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Cardiogenic shock and Diabetes Mellitus Type 2 Complicating Acute Myocardial Infarction

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Background

- The clinical definition of **Cardiogenic shock** is decreased cardiac output and evidence of tissue hypoxia in the presence of adequate intravascular volume.

https://emedicine.medscape.com/article/152191-overview

- Left bundle branch block (LBBB) is an independent negative prognostic marker in acute myocardial infarction. A diagnosis of MI with ECG is especially difficult in the setting of LBBB because of the characteristic ECG changes caused by altered ventricular depolarization.

(Bryan Wilner, etc, 2017 Americal College of Cardiology)

Epidemiology

- Epidemiologically proven, 5%-15% of patients with Acute Myocardial Infarction (AMI) develop cardiogenic shock (CS)

  D. Kalavrouziotis et all Canadian Journal of Cardiology 33 (2017) 36e43

- Even with the introduction of modern intensive care units (ICUs), advanced medical treatment, and invasive devices, in-hospital death rates remain high at 40%-50%, despite advances in early revascularization and adjunctive pharmacotherapy

  Eur Heart J 2015;36:1223-30

- In large study with 72,765 cardiogenic shock patients (J.B.Echouffo-Tcheugui, 2018) were found that pre-existing diabetes (DM) was associated with an increased risk of cardiogenic shock (5.8% vs 5.2%; adjusted odds ratio [aOR] 1.14) and it worsens outcomes (higher in-hospital mortality (37.9% vs 36.8%; aOR 1.18), with a longer hospital stay (mean ± SEM: 11.6 ± 0.16 vs 10.9 ± 0.16 days)).

  https://doi.org/10.1016/j.amjmed.2018.03.004

Clinical case

- *Patient* - S.A.P
- *Age* - 72
- *Gender* - Female
- *Occupation* - House Wife
- *Date of hospital administration* - 12-December-2017

Complaints on admission

- 12-dec-2017 - Patient was delivered by ambulance with complaints of general weakness and dyspnea on minimal physical exertion.

- Intensive chest pain bothered patient on 11-dec-2017 at 9pm for which the patient has consecutively taken 6 tablets of Nitroglycerin.

- On admission moment patient denies chest pain as complain.

Past Medical History

- **1995** – Diabetes mellitus type II, constantly receiving treatment with "Lantus" 40 IU/day and Glybenclimide 5 mg/day
- **1999** – History of Arterial hypertension controlled by:

✓Sartan

✓*Ca2+ - channel blocker*

✓*Bisoprolol*

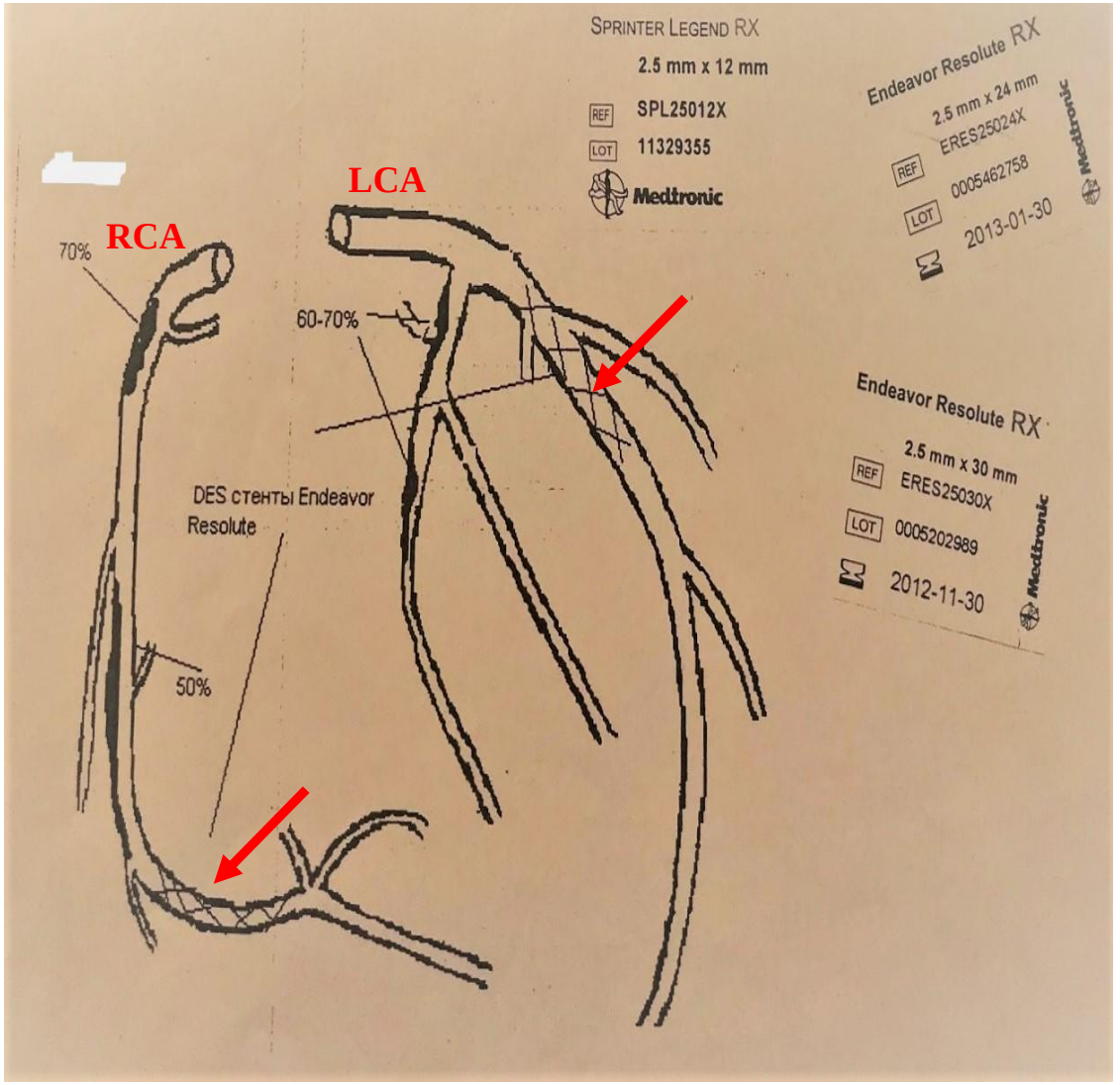
[Max BP 220/110, min 140/70 mmHg]

- **2010** – Initial appearance of retrosternal chest pain.
- Received routine double - therapy with Aspirin + Ticagrelor
- No MI or stroke in anamnesis

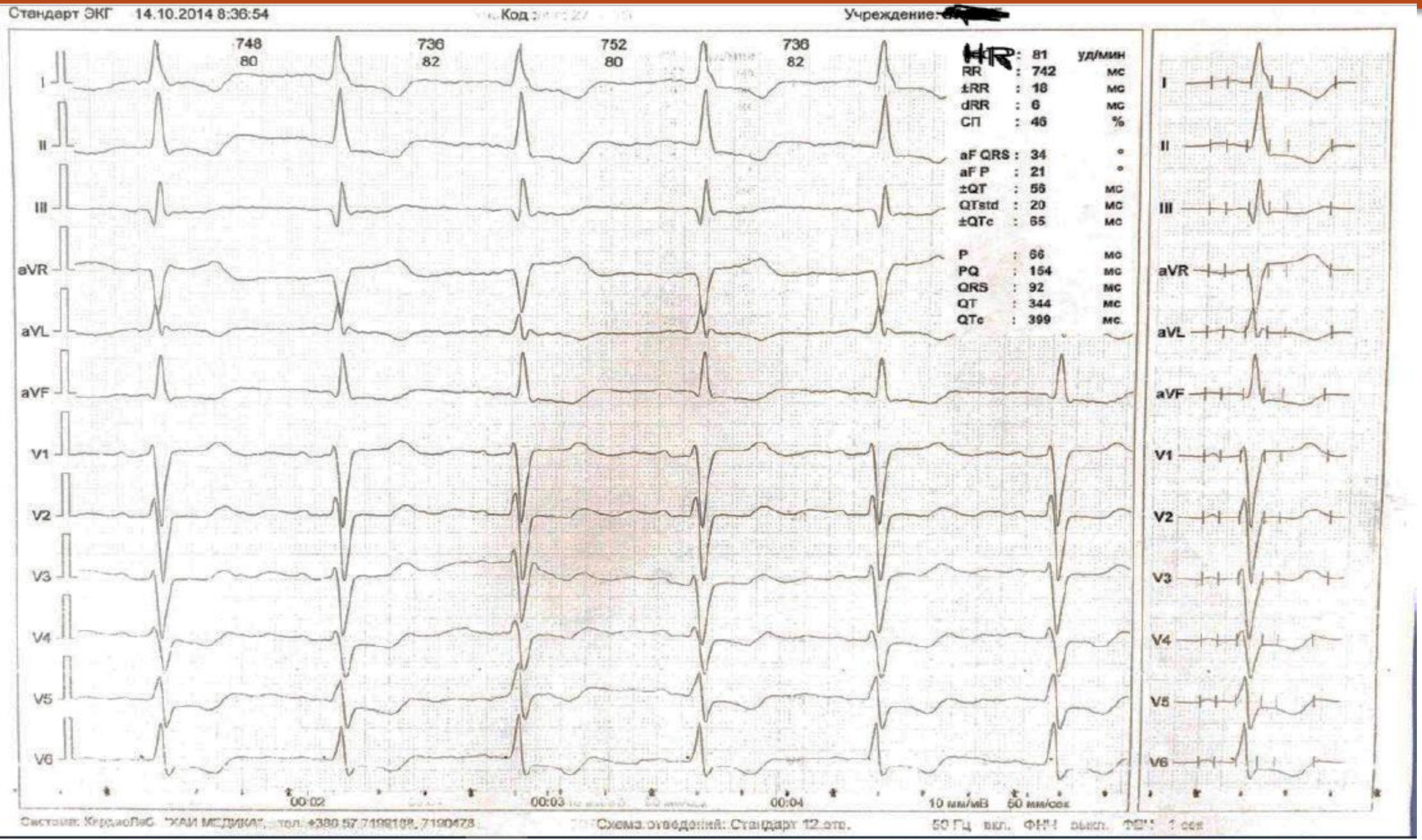
Cont.

- 2011- Coronarography revealed diffuse atherosclerosis of coronary arteries for which PCI with stenting was performed
- Post PCI - No chest pain or physical exertion intolerance
- Subsequently, symptoms reappeared elementarily in 2016
- RCA - Critical occlusion before bifurcation with stenting (arrow), TIMI - 1 before stenting, in proximal segment – stenosis 70%, in middle segment – 50%
- LCA - prolonged atherosclerotic plaque with sub-occlusion in left anterior descending branch (stenting-arrow), atherosclerosis of diagonal branches, diffuse stenosis of circumference branch – 60-70%
- Before stenting diagnosis of stable angina IV class according to NYHA
-

TIMI Grade	Description
TIMI 0 - no perfusion	no antegrade flow beyond the point of occlusion
TIMI 1 - penetration without perfusion	faint antegrade coronary flow beyond the occlusion with incomplete filling of the distal coronary bed
TIMI 2 - partial perfusion	delayed or sluggish antegrade flow with complete filling of the distal territory
TIMI 3 - complete perfusion	normal flow with complete filling of the distal territory



Electrocardiogram - 2014



- Sinus rhythm, HR 80 in min. Normal heart axis. Relative signs of LV hypertrophy.
- Repolarization alternation in anterior-lateral LV wall.

Objective examination on admission

- Conciseness – Lethargic. State –Severe, Body position - Passive
- Skin and Mucosae – pale, acrocyanosis, cyanosis of lips
- BMI – 32 kg/m2
- RR – 18 /min, SpO2 – 75%
- Pulse – Rhythmic, 90 bpm
- BP- 110 / 60 mm/Hg
- Pulmonary:

✓ Percussion- Insignificant.

✓ Auscultation:

a) Decreased breath sounds over inferior and lateral parts of lungs
b) Wheezing over both lung feilds
c) Rales below both scapular angles.

Cont.

- Cardiovascular:

✓ Percussion: Left border displaced 1.5 cm away from midclavicular line.

✓ Auscultation: Rhythmic, Heart sounds – Muffled

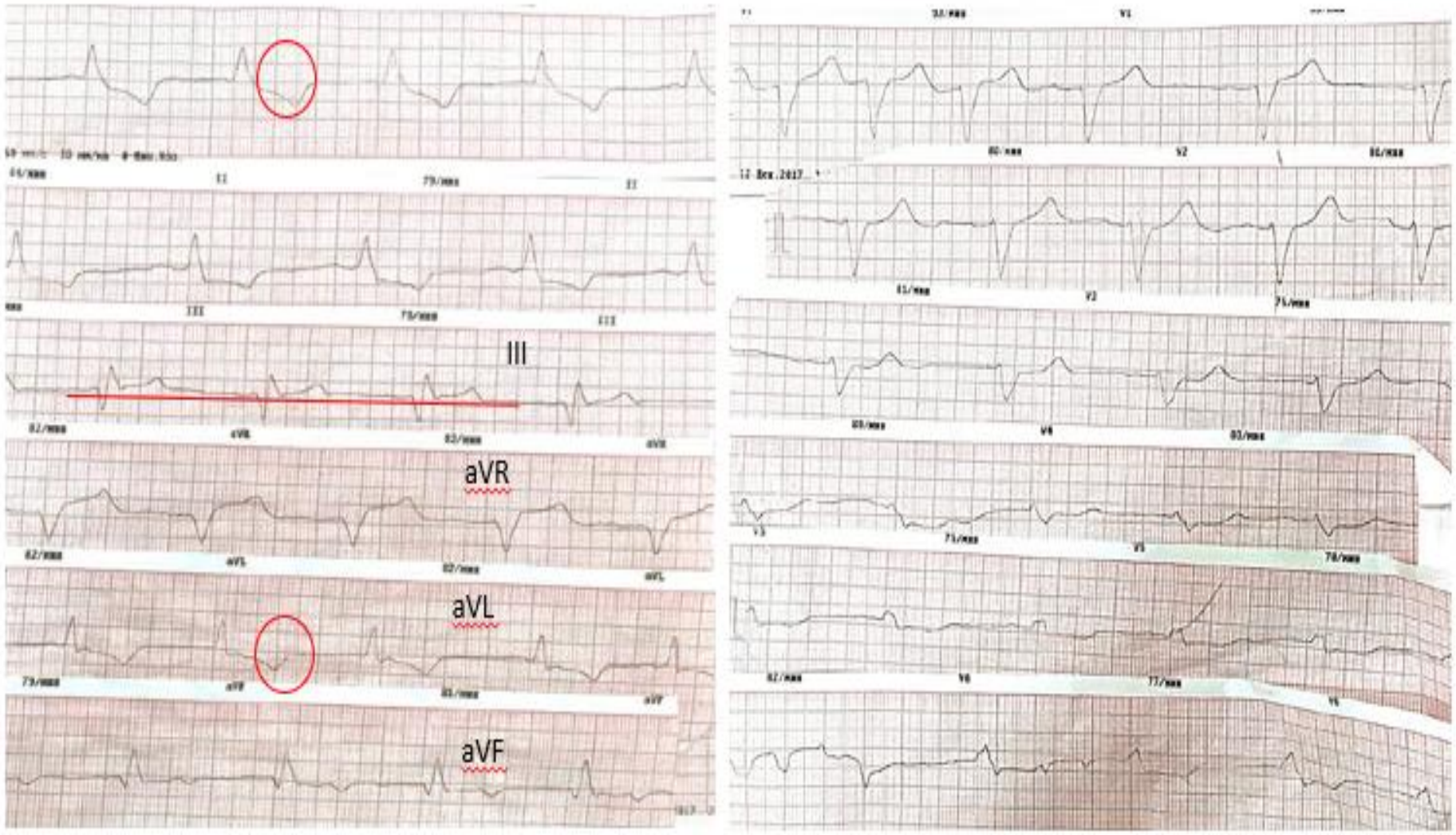
- Abdomen: Normal size, symmetric, no tenderness

✓ Liver: Enlarged, +2cm with no tenderness

✓ Spleen: Normal

- Edemas: Pitting edemas
- CVAT - Negative

Conclusion

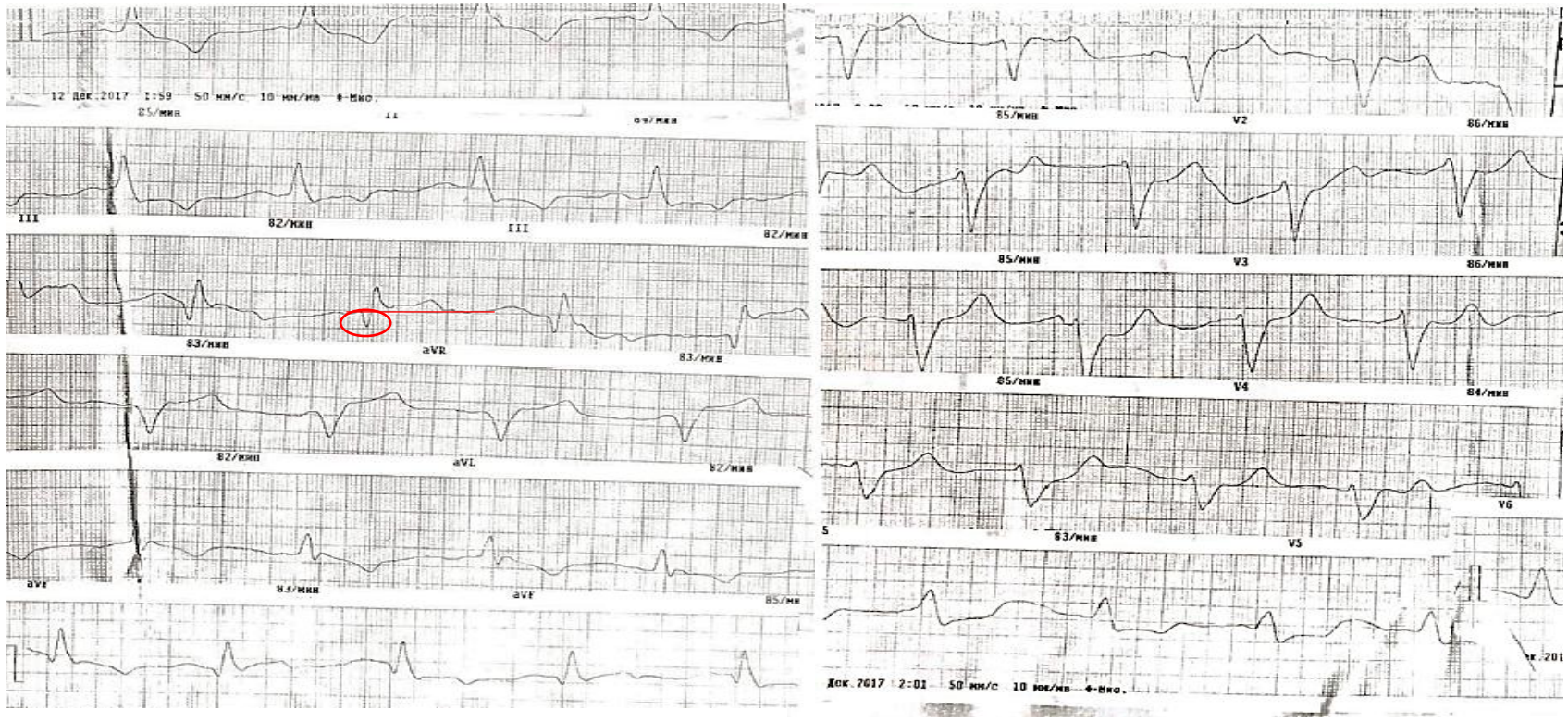


- ✓ Sinus rhythm, HR – 83 bpm.
- ✓ Left axis deviation. LBBB (QRS - 0,12s).
- ✓ Paired supraventricular extrasystoles (in V1).
- ✓ Posterior myocardial infarction (ST-segment elevation greater than or equal to 0.1 mV (1 mm) in leads with a positive QRS complex, and ST depression greater than or equal to 0.1 mV (1 mm) in leads V1 through V3, ie, leads with a dominant S wave. Negative T in 1 and aVL, Q wave start of formation).

2:01 am

✓ **Clinically:**

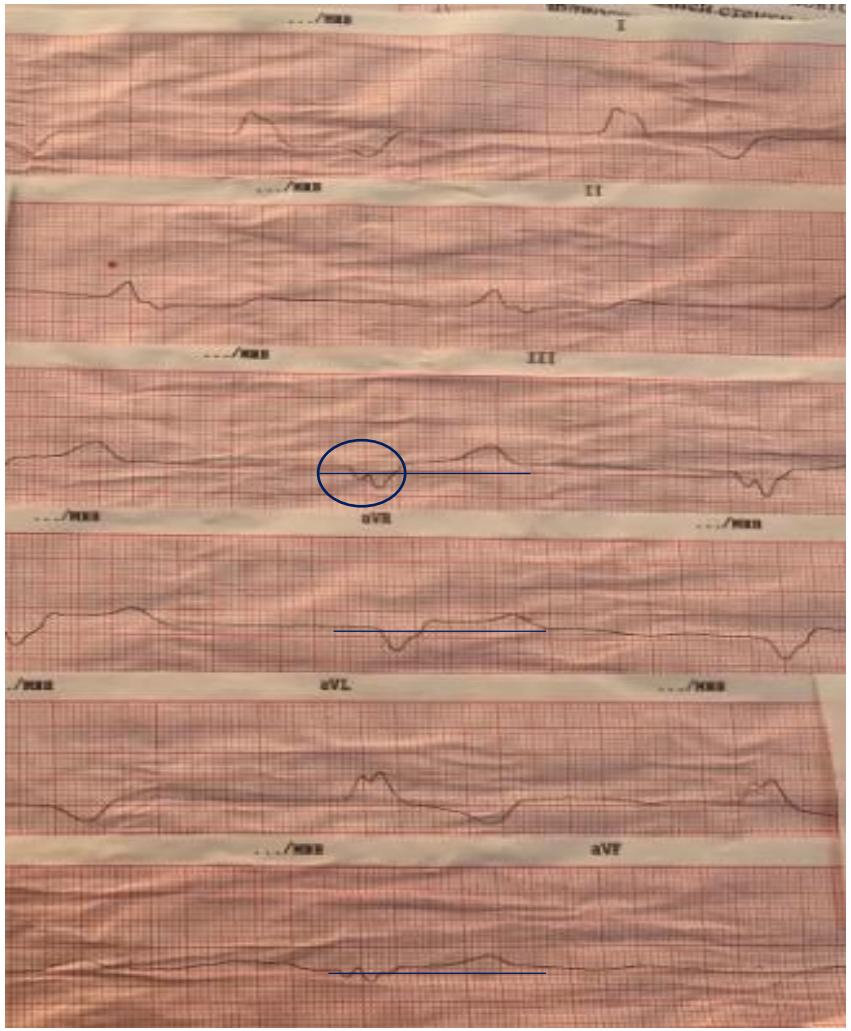
- Dyspnea, exacerbated by horizontal position and in minimal exertion.
- Conciseness, sleepy??, State –severe. Acrocyanosis, cyanosis of lips.
- RR – 26 bpm. SpO2 – 82%.
- Crackles in lower lung fields. Heart sounds are muffled, rhythmic. HR- 68 bpm.
- BP- 90/60 mm/Hg on dopamine infusion. Diuresis by catheter 8-10:00 is 10 ml

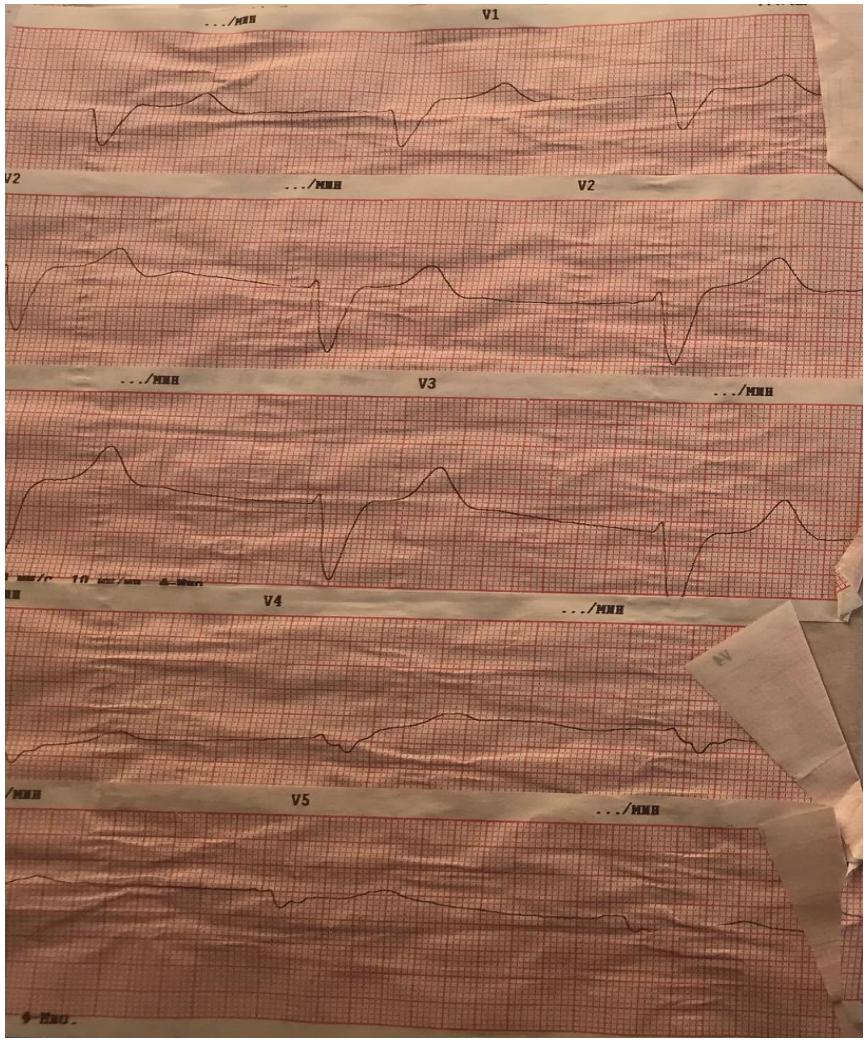


✓ **Conclusion:**

- Sinus rhythm, HR-84 bpm.
- Left axis deviation. LBBB (QRS - 0,12s).
- Positive Q wave, posterior myocardial infarction (ST-segment elevation greater than or equal to 0.1 mV (1 mm) in leads with a positive QRS complex in III and aVF, and ST depression greater than or equal to 0.1 mV (1 mm) in leads V1 through V3, ie, leads with a dominant S wave. Reciprocal negative T in 1 and aVL, Q=0.02 sec, 4mm), negative dynamics comparing with previous.

3:30 pm

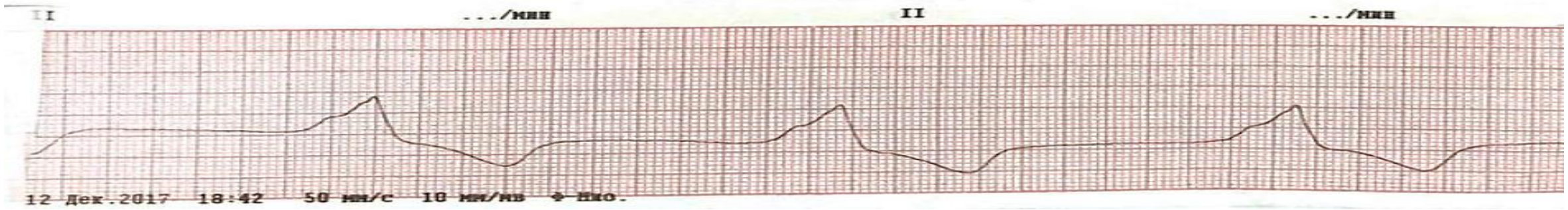




✓ **Clinically:**

- *BR – 30 in min. SO2 – 63%.*
- HR=Ps=66 in min
- *BP 85/60 mm Hg on dopamine infusion background*. Pitting edemas.
- *Diuresis* by catheter 8-14:00 is 20 ml.
- On behalf of pulmonary edema treatment was added Sol. Morphini hydrochloride 1% - 1ml in 10 ml 0,9% NaCl solution bolus, Furosemide 60mg intravenously, venous tourniquets placement, O2 inhalation.

- **18:42 (6:42pm)** unconsciousness, wide pupils, no respiration, no BP or pulse on main vessels. ECG: idioventricular rhythm



✓ Therapy with CVR, artificial lung ventilation in CMV regimen, adrenalin 0,18%-1ml and atropine 0,9%-10ml

- **18:55 (6:55 pm)** unconsciousness, wide pupils, no photoreactions. Respiration is absent, no BP or pulse on main vessels. ECG: isoline. Biological death.

Complete blood count

	Pts ranges	Normal Range
Hemoglobin, g/l	**100**	120-140
Red blood cells, 1012	**3,5**	3,9-4,7
Color index	**0,75**	0,85-1,15
White blood cells, 109	**14,7**	4-9
ESR, mm/h	3	2-15
Bands	3	1-6%
Segments	56	47-72%
Eosinophils	3	0,5-5%
Monocytes	2	3-11%
Lymphocytes	36	19-37%
Platelets	185	180-320

Conclusion: Mild hypochromic anemia and leukocytosis

Biochemical panel

	Glucose profile (3,3-5,5)
1:30	26,6 mmol/l
8:30	15,0 mmol/l
11:00	13,2 mmol/l
13:00	10,2 mmol/l

	Troponin I (till 0,5)
1:30	0,84 ng/ml

Conclusion: **Poorly controlled Hyperglycemia and Troponin I elevation**

Echocardiography on admission

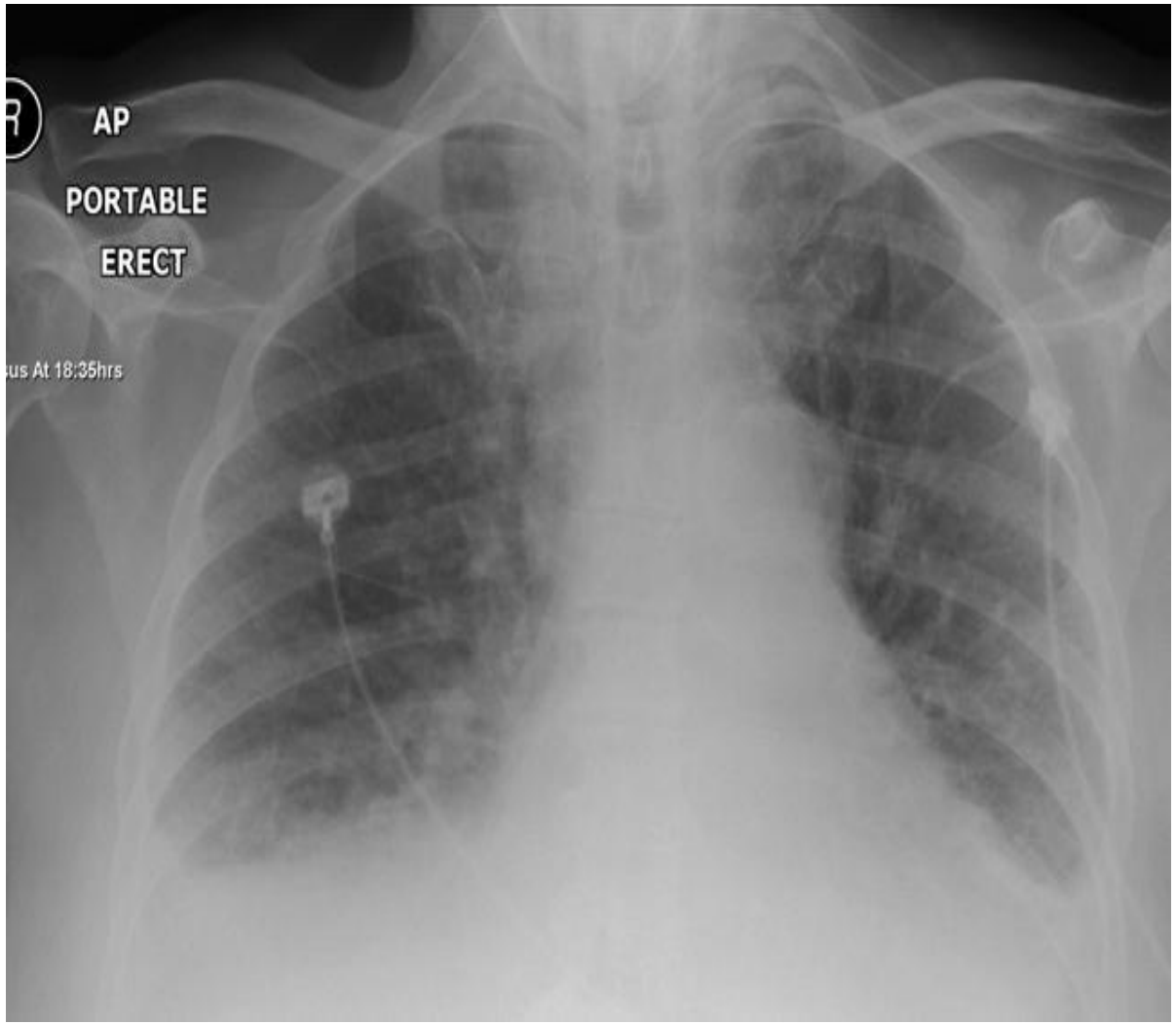
Left Ventricle: • FDD – 59 mm (N – 35 – 55mm -FSD – 48 mm (N – 23 – 38 mm) • FDV – moderately increased – 174 ml • EF – 35% (N - 55 – 78%). • Stroke volume – 62 ml - increased • Posterior wall thickness in diastole– 13 mm (N – 6 – 13mm). Mild hypertrophy of LV wall • Intraventricular wall thickness in diastole – 12 mm • Mitral regurgitation II stage	**Right Ventricle:** • Diameter – 25 mm (N – 9 – 26 mm) Wall thickness – 0,4 mm (N – 0,5 mm)changed.
Left atrium: • Dilated - 45 mm in diameter (N – till 39 mm)	**Right atrium:** • Dilated – 37 mm in diameter (N – 25-37).

Conclusion: Dilation of left heart chambers, LV hypertrophy. Diffuse contractility decline

Chest X-ray

- Decreased lung transparency.
- Lung roots – intensive, not structured.
- Diaphragmal and costal sinuses are dark, not visualized.
- Heart – increased in diameter, with non precise contours. Aorta - unchanged.

Conclusion: Congestive changes, probable pulmonary edema



Final Diagnosis

✓ **Main:**

- CAD: Acute (10.dec.2017) posterior MI type I, atherosclerotic cardiosclerosis, aorta and coronary arteries atherosclerosis.
- Stenosing coronary sclerosis (PCI 2011).
- Arterial hypertension III stage, very high risk

✓ **Complications:**

- Acute heart failure IV stage by Killip. Pulmonary edema, recurrent.
- Cardiogenic shock (12 dec 2017) III stage.
- Bilateral pleural effusion
- Complete left bundle branch block
- Asystole (12.12.2017 18:55)

✓ **Concomitant diseases:**

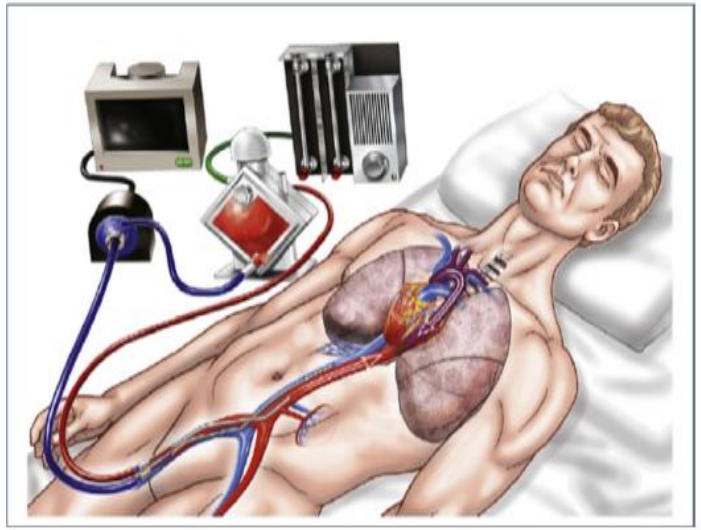
- Diabetes mellitus 2 type, insulin dependent, severe. Anemia of chronic disease, mild.

Mortality factors of Cardiogenic Shock

- Right bundle branch block
- ***Left bundle branch block*** (LBBB) is an independent negative prognostic marker in acute Myocardial infarction (AMI) (30% vs.19%, p = 0.012, OR 1.57) .
- Advanced age (75 years and more)
- Large myocardial involvement,
- ***Severe left ventricular dysfunction***
- ***Severity of end-organ injury***
- ***The glucose level at admission*** is a strong independent predictor for mortality
- **Comorbidities: STEMI, *Dyslipidemia***, Stroke, ***Diabetes mellitus***

Conclusion

- Probable causes of AMI after PCI performance in DM patients are: re-stenosis after PCI, progression of a separate untreated plaques, or the development of new ones with acceleration of negative remodeling owing to neointimal proliferation after PCI and increased platelet aggregation, small distal vessels microangiopathy and reduced collateral blood flow.
- The current management of patients with acute myocardial infarction complicated by cardiogenic shock (AMI-CS) is associated with a high rate of mortality, despite widespread regional implementation of rapid transfer to percutaneous coronary intervention-capable centers for prompt infarct-related artery reperfusion.
- In selected patients as our patient who was hemodynamically unstable, there might be a benefit associated with early institution of mechanical circulatory support before revascularization
- Unloading the left ventricle during AMI to decrease LV wall stress, stroke work, and myocardial oxygen demand might limit myocardial cellular loss and decrease the extent of infarction. The major clinical utility of short-term mechanical circulatory support (MCS) is the reversal of shock by the restoration of cardiac output for distal organ and coronary perfusion.



Any questions?