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CHEST PAIN SYNDROME

Neset Uzairi

FSHM, University of Tetova Macedonia,
Clinical Hospital Tetovo, University of Tetova, Faculty of Medicine
Corresponding Author: e-mail: neset.uzairi@unite.edu.com

Abstract

Introduction: Cardiovascular diseases are in the first place in the statistics of the western countries as the cause of death. Coronary arteries diseases are the main cause of pain in chest known as angina pectoris, a clinical syndrome characterized by retrosternal pain tending to spreading toward the shoulders, left hand, lower jaw, neck and back, often with atypical symptomatology. In addition to aa coronary disease there are other cardiovascular factors such as diseases of aortas. Mitral valve prolapse, cardiomyopathies, pericards, emboli and pulmonic hypertension and other. As neuromuscular, digestive gastrointestinal, pleurites and anxious states. Ischemia of myocardia happens because of the inability of coronary arteries to supply the heart muscle with blood and oxygen requirements because of the aa. coronary atherosclerosis that lead to their stenosis or occlusion of the spasm of a coronary. According to Statistics for 2005 from cardiovascular disease have died 17.5 million, while 7.5 million for heart attack situation similar to 2017. The Republic of Macedonia is closer to European trends regarding morbidity and mortality of these diseases.
Aim of this paper: is to present data concerning the appearance of morbidity and mortality of coronary syndrome and to bring a conservative view on the strategy and data along with invasive world authors in this area.
Materials and Methods: There were analyzed 64 patients with heart ischemic disease; 37 men and 27 women aged 25-78; the average age for women was 56.5 years old and 54.9 years old for men who were treated in the above-mentioned institution in Tetovo, clinical hospital and cardiology clinic in Skopje in the last five years. Based on methods used as objective history and examination, monitoring and screening for cardiovascular risk factors of arterial blood pressure, diabetes and hyperlipidemia, ECG with specific changes as omission of ST-T segment and negative T wave, dynamic ECG, coronary stress test Echocardiography, laboratory and enzymatic methods, AST, ALT,, LDH, CK and CK_MB, troponins, invasive methods such as selective coronagraph and myocardium coronarography with TL 201 that was done in the cardiology clinic in Skopje and Tetovo in the doubtful cases.
Results: Judging on this data anamnesis was positive for cardiovascular diseases and risk factors were present in the overwhelming part. Objective data were scarce almost negative, rapid increase of arterial blood pressure and transitional extra systolic arrhythmia; ECG changes had specific S_T segment and negative T-wave. We found coronagraph stenosis changes 50-80% of coronary 13 (20.3%) of cases with dominance of the LAD in 60% of cases, while other branches of RCA, CX-4, with LMCA with less attacks while in 2 (3.1%) cases chorography was normal. Myocardial scintigraphy of was in positive correlation with selective coronagraph in detecting the ischemic disease at 9 (14%) of cases. Most attacked age for men 50-60 years and 60-70 years for females.
Discussion and conclusion: Apart from elimination of risk factors mentioned as hypertension, diabetes hyperlipidemia should look at thyroid glands, anemia and aortas diseases. Drug therapy started with Nitrates, ASA, Beta blockaders, calcium antagonists, ACE inhibitors, diuretics, antithrombotic and others. The invasive therapy consisted of, PTCA, stenting, myocardial revascularization.
Judging by the published data in European centers and in our country, there are two strategies in the treatment of these conditions: the early conservative strategy and early invasive strategy. At patients with elevations of ST segment intervening therapy is first. Patients at risk high, increased enzyme, rhythm disorders, ST segment dispersion to be treated 48-72 hours invasively because of the hemodynamic instability.
Despite continuous screening these diseases and risk factors of these diseases are increasing.

Keywords: angina pectoris, pain, coronary syndrome, invasive treatment, revascularization.

Introduction

Syndrome of pain in the arms implies broad constellation of causes and consequences cited in medical literature, but the most common clinical syndrome is angina pectoris.

Cardiovascular Diseases take the first place in Western Country Statistics as causes of Death {1,2,4,9,10]. Clinical manifestations of ischemic heart disease are: 1. angina pectoris 2. coronary acute syndrome 3. heart rhythm disorders. 4. Decompensation of the heart 5. Sudden death. Angina pectoris clinical syndrome is characterized with retrosternal pain with the tendency to spread to the shoulders, left hand, lower jaw, neck and back. The symptomatology of this clinical entity is often atypical, so the place and character of worries often change.

Pathogenesis: The inability of coronary aa. to supply the muscle with needed amount of blood due to atherosclerosis of the coronary arteries leading to their stenotic or occlusive changes, which consequently have regional blood flow disorders and O2 deficiency, and an imbalance between supply and demand of myocardium for energy. Ischemia occurs due to the reduction of blood flow in those areas which consequently reduces the contractor's myocardial function known by some authors as perfusion-contractual matching [1,14,15 21].

The clinical manifestation of all this is severe pain in the chest known as Angina pectoris. The other explanation is that unsophisticated non-oxygen products irritate the sensory fibers in the myocard, thereby creating a pain sensation in the visceral. Angina pectoris is divided into stable, unstable forms; Prinzmeta Angina and X-ray Syndrome with normal aa coronary artery but coronary disorders of S-T during the test of the ventricle and left disorders of left ventricle are evidenced by the radionuclide method [1,2,3,11,15,19,20].

The well-known functional division of Angina pectoris patients according to the Canadian Association of Cardiologists (1976) is in four functional groups: Group I, II, III and IV, with angina pectoris based on physical activity, character and appearance of pain under pressure or in rest. There is also NYHA classification. Classical, atypical and equivalent forms of angina pectoris (dyspnea) which in the most advanced forms of heart decompensation may mask the picture of equivalent angina.

Regarding the clinical expression of angina pectoris, pain may be light, severe, chest pain, fear, breathing problems, depression, weakness, discomfort, commonly reported when walking downhill or climbing stairs, physical work, accelerating emotional crises heart failure. The pain lasts 1-5 minutes, and end after stopping physical activity or removing the factors that cause it, or after taking Tbl Nitroglycerin. During angina pectoral, a rapid increase in arterial blood pressure is transient as well as extracellular and hypothyroidism.

Physical status of the patient out of the angina attacks is in the order. Heart rate is increased during the load and it can have bradycardia at atypical forms. Objective data are almost negatives and this aggravates the diagnosis. Here, the history has great value in discrete detection of diagnosis. In the ECG we find changes as a result of ST segment, T-negative wave, ST segment leakage to standard derivatives below 2mm and most commonly in left-handed derivations. ECG out of the attacks is approximately 50% in order, the second part is cicatrix from earlier infarction, pulmonary rupture, blockage of the ventricles and left ventricular hypertrophy.

We emphasized that myocardial metabolism is aerobic and at certain heart rates and blood pressure if O2 is not in the required amount, it is developed ischemic development that is clinically expressed as angina pectoris, dyspnoea, cardiac arrhythmia or hypotension. And in ECG we have ST segment changes and T wave.

In coronarography we find narrowing of the left or right coronary arteries 50-80%;

It is a method that shows the panoanatomy of the coronary arteries in vivo, It is carried out along with left ventricular ulceration to determine its function and to make a decision for prognosis and further therapy.

Cardiovascular diseases, ischemic heart disease and myocardial infarction are on the rise in transition countries, while in decline by 25% in western countries due to the campaign for these diseases, new methods and new medications and application of pre-hospital restoration. According to WHO: 37% of people die of cardiovascular disease, of which 75% of coronary diseases [4,9,11,16,]. According to the WHO for 2004, 12.2% of deaths have been from ischemic diseases. According to statistics for 2005, 17.5 million people died from these diseases. That is 30% of total deaths, while 7.5 million from the heart attack. The Republic of Macedonia is getting closer to European trends in terms of these diseases.

Material, methods and results

According to the statistical data of the Center for Public Health and Coronary Unit Data at the Clinical Hospital Tetovo, in the period 2008-2012 and 2015-2016 from the total number of patients hospitalized with circulatory system diseases: (Ioo-I 99), with ischemic heart disease (I20-I25) were:

2008 Total hospitalized: 17479 ..... Circulatory system disease (I00-I99): 1740 ... ischemic disease: 428 m; 283, f145

2009: hospitalized (I00-I99) ... .1500 pt ... Angina pectoris: 170 pt. Infarct ac myoc: 171 pt

2010: hospitalized: (I00-I99) .... 1649 m: 845..f: 804, Angina pectoris--, Infarct ac myoc: 163, m: 103, f: 60
2011: hospitalized: (I00-I99) ... .1788 m: 936 ... f: 832, .. ischemic heart disease: 531 pt, m: 328, f: 203 pt.
Infarct ac. myocardi (I21.0_I21.9) were: 145; (102 m, and 43 f)
2012: hospitalized: (I00..I99) 1573 pt ... .. ischemic heart disease: 488 (261 and 227 f)

For 2015 with angina pectoris 1169 subjects and myocardial infarction 172 subjects, respectively 1098 versus 140 cases of myocardial infarction for 2016.

The purpose of the paper was to present data from our material related to appearance, incidence, morbidity, clinical manifestation of coronary syndrome and a look at the conservative and invasive strategy in addition to the data of the world's authors in this regard.

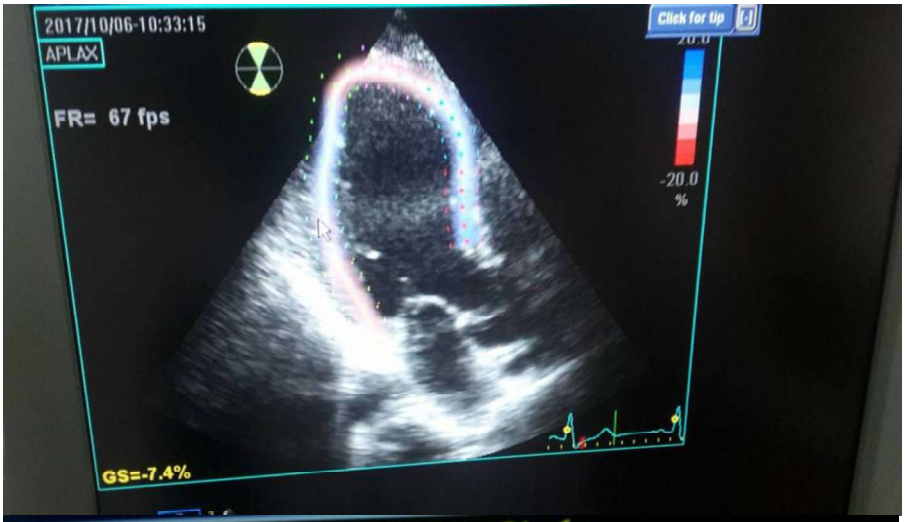
Material and methods: 64 patients were analyzed with ischemic heart disease out of 37 males and 27 females aged 25-78 years, the average age for women was 56.5 years and 54.9 years for males treated at the aforementioned institution, Hospital Clinic Tetovo and Cardiology Hospital Clinic in Skopje in the past five years. Based on the methods used as anamnesis and objective examination, following and screening of cardiovascular risk factors, arterial tension, diabetes and hyperlipidemia. ECG with specific segmental ST-T and T negative wave, dynamic ECG, coronary stress test, echocardiography, laboratory and enzymatic methods, AST, ALT, LDH, CK and CK_MB, troponin, invasive methods such as selective coronarography and myocardial scintigraphy with TL 201 done at the Cardiology Clinic in Skopje in suspicious cases.

Results

Anamnesis was important in determining the coronary artery disease and the nature of angina pain which was the leading symptom of angina pectoris. Based on the analysis, risk factors, such as hypertension, diabetes, obesity and smoking were present in most cases with a positive cardiovascular history, an important characteristic of chest angina was its connection to physical and emotional action. Patients this type of discomfort describe as anxiety that comes from their physical and psychic tension while relaxing during the break. The spread of pain on the arms and on its inner surface was a typical sign of chest angina but chest angina was not excluded even though the character and the site were atypical because myocardial ischemia is also characterized by neck, jaw, shoulder, jawbone , abdomen and spine but without complaints in the chest [1,16,17,18]. Objective data were few, rapid transient arterial tension elevation and extrasystemic arrhythmia, systolic noise of mitral regurgitation.Arteriosclerotic changes in the peripheral arteries were encountered less, as well as cardiomegaly in cases of previous infarction. In ECG there were specific changes in the ST segment and the T wave that appeared during angina pain and then disappeared, had diagnostic significance as ST segment shift with or without tidal wave T which was similar from all directions to that was caused by long coronary stress test, the ST segment can sometimes be elevated in myocardial infarction or angina Prinzmetal. The ECG out of attacks in nearly 50% of the patients was normal. Others could be found early cycatrix infarction, left ventricular hypertrophy, branch block, rhythm correction. An obviously normal ECG does not rule out coronary disease. Clinical manifestations of ischemic disease are: angina pectoris and its forms, silent angina, acute coronary syndrome, which means forms of myocardial infarction with and without elevation of the S-T segment (NSTEMI / STEMI) and unstable angina pectoris. The factual data are as follows:

Tab I.1:
Angina pectoris and acute coronary syndrome;

Cases:	**M: 37,**	**F: 27**					
(Male female cases		**Age (age group)**					
NSTEMI			**1**		**1**	**1:56**	**54 years**
STEMS			**4**	**2**	**6**	**9.3**	**39-70**
APNS / NSTEMI			**10**	**2**	**12**	**18.7**	**35-60**
Infarction happened		**10**	**4**	**14**	**21.8**	**39-77**	
APS and angina variants			**12**	**17**	**29**	**45.3**	**25-78**
Ischemia suspected in the STM			**2**	**2**	**3.1**	**52-69**	
37	**27**	**64**		**100%**			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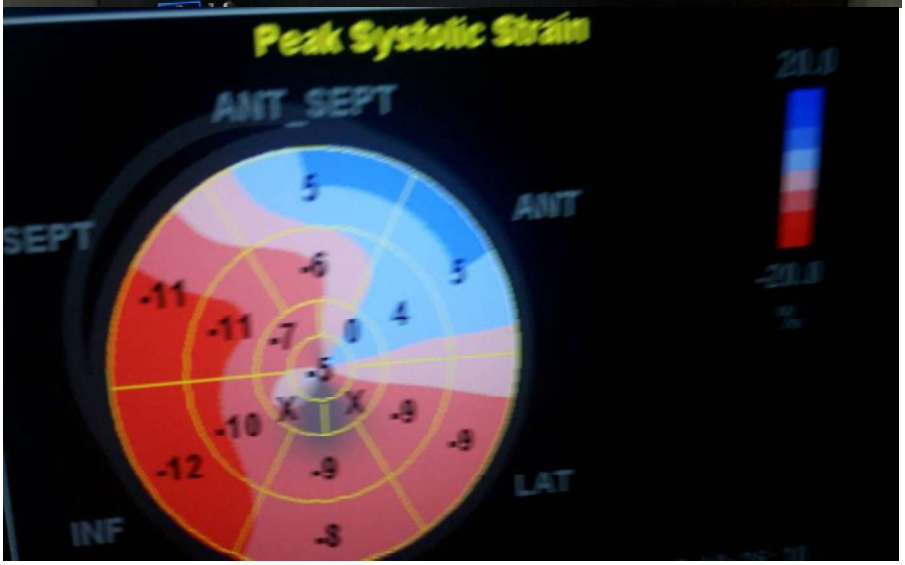
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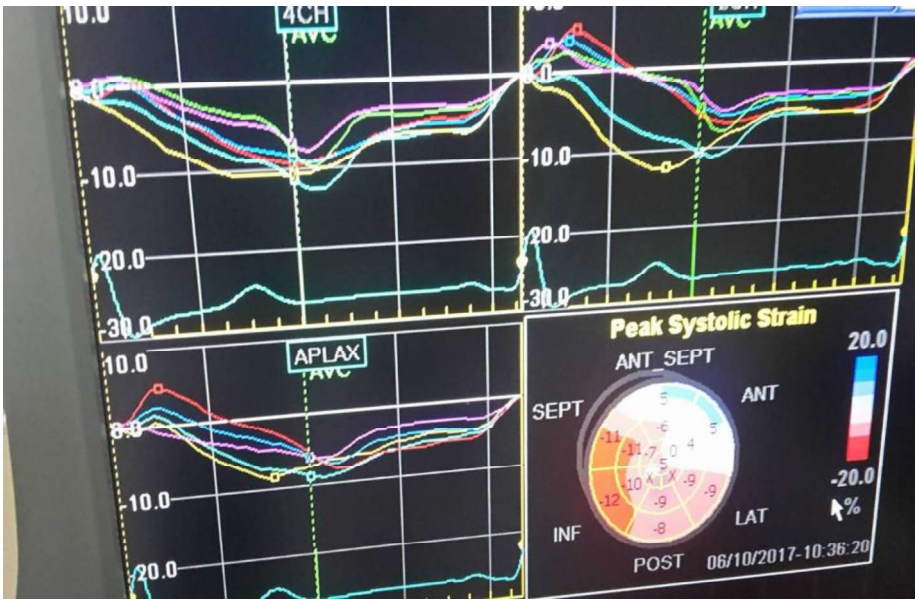
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In coronary artery we found these stenotic changes 50-80% of coronary arteries at 13 (20.3%) of cases. In 4 (6.25%) cases there was an occlusion of 99-100% RCA associated with stenosis of Cx 70-80% in 2 (3.12%), respectively stenosis of RD1 (90%) in 1 (1.56%) case. Changes in LM (99%) of 2 (3.1%) cases were associated with RCA stenosis (90%), at 2 (3.12%) where stenting was performed, and LAD (100%) bitronculatory changes , RCA 90%, OM-80%, Cx-90% in 1 (1.56%) of cases where immediate intervention was made. The changes were predominant in LAD (60%) with coronarography by stenotic at 50-90% 100% occlusion where invasive intervention was recommended as well as other authors recommend. Normal coronarography had 2 (3.12%), and minimum lesion 2 (3.12%).

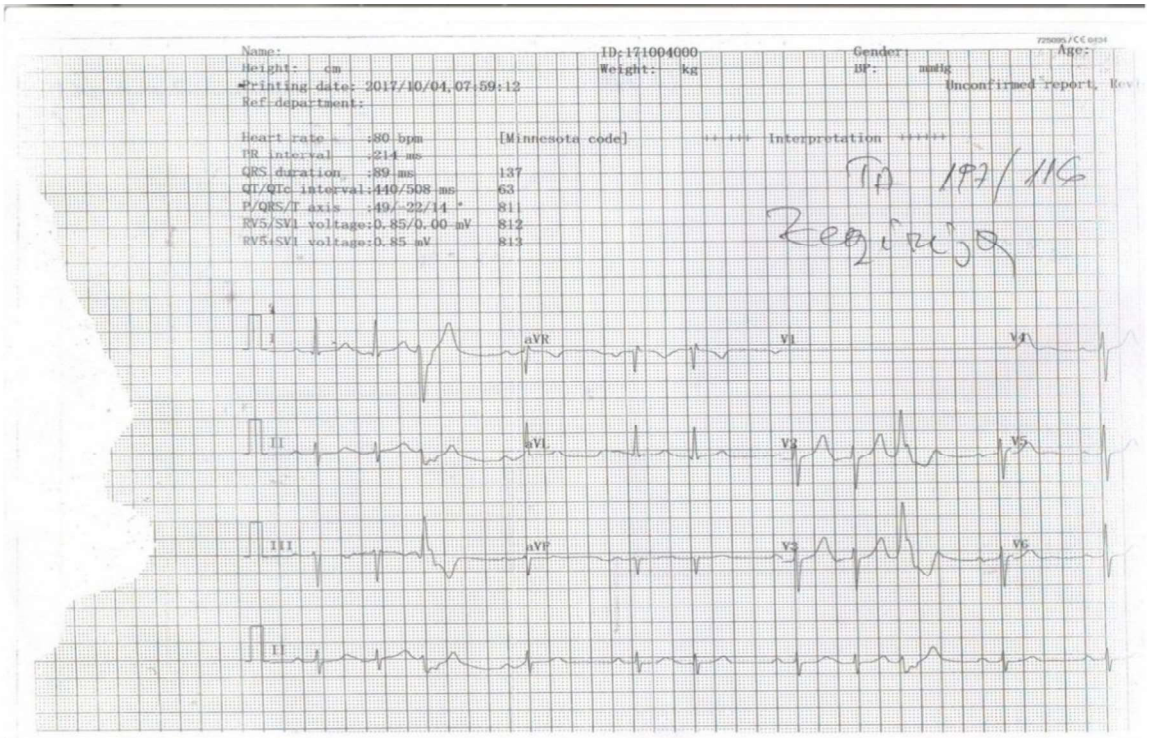
Myocardial perfusion scintigraphy was in a positive correlation with selective coronary artery detection in 9 (14%) cases of radiopreparate radiation to follow myocardial ischemia. Only in two cases (3.12%) female scintigraphy did not detect induced stress induced along the left ventricular myocardium. In invasive treatments such as PCI, PTCA, stenting had 8 (12.5%) of them and stent placement in 7 (10.9%), rupestylitis was performed in 5 (7.8%) cases followed by drug therapy. I must emphasize that according to the authors: C. Seiler and B. Meier from Bern University the cardiac diagnostic catheterization and coronary angiography as an invasive method has low risk of complications ≈1% or death ≈ 0.1% .PTCA is at risk of complications ≈ 4% and death approximately 1%. Intracoronary stenting complicates approximately 6-15%, mortality = 2%, which is devoted to their thrombogenicity and leads to acute or subcutaneous coronary artery closure.

In our cases, we had a complication in one female case 69 years of age during invasive treatment due to anorectal rupture of the thoracic aorta, respectively male patient following surgery on the left ventricle. The living was three months due to post-operative complications and pulmonary infection. Other cases of invasive treatment have good condition and survival over five years with regular cardiac follow-up that complies with the recommendations of world authors.

We judged that the number of patients with coronary artery disease was getting higher together with the age with male predominance, whereas in the more advanced ages over 70 years there was no significant difference between sexes due to hormonal changes in women, postmenopausal status, genetic factor, lifestyle, and others, judged by these statistics on ischemic diseases, the age most affected by males is age groups (50-60 years), and females 60-70 years.

Based on cardiac risk factors: Hypertension, diabetes, nicotine abuse, alcohol, overweight, hyperlipoproteinemia, physical inactivity, irregular eating and rest, psychiatric stress and accompanying comorbidities, potential candidates for ischemic heart disease are males over 45 years and women over 55 years and rarely young people. It was a global view of these statistics facts about chest pain syndrome - acute coronary syndrome.

By the term “acute coronary syndrome” we mean the combination of clinical syndromes caused by the rapid blood flow disorder in the coronary arteries which as consequence have the ischemia of the heart muscle. This syndrome includes these clinical entities: Angina pectoris nonstabilis, myocardial infarction with no "Q" at ECG, or without elevation of the ST segment, and myocardial infarction with "Q" and elevation of the segment ST to ECG.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RV5+SV1 voltage:0.85 mV
[Minnesota code]
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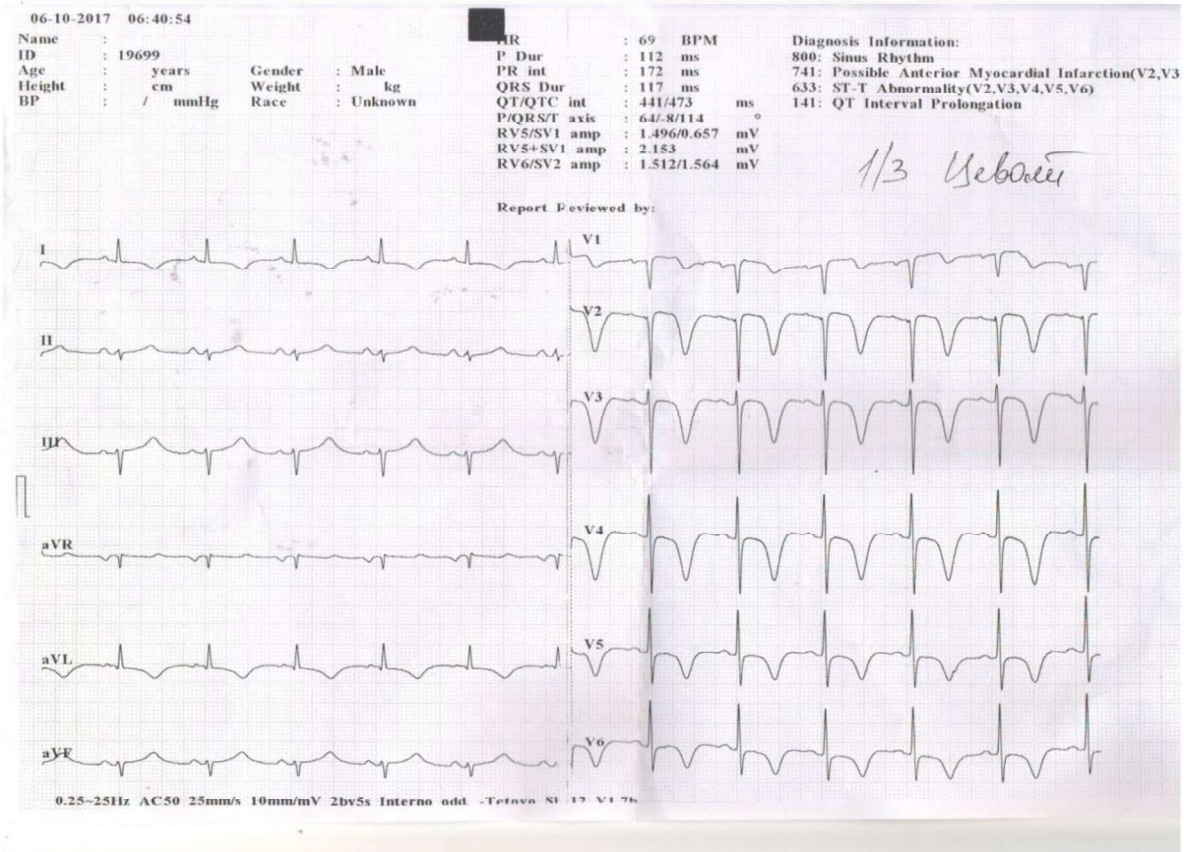
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Gender : Male
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Race : Unknown
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P Dur : 112 ms
PR int : 172 ms
QRS Dur : 117 ms
QT/QTC int : 441/473 ms
P/QRS/T axis : 64/-8/114 °
RV5/SV1 amp : 1.496/0.657 mV
RV5+SV1 amp : 2.153 mV
RV6/SV2 amp : 1.512/1.564 mV
Diagnosis Information:
800: Sinus Rhythm
741: Possible Anterior Myocardial Infarction(V2,V3
633: ST-T Abnormality(V2,V3,V4,V5,V6)
141: QT Interval Prolongation
1/3 Uebolen
Report Reviewed by:
I
II
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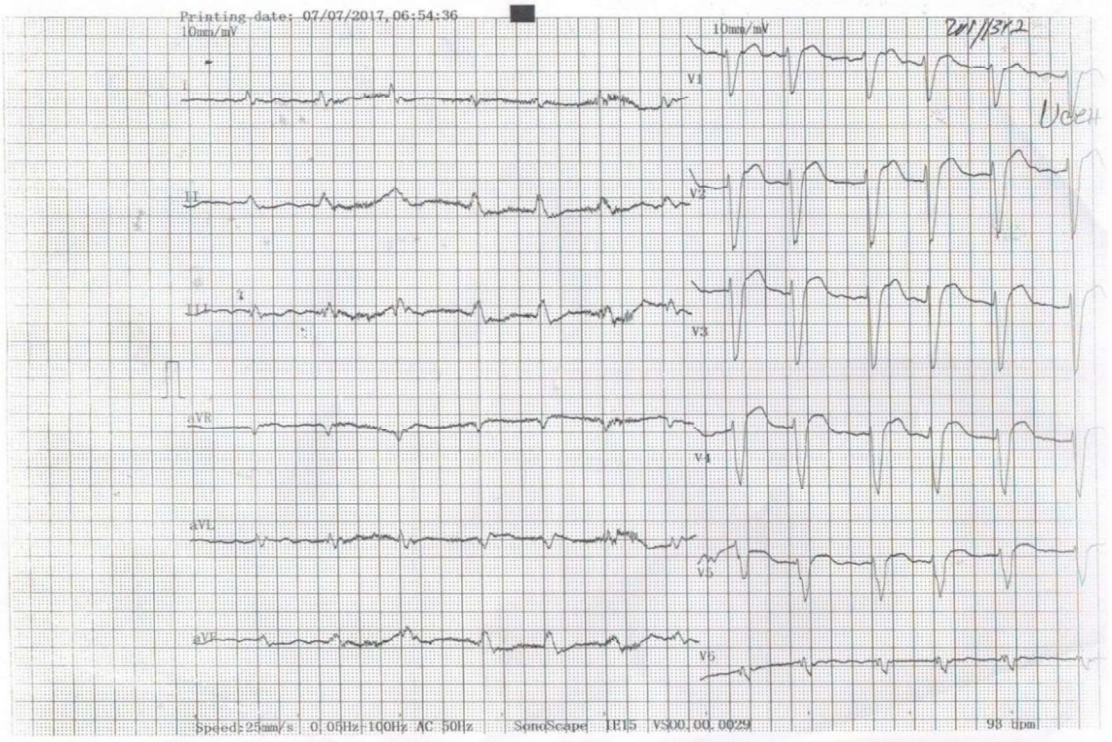
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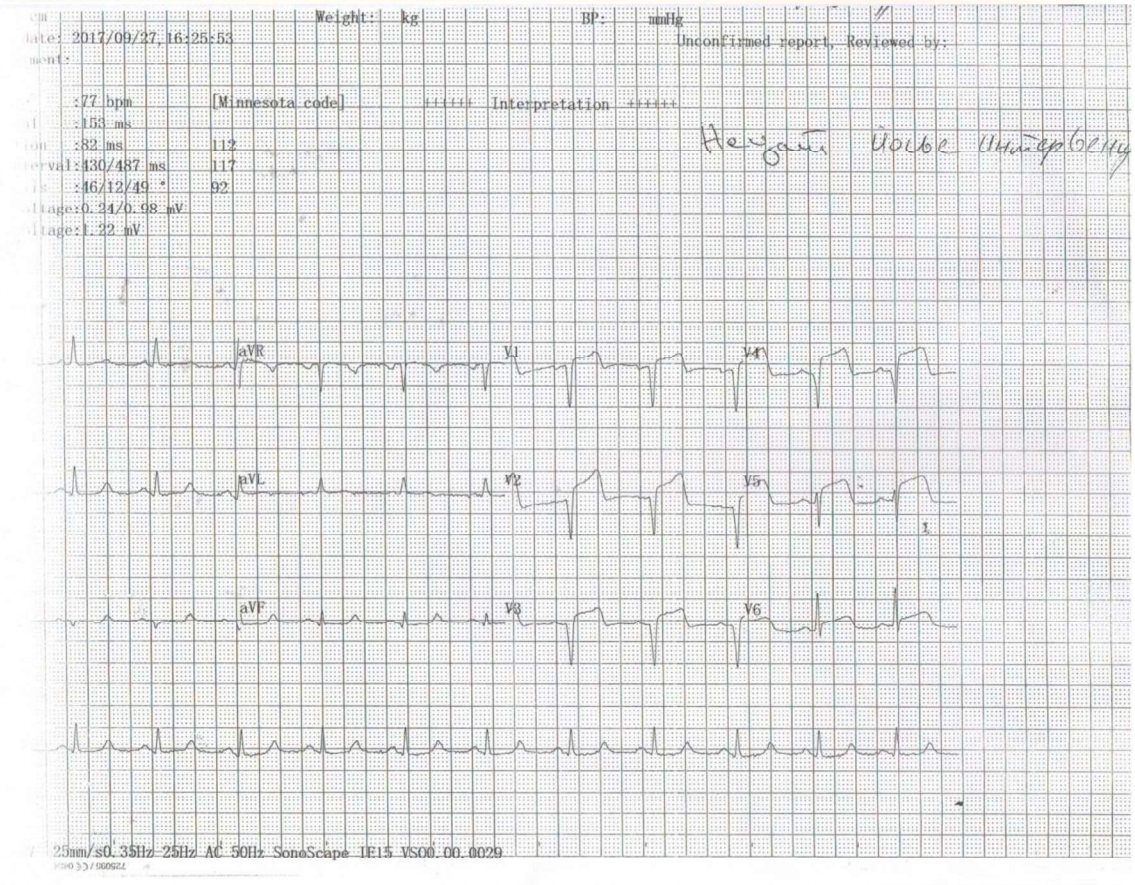
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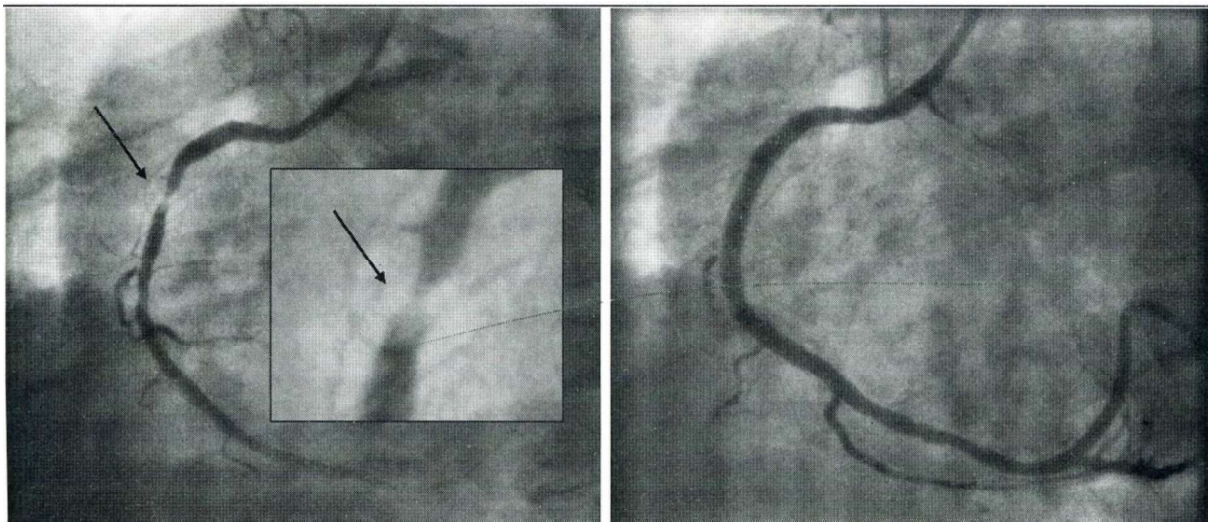
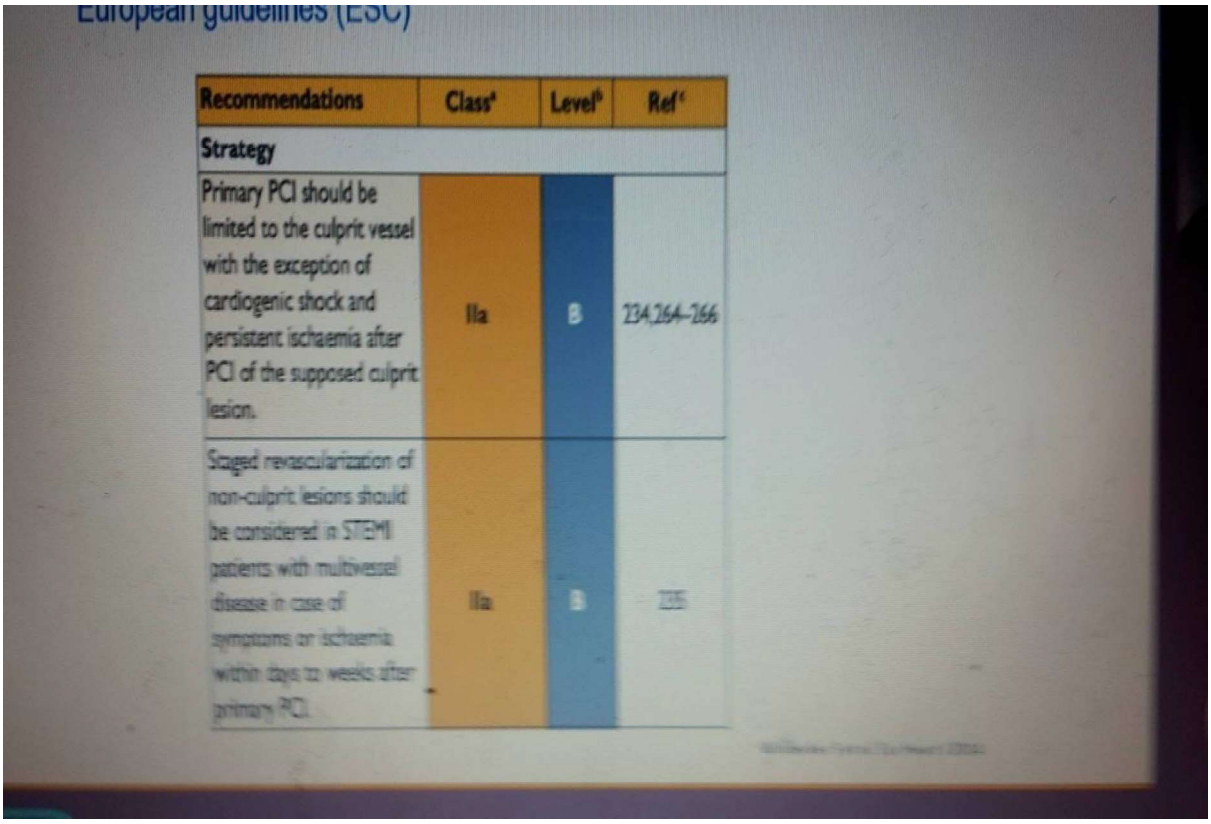


The treatment of coronary syndrome has its own strategy. It is important to stratify patients and predict the risk of myocardial infarction and sudden death as well as building a further strategy based on ESC, ACC / AHA principles. Apart from the removal of classical risk factors, the condition of the thyroid gland, anemia, aortic and other diseases should be looked at. Medication therapy was: nitrates, ASA, beta blockers, calcium antagonists, ACE inhibitors, diuretics, antithrombotic agents, statins and others. Invasive therapy such as PTCA and myocardial revascularization.



European guidelines (ESC)

Recommendations	Class[a]	Level[b]	Ref[c]
Strategy			
Primary PCI should be limited to the culprit vessel with the exception of cardiogenic shock and persistent ischaemia after PCI of the supposed culprit lesion.	IIa	B	234,264–266
Staged revascularization of non-culprit lesions should be considered in STEMI patients with multivessel disease in case of symptoms or ischaemia within days to weeks after primary PCI.	IIa	B	235

Treatment of angina pectoris and acute coronary syndrome, which means the forms of unstable angina pectoris to myocardial infarction forms without and with elevation of the ST segment have its own characteristics.

At angina Pectoris it is important to persuade the patient that the disease will not have rapid cure and fatal end. Risk factors such as diabetes, hypertension, obesity, nicotinism and others such as anemia, hyperthyroidism, and aortic stenosis should be cured or removed if they exist as comorbidities. During acute angina attacks, the patient should stop physical activity, direct the body or sit (angina decubitale), and take nitroglycerin (glyceryl trinitrate) in the sublingual route 0.3-0.6mg.

The healing effect occurs within two minutes, this dose of Nitroglycerin may be repeated in interval of several minutes. Nitroglycerin can be taken as a single prophylactic dose to prevent angina attacks in the next 30-45 minutes before the activity triggering angina attack begins. For prolonged action, oral nitroglycerin action or nitroglycerin is taken. The basic pharmaceutical action of nitrates is to reduce tonus of the lining, reducing venous blood flow to the heart, leading to lowering the tension in the left ventricle at the end of the diastole and reducing the volume of left ventricular cavity.

Nitrates dilate normal and stenosis epicardial arteries and collateral arteries. Nifedipine at a dose of 10 mg per in sublingual way can replace nitroglycerin. The isosorbide dinitrate at a dose of 10-15 mg in the sublingual way has the same action. Healing of chronic angina pectoris forms under load begins with beta blockers or calcium antagonists and, as needed, nitrates are given to cure angina pain. In the most severe cases it is necessary to give three medicines.

Prevention of further attacks: work with the patient in changing the lifestyle and teach him/her to coexist with the illness. Tell him maximally to leave the circumstances and activities that cause angina attack, while drugs for the prevention of successful anginal attacks are shown and isosorbide dinitrate or mononitrate at usual doses of 5-10 mg in the sublingual route 15 min prior to the activities that cause the risk as well as acetylsalicylic acid (ASA) in daily doses of 80-300 mg has been shown to be successful for the prevention of angina pectoris aids as well as the Molsidomine preparation at standard doses [1,18,22].

Blockers of beta-adrenergic receptors: constitute the basis of angina pectoris therapy caused by physical activity. Betablocator therapy begins with small doses, increasing the dose until the heart rate is reached 55-60 min. Heartbeat should not be more than 20 / min above the normal values during light physical activity, while during physical activity the heart rate should not be greater than 110-120 / minute. Cardioselective blockers such as Atenolol, Metoprolol according to some authors are given more often due to few side effects. [1,2,3,18].

Calcium antagonists (calcium channel blockers) are important drugs, as well as beta blockers of angina pectoris caused by physical activity. They are herbs that are used in peripheral vascular diseases as well as diabetics, pulmonary bronchospasm disease, but always taking care of blood pressure and heart rate. This includes: Verapamil: 40 to 120 mg every 8 hours, softens chest pain during physical activity, lowers blood pressure and heart rate, Nifedipine and Diltiazem are of the same effect, and younger generations such as Amlodipine, Felodipine, Lacidipine, Nikardipine.

Anti-Aging drugs inhibit the acute uptake of stable Angina and protect against myocardial infarction. Healing of chronic forms of angina pectoris occurs with sublingual nitrate and ASA or with inhibitors of ADP inhibitors that inhibit platelet aggregation (Clopidogrel) Nitroglycerin, if administered at weekly doses, include Betablocators, Ca antagonists, propranolol and others.

Myocardial Revascularization: There are two ways: Percutaneous transluminal angioplasty - PTCA and surgical revascularisation - Aortic coronary artery. The invasive treatment is based on anamnesis, selective coronarography and left ventriculography. All high risk patients such as "Ef <40%", multitronicular coronary artery disease and major aa column, left coronary artery, proximal prolapse stenosis of LAD, advanced patient age, hypertension, diabetes, angina severe pectorals, earlier infarction are candidates for surgical treatment.

According to German authors M. Weber and C Hamm, the clinical form of acute coronary syndrome is changeable. It has been achieved that on the basis of the ECG to distinguish groups with STEMI and without NSTEMI / unstable angina pectoris. ACS risk diagnosis and stratification without ST avoidance is important. We emphasized that pain as a management symptom, hidden as the "complaint of silence", in duration of 20 minutes, where angina is first presented as the acceleration of a stable symptomatic.

Data on risk factors, age, gender, kidney failure, earlier events such as myocardial infarction, surgical interventions at aka coronary increase the likelihood of acute coronary syndrome. [2] 12 channel ECG after each episode of pain, or after 6-12 hours, compared to the front EKG, ST segment monitoring [1,2], a ST root of 0.1mv in two neighboring derivations has diagnostic and prognostic value Inversion The T-wave> 0.1mV in R-tooth cases is far less specific.

Biochemical markers

At ACS without S-T elevation, biochemical markers are indispensable. CK and CK_MB, Troponin T and Troponin I, Myoglobin has low specificity [1,2,18]. Troponin has high values 3-4 hours after ischemia, secondary troponin measurement takes place after 6-12 hours. If the pain does not calm down even after 12 hours the clinical data refer to Coronary Acute Syndrome. The second measurement is excluded. After a stroke, the value of troponin remains increased for 3 weeks and the determination of Troponin is limited to post-infarct angina [1,2]. Troponin detection is the newest gold standard in determining myocardial necrosis.

Because the cardiovascular forms of isoform tropone T and I are found only in myocytes, the slower growth of troponin values with normal CK_MB values is the basis for the detection of smaller myocardial lesions [1,2,9,18]. The troponin value is> 0.1 microgram / ml. For early infarction it is better CK-MB and (myoglobin) "Wrongly" troponin increase are interpreted in kidney

insufficiency (Creatine> 2.5 mg / dl Troponin increase is also possible in myocarditis, lung embolism, hypertensive crisis , contusion cordis and so on.

Other markers such as CRP are found as increased in 40% of cases with acute coronary syndrome. Circulating ligands such as CD-40 (sCD-40) are novel markers at the cut-off point in the activation of platelets and inflammation, which in troponin possess power high prognostic value of expression [1,2] BNP (B-type natriuretic peptide) markers, namely Nt pro BNP, have prognostic significance [1,2, ..] ECG under load is contraindicated in diseased patients. In the period of observations ECG can be used with ergometer as evidence of ischemia, myocardium echocardiography and scintigraphy provides additional information, but best standard remains coronarography. Only 25% of patients with ACS have a normal coronarographic judgment or only gradual changes.

Treatment

The drugs are divided into: Antibodies: ASA, Ticlopidine, Clopidogrel, low molecular weight heparin, glycoprotein IIb / IIIa receptor inhibitors prevent further thrombosis and enable internal fibrinolysis, trumpet fusion and lower coronary artery stenosis. Long-term antithrombotic therapy aims at preventing the rupture of atherosclerotic plaque and occlusion of the coronary artery.

Anti-cancer drugs: Betablockers, Nitrates, Calcium Antagonists reduce myocardial oxygen demand. Coronary revascularization PCI, PTCA, and surgical revascularization is often necessary for the recovery of recurrent or rheumatic ischemic attacks. Patients with ACS are admitted to the coronary unit where ECG monitoring is performed to control arrhythmias and asymptomatic elevations of the ST segment, intravenous is provided, ASA is given at a dose of 75-325 mg because it inhibits cyclogenase-I, blocks thromboxane A2 and agglomeration platelets, giving oxigen in cases of hypoxia is to stop the pain is given nitroglycerin 3 lingvaleta from 0.4 mg at 5 minute intervals, if no nitroglycerin effect is given in infusion, 10 micrograms / min, and doses increase 10 micrograms every 5 min until achieved effect. Morphine is given 1-5 mg when there are no nitroglycerin effects.

Betablocators are fed at ACS in the i.v. way, continues to give for os until the heart rate reaches 50-60 rpm. The beta blocker decreases oxygen consumption and decreases the frequency by acting on the B-1 receptors to inhibit the catecholamines. They are given if there are no contraindications such as: bloc, bradycardia, hypotension, bronchospasm, nodules: They act on coronary and atherosclerotic arteries, increase collateral circulation, inhibit platelet aggregation, are given in i.v. way as the control of the symptoms continues to give nitrite providing 10-12 nitrates free of nitrate due to tolerance.

Long-acting calcium antagonists make vasodilatation by reducing the smooth muscle contractions of the blood vessel, Ca short acting antagonists such as Nifedipine and dihydroperidine are only administered by betablockers. Antithrombotics: Low molecular weight heparin or non-fractionated heparin reduces intracoronary thrombosis while thrombin inhibitors (Hirudin) are still discouraging. Antithrombotic agents include: thienopyridine, ticlopidine, and clopidogrel and glycoprotein IIb / IIIa receptor inhibitors.

Ticlopidine and Clopidrogel inhibit ADP and inhibit platelet aggregation. Clopidogrel is of primary importance in cases of acute NYC and ST elevation (COMMIT and Clarity studies), Acute Coronary Syndrome without ST segment elevation (CURE_study), and CREDO - study Clopidogrel affects on elective PTCA before and after surgery and stent implantation by recurrence of death, myocardial infarction by 26.9%. The advantage of clopidogrel lies in cases where acetyl salicylic acid is contraindicated due to gastrointestinal disorder.

Inhibitors of glycoprotein IIa / IIIb inhibitors inhibit these receptors on the platelet surface to bind fibrinogen to them, which at the other end binds to GP IIb / IIIA receptors of paired platelet formation by platelet aggregation. In treating acute coronary syndrome, three inhibitors of GP IIa / IIIb: Abciksimab, Tinofiban, Eptifibatide are given intravenously, outpaced by pharmacokinetics and their pharmacodynamics and are given alongside acetyl salicylic acid and low heparin doses. Many authors testify to their clinical behavior, especially in cases of high risk, permanent ischemia and raised values of Troponion T and I which are usually candidates for Percutaneous Interventions (PCIs), Thrombolytics on the basis of some studies (ISIS -] are not indicated in the recovery of acute coronary syndrome.

European recommendations for 2013 on coronary disease include additional therapies such as ivabradine, nicorandil, ranolazine, class Iia or trimetazidine (class IIb). In the United States for 2014 it is recommended ranolazine, other medicine of the second degree are not mentioned.

Discussion

In preventative cardiology the main purpose is to prevent these conditions as soon as possible, which means regulating blood pressure, lipid level LDL, HDL, triglycerides, blood sugar level, ie risk factors that determine the overall risk of cardiac identity such as people with coronary and high risk patients in the next ten years that exceeds> 20%, not forgetting to mention advanced biological markers that are not for diagnostic routines but that are made in case of signs of coronary disease such as: Dense LDLs,

Apolipoprotein B, HDL2b, Fibrinogen, Chlamydia Pneumonia, Insulin, Homocysteine, Lipoprotein A, Apolipoprotein E and C-Reactive Protein, which predicts not only the first cardiac attack but also the aches repeatedly.

Determining the risk is the first step in combating coronary heart disease. Risk factors are divided into fixed factors, medical factors, genetic factors, physical and life-style factors. Fixed factors like gender age and ethnicity do not change. Does the genetic factor change?. Genes are invariable and situations that have a degenerative effect on the artery as raised triglycerides insulin resistance high arterial pressure are genetically defined but we have an impact on them: we reduce weight, physical exercise, we follow following our cardiac and metabolic profile and dangers decreses, we can change the mass of lipoproteins and here we go beyond the genes [21].

There are also additional factors such as inflammation, vascular activity, blood flow tendency - thrombogenic factor, and so on. Experimental and clinical data on "classical" risk factors are conditioned by disproportion and disordered interaction of sympathetic and nutritional growth where the increase in sympathy due to psycho-social stress with hormone activation - renin - angiotensis - aldosterone and insulin, and regulation of endothelial balance leads to hypertension, heart vessel restenction and atherosclerosis, discrete insulin induces increased LDL / HDL cholesterol levels.

Genetic predisposition which may be a consequence of these conditions, smoking as exogenous factors of sympathy. The interaction of all these processes take place in the cellular, but partly in the molecular field. In this spectrum of arterial hypertension, fat metabolism disorders, melitus diabetes, and inhalative smoking are highly active factors of coronary heart disease, myocardial infarction, apoplexy [1, 4, 5, 14, 21].

Conclusion

- Judged according to data at European Centers and the way we treat acute coronary syndrome there are two main strategies known. Early conservative strategy and early invasive strategy. The first foresees the making of coronarography in all cases with ischemia or angina pectoris with S-T segm changes in re or during the smallest physical activity but with highly coronary stress test versus drug therapy.
- The invasive strategy foresees the making of coronarography and revascularization in all patients with recurrent angina pectoris in calm and minimal activity when coronarography is not contraindicated and revascularization if indicated. Candidates for invasive treatment are patients with decreased left ventricular function, heart failure symptoms, galloping swelling, pulmonary edema, hemodynamic instability, ventricular tachycardia and PCI-treated patients, or myocardium revascularization during the first six months.
- In patients with elevation of the segment S-T the invasive therapy is the first. High risk patients, elevated enzymes, rhythm cremations, S-T hemodynamic instability dispersion, should be treated with invasive methods 48-72 hours before. Despite the persistent scenes of these diseases and the risk factors, these diseases are on the rise, requiring a serious involvement of the medical factor such as cardiologists, internists, and that of other profiles in general.

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