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The Role of Acetylsalicylic Acid in the Prevention of Cardiovascular Complications in Patients with Stable Ischemic Heart Disease

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Annotation: The historical development and therapeutic significance of acetylsalicylic acid (ASA) have made it one of the most important drugs in modern medicine. From its origins in ancient Sumerian medicine to its synthesis in the 19th century, ASA has evolved from a simple antipyretic agent to a cornerstone in cardiovascular disease (CVD) prevention. The discovery of its antiplatelet properties in the 20th century marked a turning point in the secondary prevention of ischemic heart disease (IHD). Numerous large-scale studies and meta-analyses, including the Antithrombotic Trialists' Collaboration, ISIS-2, SAPAT, and the Swedish national registry, have demonstrated ASA's ability to significantly reduce the risk of myocardial infarction, stroke, vascular death, and graft thrombosis. ASA remains a "gold standard" for the prevention of atherothrombotic events and is endorsed by leading international cardiology societies. Recent research also suggests its potential anticancer effects, particularly in reducing colorectal and postmenopausal breast cancer incidence. Despite its proven efficacy, long-term ASA use requires careful monitoring due to the risk of adverse effects, underscoring the need for individualized therapy and continued investigation into safer and more effective regimens.

Keywords: acetylsalicylic acid,

cardiovascular diseases, ischemic heart disease, atherothrombosis, cyclooxygenase inhibition, myocardial infarction.

The history of salicylate use dates back to around 3000 BC. At that time, an ancient Sumerian clay tablet was discovered containing a text that mentioned the use of a remedy derived from willow bark [1]. Salicylates were recommended for therapeutic purposes by ancient scholars such as the Greek physician Hippocrates (460–370 BC), the Roman encyclopedist Celsus (25 BC – 10 AD), and the Roman philosopher Pliny the Elder (23–79 AD) [2]. In 1763, the English physician E. Stone, speaking before the Royal Society, recommended the use of an infusion made from willow bark for the treatment of febrile conditions. The first form of acetylsalicylic acid (ASA) was obtained in the mid-19th century by the French chemist Charles Gerhardt. In 1897, the modern form of ASA was synthesized, and in 1904, it was released as the original powder under the name "aspirin". Over the past hundred years, ASA has remained one of the most widely used drugs in the world [3]. In addition to its antipyretic effect, approximately fifty years ago its antiplatelet (disaggregant) properties were discovered [4], which led to its use in patients with cardiovascular diseases (CVDs).

At the present stage, CVDs constitute one of the most significant challenges for the medical community. Among them, ischemic heart disease (IHD) is one of the leading conditions [65]. In Russia, mortality from acute cardiovascular events against the background of chronic IHD (CIHD) remains the most common cause of death among the population [5]. Therefore, the issue of preventing acute cardiovascular events and reducing mortality in patients with cardiovascular risk is of critical importance. In the 1960s, a concept was developed that laid the scientific foundation for the prevention of cardiovascular diseases (CVD), particularly ischemic heart disease (IHD). According to this concept, there are three main strategies for IHD prevention: population-based prevention, high-risk strategy, and secondary prevention. One of the key components of secondary prevention is the mandatory use of acetylsalicylic acid (ASA) [6]. Since 1971, ASA has been actively used for the prevention of cardiovascular events . Over the past year, significant changes have occurred in the understanding of stable or chronic IHD. These changes were reflected in the new European Society of Cardiology (ESC) guidelines (2019), where a new term — chronic coronary syndrome (CCS) — was introduced. Approaches to the diagnosis of IHD have been refined, with an increased role of selective coronary angiography. The assessment of pre-test probability (PTP) of IHD has been revised due to a decrease in typical manifestations among symptomatic patients. A new term, "clinical probability" of IHD, was introduced, reflecting various IHD risk factors as modifiers of PTP [7].

Chronic IHD and acute cardiovascular events primarily result from the development and progression of atherosclerosis, which causes alterations in the vascular wall and blood flow characteristics [8]. Endothelial damage in vessels of various localizations leads to regional and systemic changes in the blood's coagulation potential. This increases platelet and erythrocyte aggregation and adhesion activity, activates plasma coagulation factors, and depletes the activity of the blood's anticoagulant components. Platelet production of thromboxane A_2 increases under the influence of the cyclooxygenase (COX) enzyme, which promotes platelet aggregation [9]. Atherosclerotic changes in arteries narrow their lumen, creating conditions for turbulent blood flow, which in turn exposes collagen and leads to endothelial dysfunction. These processes initiate platelet aggregation with the formation of platelet conglomerates and a white thrombus [57]. It is believed that ASA not only inhibits COX and thus prevents thrombus formation, but also, to some extent, may inhibit thrombin formation and the conversion of fibrinogen to fibrin [203]. Therefore, the need for ASA administration in such patients is beyond doubt [10].

The effectiveness of ASA therapy in patients with various levels of atherothrombotic risk has been confirmed in numerous studies [11]. The **Antithrombotic Trialists' Collaboration** meta-

analysis (2002) presented results from 287 randomized studies involving over 200,000 patients at high risk for cardiovascular complications. A significant reduction was demonstrated in the risk of recurrent cardiovascular events overall by 25%, myocardial infarction (MI) by 30%, stroke by 25%, and cardiovascular mortality by 17%. The effectiveness of antiplatelet therapy was confirmed for each high-risk category in individual placebo-controlled trials [12]. In 2009, the same collaboration published the next phase of its research on the efficacy of ASA. This meta-analysis included 16 clinical trials with 17,000 patients who underwent secondary prevention of cardiovascular complications. The findings conclusively confirmed the high efficacy of ASA in preventing atherothrombosis. The results showed that ASA significantly reduced the risk of a first myocardial infarction in patients with high cardiovascular risk by 32% and the total number of cardiovascular events by 15% [13].

Significant evidence of the preventive effect of ASA was also obtained in the large prospective randomized **Swedish Angina Pectoris Trial (SAPAT),** conducted in 94 clinics across Sweden. The study included 2,035 patients with stable angina pectoris who received 75 mg of ASA or placebo. The trial demonstrated a significant reduction in the risk of myocardial infarction and sudden death by 34% compared to the placebo group ($p = 0.003$), as well as a 22–32% decrease in vascular death, stroke, and overall mortality [14]. The **U.S. Physicians' Health Study,** involving 333 participants with stable angina who received 325 mg of ASA for 5 years, showed an 87% reduction in the incidence of primary myocardial infarction ($p < 0.001$). Among healthy volunteers taking an equivalent dose for the same period, this reduction was 44% [15].

In patients after coronary artery bypass grafting (CABG), the use of acetylsalicylic acid (ASA) reduced the incidence of graft thrombosis by 50% [16]. It was also shown that in patients who underwent coronary angioplasty, ASA administration reduced the risk of cardiovascular events by 53%. This finding was confirmed by nine clinical trials involving a total of 3,000 patients [17]. The **ISIS-2 (Second International Study of Infarct Survival),** which included 17,187 patients with acute myocardial infarction (AMI), demonstrated that ASA administration in the acute phase of AMI increased one-month survival, preventing 38 fatal or nonfatal cardiovascular events per 1,000 treated patients [18]. Further follow-up of these patients showed that the improvement in survival achieved during the first month persisted in subsequent years . Two large contemporary studies have recently been completed evaluating the effectiveness of ASA under modern clinical conditions. The **Sundström study** analyzed data from the Swedish national registry between 2005 and 2009. The objective was to determine how cardiovascular risks change after discontinuation of ASA .

The registry included 601,527 patients who were taking ASA in cardioprotective doses, both for primary and secondary prevention. The results revealed that patients who discontinued ASA had a significantly higher cardiovascular risk (HR = 1.37; 95% CI, 1.34–1.41). Those who stopped taking ASA experienced a more than 30% increased risk of major cardiovascular events. Notably, this elevated risk appeared immediately after discontinuation and did not decrease over time . ASA is considered the **"gold standard"** for the prevention of cardiovascular events [19]. Its use as an antiplatelet agent is included in the recommendations of most major professional societies of cardiologists, neurologists, and other specialists, including the **American College of Cardiology, American Heart Association, European Society of Hypertension, American Diabetes Association, European Society of Cardiology, American College of Thoracic Surgeons,** and the **Russian Society of Cardiology,** among others. ASA is one of the oldest and most extensively studied pharmaceutical agents. Nevertheless, new properties of this drug continue to be discovered. For example, **Rothwell P.M. et al.** conducted a meta-analysis of four clinical trials involving 14,033 patients taking ASA in cardioprotective doses. The results demonstrated that ASA in such doses significantly reduced the incidence and mortality from colorectal cancer [20]. Regular use of ASA, but not other nonsteroidal anti-inflammatory drugs (NSAIDs), was also associated with a 20% reduction in the risk of postmenopausal breast cancer, regardless of estrogen or progesterone receptor status of the tumor . All of the above

evidence indicates that patients should receive ASA for an indefinite period; however, this long-term use often leads to the development of adverse side effects [21].

Conclusion. Acetylsalicylic acid (ASA) has a long and distinguished history in medicine, evolving from a natural remedy to a vital pharmacological agent in cardiovascular protection. Its proven antiplatelet and cardioprotective effects have solidified its role in the prevention of ischemic events and improved survival outcomes in high-risk patients. Modern clinical trials and meta-analyses continue to validate ASA's efficacy not only in secondary prevention of cardiovascular diseases but also in potential cancer risk reduction. However, prolonged ASA administration must be balanced against the risk of adverse effects, emphasizing the importance of individualized dosing strategies and patient selection. ASA remains a cornerstone of evidence-based preventive cardiology and continues to reveal new therapeutic potentials.

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