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# Prevention and Treatment of Acute Coronary Artery Syndrome

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**Abstract.** Acute coronary syndrome(ACS) refers to a group of clinical symptoms caused by acute myocardial ischemia, including unstable angina pectoris, acute myocardial infarction, and sudden ischemic death. In recent years, studies have shown that the main mechanism of its occurrence is the rupture of plaque and the consequent intravascular thrombosis. Multiple factors may lead to the occurrence of the disease. However, the mechanism of the histomorphological change from pre-fragmentation to post-fragmentation is still unclear. Vasospasm may also play an important role. Prevention of plaque formation and rupture or prevention of thrombosis by antithrombotic therapy is effective in reducing the incidence of acute coronary syndrome, and coronary intervention also reduces the mortality of acute coronary syndrome. Some new therapies, such as gene therapy, are still in the experimental stage. At the same time, people should improve the awareness of disease prevention, and the most active prevention is the best means of disease treatment.

**Keywords:** acute coronary syndrome, mechanism, prevention, treatment.

## 1. Introduction

Acute coronary syndromes (ACS) is the acute ischemic syndrome of the heart caused by the rupture or erosion of an unstable atherosclerotic plaque in the coronary artery. ACS includes ST elevation myocardial infarction (STMEI), non-ST elevation myocardial infarction (NSTEMI), and unstable angina pectoris (UA). Among them, ST elevation myocardial infarction is also known as ST elevation coronary artery syndrome. The combination of non-ST elevation myocardial infarction and unstable angina pectoris is known as non-ST elevation acute coronary syndrome (NSTEM-ACS). Approximately 70% of the ACS is caused by arterial part or intermittent occlusion, accompanied by ST segment depression (about 31%), T wave inversion (about 12%), ST segment depression combined with T wave inversion (16%), or both (about 41%). Typical manifestations are recurrent retrosternal pain, tight squeezing, compression or burning. It can radiate to the left upper arm, jaw, neck, back, shoulder, or the ulnar side of the left forearm, as intermittent or persistent, which has a particularly negative impact on people's lives.

It is estimated that over 7 million people are annually diagnosed with ACS worldwide [1]. Despite the rapid development of medical conditions and technology, but the incidence of acute coronary syndrome (ACS) remains high, and the case fatality rate is high, becoming one of the major diseases threatening human health and health care systems. In order to improve patients' symptoms and

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improve their quality of life, it is necessary to find effective treatment methods. This paper aims to discuss the incidence of ACS and to reduce the recurrence of patients with already ACS through the relevant defense and treatment of ACS.

## 2. The occurrence mechanism of acute coronary artery syndrome

The pathogenesis of ACS is complex and diverse, which has been found to include smooth muscle cell changes, vascular remodeling, plaque and inflammation.

### *2.1 Ons in smooth muscle cells*

Instability of the formation of atherosclerotic plaque and fissure atherosclerosis, the initial stage of the lesions mainly by smooth muscle cells to intima and proliferation began, early for very small yellow bulge fat stripes, then evolved into white bulge of hardening spots, fiber spots, part of the lesions evolved with thrombosis calcified ulcer complex lesions. Generally easy to cause rupture of the porridge plaque is called soft porridge plaque, its plaque is large and covered with its thin membrane, and not easy to cause rupture for the hard porridge plaque, which is often accompanied by calcification. Unstable plaques often contain a crescent lipid mass that is separated from the lumen by the fiber cap, and the fiber cap at the junction with the normal inner membrane may become thin before rupture, and the rupture mouth is often located here [2].

### *2.2 Vascular remodeling*

Atheromatous plaque does not initially bulge into the endovascular lumen,but develops to the outer vascular membrane. With the increase of the plaque, the inner diameter of the blood vessel also increases. By coronary angiography, no meaningful stenosis can be found. In the vascular section, when the plaque develops to about half of the vessel wall, it begins to bulge to the inner lumen, then coronary angiography will find meaningful stenosis, and such vascular structural changes during the development of atherosclerosis are called vascular remodeling. Due to the application of new techniques such as endovascular ultrasound and vascular endoscopy, it is easier to grasp the characteristics of atherosclerotic plaque carefully than in coronary angiography alone [3].

### *2.3 Plaque rupture*

The good hair site of the atherosclerotic plaque fissure is the fibrous capsule called / shoulder 0 and the normal artery junction. Pathology suggests that this site has more macrophages and lymphocytes aggregation, macrophages secretion of collagen tissue breakdown is mainly matrix metalloproteinase enzymes, T lymphocytes secretion to promote macrophages MMP secretion of C-interferon, due to the secretion of MMP, fibrous tissue is decomposed, atherosclerotic plaque fibrous membrane thinning, cause to become brittle atherosclerotic plaques. And plaque macrophages and foam cells contain a lot of tissue factors, atherosclerotic plaque rupture form fissure, because of the relationship of pressure, blood first into atherosclerotic plaque, atherosclerotic plaque contact with blood and white thrombosis, blood continue to fill to the plaque to form a red thrombus, atherosclerotic plaque pressure increase, thrombosis to the blood vessels, sometimes can block the whole blood vessels [4].

Most studies have shown that unstable plaques that can cause serious consequence plaque rupture are mostly severity stenosis lesions without collateral circulation, while severe stenosis plaques tend to be fibrosis and are not easy to rupture [5].

### *2.4 Foam formation of foam cells*

Koch examined the atherosclerotic plaques obtained in 1985 and confirmed the presence of inflammatory cells on the arterial wall, the invasion of monocytes, macrophages, and T lymphocytes. Anatomically, van der Wahr found that the most common plaque rupture in acute coronary syndrome was located in the shoulder region, which is also the most active area where inflammatory cells are active [6]. In 1988, Siku reported that the serum evidence of Chlamydia pneumonia Cpn was associated with an acute myocardial infarction of coronary heart disease. Subsequently, the

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relationship between CMV, Helicobacter pylori and coronary heart disease was also reported successively. Cpn was used as an example to illustrate the possible mechanism by which pathogens cause AS. Cpn is released by respiratory tract infection in the damaged vascular endothelium, which reinfects distant vascular endothelial cells, smooth muscle cells and mononuclear macrophages and proliferate inside the cells [7]. Cpn also stimulates smooth muscle cell hyperproliferation and decreased apoptosis; aggregation of inflammatory cells; enhanced endothelial cell coagulation and weakened fibrinolytic capacity [8]. When macrophages are activated, their cell-surface phagocytic receptors are expressed to engulf oxidized LDL like phagocytotic microorganisms, resulting in atherosclerotic plaque markers, foam cell formation [9].

### *2.5 Formation of thrombosis*

The above effect results in the proliferation of atherosclerotic plaques [10]. Libby believes that infection can also make AS progress through the action of procoagulants (fibrinogen, tissue prothrombin agonist, prothrombin agonist inhibitor PAI-I, platelets), causing thrombosis and coronary artery obstruction [11]. In the process of its disease, leukocyte-endothelial cell adhesion molecules and heat shock protein 60 expression, C reaction protein increase, cytokines (such as TNF-IL-2IL-6, etc.) secretion, more coagulation factors, oxidative modification of low-density lipoprotein and the formation of pathogenic microbial antibodies, complement system activation may play a key role, the cause of its disease still need to be further explored [12].

## 3. Predisposition

Triggers for atherosclerotic plaque rupture Regardless of whether the trigger is present or not, the vulnerable plaque will rupture sooner or later, but unusual activity may occasionally induce the rupture of the plaque [13], Myocardial infarction occurs in moderate to severe physical activity, emotional stimulation or excitement. There is still controversy about the trigger factor of myocardial infarction after activity, because many myocardial infarction occurs in daily life without significant increase in activity, and many patients with coronary heart disease do not have myocardial infarction in exercise trials [14].

### *3.1 Nerve and endocrine system*

In recent years, studies have shown that the occurrence of acute coronary syndrome with circadian changes, good at 7~12h in the morning, considering the nerve and endocrine activity circadian cycle changes, early morning sympathetic activity increases, catecholamine levels increase, and heart rate speed up myocardial contractility increases, and the vasoconstriction, aggravating the imbalance of myocardial supply and demand, morning plasmin activity decreases, platelet aggregation increases, antithrombin O activity decreases, the above factors promote the development of plaque rupture and the formation of local thrombosis[15]. In addition, the coronary artery curvature and torsion produced by each diastole may also trigger plaque rupture.

### *3.2 Catecholamines*

Thrombosis formation platelet activation is critical to the formation of acute coronary syndrome, and an increase in circulating catecholamines may activate platelet aggregation and stimulate thrombin formation, thereby prompting the formation of platelet thrombosis at vascular injury. Emotional excitement, circadian changes and smoking are related to the increase of catecholamines.

### *3.3 Fixed thrombus*

Shinal vascular intima damage only causes a variable and partially self-disintegrated unstable thrombus; and deep damage (exposure of collagen fibers) will cause insoluble compact platelet thrombus, in addition, deep damage due to the exposure of tissue factors, will activate the coagulation system, produce thrombin, further fixed the thrombus, namely fixed thrombus [16].

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*3.4 Dyslipidemia*

Abnormal blood lipids in the blood can also lead to the imbalance of the fibrinolytic system, t-PA activation has decisive significance for thrombus development or activation, t-PA acts on plasminogen and fibrinogen complex, prompting fibrinolysis, the continuous decline of t-PA and the rise of its inhibitor PAI lead to the formation of coronary artery thrombosis. Reports have shown that increased circulating fibrinogen concentration, increased activity of coagulation factors, and decreased antithrombin activity are closely related to the occurrence of acute coronary artery syndrome.

*3.5 Vascular constriction (spasm) coronary artery spasm is possible*

Vascular constriction (spasm) Coronary artery spasm may be associated with deeper vascular damage or abnormal endothelial function. Vascular endothelium is important in maintaining the relaxation and integrity of arteries. NO secreted by endothelial cells is a local hormone that can relax middle smooth muscle cells, and ET is a vasoconstrictor material secreted by endothelial cells. When the atherosclerotic lesion endothelium is damaged, NO / ET is dysregulated, leading to damaged vasoconstriction. Studies have pointed out that after vascular injury, the local release of serotonin and thromxin (TXA2) and local thrombin are mediated by vasospasm.

## 4. The prevention of acute coronary artery syndrome

*4.1 Regulation of blood lipids*

In recent years, a series of large-scale clinical trials fully proved that regulating lipid has very important clinical significance for the prevention of acute coronary syndrome famous Nordic simvastatin (Shu drop) survival study namely / 4S0 study, proved that long-term simvastatin, low total cholesterol, 25% and 35%, acute coronary syndrome events by 34%, by coronary angiography further confirmed that privastatin, simvastatin and other hydroxymeprotaryl coenzyme A (HMG-CoA) reductase inhibitors can make coronary atherosclerosis, reduce or even disappear [17].

*4.2 Reduce the High-HDL*

The HDL reduction is a risk factor for coronary heart disease, and the benforbet coronary angiography intervention trial is the first applied coronary angiography observational randomized trial to prove that benforbet reduces high TG lipoprotein, independent of LDL-C changes, and can slow down the development of coronary atherosclerosis. The gefbert CHD secondary prevention trial suggested that gefbate mainly treated CAD patients with HDL-C reduction, which reduced major coronary heart events by improving HDL-C and reducing TG, while LDL-C was unchanged. The above data show that if statins are mainly used for TG and LDL-C increase, fibrins are mainly used for TG increase, and betates should be used for HDL-C decrease in dyslipidemia [18].

*4.3 Anti-thrombotic*

Theoretically speaking, although antithrombotic treatment cannot prevent plaque rupture, but it prevents plaque progression and the occurrence of acute coronary artery syndrome.

Common anti-thrombotic drugs include aspirin, heparin (regular heparin and low molecular weight heparin) and so forth. In a primary prevention trial, the incidence of myocardial infarction was significantly reduced after a group of asymptomatic American health physicians took low-dose aspirin (325mg, once the other day) [19]. However, aspirin could not significantly reduce the overall mortality rate. It affected only one of the three pathways of platelet activation, and it had no effect on the coagulation system. Thiclopidogrel and clopidogrel are diphosphate gland acid receptor antagonists, and a recent large trial of CAPRIE data showed that clopidogrel (25 mg/d) is more effective than aspirin 325 mg/d. Due to its differences from aspirin antiplatelet mechanism, the combination can improve the efficacy.

Heparin shows its anticoagulant effect by accelerating the activation of antithrombin in the circulating blood, including common heparin and LMWH, which is less bound to plasma protein

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endothelial cells and has a long half-life, and, in addition, requires no laboratory monitoring of its activity. The comparison conclusions of the two large-scale clinical randomized trials are different and need to be considered. Direct thrombin inhibitors can highly specifically block thrombin, hirudin directly binds to the anion binding site and hydrolysis site of thrombin, has a strong and predictable anticoagulant effect, small-scale test, compared with ordinary heparin, the incidence of acute coronary syndrome and bleeding is reduced [20]. There are large amounts of the GPOb / Oa receptor on the platelet surface, and when the platelets are activated, the fibrinogen molecule binds to this receptor, causing platelet condensation. Aximab has a strong affinity for this receptor, returning platelet aggregation to normal. Data from more than 15,000 patients with acute coronary syndrome showed that the use of glycoprotein Ob / Oa inhibitors reduced the incidence of ischemic complications. Currently, it is most commonly used clinically with oxiximab. New antithrombotic pathways are being investigated with the aim of enhancing the antithrombotic effect without increasing complications such as bleeding. For example, the TXA2 receptor and serotonin receptor blockers simultaneously block both platelet activation pathways, and block the early stages of coagulation with anti-tissue factors.

### *4.4 Anti-infection*

Anti-infection route in recent years found the occurrence of coronary heart disease with systemic inflammatory response. Patients with coronary heart disease were treated with roxithromycin, measured by serum chlamydia pneumonia TWARIgG, IgM antibody titer, high level of antibody titer, the incidence of acute coronary syndrome was significantly increased, and roxithromycin can prevent the incidence of acute coronary syndrome [21].

## 5. Treatment of ACS

### *5.1 Treatment of the STEMI*

Patients with STEMI should undergo cardiac catheterization and coronary angiography immediately, followed by percutaneous coronary intervention (PCI) with a drug-eluting stent implantation. When the ECG suggested STEMI, rapid reperfusion with direct PCI within 120 min reduced mortality by 2% (from 9% to 7%) as compared to thrombolytic therapy. The DANAMI-2 trial enrolled 1,572 patients with STEMI, and were randomly assigned to receive thrombolytic therapy or direct PCI. The 16-year follow-up showed that direct PCI had significant benefits over thrombolytic therapy (cardiac mortality: 18.3% vs 22.7%). If PCI is not performed in STEMI patients within 120 minutes of the first onset, thrombolytic therapy (roteplase, or tenepplase) is given in the absence of contraindications. Patients under the age of 75 should receive full-dose thrombolysis, and those above 75 should receive half-dose thrombolysis. Active or recent bleeding, a recent stroke, significant head trauma, a propensity to bleed readily, and uncontrolled hypertension are all contraindications to thrombolytic therapy. Patients should be brought to the hospital where angiography and PCI can be done within 6 to 24 hours after receiving thrombolytic treatment.

The COMPLETE trial included 4041 patients with STEMI with multivasculopathy, randomized to complete revascularization for all coronary stenosis or revascularization for culprit vessels only. With a median follow-up of 3 years, 7.8% of cardiovascular death or new myocardial infarction occurred in any one of the patients in the complete revascularization group, and 10.5% in the culprit lesion revascularization group (HR=0.74; 95% CI,0.60-0.91; P=0.004). The incidence of revascularization resulting from cardiovascular death, new myocardial infarction, or ischemia was 8.9% in the complete revascularization group and 16.7% in the culprit lesion revascularization group (HR=0.51; 95% CI,0.43-0.61; P <0.001). The results of the Meta-analysis in some randomized trials were consistent with the COMPLETE trial results. However, the optimal timing for targeting the nonculprit coronary artery for revascularization still needs uncertain and should be determined based on the patient's characteristics(coronary anatomical features and renal function.) Only the occluded arteries that cause STEMI should be treated when accompanied with cardiogenic shock, according to clinical trial

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findings, as treating numerous coronary arteries in STEMI patients with cardiogenic shock may have negative side effects.[22].

*5.2 Treatment of the NSTEMI*

For patients with NSTEMI, the initial treatment option, if the first or later troponin measurement exceeds the upper reference 99th percentile, includes conservative treatment (pharmacological treatment) or invasive treatment (prior coronary angiography, in the next place revascularization), determined by outcome of coronary angiography. Basically, drug-eluting stent PCI therapy is used in the patient with NSTEMI, and CABG is used in patients with complex multiangiopathy at low risk of surgical complications. Previous stroke with patients multiple comorbidities, or weakness are at high risk of surgical complications.

The effectiveness of conservative versus invasive treatment was assessed in many randomized studies and meta-analyses. Most of the results support early invasive treatment in intermediate-and high-risk patients, with specific choices depending on the abnormal troponin levels, the ECG change, or risk scores. For low-risk patients, patient preference and doctor-patient decisions are particularly important when deciding whether to treat conservatively or invasively.

A Meta-analysis (8,375 NSTEMI patients) including seven randomized trials showed an association with lower mortality for early invasive treatment of PCI at 24-48 hours after presentation, compared with conservative treatment (2 years of follow-up, 6.5% vs 4.9%). Approximately 60% of NSTEMI patients undergoing coronary angiography receive PCI, 10% undergo coronary artery bypass grafting, and 30% receive initial medication only.

There was an association between non-obstructive coronary plaque and a high ischemic risk in patients who did not receive coronary revascularization. The TRILOGY ACS trial showed that among the 9294 included NSTE-ACS patients treated with pragrel versus clopidogrel, approximately 11% had another ischemic events at two and a half years. In patients with severe bicin orbidities (COVID-19, heart failure or renal failure), probably due to reasons other than plaque rupture.[22]

Oral antiplatelets therapy (aspirin and P2Y12 inhibitors) and parenteral anticoagulants (common heparin, low molecular hepweight heparin, direct thrombin inhibitors, or factor Xa inhibitors) are the recommended initial treatments for patients with ACS, whether invasive or noninvasive. A common complication of the aforementioned treatment is bleeding.

*5.3 Gene therapy*

Gene therapy In 1990, some scholars did direct myocardial injection of gene therapy research, such as vascular endothelial growth factor, to promote the establishment of new blood vessels and collateral circulation, but the gene transfer efficiency of this method is not high. Later, some scholars used the adenoviral vector to introduce exogenous therapeutic genes into the myocardium and blood vessels. The gene transfer efficiency of this method was high, and the target gene could be expressed for a long time, and the number of collateral vessels in the ischemic myocardium increased significantly. The genetic instability of the above gene carriers exists, making the gene efficacy uncertain. Academician Xia Jiahui's discovery of human gene carrier has gained new methods for the treatment of acute coronary artery syndrome, and this progress needs to be further studied [23].

## 6. Conclusion

ACS is a major disease endangering human health worldwide at present, with a high fatality rate. Many factors may lead to the occurrence of this disease. Early identification of high-risk factors and timely, appropriate and optimal treatment are the key to reduce the mortality of ACS patients and improve the prognosis of patients. However, the treatment of the disease should not be limited to new drugs or surgical treatments, but more attention should be paid to the prevention of the disease. Proactive prevention is the best management strategy for disease treatment. Therefore, in clinical practice, we should improve our awareness of prevention and avoid the disease from progressing to an

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uncontrollable stage. In the future, scholars need to conduct more in-depth and sample size research to provide more evidence for the diagnosis and treatment of the disease.

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