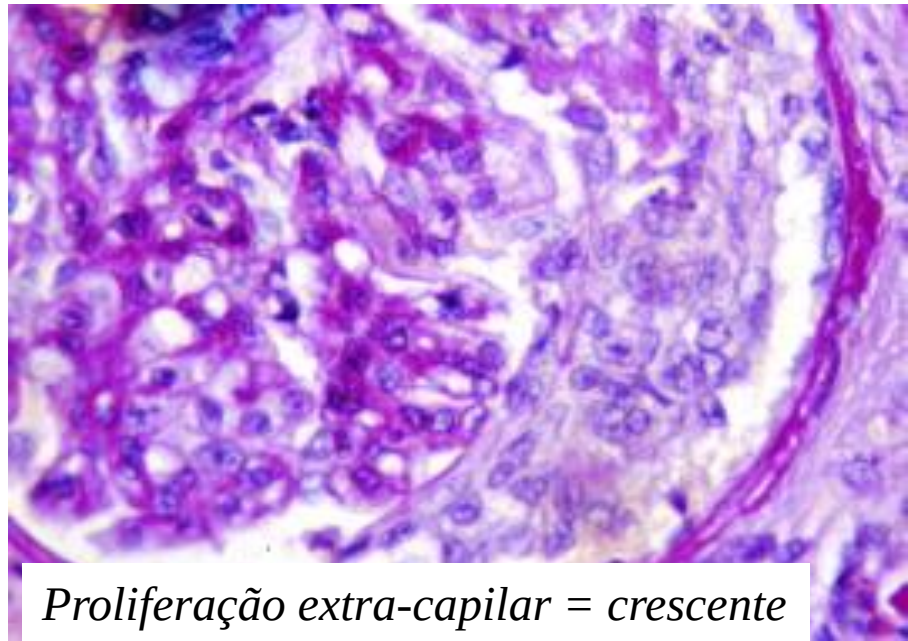
Hipercelularidade



Proliferação endocapilar global



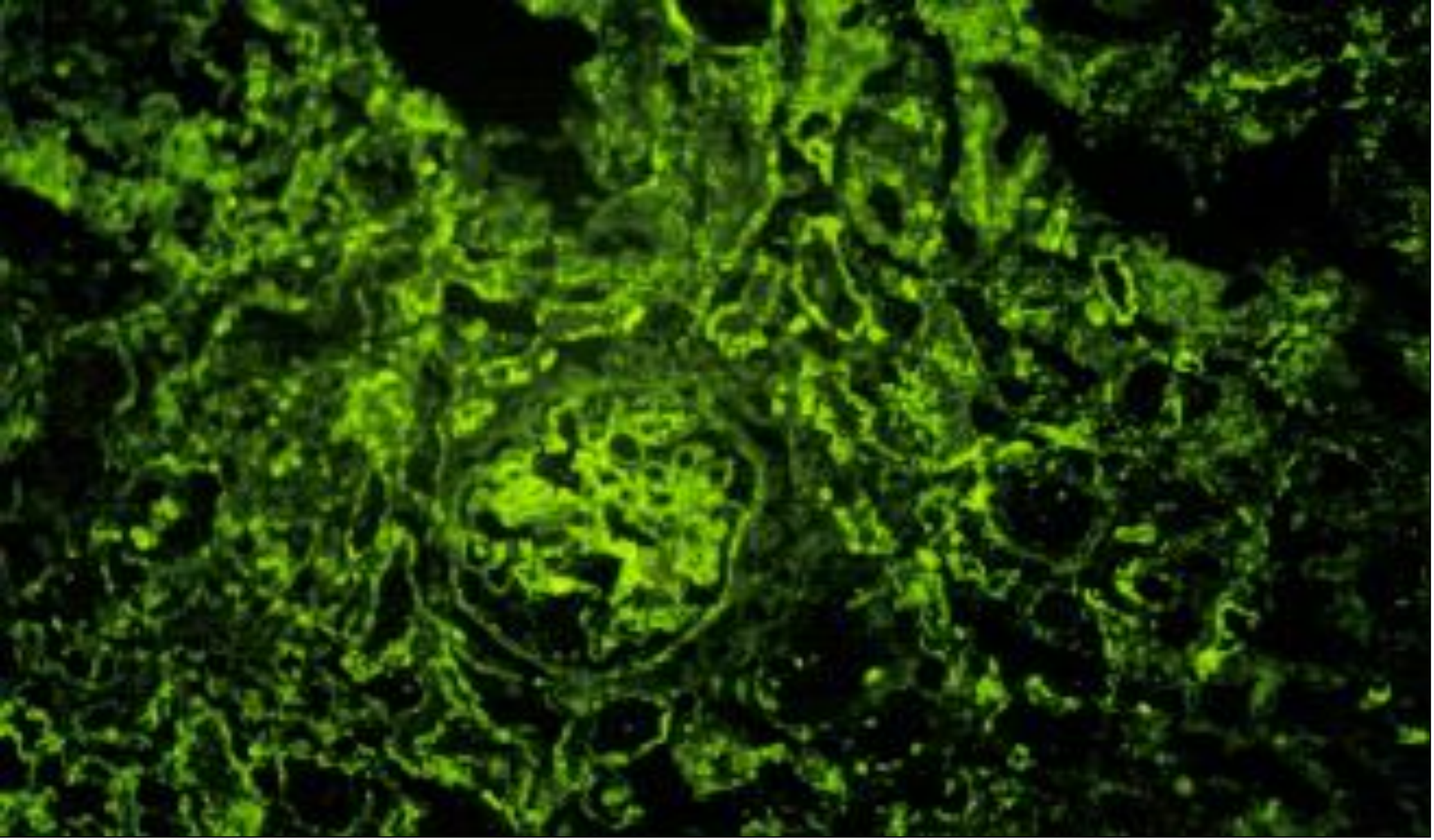
Infiltração leucocitária global



Proliferação extra-capilar = crescente

Nefrite Lúpica

1. Alt urinárias assintomáticas
   a) hematúria glomerular isolada
   b) Proteinúria isolada
2. síndrome nefrótica
3. síndrome nefrítica
4. IRA (insuficiência aguda – rapidamente progressiva)
5. Doença Renal Crônica

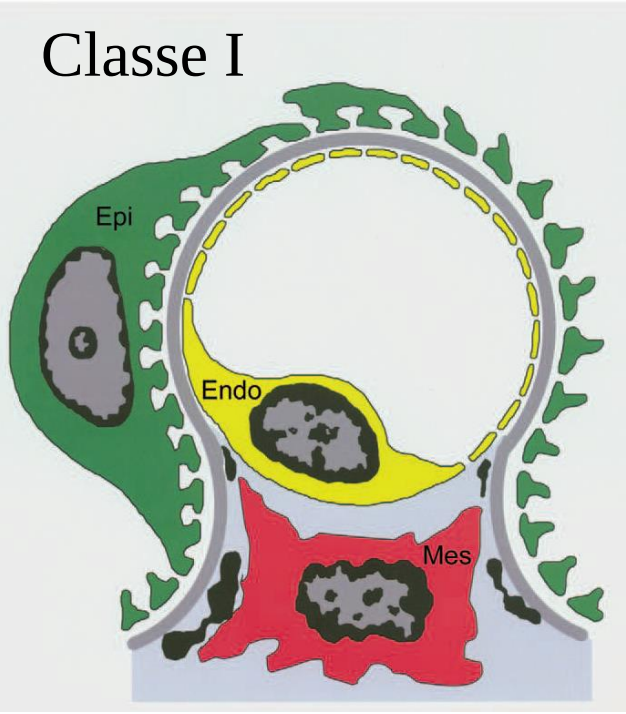


Nefrite Lúpica

IgA, IgG, IgM, C3, C1q, *kappa*, *lambda*

Nefrite Lúpica

Classe I



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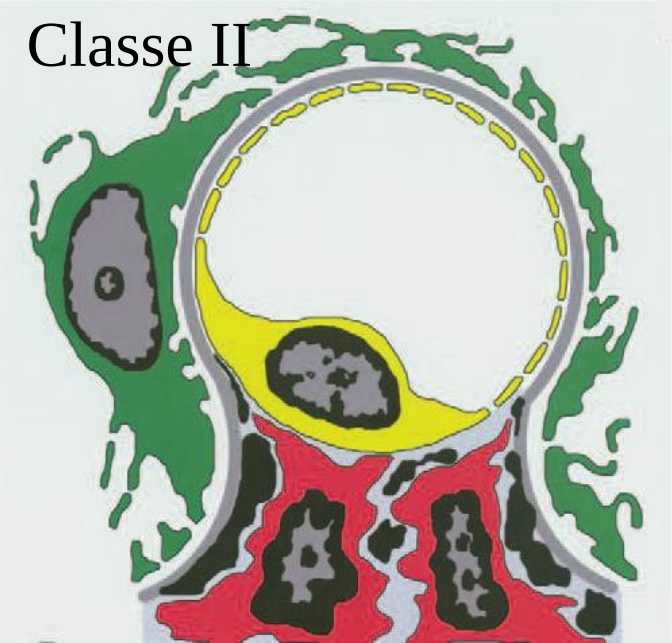
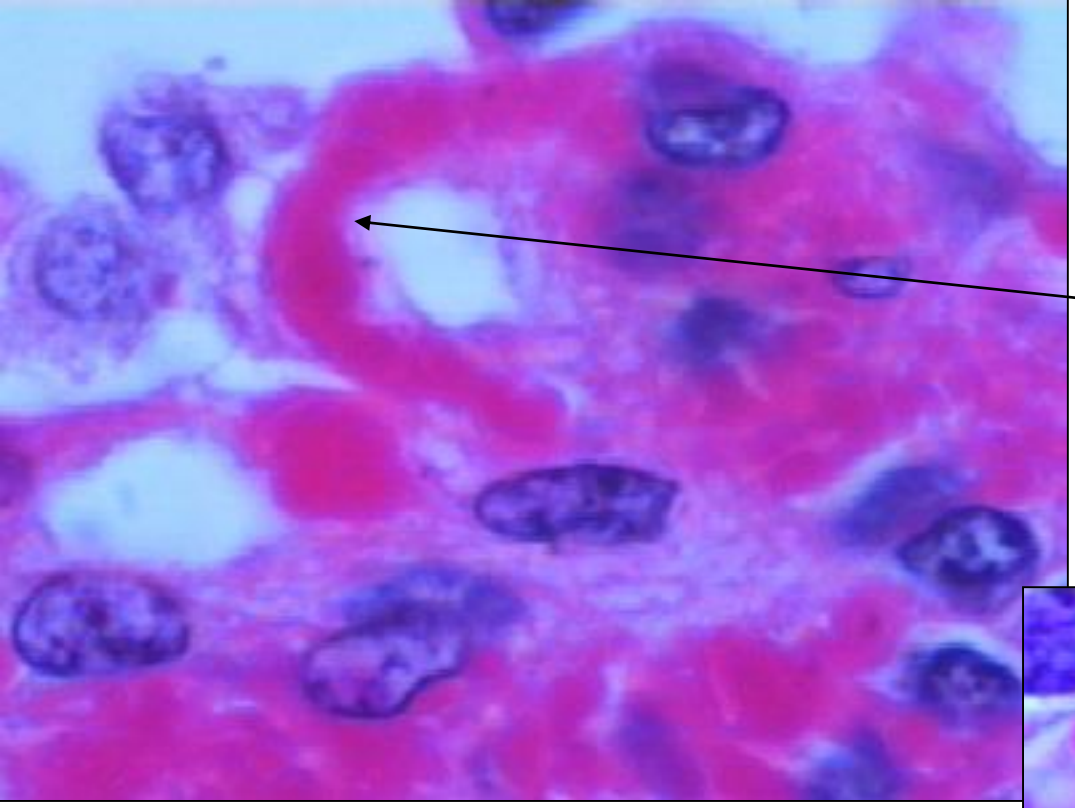
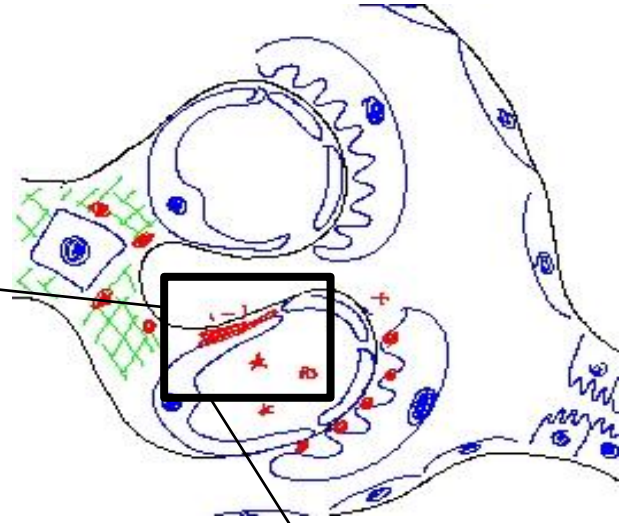


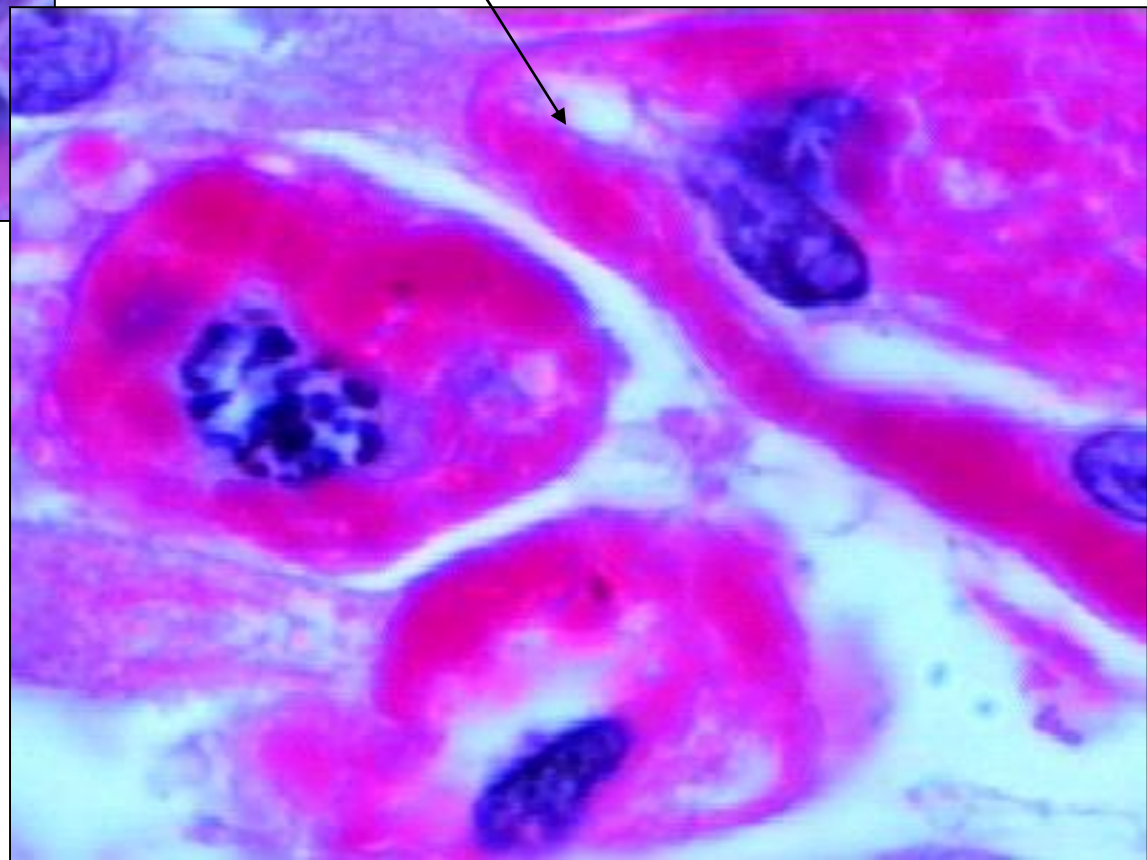
Classe III – Focal

Classe IV - Difuso

Seshan & Jennette, Arch Pathol Lab Med 133:233-248;2009



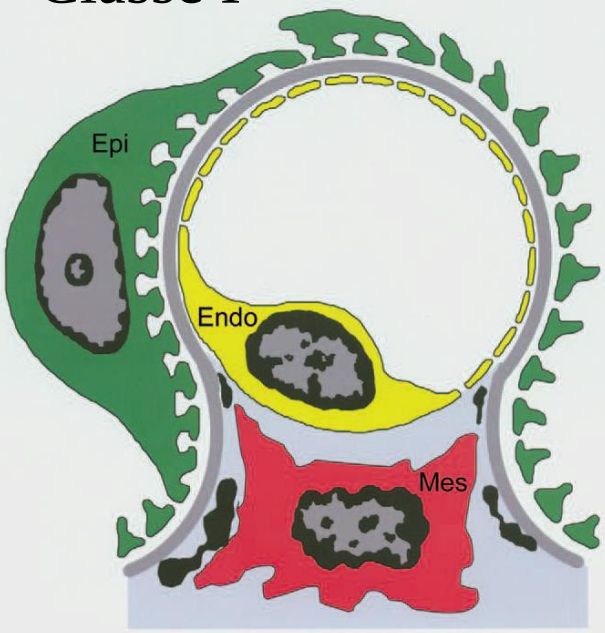




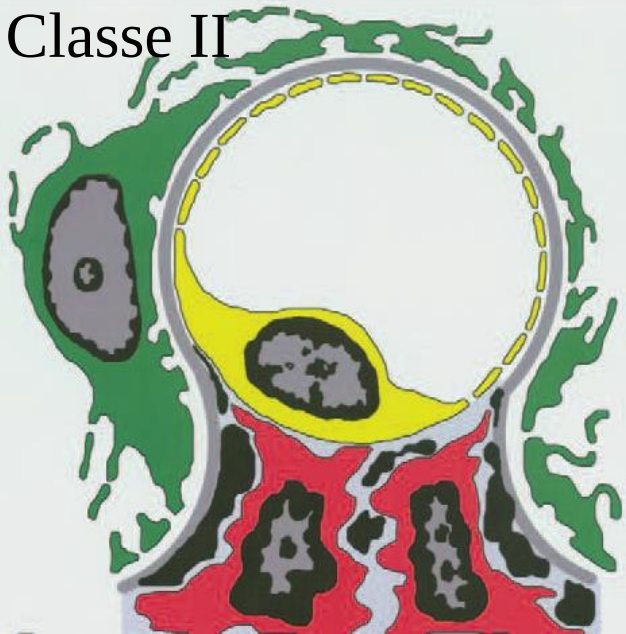
Lupus
“wireloops”
trombos hialinos

Nefrite Lúpica

Classe I

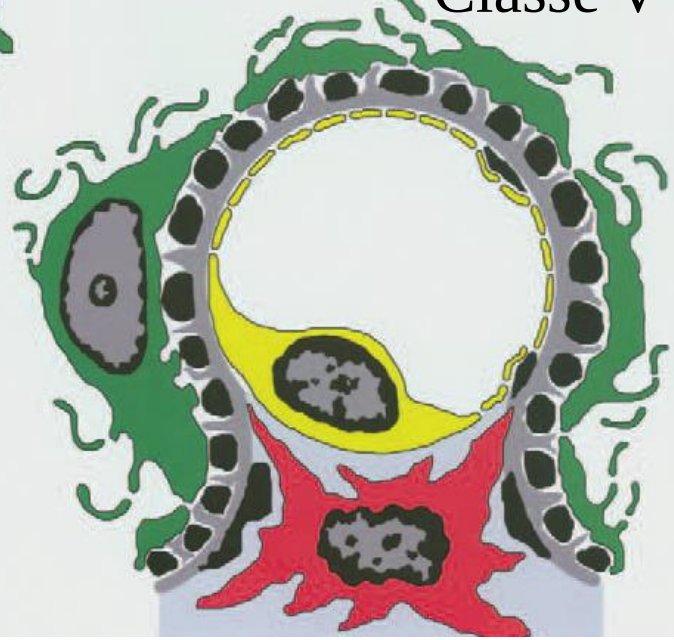


Classe II



Classe III – Focal
Classe IV - Difuso

Classe V

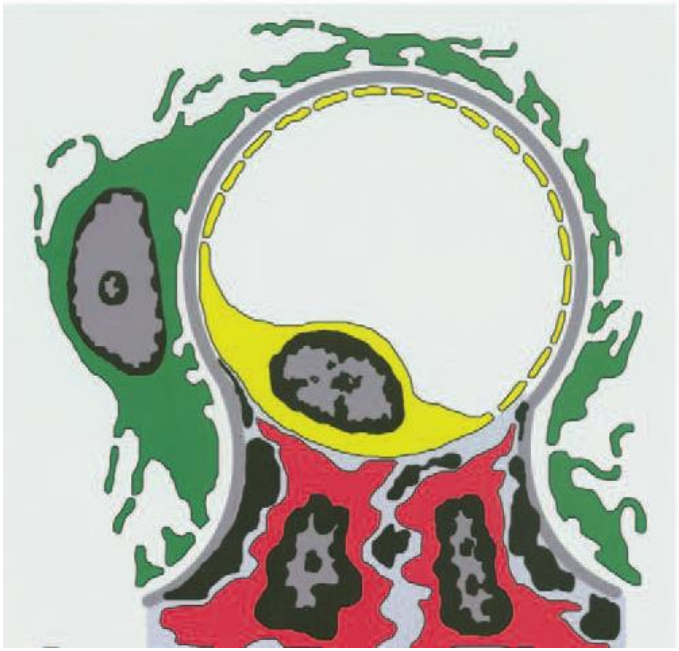


Seshan & Jennette, Arch Pathol Lab Med 133:233-248;2009

Nefropatia IgA primária
Doença de Berger

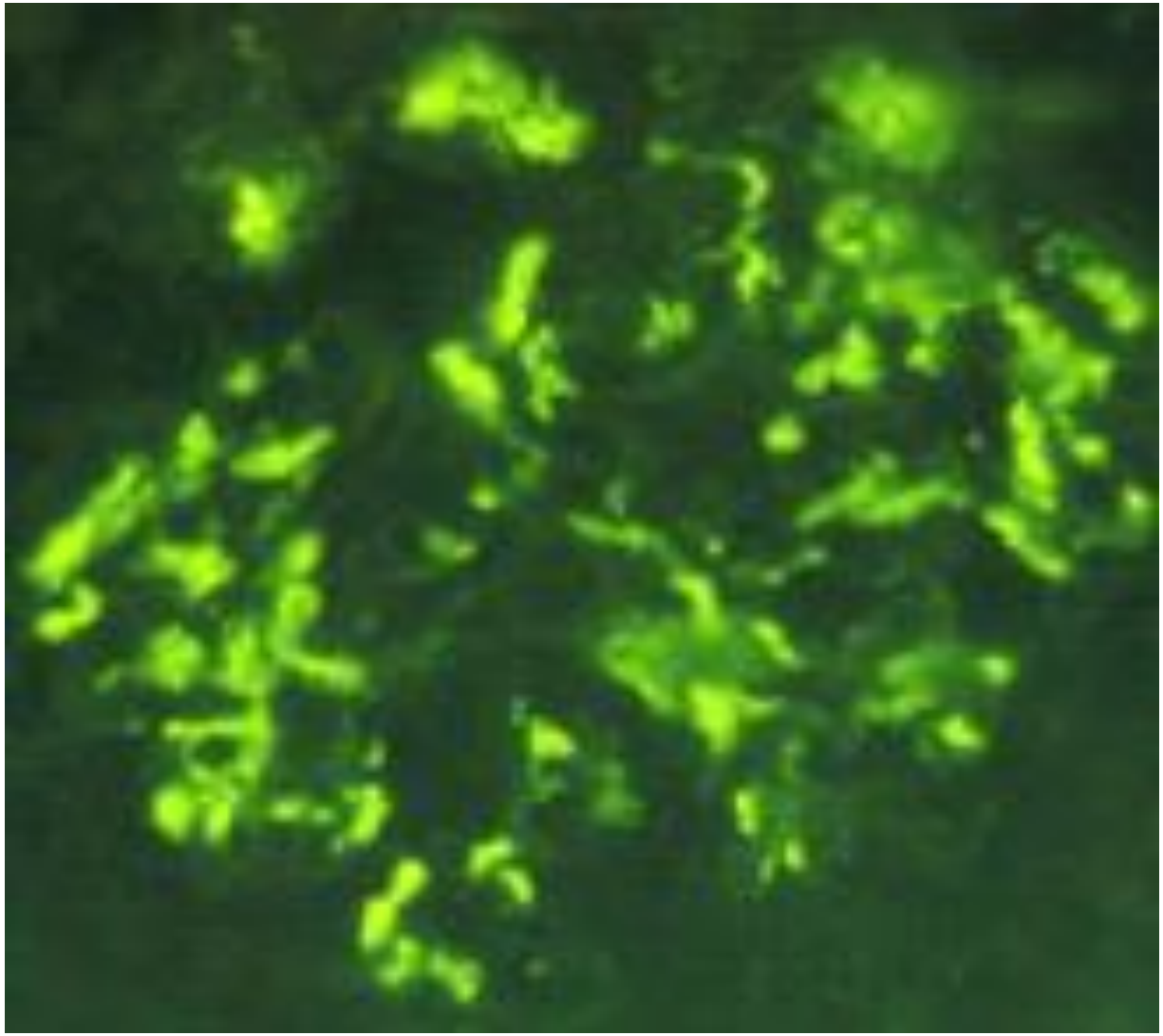
1. Alt urinárias assintomáticas
   a) **hematúria** glomerular isolada
   b) Proteinúria isolada
2. síndrome nefrótica
3. síndrome nefrítica
4. IRA (insuficiência aguda – rapidamente progressiva)
5. Doença Renal Crônica

Classificação de Oxford:
Trimarchi H et al., Kidney Int 91:1014-1021;2017
Roberts ISD et al. Kidney Int 76:546–556;2009



Eixo mesangial

Nefropatia IgA



Mesângio

Identificação

- 11 anos de idade
- sexo masculino
- cor branca
- profissão: estudante

História

- Faringite purulenta há 15 dias
- há um dia:
    - **hematúria**
    - **oligúria**
    - edema

Exame físico

- edema generalizado
- **PA = 160x100mmHg**

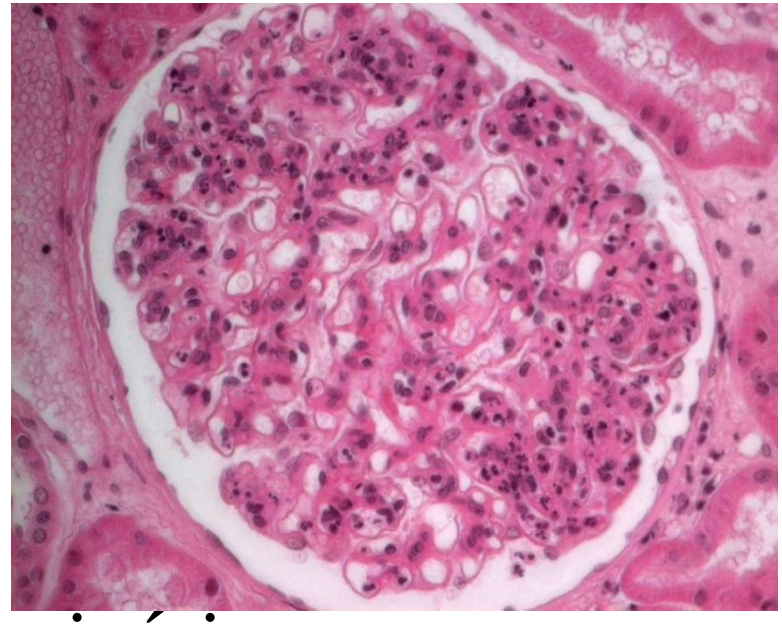
Exames complementares

- **Creatinina = 4,6mg%**
- Proteinúria de 24h = 1.500mg
- Proteínas totais = 6,1g%
- Albumina = 4,1
- ASO = 800UTodd
- FAN = negativo

Manifestações Clínicas - Glomerulopatias

1. **Alt urinárias assintomáticas**
   a) **hematúria glomerular isolada**
   b) **Proteinúria isolada**
2. **síndrome nefrótica**
3. **síndrome nefrítica**
4. **IRA (insuficiência aguda – rapidamente progressiva)**
5. **Doença Renal Crônica**

Glomerulopatias
Alterações clínicas

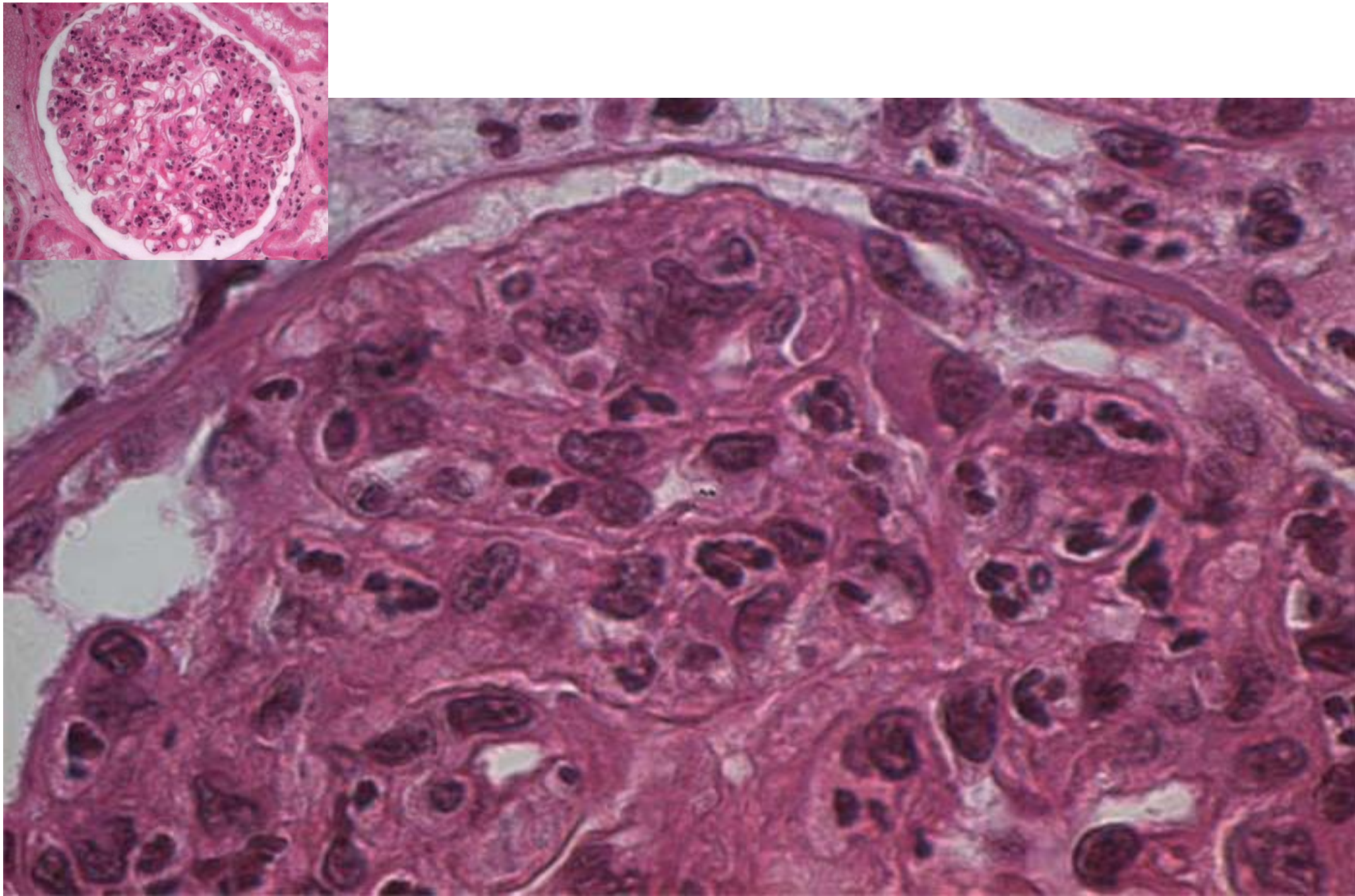


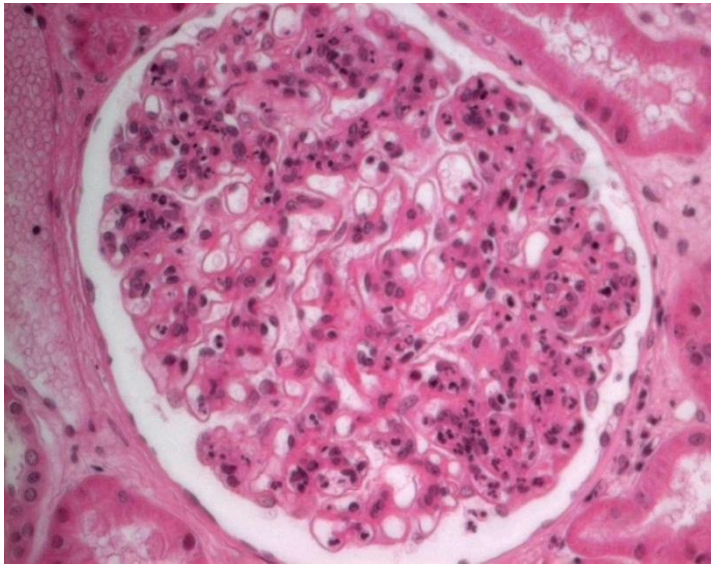
Síndrome Nefrítica

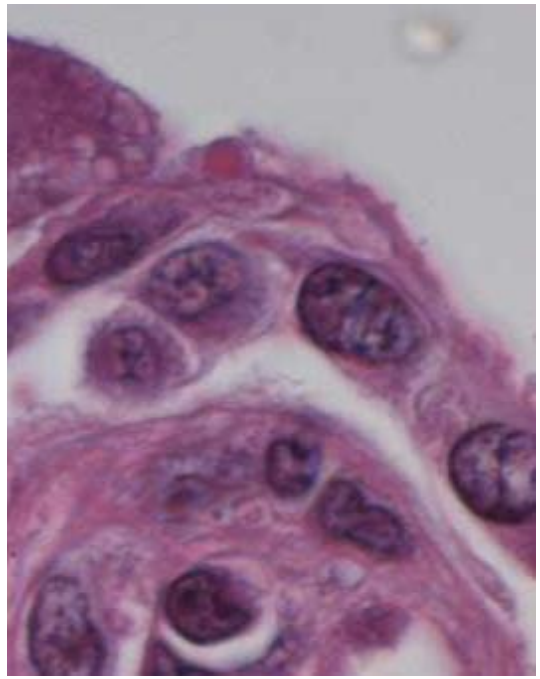
diminuição da TFG

- Oligúria – diminuição do volume urinário
- Hipertensão arterial - retenção de líquido
- Azotemia: retenção de elementos nitrogenados

- Hematúria
- proteinúria variável
- edema (discreto)



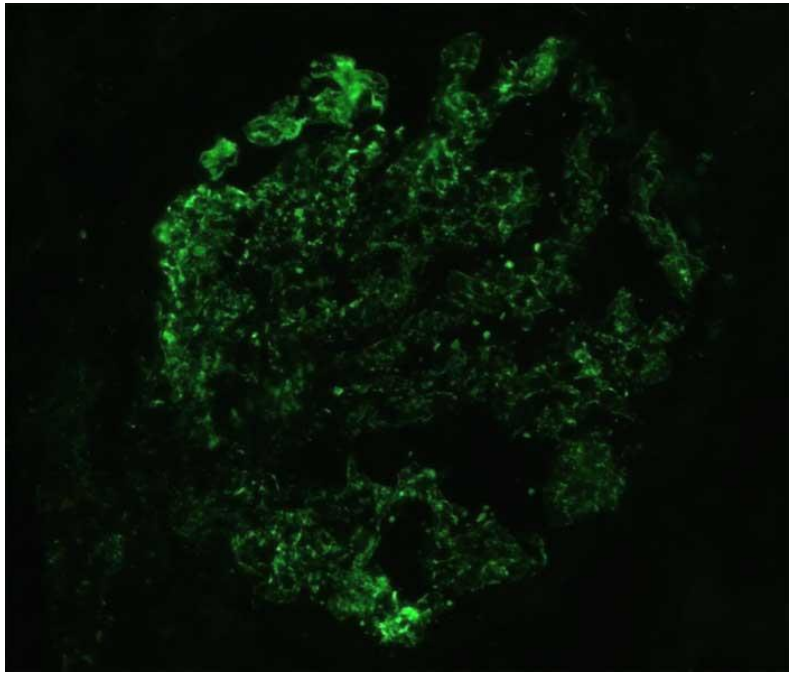




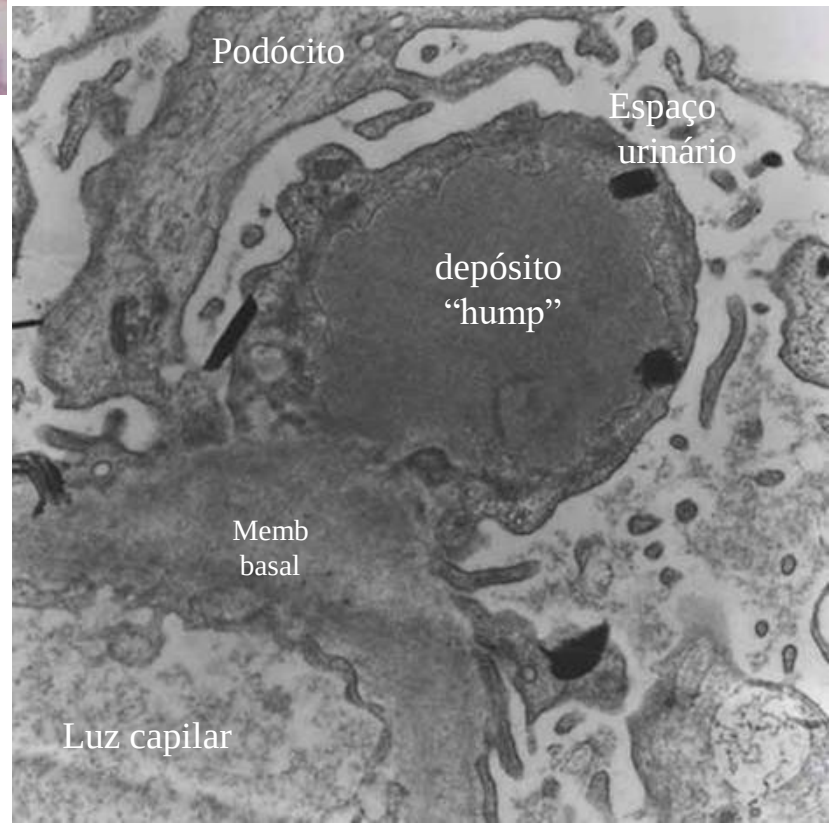
Alça Capilar

granular grosseiro

“humps”

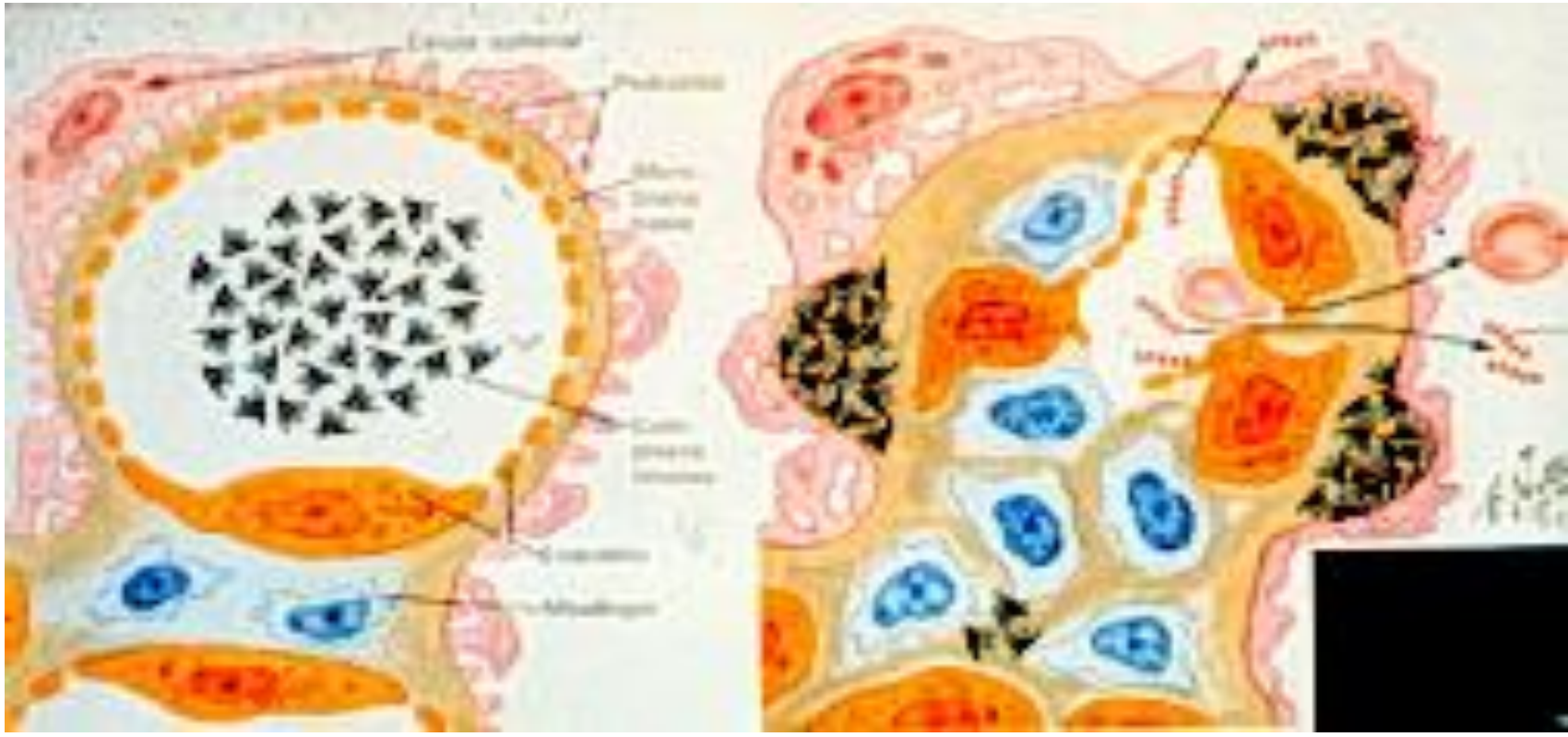


C3, IgG
GNDA



GNDA - Patogenia

Doença do soro aguda
- Von Pirket
soro albumina-bovina
injetou em coelho

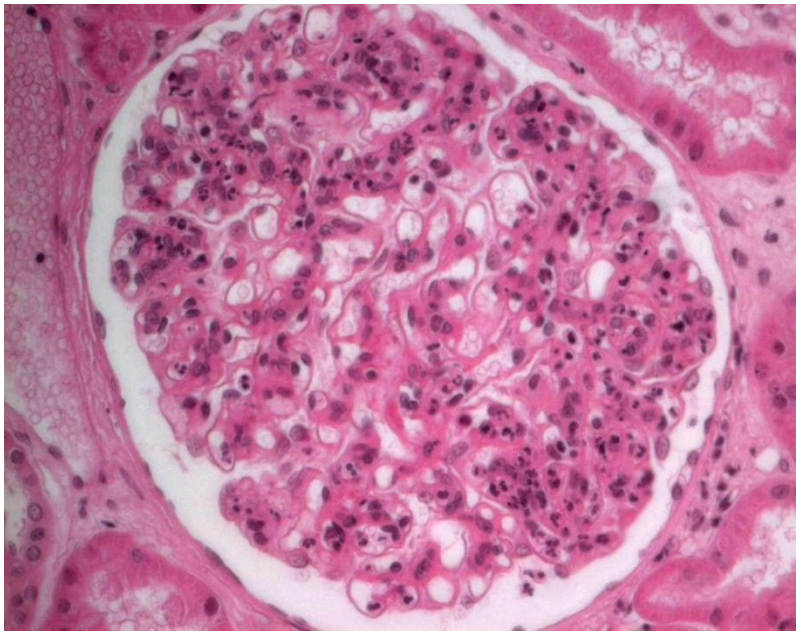


Glomerulopatias

Patogenia

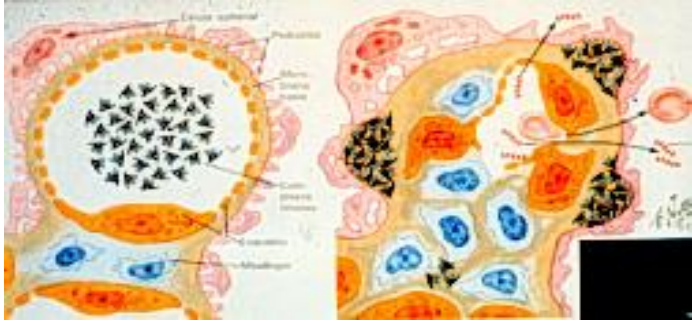
Modelos experimentais

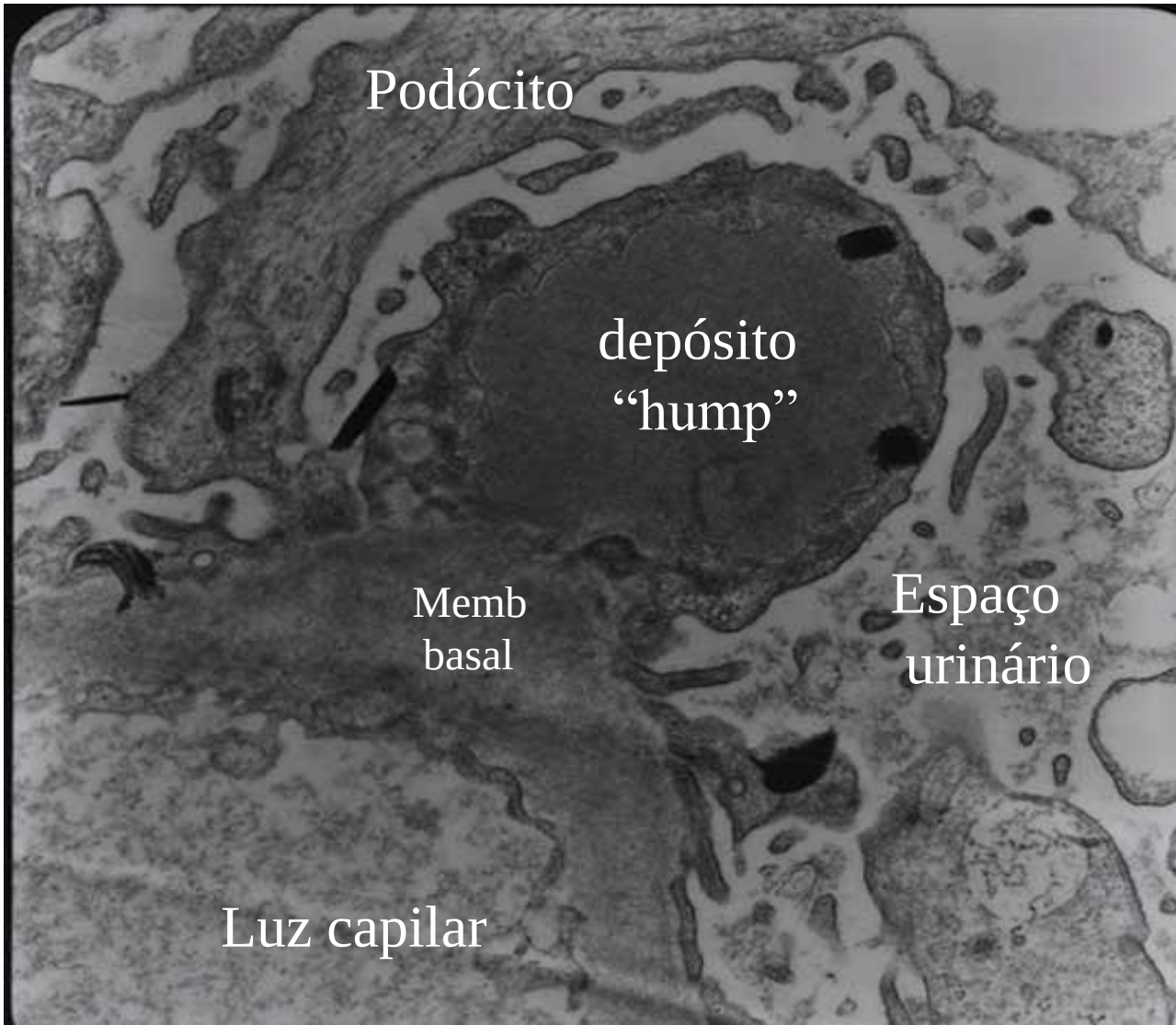
Doença do soro aguda

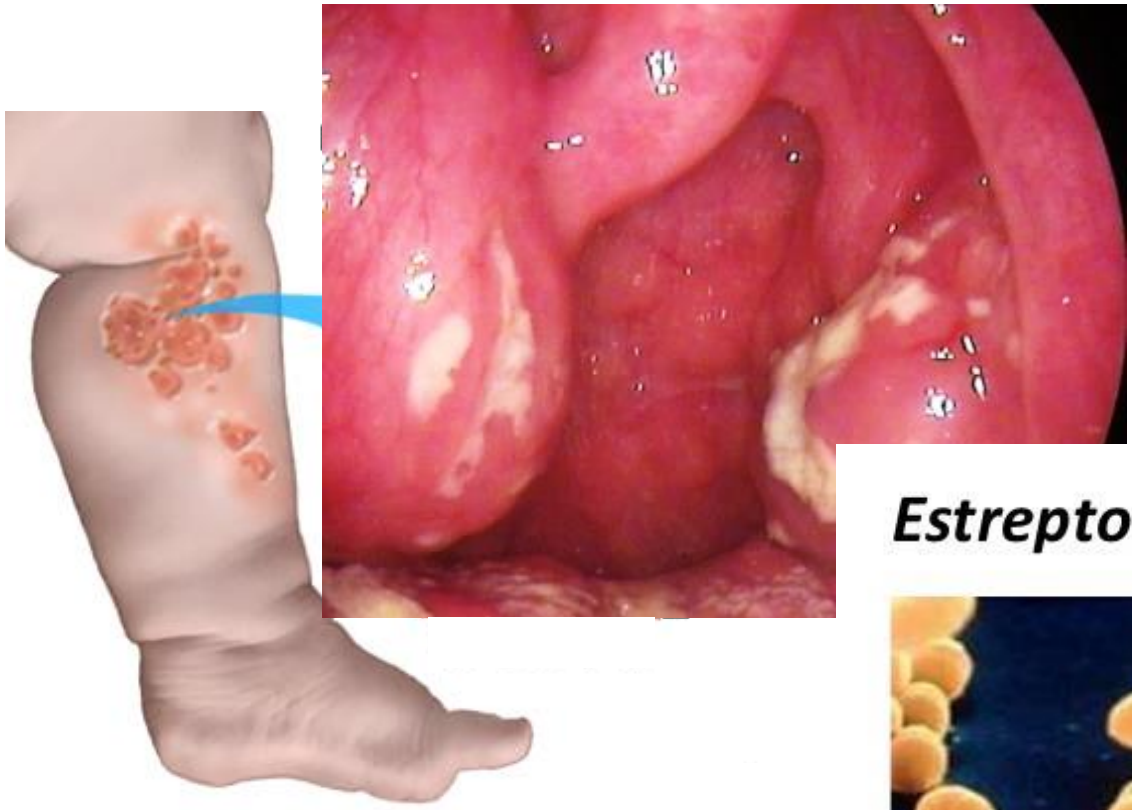


hipercelularidade

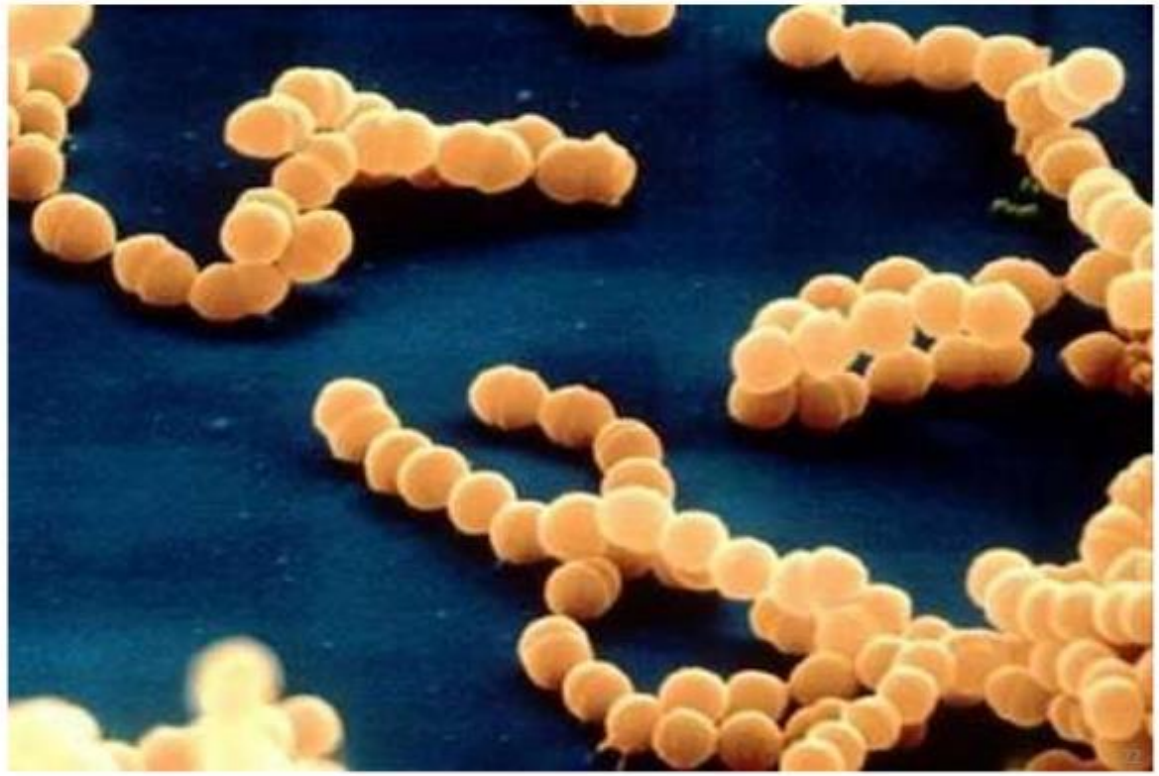
- IC - “Humps”





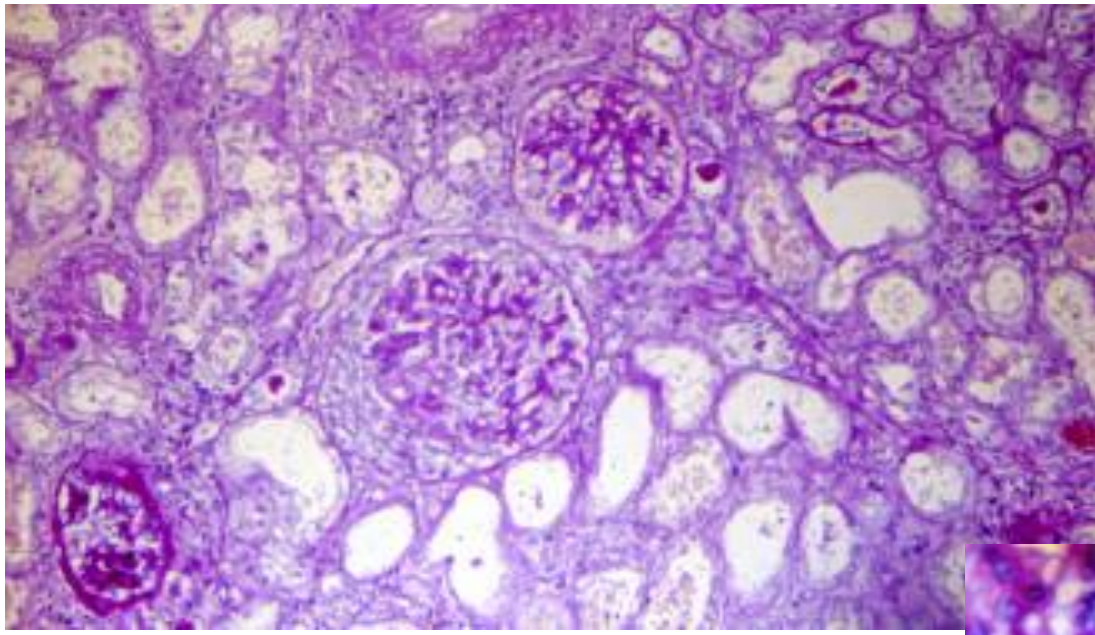


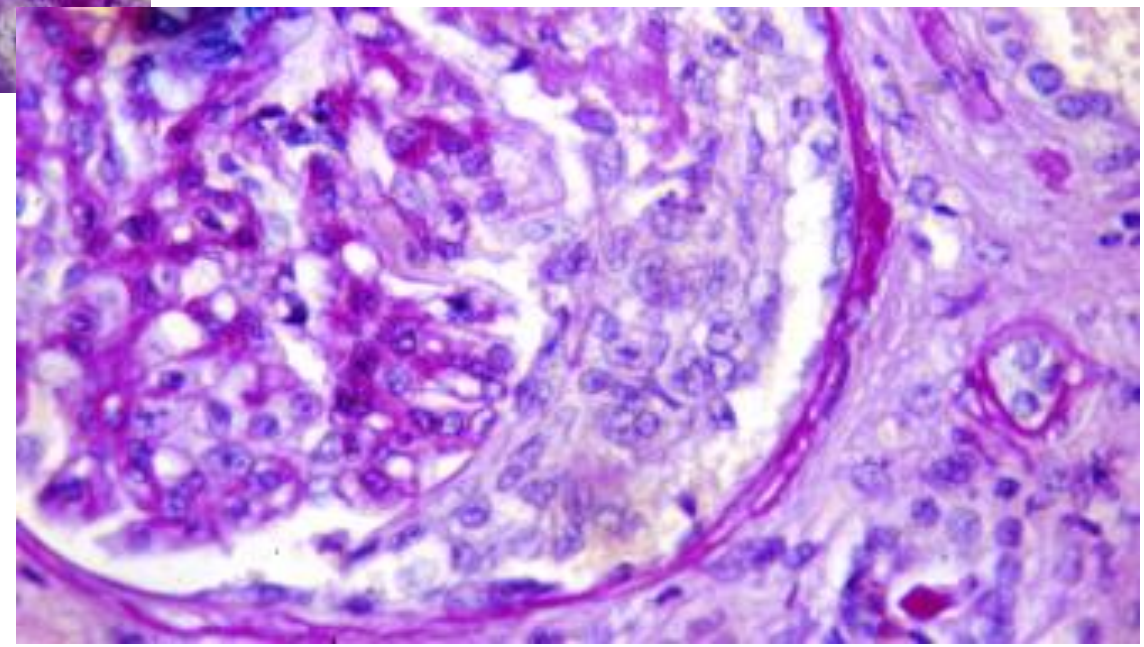
Estreptococo beta-hemolítico do grupo A

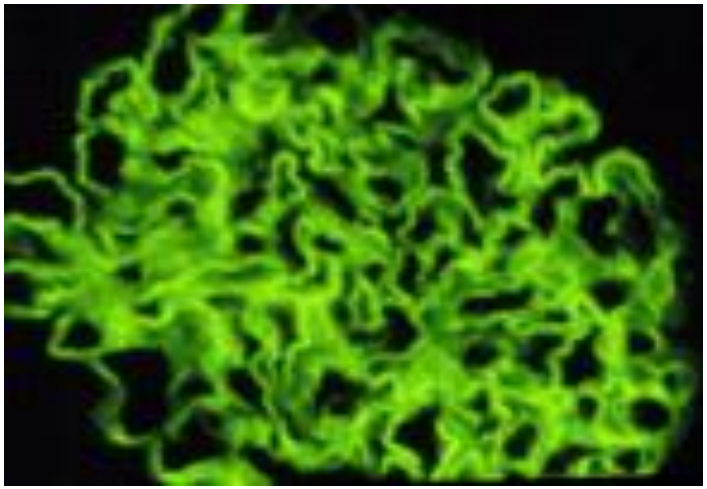


Manifestações Clínicas - Glomerulopatias

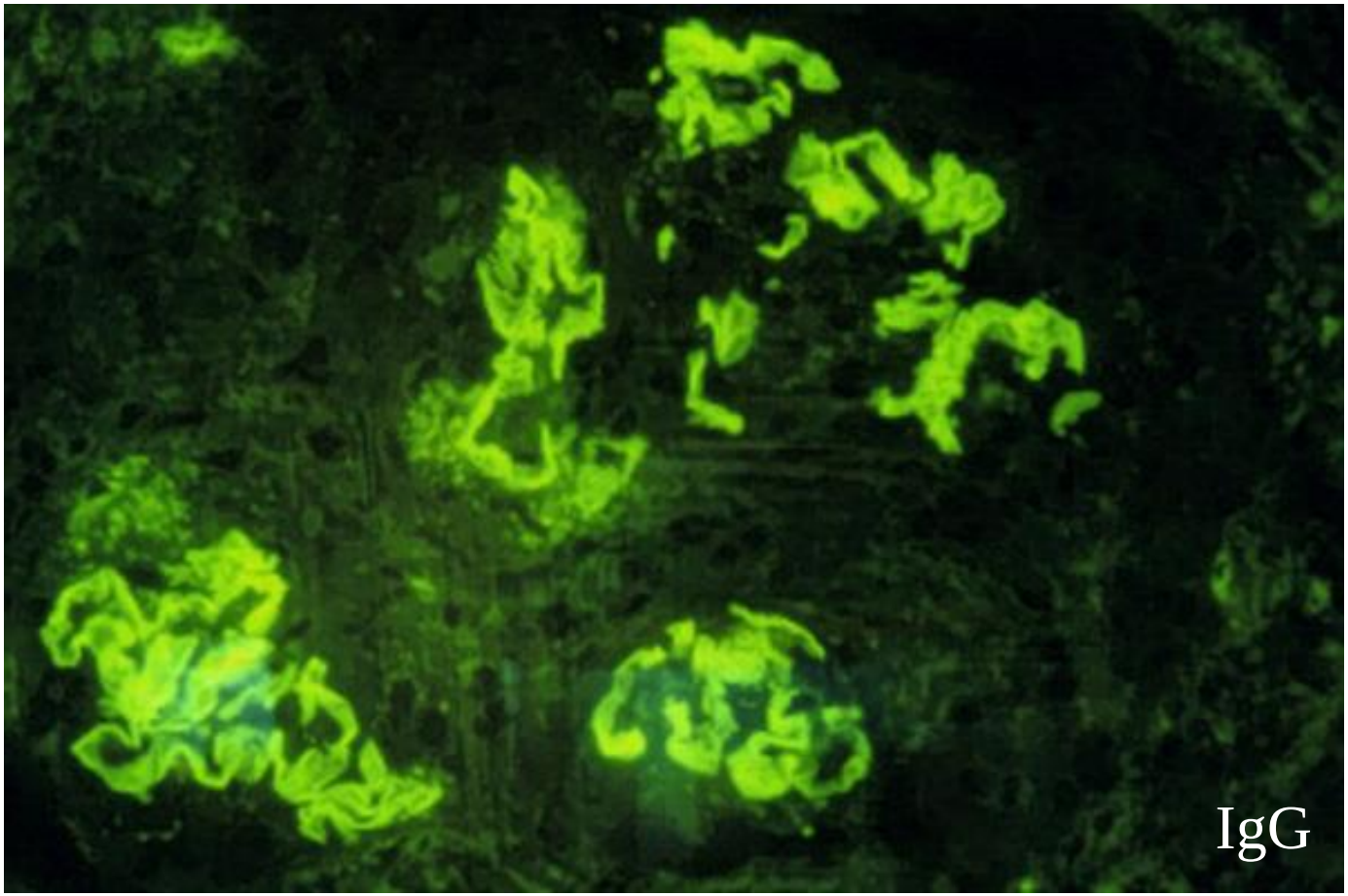
1. **Alt urinárias assintomáticas**
   a) **hematúria glomerular isolada**
   b) **Proteinúria isolada**
2. **síndrome nefrótica**
3. **síndrome nefrítica**
4. **IRA (insuficiência aguda – rapidamente progressiva)**
5. **Doença Renal Crônica**







Linear = membrana basal



Diagnóstico Clínico

IRA – GN rapidamente progressiva

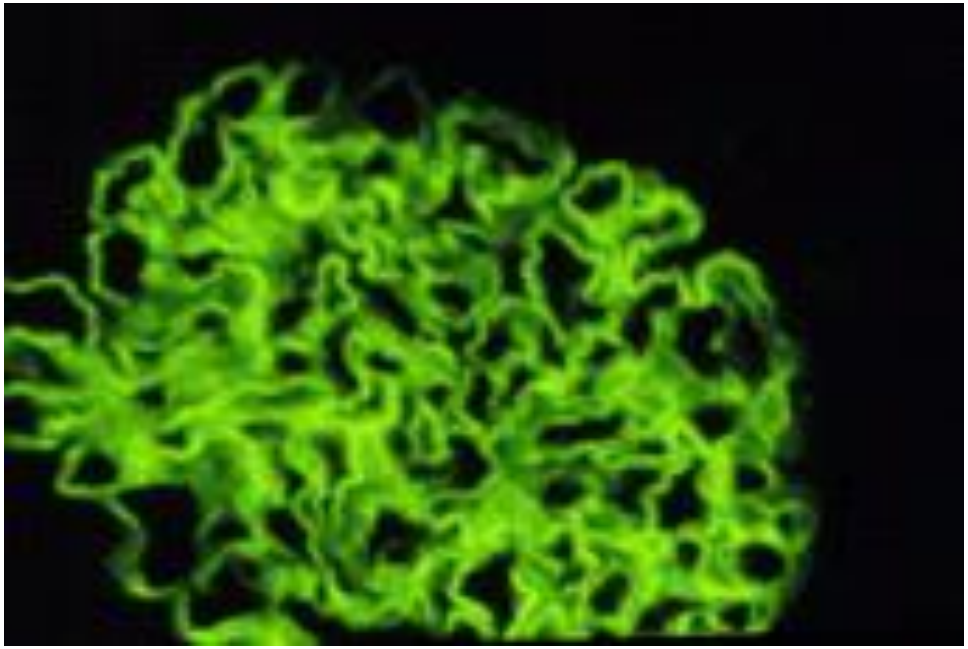
Diagnóstico Morfológico

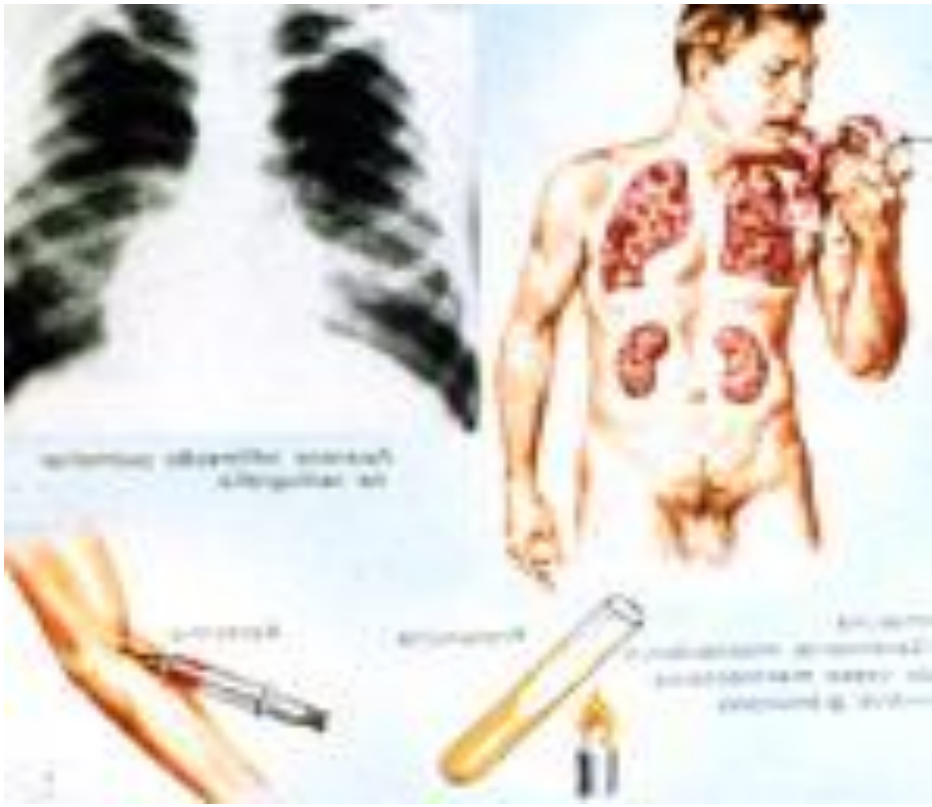
Glomerulonefrite Crescêntica do tipo I
Glomerulonefrite anti-membrana basal glomerular

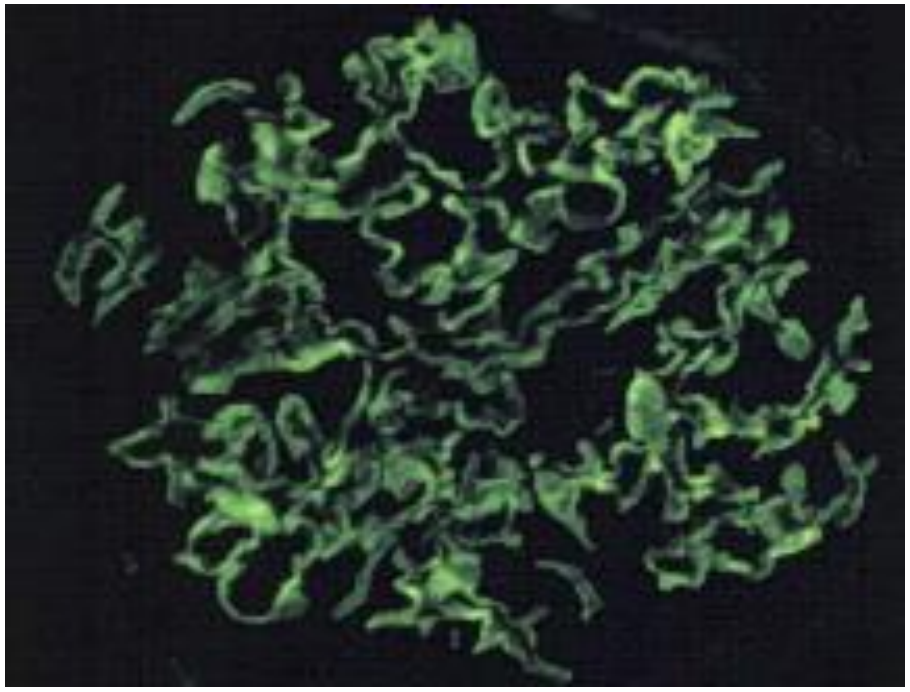
Glomerulonefrite crescêntica

> 50% dos glomérulos

- Tipo I - ac anti MBG
  - Síndrome de Goodpasture
    com hemorragia pulmonar
  - GN anti MBG
    sem hemorragia pulmonar





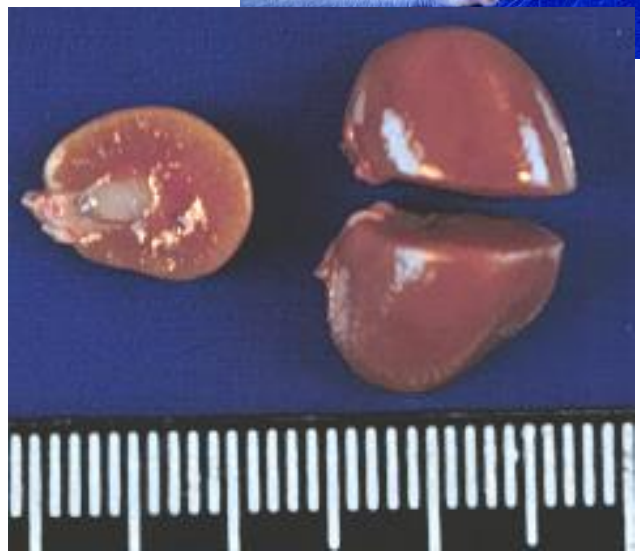


Síndrome de Goodpasture

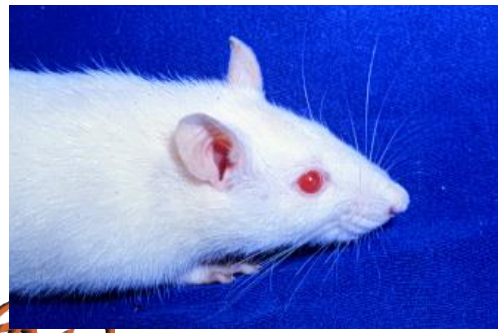
3. Nefrite Nefrotóxica

- Landerman (início século)
- 1934 - Masugi - Nefrite de Masugi

macerado de rim de rato (MBG)
injeta em coelho

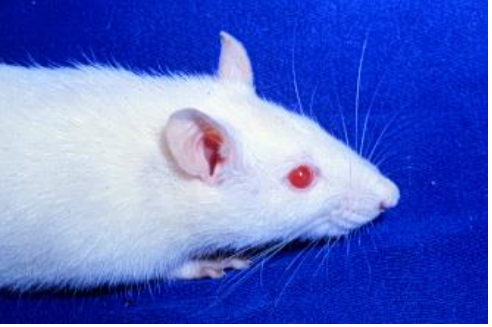


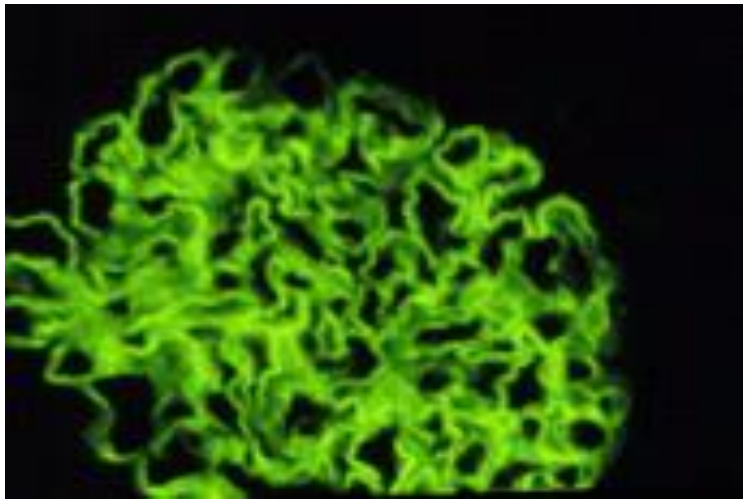
soro do coelho
(ac anti MBG do rato)



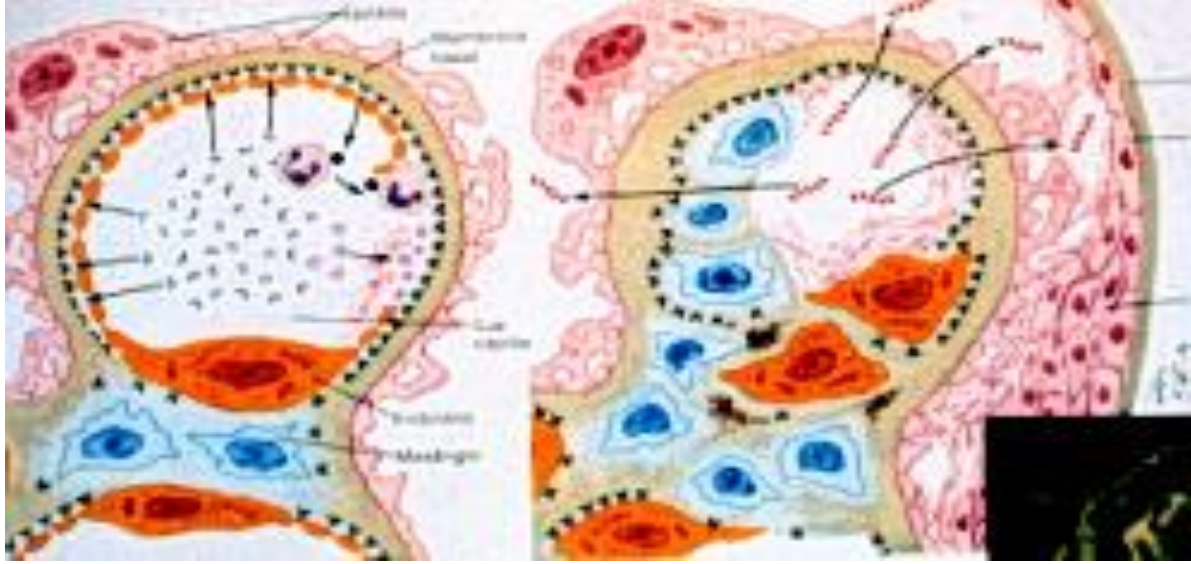
injeta no rato

GN anti membrana basal glomerular

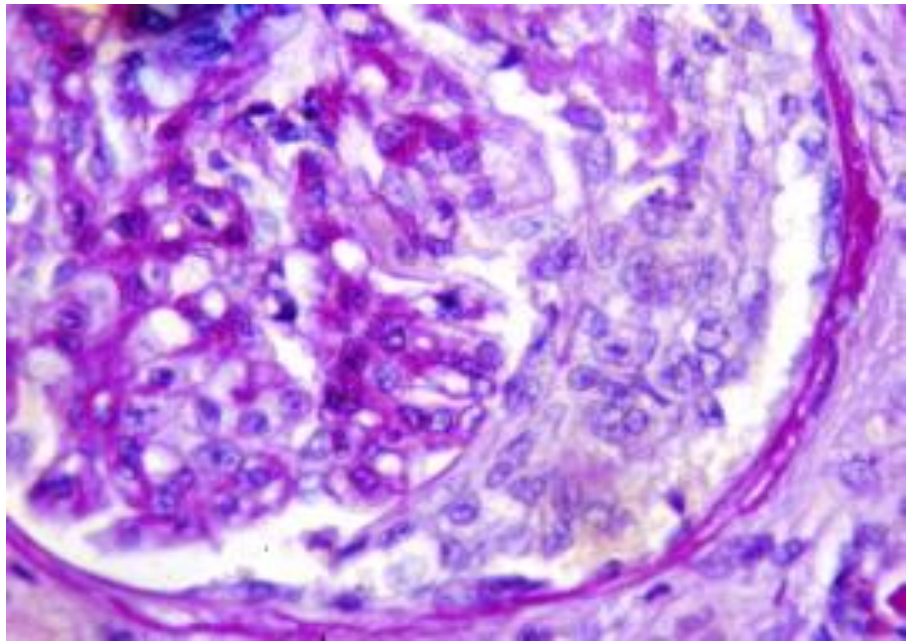




IgG



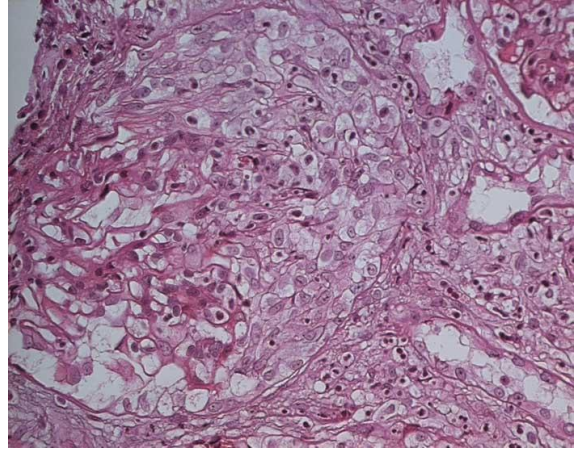
Hipercelularidade
ac na MBG - padrão linear

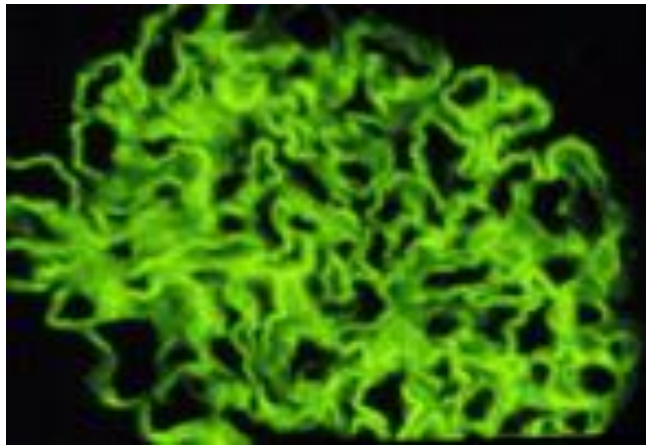


GN anti MBG

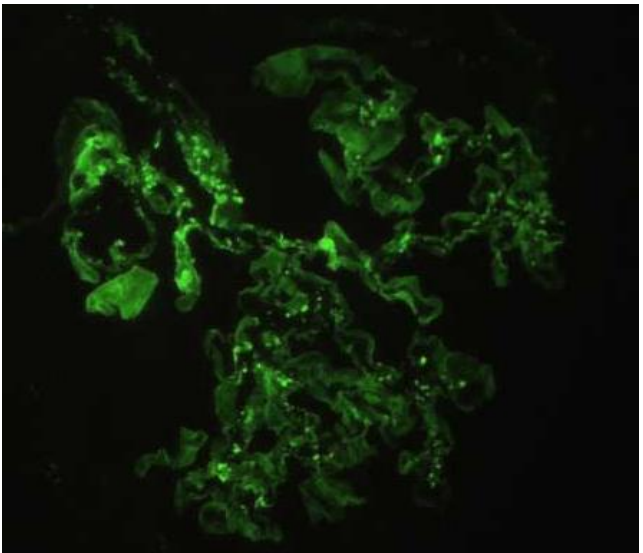
Glomerulonefrite crescêntica

≥ 50% dos glomérulos



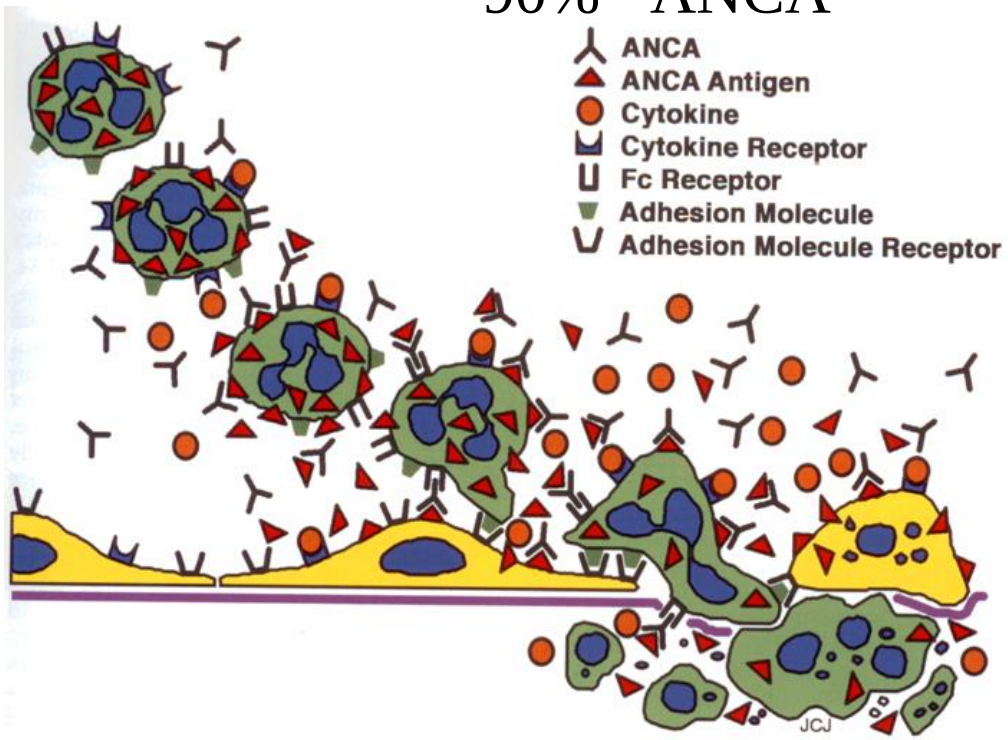


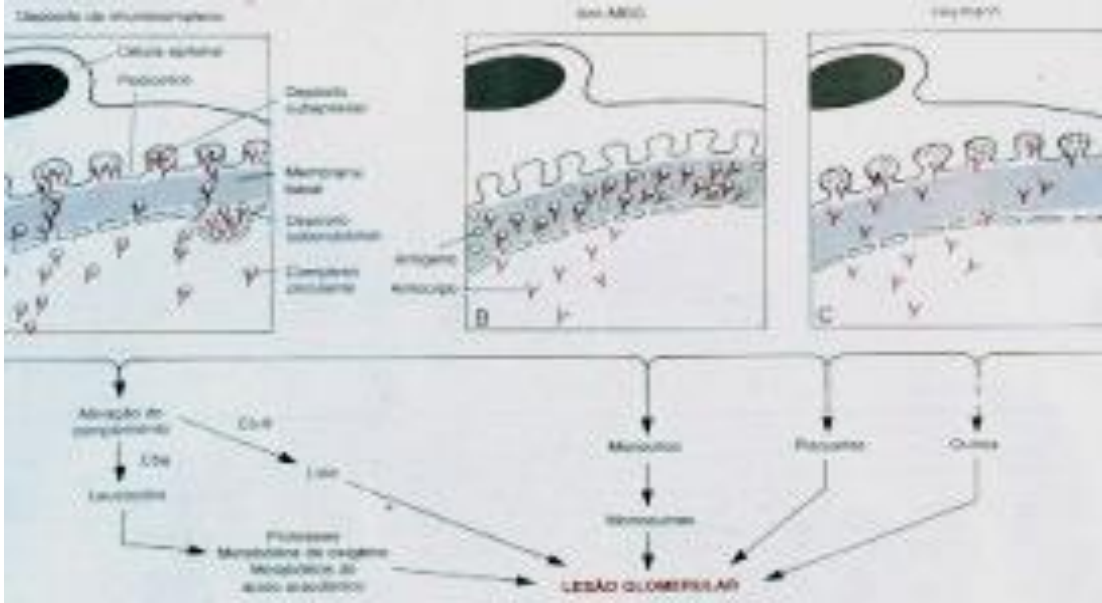
Tipo I – linear - ac anti MBG -10%



Tipo II – 40%
não linear
depósito IC/C

Tipo III - pauci-imune – 50%
90% - ANCA





I. lesão mediada por anticorpo

1. formação “in situ” de imunocomplexo
2. depósito de imunocomplexo circulante
3. Anticorpo anti-citoplasma de neutrófilo - ANCA

II. Lesão mediada por células

III. Ativação da via alternativa do complemento

Glomerulopatias

Mecanismos imunológicos

I. lesão mediada por anticorpo

- neutrófilo (quimiotaxia):
  - proteases, metabólitos do ác aracdônico, radicais livres de O2
- complemento (fagocitose/quimiotaxia/ataque à membrana)
- monócitos e macrófagos
- plaquetas
- mesângio: IL1, radicais livres de O2, metab ácido aracdônico
- sistema da coagulação

Glomerulopatias
correlação clínico-morfológica

Síndrome nefrótica

Glomerulopatias:

- Lesões mínimas
- Membranosa
- Esclerose segmentar focal - GESF

Glomerulonefrites:

- Membranoproliferativa - GNMP
- Proliferativa difusa aguda - GNDA

- Crescêntica

Síndrome nefrítica

IRA

Manifestações Clínicas - Glomerulopatias

1. Alt urinárias assintomáticas
   a) hematúria glomerular isolada
   b) Proteinúria isolada
2. síndrome nefrótica
3. síndrome nefrítica
4. IRA (insuficiência aguda – rapidamente progressiva)
5. Doença Renal Crônica

Arthur Cecil Alport (1880-1959)
25.01.1880 – África do Sul
17.04.1959 – Londres - Inglaterra

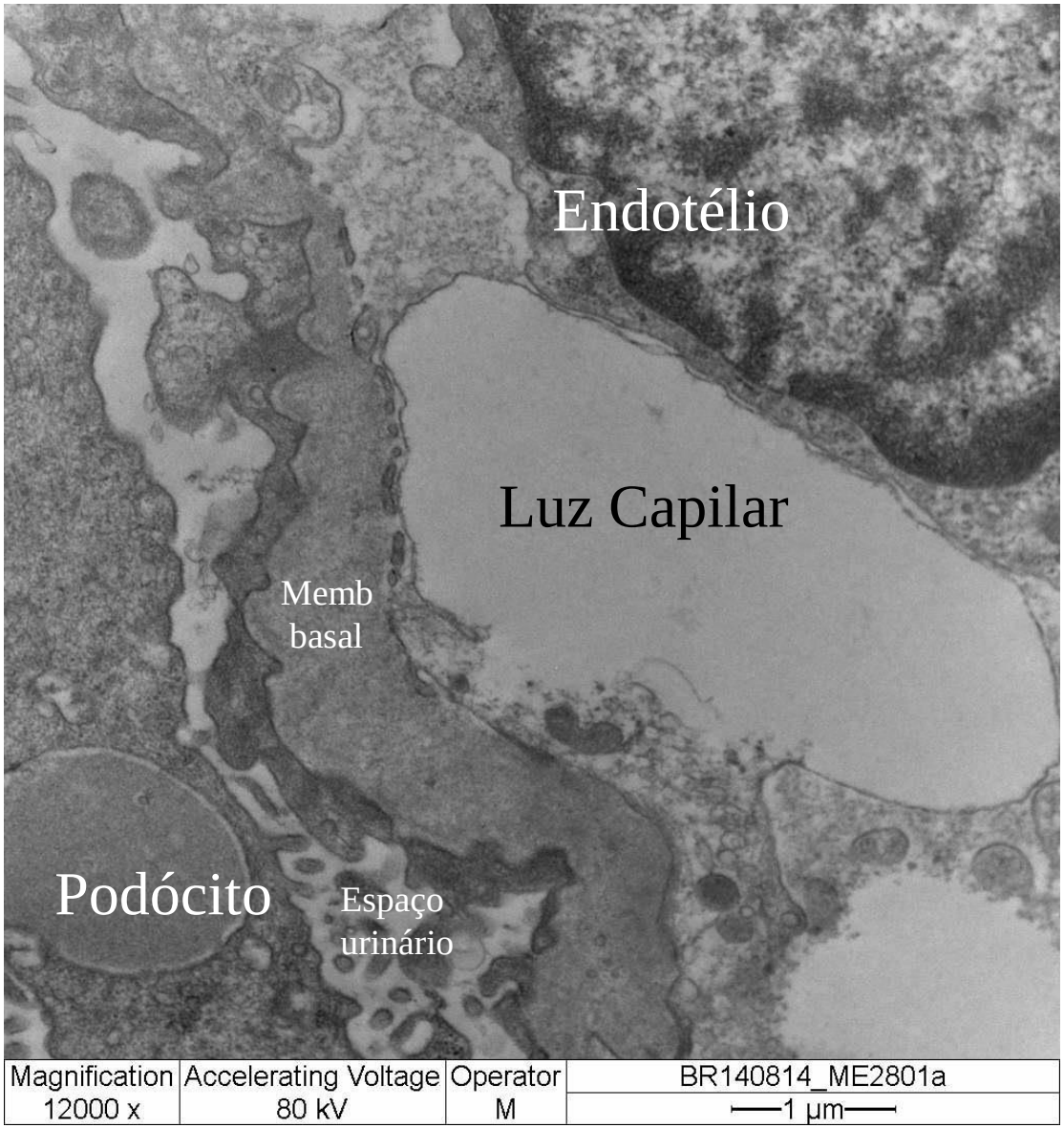
1902 – Guthrie
1912 – Kendall & Hertz

1927 – associação de surdez com a “nefrite hemorrágica familiar hereditária”

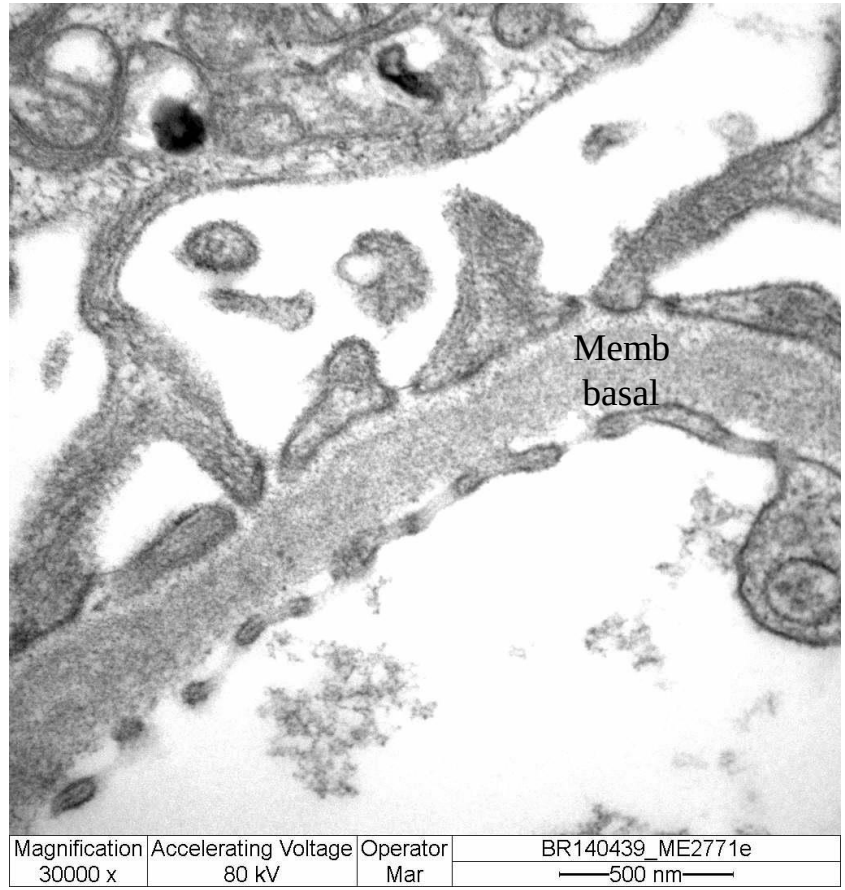
Homem: mais grave

Mulher: menos grave

1972 – Spear & Slusser – defeito colágeno da membrana basal glomerular

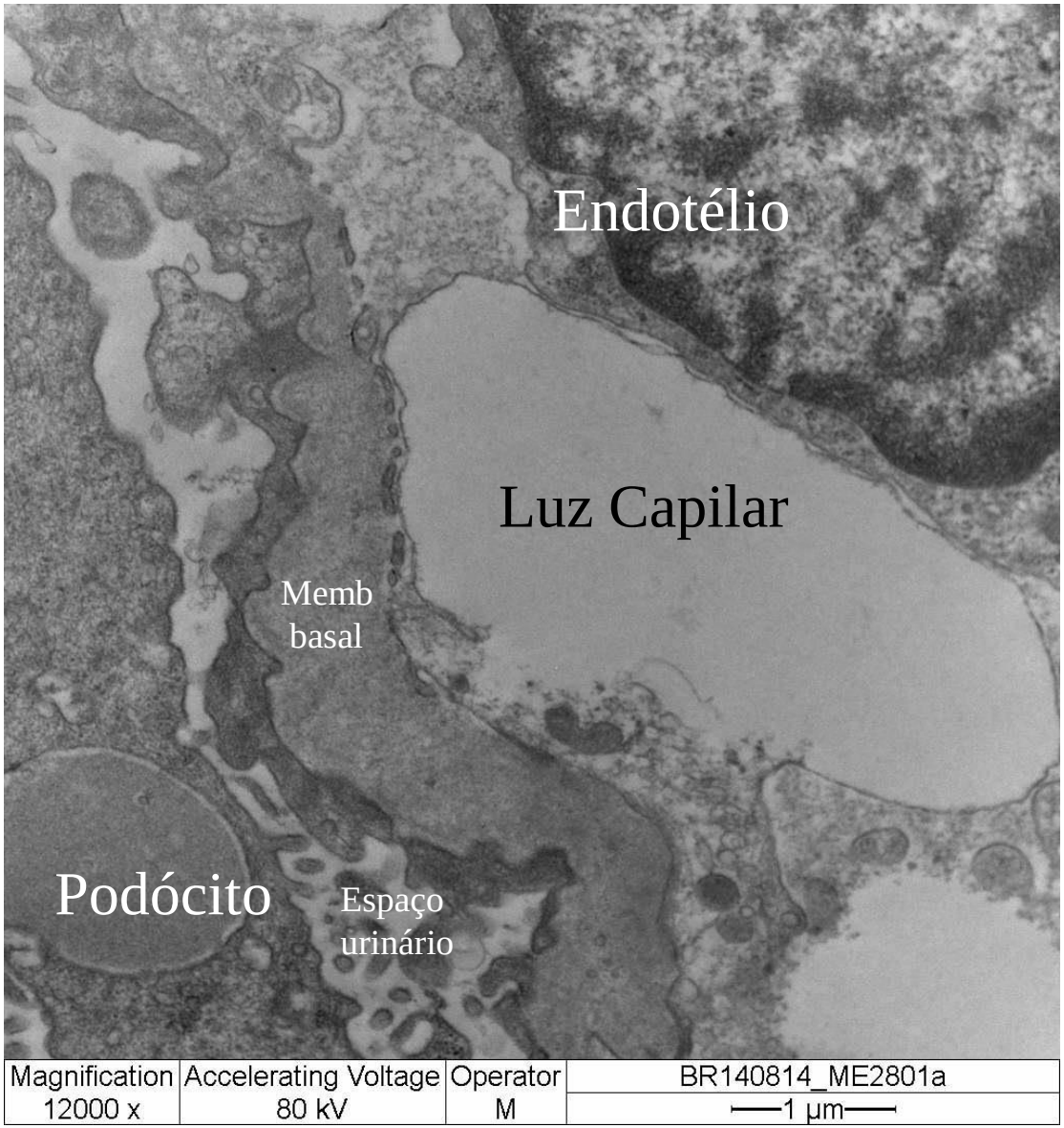


Síndrome de Alport



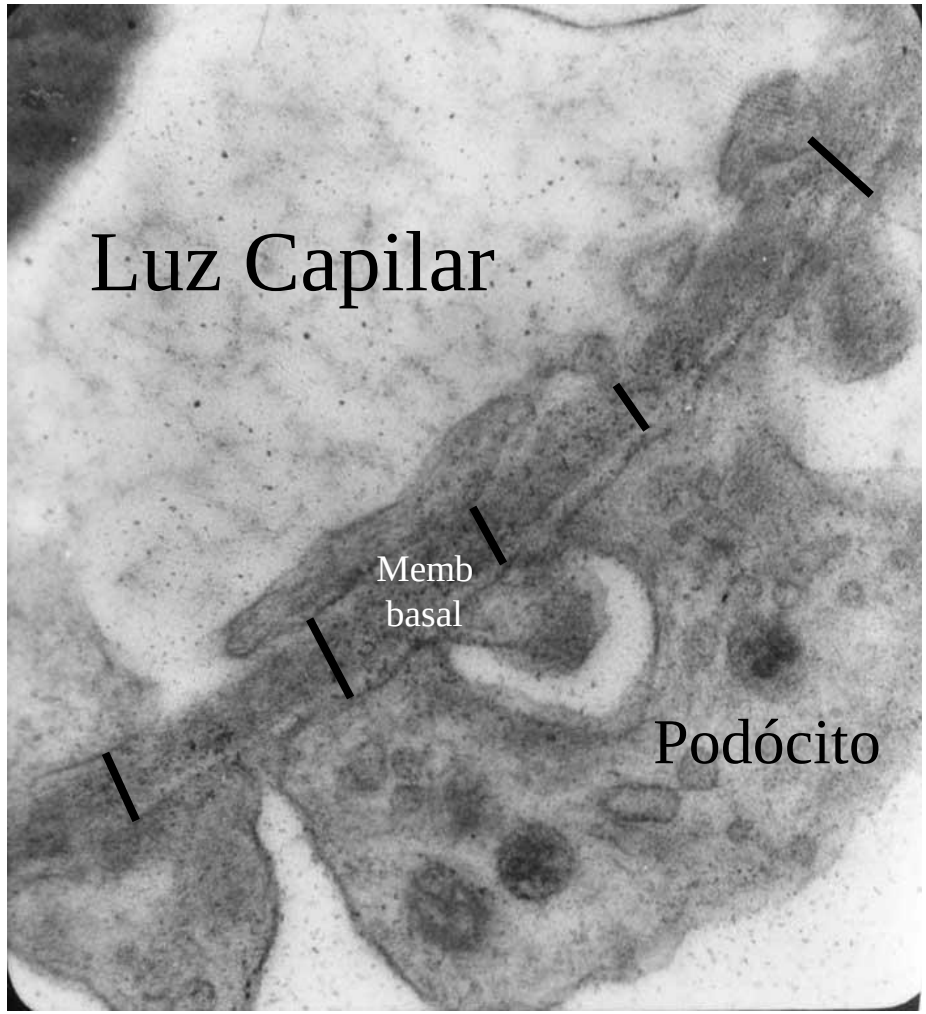
Normal

Hematúria

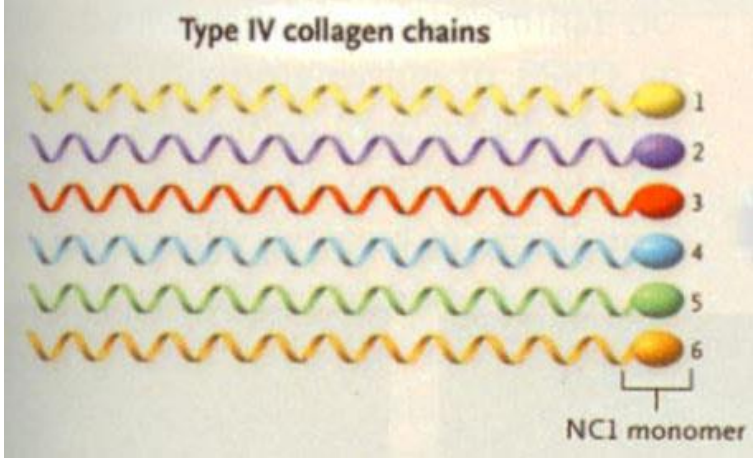


Síndrome de Alport

Hematúria

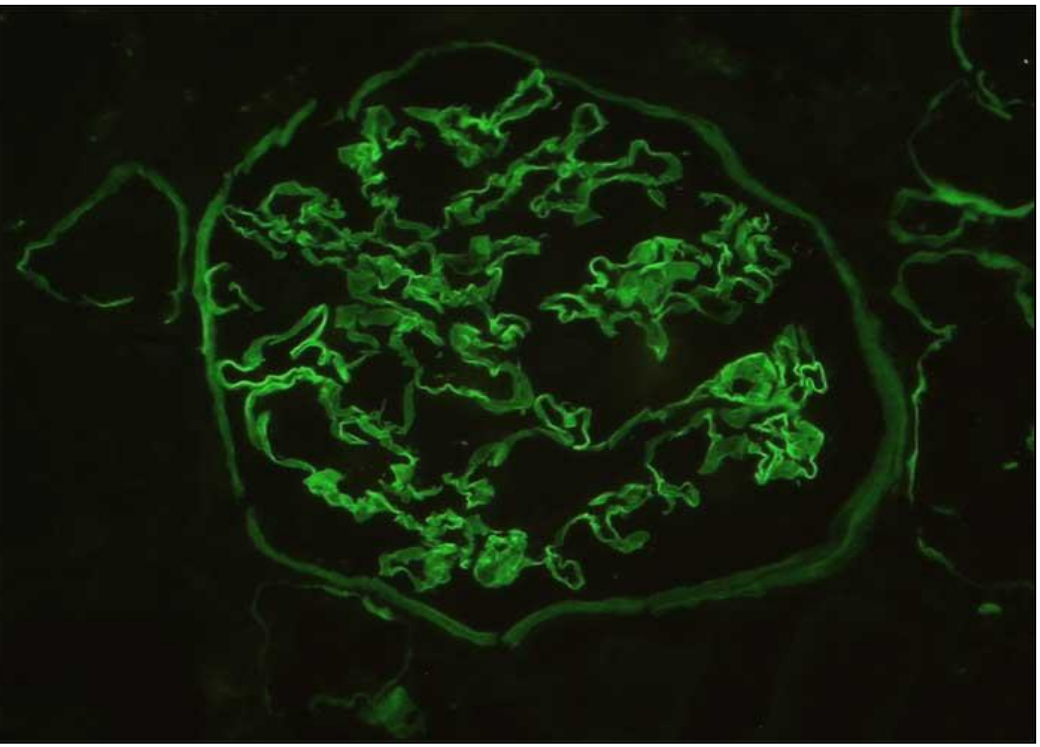


Doença da Membrana Fina



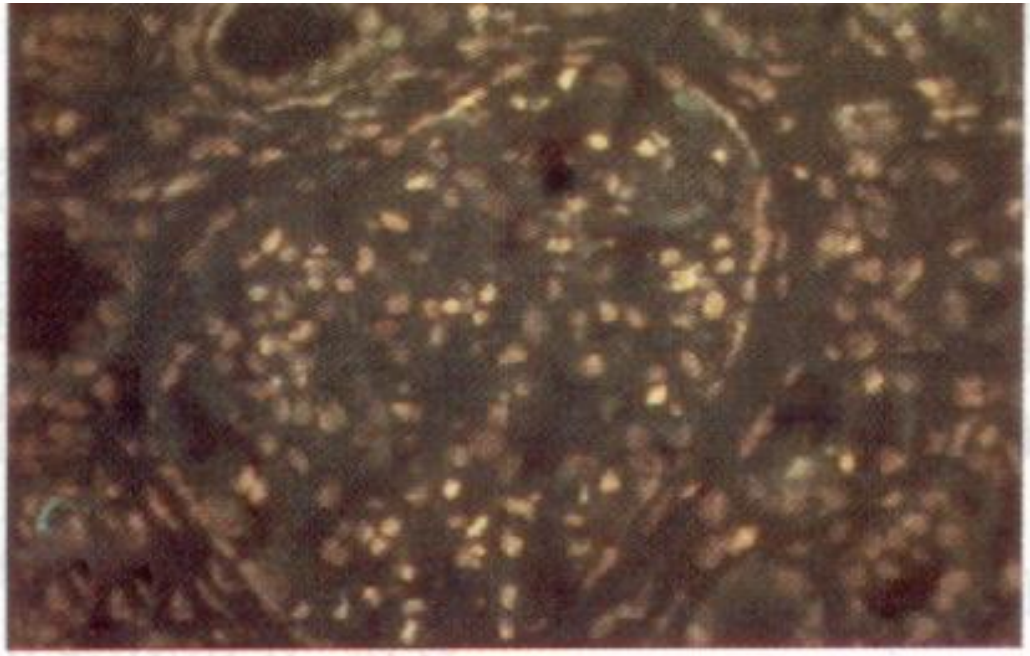
Colágeno IV

Cadeia	cromossomo	gene
α1	13q34	COL4A1
α2	13q34	COL4A2
α3	2q35	COL4A3
α4	2q35	COL4A4
α5	Xq22	COL4A5
α6	Xq22	COL4A6



α5col IV

Alport
Ausência α5col IV



Manifestações Clínicas - Glomerulopatias

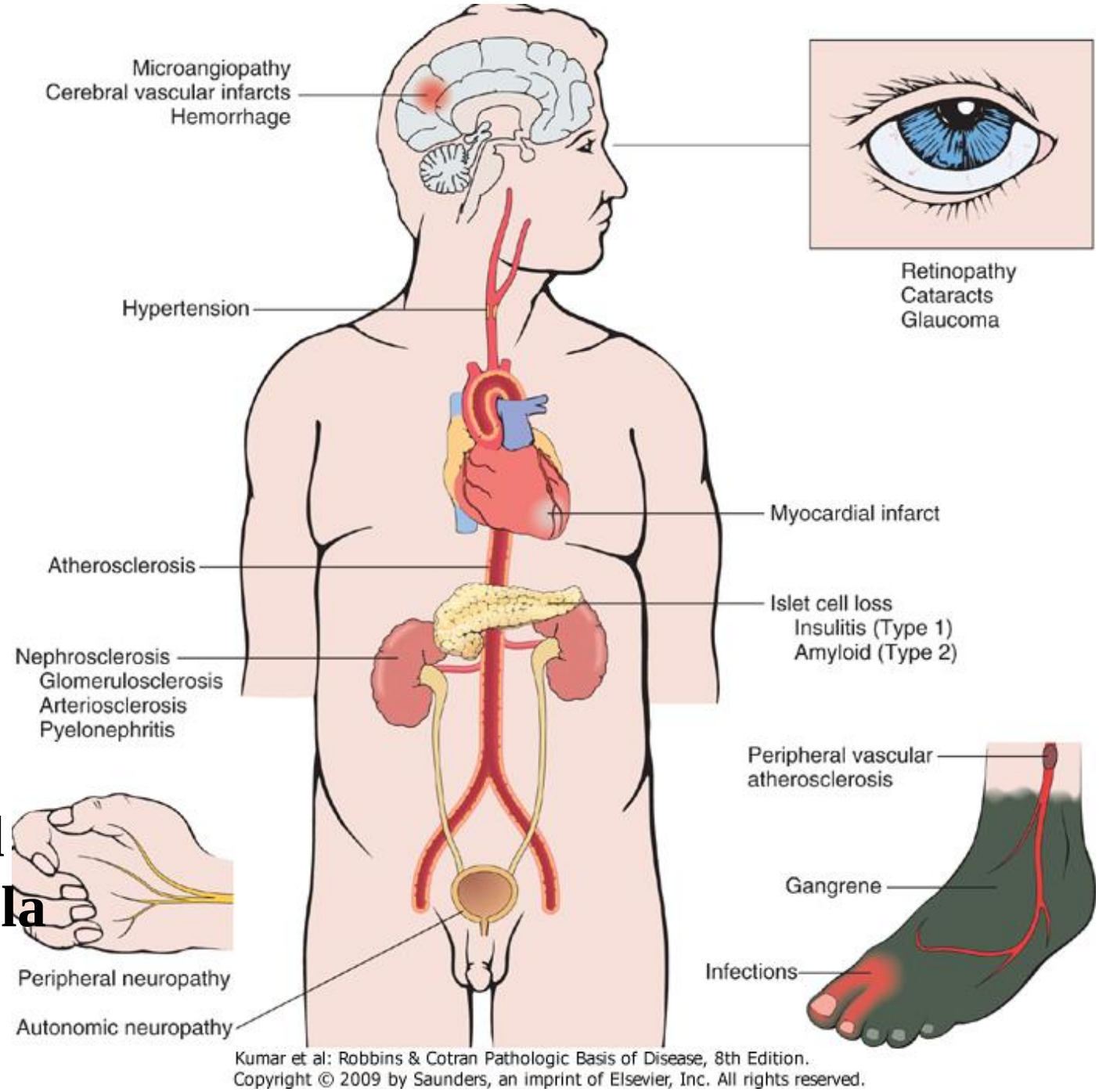
1. Alt urinárias assintomáticas
   a) hematúria glomerular isolada
   b) Proteinúria isolada
2. síndrome nefrótica
3. síndrome nefrítica
4. IRA (insuficiência aguda – rapidamente progressiva)
5. Doença Renal Crônica

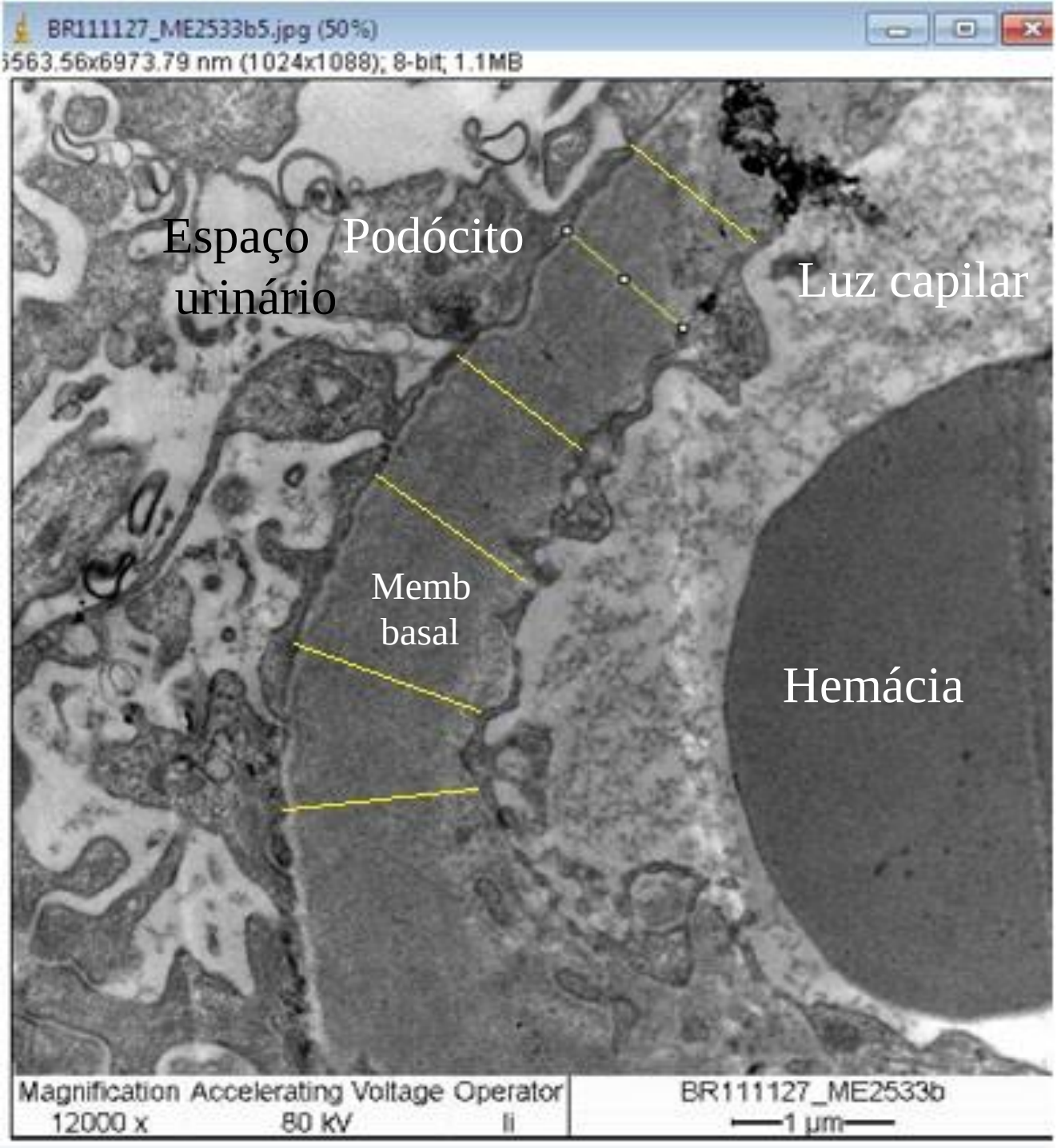
Diabetes *Mellitus*
Hiperglicemia

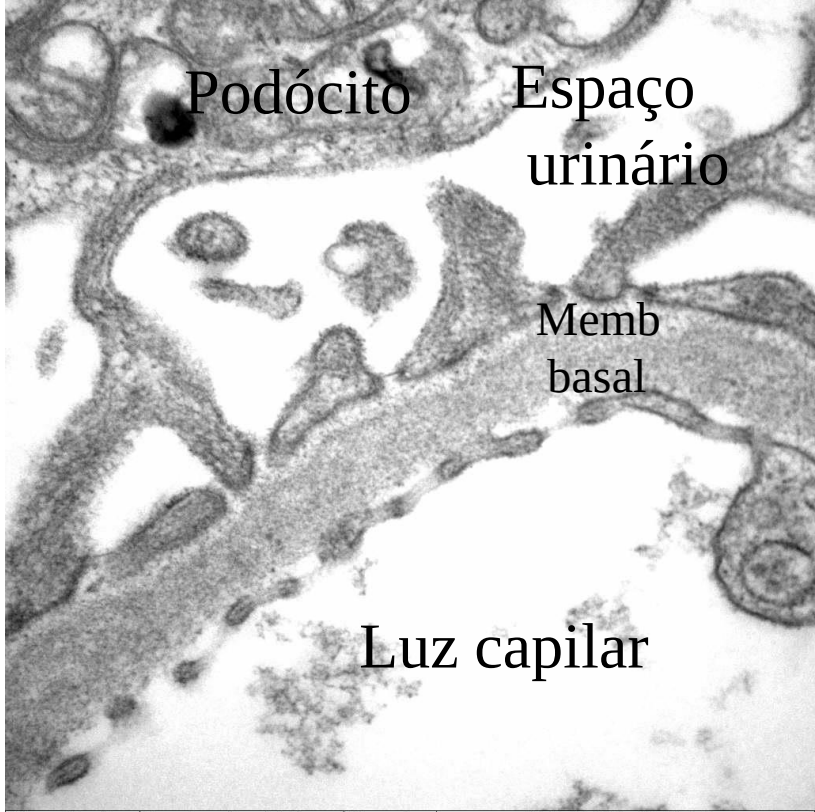
Nefropatia
Retinopatia
Neuropatia
Vaculopatia

Vasculares

↑ **Membrana basal**
Hialinose arteríola
Aterosclerose







MBG = 300 a 400nm normal
(mulher-395nm; homem-430nm)
Diabetes:

1,5 a 2,5 anos - aumento da espessura
- fases iniciais=1200nm.
- avançado: 3000 a 4000nm

Nefropatia Dibética

SPECIAL ARTICLE www.jasn.org

Sociedade Patologia Renal

Pathologic Classification of Diabetic Nephropathy

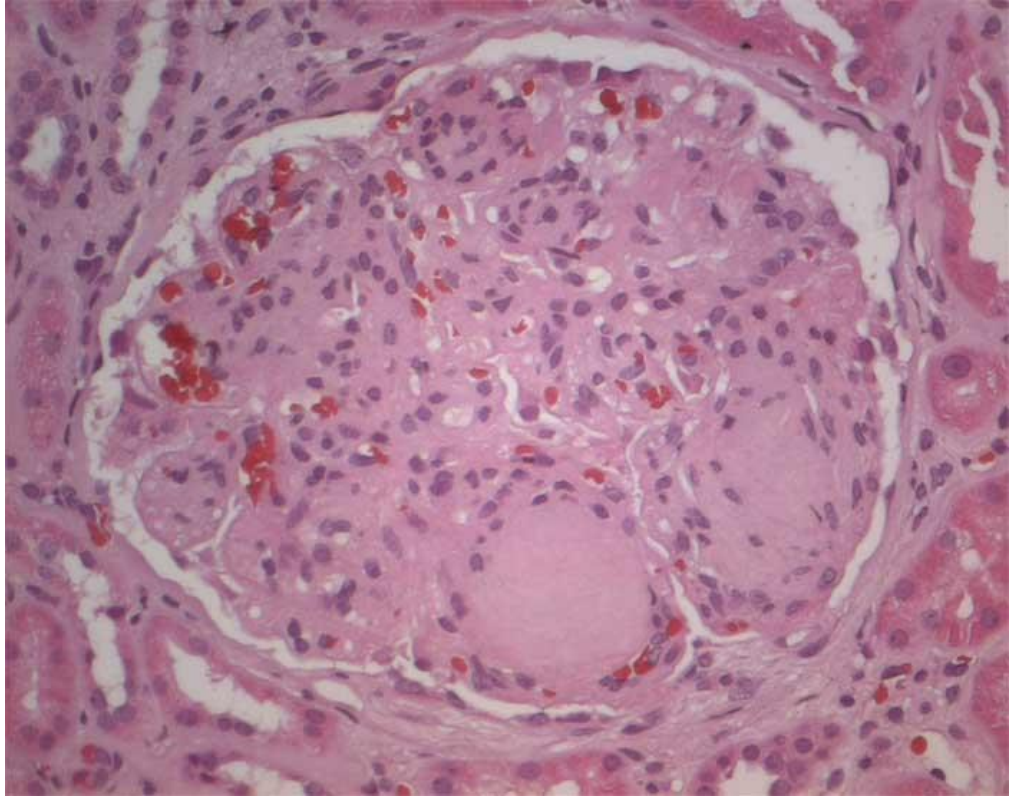
Thijs W. Cohen Tervaert,* Antien L. Mooyaart,* Kerstin Amann,† Arthur H. Cohen,‡ H. Terence Cook,§ Cinthia B. Drachenberg,‖ Franco Ferrario,¶ Agnes B. Fogo,** Mark Haas,‡ Emile de Heer,* Kensuke Joh,†† Laure H. Noël,‡‡ Jai Radhakrishnan,§§ Surya V. Seshan,‖‖ Ingeborg M. Bajema,* and Jan A. Bruijn,* on behalf of the Renal Pathology Society

J Am Soc Nephrol 21: 556–563, 2010.

Table 1. Glomerular classification of DN

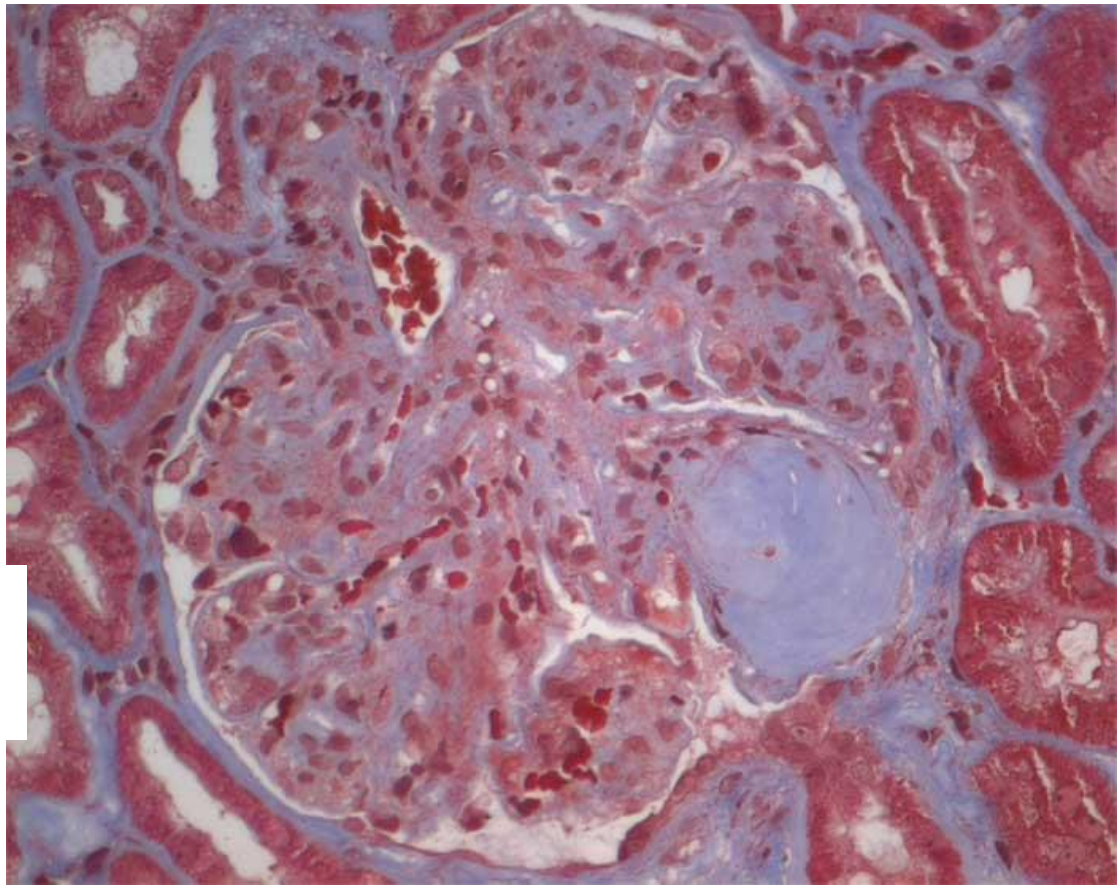
Class	Description	Inclusion Criteria
I	Mild or nonspecific LM changes and EM-proven GBM thickening	Biopsy does not meet any of the criteria mentioned below for class II, III, or IV GBM > 395 nm in female and >430 nm in male individuals 9 years of age and older[a]
IIa	Mild mesangial expansion	Biopsy does not meet criteria for class III or IV Mild mesangial expansion in >25% of the observed mesangium
IIb	Severe mesangial expansion	Biopsy does not meet criteria for class III or IV Severe mesangial expansion in >25% of the observed mesangium
III	Nodular sclerosis (Kimmelstiel–Wilson lesion)	Biopsy does not meet criteria for class IV At least one convincing Kimmelstiel–Wilson lesion
IV	Advanced diabetic glomerulosclerosis	Global glomerular sclerosis in >50% of glomeruli Lesions from classes I through III

Nefropatia Diabética Classe III



BR110660_HE, 20x2,0

BR110660_TM, 20x2,0



Sem IC

Vermelho congo negativo

SPECIAL ARTICLE www.jasn.org

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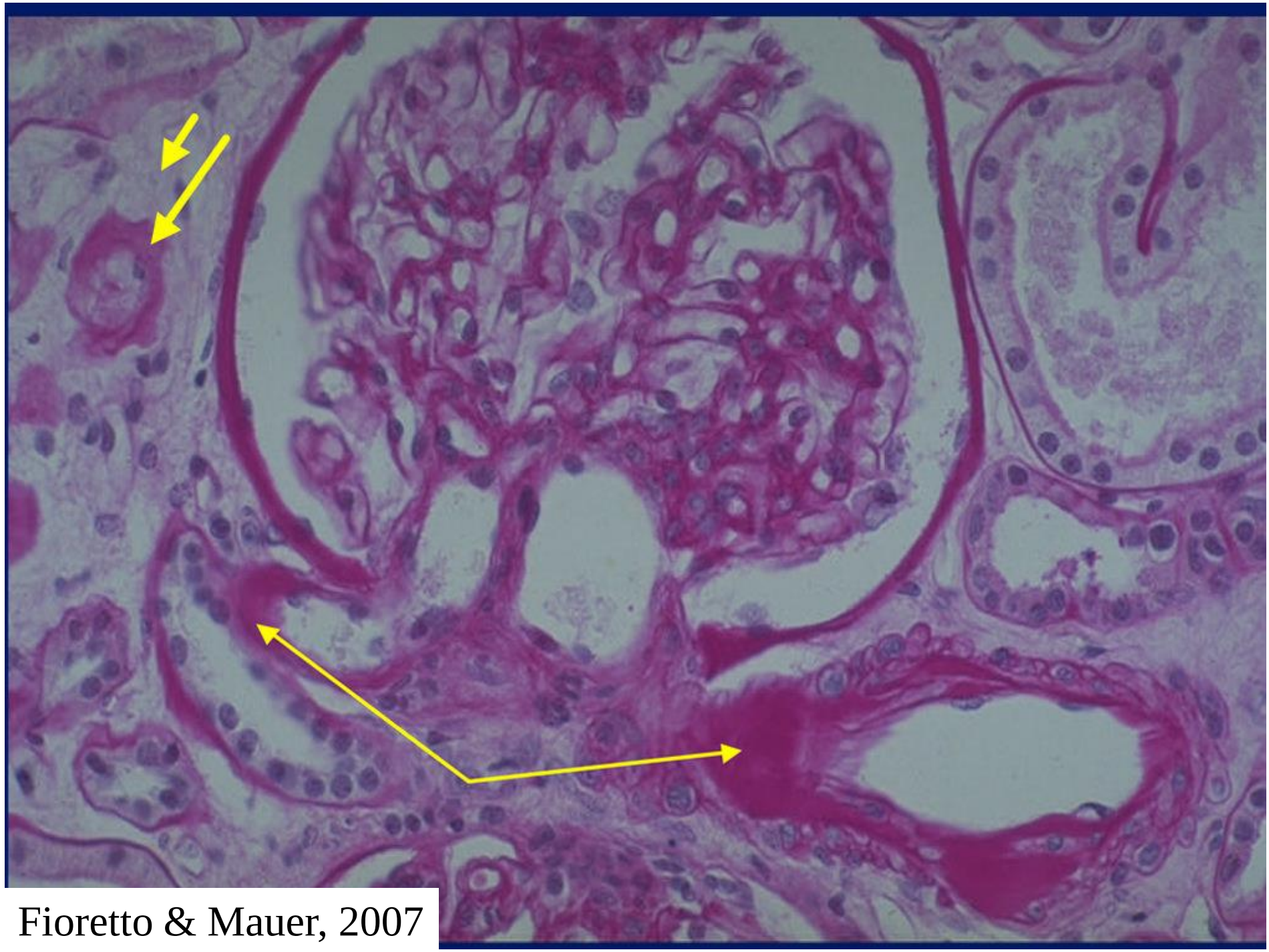
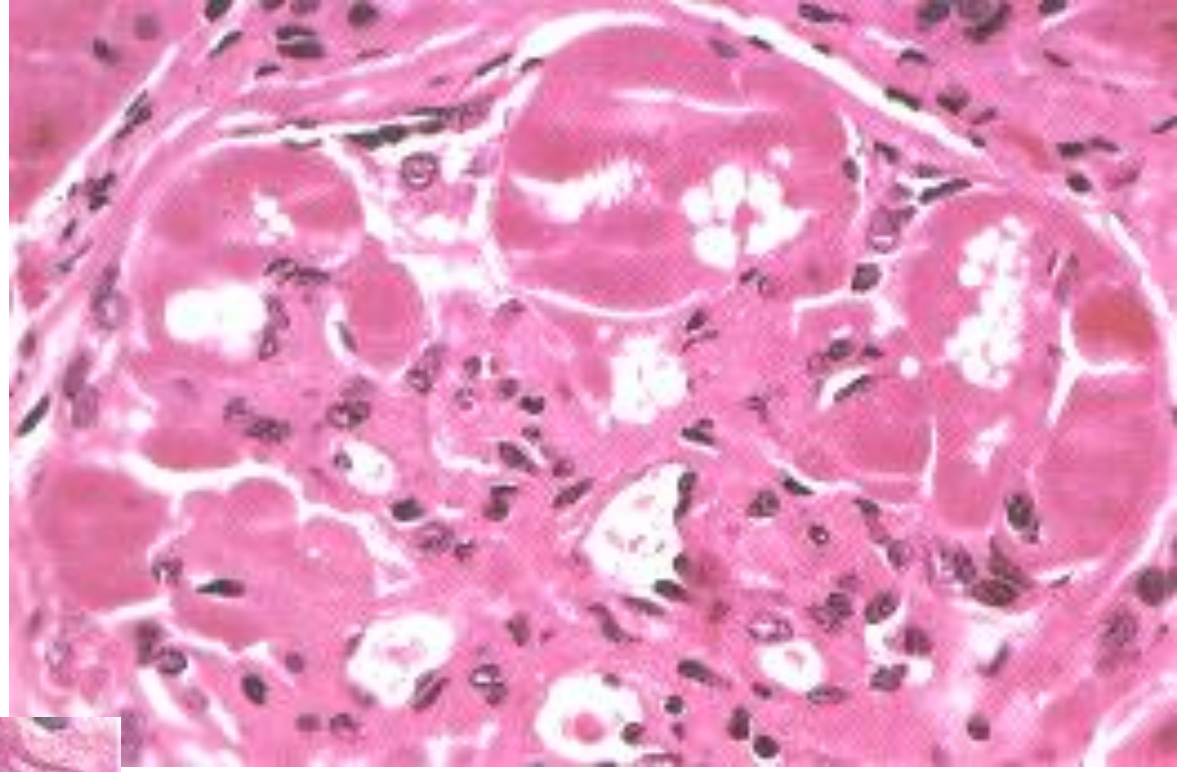


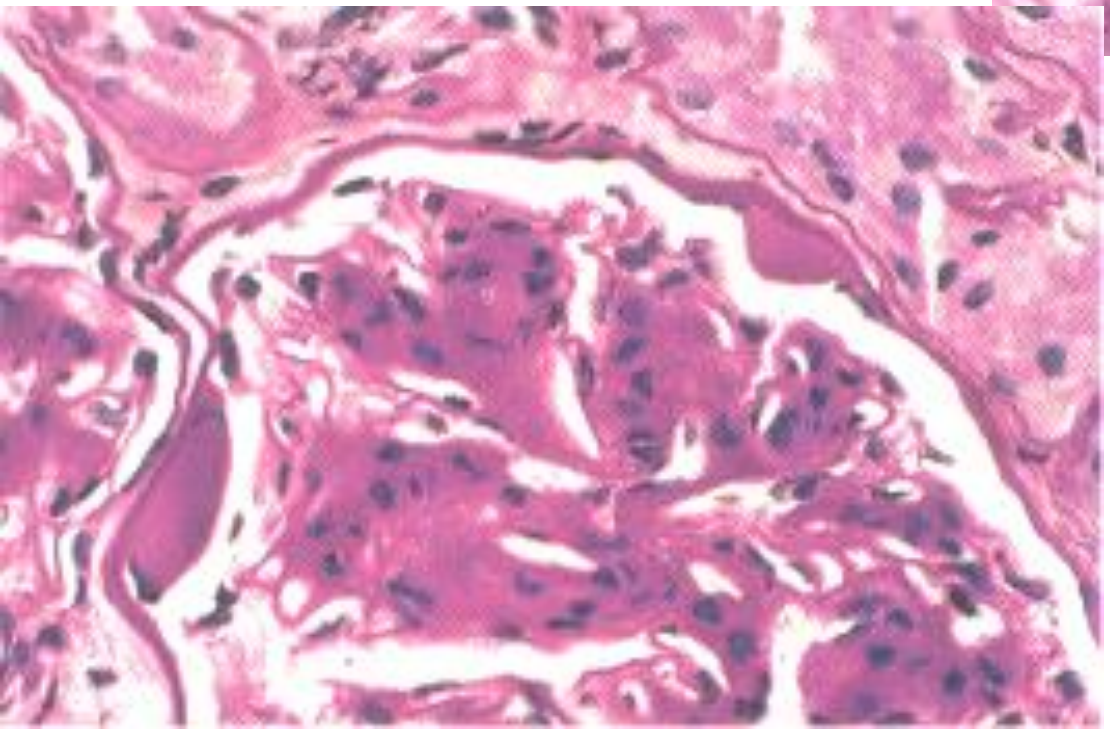
Fioretto & Mauer, 2007

Diabetes *Mellitus*

“capsular drop”

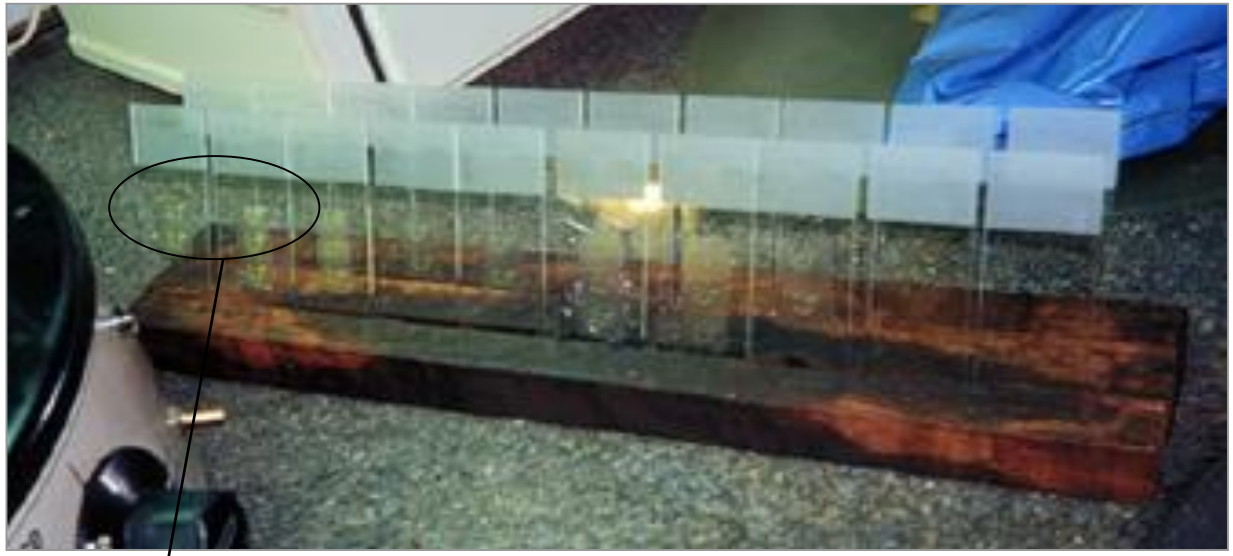
- fortemente sugestivo de diabetes
- hialinose entre MB e epitélio da cápsula de Bowman
- prata negativo



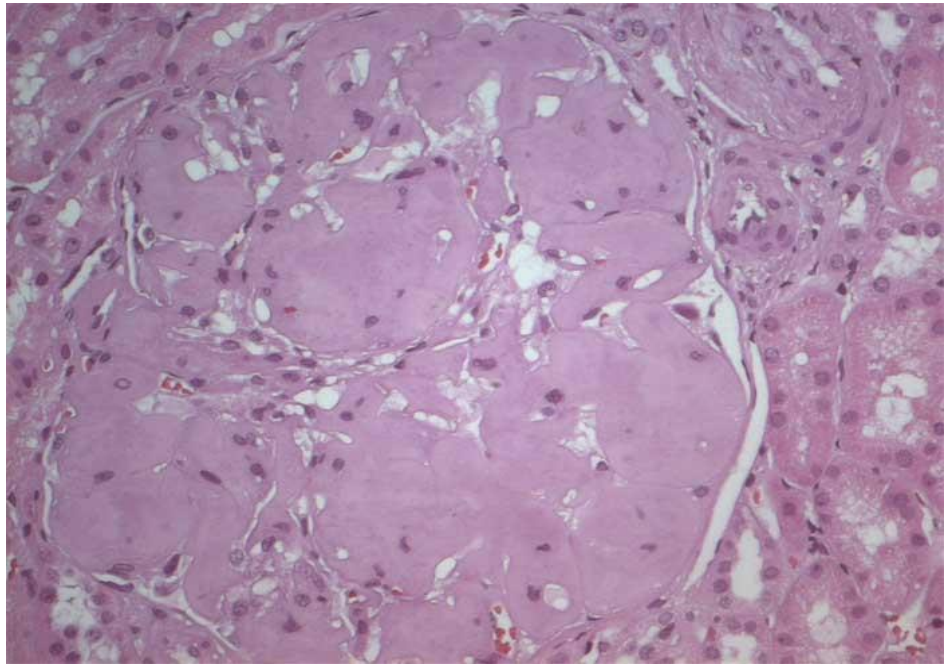


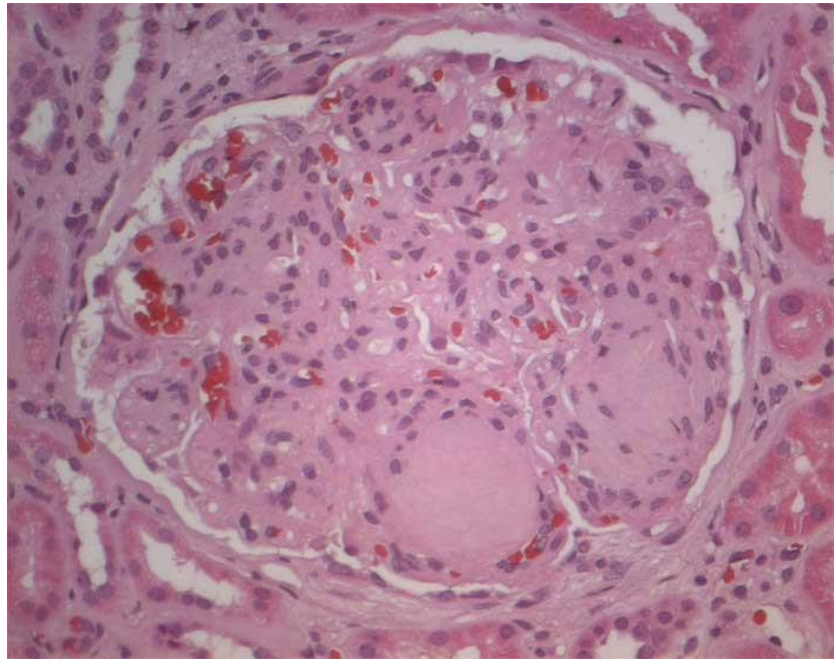
“fibrin cap”

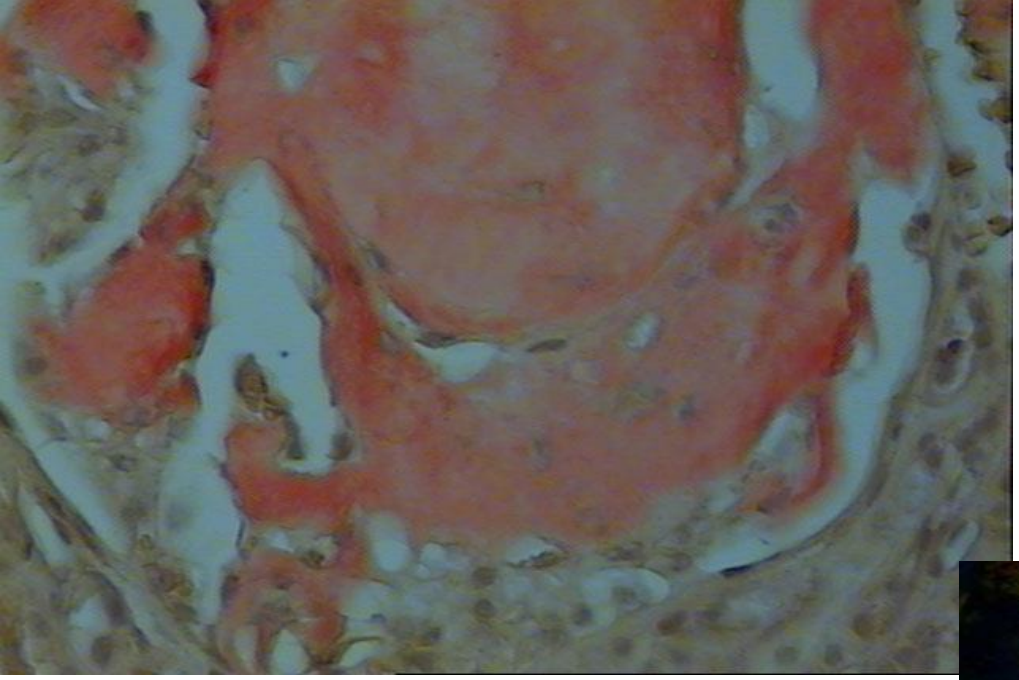
- entre MBG e endotélio
- proteinas e lipideo



10 µm - Vermelho Congo



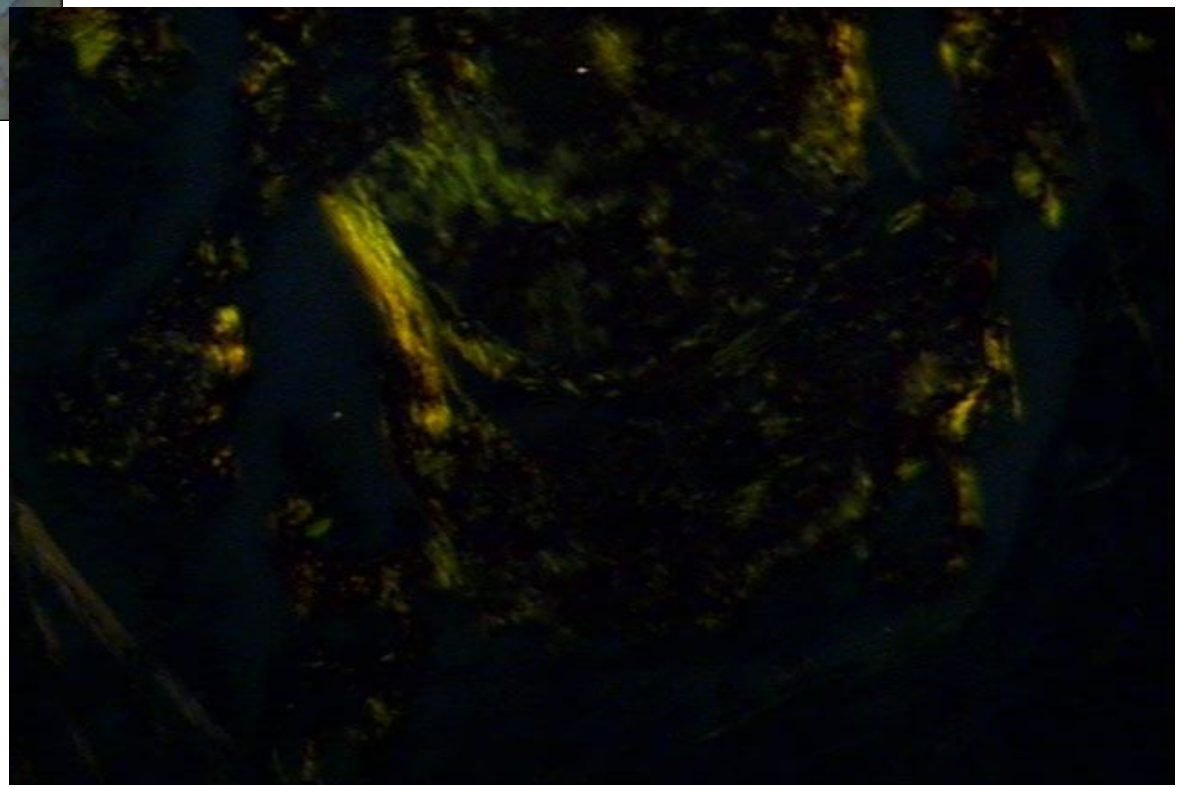




Vermelho Congo

Luz comum

Amiloidose



Luz Polarizada

β-fibrilose

Amiloidose

8-12nm

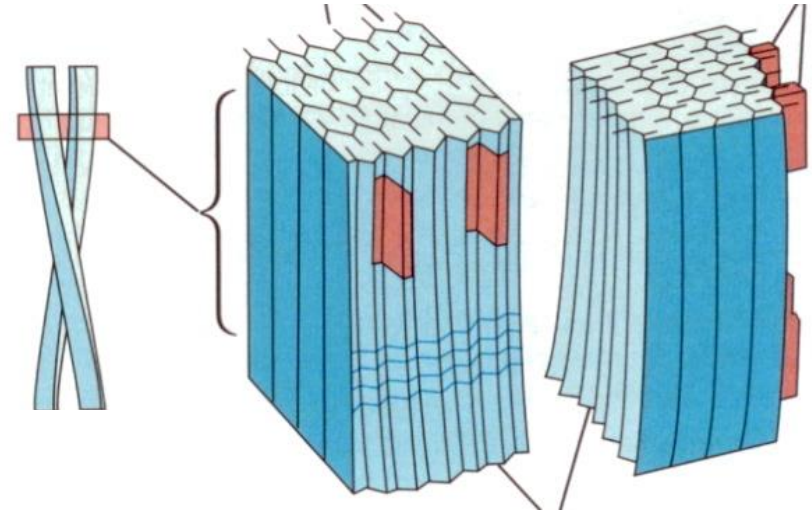
EM – 30.000X

Amiloidose

produção

degradação

- Doenças Inflamatórias (com ou sem infecção)
- Alt Imune
- Hereditárias (mutação)



Amiloidose Renal – Amiloidose sistêmica

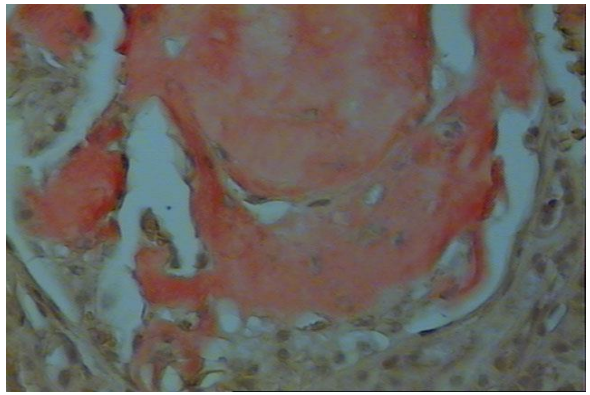
> 30
Proteínas amiloidogênicas
São conhecidas

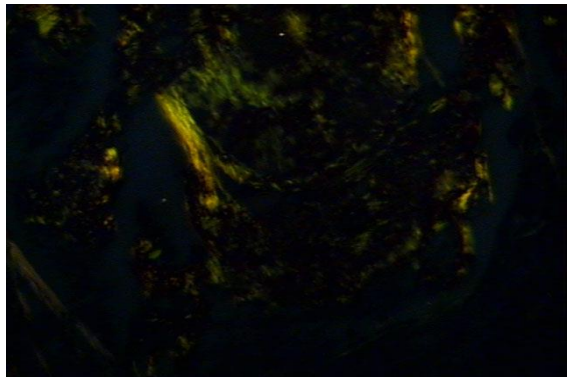
AL	**light chain Ig**
AA	**Serum Protein A**
Aβ2M	**β2 microglobulin**
ALECT2	**Chemotactic Factor Leucocyte 2**
AFib	**A alfa-chain fibrinogen**
ATTR	**transtiretin**
AApoAI	**Apolipoprotein AI**
AApoAII	**Apolipoprotein AII**
ALys	**lysozyme**
AGel	**Gelsolin**
ACys	**Cystatin C**

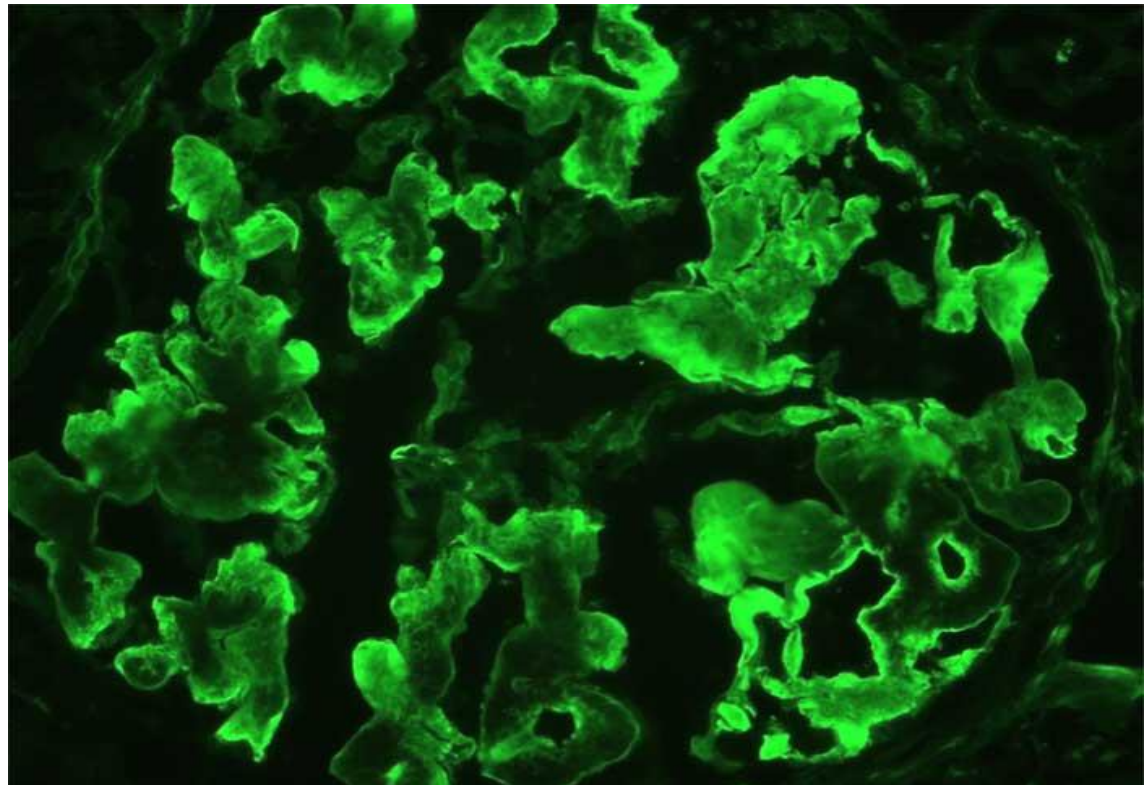
Picken MM - Arch Pathol Lab Med 134:545-551;2010 - Herrera GA et al., Arch Pathol Lab Med 134:512-531;2010 - Matsumoto S et al. Amyloid. Mar;24(sup1):97-98;2017

BR100550

Glu526Val
Mutação
um alelo







Amyloid
The Journal of Protein Folding Disorders

http://informahealthcare.com/amy
ISSN: 1350-6129 (print), 1744-2818 (electronic)

Amyloid, Early Online: 1–4

 DOI: 10.3109/13506129.2012.763029

informa
healthcare

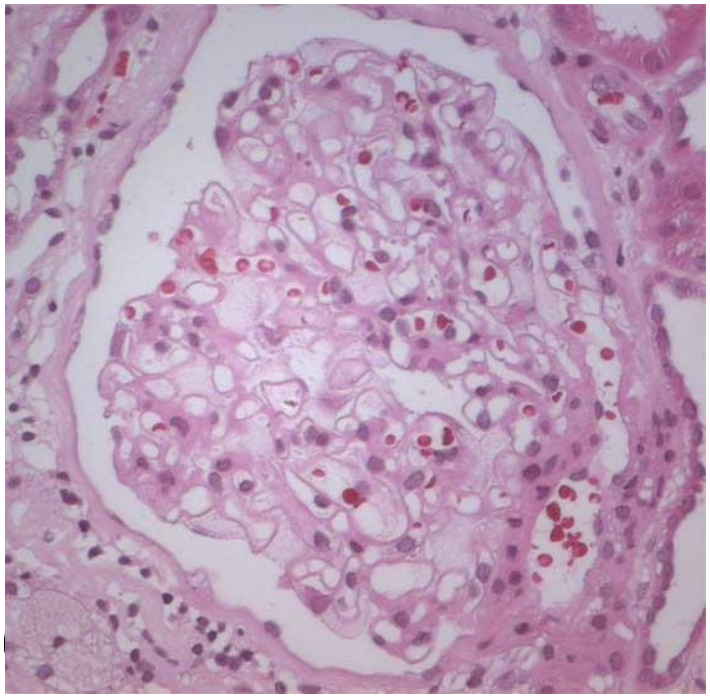
CASE REPORT

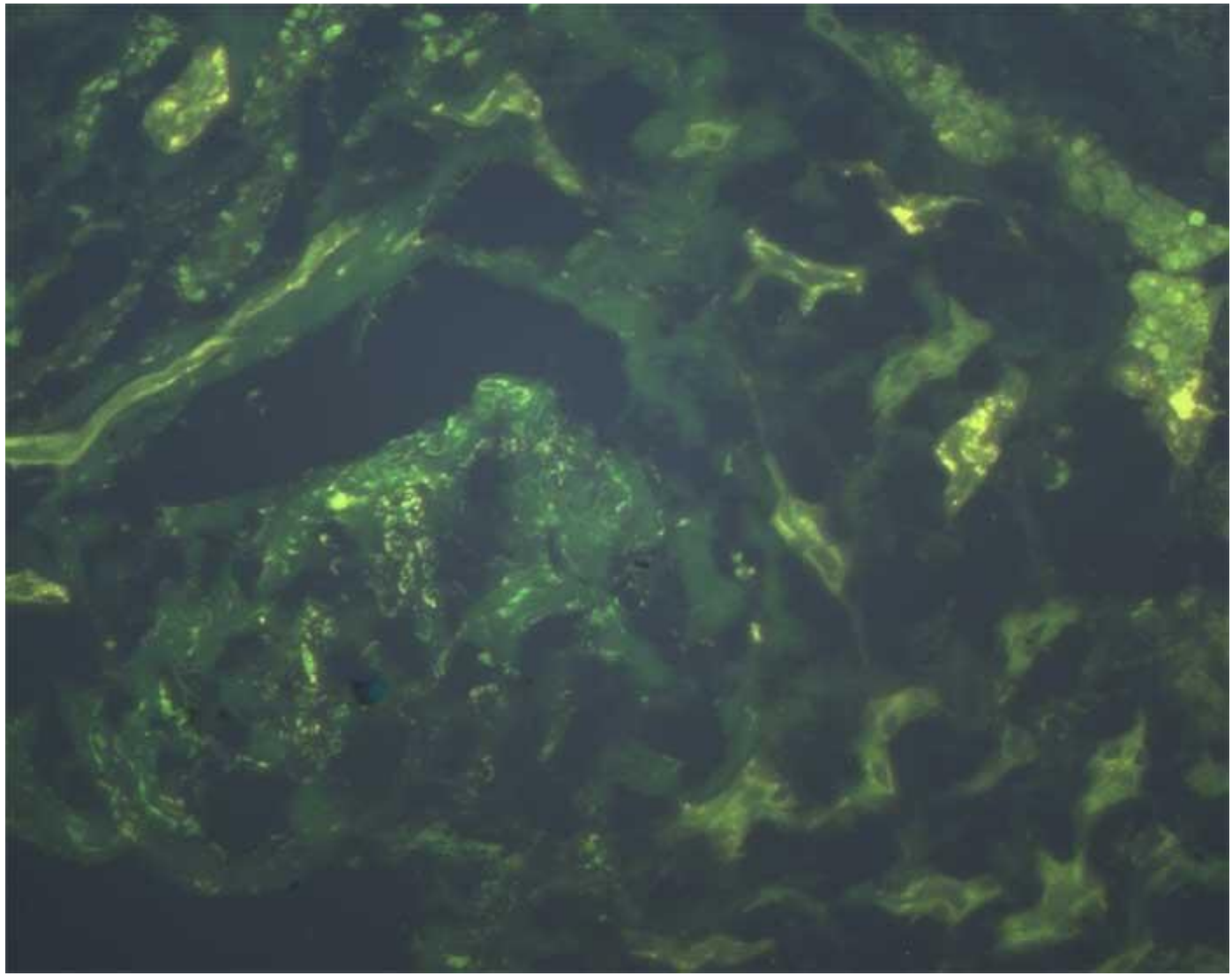
Fibrinogen A alpha-chain amyloidosis: report of the first case in Latin America

Juliana Reis Machado[1], Marcos Vinícius da Silva[2], Precil Diego Miranda de Menezes Neves[1], Flavia Aparecida de Oliveira[3], Rosana Rosa Miranda Corrêa[1], Willians Vinícius Dutra Rodrigues[4], Merril Benson[5], and Marlene Antônia dos Reis[1]

Pesquisa de Imunocomplexos - IC

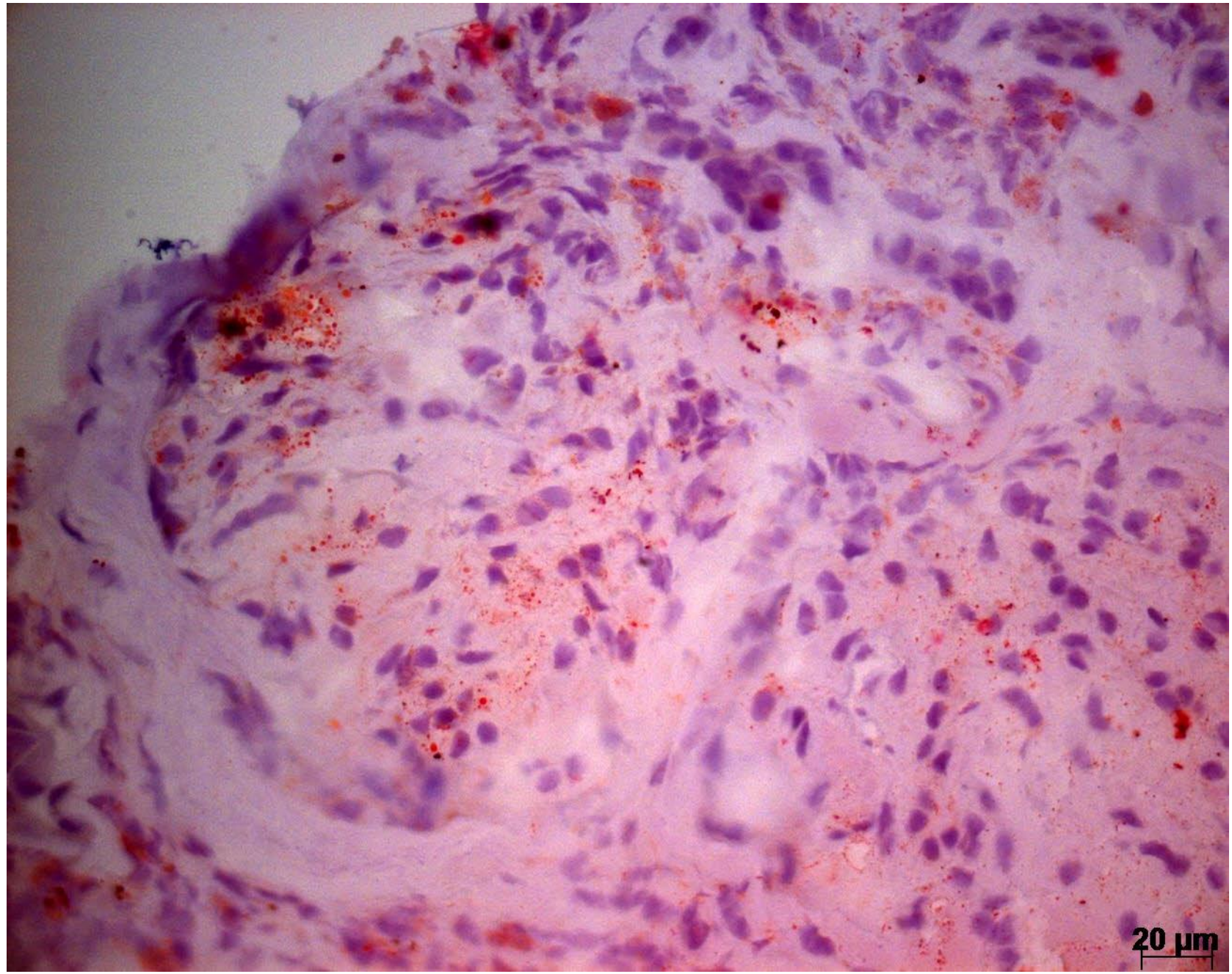
Fluorescência natural
Controle negativo





Lipídeos

Sudan

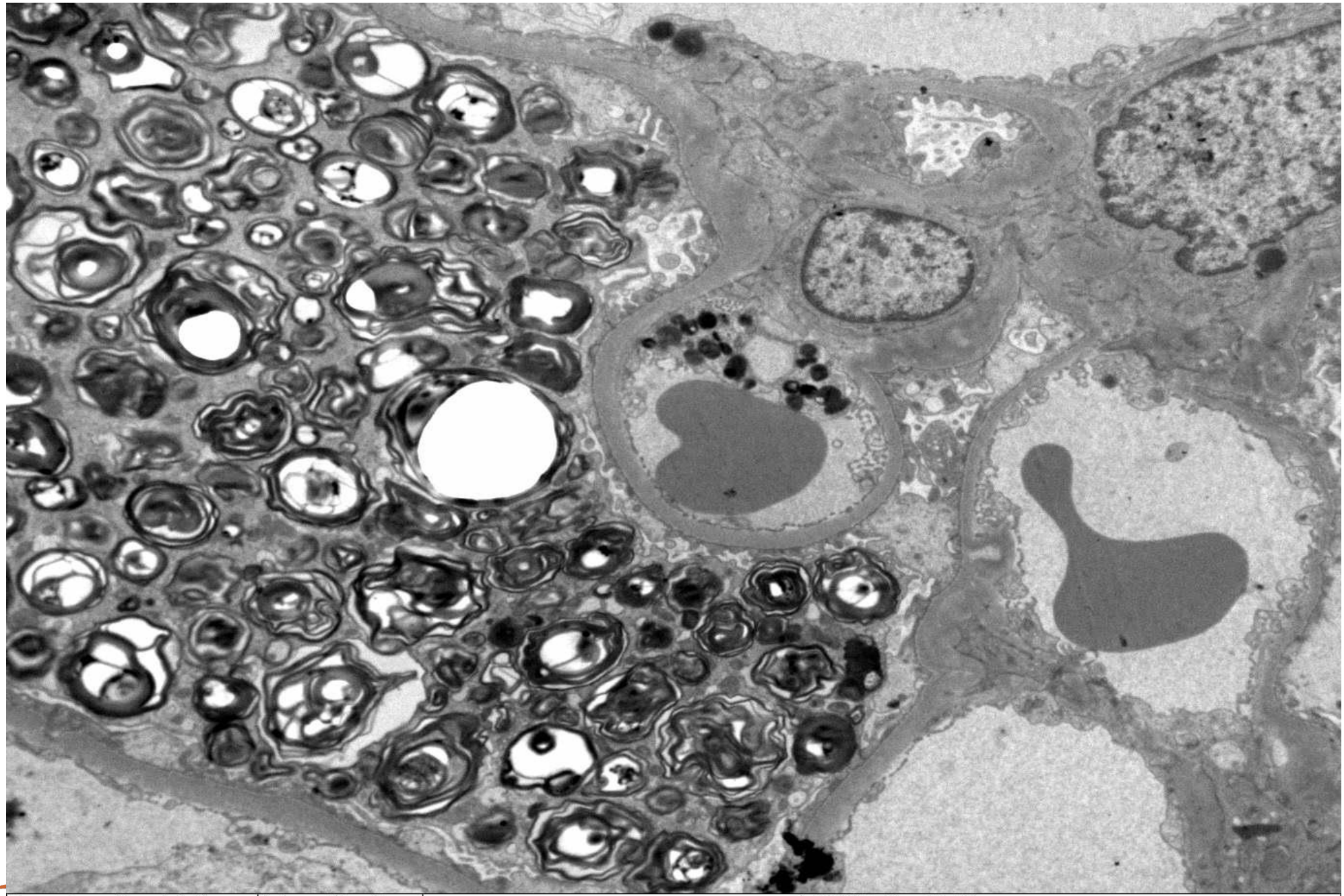


Doenças de Depósitos em Lisossomos

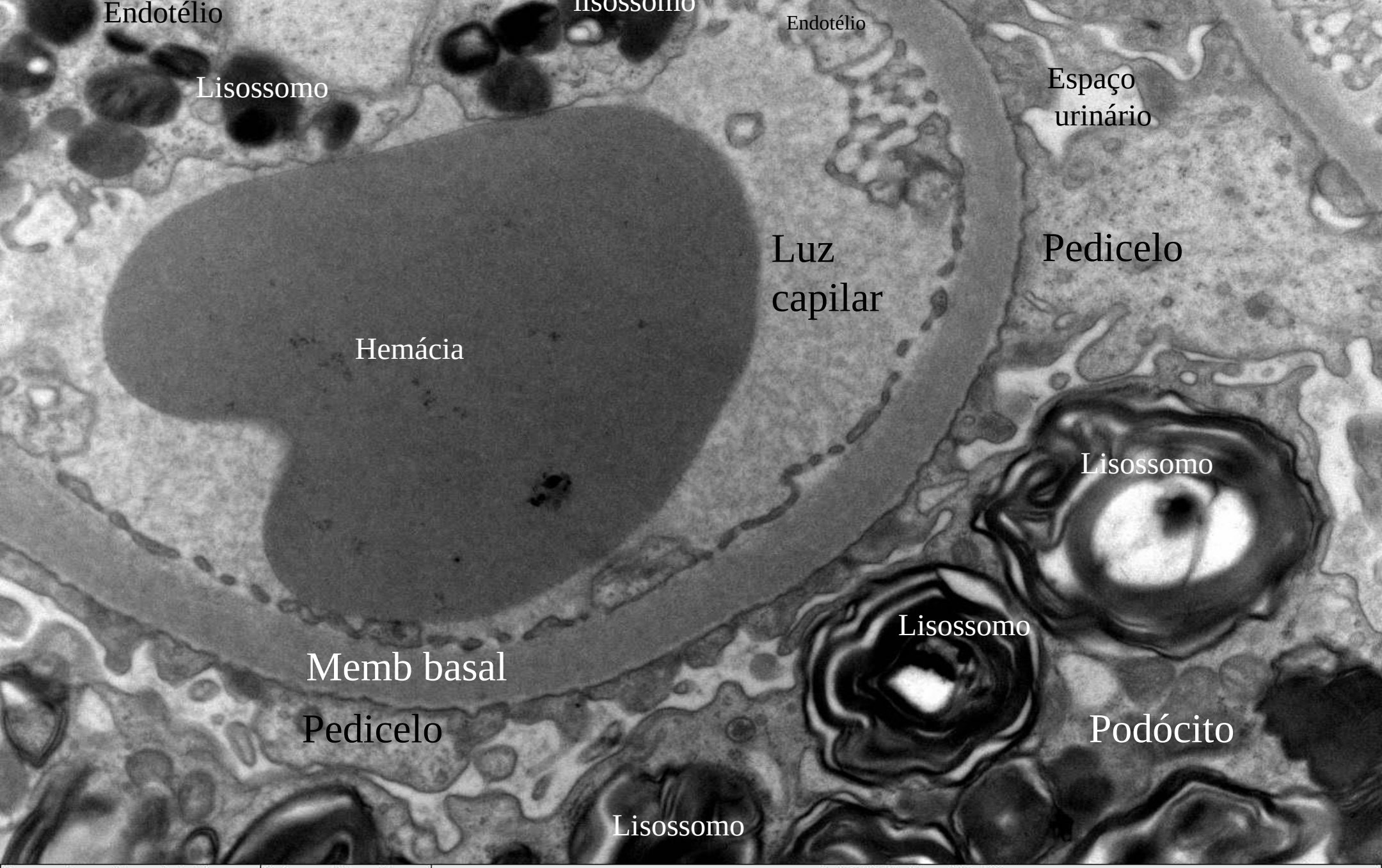
(deficiência enzimática)

- Doença de Gaucher - glicosilceramidase
- Doença de Fabry – alfa-galactosidase
- Doença de Niemann-Pick - esfingomielinase
- Doença de Tay-Sachs - hexosaminase A

Finn LS. Renal disease caused by inborn errors of metabolism, storage diseases ...; Heptinstall's, 223-1279; 2014



Accelerating Voltage	Magnification	Br090472_1819
80000 kV	3000 x	5 µm

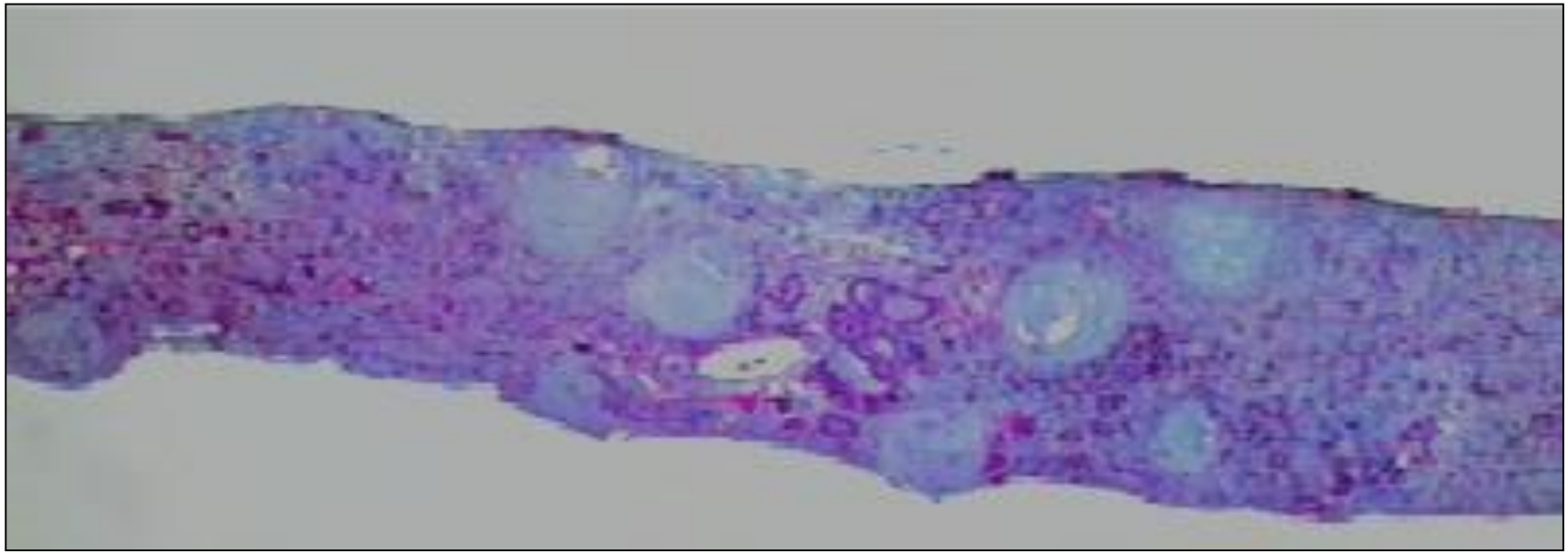


Accelerating Voltage	Magnification	Br090472_1819
80000 kV	12000 x	2 µm

Doença Renal Crônica
Glomerulopatia crônica

≥90% glomérulos com esclerose global

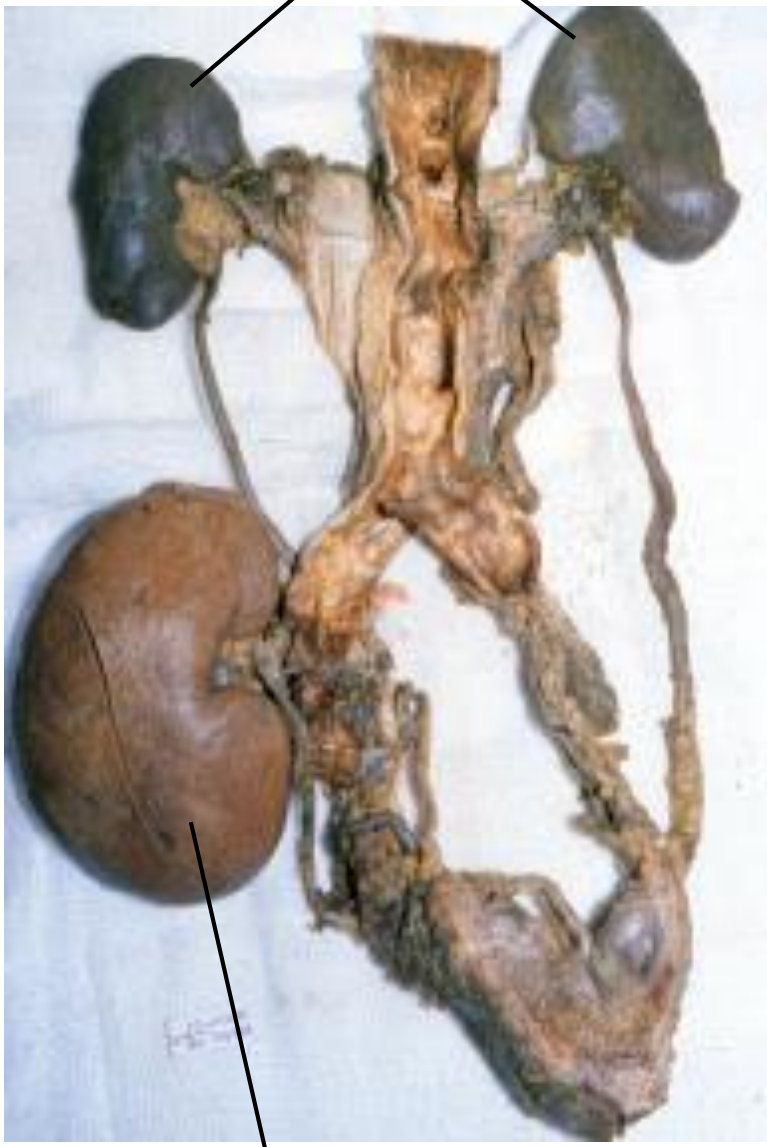
Fibrose intersticial e atrofia tubular



Transplante renal ao longo do tempo

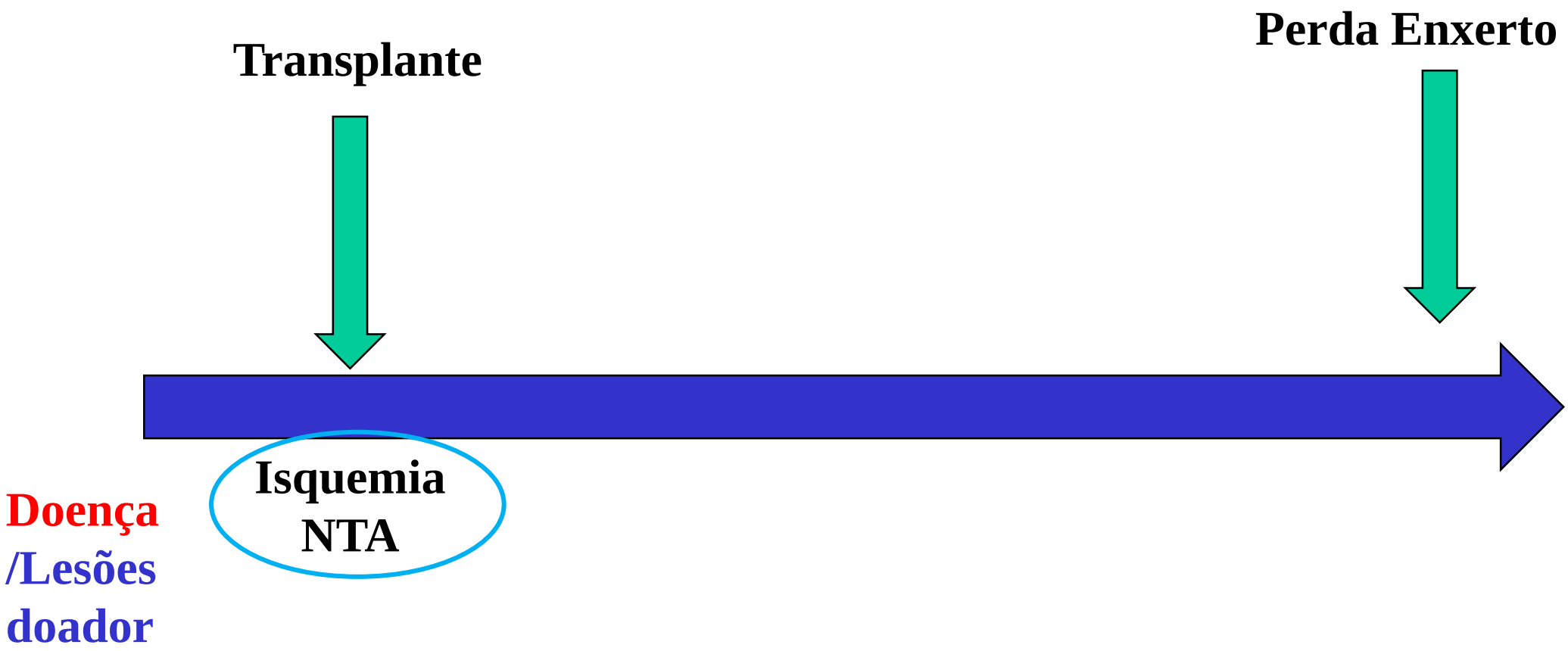
Transplante

Perda Enxerto

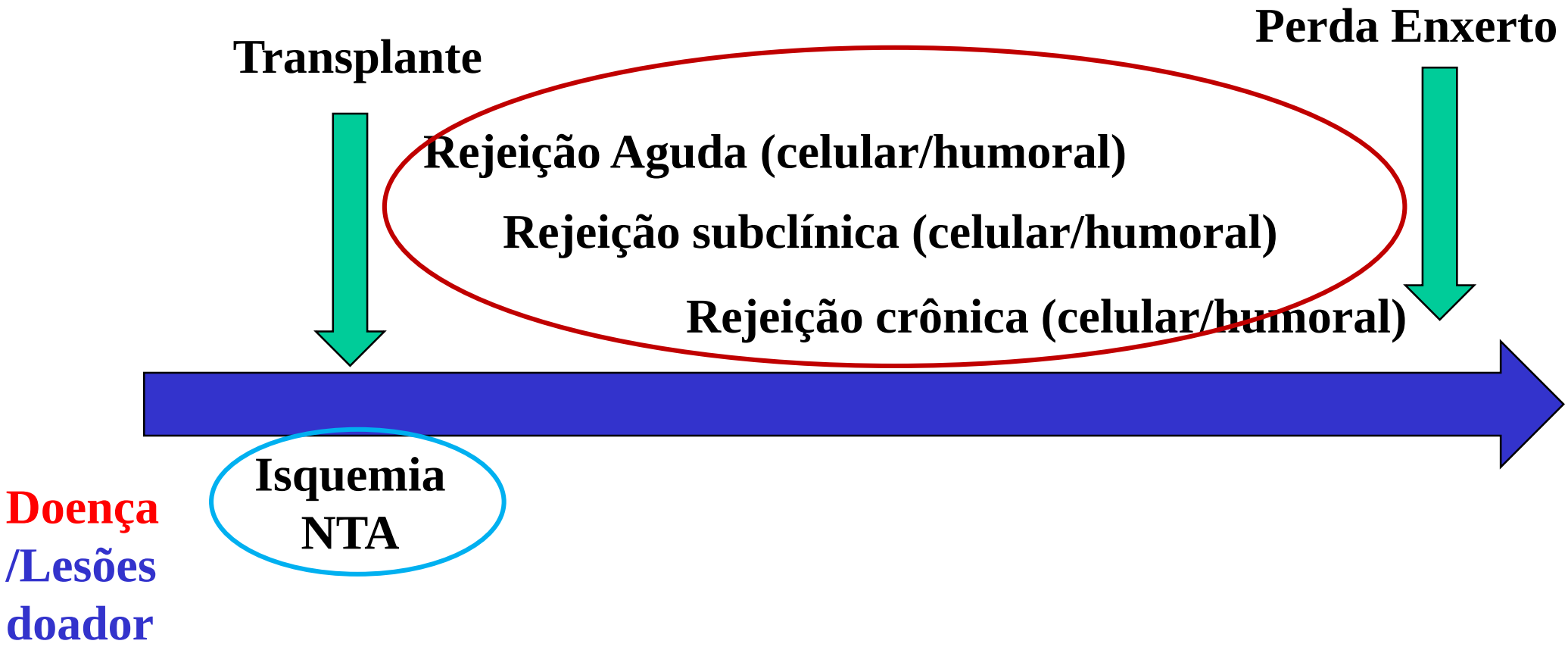


Lesões provenientes do doador

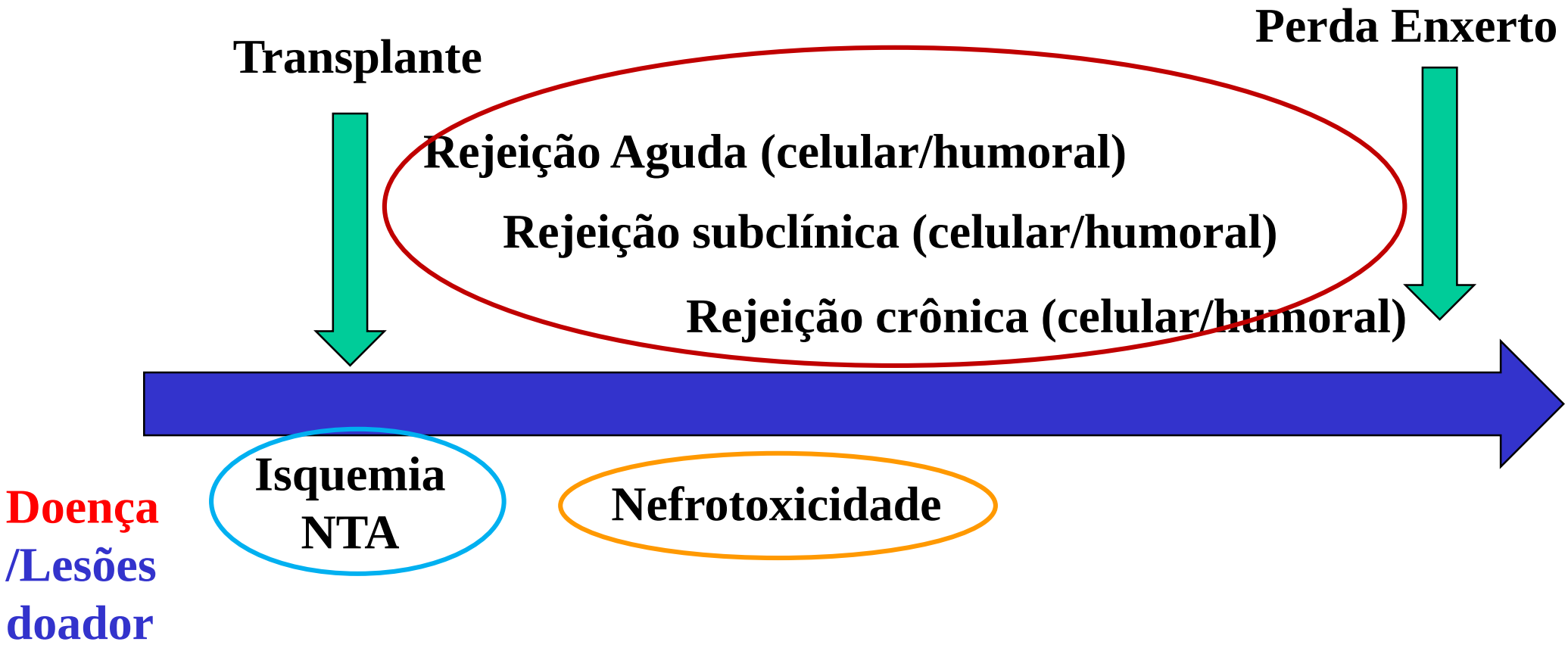
Transplante renal ao longo do tempo



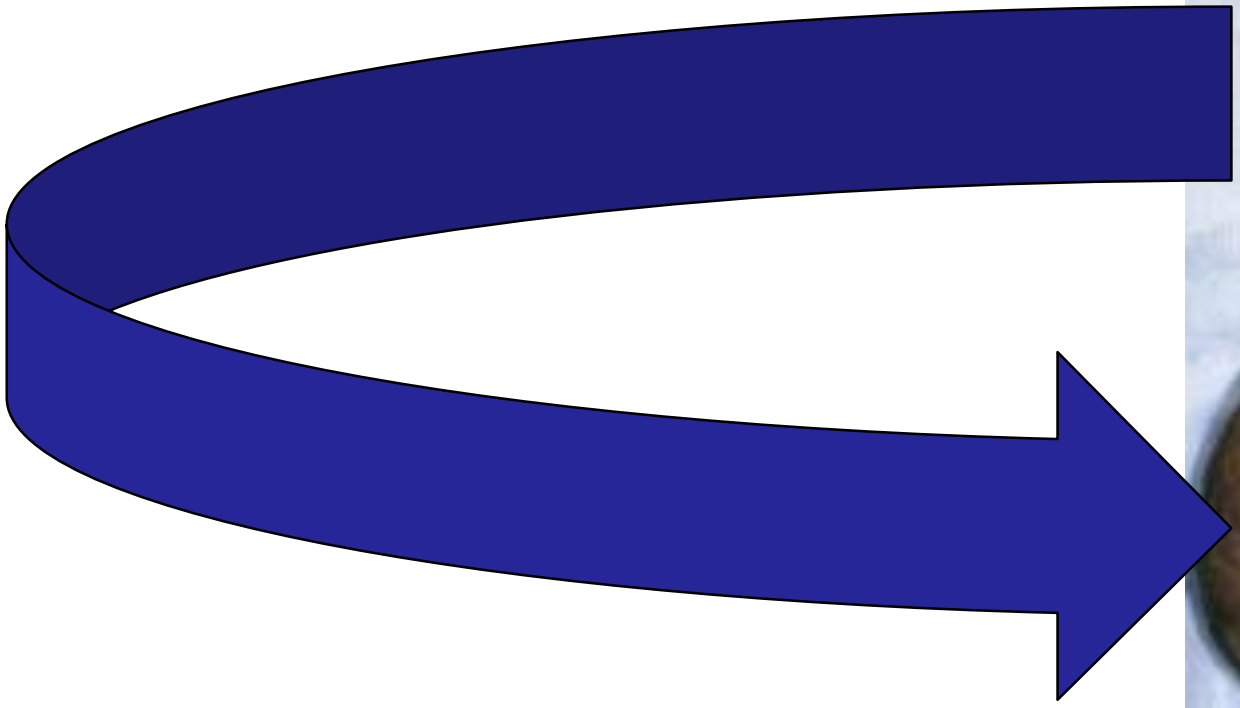
Transplante renal ao longo do tempo

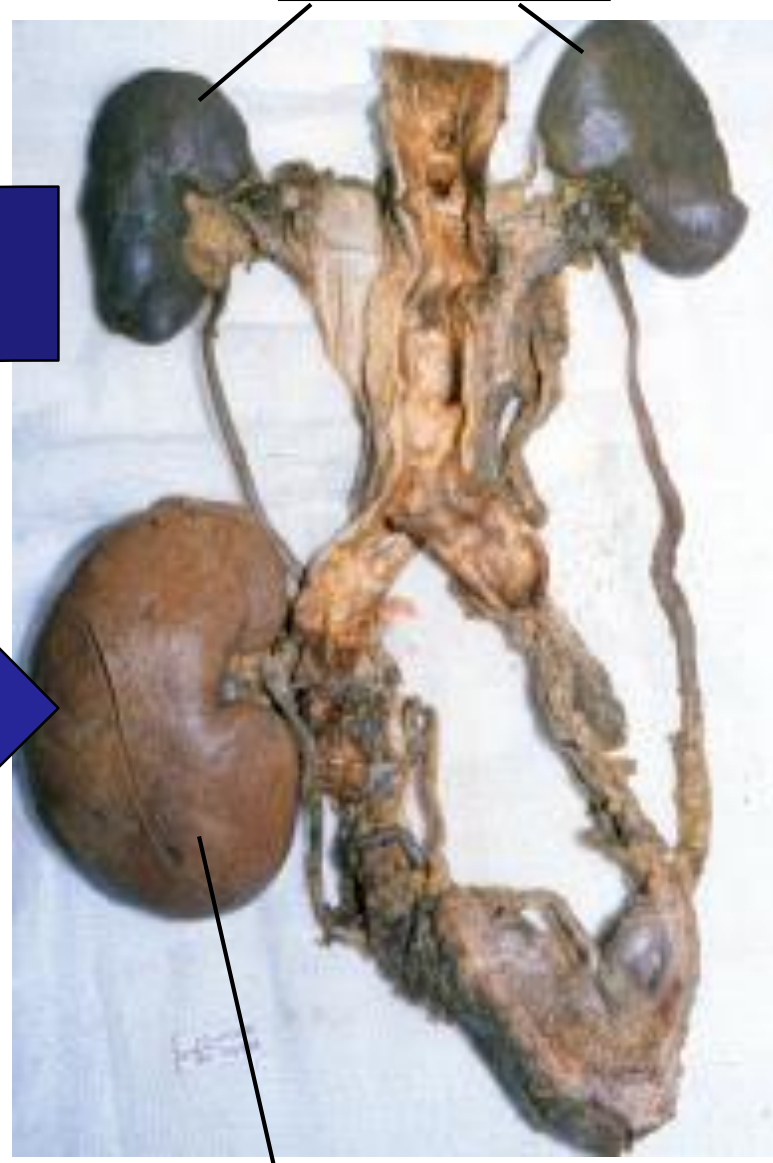


Transplante renal ao longo do tempo



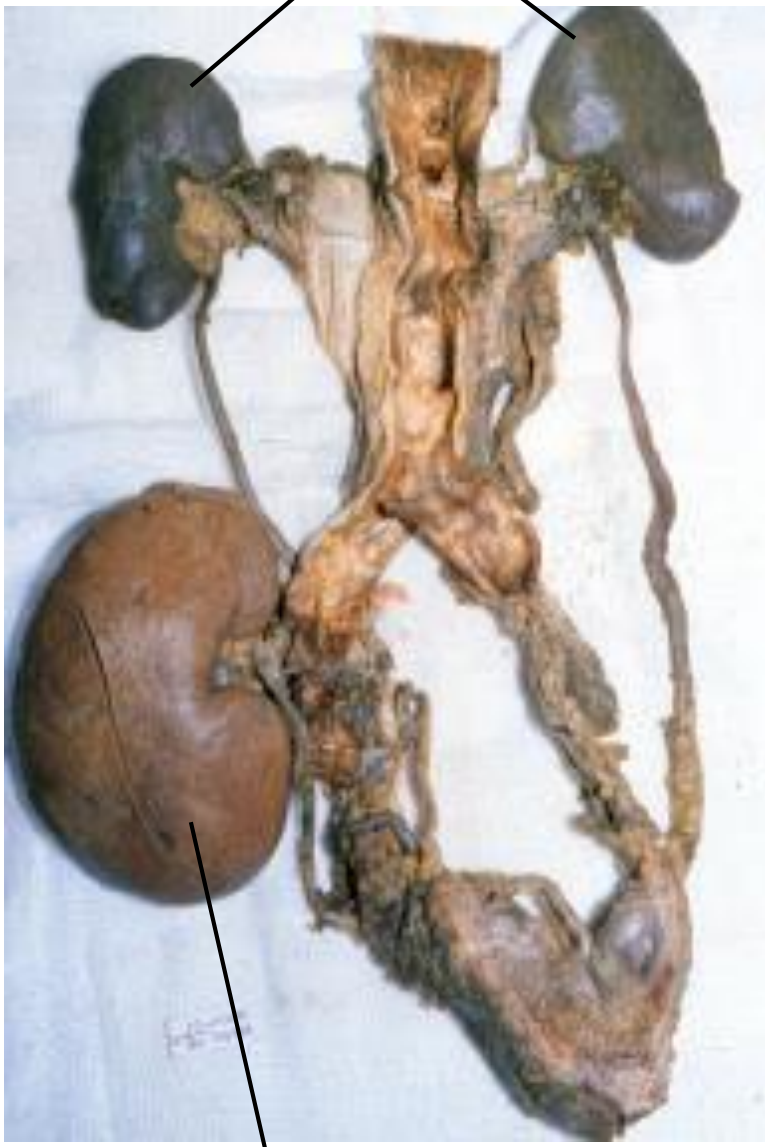
Doença recorrente





doença do rim nativo
aparece no transplante

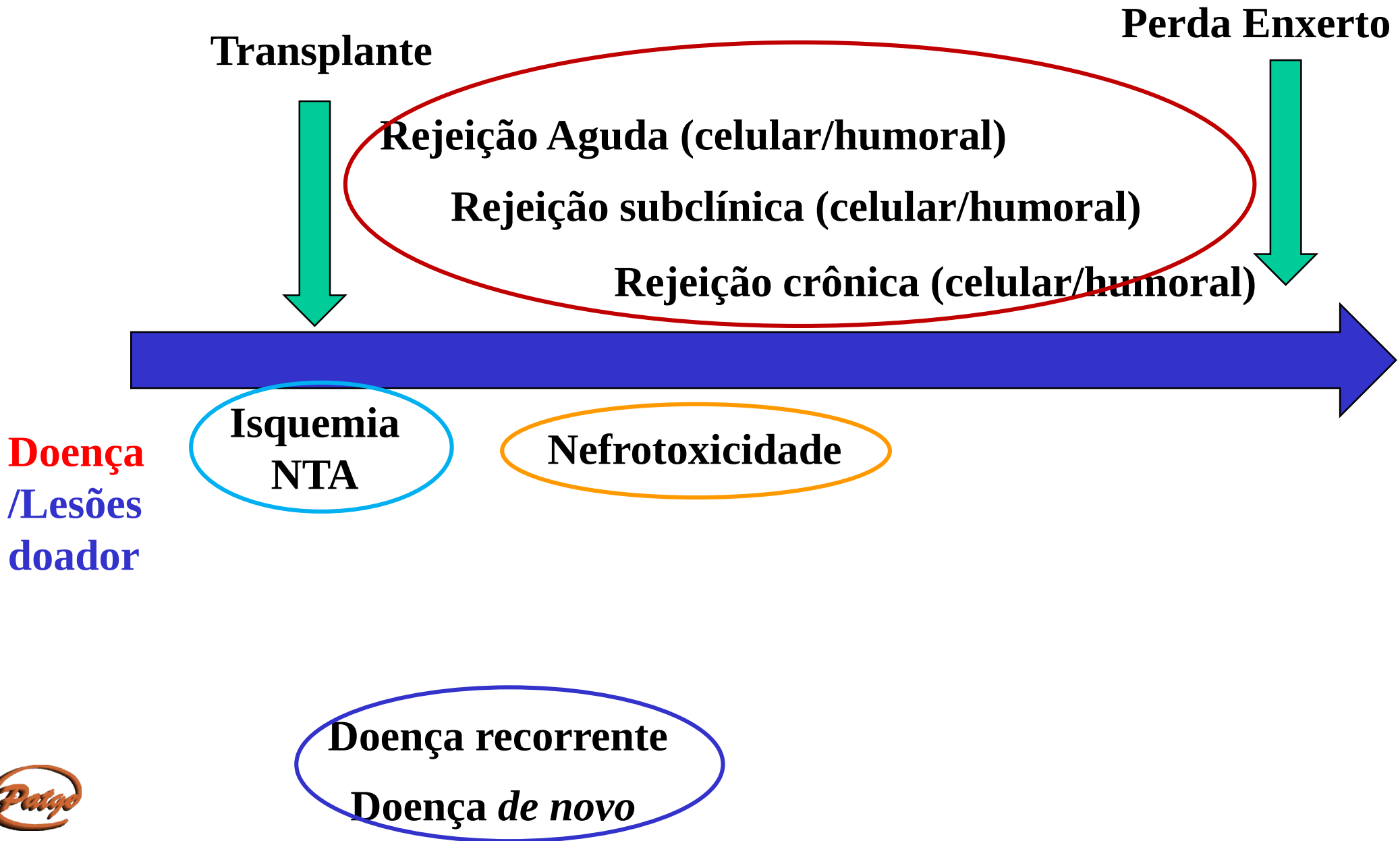
GESF
DDD



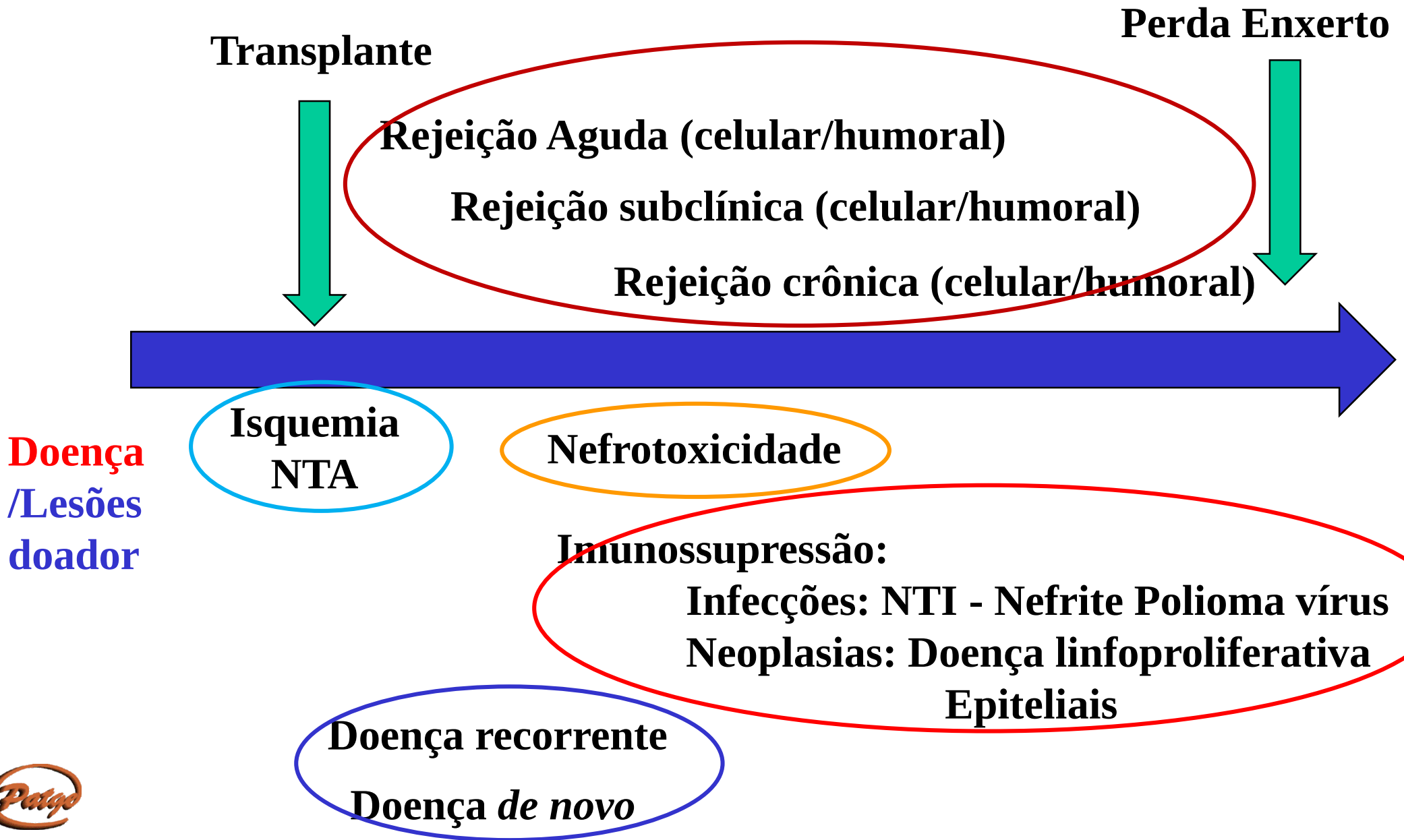
Doença *de novo*

doença no transplante diferente da doença no rim nativo

Transplante renal ao longo do tempo



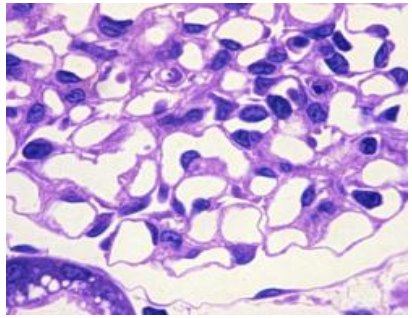
Transplante renal ao longo do tempo



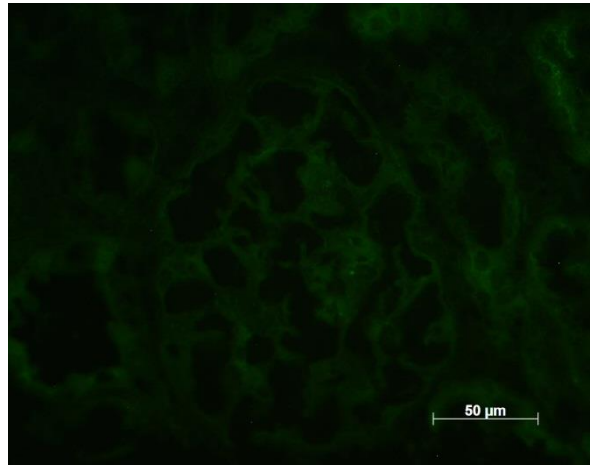
Biópsia Renal

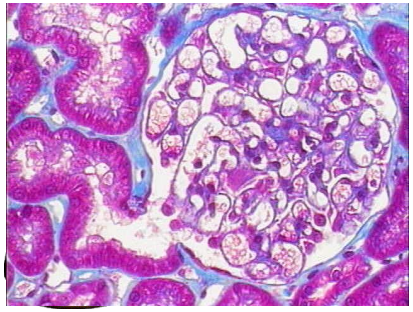
ML-10 gl – paraformaldeido
Formaldeído / Bouin / Metacarn

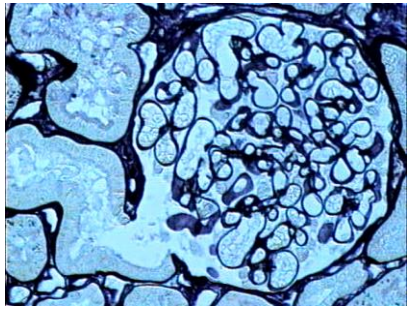
IF-10gl – IC/compl
A fresco / Meio de transporte



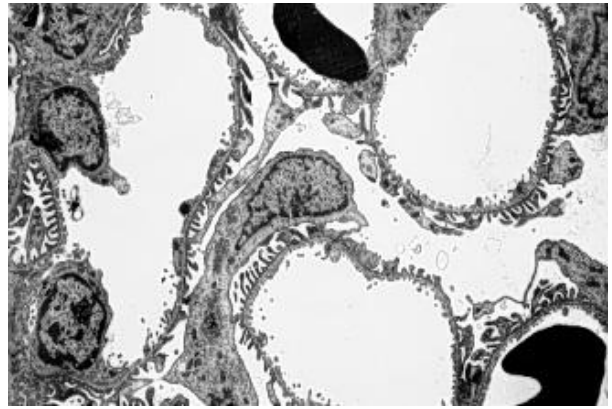
- IgA
- IgG
- IgM
- *Kappa*
- *Lambda*
- Fibrinogênio
- C1q
- C3

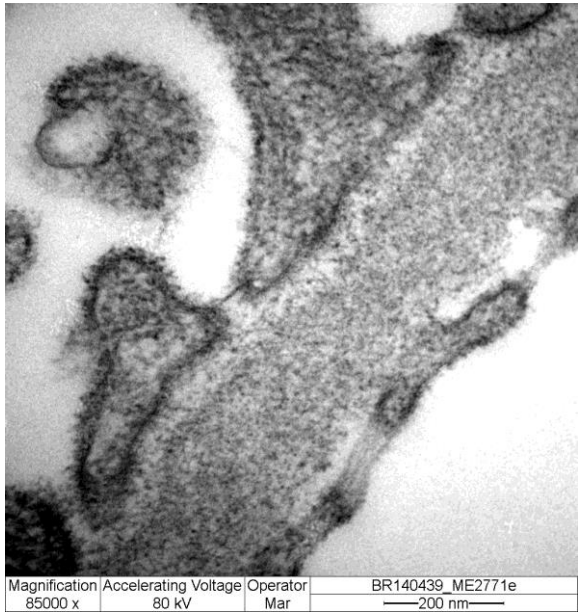




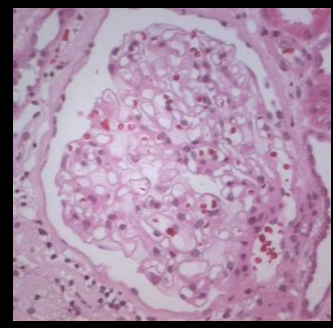


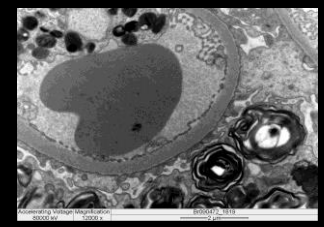
ME-2gl – Glutaraldeído
/ Karnovsky + Vermelho rutênio

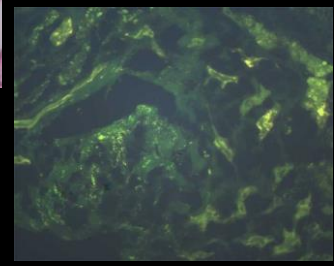


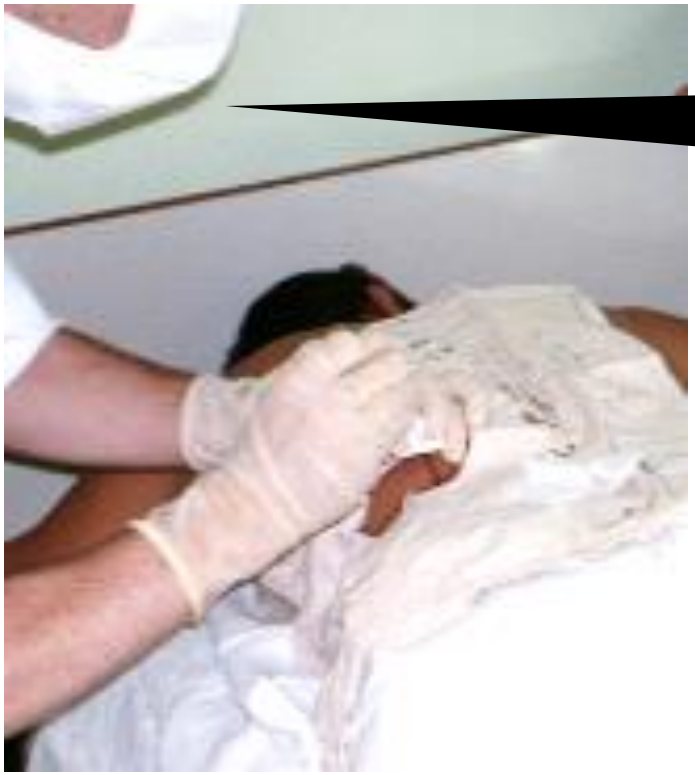


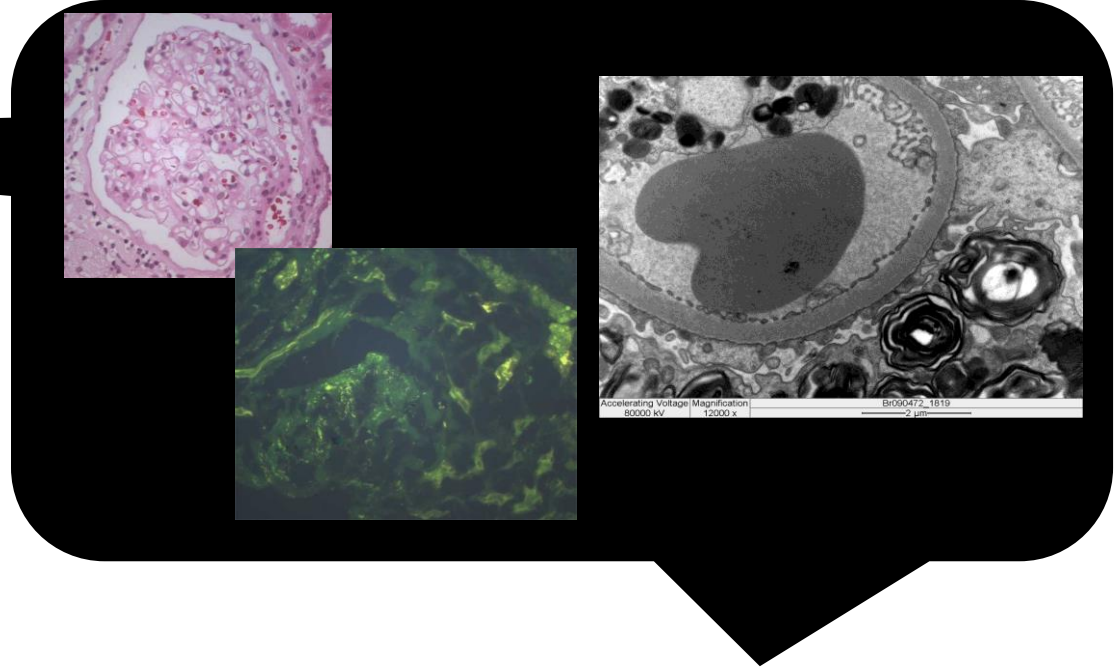
Informações Clínicas










Accelerating Voltage
80000 kV
Magnification
12000 x
Br090472_1819
2 µm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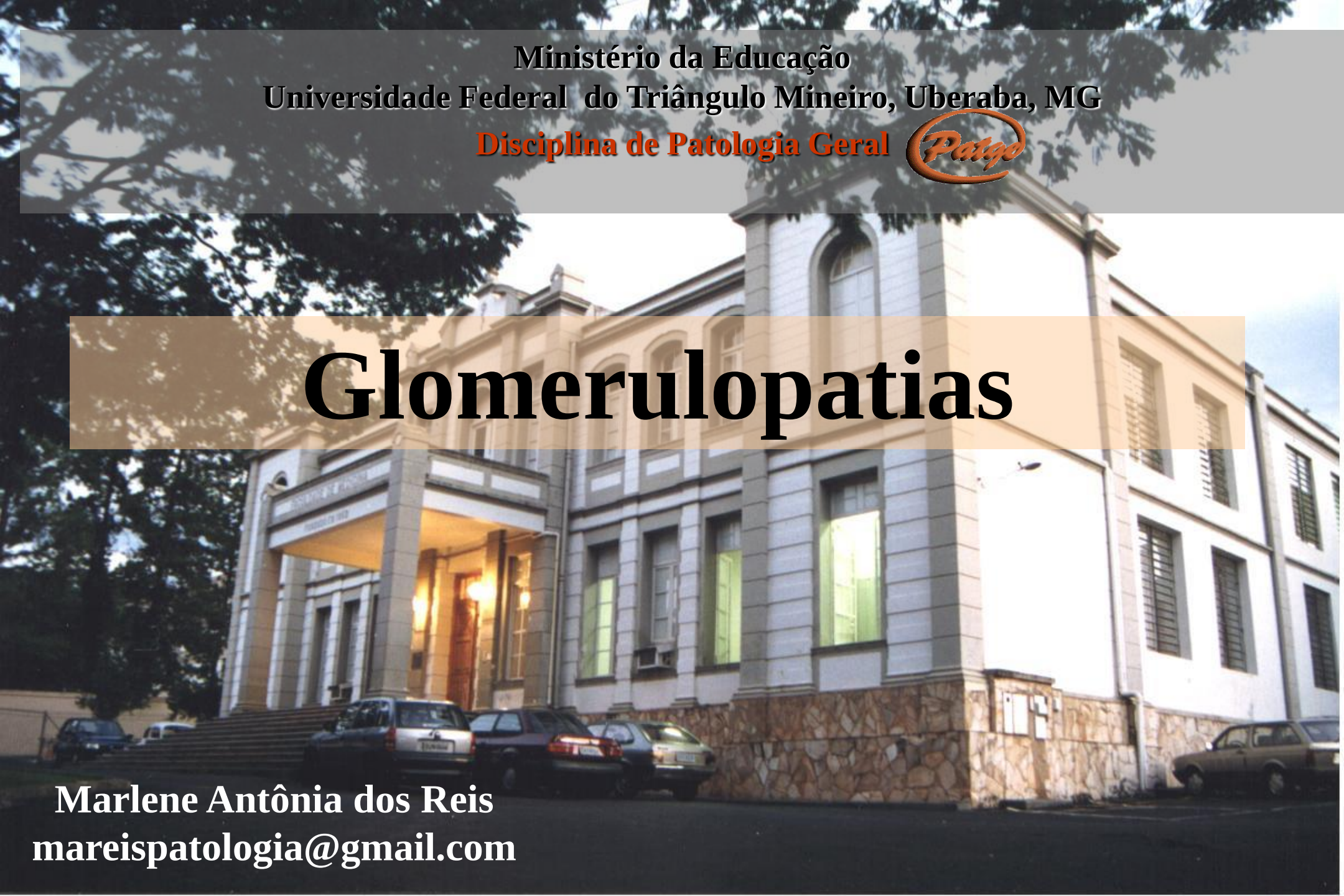
Glomerulopatias



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