



From simple to complex reaction in hypertension emergency

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Disclosure

Nothing to disclose

Different BP levels!

M/28 y – CVI, dissection a. vert.
160/100 mmHg
Her usuall BP 138/85 mmHg



F/37 y – aortic dissection
270/130 mmHg

Her usuall BP 160/100 mmHg

M/52 y – kidney failure,
TIA

190/110 mmHg

His usuall BP 150/90 mmHg

F/93 y – NSTEMI,
Pulomonray oedema
175/75 mmHg

His usuall BP 130/60 mmHg

Case 1: Outpatient clinic

- 28yrs old woman

With..

- In several occasions at clinical visit with family doctor BP 142/86mmHg
- Shortness of breath on severe exertion for 2 months
- Diabetes for 1 year, on diet
- No other symptoms

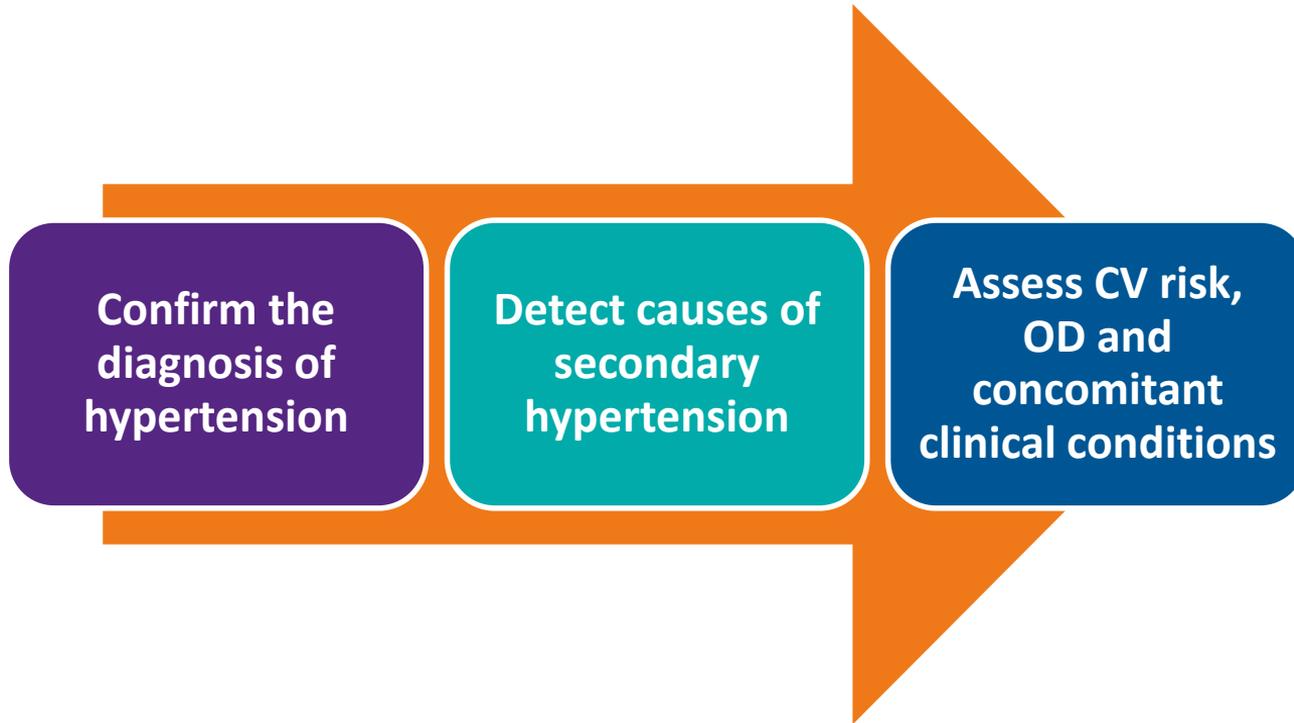
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Hematološke preiskave				
Preiskava	Rezultat	Referenčni	Enota	Opomba
K-SR	H 24	do 15	mm/h	
K-Levkociti	7.5	4.0 - 10.0	10 ⁹ /L	
K-Eritrociti	4.3	4.2 - 6.3	10 ¹² /L	
K-Hemoglobin	L 101	120 - 180	g/L	
K-Hematokrit	L 0.32	0.37 - 0.54	l	
K-MCV	L 74	81 - 94	fl	
K-MCH	L 23	26 - 32	pg	
K-MCHC	317	310 - 350	g/L	
K-RDW	H 0.149	do 0.145	%	
K-Trombociti	340	140 - 340	10 ⁹ /L	
K-DKS- aparat				
K-nevtrofili	50	40 - 70	%	
K-limfociti	38	20 - 60	%	
K-eozinofili	10	0 - 10	%	
K-bazofili	1	1 - 6	%	
K-bazofili-a	1	0 - 1	%	
K-nevtrofili-a	3.71	1.60 - 7.50	10 ⁹ /L	
K-limfociti-a	2.83	0.80 - 5.00	10 ⁹ /L	
K-monociti-a	0.78	0.08 - 1.00	10 ⁹ /L	
K-eozinociti-a	0.05	0.00 - 0.10	10 ⁹ /L	
K-bazofili-a	0.05	0.00 - 0.10	10 ⁹ /L	

Patient history

- **Family history:**
 - **Father: hypertension, hyperlipidemia, CVI at the age of 54**
 - **Mother: myocarditis at the age of 32**
 - **Grandmother: diabetes mellitus type 2, carcinoma uteri**
- **Past history:**
 - **Op. due to apendicitis 5y ago**
- **Allergies: amoxicillin with clavulanic acid**
- **Social history: not smoking, denies alcohol abuse**

Evaluation of patient with hypertension:



Physical examination Treatment??

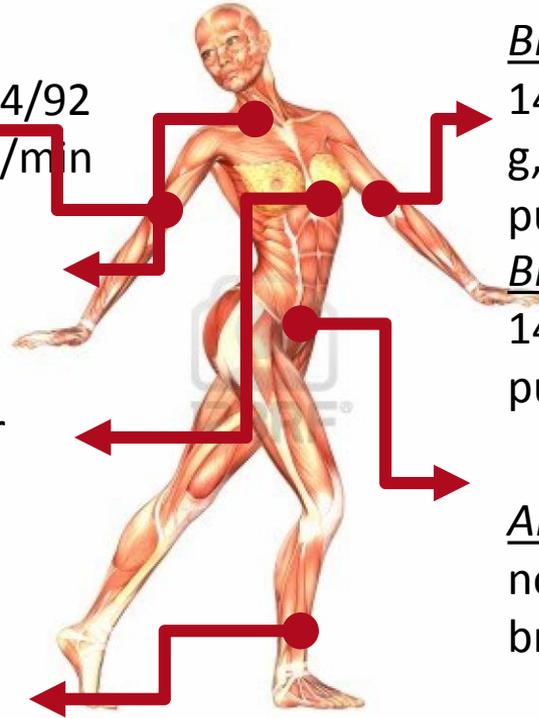
BMI=29 kg/m²

BP right arm: 144/92
mmHg, pulse 76/min

Neck: no bruits

Heart: no murmur

Extremities: no edema,
normal palpable
peripheral pulses



BP left arm:
142/93mmH
g,

pulse 71 bpm

BP on standing:
144/90 mmHg,
pulse 89/min

Abdomen:
no abdominal
bruit

Complex reaction in hypertension emergency

- **Many** of patients who present to the Emergency Department (ED) may have elevated blood pressures recorded during their visit. This can be due to a **number of reasons** including pain, anxiety, missing their normal anti-hypertensive medications and "white coat hypertension".
- No current evidence based or guideline recommendations exist for a **blood pressure cut-off** at which point an asymptomatic patient must be acutely treated.
- So who can we **safely send home** and who needs more urgent treatment? The concern is not so much with the absolute numbers of the blood pressure readings but the **effects** of severe hypertension on the **rest of the body**. Patients with severe hypertension but no organ damage are often considered to be hypertensive urgencies rather than emergencies. They will require commencement of oral anti-hypertensive agents or alteration of existing regimen but can usually be discharged home with appropriate follow-up.
- Hypertensive emergencies will usually require **intravenous anti-hypertensive** therapy aiming for a 25% reduction in blood pressure over 1-4 hours.



ESC

European Society
of Cardiology

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POSITION PAPER

ESC Council on hypertension position document on the management of hypertensive emergencies

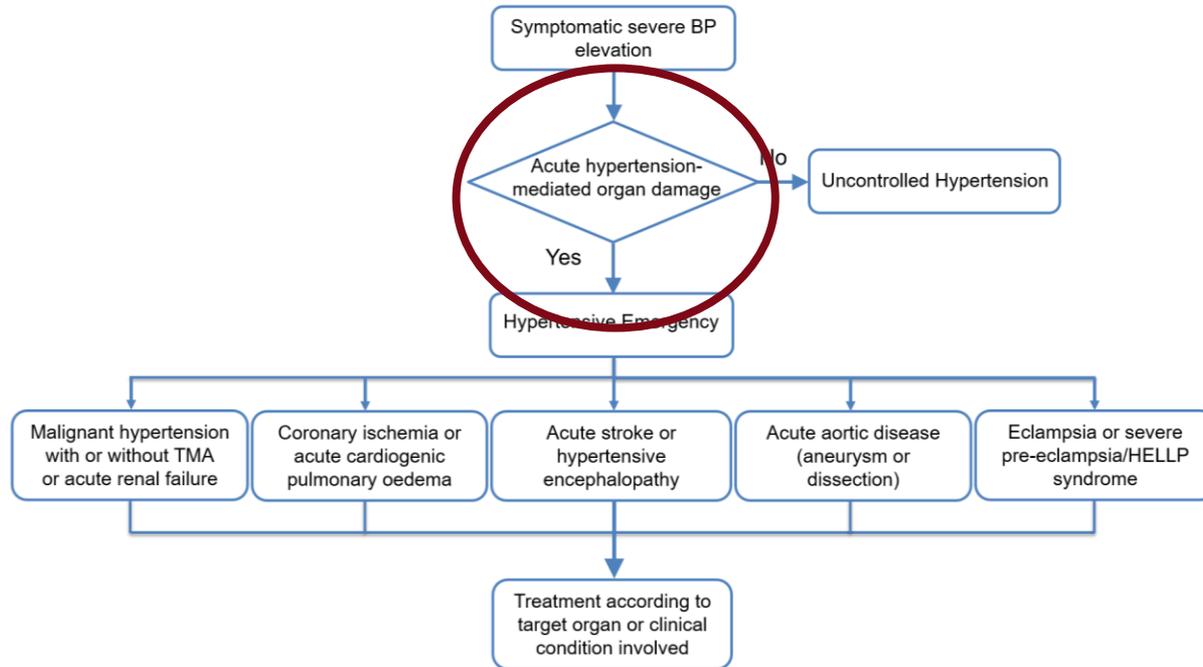
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doi:10.1093/ehjcvp/pvy032

Definition of Hypertensive Emergencies

- **Very high BP values associated with acute hypertension-mediated organ damage**
- **Key target organs: heart, retina, brain, kidneys, and large arteries**
- **Immediate BP reduction required to limit extension or promote regression of target organ damage**
- **The type of target organ damage is the principal determinant of the choice of treatment, target BP, and timeframe by which BP should be lowered**

Stratification of hypertensive emergencies according to the condition or target organ involved



Important !

- **Patients with a hypertensive emergency should be admitted for close monitoring and, in most cases, treated with intravenous BP-lowering agents to reach the recommended BP target in the designated time-frame.**
- **Patients that have no hypertensive emergency can usually be treated with oral BP-lowering agents and usually discharged after a brief period of observation.**

Epidemiology

- **Coding differences among institutions**
- **One in every 200 patients presents at the emergency department with a suspected hypertensive emergency**
- **Despite improved treatment for hypertension in the past decades, the incidence of hypertensive emergencies has not declined**
- **Acute pulmonary oedema, stroke, myocardial infarction, heart failure, acute aortic dissection, acute renal failure and hypertensive encephalopathy.**

Pathophysiology

- Many pathophysiological mechanisms are involved in the development of hypertension emergencies, but the initiating events for the sudden escalation in BP are not completely understood.
- Among patients presenting at the Emergency Dept. with malignant hypertension, secondary causes can be found in 20-40% and most often consist of **renal parenchymal disease** and **renal artery stenosis**, whereas endocrine causes appear to be rare.
- The majority of patients with malignant hypertension have **unrecognized or uncontrolled** essential hypertension.

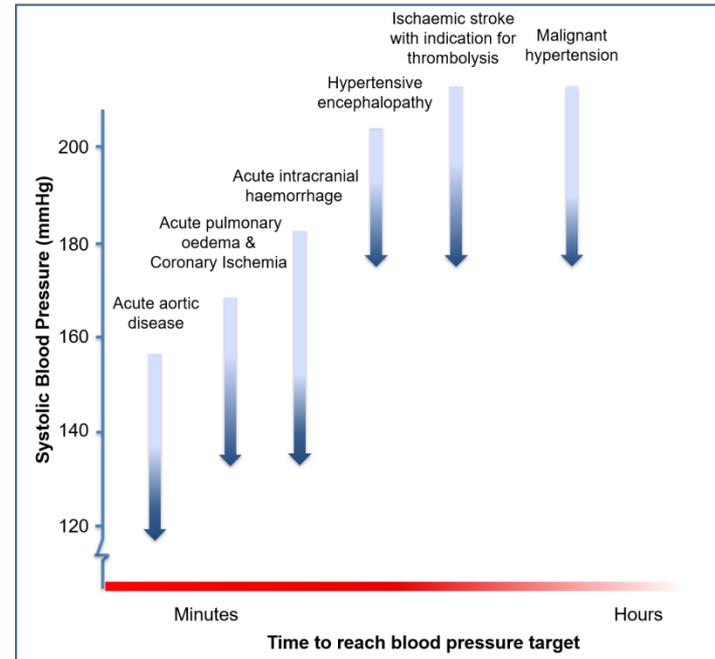
Acute HT mediated organ damage

Aorta – dissection, aneurysm

Heart – MACE, acute pulmonary oedema

Brain – stroke, hypertensive encephalopathy

Retina & Kidneys – malignant hypertension



Case 2

- **39 y old lady admitted to the emergency department due to chest pain which started in late afternoon and escalated till evening so relatives called an ambulance.**
- **At the admission she described her pain as sharp abrupt onset in her chest, tearing character radiating towards back**
- **Few days before her blood pressure was not well controlled and she measured SBP around 200mmHg**

Examination list:

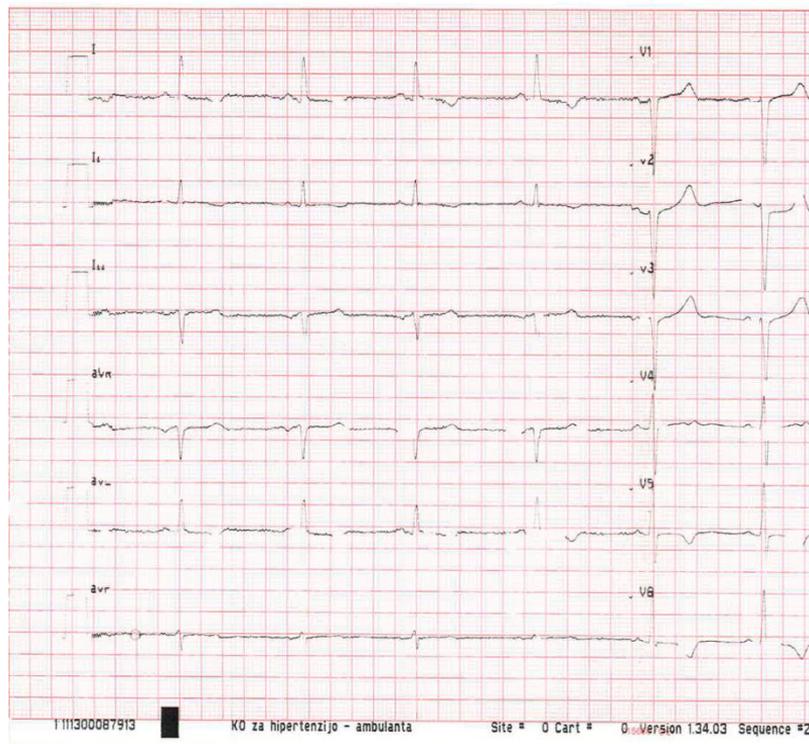
- History
- Physical examination
- Haemoglobin, platelet count
- Creatinine, GFR, sodium, potassium, lactic dehydrogenase (LDH), haptoglobin
- Quantitative urinalysis for protein, urine sediment for erythrocytes, leukocytes, cylinders and casts
- ECG
- Chest X Ray

- **Family history:** Grandfather died because of CVI, father survived CVI and is treated for AH, grandmother is treated for DM and AH
- **Past history:** DM for few years on diet, chronic kidney disease stg 4 for 3 y, extreme obesity, hypertensive retinopathy
- **Present history:** Treated for hypertension for 4 years, she delivered 3 times, 2 times normal pregnancy, 1 spontaneous abortion and 1 on hypertensive therapy
- She was obese since her childhood, BMI in y2017 was 50.1 (163cm, 133kg)
- She was tested for secondary hypertension but was not discovered. Was hospitalised many times and often her SBP was measured even 280mmHg
- On medications: bisoprolol 10mg in the evening, furosemide 40mg , amlodipine 5mg, perindopril 4mg, moxonidine 0.3mg
- Smoker 10y 30 cig/day, now 10 cig/day

Physical examination

- Extreme obesity, BMI 43
- On admission, her oxygen saturation was 92%
- BP 250/130mmHg D arm, 220/112mmHg L arm, HR 74/min
- Jugular venous pressure was normal
- Lungs: some inspiratory crackles were heard at pulmonary basis
- Heart: rhythmic action fr 74/min, no murmurs
- No abnormalities in abdomen
- No peripheral oedema

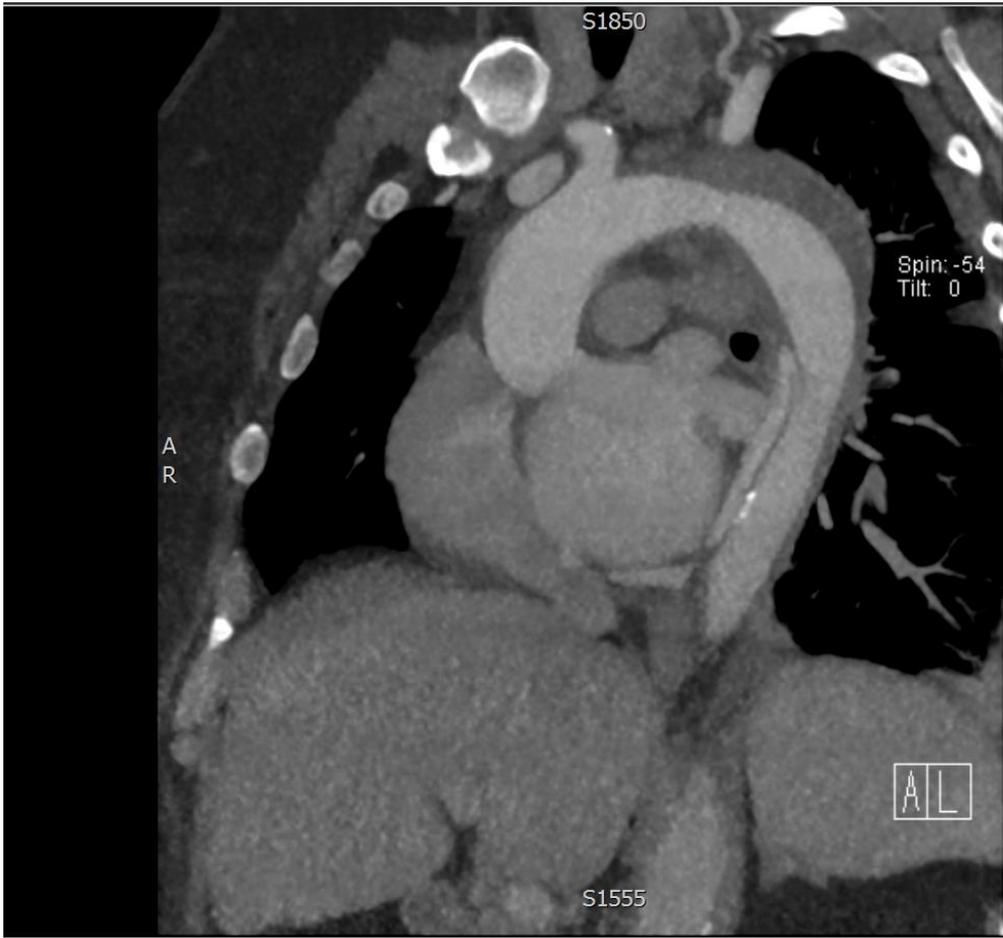
variable	result	variable	result
RBC	4.2 (10 ¹² /L)		
WBC	9.7 (10 ⁹ /L)	pH	7.39
Hb	120 g/L	pO ₂	11.1 kPa
Creatinine	308 μmol/L (3.5 mg/dL)	pCO ₂	5.2 kPa
GFR	16 (CKD-EPI)/1.73m²	HbO ₂	0.94
K	3.6 mmol/L		
Na	141 mmol/L	urine	Proteinuria 2
LDH	4.68 μkat/L		
CRP	109 mg/L		
Trop I ultra	0.02 μg/L		
NT-pro BNP	2878.5 ng/L		



Enlarged heart, mild interstitial oedema

Differential diagnosis of acute chest pain

- **Nonvascular pathology:** pulmonary embolus, spontaneous pneumothorax, aortic regurgitation without dissection, esophageal rupture, pericarditis and pleuritis
- **Vascular pathology:** acute coronary syndrome, acute aortic dissection, other acute aortic pathologies as intramural hematoma without dissection, aortic aneurysm, aortic injury without dissection, peripheral artery diseases, chronic aortic dissection



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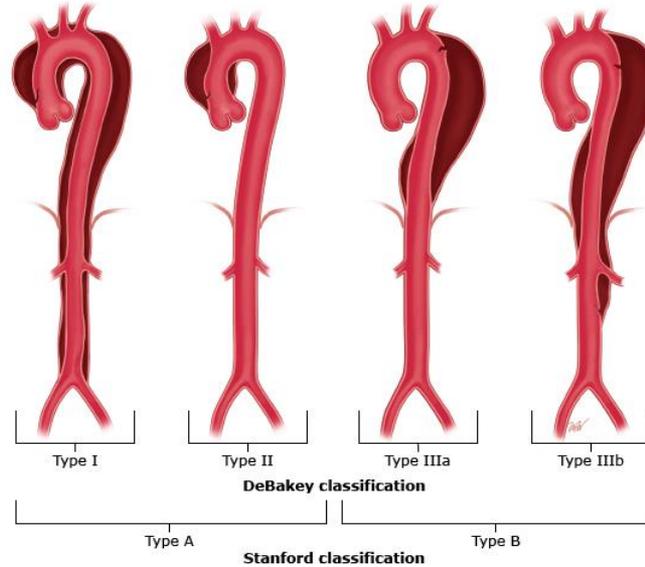
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TOR AORTA VRT
CTA TORAKALNE AORTE

Classification of aortic dissection



In the DeBakey classification of aortic dissection:

- Type I involves the ascending aorta, arch, and descending thoracic aorta and may progress to involve the abdominal aorta.
- Type II is confined to the ascending aorta.
- Type IIIa involves the descending thoracic aorta distal to the left subclavian artery and proximal to the celiac artery.
- Type IIIb dissection involves the thoracic and abdominal aorta distal to the left subclavian artery.

In the Stanford classification of aortic dissection:

- Type A involves the ascending aorta and may progress to involve the arch and thoracoabdominal aorta.
- Type B involves the descending thoracic or thoracoabdominal aorta distal to the left subclavian artery without involvement of ascending aorta.

Acute aortic dissection

- Relatively uncommon, estimated 2.6 to 3.5 per 100.000 person-years, 66% men
- Hypertension is the most important predisposing factor, more common in distal aortic dissection
- Important to define ascending vs descending aortic involvement (surgical vs medical management)

Drug choice according the cause and mechanism of the HTN Emergency

Table 3 Hypertensive emergencies requiring immediate BP lowering

Clinical presentation	Time line and target BP	1st line treatment	Alternative
Malignant hypertension with or without TMA or acute renal failure	Several hours, MAP -20% to -25%	Labetalol Nicardipine	Nitroprusside Urapidil
Hypertensive encephalopathy	Immediate, MAP -20% to -25%	Labetalol Nicardipine	Nitroprusside
Acute ischaemic stroke and BP >220 mmHg systolic or >120 mmHg diastolic	1 h, MAP -15%	Labetalol Nicardipine	Nitroprusside
Acute ischaemic stroke with indication for thrombolytic therapy and BP >185 mmHg systolic or >110 mmHg diastolic	1 h, MAP -15%	Labetalol Nicardipine	Nitroprusside
Acute haemorrhagic stroke and systolic BP >180 mmHg	Immediate, systolic 130<BP <180 mmHg	Labetalol Nicardipine	Urapidil
Acute coronary event	Immediate, systolic BP <140 mmHg	Nitroglycerine Labetalol	Urapidil
Acute cardiogenic pulmonary oedema	Immediate, systolic BP <140 mmHg	Nitroprusside or Nitroglycerine (with loop diuretic)	Urapidil (with loop diuretic)
Acute aortic disease	Immediate, systolic BP <120 mmHg and heart rate <60 b.p.m.	Esmolol and Nitroprusside or Nitroglycerine or Nicardipine	Labetalol or Metoprolol
Eclampsia and severe pre-eclampsia/HELLP	Immediate, systolic BP < 160 mmHg and diastolic BP <105 mmHg	Labetalol or Nicardipine and Magnesium sulphate	

Case-Treatment

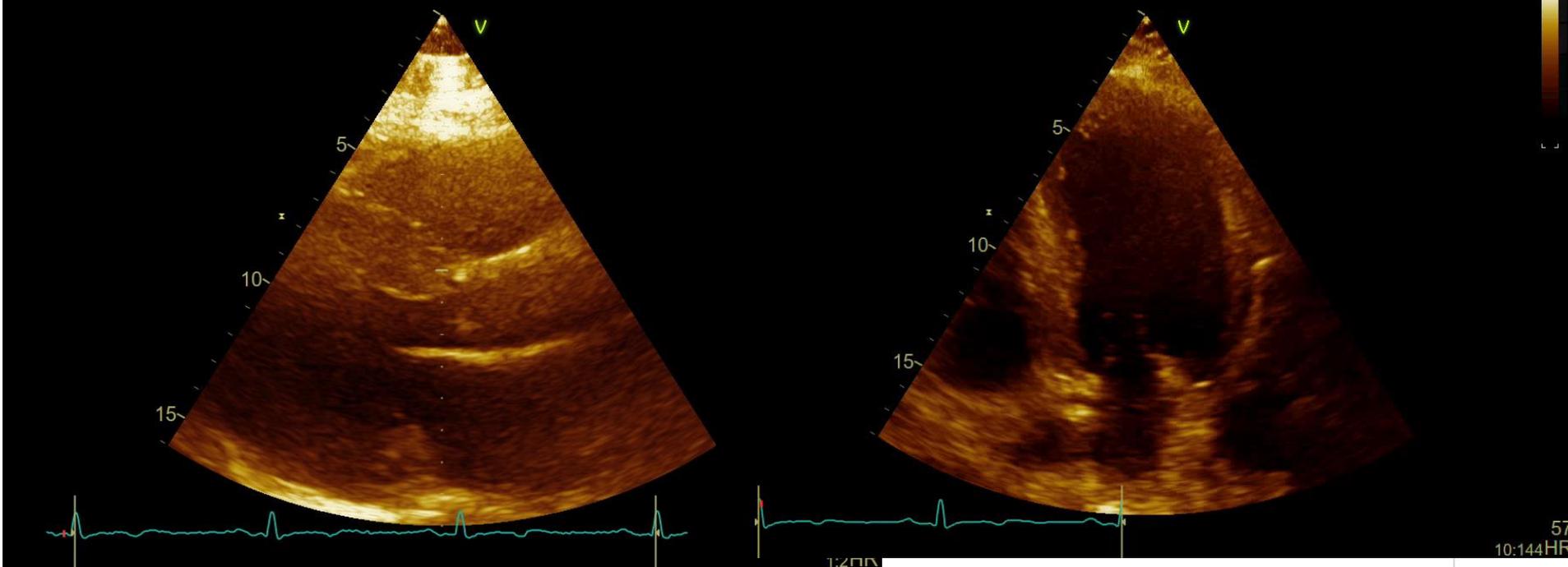
- Continuous perfusion of anti-hypertensive is mandatory, our case urapidil i.v. (25mg till 75mg i.v.) and gliceriltrinitrat infusion, furosemid 40mg i.v.
- Pain control - analgesia

Admission to the ward

- Intensive care unit, regulating BP, ACE inhibitor creat 455
- Doppler of renal arteries: bad visibility, no obvious stenosis, RK: 10.3cm, RI 0.78, LK 10.4cm, RI 0.8,
- Echocardiography

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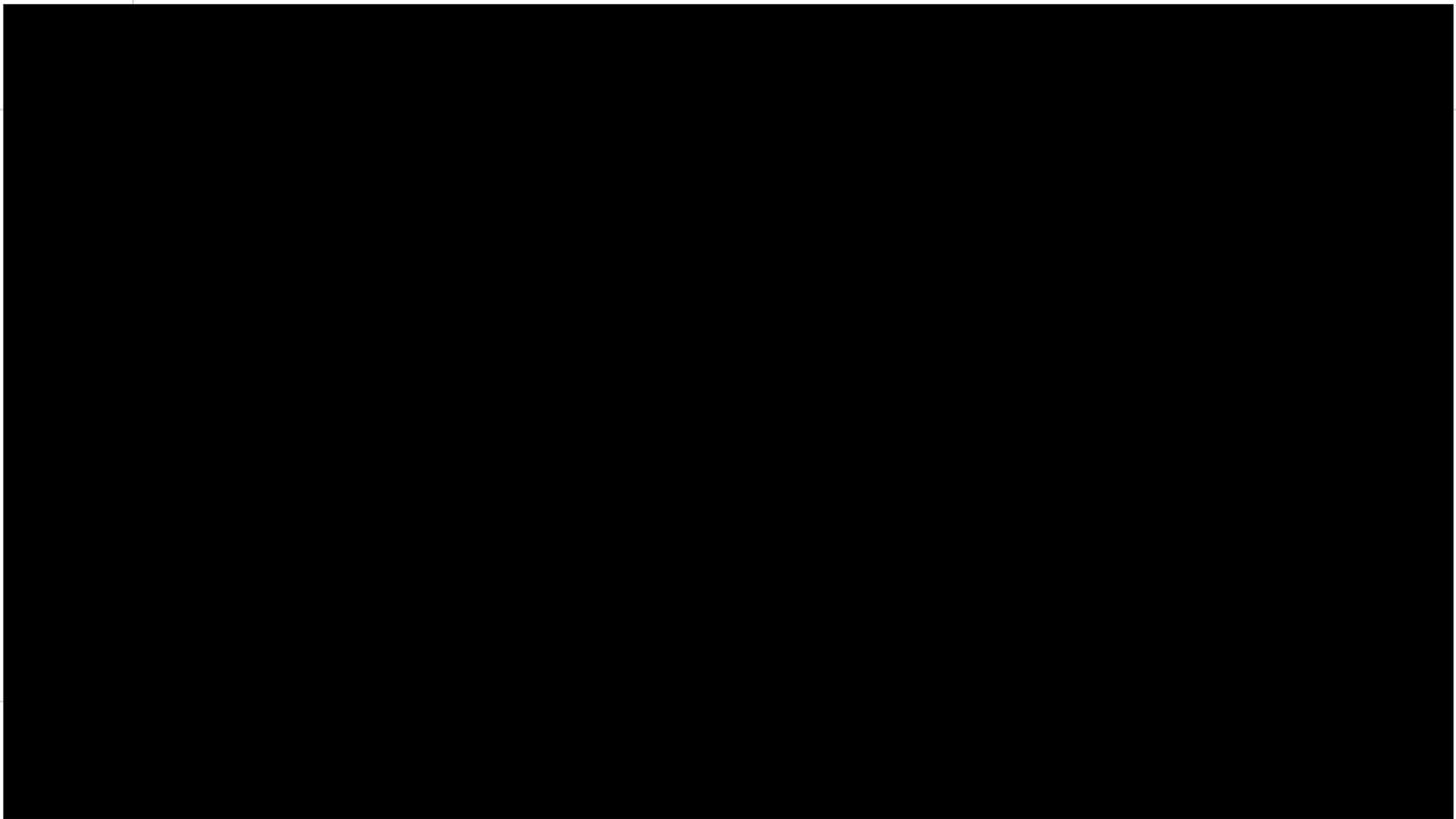
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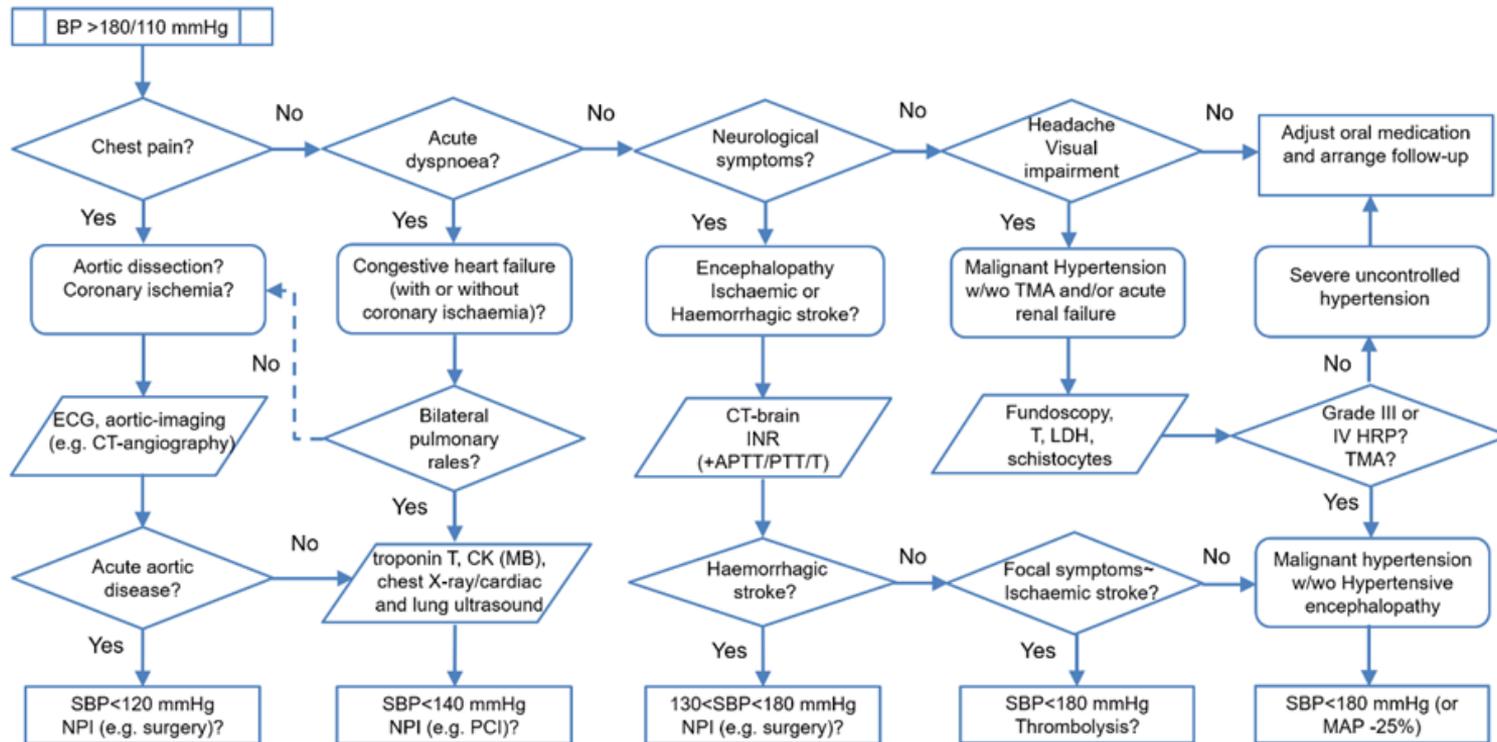
LV EDD 5.1cm, ESD 3.3cm, IVS 1.8cm, INF LAT 1.6cm, LVMI (ASE) 201g/m², LVMI (Ht) 115g/m, RWT 0.60, EDV 116ml, EDVI 53ml/m², SV 2D 77ml, EF 2D 66%. LVOT premer 2.5cm. LA premer (PLAX) 4.9cm,

Discharge from the ward

- Her th. on discharge was: amlodipine 10mg/24h, furosemide 40mg 2 times daily, minoxidil 2.5mg/12h, bisoprolol 10mg, doxazosin 4mg/12h, moxonidine 0.3mg/12h, rosuvastatin 10mg, aspirin 100mg, sodium bicarbonate 2x1g, CaCo₃ 3x 1g
- Advised to continue loosing weight







Case 3

- 59 y old lady admitted to the emergency department due to heavy breathing which started in late afternoon and escalated till late evening so relatives called an ambulance.
- Rescue team measured blood pressure 240/100mmHg oxygen saturation 70% and put her on OHIO mask where oxygen saturation elevated to 90%, administered furosemide 40mg i.v., Morphine 2mg plus 2mg i.v., metoclopramide i.v. and fenoterol and ipratropium bromide in inhalation.
- They transferred her to emergency department of our university hospital.

Diagnostic examination:

- Detail history and physical examination with BP measured on both arms
- ECG (ischemia, arrhythmias, left ventricular hypertrophy)

On indication:

- Troponin-T, CK, CK-MB
- Chest X-ray (fluid overload)
- Fundoscopy
- Transthoracic echocardiography (cardiac structure and function) or
- point of care cardiac and lung ultrasound (cardiac pulmonary oedema)
- CT (or MRI)-brain (intracranial haemorrhage)
- CT-angiography of thorax and abdomen (acute aortic disease)
- Renal ultrasound (postrenal obstruction, kidney size, left to right difference)

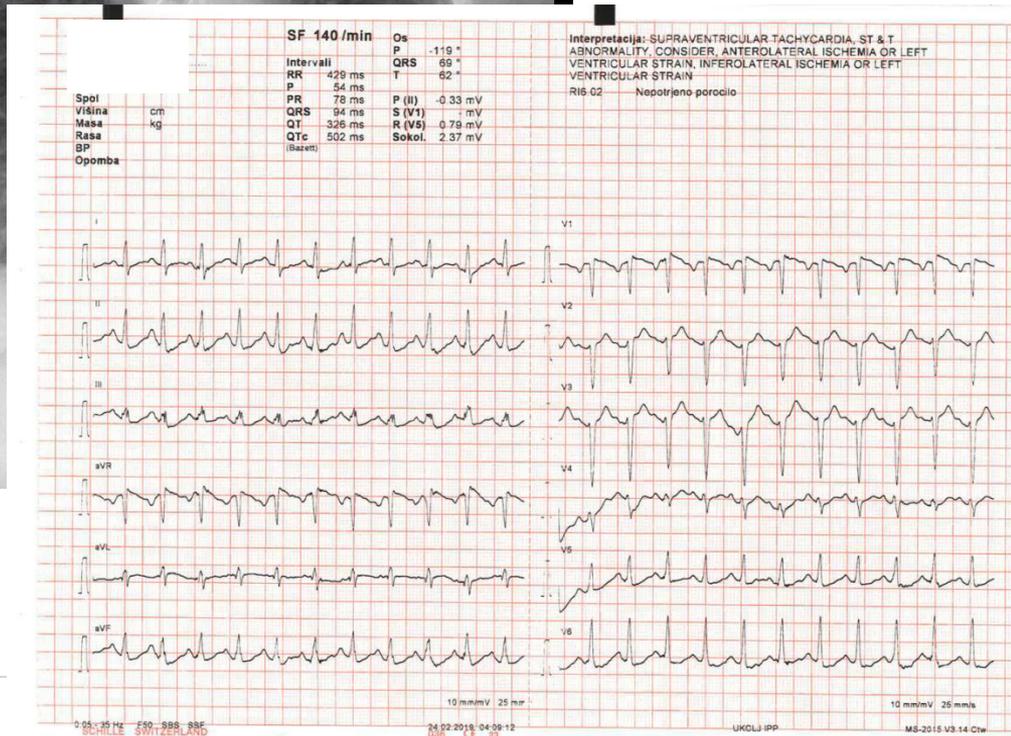
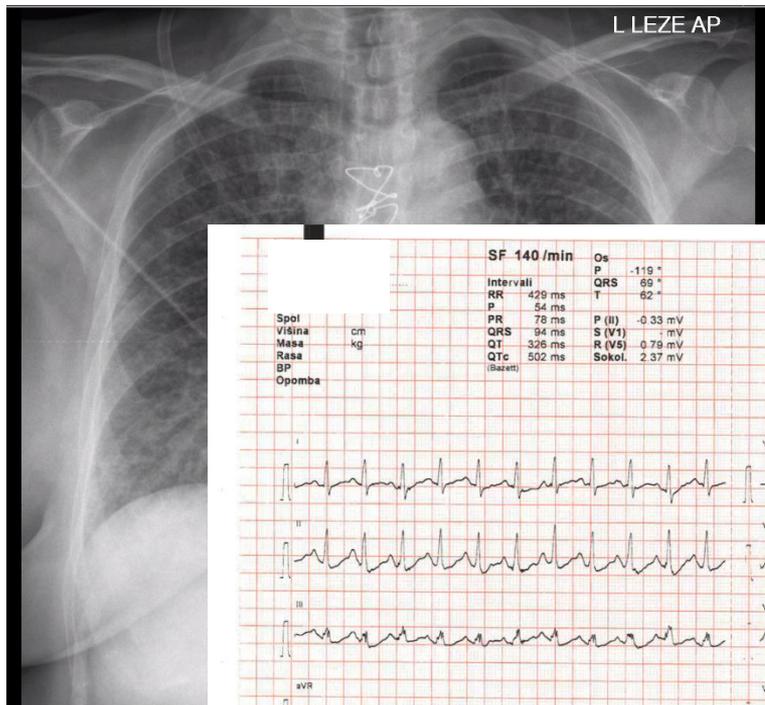
- **Family history:** Father CVI, mother operated on heart but no details known
- **Past history:** Hypothyreosis from 2001 on therapy,
- PTA AIC bilateral with stenting in year 2013,
- Known three vessel coronary disease. CABG on February 2018 (LIMA-LAD, SVG-OMV, RCA off pump)
- Aorto-bifemoral bypass and TEA AFC sin on May 2018
- **Present history:** Treated for hypertension for 15 years, two days before admission she had breathing problems which worsened the night of admission
- On medications: bisoprolol 2x5mg, Aspirin 100mg, perindopril/indapamide/amlodipine 10/2.5/10mg, rosuvastatin 40mg, levothyroxine 50microg
- Smoker 30y 15 cig/day, now 6 cig/day

- On admission, her oxygen saturation on OHIO oxygen mask was 94%
- BP 233/125mmHg, HR 141/min
- Jugular venous pressure was measured as 20 cm
- Lungs: diffuse inspiratory crackles and expiratory wheezing were heard
- Heart: rhythmic action fr 130/min, no murmurs
- No abnormalities in abdomen
- Moderate peripheral oedema

Lab. test

variable	result	variable	result
RBC	5.22 (10 ¹² /L)	OHIO mask	
WBC	20.1 (10 ⁹ /L)	pH	7.29
Hb	143 g/L	pO ₂	9.1 kPa
Creatinine	120 μmol/L (1.36 mg/dL)	pCO ₂	6.0 kPa
GFR	43 (CKD-EPI)/1.73m ²	HbO ₂	0.91
K	4.1 mmol/L		
Na	141 mmol/L		
LDH	4.68 μkat/L		
CRP	117 mg/L		
Trop I ultra	0.289 μg/L		
NT-pro BNP	26926 ng/L		

Case 1: admission



Case 3: treatment

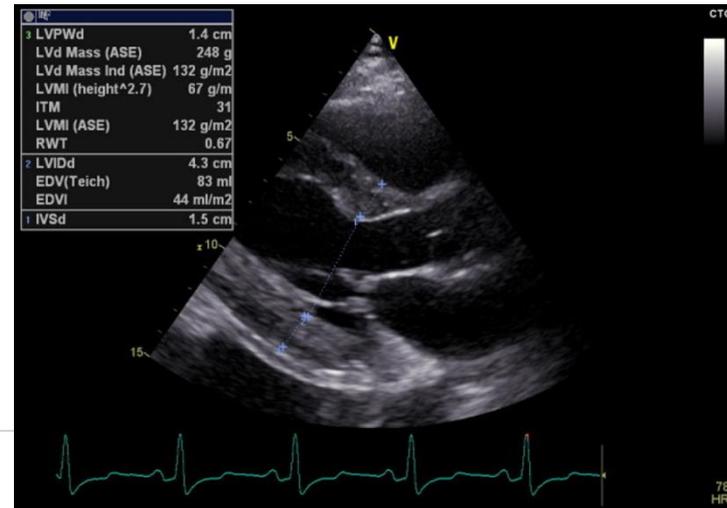
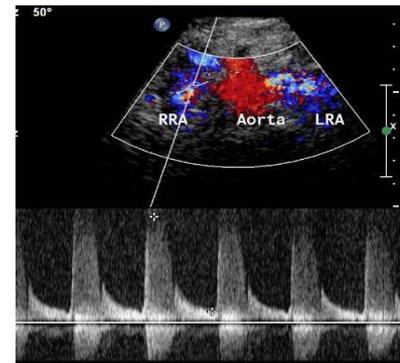
- OHIO oxygen mask
- NTG 2x subl, Nitroglycerine infusion, furosemide 40mg iv, MO 3mg,
- Beside cefotaxime, azithromycin were suggested from our infectologists

- BP dropped to 150/82, HR 120/min
- She was feeling better and admitted to our department
- During hospitalization we noticed, murmur in abdomen, transitional decline in renal function, SBP was 100-110 mmHg
- Abdomen US and renal doppler was performed

Case 3: hospitalization

- Sizes: right kidney 11.6 cm, left kidney 9 cm
- Resistance indexes: right 0.61 left 0.55
- Stenosis of left renal artery
- **ECHO**: normal EF, contractility abnormalities, elevated LVMI

Dg.: Pulmonary oedema
Acute respiratory infection
Renovascular hypertension
CABG 2018
Peripheral Arterial Disease
Hypothyreosis on th.



Hypertension

Therapy:

bisoprolol 2x5mg, Aspirin 100mg,
amlodipine 10mg,
rosuvastatin 40mg,
levothyroxine 50microg

Woman, 59 y

24-h average:

SD (weighted):

Awake:

Sleeping:

BP dipping:

Max. (last hour):

HR (24 h/awake/sleep):

Succ. 95%

139 / 69 mmHg

17 / 9 mmHg

133 / 69 mmHg

148 / 69 mmHg

+11 / 0 %

169/81 mmHg

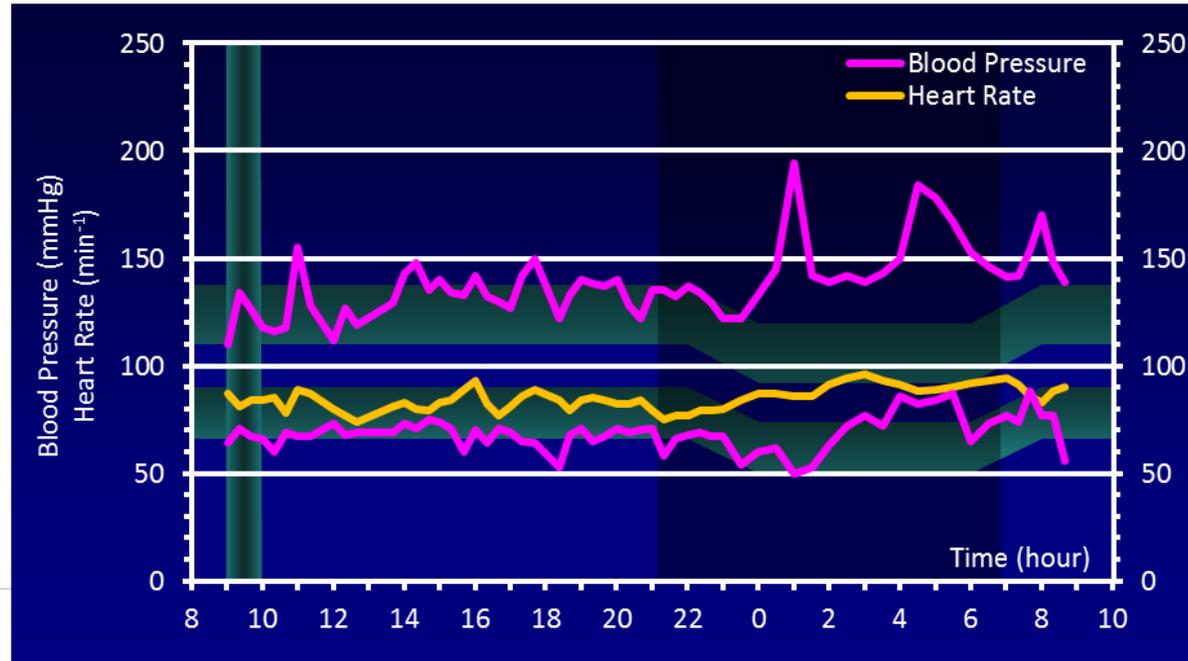
85 / 84 / 87 min⁻¹ (+4%)

ARV_d = 15/7 mmHg
ARV₂₄ = 13/7 mmHg



24-hour
Ambulatory
Blood
Pressure
Monitoring
2019-03-13

KOHip 2019



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Eclampsia and severe pre-eclampsia/HELLP	Immediate, systolic BP < 160 mmHg and diastolic BP <105 mmHg	Labetalol or Nicardipine and Magnesium sulphate	

Table 1. Hypertensive Emergency versus Asymptomatic Markedly Elevated Blood Pressure

Term	Criteria
Hypertensive Emergency	SBP > 180 or DBP > 120 mmHg AND End-Organ Dysfunction
Asymptomatic Markedly Elevated Blood Pressure (formerly hypertensive urgency)	SBP \geq 160-180 mmHg or DBP \geq 100-120 mmHg

Table 2. Symptoms Associated with Hypertension

Hypertensive Emergency	Blood Pressure Target	Treatment
Aortic Dissection	Reduce SBP to 100-120 mmHg Lower heart rate to <60 bpm	1. Labetalol or Esmolol 2. Nitroprusside or nicardipine after beta-blocker
Acute Myocardial Infarction	Reduce BP < 160 mmHg	Nitroglycerin Labetalol or metoprolol (if no heart failure)
Acute Pulmonary Edema	Reduce BP by 20%–30%	Nitroglycerin sublingual/topical/IV or Nitroprusside IV or Nicardipine IV and Furosemide if volume overloaded
Acute Renal Failure	Acute reduction in BP by < 20%	Nicardipine IV or Clevidipine IV
Hypertensive Encephalopathy	Reduce BP 20-25% in first hour	Nicardipine IV or Clevidipine IV or Labetalol IV
Subarachnoid Hemorrhage	Reduce SBP to <160 mmHg	
Intracerebral hemorrhage	SBP goal 140-180 mmHg	
Acute Ischemic Stroke	Receiving tPA: Reduce < 185 mmHg with goal 140-150 mmHg No tPA: Reduce to <220/120 mmHg	

Definition

- We should abandon the term ‘hypertensive crises’, which may not precisely characterize the clinical picture.
- We should use the terms:
- Hypertensive emergencies,
or
- Uncontrolled hypertension
- depending on the presence (emergencies) or absence of **acute and rapidly progressing end-organ dysfunction/damage**

Important points

- HT emergencies 1 in 200 pts at the emergency department
- A correct diagnosis and appropriate treatment are critical.
- Blood pressure should be reduced:
- within **minutes to hours** (target BP depending on the clinical diagnosis) in patients with hypertensive emergencies
- **within 24 to 48 hours** in patients with hypertensive urgencies.



