Original Article: Applications of PTCA

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ABSTRACT

The purpose of coronary artery angioplasty is to revascularize the heart muscle, which is responsible for reducing angina pain and increasing life expectancy. PTCA is an invasive intervention method used in patients with angina pectoris as well as acute MI. Unstable angina that does not respond to drug treatment and lasts less than 1 year affects the patient's quality of life. Chronic and debilitating angina and new ECG changes include ST-segment elevation and pathological Q-wave. Acute MI that lasts less than 6 hours with or without concomitant use of thrombolytic agents and patients for whom CABG is dangerous. The patient should trust the treatment team, as fear can lead to vagal attacks of PTCA failure. Initially used as an alternative non-surgical invasive treatment for patients requiring coronary artery bypass grafting, today it is an adjunctive therapy for the treatment of Acute MI with fibrinolytic drugs. Acute pulmonary edema is a condition of abnormal accumulation of fluid in the lungs. Fluid may accumulate in the interstitial space or alveoli. Acute pulmonary edema is an acute event caused by heart failure, it can be acutely caused by factors such as myocardial infarction and chronic heart failure. Myocardial infarction as an ischemic effect can reduce ventricular dilatation and its function can significantly increase cardiac output. As resistance increases, left ventricular filling causes the blood to return to the pulmonary circulation.

Introduction

A variety Angioplasty is used with great caution. Complete artery occlusion, unstable angina, multiple plaques, left ventricular failure and extensive coronary stenosis with more than 40% reduction in cardiac blood flow [1-3]. Because in such cases, there is a possibility of MI, cardiac tamponade, coronary artery rupture and other complications [4-6].

Factors influencing the success of angioplasty

The experience of the physician performing the procedure, the lack of calcification in the coronary arteries, the presence of stenosis at the beginning of the artery and the disease of a single vessel involving arteries other than the garlic complex [7-9].

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Complications of PTCA: Complications may be due to the severity and characteristics of the disease process, the materials used, and the aggressiveness of the procedure [10-12].

Complications of angioplasty may occur later in the week or weeks after the procedure, including:

A) Restenosis: The main complication is PTCA and delayed complication [13-15].

Note: Aspirin and presantine, or both, should be given before angioplasty to reduce the risk of recurrence [16-18].

B) Arterial rupture, acute obstruction, coronary artery spasm: occurs in 5% of cases. Myocardial infarction, response and ozone with arrhythmia, contrast sensitivity, blood flow insufficiency in the limb used [19-21], neurological events due to embolus removal, arterial trauma or extensive bleeding, hematoma at pod entry site, rupture Coronary artery, which causes cardiac tamponade [22-24], prolonged angina during angioplasty, hypotension, decreased heart rate, and death are rare. In women, patients older than 60 years and patients who have had coronary artery bypass grafting have an increased risk of death [26-28].

Note: A successful angioplasty should reduce stenosis by 20% or more.

If the stenosis is not corrected, either the balloon dilation is repeated or a larger diameter balloon catheter is used. The risk of bleeding increases if the sheet is removed. Leaving the sheet allows the artery to be readmitted for re-dilation if necessary [29-31]. The pod is removed as soon as the anticoagulant effect wears off. To repair the area, the patient is placed in a supine position for 6 hours, checked for signs of bleeding [32-34].

Nursing care before PTCA

Psychological preparation to reduce the patient's anxiety and fear, physical fitness, consent form is morally and legally necessary. The consent form should include a description of the procedure and risks with possible side effects, review of history of drug sensitivity [35-37], especially to seafood iodide compounds, blood sampling for routine tests including blood type, cross-match, coagulation factors, electrolytes, complete blood cell count, for prevention Vomiting and aspiration of the patient should be 6-8 hours for solids and at least 4 hours for NPO fluids [38-40]. For the amount of Haji material to be administered, the patient's weight and height should be measured and recorded. Pre-procedure medications include mild sedatives and sometimes prophylactic antibiotics. For the procedure that uses the femoral artery, the pulse of the back of the foot, behind the ankle [41-43], and for the procedure that uses the brachial artery. The pulse of the radial and ulnar should be marked with a form pen [44-46]. In both limbs, this is to determine and compare the pulse before the procedure to determine the possible blockage of the cannulated artery. After the patient lies on the bed, the monitor electrocardiogram leads should be connected to the patient in the appropriate place [47-49].

Nursing care during PTCA

The first and greatest necessary responsibility for the people participating in the procedure is to be constantly aware of the patient's condition. The patient should be monitored for comfort, pain, etc [50-52]. Careful monitoring of the patient is essential to reduce possible complications. In case of ventricular fibrillation, the nurse charges the fibrillator according to the doctor's instructions, prepares the pedals and shocks the patient [53-55]. Another important task of the nurse is to accurately record the flow sheet of the procedure, including the time of incision, the type of catheter, the time of recording the pressure in specific areas, the time and place of sampling, the time of angiography, the time and amount of medication [56-58], the patient's responses. Controlling the patient for

signs of an allergic reaction to the contrast agent, such as burning, feeling warm. Educate the patient to report any chest pain during the procedure [59-61].

Important responsibility of the nurse after PTCA

A) Control vital signs every 15 minutes to 1 hour, then every 5 hours until balanced, and then every 4 hours for 24 hours [62-64].

B) ECG control, reporting any chest pain the patient should be monitored for cardiac arrhythmia by controlling the monitor screen or listening to the tip of the heart and touching the peripheral pulses [65-67]. In cases where the vasovagal reaction includes bradycardia, hypotension, nausea due to pain and dilatation of the bladder, especially when entering the femoral artery, in which case the patient's foot should be placed immediately above the head and fluids and sometimes intravenous atropine should be prescribed [68-70].

C) Educate the patient to inform in case of any chest pain.

D) Evaluate the area below the organ used to insert the catheter for signs of insufficient blood supply, such as arterial cyanosis, skin blemishes, paleness, coldness, a weak pulse, and changes in sensation and movement [71-73]. To diagnose arterial insufficiency, temperature, limb color, any patient complaints of pain, numbness or tingling in the limb involved in the assessment should be reported to the physician in case of any change. If the femoral artery is used for PTCA, the patient is kept in bed for 6-24 hours.

E) The organ used to insert the catheter should be kept upright for 4-6 hours after the procedure. If the femoral artery is used, to avoid bending the femur, the head of the bed should not be raised more than 20 degrees. During the patient's rest in the nurse's bed, he should try to be comfortable [74-76], which includes turning the patient from one pack to the other and, if necessary, prescribing analgesics according to the doctor's prescription [77-79].

F) Compression dressing should be done with a 2-5 kg sand bag on the arterial cannulation site according to the instructions. In case of bleeding and direct pressure on the site for at least 10 minutes to control bleeding and then a bag of 1-2 kg of sand can be used for continuous pressure.

G) To prevent bleeding at the catheter entrance, he should be trained to protect the area with his hand when coughing [80].

H) The site of catheter and dressing should be evaluated for signs of bleeding, inflammation, inflammation, and hemorrhage, as the patient becomes heparinized and should be monitored for other signs of bleeding [81].

I) Control of absorption and excretion, encouraging the client to receive fluids, because renal excretion of contrast material is in the form of urine [82].

J) Control the patient for allergy symptoms such as nausea, vomiting, rash [83].

K) Advise the client that after a long rest to get out of bed there is a possibility of hypotension, be sure to seek help [84].

L) If the pod is to remain in place, it should be held open by a solution infusion or an infusion pump [85].

M) When removing the pod to prepare for the vasovagal reaction, the patient should be monitored and have IV Line. Atropine and other devices should be available, heparin injection should be discontinued to bring PT closer to normal [86].

N) After removing the sheath, be prepared to apply direct pressure to the catheter entry site

until the bleeding stops. The patient should lie down for 4-6 hours after removing the pod [87]. **Note:** It should be noted that after angioplasty and before discharge, the patient should be able to perform and endure exercise testing [88].

Vasovagal response treatment protocol

If a vasovagal response occurs, if blood pressure is still present, the nurse should follow the steps below in order. If the patient is taking intravenous nitroglycerin, it should be stopped immediately unless instructed to do so [89]. As soon as the systolic blood pressure reaches more than 100 mm Hg, the nitroglycerin drops should be resumed to the required level. If blood pressure does not change, the patient should be in the Trendelenburg position. 0.5 to 1 mg of atropine sulfate is injected intravenously and 250 normal salines should be infused intravenously [90], 125 ml can be infused rapidly, the patient should be informed of the patient's condition. Atropine 0.5 mg may be repeated intravenously every 5 minutes and may be increased to 2 mg if the patient's symptoms persist [91].

Intra-aortic pump balloon (IABP)

The device has a polyethylene balloon that is inserted into the femur and placed in the descending thoracic aorta farther away from the left subclavian artery and connected to the external air pumping system. The balloon is automatically inflated early in diastole by helium gas and carbon dioxide, thereby increasing coronary blood flow and decreasing afterload in early emptied systole. The balloon filling and emptying period is adjusted based on the client's ECG, especially by the R-wave, which indicates the onset of systole. Due to the fact that the coronary arteries become liquefied during diastole, perfusion of the coronary arteries increases and the myocardial blood flow improves during balloon filling during the diastolic phase. The aorta gets better. Dilation of the balloon during diastole, when the aortic valve closes, causes blood to flow into the coronary artery.

Important points about IABP

The time of filling and emptying the balloon is a very important factor in maintaining the efficiency of the device. Properly timed, the IABP-induced aortic end-diastolic pressure should be less than the patient's own aortic end-diastolic pressure, which helps reduce afterload and improve cardiac output.

Common and dangerous complications

Including myocardial infarction, which is the most common complication of arrhythmic myocardial infarction. It has many complications of death risk, including cardiogenic shock, ruptured heart, and ruptured interventricular septum. The first goal of nursing is to prevent the complications of myocardial infarction.

Arrhythmia

It is the most common and early supply. The most common arrhythmias that occur in the first hours and can cause death in the same hour include premature ventricular fibrillation, ventricular tachycardia, and especially ventricular fibrillation.

Cardiogenic shock

It is a form of left ventricular failure and about 10% of all patients with myocardial infarction suffer from cardiogenic shock. The cause of damage is a large part of the myocardium. More than 40% of the left ventricle is damaged in cardiogenic shock, and systemic blood flow is reduced due to low cardiac output, and coronary artery blood flow is reduced as the shock continues. In areas of ischemia and necrosis that spread to each ventricle, ischemia and progressive infarction cause further loss of

function in the left ventricle and, if left unchecked, lead to death.

Clinical manifestations

Hypotension (systolic pressure less than 80 mm Hg) Tachycardia, decreased urine output to less than 30 cc per hour, decreased level of consciousness, drowsiness, cold and wet skin, metabolic acidosis (due to anaerobic metabolism and production of lactic acid by tissues Decreased blood supply), decreased cardiac output, increased left ventricular filling (PCWP becomes more than 18 mm Hg). Factors that predispose to cardiogenic shock include old age, decreased discharge fraction, severe left ventricular failure, history of myocardial infarction, and diabetes.

Treatment of cardiogenic shock

It has been a medical emergency and requires immediate intervention and treatment. The main treatment is to reduce any further demand of the heart, improve oxygen delivery and maintain blood supply. In these patients, a Swan Gonz catheter is usually placed and the pulmonary artery pressure and pulmonary edema of the pulmonary capillaries are monitored regularly to check left ventricular function. ABG ventricular control and metabolic acidosis correction are performed using artificial ventilation and buffering materials such as sodium bicarbonate. Soothing piping may be necessary to maintain oxygen balance. The setting of mechanical ventilation depends on the oxygen supply situation and the need to store the patient's energy. A catheter is inserted into a radial artery to accurately measure arterial blood pressure, a bladder catheterization to accurately control urinary output, including (normal saline solution, lactate Ringer's solution, or albumin). Medications such as vasopressin (a vasoconstrictor) including dopamine (chronotropic and positive inotropic) that increase both strength and heart rate. Isoproterenol is a drug that increases the strength of the myocardium, but due to the dilation of peripheral arteries, the effect on heart rate, increased myocardial oxygen demand and thus reduced coronary blood flow can reproduce the size of the ischemic area, so now less Is used. One of the positive inotropic factors is that its vasodilator effect is stronger. This drug increases renal blood flow and glomerular filtration rate. The initial dose of the drug is 7.5 mg / kg / min, after 2 to 3 minutes it is increased to 10 and then, if necessary, to 15 mg / kg / min, and vasodilators are used in these cases. Such as sodium nitroprusside, which can be given in small amounts to other therapies or alone to reduce afterload and reduce systemic vascular resistance. Every effort should be made to maintain systemic arterial pressure at least above 60 minHg and preferably 90 mmHg. Vasopressor drugs are used to raise blood pressure and cardiac output. They are used to improve blood flow to the heart and brain, but are not given in the early stages of the cardiogenic process due to increased cardiac output by increasing oxygen demand.

Note: Complete coronary artery collapse occurs at a pressure of 40 mmHg. Intravenous nitroglycerin, captopril, enalapril can be used to reduce afterload. If the shock does not improve with cardiac output and tissue oxygenation, the patient's condition worsens and an intra-aortic balloon pump may be used.

Note: Selection and continuation of drug treatment is based on cardiac output, cardiac criteria and moderate arterial pressure. Diuretics and vasodilators may be carefully prescribed to reduce heart rate, which exacerbates tissue loss. They dilate the arteries and veins, thereby reducing further return of intravascular volume to the environment and ultimately reducing postload and preload.

Ventricular aneurysm

It is mainly caused by Q-wave strokes. Complications of a left ventricular aneurysm usually do not occur for weeks or months. The most common complications are congestive heart failure, arterial embolism, and ventricular arrhythmias.

Note: The most common type of aneurysm is aneurysms of the tip of the heart. Post-myocardial infarction syndrome or Dresler syndrome in a small number of patients, 16 weeks after acute myocardial infarction, symptoms appear as pleural pain, joint pain, which is called Dresler syndrome. This syndrome occurs due to the autoimmune reaction of the necrotic area. Salicylates and steroid medications such as aspirin usually relieve the discomfort, and in more severe cases, a course of corticosteroids is given.

Pericarditis

Occurs in a patient with QWave. If the pericardial effusion is rapid and the pericardial sac is stretched, it can reduce cardiac output and venous blood flow, leading to cardiac tamponade.

Symptoms of tamponade

Feeling of pressure in front of the heart due to pericardial effusion, shortness of breath, abnormal falls and changes in blood pressure, paradoxical pulse, increased venous pressure, protrusion of cervical veins, decreased pulse pressure.

Pulse pressure

The difference between systolic and diastolic pressure is called pulse pressure. The normal value is 40 mmHg. If it is less than 30 mmHg, it indicates a decrease in cardiac output. In severe cases, the radial pulse disappears during inhalation. In cases of pericarditis, pain and fractionation are heard, which can be relieved by administering aspirin four times a day. If heart function is severely impaired, some of the fluid in the pericardial sac is drained by perforation and draining the pericardial synthesis. To do this, the patient is monitored by cardiogram monitoring, CVF is placed on the patient, and cardiopulmonary resuscitation devices are brought to the patient's bedside. It is raised 45 to 60 degrees below the patient's head so that the needle can be easily inserted into the pericardial sac. A venous route is taken and 5% glucose solution is injected as KV0 (for immediate injection, if necessary, an aspiration needle is attached to a CC 50 syringe). The person is monitored and enters the heart from the angle between the rib of the left rib and the sternum near the tip of the heart or from the right side of the fourth intercostal space. The needle is slowly pushed forward to reach the fluid (a rapid drop in central venous pressure, accompanied by an increase in blood pressure, and indicates an improvement). Pericardial fluid is usually sent to a laboratory for microbiological analysis of chemical analysis and tumor cells.

Early symptoms of cardiac tamponade

Increased CVP except in patients with hypovolemia, tachycardia, tachypnea, prominent jugular vein, anxiety, weakening of peripheral pulses, sudden cessation of discharge or bleeding from the chest tube, decreased urine volume, right ventricular collapse.

Late symptoms of cardiac tamponade

Decreased cardiac index, hypotension, blinding of the heart sounds (on listening by phone), equalization of CAP and PAD pressure, flattening of the mediastinum, bradycardia, cardiac arrest, sudden decrease in ECG voltage on the monitor.

Triple and classic symptoms of cardiac tamponade

A) Increased atrial pressure.

B) Lower blood pressure.

C) The hearing loss of the heart is called the classic symptoms of Beck's triple symptoms. It should not be overlooked that not all patients may have all three manifestations in those critical moments.

Cnclusion

It is a relatively common complication that if diagnosed in the early stages with proper and timely treatment, infarction and ischemia can be prevented. If left undiagnosed in the first few days, the patient develops pulmonary edema, which increases the potency of catecholamines from myocardial insufficiency, which, even with proper treatment of pulmonary edema, increases the size of the infarct and the large amount of tissue lost. The first sign of heart failure is an increase in heart rate compared to the previous day and the hearing of wet pulses in both lungs. If the patient is not treated, pulmonary edema is observed. Treatment is based on the degree of heart failure and is usually done with diuretics (in the early stages). Digoxin should be used with caution if heart failure is exacerbated due to increased oxygen consumption.

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