V.N. Karazin Kharkov National University
Faculty of Medicine
Department of Internal Medicine

Case Study of Renovascular Hypertension

Students:
Aws Amer Sulaiman
Anyasi C. Island
Suhaib Al-Shorman

Supervision:
Dr. Bogun
Dr. Yabluchanskiy

Renovascular Hypertension(RVH)

Renovascular hypertension is high blood pressure (greater than 140/80 mmHg) caused by renal artery disease.

Proper kidney function is disrupted, however, when the arteries that provide blood to the kidneys become narrowed, a condition called renal artery stenosis.

When stenosis results in reduced blood-flow, the kidney compensates by producing hormones that increase blood pressure.

This response is a healthy one under normal circumstances. But when the reduction in blood-flow is due to stenosis, blood pressure is increased unnecessarily.

ICD 10 code for (RVH)

Home > 2014 ICD-10-CM Diagnosis Codes > Diseases of the circulatory system I00-I99 > Hypertensive diseases I10-I15 >

Secondary hypertension I15- >

Code Also

- underlying condition

Type 1 Excludes

- postprocedural hypertension (I97.3)

Type 2 Excludes

- secondary hypertension involving vessels of brain (I60-I69)
- secondary hypertension involving vessels of eye (H35.0-)

- I15 Secondary hypertension
  - I15.0 Renovascular hypertension
  - I15.1 Hypertension secondary to other renal disorders
  - I15.2 Hypertension secondary to endocrine disorders
  - I15.8 Other secondary hypertension
  - I15.9 Secondary hypertension, unspecified

ESC Grading of Arterial Hypertension

2013 ESH/ESC Guidelines for the management of arterial hypertension

Table 3
Definitions and classification of office blood pressure levels (mmHg)[a]

Category	Systolic		Diastolic
Optimal	<120	and	<80
Normal	120–129	and/or	80–84
High normal	130–139	and/or	85–89
Grade 1 hypertension	140–159	and/or	90–99
Grade 2 hypertension	160–179	and/or	100–109
Grade 3 hypertension	≥180	and/or	≥110
Isolated systolic hypertension	≥140	and	<90

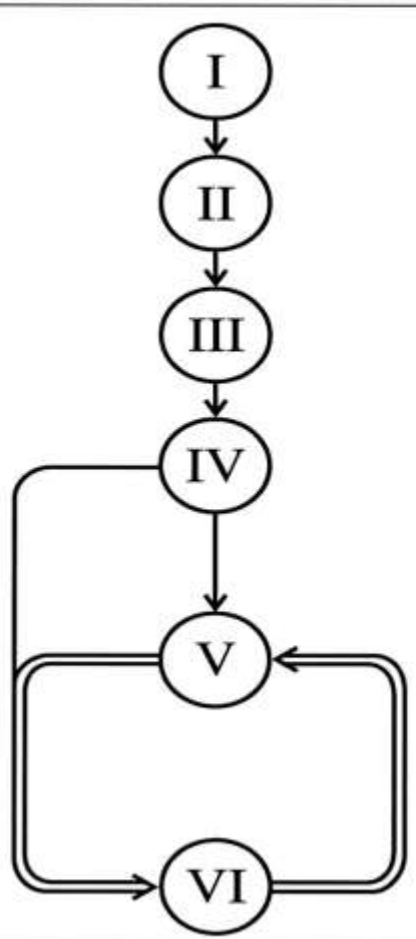
[a]The blood pressure (BP) category is defined by the highest level of BP, whether systolic or diastolic. Isolated systolic hypertension should be graded 1, 2, or 3 according to systolic BP values in the ranges indicated.

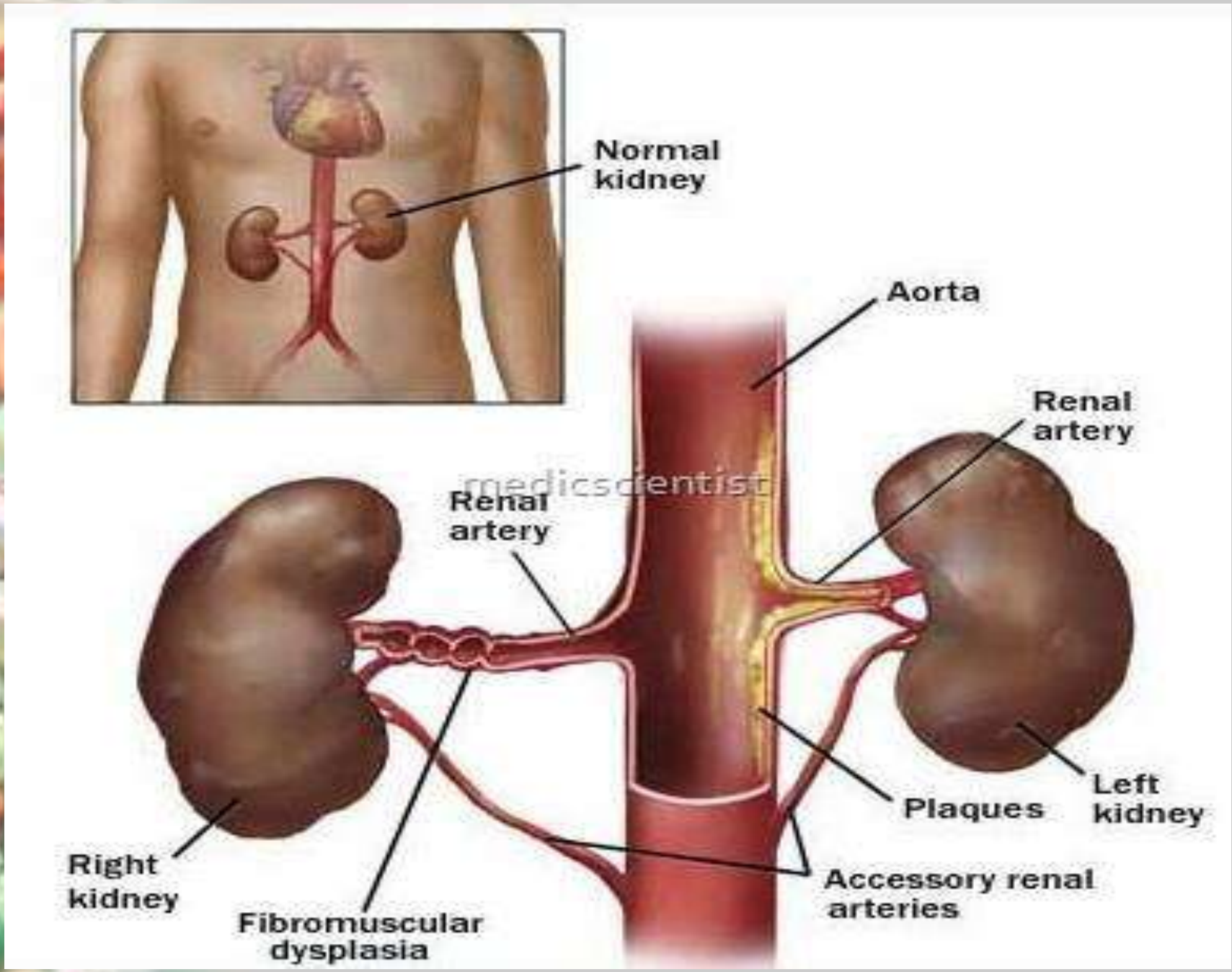
Atherosclerosis

Artherosclerosis, or hardening of the arteries, is a condition in which plaque builds up inside the arteries. Plaque is made of cholesterol, fatty substances, cellular waste products, calcium and fibrin (a clotting material in the blood).

Plaque may partially or totally block the blood's flow through an artery in the heart, brain, pelvis, legs, arms or kidneys. Some of the diseases that may develop as a result of atherosclerosis include coronary heart disease, angina (chest pain), carotid artery disease, peripheral artery disease (PAD) and chronic kidney disease.

Histological classification of artherosclerosis

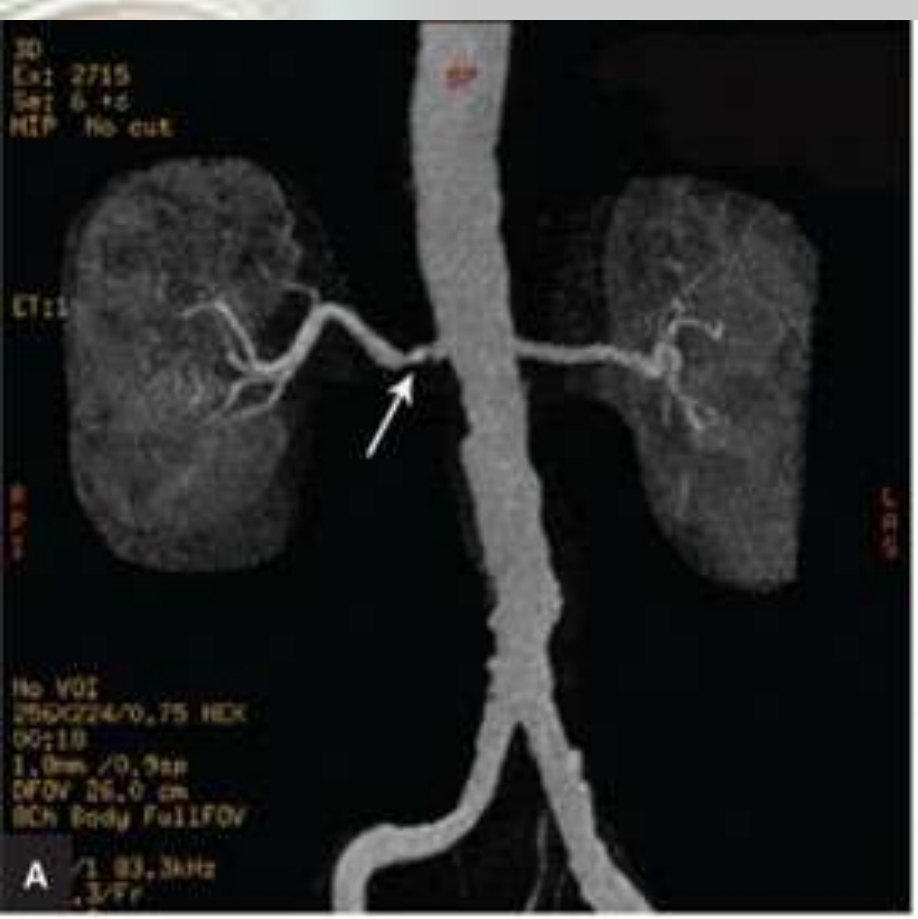
Nomenclature and main histology	Sequences in progression	Main growth mechanism	Earliest onset	Clinical correlation
Type I (initial) lesion isolated macrophage foam cells		growth mainly by lipid accumulation	from first decade	clinically silent
Type II (fatty streak) lesion mainly intracellular lipid accumulation				
Type III (intermediate) lesion Type II changes & small extracellular lipid pools			from third decade	
Type IV (atheroma) lesion Type II changes & core of extracellular lipid				clinically silent or overt
Type V (fibroatheroma) lesion lipid core & fibrotic layer, or multiple lipid cores & fibrotic layers, or mainly calcific, or mainly fibrotic		accelerated smooth muscle and collagen increase	from fourth decade	
Type VI (complicated) lesion surface defect, hematoma-hemorrhage, thrombus		thrombosis, hematoma		


Normal kidney
Aorta
Renal artery
Renal artery
Left kidney
Plaques
Right kidney
Accessory renal arteries
Fibromuscular dysplasia

RVH

High blood pressure caused by renal artery disease may be difficult to control with medication.

The good news is that renovascular hypertension is one of the few identifiable and treatable causes of high blood pressure — a condition that, if left untreated, can lead to heart attack, stroke or kidney failure.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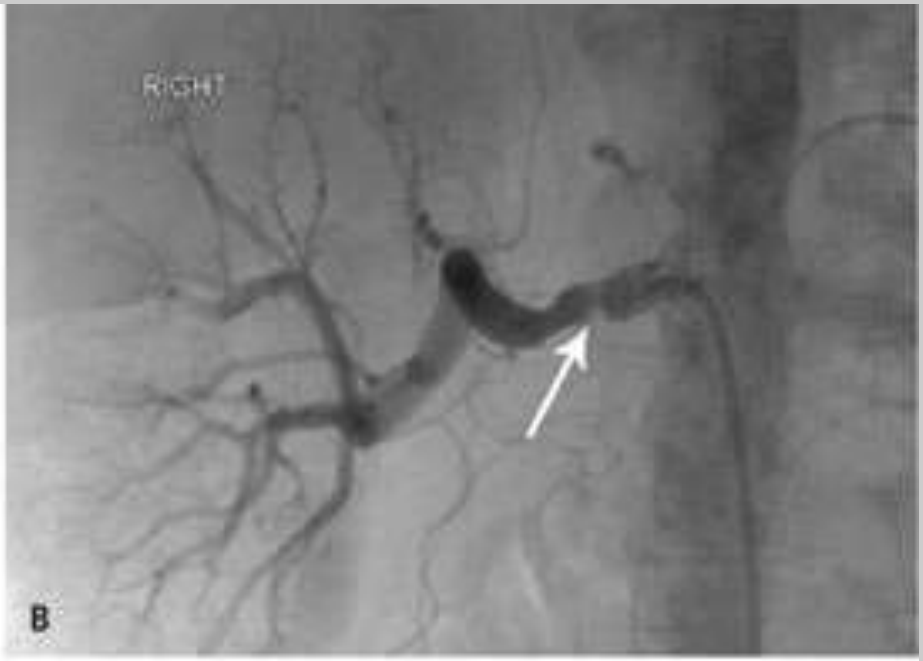
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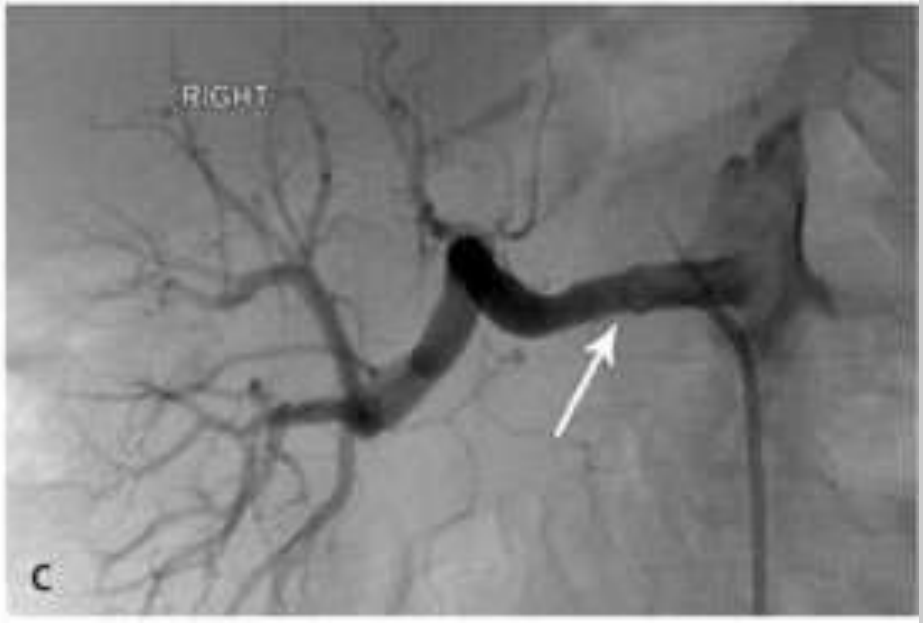
B


RIGHT
C

Epidemiology of Hypertension

Hypertension is a common condition affecting approximately 20% of adults.

- Secondary hypertension (i.e., hypertension with a demonstrable cause) accounts for only 5% to 10% of all cases of hypertension, with the remaining cases considered essential hypertension.
- Renovascular hypertension is the most common type of secondary hypertension and is estimated to have a prevalence between 0.5% and 5% of the general hypertensive population, and it has an even higher prevalence among patients with severe hypertension and end-stage renal disease, approaching 25% in elderly dialysis patients.

Aetiology of RVH

There are varied causes of reduced renal perfusion with resultant renovascular stenosis secondary to either atherosclerotic disease (90%) or fibromuscular dysplasia (10%). Other less common etiologies include;

- vasculitis,
- embolic disease,
- dissection,
- post traumatic occlusion,
- and extrinsic compression of a renal artery

Diagnostic criteria for RVH

Ultimately, the defining criterion for renovascular hypertension is a fall in blood pressure after intervention (angioplasty, intravascular stent placement, or surgery).

Because of the low prevalence of renovascular hypertension among hypertensive patients in general, screening examinations on an unselected population without clinical features suggestive of renovascular hypertension are prone to false-positive results. To improve the predictive value of diagnostic imaging examinations, imaging ideally is performed for those persons having clinical features associated with an increased hypertension

Diagnostic Criteria cont'd

likelihood of renovascular hypertension, such as;

- An abdominal bruit
- Malignant or accelerated hypertension,
- Significant (diastolic pressure >110 mm Hg) hypertension in a young adult (<35 years of age),
- Sudden development or worsening of hypertension,
- Deterioration of renal function in response to angiotensin converting enzyme (ACE) inhibitors, and
- generalized arteriosclerotic occlusive disease with hypertension.

Signs and symptoms of RVH

- Symptoms and Signs

Renovascular hypertension is usually asymptomatic. A systolic-diastolic bruit in the epigastrium, usually transmitted to one or both upper quadrants and sometimes to the back, is almost pathognomonic, but it is present in only about 50% of patients with fibromuscular dysplasia and is rare in patients with renal atherosclerosis.

Signs and Symptoms of RVH

Renovascular hypertension should be suspected if;

- Diastolic hypertension develops abruptly in a patient <30 or>50
- New or previously stable hypertension rapidly worsens within 6 months
- Hypertension is initially very severe associated with worsening renal function or highly refractory to drug treatment
- Abrupt onset of severe(stage II) hypertension(>160/100mmHg in patients older than 55 years
- Severe or resistent hypertension despite appropriately dosed multidrug(>3 agents) antihypertensive therapy
- Negative family history of Hypertension
- Smoking tobacco products
- Recurrent pulmonary edema in the setting of moderate to severe hypertension

Signs and symptoms of RVH

- Abrupt increase in blood pressure over previously stable baseline in patients with previously well-controlled essential hypertension as well as patients with known renal artery stenosis (RAS)
- Acute sustained rise in serum creatinine levels with angiotensin-converting enzyme (ACE) inhibitor therapy
- Unprovoked hypokalemia (serum potassium level < 3.6 mEq/L, often associated with metabolic alkalosis)
- Symptoms of atherosclerotic disease elsewhere in the presence of moderate-to-severe hypertension, particularly in patients older than 50 years
- Moderate-to-severe hypertension in a patient with an unexplained atrophic kidney, significantly asymmetric kidneys (>1.5 cm difference), or diffuse atherosclerosis

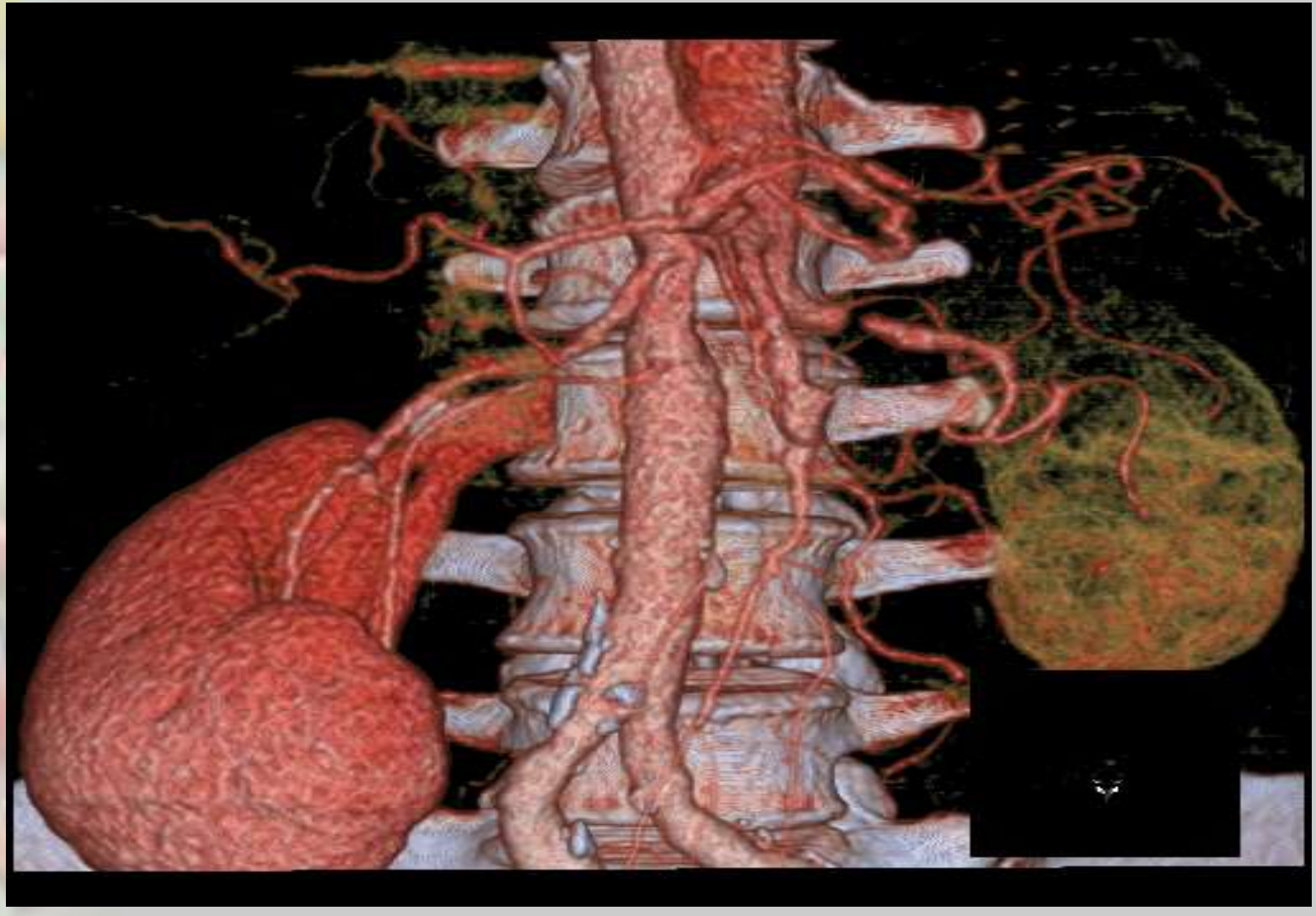
Laboratory Diagnostics of RVH

- Complete blood count (CBC)
- Urinalysis
- Urine culture (all girls, selected boys)
- Serum electrolyte levels (sodium, potassium, chloride, and total carbon dioxide)
- Blood urea nitrogen (BUN) levels
- Serum creatinine levels
- Lipid profile
- Blood glucose and Glucose tolerance test

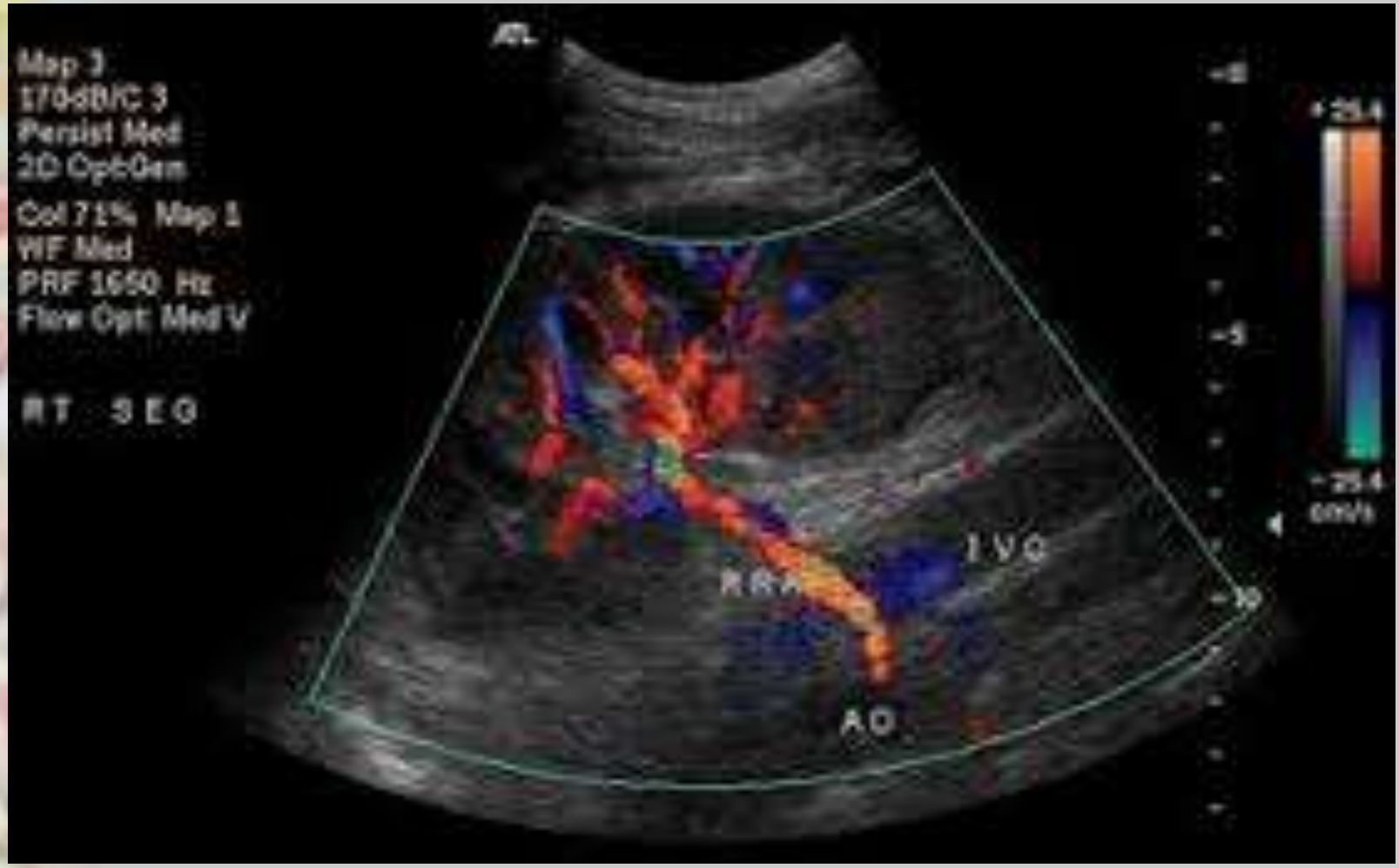
Instrumental Diagnotics of RVH

- Initial identification with ultrasonography, magnetic resonance angiography, or radionuclide imaging
- Confirmation with renal angiography (also may be therapeutic)
- If renovascular hypertension is suspected, ultrasonography, magnetic resonance angiography (MRA), or radionuclide imaging may be done to identify patients who should have renal angiography, the definitive test.
- Renal doppler ultrasound is most affordable method for diagnosis

CT Scan Kidneys and Renal arteries



Doppler Ultrasound of Renal Artery Stenosis



Indications for diagnosis of (RVH)

TABLE 13. Clinical indications and diagnostics of secondary hypertension

	Clinical indications		Diagnostics		
Common causes	**Clinical history**	**Physical examination**	**Laboratory investigations**	**First-line test(s)**	**Additional/ confirmatory test(s)**
Renal parenchymal disease	History of urinary tract infection or obstruction, haematuria, analgesic abuse; family history of polycystic kidney disease.	Abdominal masses (in case of polycystic kidney disease).	Presence of protein, erythrocytes, or leucocytes in the urine, decreased GFR.	Renal ultrasound	Detailed work-up for kidney disease.
Renal artery stenosis	Fibromuscular dysplasia: early onset hypertension (especially in women). Atherosclerotic stenosis: hypertension of abrupt onset, worsening or Increasingly difficult to treat; flash pulmonary oedema.	Abdominal bruit	Difference of >1.5 cm in length between the two kidneys (renal ultrasound), rapid deterioration in renal function (spontaneous or in response to RAA blockers).	Renal Duplex Doppler ultrasonography	Magnetic resonance angiography, spiral computed tomography, intra-arterial digital subtraction angiography.
Primary aldosteronism	Muscle weakness; family history of early onset hypertension and cerebrovascular events at age <40 years.	Arrhythmias (in case of severe hypokalaemia).	Hypokalaemia (spontaneous or diuretic-induced); incidental discovery of adrenal masses.	Aldosterone–renin ratio under standardized conditions (correction of hypokalaemia and withdrawal of drugs affecting RAA system).	Confirmatory tests (oral sodium loading, saline infusion, fludrocortisone suppression, or captopril test); adrenal CT scan; adrenal vein sampling.
Uncommon causes					
Pheochromocytoma	Paroxysmal hypertension or a crisis superimposed to sustained hypertension; headache, sweating, palpitations and pallor; positive family history of pheochromocytoma.	Skin stigmata of neurofibromatosis (café-au-lait spots, neurofibromas).	Incidental discovery of adrenal (or in some cases, extra-adrenal) masses.	Measurement of urinary fractionated metanephrines or plasma-free metanephrines.	CT or MRI of the abdomen and pelvis; 123 I-labelled meta-iodobenzyl-guanidine scanning; genetic screening for pathogenic mutations.
Cushing's syndrome	Rapid weight gain, polyuria, polydipsia, psychological disturbances	Typical body habitus (central obesity, moon-face, buffalo hump, red striae, hirsutism).	Hyperglycaemia	24-h urinary cortisol excretion	Dexamethasone-suppression tests

Pharmacological treatment of RVH

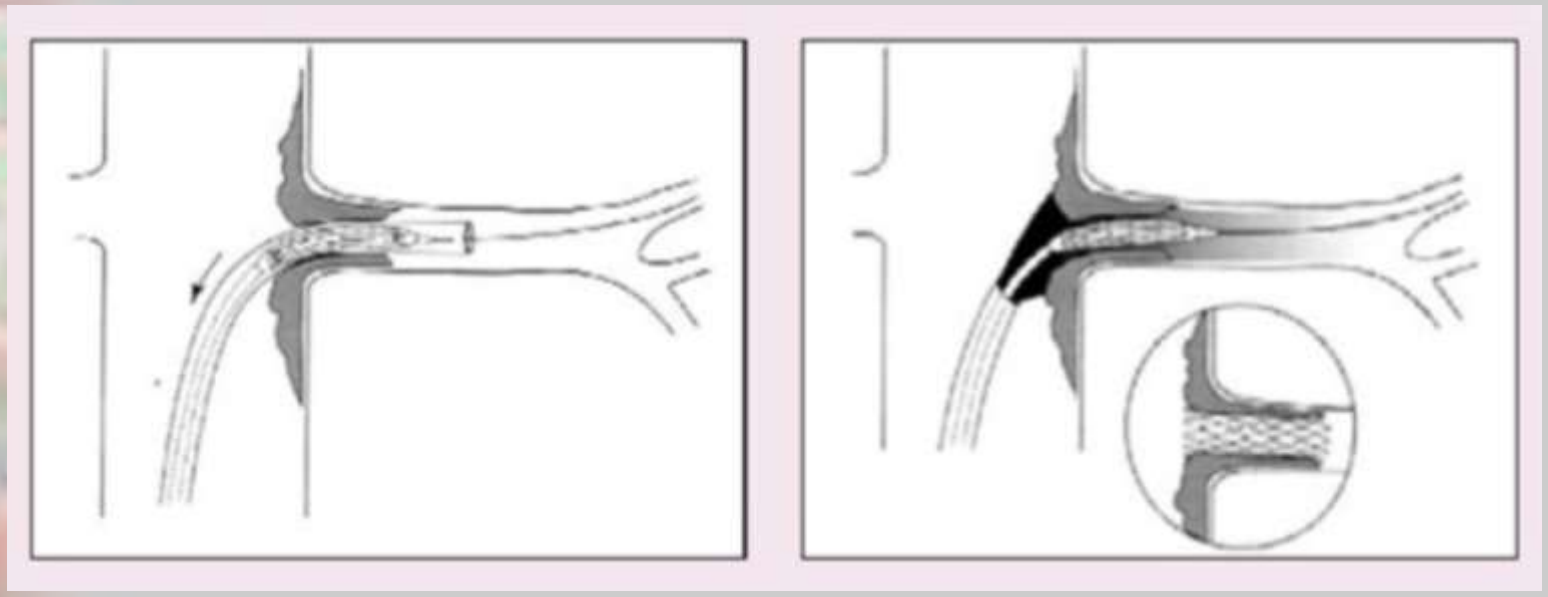
•The obvious goal of treatment of renovascular hypertension is control of blood pressure, and prevention of the long-term sequelae of poorly controlled hypertension, including renal and cardiac failure.

• With the advent of medicines such as angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) control of blood pressure can almost uniformly be maintained. However, because these medications can alter renal blood flow and function, close monitoring of overall renal function is paramount. If control of blood pressure sacrifices renal function, direct renal artery intervention may be warranted

Surgical Treatment of RVH

This may involve percutaneous intervention with a renal stent or an open surgery with a renal bypass or renal artery reconstruction. Lifestyle changes may be recommended, including weight loss, exercise, and dietary adjustments. Stop smoking. Stop drinking alcohol. These habits add to the effects of hypertension in causing complications.

Stenting Procedure



Case study

Patient profile :

- Name; M.V
- Gender; Male
- Age; 64
- Address; Kharkiv region, Kharkiv, Ukraine

Complaints

- Poorly controlled blood pressure since November 2013
- Hot feeling in the head, noise in the ears
- Headache
- Blurred vision and color blindness during episodes of dizziness

Anamnesis Morbi

- From young age registered blood pressure (BP) up to 130/100mmHg.
- BP persistent increase to 160/100mmHg.
- From November 2013 BP persistent increases up to 240/140mmHg - 270/150mmHg. Was treated in Outpatient department with multidrug therapy:
- B-blockers
- Calcium channel blockers
- ACE inhibitors

without improvement and was referred to Central Hospital No 5.

Anamnesis Vitae

- Patient denies any history of chronic diseases; diabetes, rheumatic disease .
- No history of chronic infections; hepatitis B, Tuberculosis, Syphilis.
- There is no family history of premature cardiovascular diseases
- He is a non- smoker and says he does not drink.
- Patient denies eating salty and fatty foods
- Mr is not on any drugs other than his antihypertensive medications
- He denies any history of drug allergies

Physical Examination

Somatic status:

Patient is active and alert, musculoskeletal system is well developed. Xanthelazma palbebrarum on left upper eyelid. Auscultation of breath sounds is without abnormalities. Heart activity is rhythmic, heart sounds are muted. Pulse 78bpm, Blood pressure 200/90mmHg. Abdomen enlarged because of increased fat. Liver is located at the subcostal margin and is not tender.

Systolic bruit above projection of right renal artery. Physiological functions are normal. Negative Pasternatsky sign on both sides, no edema of the legs.

Risk factors for CVD: arterial hypertension, signs of hyperlipidemia, excess body weight.

Plan of Management

Basic investigations

- Full Blood Count
- Blood Urea, Electrolytes and Glucose
- 12 leads Electrocardiogram
- Lipid Profile
- Heart ultrasound

Additional investigations

- Test for Syphilis and Helminths
- Stress test
- Renal artery angiography
- Ophtamologist Consultation
- Endovascular Surgeon Consultation

Complete Blood Count (CBC)

Hemglobin	153	120-140(g/l)
Erythrocytes	5.11	3.9-4.7(T/l)
Color index: 0.89	0.89	0.85-1.15
Leucocytes: (4.0-9.0 g/L)	7.3	4.0-9.0 (g/l)
Neutrophils: 45.1	45.1	47-72%
Eosinophils:	9	(0.5-5.0%)
Basophils	0.7	(0-1.0%)

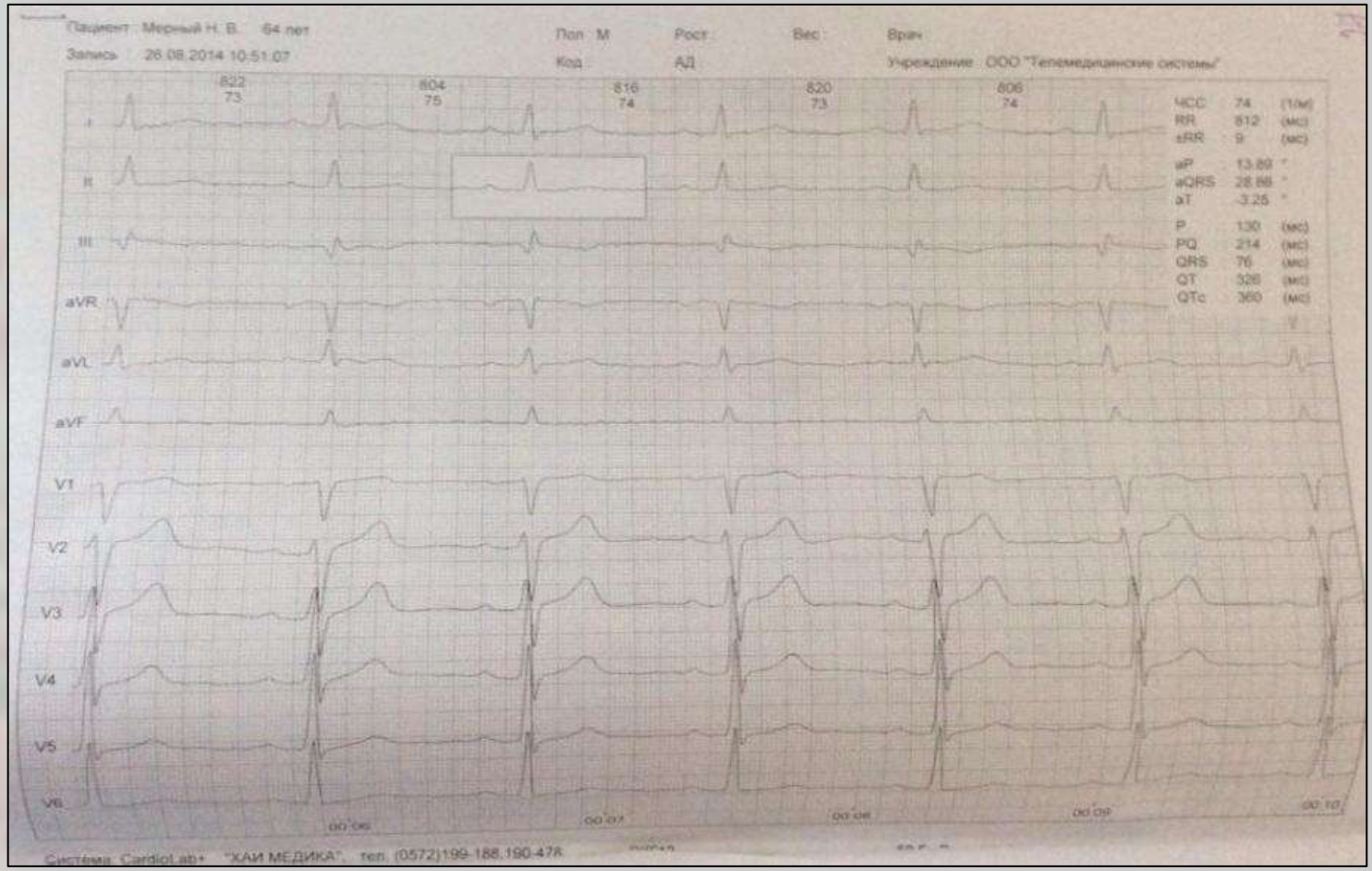
CBC cont'd

Lymphocytes	42.3	(19.0-37.0%)
Monocytes	7.0	(3-11%)
Thrombocytes	196	(180-320 g/L)
Hematocrit	64.9	(36-42%)
Urinalysis Specific gravity: pH: 6.5 (5-7) Protein Glucose	 1.018 6.5 - -	 (0.001-1.040) (5-7) - -

Lab test

Blood biochemistry 27-08-14		
Glucose	8.93	4.2-6.1(mmol/l)
Creatinine	145	53-97(mmol/l)
Blood investigation by ELFA 2		
HbsAg	Negative	
HBCT	Negative	
HCV	Negative	
Glycylated Hb	5.41	4-6%

Electrocardiogram



Sinus rhythm, heart rate 74, left axis deviation, hypertrophy of the myocardium of the left ventricle

Cardiac Ultrasonography

1- Acoustic Window	Normal
2- Aorta	D.; 34.2 mm (normal (20-37 mm). Aortic walls are of increased echo density with a folded valve.
Ascending Aorta	D; 39.3 mm – widened
3- Aortic Valve	Ampl. of opening of leaflets; 15.8 (17-26mm) regurgitation findings; Physiologic. Vmax; 205.5 cm/sec. P.G. max 16.9 mmHg. P.G. avr.; 4.5 mmHg.
4- Left Atrium	Anteroposterior size; 32.2 (21-37). Leaflets of the Mitral Valve are of increased echo density and thickened. Movements are of different directions.
5- Mitral Valve	Ampl. of opening of leaflets; 29.2 (26-35) VmaxE; 76.6 cm/sec VmaxA; 91.1 cm/sec. P.G. maxE; 2.3 mmHg. P.G.maxA; 3.3 mmHg.
6- Speed of the Diastolic Opening	69.0 (50-180 cm/sec). regurgitation findings; Physiological. Mitral-Septal (E-septum); 5.9 (0-10 mm).
7- Left Ventricle	End-Diastolic pressure; 36.3 (35-55 mm). End-Systolic pressure; 32.9 (23-38 mm). Posterior wall of Left Vent.; 16.2 (6-11mm) EF; 56% (55-78%). FS; 29% (28-44%). Ampl. of post. Wall. Of LV; 8.6 (7-13 mm). LV cavity – not widened. Contraction – normokinetic. Presence of aneurism – N/A.

Cardiac Ultrasonography cont'd

8- Interventricular Septum	**Thickness; 16.0 (6-11 mm).** **Ampl. of movement; 4.6 (4-10mm).**
9- Right Ventricle	D.; 35.4 mm (9-26 mm). Thickness of the wall 6.0 (3-6 mm).
10- Interauricular Septum	normal
11- Tricuspid Valve	movement of leaflets are of different directions. regurgitation; 1 leaflet.
12- Pulmonary Valve	movment of leaflets are of different directions. regurgitation; 1 leaflet. Avr. P. PA; 21.7 mmHg. Wave ampl. 4.1 (4-10 mm). Vmax; 80.3 cm/sec. P.G. max; 2.6 mmHg. P.G. med; 0.6 mmHg.

Cardiac Ultrasonography cont'd

CONCLUSION

- Sclerotic changes in the walls of the aorta, Leaves of aortic and mitral valve. Dilatation of the ascending aorta.
- Mild atherosclerotic aortic stenosis.
- Hypertrophy of left ventricle (concentric type).
- Increased diastolic stiffness of the myocardium of the left ventricle.
- Tricuspid regurgitation 1st stage
- Pulmonary valve regurgitation 1st stage
- Signs of pulmonary hypertension 1st stage.

Treadmill Stress test

- Maximum oxygen consumption 7.0 mets.
- No signs of coronary insufficiency according to effort. Test is negative.
- Although there was no evidence for the need to provide the treadmill stress test, but it was carried out anyway.

Preliminary Diagnosis

- Atherosclerotic disease with peripheral artery involvement
- Right renal artery stenosis
- Renovascular hypertension stage II, Level 3
- Heart failure stage IIA, FC II

Stratification of total CV risk in categories of low, moderate, high and very high risk according to SBP and DBP and prevalence of RFs, asymptomatic OD, diabetes, CKD stage or symptomatic CVD. Subjects with a high normal office but a raised out-of-office BP (masked hypertension) have a CV risk in the hypertension range.

Other risk factors, asymptomatic organ damage or disease	Blood Pressure (mmHg)			
	High normal SBP 130–139 or DBP 85–89	Grade 1 HT SBP 140–159 or DBP 90–99	Grade 2 HT SBP 160–179 or DBP 100–109	Grade 3 HT SBP ≥180 or DBP ≥110
No other RF		Low risk	Moderate risk	High risk
1–2 RF	Low risk	Moderate risk	Moderate to high risk	High risk
≥3 RF	Low to Moderate risk	Moderate to high risk	High Risk	High risk
OD, CKD stage 3 or diabetes	Moderate to high risk	High risk	High risk	High to very high risk
Symptomatic CVD, CKD stage ≥4 or diabetes with OD/RFs	Very high risk	Very high risk	Very high risk	Very high risk

BP = blood pressure; CKD = chronic kidney disease; CV = cardiovascular; CVD = cardiovascular disease; DBP = diastolic blood pressure; HT = hypertension; OD = organ damage; RF = risk factor; SBP = systolic blood pressure.

Lifestyle Change

Modification	Recommendation	SBP ↓
Weight reduction	BMI 18.5-24.9 kg/sq m	50-20 mm Hg/10kg
DASH eating plan	Fruits, vegetables, low fat dairy products	8-14 mm Hg
Na restriction	2.4 gm sodium or 6 gm NaCl/day	2-8 mm Hg
Physical activity	Brisk walking (at least 30 min/day)	4-9 mm Hg
Avoidance of tobacco	-	-
Moderation of alcohol consumption	No more than 2 drinks/day	2.5-4 mm Hg

Braunwald's Heart Disease, 2005

Medication' treatment

Received in hospital	Doctor's recommendation	Our recommendation
1. Bisoprolol (5mg)	• Lorista 100mg in morning	Atorvastatin 20 mg od after supper (titration up to target levels of LDL-C 1,7 mmol/L)
2. Losartan (25mg)	• Korenfar 40mg evening	Losartan 25 mg od at bedtime
3. Nifedipine (10mg)		Bisoprolol 5 mg in the morning (target HR=60/min)
4. Betaxolol (lorken) (20mg)	• Lorken 10mg in morning	Aspirin 100 mg od
5. Pantprozole (nolpaza) (40mg)	• Nolpaza 40mg at night 2 weeks, small doses as needed for epigastric pain	Clopidogrel 75 mg od

Renal Angiography and Stenting

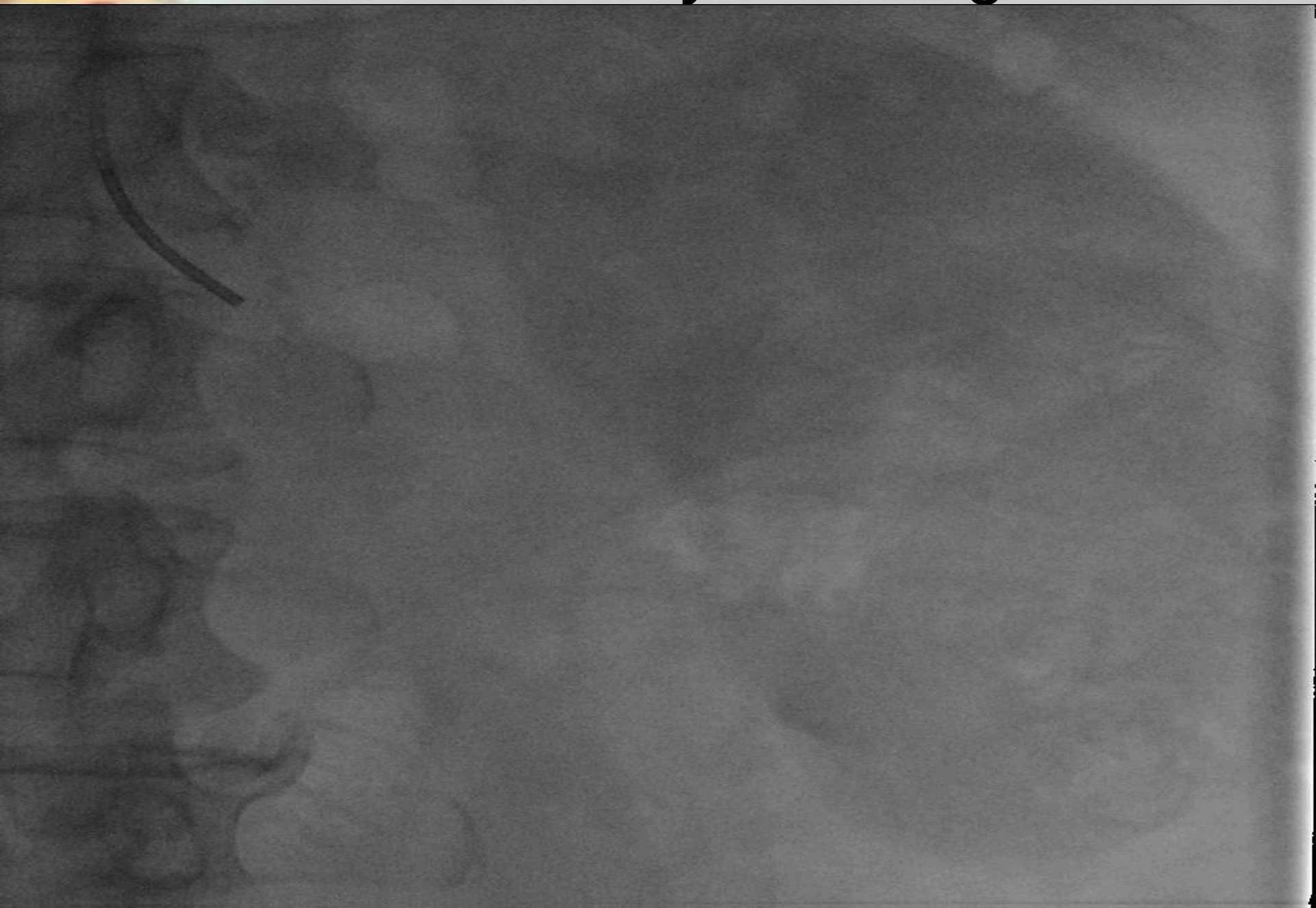
- Angiography of renal arteries

Renal arteries exit at the level of L1.

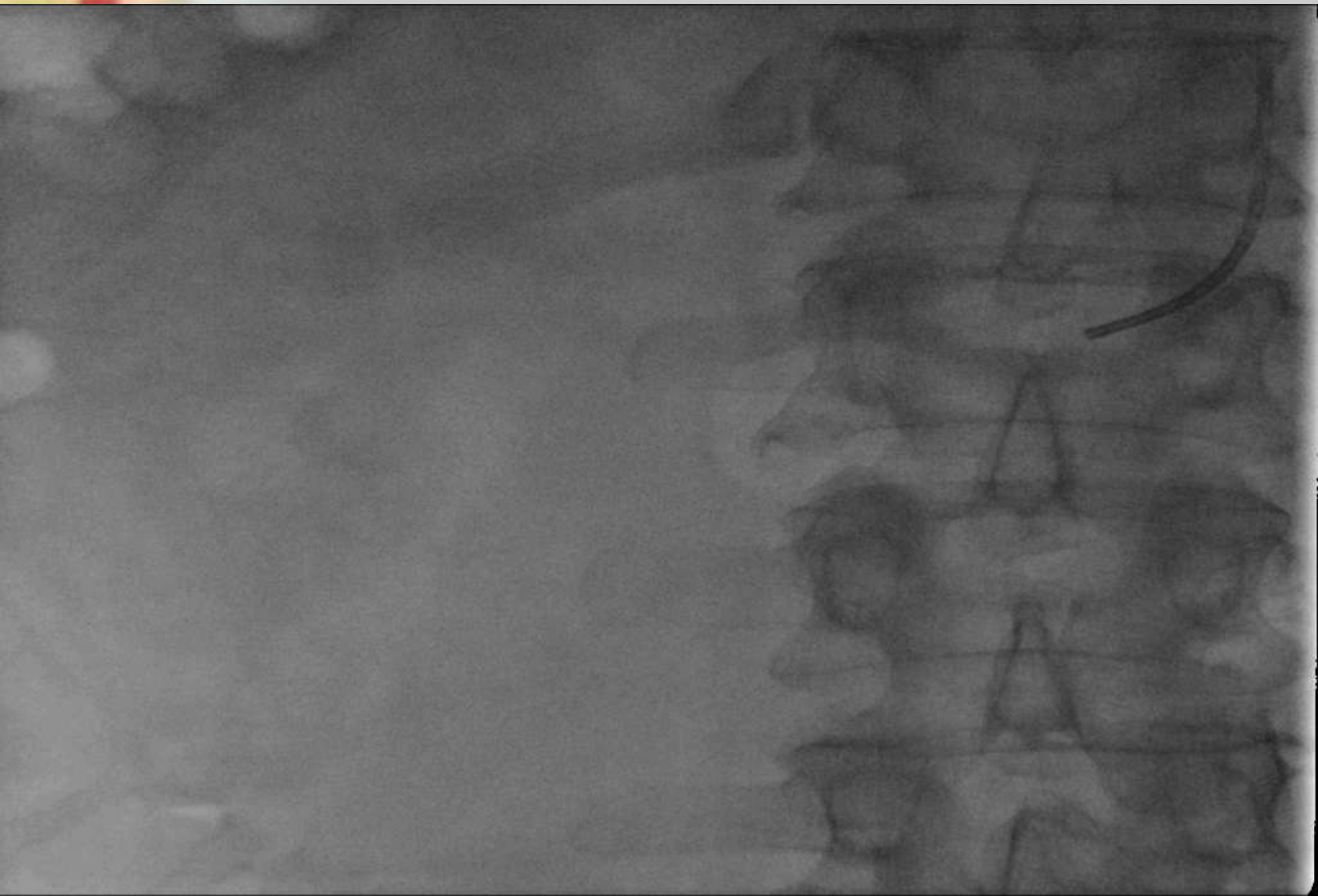
Left renal artery without abnormalities.

80% stenosis of the right renal artery. Stenting of right renal artery was performed (tsunami peripheral) 5.0x18mm.

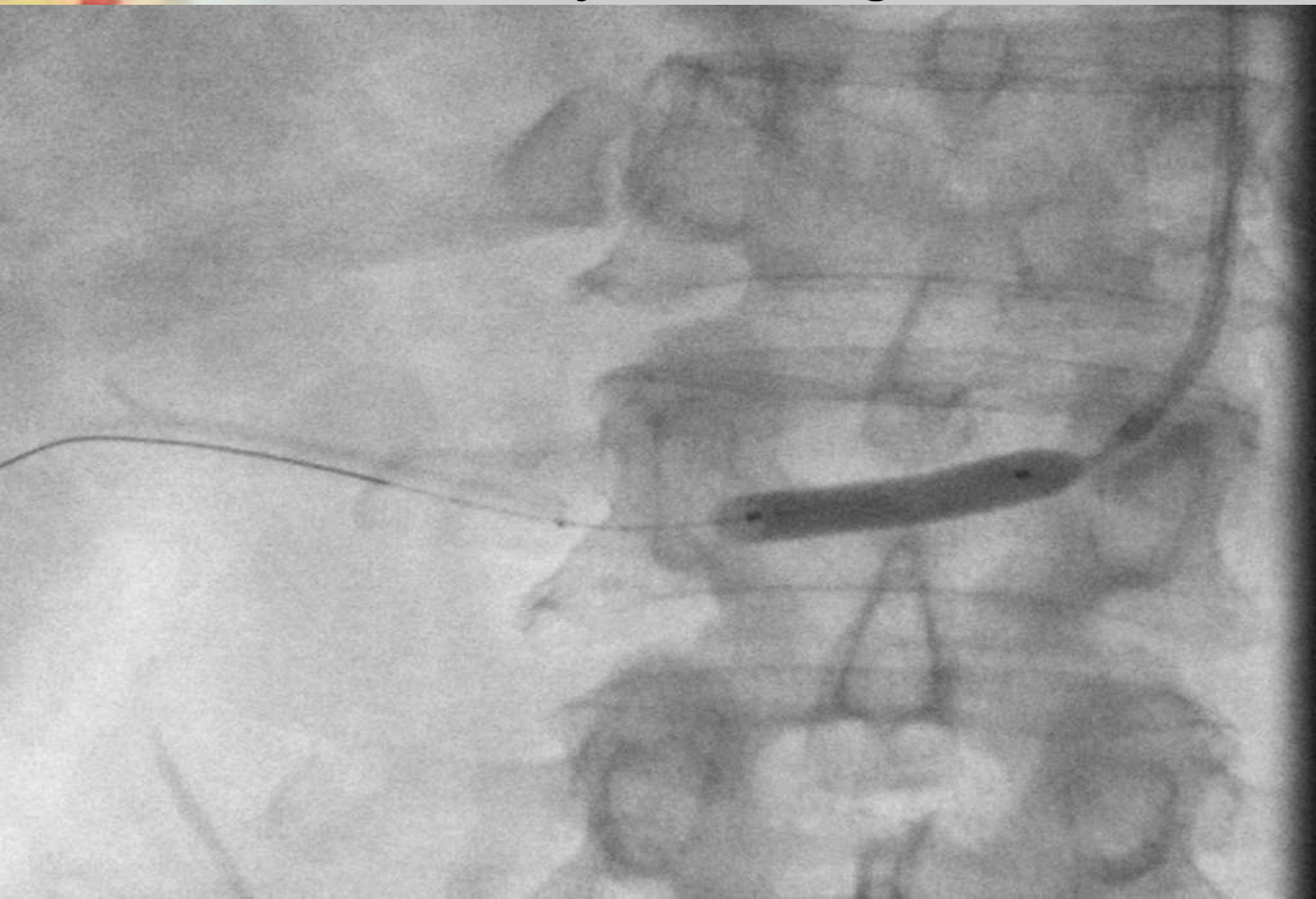
Renal Artery Stenting



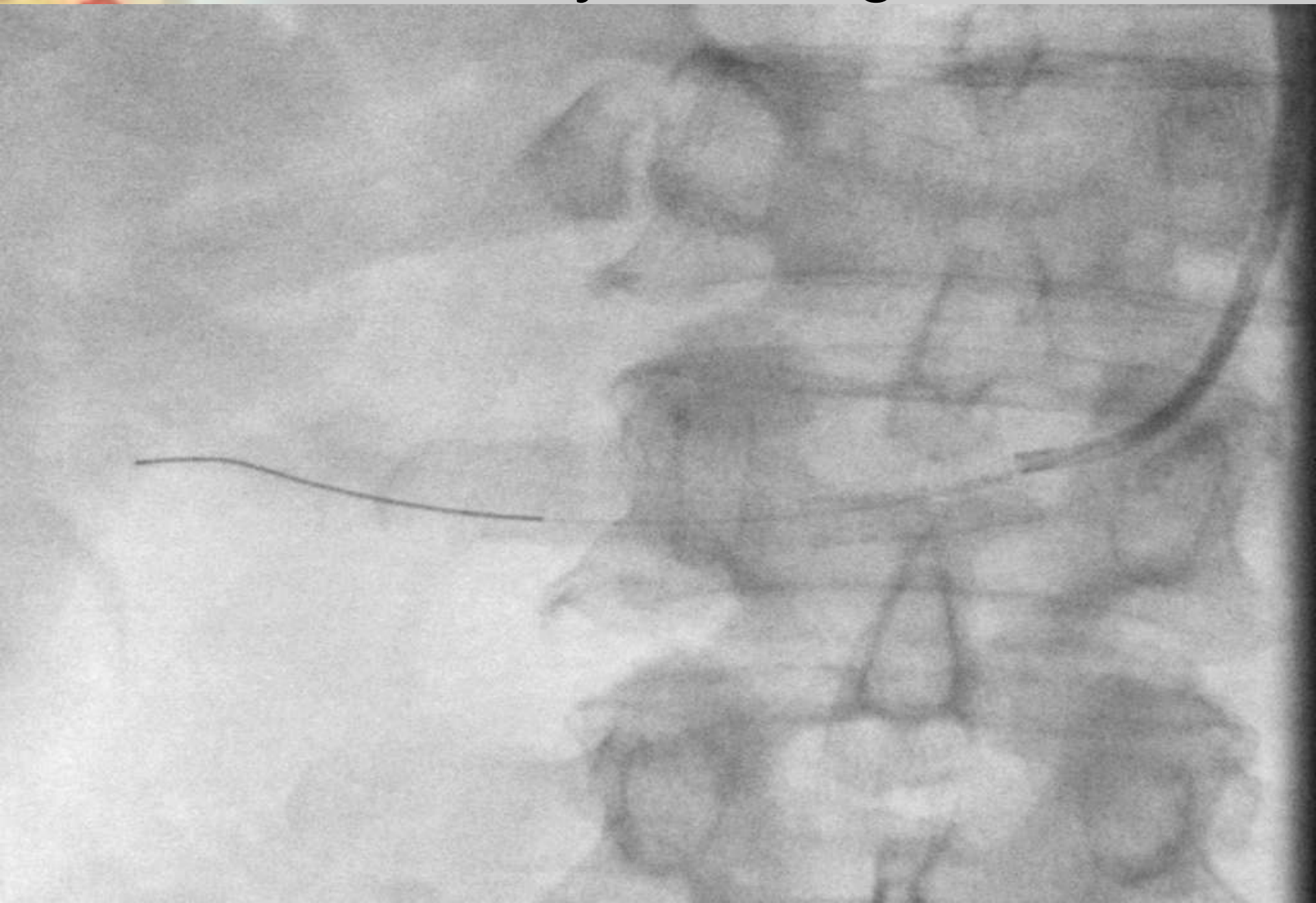
Renal Artery Stenting cont'd



Renal Artery Stenting cont'd



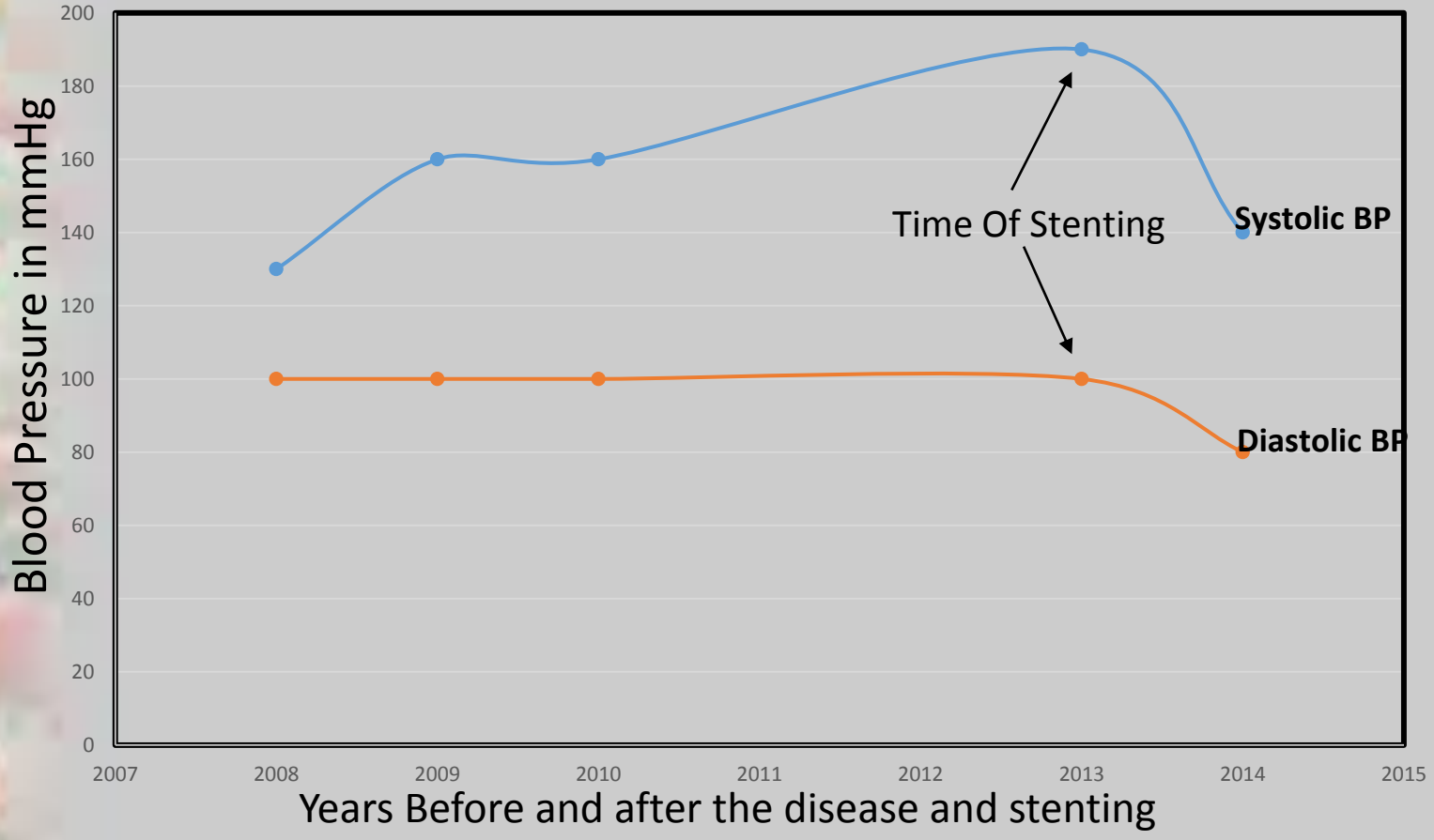
Renal Artery Stenting cont'd



Treatment

- Condition
- Improved, pressure was lowered to 120-140/80mmHg with the help of the medication. Periodic bouts of dizziness.Unfit for work because of the renal artery stenting. Discharged but indicated for followup.

Changes in BP of Mr M.V



Final Diagnosis

Atherosclerotic disease. Atherosclerosis of aorta. Mild aortic valve stenosis of atherosclerotic genesis. Occlusive (80%) atherosclerosis of the right renal artery (angiography of renal arteries 28/08/2014). Condition after stenting of the right renal artery (28/08/2014).
Secondary renovascular hypertension stage II, Level 3, Total Cardiovascular Risk Very High.
Hypertensive heart (LVH).
Heart failure stage IIA, with preserved systolic function of the left ventricle, II Functional Class

Peculiarities of the Given Clinical Case.

In this case we can see so called vicious circle:

Arterial hypertension as well-known potent risk factor promoted accelerated progression of atherosclerosis.

Atherosclerosis involved renal artery with its occlusion and development of secondary renovascular arterial hypertension yet.

Secondary hypertension as more resistant to therapy further increased the total cardiovascular risk and accelerated progression of atherosclerosis

Possibly timely and aggressive correction of cardiovascular risk (first of all optimal control of BP, hypolipidemic therapy with statins) would be able to slow down the progression of atherosclerosis and even to prevent the development of secondary hypertension

Prognosis

Untreated atherosclerotic RVH will likely progress to a greater degree of stenosis within 2 years. Typically, it progresses at a rate of 7% per year. It is also likely to become bilateral, impair renal function, and develop into ischemic nephropathy, pulmonary edema, or congestive heart failure. Pulmonary edema and congestive heart failure are more likely with bilateral disease.

Studies indicate that atherosclerotic renovascular hypertension treated with revascularization surgery or angioplasty has a cure rate of 15% to 44%, improvement rate of 50% to 75%, failure rate of 21%, and with follow-up of 8 to 9 years, cure or improvement rate of 79% (Novick 253). Likelihood of cure or improvement is greater with unilateral disease.

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