A 32 y/o male with Hypertension and Hypokalemia

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Outlines

- Case presentation
- Approach to hypertension and hypokalemia
- What is happen for the patient?
- Approach to patient with renovascular hypertension?
- What is happen for the patient?

Case presentation

- A 32 years old male was consulted for CT-Angiography of renal vasculature.
- He was admitted in the Semirom hospital about 2 months ago due to COVID-19 infection.
- He had high blood pressure around 200-220 mmHg in systol
- He recieved hemodialysis for 11 sessions due to volume overload besides rising in serum creatinine.
- He was discharged with serum creatinine= 1.7mg/dL

Past medical history

- He had a history of hypertension for about 1 year.
- In his laboratory data:
- Serum creatinine= 1.5-1.8mg/dL. K=3.9-4.2meq/L
- U/A: no protein or blood.
- Ultrasonography: R kidney: 110mm. L kidney: 105mm
- Any extra evaluation was done by physician.

In clinic visit:

- He had no symptoms except dyspnea and history of DOE(FC II)
- PH.Ex:
- His blood pressure: in Rt arm: 180/110mmHg. And in Lt arm: 160/110mmHg.
- Low extrimity blood pressure was not measured.
- Peripheral pulses was palpable and symmetric in upper and lower extrimities.

Case presentation

- Heart examination: S1 & S2 with normal intensity without any murmur also no coarctation murmur was heard.
- Abdominal auscultation: No bruit was heard.
- Lung auscultation: decreased lung sounds in base of both lungs.
- He had no rales in both lungs.

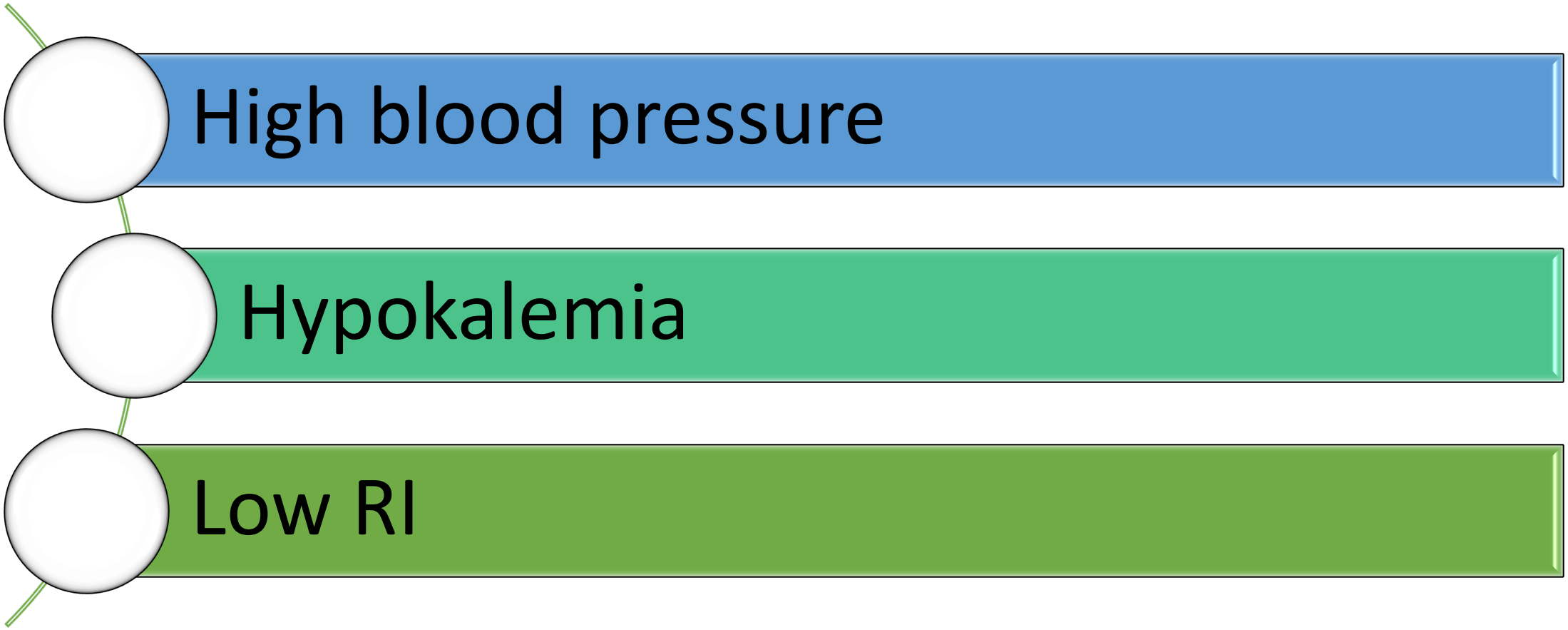
In the presentation day:

- Serum Cr=1.7mg/dL K=3.1meq/L. U/A: Nl in laboratory reports 1 week before and serum Cr= 1.7mg/dL K= 2.5meq/L. in a day before.
- Drug history: Amlodipin 5mg BID, Carvedilol 12.5mg BID, Spironolactone 25mg daily- did not use from 1 weeks ago- Furosemide 40mg daily.

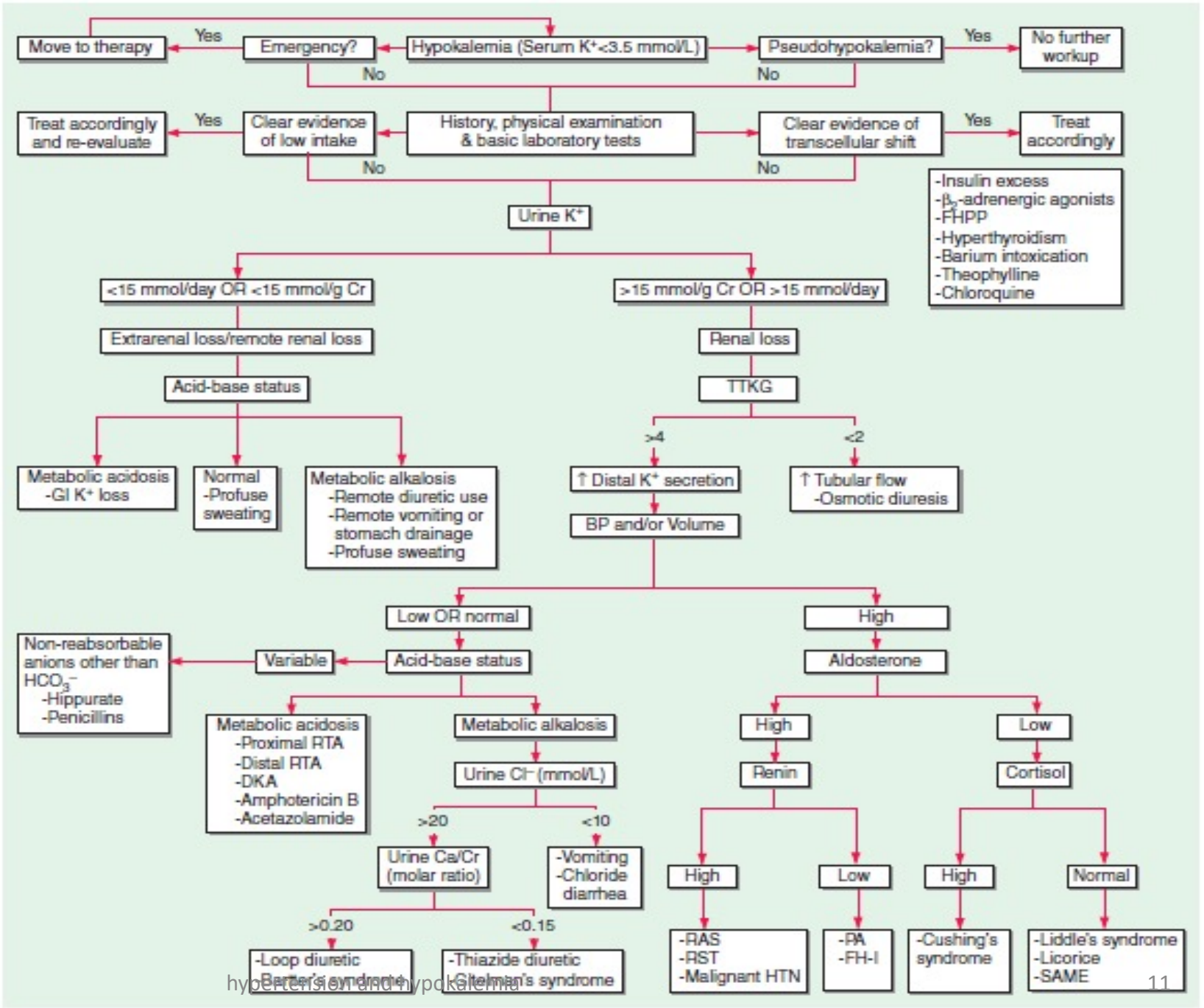
In the presentation day:

- Doppler sonography: decreased RI in left kidney 0.49. and in right kidney. 0.82: RAS in left kidney
- Echocardiography: EF= 35%. No valvular heart disease

What should I do?



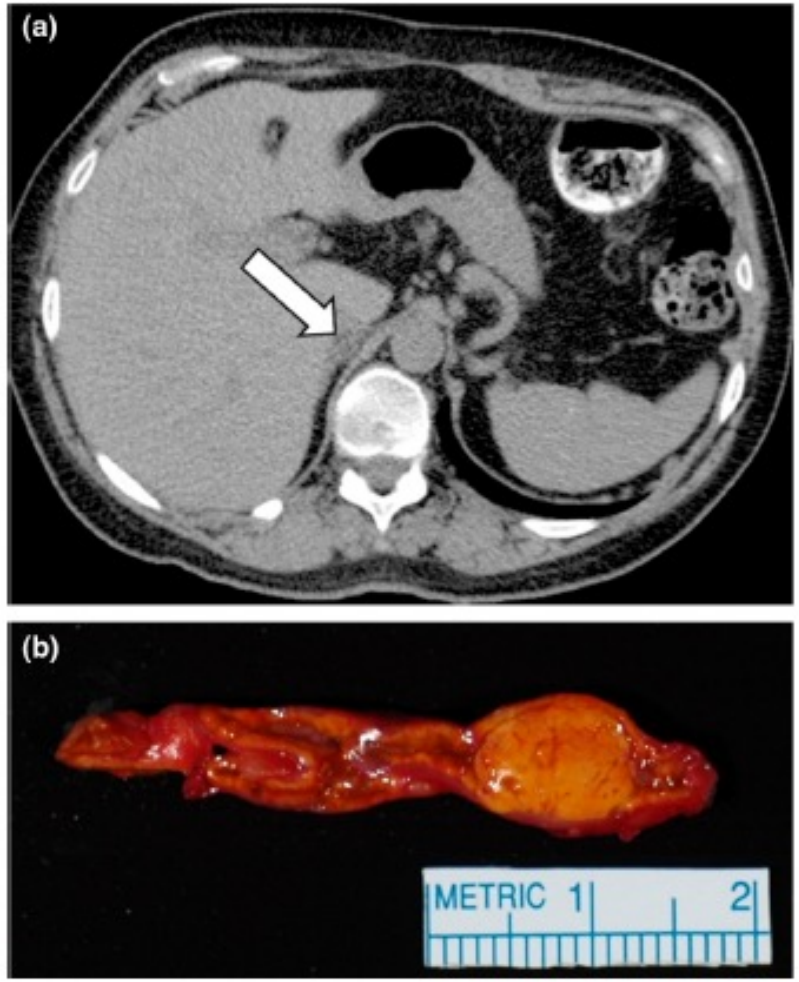
Approach to patient with HTN and hypokalemia



Endocrine cause of Hypertension & Hypokalemia	
Low Renin and High Aldosterone	**Primary Aldosteronism** Aldosterone-producing adenoma (APA)—30% of cases Bilateral idiopathic hyperplasia (IHA)—60% of cases Primary (unilateral) adrenal hyperplasia—2% of cases Aldosterone-producing adrenocortical carcinoma—<1% of cases Familial hyperaldosteronism (FH) Ectopic aldosterone-producing adenoma or carcinoma—<0.1% of cases
Low Renin and Low Aldosterone	Congenital adrenal hyperplasia 11β-Hydroxylase deficiency 17α-Hydroxylase deficiency Deoxycorticosterone-producing tumor Primary cortisol resistance Apparent mineralocorticoid excess (AME)/11β-HSD 2 deficiency Genetic Acquired
Cushing Syndrome	

Primary aldosteronism

- The most common form of secondary HTN
- Surgically cured or
- Pharmacotherapy



Primary aldosteronism (PA)

- PA is frequently **undiagnosed** and untreated, leading to aldosterone-specific **cardiovascular morbidity** and **nephrotoxicity**.
- Thus, clinicians should perform case detection testing for PA at least once in **all patients** with hypertension.

Who should be screened for PA?

- Unlike other adrenal disorders (e.g. Cushing syndrome), there is **no typical PA phenotype** to guide the clinician to suspect PA.
- Serum potassium status is not a reliable guide for screening for PA because 72% of patients with PA are normokalemic.

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Society guidelines on PA recommend testing high-risk groups for PA.

1. Patients with sustained BP > 150/100 mmHg on each of 3 measurements obtained on different days
2. Patients with HTN resistant to 3 conventional antihypertensive drugs (including a diuretic) or controlled BP on 4 or more antihypertensive drugs
3. Patients with HTN and spontaneous or diuretic-induced hypokalaemia
4. Patients with HTN and adrenal incidentaloma
5. Patients with HTN and sleep apnoea
6. Patients with HTN and a family history of early onset HTN or cerebrovascular accident at a young age (<40 years)
7. All hypertensive first-degree relatives of patients with PA .

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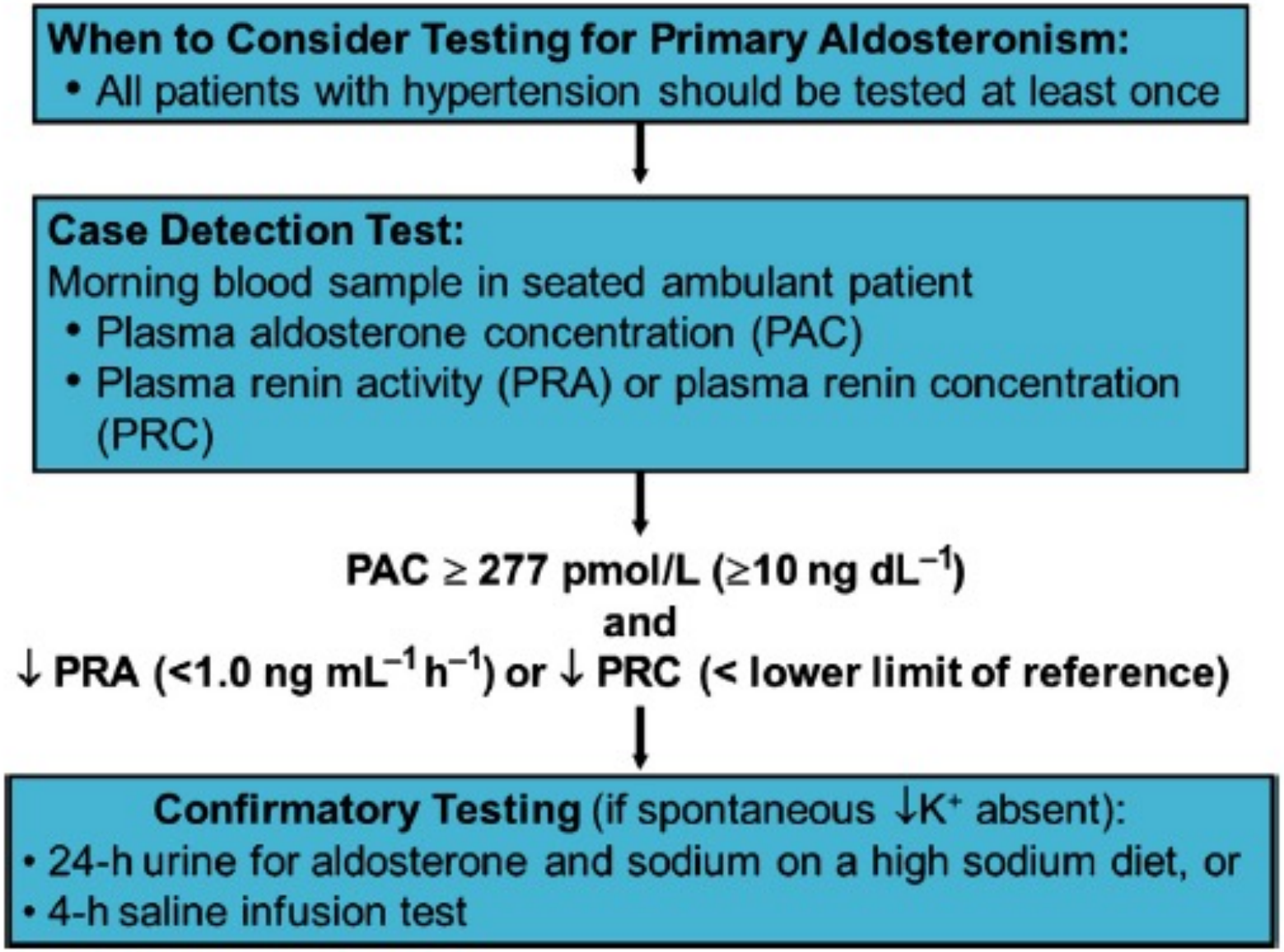

Williams textbook of endocrinology 14 th

Confirmatory

- Oral sodium loading test
  - lAdosterone secretory autonomy with aldosterone-suppression testing, which can be performed with orally administered sodium chloride and measurement of urinary
- Aldosterone excretion Intravenous saline infusion test
  - Intravenous sodium chloride loading and measurement of PAC

Restoring the serum potassium level before performing diagnostic studies ??

- Although **hypokalaemia reduces** the secretion of **aldosterone**, it rarely normalizes aldosterone secretion in patients with PA; these patients are hypokalaemic because of excess aldosterone secretion.
- Nevertheless, restoring the serum potassium level to normal before performing diagnostic studies is optimal (although not necessary in most cases).

Medications used to treat hypertension

- Can potentially cause **false-negative** testing results in patients with **mild PA**, there is no medication that causes false-positive results, as long as a cut-off level for aldosterone is used.

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PA

- **Calcium channel blockers** and **α1-adrenergic receptor blockers** do not affect the diagnostic accuracy in most cases.

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ACE & ARBs

- ACE inhibitors and ARBs have the potential to **elevate PRA** in patients with **mild PA**.
- Therefore, the finding of a PRA level ≥1.0 ng/ ml/ h or a PRC that is not suppressed in a patient taking an ACEI or ARB does not exclude the diagnosis of PA. (withdraw at least **2 weeks**)
- However, a **PRA level <1.0** ng /mL/ h or a **PRC** below the reference range in a patient taking an ACE inhibitor or ARB is **diagnostic** of low-renin hypertension and possible PA.

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MRAs (e.g. spironolactone and eplerenone)

- Resulting sequentially in sodium loss, a decrease in plasma volume and an **elevation in renin**.
- A should be **discontinued for 6 weeks** before re-testing
- However, if the patient is **hypokalaemic** despite treatment with an MRA, then the mineralocorticoid receptors are not fully blocked and PRA or PRC should be **suppressed** in such a patient with PA.

Table 3. Factors That May Lead to False-Positive or False-Negative ARR Results

Factor	Effect on Aldosterone Plasma Levels	Effect on Renin Levels	Effect on ARR
Medications[a]			
β-Adrenergic blockers	D	D D	U (FP)
Central agonists (eg, clonidine, α-methyldopa)	D	D D	U (FP)
NSAIDs	D	D D	U (FP)
K^+-wasting diuretics	R U	U U	D (FN)
K^+-sparing diuretics	U	U U	D (FN)
ACE inhibitors	D	U U	D (FN)
ARBs	D	U U	D (FN)
Ca^{2+} blockers (DHPs)	R D	U	D (FN)
Renin inhibitors	D	D U	U (FP) D (FN)
Potassium status			
Hypokalemia	D	R U	D (FN)
Potassium loading	U	R D	U
Dietary sodium			
Sodium restriction	U	U U	U (FN)
Sodium loading	D	D D	U (FP)
Advancing age	D	D D	U (FP)
Premenopausal women (vs males)[b]	R U	D	U (FP)
Other conditions			
Renal impairment	R	D	U (FP)
PHA-2	R	D	U (FP)
Pregnancy	U	U U	D (FN)
Renovascular HT	U	U U	D (FN)
Malignant HT	U	U U	D (FN)

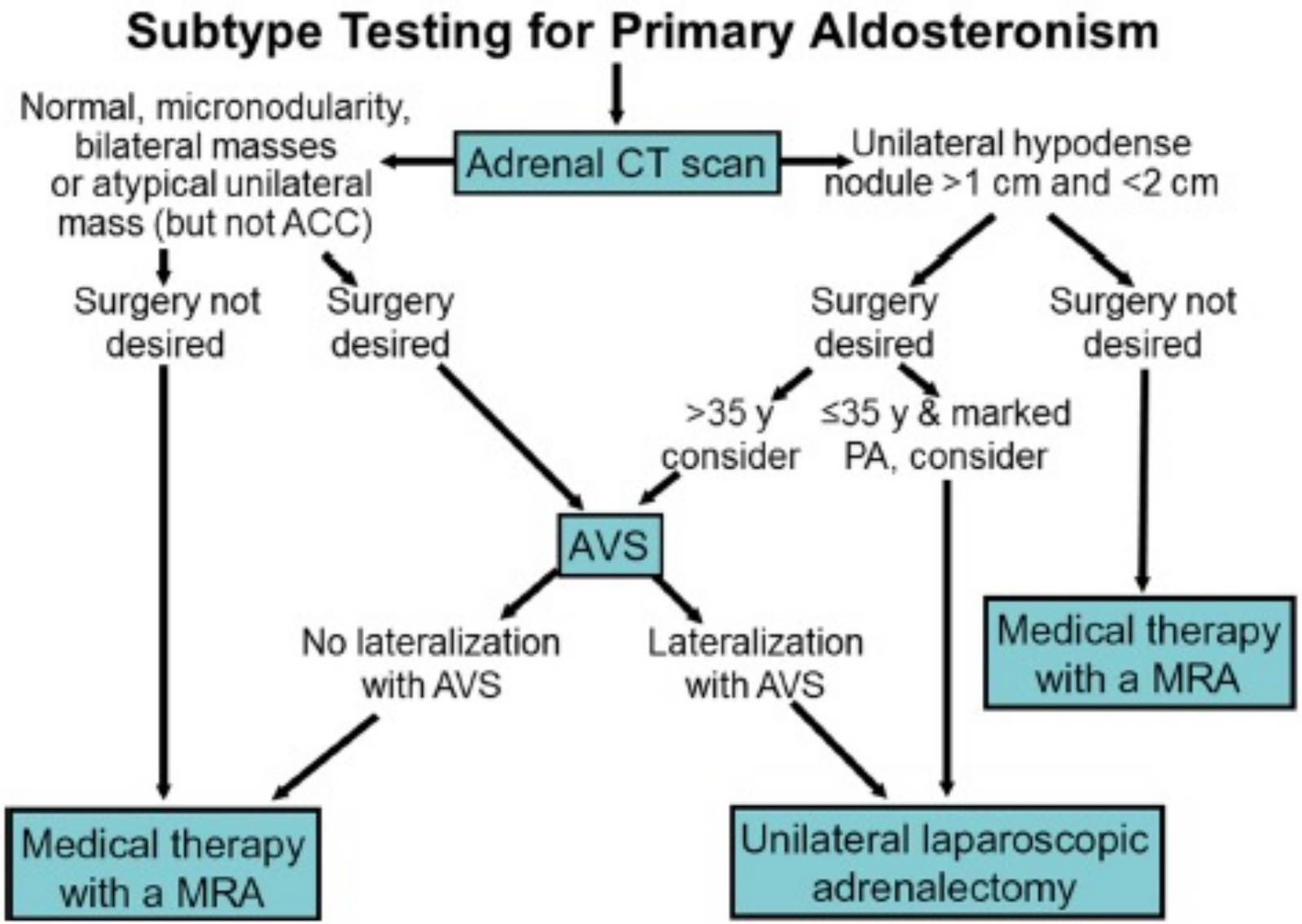
Guidelines on Primary Aldosteronism
J Clin Endocrinol Metab, May 2016, 101(5):1889–1916

Secondary hyperaldosteronism (e.g. renovascular hypertension)

- Hypertension and hypokalemia
- PAC/PRA ratio is <277 (with PAC measured in pmol /L and PRA in ng/ mL/ h;
- PAC/PRA ratio <10 if PAC is measured in ng/dL and PRA in ng/mL/ h).

PAC and PRA (or PRC) are suppressed

- Hypertension and hypokalaemia,
- An alternate source of agonism at the mineralocorticoid receptor should be considered (e.g. hypercortisolism, licorice use).


Subtype Testing for Primary Aldosteronism
Adrenal CT scan
Normal, micronodularity, bilateral masses or atypical unilateral mass (but not ACC)
Unilateral hypodense nodule >1 cm and <2 cm
Surgery not desired
Surgery desired
Surgery desired
Surgery not desired
>35 y consider
≤35 y & marked PA, consider
AVS
No lateralization with AVS
Lateralization with AVS
Medical therapy with a MRA
Medical therapy with a MRA
Unilateral laparoscopic adrenalectomy

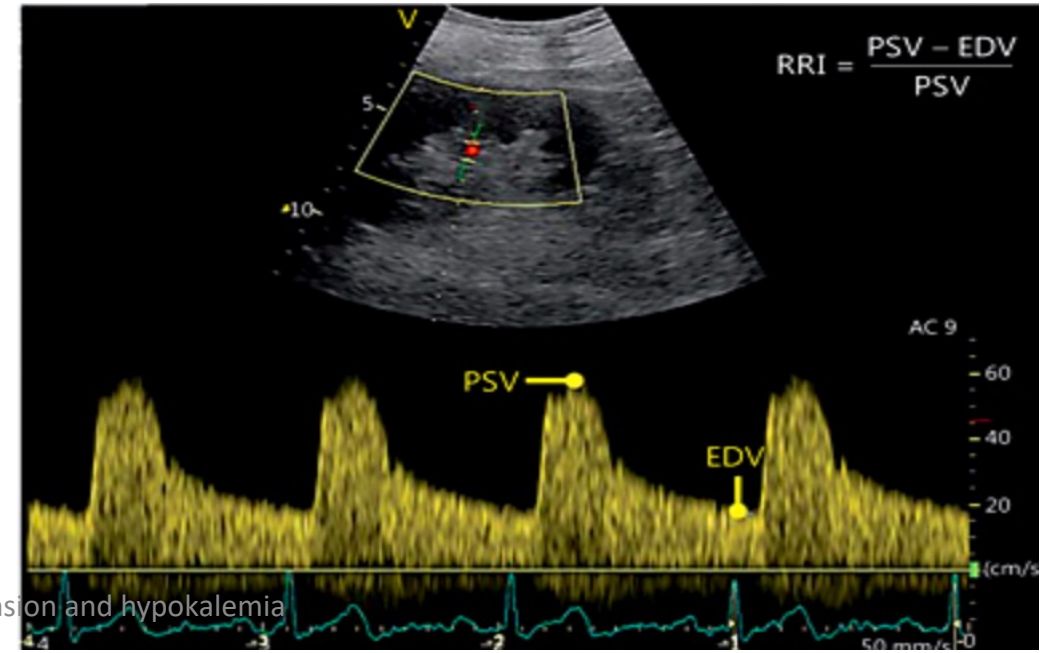
In admission of the patient:

- He was tachypneic, at sitting position in all times.
- Serum Cr= 1.7mg/dL. K= 4.2-4.5meq/L without any hypokalemic episodes.
- He received: carvedilol, amlodipin, hydralazin for HTN control.

- In this situation we consider his Doppler sonography of patient:
- **Decreased RI in left kidney 0.49 and in right kidney. 0.82: RAS in left kidney**

Arterial resistive index

- **RI = (PSV - EDV) / PSV**
- An RRI value 0.60±0.01 (mean±SD) is usually taken as normal with a value of 0.70 being considered the upper normal threshold.
- RRI >0.80 : reliable indicator of poor therapeutic response to revascularization treatment.



Factors influence intrarenal RI

- (i) the extent of stenosis
- (ii) the distensibility/stiffness of the vascular system
- (iii) non-renal factors
- (iv) the location of intrarenal Doppler measurement

Nephrol Dial Transplant (2007) 22: 692–696
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Advance Access publication 27 December 2006

Doppler sonography in renal artery stenosis—does the Resistive Index predict the success of intervention?

Bernd Krumme[1] and Markus Hollenbeck[2]

Extent of stenosis

- Significant narrowing of the vessel induces a reduction of the peak systolic flow velocity, including a loss of the so-called ‘early systolic peak’.
- While end diastolic velocity increases in stenoses, RI decreases

The distensibility/stiffness of the vascular system

- A higher RI is measured in vessels with low compliance than in those with excellent compliance.
- The stiffness of the supplying arteries, e.g. the aorta or the iliac artery, have a significant impact on the RI derived in renal allografts.

Non-renal factors

Tachycardia: **low values of RI**, simply because the systolic peak begins earlier than in the case of normal heart rate.	Arrhythmias, **RI** does not give any information on renal perfusion. Especially in patients with **AF**, RI should not be used for the diagnosis of RAS.
Bradycardia (heart rate <60 beats/min) induces **high values of RI** due to later beginning of the next systolic peak with less endiastolic velocity.	Valvular heart disease: **AI** induce **high intrarenal RI.**
	Significant **AS**, **low RI** is registered in the kidneys

The location of intrarenal Doppler measurement

- Intrarenal RI decreases from the hilum of the kidney towards the renal cortex.
- If intrarenal RI is calculated from the flow pattern of the hilar artery, higher values of RI are expected

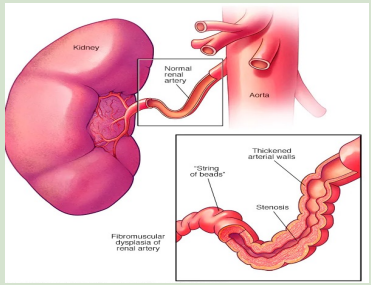
Reasons for elevated values	Reasons for elevated values in a transplant kidney	Reasons for decreased values
Medical renal disease	Acute tubular necrosis (ATN)	**Renal artery stenosis**
Ureteric obstruction	Acute or chronic transplant rejection	
extreme hypotension	Renal vein thrombosis	
Perinephric fluid collection	Drug toxicity	
Abdominal compartment syndrome	Ureteric obstruction	
very young children	Perinephric fluid collection	

Renovascular hypertension definition

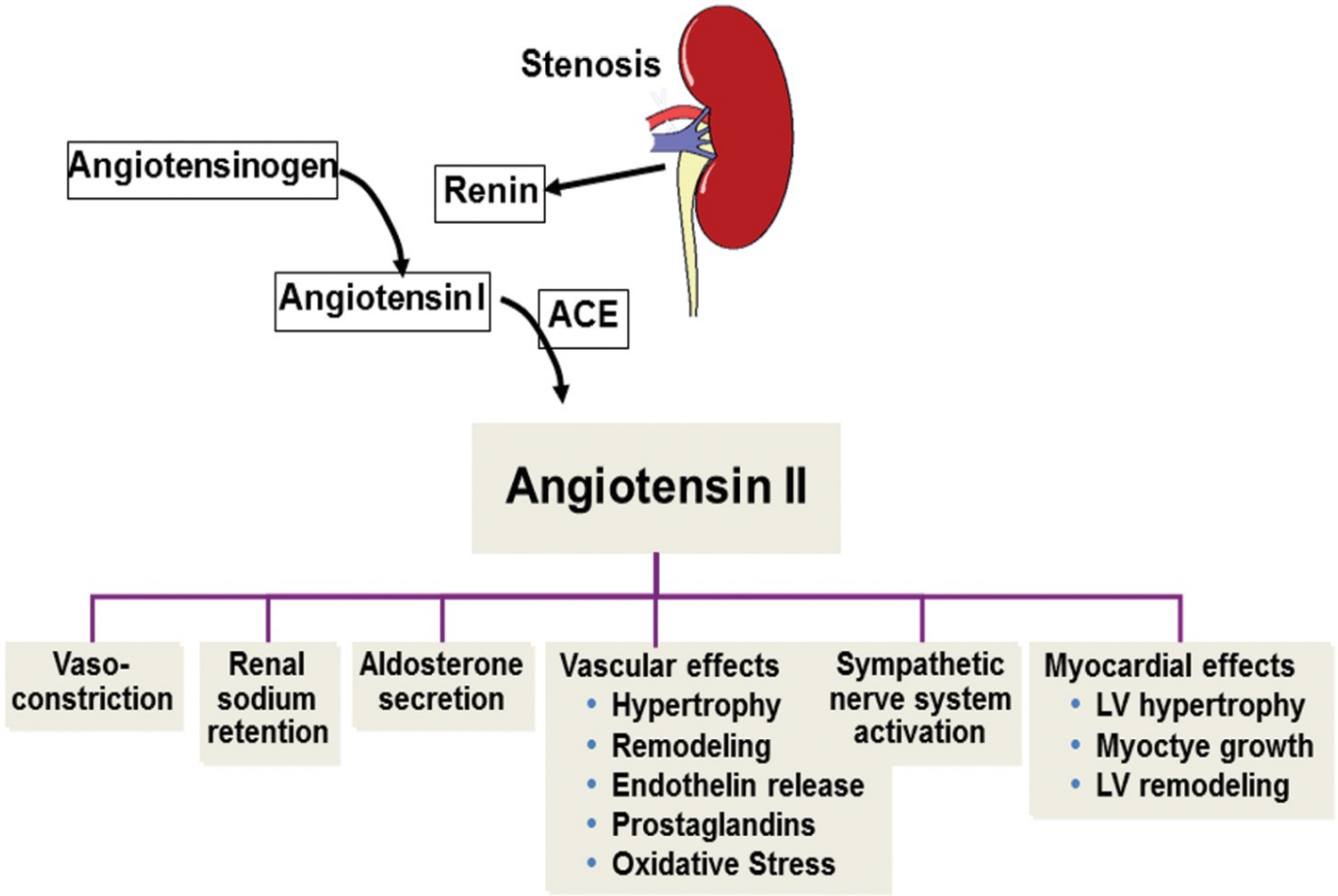
- Systemic hypertension resulting from renal arterial compromise, often due to occlusive lesions of the main renal arteries.
  - Prevalence:
- in the general population : 1% - 2% of all cases of HTN
- In >65 years of age: up to 6.8%
- In cases of secondary HTN: 5.8%.

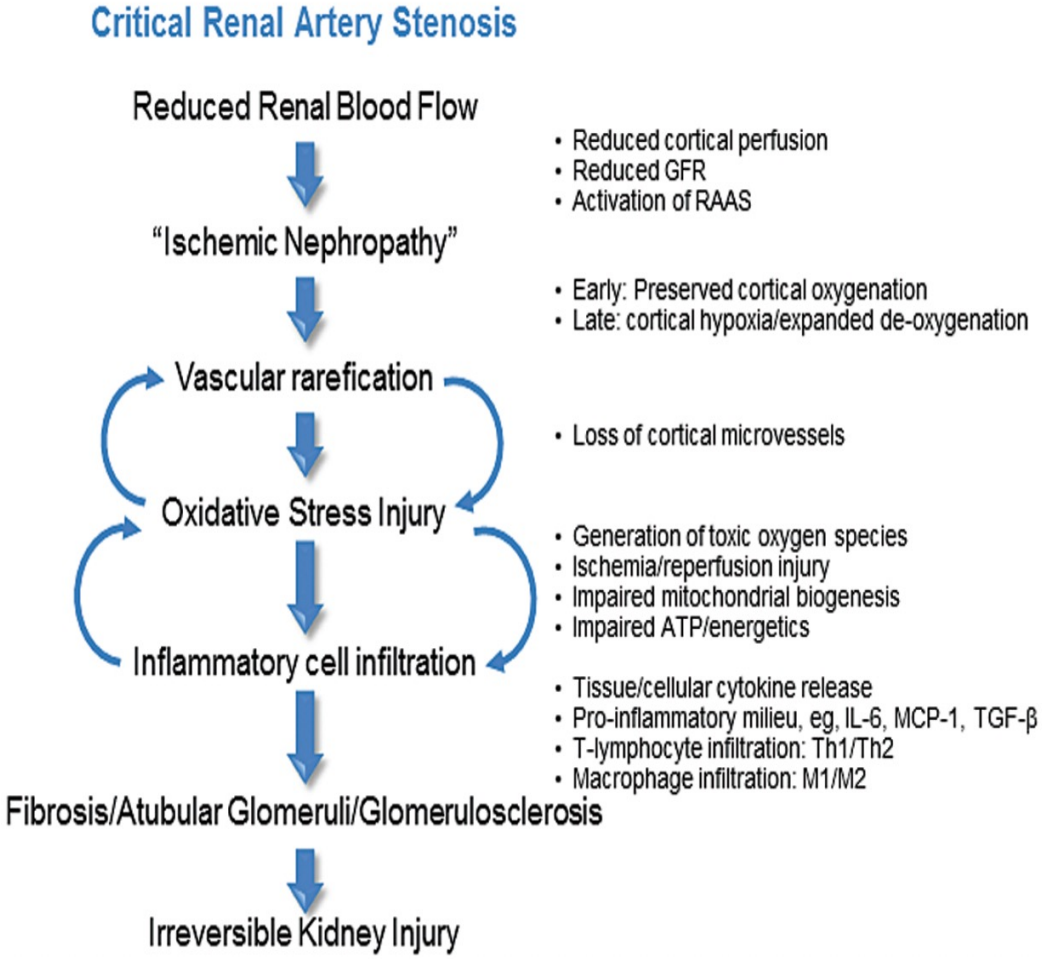
American Journal of Hypertension 31(2) February 2018

Causes of RVH

Atherosclerotic renal artery stenosis	**Extrinsic fibrous band**
Fibromuscular disease: • Medial fibroplasia • Perimedial fibroplasia • Intimal fibroplasia • Medial hyperplasia 	**Renal trauma** • Arterial dissection • Segmental renal infarction • Page kidney (perirenal fibrosis)
Aortic dissection occluding the renal artery	**Miscellaneous:** Hypercoagulable state with renal infarction (e.g., Lupus anticoagulate) Autoimmune diseases (e.g., Takayasu's arteritis, Polyarteritis nodosa) Malignancy encircling the renal artery (e.g., Renal cell carcinoma, pheochromocytoma)
Arterial embolus	
Aortic endograft	

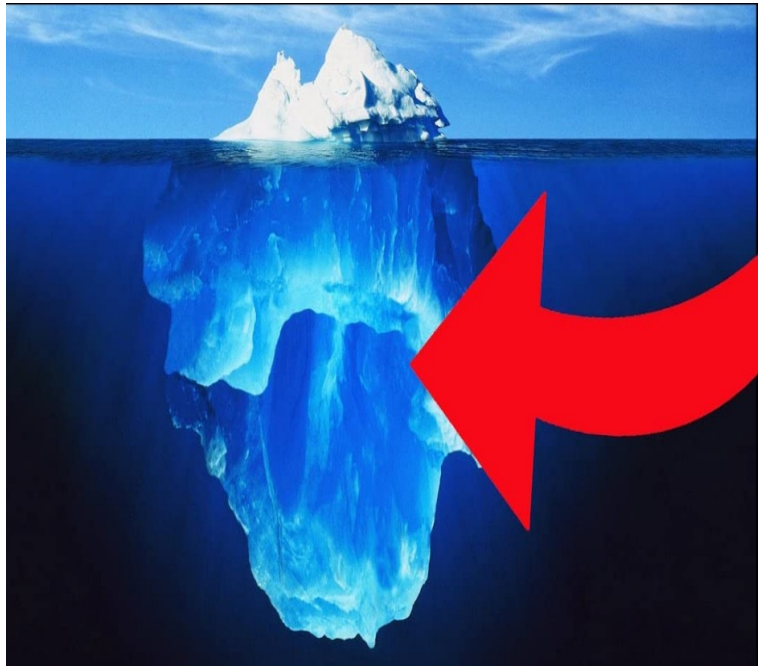
PATHOPHYSIOLOGY OF RVH

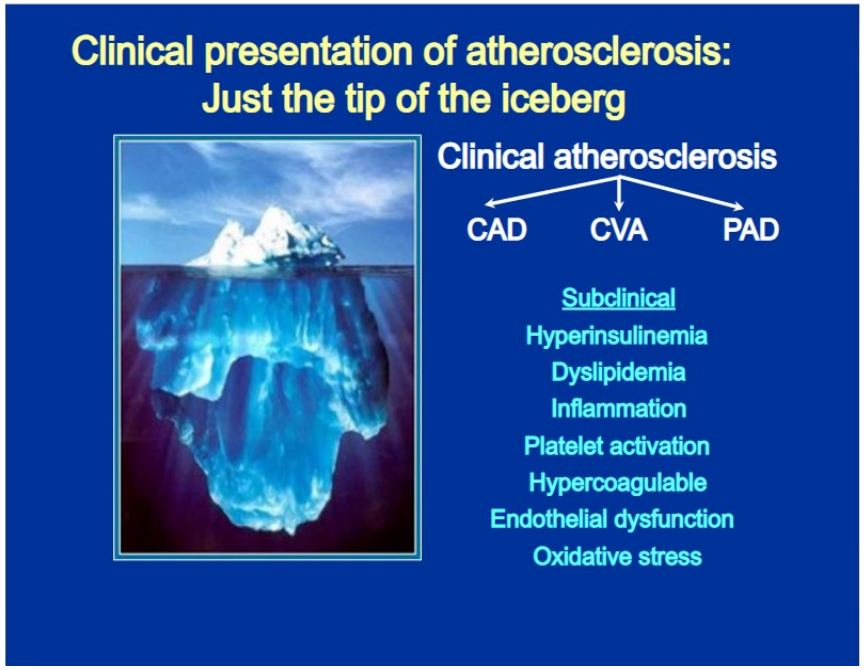




Mechanisms of atherosclerosis formation:

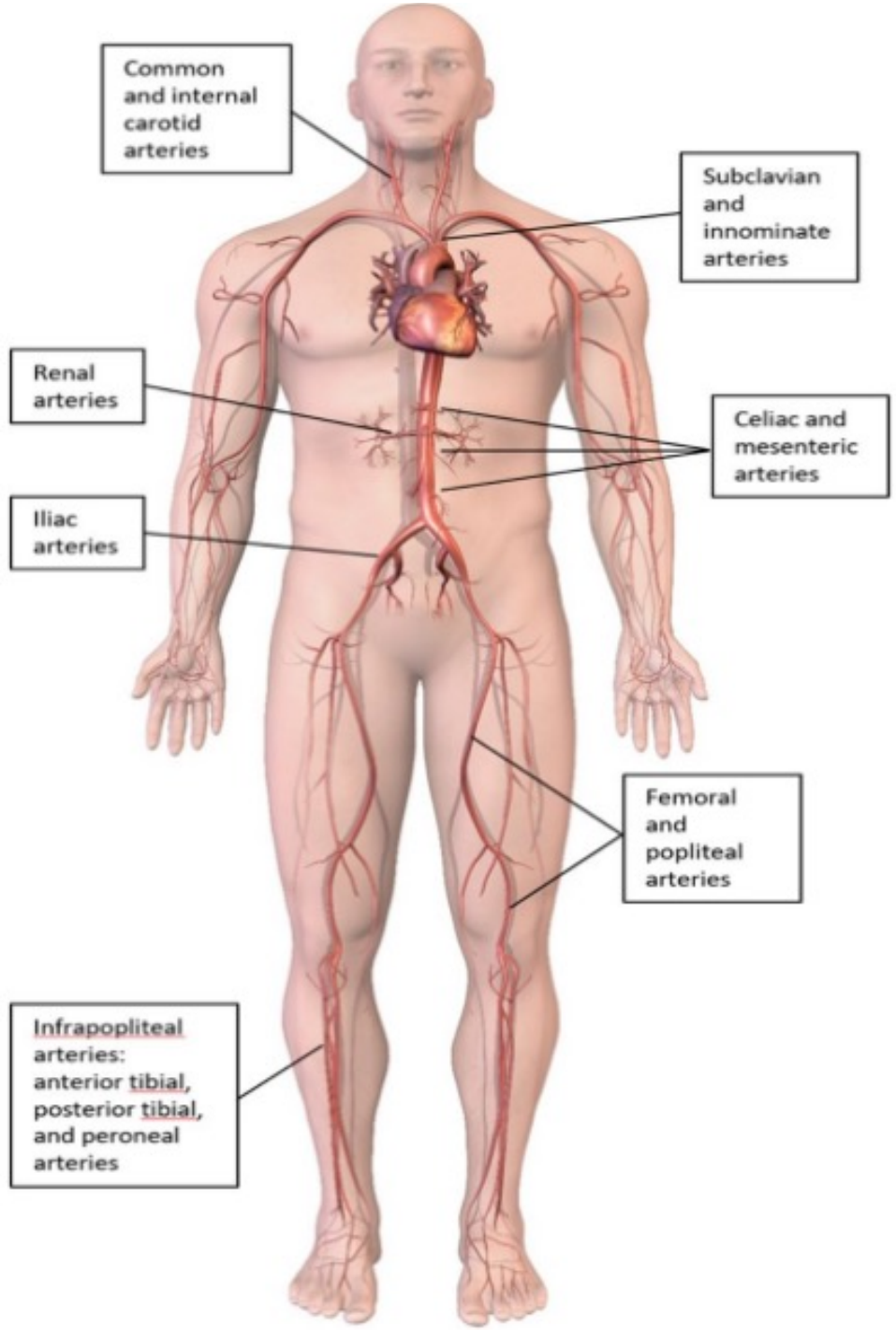
- **Inflammation of vascular smooth muscle cells(SMC)** leads to an **increase in calcium deposits**, which turn into **atherosclerotic plaques** in the inner wall of the arteries.





Mechanisms of….

- **Atherosclerosis causes clinical disease** through *luminal narrowing* **or** by *precipitating thrombi* that **obstruct blood flow to the heart (coronary heart disease), brain (ischemic stroke)**, or **peripheral arteries(peripheral vascular disease)**.



Peripheral Vascular Disease(PVD):

- **Renovascular disease** is an important, **potentially correctable** cause of **secondary** and **treatment-resistant hypertension**.
- **Most cases** are related either to **atherosclerotic renal artery stenosis (ARAS)** or **fibromuscular dysplasias (FMD)**, but a variety of other causes including **arterial dissection**, **stent occlusion**, and **embolic disease,……**can produce the same syndrome.
- **Atherosclerotic disease commonly develops at the origin of the renal artery**, sometimes linked to areas of flow turbulence.
- These lesions **commonly develop beyond the fifth decade** and are **linked to atherosclerotic risk factors**, including **tobacco use**, **dyslipidemias**, **diabetes**, and **hypertension**.

Table 5 Clinical situations raising suspicion for renal artery disease

Onset of hypertension before the age of 30 years
Onset of severe hypertension after the age of 55 years, when associated with CKD or heart failure
Hypertension and abdominal bruit
Rapid and persistent worsening of previously controlled hypertension
Resistant hypertension (i.e. other secondary form unlikely and target not achieved despite four drug classes including a diuretic and a mineralocorticoid-receptor antagonist in appropriate doses)
Hypertensive crisis (i.e. acute renal failure, acute heart failure, hypertensive encephalopathy, or grade 3–4 retinopathy)
New azotaemia or worsening of renal function after treatment with RAAS blockers
Unexplained atrophic kidney or discrepancy in kidney size, or unexplained renal failure
Flash pulmonary oedema

CKD = chronic kidney disease; RAAS = renin-angiotensin-aldosterone system.

➢ Imaging:

Digital subtraction angiography is the **gold standard** for characterizing these lesions, but is an invasive and expensive procedure, usually combined with endovascular procedures including dilation and stent placement.

Recommendations for diagnostic strategies for renal artery disease

Recommendations	Class[a]	Level[b]
DUS (as first-line), CTA[c] and MRA[d] are recommended imaging modalities to establish a diagnosis of RAD.[204,212]	I	B
DSA may be considered to confirm a diagnosis of RAD when clinical suspicion is high and the results of non-invasive examinations are inconclusive.[212,215]	IIb	C
Renal scintigraphy, plasma renin measurements before and after ACEI provocation and vein renin measurements are not recommended for screening of atherosclerotic RAD.[204]	III	C

ACEI = angiotensin-converting enzyme inhibitor; CTA = computed tomography angiography; DSA = digital subtraction angiography; DUS = duplex ultrasound; eGFR = estimated glomerular filtration rate; MRA = magnetic resonance angiography; RAD = renal artery disease.

[a]Class of recommendation.

[b]Level of evidence.

[c]When eGFR is ≥ 60 mL/min.

[d]When eGFR is ≥ 30 mL/min.

Treatment of RVHT:

- As with all forms of hypertension, the **overall goal** of managing RVH is to reduce the morbidity and mortality associated with elevated BP.
- A **second goal** is to protect the circulation and function of the kidneys.
- As noted above, many of the pressor pathways, including activation of the RAAS, are activated at reductions of post-stenotic pressures and blood flows that are well tolerated by the kidney itself.
- **Antihypertensive drug therapy** often can be implemented with effective BP reduction and minimal adverse effects upon the post-stenotic kidney(s).

Treatment of …

- Because **atherosclerotic disease is a systemic disorder**, RVH patients should **routinely be treated with statin therapy** and **lifestyle measures** including **withholding tobacco products**.
- **Statins** have been shown experimentally to modify the microvascular milieu with the **kidney** and limit fibrosis and inflammatory damage.
- **Statin-treated patients** subjected to nephrectomy of completely occluded kidneys demonstrate **reduced activation of transforming growth factor-beta** and interstitial fibrosis **as compared to those not treated with statins**.
- Hence, **statins routinely should be part of medical therapy of patients with ARAS**.

ESC European Society of Cardiology
European Heart Journal (2018) 39, 3021–3104
doi:10.1093/eurheartj/ehy339

2018 ESC/ESH Guidelines for the management of arterial hypertension

The Task Force for the management of arterial hypertension of the European Society of Cardiology (ESC) and the European Society of Hypertension (ESH)

Authors/Task Force Members: Bryan Williams* (ESC Chairperson) (UK), Giuseppe Mancia* (ESH Chairperson) (Italy), Wilko Spiering (The Netherlands), Enrico Agabiti Rosei (Italy), Michel Azizi (France), Michel Burnier (Switzerland), Denis L. Clement (Belgium), Antonio Coca (Spain), Giovanni de Simone (Italy), Anna Dominiczak (UK), Thomas Kahan (Sweden), Felix Mahfoud (Germany), Josep Redon (Spain), Luis Ruilope (Spain), Alberto Zanchetti† (Italy), Mary Kerins

Recommendations for treatment strategies for renal artery disease

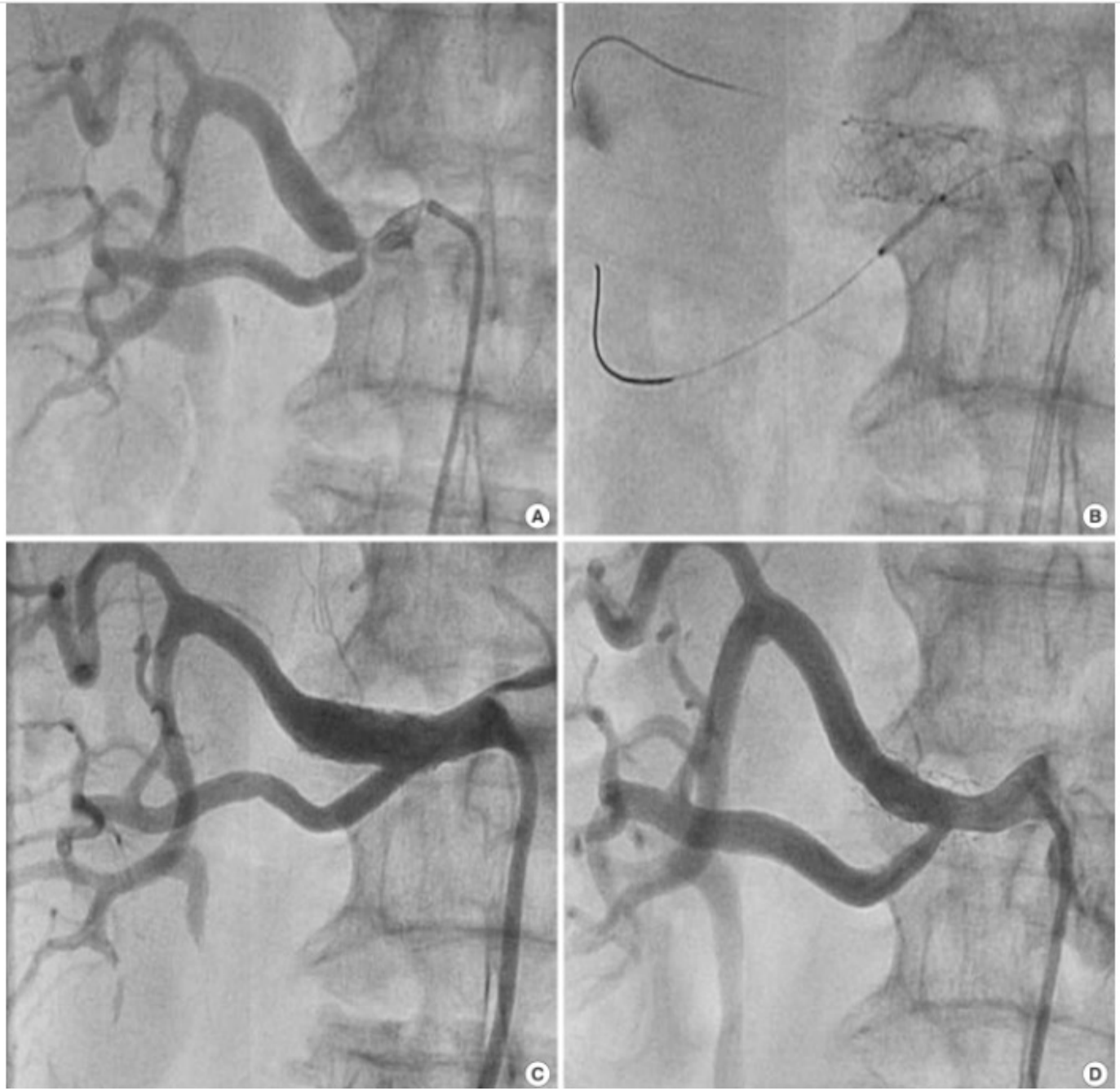
Recommendations	Class[a]	Level[b]
Medical therapy		
ACEIs/ARBs are recommended for treatment of hypertension associated with unilateral RAS.[219–222,240]	I	B
Calcium channel blockers, beta-blockers and diuretics are recommended for treatment of hypertension associated with renal artery disease.	I	C
ACEIs/ARBs may be considered in bilateral severe RAS and in the case of stenosis in a single functioning kidney, if well-tolerated and under close monitoring.[219,221]	IIb	B

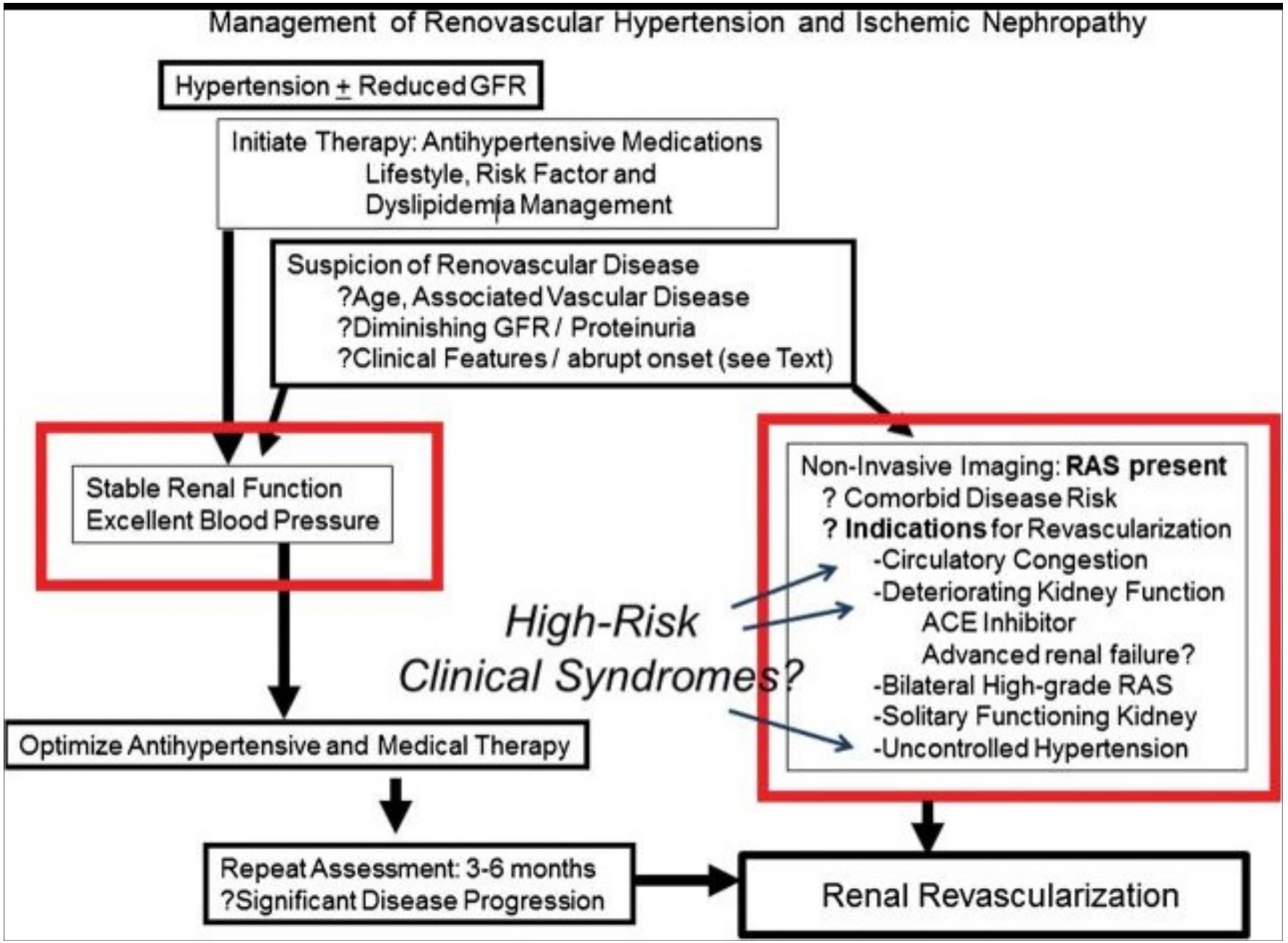
Renal revascularization in the Management of RVHT:

- **Renal-artery stenting did not confer a significant benefit with respect to the prevention of clinical events** when added to comprehensive, multifactorial medical therapy in people with atherosclerotic renal-artery stenosis and hypertension or chronic kidney disease.
- **Prospective randomized trials** reported between **1998 and 2015 fail to identify additional benefits from endovascular stent revascularization for ARAS** when added to medical therapy.

✓**When should revascularization be applied for RVH?**

- **Numerous observational series report improved BP control and occasional dramatic recovery of kidney function** in patients with **both FMD** and **ARAS** as the basis for RVH.


A
B
C
D


Management of Renovascular Hypertension and Ischemic Nephropathy
Hypertension ± Reduced GFR
Initiate Therapy: Antihypertensive Medications
Lifestyle, Risk Factor and
Dyslipidemia Management
Suspicion of Renovascular Disease
?Age, Associated Vascular Disease
?Diminishing GFR / Proteinuria
?Clinical Features / abrupt onset (see Text)
Stable Renal Function
Excellent Blood Pressure
Non-Invasive Imaging: RAS present
? Comorbid Disease Risk
? Indications for Revascularization
-Circulatory Congestion
-Deteriorating Kidney Function
ACE Inhibitor
Advanced renal failure?
-Bilateral High-grade RAS
-Solitary Functioning Kidney
-Uncontrolled Hypertension
High-Risk
Clinical Syndromes?
Optimize Antihypertensive and Medical Therapy
Repeat Assessment: 3-6 months
?Significant Disease Progression
Renal Revascularization

Am J Hypertension recommendation:

- **If 1)adequate BP cannot be readily achieved**
- and/or **2) conditions develop defining "high-risk" clinical syndromes** with **refractory hypertension**, **progressive renal dysfunction** and/or **episodes of circulatory congestion**,
- **We strongly recommend moving forward with further characterization and restoration of the renovascular supply**.

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Revascularization		
Routine revascularization is not recommended in RAS secondary to atherosclerosis.[229,231,232]	III	A
In cases of hypertension and/or signs of renal impairment related to renal arterial fibromuscular dysplasia, balloon angioplasty with bailout stenting should be considered.[234–236]	IIa	B
Balloon angioplasty, with or without stenting, may be considered in selected patients with RAS and unexplained recurrent congestive heart failure or sudden pulmonary oedema.[229,237,238]	IIb	C
In the case of an indication for revascularization, surgical revascularization should be considered for patients with complex anatomy of the renal arteries, after a failed endovascular procedure or during open aortic surgery.[241–243]	IIa	B

CANADA Hypertension Guideline recommendation:

❑**Treatment of hypertension in association with renovascular disease:**

- **1)** Patients with **hypertension attributable to atherosclerotic renal artery stenosis** should be **primarily medically managed** because renal angioplasty and stenting offers no benefit over optimal medical therapy alone (Grade B).
- **2) Renal artery angioplasty and stenting for atherosclerotic hemodynamically significant renal artery stenosis** could be considered for patients **with any of the following** (Grade D; revised recommendation):

  I) Uncontrolled hypertension resistant to maximally tolerated pharmacotherapy,

  II) Progressive renal function loss,

  III) Acute pulmonary edema

CANADA Hypertension Guideline…

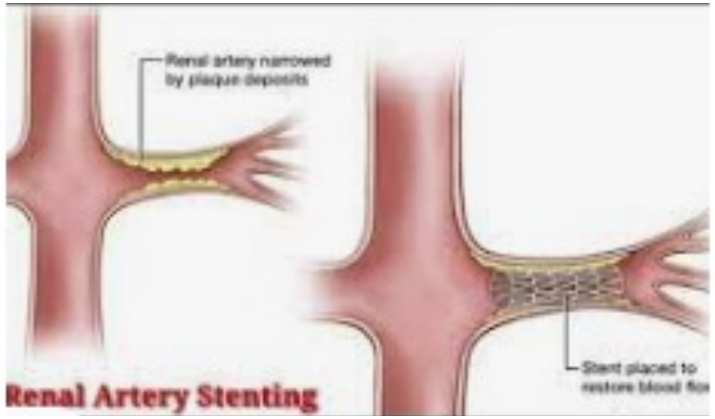
- **3) Renal artery angioplasty without stenting is recommended for treatment of FMD-related renal artery stenosis.**

➢ **Stenting is not recommended unless needed** because of a **periprocedural dissection**.

➢ **Surgical revascularization** should be considered in case of **complex lesions less amendable to angioplasty**, **stenosis associated with complex aneurysm**, and **restenosis despite 2 unsuccessful attempts of angioplasty** (Grade D).

Novel Insights 2020:

- **Today** **revascularization** is **only recommended** for patients with
  1) **progressive worsening of renal function**,
  2) **recurrent 'flash pulmonary edema'** and
  3) **rapid increase in antihypertensive requirement** in patients with previously well-controlled hypertension.



Recurrent disease and follow-up after Stenting:

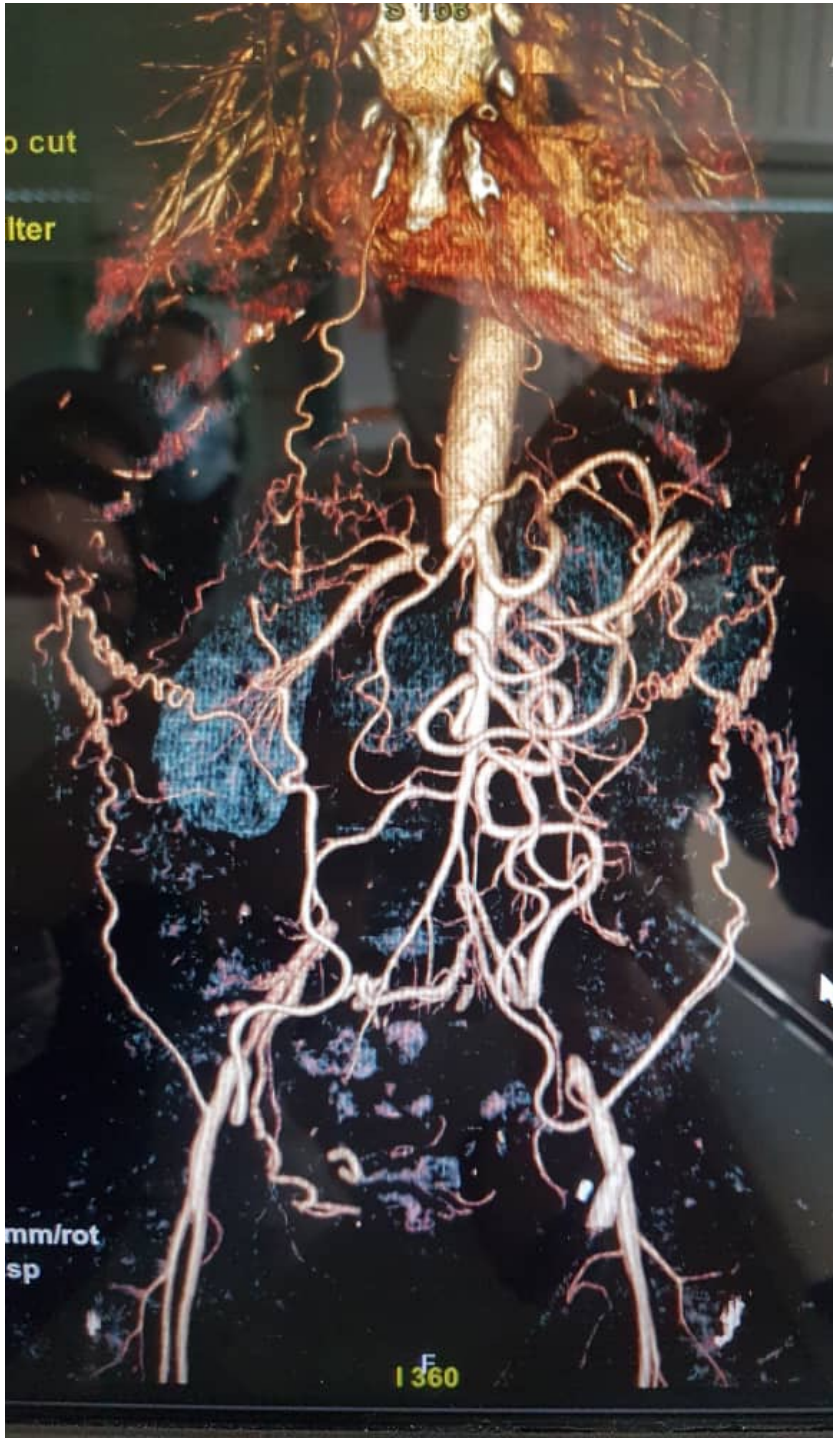
- Despite restoring main vessel patency, **restenosis** can develop in up to **14–18% of subjects followed for a year**.
- Recommendations for **follow-up include surveillance ultrasound** as well as BP and **renal function**.
- Some institutions **favor antiplatelet agents such as clopidogrel for several months** after stenting, although data are limited to support this.

Peripheral arterial disease (PAD):

- One subset of PAD is **aortoiliac occlusive disease (AIOD)**. AIOD can occur anywhere from the distal aorta to the common femoral arteries and is therefore called an **"inflow lesion."**
- More commonly, distal aortic occlusive disease extends into the common iliac arteries.
- Leriche Syndrome presents with a **triad of claudication, impotence, and absence of femoral pulses**.

Peripheral arterial disease (PAD):

- **Risk factors for development of AIOD** include nonwhite ethnicity, smoking, diabetes mellitus, dyslipidemia, hypertension, age, male gender, C-reactive protein elevation, hyperhomocystinemia, hyperviscosity/hypercoagulability, and chronic renal insufficiency.
- **Optimization of modifiable risk factors** is an important component of successful management of patients with AIOD.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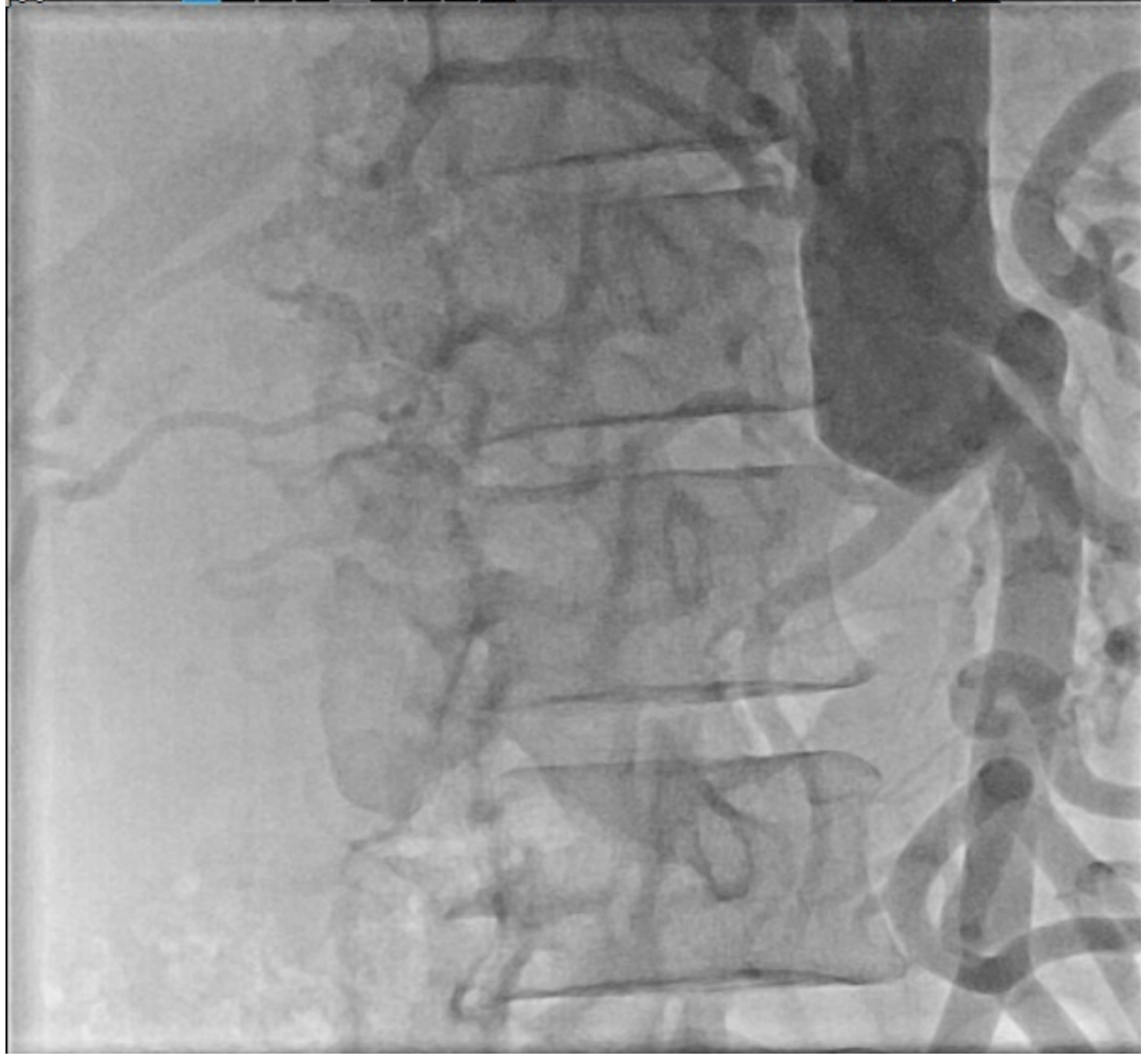

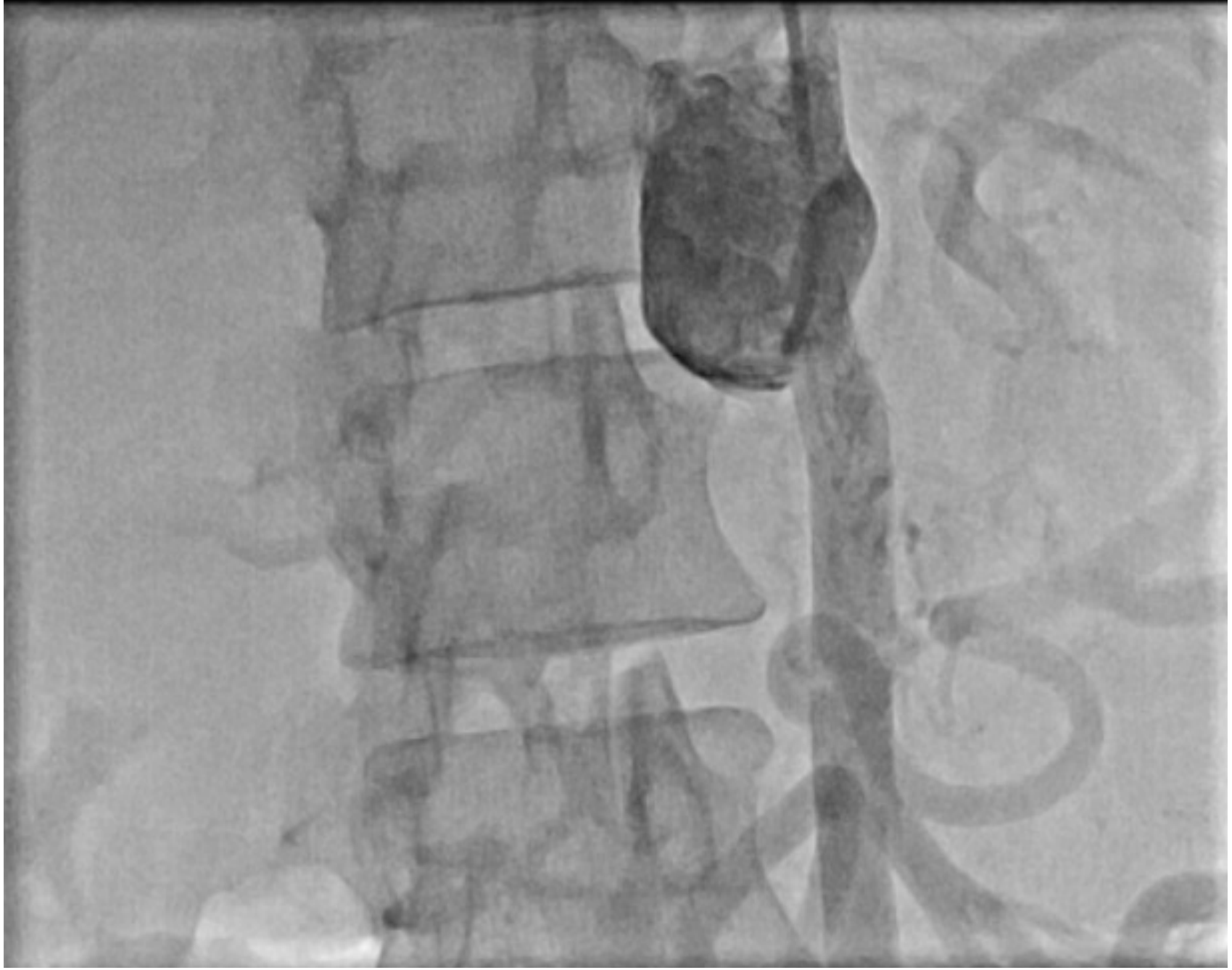

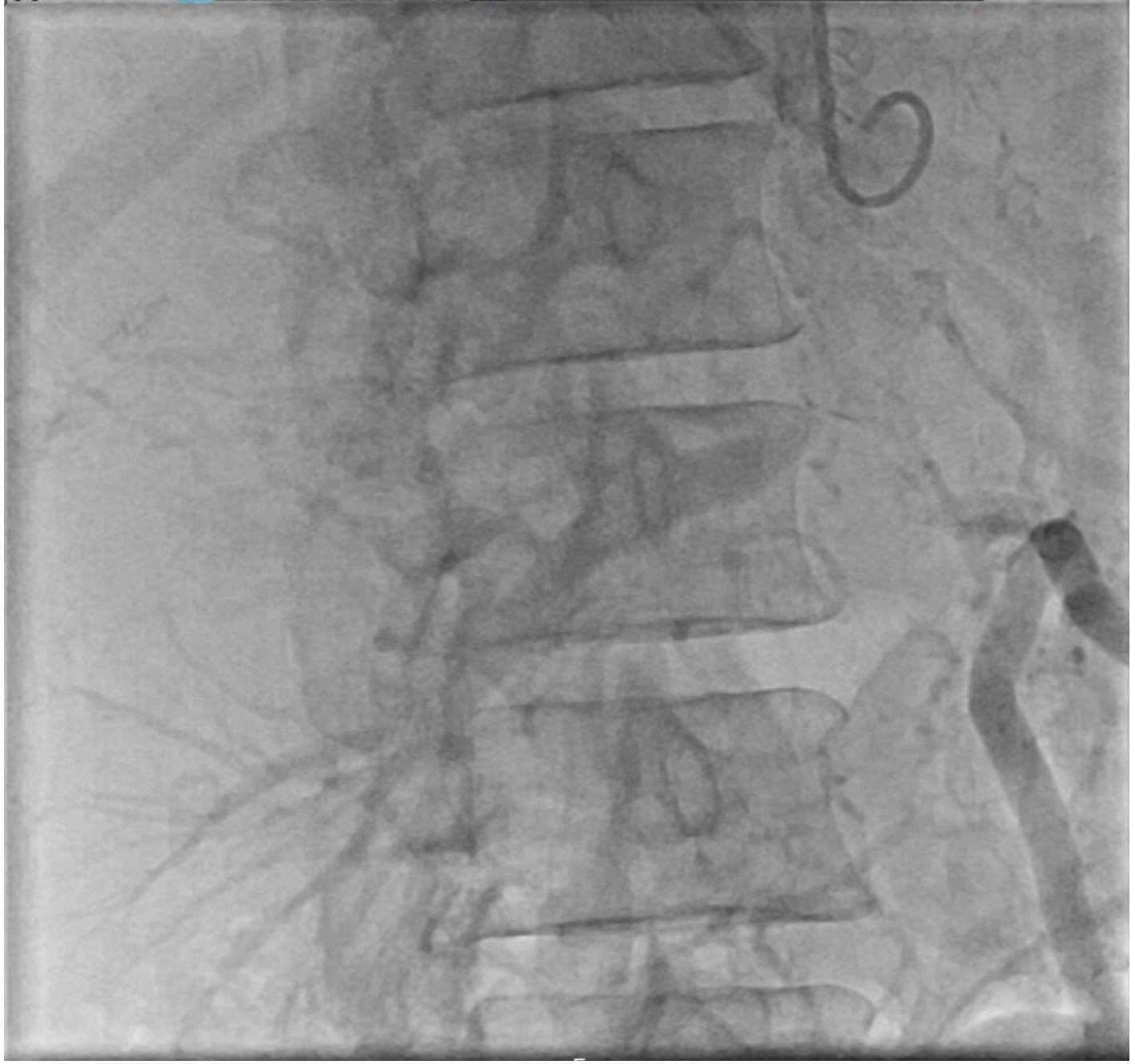

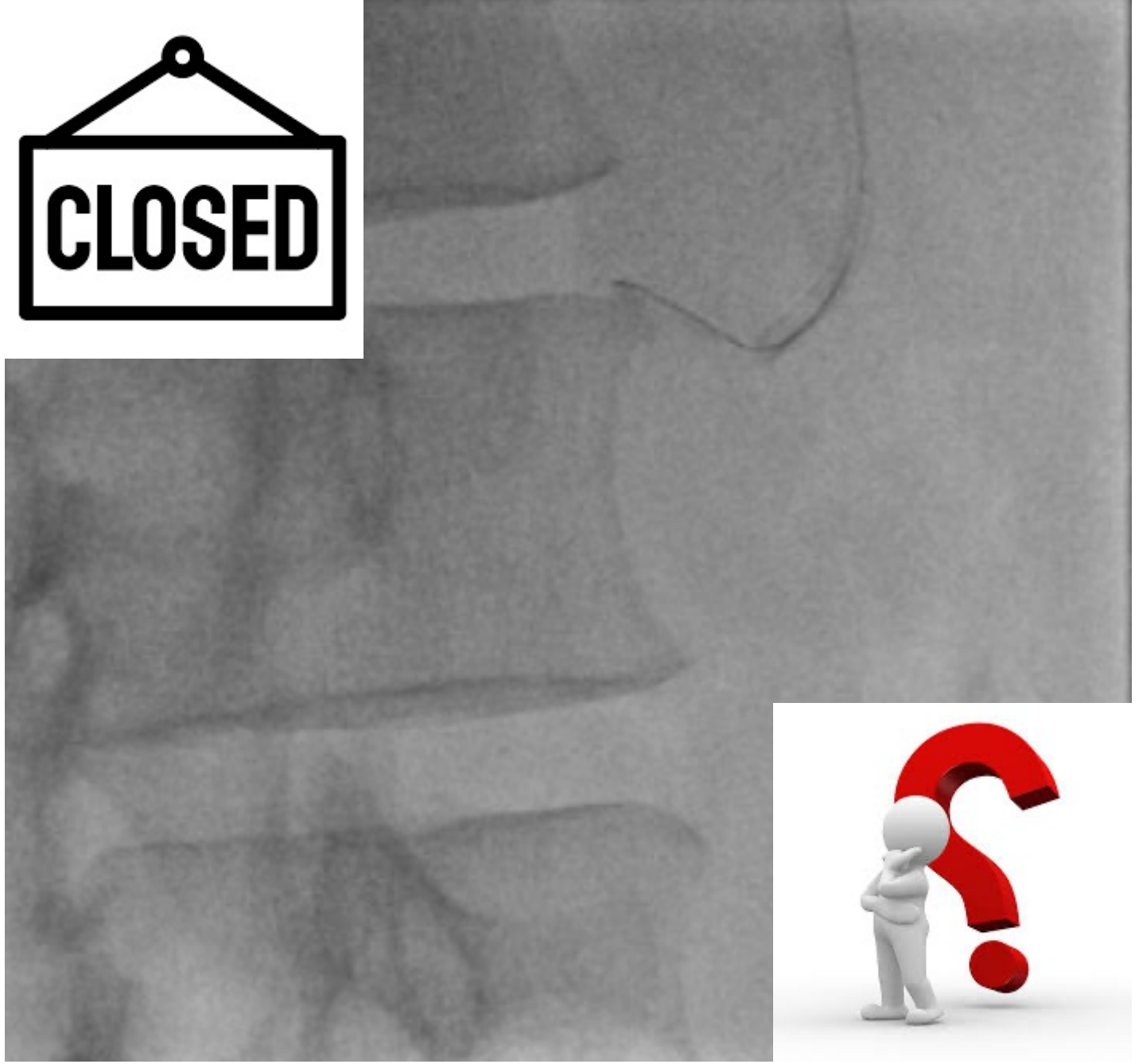
CLOSED

Conclusion

- Clinicians should perform case detection testing for PA at least once in **all patients** with hypertension.
- Serum potassium status is not a reliable guide for screening for PA.
- RRI >0.80 : reliable indicator of poor therapeutic response to revascularization treatment
- High or low RI could indicate RAS.
- Statins routinely should be part of medical therapy of patients with ARAS

Conclusion

- **Revascularization** is **only recommended** for patients with
  1)**progressive worsening of renal function**,
  2)**recurrent 'flash pulmonary edema'** and
  3)**rapid increase in antihypertensive requirement** in patients with previously well-controlled hypertension

Thanks For Your Attention

به امید روزها و سالهای پر بارش..