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Thromboembolic Complications in Cardiovascular Diseases: Pathophysiological Mechanisms and Modern Approaches to Prevention

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Resume: The development of thromboembolic complications in cardiovascular diseases (CVDs) is primarily associated with atherothrombosis, characterized by the rupture, fissure, or erosion of atherosclerotic plaques and subsequent thrombus formation. Thromboembolic events can occur in both arterial and venous circulations, significantly increasing morbidity and mortality among CVD patients. Intracardiac thrombosis frequently accompanies atrial fibrillation, myocardial infarction, and valvular defects. The main strategy for preventing atherothrombosis and thromboembolic complications involves the rational use of antiplatelet agents and anticoagulants. Numerous clinical studies and meta-analyses have confirmed the efficacy of antiplatelet therapy, particularly dual antiplatelet therapy (DAPT) combining acetylsalicylic acid (ASA) and clopidogrel, in reducing cardiovascular events by approximately 25%. The combined use of anticoagulants (such as warfarin) and antiplatelet drugs is especially indicated in patients with high thromboembolic risk, including those after stent implantation or with atrial fibrillation. However, the intensification of antithrombotic therapy increases the risk of gastrointestinal adverse reactions, including erosive and ulcerative lesions and bleeding. Therefore, an individualized, risk-balanced approach to combination therapy is essential to optimize efficacy and minimize complications.

Keywords: Atherothrombosis, cardiovascular diseases, antiplatelet therapy, anticoagulants, dual antiplatelet therapy, thromboembolic complications.

The pathogenesis of most cardiovascular diseases (CVDs) is based on atherothrombosis. Morphologically, atherothrombosis is characterized by the presence of a rupture, fissure, or erosion on the surface of an atherosclerotic plaque, which is "covered" by thrombi of various sizes — from mural to completely occlusive thrombi that block the arterial lumen. The clinical manifestations of atherothrombosis may be stable (chronic) or unstable (acute) and depend on the location of the atheroma and the size of the thrombus. Involvement of the brachiocephalic arteries leads to cerebrovascular disorders of varying severity; coronary localization manifests as ischemic heart disease (IHD), ranging from stable angina to acute coronary syndromes; and involvement of the lower limb arteries results in intermittent claudication [1].

At the same time, thromboembolic complications in patients with CVDs can occur not only in the arterial, but also in the venous circulation. Venous thromboembolic complications (VTECs) — combining pulmonary embolism and deep or superficial vein thrombosis — often develop as complications in hospitalized CVD patients during exacerbation of the underlying disease or after surgical and other invasive procedures [2]. Furthermore, certain cardiovascular diseases such as arterial hypertension, myocardial infarction (MI), and chronic heart failure are independent risk factors for venous thromboembolic events . In addition, intracardiac thrombosis frequently occurs in patients with CVD. Atrial fibrillation, chronic heart failure, myocardial infarction, infective

endocarditis, valvular defects, and cardiac tumors predispose to intracardiac thrombus formation. In many cases, patients exhibit combined cardiovascular pathology.

For primary and secondary prevention of atherothrombosis in patients with various cardiovascular disorders, antiplatelet agents and anticoagulants are widely used. Some patients have clear indications for long-term anticoagulant therapy, while others require prolonged or even lifelong antiplatelet therapy, often in the form of dual or combined regimens. The combined use of antiplatelet and anticoagulant drugs is now common worldwide, especially in the United States and Europe [3]. Each year, the need for such an intensive antithrombotic strategy in cardiology increases. According to S.G. Johnson et al. (2007), about 4 out of 10 American patients taking warfarin also receive antiplatelet therapy — most commonly acetylsalicylic acid (ASA), clopidogrel, dipyridamole, or combinations such as ASA + clopidogrel or ASA + dipyridamole. The combined use of antiplatelet agents and warfarin is particularly frequent among patients with heart failure and ischemic heart disease [4].

The large Antithrombotic Trialists' Collaboration meta-analysis, which combined data from 145 clinical trials, demonstrated that antiplatelet therapy in high-risk patients reduces cardiovascular complications by 25% [5]. The most significant benefits of antiplatelet therapy are observed in patients who have experienced an acute coronary syndrome or undergone coronary intervention, particularly stent implantation . Moreover, for many categories of high-risk cardiovascular patients, dual antiplatelet therapy (DAPT) with two agents of different mechanisms of action has proven superior to monotherapy. The most robust evidence supports the combination of ASA and clopidogrel, which has demonstrated higher efficacy than ASA, clopidogrel, or other single antiplatelet agents in numerous large randomized trials [6]. Patients with atrial fibrillation, valvular defects, mechanical valve prostheses, mural thrombi of the left ventricle, and post-myocardial infarction patients at high risk of intracardiac thrombosis also require short-term or long-term oral anticoagulant therapy. Warfarin use in such patients significantly and reliably reduces the risk of cardioembolic stroke.

In recent years, both new pharmacological strategies and expanded invasive treatment options have emerged for patients with stable and unstable angina. Percutaneous coronary interventions (PCI) have become among the most frequently performed medical procedures worldwide. However, thrombosis and restenosis following coronary interventions remain major clinical challenges [7].

Stent thrombosis most commonly occurs within the first month after stenting and often results in Q-wave myocardial infarction or death. With improvements in stent technology and the introduction of mandatory dual antiplatelet therapy (ASA + thienopyridine) for one month, followed by indefinite ASA use, the incidence of stent thrombosis has fallen to approximately 1%. This issue of combination therapy has become even more relevant following recent guideline updates on acute coronary syndrome (ACS) management, which emphasize the long-term benefits of dual antiplatelet therapy after coronary stenting. The recommended duration of combined ASA and clopidogrel therapy has increased to 12 months for most patients with coronary stents[8].

According to the European Society of Cardiology (ESC) guidelines (2008), patients at high risk of thromboembolic events after ST-segment elevation myocardial infarction (STEMI) may receive oral anticoagulants in combination with low-dose ASA [9] (class IIA, level B), clopidogrel (class IIB, level C), or dual antiplatelet therapy (ASA + clopidogrel) (class IIB, level C). The combination of warfarin and ASA is indicated for patients at high thromboembolic risk, while the triple therapy (warfarin + ASA + clopidogrel) is recommended after stent implantation. Recent evidence has also raised concerns about late stent thrombosis following drug-eluting stent implantation, highlighting the critical importance of adherence to antiplatelet therapy [10]. Thus, for most patients with cardiovascular diseases, combined antiplatelet and oral anticoagulant therapy is necessary for the prevention of thromboembolic complications. However, it must be noted that antiplatelet and anticoagulant drugs are associated with an increased risk of gastrointestinal adverse events, including erosive-ulcerative lesions and gastrointestinal bleeding.

Conclusion: Thromboembolic complications remain a major clinical problem in patients with cardiovascular diseases. Atherothrombosis underlies most ischemic events, and its prevention is a key goal of modern cardiology. The combination of antiplatelet and anticoagulant therapy significantly reduces the risk of arterial and venous thromboembolic events and improves patient outcomes. Dual antiplatelet therapy with ASA and clopidogrel remains the gold standard following coronary stenting and acute coronary syndromes. However, this benefit must be balanced against the increased risk of gastrointestinal bleeding and other complications. Optimal prevention and management require a personalized approach based on individual thrombotic and hemorrhagic risk factors, strict adherence to treatment protocols, and continuous clinical monitoring.

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