

# ATYPICAL CASE OF SYNCOPE AS A CHEST PAIN EQUIVALENT IN PATIENT WITH ACUTE MYOCARDIAL INFARCTION

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# INTRO

- Syncope may be the initial complaint in 5-12% of patients with acute myocardial infarction (MI) and is related to arrhythmia or pump failure.
- Vasovagal reactions, bradyarrhythmia and atrioventricular blocks can be caused by ischemia.
- Patients suffering from acute mi are susceptible to neurally mediated syncopal or presyncopal attacks.
- Sympathetic withdrawal seems to be one of the most likely mechanism of syncope in patients with acute mi.

In the general population, frequent premature ventricular contractions (PVCs), which are defined as the presence of at least 1 PVC on a 12-lead ECG or >30 PVCs per hour, are associated with increased cardiovascular risk and increased mortality.

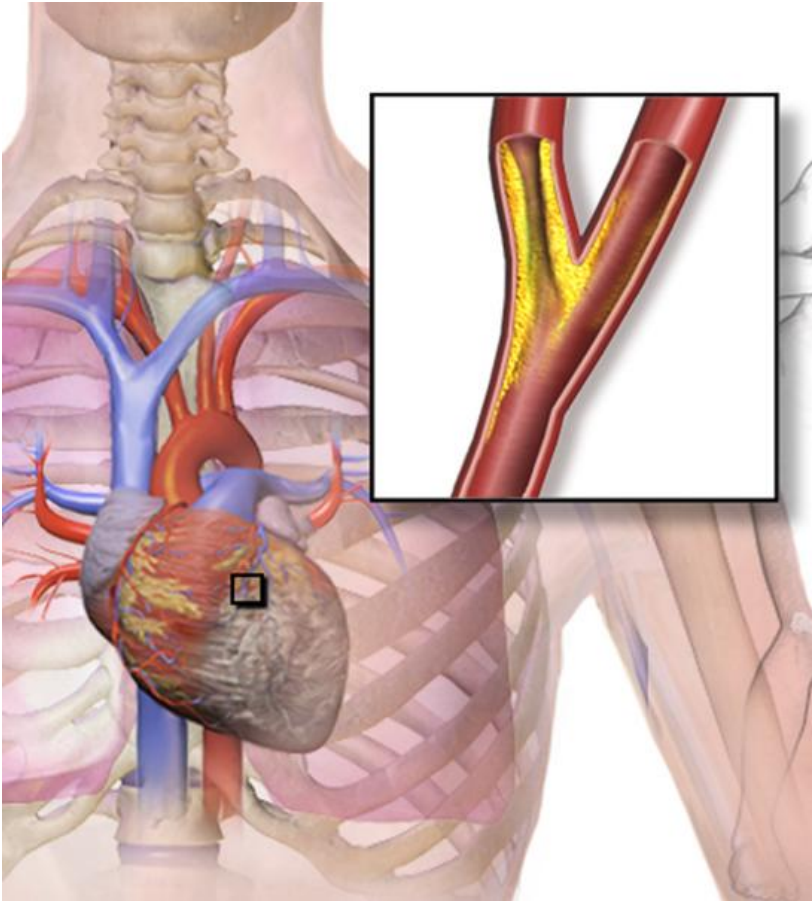
PVC and nonsustained ventricular tachycardia (NSVT) in patients with cardiovascular disease are common and have been associated with adverse outcomes.

# OUR PATIENT

- ▶ Patient M
- ▶ 80 year old
- ▶ Scientist
- ▶ City resident



# COMPLAINTS

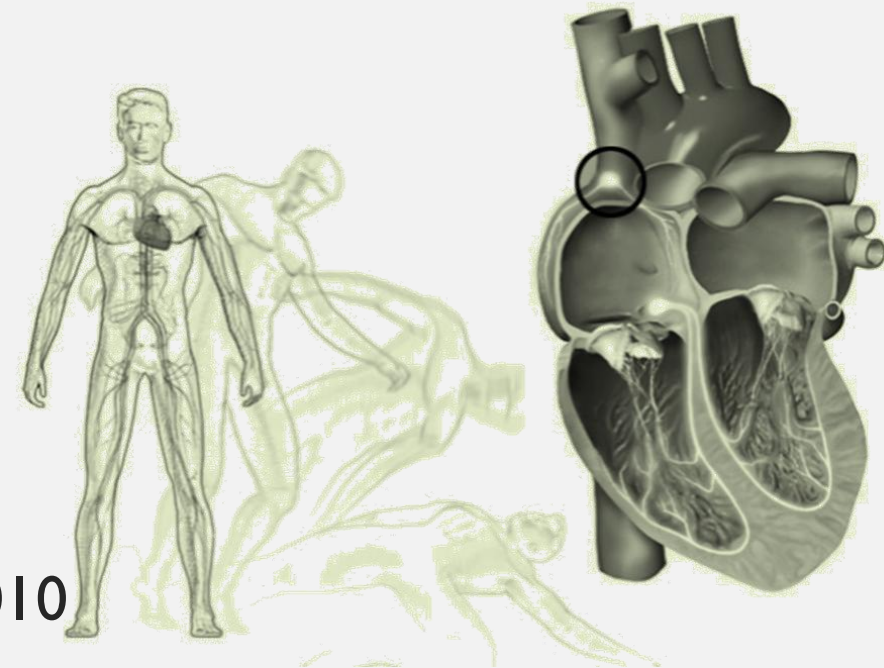


Was hospitalized 15/01/2019 with complains of:

- ▶ Pressing chest pain with irradiation to the left shoulder after moderate physical activity
- ▶ Chest pain was relieved by taking nitroglycerin
- ▶ Dyspnea
- ▶ Pitting edemas
- ▶ Deterioration over 2 month

# ANAMNESIS MORBI

- ▶ In 2005 suffered – acute Q-positive posterior-diaphragmal MI and acute anterior-septal MI- in 2006. In both cases during daily routine activity the onset of acute cardiovascular event was presented only by syncope episode.
- ▶ Ischemic heart disease since 2001
- ▶ First experience of retrosternal pains in 2006
- ▶ Blood pressure levels usual are 130/70, max-170/100
- ▶ Hypertensive retinal angiopathy since 2005
- ▶ Dyscirculatory encephalopathy of 2<sup>nd</sup> degree since 2010



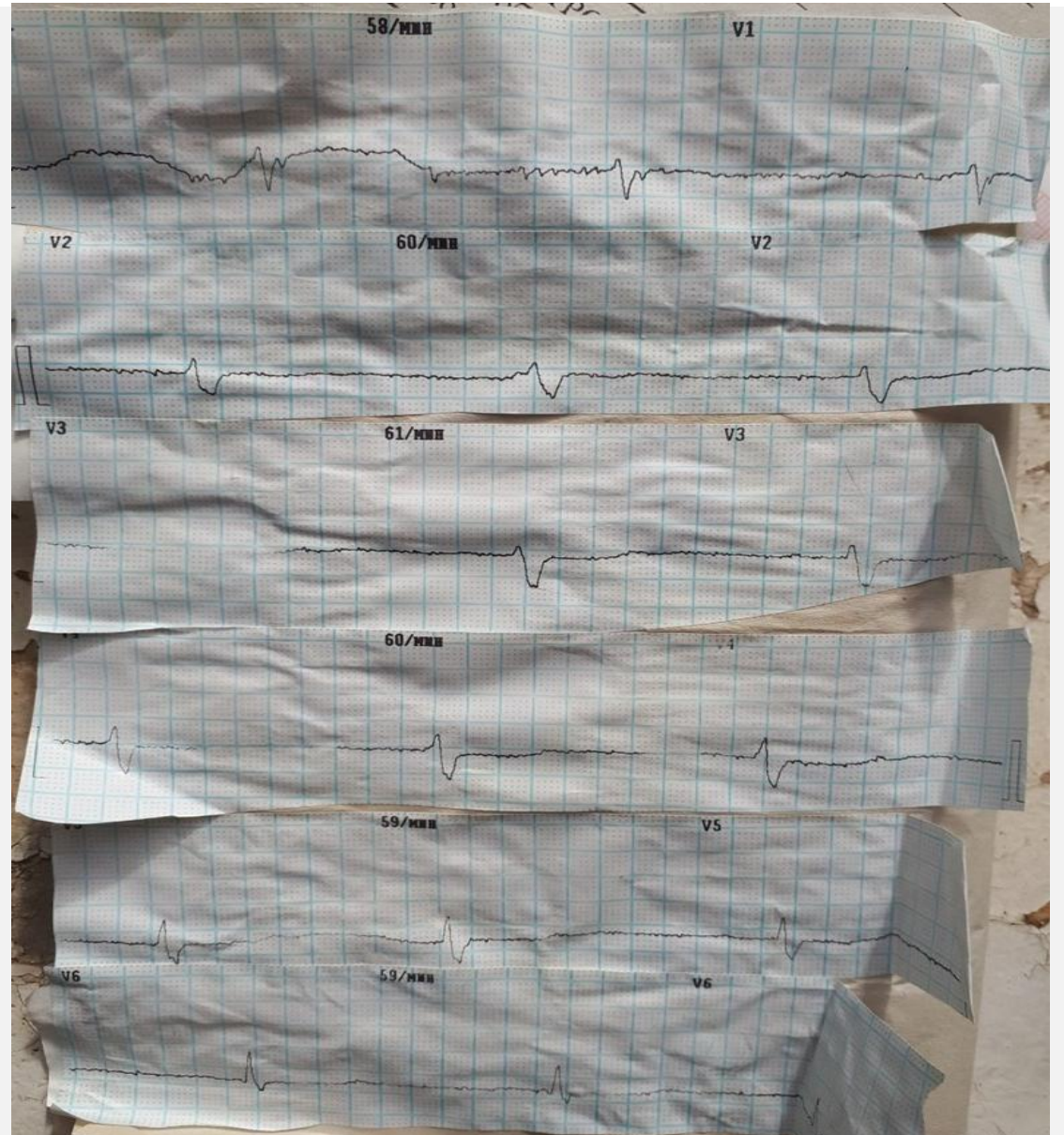
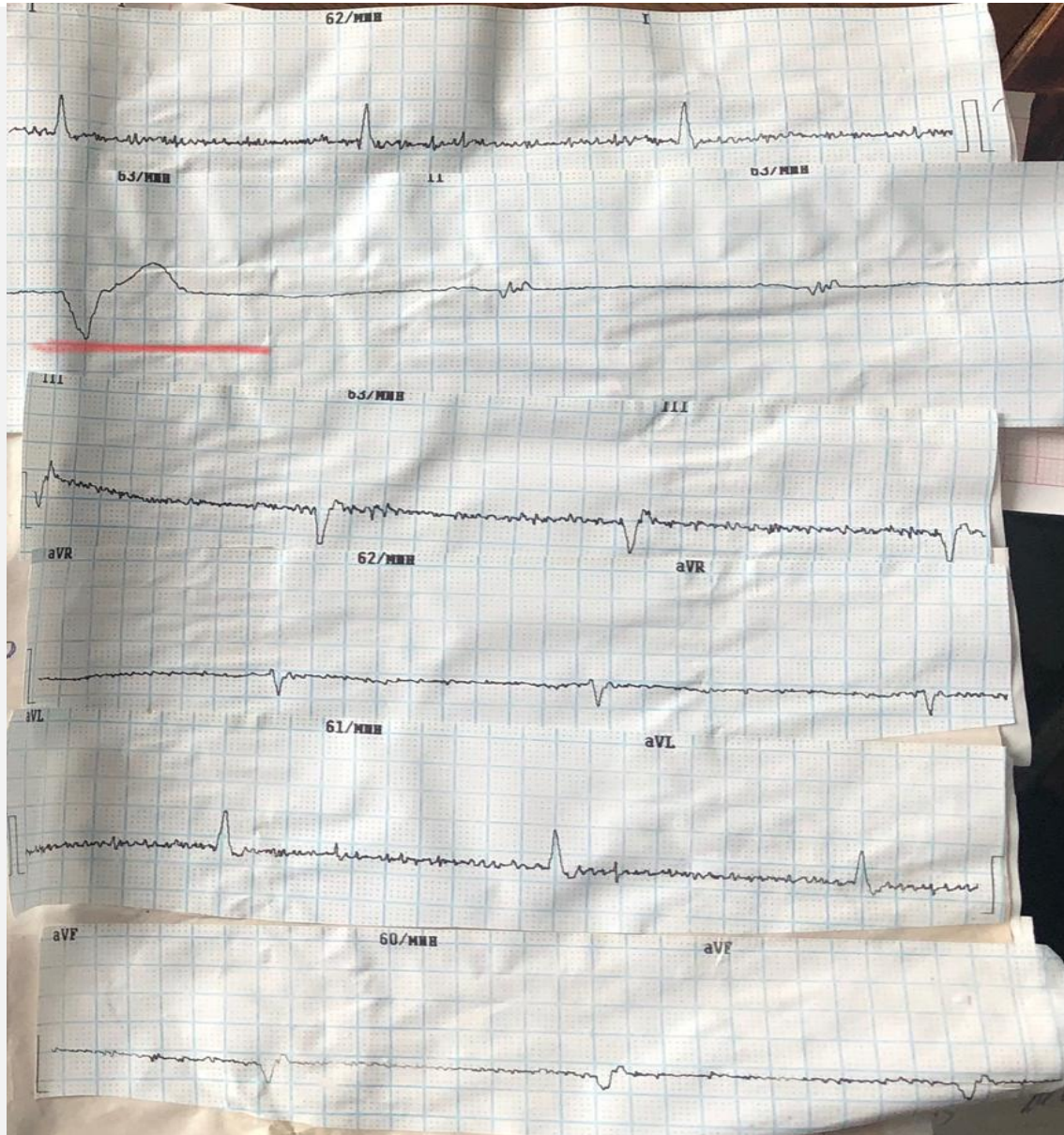
# ANAMNESIS VITAE

- ▶ Non diabetic
- ▶ No history head trauma or epilepsy
- ▶ Hereditary diseases were not identified
- ▶ Allergic history is not burdened
- ▶ Sexually transmitted diseases were denied
- ▶ CV disease family history: absent
- ▶ Chronic bilateral hearing loss since 2010
- ▶ Chronic non-calculous cholecystopancreatitis

# OBJECTIVE STATUS

- Consciousness - clear, state – moderate severe, body position – active
  - Patient can orient himself in place, time, his personality
- Weight -90 kg, height- 167 cm, waist circumference-105, **BMI- 33**
- Pale skin and mucosae; Musculoskeletal system – no changes
- BR (breathing rate) – 18 /min
- Lung percussion: no clinically significant changes
- Lung auscultation: Vesicular breathing weakened in the lower parts
- **Heart borders: right - right edge of the sternum; left - 1.5 cm left of middle clavicle line; upper - in 3<sup>rd</sup> intercostal space**
- **Heart auscultation: arrhythmic, extrasystoles 2-3 in min, heart tones –deaf,**
- **Pulse – arrhythmic, 60 bpm;** Blood pressure (BP) 110/80 mm Hg (over background of antihypertensive therapy)
- Abdomen - enlarged, symmetric, unpainful; Edemas - moderate
- **Liver: + 1.5-2 cm,** no pain during palpation of right hypochondrium; Spleen: normal
- Pasternatsky symptom – negative from both sides

- 15.01.19 ECG revealed sinus rhythm, heart rate 60 in min, the electrical axis of the heart is deflected to the left, ventricular premature contraction, repolarization abnormalities in V4-V6.



# 24HR-ECG MONITORING

- During observation displacement of ST segment not detected.
- Circadian oscillations persist - a tendency to slow down the rhythm
- **Bradycardia** at night - up to 40 per minute
- Up to 30 pauses 1700 ms/day, mainly at night
- Signs of AV blockade is not revealed



## Rhythm disturbances:

- Supraventricular extrasystoles up to 31 per hour
- Short-term allorhythmias such as bigeminies, couplets rare,
- Triplet on 01/17/19 (during activity)
- Ventricular extrasystoles - 31 per hour, predominantly single, short-term allorhythmias of the bigeminy type, monomorphic, group and early extrasystoles were not detected - grade I by Laun.



# COMPLETE BLOOD COUNT



**Conclusion:** no clinically important changes

	<u>15/01/19</u>	Normal Range
Hemoglobin, g/l	142	130 - 160
Red blood cells, 10 <sup>12</sup>	4.63	4.0 - 5.0
Ht	46	40-48%
White blood cells, 10 <sup>9</sup>	7.8	4 - 9
ESR, mm/h	7	1 -10
Bands	2	1.06 - 6%
Segments	68	47 - 72%
Eosinophils	3	0.5 - 5%
Monocytes	2	0.1 - 3%
Lymphocytes	25	19 - 37 %
Platelets	263	180-320

# BIOCHEMISTRY TEST DATA

**Conclusion:** no clinically important changes

	Patient's ranges,	N
Glucose (mmol/l)	3.8	3.38 - 5.55
Total bilirubin, mkmol/l	17.7	8.6 - 25.5
AST, $\mu\text{mol/h*ml}$	0.2	0.1-0.445
ALT, U/l	0.4	0.1-0.68
Prothrombin index (%)	87%	85-105
Creatinine, $\mu\text{mol/l}$	84	80 - 115
K (mmol/l)	4.2	3.5-5.1
Ca (mmol/l)	2.13	2.15–2.50
Na (mmol/l)	136	135-145
CL (mmol/l)	100	98–107
C-reactive protein	negative	
Total cholesterol	3.3	< 6.2

# HEART ULTRASOUND

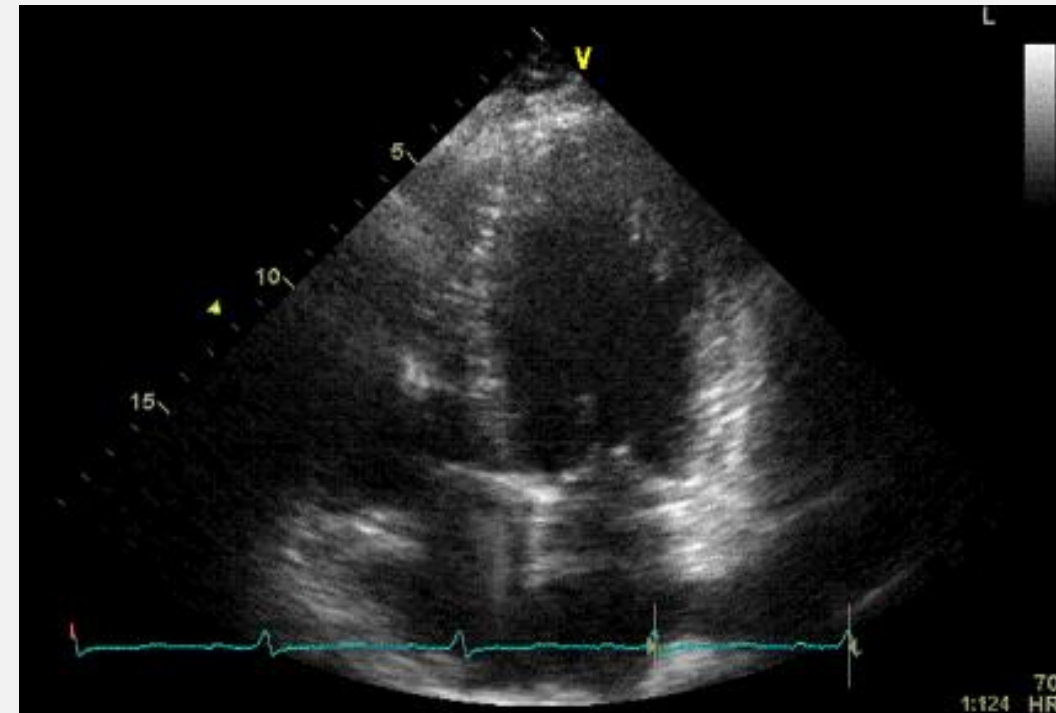
During an heart ultrasound was found

- ▶ Hypokinesia of left ventricle posterior wall
- ▶ Global contractility decline with EF -44%
- ▶ Dilation of left atrium

No pulmonary hypertension,

- ▶ Atherosclerotic changes of aorta present

No ventricular septal defect or mitral valve significant changes were noted.



# COMPLETE DIAGNOSIS

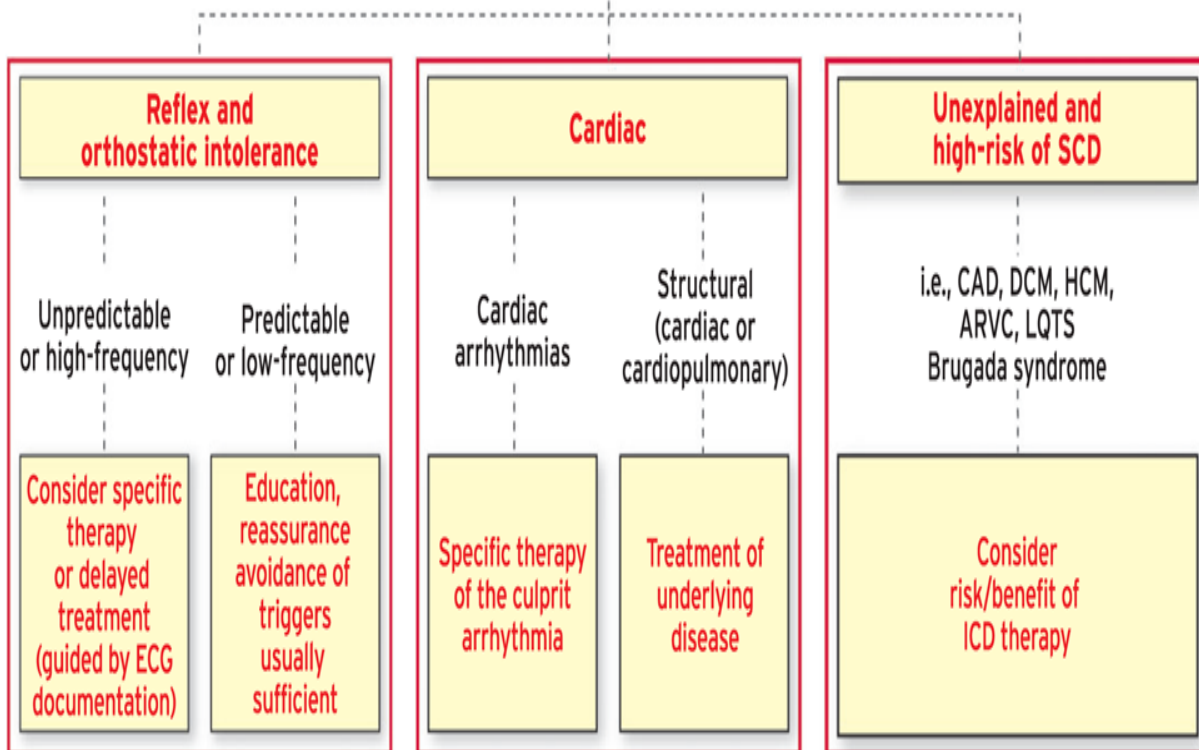
- ▶ **Main:** *Ischemic Heart Disease*:- stable angina III functional class, postinfarction (2005, 2006 – Q-positive) and diffuse cardiosclerosis.
  - Aortic sclerosis . Arterial Hypertension III degree, 2 stage, very high risk.
  - Extrasystolic arrhythmia grade I by Laun.
- ▶ **Complications:** Chronic Heart Failure IIB stage with left ventricle systolic dysfunction (EF – 44%)
- ▶ **Concomitant diseases:**
  - Dyscirculatory encephalopathy of 2<sup>nd</sup> degree
  - Chronic bilateral hearing loss, hypertensive retinal angiopathy
  - Chronic non-calculous cholecystopancreatitis

# GENERAL PRINCIPLES OF TREATMENT OF SYNCOPE

- General framework of treatment is based on risk stratification and the identification of specific mechanisms when possible.
- In our clinical case - it is impossible to establish the real cause of the syncope.
- Since the changes in the available ECG reduce the structural changes in the myocardium, which occurred due to two myocardial infarctions.
- And this patient can be associated with a high risk of sudden death

## Treatment of syncope

### Diagnostic evaluation



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ARVC = arrhythmogenic right ventricular cardiomyopathy; CAD = coronary artery disease; DCM = dilated cardiomyopathy; ECG = electrocardiographic; HCM = hypertrophic cardiomyopathy; ICD = implantable cardioverter defibrillator; LQTS = long QT syndrome; SCD = sudden cardiac death.

# FOR PREVENTION OF FURTHER SUDDEN CARDIAC DEATH PT SHOULD BE RECOMMENDED

- ▶ For secondary prevention, individuals aged 75 years with clinical atherosclerotic cardiovascular disease (ASCVD) should be started on high-intensity statin therapy unless contraindicated

## The goals

- ▶ BP <140/90 mm Hg in all patients - initiate or maintain lifestyle modification, weight control, increased physical activity, alcohol moderation, sodium reduction, and increased consumption of fresh fruits, vegetables, and low-fat dairy products.
- ▶ achieving low-density lipoprotein cholesterol (LDL-C) levels below 100 mg/dL; if triglyceride levels are 200 mg/dL or above, non-HDL-C (non-high-density lipoprotein cholesterol) levels should be below 130 mg/dL. (Non-HDL-C = total cholesterol – HDL-C.)

## Therapies

- ▶ Initiate 12 month course of clopidogrel 75 mg/day in combination with aspirin in patients after acute coronary syndrome
- ▶ Initiate indefinite course of beta-blockers in all patients who have had MI, ACS or LV dysfunction with or without heart failure symptoms, unless contraindicated.
- ▶ Beta blockers reduce all-cause mortality and SCD in patients with HF with reduced EF and have long been proven to reduce mortality after MI
- ▶ Treatment of PVCs with antiarrhythmic medications has not been shown to reduce mortality and An electrophysiological study may be needed
- ▶ In patients with heart failure with reduced ejection fraction (HFrEF) (LVEF  $\leq$ 40%) - treatment with a beta blocker, a mineralocorticoid receptor antagonist and either an ACE inhibitor, an angiotensin-receptor blocker, or an angiotensin receptor-neprilysin inhibitor is recommended to reduce SCD and all-cause mortality

*The End*

