Original Article: Myocardial infarction

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ABSTRACT

The EKG electrocardiogram should be taken within 10 minutes of the patient's pain reporting on arrival at the emergency department. With the help of this device, the location and causes of MI solutions can be identified and followed up. Early signs of EKG in acute MI are due to ischemia and myocardial injury. Myocardial revolarization is altered and causes a delay and reversal of the T wave, and the ischemic zone may remain repolarized until the myocardium returns to rest. Myocardial injury causes changes in the ST segment. Damaged cells naturally depolarize, but depolarize faster than normal cells. This causes the ST segment to rise at least 1 mm above the isoelectric line (the beginning of the P wave is used as a source for the isoelectric line). Myocardial injury causes an increase in the ST segment and long symmetrical T waves. The affected area will become a necrotic area if blood flow continues to decrease. ST segment changes return to baseline within a few days to 2 weeks. When ST elevation is greater than 1 mm (at least in 2 of 14 leads), MI Cute is considered. It causes the wave to be reversed to the region due to a change in repolarization, but in acute ischemia the T wave becomes long and sharp, and after this stage the flat is finally reversed. The big wave and symmetry are created within 24 hours. Reversed within 1 to 3 days with a final 1 to 2 weeks, T-wave reversal may persist for several months. Inverse T The deeper, more symmetrical, the more severe the ischemia.

Introduction

Areas that are necrotic in MI are called infarct areas, where the scar tissue eventually replaces the dead tissue, which is irreversible damage. The necrotic region leads to a permanent pathological Q wave due to lack of repolarization [1-3].

If MI does not cause a Q wave, it is called non-Q vawe MI. The wave appears a few hours or days after MI. When a wave appears, the term transmoral myocardial infarction is used to denote involvement of the entire myocardial wall [4-6]. If the patient has clinical manifestations of ischemia, but the ECG and vital signs of the heart do not show evidence of Acute MI, the person has unstable angina [7-9].The patient has symptoms of ischemia and unstable angina with enzymatic changes without pathological Q, subendecardi or non-Q ware [10-

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12]. Diagnosis of the location of MI is a vital factor in determining appropriate treatment and prevention of possible complications [13].

Anterior surface infarction

LAD artery provides blood supply to the anterior part of the left ventricle, blood supply to the anterior apex and part of the posterior ventricular apex and part of the left and right bundles. Occurs in case of anterior MI blockage [14-16]. An anterior stroke is often accompanied by the loss of a significant portion of the left ventricular muscle mass. Due to the fact that the left ventricle has a great role in pumping blood. As a result, it causes severe hemodynamic disturbances [17].

Symptoms on the EKG

Changes in leads V1 to V6 are evident. In cardiac leads, there is a slight increase in the R-wave, as the left ventricle is normally repolarized. The ST segment rises and the T wave reverses, and mirror changes are observed in the anterior MI and in the lower leads, namely leads D2, D3, and avf, which are observed as R as the ST segment rising and falling [18].

Complications of anterior MI

Severe hemodynamic abnormalities include different types of AV block, degree of bundle block, ventricular excitability, and left heart failure [19]. If all leads from AVL, V1, V6, and D1 change, it is called extensive Anterior MI.

Lower surface infarction

Occurs due to right coronary occlusion. EKG changes are seen in the D3 and AVF leads, also known as diaphragmatic infarction [20-23]. Because the lower surface of the heart is located above the diaphragm. Patients with inferior infarction are in the line of sinus bradycardia progression of myocardial infarction and PVC [24-26]. This type of MI can occur alone with lateral MI or with right ventricular infarction. The right coronary artery supplies blood to the SA node, the lower wall of the heart, the right heart wall, the septum between the atria, and the posterior part [27-29]. In 67% of people in the crux area where AV is located and is supplied by this artery [30].

Complications of diaphragmatic infarction

SA node disorders: One of the complications of right coronary artery (RCA) closure is that blood does not reach the SA and AV nodes suddenly, causing sudden cardiac arrest and sudden death. In this complication, SA node disorders are seen as SA Arrest [31].

AV node disorders: includes ventricular atrial block in the form of a degree block of mobbits [32].

Sinus bradycardia: One of the most dangerous arrhythmias caused by myocardial infarction is infarction, because bradycardia causes the onset of ventricular impulses such as ventricular tachycardia, ventricular fibrillation and death.

Pulmonary embolism: It is possible that thrombosis has formed at the site of infarction and separates from the wall of the right ventricle and goes to the lungs and causes emboli [33].

Atrial infarction

Right ventricular failure: which is characterized by hypotension, rapid heart rate due to decreased cardiac output, prominent jugular veins, pain and pressure in the right hypochondriac area due to hepatic congestion.

Rupture of the right ventricle: the symptoms of which are seen as bulging of jugular veins, drop in blood pressure, symptoms of cardiac tamponade and electromechanical separation rhythm (EMD) [34-36].

Pericarditis: This complication is caused by right ventricular infarction, because necrosis extends to the visceral layer of the pericardium, causing inflammation of the pericardium and accumulation of pericardial fluid, which can be detected by hearing the sound of friction [37].

Vaginal phrenic stimulation: Vaginal nerve stimulation during lower infarction activates severe vomiting, which increases the risk of cardiac arrest during vomiting (due to slow heart rate and arrhythmia) [38-40]. Prolonged contractions of the diaphragm can sometimes lead to prolonged hiccups. Atropine injections can prevent the stimulation of the pentogastric system [41].

Right ventricular aneurysm: is more common in pathological Q-wave stroke [42].

Lateral surface infarction: is caused by obstruction of the left rotational coronary artery. If a large mass of myocardium is involved, it causes hemodynamic changes [43].

Lateral infarction: There is involvement in the left side of the heart, near the base of the heart. ECG changes are seen in lead D1, AVL [44].

Posterior myocardial infarction: occurs as a result of obstruction of the right coronary artery or left CCA. This type of MI causes reciprocal mirror changes in leads V and V2 [45-47]. Classic MI-negative changes include: R wave, long ST segment drop, and wave flattening [48].

Right ventricular infarction: usually occurs following RCA obstruction. EKG changes as the ST segment rises in the right pericardial leads of the V 3R and V 4R leads on the right side of the heart [49-51]. If we cannot use the right leads, we can use leads II, III, avf, and leads V1, V2, and V5, and check for ST-segment elevation. II can be used to check for myocardial damage [52].

Note: The most common cause of death in patients with MI outside the hospital is arrhythmia (ventricular fibrillation). The most common cause of death in patients with MI in the hospital: Complications of MI such as shock cardiac failure [53-55].

The use of calcium blockers in Acute MI is of little value, it can even increase the risk of death. Catheter oxygen administration increases arterial oxygen pressure, thus preventing myocardial infarction [56-58]. Pulse oximetry to determine that his arterial blood pressure is normal and do not need to give oxygen. Therapeutic action is to limit the size of the infarct area [59-61]. By restoring blood flow, the myocardium needs reduced oxygen to disrupt the accumulation of harmful metabolism and mediators. The use of steroidal and non-steroidal anti-inflammatory drugs other than aspirin should be strictly avoided, as in addition to delaying the healing of the infarct area, they increase the risk of myocardial rupture. It also reduces ischemic myocardial blood flow by increasing vascular resistance [62-64]. Thrombolytics are drugs that are usually given intravenously. However, some may inject it directly into the coronary artery during cardiac catheterization [65].

The purpose of treatment with thrombolytics

Includes the elimination and destruction of thrombosis in the coronary artery, which restores blood flow in the coronary artery and minimizes the size of the infarction and maintains ventricular function. Thrombolytic therapy can reduce the relative risk of nosocomial mortality by up to 50%, provided it is given within the first few hours after a heart attack. Patients who are treated with thrombolytics within 1 to 3 hours after the onset of symptoms will benefit most. Of course, the effect of this treatment has also been observed in patients who have had MI for up to 3 to 6

hours. Its effects were observed even up to 12 hours after MI [66-68]. Especially when the patient complains of severe pain and the ST-segment is not high yet [69-71]. Thrombolytics dissolve not only the clots in the coronary artery but all clots. Therefore, these drugs should not be used in patients with protective clots such as after major surgery or hemorrhagic stroke. Thrombolytics reduce the patient's ability to form a fixed clot, so that the patient is exposed to bleeding [72-75].

Thrombolytic Indications

Chest pain for more than 20 minutes and not relieved by nitroglycerin, ST-segment elevation at least 2 leads indicating the area involved in the heart [76].

Prohibition of definitive use

active internal bleeding, recent history of use of these drugs 2 weeks ago, ocular bleeding disorders (such as retinopathy), active peptic ulcer and major surgery and recent injury systolic blood pressure above 180 mmHg and increased control blood pressure No pressure of 110 mmHg, pregnancy in patients treated with streptokinase in the last 5 days to 2 years, liver problems, age over 75 years [77-79]. The most commonly used thrombolytic agents are streptococci and tissue plasminogen activators.

Streitokinase

This drug, which is made from streptococcus, can systematically affect blood coagulation function. Its positive effect on the clot has been proven, but the risks of systemic bleeding are observed with its consumption [80-82]. The most serious complication is cerebrovascular hemorrhage, which increases with age. The rate of bleeding in people over 70 is twice as high as in people under 65. The next complication is an allergic reaction, which is seen in 2% of people, a slight decrease in blood pressure is observed in patients [83-85]. This drug is most effective when injected directly into the coronary artery, so it requires coronary catheterization facilities, a skilled physician, and a team ready for heart surgery. Dosastreptokinase is also given in 1.5 million units intravenously over one hour [86].

Nursing measures in the treatment of thrombolytics

In general, in patients treated with thrombolytic drugs, unnecessary intravenous and intramuscular injections should be avoided. Provide longer than usual pressure at the injection site [87-89]. Electrocardiogram monitoring and control of vital signs and coagulation tests (PT, CT and PTT) should be performed before if bleeding is observed during and after administration. Avoid continuous use of non-invasive blood pressure cuff. Control heart rate and blood pressure drop [90].

Note: If thrombolytics are used and coronary artery occlusion is removed due to coronary blood flow, the risk of arrhythmia is high, but the patient should be monitored.

Note: In case of large bleeding, discontinue treatment with thrombolytics and any kind of anticoagulant, apply direct pressure on the area and inform the doctor immediately. If there is no small bleeding, control the bleeding with direct pressure and monitor it continuously [91].

Therapeutic measures in the CCU ward

All treatments that are performed in emergencies are also performed in the CCU ward. In this ward, the patient is closely monitored and any abnormalities are identified and reported to the physician. Intravenous injection of morphine sulfate is used to relieve chest pain. The reason for intravenous injection is faster absorption and prevention of false increase in enzyme and reduction of anxiety in the patient as soon as possible. It is important to control the side effects of morphine, such as hypotension, impaired breathing, and decreased level of consciousness. In case of morphine

poisoning, naloxone or talorphine should be used.

Conclusion

fluid status check, electrolyte, hemodynamic status check, heart rhythm check, control and taking ECG12 Lady, control of cardiac enzymes, reduce patient and family anxiety, create a relaxing environment in CCU environment, control VS and hemodynamics and rest Absolute at least 12 hours, arterial oxygen pressure control by pulse oximetry or ABG.

Maintaining an open vessel prevents Valsalva maneuvering because of the risk of changes in heart rate and blood pressure and exacerbation of ischemia. Start of activity if there is no complication, 24 hours after the bed, which first hangs only the legs and walks beside the bed from the third day, on the 4th and 5th day, 180 meters, at least 3 times a day, can be walked if tolerated. The most important point is to start the appropriate gradual activity in the patient. Diet to prevent aspiration 4 to 6 hours after Acute MI is better for the NPO patient, as therapeutic interactions may be required. After 12 hours, the liquids should be followed by puree and 24 minutes of low-volume, low-salt, low-fat food, a total of 30% high in protein, rich in potassium, magnesium and fiber. It is best to give potassium by controlling serum potassium.

Note: In patients with MI, the use of angiotensin-converting inhibitors can reduce mortality and prevent congestive heart failure. When prescribing this drug, you should make sure that there is no drop in blood pressure, decrease in blood sodium, and decrease in blood volume or increase in potassium. If these drugs are prescribed, serum creatinine, blood pressure, urinary output, serum sodium and potassium levels should be checked. Increased blood supply to the heart in a patient with MI can prevent further damage to the heart. In addition to medication, the following methods can be used to correct blood circulation. Intra-aortic balloon pump, atheroma removal, laser treatment, coronary artery bypass grafting, stent placement, coronary artery bypass grafting.

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