



DANNO RENALE ACUTO (AKI)



Carmelo
Libetta

Capitolo 5°

<u>Termine</u>	<u>Autore</u>	<u>Anno</u>
Iscuria renale	William Heberden	1802
Malattia di Bright	Richard Bright	1827
Nefrite di guerra	-	1918
Sindrome da schiacciamento	Bywaters e Beall	1945
Insufficienza Renale Acuta (Acute Renal Failure)	Homer Smith	1951
Danno Renale Acuto	ADQI group	2002

**Acute Dialysis Quality Initiative (ADQI) group*



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Terminologia

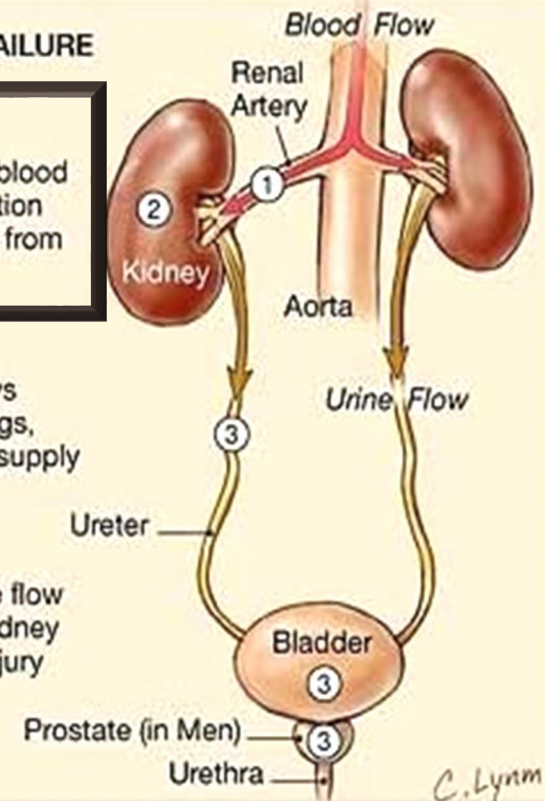


ACUTE KIDNEY INJURY

Acute renal failure

CAUSES OF ACUTE RENAL FAILURE

- ① **Prerenal**
Sudden and severe drop in blood pressure (shock) or interruption of blood flow to the kidneys from severe injury or illness
- ② **Intrarenal**
Direct damage to the kidneys by inflammation, toxins, drugs, infection, or reduced blood supply
- ③ **Postrenal**
Sudden obstruction of urine flow due to enlarged prostate, kidney stones, bladder tumor, or injury

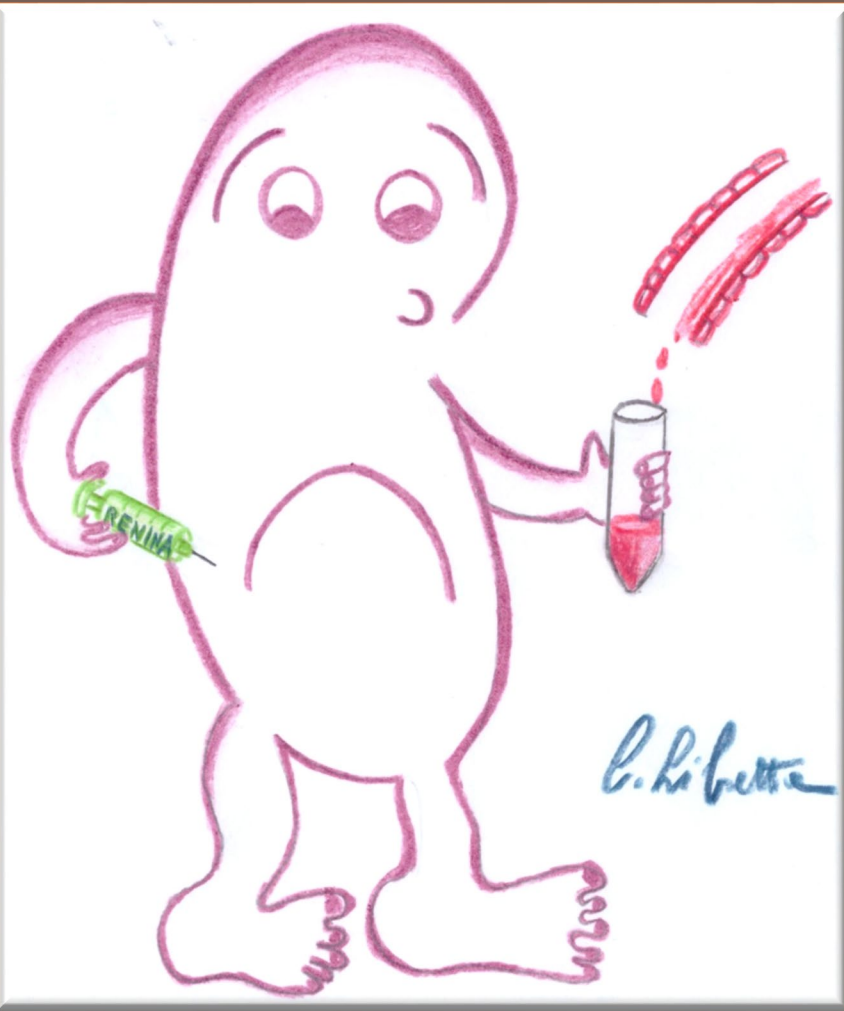


C. Lynn

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Terminologia

ACUTE KIDNEY INJURY (AKI)



- **AUMENTO CREATININEMIA**
- **AUMENTO CATABOLITI AZOTATI**
- **ALTERAZIONI IDROELETTROLITICHE**
- **RIDUZIONE DIURESIS**

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Definizione

- \approx 7% pazienti ospedalizzati.
- 36 – 67% dei pazienti critici.

*5–6% dei pazienti sono sottoposti a
terapia sostitutiva dialitica.*





100,000 deaths are
year are associated
with AKI.

Costs to the NHS
estimated to be £1
billion per year.
(Kerr et al 2014)

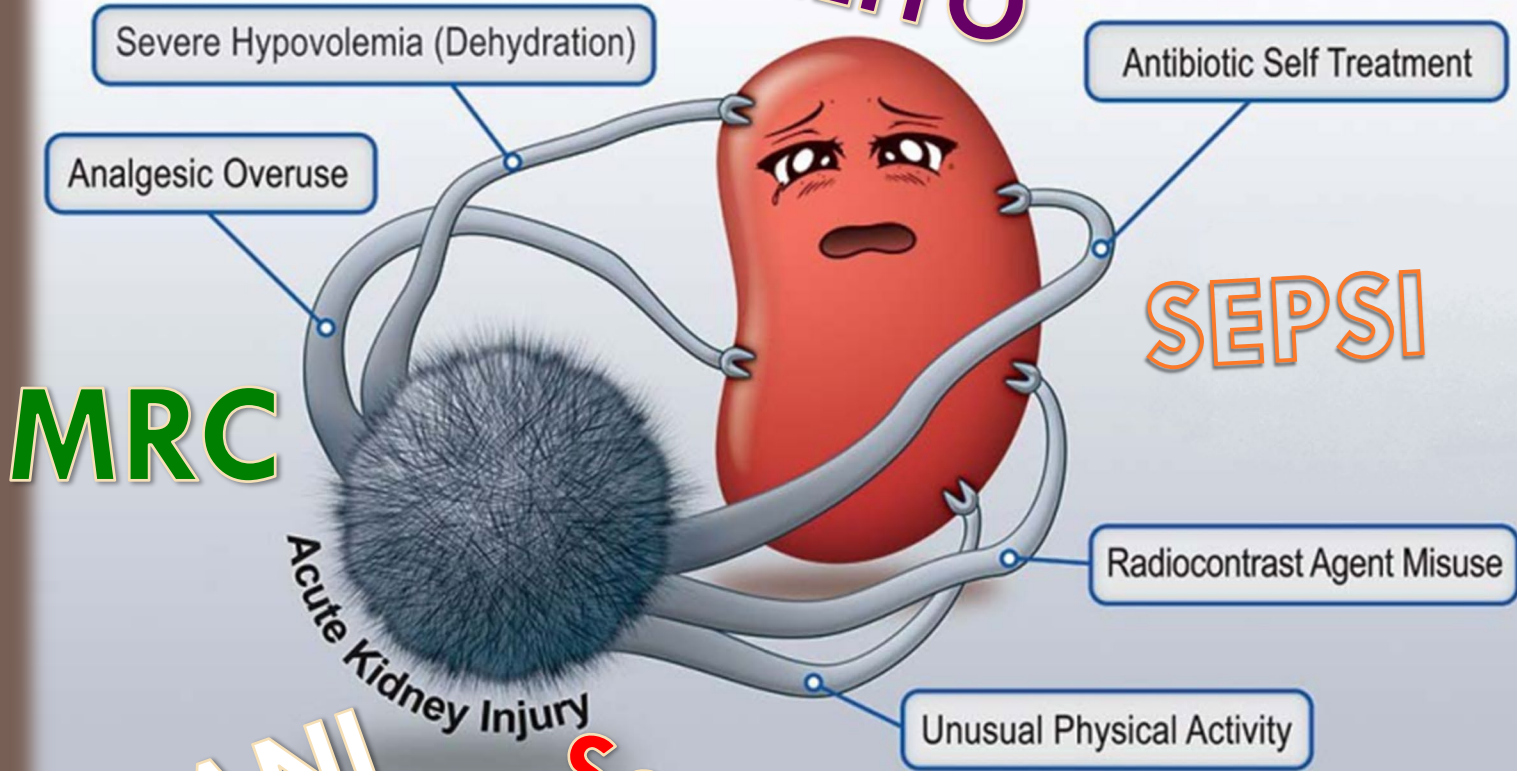
AKI



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Epidemiologia

DIABETE MELLITO



MRC

SEPSI

ANZIANI

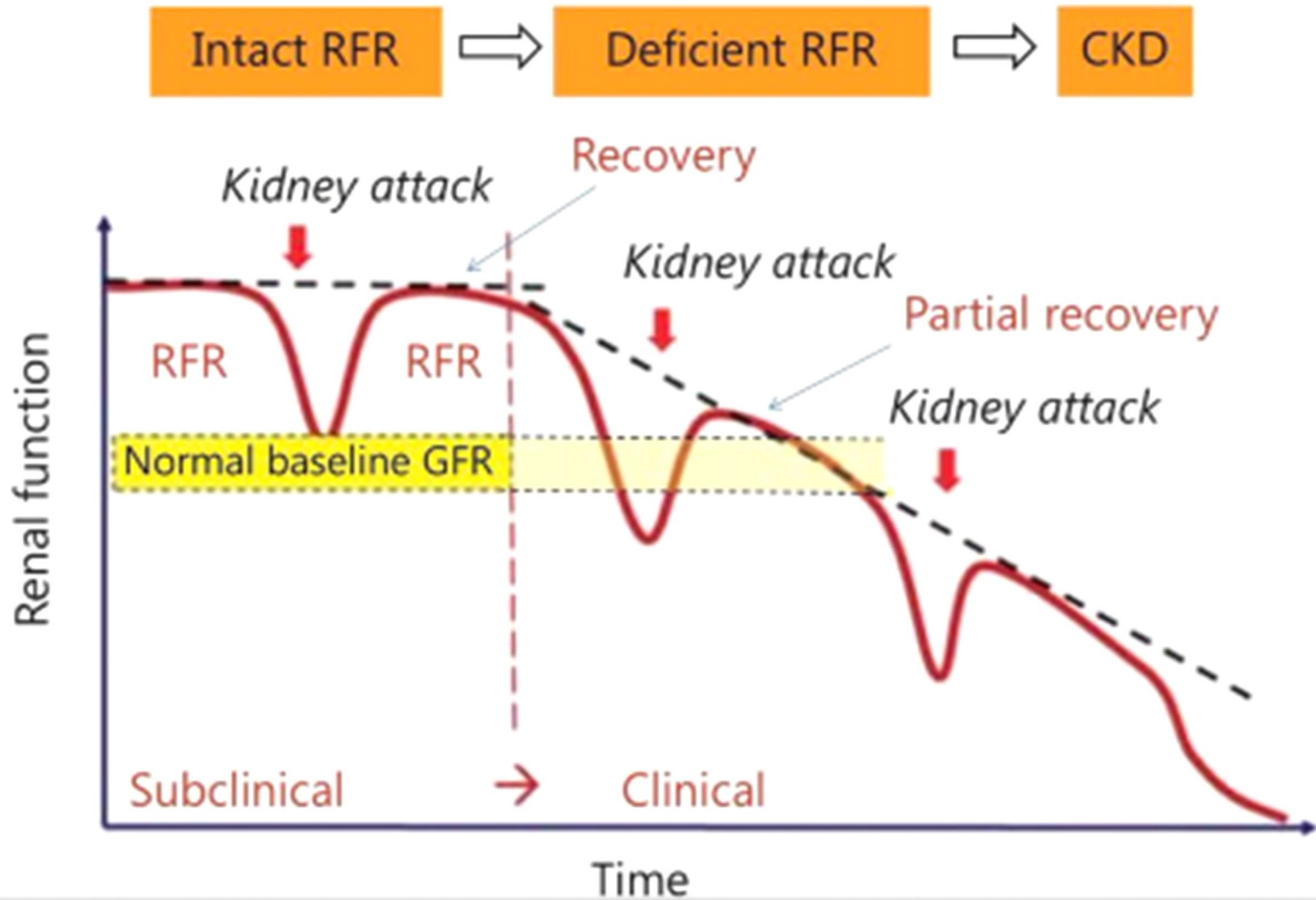
Sompensso cardiaco



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FATTORI DI RISCHIO

RISERVA RENALE FUNZIONALE (RFR)



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FATTORE PROTETTIVO



1. **DIAGNOSI DI AKI**

2. **DIAGNOSI EZIOLOGICA**



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DIAGNOSI

DIAGNOSI AKI



➤ PAZIENTI CON PREGRESSA

NO

**Creatininemia
anamnestica**

MIA

➤

A MRC

○ AUMENTO DEL 50%

CREATININEMIA



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Quando ero studente...

	Creatinine/GFR	UOP
<u>R</u> isk	1.5-fold ↑ in Cr <i>OR</i> GFR ↓ by 25%	< 0.5 ml/kg/hr x 6 hrs 0,4=168 ml
<u>I</u> njury	2-fold ↑ in Cr <i>OR</i> GFR ↓ by 50%	< 0.5 ml/kg/hr x 12 hrs
<u>F</u> ailure	3-fold ↑ in Cr <i>OR</i> GFR ↓ by 75%	< 0.5 ml/kg/hr x 24 hrs <i>OR</i> 672 ml Anuria x 12 hrs
<u>L</u> oss	Complete loss of function > 4 weeks (needs RRT)	
<u>E</u> SRD	Complete loss of function > 3 months	



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RIFLE

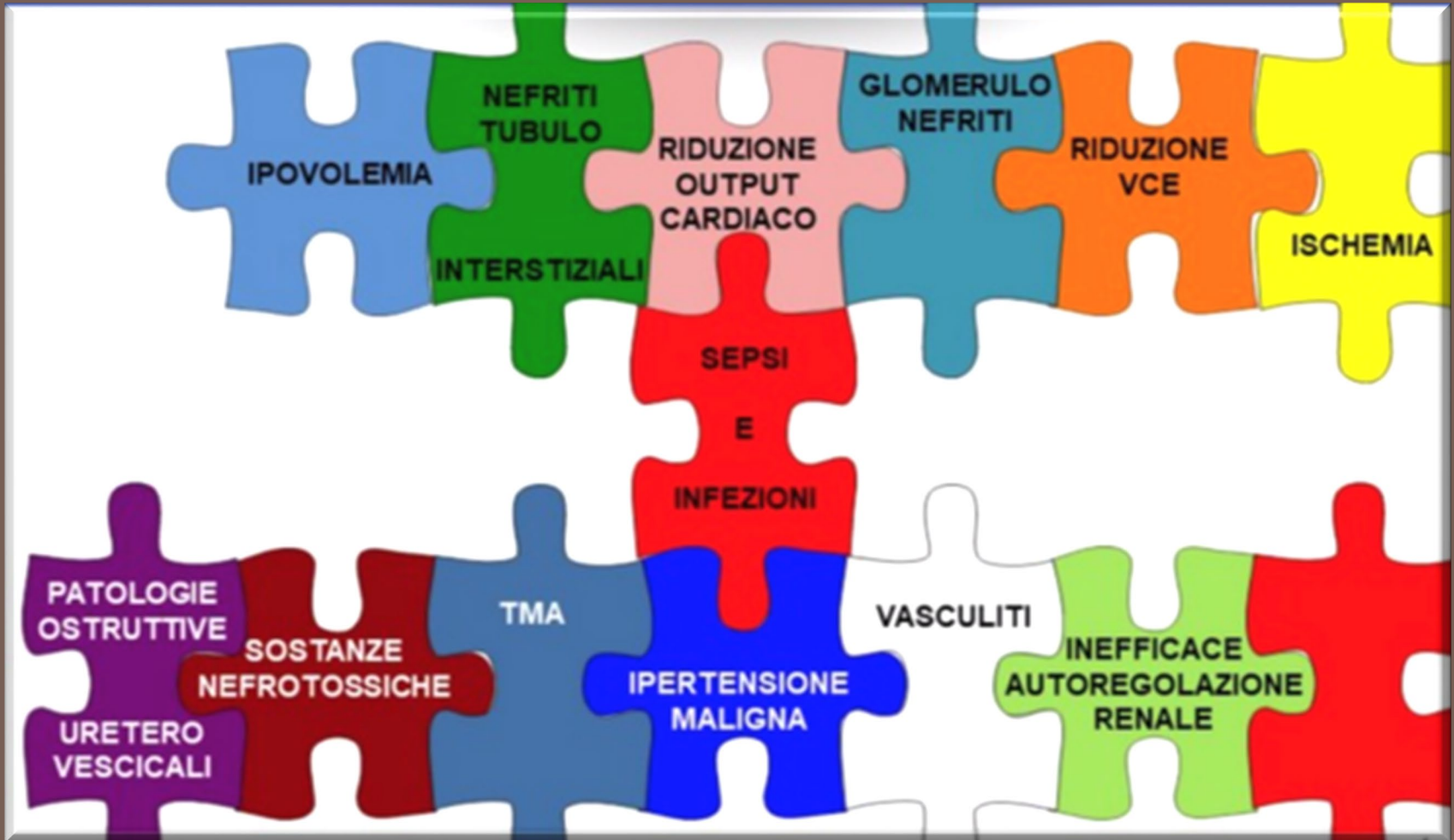
DIAGNOSI EZIOLOGICA

...e adesso viene il bello



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PUZZLE EZIOLOGICO



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PUZZLE EZIOLOGICO



PRERENAL *hypo, perfusion*

BUN: Creatinine > 20

↓ GFR causes both urea & creatinine to back up into blood. ↓ RPF causes more urea to be absorbed in PCT while creatinine is excreted

- ★ CHF
- ★ HEMORRHAGE
- ★ SHOCK
- ★ VOLUME DEPLETION
- ★ ADRENAL INSUFFICIENCY
- ★ RENAL ARTERY STENOSIS

Stimulates salt & water retention to restore volume & pressure by baroreceptor reflex mediated sympathetic (β₁) vasoconstriction of AFFERENT arteriole & renin release, which via effects of ATII stimulates aldosterone release to ↑ Na⁺ & H₂O reabsorption in distal collecting tubule

Stimulates hypothalamic production of ADH which acts on medullary collecting duct to cause increased H₂O reabsorption

stimulates enhanced PCT reabsorption of Na⁺, H₂O, urea, calcium, urate, and bicarb

URINE OSMOLARITY > 500 mOsm/kg

URINE Na⁺ < 20 mEq/L

Fe_{Na} < 1% (reduced)

RENAL uremia → ACUTE RENAL FAILURE

BUN: Creatinine < 15

↓ GFR causes both urea & creatinine to back up into blood, leading to ↑ excretional loss of urea. In renal failure, PCT cells are sloughed off and urea is not reabsorbed

URINE OSMOLARITY < 350 mOsm/kg

URINE Na⁺ > 40 mEq/L

Fe_{Na} > 2% (increased)

- ★ ACUTE TUBULAR NECROSIS
- ★ RENAL ISCHEMIA
- ★ INTERSTITIAL NEPHRITIS
- ★ GLOMERULONEPHRITIS
- ★ VASCULITIS
- ★ CRUSH INJURY

POSTRENAL obstruction

BUN: Creatinine > 15

↓ GFR causes both urea & creatinine to back up into blood, & subsequent ↑ in tubular pressure promotes further diffusion of urea back into blood. Persistent obstruction leads to kidney damage & findings of renal azotemia

URINE OSMOLARITY < 350 mOsm/kg

URINE Na⁺ > 40 mEq/L

Fe_{Na} > 1%

Obstruction of flow leads to reversal of Starling forces responsible for GFR. Hydronephrosis can occur w. increase in hydrostatic pressure in Bowman's capsule and tubular blockage, which can lead to cessation of filtration, azidosis, fluid overload, & hyperkalemia

- ★ NEPHROLITHIASIS
- ★ VESICoureTERAL REFLUX
- ★ PROSTATIC HYPERTROPHY
- ★ PREGNANCY

AZOTEMIA

NAUSEA, VOMITING, PRURITUS

↑ Blood urea nitrogen & creatinine



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Apparato urinario/Apparato cardiovascolare

PRE-RENALE

55-60%

POST-RENALE

5-10%

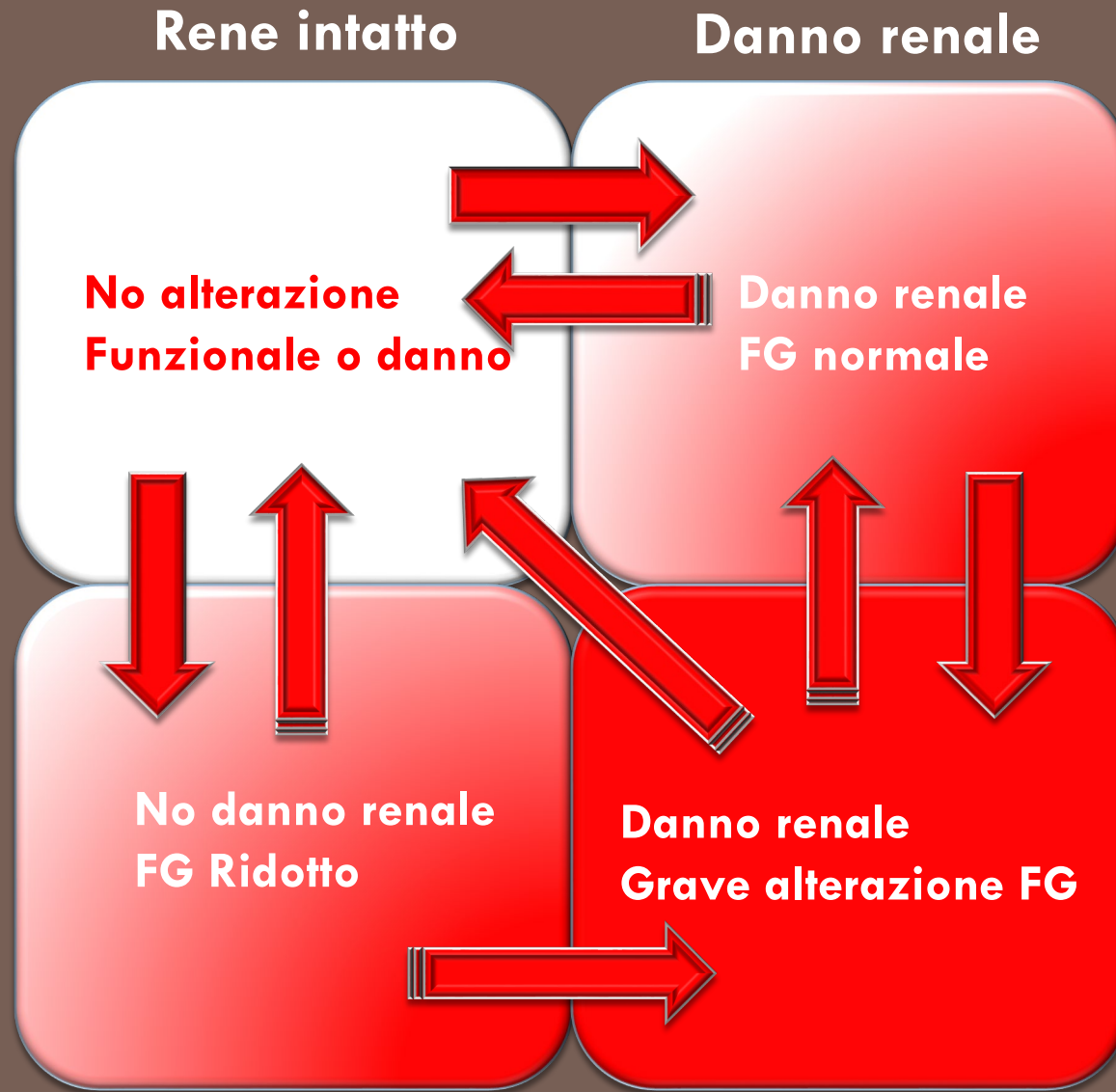
RENALE

30-35%



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EPIDEMIOLOGIA



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Progressione AKI



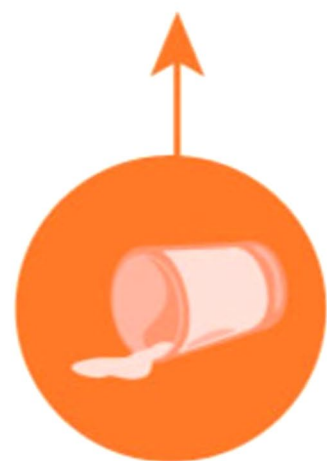
AKI (Acute Kidney Injury)



Recent infection



Certain medications



Severe dehydration



Exposure to heavy metals or toxic solvents



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ANAMNESI



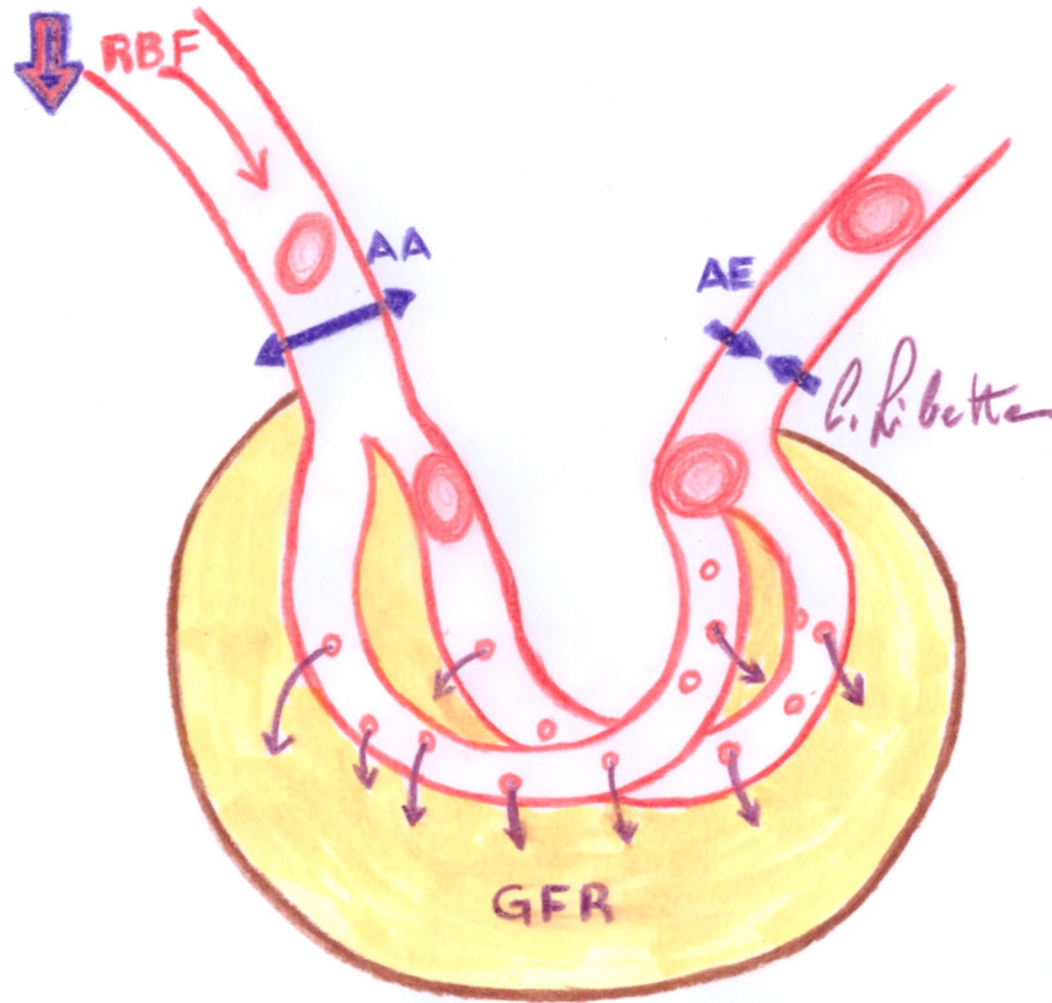
AKI

ELETTROLITI URINARI



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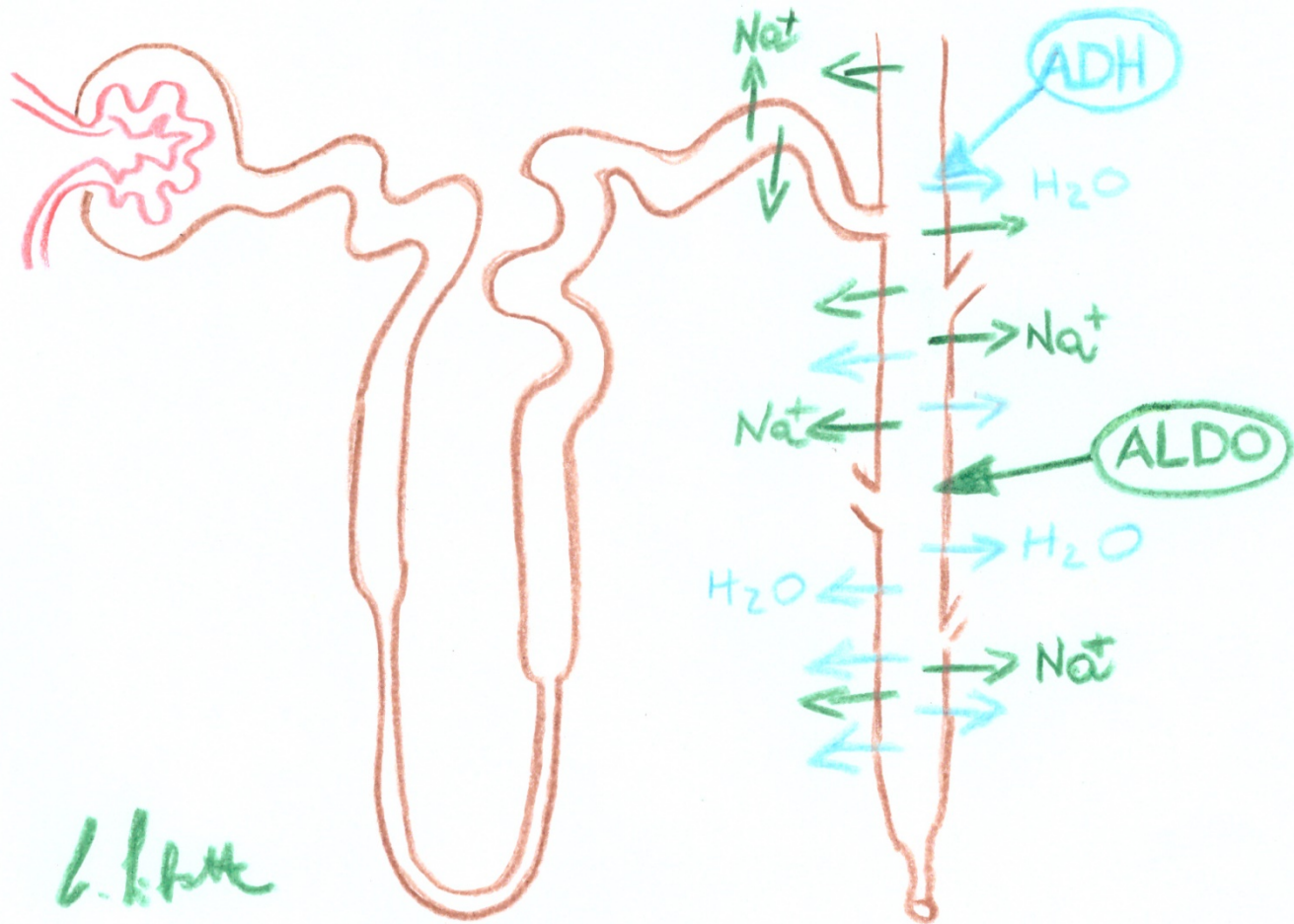
DIAGNOSTICA



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Iperperfusion Renale

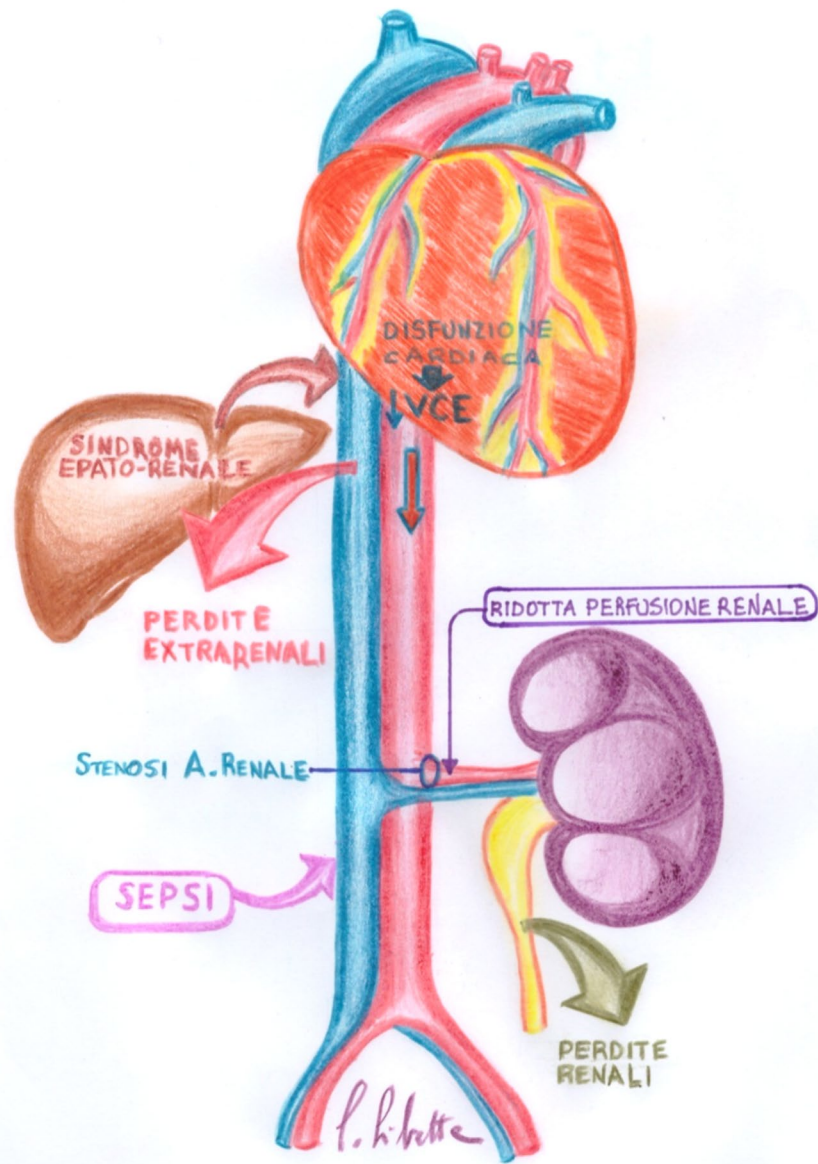




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Ipoperfusione Renale





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AKI PRE-RENALE



1. DEPLEZIONE DEL VEC

2. RIDUZIONE DEL VCE

3. VASODILATAZIONE SISTEMICA

4. VASOCOSTRIZIONE RENALE SELETTIVA

5. FARMACI



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Eziologia AKI Pre-renale

1. DEPLEZIONE DI VOLUME

- **Disidratazione**
 - **Perdite extrarenali**
 - *Sudorazione*
 - *Diarrea*
 - *Vomito*
 - **Perdite renali**
 - *Diuretici*
- **Perdita di plasma**
 - *Traumi estesi*
 - *Ustioni*
- **Emorragie**



**DOTTORE
MI GIRA LA
TESTA, FACCIO
POCA PIPI' E
HO LA BOCCA
SECCA**



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AKI Pre-renale da deplezione





Ipotensione ortostatica

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AKI Pre-renale da deplezione



1. DEPLEZIONE DI VOLUME

- Emorragie
 - Trasfusione
- Perdita di plasma
 - *Plasma*
 - *Soluzione Fisiologica*
- Disidratazione
 - *Soluzione Fisiologica*



2. Riduzione del VCE

- ❖ Scompenso cardiaco acuto (Infarto del miocardio)
- ❖ Aritmie



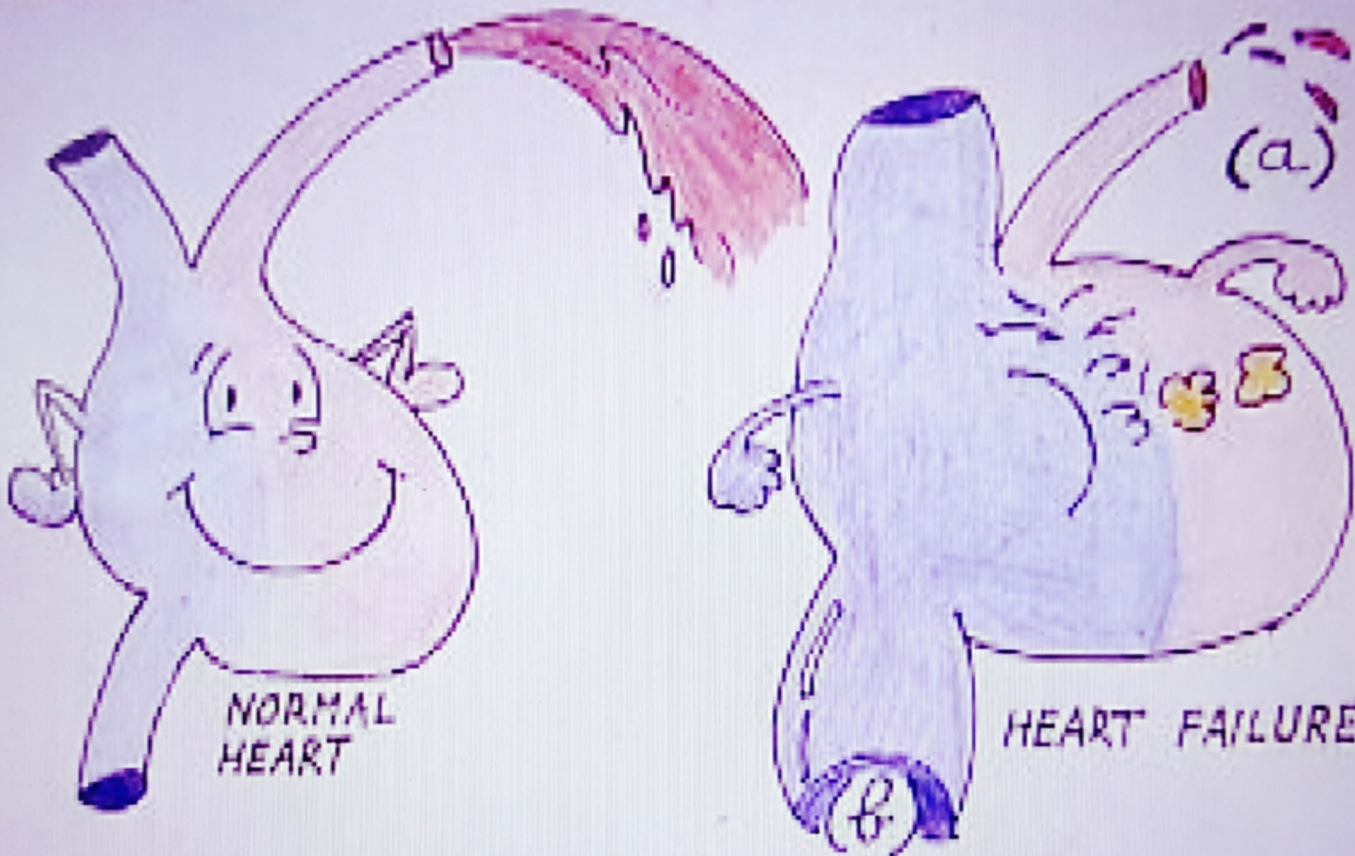
**DOTTORE
FACCIO POCA
PIPI',
HO I PIEDI
GONFI E
RESPIRO MALE**



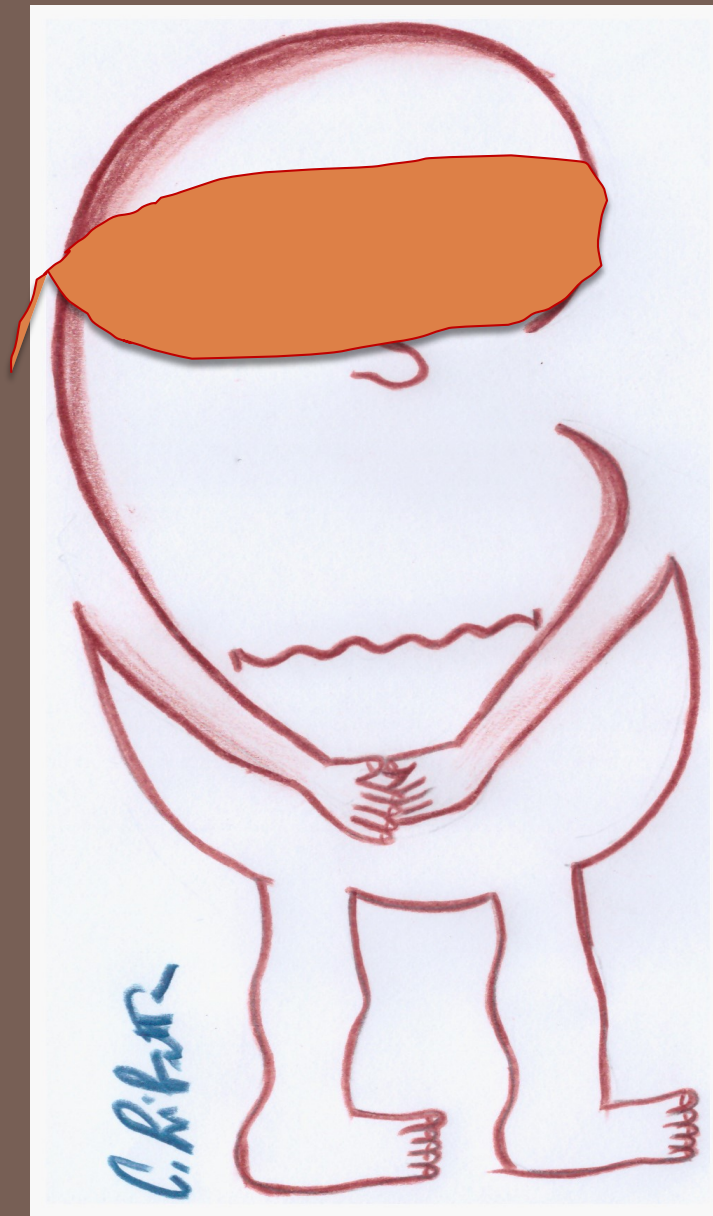
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AKI Pre-renale



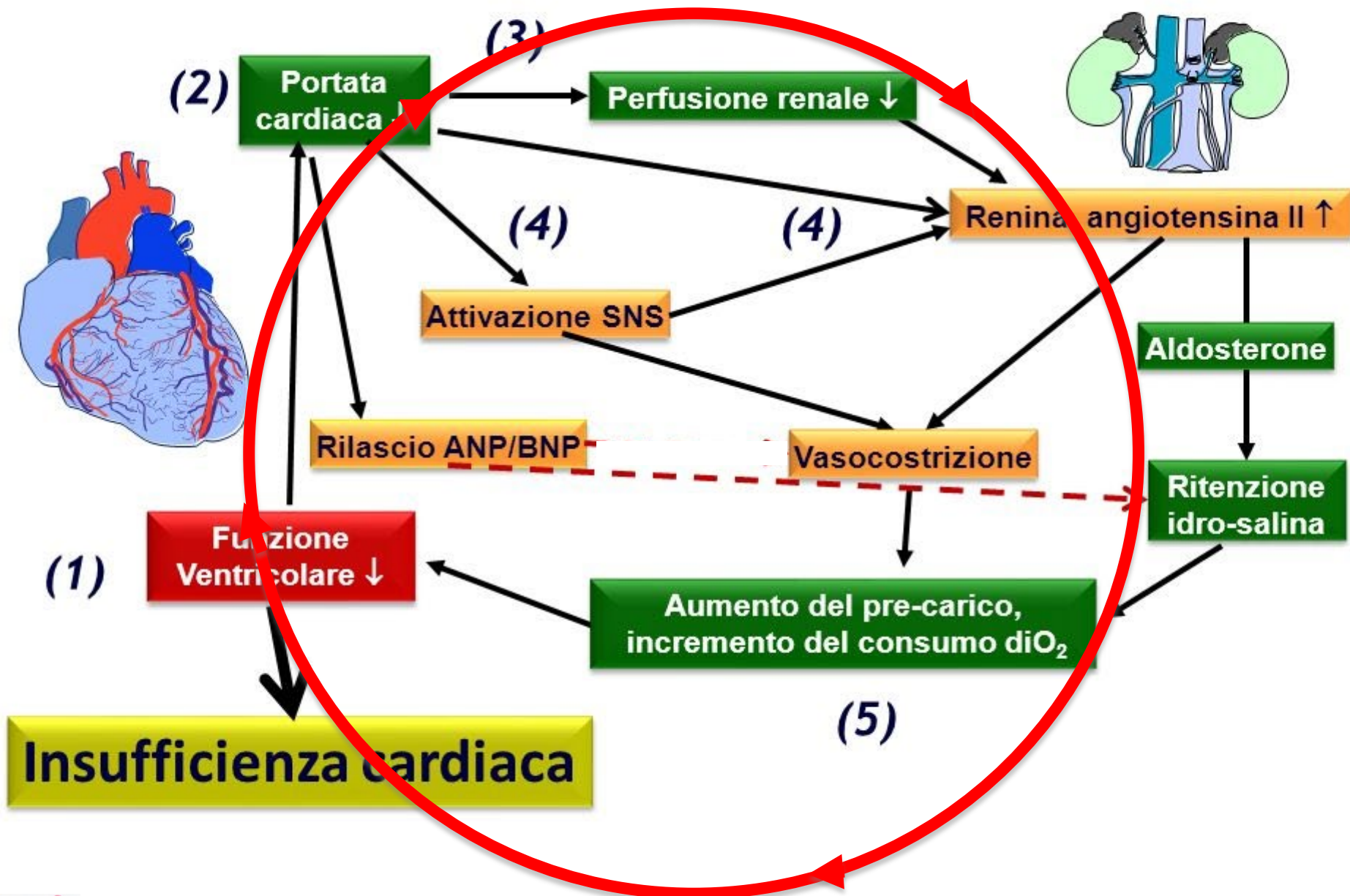


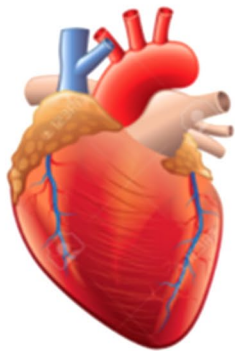
Hemodynamic effects of heart failure:
(a) low effective blood volume
(b) high peripheral venous pressure



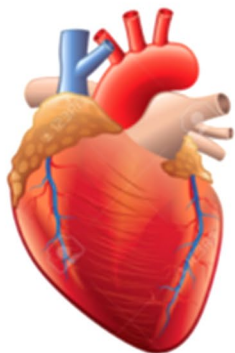

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AKI Pre-renale da Scompenso cardiaco

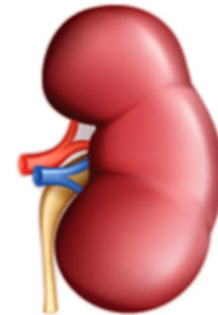




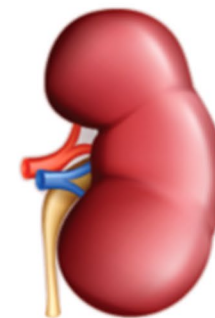
**Scompenso
cardiaco acuto**



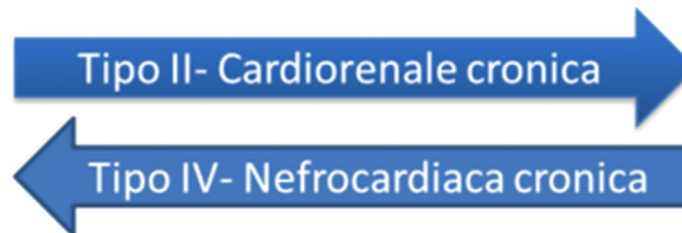
**Scompenso
cardiaco cronico**



**Insufficienza
renale acuta**



**Insufficienza
renale cronica**



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Sindrome Cardio-renale acuta



2. Riduzione del VCE

❖ Farmaci che riducono il Pre-carico

- Diuretici
- Nitroderivati



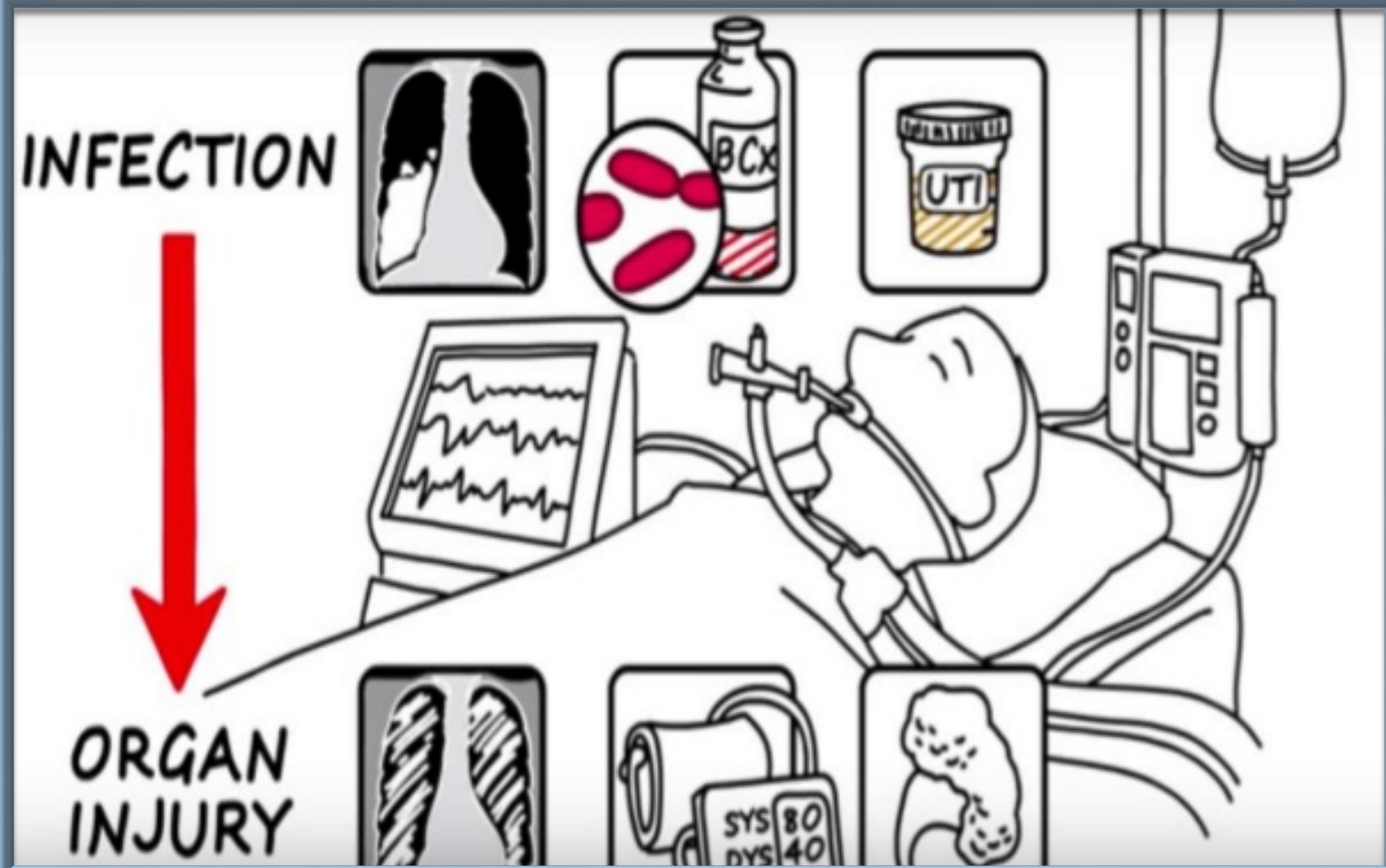
3. Vasodilatazione sistemica

❖ Sepsi

❖ Anafilassi



VASODILATAZIONE ART. EFF.



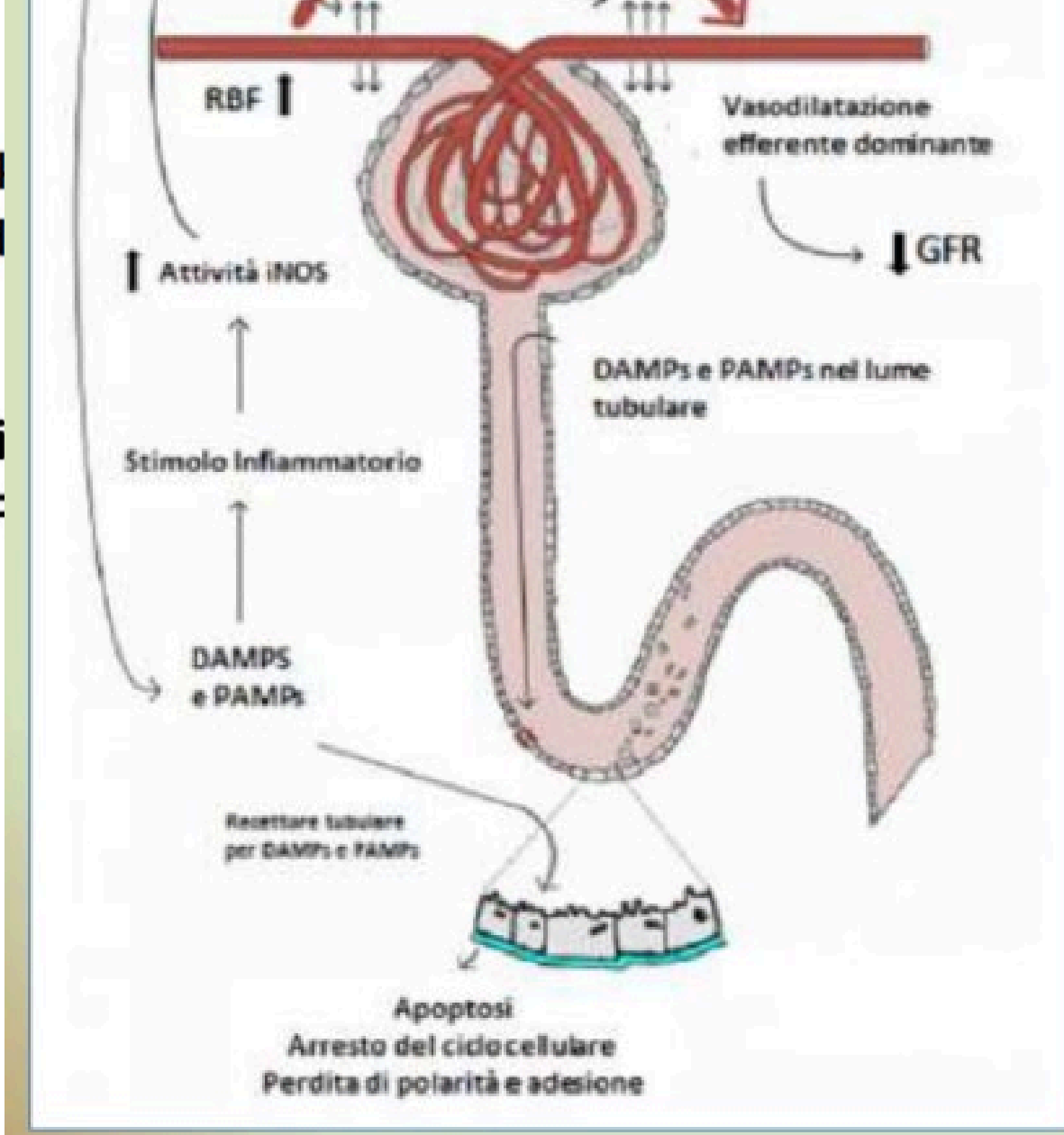
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SEPSI



Shock
iperd

Riduzi
del GF



ate



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AKI e SEPSI

3. Vasodilatazione sistemica

- ❖ AMINE
- ❖ ANTIBIOTICI
- ❖ CRRT



4. Vasocostrizione renale

- ❖ Sindrome epatorenale (SER)
- ❖ Farmaci: catecolamine





Stasi Splancnica

Vasodiazione sistemica



Paracentesi



Riduzione Volemia Efficace



Diuretici



↓ **Perfusione Renale**



↑ **Attivazione SRAA**

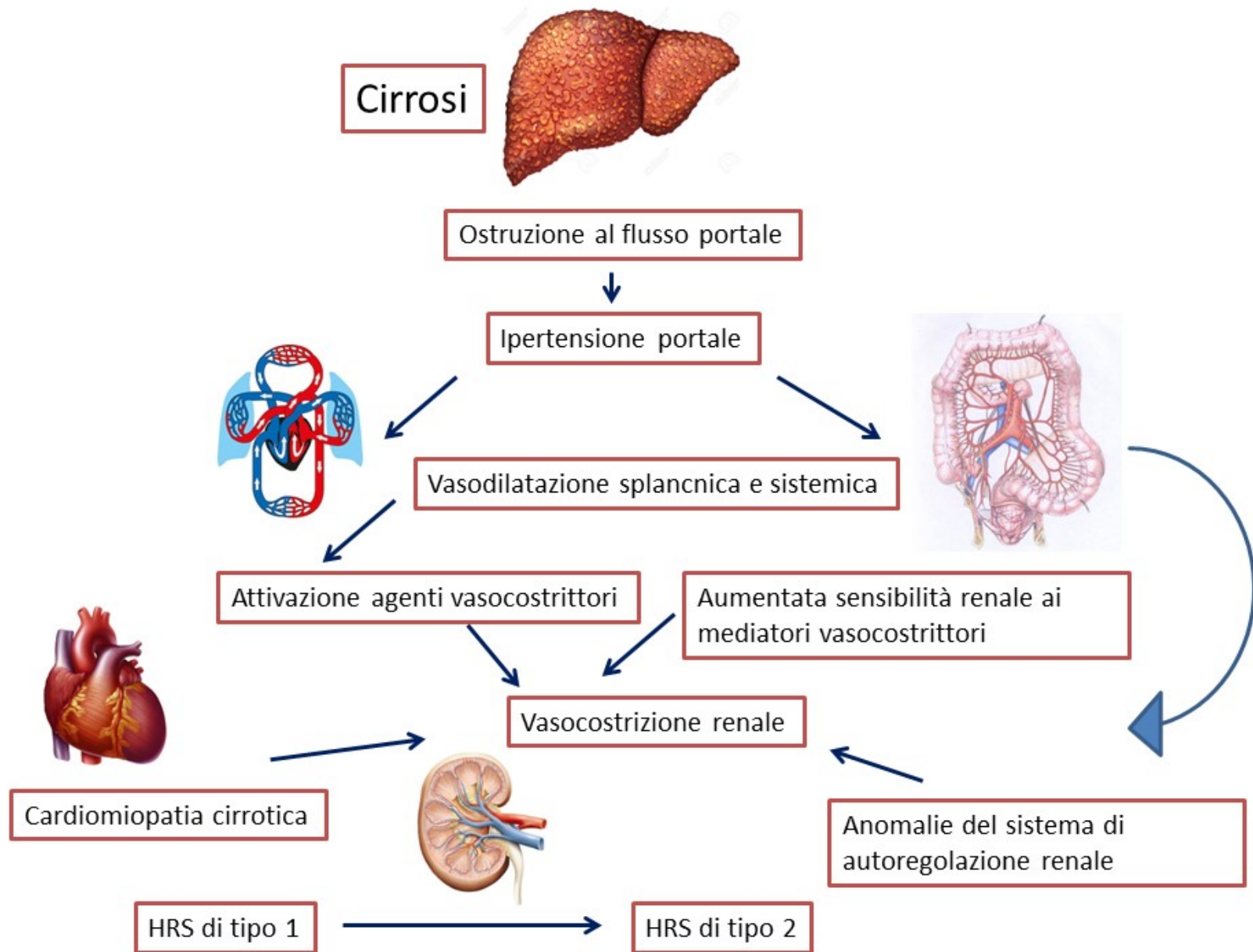


↑ **Attivazione Sistema Simpatico**



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Sindrome Epato-renale



AKI si sviluppa in 2 settimane o meno



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HRS tipo 1

Il danno renale si sviluppa gradatamente, con lenta progressione dell'azotemia e della creatininemia che rimane nel range di 2-4 mg/dl. Si associa frequentemente a patologie epatiche croniche, come l'epatite B, epatite C, la cirrosi biliare primaria.



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HRS tipo 2

Criteri diagnostici maggiori (devono esserci tutti)

- **Epatopatia** acuta o cronica con insufficienza epatica avanzata ed ipert. portale
- **Ridotta GFR** = creat. > 1,5 mg/dl o clearance creat. <40 ml/min
- **Assenza di cause precipitanti** definite (shock, infezione batterica in atto, recente trattamento con farmaci nefrotossici, perdite gastrointestinali o renali di liquidi (>500 g/d per alcuni gg in pazienti senza edemi o ascite, >1000 g/d in pazienti con edemi o ascite)
- **Nessun miglioramento della funzione renale dopo la sospensione della terapia diuretica e espansione con 1500 cc di sol. fisiologica**
- **Proteinuria <500 mg/dl e assenza di uropatia ostruttiva o nefropatia parenchimale**

Criteri diagnostici minori

1) Volume urinario <500 ml/die; 2) Sodio urinario <10 mEq/l; 3) Osmolarità urinaria > osmolarità plasmatica; 4) Sodiemia <130 mEq/l; 5) numero di emazie nel sedimento urinario <50 per campo.



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Sindrome Epato-renale

4. Vasocostrizione renale

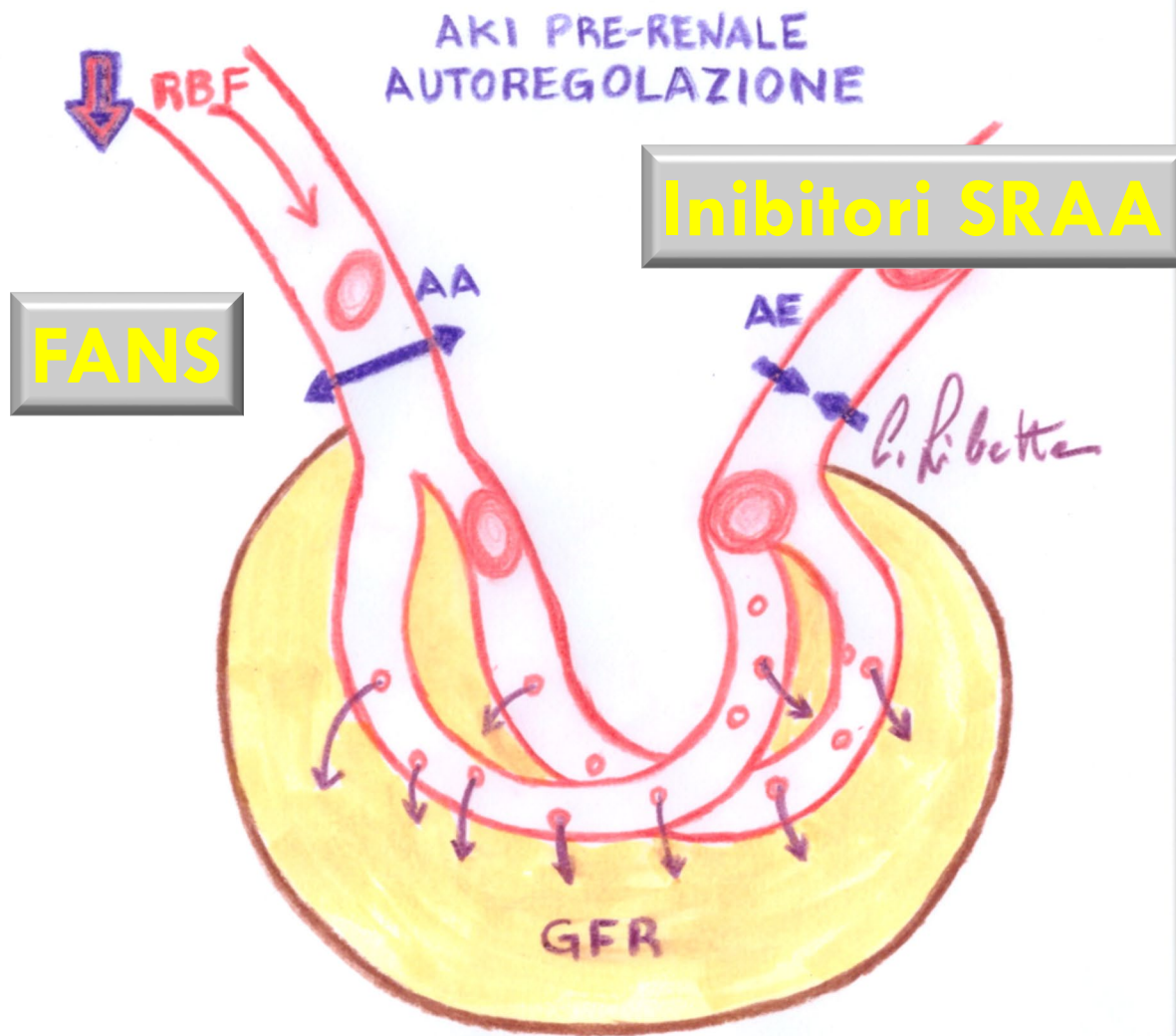
- ❖ Dopamina dosaggio renale
- ❖ Albumina
- ❖ Blanda terapia diuretica
(Risparmiatori di potassio)



5. Farmaci

- **INIBITORI SRAA**
- **FANS**
- **INIBITORI CALCINEURINE**
- **IL-2**
- **ANESTETICI**

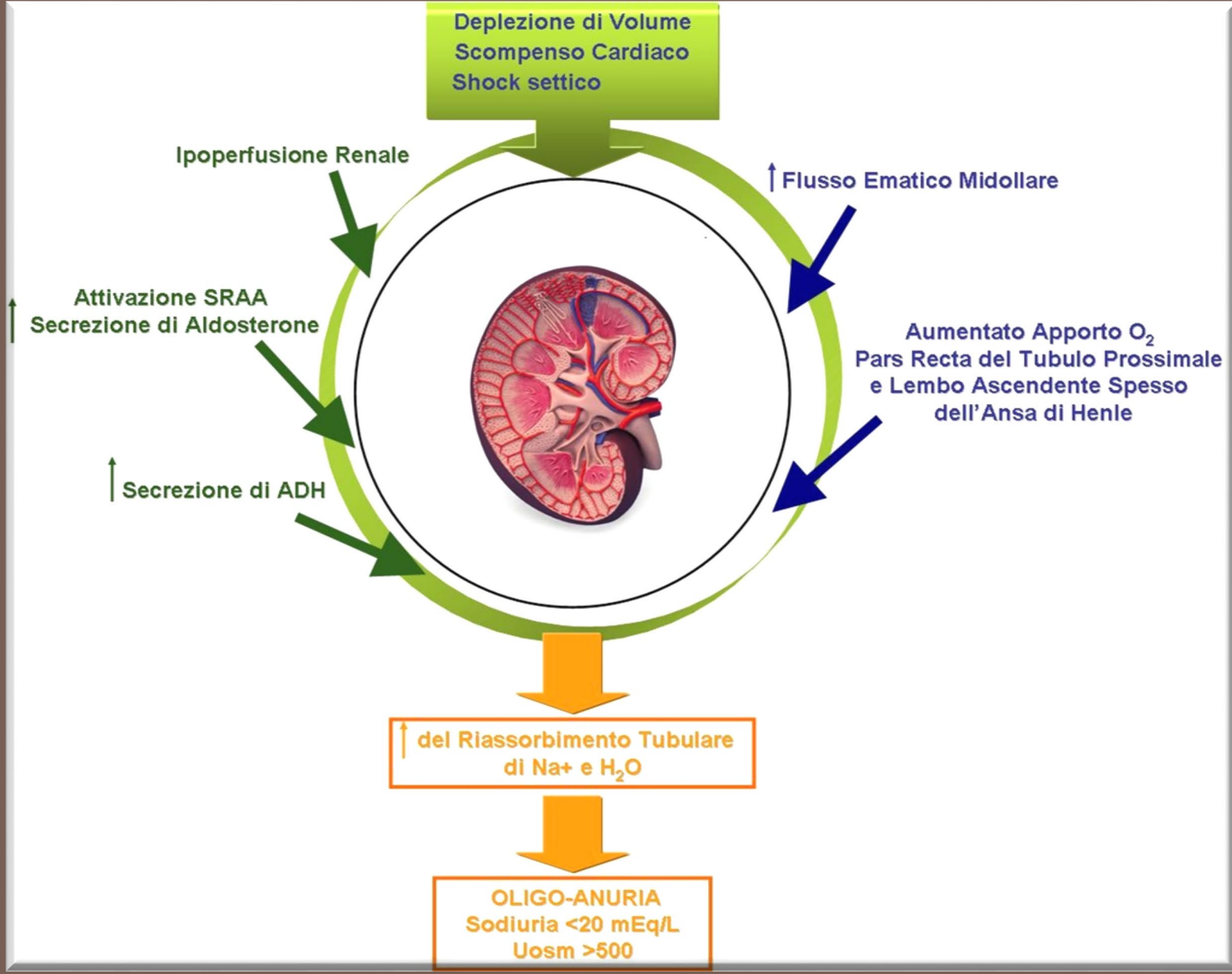




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Farmaci e AKI Pre-renale





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Fisiopatologia AKI Pre-renale

- **DANNO TUBULARE (70%)**

- **Necrosi tubulare acuta**

- **DANNO GLOMERULARE (15%)**

- **Glomerulonefrite acuta**
- **Malattie sistemiche**

- **DANNO VASCOLARE (10%)**

- **Malattia renale aterotrombotica**
- **Cause microvascolari**
- **Stenosi**

- **DANNO TUBULO-INTERSTIZIALE(5%)**

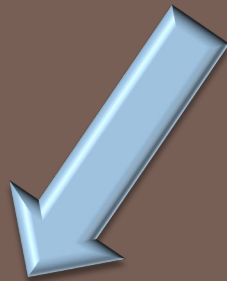
- **Allergico**
- **infettivo**



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AKI RENALE

Necrosi Tubulare Acuta



Ischemica

- Riduzione VEC
- Shock settico
- Arresto Cardiaco
- Scompenso Cardiaco



Tossica Endogena

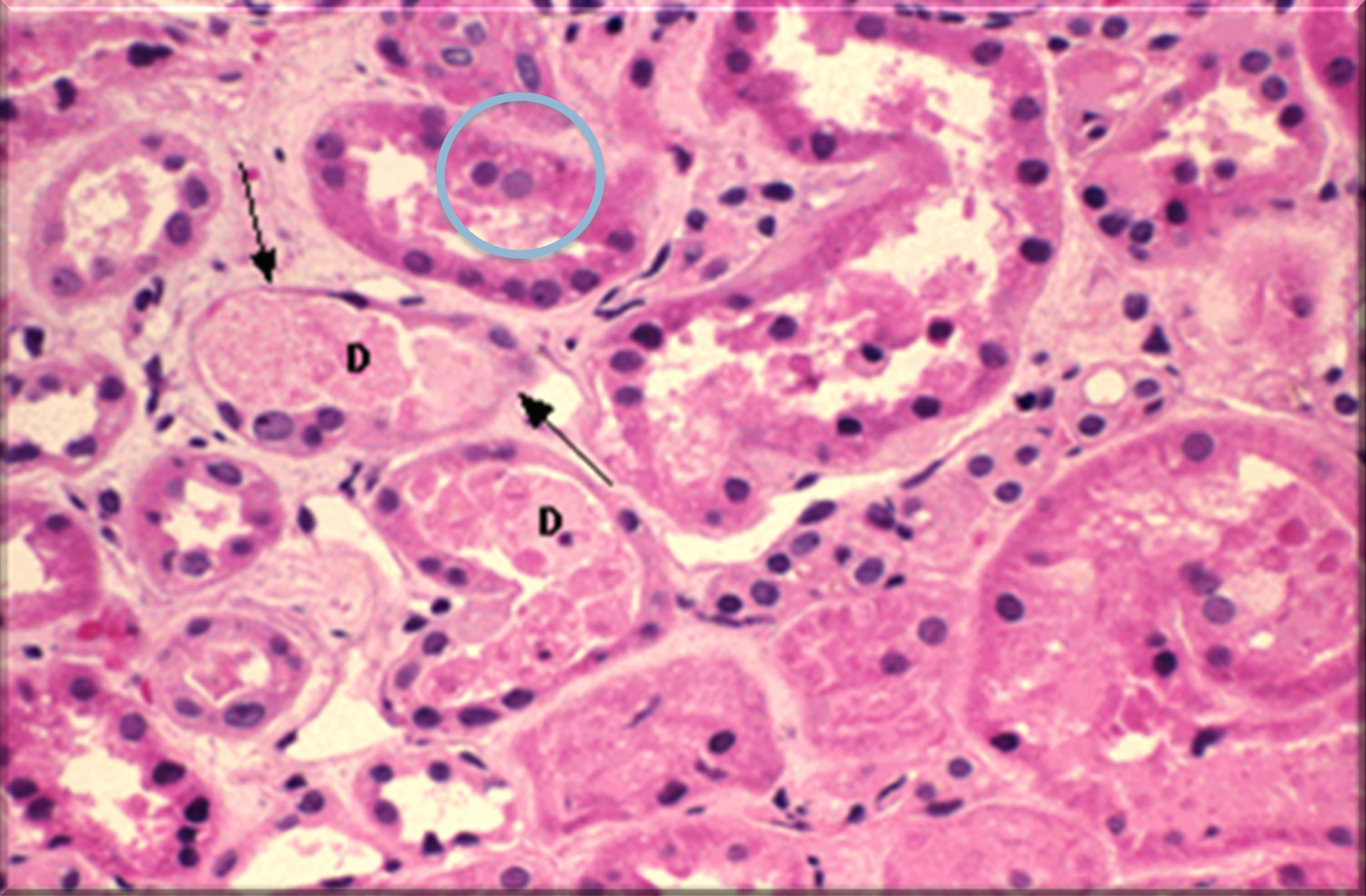
- Mioglobina
- Bilirubina
- Catene leggere



Tossica Esogena

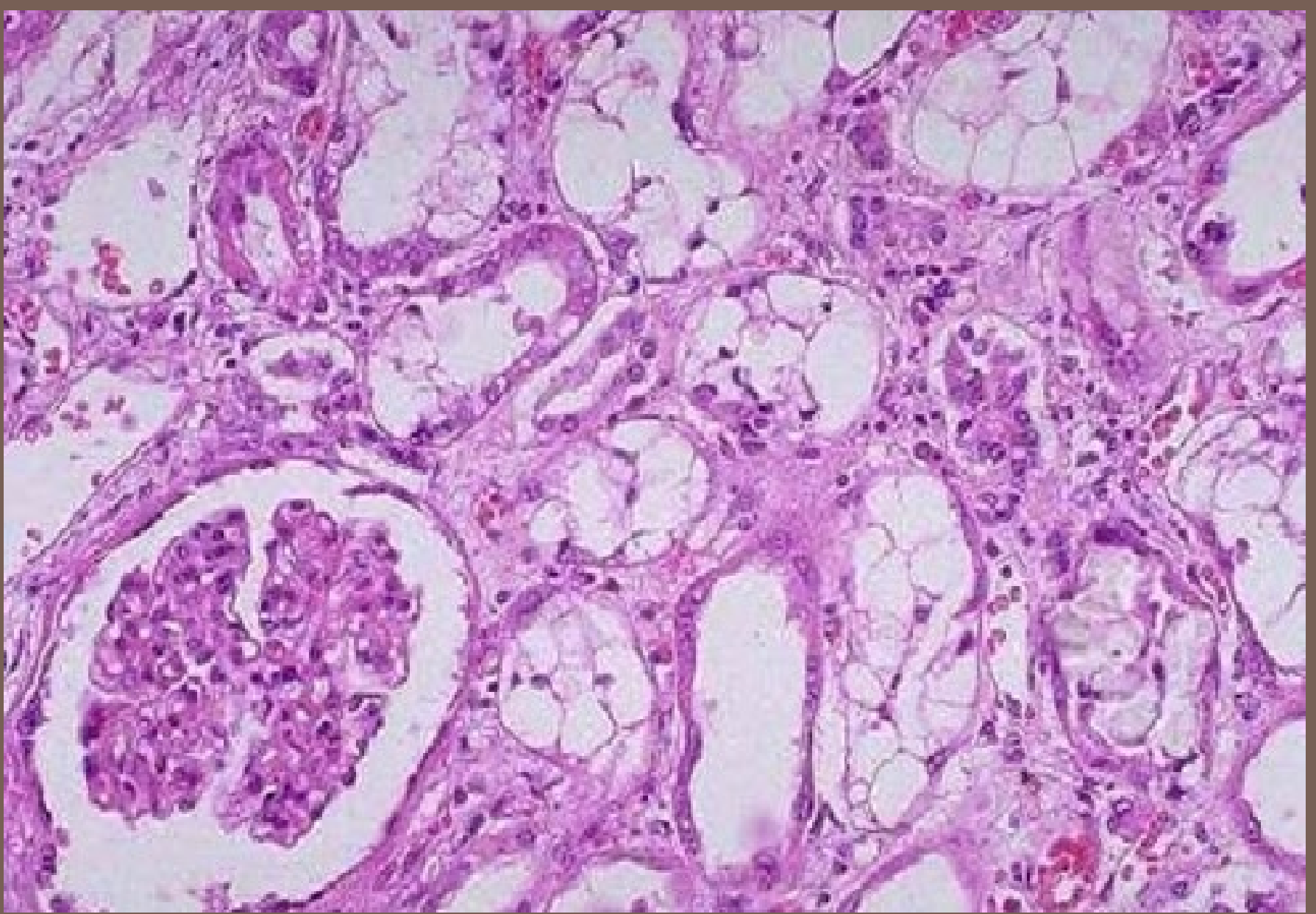
- Mezzo di contrasto
- Aminoglicosidi
- FANS
- CIS-Platino
- Anfotericina-B





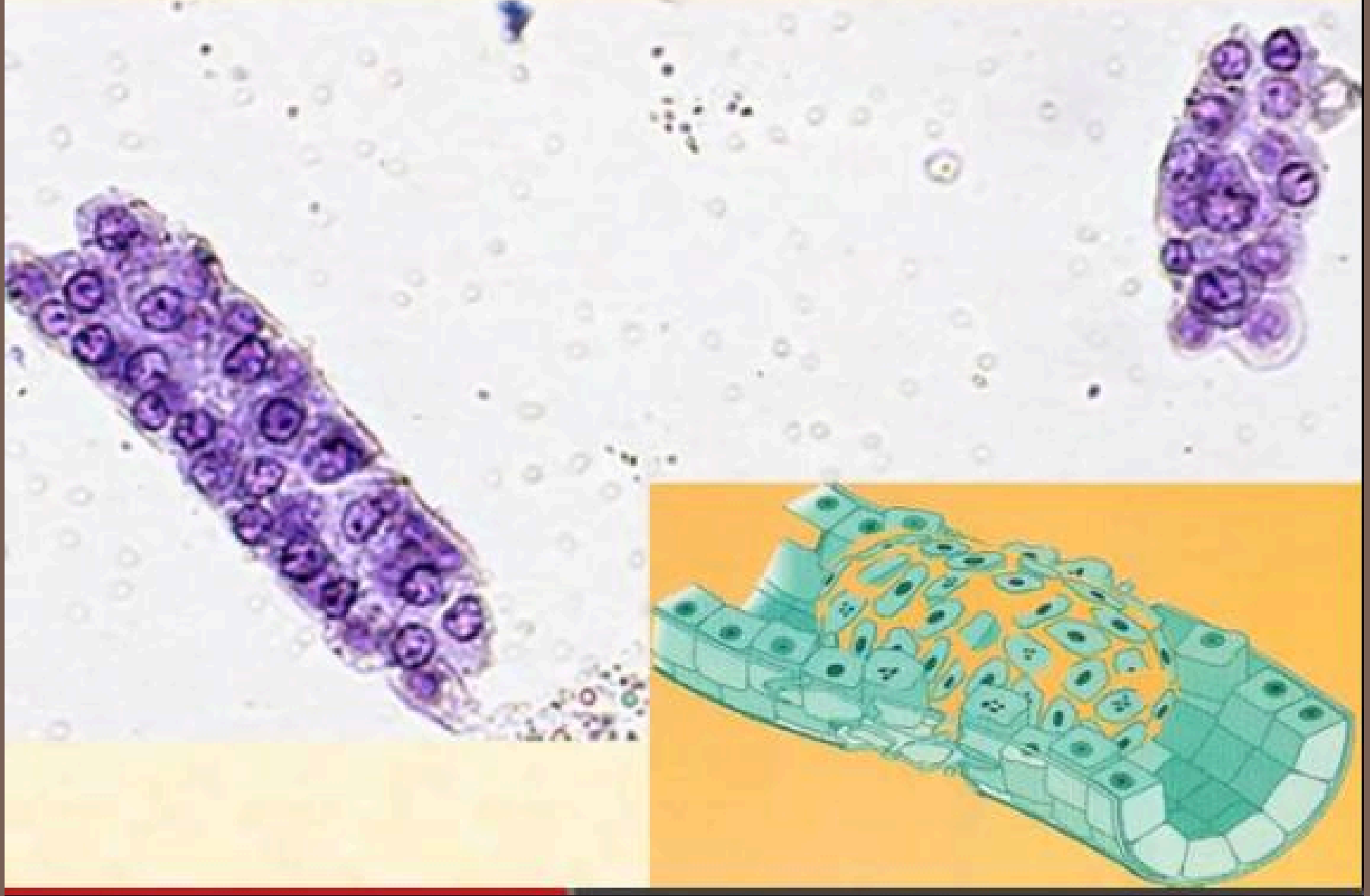
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Istologia ATN



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Istologia ATN



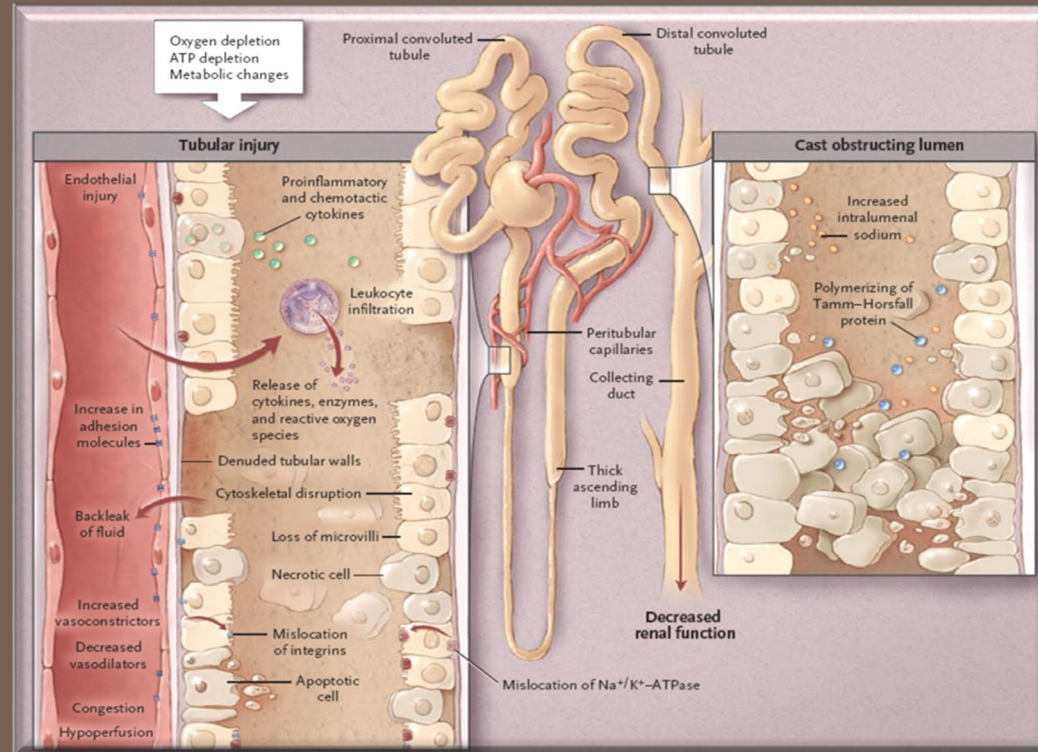
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Cast cellule tubulari



IPO-PERFUSIONE

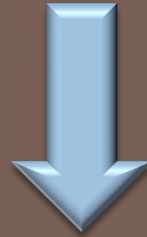
TCP



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AKI ISCHEMICO

Necrosi Tubulare Acuta



Tossica
Endogena

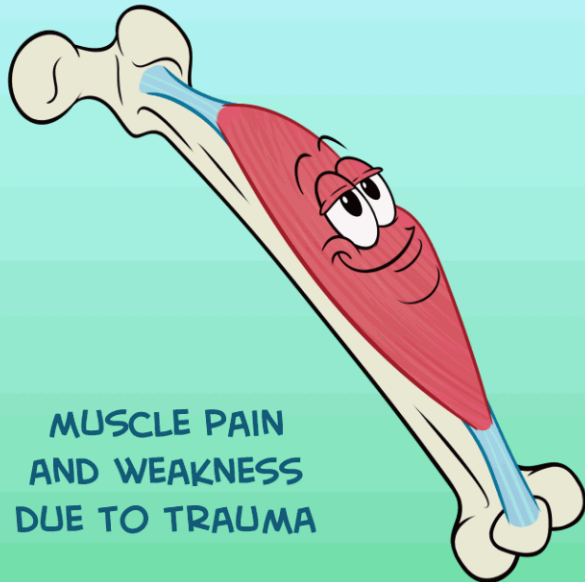


- **Mioglobina**
- **Bilirubina**
- **Catene leggere**



RHABDOMYOLYSIS

MUSCLE TISSUE BREAKDOWN WITH
RELEASE OF INTRACELLULAR CONTENTS
(MYOGLOBIN) INTO CIRCULATION



MUSCLE PAIN
AND WEAKNESS
DUE TO TRAUMA

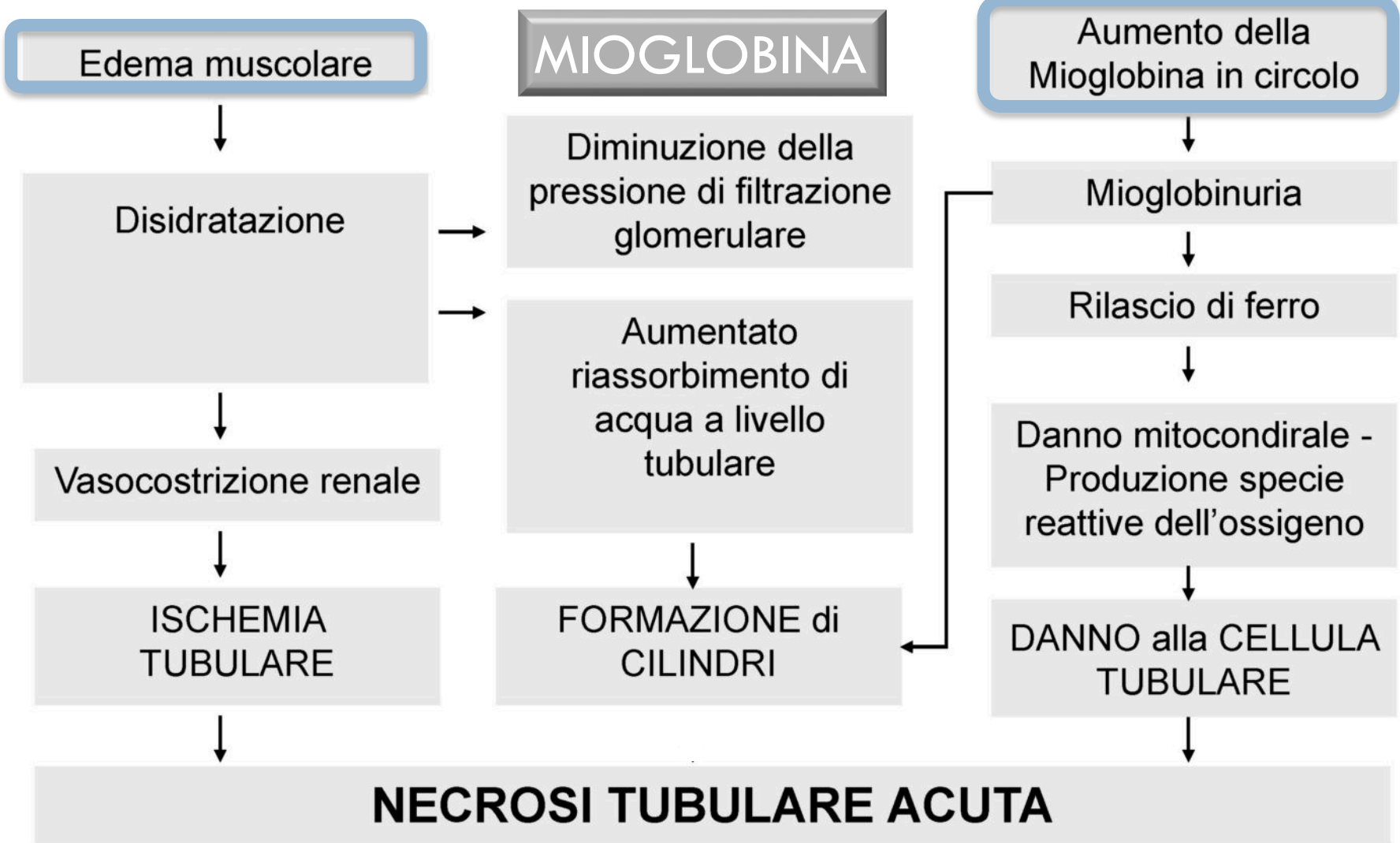
ELEVATED CREATINE
KINASE (CK) LEVELS

DARK, REDDISH
BROWN URINE DUE
TO MYOGLOBINURIA



MYOGLOBIN MAY OCCLUDE THE STRUCTURES OF THE KIDNEY
AND BREAK DOWN INTO TOXIC COMPOUNDS LEADING TO
ACUTE TUBULAR NECROSIS OR ACUTE RENAL FAILURE



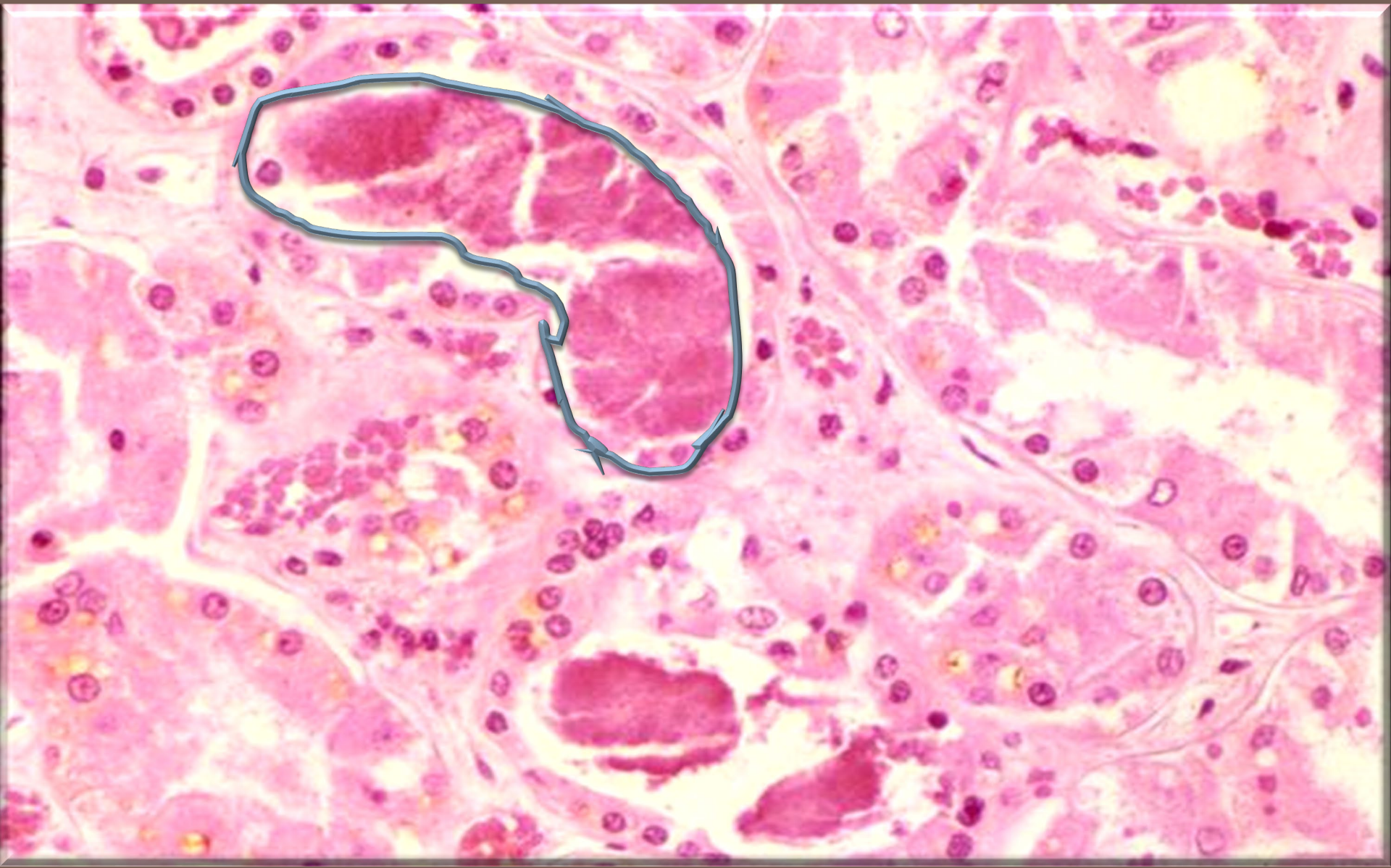


AKI-Renale Tossica endogena



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AKI/Rabdomiolisi



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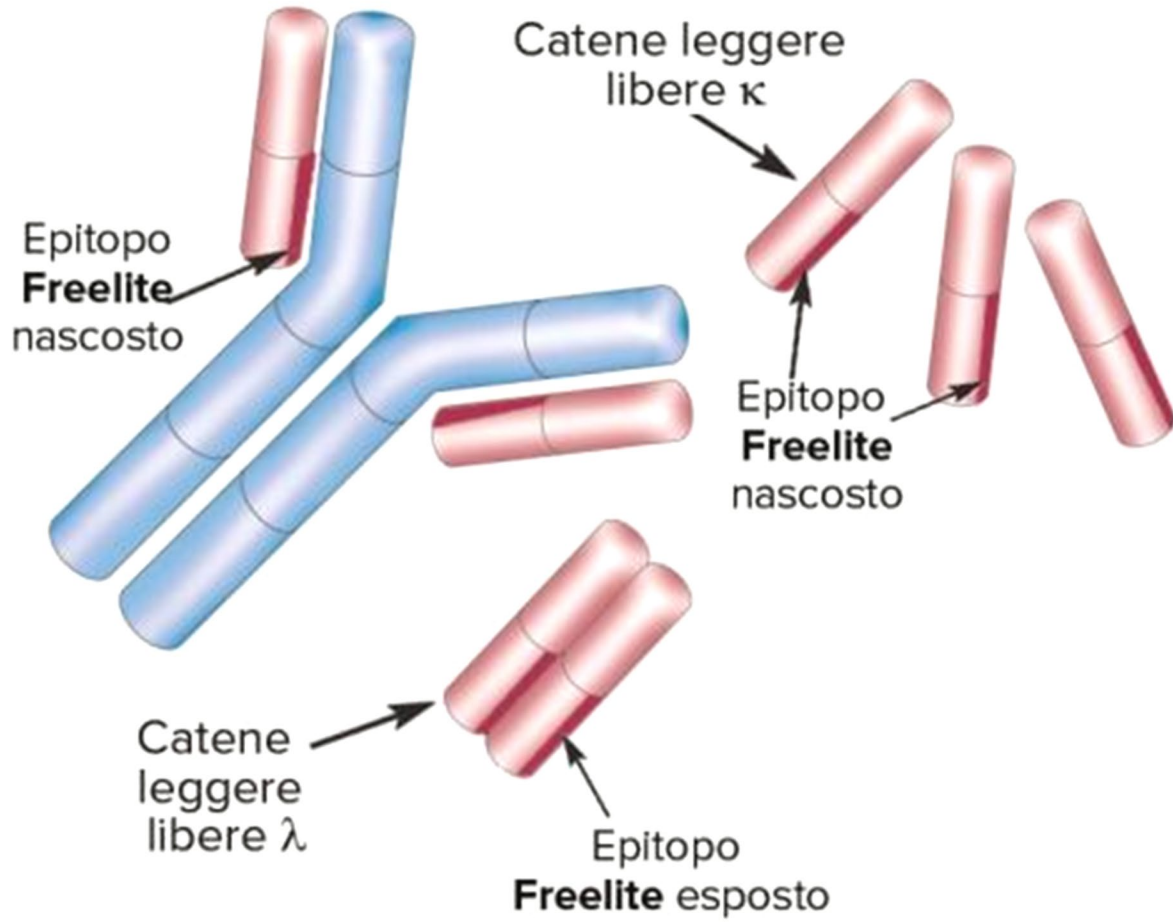
AKI/Rabdomiolisi

RHABDO



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AKI/Rabdomiolisi

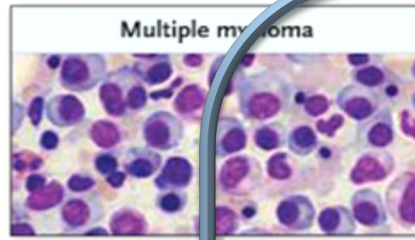
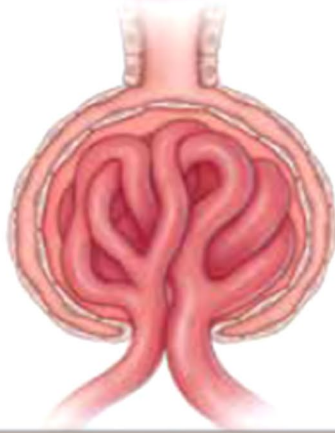


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MIELOMA MULTIPLO



Glomerular manifestations



Multiple myeloma

Overproduction

Kappa or lambda light chains

Heavy immunoglobulin chains

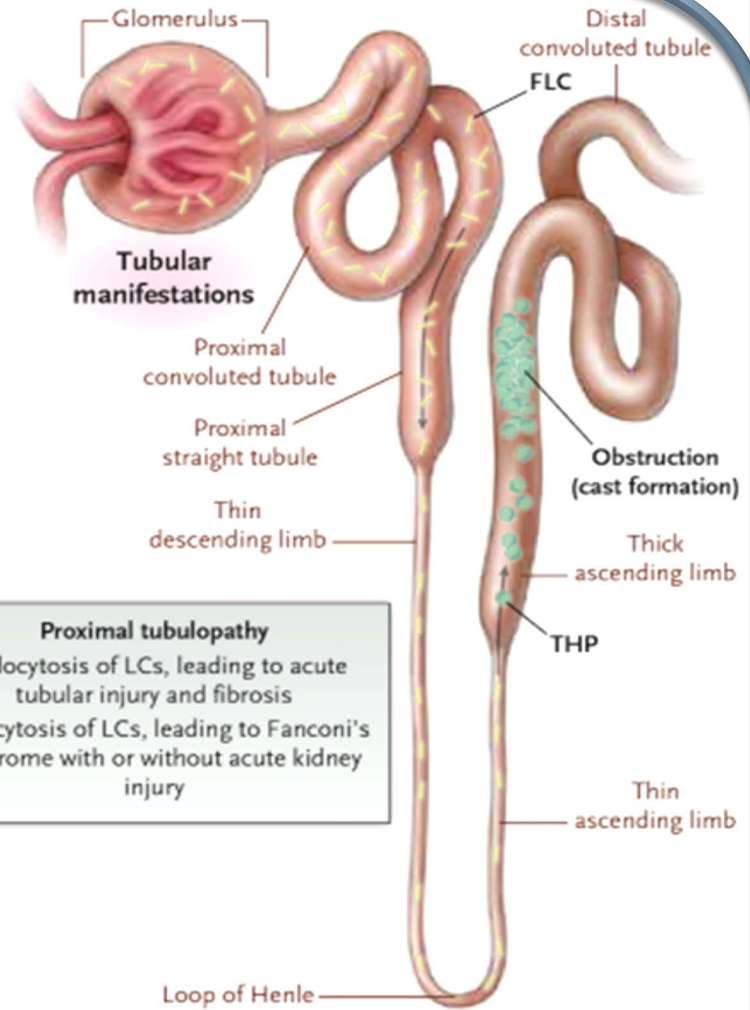
Urine albumin >2 g/day

Urine albumin ≤2 g/day

Deposition of light chains or monoclonal immunoglobulins, leading to glomerulopathy and proteinuria (urine albumin typically >2 g/day)

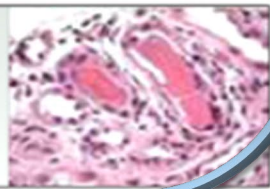
Glomerulopathy

- AL amyloidosis
- AH amyloidosis
- Monoclonal immunoglobulin deposition disease (light-chain, heavy-chain, or both)
- Proliferative GN with monoclonal IgG deposits
- Monoclonal cryoglobulinemia
- Membranoproliferative GN
- C3 glomerulopathy
- Fibrillary GN
- Immunotactoid glomerulopathy



Proximal tubulopathy
Endocytosis of LCs, leading to acute tubular injury and fibrosis
Endocytosis of LCs, leading to Fanconi's syndrome with or without acute kidney injury

Cast nephropathy
LCs bind with THP, forming insoluble casts that obstruct tubular lumen and elicit local inflammation, leading to acute kidney injury with or without chronic kidney disease

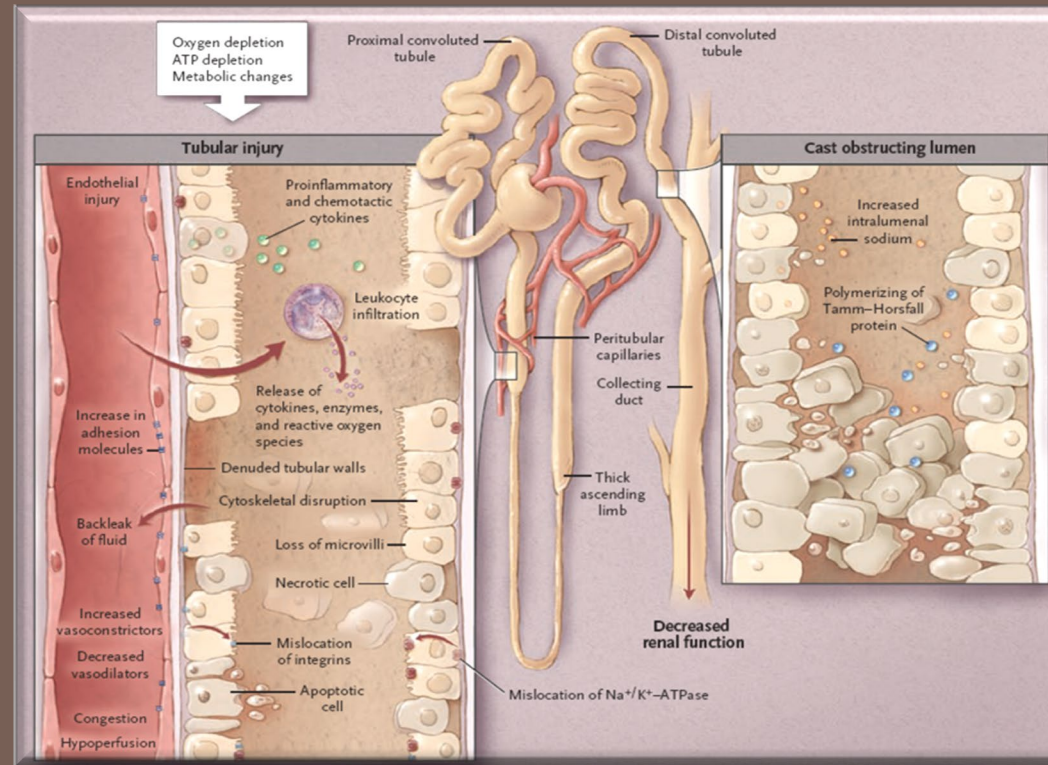


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CAST NEPHROPATHY



DANNO TOSSICO ESOGENO



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AKI Tossica esogena

Sostanze Esogene

IATROGENE



MEZZO DI CONTRASTO

FARMACI

ALTRE

- **Aminoglicosidi**
- **Vancomicina**
- **FANS**
- **CIS-Platino**
- **Anfotericina-B**

- **Mercurio**
- **Arsenico**
- **Uranio**
- **Eroina**
- **Anfetamine**



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AKI-TOSSICA ESOGENA

Hemodynamic AKI (capillary leak syndrome)

IL-2, denileukin difitox

Thrombotic microangiopathy

Antiangiogenesis drugs (bevacizumab and tyrosine kinase inhibitors)

Gemcitabine and cisplatin

Mitomycin C and IFN

Glomeruli

Minimal change disease

IFN

Pamidronate

FSGS

IFN

Pamidronate

Zoledronate (rare)

Tubulointerstitium

Acute tubular necrosis

Platinums, zoledronate, ifosfamide, and mithramycin

Pentostatin, imatinib, diaziquone, and pemetrexed

Tubulopathies

Fanconi syndrome

Cisplatin, ifosfamide, and azacitidine

Diaziquone, imatinib, and pemetrexed

Salt wasting

Cisplatin and azacitidine

Magnesium wasting

Cisplatin, cetuximab, and panitumumab

Nephrogenic diabetes insipidus

Cisplatin, ifosfamide, and pemetrexed

Syndrome of inappropriate antidiuresis

Cyclophosphamide and vincristine

Acute interstitial nephritis

Sorafenib and sunitinib

Crystal nephropathy

Methotrexate



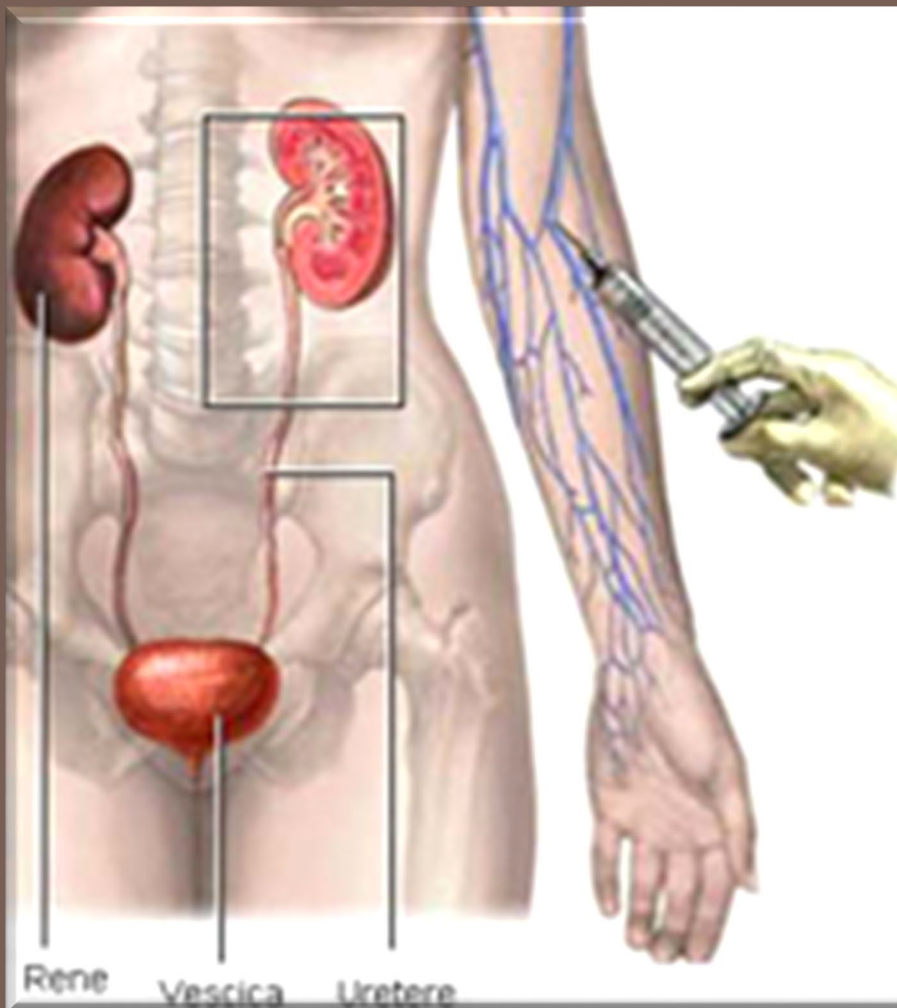
- A. Disidratazione (nausea/vomito, diarrea, asciti e versamenti pleurici massivi ,sepsi, poliuria..)
- B. Alte dosi o dosi prolungate : monitoraggio attento delle condizioni cliniche e dei fattori di rischio
- C. Età avanzata



Carmelo
Libetta

PREVENZIONE

AKI- Tossica esogena



Carmelo
Libetta

NFROPATIA DA MEZZO DI CONTRASTO (NMC)

AKI- Tossica esogena

Patogenesi

- Ridotta perfusione della midollare renale con ipossia delle cellule tubulari (aumentata produzione di endotelina e adenosina, ridotta sintesi di prostaglandine e di ossido nitrico)
- Elevata escrezione di acido urico che precipita a livello tubulare
- Tossicità del mezzo di contrasto diretta verso la cellula tubulare



Carmelo
Libetta

NFROPATIA DA MEZZO DI CONTRASTO (NMC)

Incidenza

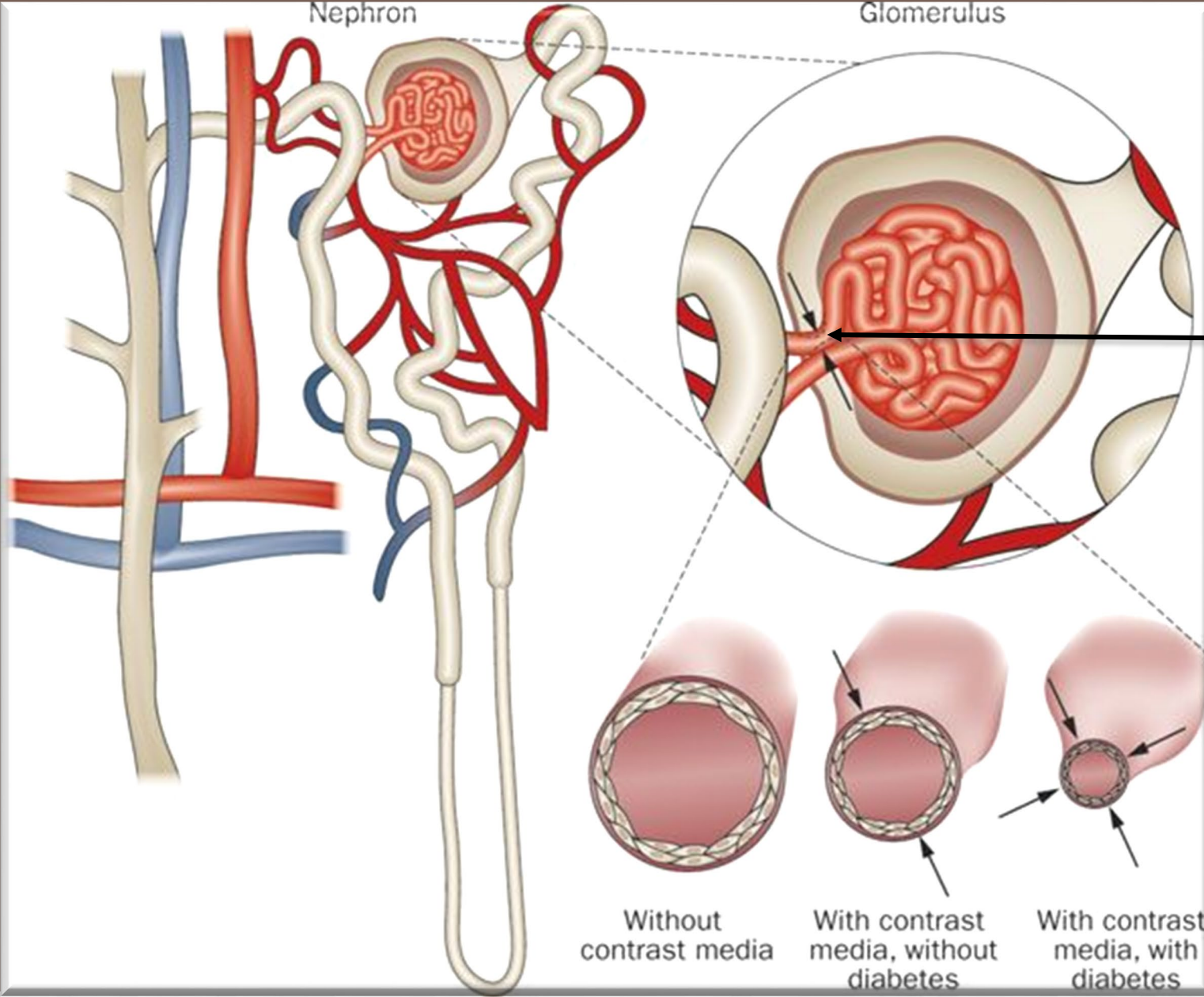
- 20% per pazienti con creatininemia di 2,0 mg/dl
- 50% per pazienti con creatininemia > di 5,0 mg/dl

Fattori di Rischio

- **Età avanzata**
- **Diabete Mellito**
- **Scompenso cardiaco congestizio**
- **Mieloma multiplo**
- **Ipovolemia**
- **Scompenso cardiaco a bassa portata**
- **Studi contrastografici ad alte dosi**

FATTORI DI RISCHIO





MC

Without contrast media

With contrast media, without diabetes

With contrast media, with diabetes

Carmelo Libetta

VASOCOSTRIZIONE AA



❖ **Sospendere INIBITORI SRAA e DIURETICI**

❖ **IDRATAZIONE**

❖ **Acetilcisteina come antiossidante, contro la produzione di radicali liberi prodotti dall'azione tossica del m.d.c. sulle cellule tubulari (la dose è di 600 mg 2 volte al giorno prima, dell'esame e il giorno dopo)**

Mezzi di contrasto

- **Bassa viscosità**
- **Bassa osmolarità**



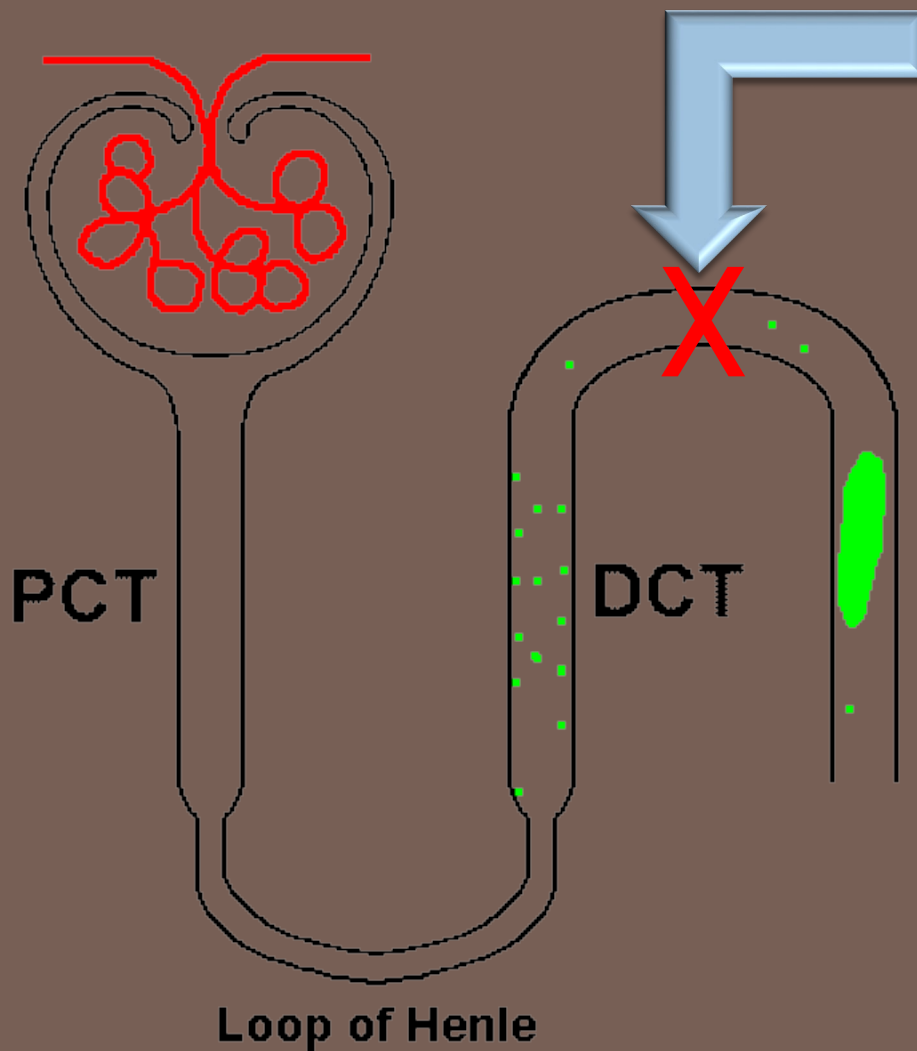
creatininemia

- subito prima dell' esame
- dopo 48 h e 6 giorni



Carmelo
Libetta

Sorveglianza Danno NMC



ANTIBIOTICI

- Aminoglicosidi
- Vancomicina

FANS

METALLI PESANTI

- CIS Platino
- Litio

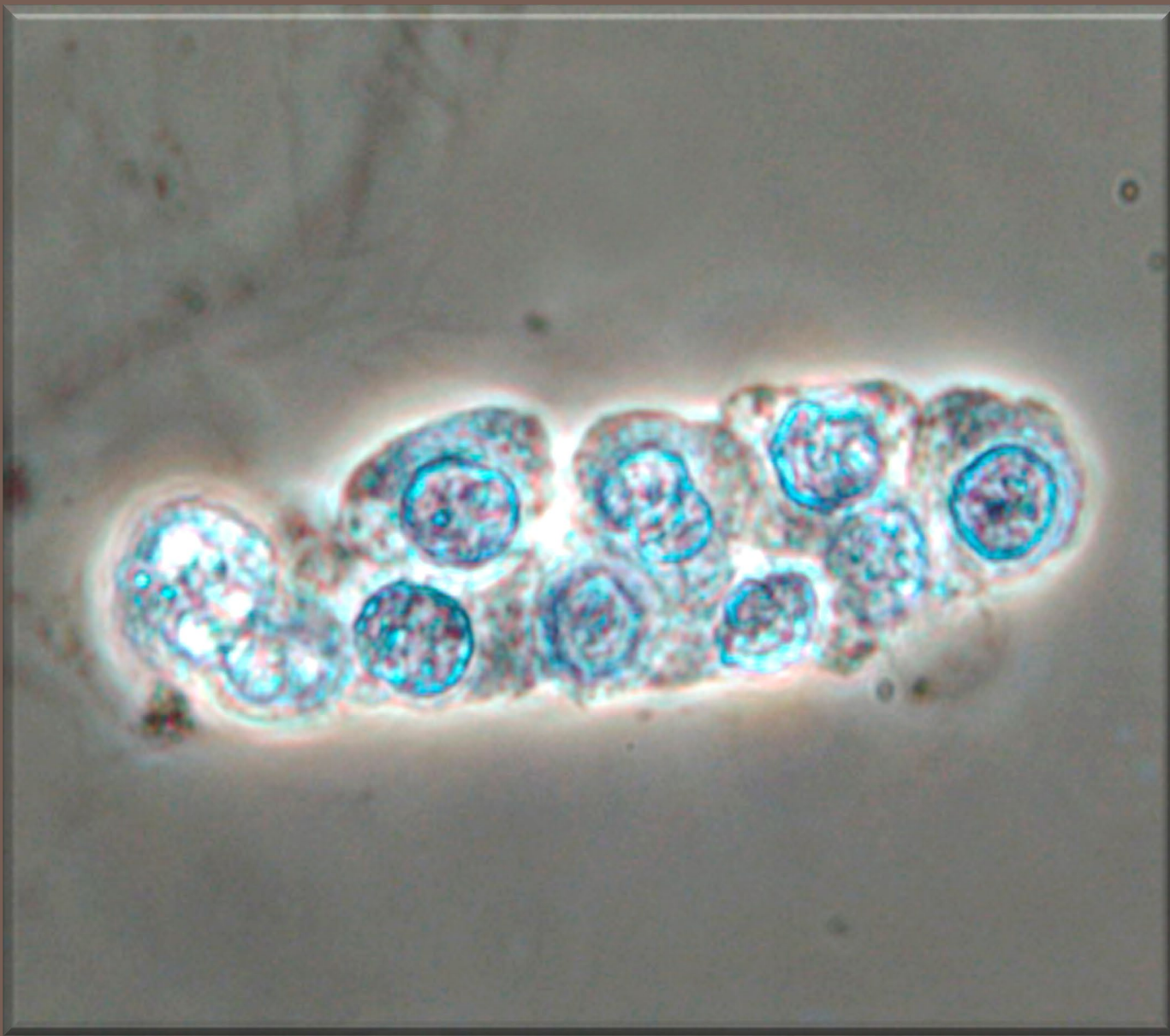
INIBITORI CALCINEURINE

- Ciclosporina
- Tacrolimus



Carmelo
Libetta

FARMACI NEFROTOSSICI

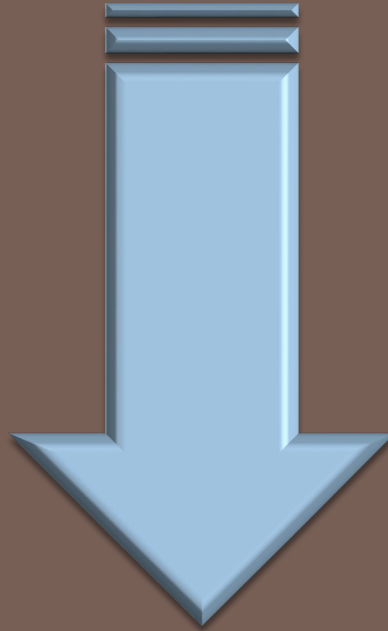


Carmelo
Libetta

Es. urine: cast di cellule tubulari



NECROSI TUBULARE ACUTA

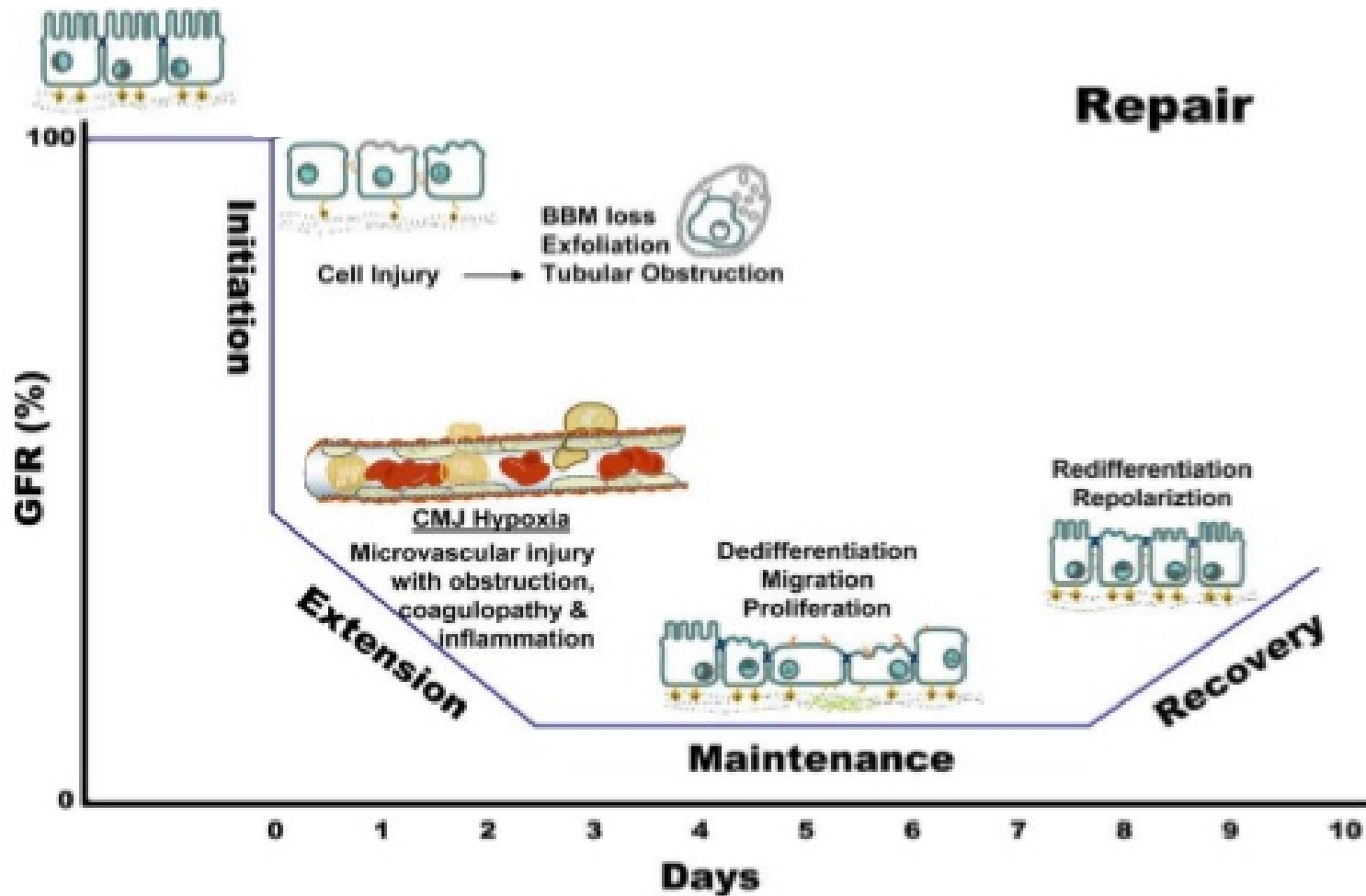


RIGENERAZIONE TUBULARE



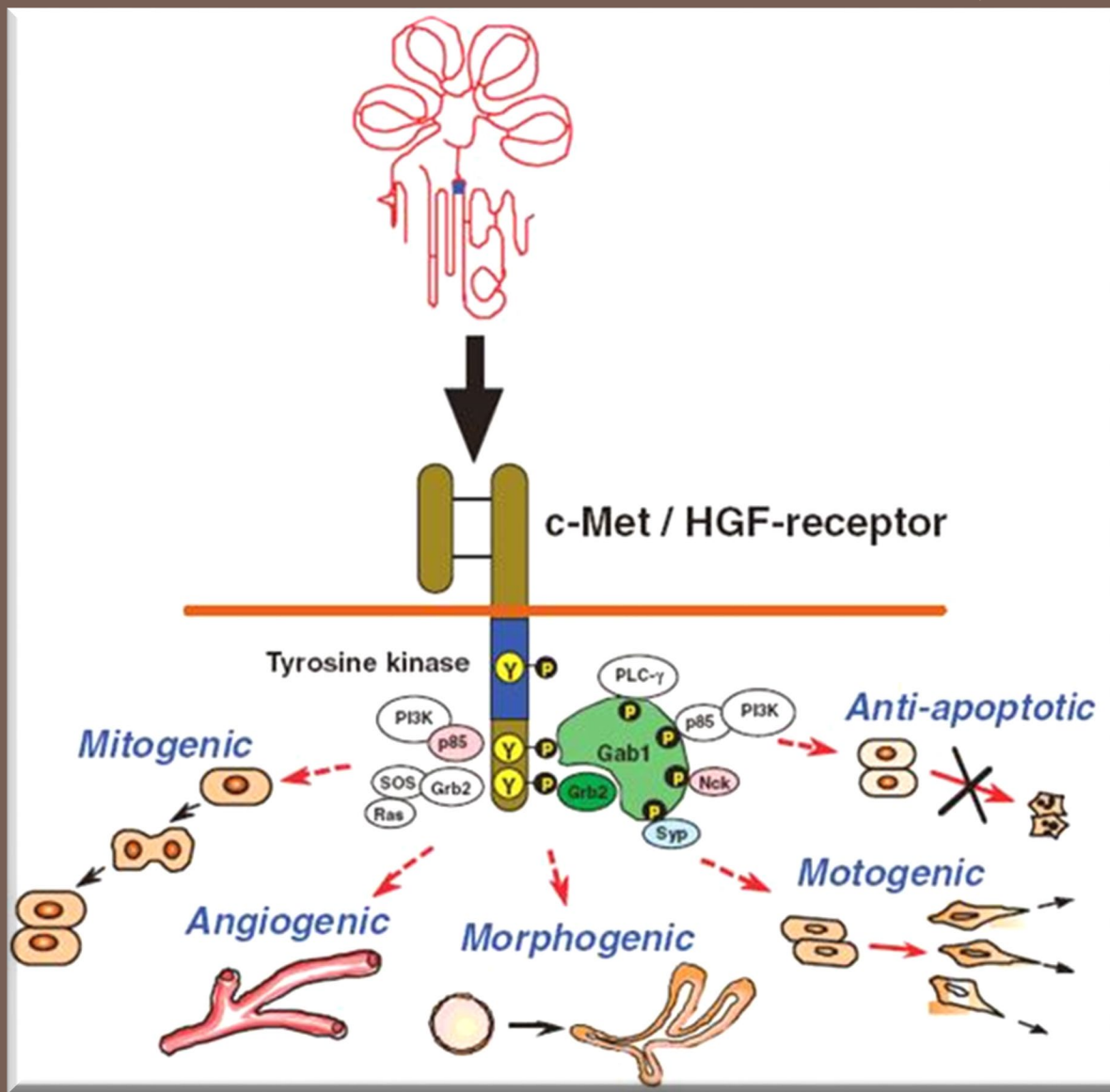
**Carmelo
Libetta**

AKI Danno reversibile



Carmelo
Libetta

RIGENERAZIONE TUBULARE



Carmelo
Libetta

HGF

HGF Biological effects

Source of HGF

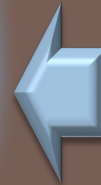
Target cells



Mesenchymal
cell



Tubular
cell



Mitogenic

Tubulogenesis

Migration

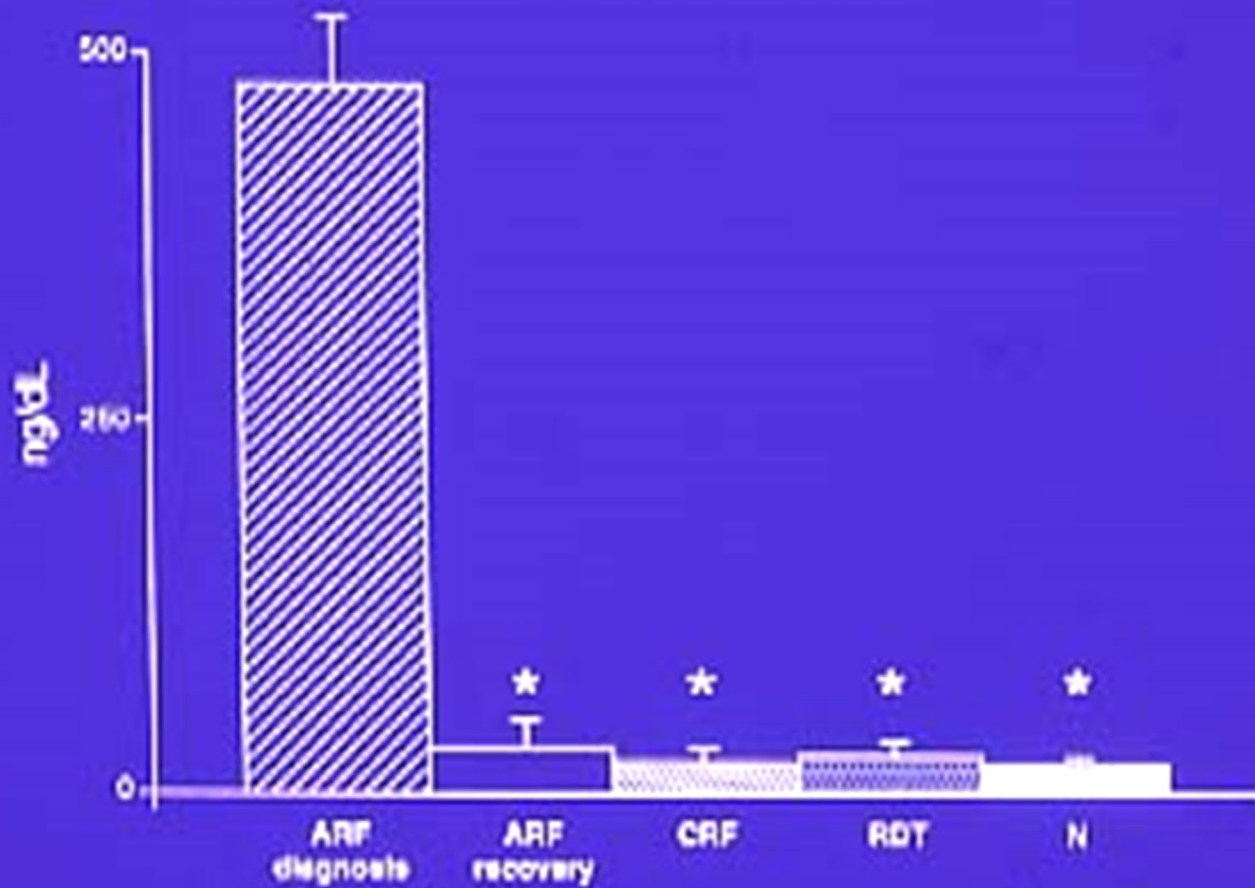
Anti-apoptosis



Carmelo
Libetta

ATN/HGF

Serum level of HGF in patients with acute renal failure and controls



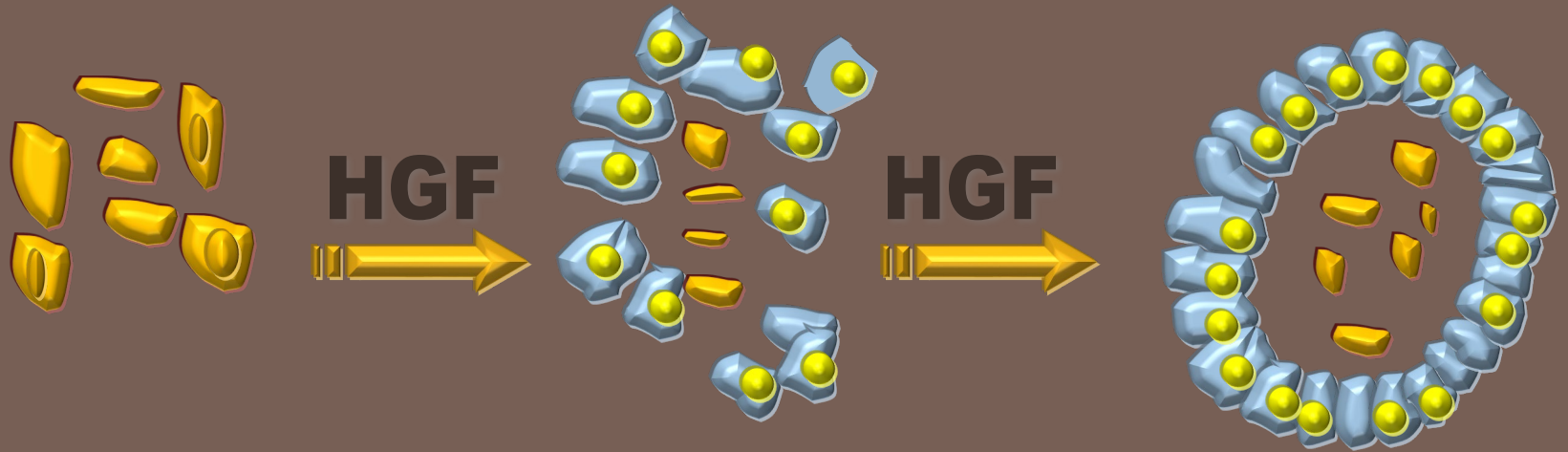
* $p < 0,001$ vs ARF (diagnosis)

Libetta et al. *Nephron* 80:41, 1998



Carmelo
Libetta

AKI/HGF



Tubulo necrotico

Proliferaazione cellule
Mesenchimali

Allineamento
cellule tubulari



Carmelo
Libetta

HGF e riparazione tubulo



Carmelo
Libetta

Diagnosi differenziale AKI

- **Nausea? Vomito? Diarrea? (Da quanti giorni)**
- **Alterazioni cardiologiche?**
- **Alterazioni della diuresi? (Da quanti giorni)**
- **Assunzione di farmaci?**
- **Esami strumentali con mezzo di contrasto?**



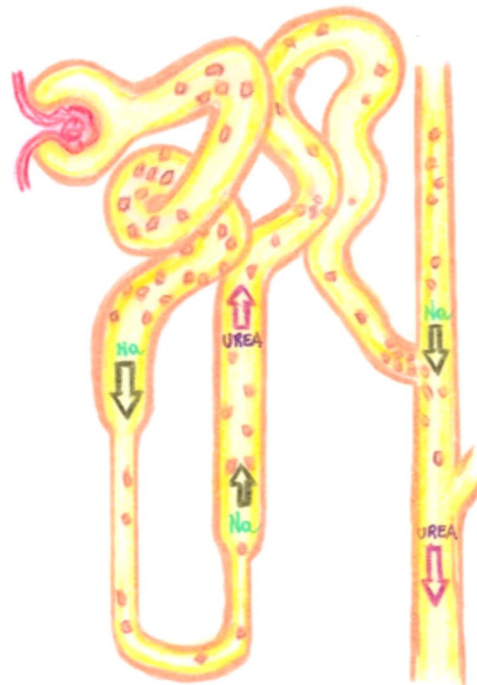
**Carmelo
Libetta**

Anamnesi

AKI PRE-RENALE



AKI RENALE



C. Libetta

$UNa < 20 \text{ mEq/L}$

$FeNa < 1\%$

$UOsm > 500 \text{ mOsm/Kg}$

$BUN/Cr \gg 20:1$

$UNa \geq 50 \text{ mEq/l}$

$FeNa > 1\%$

$UOsm < 500 \text{ mOsm/Kg}$

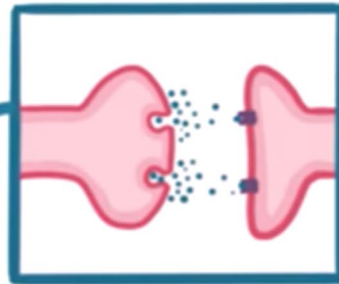
$BUN/Cr \leq 10:1$

Carmelo
Libetta

Diagnosi differenziale AKI

Dopamina

Basse dosi



NEUROTRANSMITTER



← SYNTHESIZED →



KIDNEY

CONSTRICTS
CAPILLARIES IN

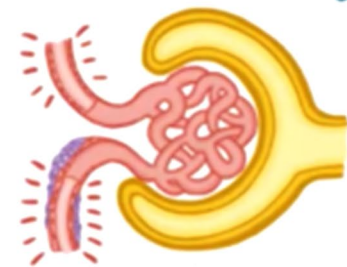
SKIN

MUSCLE

DILATES
SMALL VESSELS IN

D1

HEART



Carmelo
Libetta

Terapia



Basse-dosi di dopamina (2 $\mu\text{g}/\text{kg}/\text{min}$ e.v.)

- Incremento flusso renale per attivazione recettori dopaminergici dei vasi renali
- Incremento dell'escrezione di Na^+ e H_2O
- Inibizione riassorbimento Na^+ tubulo prossimale, branca spessa ascendente ansa Henle, dotto collettore corticale con riduzione consumo di O_2
- Incremento produzione renale di PgE_2 vasodilatatorie



Carmelo
Libetta

Terapia AKI renale

DANNO VASCOLARE

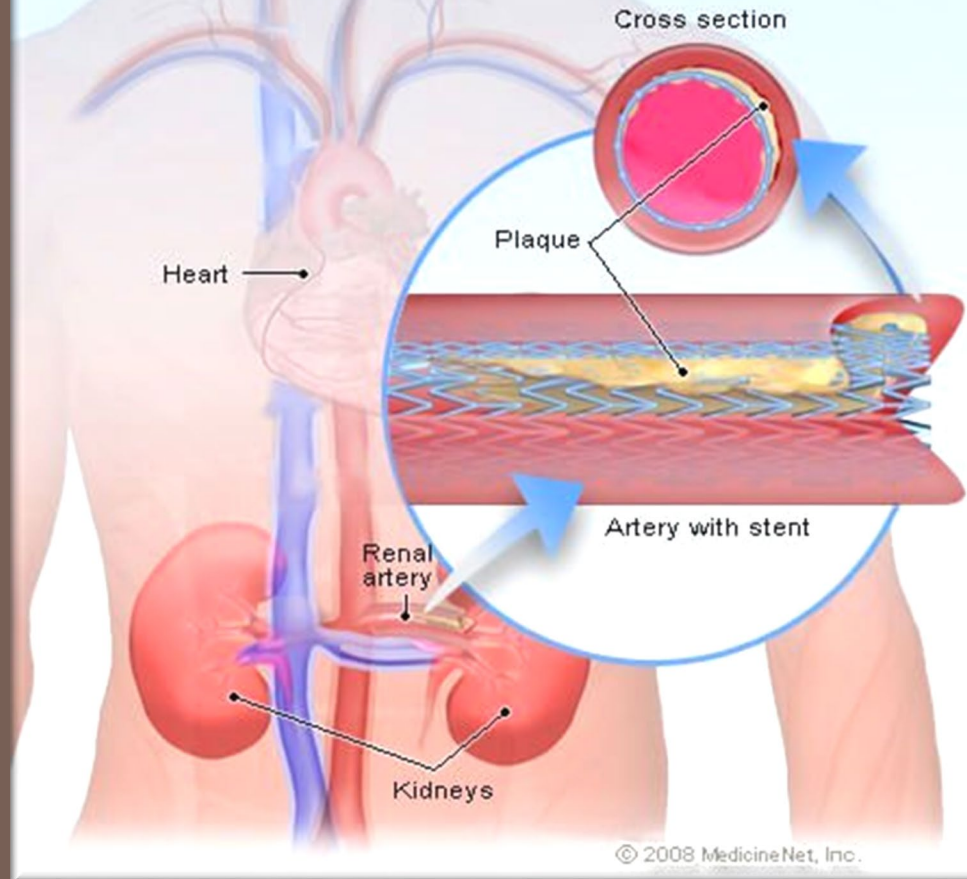
- ARTERIE RENALI
- PICCOLI VASI



Carmelo
Libetta

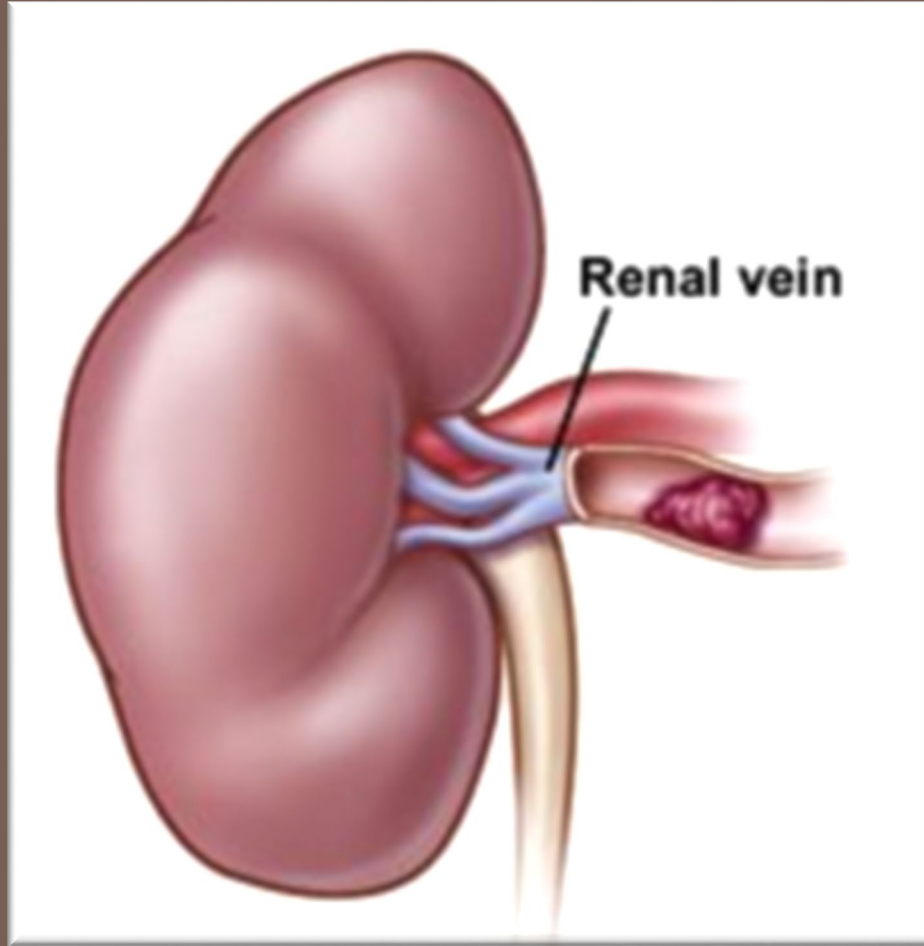
AKI ORGANICO

Renal Artery Stenosis



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Libetta

AKI ORGANICO



Carmelo
Libetta

AKI ORGANICO VASCOLARE

Necrosi Corticale Acuta

Eziologia

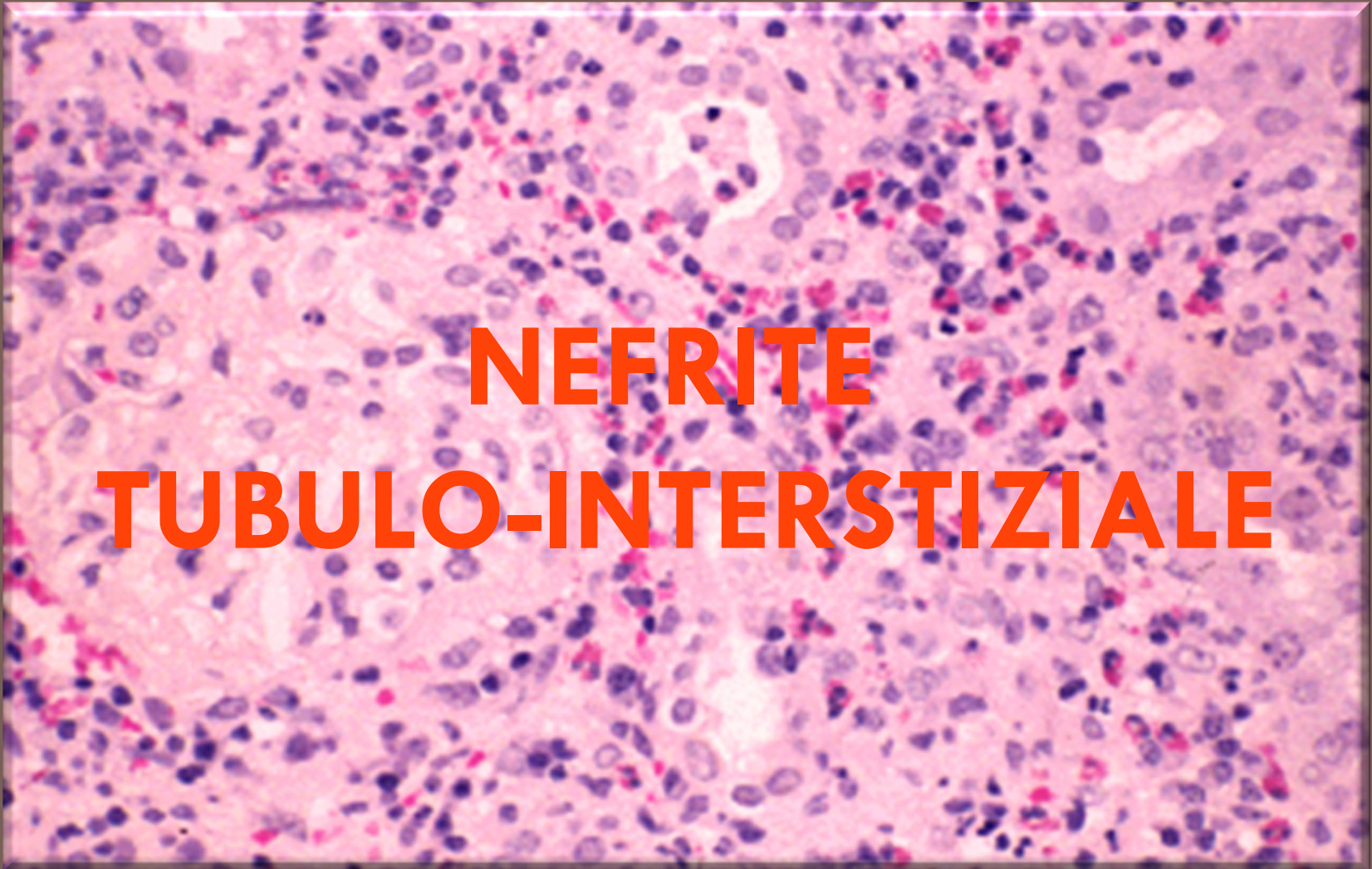
- **Gravidanza (distacco precoce di placenta, emorragia post-partum, aborto settico)**
- **Infezioni (Sepsi da batteri GRAM negativi)**
- **Pancreatiti**
- **Gastroenteriti acute in età pediatrica**
- **Crisi emolitiche, Sindrome Emolitico-Uremica**
- **Rigetto Iperacuto nel Trapianto Renale**



Patogenesi

- **Ischemia prolungata con ipoperfusione della corticale**
- **Attivazione del sistema coagulativo (esempio: tromboplastina placentare in corso di gravidanza)**
- **Deposizione di fibrina e formazione di trombi intravascolari alle arteriole, piccole arterie e fino alle arterie di calibro maggiore**
- **Necrosi massiva di tutte le strutture componenti il parenchima della corticale renale**





**NEFRITE
TUBULO-INTERSTIZIALE**

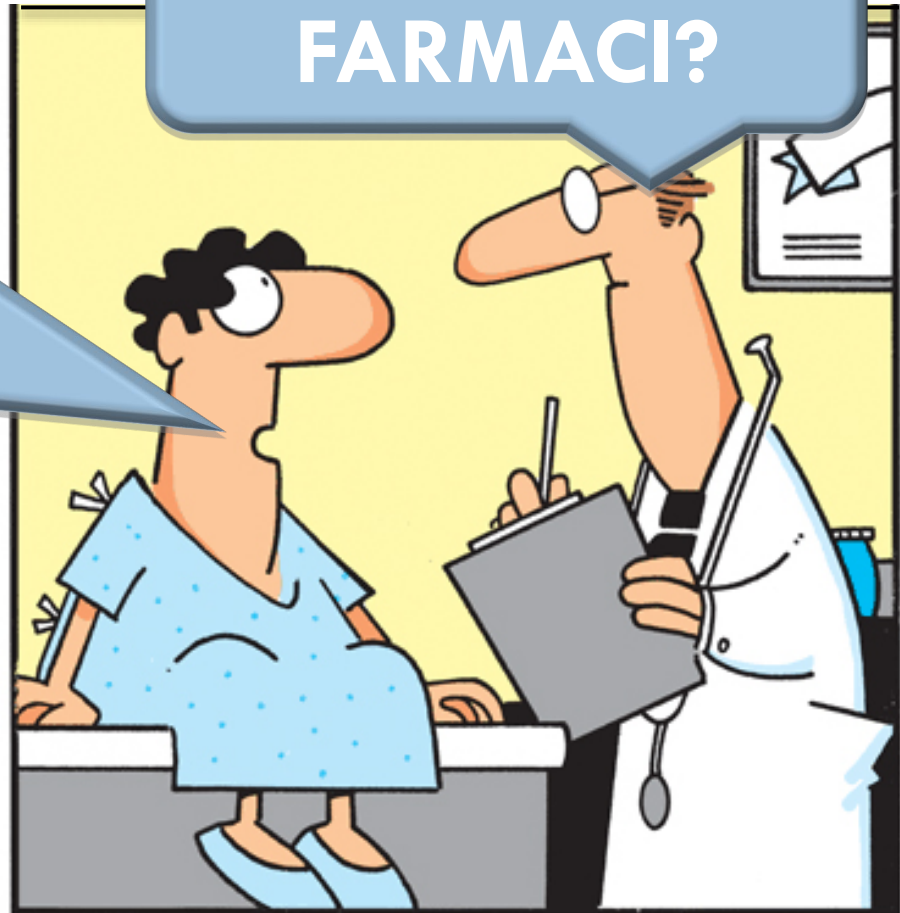


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AKI RENALE

**DOTTORE HO
FEBBRICOLA E
MACCHIOLONE
ROSSE SULLA
PELLE**

**HA ASSUNTO
FARMACI?**



**Carmelo
Libetta**

Sintomi Nefrite Interstiziale



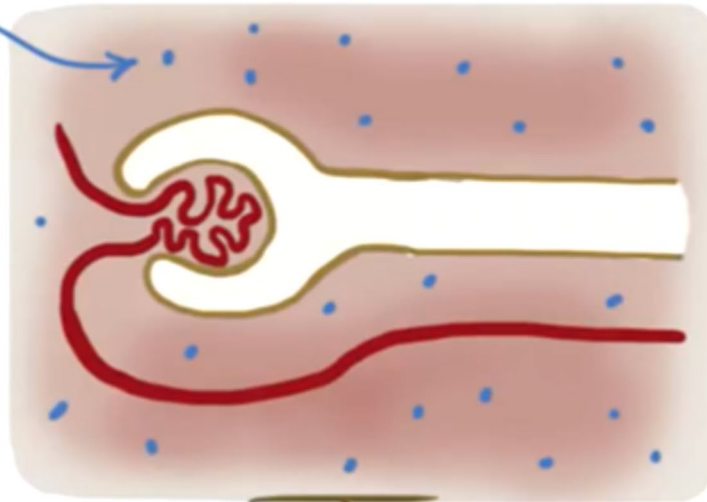
* ACUTE INTERSTITIAL NEPHRITIS *

days to weeks

inflammation

INFILTRATION of IMMUNE CELLS

- ↳ neutrophils
- ↳ eosinophils



INTERSTITIUM

MEDICATIONS

- ↳ NSAIDS
- ↳ PENICILLIN
- ↳ DIURETICS

↓
TYPE I or IV
HYPERSENSITIVITY

- ↓
- * OLIGURIA
 - * EOSINOPHILURIA

Carmelo
Libetta

NEFRITE INTERSTIZIALE

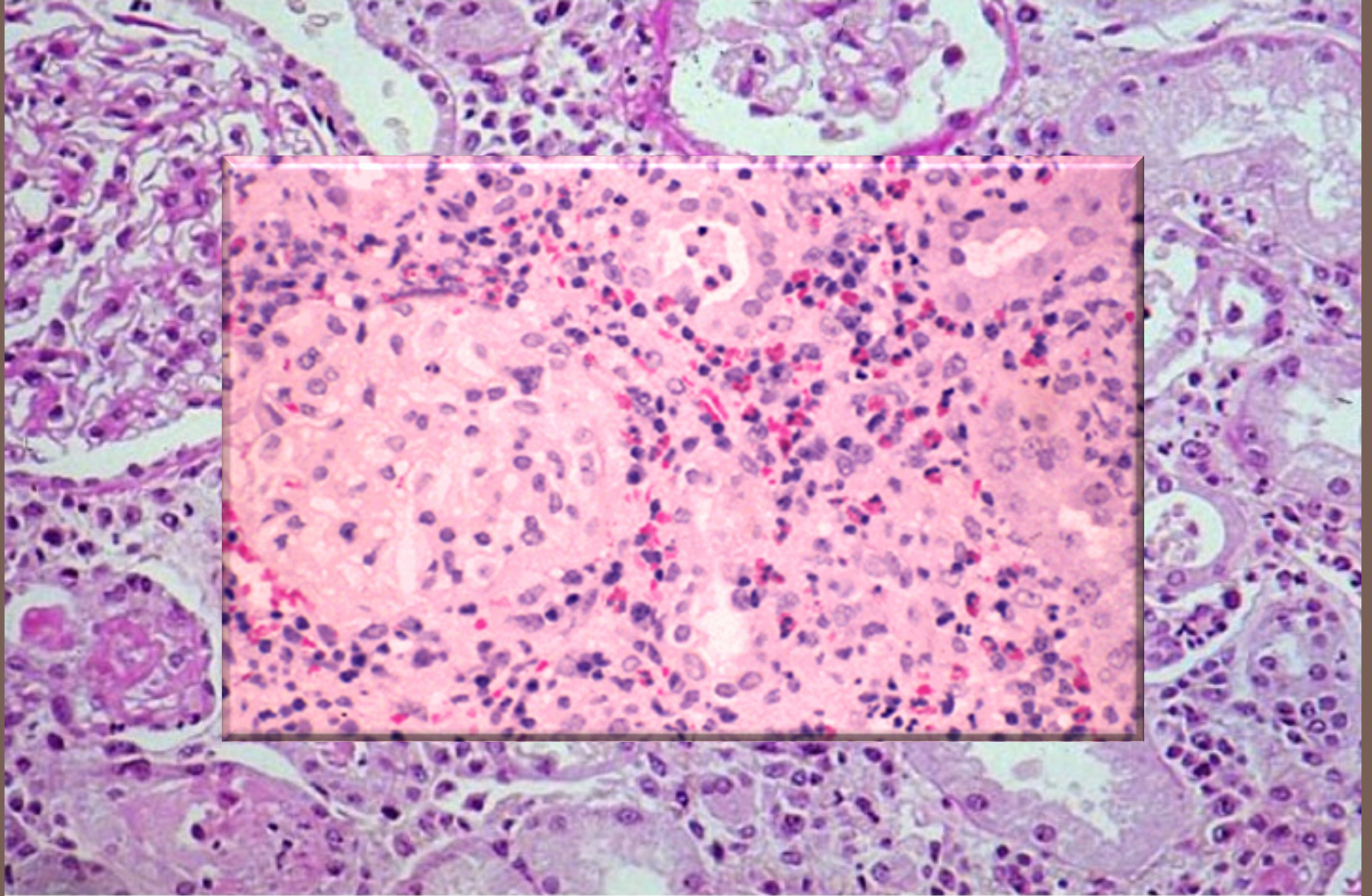


- PENICILLINS**
- SULFONAMIDES**
- CEPHALOSPORIN**
- NSAID**
- ALLOPURINOL**
- PHENYTOIN**
- THIAZIDES**
- FUROSEMIDE**
- CIMETIDINE**



- Fever**
- Rash**
- Eosinophilia**
- Eosinophiluria**
- WBC Casts**



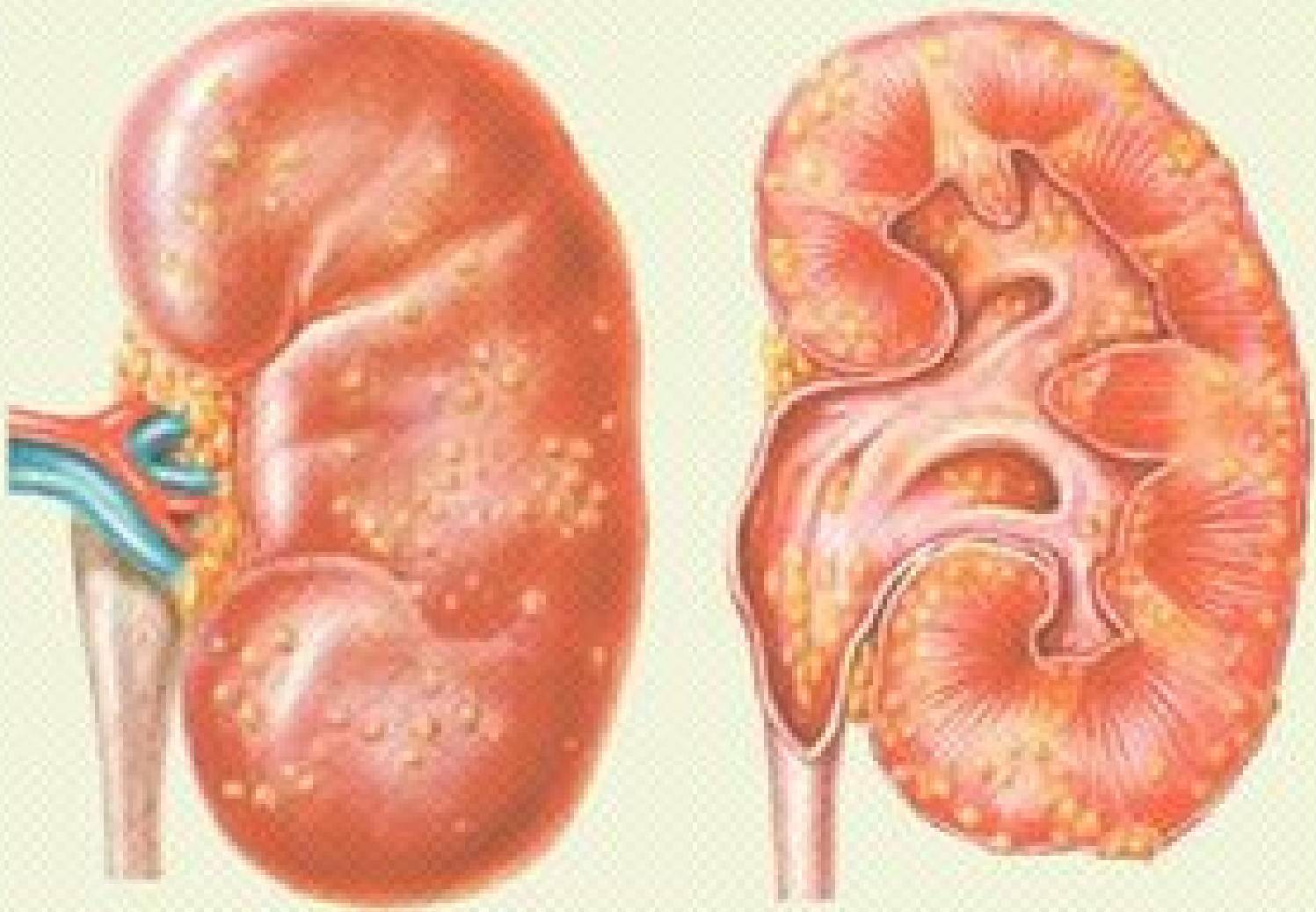


Carmelo
Libetta

Nefrite interstiziale



Pielonefrite



Carmelo
Libetta

Nefrite Tubulo-interstiziale acuta



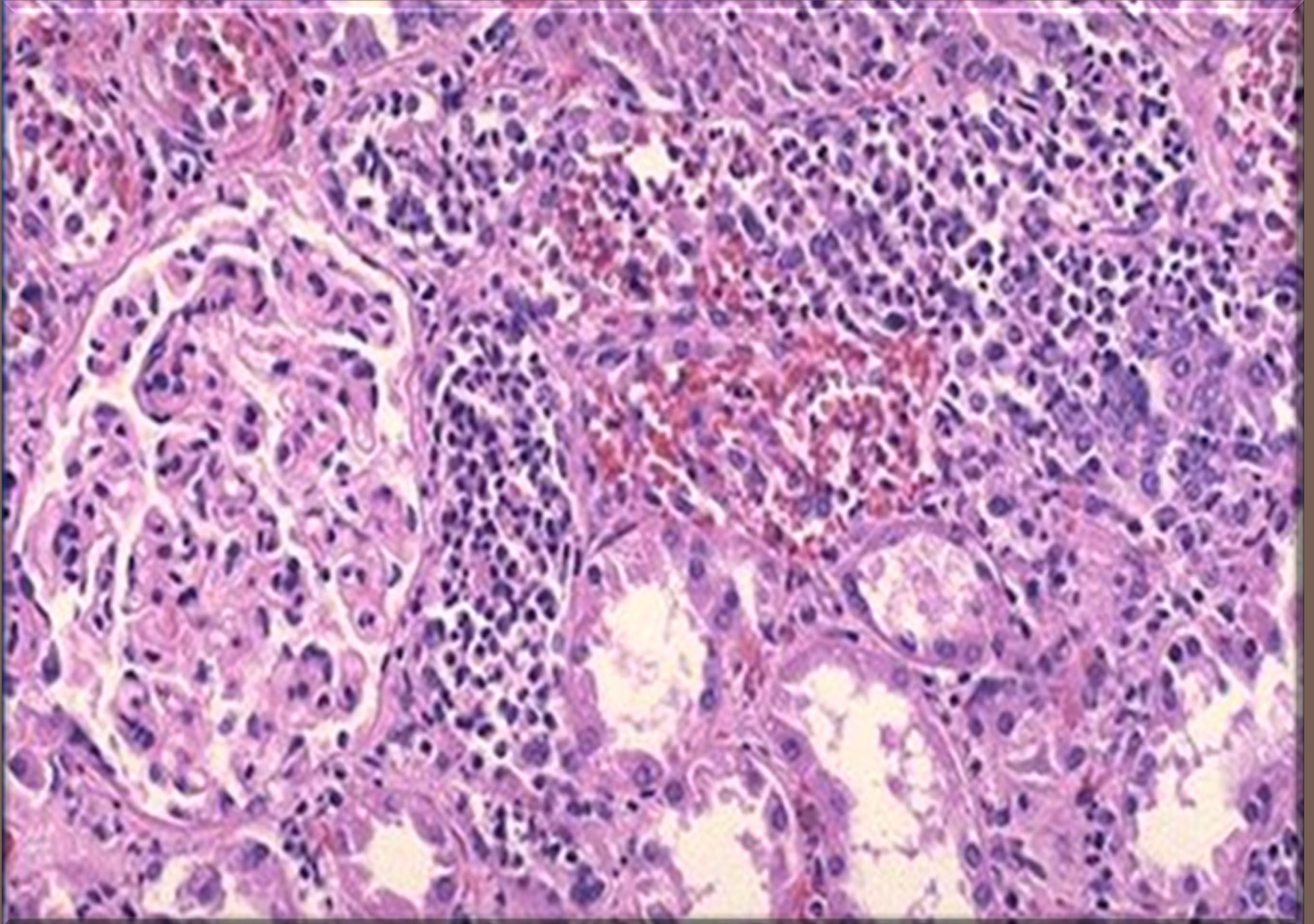
DOTTORE
DA QULCHE GIORNO
HO UN DOLORE AL
FIANCO DX, FEBBRE
ALTA E NAUSEA.



Carmelo
Libetta

Sintomi Pielonefrite





Carmelo
Libetta

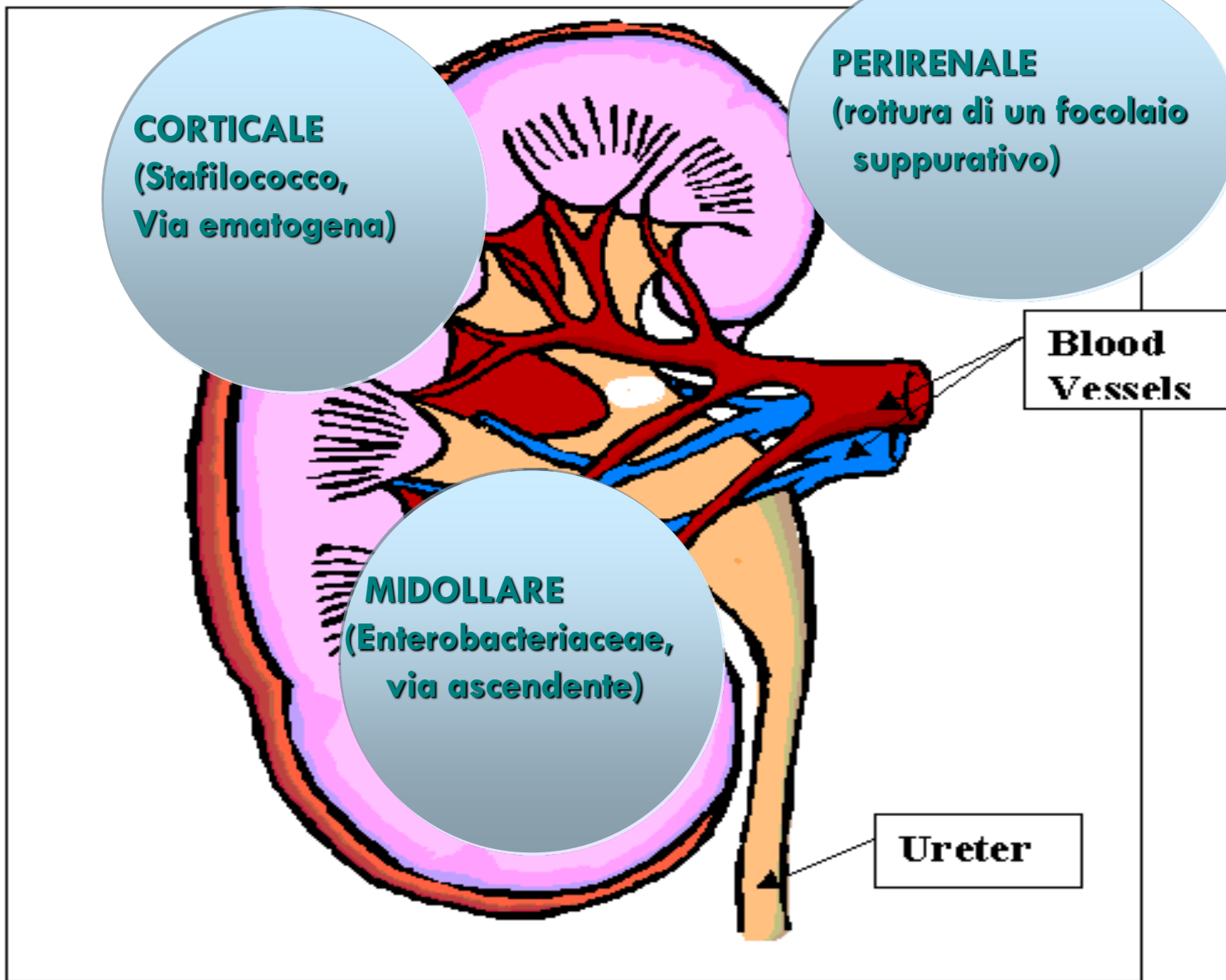
Pielonefrite





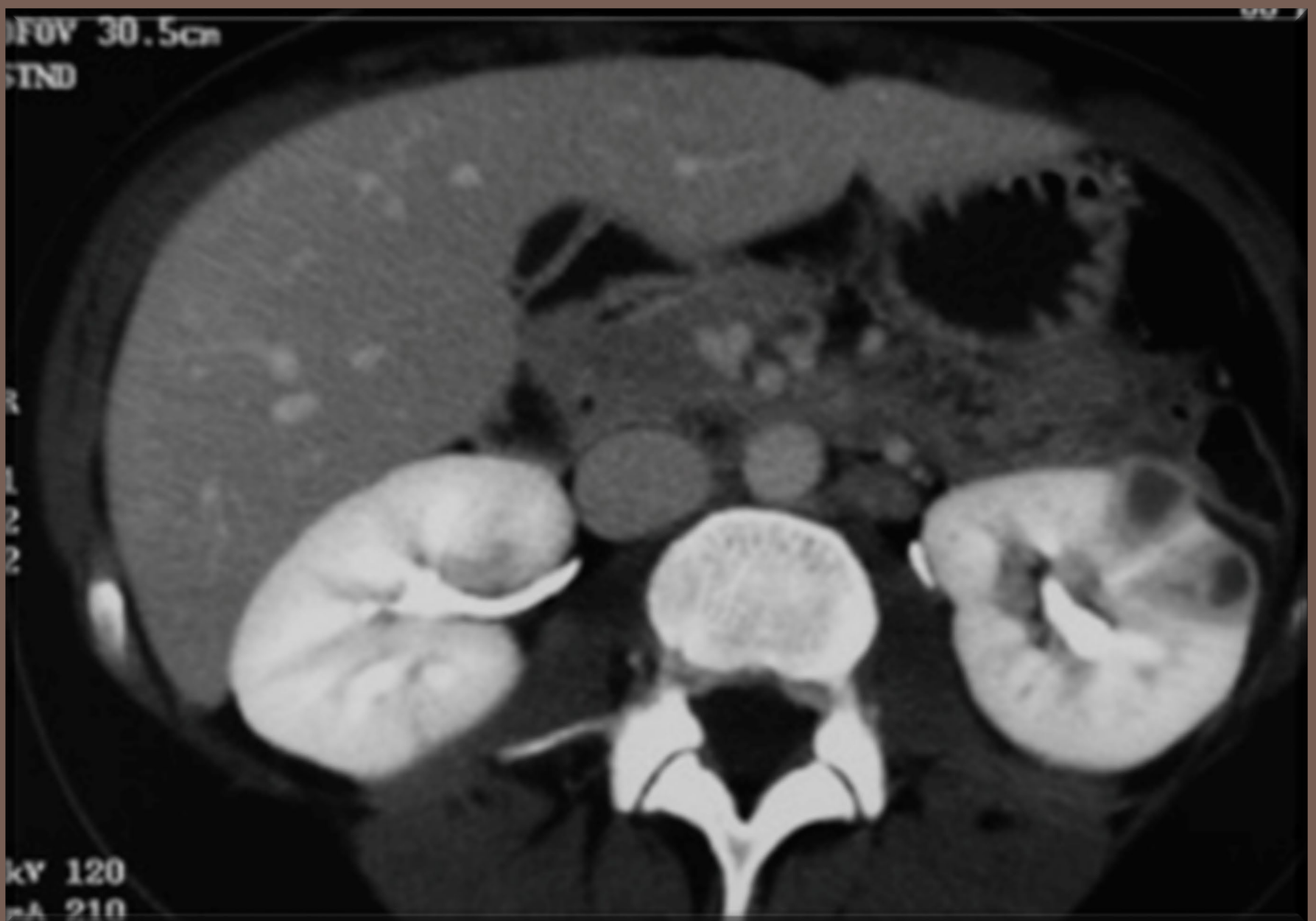
Carmelo
Libetta

Pielonefrite



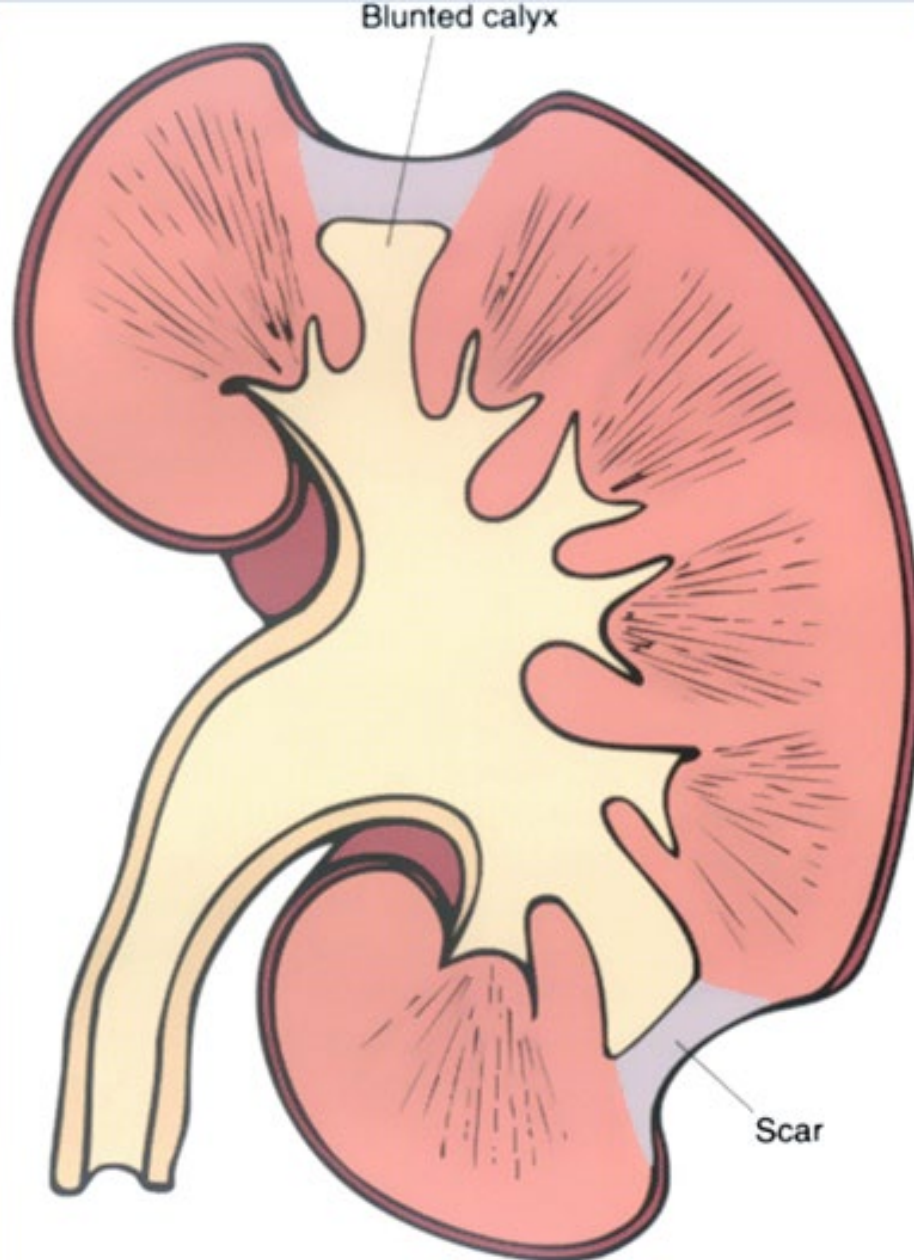
Carmelo
Libetta

Ascesso renale



Carmelo
Libetta

Ascessi renali



Carmelo
Libetta

Cicatrici parenchimali



CAUSE GLOMERULARI



Carmelo
Libetta

AKI RENALE

Glomerulonefrite Acuta



Primitive

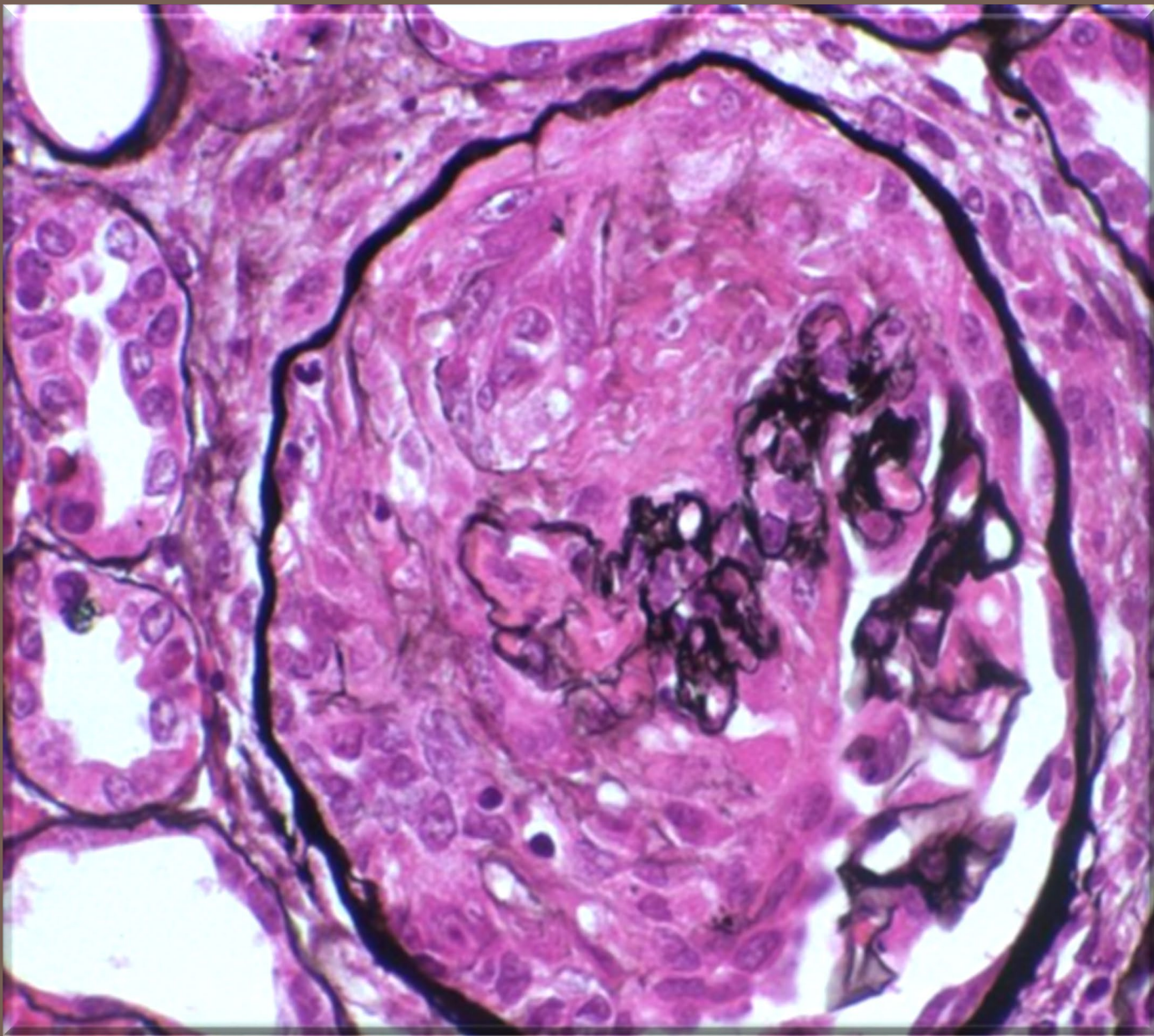
- **Glomerulonefrite Post-streptococcica**
- **Nefropatia da IgA**
- **Sindrome di Goodpasture**



Secondarie

- **Vasculiti**
- **LES**
- **Porpora di Schonlein Henoch**





Carmelo
Libetta

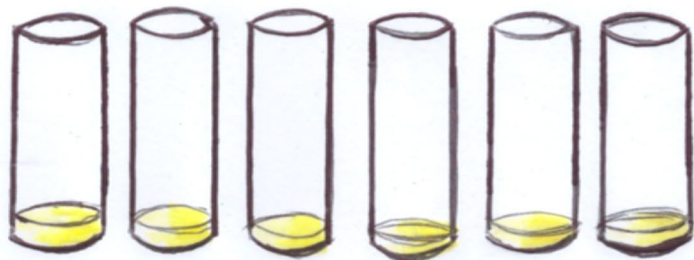
DANNO GLOMERULARE



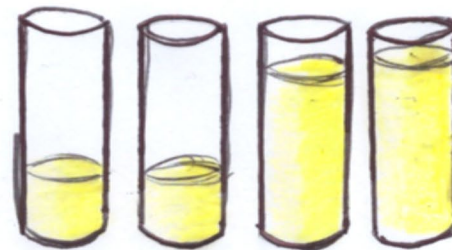
DIURESI



↑
DANNO
RENALE



1-4 SETTIMANE



RIPRESA
FUNZIONALE



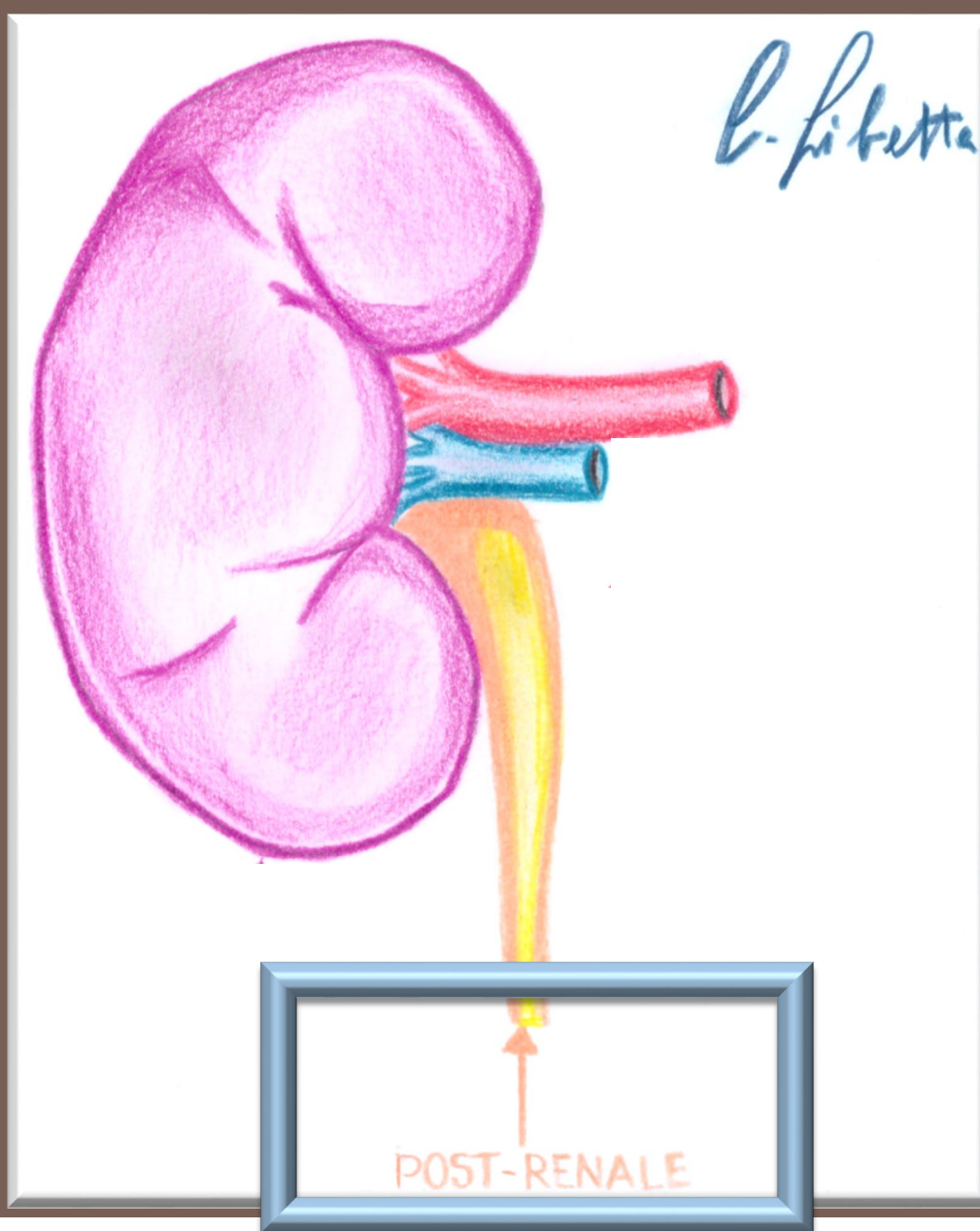
DIALISI

P. Libetta



Carmelo
Libetta

AKI RENALE (recovery)



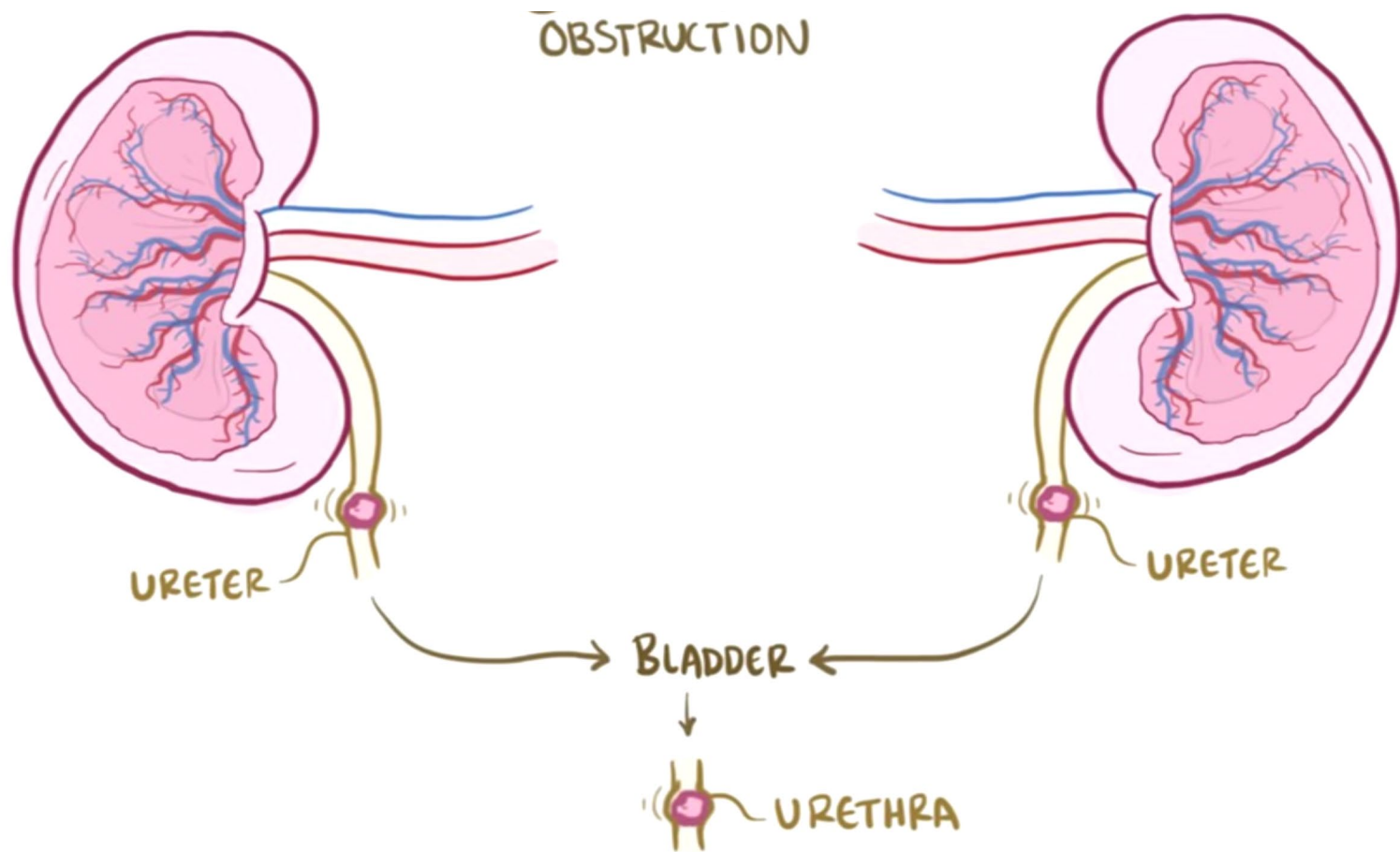
L. Libetta

POST-RENALE



Carmelo
Libetta

AKI POST-RENALE



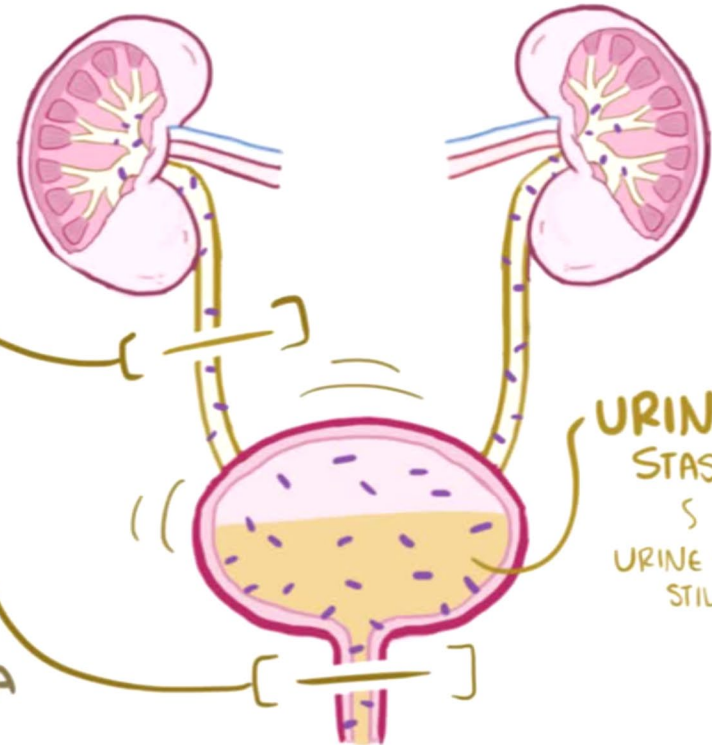
Carmelo
Libetta

AKI POST-RENALE



KIDNEY OBSTRUCTION

MONOLATERAL



BILATERAL OBSTRUCTION

↳ affects BOTH KIDNEYS

* CONGENITAL MALFORMATION

↳ POSTERIOR URETHRAL VALVE

* BENIGN PROSTATIC HYPERPLASIA

* CERVICAL CARCINOMA

MEN

WOMEN

URINARY
STASIS

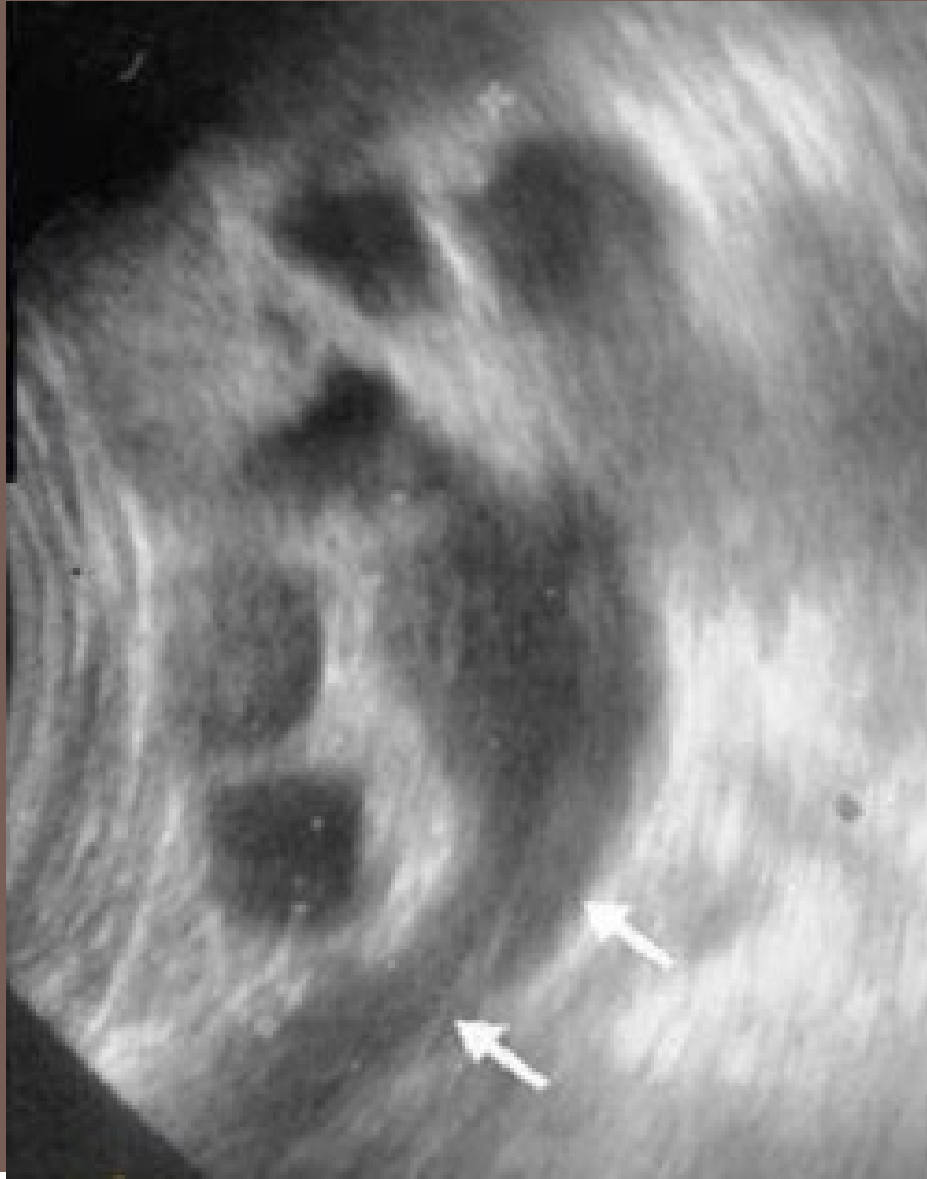
↳
URINE STANDS
STILL

Carmelo
Libetta

AKI Post-renale



DIAGNOSI



Carmelo
Libetta

AKI POST-RENALE



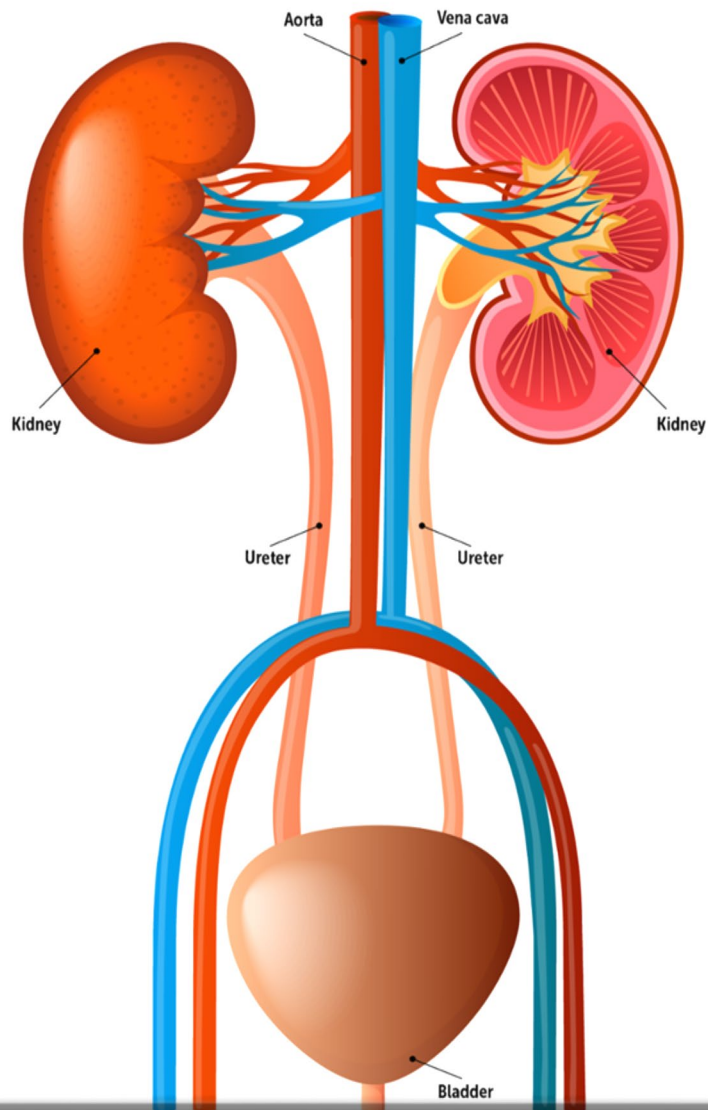


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Libetta

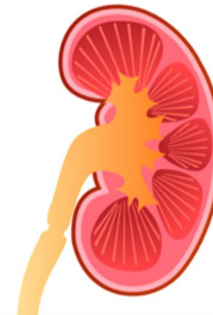
Dilatazione vie urinarie



Normal



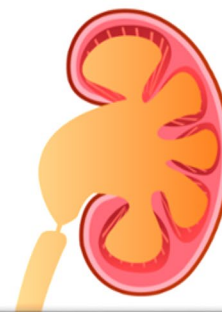
Mild



Moderate



Severe



Carmelo
Libetta

IDRO-URETERO-NEFROSI



- **Anuria se ostruzione completa**
- **Diuresi conservata se ostruzione parziale monolaterale**
- **Poliuria post-disostruzione**

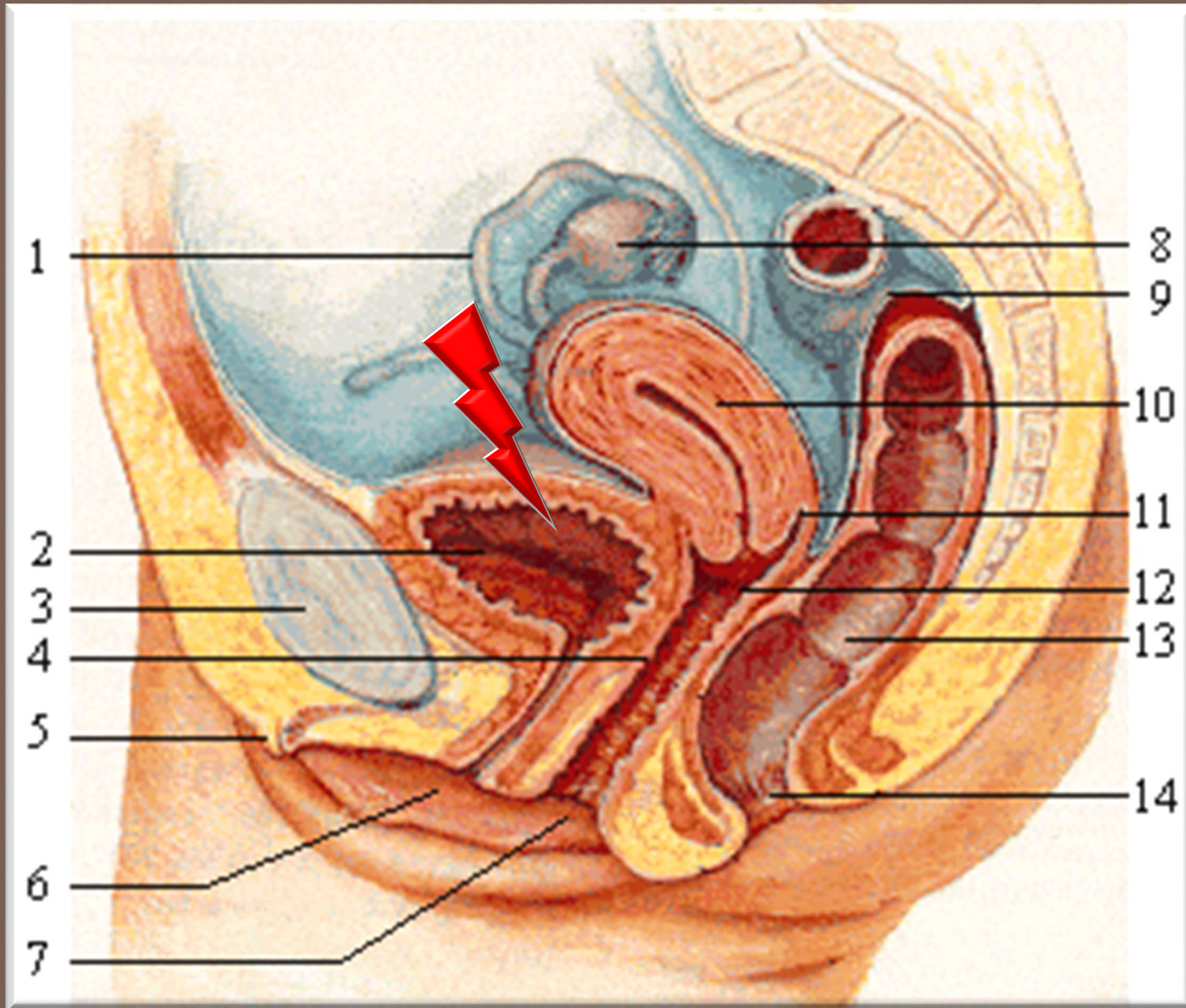


- **La ripresa della funzione renale dopo disostruzione dipende dalla rapidità di intervento.**
- **L'incidenza nel sesso maschile è maggiore.**



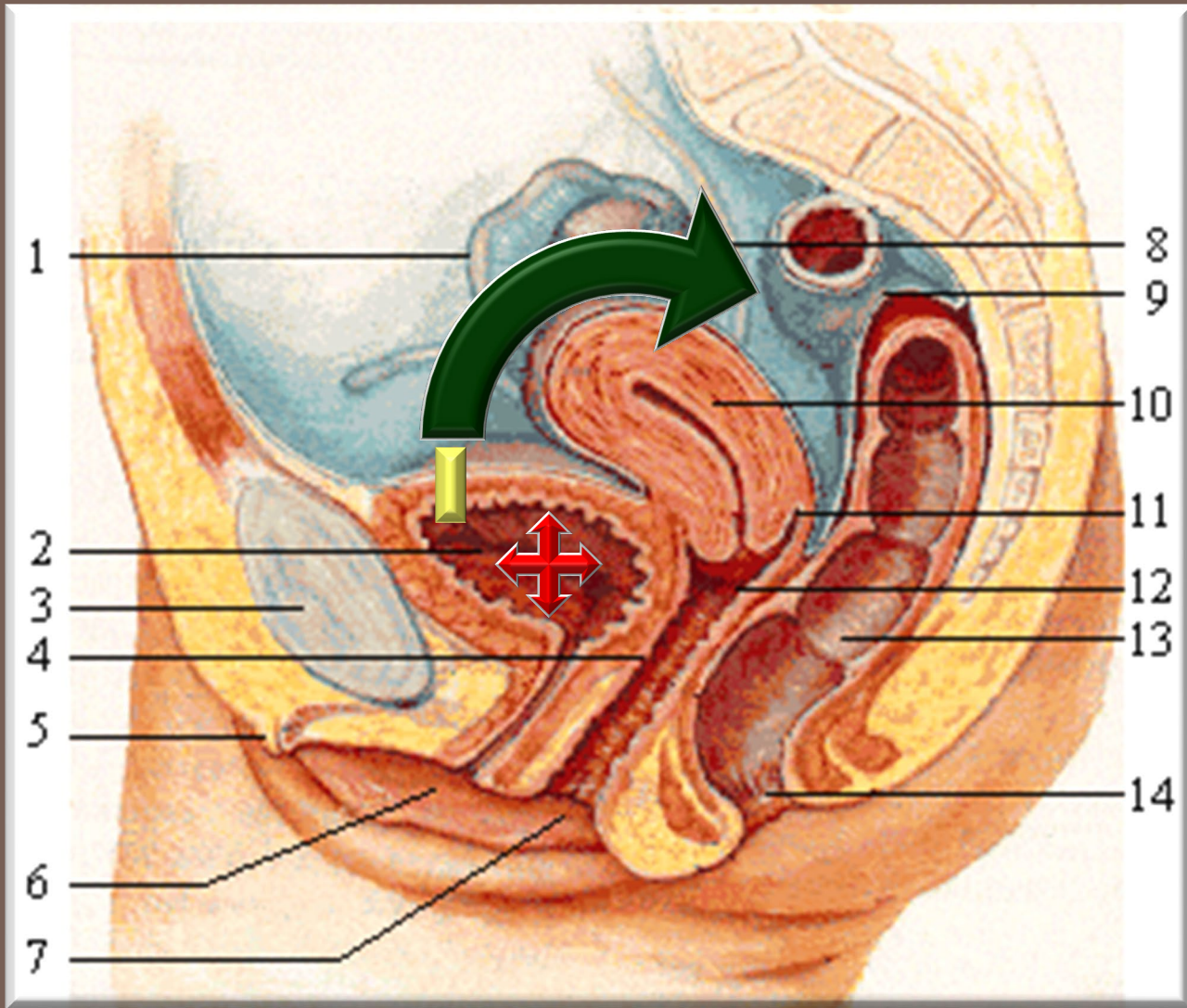
**Carmelo
Libetta**

Caratteristiche AKI Post-renale



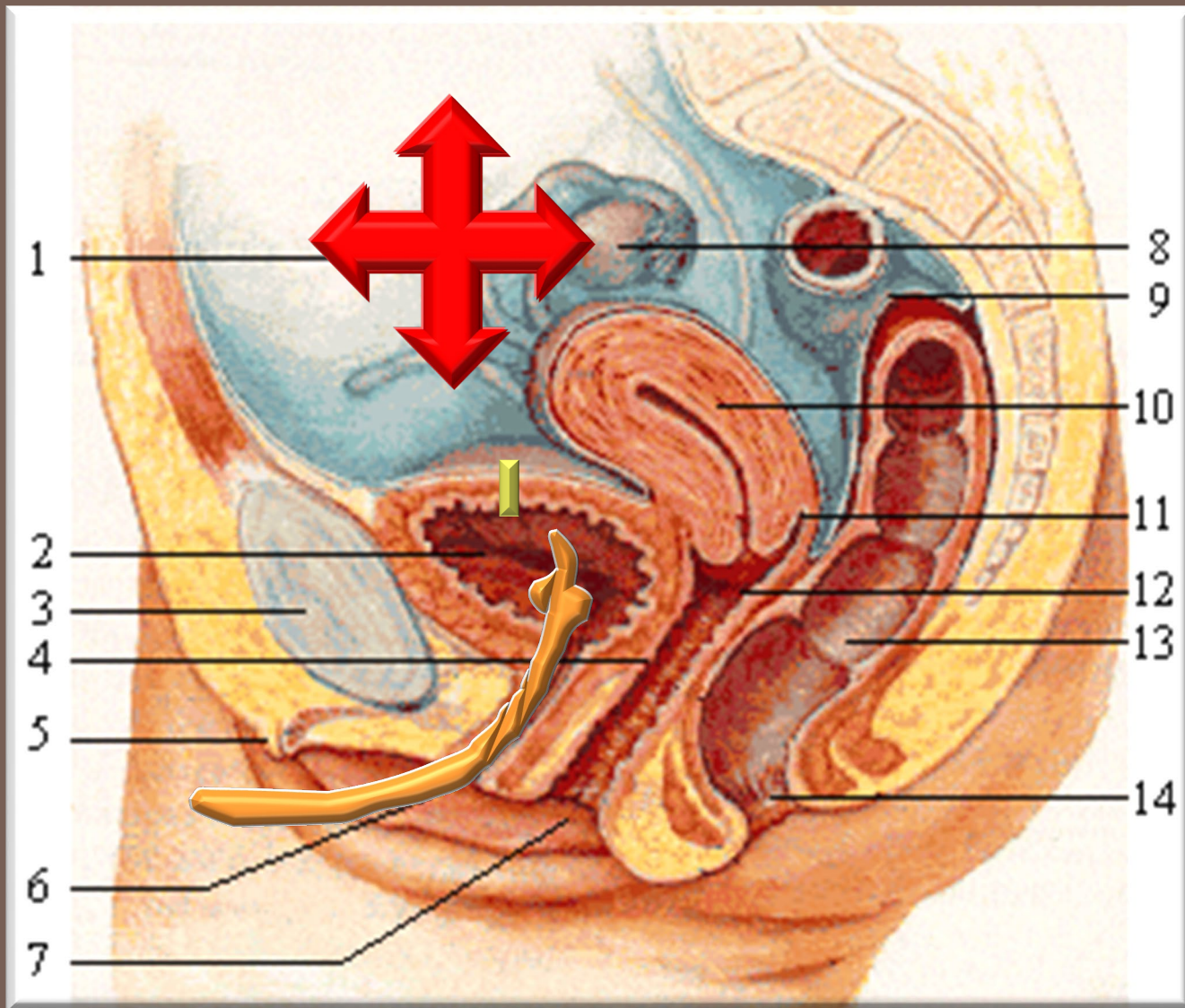
Carmelo
Libetta

PSEUDO-AKI



Carmelo
Libetta

PSEUDO-AKI



Carmelo
Libetta

PSEUDO-AKI

Anamnesi:

traumi o interventi addominali, seguiti ascite e alterazione della funzione renale.



Carmelo
Libetta

QUANDO SOSPETTARLA



Carmelo
Libetta

CISTOGRAFIA RETROGRADA





5-6%



Carmelo
Libetta

AKI: Quando la terapia sostitutiva

Anuria (diuresi < 50 ml/12 ore)

Iperpotassiemia grave ($K^+ > 7$ mEq/L)

Iperazotemia (urea > 200 mg/dl)

Acidosi lattica

Edema polmonare acuto (non responsivo alla terapia diuretica)

Intossicazione da sostanze dializzabili



- ✓ **CRRT (pazienti critici)**
- ✓ **Dialisi intermittente**



**Carmelo
Libetta**

Quale terapia sostitutiva?

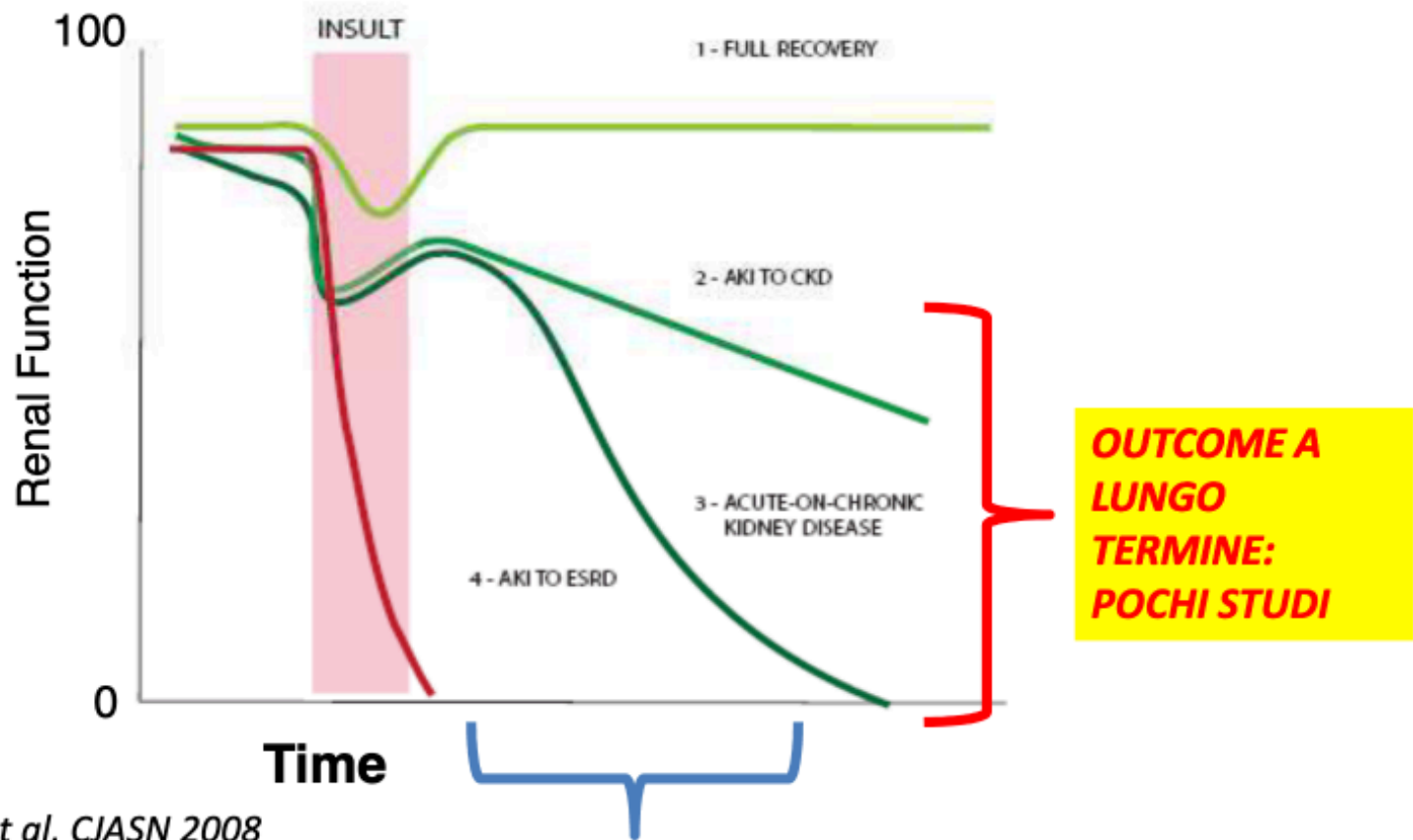
Meccanismo fisico	acronimo	definizione
convezione	SCUF	Slow continuous ultrafiltration
	CAVH	Continuous arterio-venous hemofiltration
	CVVH	Continuous veno-venous hemofiltration
diffusione	CAVHD	Continuous arterio-venous hemodialysis
	CVVHD	Continuous veno-venous hemofiltration
convezione + diffusione	CAVHDF	Continuous arterio-venous hemodiafiltration
	CVVHDF	Continuous veno-venous hemodiafiltration



Carmelo
Libetta

CRRT

STORIA NATURALE DELL'AKI

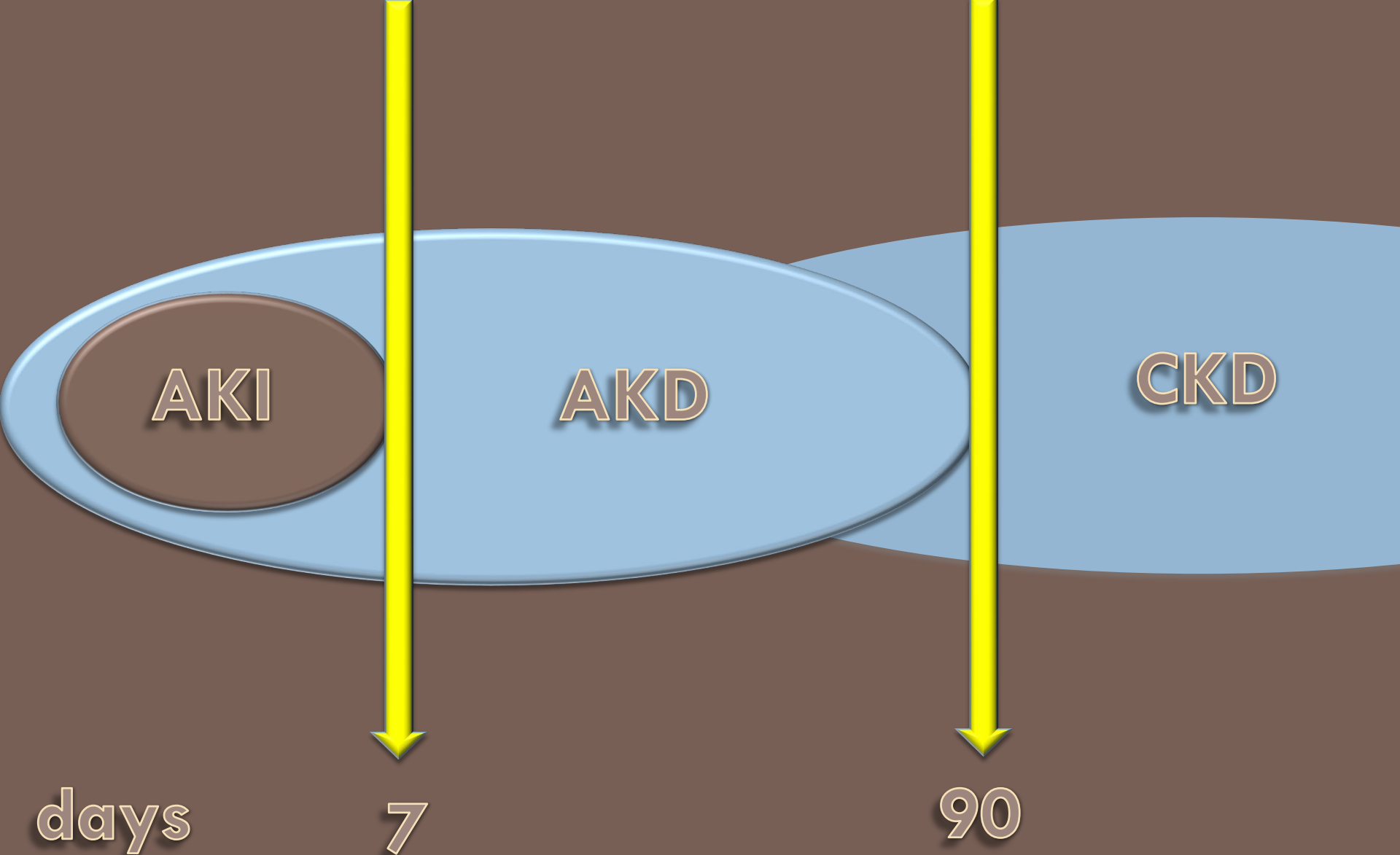


Cerda et al, CJASN 2008

Carmelo
Libetta

AKD





days

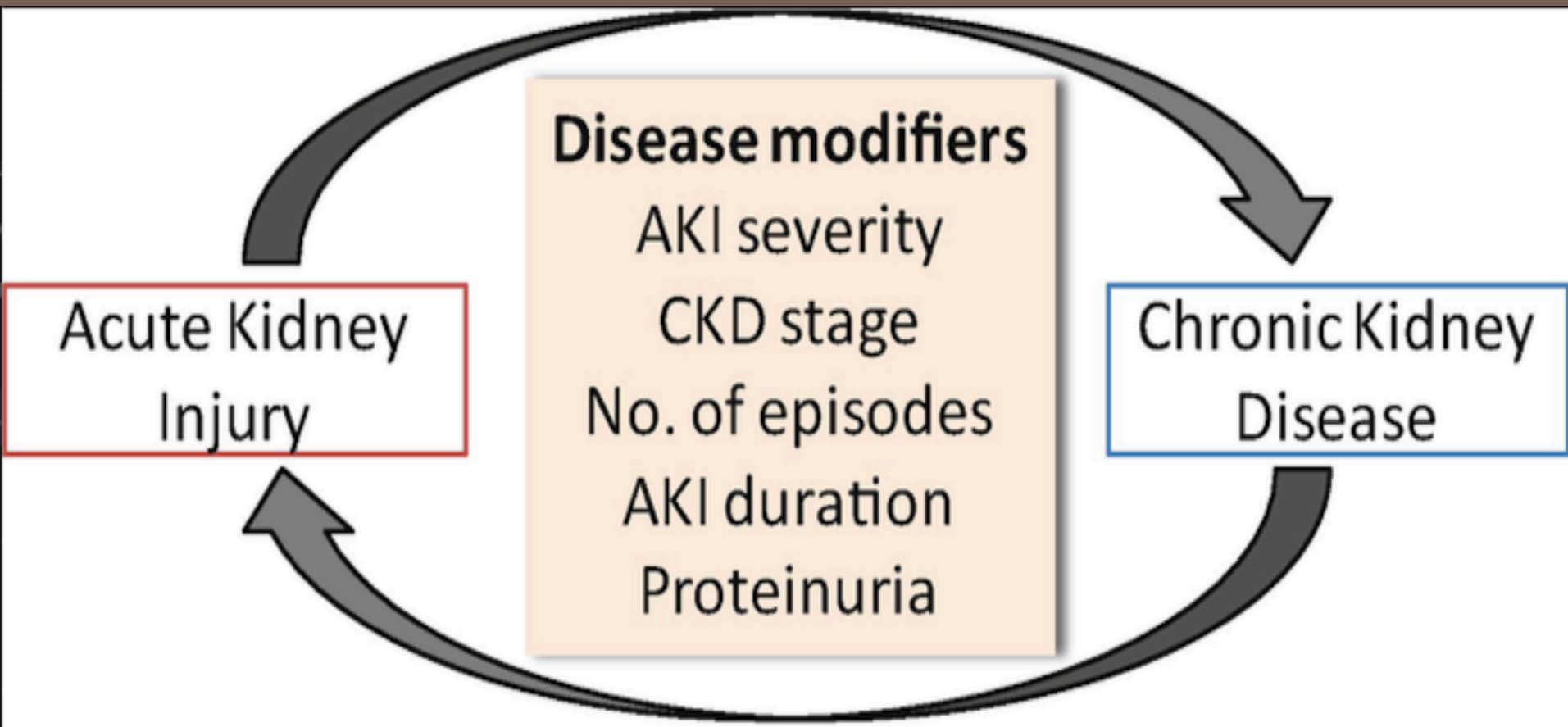
7

90



Carmelo
Libetta

AKD



Acute Kidney
Injury

Disease modifiers

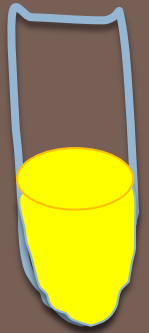
AKI severity
CKD stage
No. of episodes
AKI duration
Proteinuria

Chronic Kidney
Disease

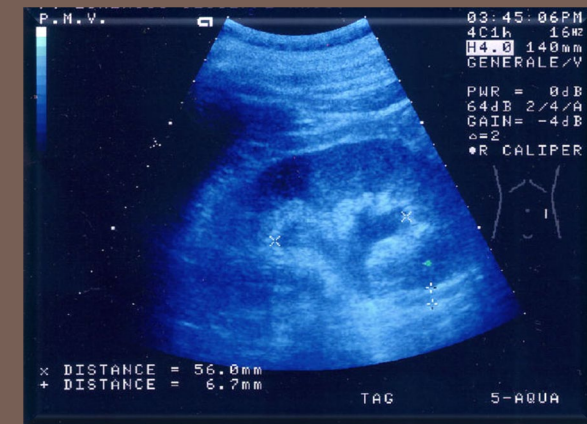
Carmelo
Libetta

AKD: FATTORI PREDISPONENTI

✓ **ELETTROLITI URINARI**



✓ **ECOGRAFIA RENALE**



**Carmelo
Libetta**

AKI

THE END



Chapter 5°