DOI 10.64108/imh.2026.2.6.12
UDC 616.12-005.4-06:616.12-008.331.1]-053.9:615.273.5

THE EFFECT OF ANTIPLATELET THERAPY ON PLATELET FUNCTION IN ELDERLY PATIENTS WITH CORONARY ARTERY DISEASE AND ESSENTIAL HYPERTENSION

V. S. Pasko

State institution of science «Center of innovative healthcare technologies" State administrative department, Kyiv, Ukraine

ORCID ID:0000-0002-4124-3100, email: victoriapasko@ukr.net

Abstract. Functional whole blood haemostatic assays are used increasingly to monitor medical treatment, platelet reactivity and are also applied for in-vitro evaluations of the haemostatic potential of stored platelets. Antiplatelet therapy is the cornerstone in the management of coronary artery disease and essential hypertension. Platelet function testing might not only yield clinical benefits for the patients but also economical advantages by identifying a right drug at a right dose for a right patient.

Purpose to evaluate the effect of antiplatelet therapy on platelet function in elderly patients with coronary artery disease and essential hypertension.

Materials and methods. 19 elderly patients with coronary artery disease and essential hypertension, 15 healthy individuals were included in the study and venous blood samples were obtained. All patients had an indication for antiplatelet therapy: mono antiplatelet therapy with aspirin or clopidogrel or dual antiplatelet therapy with aspirin and clopidogrel in standard doses. Platelets were activated with adenosine diphosphate (20 μM), arachidonic acid 0.5 mM), ristocetin (1 mg/mL), collagen (2 μg/mL) and analysed by whole blood impedance aggregometry (CHRONO-LOG 700).

Results. It was noted that amplitude of the aggregation with adenosine diphosphate and arachidonic acid in elderly patients was significantly reduced relative to control group by 2.0 ($p<0.05$) and 3.6 ($p<0.05$) times, respectively.

Slope of the aggregation was reduced in the elderly by 2.5 ($p<0.05$) and 1.5 ($p<0.05$) following induction with arachidonic acid and collagen compared to normative values, respectively.

Area under the curve when using adenosine diphosphate, arachidonic acid and collagen was therefore 2.3 times ($p<0.05$), 3.6 times ($p<0.05$) and 1.8 times lower ($p<0.001$) in patients, respectively.

63.2% patients showed decrease in adenosine diphosphate-induced aggregation compared to the control group, while 36.8% showed no decrease.

The direct correlation ($r=0.47$; $p<0.05$) was found between the slope of adenosine diphosphate-induced aggregation and the level of total serum cholesterol.

There is also the direct correlation ($r=0.597$; $p<0.05$) between the amplitude of adenosine diphosphate-induced platelet aggregation and platelet count.

The direct correlation ($r=0.57$; $p<0.05$) was found between the amplitude of adenosine diphosphate-induced platelet aggregation and level of gamma-glutamyltranspeptidase.

There is also the direct correlation ($r=0.60$; $p<0.05$) between the amplitude of the aggregation induced by arachidonic acid and level of activated partial thromboplastin time.

Conclusions. The data demonstrate the overall high efficacy of antiplatelet therapy in elderly patients with coronary artery disease and essential hypertension, particularly with regard to its effect on arachidonic acid pathway. However, the fact that nearly 37% of elderly patients do not show a reduction in adenosine diphosphate-induced platelet aggregation indicates the need for a personalised approach to dosage selection or a change in antiplatelet therapy.

Keywords: coronary artery disease, essential hypertension, platelet function, elderly patients, antiplatelet therapy, mono antiplatelet therapy, dual antiplatelet therapy, aspirin (acetylsalicylic acid), clopidogrel.

Introduction. Although need to assess platelet function (PF) is still debated, the need for platelet inhibition in patients suffering from cardiovascular and cerebrovascular diseases is well established and proven. Acute coronary syndrome (ACS), a condition characterized by acute cardiac ischemia, is among the major causes of death from cardiovascular diseases. However, whether there is a correlation between platelet reactivity and major adverse cardiovascular events remains debatable, and whether tests should be tailored for ACS patients after percutaneous coronary intervention (PCI) is still under discussion. The meta-analysis outcomes indicate that in ACS patients re-

ceiving PCI and using dual antiplatelet therapy (DAPT) for 1-2 years, high platelet reactivity was independently positively correlated with major adverse cardiovascular events, all-cause (or cardiac) mortality, recurrent myocardial infarction (MI), in-stent restenosis, and stroke. This suggests that platelet reactivity testing has clinical and translational significance in predicting patients' risk of adverse cardiovascular events [6]. There is a wide variety of tests available, often using different methodologies, a clear lack of standardisation, and, finally, evidence is emerging on the clinical significance of PF testing.

Assessment of PF may not only provide clinical benefits for patients, but also economic benefits by identifying a right drug at a right dose for a particular patient.

Research justification. Since antiplatelet therapy (APT) is one of the most important components of treatment for patients with various forms of coronary artery disease (CAD), search for methods of objective control over its implementation is an important scientific task.

The era of aspirin (A) use as an antiplatelet agent began in 1946, when its accidental prescription as an anti-inflammatory agent made it possible to establish the reduction in the risk of MI in men. Today, A is recognised as the gold standard of antiplatelet strategy for primary and secondary prevention of cardiovascular events. Recently, it has been established that A not only has traditional anti-inflammatory and antiplatelet properties, but also improves endothelial function and has a hypotensive effect in untreated arterial hypertension (AH). The hypotensive effect is time-dependent and more pronounced when A is taken in the evening compared to when it is taken after waking up.

However, A does not always solve all APT problems. Despite treatment with acetylsalicylic acid (ASA), 10–15% of patients with ACS die or suffer a MI with ST elevation. A is widely used for preventing ischaemic events. About 20%-40% of patients have A resistance (AR), which prevents them from benefiting from A medication [3]. 20% of patients who have had ACS require rehospitalisation, and one-third of patients with atherosclerotic lesions of lower extremity arteries experience various complications and diseases within four years, half of which are fatal. A possible explanation for insufficient effect of ASA is that the drug blocks only one of the pathways of platelet activation, associated with the inhibition of cyclooxygenase (COX) and the formation of thromboxane A_2. All this determines the advisability of enhancing ASA therapy in patients with various manifestations of atherothrombosis. Thus, in patients with ACS without ST segment elevation, combination therapy with simultaneous blockade of platelet COX-1 with A and P2Y12 receptors to adenosine diphosphate (ADP) with clopidogrel (C) has an additive effect and reduces the incidence of the first primary composite endpoint, cardiovascular mortality, non-fatal MI and stroke by 20% compared with ASA monotherapy [3]. However, data comparing P2Y12 inhibition with C versus COX inhibition by ASA is missing. It is well known that the antiplatelet effects of ASA and C are frequently impaired (high on-treatment platelet reactivity [HTPR]. Impaired pharmacodynamic response to C was more frequent as HTPR to ASA [4]. To date, the efficacy and safety of this combination strategy has been proven in patients with ACS with ST segment elevation (Chinese Cardiac Study). C is the drug of choice in patients with ACS who are intolerant to ASA, and the dose of A when combined with C should not exceed 100 mg/day.

C is a thienopyridine derivative that inhibits ADP-dependent platelet activation, leading to inhibition of fibrinogen (FG) binding to GP IIb/IIIa complex. Recent studies indicate the existence of at least two types of ADP receptors on the platelet membrane. One of them is a high-affinity (P2Y1) receptor, whose activation leads to a change in the shape of platelets from discoid to spherical and ensures rapid entry of calcium ions through the outer platelet membrane. Another platelet receptor for ADP, a low-affinity purinergic receptor of the second type, is responsible for the mobilisation of calcium ions from platelet depots, which leads to conformational changes in the GP IIb/IIIa complex, its binding to FG and other ligands, and platelet aggregation. There is evidence that thienopyridines affect the type 2 ADP receptor [1].

C is the modern specific antagonist of ADP receptors that selectively inhibits ADP-induced platelet aggregation, slows down platelet aggregation by inhibiting binding of ADP to P2Y12 receptor of platelet membrane, which leads to a decrease in adenylate cyclase activity, resulting in the antithrombotic effect of the drug [1]. This inhibition is irreversible, and platelets exposed to the drug remain inactive throughout their entire lifespan (on average 7–10 days). Platelet inhibition is specific and does not significantly affect COX and arachidonic acid metabolism. Function of new platelets formed after end of C administration is not affected. The irreversible binding of active metabolite of C to P2Y12 receptor on platelet membrane leads to inhibition of ADP-dependent release of contents of dense platelet granules (ADP, calcium and serotonin) and its alpha granules (FG and thrombospondin) responsible for platelet aggregation. Although C blocks ADP-induced binding of FG to GP IIb/IIIa receptors, it has no direct effect on expression of this glycoprotein. Unlike ASA, C does not block COX and, accordingly, synthesis of thromboxane A_2 and prostacyclin. Regarding use of C in a special category of patients, it should be noted that in people over 75 years of age, concentration of C metabolite in blood plasma is higher than in younger people, but this does not require changes in dosage. In patients with concomitant reduced renal function, level of C metabolite is significantly lower than in healthy individuals, and platelet aggregation inhibition is reduced by 25%. However, no dependence of bleeding time on renal function was found, which served as basis for recommendations not to change dose of C in patients with renal impairment. There is virtually no experience with use of C in patients with severe hepatic impairment. In patients with gastrointestinal tract impairment, according to consensus guidelines for the management of patients with various forms of CAD, C is recognised as an alternative to contraindicated A. For patients with diabetes mellitus (DM), the combination of ASA and C is indicated

due to the synergistic antiplatelet effect. The efficacy of C has been established in a number of studies [1]. It has been found that C significantly reduces risk of vascular ischemic events and overall mortality in patients with various (but stable) manifestations of atherothrombosis. The multicentre CURE study confirmed advantages of C in combination with ASA in reducing cardiovascular mortality and recurrent coronary events in unstable angina, albeit at the expense of an increase in major (but not fatal) bleeding. Today, C is mandatory in antithrombotic regimens in cases of coronary artery stenting, as it has been shown to be effective in preventing post-interventional thrombosis.

Intensive research into effects of C, particularly on PF and other haemostasis parameters in patients with ACS and acute MI, began only a few years ago [1]. Although thienopyridines do not alter coagulation tests that integrally reflect shifts in coagulation cascade (activated partial thromboplastin time (APTT), blood clotting time) [1], they nevertheless have a hypocoagulant effect by inhibiting platelet contribution to plasma clotting potential, as recorded in relevant studies on animal models using platelet plasma. According to the recommendations of the American College of Cardiology for the treatment of ACS without ST segment elevation (2025) [5] and the European Society of Cardiology on the use of antiplatelet drugs (2023) [2], C is the recommended antiplatelet drug with proven efficacy and safety for the treatment of patients with ACS [1]. Today, C is recognised as the drug of choice in the treatment of not only ACS but also stable angina. The CAPRIE and CURE studies have demonstrated the advantages of long-term C over ASA in high-risk patients with the history of MI, stroke, atherosclerotic disease of lower extremities, DM, and duodenal and gastric ulcers, which make it impossible to take ASA [1]. However, despite long-term use of antiplatelet drugs, not all patients are protected from development of cardiovascular events. Recently, issues of clinical and biochemical resistance to antiplatelet drugs have been widely discussed. According to the literature, in 5–40% of patients, despite long-term use of low-dose ASA, its antiplatelet effect is absent, and in 8–30% there is no effect from the use of C [1]. The highest number of aspirin-resistant (AR) patients is found among elderly people with DM. It has recently been established that women, especially those who smoke, predominate among AR patients. In addition, women have lower haemoglobin levels, which may explain lower cardioprotective effect of ASA in development of the first non-fatal MI. AR patients have higher levels of follicle-stimulating hormone. Patients with DM who are subject to interventional PCI, in particular stenting, were found to be most resistant to ASA and C; in addition, they have higher body mass index. C resistance (CR) correlates closely with insulin resistance and glycated haemoglobin levels. The data on the increased efficacy of C in patients who smoke were unexpected. The authors explain the increased antiplatelet effect of the drug by its conversion to an active form during metabolism by cytochrome P450 enzymes, which is activated by polycyclic aromatic hydrocarbon released by cigarette smoke. CR is associated with higher risk of developing ACS with ST segment elevation. Thus, half of AR patients were also resistant to C. They had higher MB-CFC levels. Given that one of its metabolites is considered active when C is used, due to transformational changes in liver, combining it with atorvastatin negates both the antiplatelet and hypolipidemic effects of each of them [1].

Purpose of the research. To evaluate the effect of APT on PF in elderly patients with CAD and essential hypertension (EH).

Materials and organization of the research. 19 elderly patients (mean age 70.2±5.5) with CAD and EH were included in the study and venous blood samples were obtained. The control group consisted of 15 healthy individuals without CAD and EH who were not prescribed antiplatelet drugs. All patients had an indication for APT: mono APT with A or C or DAPT with A and C in standard doses. DAPT was the treatment of choice in patients with ACS who underwent PCI or coronary artery bypass grafting. Platelets were activated with ADP (20 μM), AA (0.5 Mm), ristocetin (1 mg/mL), collagen (2 μg/mL) and analysed by whole blood impedance aggregometry (CHRONO-LOG 700, CHRONO-LOG Corporation, United States of America). The following indices were calculated: amplitude of the aggregation (AA) (Ohm); slope of the aggregation (SA) (Ohm/min), lag phase (L-P) (sec) and area under the curve (AUC) (Ohm/min^2).

Statistical data processing was performed using MedStat software package and Microsoft Excel software.

Results of the research. Differences in platelet haemostasis were found in elderly patients with CAD and EH when taking APT. When comparing aggregation ability, it was noted that AA with ADP and arachidonic acid in patients was significantly reduced relative to control group by 2.0 ($p<0.05$) and 3.6 ($p<0.05$) times, respectively (table 1).

Thus, SA was reduced in the elderly by 2.5 ($p<0.05$) and 1.5 ($p<0.05$) following induction with arachidonic acid and collagen, respectively.

AUC when using ADP, arachidonic acid and collagen was therefore 2.3 times ($p<0.05$), 3.6 times ($p<0.05$) and 1.8 times lower ($p<0.001$) in patients with CAD and EH, respectively.

63.2% showed decrease in ADP-induced aggregation compared to control group, while 36.8% showed no decrease.

When studying the relationship between platelet aggregation capacity indicators and certain biochemical indicators, the direct correlation ($r=0.47$; $p<0.05$) was found between the slope of ADP-induced aggregation and the level of total serum cholesterol. This indicates that hypercholesterolaemia increases the risk of thrombosis by activating the platelet-vascular link of haemostasis.

There is also the direct correlation ($r=0.597$; $p<0.05$) between the amplitude of ADP-induced platelet aggregation and platelet count.

Table 1

Indicators of induced platelet aggregation in elderly patients with CAD and EH when taking APT compared to control group

Indicator		Control n=15 -1-	Patients with CAD and EH when taking APT n=19 -2-	P_{1-2}
AA, Ohm	ADP	5.3±1.9	2.6±2.8	<0.01
	Arachidonic acid	5.8±3.6	1.6±3.2	<0.05
	Ristocetin	12.8±5.0	8.5±10.0	>0.05
	Collagen	9.9±4.7	5.9±4.8	>0.05
SA, Ohm/min	ADP	4.7±2.0	3.5±2.5	>0.05
	Arachidonic acid	5.9±3.8	2.4±3.0	<0.05
	Ristocetin	11.0±5.8	9.4±11.5	>0.05
	Collagen	7.1±3.8	4.6±2.5	<0.05
L-P, sec	ADP	63.7±24.9	43.6±45.2	>0.05
	Arachidonic acid	46.8±29.2	21.1±48.8	>0.05
	Ristocetin	39.7±22.3	42.2±25.8	>0.05
	Collagen	80.1±14.9	82.5±54.2	>0.05
AUC, Ohm/min^2	ADP	24.4±15.7	10.8±12.0	<0.05
	Arachidonic acid	17.0±12.2	4.7±11.4	<0.05
	Ristocetin	47.2±18.0	34.0±46.2	>0.05
	Collagen	31.1±13.4	17.0±11.5	<0.001

Note: P_{1-2} – statistical significance of difference between control and elderly patients with CAD and EH.

The direct correlation (r=0.57; p<0.05) was found between the amplitude of ADP-induced platelet aggregation and level of gamma-glutamyltranspeptidase (GGT). It is known that GGT is the liver enzyme (protein) whose activity in blood increases in liver diseases (hepatitis, fatty hepatosis, cholestasis) and alcohol abuse. All these conditions are accompanied by the disturbance in the serum lipid spectrum. Thus, the combination of EH and CAD with liver pathology increases the thrombogenic potential of blood.

There is also the direct correlation (r=0.60; p<0.05) between AA induced by arachidonic acid and level of APTT. This may indicate compensatory activation of plasma haemostasis factors in response to inhibition of its platelet link, since APTT is the integral indicator of plasma haemostasis.

Discussion of the results. The results presented indicate significant changes in platelet function in elderly patients with CAD and EH undergoing APT.

1. Efficacy of APT

The key observation is the significant reduction in platelet aggregation under the influence of various inducers compared with control group:

Arachidonic acid: the most pronounced inhibition — AA fell by 3.6, and AUC also decreased by 3.6. This is the direct indication of patients' high sensitivity to aspirin-type drugs (COX-1 inhibitors), as it is this pathway that blocks the aggregation cascade induced by arachidonic acid.

ADP and collagen: 2.3-fold and 1.8-fold reduction in AUC, respectively, indicates a complex inhibition of platelet receptor activity (particularly $P2Y_{12}$ receptors, if patients were taking C).

2. Age-related and structural characteristics

Elderly age: the slowing of SA (particularly for collagen and arachidonic acid) in elderly patients suggests that therapy in this group achieves the desired effect by reducing the risk of thrombus formation. However, such a significant reduction (by 2.5–3.6 times) may also signal the increased risk of haemorrhagic complications (bleeding), which requires careful monitoring.

Correlation (r=0.597): the presence of the direct link between platelet count and the amplitude of ADP-induced aggregation is expected but important for clinical practice. This confirms that the intensity of the response to ADP stimulation depends in part on the quantitative composition of blood, and not solely on functional state of individual cell.

3. Heterogeneity of response to treatment

The distribution of patients according to their response to ADP deserves particular attention:

63.2% — «responders»: patients in whom the expected reduction in aggregation is observed.

36.8% — «non-responders» or those with a low response: a fairly high percentage of patients in whom ADP-induced aggregation has not decreased. This may indicate: genetic resistance to antiplatelet agents [1],

clinical ineffectiveness of the current dose of the drug, high residual risk of thrombotic events (stroke, heart attack) despite treatment [6].

Conclusions. The data demonstrate the overall high efficacy of APT in elderly patients with CAD and EH, particularly with regard to its effect on the arachidonic acid pathway. However, the fact that nearly 37% of elderly patients do not show a reduction in ADP-induced platelet aggregation indicates the need for a personalised approach to dosage selection or a change in APT.

Prospects for further research. Comparison of platelet aggregation indices in patients with CAD and EH receiving monotherapy with A or C or DAPT depending on response to treatment.

Conflict of interest. The author declares that she has no conflict of interest regarding this study, including financial, personal, authorship or other nature, which could affect the study and its results presented in this article.

Funding. The study was conducted without financial support.

The manuscripts provided were produced with state funding as part of the research project "Development and improvement of methods for preventing complications of AH and CAD with concomitant diseases in civil servants" (state registration number 0114U002119).

This work was carried out in accordance with the Declaration of Helsinki. The study protocol was approved by the Local Ethics Committee for all participants. Informed consent was obtained from patients to conduct the research.

References:

1. Dean L., Kane M., Pratt V. M., Scott S. A., Pirmohamed M., Esquivel B., Kattman B. L., Malheiro A. J. (2022). Clopidogrel therapy and CYP2C19 genotype.
2. Byrne, R. A., Rossello, X., Coughlan, J. J., Barbato, E., Berry, C., Chieffo, A., Claeys, M. J., Dan, G.-A., Dweck, M. R., Galbraith, M., Gilard, M., Hinterbuchner, L., Jankowska, E. A., Jüni, P., Kimura, T., Kunadian, V., Leosdottir, M., Lorusso, R., Pedretti, R. F. E., ... Zeppenfeld, K. (2023). 2023 ESC Guidelines for the management of acute coronary syndromes. *European Heart Journal*. https://doi.org/10.1093/eurheartj/ehad191
3. Liu, Q., Guo, S., Wang, N., Wang, K., Mo, S., Li, X., Zhang, Y., He, H., Wang, S., & Wu, J. (2023). Model based on single-nucleotide polymorphism to discriminate aspirin resistance patients. *Stroke and Vascular Neurology*, svn—2022–002228. https://doi.org/10.1136/svn-2022-002228
4. M'Pembele, R., Ahlbrecht, S., Helten, C., Mourikis, P., Naguib, D., Zako, S., Trojovsky, K., Huhn, R., Petzold, T., Hohlfeld, T., Zeus, T., Kelm, M., Dannenberg, L., & Polzin, A. (2022). High on-treatment platelet reactivity: Aspirin versus clopidogrel. *Pharmacology*, 1–7. https://doi.org/10.1159/000527816
5. Rao S. V., O'Donoghue M. L., Ruel M., Rab T., Tamis-Holland J. E., Alexander J. H., Baber U., Baker H., Cohen M. G., Cruz-Ruiz M., Davis L. L., de Lemos J. A., DeWald T. A., Elgendy I. Y., Feldman D. N., Goyal A., Isiadinso I., Menon V., Morrow D. A., Mukherjee D., Platz E., Promes S. B., Sandner S., Sandoval Y., Schunder R., Shah B., Stopyra J. P., Talbot A. W., Taub P. R., Williams M. S. (2025). ACC/AHA/ACEP/NAEMSP/SCAI Guideline for the management of patients with acute coronary syndromes: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. JACC 85(22).
6. Wang, J., Shi, X., Chen, L., Li, T., Wu, C., & Hu, M. (2024). Platelet reactivity with MACE in acute coronary syndrome patients post-pci under dual antiplatelet therapy: A meta-analysis. *British Journal of Hospital Medicine*, 1–17. https://doi.org/10.12968/hmed.2024.0370

УДК 616.12-005.4-06:616.12-008.331.1]-053.9:615.273.5

ВПЛИВ АНТИТРОМБОЦИТАРНОЇ ТЕРАПІЇ НА ФУНКЦІЮ ТРОМБОЦИТІВ У ХВОРИХ ПОХИЛОГО ВІКУ НА ІШЕМІЧНУ ХВОРОБУ СЕРЦЯ З ГІПЕРТОНІЧНОЮ ХВОРОБОЮ

В. С. Пасько

Державна наукова установа «Центр інноваційних технологій охорони здоров'я» Державного управління справами, м. Київ, Україна,

ORCID ID: 0000-0002-4124-3100, email: victoriapasko@ukr.net

Резюме. Функціональні гемостатичні аналізи в цільній крові все частіше використовуються для моніторингу медичного лікування та реактивності тромбоцитів, а також застосовуються для in vitro оцінки гемостатичного потенціалу тромбоцитів. Антитромбоцитарна терапія є основою лікування ішемічної хвороби серця та гіперто-

нічної хвороби. Дослідження функцій тромбоцитів може принести не лише клінічну користь пацієнтам, а й економічні переваги завдяки підбору необхідного препарату в правильній дозі для конкретного пацієнта.

Мета дослідження – оцінити вплив антитромбоцитарної терапії на функцію тромбоцитів у пацієнтів похилого віку з ішемічною хворобою серця та гіпертонічною хворобою.

Методи. Група обстежених була сформована з урахуванням 19 хворих похилого віку на артеріальну гіпертензію, у тому числі поєднану з ішемічною хворобою серця, яким призначена антитромбоцитарна терапія: монотерапія аспірином чи клопідогрелем або подвійна антитромбоцитарна терапія (аспірин і клопідогрель) у рекомендованих протоколом дозах. Групу контролю склали 15 здорових осіб. Як індуктори агрегації були застосовані аденозиндифосфат у кінцевій концентрації 20 мкмоль/л, арахідонова кислота у кінцевій концентрації 0,5 ммоль, ристоцетин – 1 мг/мл та колаген – 2 мкг/мл відповідно. Нами проводилось визначення агрегації в цільній крові імпедансним методом (CHRONO-LOG 700).

Результати. Привернуло увагу те, що показник ступеня агрегації з аденозиндифосфатом і арахідоновою кислотою у пацієнтів похилого віку значно зменшився відносно контролю у 2,0 ($p<0.05$) і 3,6 рази ($p<0.05$) відповідно.

Уповільнювалась швидкість агрегації при індукції арахідоновою кислотою та колагеном у похилих у 2,5 ($p<0.05$) та 1,5 рази ($p<0.05$), порівняно з нормативними показниками відповідно.

Площа агрегаційної кривої при використанні аденозиндифосфату, арахідонової кислоти та колагену, відповідно, була нижчою у пацієнтів у ,.3 ($p<0.05$), 3,6 ($p<0.05$) та 1,8 разів ($p<0.001$).

У 63,2% пацієнтів відзначено зниження аденозиндифосфат-індукованої агрегації, порівняно з групою контролю, а у 36,8% хворих його не спостерігалося.

Виявлено прямий кореляційний зв'язок ($r=0.47$; $p<0.05$) між швидкістю аденозиндифосфат-індукованої агрегації та рівнем загального холестеролу сироватки.

Також простежується прямий кореляційний зв'язок ($r=0.597$; $p<0.05$) між ступенем аденозиндифосфат-індукованої агрегації тромбоцитів і числом тромбоцитів.

З'ясовано прямий кореляційний зв'язок ($r=0.57$; $p<0.05$) між ступенем аденозиндифосфат-індукованої агрегації тромбоцитів і рівнем гама-глутамілтранспептидази.

Відзначено прямий кореляційний зв'язок ($r=0.60$; $p<0.05$) між ступенем агрегації тромбоцитів, індукованої арахідоновою кислотою, та рівнем активованого часткового тромбопластинового часу.

Висновки. Дані демонструють загальну високу ефективність антитромбоцитарної терапії у пацієнтів похилого віку з ішемічною хворобою серця та гіпертонічною хворобою, особливо щодо впливу на шлях арахідонової кислоти. Проте наявність майже 37% хворих похилого віку, які не демонструють зниження аденозиндифосфат-агрегації, вказує на необхідність персоніфікованого підходу до вибору дозування або зміни антиагреганта.

Ключові слова: ішемічна хвороба серця, гіпертонічна хвороба, функція тромбоцитів, похилий вік, антитромбоцитарна терапія, монотерапія антитромбоцитарними препаратами, подвійна антитромбоцитарна терапія, аспірин (ацетилсаліцилова кислота), клопідогрель.

Дата першого надходження до видання 03.04.2026
Дата прийняття статті до друку після рецензування 08.05.2026
Дата публікації (оприлюднення) статті 28.06.2026