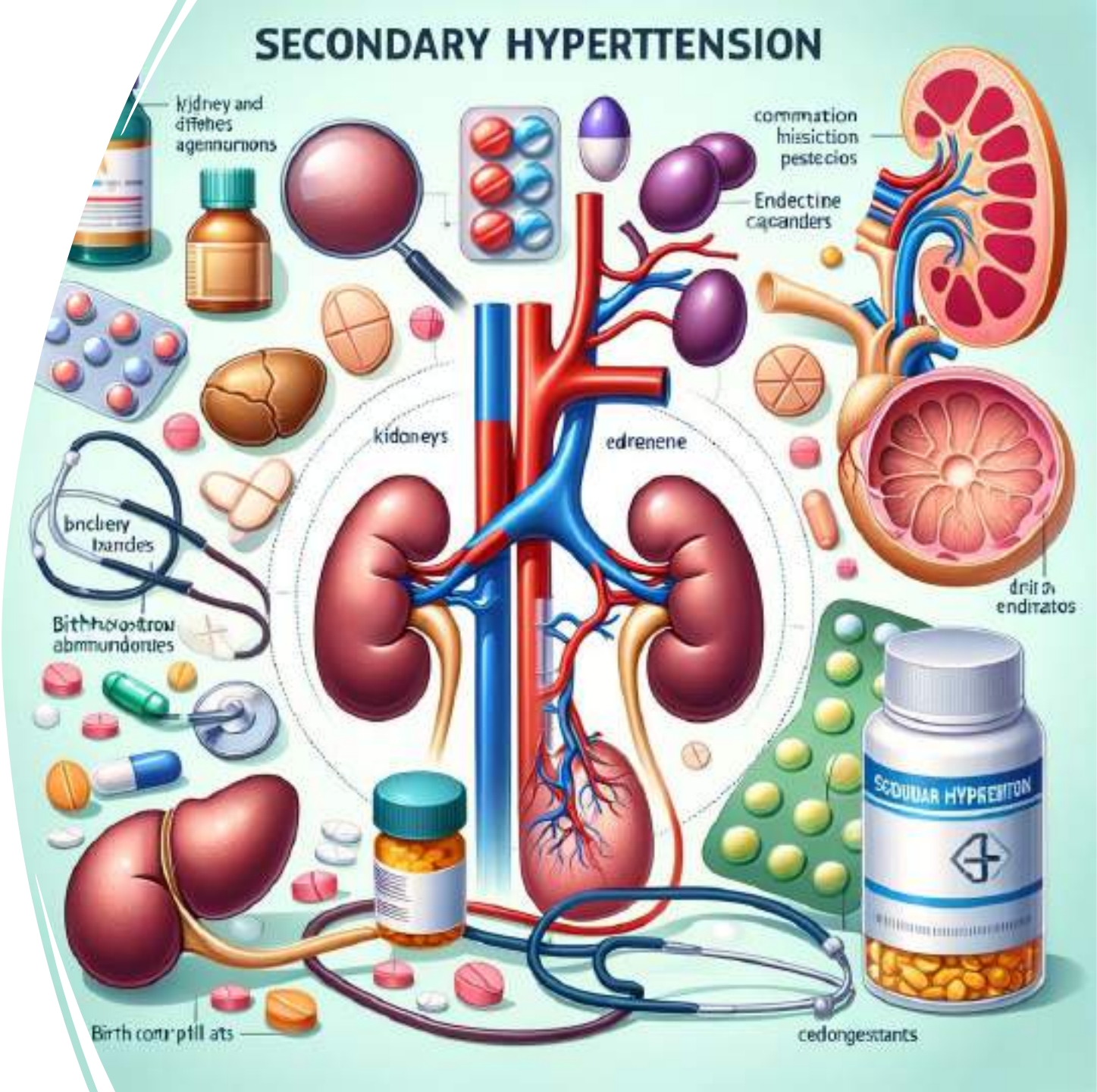
Azərbaycan
Kardiologiya
Cəmiyyəti

Secondary Hypertension

Ulvi Mirzoyev




30%
HYPERTENSION

Secondary Hypertension

- Essential hypertension (hypertension without an identifiable cause) is found as the main reason for hypertension,
- 10% of patients with hypertension are found to have secondary hypertension.
- Secondary hypertension is defined as elevated blood pressure (BP), secondary to an identifiable cause.
- Since its prevalence is relatively low, performing routine evaluations in every case of hypertension is not cost-effective and is also time-consuming

Patient characteristics that should raise the suspicion of secondary hypertension

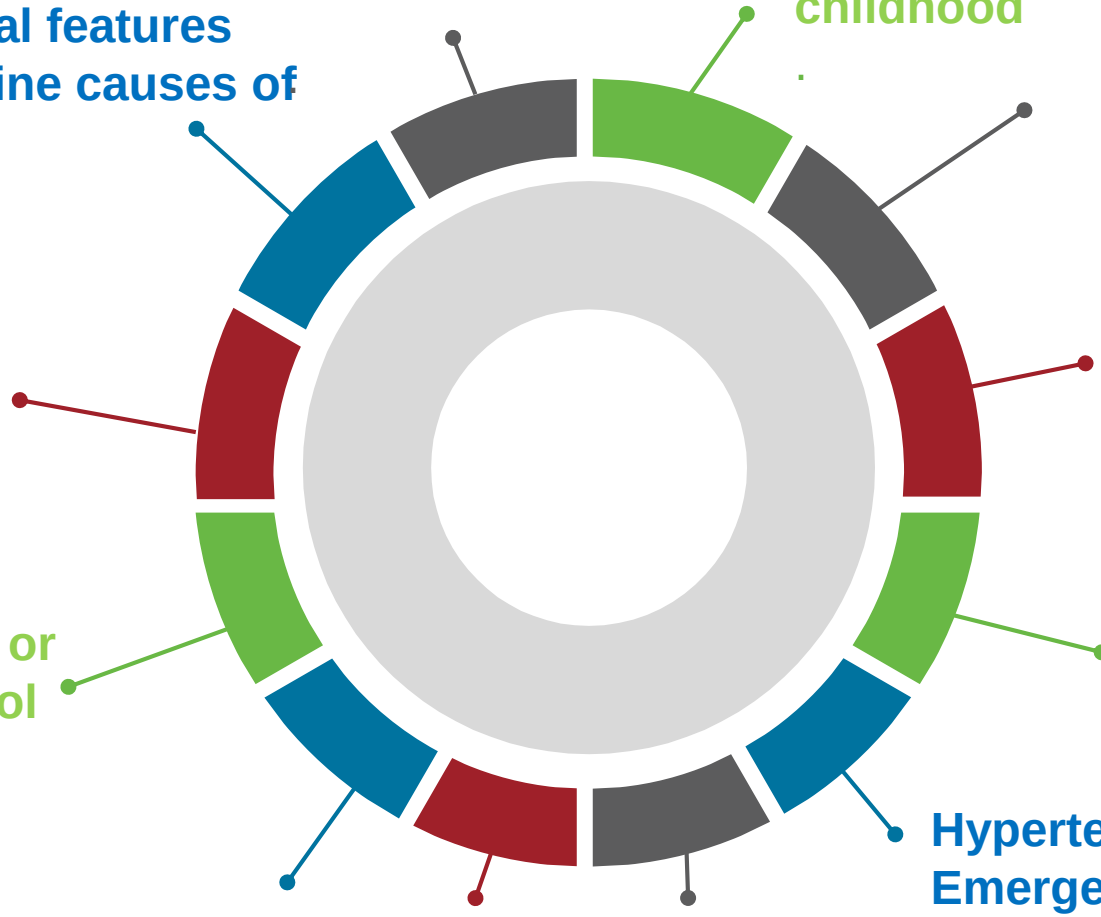
Clinical features suggestive of atherosclerotic renovascular disease or fibromuscular dysplasia

Younger patients (<40 years) with grade 2 or 3 hypertension or hypertension of any grade in childhood

Clinical or biochemical features suggestive of endocrine causes of hypertension

Sudden onset of HT in people previously documented normotension

Severe and/or extensive HMOD, particularly if disproportionate for the duration and severity of the BP elevation



Acute worsening of BP control in patients with previously well controlled by treatment

Severe hypertension in pregnancy (>160/110 mmHg) or acute worsening of BP control in pregnant women with preexisting hypertension

True resistant hypertension

Hypertensive Emergency

Clinical features suggestive of obstructive sleep apnea

Severe (grade 3) or malignant hypertension

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Causes of secondary hypertension

Endocrine

- Primary aldosteronism (PA)
- Cushing's syndrome (CS)
- Pheochromocytoma / paraganglioma (PPGL)
- Primary hyperparathyroidism (PHPT)
- Hypothyroidism
- Thyrotoxicosis
- Acromegaly
- Apparent Mineralocorticoid Excess (congenital)

Renal

- Renal parenchymal disease
- Renin-producing tumor
- Primary sodium retention (Liddle's syndrome)
- Gordon' syndrome (hyperkalemia with metabolic acidosis, normal renal function, low or low-normal plasma renin activity, and normal or elevated plasma aldosterone concentration)

Obstructive sleep apnea

Reno-vascular hypertension (RVH)

- Atherosclerotic (ATS-RVH)
- Fibromuscular dysplasia (FMD-RVH)

Coarctation of the aorta

- Arteritis

Intrarenal (i.e. microscopic polyangiitis, granulomatosis with polyangiitis)

Schönlein-Henoch purpura

Cryoglobulinemic vasculitis

Iatrogenic

Drugs and exogenous hormones (i.e. contraceptive pills, immunosuppressive, non-steroidal anti-inflammatory drugs, etc.)

Acquired Apparent Mineralocorticoid Excess (licorice, etc.)

Cancer therapies (angiogenesis inhibitors as bevacizumab, and others)

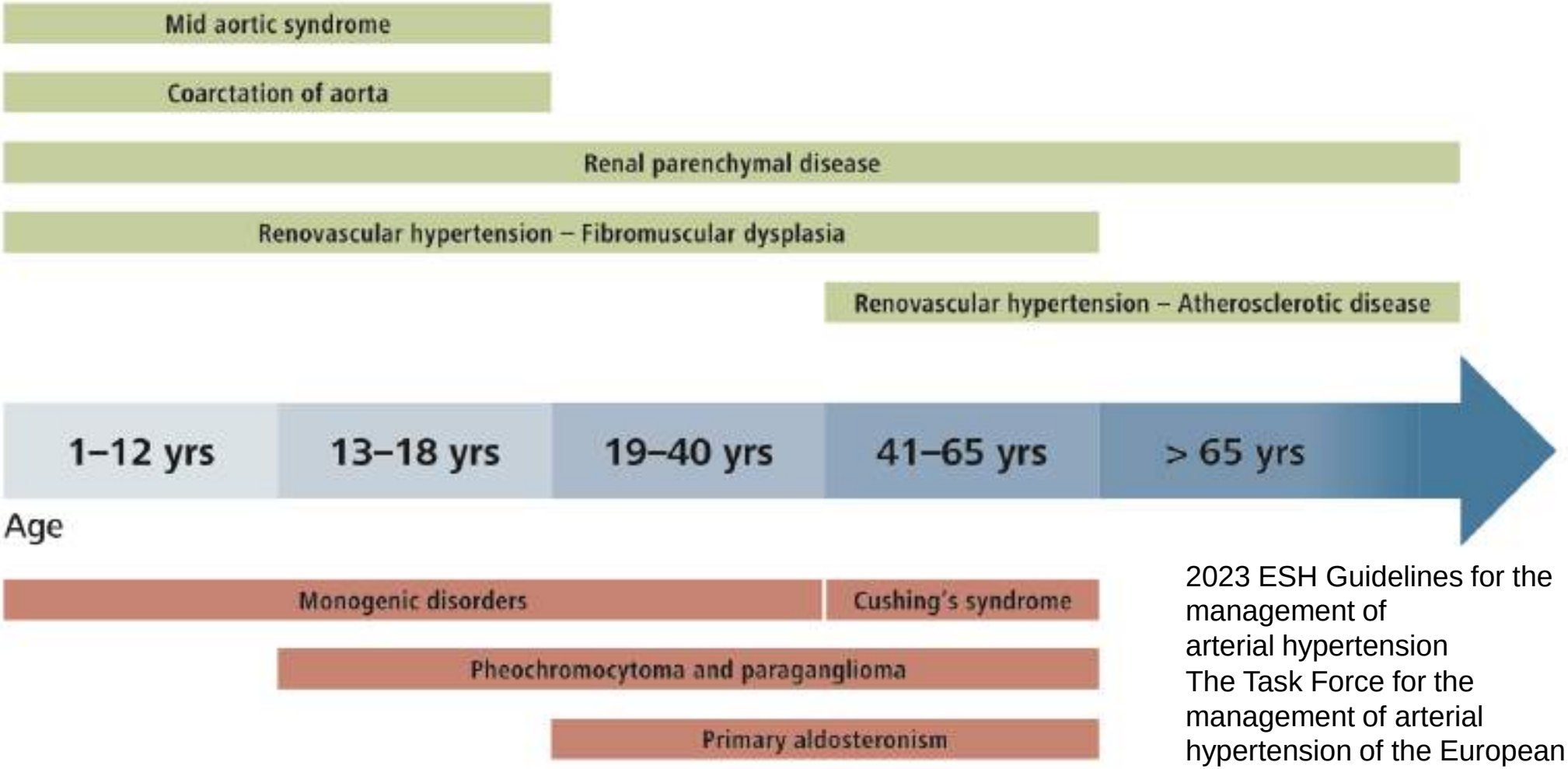
Rare genetic causes of secondary hypertension

Condition	Phenotype	Mechanism and Treatment
Liddle syndrome	Hypokalemia, metabolic alkalosis, low PRA or PRC, low PAC	Increased renal tubular ENaC activity; responds to treatment with amiloride
Apparent mineralocorticoid excess	Hypokalemia, metabolic alkalosis, low PRA or PRC, low PAC	Decreased 11β-hydroxysteroid dehydrogenase isoenzyme 2; responds to spironolactone
Gordon syndrome	Hyperkaliemia, metabolic acidosis, low PRA or PRC, low/normal PAC	Overactivity of the sodium-chloride cotransporter; responds to thiazides
Geller syndrome	Pregnancy-exacerbated hypertension, low PRA or PRC, low PAC	Agonist effect of progesterone on the mineralocorticoid receptor (which is constitutively active); responds to amiloride, spironolactone activates instead of blocking the receptor
Glucocorticoid-remediable aldosteronism (familial hyperaldosteronism type 1)	Hypokalemia, metabolic alkalosis, low PRA or PRC, increased PAC	Chimeric *CYP11B1/CYP11B2* gene; responds to glucocorticoids
Familial hyperaldosteronism type 2	Hypokalemia, metabolic alkalosis, low PRA or PRC, increased PAC	Increased activity of CLCN2 chloride channel; responds to steroidal MRA
Familial hyperaldosteronism type 3	Hypokalemia, metabolic alkalosis, low PRA or PRC, increased PAC	Loss of selectivity of KCNJ5 potassium channel; patients who do not respond to steroidal MRA require bilateral adrenalectomy
Familial hyperaldosteronism type 4	Hypokalemia, metabolic alkalosis, low PRA or PRC, increased PAC	Increased activity of CACNA1H calcium channel; responds to steroidal MRA
PASNA syndrome (primary aldosteronism, seizures and neurological abnormalities)	Hypokalemia, metabolic alkalosis, low PRA or PRC, increased PAC; neurological defects coexists	Increased activity of CACNA1D calcium channel; responds to steroidal MRA and CCB
11beta-hydroxylase deficiency	Hypokalemia, metabolic alkalosis, low PRA or PRC, low PAC, virilization of female individuals	Reduced activity of 11β-hydroxylase with increase of DOC and androgens; responds to glucocorticoids
17alpha-hydroxylase deficiency	Hypokalemia, metabolic alkalosis, low PRA or PRC, low PAC, pseudohermaphroditism in male individuals	Reduced activity of 17α-hydroxylase with increase of DOC and reduction of androgens; responds to glucocorticoids
Autosomal dominant hypertension with brachydactyly [342]	Brachydactyly type E (BDE), short stature, severe hypertension (salt-independent, age-dependent), high risk of death from stroke before age 50	PDE3A mutations upregulated the cAMP-hydrolytic activity that results in lower cAMP levels in vascular smooth muscle cells

Drugs that can cause hypertension

- Non-steroidal anti-inflammatory drugs
- Sodium-containing antacids
- Drugs used to treat attention-deficit/hyperactivity disorder(ADHD): Methylphenidate, amphetamine, dexmethylphenidate, and dextroamphetamine
- Anti-depressants: Monoamine oxidase inhibitors, tricyclic antidepressants, and serotonin-norepinephrine reuptake inhibitors
- Atypical antipsychotics like clozapine and olanzapine
- Decongestants that have phenylephrine or pseudoephedrine
- Appetite suppressants
- Herbal supplements like St John wort, ephedra, and yohimbine
- Systemic corticosteroids like dexamethasone, methylprednisolone, prednisone, prednisolone, and fludrocortisone
- Mineralocorticoids like carbenoxolone, licorice, 9-alpha fludrocortisone, and ketoconazole
- Estrogens, androgens, and oral contraceptives
- Immunosuppressants like cyclosporine
- Chronic recombinant human erythropoietin
- Recreational drugs: cocaine, methamphetamine, MDMA, bath salts
- Nicotine, alcohol
- Chemotherapeutic agents like gemcitabine (which causes microvascular injury)

Incidence of selected forms of secondary hypertension according to age



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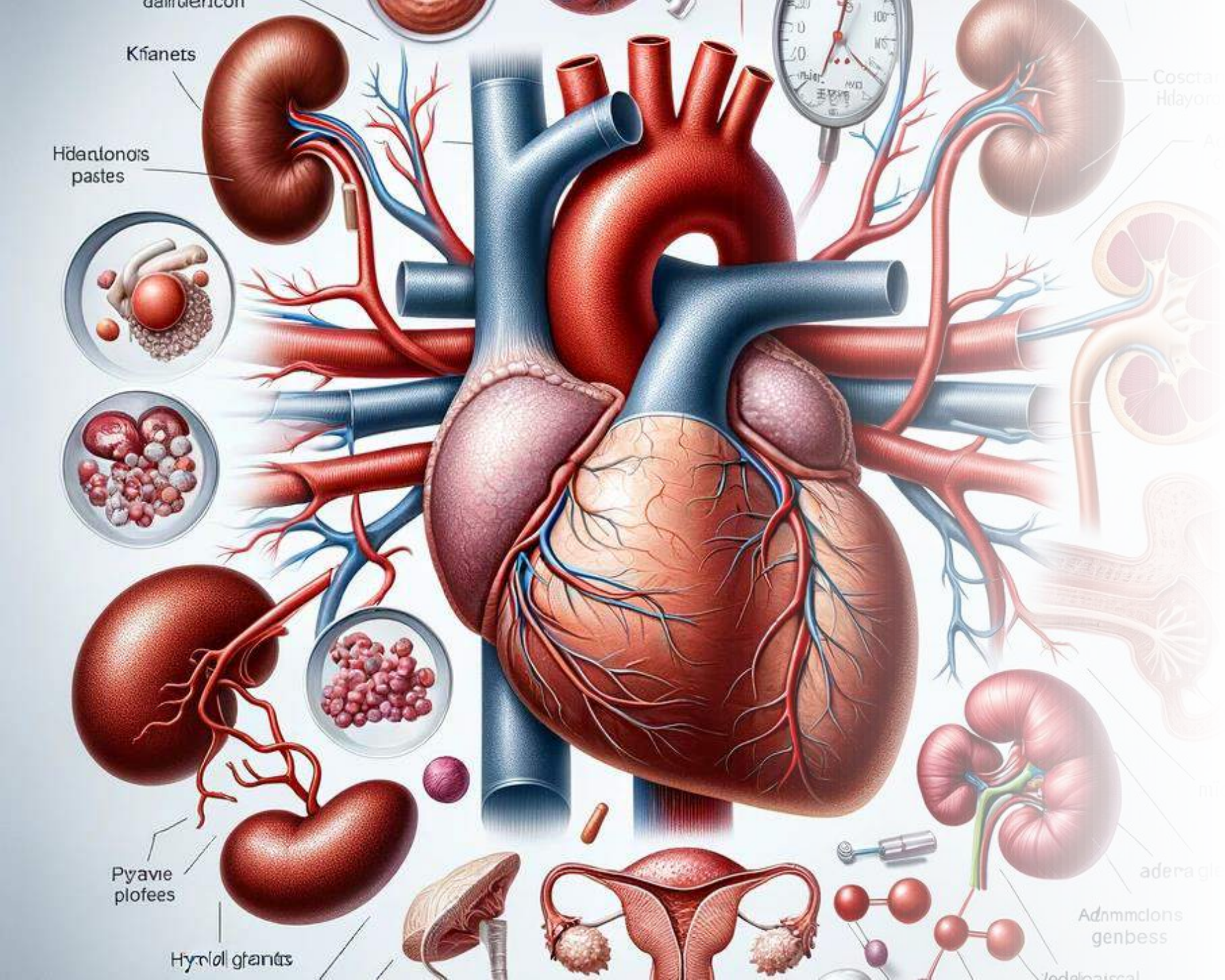
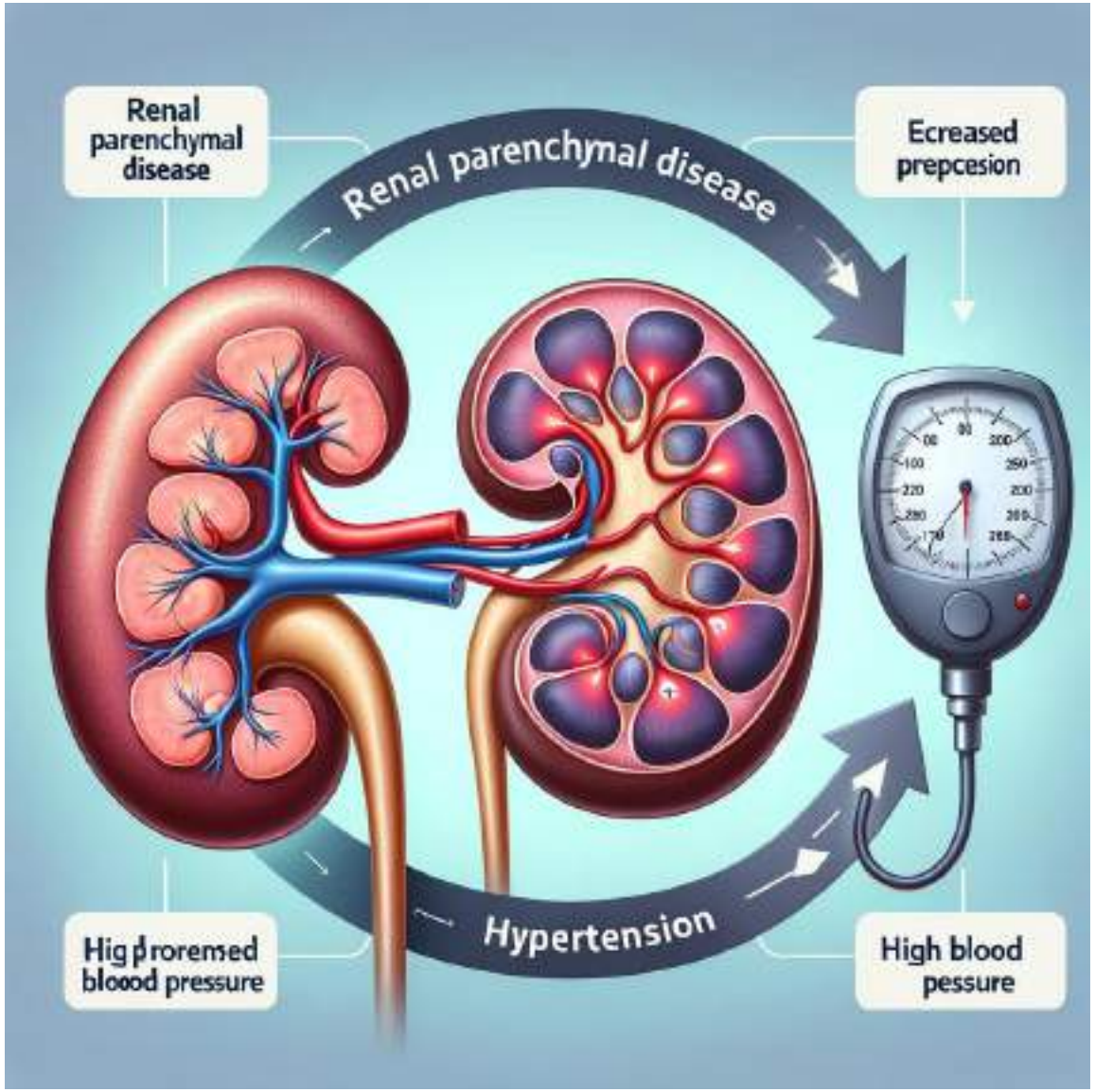


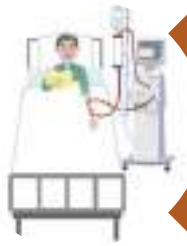
Etiology

Renal Parenchymal disease (RPD)

- the most common cause of secondary hypertension
- diabetic nephropathy, glomerulonephritis, interstitial renal parenchymal diseases, and polycystic kidney diseases
- > ½ of patients have HT
- HT has a negative effect on RPD and it accelerates the worsening of renal function and leads to ESRD



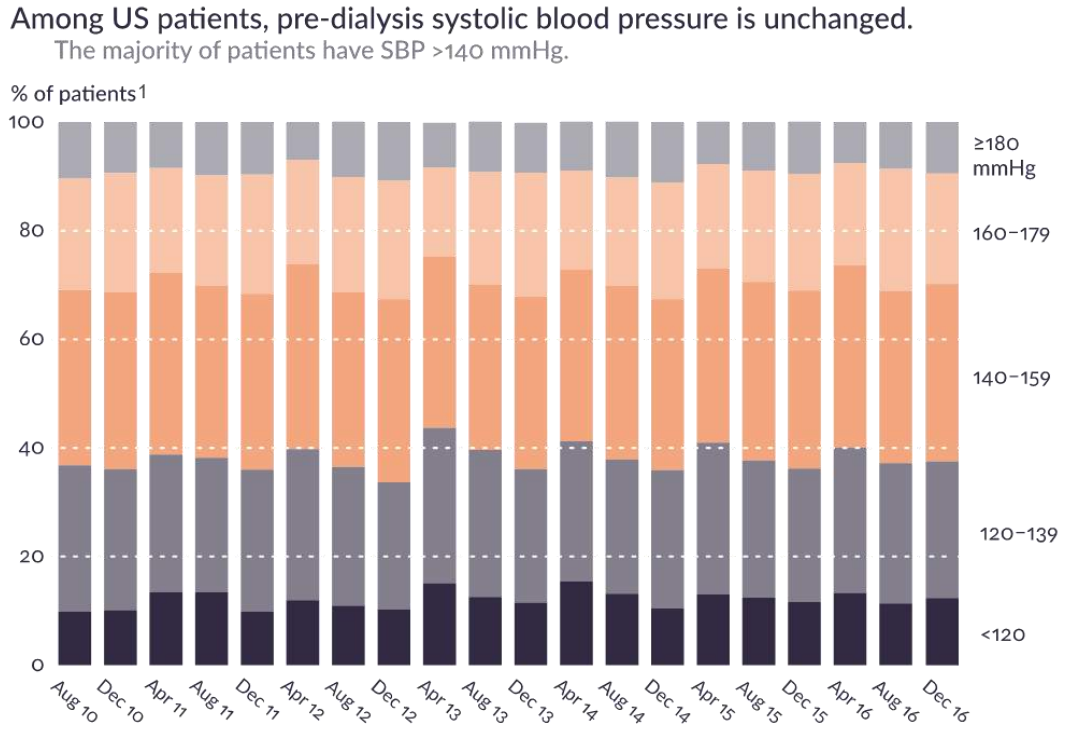
ESRD



Hypertension is common in dialysis patients[1]

Elevated blood pressure, measured by ambulatory blood pressure monitoring, is clearly associated with shorter survival[2-5]

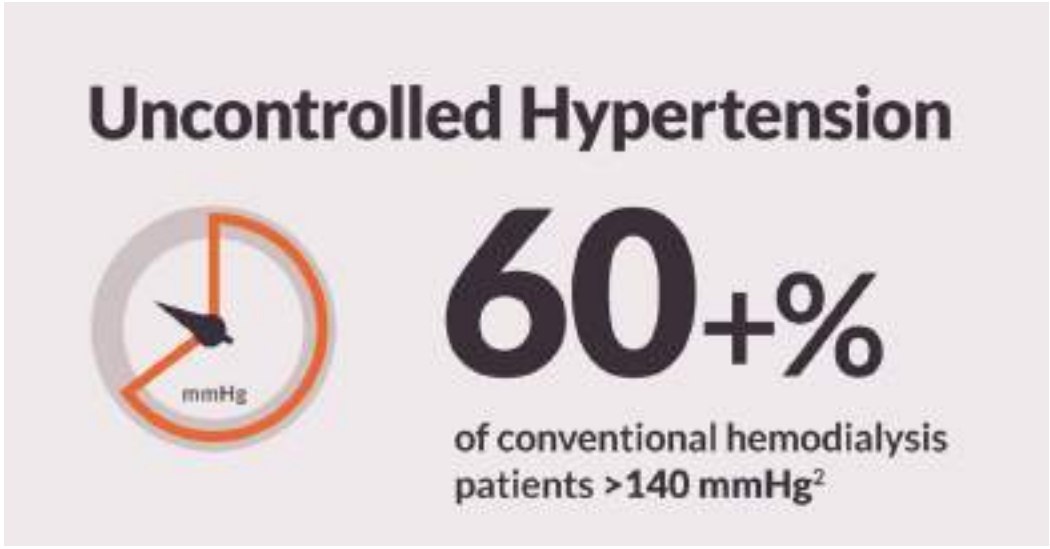
Appropriate treatment requires understanding of the principal causes of hypertension[6]



[1]DOPPS http://www.dopps.org/DPM\
[2]Alborzi, P., Patel, N., and Agarwal, R. Home blood pressures are of greater prognostic value than hemodialysis unit recordings. Clin J Am Soc Nephrol. 2007; 2: 1228–1234.
[3]Agarwal, R. Blood pressure and mortality among hemodialysis patients. Hypertension. 2010; 55: 762–768.
[4]Agarwal, R. The controversies of diagnosing and treating hypertension among hemodialysis patients. Semin Dial. 2012; 25: 370–376.
[5]Agarwal, R. Pro: Ambulatory blood pressure should be used in all patients on hemodialysis. Nephrol Dial Transplant. 2015; 30: 1432–1437.
[6]Sarafidis PA, et al. Hypertension in dialysis patients. J Hypertens. 2017;35(4):657-676

Numerous Causes of Hypertension[1]

- Volume overload
- Arterial stiffness increase
- Sympathetic nervous system activation
- Renin-angiotensin-aldosterone system activation
- Endothelial dysfunction
- Sleep apnea
- Erythropoietin-stimulating agents

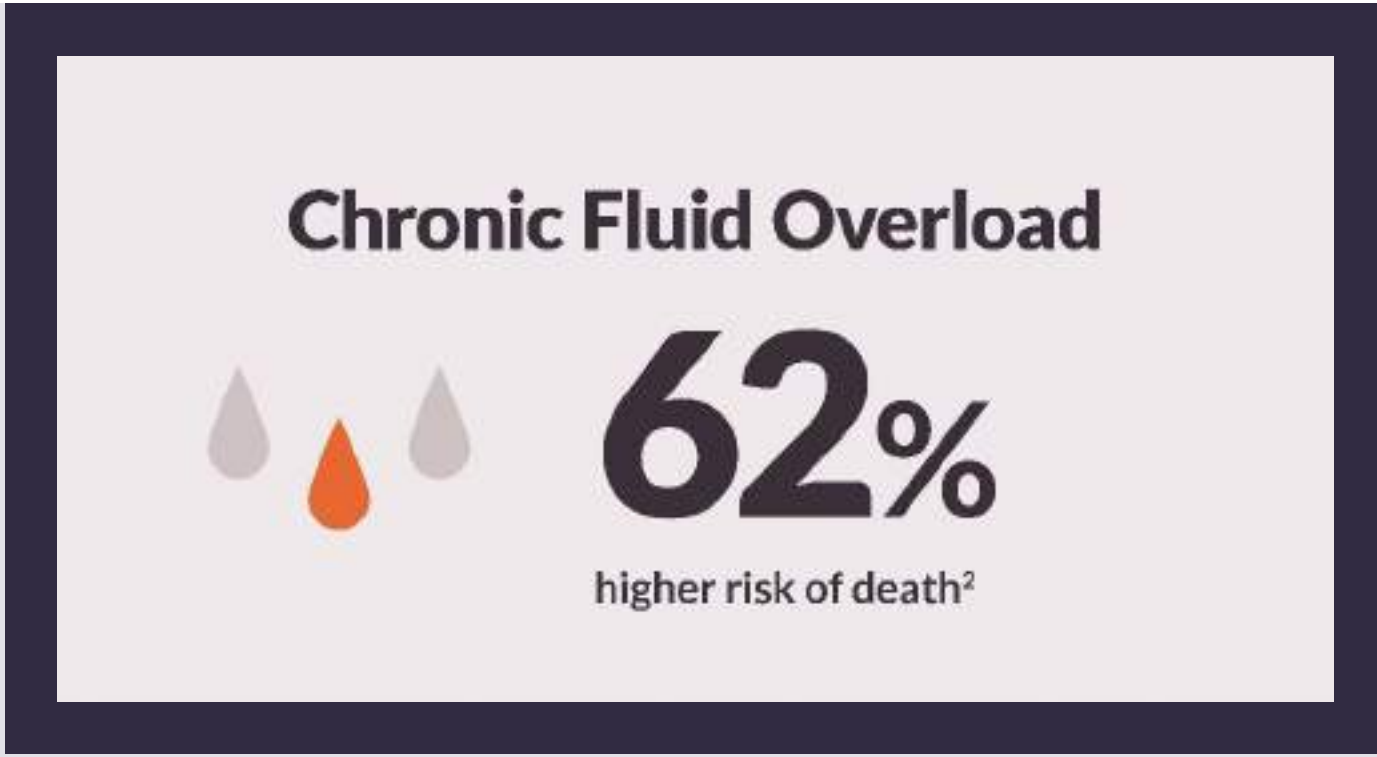


Bottom Line

"Sodium and volume excess appear to be the most important causes of hypertension in dialysis patients" [1]

[1]Sarafidis PA, et al. Hypertension in dialysis patients. J Hypertens. 2017;35(4):657-676

“Until fluid and sodium overload is removed during dialysis, a rise in peripheral vascular resistance will sustain hypertension in these individuals.”[1]



[1]Sarafidis PA, et al. Hypertension in dialysis patients. J Hypertens. 2017;35(4):657-676
[2]Zoccali C et al. Chronic fluid overload and mortality in ESRD. J Am Soc Nephrol. 2017 Aug;28(8):2491-2497.

Treatment Strategies[1]

Non-pharmacological strategies	Pharmacological strategies

Management of hypertension in dialysis patients should focus at correction of the primary pathogenetic mechanisms, that is sodium and volume excess.

[1]Sarafidis PA, et al. Hypertension in dialysis patients. J Hypertens. 2017;35(4):657-676.

Non-pharmacological Strategies to Reduce Blood Pressure

Reduce salt intake

Individualize dialysate sodium

Increase treatment length and frequency

Reduce salt intake[1]

- Reducing the amount of sodium gained from diet or dialysate fluid is critical to achieve BP control
- Dietary sodium restriction appears to be an effective approach to limit sense of thirst, reduce interdialytic weight gain and facilitate achievement of dry-weight and BP control

[1]Sarafidis PA, et al. Hypertension in dialysis patients. J Hypertens. 2017;35(4):657-676.

Non-pharmacological Strategies to Reduce Blood Pressure

Reduce salt intake

Individualize dialysate sodium

Increase treatment length and frequency

Individualize dialysate sodium[1]

- Recent research has emphasized that a high-dialysate sodium concentration may increase thirst and interdialytic weight gain
- A consensus document by the Chief Medical Officers of US Dialysis Providers warns against the use of dialysate with a sodium concentration exceeding predialysis serum sodium

[1]Sarafidis PA, et al. Hypertension in dialysis patients. J Hypertens. 2017;35(4):657-676.

Non-pharmacological Strategies to Reduce Blood Pressure

Reduce salt intake

Individualize dialysate sodium

Increase treatment length and frequency

Increase treatment length and frequency[1]

- Length of dialysis session **must not** be decided only on the grounds of optimal Kt/V
- Hemodialysis patients should receive at least 3 dialysis sessions of **4 hours each** per week
- Increasing duration of dialysis may represent an additional approach to control BP
- Patients assigned to **longer** or **more frequent** dialysis regimens achieve better BP control with reduced requirements for antihypertensive medications

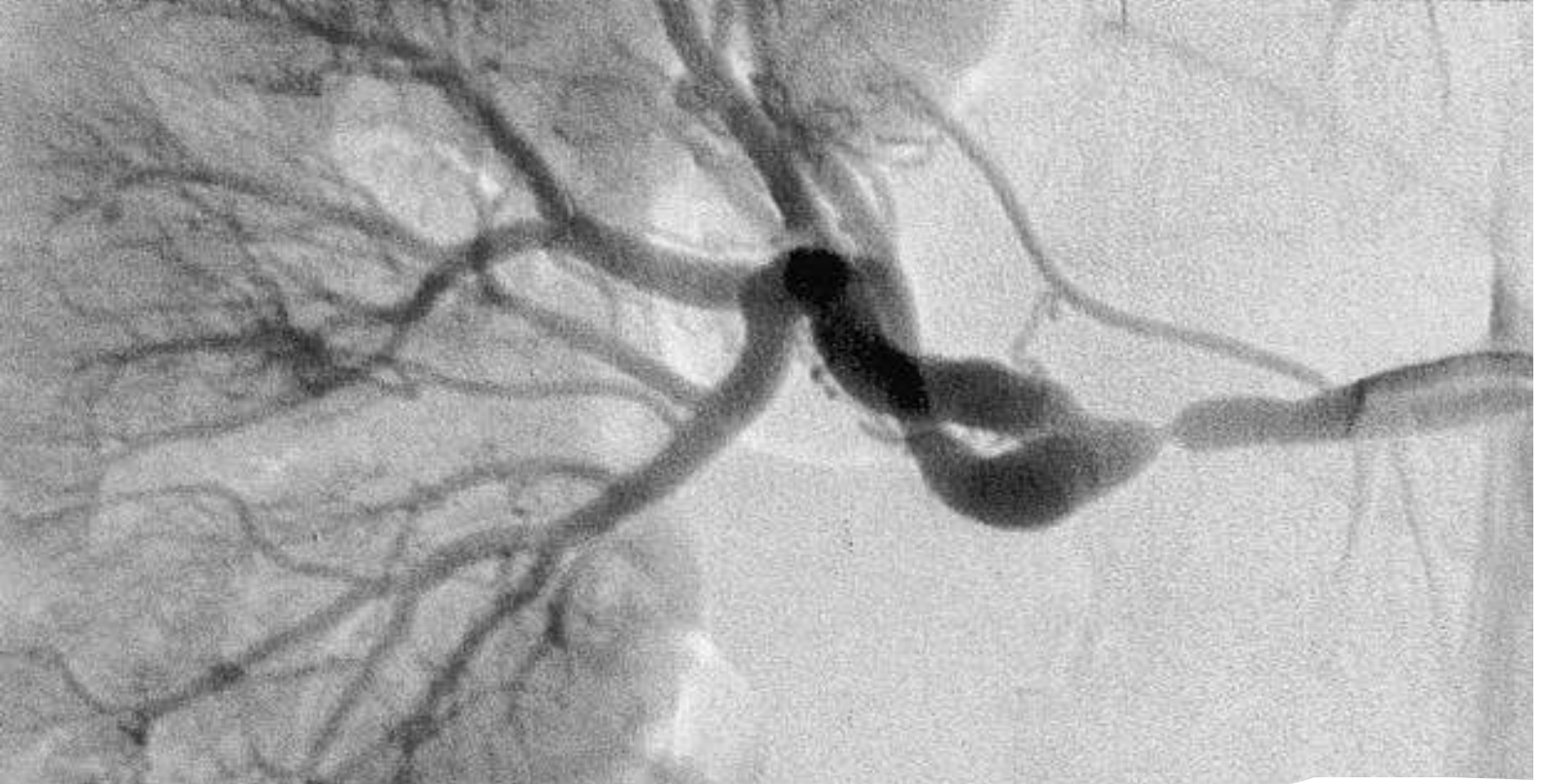
[1]Sarafidis PA, et al. Hypertension in dialysis patients. J Hypertens. 2017;35(4):657-676.

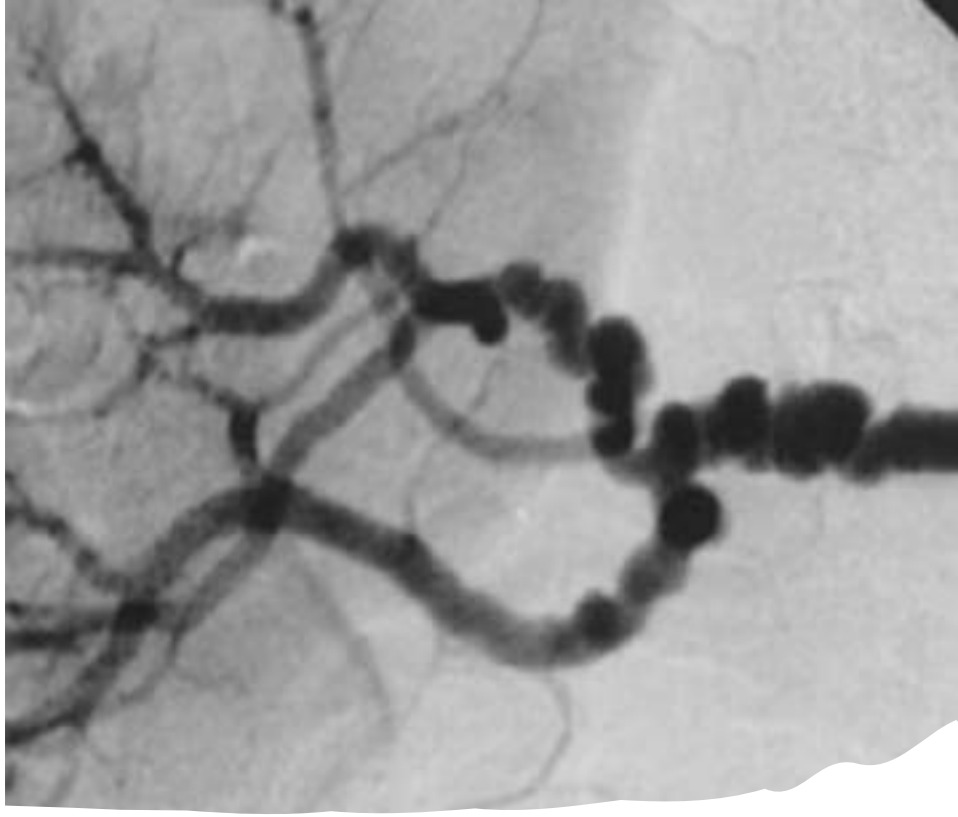
Pharmacological Strategies[1]

Beta blockers	Calcium channel blockers
ACE inhibitors & ARBs	Mineralocorticoid receptor antagonists

Administration of antihypertensive drug therapy in dialysis patients considered to be volume overloaded should follow the attainment of dry-weight.

[1]Sarafidis PA, et al. Hypertension in dialysis patients. J Hypertens. 2017;35(4):657-676.





Renovascular Hypertension (RVH)

(A) Atherosclerotic renovascular disease

Prevalence: 6–14%[a]

Suggestive symptoms, signs and findings

Resistant hypertension
Flash pulmonary edema
Rapidly declining kidney function
Acute renal function degradation on ACEi or ARB
Generalized atherosclerosis[b]

1st choice screening test

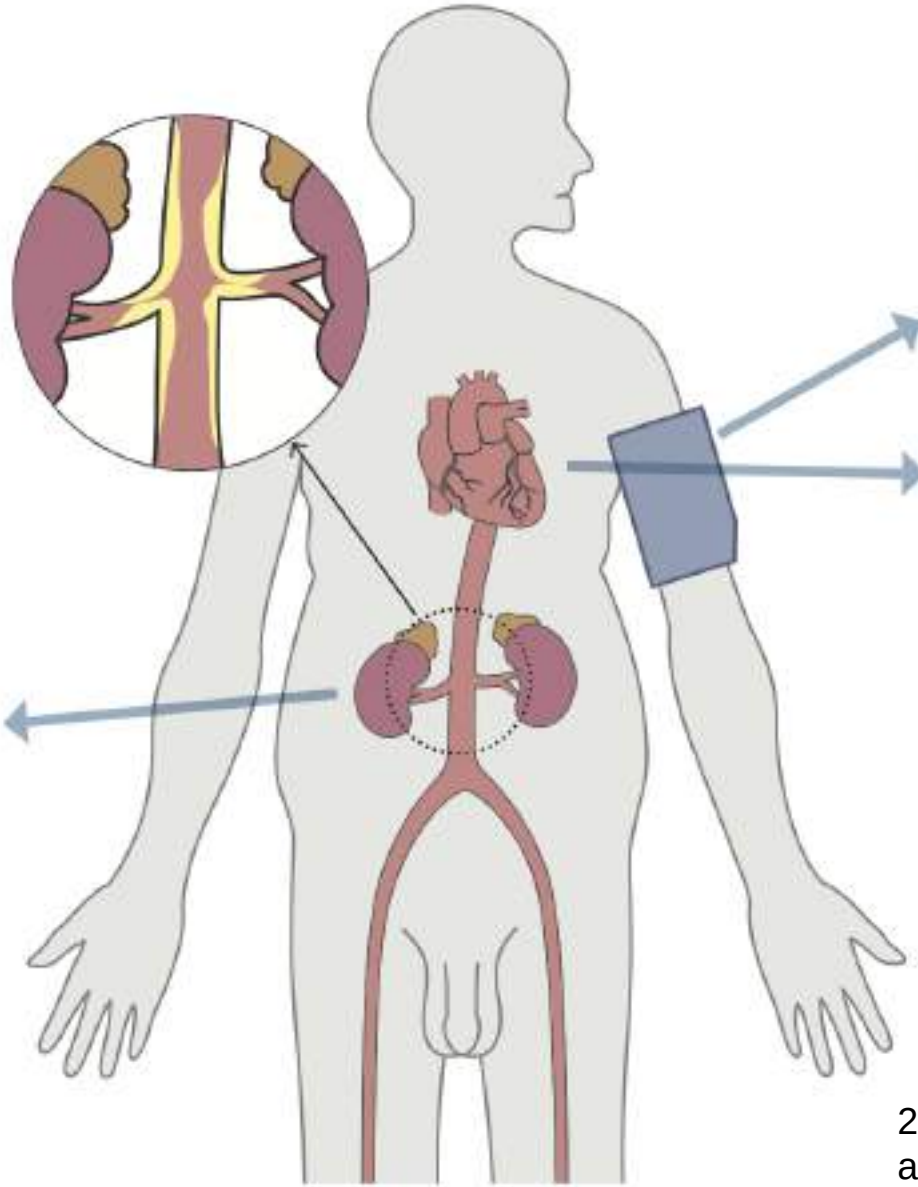
Renal artery duplex ultrasound; otherwise angio-CT or angio-MR

Further work-up

Angio-CT or angio-MR
Invasive catheter angiography

Treatment[c,d]

Antihypertensive treatment
Strict control of CV risk factors
Revascularization (selected cases)



Cardiovascular phenotype

24h ABPM – resistant hypertension, frequent non-dipping

- LVH
- Decreased diastolic function
- Decreased systolic function

Increased CV Risk and mortality

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(B) Fibromuscular Dysplasia

Prevalence:
<1 to 6%[a]

Suggestive symptoms, signs and findings

Early-onset/ severe hypertension
Migraine
Pulsatile tinnitus

1st choice screening test[b]

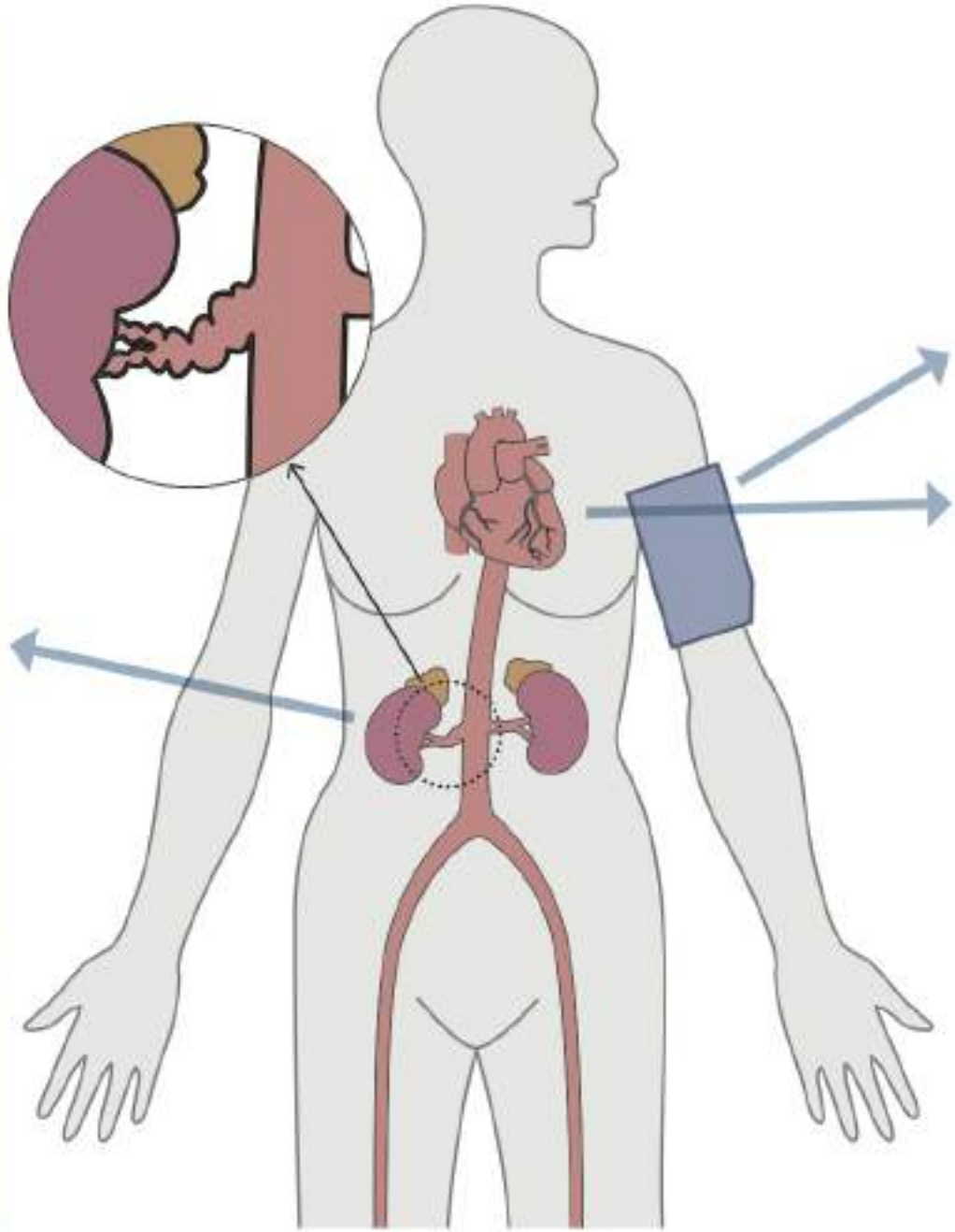
Renal artery duplex ultrasound; otherwise angio-CT or angio-MR

Treatment

Antihypertensive treatment
Angioplasty without stenting[c,d]

Follow-up

- Whole body angio-CT or angio-MR at diagnosis[e]
- Indefinite follow-up



Cardiovascular phenotype

24h ABPM – early onset or resistant hypertension

Frequent in patients with Spontaneous Coronary Artery Dissection (SCAD)

May affect all medium sized arteries (most frequent: renal and cervical arteries)

Often associated with arterial dissections and aneurysms

Cardiovascular phenotype:
From asymptomatic to resistant hypertension, stroke, renal, mesenteric or myocardial infarction

Clinical findings suggestive of renovascular hypertension (RVH) according to the etiology

FMD-RVH

Early onset of HT (< 30 years old), especially in women

Unilateral small kidney without a causative urological abnormality

Abdominal bruit in the absence of atherosclerotic disease or risk factors for atherosclerosis

Suspected renal artery dissection/infarction

Presence of FMD in at least 1 other vascular territory

History of stroke, headaches, neck pain, and a pulsatile ringing or swooshing sound in the ears when carotid and/or vertebral arteries is involved

History of ACS caused by spontaneous coronary artery dissection

Weight loss, abdominal pain and ischemia, in case of abdominal artery involvement

Typical symptoms and signs of peripheral artery diseases

ATS-RAS

Severe HT > 55 years old

Most frequent in older people, smokers, obese and diabetic

History of generalized atherosclerosis (coronary artery disease, peripheral vascular disease, etc.)

Shared by the two conditions

Accelerated, or malignant or grade 3 (> 180/110 mmHg) HT

Drug-resistant hypertension (blood pressure target not achieved despite 3 drug-therapy at optimal doses including a diuretic)

Development of new azotemia or worsening renal function after administration of ACE-inhibitors or ARBs

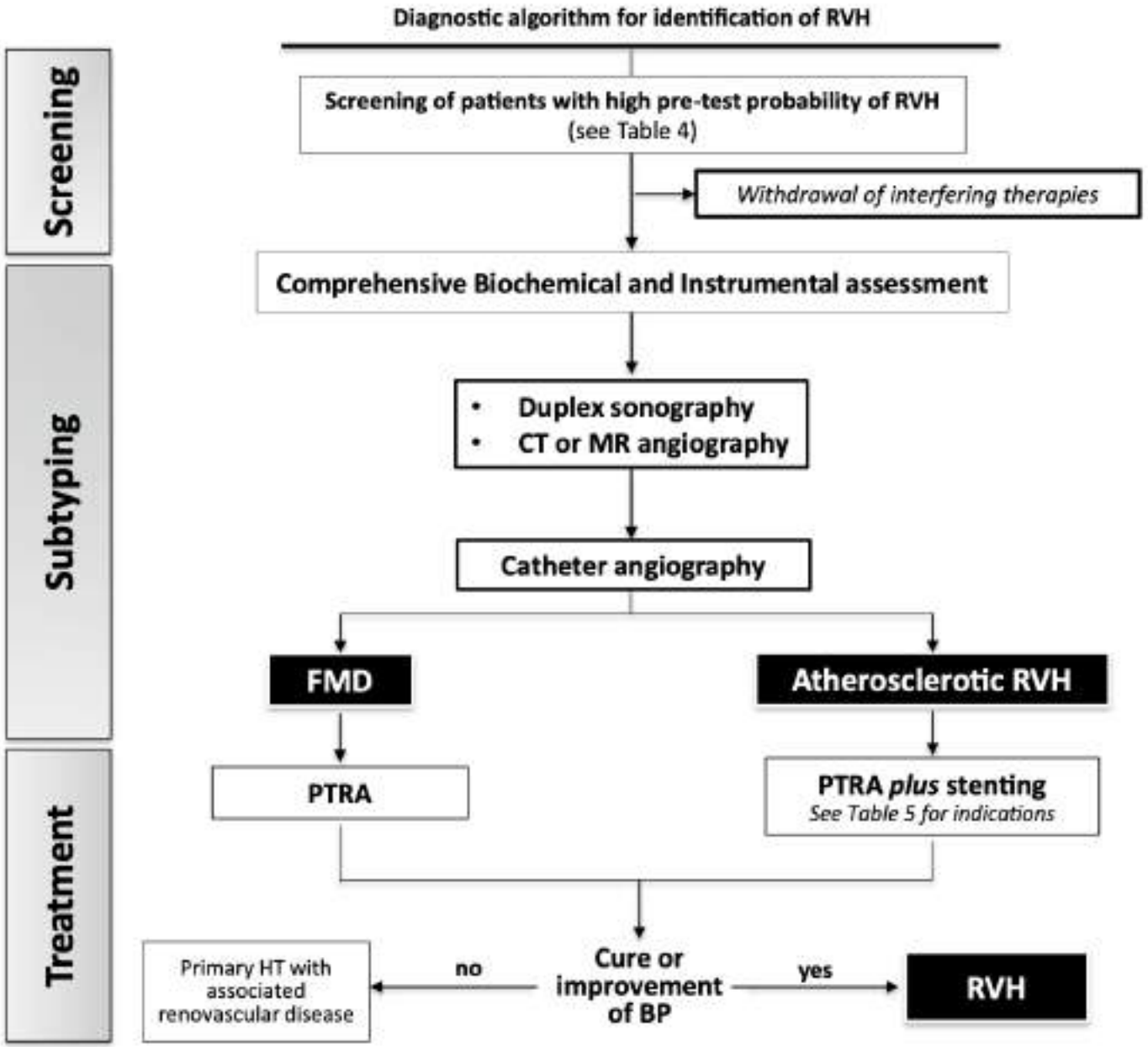
Unexplained atrophic kidney or size discrepancy between kidneys of greater than 1.5 cm

Sudden, unexplained pulmonary edema

Multi-vessel coronary artery disease

Unexplained congestive heart failure

Refractory angina



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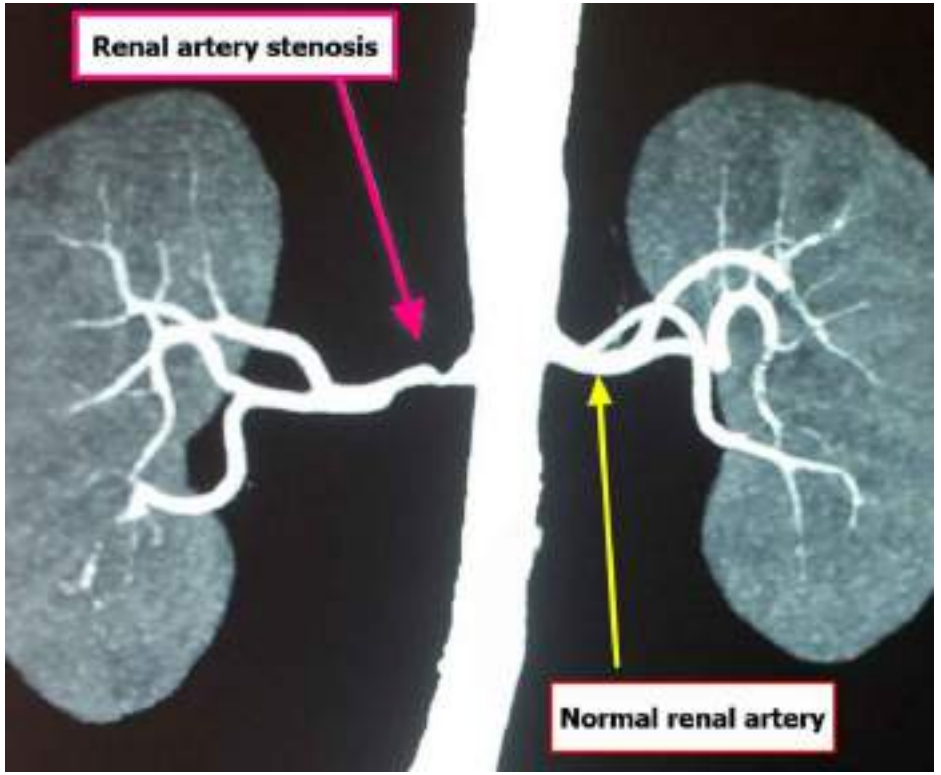
https://doi.org/10.1007/s40292-020-00415-9

ACC/AHA recommendations to perform angioplasty and stent in ATS-RVH

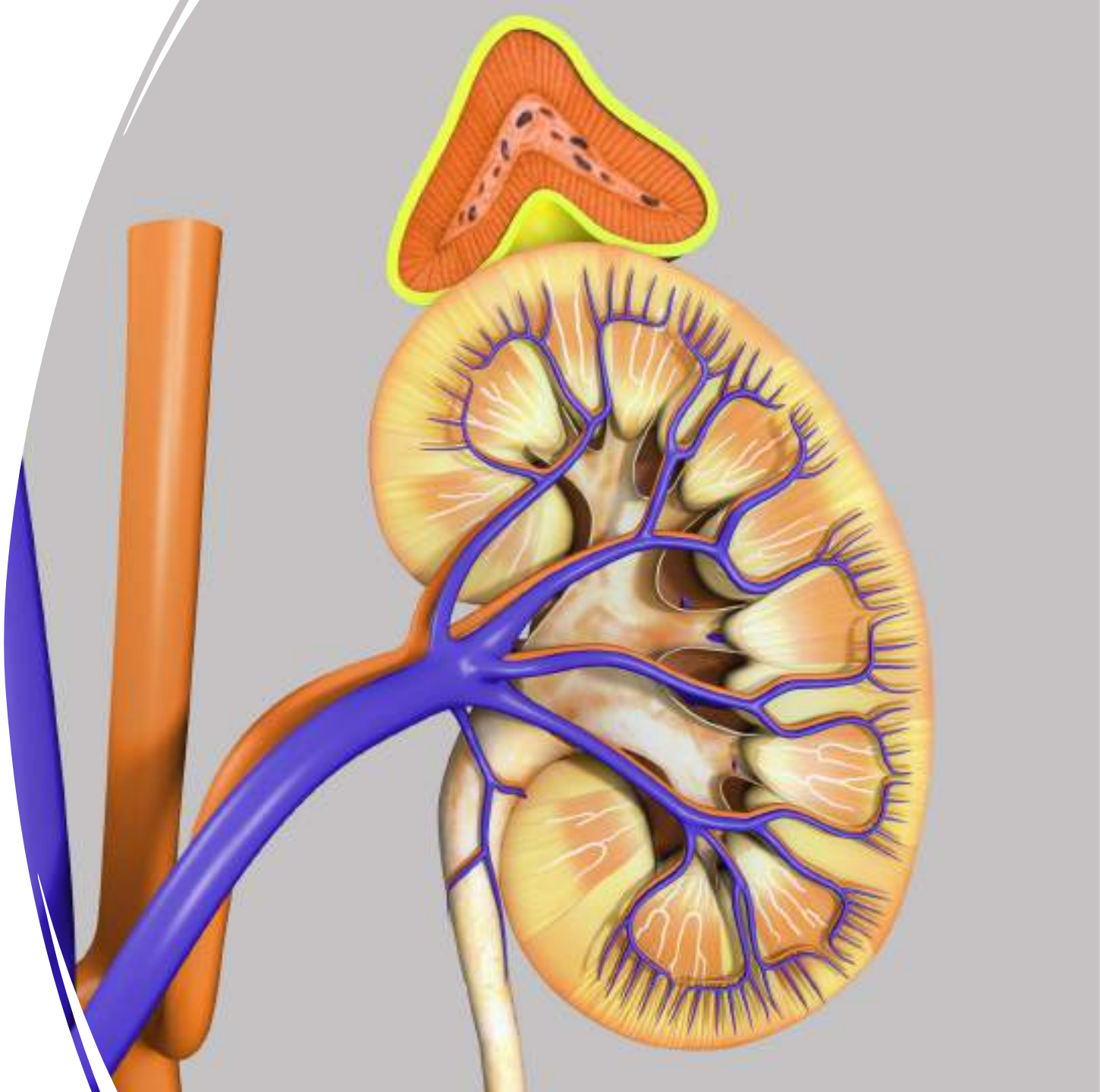
Indication	Class of recommendation/level of evidence
An asymptomatic bilateral or viable kidney with a hemodynamically significant RAS	IIb/C
Patients with hemodynamically significant RAS and accelerated HT, resistant HT, malignant HT, HT with an unexplained unilateral small kidney, and HT with intolerance to medication	IIa/C
RAS and progressive chronic kidney disease with bilateral RAS or a RAS to a solitary functioning kidney	IIa/B
Patients with RAS and chronic renal insufficiency with unilateral RAS	IIb/C
Patients with hemodynamically significant RAS and recurrent, unexplained congestive heart failure or sudden, unexplained pulmonary edema	I/B
Patients with hemodynamically significant RAS and unstable angina	IIa/B
Renal stent placement is indicated for ostial atherosclerotic RAS lesions that meet the clinical criteria for intervention	I/B

Renal artery angioplasty would be ‘rarely appropriate’ in:

- (1) asymptomatic unilateral hemodynamically significant RAS in a viable kidney;
- (2) patients with small (< 7 cm pole to pole) nonviable kidneys;
- (3) patients with moderate RAS (50–69% diameter stenosis) with translesional gradients that fail to achieve the threshold



Primary Aldosteronism



Clinical characteristics suggestive of primary aldosteronism

Sustained BP > 150/100 mmHg on each of 3 measurements obtained on different days

Hypertension (BP > 140/90 mmHg) resistant to 3 conventional antihypertensive drugs (including a diuretic)

Spontaneous or diuretic-induced hypokalemia

Hypertension and adrenal incidentaloma

Obstructive sleep apnea

Family history of primary aldosteronism and/or early-onset hypertension or cerebrovascular accident at a young age (< 40 years)

Atrial fibrillation not explained by other causes (i.e. valvular disease)

HMOD (i.e. LVH, diastolic dysfunction, microalbuminuria, CKD) in excess of what expect based on BP values

- **PA is the most common curable form of HT**
- **PA is associated with an excess rate of HMOD and CV complications as compared to primary essential HT with a similar degree of BP elevation**

(C) Primary aldosteronism

Prevalence: 6–20%[a]

Suggestive symptoms, signs and findings

- Resistant hypertension
- Grade 2 or 3 hypertension
- Hypokalemia/Potassium in the low-normal range
- Atrial fibrillation
- OSA
- Adrenal incidentaloma[b]
- Family history of PA/early stroke

1st choice screening test[c]

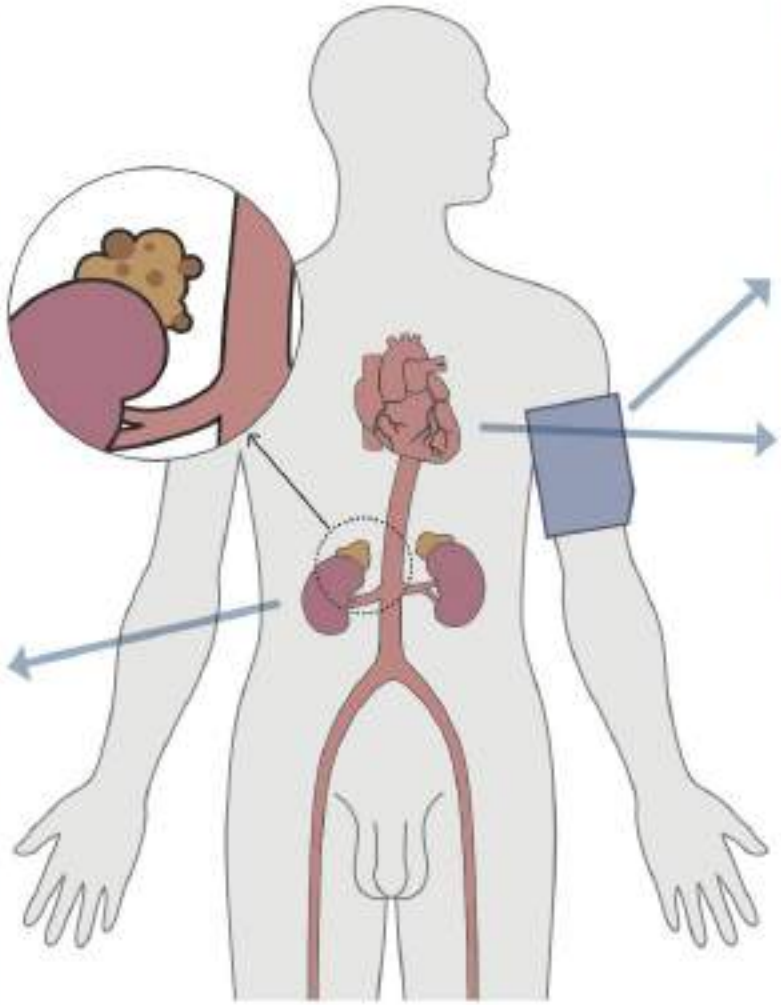
Plasma aldosterone to renin ratio (ARR)

Further work-up[d]

- CT scanning
- IV saline infusion test (SIT)
- Fludrocortisone suppression test (FST)
- Oral sodium loading test (SLT)
- Captopril challenge test (CCT)
- Adrenal vein sampling
- Genetic testing in selected cases[e]

Treatment

- Surgical treatment (laparoscopic adrenalectomy) – unilateral PA
- Medical treatment – bilateral adrenal disease[f]



Cardiovascular phenotype

24 ABPM – true resistant hypertension, frequent non-dipping

- LVH
- Decreased diastolic function
- Myocardial fibrosis (MRI)

Increased CV Risk and mortality

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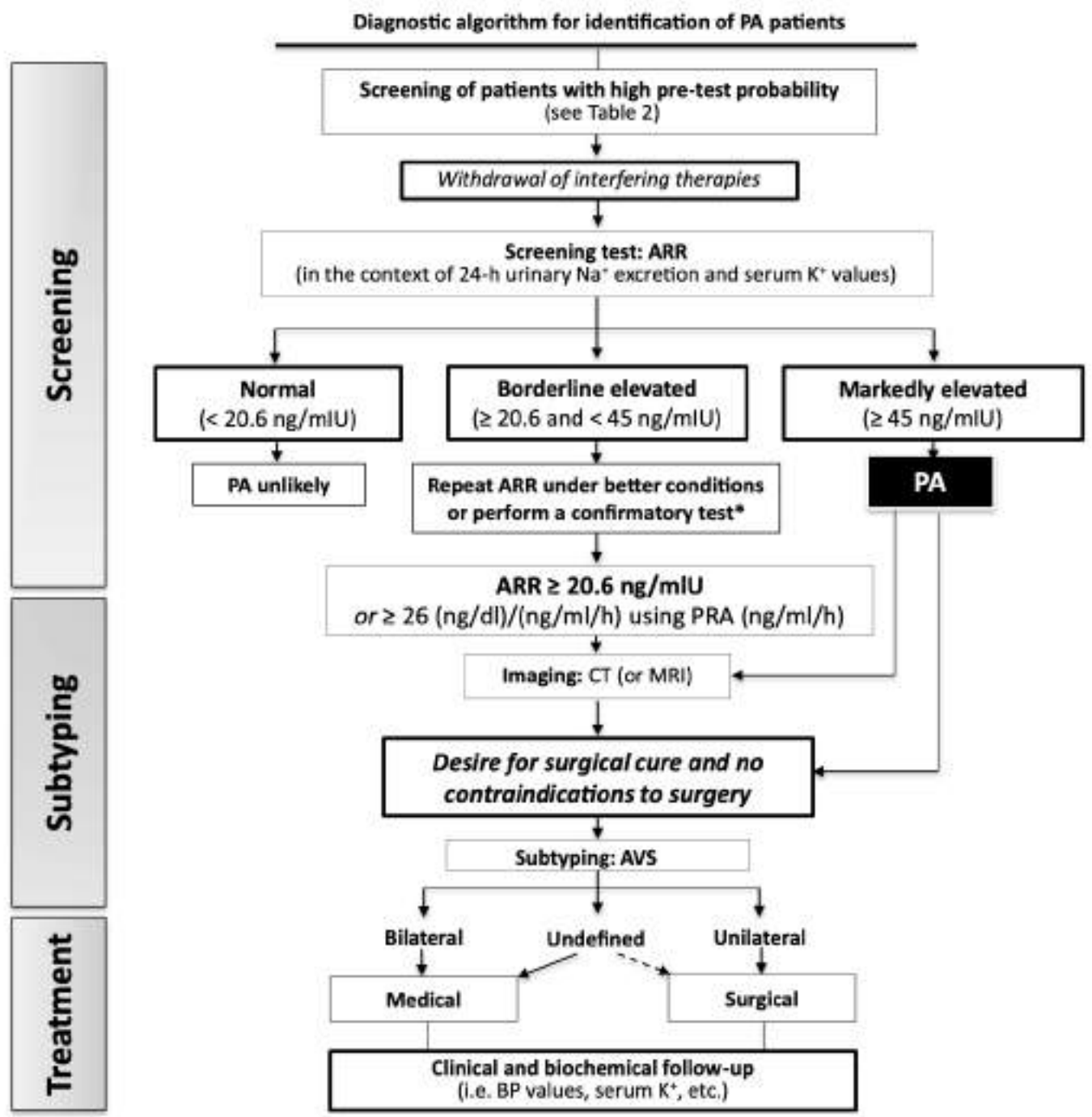

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Pheochromocytoma/Paraganglioma

- About 80–85% of chromaffin-cell tumors are pheochromocytomas (PHEO), and 15–20% are paragangliomas (PGL)
- Owing to their common embryologic origin and clinical similarities, they are usually jointly defined as pheochromocytomas and paragangliomas, in short PPGLs
- PPGLs have been defined as ***'the great simulators'*** as symptoms can range from none to many. Symptoms are usually due to a mass effect and/ or derive from catecholamines excess
- Paroxysmal or sustained high BP is the most common sign of PPGLs and involves up to 80% of patients

(D) Pheochromocytoma and paraganglioma

Prevalence: <1%[a]

Suggestive symptoms and signs[b]

- paroxysmal symptoms (such as headache, sweating, palpitation, increased HR)
- large BP variation
- CV manifestations (e.g. MI, arrhythmias, Takotsubo cardiomyopathy)

1st choice screening test

Plasma or urinary free metanephrines

Further work-up

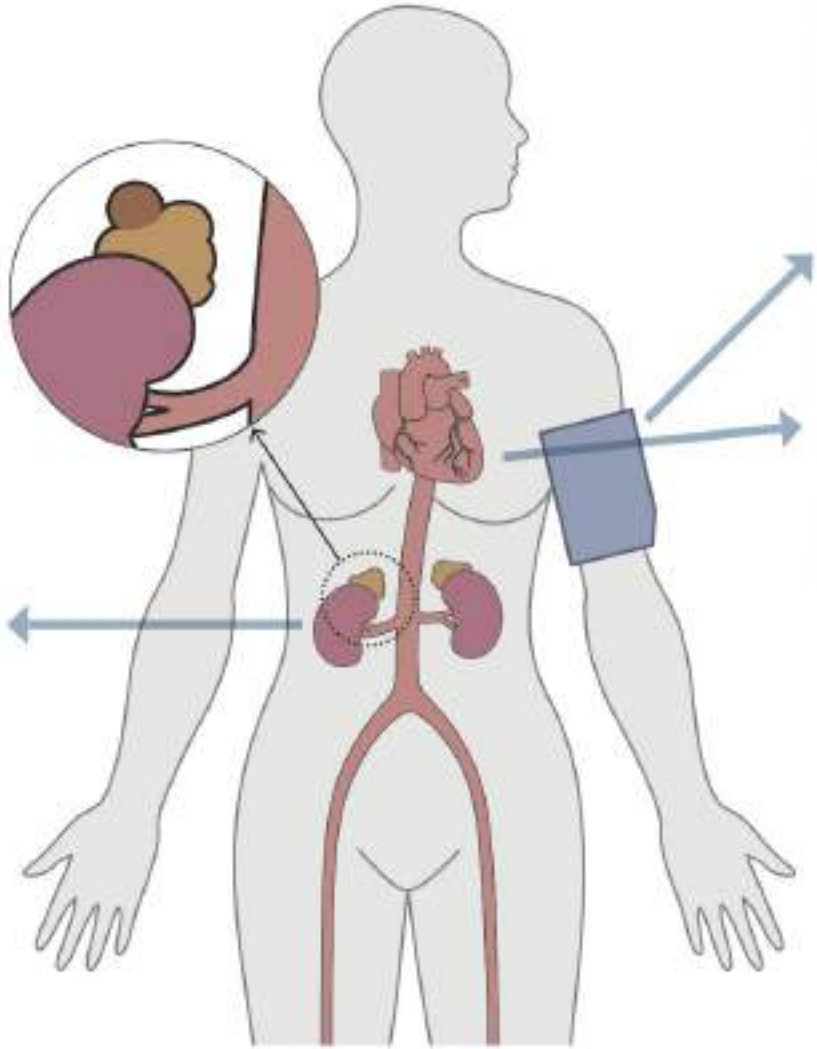
Contrast enchanced CT or MRI
Functional imaging
Genetic testing[c]

Treatment[d]

Surgical resection
(Pheochromocytoma: minimally invasive laparoscopic adrenalectomy)

Follow-up[e]

In most cases > 10 yrs



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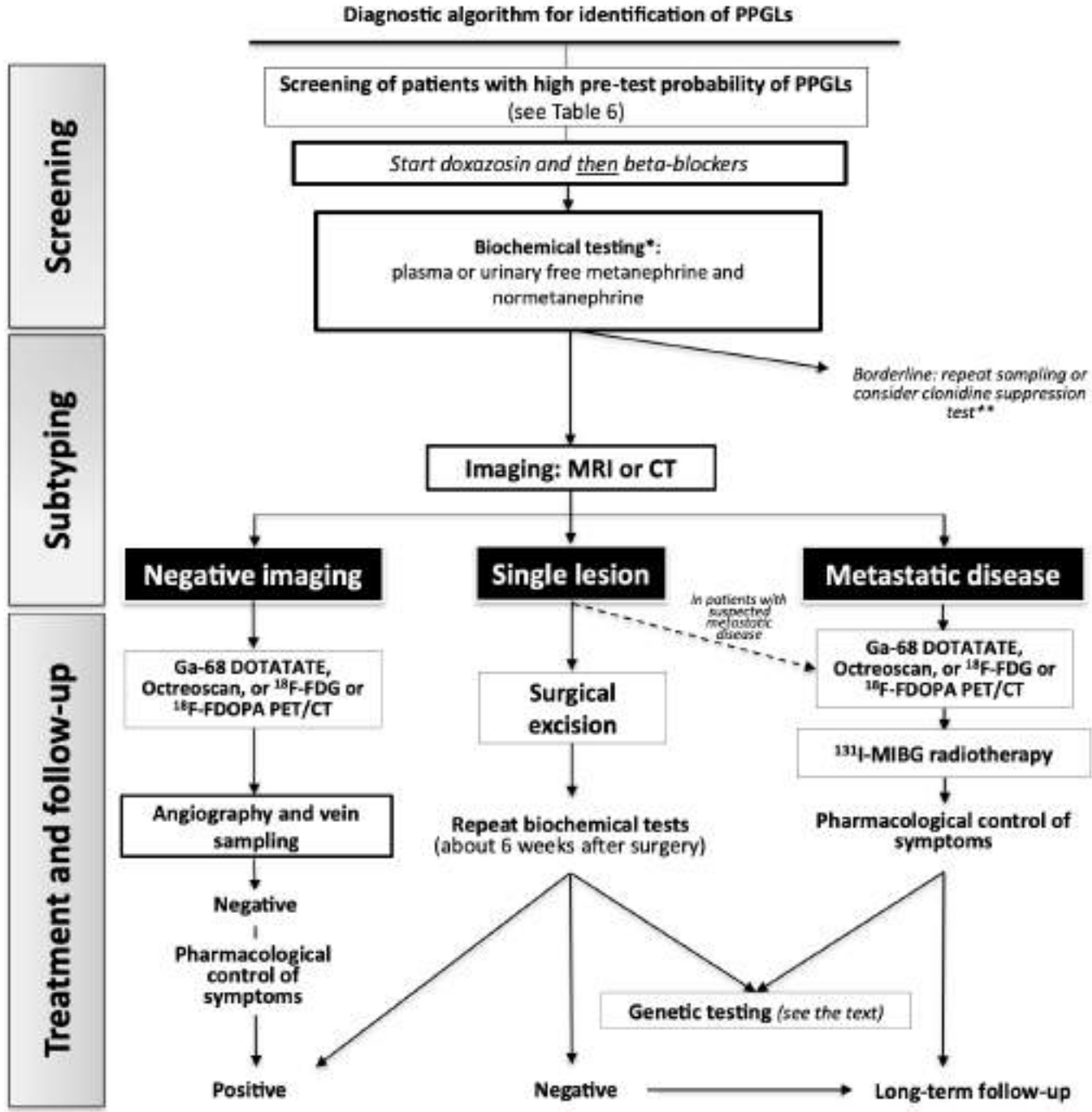

Diagnostic algorithm for identification of PPGLs
Screening
Subtyping
Treatment and follow-up
Screening of patients with high pre-test probability of PPGLs
(see Table 6)
Start doxazosin and then beta-blockers
Biochemical testing*:
plasma or urinary free metanephrine and normetanephrine
Borderline: repeat sampling or consider clonidine suppression test**
Imaging: MRI or CT
Negative imaging
Single lesion
Metastatic disease
In patients with suspected metastatic disease
Ga-68 DOTATATE, Octreoscan, or 18F-FDG or 18F-FDOPA PET/CT
Surgical excision
Ga-68 DOTATATE, Octreoscan, or 18F-FDG or 18F-FDOPA PET/CT
131I-MIBG radiotherapy
Angiography and vein sampling
Repeat biochemical tests
(about 6 weeks after surgery)
Pharmacological control of symptoms
Negative
Pharmacological control of symptoms
Genetic testing (see the text)
Positive
Negative
Long-term follow-up

(E) Cushing's syndrome

Prevalence: 2–5%[a]

Suggestive symptoms and signs

Resistant hypertension
Easy bruising, facial plethora, ‚moon' face, skin thinning
Proximal myopathy
Weight gain with centripetal distribution of body fat
Diabetes mellitus

1st choice screening test[b]

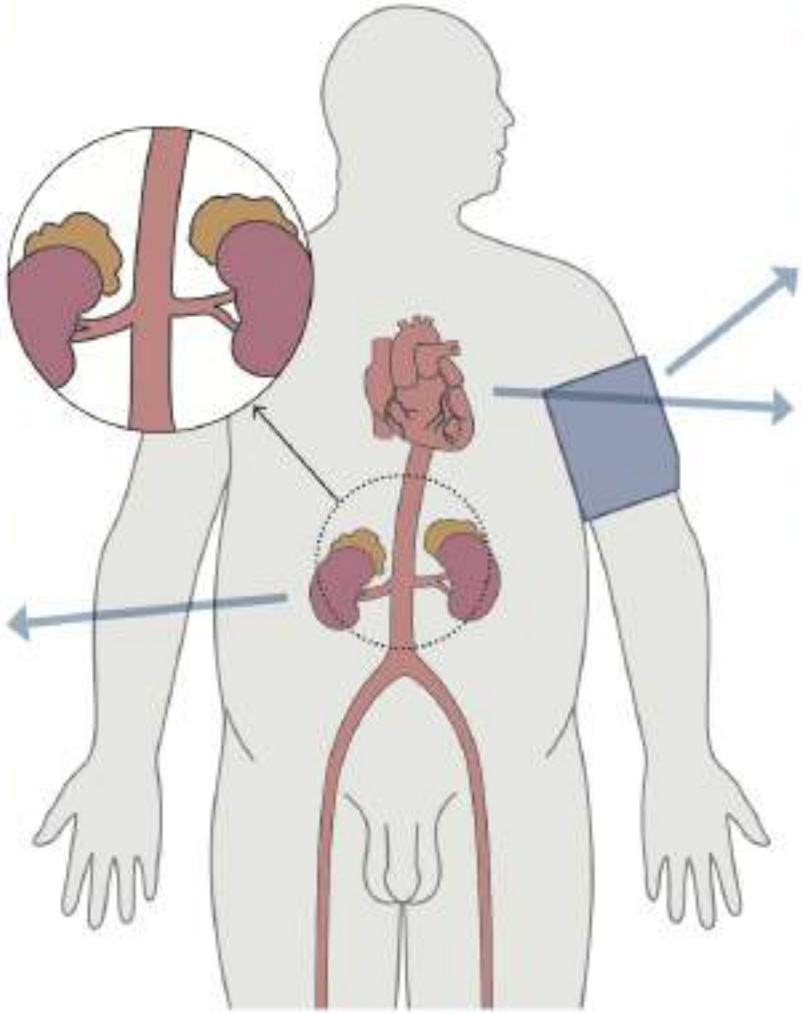
Overnight 1 mg dexamethasone suppression test
24-h urinary free cortisol
Late-night salivary cortisol

Further work-up

Morning plasma ACTH
ACTH stimulation by CRH or desmopressin
CT

Treatment

Medical – normalization of cortisol levels
Surgical – first line treatment for Cushing's disease , ectopic Cushing's syndrome and ACTH-independent hypercortisolism



Cardiovascular phenotype

24h ABPM – frequent non-dipping
Short-term BP variability

- LVH
- Decreased systolic function
- Decreased diastolic function

Increased CV Risk and mortality

2023 ESH Guidelines for the management of arterial hypertension
The Task Force for the management of arterial hypertension of the European Society of Hypertension

Screening and Diagnostic Tests for CS

- (1) patients with unusual features, for example, osteoporosis at a young age
- (2) patients with suggestive signs and symptoms
- (3) adrenal incidentaloma compatible with adenoma

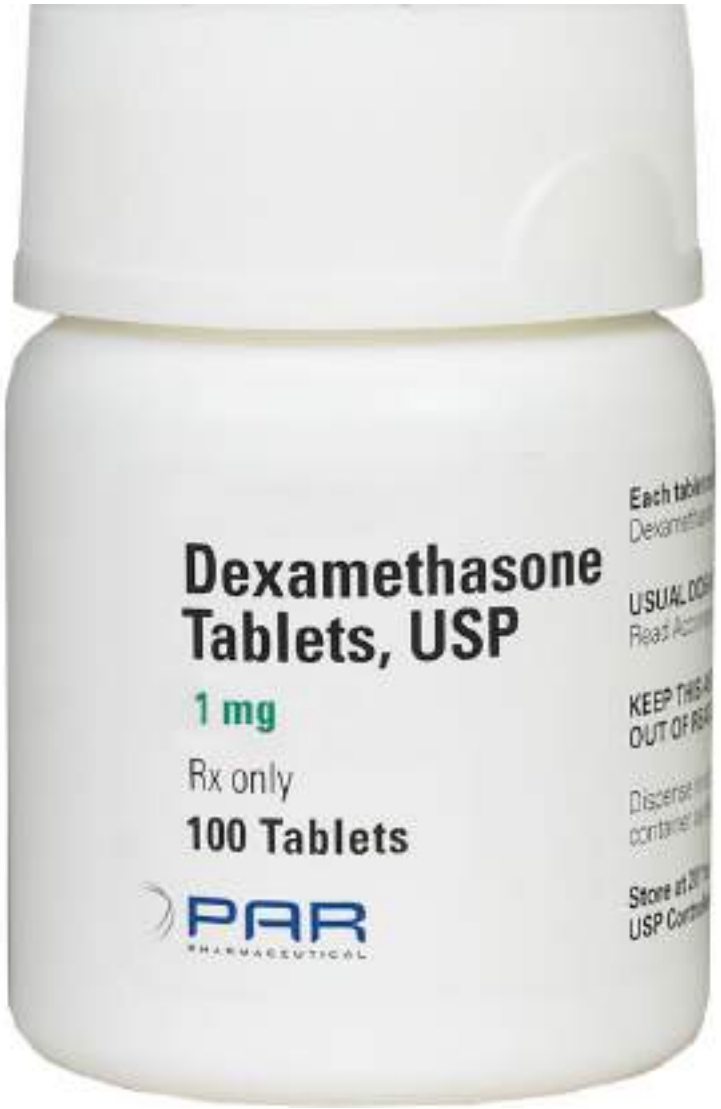
Decreased libido	91–100%
Facial rounding ("moon faces")	
Obesity	
Impaired glucose tolerance/diabetes	71–90%
Menstrual changes	
Hirsutism	
Striae rubre	
Muscle weakness	51–70%
Osteopenia/osteoporosis/fractures	
Psychiatric disturbances (especially lethargy, depression)	
Atherosclerosis	
Easy bruising	
Impaired wound healing	
Headaches	
Backache	21–50%
Recurrent infections	
Edema	
Hypokalemic alkalosis	
Acne	11–20%
Hair loss	

1-mg overnight dexamethasone suppression test

Serum cortisol < 1.8 µg/dL - normal response

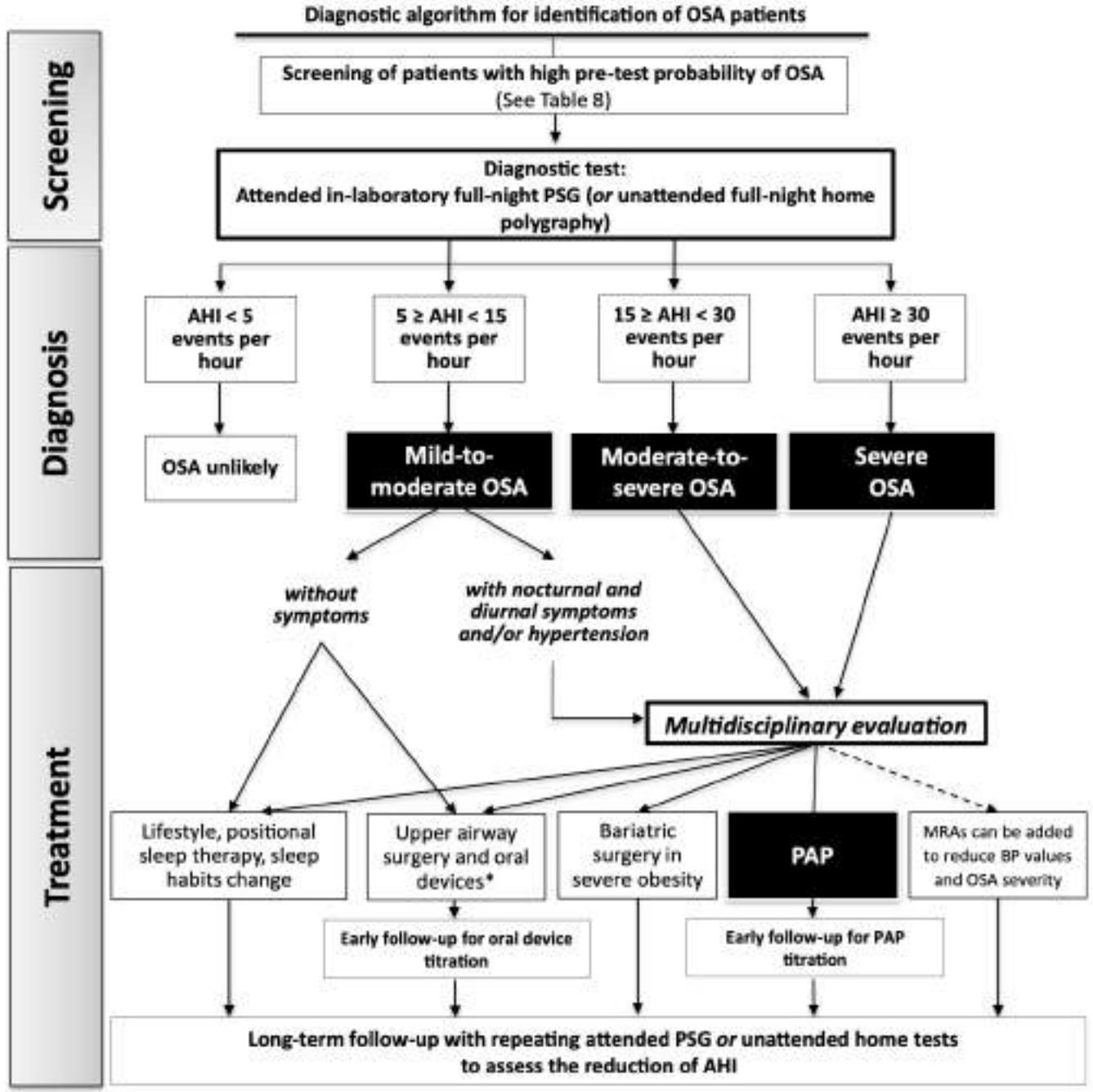
Between 1.8 and 5 µg/dL- subclinical CS.

Serum cortisol >5 µg/dL (or >140 nmol/L) : **CS diagnosis.**



Obstructive Sleep Apnea

OSA is a frequent cause of SH and ‘difficult-to-treat’ HT. Patients with obesity, snoring, upper airway abnormalities, excessive daytime sleepiness, reduced sleep-related quality of life should be screened for OSA. The effect of PAP on BP is still controversial but, if associated with sleep hygiene and specific antihypertensive drugs, it can ameliorate BP control with the largest effect on nocturnal BP


Diagnostic algorithm for identification of OSA patients
Screening
Diagnosis
Treatment
Screening of patients with high pre-test probability of OSA
(See Table 8)
Diagnostic test:
Attended in-laboratory full-night PSG (or unattended full-night home polygraphy)
AHI < 5 events per hour
5 ≥ AHI < 15 events per hour
15 ≥ AHI < 30 events per hour
AHI ≥ 30 events per hour
OSA unlikely
Mild-to-moderate OSA
Moderate-to-severe OSA
Severe OSA
without symptoms
with nocturnal and diurnal symptoms and/or hypertension
Multidisciplinary evaluation
Lifestyle, positional sleep therapy, sleep habits change
Upper airway surgery and oral devices*
Bariatric surgery in severe obesity
PAP
MRAs can be added to reduce BP values and OSA severity
Early follow-up for oral device titration
Early follow-up for PAP titration
Long-term follow-up with repeating attended PSG or unattended home tests to assess the reduction of AHI

To sum up

- The **detection of SH** can be simplified and made cost-effective by estimating the patient's prior **(pre-test) probability** of the disease, which can be established by knowing the **prevalence** of the most common forms and by following few rules

- Once a form of SH has been detected, knowledge of the **underlying pathophysiology** warrants a more rational and more **effective treatment**, which can allow to achieve long-term **cure** of arterial hypertension or a better control of the high BP values when cure is not accomplished.

- These outcomes are rewarding for the doctor, but even more so for the patient, who will avoid the complications of life-long exposure to high BP values, will enjoy a **better quality of life**


THANK YOU