Case-Based Approach in Emergency Medicine

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Disclosure information

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- **Prof. Bancha Satirapoj, M.D.**
- **Scientific Advisor/Honoraria:**
  - **Astra Zeneca, Abbott Laboratories, Boehringer Ingelheim, Celltrion Healthcare, Fresenius Kabi, LG Life Sciences, Janssen-Cilag, Menarini, MSD, Novo Nordisk, Osotspa Taisho, Sanofi Aventis, Servier, Viatris and Zuellig Pharma**
- **DISCLAIMER**
  - **This presentation is intended for educational purpose for HCPs only. It may contain new science data which is currently not in approved package insert information and is not intended for off-label promotion.**

Hypertension emergencies

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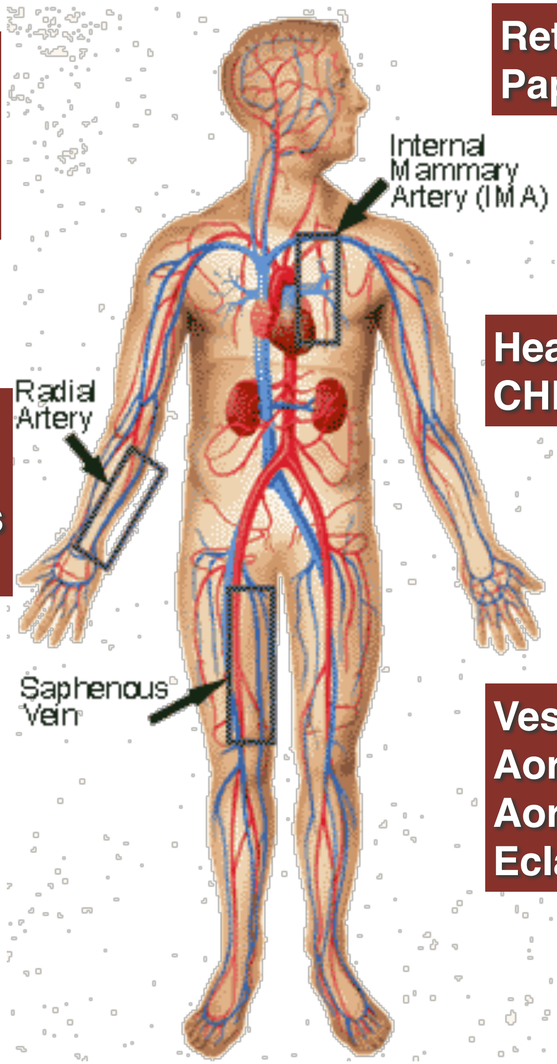
- Severe hypertension (grade 3: 180/110 mmHg) is associated with acute symptomatic HMOD

- Hypertension emergencies, can be life-threatening and require immediate intervention to lower BP, usually with IV therapy

CNS
HT encephalopathy
ICH
Cerebral infarction

Retina hemorrhage
Papilledema



Heart
CHF, MI, angina

Kidney
Acute kidney injury
Acute glomerulonephritis
RAS

Vessels
Aortic dissection
Aortic aneurysm
Eclampsia

Mancia G, et al. J Hypertens. 2023; 41(12):1874-2071.

Malignant hypertension with thrombotic microangiopathy and persistent acute kidney injury

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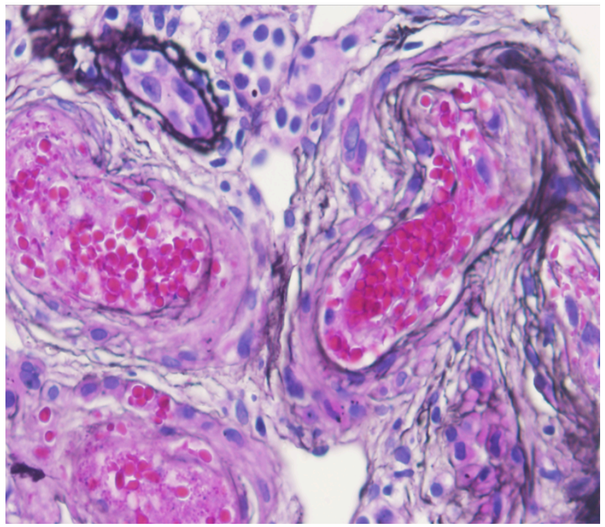
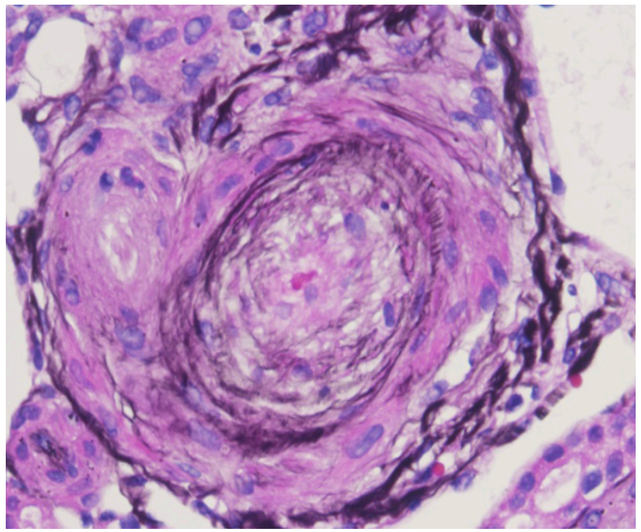
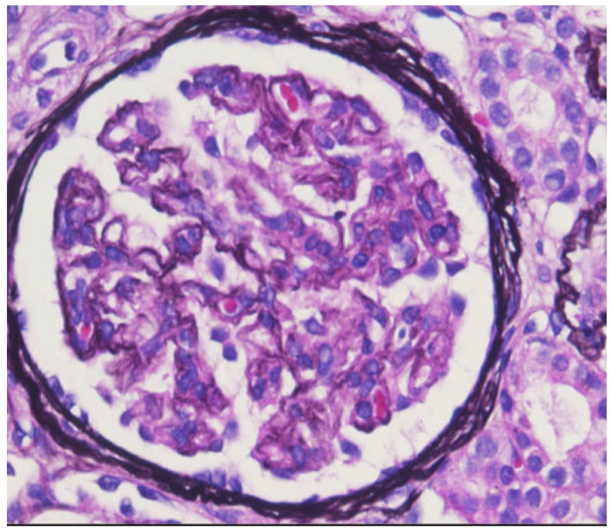
- Severe BP elevation with hemolysis and thrombocytopenia in the absence of other causes and improvement with BP-lowering therapy



RBC and fragments within lumen



Mucoid change with intimal proliferation



Extensive corrugation of GBM

Nzerue C, et al. Clin Kidney J. 2014 Dec;7(6):586-9.

Keith-Wagener-Barker Classification

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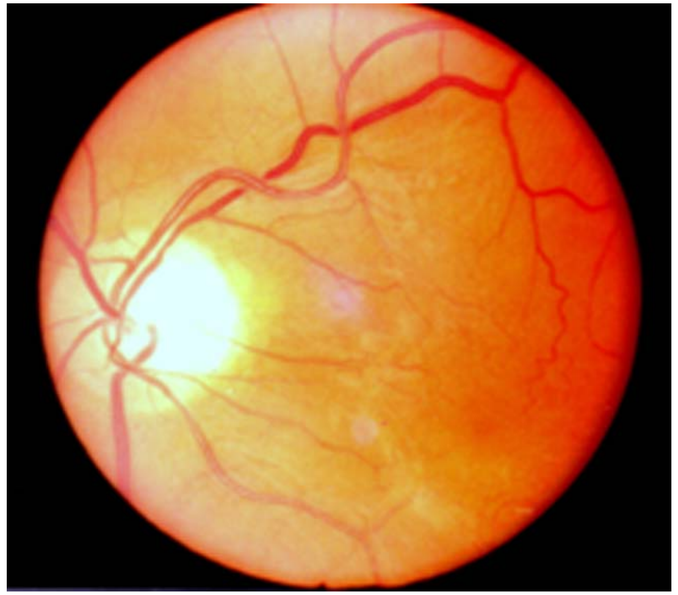

- Grade 1
  - Mild narrowing of the arterioles
  - “Copper Wire”
- Grade 2
  - Moderate narrowing -Copper wire and AV nicking
- Associated with long standing essential hypertension

Keith-Wagener-Barker Classification

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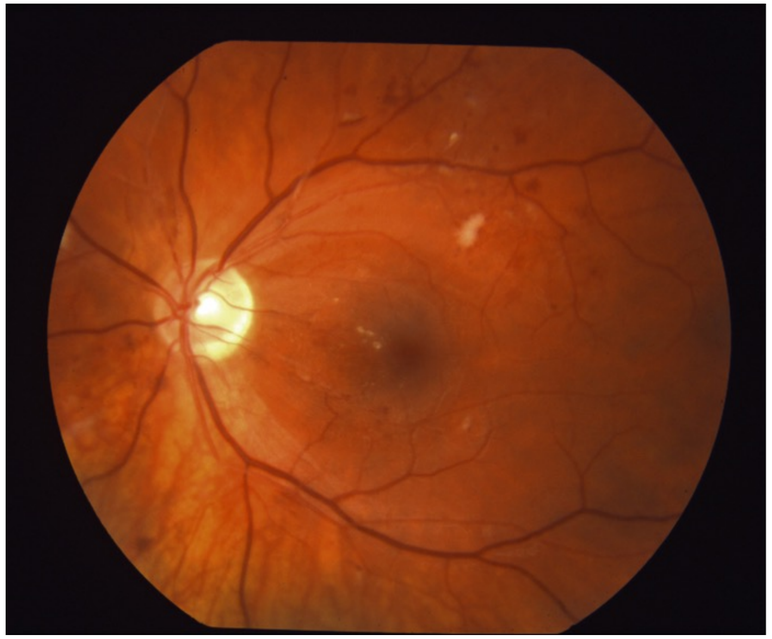
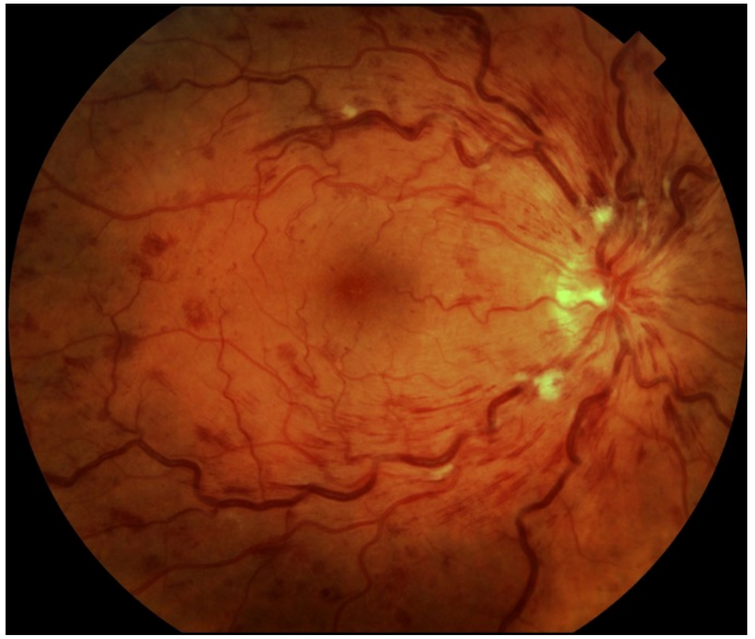
- Grade 3
- Severe hemorrhage, cotton wool spots, hard exudates





Accelerated hypertension

Keith-Wagener-Barker Classification

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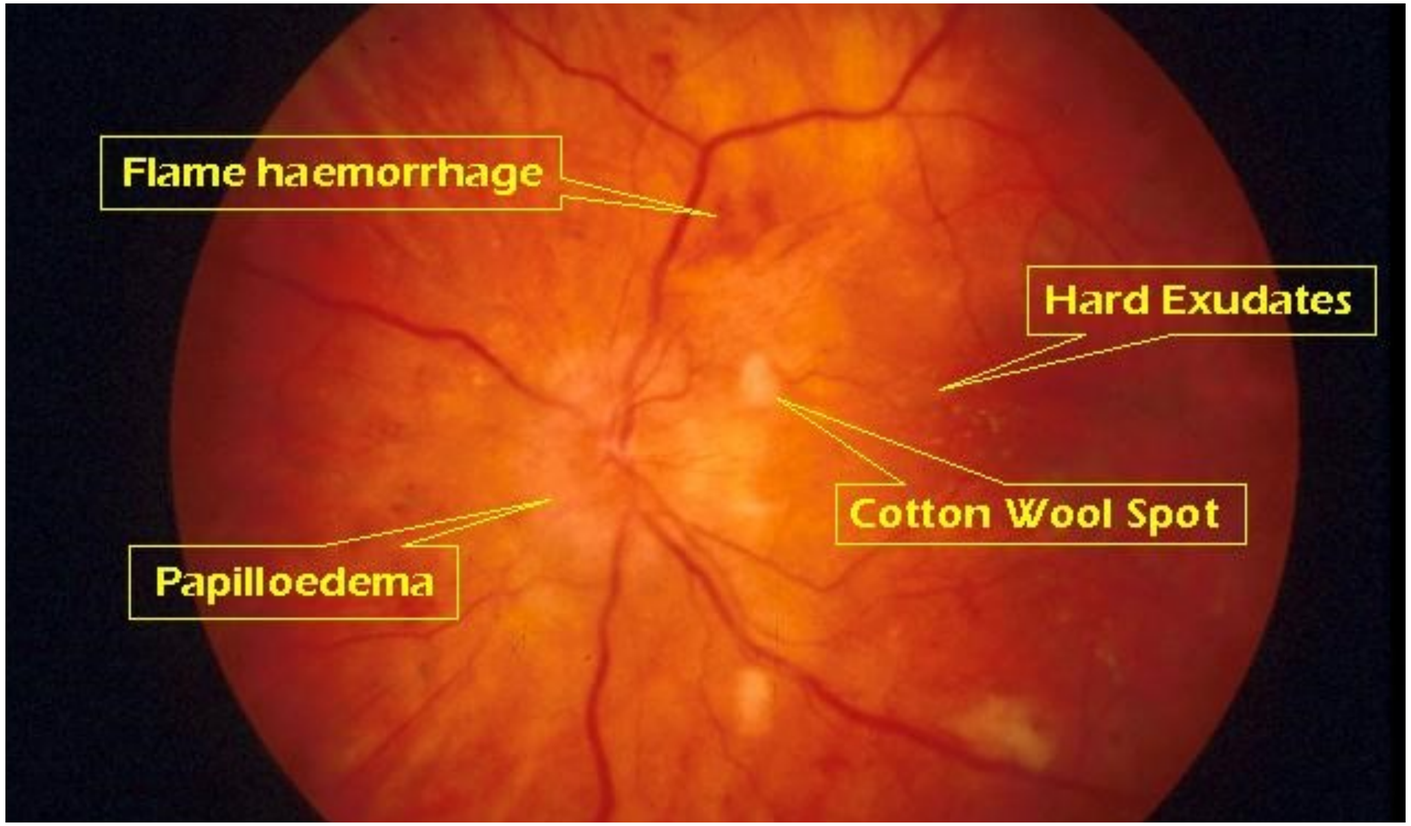
- **Grade 4: Grade 3 + Papilledema**



Malignant hypertension

Grade 3 and 4 highly correlated with progression to end organ damage and decreased survival

Renal Autoregulation

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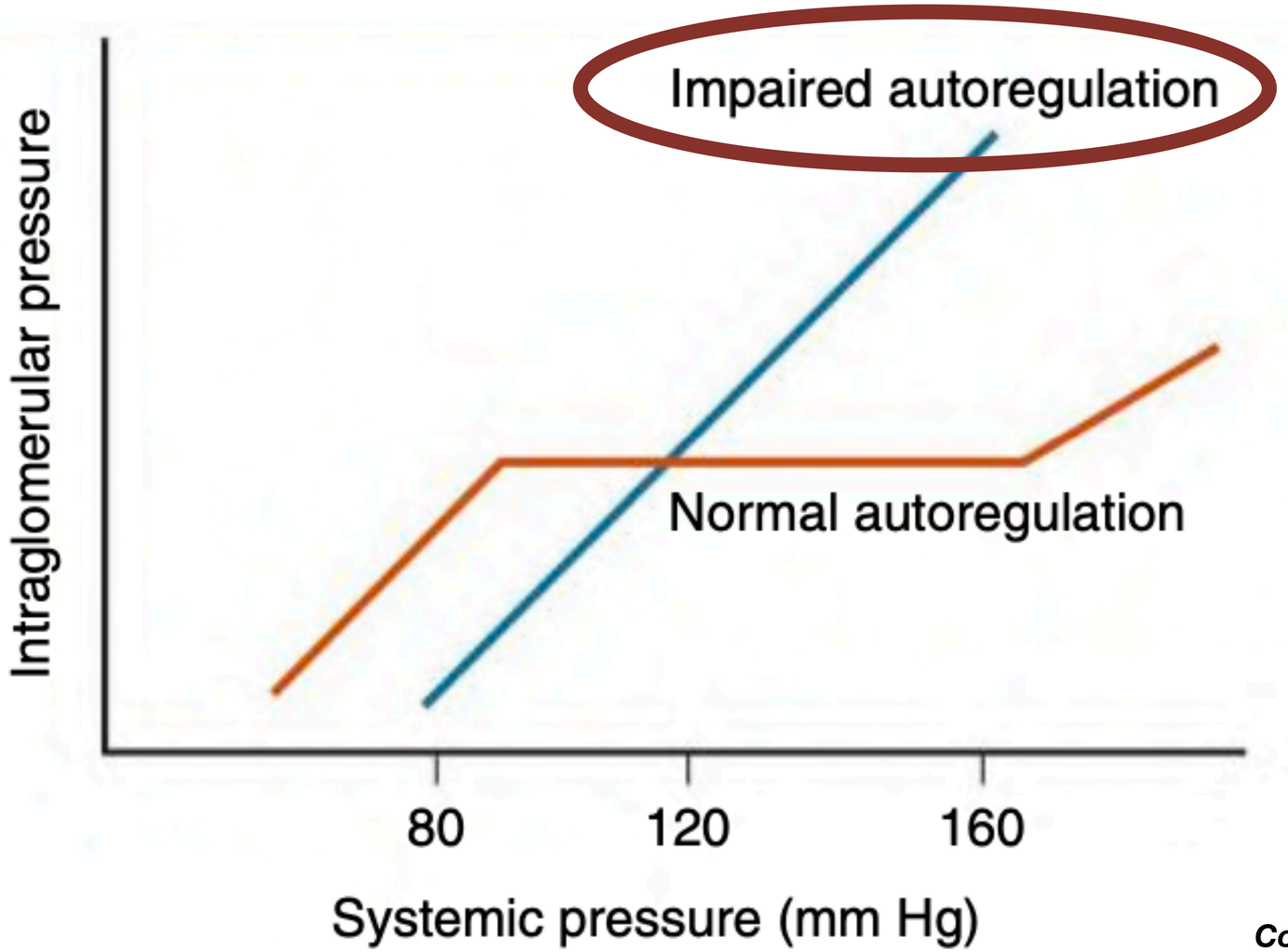

- **Renal Autoregulation.**
- **Relationship of systemic to glomerular pressure in the setting of normal or abnormal renal autoregulation.**

Comprehensive Clinical Nephrology (Johnson) 7 ed (2023)

Hypertensive emergencies requiring immediate BP lowering with i.v. drug therapy

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Clinical presentation	Timing and BP target	First-line treatment
Malignant hypertension with or without acute renal failure	Several hours Reduce MAP by 20–25%	Labetalol[a] Nicardipine
Hypertensive encephalopathy	Immediately reduce MAP by 20–25%	Labetalol[a] Nicardipine
Acute coronary event	Immediately reduce SBP to <140 mmHg	Nitroglycerine Labetalol[a]
Acute cardiogenic pulmonary edema	Immediately reduce SBP to <140 mmHg	Nitroprusside or nitroglycerine (with loop diuretic)
Acute aortic dissection	Immediately reduce SBP to <120 mmHg and heart rate to <60 bpm	Esmolol AND nitroprusside or nitroglycerine or nicardipine
Eclampsia and severe preeclampsia/HELLP	Immediately reduce SBP to <160 mmHg and DBP to <105 mmHg	Labetalol[a] or nicardipine and magnesium sulphate

Mancia G, et al. J Hypertens. 2023; 41(12):1874-2071.

Thrombotic Microangiopathy (TMA)

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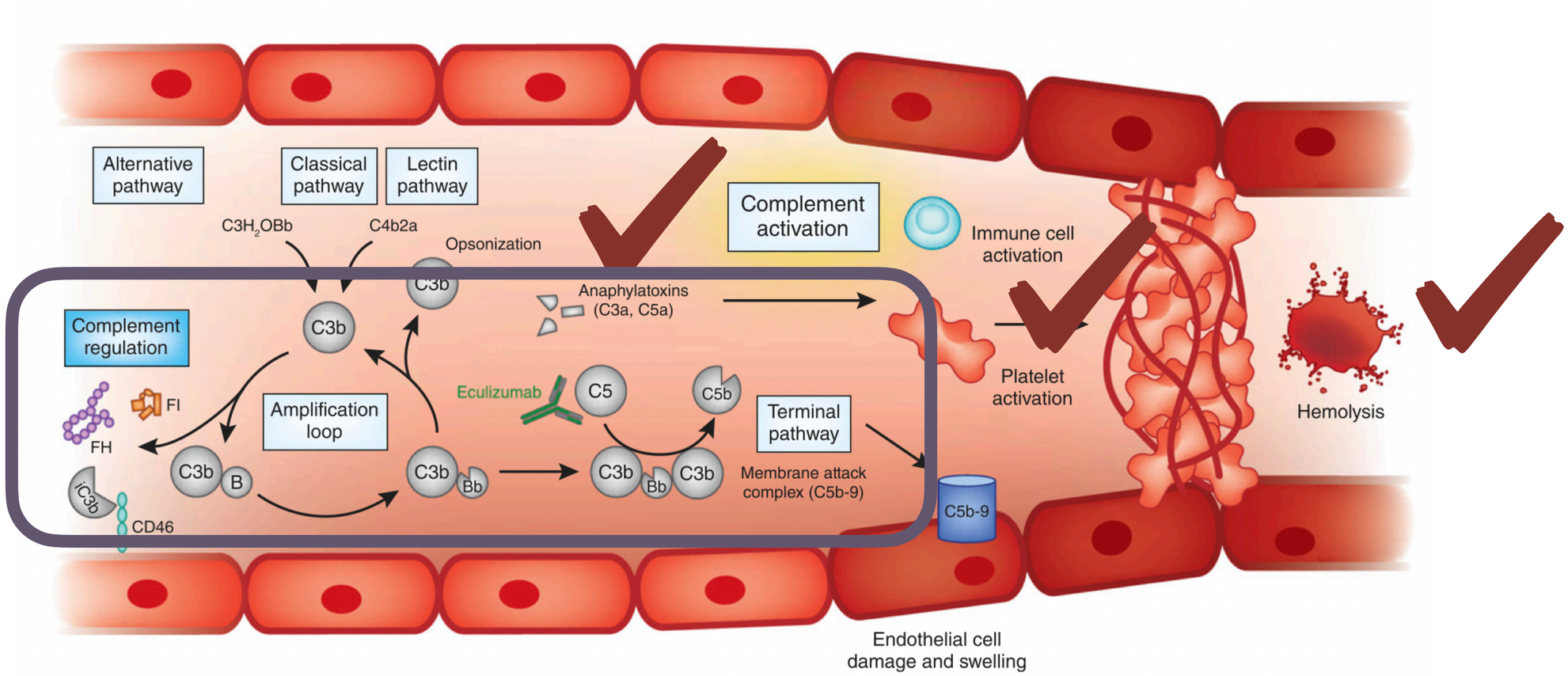
Complement activation ultimately results in thrombus formation, platelet consumption, vascular occlusion and mechanical hemolysis



Brocklebank V, et al. Clin J Am Soc Nephrol. 2018; 13(2):300-317.

What is Thrombotic Microangiopathy (TMA) ?

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T hrombotic

Clot comprising various blood cells and proteins within the vasculature

M icro

Clots form in the small blood vessels, such as the capillaries and arterioles

A ngiopathy

Angiopathy is a disease of the blood vessels, evident if vessel lesions are in histologic sections

The diffuse and systemic nature of TMA has the potential to affect the microvasculature of many organ systems, causing ischemia and eventual organ failure

1. Pendleton RC, Rodgers GM. Thrombosis and antithrombotic therapy. In: Greer JP, et al, eds. Wintrobe's Clinical Hematology. 13th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2014:1218-1257. 2. Campistol JM, et al. Nefrologia. 2015;35(5):421-447.

Cardiac

Acute myocardial infarction, cardiomyopathy (heart failure)

Neurological

Headache, visual disturbance, hyperreflexia, encephalopathy, syncope, focal deficit, seizure, coma

Gastrointestinal

(Bloody) diarrhoea, vomiting, abdominal pain, melaena, pancreatitis, cholecystitis

Renal

Oliguria, acute kidney injury, hypertension, haematuria, proteinuria

Skin

Purpura, petechiae, digital ischaemia

Lungs

Dyspnoea, pulmonary embolism

Adrenal

Hypotension

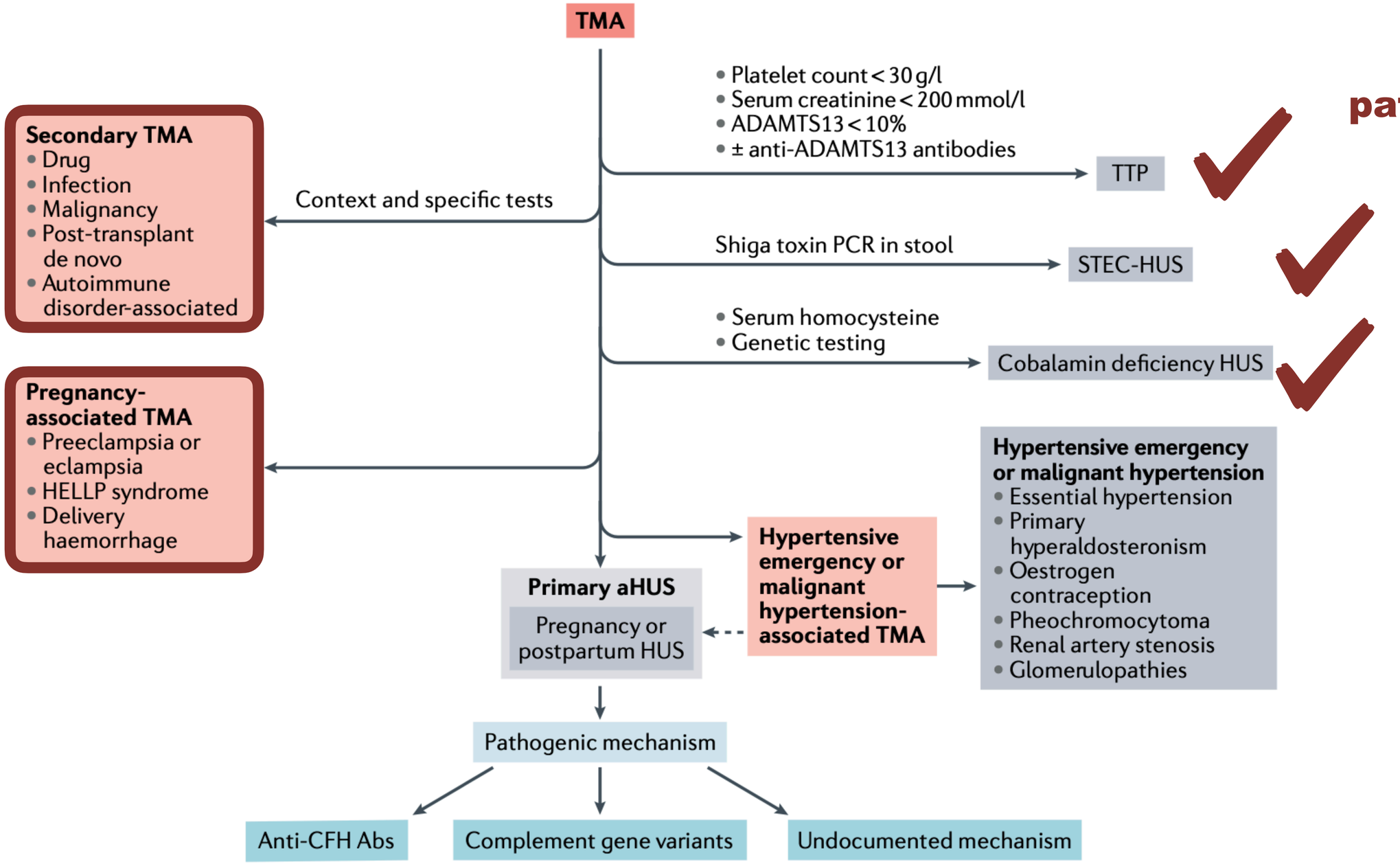
Pregnancy complications

Placental insufficiency, fetal distress

TMA induced organ dysfunction

Fox LC, et al. Nephrology (Carlton). 2018; 23(6):507-517.

A diagnostic algorithm for patient presenting with TMA



Fakhouri F, et al. Nat Rev Nephrol. 2021; 17(8):543-553.

Thrombotic thrombocytopenic purpura

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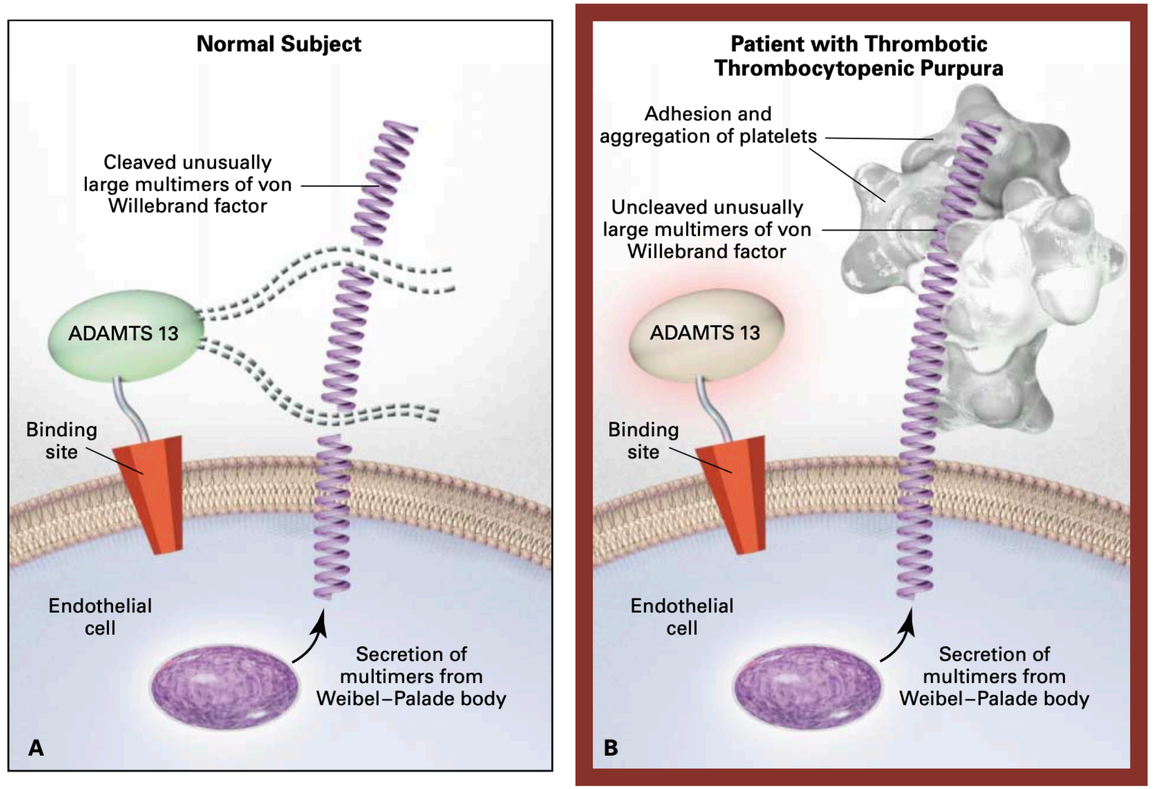
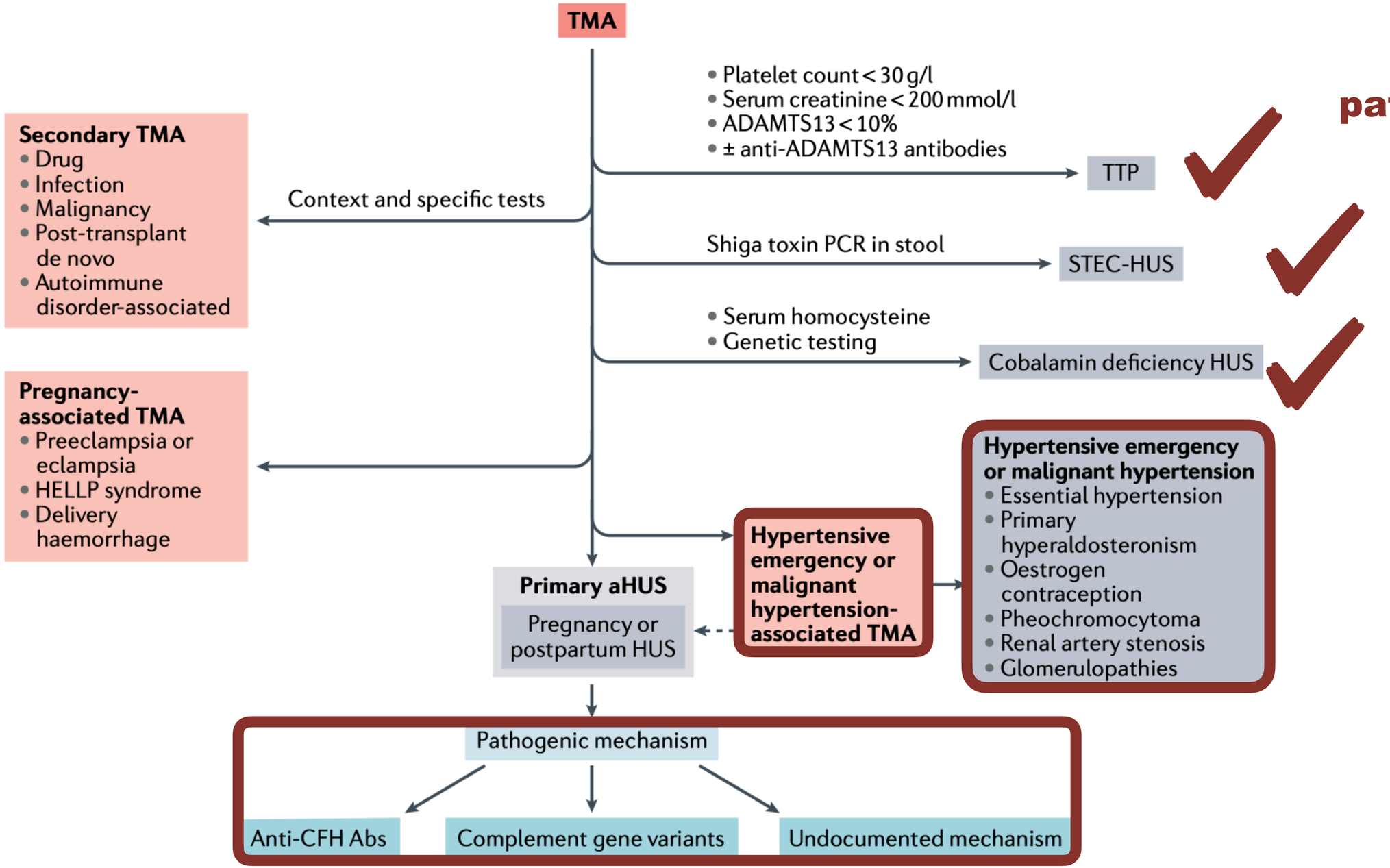
- Microvascular thrombosis
  - MAHA
  - Thrombocytopenia
  - Purpura and no severe bleeding
  - Fever
  - Acute kidney injury
  - Neurologic abnormalities
  - Low ADAMTS-13 activity <10%



Idiopathic TTP

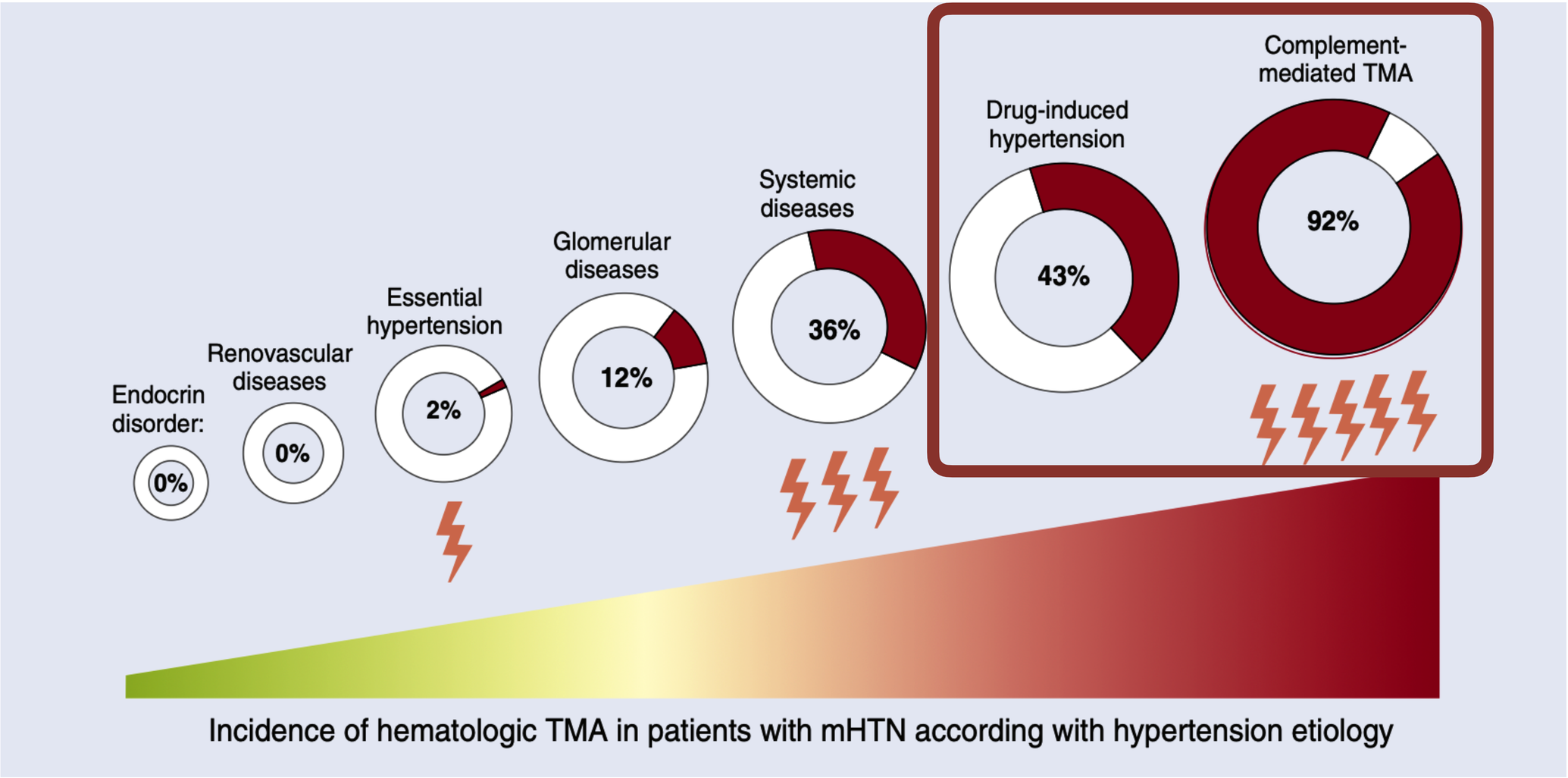
Moake JL. Thrombotic microangiopathies. N Engl J Med 2002; 347:589.

A diagnostic algorithm for patient presenting with TMA



Fakhouri F, et al. Nat Rev Nephrol. 2021; 17(8):543-553.

Incidence of TMA in patients with malignant hypertension according to hypertension etiology



Juárez G, et al. Kidney Int. 2025: S0085-2538(25)00879-8.

TMA in patients with malignant hypertension

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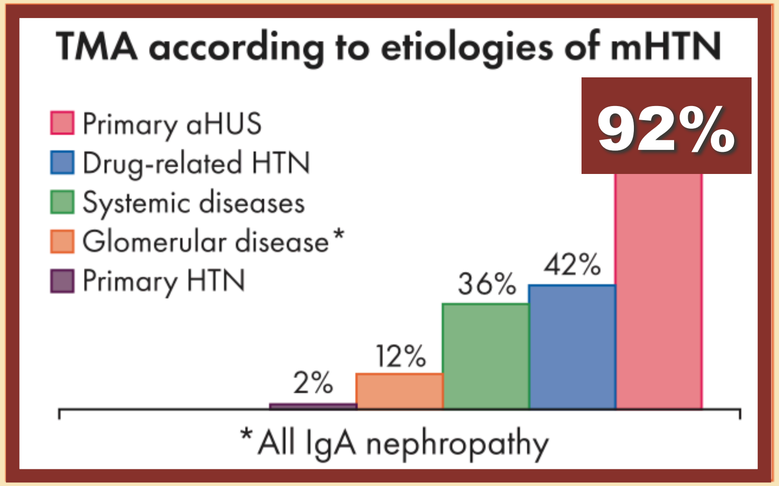
Background

No study has investigated whether the presence of thrombotic microangiopathy (TMA) is associated with specific causes of malignant hypertension (mHTN) or if TMA can be the consequence of extremely high blood pressure regardless of its cause.

Methods

- 8 Spanish hospitals
- Patients with mHTN
  n =199
- 2000–2020
- Outcome:
  Kidney recovery and/or failure

Results



Kidney survival

TMA vs. without TMA

1 year	59% vs. 79%
2 year	51% vs. 73%
5 year	43% vs. 67%

Characteristics of patients with TMA, n = 40 (20%)

Younger · Female · Lower BP · Worse kidney function

Cavero T, et al. Nephrol Dial Transplant. 2023 May 4;38(5):1217-1226.

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Acute phase of atypical HUS

TMA + Hypertensive emergencies (HE) (malignant hypertension)

Main issue

Overlap between malignant hypertension and atypical HUS:

a) HE complicates more than half of atypical HUS cases
b) HE related to essential and secondary (non atypical HUS) hypertension may, albeit rarely (~15%), lead to TMA.
c) Underlying glomerulonephitis, mainly IgA nephropathy, may be complicated by HE leading to TMA

Features that help distinguish atypical HUS from other causes of HE	Weight
At diagnosis	
Female	+ 1
Age < 45 years	+ 1
Familial history of atypical HUS	+4
Absence of history of hypertension or of antihypertensive treatment (cessation)	+ 1
Absence of left ventricular hypertrophy	+ 1
Requirement for dialysis	+ 1
Rapid (< 48 h) resolution of hematological TMA after strict blood pressure control	-2
Low C3 plasma level	+2
During follow-up	
Absence of thrombi in glomeruli (kidney biopsy)	-2
Persistent low C3, Factor H and/or factor I plasma levels.	+3
The higher the score, the higher the probability of atypical HUS complicated by HE.	

Fakhouri F, et al. Blood. 2023 Mar 2;141(9):984-995.

Initiating eculizumab within 7 days of a TMA significantly improved eGFR vs delayed initiation

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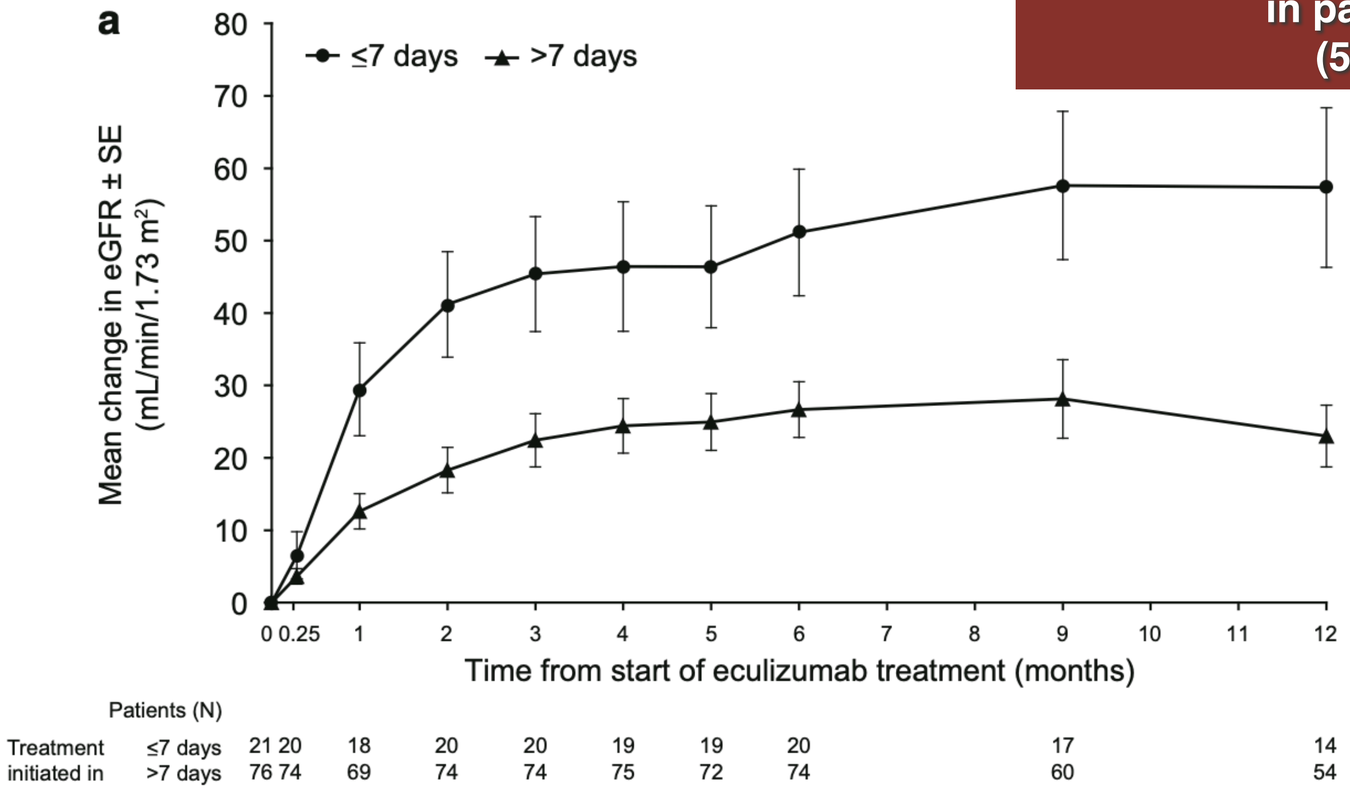
- **In a pooled prospective analysis of four Phase 2 studies (N=97)**

Mean eGFR change from baseline at 1 year was significantly higher in patients treated in <7 days than >7 days (57 vs. 23 ml/min/1.73 m2, p= 0.0098).



Proportion of patients with increased eGFR ≥15 mL/min/1.73 m² at 12 months

81% Earlier treatment ≤7 days*

VS

47% Later treatment >7 days*

Vande Walle J, et al. J Nephrol. 2017;30:127-134.

Atypical HUS

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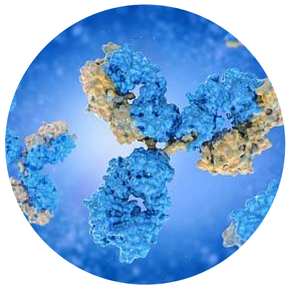
Incidence 0.23-1.9:1,000,000[1]
Thrombocytopenia + MAHA + End Organ Damage
ADAMTS-13 ≥ 10%



Treatment options

Plasma Therapy: PE/PI

- Traditional supportive treatment
- Risk of fluid and electrolyte imbalance

Specific Treatment: Eculizumab[4-7]

- ✓ Inhibits C5 specifically
- ✓ Rapid hematological response followed by normalization
- ✓ Improvement or stabilization of renal function
- ✓ Prevention of TMA recurrence and improved renal outcomes

Legendre CM, et al. N Engl J Med. 2013;368:2169-2181. Licht C, et al. Kidney Int. 2015;87(5):1061-1073. Fakhouri F, et al. Am J Kidney Dis. 2016;68(1):84-93. Greenbaum LA, et al. Kidney Int. 2016;89(3):701-711.

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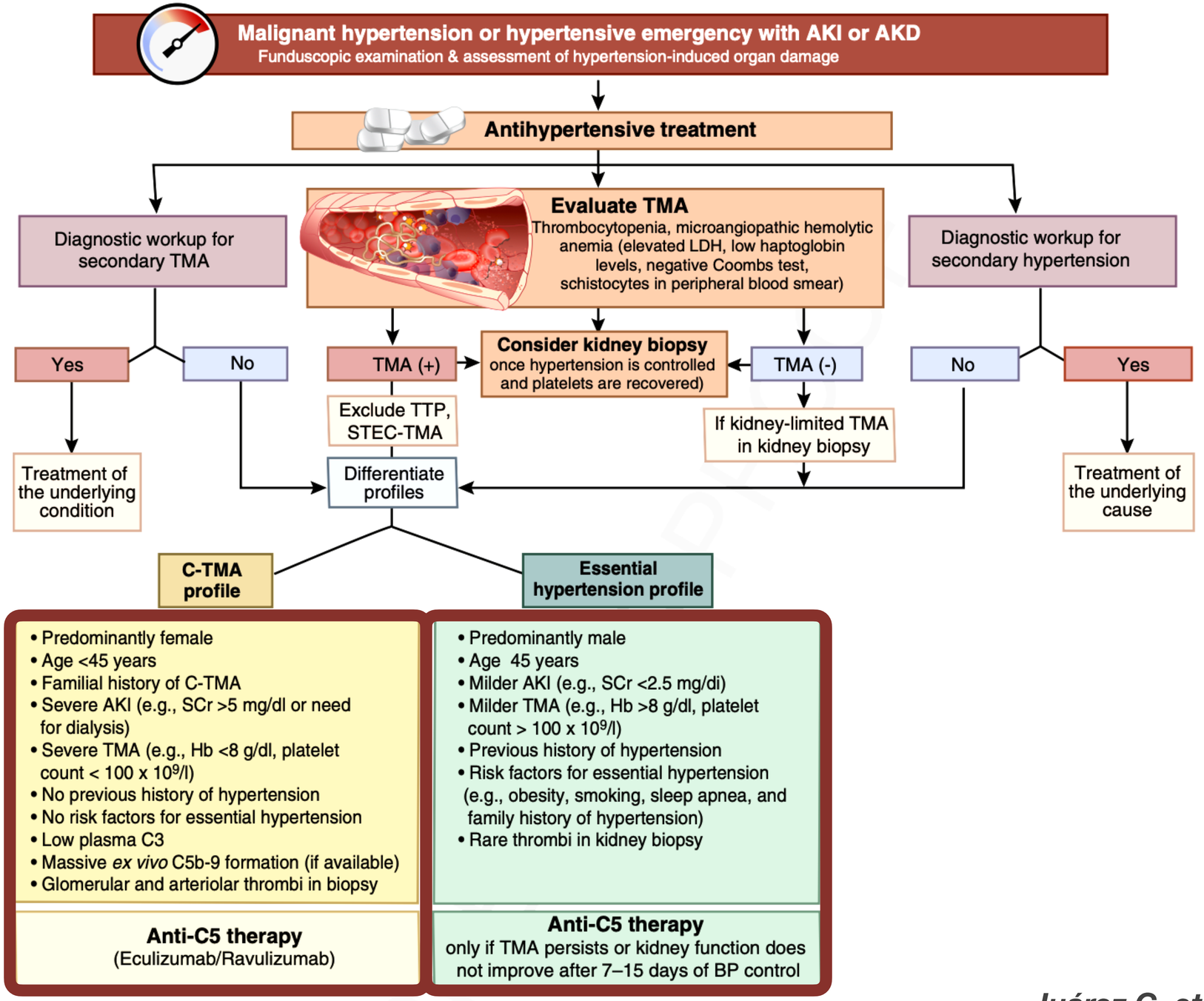
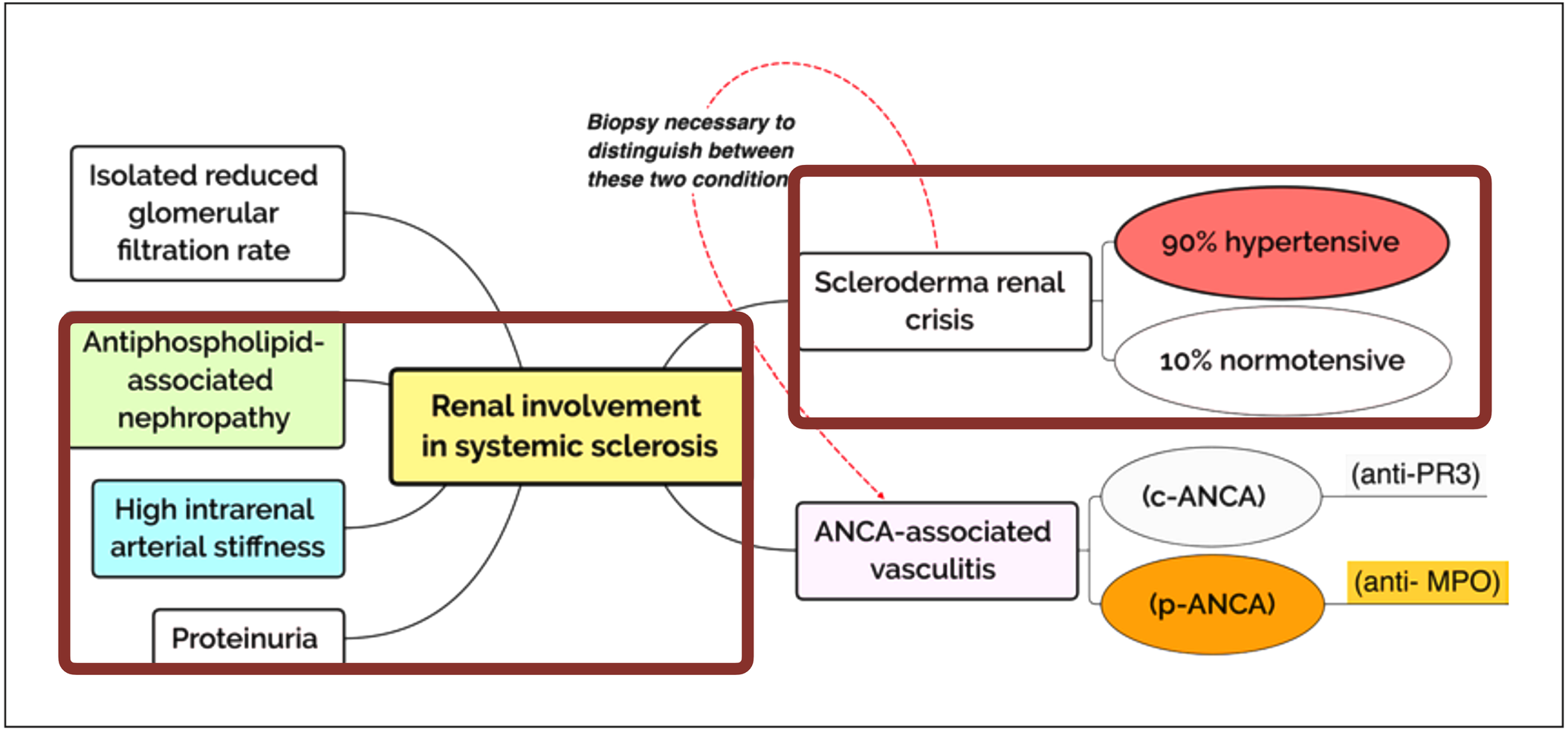
Diagnostic and therapeutic algorithm for patients with severe hypertension and AKI



Juárez G, et al. Kidney Int. 2025: S0085-2538(25)00879-8.

Spectrum of kidney involvement in systemic sclerosis



Chrabaszcz M, et al. Kidney Blood Press Res. 2020;45(4):532-548.

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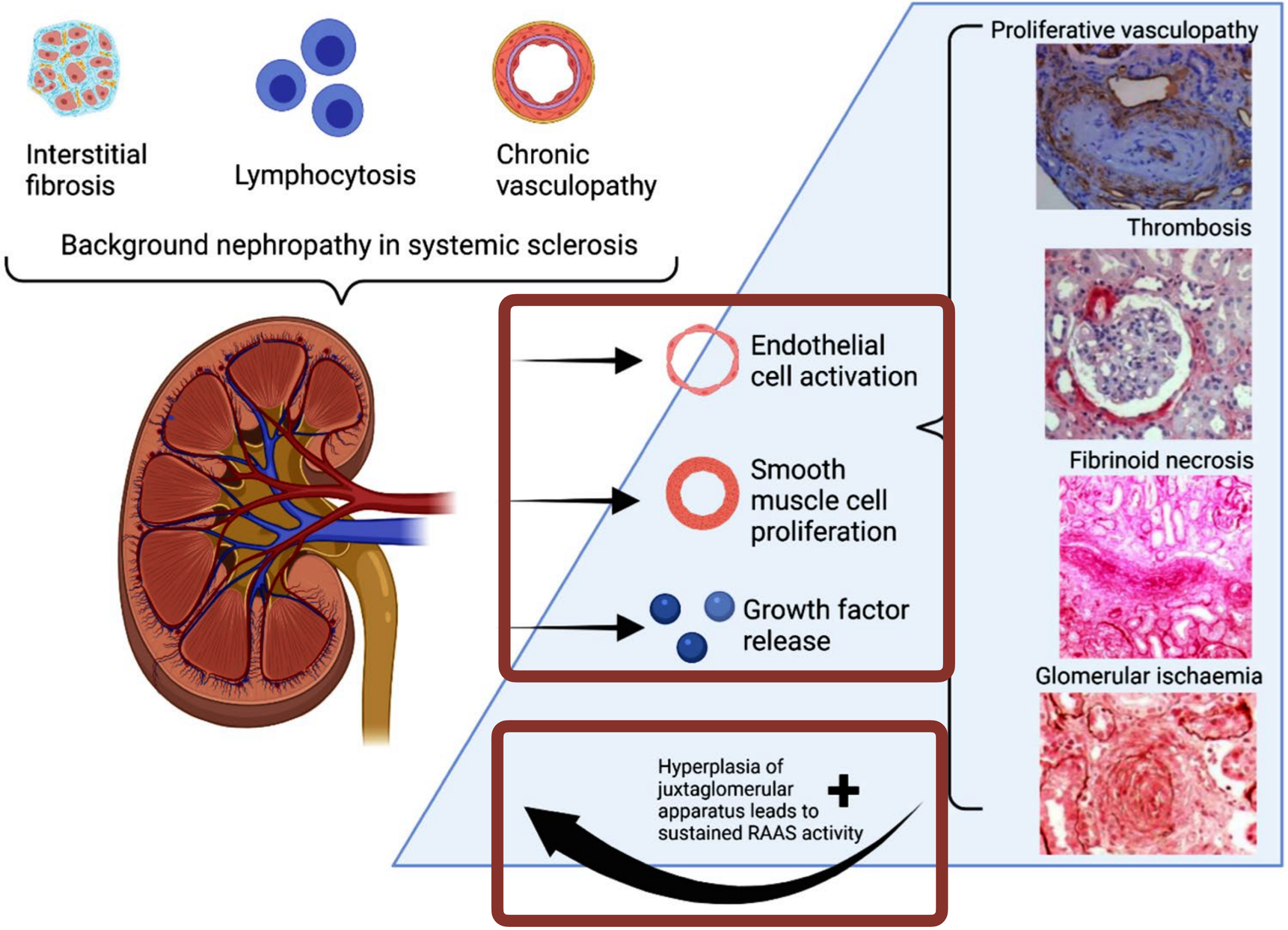
Proposed pathogenesis of SRC



Cole A, et al. Clin Rev Allergy Immunol. 2023; 64(3):378-391.

UKSSG Diagnostic criteria for SRC 2016

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Diagnostic criteria (essential)
New onset BP > 150/85 mmHg or obtained at least twice over 24 h
Increase ≥ 20 mmHg from usual systolic BP
Acute kidney injury stage 1 or higher:
(> 50% increase in serum creatinine from stable baseline or an absolute increase of 26.5 μmol/L)
Supportive evidence (desirable)
MAHA on blood film, thrombocytopaenia and other biochemical findings consistent with haemolysis
Findings consistent with accelerated hypertension on retinal examination
Microscopic haematuria on urine dipstick and/or red blood cells on urine microscopy
Oliguria or anuria
Renal biopsy with typical features of SRC including onion skin proliferation within the walls of intrarenal arteries and arterioles, fibrinoid necrosis, glomerular shrinkage
Flash pulmonary oedema

Cole A, et al. Clin Rev Allergy Immunol. 2023; 64(3):378-391.

Kidney biopsy findings in scleroderma renal crisis

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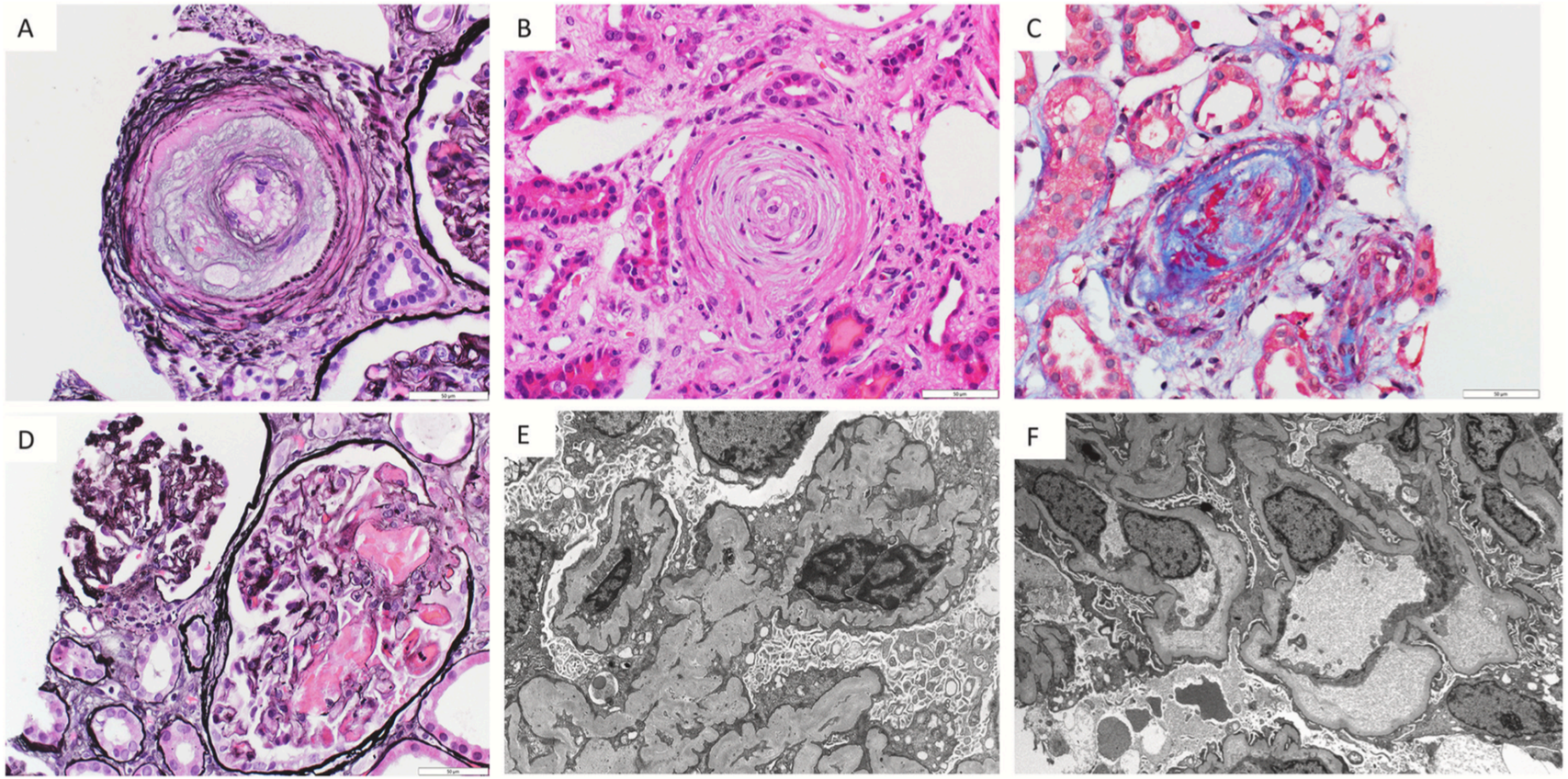

Onion skin proliferation within the walls of intrarenal arteries and arterioles, fibrinoid necrosis, glomerular shrinkage

Fig. 3. Kidney biopsy findings in scleroderma renal crisis
A) A small interlobular artery showing severe mucoid intimal edema which stains light blue on silver stain (X400).
B) An interlobular artery showing mucoid intimal thickening with concentrically arranged myointimal cellular proliferation ("onion skin" lesion)(hematoxylin and eosin)(X400)
C) An arteriole showing severe occlusive intimal fibrosis and intimal and luminal deposition of fibrin, which appears dark red on trichrome stain (X400)
D) The glomerulus on the left exhibits global ischemic type wrinkling of the glomerular basement membrane. The glomerulus on the right shows large intracapillary fibrin thrombi, which appear pink on silver stain (X400)
E) The glomerular basement membrane demonstrates severe wrinkling with a collapsed glomerular tuft (electron microscopy, X4800).
F) There is prominent widening of the subendothelial zone by electron lucent "fluffy" material (electron microscopy, X4000)

Scheen M, et al. Autoimmun Rev. 2023; 22(6):103330.

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SRC is a medical emergency:

- Admit all patients where SRC suspected (i.e. acute hypertension + AKI with clinical features or serological evidence of systemic sclerosis)
- Immediate referral to renal team
- Consider early critical care involvement where appropriate (see below)

Assess severity

Blood pressure

SBP >180 or DBP >110 mm Hg?

No

Aim for a reduction in SBP of 20 mm Hg and DBP 10 mm Hg *per day* with oral antihypertensives (see box)

Oral antihypertensives

1st line
Long-acting ACEi (e.g. ramipril).
Start at moderate dose (e.g. ramipril 5mg) and double dose daily.
Short-acting ACEi (e.g. captopril) only required if haemodynamically unstable.
2nd line ARB (e.g. losartan)
3rd line Calcium antagonist (preferably short acting)
4th line Doxazocin

Yes

Admit to HDU/ITU for haemodynamic monitoring
Aim to reduce MAP by 10-20% within one hour.
Target DBP 100-110 mm Hg within 24hours.
Use oral therapy + IV vasodilators (see box) to achieve targets.

IV vasodilators
IV nitrates (e.g. GTN)
Continuous low-dose iloprost (e.g. 0.9ml/kg/hr)
in addition to standard oral therapy

ACEI: captopril

Other clinical features

Early ITU/HDU referral if any of the following warning signs are present

Seizures
IV phenytoin, brain imaging and neurology opinion

Pulmonary oedema
IV vasodilators (see box) and IV loop diuretic

Tachyarrhythmias
Beta-blockers are relatively contraindicated

Severe AKI
If creatinine 3 times baseline (or >200 µmmol/l where baseline unknown), oligoanuria or hyperkalaemia, may require early renal replacement therapy

Cole A, et al. Clin Rev Allergy Immunol. 2023; 64(3):378-391.

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Renal dysfunction in SSc	Management
SRC	ACE-I are the initial choice of therapy Monitoring BP several times per day with a target of <130/90 mm Hg Other antihypertensive medications (e.g., calcium channel blockers) as needed In case of severe renal failure and/or end-stage renal disease, consider dialysis as required Consider renal transplantation in dialysis-dependent eligible patients (usually within 2 years)
	No evidence to support prophylactic use of ACE-I In cases with dramatic clinical and histological severity, and in those that do not respond to conventional treatment, eculizumab has recently been proposed
Normotensive SRC	Earlier need for renal replacement therapy ACE-I or dialysis seems to be less effective in this group

Chrabaszcz M, et al. Kidney Blood Press Res. 2020;45(4):532-548.

EULAR recommendations for the treatment of systemic sclerosis: 2023 update

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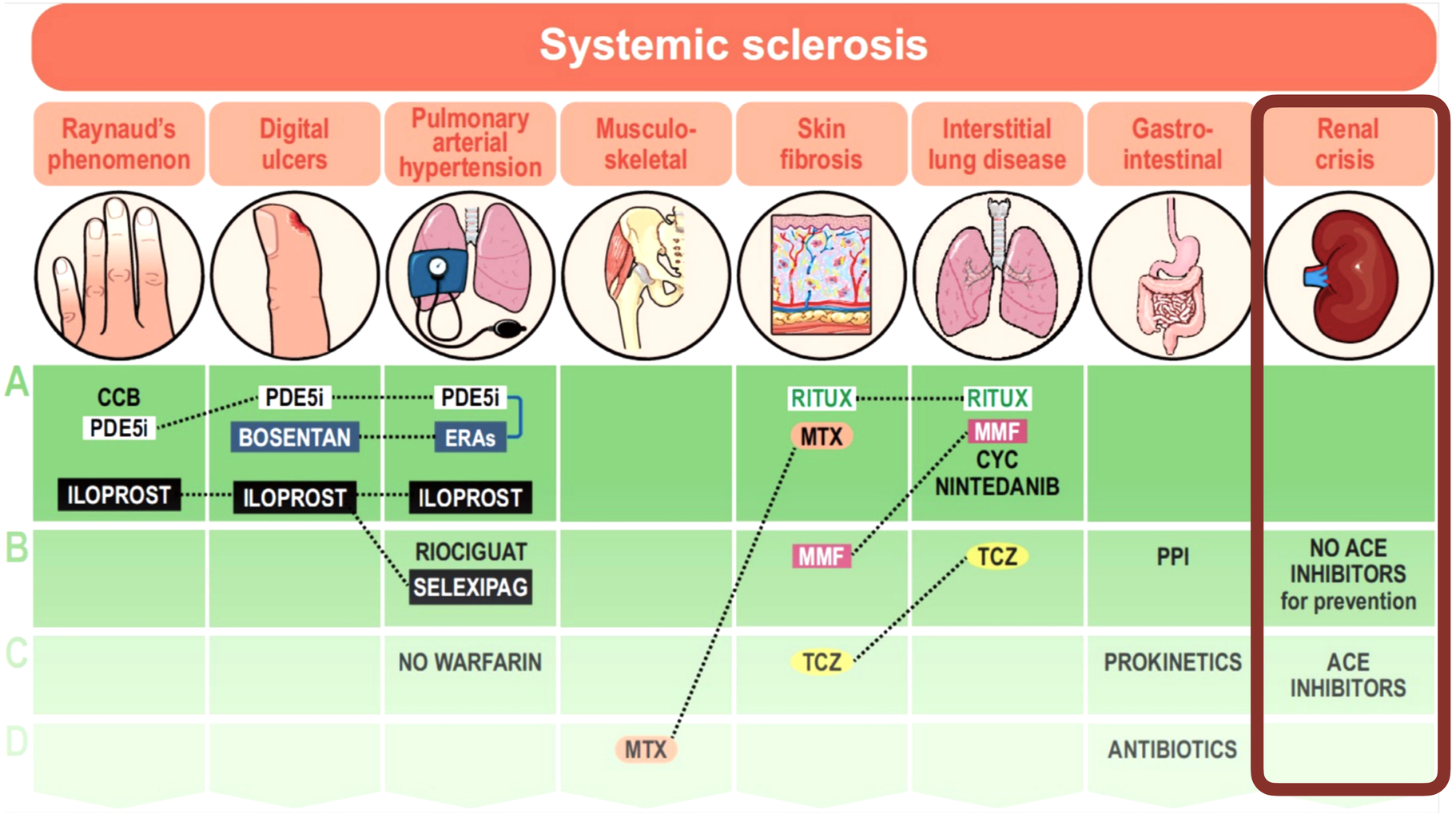

ACE inhibitors should be used immediately at diagnosis of scleroderma renal crisis

SSc patients treated with glucocorticoids should have regular monitoring of blood pressure to detect scleroderma renal crisis

Del Galdo F, et al. Ann Rheum Dis. 2025; 84(1):29-40.

Odds ratio (OR) and hazard ratio (HR) in cohort studies analysing independent risk factors for development of SRC

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	p value	OR	HR	CI	Study
anti-RNA pol III	<0.001	5.86		[2.6–13.2]	Moinzadeh et al. 2020[a]
Chronic kidney disease	<0.004	2.5		[1.34–4.6]	
	<0.001		20.7	[2.2–190.7]	Gordon et al. 2019[b]
Proteinuria	<0.001		183	[19.1–1750]	
	<0.001	5.55		[3.4–8.9]	Moinzadeh et al. 2020[a]
DcSSc vs. LcSSc	0.002	2.54		[1.42–4.5]	
DLCO	<0.001	4.41		[2.01–9.6]	
Glucocorticoid use	0.007	1.93		[1.20–3.1]	
	0.014	3.63		[1.30–10.05]	De Marco et al. 2002[c]
	0.49		1.32	[0.60–2.87]	Butikofer et al. 2020[d]
Hypertension	0.002		2.22	[1.34–3.6]	
	<0.001	13.1		[4.7–36.6]	Gordon et al. 2019[b]
mRSS > 14		3.08		[1.24–7.61]	Avouac et al. 2016[e]
	0.003	10		[2.21–45.9]	De Marco et al. 2002[c]
ACE inhibitors	0.003		2.07	[1.28–3.36]	Butikofer et al. 2020[d]
Tendon friction rub	0.15		1.7	[0.83–3.48]	
	0.0007		2.33	[1.03–6.19]	Avouac et al. 2016[e]
Large joint contracture	0.008	16.12		[2.07–125.2]	De Marco et al. 2002[c]
Heart involvement	0.048	2.93		[1.01–8.4]	

Anti-RNA polymerase III antibody

CKD

Proteinuria

Hypertension

Large joint contracture

Cole A, et al. Clin Rev Allergy Immunol. 2023; 64(3):378-391.

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Renal dysfunction in SSc	Risk factors
SRC	Diffuse skin disease Rapidly progressive skin disease Large joint contractures Prednisolone at a dose >15 mg/day <4 years since scleroderma onset Presence of anti-RNA polymerase antibody Proteinuria Anemia Recent cardiac events Digital pitting scars Myalgias and myopathy Cyclosporin therapy HLA-DRB1*0407 and HLA-DRB1*1304 High serum CD147 levels
Normotensive SRC	Cardiac involvement Previous treatment with glucocorticoid Absence of elevated BP may delay treatment, leading to disease progression

Anti-RNA polymerase III antibody

Chrabaszcz M, et al. Kidney Blood Press Res. 2020;45(4):532-548.

High risk and diagnosis of preeclampsia

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Box 1. Risk Factors for Preeclampsia

Nulliparity
Multifetal gestations
Preeclampsia in a previous pregnancy
Chronic hypertension
Pregestational diabetes
Gestational diabetes
Thrombophilia
Systemic lupus erythematosus
Prepregnancy body mass index greater than 30
Antiphospholipid antibody syndrome
Maternal age 35 years or older
Kidney disease
Assisted reproductive technology
Obstructive sleep apnea

Box 2. Diagnostic Criteria for Preeclampsia

Blood pressure

- Systolic blood pressure of 140 mm Hg or more or diastolic blood pressure of 90 mm Hg or more on two occasions at least 4 hours apart after 20 weeks of gestation in a woman with a previously normal blood pressure
- Systolic blood pressure of 160 mm Hg or more or diastolic blood pressure of 110 mm Hg or more. (Severe hypertension can be confirmed within a short interval (minutes) to facilitate timely antihypertensive therapy).

Proteinuria

- 300 mg or more per 24 hour urine collection (or this amount extrapolated from a timed collection) or
- Protein/creatinine ratio of 0.3 ~~mg/dL~~ or more or
- Dipstick reading of 2+ (used only if other quantitative methods not available)

Or in the absence of proteinuria, new-onset hypertension with the new onset of any of the following:

- Thrombocytopenia: Platelet count less than 100~~,000~~ $\times 10^9$/L
- Renal insufficiency: Serum creatinine concentrations greater than 1.1 mg/dL or a doubling of the serum creatinine concentration in the absence of other renal disease
- Impaired liver function: Elevated blood concentrations of liver transaminases to twice normal concentration
- Pulmonary edema
- New-onset headache unresponsive to medication and not accounted for by alternative diagnoses or visual symptoms

Gestational Hypertension and Preeclampsia: ACOG Practice Bulletin, Obstet Gynecol. 2020; 135(6):e237-e260.

Diagnostic Criteria for Preeclampsia

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Always necessary. . .

Hypertension

- SBP ≥140 mm Hg or DBP ≥90 mm Hg on 2 occasions at least 4 h apart after 20 weeks' gestation in a woman with previously normal BP
- SBP ≥160 mm Hg or DBP ≥110 mm Hg on 1 occasion

. . .And 1 of the following

Proteinuria

- ≥300 mg per 24-h urine collection (or extrapolated from timed collection), or
- Protein/creatinine ratio of ≥0.3 mg/dl, or
- Dipstick reading of 2+ (used only when other methods not available)

Hypertension (≥140/90 mmHg at lease 2 occasion or ≥160/110) and proteinuria (>300 mg/day)

OR any 1 of the following (in the absence of proteinuria)

Thrombocytopenia

- Platelet count <100,000/mm^3

Renal insufficiency

- Serum creatinine concentration >1.1 mg/dl or a doubling of serum creatinine concentration in the absence of other renal disease

Impaired liver function

- Elevated concentration of liver transaminases to 2× normal
- Severe persistent right upper quadrant or epigastric pain unresponsive to medication

Pulmonary edema

- Diagnosed by physical examination or chest x-ray

Neurological signs

- New-onset headache unresponsive to medication and not accounted for by alternative diagnoses or visual symptoms
- Visual disturbances

Fetal growth restriction*

- Estimated fetal weight <10th percentile

Severe organ damage

Ives CW, et al. J Am Coll Cardiol. 2020; 76(14):1690-1702.

Pathogenesis of Preeclampsia

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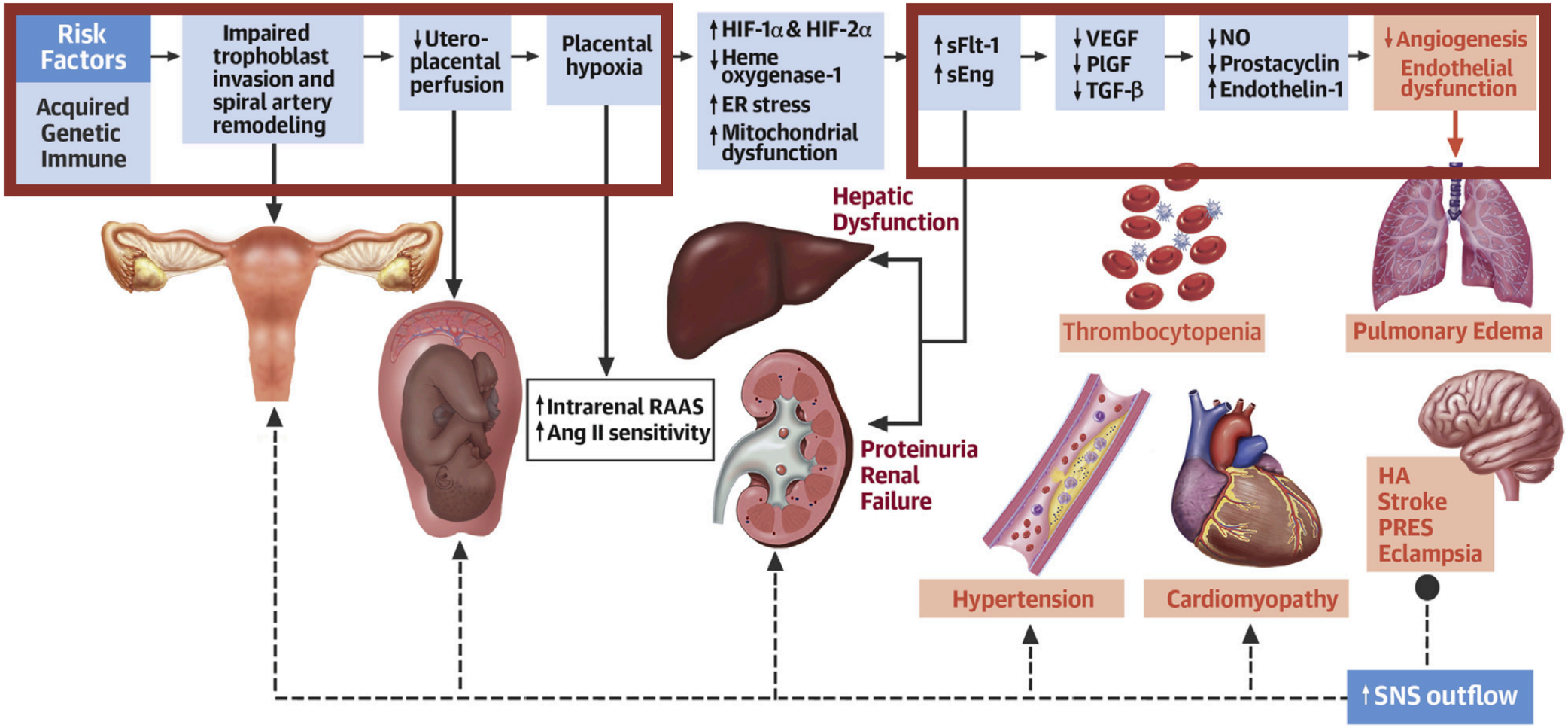

Ives CW, et al. J Am Coll Cardiol. 2020; 76(14):1690-1702.

Role of sFlt1 (soluble fms-like tyrosine kinase 1) and PlGF (placental growth factor) in preeclampsia development

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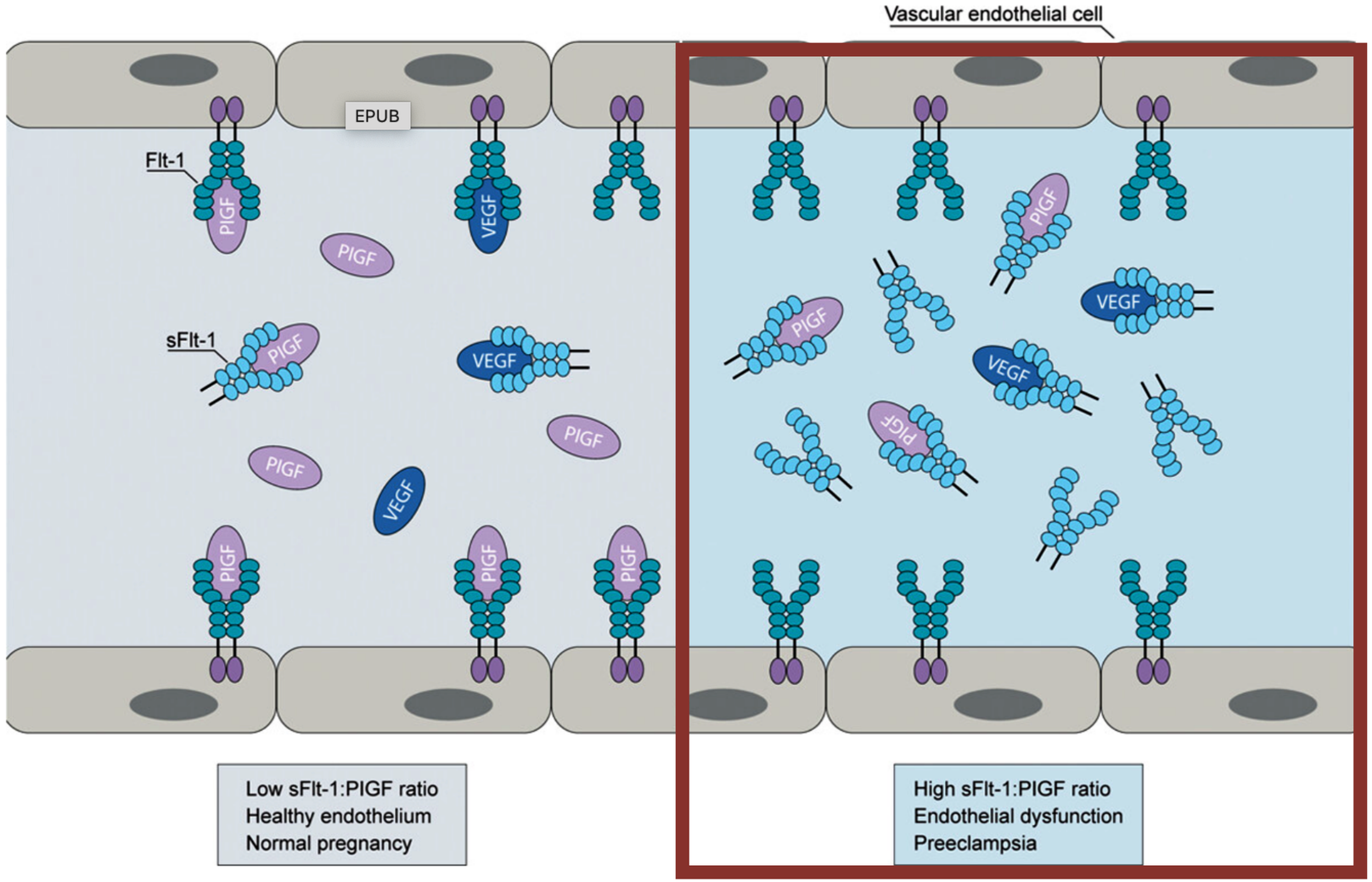


- In pregnant women, sFlt-1 binds and inhibits VEGF (vascular endothelial growth factor) and PlGF in the circulation.
- A high sFlt-1/PlGF ratio, therefore, potentially results in endothelial dysfunction and development of preeclampsia

Abundant sFlt1 bind PlGF: growth factors cannot occupy FLT-1 on the cell surface

Stepan H, et al. Hypertension. 2020; 75(4):918-926.

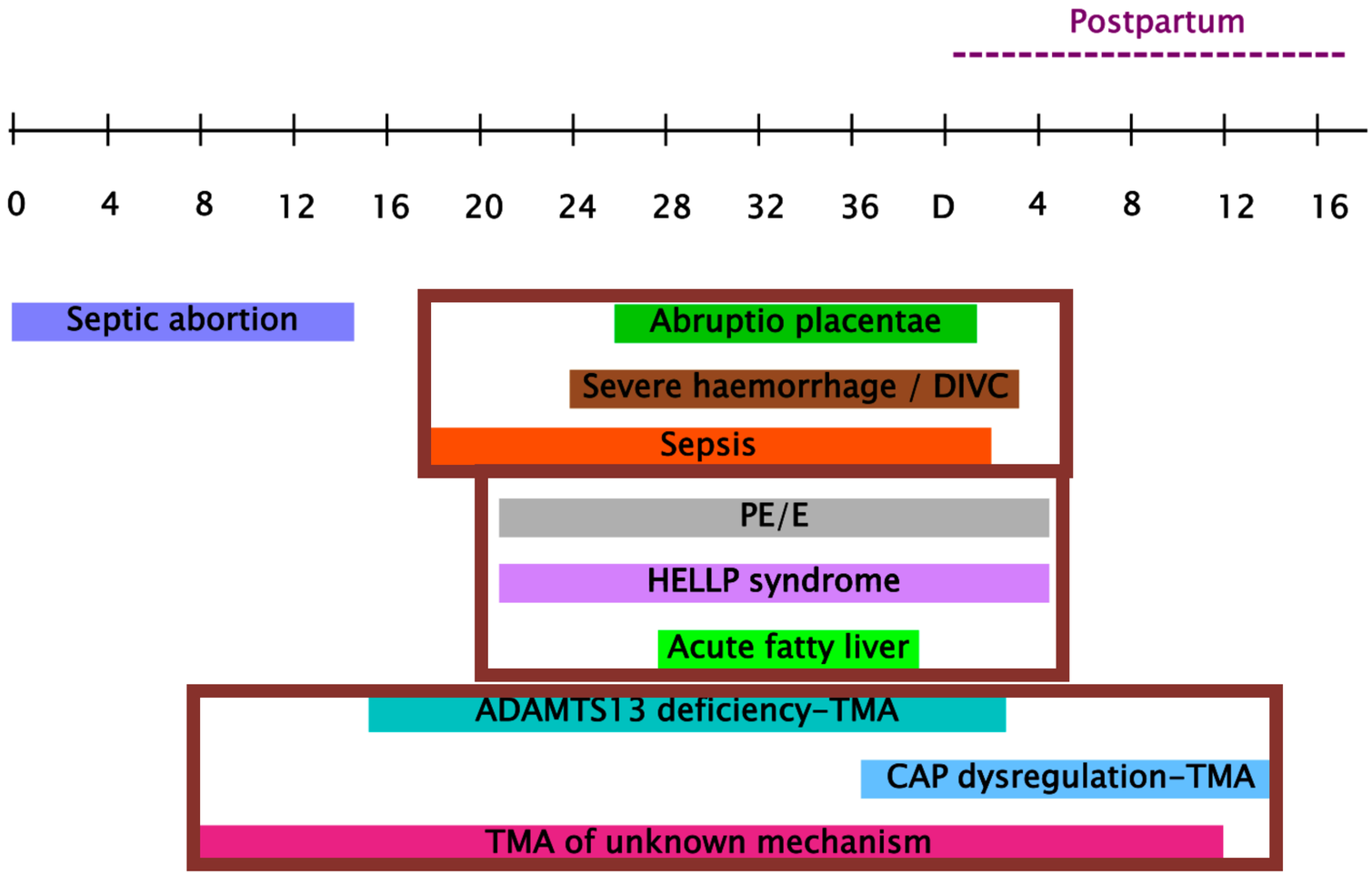
Angiogenic markers used in the prediction and diagnosis of preeclampsia

Angiogenic Marker	Gestation Used	Unlikely to Develop Preeclampsia in the Next Week[a]	At Risk of Developing Preeclampsia within 1–4 wk[a]	Assess as Preeclampsia[a]
PlGF (Triage, Alere/Quidel)	20 to <37 wk	>100 pg/ml	12–100 pg/ml	<12 pg/ml
Ratio sFLT-1-to-PlGF (Elecsys, Roche)	24 to <34 wk	<38	38–85	>85

PlGF, placental growth factor; sFlt-1, soluble fms-like tyrosine kinase receptor 1. Modified from reference 19 and 76, with permission.
[a]Or another form of placental insufficiency, *e.g.*, fetal growth restriction.

Wiles K, et al. Clin J Am Soc Nephrol. 2020; 15(9):1371-1380.

Pregnancy-related AKI depending on their predominant timing of occurrence during pregnancy



Clin J Am Soc Nephrol 7: 2100–2106, 2012.

Phenotype between normal pregnancy, preeclampsia/ HELLP, and kidney disease presenting in pregnancy

Features	Normal Pregnancy	Preeclampsia/HELLP	Lupus Nephritis	Atypical HUS
Gestation	All	>20 wk	Any	Typically peri-/ postpartum
Skin changes/rash	+	—	+	—
Hair loss	+	—	+	—
Edema	+	++	+	+
Hypertension (BP>140/90 mm Hg)	—	++	+	+
UPCR≥0.3 mg/mg (>30 mg/mmol)	—	+	+	+
Hematuria	Trace	—	+	−/+
Anemia	+	+	+	−/+
Thrombocytopenia ($<100\times10^9$/L)	—	+	+	++
Hemolysis	—	+ Improves 48–72 h postdelivery	Rare	++ Persists >72 h after delivery
Serum creatinine	⇓ in T1, ⇓⇓ in T2, ⇑ in T3	⇑	⇑	⇑⇑
Transaminitis	No	Yes	No	No
ESR	⇑	—	⇑	⇑
dsDNA	—	—	⇑	—
Complement	Can ⇑	—	⇓ (within normal range)	⇓ in 30%−50%

Wiles K, et al. Clin J Am Soc Nephrol. 2020; 15(9):1371-1380.

Hypertensive emergencies requiring immediate BP lowering with i.v. drug therapy

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Clinical presentation	Timing and BP target	First-line treatment
Malignant hypertension with or without acute renal failure	Several hours Reduce MAP by 20–25%	Labetalol[a] Nicardipine
Hypertensive encephalopathy	Immediately reduce MAP by 20–25%	Labetalol[a] Nicardipine
Acute coronary event	Immediately reduce SBP to <140 mmHg	Nitroglycerine Labetalol[a]
Acute cardiogenic pulmonary edema	Immediately reduce SBP to <140 mmHg	Nitroprusside or nitroglycerine (with loop diuretic)
Acute aortic dissection	Immediately reduce SBP to <120 mmHg and heart rate to <60 bpm	Esmolol AND nitroprusside or nitroglycerine or nicardipine
Eclampsia and severe preeclampsia/HELLP	Immediately reduce SBP to <160 mmHg and DBP to <105 mmHg	Labetalol[a] or nicardipine and magnesium sulphate

Mancia G, et al. J Hypertens. 2023; 41(12):1874-2071.

Recommendations for acutely managing BP in patients with severe hypertension in pregnancy and pre-eclampsia

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Recommendation	Class[a]	Level[b]
In pre-eclampsia or eclampsia with hypertensive crisis, drug treatment with i.v. labetalol or nicardipine and magnesium is recommended.[971]	I	C
In pre-eclampsia or eclampsia associated with pulmonary oedema, nitroglycerin given as an i.v. infusion is recommended.[242]	I	C
In severe hypertension in pregnancy: • drug treatment with i.v. labetalol, oral methyldopa, or oral nifedipine is recommended. Intravenous hydralazine is a second-line option.[666–668,969,971]	I	C

- **Magnesium sulfate 4 g i.v. over 5 min, then 1 g/h i.v.; or 5 g i.m. into each buttock, then 5 g i.m. every 4 h**
  - **Recommended for eclampsia treatment and pre-eclampsia who have severe hypertension and proteinuria or hypertension and neurological symptoms or signs**
- **A risk of hypotension when magnesium is given concomitantly with nifedipine.**

McEvoy JW, et al. Eur Heart J. 2024; 45(38):3912-4018.

ESC European Society of Cardiology
European Heart Journal (2024) **45**, 3912–4018
https://doi.org/10.1093/eurheartj/ehae178

Recommendations for managing hypertension in pregnancy

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Recommendations	Class[a]	Level[b]
In women with gestational hypertension, starting drug treatment is recommended for those with confirmed office systolic BP ≥140 mmHg or diastolic BP ≥90 mmHg.[661]	I	B
In pregnant women with chronic hypertension, starting drug treatment is recommended for those with confirmed office systolic BP ≥140 mmHg or diastolic BP >90 mmHg.[88,660,661,678]	I	B
In women with chronic and gestational hypertension, it is recommended to lower BP below 140/90 mmHg but not below 80 mmHg for diastolic BP.	I	C
Systolic BP ≥160 mmHg or diastolic BP ≥110 mmHg in pregnancy can indicate an emergency, and immediate hospitalization should be considered.	IIa	C
HBPM and ABPM should be considered to exclude white-coat and masked hypertension, which are more common in pregnancy.[679]	IIa	C

ESC European Society of Cardiology
European Heart Journal (2024) **45**, 3912–4018
https://doi.org/10.1093/eurheartj/ehae178

McEvoy JW, et al. Eur Heart J. 2024; 45(38):3912-4018.

Recommendations for managing hypertension in pregnancy

NEPHROLOGY
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Recommendations	Class[a]	Level[b]
Dihydropyridine CCBs (preferably extended-release nifedipine), labetalol, and methyldopa are recommended first-line BP-lowering medications for treating hypertension in pregnancy.	I	C
RAS blockers are not recommended during pregnancy.[680,681]	III	B

A meta-analysis suggests that beta-blockers and CCBs are more effective than methyldopa in preventing severe hypertension

Methyldopa has been associated with an increased risk of post-partum depression and caution is therefore advised both intra-partum and post-partum

ESC European Society of Cardiology
European Heart Journal (2024) **45**, 3912–4018
https://doi.org/10.1093/eurheartj/ehae178

McEvoy JW, et al. Eur Heart J. 2024; 45(38):3912-4018.

Recommended Pharmacotherapy for the Treatment of Nonsevere and Severe Antepartum Hypertension

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Drug name and class	Starting dose, mg	Maximum daily dose, mg	Titration interval in stable patients, d	Special considerations
Nonsevere hypertension				
First-line agents				
Nifedipine XL (calcium channel blocker)	30 daily	120 or 60 BID	5–7	Contraindicated in heart failure; flushing, headache, and edema are common
Labetalol (β-blocker)	200 BID	2400	2–3	Avoid in patients with bradycardia, bronchospasm, or asthma

Countouris M, et al. Circulation. 2025; 151(7):490-507.

Recommended Pharmacotherapy for the Treatment of Nonsevere and Severe Antepartum Hypertension

NEPHROLOGY
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Drug name and class	Starting dose, mg	Maximum daily dose, mg	Titration interval in stable patients, d	Special considerations
Alternative agents				
Methyldopa (α agonist)	250 BID	3000	2–3	Poorly tolerated: peripheral edema, dry mouth, lightheadedness, drowsiness, and effects on mood; limited availability
Amlodipine (calcium channel blocker)	5 daily	10	5–7	Peripheral edema is common
Hydrochlorothiazide (thiazide diuretic)	12.5 daily	50	3–5	
Furosemide (loop diuretic)	10 daily	160 (can be BID, TID dosing)	3–5	Monitor volume status to minimize risk of placental hypoperfusion
Hydralazine (direct vasodilator)	10 QID	200	2–3	
Carvedilol (β-blocker)	6.25 BID	25 BID (or 50 BID if weight >100 kg)	2–3	Outcome data are limited in pregnancy
Metoprolol tartrate (β-blocker)	12.5 BID	200 BID	2–3	Avoid in patients with bradycardia, bronchospasm, or asthma
Pindolol (β-blocker)	5 BID	60	7–14	
Clonidine (α agonist)	0.1 BID, or 0.1 patch weekly	2.4 or two 0.3-mg patch/24 hrs	7	Can have withdrawal or rebound hypertension

Countouris M, et al. Circulation. 2025; 151(7):490-507.

Interventions to Reduce Preeclampsia

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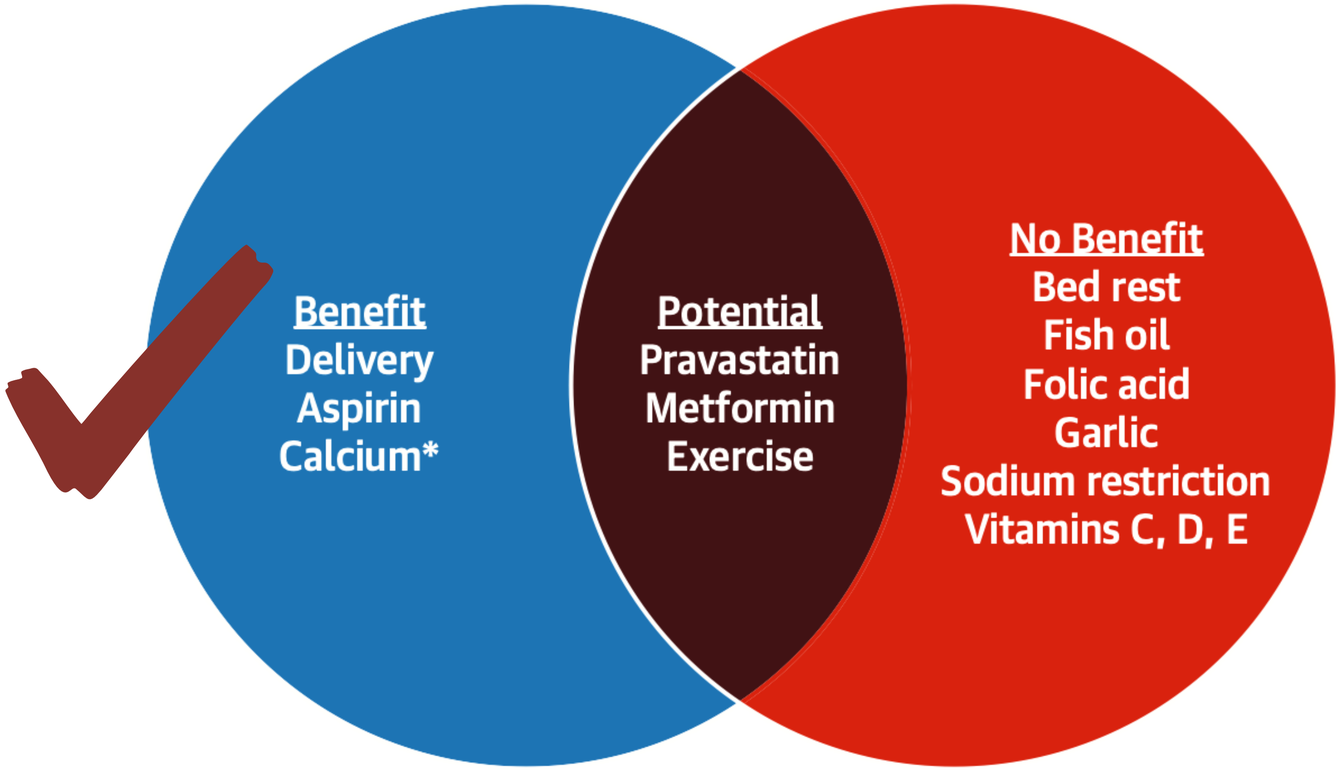
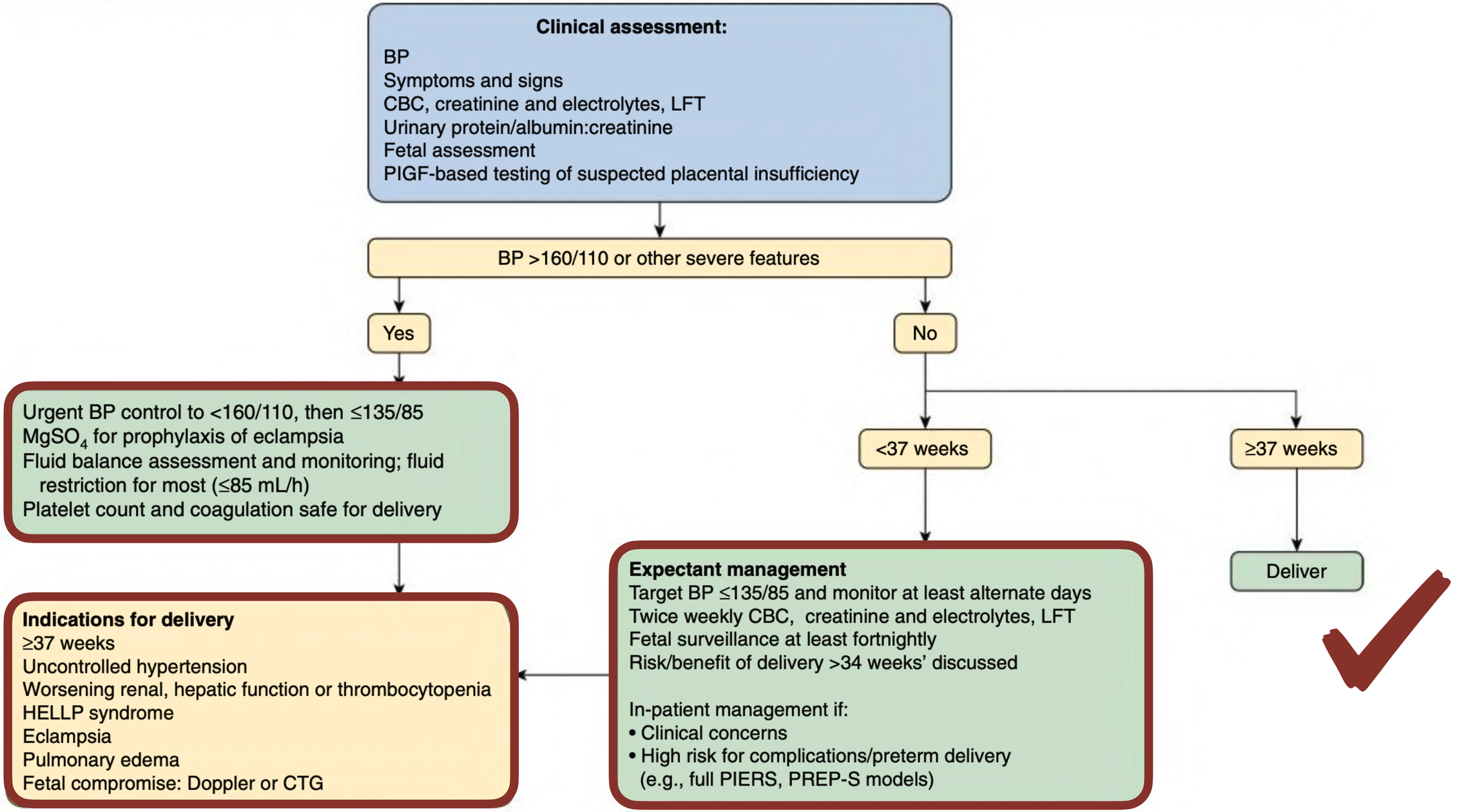


Overview of evidence of treatments to reduce risk of preeclampsia. Interventions with proven benefit are delivery for treatment, and aspirin and calcium for prophylaxis. Pravastatin, metformin, and exercise are currently being investigated and are showing promise. *Only in nutritional deficiency in low-middle income countries.

Ives CW, et al. J Am Coll Cardiol. 2020; 76(14):1690-1702.



Comprehensive Clinical Nephrology 7th edition

Medications in lactation

Drug	Comments
Labetalol	Low levels in breastmilk. Not expected to cause any adverse effects in full term infants.
Nifedipine	Low levels in breastmilk. Not expected to cause any adverse effects in infants.
Methyldopa	Low levels in breastmilk. Not expected to cause any adverse effects in infants.
Hydrochlorothiazide	Low levels in breastmilk. High doses (>25 mg) may cause more intense diuresis and decrease breastmilk production.
Hydralazine	Limited levels in breastmilk. Not expected to cause any adverse effects in infants.
Clonidine	High levels in infant serum with possible side effects in infants and possible effects in breastmilk production. Other agents preferred.
Amlodipine	Limited information shows low levels in breastmilk. Reasonable to use if necessary for maternal treatment.
Azathioprine	Most experts consider acceptable. Low levels in breastmilk. Long-term data are lacking. Possibility of mild neutropenia in the infant.
Calcineurin inhibitors (tacrolimus, cyclosporine)	Limited data, experts agree it is probably safe and acceptable to use.
Prednisone	Low levels in breastmilk. Not expected to cause any adverse effects in infants.
Rituximab	Not well-studied, no long-term data available. Use with caution, if required by the mother, may not be a reason to discontinue breastfeeding.
Mycophenolate Mofetil	Limited data, alternate agent may be preferred.
Cyclophosphamide	Potentially toxic levels in the breastmilk which can cause adverse infant effects. Some experts consider contraindicated.
mTOR inhibitors (sirolimus, everolimus)	Limited data, alternate agent may be preferred.
Belatacept	No information, alternate drug preferred.

Reynolds ML, et al. Adv Chronic Kidney Dis. 2020; 27(6):461-468.

Immunosuppression Medications in Pregnancy

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Drug	Comments
Azathioprine	Generally safe and first-line immunosuppressive agent in pregnancy
Calcineurin inhibitors (tacrolimus, cyclosporine)	Consider early gestational diabetes mellitus (GDM) screen; upward dose adjustment may be necessary to achieve therapeutic level; case reports of reversible fetal nephrotoxicity and hyperkalemia
Prednisone	Consider early GDM screen; in those maintained on long-term steroids, consider providing stress doses during labor and delivery
Rituximab	No teratogenicity described; neonatal B cell depletion possible; long-term neonatal effect is unknown
Mycophenolate Mofetil	Avoid; high rates of fetal loss and congenital anomalies; transition to alternative agent 12 weeks before conception
Cyclophosphamide	Avoid; high rates of fetal loss and congenital anomalies; use in late pregnancy if critical to maternal survival
Mammalian target of rapamycin (mTOR) inhibitors (sirolimus, everolimus)	Limited data; consider transition to alternative agent 12 weeks before conception
Belatacept	Limited human data; weigh risks/benefits in pregnancy

Reynolds ML, et al. Adv Chronic Kidney Dis. 2020; 27(6):461-468.

Consensus definitions for sepsis and septic shock (Sepsis-3)

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Current Guidelines and Terminology	Sepsis	Septic Shock
1991 and 2001 consensus terminology[9,10]	Severe sepsis Sepsis-induced hypoperfusion	Septic shock[13]
2015 Definition	Sepsis is life-threatening organ dysfunction caused by a dysregulated host response to infection	Septic shock is a subset of sepsis in which underlying circulatory and cellular/metabolic abnormalities are profound enough to substantially increase mortality

Singer, M. et al. JAMA. 2016: 315, 801–810.

Consensus definitions for sepsis and septic shock (Sepsis-3)

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Current Guidelines and Terminology	Sepsis	Septic Shock
2015 Clinical criteria	Suspected or documented infection and an acute increase of ≥2 SOFA points (a proxy for organ dysfunction)	Sepsis[a] and vasopressor therapy needed to elevate MAP ≥65 mm Hg and lactate >2 mmol/L (18 mg/dL) despite adequate fluid resuscitation[13]

SOFA score (Sequential Organ Failure Assessment)

Singer, M. et al. JAMA. 2016: 315, 801–810.

Sepsis-associated AKI: consensus report of the 28th ADQI workgroup

Definition = Sepsis-3 criteria + AKI criteria KDIGO

Consensus statement 1a

We propose that sepsis-associated acute kidney injury (SA-AKI) be characterized by the presence of both consensus sepsis criteria (as defined by Sepsis-3 recommendations) and AKI criteria (as defined by Kidney Disease: Improving Global Outcomes recommendations) when AKI occurs within 7 days from diagnosis of sepsis (not graded).

Zarbock A, et al. Nat Rev Nephrol. 2023; 19(6):401-417.

Proposed New Definition of Acute Kidney Injury

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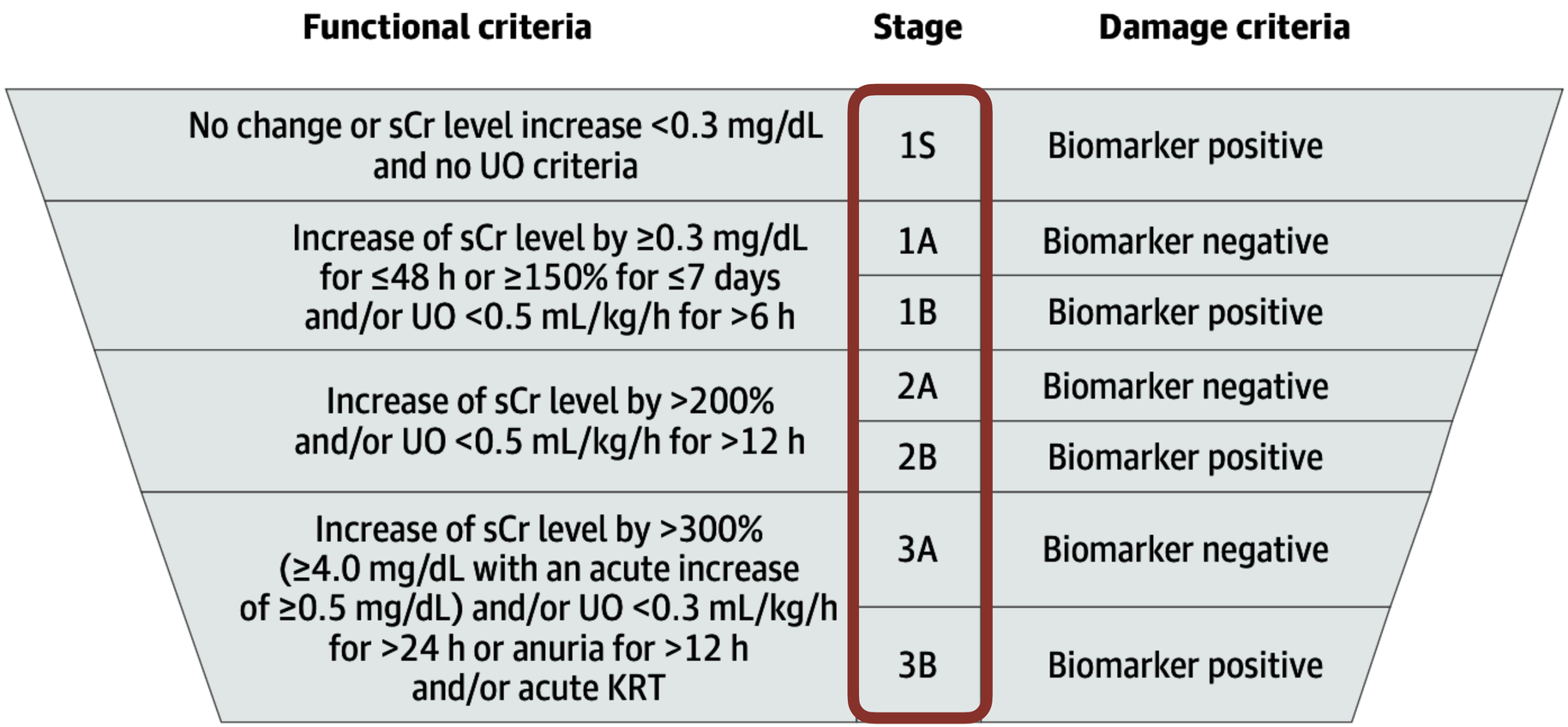
Figure 2. Proposed New Definition of Acute Kidney Injury



Ostermann M, et al. JAMA Netw Open. 2020; 3(10):e2019209.

Markers and characteristics of kidney disease

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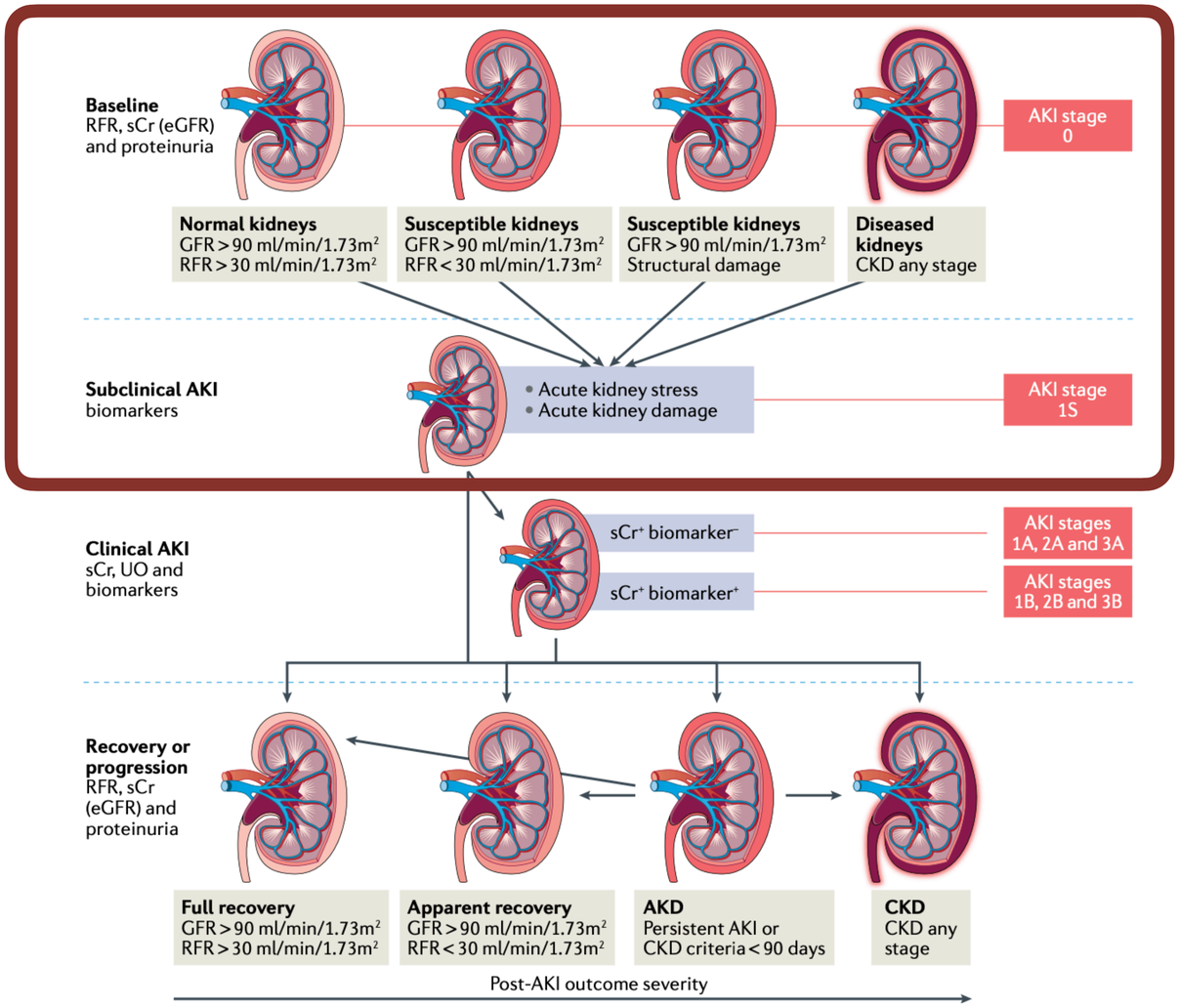

Kellum JA, et al. Nat Rev Nephrol. 2021; 17(7):493-502.

Markers and characteristics of kidney disease

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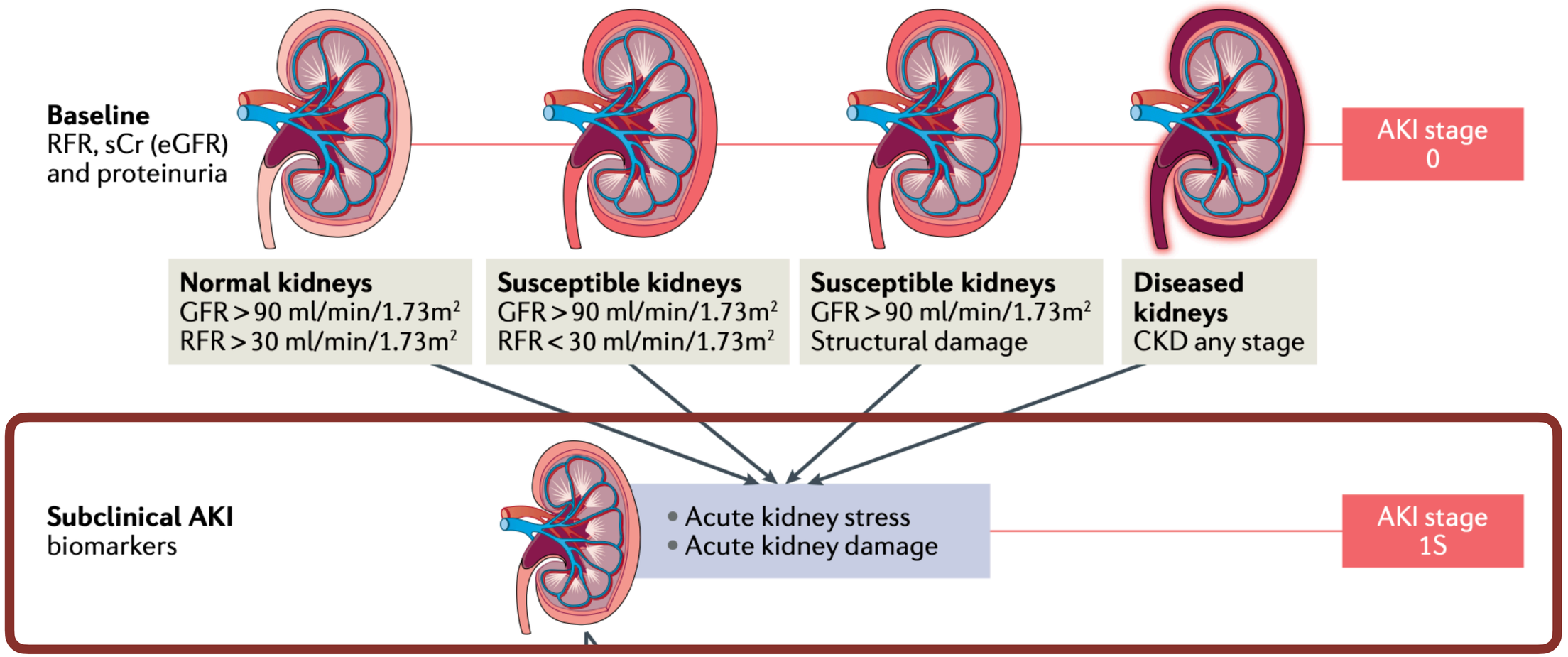

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Markers and characteristics of kidney disease

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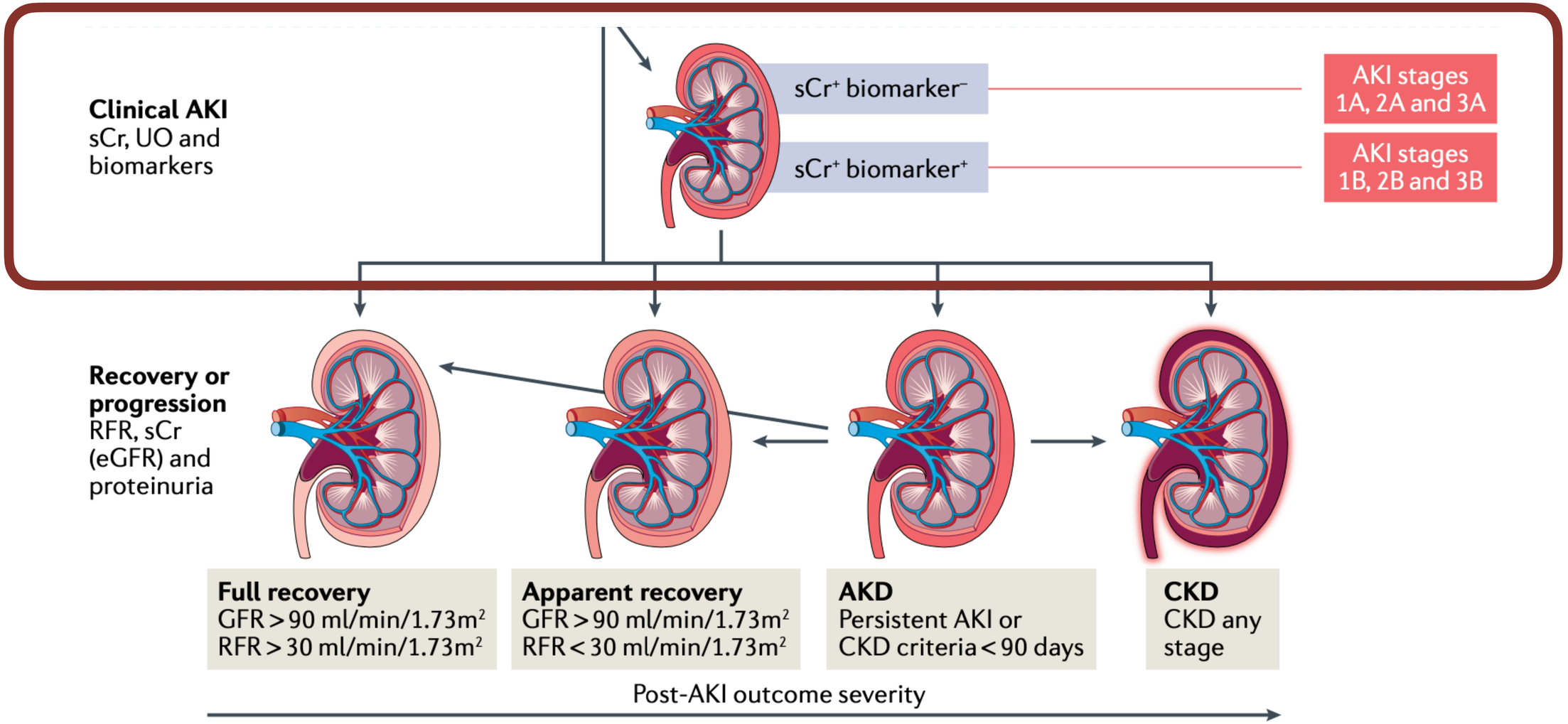

Kellum JA, et al. Nat Rev Nephrol. 2021; 17(7):493-502.

Biomarkers	Sample type or application	Clinical utility
AKI stress marker[a]		
(TIMP-2)•(IGFBP7)	Urine	FDA-approved and CE-marked for clinical use (≥21 and ≥18 years of age, respectively); test designed to predict the risk of developing stage 2–3 AKI within 12 h of assessment
AKI damage markers[a]		
CCL14	Urine	CE-marked for clinical use (≥18 years of age); test designed to predict persistent stage 2–3 AKI
Dipstick albuminuria	Urine	Widely used as an initial screening tool for the evaluation of kidney disease because of its low cost, wide availability and ability to provide rapid point-of-care information
KIM-1	Urine	KIM-1 levels increase 12–24 h after tubular injury, peaking at 2–3 days[174]; FDA-approved and CE-marked for preclinical drug development
Low-molecular-weight proteins	Urine	Widely used to assess proximal tubule cell dysfunction[175]; α_1-microglobulin has been studied for the prediction of AKI-KRT[176], but validation is pending
L-type fatty acid-binding protein	Urine	Japanese MHLW-approved for clinical use (early diagnostic of kidney disease or predicting kidney prognosis)[177]
NGAL	Urine or serum	Levels peak 4–6 h after tubular injury; elevated in sepsis and inflammation[112,178] (thus, clinical use is limited in the ICU setting); commercially available NGAL assays can measure different molecular forms depending on their antibody combination; CE-marked (but not FDA-approved) for clinical use
Urine microscopy	Urine	Oldest and one of the most commonly used tests to differentiate kidney disease aetiology; prone to inter-observer variability[179]; a urine microscopy score based on the number of granular casts and/or kidney tubular epithelial cells per high-powered field[180] has been proposed for sepsis-associated AKI[180] but validation is pending

Kidney tubular epithelial cells

Zarbock A, et al. Nat Rev Nephrol. 2023; 19(6):401-417.

Prospective observational cohort studies with discovery and validation cohorts for AKI cast scoring index[51]

Grade 1—no casts or RTE

Grade 2—≥1 cast or RTE but <10% of LPF

Grade 3—many casts or RTEs (10-90% of LPF)

Grade 4—sheet of muddy brown casts and RTEs in >90% of LPF

Prospective observational cohort for development of urinary sediment scoring system[52]

0 points—no casts or RTE seen

1 point each—1-5 casts per LPF or 1-5 RTEs per HPF

2 points each—≥6 casts per LPF or ≥6 RTEs per HPF

Prospective multicenter observational cohort for derivation of urine microscopy score[53]

0 points—no casts or RTE seen

1 point each—1 cast or 1 RTE per HPF

2 points each—2-4 casts or RTEs per HPF

3 points each—≥5 casts or ≥5 RTEs per HPF

HPF=high power field; LPF=low power field; RTE=renal tubule epithelial cells

stems

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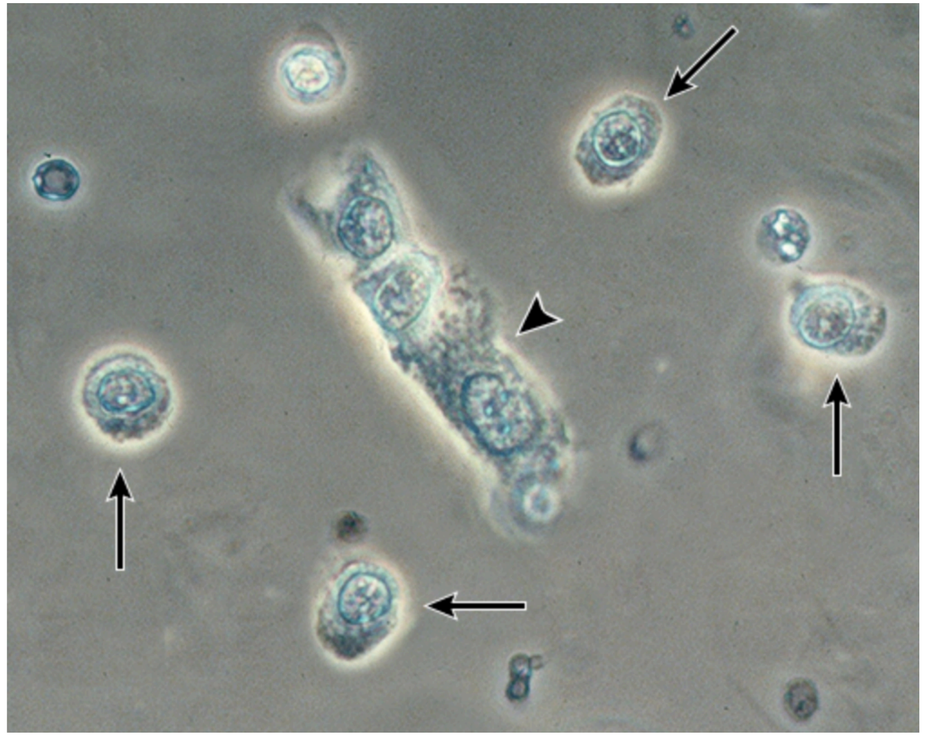
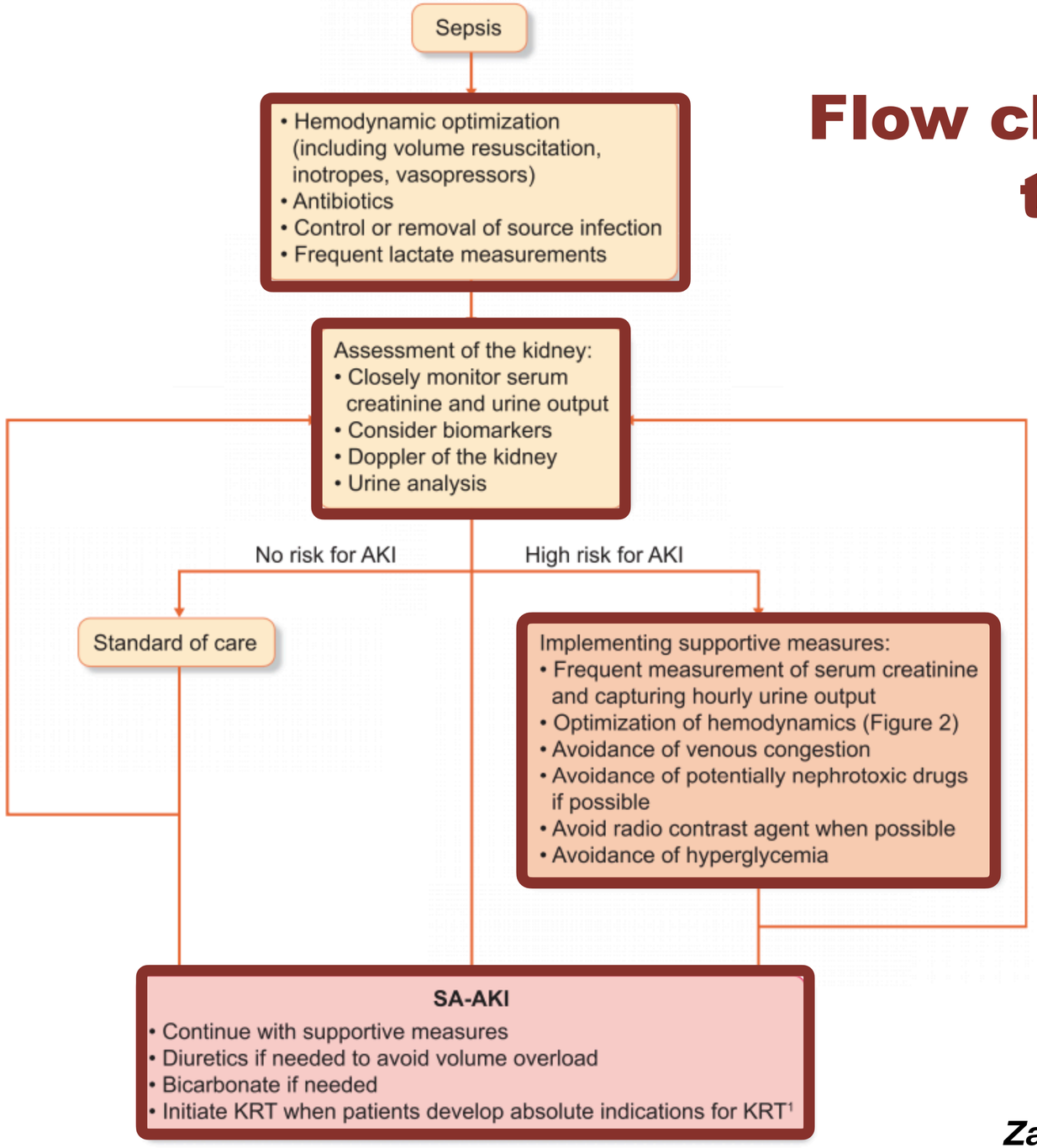
Urinalysis score above 3 was predictive of severe AKI and was highly correlated with biomarkers of tubular injury



Poston TJ, et al. BMJ 2019: 364: K4891.

Flow chart of the diagnostic and treatment algorithm

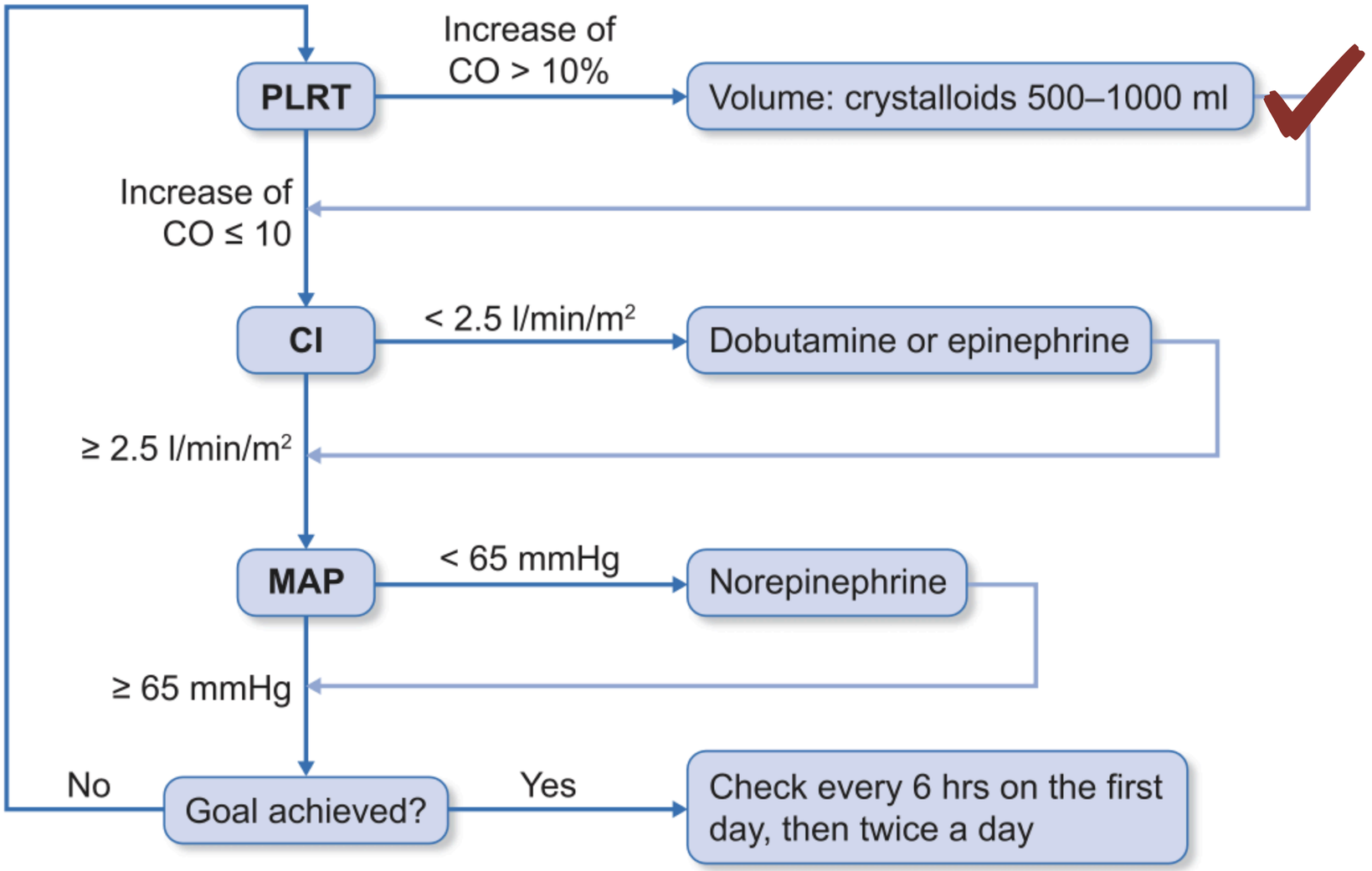


Optimization of the volume and hemodynamic status

Avoidanced of venous congestion

Zarbock A, et al. Nephrol Dial Transplant. 2024 ;39(1):26-35.

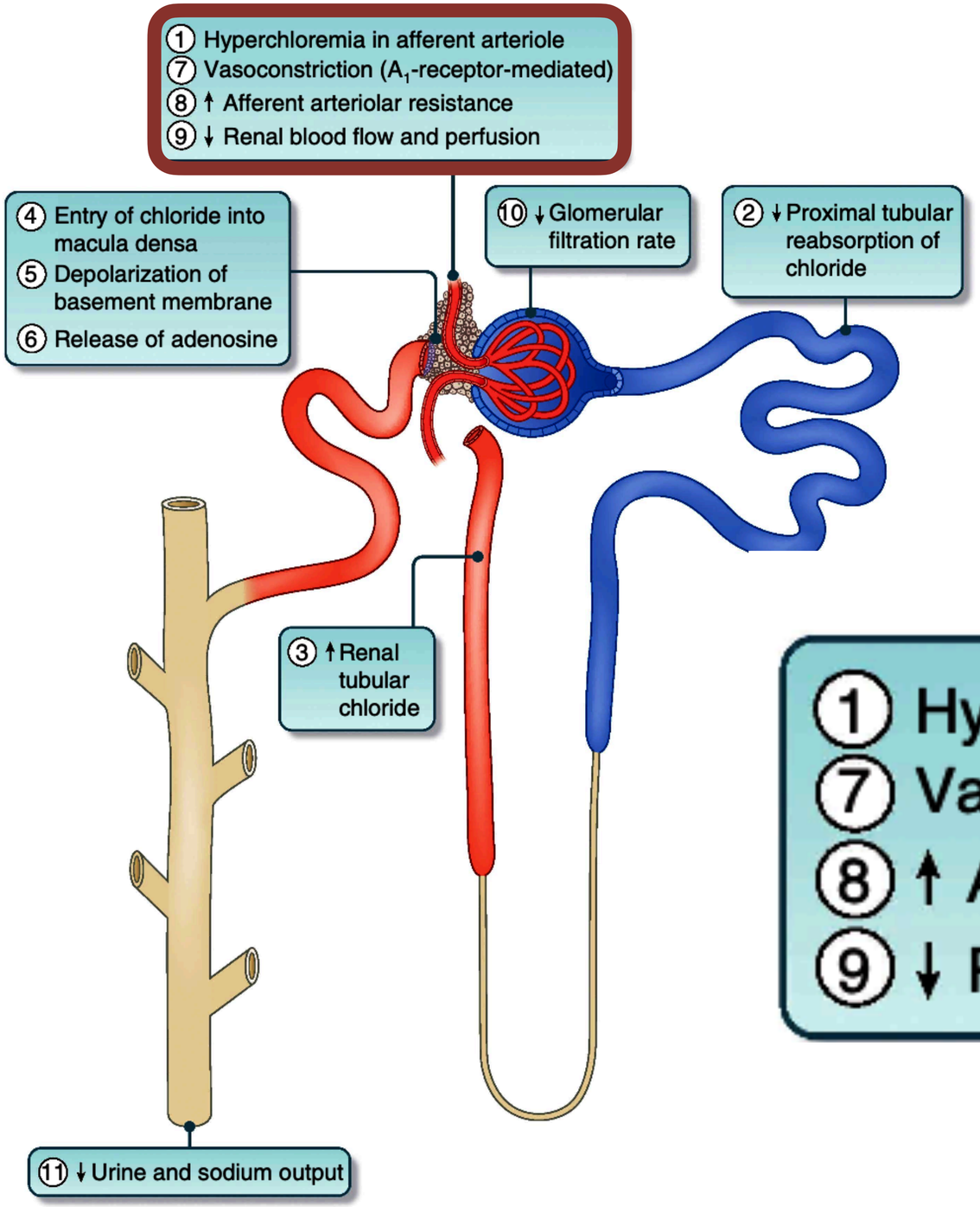
Optimization of the volume and haemodynamic status



Balanced crystalloid solutions are the first choice for fluid replacement and are preferred over chloride-rich solutions and synthetic colloids

Zarbock A, et al. Nephrol Dial Transplant. 2024 ;39(1):26-35.

Sequential effects of hyperchloremia on the kidney



① Hyperchloremia in afferent arteriole
⑦ Vasoconstriction (A_1-receptor-mediated)
⑧ ↑ Afferent arteriolar resistance
⑨ ↓ Renal blood flow and perfusion

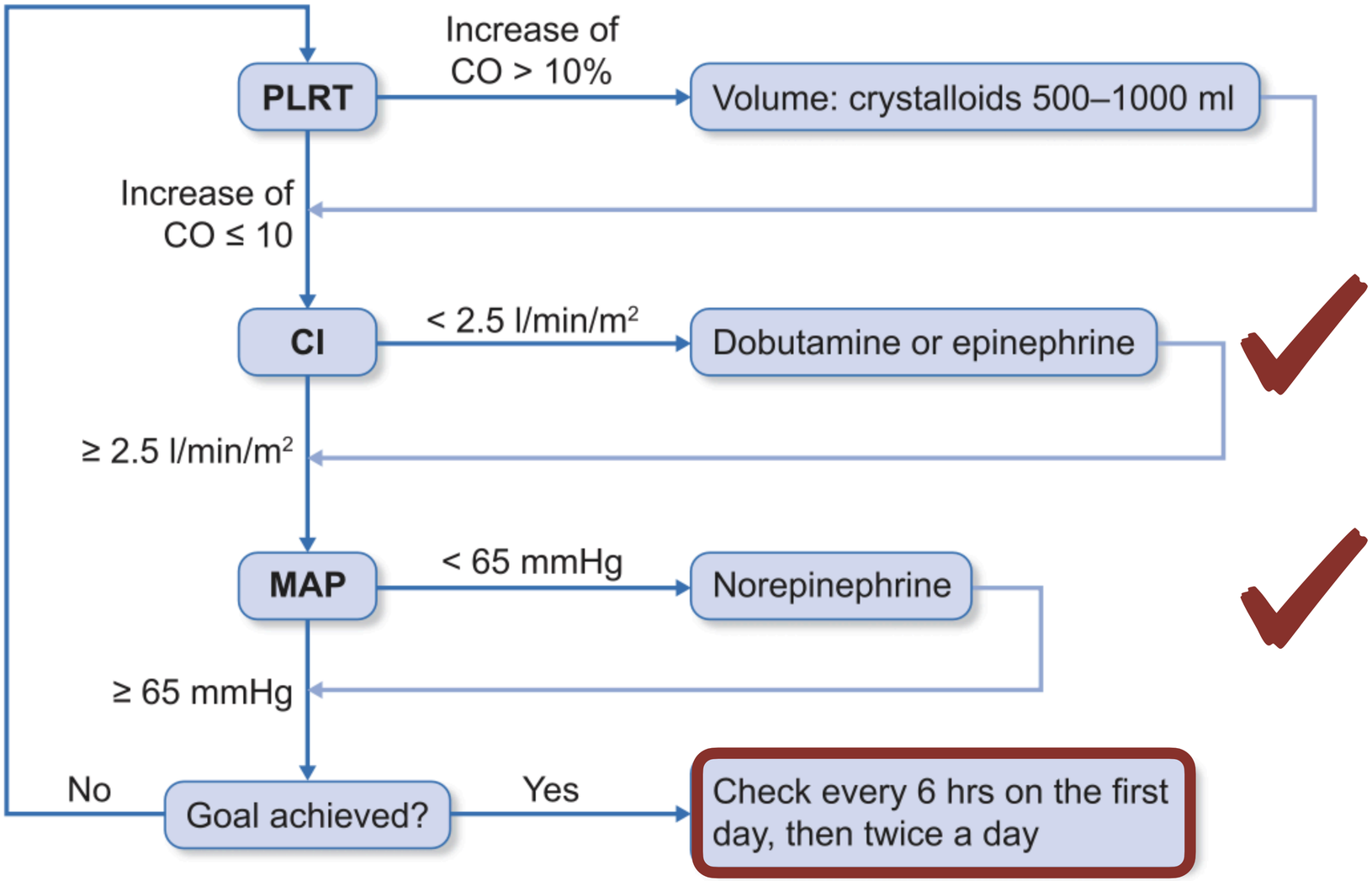
Lobo DN, et al. Kidney Int. 2014; 86(6):1096-105.

Sequential effects of hyperchloremia on the kidney

Metabolic	• Hyperchloremic acidosis • ↑Need for buffers to correct acidosis
Body water	• Possible damage to the endothelial glycocalyx • ↑Interstitial fluid volume leading to edema
Renal	• Renal edema and capsular stretch leading to intrarenal tissue hypertension • Renal vasoconstriction, ↓renal blood flow and renal tissue perfusion • ↓Glomerular filtration rate, urine volume, and sodium excretion

Lobo DN, et al. Kidney Int. 2014; 86(6):1096-105.

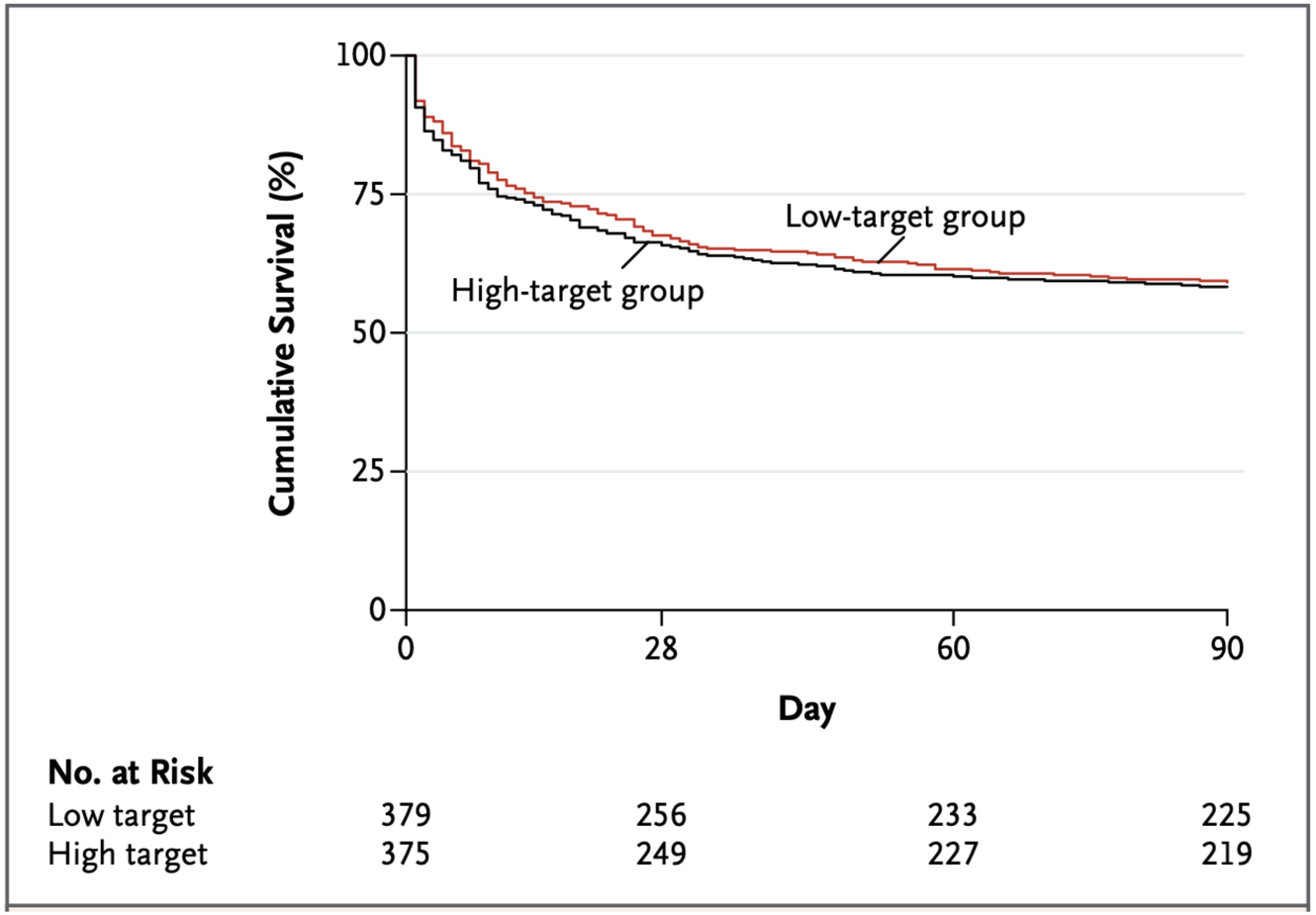
Optimization of the volume and haemodynamic status



Norepinephrine and vasopressin remain first-line agents for the treatment of septic shock.

Zarbock A, et al. Nephrol Dial Transplant. 2024 ;39(1):26-35.

High versus low BP target in patients with septic shock



- In a multicenter, open-label trial, we randomly assigned 776 patients with septic shock to undergo resuscitation with a mean arterial pressure target of either 80 to 85 mm Hg (high-target group) or 65 to 70 mm Hg (low-target group). The primary end point was mortality at day 28.

Targeting a MAP of 80 to 85 mmHg, as compared with 65 to 70 mmHg, in patients with septic shock undergoing resuscitation did not result in significant differences in mortality at either 28 or 90 days.

Asfar P, et al. N Engl J Med. 2014; 370(17):1583-93.

High versus low BP target in patients with septic shock

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Variable	Low-Target Group (N=388)	High-Target Group (N=388)	P Value
Duration of catecholamine infusion — days	3.7±3.2	4.7±3.7	<0.001
Primary outcome: death at day 28 — no. (%)*	132 (34.0)	142 (36.6)	0.57
Secondary outcomes — no./total no. (%)			
Death at day 90†	164 (42.3)	170 (43.8)	0.74
Survival at day 28 without organ support‡	241 (62.1)	235 (60.6)	0.66
Doubling of plasma creatinine	161 (41.5)	150 (38.7)	0.42
No chronic hypertension	71/215 (33.0)	85/221 (38.5)	0.32
Chronic hypertension	90/173 (52.0)	65/167 (38.9)	0.02
Renal-replacement therapy from day 1 to day 7	139 (35.8)	130 (33.5)	0.50
No chronic hypertension	66/215 (30.7)	77/221 (34.8)	0.36
Chronic hypertension	73/173 (42.2)	53/167 (31.7)	0.046

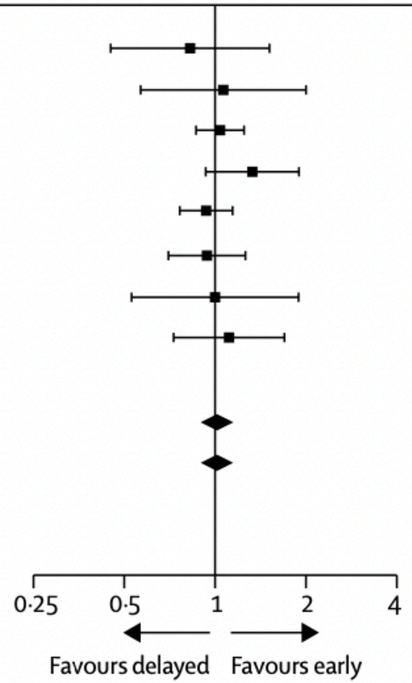
Among patients with chronic hypertension, those in the high-target group required less renal-replacement therapy than did those in the low-target group

Asfar P, et al. N Engl J Med. 2014; 370(17):1583-93.

Delayed versus early initiation of renal replacement therapy for severe acute kidney injury: a systematic review and individual patient data meta-analysis of randomised clinical trials

A

	28-day mortality, n/N (%)		Risk ratio (95% CI)	Weight (%)
	Delayed RRT	Early RRT		
Combes et al, 2015	10/22 (45%)	11/20 (55%)	0·83 (0·45–1·51)	3·1
Wald et al, 2015	15/52 (29%)	13/48 (27%)	1·07 (0·57–2·00)	2·9
Gaudry et al, 2016	134/308 (44%)	129/308 (42%)	1·04 (0·87–1·25)	34·2
Zarbock et al, 2016	48/119 (40%)	34/112 (30%)	1·33 (0·93–1·90)	9·1
Barbar et al, 2018	102/241 (42%)	111/245 (45%)	0·93 (0·76–1·14)	28·2
Lumlertgul et al, 2018	35/60 (58%)	36/58 (62%)	0·94 (0·70–1·26)	13·3
Srisawat et al, 2018	9/18 (50%)	10/20 (50%)	1·00 (0·53–1·89)	2·8
Geri et al, 2019	13/17 (76%)	11/16 (69%)	1·11 (0·73–1·70)	6·4
Total	**366/837 (44%)**	**355/827 (43%)**		
Random-effects one-step model			1·01 (0·91–1·13)	
Random-effects two-step model			1·01 (0·91–1·13)	
I^2=0·0%				
τ^2=0·0				



The timing of RRT initiation does not affect survival in critically ill patients with severe AKI in the absence of urgent indications for RRT.

Gaudry S, et al. Lancet. 2020; 395(10235):1506-1515.

Indications for dialysis

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- A Refractory Acidosis
- E Refractory hyperkalemia
- I Intoxication : methanol, ethylene glycol, lithium
- O Refractory volume Overload
- U Uremia : uremic pericarditis, encephalopathy

- OR BUN>100, Cr>10 in non-hypaercatabolic state
- BUN>70,Cr >7 in hypercatabolic state

Comparison of two delayed strategies for renal replacement therapy initiation for severe acute kidney injury (AKIKI 2): a multicentre, open-label, randomised, controlled trial

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- **More-delayed strategy: RRT initiation was postponed until mandatory indication (hyperkalemia or metabolic acidosis or pulmonary edema) or until BUN to 140 mg/dL**

	Univariable analysis		Multivariable analysis	
	Hazard ratio (95% CI)	p value	Hazard ratio (95% CI)	p value
More-delayed strategy	1·34 (0·96–1·89)	0·13	1·65 (1·09–2·50)	0·018
Simplified Acute Physiology Score III	1·03 (1·02–1·05)	<0·0001	1·03 (1·01–1·05)	0·0005

Severe AKI patients with oliguria > 72 h or BUN > 112 mg/dL would mandate immediate RRT.

Gaudry S, et al. Lancet. 2021; 397(10281):1293-1300.

Controversies in AKI: KDIGO Conference

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- **The 2012 KDIGO AKI guideline suggested initiating RRT emergently in the presence of life-threatening changes in fluid, electrolyte, and acid–base balance.**
- **Optimal timing for short-term KST remains unknown**
- **KST initiation should be considered when metabolic and fluid demands exceed the kidney's capacity**
- **Risk of complications, global prognosis, potential for recovery, and patient preferences should be considered when taking the decision**

Ostermann M, et al. Kidney Int. 2020;98(2):294-309.

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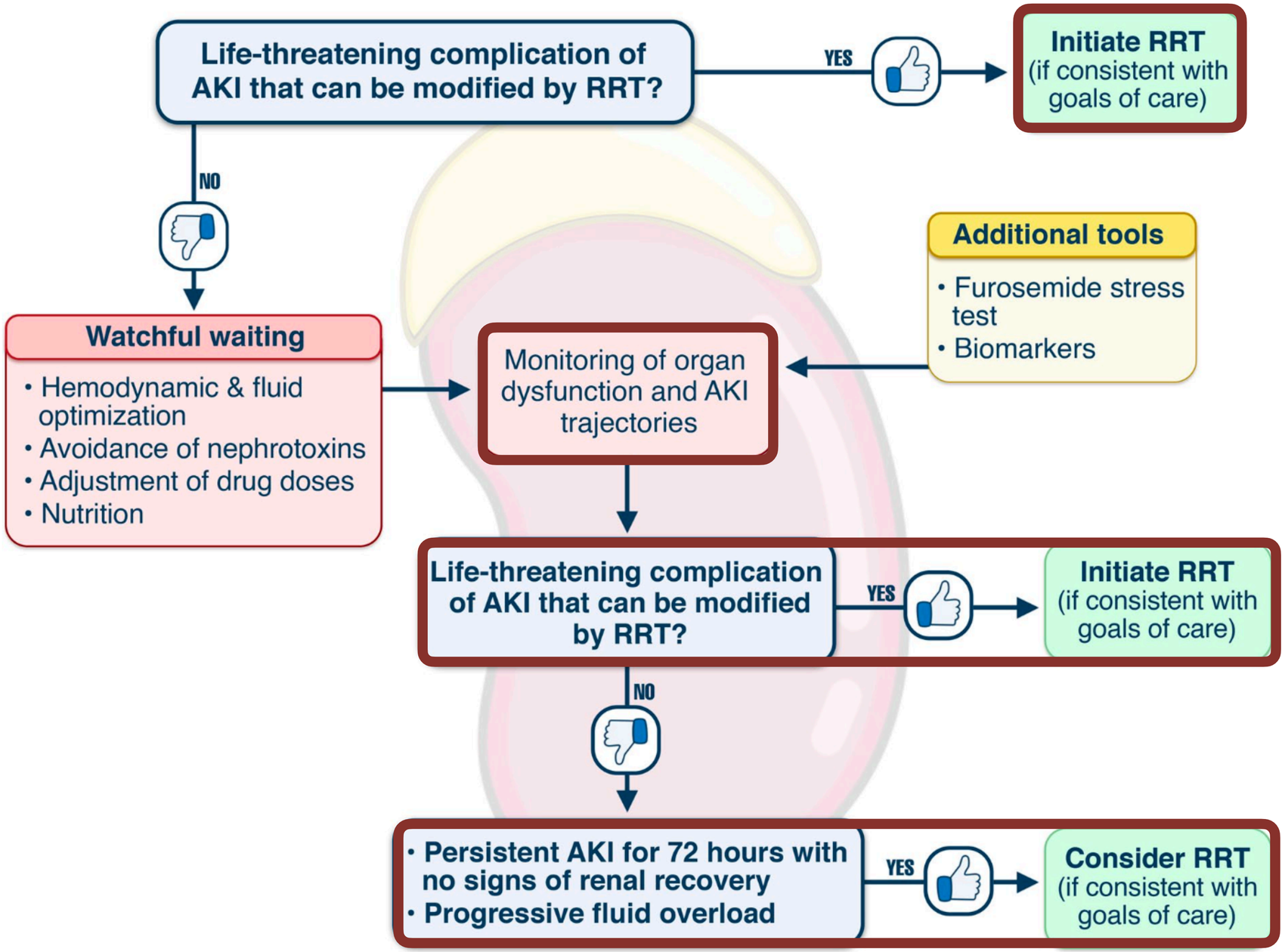
A proposed algorithm for the initiation of renal replacement therapy



Wald R, et al.. Intensive Care Med. 2022; 48(10):1368-1381.

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Table 2 Characteristics of renal replacement therapy modalities used to treat acute kidney injury in the ICU

Characteristic	RRT modality			
	IHD	SLED	CRRT	PD
Treatment duration (h)	3–6	6–18	24	24
Frequency	3 times per week plus additional treatments as indicated	3 times per week plus additional treatments as indicated	Daily	Daily
Mode of solute transport	Diffusion/convection/both	Diffusion/convection/both	Diffusion/convection/both	Diffusion
Blood flow (ml/min)	200–350	100–300	100–250	N/A
Dialysate flow (ml/min)	300–800	100–300	0–50	N/A
Filter size (m^2)	1.7–2	0.4–1.7	0.6–1.5	N/A
Urea clearance (ml/min)	150–180	90–140	20–45	15–35
Need for anticoagulation	Can be readily delivered without anticoagulation	Can be readily delivered without anticoagulation	Generally required	No

RRT renal replacement therapy, *IHD* intermittent hemodialysis, *SLED* sustained low efficiency dialysis, *PD* peritoneal dialysis, *h* hours, *N/A* not applicable, m^2 square-meters, *ICP* intracranial pressure, *DDS* dialysis disequilibrium syndrome, *CVC* central venous catheter, *PDC* peritoneal dialysis catheter, *CKD* chronic kidney disease *Rx* prescription, *ICU* intensive care unit, *RO* reverse osmosis, *HD* hemodialysis

Wald R, et al.. Intensive Care Med. 2022; 48(10):1368-1381.

Continuous Renal Replacement Therapy

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- **Potential setting in AKI**

- **Using CRRT > standard intermittent RRT, for hemodynamically unstable patients. (2B)**
- **Using CRRT > intermittent RRT, for AKI patients with acute brain injury or other causes of increased intracranial pressure or generalized brain edema. (2B)**

KDIGO. Kidney Int Supplements. 2012: 2: 8–12.

Continuous Renal Replacement Therapy (+/IV)

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- ผู้ที่ยังมีความดันโลหิตต่ำแม้จะได้รับยากระตุ้นความดันโลหิตในขนาดสูง และจำเป็นต้องได้รับการบำบัดทดแทนไตควรได้รับการพิจารณาว่าน่าจะได้ประโยชน์จากวิธีการบำบัดทดแทนไตชนิดต่อเนื่อง (CRRT)
  - Dopamine> 15 ug/kg/min or
  - Epinephrine or norepinephrine > 0.1 ug/kg/min

CRRT or SLED (+/IV)

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- ภาวะสมองบวมหรือมีความเสี่ยงที่จะเกิดภาวะสมองบวม เช่น ภาวะตับวายเฉียบพลัน เส้นเลือดในสมองอุดตันเฉียบพลัน สมองขาดออกซิเจน เนื่องจากหัวใจหยุดเต้น (Hypoxic ischemic encephalopathy)
- ความผิดปกติทางเมตาบอลิกที่ยังเกิดต่อเนื่องที่ไม่สามารถแก้ไขได้ด้วยวิธีการฟอกเลือดชนิดชั่วคราว ได้แก่ ภาวะเลือดเป็นกรดอย่างรุนแรง ภาวะโพแทสเซียมในเลือดสูง
- ปริมาณสารน้ำในร่างกายเกินที่ไม่สามารถขจัดออกด้วยการใช้ยา หรือวิธีการฟอกเลือดชนิดชั่วคราว โดยเฉพาะในผู้ป่วยที่มีส่วนเกินมากกว่าร้อยละ 10 ของน้ำหนักเดิม
- มีความจำเป็นที่จะต้องควบคุมปริมาณน้ำและสมดุลกรดด่างรวมถึงเกลือแร่ในเลือดอย่างต่อเนื่อง

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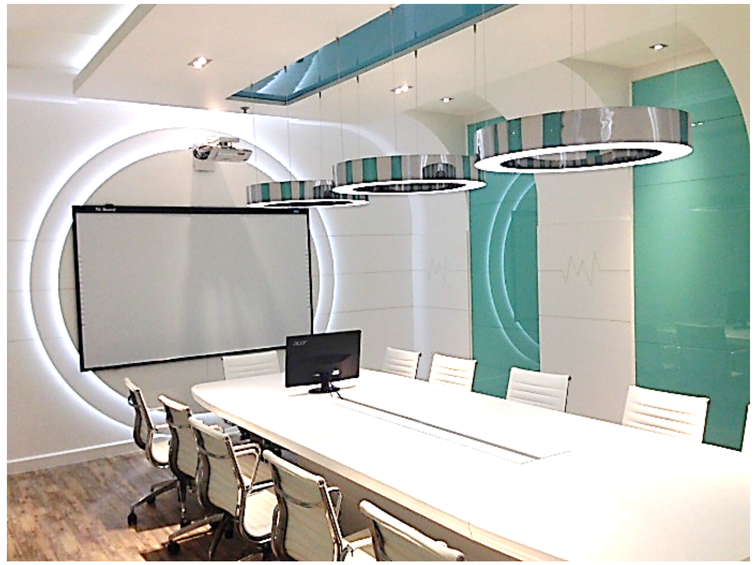

Intelligence Dialysis Center
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