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*Review Article*

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# Collaboration Between Physician and Clinical Pharmacist in Pharmacotherapy of Ischemic Heart Disease

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**ABSTRACT**
This study addresses the importance of clinical pharmacist-physician collaboration in Georgia in the process of proper management of ischemic heart disease. Ischemic heart disease is one of the leading causes of mortality in the world, its management is complex, and pharmacotherapy is diverse.

The aim of the study was to analyze drug interactions in ischemic heart disease. The task of the study was to analyze prescriptions in the intensive care unit, taking into account the therapeutic properties of drugs and the problem of polypharmacy, and to determine the expected side effects and complications, and to correct the prescription in collaboration with a doctor. The research method was to discuss patient cases, record cases of polypharmacy in prescriptions, determine the expected drug interactions and pharmacological analysis taking into account the basic principles of pharmacokinetics and pharmacodynamics. Within the scope of the study, we will discuss one case, and in the process of collaboration with a cardiologist, recommendations for correcting the prescription are given in the conclusions.

The study also highlighted the role and importance of clinical pharmacists in the process of collaboration with doctors in Georgia. The involvement of clinical pharmacists in the decision-making process with doctors increases the effectiveness of treatment, and this collaboration also increases patient education and awareness. Taking the above into account, ultimately, the number of cases of polypharmacy and the expected negative consequences are reduced.

Ischemic heart disease (IHD), also known as coronary artery disease (CAD), is one of the most common diseases worldwide, and its preventive, diagnostic, and therapeutic options are increasingly being developed. Coronary artery disease (CAD) refers to inadequate blood supply to the coronary arteries of the heart, which is caused by atheroma. Clinical manifestations of coronary artery disease include so-called silent angina, sudden cardiac arrest, exercise or stable angina, acute coronary syndrome (unstable angina, myocardial infarction). General diagnosis of the disease can be made by various methods: pronounced symptoms, exercise tests, electrocardiography (ECG), blood cardiac marker studies, coronary angiography, and others [1].

the American Heart Association (AHA), 244.1 million people were carriers of this disease in 2020, with a higher prevalence in men than in women (in men - 141.0 million, among women - 103.1 million cases) In 2020, countries in North Africa, Central and South Asia, and Eastern Europe had the highest incidence of ischemic heart disease. The mortality rate in 2020 was 112.37 per 100,000 people. This rate was highest for countries in North Africa, Eastern Europe, and Central Asia [2].

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The main cause of ischemic heart disease is the development of atherosclerosis and the formation of atherosclerotic plaques. How does this process occur? The process involves the deposition of cholesterol and various substances in the coronary arteries - the formation of atheroma, which narrows the lumen of the blood vessels. As atheroma grows in size, it further clogs the blood vessel and further disrupts the normal process of blood flow. When is it most noticeable? Of course, during physical exertion, when the heart's demand for oxygen consumption increases sharply. As a result, we get a process called myocardial ischemia, the heart cannot receive an adequate amount of oxygen-rich blood and cannot perform its normal function - pumping blood. After a certain period of time, the formed atheroma may rupture, the released substances cause platelet aggregation and thrombus formation, which further aggravates the situation and may completely occlude the lumen of the blood vessel, followed by the development of acute coronary syndrome, which includes the following: unstable angina, ST-segment elevation myocardial infarction (STEMI) or non-ST-segment elevation myocardial infarction (NSTEMI). The severity of the resulting picture depends on various factors: the type of blood vessel, its location, the degree of occlusion, the extent of the damaged area, the time elapsed since the onset of the lesion, etc [3].

In addition, a relatively rare cause of coronary artery disease can be a spasm of the coronary arteries, which involves their sudden, short-term narrowing. This condition is known as Prinzmetal's angina: the spasm causes pain in the chest area, which is not associated with physical exertion and mainly develops at rest, at night or in the early morning. The causes may be: stress, smoking, cold weather, trauma, viral infections, use of harmful substances (e.g. cocaine), etc. It is characteristic that in most cases it is a process without atherosclerotic plaque.

The ECG shows a temporary, transient elevation of the ST segment. After the spasm, the ECG normalizes [3].

For the prevention of any disease, it is important to know its risk factors. Risk factors for ischemic heart disease are: gender - male, age - over 45 years in men, over 55 years in women, family history (in case of confirmed lethality in first-degree relatives, the risk increases for men over 55 years, for women over 65 years), low levels of high-density lipoproteins (HDL) in the blood, High levels of low-density lipoprotein (LDL) in the blood, type 2 diabetes, obesity, smoking, physical inactivity, high levels of C-reactive protein (CRP) in the blood, high levels of lipoprotein a, high blood pressure, dietary factors, etc. It is important that the American Heart Association has created an algorithm according to which, after filling out a specially designed questionnaire, it is possible to assess the risks of developing the disease at 10 and 30-year intervals and calculate the percentage [4].

Taking into account the pathogenesis of ischemic heart disease, the main approaches and drugs in the treatment scheme of GID have been developed. The main goals of treatment are: improving blood flow in the coronary arteries, reducing the load on the heart, delaying and stopping atherosclerotic processes. For this, various groups of drugs are used: anticoagulants, antiplatelet drugs, beta-blockers, calcium channel blockers, nitrates, opioids, thrombolytic drugs, angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, statins. To improve blood flow in the coronary arteries, revascularization may also be necessary: coronary artery bypass grafting or coronary artery bypass grafting [1].

**Clinical Case**

**Patient - J.Kh. 67 years old.**

**Diagnosis:** heart failure, ischemic heart disease Prescribed medications:

1. Depre Fix. Tab. Hypericum (St John's Wort)
2. Brovensin syrup 100 ml - (bromhexine + terbutaline + guaifenesin)
3. Nolpaza. Tab. 40 mg - (Pantoprazole)
4. Berodual. Aerosol. Berodual - (comb. Drug fenoterol + ipratropium bromide)
5. Magneo. Tab. 500 mg - (comb. drug potassium aspartate + magnesium aspartate)
6. Toragamma. Tab. 200 mg - (Torsemide)
7. Amprilan. Tab. 1.25 mg - (Ramipril)
8. Coraxan. Tab. 5 mg - (ivabradine)
9. Sorvasta. Tab. 15 mg - (rosuvastatin)
10. Eplerenone. Tab. 25 mg - (Eplerenone)
11. Pectrol. Tab. 40 mg - (isosorbide mononitrate) (Isosorbide mononitrate)
12. Sobicor. Tab. 5 mg - (bisoprolol)
13. Co-Plavix. Tab. 100/75 mg - (clopidogrel + acetylsalicylic acid)

**Drug Interactions**

**Serious Interaction**

**Aspirin + Ramipril**

The interaction of aspirin and ramipril is an example of pharmacodynamic antagonism. Their co-administration should be avoided and alternatives should be sought for a number of reasons: Co-administration may lead to a sharp decrease in renal function. NSAIDs may also reduce the antihypertensive effect of ACE inhibitors. The reasons for this interaction are related to the ability of NSAIDs to reduce the synthesis of renal prostaglandins with vasodilatory function. Prostaglandins are arteriodilators and play an important role in the tone of the renal arteries, normal hemodynamics and normal filtration. These effects are inhibited by NSAIDs.

**Depre Fix (Hypericum. St John's Wort) + Ivabradine**

The herbal antidepressant Depre Fix (Hypericum. St John's Wort) reduces the level of ivabradine in the blood, since it induces the liver enzyme CYP3A4 involved in its metabolism (increases its activity), as a result of which the metabolism of ivabradine is enhanced and its level in the blood is sharply reduced. It is better to avoid this interaction or use an alternative remedy. In general, ivabradine should not be taken together with other drugs that have the ability to induce CYP3A4, since its effect is sharply reduced.

**Depre Fix (Hypericum. St John's Wort) + Clopidogrel**

As already mentioned, Depre Fix (Hypericum. St John's Wort) is an antidepressant of herbal origin. It has also been proven that it is an inducer of liver enzymes CYP3A4, CYP2C19, that is, it increases their activity. Clopidogrel is a prodrug that requires metabolism to be converted into active metabolites.

The above-mentioned enzymes are involved in this metabolism. Accordingly, when taken together with Depre Fix (Hypericum. St John's Wort), the activity of liver enzymes involved in the conversion of clopidogrel into active metabolites increases, respectively, its antiplatelet effects increase and the risk of bleeding increases. It is necessary to use another alternative drug. If these medications are nevertheless prescribed in combination, then constant assessment and control of the risk of bleeding is necessary.

**Attentional Interaction**

**Bisoprolol + terbutaline**

Concomitant administration of bisoprolol with terbutaline may reduce the effects of both drugs through a mechanism of pharmacodynamic antagonism. Caution/monitoring is required.

**Ramipril + Eplerenone**

Their combination is an example of pharmacodynamic synergism. The risk of hyperkalemia is increased. Caution/monitoring is required. In general, when eplerenone is administered in combination with angiotensin-converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARBs), the risk of hyperkalemia is increased, which may even lead to serious arrhythmias. The risk is higher in patients with: renal insufficiency, diabetes, or dehydration. Therefore, periodic monitoring of blood potassium levels is important in such a combination.

**Ramipril + potassium citrate**

Ramipril increases the level of potassium citrate by reducing its excretion. Caution/monitoring is required. The risk of hyperkalemia increases, which in severe cases leads to renal failure, muscle paralysis, arrhythmias, and heart attack. The risk of hyperkalemia increases in elderly patients. Dehydration, kidney disease, diabetes, and a history of heart failure also increase the risk. Continuous monitoring of potassium in the blood and kidney function is required.

**Aspirin + Bisoprolol**

Salicylates in high doses reduce the antihypertensive effects of beta-blockers. This is associated with inhibition of prostaglandin synthesis. In low doses, aspirin does not significantly affect the antihypertensive effects of beta-blockers. However, caution/monitoring is still required.

**Aspirin + Clopidogrel**

Aspirin and clopidogrel increase each other's toxicity through a mechanism of pharmacodynamic synergism. The combination may cause bleeding, acute abdominal pain, weakness, and bloody stools. Caution/monitoring for these symptoms is required. Dose adjustment may be necessary.

**Ramipril + Torsemide**

This combination is an example of pharmacodynamic synergism. Caution/monitoring is required. The risk of acute hypotension and renal failure is increased. Combined administration may cause hypotension and hypovolemia to a greater extent than either drug alone. Therefore, monitoring of blood pressure, diuresis, electrolytes, and renal function is required when used in combination.

**Ivabradine + bisoprolol**

Increase each other's effects by the mechanism of pharmacodynamic synergism. Caution/monitoring approach is required. In general, ivabradine in combination with any bradycardia agent (e.g. beta-blockers, calcium channel blockers, digoxin) causes severe bradycardia and increases the risk of its complications. Therefore, caution is required when combining such drugs. Constant monitoring of heart rate is required, and ivabradine dose adjustment may also be necessary. Patients should be alert to symptoms of bradycardia: dizziness, lightheadedness, fatigue, hypotension, etc.

**Potassium citrate + eplerenone**

Both potassium citrate and eplerenone increase the level of potassium in the blood, therefore, the use of this combination is not recommended. The risk of hyperkalemia increases, which in severe cases leads to kidney failure, muscle paralysis, arrhythmias, heart attack. The risk of hyperkalemia increases in elderly patients, as well as dehydration, kidney disease, diabetes, and a history of heart failure.

**Potassium citrate + terbutaline**

Potassium citrate increases, while terbutaline decreases, blood potassium levels. Caution/monitoring approach required.

**Potassium citrate + torsemide**

Potassium citrate increases, while torsemide decreases, blood potassium levels. Caution/monitoring approach required.

**Terbutaline + Torsemide**

Both terbutaline and torsemide lower blood potassium levels. Caution/monitoring is required.

**Terbutaline + pseudoephedrine**

Terbutaline and pseudoephedrine also increase sympathetic (adrenergic) effects - hypertension, tachycardia, irregular heart rhythm. It may be necessary to prescribe alternative drugs or adjust the dose. If they are used in combination, caution/monitoring is required. Special attention is required in patients with cardiovascular disorders: coronary artery disease, arrhythmias, hypertrophic obstructive cardiomyopathy, hypertension. Frequent monitoring of blood pressure and pulse is required.

**Pantoprazole + clopidogrel**

Clopidogrel is a prodrug that requires the liver enzyme system, specifically CYP2C19, to be converted to active metabolites and exert its antithrombotic effect. Inhibitors of this enzyme are proton pump inhibitors PPIs - omeprazole, esomeprazole, pantoprazole. However, it has been established that of these three drugs, pantoprazole has the least effect on CYP2C19. Accordingly, it is the drug of choice among proton pump inhibitors in combination with clopidogrel. Of course, there are still small risks, and the risks increase with prolonged and frequent use of pantoprazole, so caution/monitoring is still necessary.

**Depre Fix (Hypericum. St John's Wort) + Eplerenone**

Depre Fix (Hypericum. St John's Wort) increases the activity of the CYP3A4 enzyme. This enzyme is involved in the metabolism of eplerenone. Accordingly, its metabolism may be increased and

the pharmacological effect may be reduced. Caution/monitoring is required.

**Bisoprolol + potassium citrate**
Bisoprolol and potassium citrate increase blood potassium levels. Therefore, caution/monitoring of blood electrolyte levels is required.

**Bisoprolol + torsemide**
Although beta-blockers and diuretics are often used in combination in the clinic, they may increase the risk of hyperglycemia and hypertriglyceridemia in some patients, especially in diabetics. Also, bisoprolol increases and torsemide decreases blood potassium levels. Caution/monitoring is required - control of blood pressure and blood potassium, glucose.

**Aspirin + potassium citrate**
Aspirin and potassium citrate both increase blood potassium levels. Therefore, caution/monitoring of blood electrolyte levels is necessary. Also, the alkalinization of urine caused by potassium citrate may lead to decreased reabsorption of acidic aspirin in the renal tubules, increasing its excretion and reducing its amount in the blood, and therefore its effect. This method, known as "ion trapping", can be used to reduce salicylate toxicity.

**Aspirin + Terbutaline**
Aspirin increases and terbutaline decreases serum potassium levels. Caution/monitoring of blood electrolyte levels is required. Caution/monitoring is required.

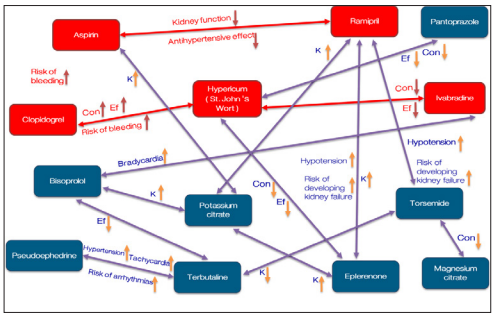
**Aspirin + Torsemide**
Aspirin increases and torsemide decreases serum potassium levels. Caution/monitoring of blood electrolyte levels is required. Caution/monitoring is required.

**Depre Fix (Hypericum. St John's Wort) + Pantoprazole**
Depre Fix (Hypericum. St John's Wort) activates the activity of liver enzymes involved in the metabolism of pantoprazole - CYP2C19, CYP3A4. Pantoprazole metabolism is enhanced, its level in the blood decreases, and consequently its pharmacological effects decrease. Caution/monitoring is required.

**Aspirin + eplerenone**
Aspirin reduces the effects of eplerenone by a mechanism of pharmacodynamic antagonism. Caution/monitoring required.

**Torsemide + magnesium citrate**
Torsemide reduces magnesium citrate levels by increasing its renal clearance. Caution/monitoring required.

**Clopidogrel + torsemide**
Clopidogrel increases blood levels of torsemide by decreasing its metabolism. Caution/monitoring is required.




**Recommendations**
In consultation with a cardiologist, we discussed the above-mentioned scheme. We identified issues of concern and recommendations.

As we can see, the herbal antidepressant Hypericum (St. John's Wort) has a serious interaction, on the one hand, with ivabradine and reduces its effects, and on the other hand, it interacts with clopidogrel, increasing its concentration and toxicity, and the risk of bleeding. Accordingly, it is recommended to discontinue the use of this drug and replace it with another antidepressant.

The combination of ivabradine and bisoprolol has been shown to cause severe bradycardia and arrhythmias, and it is recommended that the need for ivabradine be assessed. If heart rate is well controlled with bisoprolol, ivabradine may be discontinued, thereby reducing the risk of severe bradycardia and arrhythmias.

The need for potassium and magnesium citrate may need to be reconsidered after checking blood electrolyte levels.

The patient is prescribed Berodual for bronchodilator therapy, the fenoterol in which causes tachycardia and arrhythmias. If bronchodilator therapy is necessary for the patient, it is recommended to use an alternative agent with fewer cardiovascular complications, such as ipratropium alone (a muscarinic antagonist that does not significantly affect heart rate). This will prevent fenoterol-induced tachycardia.

It is also recommended to assess the need for co-Plavix as a combination drug, since aspirin and clopidogrel in combination increase the risk of bleeding. If the patient does not currently have any coronary artery disease, or has recently undergone angiosurgical intervention, the duration of combined antiplatelet therapy should be reduced or only one, for example, clopidogrel, should be used.

As for the interaction of ramipril and aspirin, aspirin in low doses is generally an effective, safe antiplatelet agent with minimal side effects. In our patient, the toxicity of this combination may be increased because the patient is also taking eplerenone, torsemide, and potassium citrate, which increase the level of potassium in the blood. Regular monitoring of renal function and electrolytes will be necessary. The above should be checked 1-2 weeks after the start of administration. No other NSAIDs should be added to the background of ramipril and aspirin.

It is also possible that the dose of torsemide may be reduced if the patient is euvolemic and there are no signs of fluid overload. The patient has been prescribed 200 mg of torsemide, which

is considered a high dose, which may cause hypovolemia, hypotension, and electrolyte imbalance. It is important to monitor this.

The patient has been prescribed rosuvastatin at a daily dose of 15 mg. It is advisable to reduce the dose to 10 mg, since rosuvastatin at doses greater than 10 mg in the elderly and patients with polypharmacy causes an increased risk of myopathy, rhabdomyolysis, and elevated liver enzymes.
After reviewing and reconciling the above information, appropriate changes were made to the designation.

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