

Leziune acuta de rinichi (LAR)

Acute Kidney Injury (AKI)

Insuficienta renala acuta (IRA)

DEFINITII

Pierdere brutala a functiei renale care duce la:

- **incapacitatea rinichiului de a participa la reglarea echilibrului hidro-electrolitic si acido-bazic**
- **incapacitatea rinichiului de a elimina produse de degradare ai metabolismului protidic**

- **OLIGURIE – diureza < 500 ml/24h**
- **ANURIE – diureza < 50 ml/24h**
- **RETENTIE AZOTATA (cresterea creatininei ser, ureei, ac uric)**

Solomon et al. , Tepel et al. , Schwab et al. , Weisberg et al. , Stevens et al. , and others 0.5 mg/dl increase in (SCr) within 48 h

Hou et al. 0.5 mg/dl increase in SCr if baseline SCr 1.9 mg/dl, or 1.0 mg/dl increase in SCr if baseline SCr 2.0 to 4.9 mg/dl, or 1.5 mg/dl increase in SCr if baseline SCr 5.0 mg/dl

Shusterman et al. 0.9 mg/dl increase in SCr if baseline SCr <2.0 mg/dl, or 1.5 mg/dl increase in SCr if baseline SCr 2.0 mg/dl, and "remained elevated for at least one additional consecutive determination"

Liaño and Pascual "Sudden" rise of >2 mg/dl in subjects with prior "normal" renal function, or "sudden" increase in SCr of 50% with "mild to moderate" basal chronic renal failure with SCr <3.0 mg/dl, or "elevation of SCr at admission with normal or increased renal size (except with myeloma or hydronephrosis with cortical atrophy)"

Bates et al. 50% increase in SCr to at least SCr of 2.0 mg/dl ("ARF") 100% increase in SCr to at least SCr of 3.0 mg/dl ("severe ARF")

Levy et al. 25% increase in SCr to at least SCr of 2.0 mg/dl within two days

Behrend and Miller 0.9 mg/dl increase in SCr if baseline SCr <2.0 mg/dl to at least 2.0 mg/dl, or 1.5 mg/dl increase in SCr if baseline SCr 2.0 mg/dl (baseline defined as lower of most recent SCr in past 3 mo or lowest value during hospitalization)

Obialo et al. 0.5 mg/dl increase in SCr to at least 2.0 mg/dl, or admission SCr 2.0 mg/dl with no history of renal disease

Kurnik et al. 0.5 mg/dl increase in SCr or 25% increase from baseline within 48 h Wang et al. (42)

Hirschberg et al. SCr 3.0 mg/dl with baseline SCr <1.8 mg/dl, or "acute decrease" in creatinine clearance to 25 mL/min after surgery, trauma, hypotension, or sepsis

Allgren et al. 1.0 mg/dl increase in SCr over 2 days

Parfrey et al. >50% increase in SCr to at least 1.4 mg/dl

Cochran et al. >0.3 mg/dl and >20% increase in SCr

Eisenberg et al. 1.0 mg/dl increase in SCr, or 20 mg/dl or 50% increase in BUN

Lautin et al. 6 graded criteria

>0.3 mg/dl and >20% increase in SCr on day 1, 2, or 3, and day 5, 6, or 7, or >0.3 mg/dl increase in SCr on day 1, 2, or 3, or >0.3 mg/dl and >20% increase in SCr on day 1 or 2, or 2.0 mg/dl increase in SCr on day 1 or 2, or 1.0 mg/dl increase in SCr on day 1, or 20 mg/dl or 50% increase in BUN on day 1

Fiaccadori et al. >50% increase in SCr in absence of "volume responsive prerenal status," or >1 mg/dl increase in SCr with known renal insufficiency

Taylor et al. 0.3 mg/dl increase in SCr a ARF, acute renal failure; SCr, serum creatinine.

L. A. R.

Definitie operational

DEFINITION

RIFLE

An increase in serum creatinine of $\geq 50\%$ developing over < 7 days

or

A urine output of < 0.5 mL/kg/hr for > 6 hr

AKIN

An increase in serum creatinine of ≥ 0.3 mg/dL or $\geq 50\%$ developing over < 48 hr

or

A urine output of < 0.5 mL/kg/hr for > 6 hr

70 Kg < 35 ml/h timp de > 6 h sau < 210 ml/6h

STAGING CRITERIA

RIFLE STAGE	INCREASE IN SERUM CREATININE	URINE OUTPUT CRITERIA	INCREASE IN SERUM CREATININE	AKIN STAGE
<i>Risk</i>	≥50%	<0.5 mL/kg/hr for >6 hr	≥0.3 mg/dL; or ≥50%	Stage 1
<i>Injury</i>	≥100%	<0.5 mL/kg/hr for >12 hr	≥100%	Stage 2
<i>Failure</i>	≥200%	<0.5 mL/kg/hr for >24 hr or anuria for >12 hr	≥200%	Stage 3
<i>Loss</i>	Need for renal replacement therapy for >4 wk			
<i>End stage</i>	Need for renal replacement therapy for >3 mo			

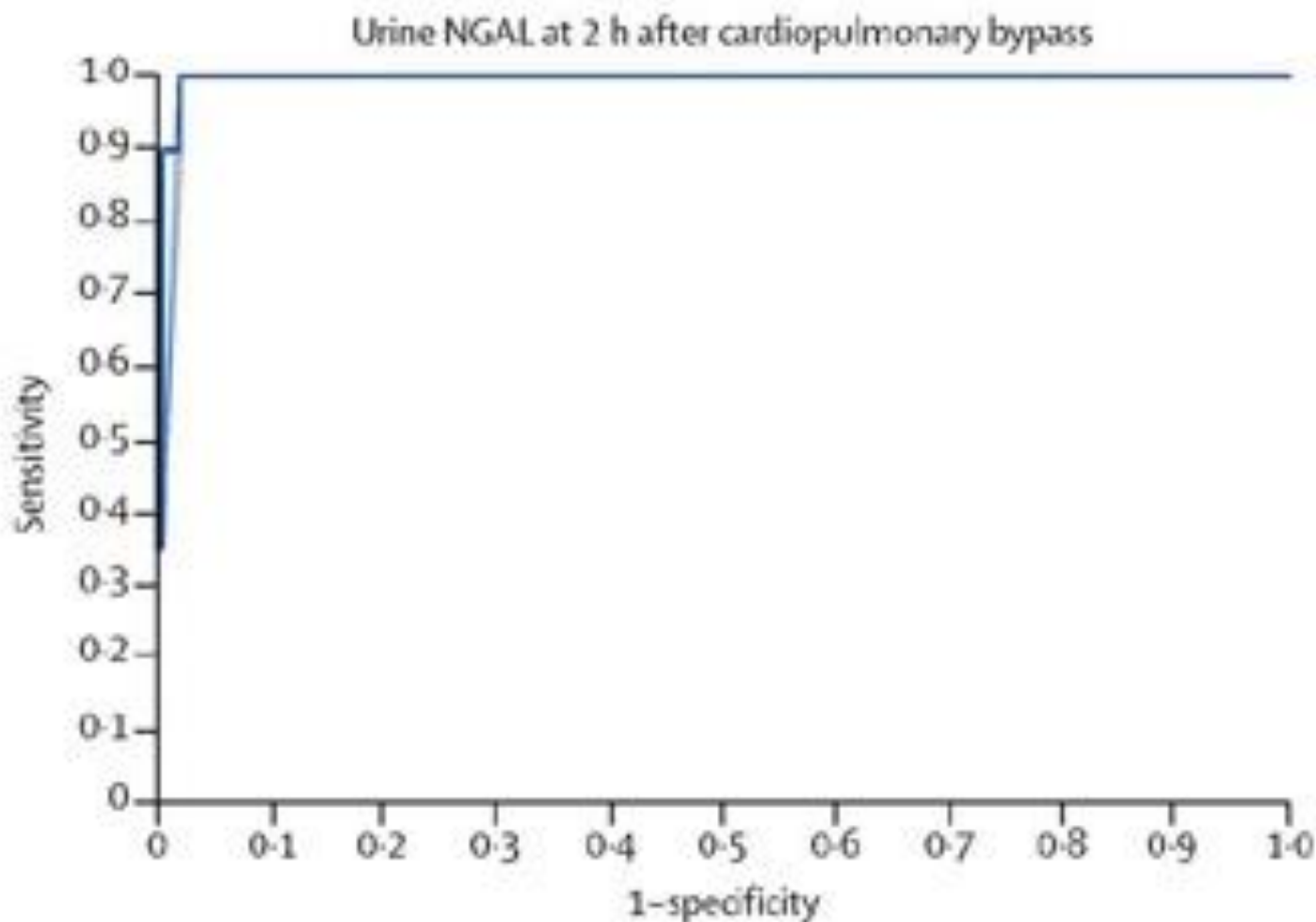
	AMI	ARF
1960	LDH	Serum Cr
1970	CPK, mioglobin	Serum Cr
1980	CK-MB	Serum Cr
1990	Troponin T	Serum Cr
2000	Troponin I	Serum Cr

BIOMARKERS

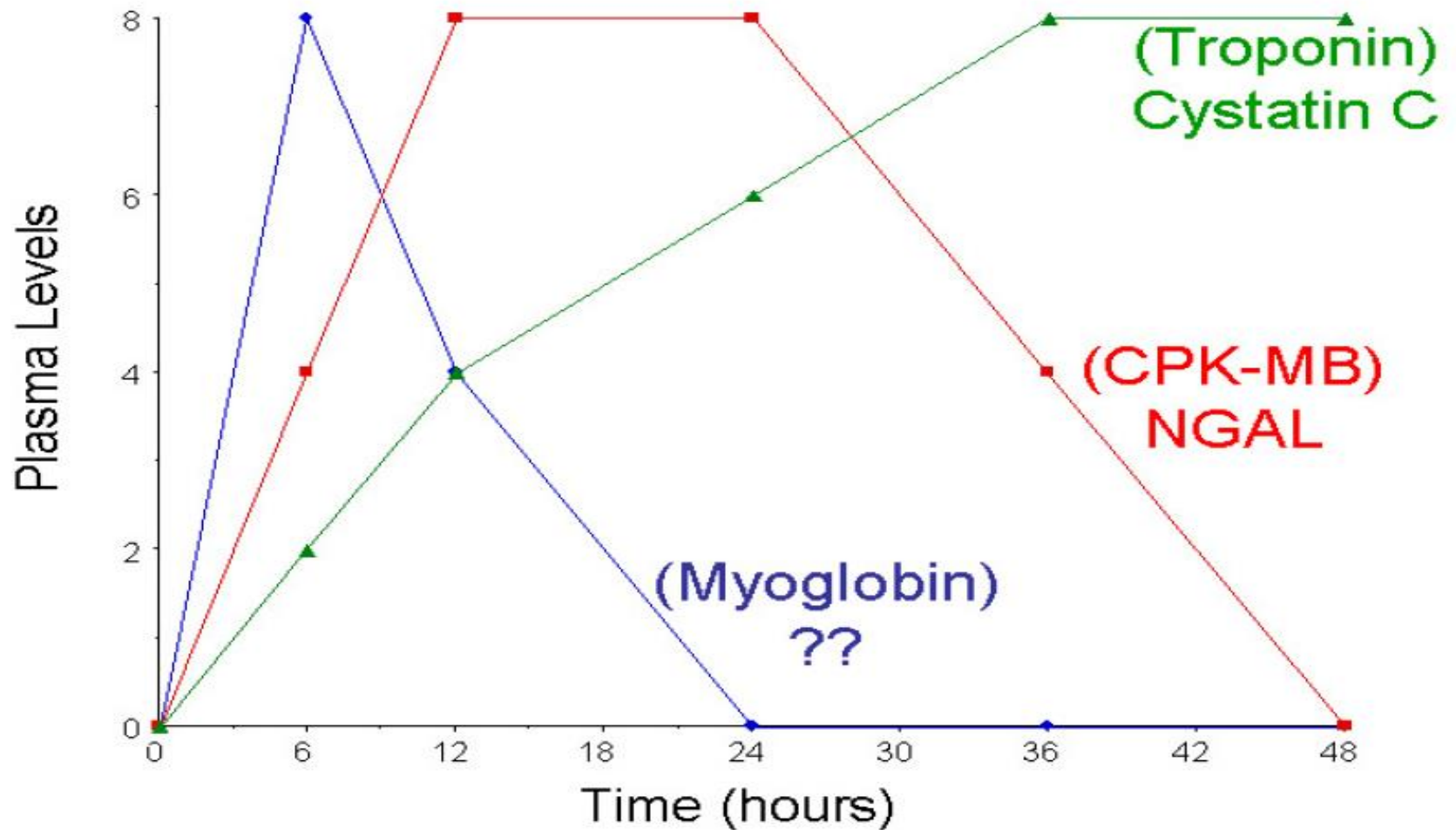
- **PROTEINES** : β 2-microglobulin, α 1-microglobulin, adenosine deaminase-binding protein, retinol-binding protein, cystatin C , renal tubular epithelial antigen-1, **neutrophil-gelatinase associated lipocaline (NGAL)**
- **ENZYMES** (N-acetyl- β -D-glucosaminidase, alanine-aminopeptidase, alkaline phosphatase; lactate dehydrogenase, glutathione-S-transferase , glutamyl transpeptidase)
- **CYTOKINES** (platelet activating factor, **interleukin-18**)
- **kidney injury molecule-1** and **Na/H exchanger isoform- 3 (NHE3)**,

Neutrophil gelatinase-associated lipocalin as a biomarker of acute renal injury after cardiac surgery

Mishra, Lancet, 2005; 365: 1231



BIOMARKERS



CLASIFICARE

- **CLASIC**
 - IRA de origine prerenala
 - IRA de origine renala
 - IRA de origine renala
- **CLASIFICARE ACTUALA**
 - **L.A.R PRERENALA** – AZOTEMIE PRERENALA (IRA functionala)
 - **L.A.R. INTRINSECA** - AZOTEMIE RENALA INTRINSECA (IRA renala)
 - Prin mecanism ischemic si toxic – NECROZA TUBULARA ACUTA
 - Prin afectarea vaselor mari
 - Prin afectarea vaselor mici si glomerulilor
 - prin afectare tubulo-interstitiala
 - **L.A.R. POSTRENALA** - AZOTEMIE POSTRENALA – (IRA obstructiva)

ETIOLOGIE

1. – HIPOPERFUZIE RENALA

- a. – Scaderea volumului circulant datorat pierderilor**
- b. - Scaderea debitului cardiac**
- c. - Prin modificarea raporturilor intre rezistentele vasculare sistemice / periferice**
 - Vasoconstrictie renala**
 - Vasodilatatie sistemica**
 - Cresterea vascozitatii sanguine**
 - Interferarea mecanismelor de autoreglare renala in contextul unei hipoperfuzii preexistente**
- d. – Obstructie reno-vasculara bilaterala**
- e. – Sindromul de compartiment intraabdominal**

2. – NEFROTOXICE

a. Toxine exogene

- **Medicamente – antibiotice, analgetice, antiinflamatorii, etc**
- **Solventi organici**
- **Otravuri sintetice si naturale**
- **Metale grele**
- **Toxine bacteriene**

b. Toxine endogene

- **Pigmenti – Hb, Mgb, MetHb**
- **Cristaloizi – Ac.uric, Ca, Oxalati**
- **Substante ce apar in cursul neoplaziilor**

3. – VASCULITE SI GLOMERULONEFRITE

a. Glomerulonefrite sau vasculite

- **Asociate cu anticorpi anti MBG**
- **Asociate cu ANCA**
- **Asociate maladiilor prin complexe imune**
- **Asociate GNRP pauci imune**

b. Sindroame de hipervascozitate

c. SHU

d. Preeclampsia

e. HTA maligna

4. – NTIA

- **NTIA induse de medicamente**
- **NTIA infectioase**
- **NTIA din cadrul bolilor de sistem**
- **Rejetul acut de grefa**
- **Boala cu anticorpi anti MBT**
- **Idiopatice**

5. UROPATII OBSTRUCTIVE

- **Compresiuni extrinseci bilaterale**
- **Obstructii intrinseci bilaterale**

6. – OBSTRUCTIA VASELOR MARI

- **Obstructie bilaterala a arterelor renale : embolie ateromatoasa, tromboembolism, anevrism disecant al aortei abdominale, boala Takayasu**
- **Obstructia bilaterala a venelor renale : prin tromboza – glomerulonefrita membranoasa**

FIZIOPATOLOGIE

MECANISMUL TOXIC SI ISCHEMIC – 80-90% din LAR

INSUFICIENTA CIRCULATORIE

- Vasoconstrictie simpatica periferica
- Sinteza crescuta de ADH (AVP)

- Activarea RAA

- Reabsorbție renală de Na și H₂O
- **Hipoperfuzie renală**

Gradul hipoperfuziei conditioneaza severitatea sindromului clinic

LAR de origine PRERENALA – hipoperfuzie renala

- Reducerea usoara a FG – scaderea diurezei + retentie azotata
- Excretia fractionata a Na (FE_{Na}) $< 1\%$ (procentul de Na care se excreta din pool-ul de Na filtrat)
- $Cru / Cr. P > 40$
- Indicele IR < 1 $NaU / (CrU / CrP)$
- Nu exista leziuni renale morfologice

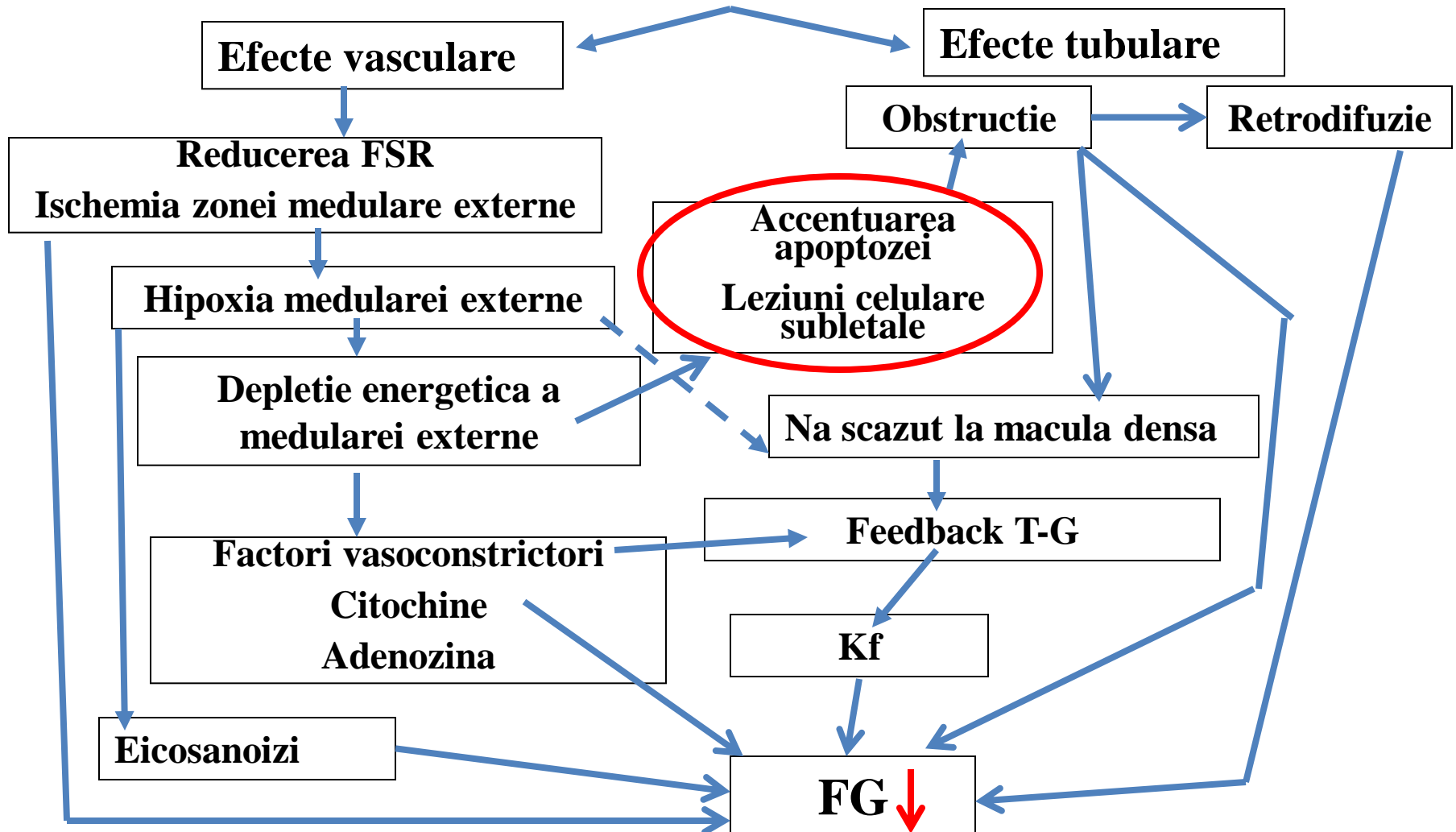
$$FE_{Na} = \frac{NaU \times CrP}{NaP \times CrU}$$

NECROZA TUBULARA ACUTA – ischemie renala

- **Diureza pastrata** – ischemie medulara
 - diureza $> 500\text{ml/zi}$ – retentie azotata
 - reversibilitate posibila in 1 – 2 saptamani
- **Forma oligurica** – ischemie medulara severa
 - Diureza 50 – 500 ml/zi + retentie azotata severa
 - Reversibilitate posibila in 2 – 3 saptamani
- **Forma anurica** – ischemie corticala severa
 - diureza $< 50\text{ ml/zi}$ + reversibilitate improbabila

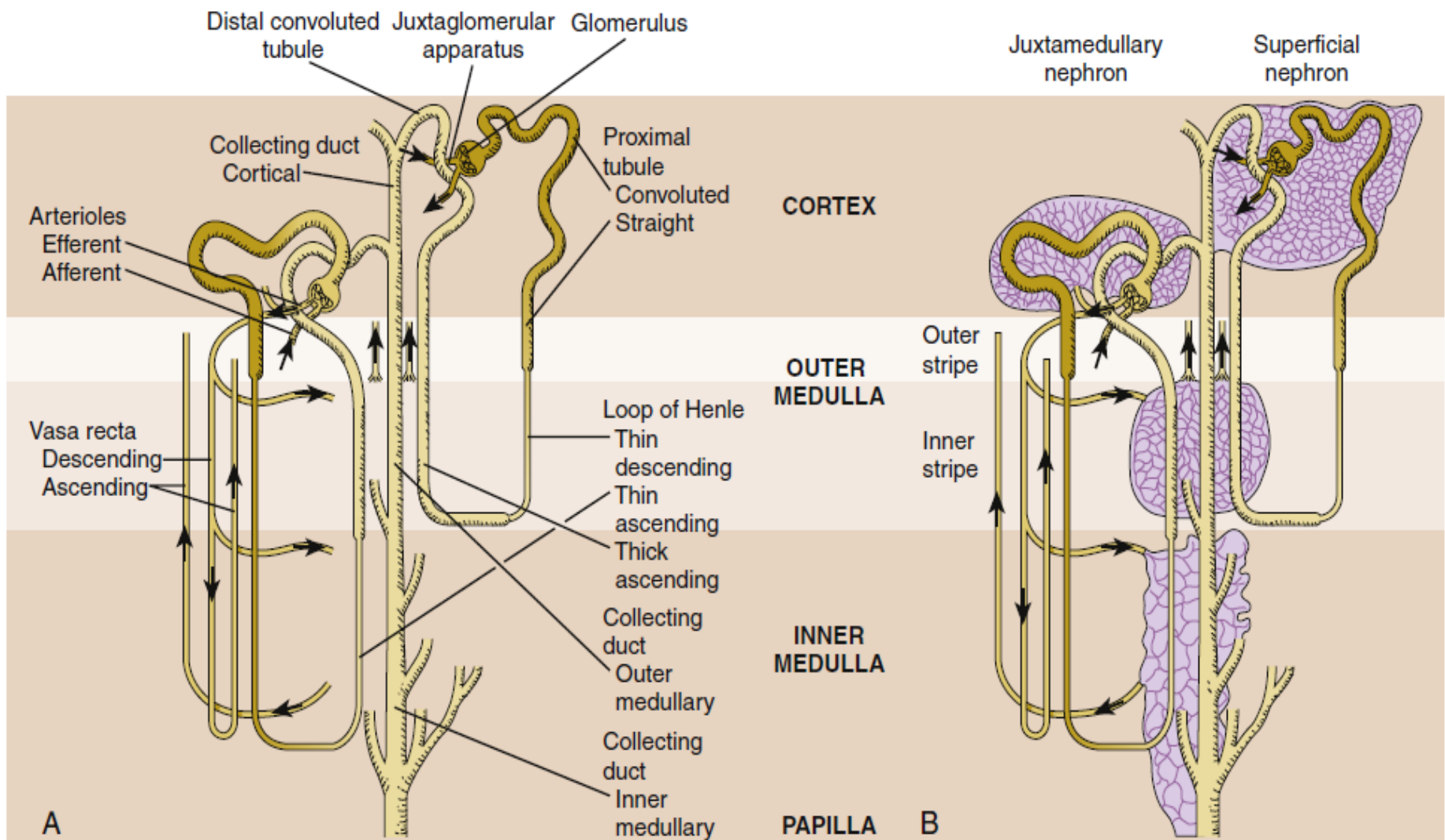
FIZIOPATOLOGIE

AGRESIUNE ISCHEMICA SAU TOXICA

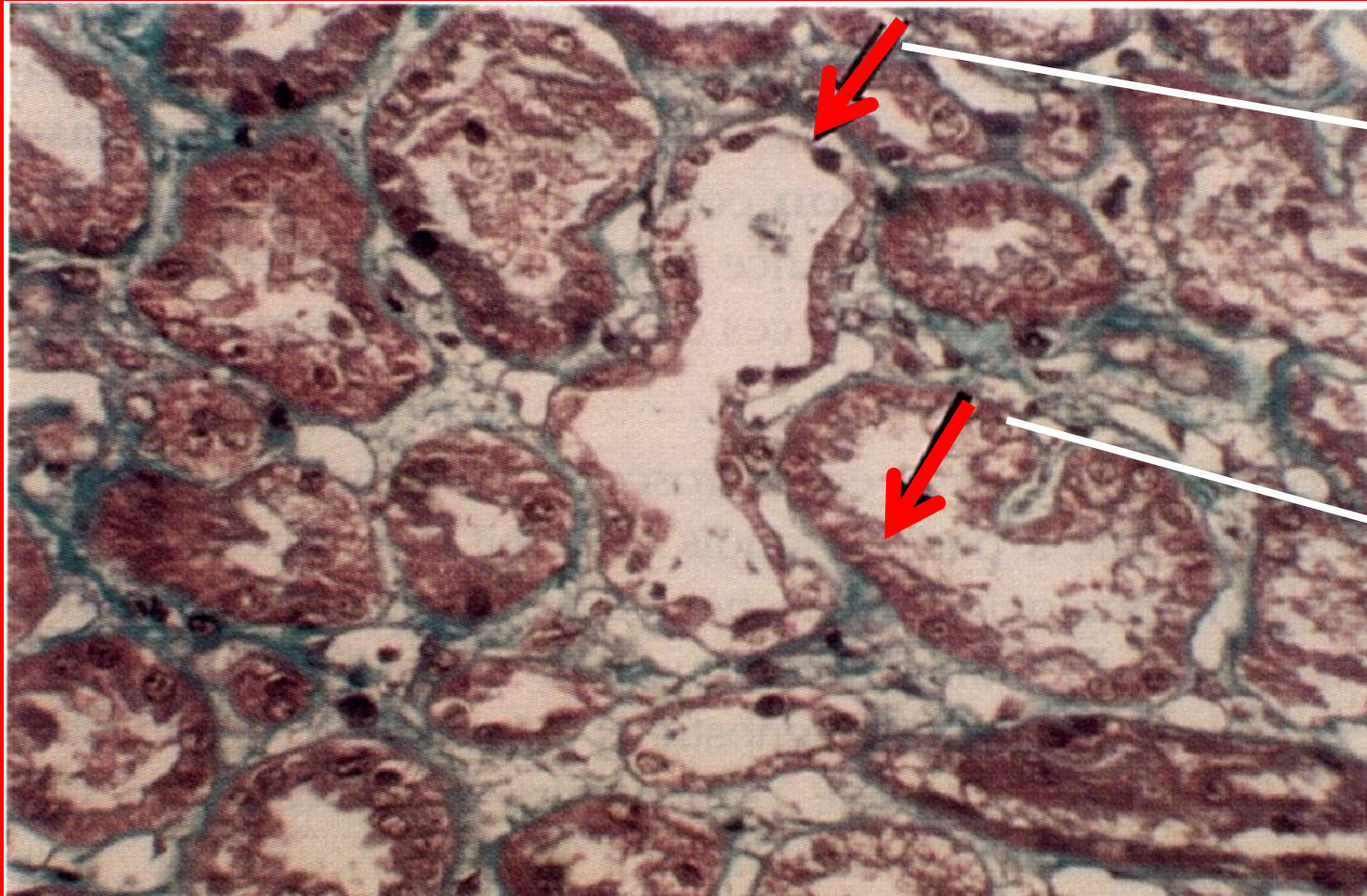


MORFOPATOLOGIE

- **MACROSCOPIC** – Rinichi mari, greutate crescuta
- **MICROSCOPIC**
 - edem si infiltrat interstitial
 - acumulari de leucocite in vasa recta
 - pierderea marginii in perie a epiteliului tubular
 - necroze ale celulelor tubulare in partea dreapta a TCP si partea ascendenta a ansei Henle
 - rupturi ale MBT
 - obstructii tubulare prin cilindri: proteina TH, hematii, detritusuri celulare
 - regenerari celulare



MORFOPATOLOGIE



**Detasarea
epiteliului
de MBT**

**Pierderea
marginii
in perie la
nivelul
TCP**

TABLOU CLINC-BIOLOGIC

LAR PRERENALA

- **ANAMNEZA** – pozitiva pentru elemente ce genereaza hipoperfuzie \pm reducerea diurezei
- **CLINIC**
 - bolnav deshidratat sau sever anemic
 - tahicardic, scaderea valorilor TA +/- hipotensiune ortostatica, tahipnee
- **BIOLOGIC**
 - Retentie azotata
 - Examen de urina – densitate crescuta, sarac in elemente, Osm > 400 mOsm, Na urie < 20mEq/l,
 - FeNa<1%, IIR < 1
- **PARACLINIC**
 - Eco – rinichi mari, fara semne de obstructie

REECHILIBRAREA HE, REFACEREA VOLEMICA DUCE LA AMORSARE IMEDIATA A DIUREZEI SI REDUCERE RAPIDA A RETENTIEI AZOTATE

TABLOU CLINC-BIOLOGIC

LAR intrinseca NECROZA TUBULARA ACUTA

• ANAMNEZA

- (+) pentru pierderi lichidiene
- (+) ingestie , contact cu toxice
- (+) diverse stari de soc
- Debut la 1 – 7 zile de la momentul actiunii factorului agresor (mascot)

• CLINIC SI BIOLOGIC

- Reducerea diurezei (sau oligoanurie) + Retentie azotata
- Tulburari cardio-circulatorii
- Anemie si tulburari de craza sanguina
- HDS

1. Tulburari cardio-vasculare

- **Tulburari de ritm – 18% din cazuri**
- **Insuficienta cardiaca congestiva**
- **HTA prin hiperhidratare**
- **Pericardita – 1-18% din cazuri**

2. Tulburari gastro-intestinale

- **Anorexie, greturi, varsaturi, dureri abdominale**
- **HDS – gastrita acuta eroziva**

3. Tulburari hematologice

- **Anemie**
- **Leucocitoza**
- **Defecte de hemostaza prin alterari trombocitare scaderea sintezei factorilor coagularii**

4. Infectii – 10 – 25% dezvolta septicemii diverse

5. Tulburari neuro-psihice

- **Confuzie, stupor, letargie, coma**
- **Agitatie, hiperreflectivitate, anxietate, paranoia**
- **Sindrom convulsivant (hiperhidratare intracelulara)**

6. Modificari urinare

- **Densitate urinara redusa - <1015 osmolaritate redusa $<300\text{mOsm}$**
- **Na urie $> 30\text{mEq/l}$, IIR > 1 , FeNa $>1\%$**
- **Diureza:**
 - **Forma cu diureza pastrata $> 400\text{ml}/24\text{h}$**
 - **Forma oligo-anurica (oligurie $<400\text{ml}/24\text{h}$, anurie $< 50\text{ml}/24\text{h}$)**

7. Retentia azotata

- **Forme necomplicate –rata zilnica de crestere - uree – 10 – 20 mg%/zi
- Cr. 0,5 – 1mg%/zi**
- **Forme complicate –rata zilnica de crestere – uree 20 – 50mg%/zi
- Cr. 1 – 2mg%/zi**

8. Tulburari hidro-electrolitice

- **Retentie hidro-salina**
- **Hiperpotasemie**
- **Hipocalcemie**

9. Acidoza metabolica

STADIUL DE RELUARE A DIUREZEI

- se instaleaza dupa 7-14 zile de la debutul bolii
- Evolueaza in 2 faze
 - Faza precoce – 4-7 zile
 - Faza tardiva – 10 – 15 zile

STADIUL DE RECUERARE

- Dureaza 3 – 12 luni
- Vindecari sechelare 50% din cazuri

TABLOU CLINC-BIOLOGIC

LAR INTRINSECA PRIN NTIA

• ANAMNEZA

- (+) pentru medicamente potential generatoare de NTIA
- (+) pentru infectii urinare repetate
- (+) pentru alte boli infectioase (stafilococii, pneumonie pneumococica, difterie, etc)

• EXAMEN CLINICO-BIOLOGIC

• NTIA MEDICAMENTOASE

- reducerea marcata a diurezei, oligo-anurie
- febra, dureri articulare
- “rush” cutanat
- eozinofilie si eozinofilurie

- **NTIA INFECTIONASE**

- febra
- semne clinice de infectie (pneumonie, stafilococie, etc)
- Semne clinice de ITU (durere lombara, febra, polachidisurie)
- leucociturie +/- cilindrurie
- +/- uroculturi (+)
- teste inflamatorii (+) – VSH, CRP, fibrinogen

[+]

SEMNE CLINICE DE LAR

TABLOU CLINC-BIOLOGIC

LAR INTRINSECA PRIN VASCULITE SAU GLOMERULONEFRITE

1. GNA

- sindrom nefritic acut (edem, HTA, hematurie, proteinurie)
- retentie azotata

2. GNRP

- sindrom nefritic
- retentie azotata ce creste lent
- +/- hemoptizii, purpura, necroze cutanate, etc

3. SHU

- greturi, varsaturi incoercibile, colici abdominale
- febra
- IRA
- Anemie hemolitica microangiopatica, trombocitopenie, , microhematurie, proteinurie

TABLOU CLINC-BIOLOGIC

LAR INTRINSECA PRIN LEZIUNI ALE VASELOR MARI

TROMBOZA DE VENA RENALA

- sindrom nefrotic, deshidratare, tratament cu steroizi
- durere lombara sau in flancuri
- tensiune lombara decelata palpator
- microhematurie, proteinurie nefrotica
- rinichi mult crescuti in volum (ziua 1-7)
+prezenta trombului ECO si CT

EMBOLIE DE ARTERA RENALA

- traumatisme abdominale, cardiopatii emboligene
- durere lombara sau in flancuri
- greturi varsaturi
- boala cardiaca emboligena
- tensiune lombara decelata palpator
- anurie
- hematurie, leucocitoza, proteinurie, GOT, GPT, LDH crescute
- urografie, nefrograma – absenta opacifierii renale

TABLOU CLINC-BIOLOGIC

LAR POSTRENALA

- Obstructie postrenala bilaterala sau unilaterala pe rinichi unic functional

ANAMNEZA

- Colici renale in antecedente, eventual eliminare de calculi

CLINIC

- glob vezical – obstructie subvezicala
- loje renale dureroase la palpare bilateral +/- febra – obstructie supravezicala

PARACLINIC

- ECO si CT confirma diagnosticul precizand distensia supraiacentă obstructiei

STRATEGII DE DIAGNOSTIC

Diagnosticul diferential se impune cu BCR - ECO

- **Rinichi mari – LAR**
- **Rinichi mici – BCR – exceptie de la regula (rinichi mari si IRC – amiloidoza renala, polichistoza renala, rinichiul tumoral bilateral)**



PLEDEAZA PENTRU LAR

- **functie renala normala antecedent episodului actual**
- **alterarea functiei renale in prezenta unor factori cunoscuti ca declansanti de IRA : insuficienta cardiaca, pierderi de lichide, hemoragii, consum de IEC sau AINS, ciroza hepatica, reactie anafilactica, anestezie sau interventii chirurgicale, traumatisme, arsuri, septicemii, consum de nefrotoxice, sindrom nefrotic instalat recent, colica renala**



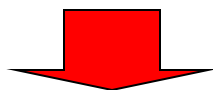
PLEDEAZA PENTRU BCR

- **Retentie azotata decelata anterior episodului actual**
- **antecedente de patologie renala**
- **antecedente de polichistoza renala sau boli ce pot afecta rinichiul**

STRATEGII DE DIAGNOSTIC

STRATEGII DE DIAGNOSTIC POZITIV

IN CONTEXTUL CLINIC DE MAI SUS
RECOLTEAZA - UREE SI CREATININA



UREE SI CREATININA CRESCUTE



ECO RENAL

Rinichi mari bilateral = LAR



Fara distensie pielocaliciala –
evalueaza in continuare



**Rinichi mici bilateral =
BCR**

**Cu distensie pielocaliciala =
LAR POSTRENALA**

URO



DACA E PREZENT

- **Sindrom nefritic sau nefrotic (edem, HTA, proteinurie, hematurie)**
- **SHU (greturi, varsaturi, colici abdominale, febra, anemie hemolitica microangiopatica, trombocitopenie, microhematurie, proteinurie)**



**LAR/GN
SAU V**



DACA NU – CAUTA MAI DEPARTE

- **sindrom nefrotic, durere lombara bilaterala, hematurie microscopica sau oprirea diurezei, eco-rinichi mari, evidentiarea trombului (CT)**
- **traumatism abdominal sau cardiopatii emboligene, anurie, dureri lombare, absenta circulatiei renale (paraclinic)**



**LAR/
TVR
SAU
EAR**





DACA

- consum de medicamente, febra, rush, eozinofilie, eozinofilurie
- dureri lombare, febra, polachidisurie, uroculturi pozitive, microabcese renale



LAR / NTIA

DACA NU



LAR / NTA

- Identifica si corecteaza cauzele de hipoperfuzie renala

COMPLICATII

- **Pericardita**
- **Embolia pulmonara – legat de etiologia IRA**
- **CID**
- **Hemoragia digestiva superioara**
- **Alterarile starii de constienta**
- **Complicatii infectioase**
- **Infarct miocardic acut – uzual emboligen**
- **Creste riscul de mortalitate pe timp scurt mediu si lung**

PROGNOSTIC

MORTALITATE

- **LAR chirurgical 50%**
- **LAR medical 30%**
- **LAR obstetrical 20 – 30%**
- **LAR la arsi – 80%**
- **La asociere de IMOS – 3 sisteme – 95%**
- **La batrani mai mare decat la tineri**

PROFILAXIA IRA

PROFILAXIA SCADERII PERFUZIEI RENALE

- prevenirea instalarii starilor de soc in septicemii, hipovolemii
- combaterea agresiva a starii de soc
- hidratare corespunzatoare inaintea interventiilor mari

PROFILAXIA ACUTIZARII STARILOR DE HIPOPERFUZIE RENALA CRONICA

- Stari de hipoperfuzie renala cronica : sindrom nefrotic, insuficienta cardiaca, stenoza de artera renala, ciroza hepatica
- In aceste situatii se vor evita: abuzul de diuretice, tratamente cu AINS, vasodilatatoare, IEC, pierderile de volum

PROFILAXIA IRA PRIN NEFROTOXICE

- nespecifica : se contraindica utilizarea nefrotoxicelor la bolnavii cu hipoperfuzie; se recomanda o buna hidratare la persoane cu hemoglobinurii, mielom etc
- specifica: metale grele – chelatori; metotrexat – fortarea diurezei; mioglobinurii – alcalinizarea urinii; dializa precoce la cei intoxicati cu substante dializabile

TRATAMENT CURATIV

TRATAMENT CONSERVATOR

- Scop
 - fortarea reversibilitatii formelor incipiente de IRA
 - transformarea formelor oligo-anurice in forme cu diureza pastrata
 - scurtarea duratei anuriei
- Metode
 - REECHILIBRARE HIDRO – ELECTROLITICA
 - Deshidratare hipotona – ser fiziologic (eventual solutii hipertone de NaCl)
$$\text{mEqNa} = (\text{Na ideal} - \text{Na actual}) \times 0,2 \times \text{GC kg}$$
 - Deshidratarea izotona – ser fiziologic
 - forme usoare – 1,5l/m² suprafata corporala
 - forme medii – 2,5 l/m²
 - forme severe – 3l/m²

TRATAMENT CURATIV

- **Deshidratarea hipertona – glucoza + ser fiziologic**

litri solutie = $\left[\frac{\text{Na actual} - \text{Na ideal}}{\text{Na ideal}} \right] \times \text{GC} \times 0,2$

- **Corectarea acidozei**

ml NaHCO₃ 8,4% = BE x 0,3 x GC

- **TRATAMENT MEDICAMENTOS**

- **MANITOL – 12,5 – 25 g (sol 20%) – IV in perfuzie**
- **DIURETICE DE ANSA – Furosemid, Torasemid**
 - **2g/24h in perfuzii - < 4mg/min**
- **DOPAMINA – 1 – 5 µg/kg/min in asociere cu furosemid**
- **BCCa – Diltiazem**
- **AA ESENTIALI**
- **ALTE : FNA, analogi de prostaglandine (misoprostol)**

TRATAMENT CURATIV

• REGM IGIENO DIETETIC

- Aport energetic – 30 – 40kcal/kg/zi
- Repartitia principiilor (glucide 55,5%; lipide 40,2%; proteine 4,3%)
 - GLUCIDE - 8 – 14g/kg/zi; parenteral glucoza hipertona 20-70%
 - LIPIDE – 0,5 – 1g/kg/zi; 2 x /sapt 2g/kg (0,15g/kg/h)
 - PROTEINE
 - tratament conservator – 0,35 – 0,40 g/kg/zi
 - bolnav dializat - 0,5 – 0,6 g/kg/zi
 - faza poliurica - 0,5 – 0,6 g/kg/zi
- Aportul de lichide

aport hidric = diureza + perspiratie insensibila + alte pierderi

TRATAMENT CURATIV

• TRATAMENT ETIOLOGIC

- **epurarea toxicului**
- **tratament antiinfectios**
- **combaterea starii de soc**
- **tratament imunomodulant**

• TRATAMENTUL COMPLICATIILOR

- **profilaxia septicemiilor**
- **profilaxia hiperpotasemiilor**
- **profilaxia HDS**
- **combaterea energica a acidozei metabolice**

TRATAMENTUL PRIN EPURARE EXTRARENALA

- 1. Oligurie – diureza < 500 ml/24h**
- 2. Anurie – absenta diurezei > 12h (<50 ml /24h)**
- 3. Uree > 200 mg%**
- 4. Creatinina >10 mg/%**
- 5. Hiperpotasemie (K^+ >6.5 mEq/l)**
- 6. Edem pulmonar acut care nu raspunde la tratament**
- 7. Acidoza metabolica - pH <7.2**
- 8. Encefalopatie uremica**
- 9. Pericardita uremica**

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TRATAMENTUL PRIN EPURARE EXTRARENALA

- **DIALIZA PERITONEALA**
- **HEMODIALIZA INTERMITENTA**
- **DIALIZELE CONTINUI : A – V , V - V**