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PATHOPHYSIOLOGY, INVESTIGATIONS, AND MANAGEMENT OF UNSTABLE ANGINA: A REVIEW

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Review Article

ABSTRACT

Unstable angina pectoris is a phenomenon that disrupts the early recovery phase of acute myocardial infection, the emergence of new ischemic symptoms, the development of more severe pain, usually at rest, or the formation of intermittent ischemic events. - Acute myocardial infarction is the most serious complication of unstable angina pectoris, which can affect up to 25% of patients within three months of the onset of symptoms. Physically, changes in ECG and hemodynamics usually occur before the onset of pain. The following hemodynamic changes and an increase in oxygen demand in the already ischemic heart respond positively, leading to further instability in unstable angina. Hemodynamic changes may be the result of abnormal stress response or excessive chemotherapeutic discharge. While coronary spasm may play a role in the pathogenesis of unstable angina, other factors such as bleeding in the wall of the atherosclerotic plaque, peripheral embolization, alteration, or platelet aggregation in the coronary artery from nearby soft cholesterol "abscess". Feedback should also be considered. Along with medications such as nitrates, calcium antagonists, and warnings, beta-adrenergic blockers are used to stimulate coronary and peripheral vasodilation and reduce cardiac effort. Treatment with aortic counter-pulsating balloons is recommended for those who have failed to respond to pharmacological treatment. Emergency surgery should be performed voluntarily. The full clinical and pathological compatibility of unstable angina pectoris is still unknown. Future issues will revolve around further investigations into entity procedures, treatment and prevention. The purpose of this review is to explain the pathophysiology of unstable angina, highlight changes in ECG in the assessment of unstable angina, and improve the outcome of patients with unstable angina. Consider the need to strengthen the coordination of care within the team.

Keywords: Acute coronary syndrome; mortality; myocardial infarction; troponin; unstable angina pectoris.

1. INTRODUCTION

Acute coronary syndrome covers a wide range of symptoms, including unstable angina. This public health problem, which affects a significant portion of the population every day, continues to be a major cause of death worldwide. When it comes to patient management and outcomes, it is important to distinguish this from other types of chest pain, such as stable angina. Because patients rely on their health professionals to distinguish between different causes of chest discomfort, they need to be aware of the signs

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and symptoms of acute coronary syndrome. Patients often go to the emergency room. On the other hand, acute coronary syndrome can be observed outpatiently. Over the years, numerous studies have been conducted to determine the most appropriate and effective treatments and available diagnostic tools for assessing unstable angina and various forms of acute coronary syndrome [1].

2. ETIOLOGY

Coronary atherosclerosis is the underlying cause of unstable angina in almost all individuals with acute myocardial ischemia. The most common cause of unstable angina is coronary artery narrowing caused by a non-occlusive thrombus forming over a disrupted atherosclerotic plaque. A rarer cause is vasospasm of a coronary artery (Prinzmetal variant angina). This vasospasm is caused by dysfunction of the endothelial or vascular smooth muscle [2].

3. PATHOPHYSIOLOGY

The following are some of the factors involved in the pathophysiology of unstable angina: thrombosis, vasoconstriction and cyclic flow are all symptoms of a supply-demand mismatch. Mismatch between supply and demand: Like all tissue ischemia, myocardial ischemia of unstable angina pectoris is caused by excessive demand or insufficient supply of oxygen, glucose, and free fatty acids. Fever, tachycardia (eg, atrial fibrillation or flutter), malignant hypertension, thyrotoxicosis, pheochromocytoma, cocaine use, amphetamine use, aortic stenosis, supravalvular aortic stenosis, obstructive cardiomyopathy, aortic shunt, high-output heart failure can all be caused CHF. The following factors may contribute to decreased oxygen supply: Anemia, hypoxia, polycythemia, and hypotension are all symptoms of anemia [3].

Many of the above causes should be investigated as they are reversible. For example, anemia caused by chronic gastrointestinal bleeding is not uncommon in the elderly. This can occur in the presence of coronary heart disease (CHD). Patients, on the other hand, may not benefit or be harmed by anticoagulants and antiplatelet drugs. The importance of preventing or treating the underlying disease cannot be overstated. Almost all cases of stable angina and about a third of all attacks of unstable angina are caused by increased myocardial stress (the product of heart rate and systolic blood pressure) or wall tension [4].

4. PLAQUE DISRUPTION

Atherosclerotic plaques accumulate lipid-containing macrophages called foam cells and smooth muscle cells. Foam cells contain cytotoxic, chemotactic, and oxidized chemotactic low-density lipoprotein cholesterol (LDL-C). Production of macrophage proteases and neutrophil elastase in atherosclerotic plaques may promote weakening of the fibrous muscle caps that line the lipid core as the plaque expands. Cracking or rupture of plaque, especially at the junction of the hood and vessel wall, is caused by increased plaque instability combined with blood flow shear and circumferential wall stress (Fig. 1) [5].

Plaque disruption has a wide range of severity and effects. Small cracks are often non-invasive and are therefore medically quiet, and histological evidence of multiple occult episodes of plaque ulceration and repair with progressive increase in plaque volume has been found. Unstable angina or acute infarction are common findings of moderate to severe plaque disorders. Up to 50% of MIs are caused by angiographically small but functionally significant lesions. Because they have unstable thin-cap fibrotheroma, angiographically even mild lesions can be benign (TCFA). This suggests that targeted treatments such as percutaneous coronary intervention (PCI) are inadequate and that drug therapy is needed to protect the entire vascular system, especially in patients with ACS [7].

5. VASOCONSTRICTION AND THROMBOSIS

Due to the contraction of the blood vessels and the development of thrombus at the site of atherosclerotic plaque rupture, most patients with ACS experience a temporary reduction in coronary blood flow. These episodes result in atherosclerosis due to episodic platelet aggregation and complex interactions between the arterial wall, leukocytes, platelets and lipoproteins. Platelet adhesion and activation occur when sub-endothelial components are opened. Platelets clump together in response to open collagen in the vessel wall or local aggregates (eg, thromboxane and adenosine diphosphate). Platelets also secrete chemicals that cause blood vessels to contract and thrombin production. Thrombin is a powerful agonist for additional platelet activation and stabilizes the thrombin by converting fibrinogen to fibrin in a mutual pattern [8].

A clot in motion may be involved in ACS (ie, forming and enlarging, chipping off, and embolizing).This dynamic clot formation or lysis, together with coronary vasoreactivity and resistance in the microvascular bed, results in intermittent and alternating (or cyclical) occlusion and flow throughout time. The nonocclusive thrombus that causes unstable angina might become occlusive either temporarily or permanently. Recurrent unstable

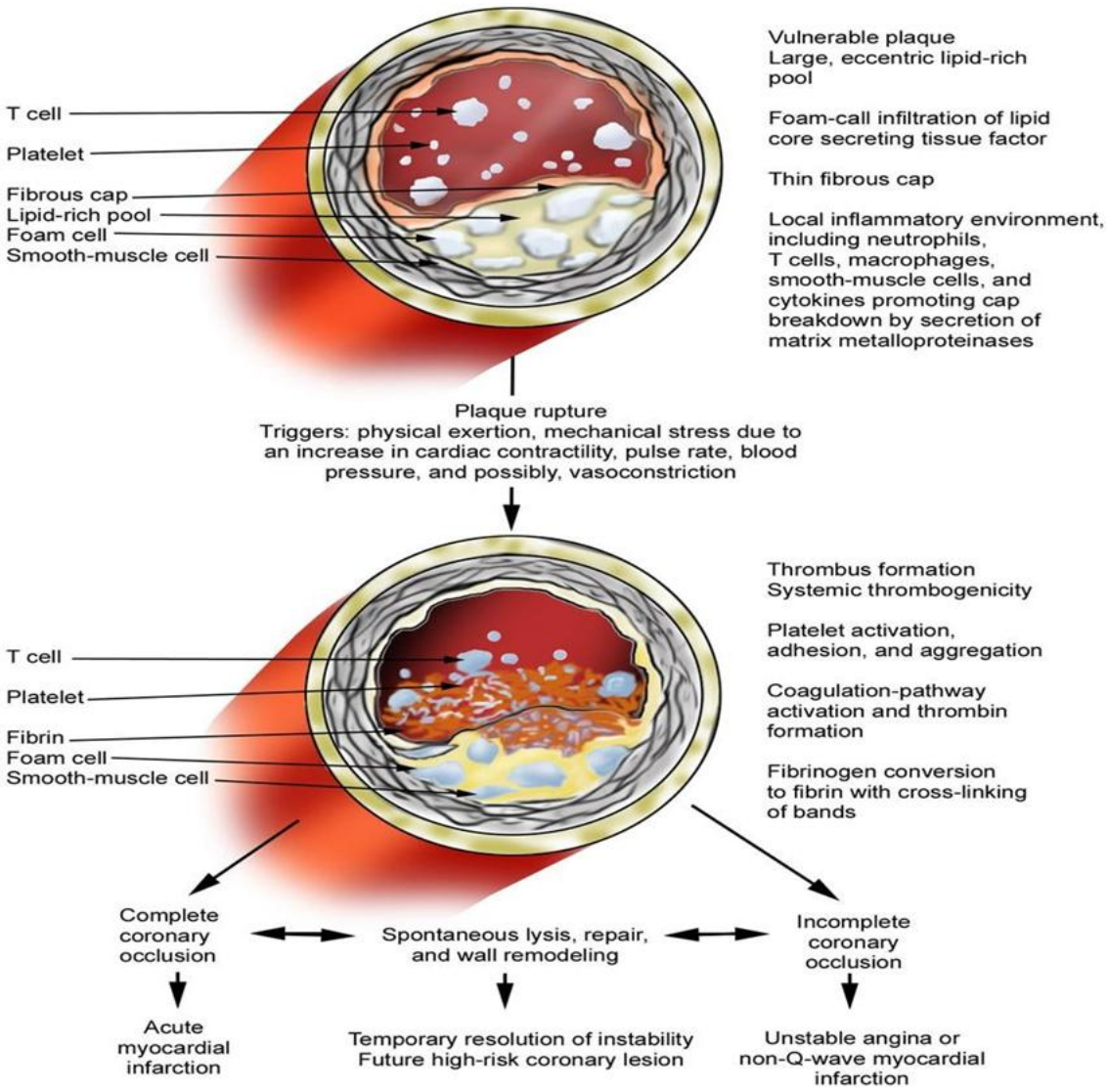

Fig. 1. Pathogenesis of acute coronary syndromes [6]

angina, non-Q-wave MI (NQMI), or Q-wave MI might occur depending on the length of the blockage, the existence of collateral arteries, and the region of myocardial perfused [9].

6. GENETICS

Although the etiology of heart disease is primarily related to variable environmental factors, genetics is known to play a role in the development of CHD and unstable angina. Much research on the genetics of cardiovascular disease has focused on the development of MI and CAD. However, there is a growing body of research on unstable angina. A variety of hereditary factors are known to play a role in unstable angina. Chromosome 2q36-q37.3, chromosome 3q26-q27, and chromosome 20q11-13 are all associated with unstable angina in Genome Wide Association Studies (GWAS). In heterozygotes for polymorphism in the Chinese population, a polymorphism in glycoprotein Ia was associated with a long time before platelet aggregation. It has been speculated that changes in platelet aggregation alter the pathophysiology of unstable angina [10].

Polymorphisms have also been found in numerous matrix metalloproteinase (MMP) genes. MMP1 with inserted guanine is associated with smaller, more stable plaques, whereas MMP9 with more than 22 "CA" microsatellite repeats is associated with a poor prognosis for unstable angina. .. Polymorphisms in interleukin (IL)-1 receptor antagonists (IL-1Ra) are thought to be involved in the development of unstable angina. Previous studies have shown that C-reactive protein (CRP) levels are higher in people who carry allele 2 of IL-2Ra. One study increased the incidence

of symptoms in young people, but there was no clear association between this polymorphism and increased risk of unstable angina [11].

Polymorphisms in the apolipoprotein E (ApoE) gene may also play a role in pathogenesis. In a study examining the association between ApoE4 and serum IL-10 levels, IL-10 levels were shown to be lower in patients with at least one copy of ApoE4. High levels of IL-10 are thought to be cardioprotective, suggesting that ApoE4 is associated with an increased risk of transient angina. Finally, the genetics of transient angina seem to be more strongly associated with inflammatory markers, and their effects on the risk of plaque rupture appear to be the most important mediator [12].

7. HISTORY AND PHYSICAL

Patients often complain of chest pain and shortness of breath. Chest pain is often described as pressure, although this is not always the case. The pain can be described as tightness, tingling and severe. Patients often report discomfort rather than pain. The pain spreads to the mouth or limbs, and can affect both the left and the right. Nausea, vomiting, dysphoria, dizziness and palpitations are all common constitutional complaints. Exercising can increase the pain, while resting can get rid of it. The use of nitroglycerin and aspirin is also helpful in relieving the discomfort [13].

The fact that the pain may not go away with these palliative variables is one of the symptoms of unstable angina. In addition, many people will already be suffering from coronary artery disease. These could be signs of established coronary artery disease or symptoms you have had for a long time. These patients may be familiar with the symptoms and may report an increase in the number of attacks of chest pain and severity of symptoms over a prolonged period of time. These signs suggest unstable angina rather than stable angina or other causes of chest pain as the most likely diagnosis. This is important because these fluctuations may indicate an impending myocardial infarction (MI) or ST-elevation myocardial infarction (STEMI), both of which should be evaluated as soon as possible. After all, the risk of morbidity and death in this condition is higher than in stable angina pectoris [14].

Although the patient may hold his chest, sweat, have difficulty breathing, his heart sounds may be tachycardic, and hemorrhages may be heard due to pulmonary edema, but most tests will be normal. - The following results indicate a high risk situation: dyskinetic apex, high JVP, S3 or S4, new apical systolic murmur, rales and crackles, and hypotension are all symptoms to look for [15].

8. INVESTIGATIONS

Patients should be examined as soon as they arrive. The patient should have an ECG to see if there is a suspicion of ischemic symptoms or STEMI. In unstable angina, ECG may show hyperactive T-wave, T-wave flattening, reverse T-wave, and ST depression. STEMI is indicated by ST elevation, and these patients should be treated with percutaneous coronary intervention or thrombolytic agents while waiting for the catheterization laboratory to become available. Acute coronary syndrome may include joint rhythm, sinus tachycardia, ventricular tachycardia, ventricular fibrillation, left bundle branch block, and other arrhythmias. However, the patient is more likely to have osteoporosis, especially if the patient has unstable angina rather than infected tissue [16].

Whole blood counts should also be performed on the patient to check for basic metabolic profiles to check for anemia, platelet counts and electrolyte imbalance. Troponin testing should be performed to determine if any myocardial infarction is present. Elevated levels are associated with an increased risk of death, so a brain natriuretic peptide (pro-BNP) test can also be performed. A clot test may be required if the patient receives or expects anticoagulation. The chest x-ray usually reveals the size of the heart and mediastinum, allowing doctors to identify the incisions of the chest pain and other causes [17].

It should be noted that chest pain, shortness of breath, pulmonary embolism, aortic dissection, esophageal rupture, pneumonia and other urgent causes of pneumothorax should be ruled out. Patients should wear a heart monitor to check for changes in rhythm. Cardiovascular stress tests (walking treadmill stress test, stress echocardiography, myocardial perfusion imaging, cardiac CT / MRI, or gold standard cardiac catheterization) can be performed. Patients and attending physicians usually order and perform them, but with the rise of observational medicine, emergency care providers may also order them [18].

Acute Coronary Syndrome Risk Assessment: If you have had a heart attack or have a family history of CHD, you are at increased risk of acute coronary syndrome. Chest discomfort may cause transient ECG or hemodynamic abnormalities. If there is evidence of angina in the chest, neck, or left arm, ST depression or elevation greater than 1 mm, and marked symmetrical T-wave inversion. Your doctor may order several tests to determine if you have angina [19].

Simply put, there are two most important questions to ask a patient with suspected angina: Is this a case of coronary artery disease? (I.e. what is the patient's diagnosis or condition?) Is it so dangerous? (In other words, what is the prognosis or what are the possibilities for something terrible to happen next?) . During the first 24 hours after examining a patient with unstable angina, the following laboratory tests are recommended: hemoglobin levels, serum chemistry panel and lipid panel, all of which are serial cardiac biomarker tests [20].

Currently, myocardial cell necrosis can be diagnosed using a variety of cardiac biomarker aces. Some of them, especially troponin tests, are also effective predictive tools and serve as important indicators of treatment aggression. When examining the metabolic pattern of unstable angina, urine protons have the ability to detect metabolic profile diagnostic biomarkers based on nuclear magnetic resonance (1H NMR) spectroscopy. Toll-like receptors 2 and 4 (TLR-2 and TLR-4) have been shown to have an effect on platelets in individuals with acute coronary syndrome, with clinical implications for preventive and curative effects. Evaluate patients with chest X-rays for signs of congestive heart failure (CHF) as well as other causes of chest discomfort such as pneumothorax, pulmonary infections or tumors, pulmonary hypertension and mediastinal enlargement (Fig. 2) [21].

9. MISSED DIAGNOSIS

Patients who missed a diagnosis of myocardial infarction (MI) or transient angina and were sent home from an emergency room (ED) had a 2-fold and 1-fold 7-fold higher risk of death compared with those who were hospitalized. Or is it a public health problem. In fact, 20% of the millions of dollars won in malpractice lawsuits against emergency physicians (ACS) are missing coronary syndrome. When patients with ACS attend the emergency department with acute chest pain, approximately one-third (32%) of them have normal levels of high-sensitivity cardiac troponin (hs-cTnT) (14 ng / L). Most of these patients suffered from transient angina. If patients with normal hs-cTnT levels died at a lower rate 1 year later than patients with higher hs-cTnT levels, their acute MI rates were significantly higher [23].

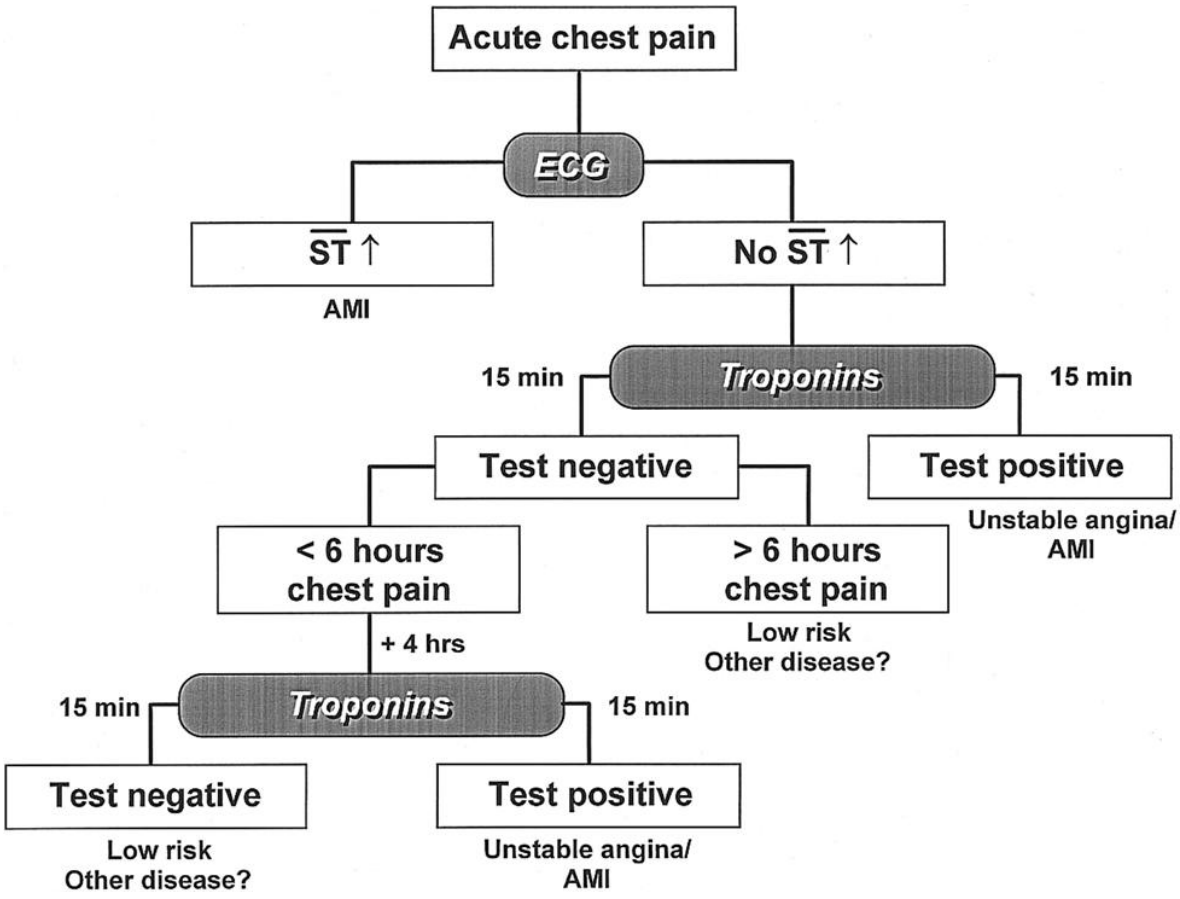

Fig. 2. Diagnostic approach for unstable angina [22]

Although it is impossible to eliminate the missed diagnosis of acute ischemia syndrome without increasing hospitalization rates and costs, the following actions can help reduce the problem: Identifying and eliminating bias or bias that affects women and non-diabetics. Leads to the correct diagnosis in white patients in ambiguous groups. The angina equivalents should be recognized, especially in elderly patients more likely to be misdiagnosed. People with unclear or normal ECG readings will have a confirmatory point-of-care cardiac enzyme test with a strong negative predictive value, with a more thorough history taking into account recent changes in the nature or course of angina symptoms. Pre-discharge exercise tests in stable, low-risk and intermediate-risk CHD patients, and since they are only snapshots taken at specific points during a dynamic procedure, the absence of ECG abnormalities or an initial increase in cardiac enzymes does not imply this. the ability to prevent an acute ischemia [24].

10. BASIC BLOOD STUDIES

Complete blood count (CBC) is used to rule out anemia as a secondary cause of acute coronary syndrome (ACS). In acute myocardial infarction, leukocytosis has prognostic value (MI). Platelet size and counting are initially variable in patients with ACS. In a prospective analysis of 134 patients with ACS (transient angina, non-ST elevation myocardial infarction [NSTEMI], STEMI), platelets and mean platelet count dropped after 3 hours of admission, although these values occurred only in acute MI patients. At 72 hours and 72 hours increased 7 days. In patients with ACS, potassium and magnesium levels should be closely monitored because low levels can cause ventricular arrhythmia. Serum potassium levels should be measured regularly and corrected as soon as possible. If cardiac catheterization is considered, the creatinine level is also required. N-acetylcysteine use and adequate fluid intake can help prevent contrast-induced nephropathy [25].

Myeloperoxidase levels can help distinguish people with ACS from those who have chest symptoms for other reasons. A previous study in the emergency department of all patients over the age of 18 who suffered from non-traumatic chest pain reviewed serial measurements of troponin and myeloperoxidase at admission and at 6 hours and found statistically significant differences not only in myeloperoxidase concentrations at admission and age 6. Hours in patients diagnosed between ACS- and non-ACS patients, but between patients with ACS and those with heart disease other than ACS [26].

11. CARDIAC BIOMARKERS

11.1 Creatine Kinase, CK-MB, and Troponin

Absolute increases in creatine kinase and its MB isoenzyme (CK-MB) or troponin levels distinguish non-ST-elevation MI (NSTEMI) from unstable angina (Fig. 3) [27].

Furthermore, biomarkers, alone or in combination with accelerated diagnostic procedures (ADPs), can reduce the number of patients who have missed an NSTEMI diagnosis who are at risk for significant adverse cardiovascular events. According to scientists at ASPECT (Asia-Pacific Evaluation of Chest Pain Trial), such techniques could promote early evacuation from the emergency department in patients who have a low short-term risk of a catastrophic cardiac event. However, the experiment did not

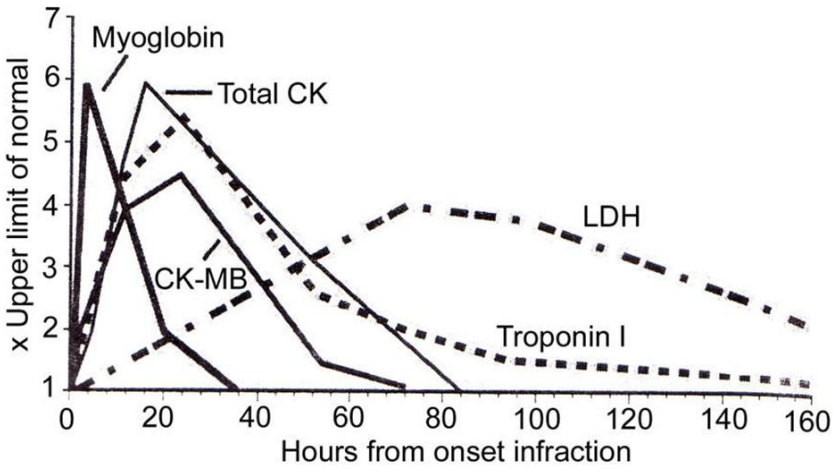

Fig. 3. Time course of elevations of serum markers after acute myocardial infarction, CK = creatine kinase; CK-MB = creatine kinase MB fraction; LDH = lactate dehydrogenase [28]

adequately assess the potential effect of cultural differences on chest pain perception and timing of presentation. Taking blood for total CK-MB levels every 6-8 hours for the first 24 hours is the current standard of care. In addition, cardiac troponin levels (T or I) should be determined at least twice, 6–8 hours apart, as these markers may be negative initially, especially after 2–4 hours of chest discomfort. within. Additional CK-MB or troponin measurements should be considered if initially negative, if patients have chronic or recurrent symptoms, or if the index of suspicion is high [29].

Troponin I levels above 0.4 ng / mL and troponin T levels above 0.1 ng / mL are considered positive and are associated with an increased risk of short-term and medium-term mortality. Aggressive treatment such as early cardiac catheterization improved the outcome of troponin-positive individuals. A slight increase in CK-MB or troponin from a low baseline level followed by a decrease in levels significantly indicates the presence of myonecrosis, as well as the temporal trend of these assays in interpreting difficult cases. increase. Troponin's serum half-life is longer than that of CK-MB and may remain elevated 7-14 days after the event, thus providing evidence of cardiac events in patients who delay hospitalization. However, due to its dynamics, cardiac troponin is not very effective in assessing recurrent chest discomfort associated with myocardial damage, but CK-MB levels can detect reinfarction [30].

Because troponin measurement is a highly sensitive test for detecting non-CAD myocardial injury or necrosis (e.g. in critically ill or septic patients), one of the European Society of Cardiology (ESC) and the American College of Cardiology Foundation A joint working group was set up. The ACCF, the American Heart Association (AHA), and the World Heart Federation have proposed recent mechanical standards for the universal definition of MI (WHF). Basically, these universal standards aim to identify people who may have ACS and who may benefit from investigation or intervention (Type I or "sponaneous MI"). A patient in an intensive care unit (ICU) with type 2 myocardial infection or myocardial necrosis associated with trypone elevation due to inconsistency in supply and demand is distinguished from the individual (e.g. anemia, tachycardia, respiratory failure). , Or hypotension due to sepsis) [31].

Higher troponin predicts worse results in all these groups, but patients with the latter are less likely to benefit from ischemia testing and may even suffer. There is also evidence that troponin T may be falsely elevated in the presence of severe skeletal muscle damage. In addition, it can be difficult to interpret the results of a bedside qualitative troponin test in people with renal failure [32].

12. BRAIN NATRIURETIC PEPTIDE

The TACTICS / TIMI-18 substudy showed that brain natriuretic peptide (BNP) (B-type) is an independent predictor of short-term and long-term mortality and risk of CHF in patients with unstable angina. rice field. In patients with unstable angina, elevated BNP levels are also associated with more severe coronary artery disease, especially in patients with the more severe left anterior descending artery (LAD) artery disease. BNP levels can complement the assessment of patients with unstable angina, but should be used in combination with other cardiac markers to help clinicians make decisions. The cost-effectiveness of regular use of multiple cardiac biomarkers has not yet been determined [33].

13. COMBINATION WITH C-REACTIVE PROTEIN

In context, troponin (a biomarker for myocardial necrosis), a combination of N-terminal pro-B-type neutrophilic peptide (NT-proBNP) (indicator of elevated ventricular and diastolic pressure and wall tension) and C-reactive protein - (CRP) levels (an indication of systemic inflammation) can be used to predict outcomes in ACS patients [34].

14. NITRIC OXIDE

Endothelial nitric oxide levels are a promising new biomarker to predict coronary complexity in people with unstable angina. In a study comparing nitric oxide levels to the SYNTAX (Synergy Between Coronary Intervention percutan with taxus and cardiac surgery) score in terms of predicting coronary complexity and treatment, researchers found lower average nitric oxide levels in people with unstable angina than in control persons. Decision for unstable angina in the emergency room. Compared to those who underwent coronary artery bypass graft surgery, coronary angiography, and percutaneous coronary intervention, those who underwent coronary artery bypass graft surgery had lower levels of nitric oxide [35].

15. ELECTROCARDIOGRAPHY

The 12-lead ECG, which should be taken within 10 minutes of the patient's arrival at the ED, is the first line of investigation in any patient with suspected unstable angina. When a previous recording is available for comparison, the diagnostic accuracy of an ECG is improved. If the patient's chest pain

persists and no ECG changes are seen on the first or subsequent recordings, serial ECG recordings every 15-30 minutes are indicated. The most dangerous ECG findings (ST-segment elevation or new bundle branch block) require rapid therapeutic triage for revascularization. Early MI may also be indicated by spikes in T waves. Patients with an ECG ST depression of more than 1 mm are considered high-risk patients. Subendocardial myocardial necrosis affects about half of people with this diagnosis. Regardless of the level of cardiac biomarkers, the presence of ST-segment depression predicts fairly significant in-hospital, 30-day, and 1-year mortality [36].

In the ancillary ECG analysis of the TIMI-III registry, a new or reversible ST segment deviation of 0.5 mm or more from baseline was associated with an increased risk of death or myocardial infarction at 1 year (15.8% vs. 8.2%). Alterations in the primary T-wave are neither sensitive nor specific for ischemia, although they can be useful when the QRS T-wave angle is greater than 60 degrees and the patient is symptomatic. Isolated symmetric T-wave inversion does not appear to be associated with an increased risk of death (Fig. 4) [37].

16. WELLENS SYNDROME

The ECG anomalies in the precordial T-wave section that are linked with significant stenosis of the proximal LAD coronary artery are known as Wellens syndrome. In the early 1980s, De Zwaan, Wellens, and colleagues identified a class of unstable angina patients who exhibited particular precordial T-wave inversions and developed a substantial anterior wall mine. LAD coronary T-wave syndrome is another name for Wellens syndrome. The following are some of the criteria for a syndrome: distinctive T-wave alterations, Anginal chest discomfort in the past, Levels of cardiac enzymes that are normal or slightly increased, An ECG with typical precordial R-wave progression, no Q waves, and no substantial ST elevation. Because LAD coronary T-wave syndrome is a pre-infarction stage of CAD that commonly develops to a severe anterior wall infarction, it's critical to recognize this ECG anomaly (Fig. 5) [39].

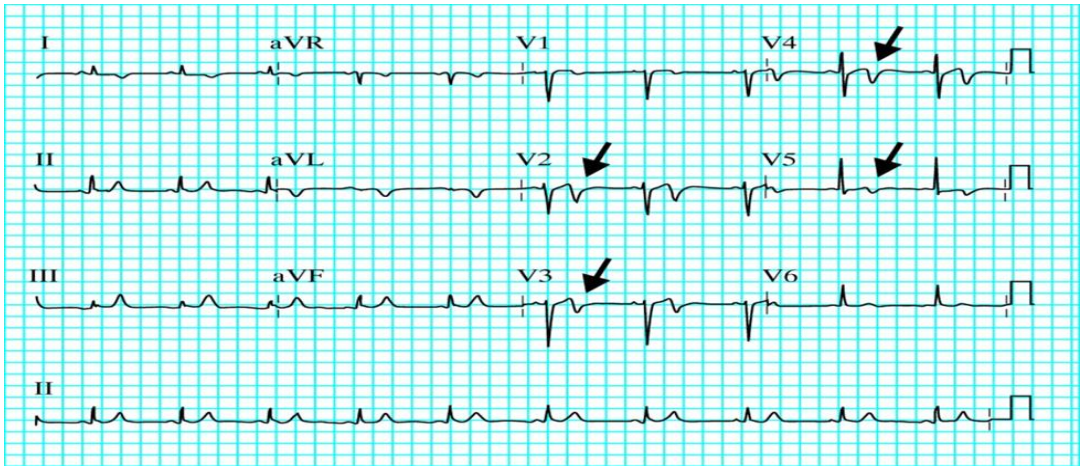

Fig. 4. Unstable Angina and the non-ST segment on ECG [38]

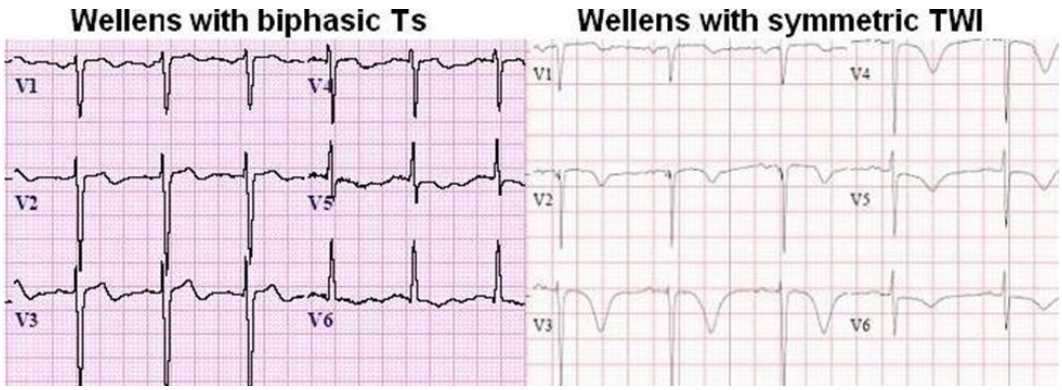

Fig. 5. Wellens syndrome on ECG [40]

17. ECHOCARDIOGRAPHY

Echocardiography, available in a timely manner, may provide a rapid assessment of left ventricular function, either for prognosis (worse when LV EF is less than 40%) or for diagnosis (e.g., if new segmental wall motion abnormalities are found). Then) (e.g., chest pain after a heart attack or revascularis where baseline left ventricular function is known). However, it should be noted that a small infarct does not appear on the echocardiography. Echocardiography can easily identify serious causes of chest pain, such as aortic stenosis and hypertrophic obstructive cardiomyopathy (HOCM). If the clinical presentation indicates a valvular or mechanical MI complication, or if the patient does not follow the hospital's proposed course, transesophageal echocardiography is strongly recommended. If aortic dissection is suspected, transesophageal echocardiography, computed tomographic angiography (CTA), or magnetic resonance angiography (MRA) may help [41].

18. SPECT AND MRI

Single-photon emission computed tomography (SPECT) has sufficient sensitivity to detect myocardial infarction of less than 10 g, although gadolinium-enhanced magnetic resonance imaging (MRI) can show infarcts as small as 1 to 5 g. In the future, MRI may be combined with MRA of the coronary artery to detect ischemia (space-time maps of reduced blood delivery), myocardial infarction (thinning of the wall, scarring, or growth retardation), and abnormalities of the heart. wall movement. The ability of MRI to detect myocardial scarring, which is a powerful prognostic indicator, of at least 1% is well documented. It has also proven to be effective in detecting and pinpointing IM problems. Due to the high resolution and complete coverage of MRI, echocardiography can miss wall motion abnormalities and myocardial infarction, either due to the high resolution and complete coverage of MRI, the omission of echocardiography of the lungs or ribs or dependence on the angle of echocardiography, which affects the affected area can be remembered as the actual top [42].

19. MYOCARDIAL PERFUSION IMAGING

In the emergency department, myocardial perfusion imaging is a useful tool for triaging patients with chest discomfort. After excluding infarcts, resting myocardial perfusion imaging is highly sensitive to the diagnosis of acute MI and can be supplemented with provocative tests. Clinical trial data, on the other hand, is only available in centers with a reputation for reliability and expertise (Fig. 6) [43].

20. EXERCISE TESTING

Exercise tests are rarely done in people who are in the acute phase of unstable angina or who have recently experienced resting angina. Subjects whose illness activity has stabilized after several days of treatment can safely undergo a stress test before discharge. Pre-discharge testing is preferable to testing weeks or months after discharge, as there is no loss of predictive value and the rate of adverse cardiovascular events occurs earlier. Pre-discharge exercise tests complement known key clinical features such as recurrent resting pain and evolutionary T-wave changes by providing independent predictive information. For example, patients with reversible defects on nuclear stress tests had a 25% chance of

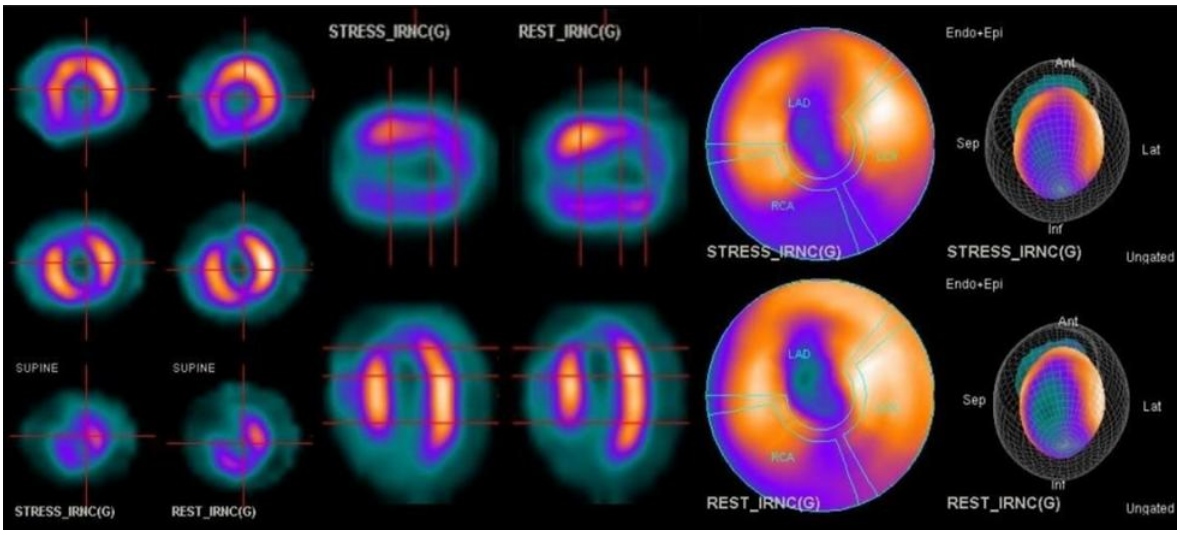

Fig. 6. Unstable angina during myocardial perfusion scintigraphy [44]

dying or having a heart attack one year later, compared to only 2% of patients with negative scans. Short exercise times, low maximum rate pressure products, and exercise-induced angina or ST depression are all associated with poor outcomes in men. Although the negative predictive value for all modalities of stress testing is on the order of 90%, the positive predictive value for exercise or adenosine stress tests is weak (16-19%) and only modestly better (31-48%) for imaging stress tests [45].

Initial stress testing is being considered by many chest pain centers as a way to speed up testing in low-risk patients. Randomized clinical trials compared patients with normal or undiagnosed ischemic ECG with those undergoing conventional care and resting perfusion scans. Non-ischemic patients who underwent initial nuclear perfusion screening had a 32% lower risk of unnecessary hospitalization without sacrificing safety. There are no extensive studies to evaluate the performance characteristics of various stress test methods in the context of unstable angina [46].

21. MANAGEMENT

The main goal of treatment is to improve coronary artery perfusion. This can be accomplished in various ways. Patients are often given aspirin (162 to 325 mg orally, or 300 mg rectal if the patient is unable to swallow) for antiplatelet therapy. Aspirin should be taken within 30 minutes. Nitroglycerin is available in various forms (intravenous, sublingual, transdermal and oral) and improves perfusion by dilating coronary arteries, leading to increased blood flow and lowering of blood pressure. As a result, the amount of work the heart has to do is reduced, which reduces the energy demand of the heart. For people who cannot take aspirin, clopidogrel is an alternative. Although Prasugrel is more effective than clopidogrel, it is associated with an increased risk of bleeding. In addition to aspirin, ticagrelor has recently been licensed to reduce the rate of thrombotic cardiac events [47].

To maintain proper oxygen saturation, you need to maintain excess oxygen from your nasal cannula. These three steps are quick and important to identify and treat the cause of transient angina. Patient response is at high risk for myocardial infarction and should be evaluated in individuals with persistent discomfort or long recovery time. Another possible treatment is anticoagulation with low or high molecular weight heparin. Beta blockers help save energy by lowering blood pressure and heart rate. The use of statins in patients with unstable angina has been proven in many studies. Cardiogenic shock, reduced ejection rate, unresponsive angina, new MR and unstable angina are all signs of cardiovascular angiography in patients with transient angina. NSTEMI early PCI (within 6 hours) has proven to have lower mortality than late PCI [48].

22. PATIENT EDUCATION

Precautionary goals include allowing the patient to resume all daily activities, maintaining myocardial function, and preventing future cardiac episodes. Most cardiac hospitals now have specialist teams, such as Cardiac Rehab, that offer more comprehensive and effective treatment. It is important to quit smoking to avoid recurrent heart attacks. This is true for everyone at home. LDL-C levels of 70 mg / dl or less, HDL levels of at least 35 mg / dl and triglyceride levels of less than 200 mg / dl should be tried to reduce lipids. The patient should follow exercise and a low fat diet. Hypertension control: The target blood pressure should be less than 140/90 mm Hg and the patient's sodium and alcohol intake should be reduced. Managing Diabetes Mellitus: Blood sugar levels can be lowered through diet, exercise or medication. Weight Management and Nutrition Counseling: Encourage the patient to lose weight and achieve a BMI of 25 kg / m3. Patients at risk of unstable angina should avoid strenuous physical exertion, especially in cold weather [49].

23. GUIDELINES

23.1 Acute Coronary Syndromes Clinical Practice Guidelines (ESC, 2020)

In late August 2020, the European Society of Cardiology (ESC) revised its guidelines for the diagnosis and treatment of Non-ST Elevation Acute Coronary Syndrome (NSTE) (ACS). The updates emphasize the need for personalized antiplatelet drugs, care systems and quality improvement, as well as a growing reliance on highly sensitive cardiac troponin (hs-cTn) tests for diagnosis. This includes coronary computed tomography (CT) to exclude low-risk patients. The main messages are listed below [50].

23.2 Diagnosis

The fundamental symptom starting the diagnostic and therapeutic series is non-persistent ST elevation chest pain (NSTE-ACS). Cardiomyocyte necrosis, determined by troponin release, or myocardial ischemia without cell damage are two types of myocardial disease (unstable angina pectoris). Patients with unstable angina had a lower risk of death and less benefit from aggressive pharmacological and aggressive treatment [51].

23.3 Troponin Assays and Other Biomarkers

hs-CTN tests are preferred by ESCs over less sensitive tests (higher diagnostic accuracy, similarly low cost). Several heart diseases, including myocardial infarction (MI), cause cardiomyocyte damage and can lead to increased CTN levels. When used with non-hs-CTN T or I (T/I), biomarkers such as creatine kinase myocardial band (CK-MB) and copeptin may be clinically meaningful in some cases. CK-MB declines rapidly after MI and may be useful in the diagnosis of early re-infarction. In rare cases where hs-cTn testing is unavailable, copeptin is recommended as an additional biomarker for early rule-out of MI [52].

23.4 Rapid "rule-in" and "rule-out" Algorithms

The use of hs-cTn tests (which have higher sensitivity and diagnostic accuracy) can reduce the time between the first and second cTn diagnoses to detect MI in the presentation. 0 h / 1 h method (best option, draw blood at 0 h and 1 h) or 0 h / 2 h algorithm is recommended (second best option, draw blood at 0 h and 2 h). 0 h / 1 h and 0 h / 2 h algorithms are used with clinical and electrocardiographic (ECG) data to help select candidates for initial discharge and outpatient treatment [53].

23.5 Hs-cTn Confounders

In addition to the presence or absence of MI, four clinical variables affect hs-CTN levels: Age: There are concentration differences of up to 300 percent between very young healthy people and very old "healthy" people. Renal dysfunction: A concentration disparity of up to 300% occurs between healthy patients with a high estimated glomerular filtration rate and healthy patients with a low estimated glomerular filtration rate (eGFR), with more than 300% experiencing chest pain at some time. moment of their lives. and more than 40 percent of people experience it because of their sexuality [54].

23.6 Ischemic and Bleeding Risk Assessments

If the clinical and ECG characteristics are too high, early cTn levels add information predicting short-term and long-term mortality (high levels of Hs-cTn increase the risk of death). Serum creatinine and EGFR should be measured in all patients with NSTE-ACS. These are prognostic indicators and an important component of the Global Registry (GRACE) risk score for severe coronary events (better than a physician's personal assessment of mortality or MI development). Nitrouretic peptides can help reduce the risk by providing additional prognostic information. The Academic Research Consortium for High Bleeding Risk (ARC-HBR) is a practical way of estimating the risk of bleeding (previously excluded from the clinical trials of duration or severity of dual antiplatelet therapy [DAPT]. HBR patients Contains the latest trials). If the prognosis of significant bleeding is low, use the PRECISE-DAPT (stent placement and subsequent bleeding complications prediction in patients with DAPT) scores to guide and teach DAPT length decisions. Can be done I can do it. Their value in enhancing patient outcomes is still in the air [55].

23.7 Noninvasive Imaging

Elective noninvasive/invasive imaging may still be necessary after a MI has been ruled out based on clinical assessment. Because a normal scan excludes coronary artery disease (CAD), coronary CT angiography (CCTA) may be an alternative for patients with a low-to-moderate clinical risk of unstable angina: It has a strong negative predictive value (NPV) for ruling out ACS (by excluding CAD) and a good outcome in patients with a low-to-intermediate pretest likelihood for ACS and a normal CCTA who present to the emergency room. In high-risk patients, CCTA imaging also minimizes the requirement for invasive coronary angiography (ICA).Stress imaging using cardiac magnetic resonance imaging (CMRI), stress echocardiography, or nuclear imaging are further imaging modalities based on risk assessment [56].

23.8 Risk Stratification for an Invasive Approach

The ESC recommends a routine intrusive strategy for NSTEMI within 24 hours of admission (based on hs-cTn levels, GRACE risk score >140, and dynamic new/presumably new ST-segment changes) to prevent severe adverse cardiac events and perhaps early survival. Highly unstable patients require urgent invasive angiography due to their hemodynamic status, arrhythmias, acute heart failure, or prolonged chest discomfort. For all other clinical presentations, a selective intrusive approach based on noninvasive testing or clinical risk assessment may be adopted [57].

23.9 Revascularization Strategies

The basic technical components of percutaneous coronary intervention (PCI) in NSTE-ACS patients are comparable to the invasive assessment and revascularization procedures for diverse CAD symptoms. Radial access is the approved and

preferable method in NSTE-ACS patients having invasive assessment with or without PCI. Consider the functional importance of all stenoses, the patient's age and comorbidities, overall clinical condition, and left ventricular function when choosing revascularization timing and completion because NSTE-ACS typically involves multivessel disease [58].

23.10 MI with Nonobstructive Coronary Arteries (MINOCA)

MINOCA refers to a group of underlying reasons that can include both coronary and noncoronary pathologic disorders, the latter of which can involve both cardiac and extra-cardiac issues. By consensus, myocarditis and Takotsubo syndrome are ruled out. CMRI, a critical diagnostic technique, identifies the underlying cause in about 85 percent of patients, allowing for correct therapy [59].

23.11 Spontaneous Coronary Artery Dissection

Spontaneous coronary artery dissection is a nonatherosclerotic, nontraumatic, or iatrogenic separation of the coronary arterial tunics caused by vasa vasorum bleeding or intimal rupture. It can account for up to 4% of all ACS cases, however it is more common in women under the age of 60. (22-35 percent of all ACS). When it comes to determining the diagnosis and treatment strategy, intracoronary MRI is extremely useful. The medical treatment will be decided later [60].

23.12 Pretreatment with P2Y12 Receptor Inhibitors

Due to a lack of evidence of benefit, the ESC does not recommend routine pretreatment with a P2Y12 receptor inhibitor in NSTE-ACS patients with unclear coronary anatomy and planned early invasive therapy. However, depending on the patient's bleeding risk, it may be considered in some cases [61].

23.13 Posttreatment APT

Unless contraindications exist, DAPT, which consists of a 12-month regimen of a strong P2Y12 receptor inhibitor combined with aspirin, is generally recommended, regardless of the stent type. DAPT length can be reduced (12 months), extended (>12 months), or modified by switching DAPT or de-escalation based on the patient's ischemia and bleeding risks, the occurrence of adverse events, comorbidities, co-medications, and the availability of the appropriate medicines [62].

23.14 Triple Antithrombotic Therapy (TAT)

Following PCI, long-term oral anticoagulation is recommended in at least 6-8 percent of patients and should be continued. For eligible patients, non-vitamin K antagonist oral anticoagulants (NOACs) are preferred over vitamin K antagonists (VKAs). The ESC recommends dual antithrombotic therapy (DAT) with a NOAC for stroke prevention for up to 12 months after a brief period of TAT (NOAC + DAPT) and single antiplatelet therapy (SAPT) (clopidogrel is preferred) as the default strategy for up to 12 months after a brief period of TAT (NOAC + DAPT). TAT can be extended up to one month when the risk of ischemia outweighs the risk of bleeding [63].

24. EPIDEMIOLOGY

Coronary artery disease affects a large percentage of the population. More over a third of deaths in those over the age of 35 are estimated to be caused by coronary artery disease. It is the leading cause of death among people in this age group. In the United States alone, this illness is estimated to affect about 18 million people. Men have a higher prevalence, but by the age of 75, the male and female rates are nearly equal. Other risk factors include obesity, diabetes, hypertension, high cholesterol, smoking history, cocaine or amphetamine abuse, family history, chronic kidney disease, HIV, autoimmune disorders, and anaemia. The average age of the presenter is 62, with women being older than men. African Americans are more likely to show up at a younger age [64].

Unstable angina is becoming more common in the United States, with an estimated 1 million hospitalised patients diagnosed each year. Unstable angina attacks are more likely to happen outside of the hospital, go unrecognised, or be treated in an outpatient setting. Despite improved public awareness, longer survival after MI, and an older population, the incidence of unstable angina should continue to rise despite main and secondary prevention initiatives. The American College of Cardiology (ACC) and the American Heart Association (AHA) have created two registries, the GUARANTEE (Global Unstable Angina Registry and Treatment Evaluation) registry and the CRUSADE (Can Rapid risk stratification of Unstable angina patients Suppress ADverse outcomes with Early implementation) registry, which provide statistical estimates for unstable angina patients (AHA) [65].

Patient demographics by age: Patients with unstable angina are on average 62 years old (range, 23-100 years). Patients in clinical studies for MI are on average 60 years old, 67 years old for carotid artery stenosis, and 63 years old for congestive heart failure,

to put this in context. When unstable angina strikes, women are on average 5 years older than men, with almost half of the women over 65, compared to just over a third of men. Persons of colour are more likely than people of other races to present at a younger age. Women experiencing unstable angina are older than men, and they are more likely to have hypertension, diabetes, CHF, and a family history of coronary artery disease. Men are more likely to have had a previous MI or revascularization, have a larger proportion of positive cardiac enzymes on admission, and have higher catheterization and revascularization rates. The severity of the sickness, rather than sex, determines the outcome [66].

Race-related demographics: There have been numerous reports of disparities in the outcome and risk factor occurrence among different ethnic groups. For example, black people have a higher prevalence of atherosclerotic risk factors (such as hypertension, diabetes, and smoking), a larger left ventricular mass, and a lower peripheral vasodilatory response than white people. MI causes more deaths in black people than it does in white people at a younger age. Fewer myocardial events but more cerebral complications have also been observed in black patients with unstable angina in randomized clinical trials of heparin versus hirudin (the Global Utilization of Streptokinase and TPA [tissue plasminogen activator] for Occluded coronary arteries II [GUSTO II] trial) or eptifibatide versus placebo (the Platelet glycoprotein IIb/IIIa in Unstable angina: Receptor Suppression Using Integrilin Therapy [PURSUIT] trial), possibly because of enhanced fibrinolytic activity and a higher prevalence of hypertension [67].

Also there are racial differences in how medical treatment is delivered and received. White people are more likely than other racial groups to undergo catheterization, angioplasty, and bypass surgery. According to studies, black persons have equal short-term (30-day) rates of death from unstable angina (including NQMI), but their long-term prognosis are consistently worse [68].

25. PROGNOSIS

The most common complications of unstable angina are myocardial infarction (MI), stroke, and death. Patients with new-onset ST-segment elevation (greater than 1 mm) have an 11 percent likelihood of having a MI or dying within a year, compared to only 7% for patients with established T wave inversion, according to studies. Low ejection fraction, persistent congestive heart failure (CHF), new or worsening MR, hemodynamic instability, protracted VT, and repeated attacks of angina despite maximal therapy are all poor prognostic indicators [69].

26. DISCUSSION

In terms of legal cases, unstable angina and other types of acute coronary syndrome account for a large portion of those brought against providers. As a result of aggressive chest pain examinations in general, over-testing, high admission rates, and many false positives have resulted in unneeded testing. Several criteria have been developed over time to prevent inappropriate admission and testing. Several of these have varying degrees of sensitivity and specificity. Physicians are often unduly active in the care and treatment of chest discomfort with the likelihood of an acute coronary syndrome due to the large number of legal claims filed [70].

Chest pain is a nebulous symptom that can result from cardiac or noncardiac causes. Acute coronary syndromes (ACSs) are a set of clinical symptoms that comprise everything from ST-segment elevation myocardial infarction (STEMI) to non-STEMI angina (NSTEMI). ACS with myocardial ischemia but no visible myocardial necrosis is described as unstable angina (i.e., cardiac biomarkers of myocardial necrosis such as creatine kinase MB isozyme, troponin, and myoglobin are not discharged into the circulation). Angina is a medical word for pain sensations induced by probable myocardial ischemia [71].

The term "unstable angina" was coined to characterise a situation that was halfway between a myocardial infarction (MI) and stable angina, a more chronic condition. The clinical goal of intervening to lessen the risk of a heart attack or mortality is referred to as "preinfarction angina." Patients with this syndrome are classified as having new-onset angina, accelerating angina, rest angina, early post-infarct angina, and early post-revascularization angina based on their symptoms, diagnostic test results, or progression over time [72].

Although the cause and definition of unstable angina are diverse, the interplay between damaged atherosclerotic plaque and overlying thrombi is often visible, resulting in hemodynamic deficit or microembolization in many cases of unstable angina. As a result, the scenario is distinct from stable angina, which is caused by a fixed coronary stenosis with restricted blood flow and slow, cumulative plaque buildup that allows collateral pathways to emerge. Other kinds of angina, such as hypertrophic obstructive cardiomyopathy (HOCM) or microvascular disease (syndrome X), cause ischemia through other mechanisms and must be treated as separate illnesses [73].

27. CONCLUSION

In emergency rooms, unstable angina is a relatively common consequence. There are a slew of treatment options for this deadly cardiac issue. According to current guidelines, this disease should be treated by a multidisciplinary team that includes primary care physicians, nurse practitioners, physician assistants, pharmacists, cardiologists, and emergency room physicians. A visit to a heart surgeon is also strongly recommended. Both the American College of Cardiology and the American Heart Foundation have developed treatment guidelines for unstable angina.

Once the situation has stabilised, prevention is critical. The nurse practitioner, pharmacist, and primary care physician should advise the patient to quit smoking, eat a good diet, exercise regularly, maintain a healthy weight, and follow medication regimens. Close monitoring is essential to verify that patients are meeting their cardiac rehab goals. In addition, lipid-lowering is required to reduce the risk of recurrent unstable angina; the pharmacist should provide guidance and monitor dose as well as any potential drug-drug interactions. Finally, the nurse and pharmacist should emphasise the need of controlling blood pressure and diabetes. Nursing will be in charge of much of the ongoing monitoring and evaluation, as well as notifying the doctor to any potential issues. This form of interdisciplinary collaboration produces the finest results.

Most institutions now have teams of doctors that specialise in treating unstable angina. Members of this group should be informed on current recommendations and educate the patient on how to avoid risk factors and gain the benefits of drug adherence.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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