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Perspective

Note on Prevention of Coronary Thrombosis

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DESCRIPTION

The mechanism of coronary thrombosis is different from the mechanism of thrombosis that occurs in blood-crowded areas such as deep vein thrombosis. Given the onset of arterial thrombus formation, soluble coagulation factors may not play an important role in the initiation, as they are diluted by the action of bloodstream and cannot reach high concentrations sufficient to form insoluble fibrin. Platelets that can attach to damaged vascular lumens, even in the presence of blood flow shear effects, can play an important role in the initiation of arterial thrombus formation. Therefore, it is necessary to evaluate the mechanism of platelet thrombus formation in the presence of blood flow. However, the current doctrine that fibrinogen binding to activated GP IIb/IIIa is the ultimate common pathway for platelet thrombus formation uses an aggregate function assay system that does not seriously consider the effects of blood flow. It was developed. This review proposes the interaction GP lb of Von Will brand's brand plasma ligand protein Factor (VWF) with its platelets. GP IIb/IIa occurs only in assay systems under the influence of high shear flow conditions such as flow chambers and cone plate viscometers and is an important event leading to the development of arterial thrombosis. A better understanding of the VWF-mediated mechanism of platelet thrombosis is important for the development of better clinical tools to prevent ischemic heart disease and for a complete understanding of the mechanism of coronary thrombosis.

Symptoms of coronary thrombosis

Some people have chest pain (angina). This indicates that atherosclerosis narrowed the coronary arteries.

Others are asymptomatic until the plaque ruptures, due to one of the following:

- Sharp pain behind the sternum or on the left side of the chest that can spread to the left arm (you may feel a lot of pressure on your chest).
- Pain can also spread to the hands, chin, ears, back, abdomen, or right arm.
- A feeling of contraction in or around the throat.
- Dyspnea.
- Sudden fainting or severe dizziness. It is often painful.

What are the danger signals?

- If you suddenly experience severe chest pain or any of the other symptoms mentioned above, it may be a sign of obstruction of the coronary arteries. If you know that you have angina and are already being treated by your doctor, you should be aware of the following:
- If glyceryltrinitrate is no longer considered sufficient for the treatment of chest pain.
- When less effort is required to cause pain than usual. These are signs that coronary artery disease is getting worse. This is because angina usually occurs only after some exercise, and the physical exercise required to induce it is generally the same overnight. I am very worried that angina will occur without any effort. If you suddenly have chest pain at rest and it lasts for more than 20 minutes, you should call the ambulance 999. At the hospital, you can tell if you have a heart attack, severe angina, or something else altogether.

PREVENTION

Primary prevention

Lifestyle: Physical activity can reduce the risk of cardiovascular disease, and it is recommended that those at risk do moderate or 75 minutes of intense aerobic exercise per week. Maintaining a healthy weight, drinking alcohol within the recommended limits, and quitting smoking can reduce your risk of cardiovascular disease.

Most guidelines recommend a combination of different preventive strategies. A 2015 Cochrane review found some evidence that such an approach could help with blood pressure, obesity index, and waist circumference. However, there was insufficient evidence to show its impact on mortality or actual cardiovascular events.

Medication: Statins, a drug that lowers blood cholesterol levels, reduce the incidence and mortality of myocardial infarction. Aspirin has been extensively studied in people at high risk of myocardial infarction. Based on numerous studies in different groups (with or without diabetes), there seems to be no strong advantage over the risk of excessive bleeding. Nevertheless, many clinical guidelines continue to recommend aspirin for primary prophylaxis, and some researchers continue to administer aspirin to individuals with a very high cardiovascular risk but a low risk of bleeding. I believe it should be.

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Secondary prevention

There is a lot of overlap between lifestyle and activity recommendations for preventing myocardial infarction and recommendations that can be adopted as secondary prophylaxis after the first myocardial infarction. Recommendations include quitting smoking, gradually returning to exercise, eating a healthy diet low in saturated fat and cholesterol, drinking alcohol within the recommended limits, and trying to exercise to achieve a healthy weight. Even if you have a stent or heart failure, exercise is safe and effective, and it is recommended that you start gradually after a week or two. Counseling should be provided on the medications used and the signs of depression. Previous studies have suggested the benefits of supplementing with omega 3 fatty acids, but this has not been confirmed.

Nitrate and ACE inhibitors taken every two or three days after a heart attack reduce the risk of death. Other medicines are: Aspirin, like other antiplatelet drugs such as clopidogrel and ticagrelor ("Double Antiplatelet Drug" or DAPT), is continued indefinitely for up to 12 months. If you have another medical condition that requires anticoagulant therapy (such as warfarin), you should adjust based on the risk of additional heart events and the risk of bleeding. Treatment with beta-blockers such as metoprolol and carvedilol should be started within 24 hours unless acute heart failure or heart block is present. The dose should be gradually increased to the maximum tolerated dose. Contrary to long-held thinking, the use of beta-blockers does not appear to affect the risk of death, probably due to improvements in other treatments for MI.