KHARKIV KARAZIN NATIONAL UNIVERSITY
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A ‘FORGOTTEN’ BRIDGE: GET BACK TO PAST

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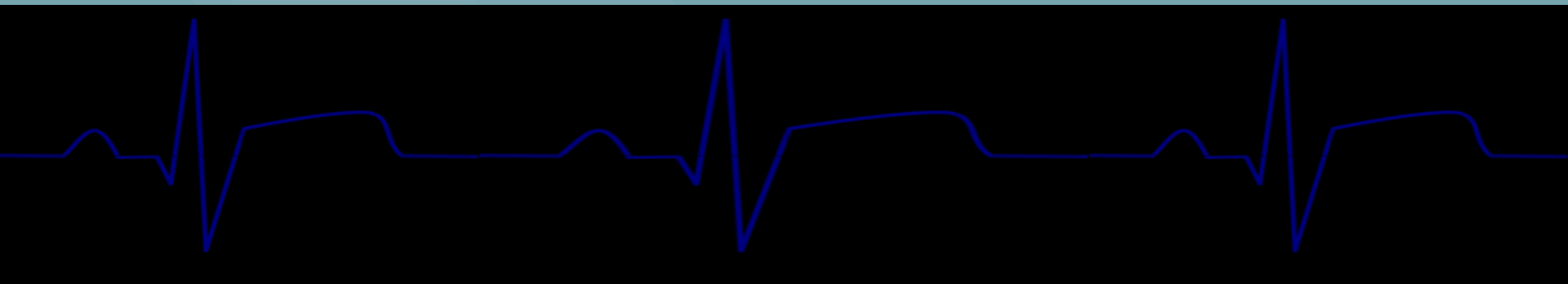


MYOCARDIAL BRIDGE

- occurs when one of the coronary arteries tunnels through the myocardium rather than resting on top of it
- in this case a band of heart tissue sits on top or surrounds of the coronary arteries
- as the heart squeezes to pump blood, the muscle exerts pressure across the bridge and constricts the artery
- are generally expressed clinically in young adult men, in whom typical or atypical chest pain appears, induced by physical exercise and appearing at rest

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2733018/

INTRODUCTION 1.12

- is an anomaly characterized by a typical intramyocardial route of a segment of one of the major coronary arteries
- it was recognized at autopsy by *Reyman* in 1737, and first described angiographically by *Portmann* and *Iwig* in 1960
- this anomaly is more frequent than previously thought; its reported incidence varies from 1.5% to 16% when assessed by coronary angiography, but can reach much higher percentages (from 40% to 80%) if estimated in the course of an autopsy series
- is generally confined to *the mid left anterior descending artery*; it is less frequently located in the circumflex artery, and is occasionally seen in the right coronary artery

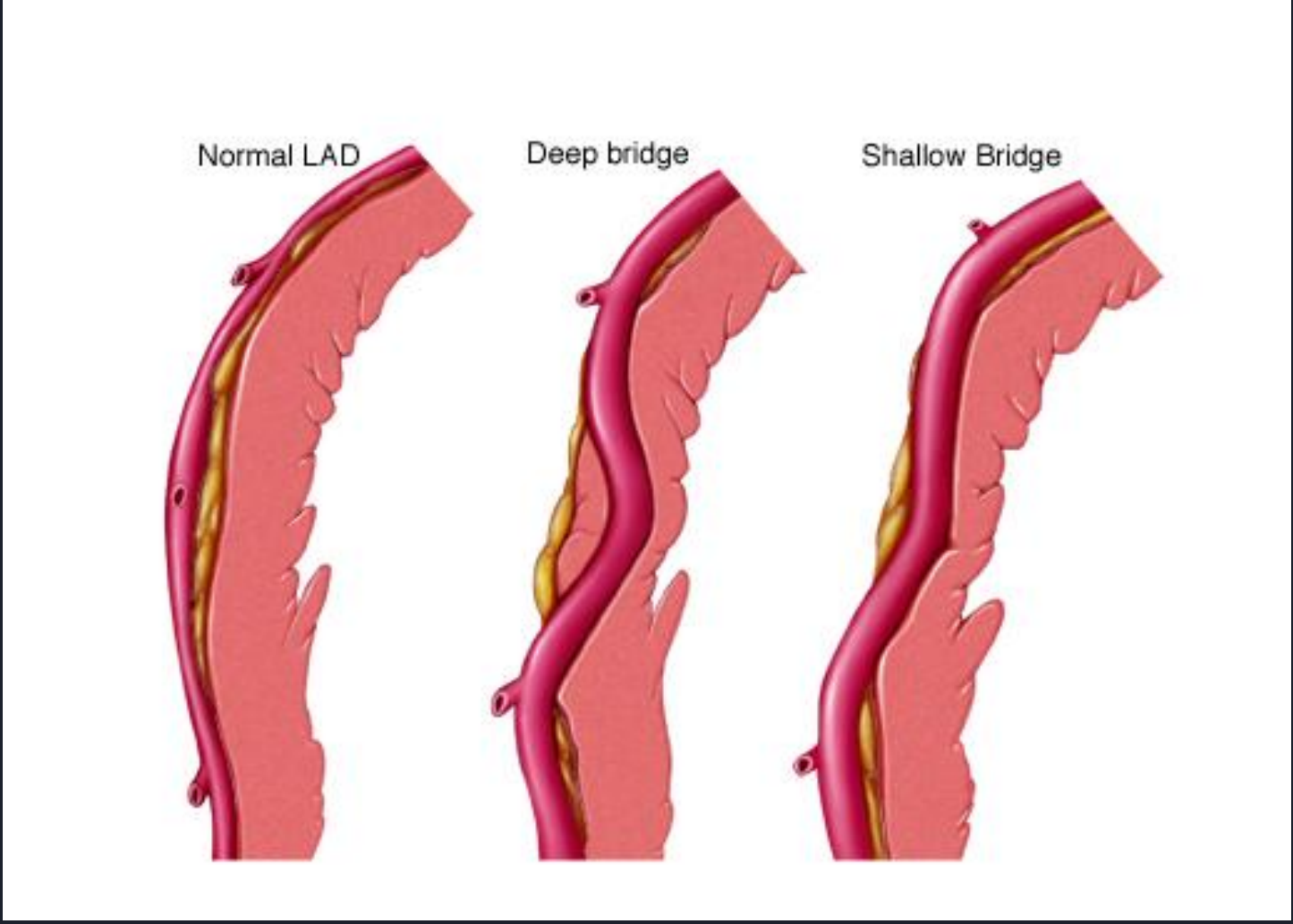
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2733018/

INTRODUCTION 2.12

- clinical complications include ischemia and acute coronary syndromes, coronary spasm, ventricular septal rupture, arrhythmias (including supraventricular tachycardia and ventricular tachycardia), exercise-induced atrioventricular conduction blocks, transient ventricular dysfunction and sudden death . The prognosis of patients with myocardial bridges, therefore, is not as benign as it was believed to be in the past

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2733018/

INTRODUCTION 3.12



https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4065198/

INTRODUCTION 4.12

The pathophysiological mechanisms of clinical manifestations:

- It has been demonstrated that the intima of the tunnelled artery is significantly thinner than that of the proximal segment of the artery
- the expression of vasoactive agents (endothelin-1, endothelial nitric oxide synthase, angiotensin-converting enzyme) and the extension of the atherosclerotic process are diminished in the myocardial bridge compared with the proximal and subsequent artery segments

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2733018/

INTRODUCTION 5.12

The typical angiographic feature is *systolic narrowing of an epicardial artery*, which is often **completely resolved** *during the diastolic phase* of the cardiac cycle.

Because only 15% of coronary flow normally occurs during systole and because **the myocardial bridge is a systolic angiographic event**, it acquires clinical relevance only in particular conditions, such as tachycardia. **Tachycardia** can provoke an ischemic effect on the myocardial bridge by shortening the diastolic phase and increasing the importance of systolic blood flow

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2733018/

INTRODUCTION 6.12

Intracoronary ultrasonography and Doppler studies

- the typical 'half-moon' phenomenon (echolucent area surrounding the myocardial bridge) - *demonstrating that the extrinsic compression of a tunnelled artery is not only a systolic event, but it also persists during a significant portion of the diastolic phase*
- characterized by a marked increase in blood flow speed during the early diastolic phase, followed by a sharp deceleration and a subsequent 'plateau' phase
- during the systolic phase of the cardiac cycle, because of the extrinsic compression of the tunnelled artery, anterograde flow is not produced or is very reduced, and retrograde flow at the proximal segment of the myocardial bridge is observed

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2733018/

INTRODUCTION 7.12

The therapeutic approaches are:

- beta-blockers
- calcium channel blockers
- coronary stents
- minimally invasive coronary artery bypass grafting
- surgical myotomy

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2733018/

INTRODUCTION 8.12

Nitrates should generally be avoided because they increase the angiographic degree of systolic narrowing and can lead to a worsening of clinical conditions

INTRODUCTION 9.12

Beta-blockers - reduce the cardiac frequency and increase the diastolic time, with a decrease in cardiac contractility and compression of the tunnelled artery

INTRODUCTION 10.12

Stenting of the myocardial bridge has, until now, given discordant results.

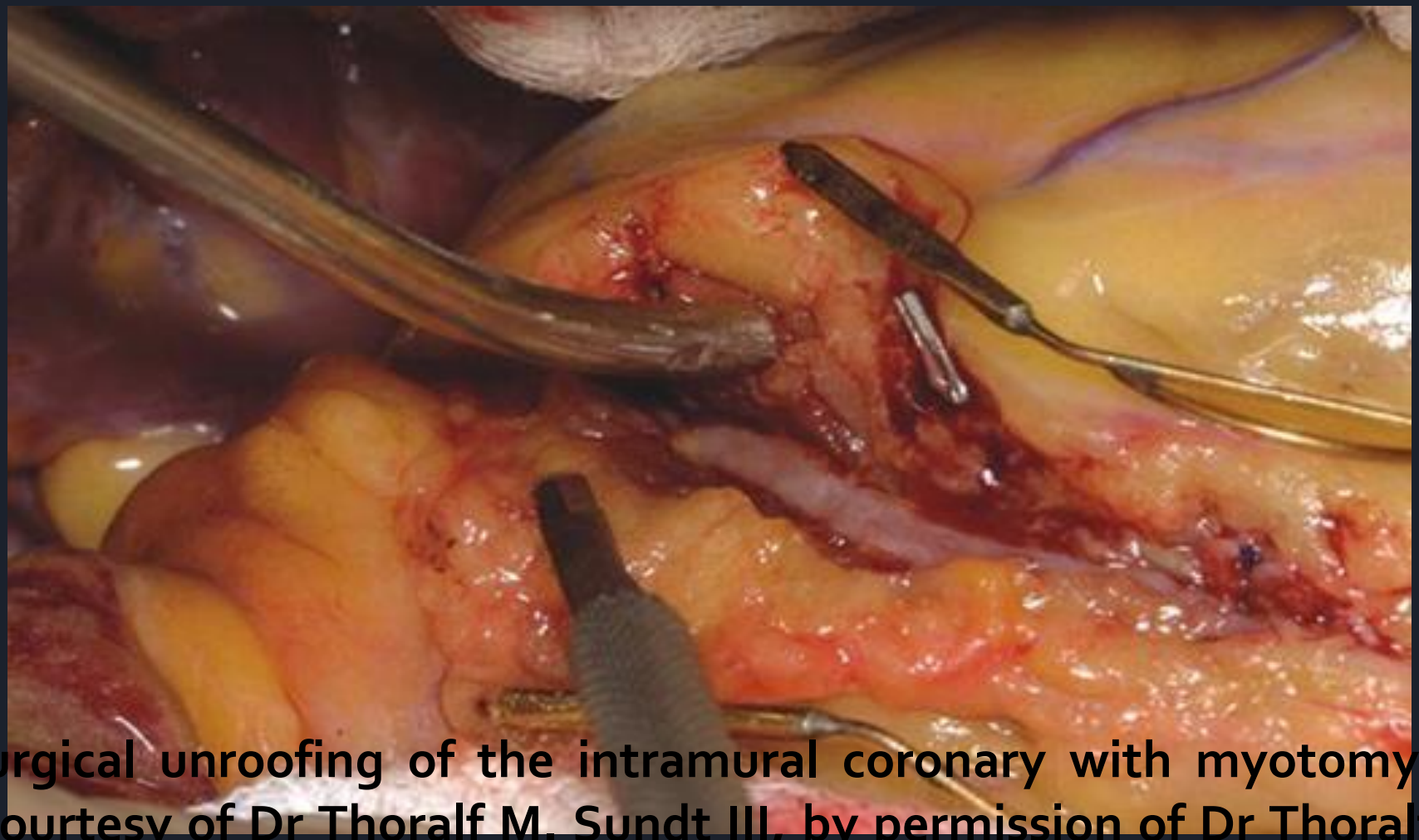
Some studies demonstrate that stenting can abolish hemodynamic alterations and improve symptoms, although no studies demonstrate normalization of myocardial perfusion when a perfusion defect was present before stenting.

Moreover, high inflation pressures may be required for optimal stent implantation, which presents a higher risk of coronary perforation

INTRODUCTION 11.12

Surgical treatment with a dissection of the myocardial fibres that surround the myocardial bridge should be limited to patients with symptoms that persist despite medical treatment. Good clinical results have been reported in a small series of these patients, although this type of procedure is extremely delicate due to the possible appearance of dangerous complications, including right and left ventricular perforation

INTRODUCTION 12.12



Surgical unroofing of the intramural coronary with myotomy. (Courtesy of Dr Thoralf M. Sundt III, by permission of Dr Thoralf M. Sundt III.)

https://academic.oup.com/eurheartj/article/26/12/1159/524878

PATIENT PROFILE

- 41 years old
- Male
- Occupation: businessman
- Date of admission - 21.02.18

MAIN COMPLAINTS

- Chest pain (onset, strong, pressing, periodical, lasting for 10 - 15 minutes, more often after physical activity)
- Dyspnea (expiratory, attack-like, after emotional overstrain, hard physical exercise)
- Palpitation (paroxismal)
- Headache (occipital region, pressing character, slight intensity
- Heartburn (periodically)

ANAMNESIS MORBI

- Listed complaints were over 10 years
- Arterial hypertension more than 10 years (adapted to 140-150/90 mm Hg) Patient took enalapril + hydrochlorothiazide 10mg/12.5 mg once a day.
- According to the patient, he complained of dyspnea after hard exercise and increase of blood pressure
- February 2018 – worsening of the disease, with complaints of chest pain and dyspnea

ANAMNESIS VITAE

- Infections, injuries, tuberculosis, sexually transmitted diseases were denied.
- Allergological history is not burdened.
- Denies smoking and alcohol abuse
- Hereditary (Father - died of a stroke; mother has arterial hypertension)
- Likes spicy food and fried fish

OBJECTIVE EXAMINATION 1.3

- GENERAL CONDITION: The general condition of the patient is satisfactory and there is no obvious physical disability or distress.
- Weight: 81 kg, height: 174 cm, BMI - 26.8 kg/m (*overweight*)
- Skin and visible mucous: membranes are clean, pale pink and acyanotic
- Peripheral lymph nodes are not palpable
- Thyroid gland is normal size, consistency, not soldered to surrounding tissues. Symptom of

swallowing is negative

OBJECTIVE EXAMINATION 2.3

- Cardiovascular system: heart rate = 68 bpm, pulse= 68 bpm;

On auscultation the apex beat is at the 5th intercostal half an inch to the left of the midclavicular line.

BP sin: 160/90 mmHg, BP dextra: 165/94 mmHg

OBJECTIVE EXAMINATION 3.3

- Respiratory system : pulmonary percussion

-resonant sound. Pulmonary auscultation

-vesicular breathing (no adventitious sounds)

- Abdomen and GI: is normal on palpation, no rebound tenderness, painless on palpation. The liver and spleen are painless on palpation
- There is no pitting edema of the lower limbs
- Pasternasky's sign is negative

PRELIMINARY DIAGNOSIS

- Unstable Angina
- Arterial Hypertension, stage II

RECOMMENDED EXAMS

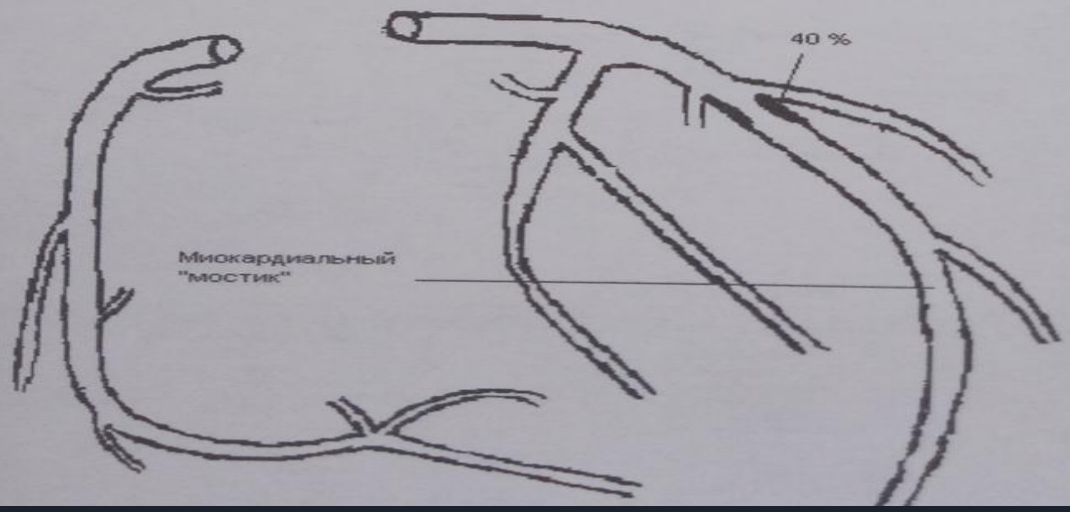
- Complete blood test
- General urine test
- ECG
- EchoCG (Intracoronary ultrasonography and Doppler studies)
- Coagulogram
- TSH, T_4, anti-thyroid gland antibodies (anti-TG Ab)
- Biochemical panel
- Coronary Angiography
- Biomarkers: troponin and BNP levels
- Renal function tests (creatinine)
- Blood lipid spectrum

ANGIOGRAPHY

Заключение:

Правый тип коронарного кровоснабжения.
Левая коронарная артерия - атеросклероз ствола, передняя нисходящая артерия - атеросклеротическая бляшка 40 % после отхождения первой диагональной артерии, протяженный миокардиальный "мостик" с систолической компрессией до 70 % в среднем сегменте, огибающая артерия с признаками атеросклеротического поражения без гемодинамической значимости.
Правая коронарная артерия с признаками атеросклеротического поражения без гемодинамической значимости.

Схема



Conclusion: shows 40% narrowing of the Left anterior descending artery by an artheroscleroctic plaque, the myocardial bridge with systolic compression up to 70 %

LIPID PROFILE

	RESULT mmol/L	NORMAL mmol/L
Cholesterol	4.45	<5.3
VLDL	0.36	<1.0
LDL	2.82	<3.5
HDL	1.27	>2.9
TG	0.80	<2.3

Conclusion: All the lipids are within the normal ranges indicating a good response to treatment and a favourable prognosis

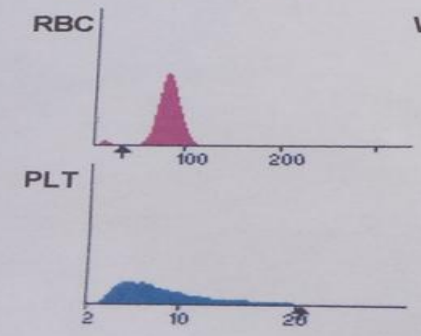
BLOOD TEST

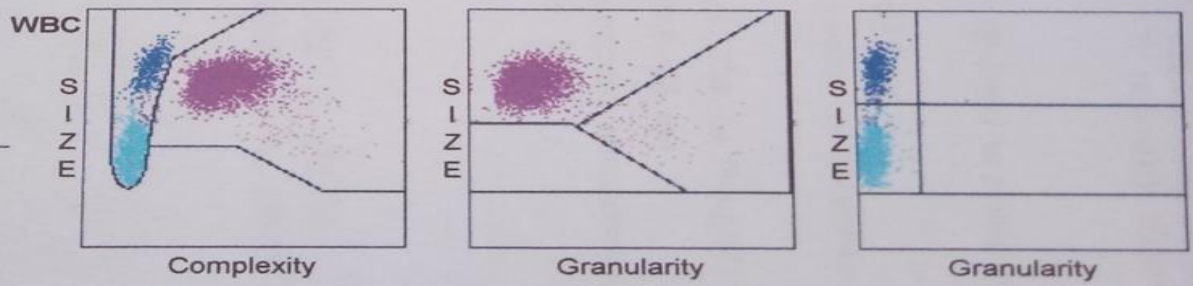
	RESULT	NORMAL
CREATININE	77 mcrmole/L	44 – 80 mcrmole/L
GLUCOSE	5.38 mmol/L	3.9 – 6.4 mmol/L

Conclusion: Blood glucose and creatine are within normal range indicating normal kidney function

CBC

WBC	7,0	[10^9/L]		(4,0 - 9,0)	[WBC FLAG]
NE	4,1	59,1	[%]	(1,7 - 7,7 / 47,0 - 72,0)	
LY	2,2	31,1	[%]	(0,4 - 4,4 / 19,0 - 37,0)	
MO	0,5	7,3	[%]	(0,0 - 0,8 / 3,0 - 11,0)	
EO	0,1	1,7	[%]	(0,0 - 0,6 / 0,5 - 5,0)	
BA	0,1	0,8	[%]	(0,0 - 0,2 / 0,0 - 1,0)	
RBC	4,93	[10^{12}/L]		(4,00 - 5,00)	[RBC FLAG]
HGB	156	[g/L]		(130 - 160)	
HCT	43,8	[%]		(40,0 - 48,0)	
MCV	88,8	[fL]		(80,0 - 100)	
MCH	31,6	[pg]		(28,0 - 36,0)	
MCHC	356	[g/L]		(310 - 370)	
RDW-CV	13,8	[%CV]		(10,0 - 16,5)	
PLT	173 L	[10^9/L]		(180 - 320)	[PLT FLAG]
PCT	0,15	[%]		(0,10 - 1,00)	
MPV	8,7	[fL]		(5,0 - 10,0)	
PDW	17,7	[%]		(12,0 - 18,0)	

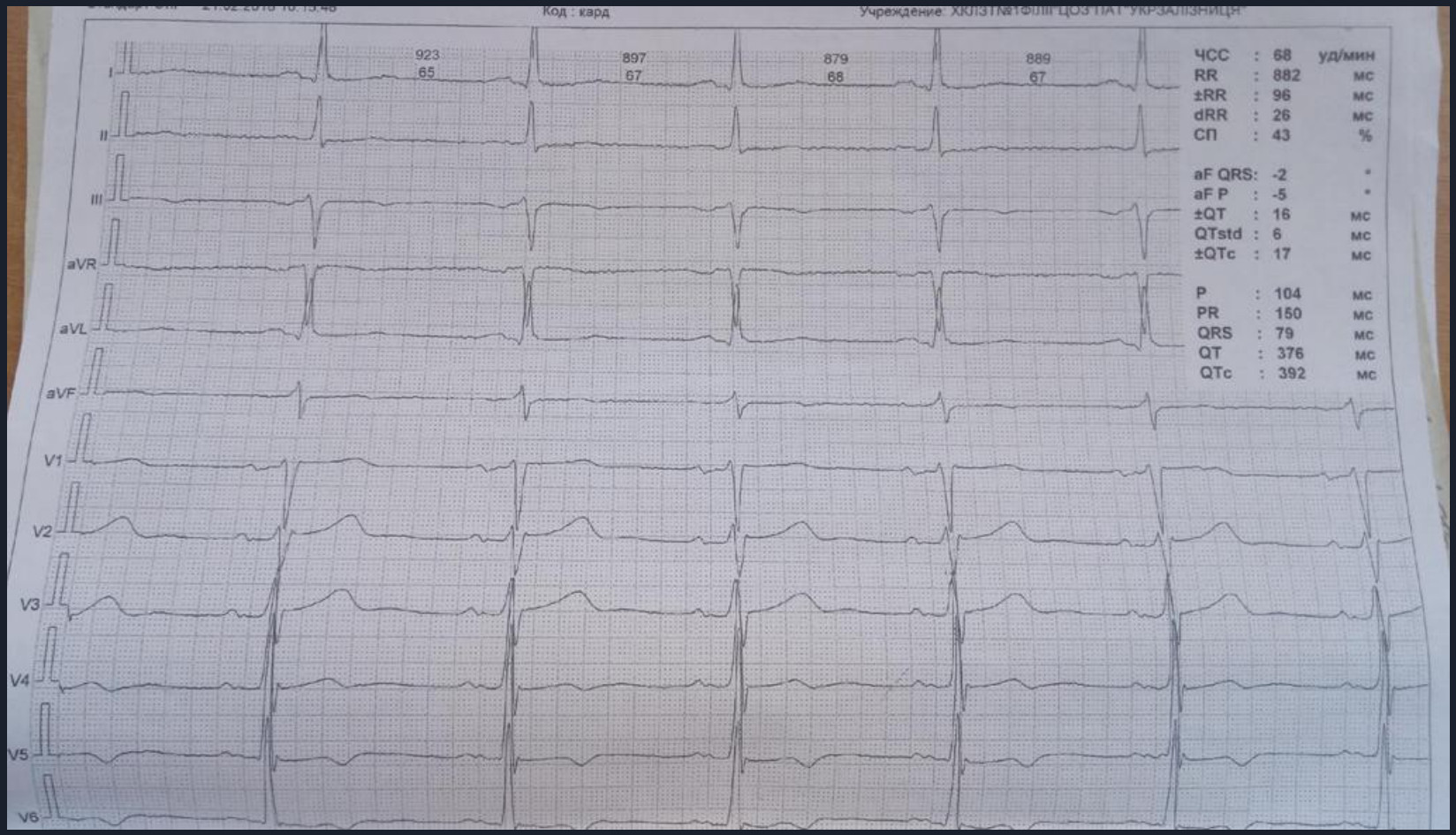




[ALARM]

Conclusion: Normal

ECG



Conclusion: Sinus rhythm. Signs of left ventricular hypertrophy

THYROID FUNCTION TESTS

Conclusion: within normal range

ECHOCARDIOGRAPHY

Conclusion: EF - 61 % (N= 55-78 %). Based on the echocardiograph the patient had asymmetric hypertrophy of the interventricular septum and hypertrophy of the left ventricle (concentric hypertrophy) and tricuspid regurgitation of the 1 st stage.

THERAPEUTIC STRATEGY FOR MYOCARDIAL BRIDGING

(adapted from Schwartz et al)

	Clinical Symptoms	Signs of Ischemia	Initial Treatment Strategy	Secondary Treatment if No Improvement
Type A	Yes	No	Reassurance	–
Type B	Yes	Yes, by non-invasive stress testing	Beta-blockers	Intracoronary hemodynamic evaluation→ surgery or stenting if abnormal
Type C	Yes	Yes, by altered intracoronary hemodynamics	Beta-blockers	Surgery or stenting

RECOMMENDED TREATMENT

- Coronary stenting of LCA
- Nebilet (Beta – Blocker + NO) 5 mg daily
- Noliprel (ACE inhibitor + Thiazide) daily
- Cardiomagnil (Aspirin + MgOH) 75 mg daily
- Plavix (clopidogrel bisulfate) 75mg
- Omeprazole 40 mg daily

FOLLOW-UP
(3 month later)

- Coronary stenting of LCA was done.
- The patient takes medication regularly.
- Patient's condition is much better:

no chest pain, no palpitation, exercise tolerance increased.

CLINICAL DIAGNOSIS

Myocardial bridge of LCA with systolic compression 70%. Coronary stenting of LCA.

Essential Arterial Hypertension, second grade, Heart failure with preserved ejection fraction II FC, stage A

OUR RECOMMENDATION

- The patient should have adequate rest to prevent working the heart as he has unstable angina.
- The patient should also avoid fatty and spicy foods as he has heartburn and was given omeprazole.

PROGNOSIS

- The patient was in good physical condition and most of the lab tests were within the reference ranges.
- There was no infarction of the heart tissue.
- The stenosis and the myocardial bridge was surgically treated
- Overall the prognosis is favourable with his chances of survival being positive

CONCLUSION

- the patient diagnosed with unstable angina, myocardial bridge and hypertension appears to be healthy and satisfied with his treatment
- considering the epidemiological prevalence of these conditions, clinical suspicion of a myocardial bridge should be considered in all cases of typical or atypical chest pain; particularly in young patients with a low probability of atherosclerosis (which is confirmed by our clinical case) there is a connection with it) who are free from traditional cardiovascular risk factors

LINKS

- https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4065198/ (myocardial bridge)
- https://en.wikipedia.org/wiki/Myocardial_bridge (myocardial bridge)
- https://en.wikipedia.org/wiki/Acute_coronary_syndrome (acute coronary syndrome)
- https://www.pinterest.com/pin/173811485331207 57/ (human heart)
- http://ctisus.com/responsive/learning/features/illustrated-coronary/anomalous-anatomy-10 (myocardial bridge)

THANK YOU FOR YOUR ATTENTION !!!