ADITUM
Enrich your Research

Journal of Clinical Cardiology Interventions

Research Article

First Results of The Use of Inclisiran in Patients with Dyslipidemia in Real Clinical Practice

E. A. Polyakova, Yu. Sh. Khalimov, S. V. Lapin, T. M. Bakher*, A. V. Mazing, A.K. Musonova, V.D. Nazarov
[1]The Pavlov First Saint Petersburg State Medical University, Saint Petersburg.

Article Info

Received: August 05, 2025
Accepted: August 20, 2025
Published: August 30, 2025

***Corresponding author:** Timur Bakher, The Pavlov First Saint Petersburg State Medical University, Saint Petersburg.

Citation: E. A. Polyakova, Yu. Sh. Khalimov, S. V. Lapin, T. M. Bakher, A. V. Mazing, A.K. Musonova, V.D. Nazarov., (2025) "First Results Of The Use Of Inclisiran In Patients With Dyslipidemia In Real Clinical Practice." journal of clinical cardiology interventions, 5(2). DOI: 10.61148/2836-077X/JCCI/054.

Abstract:

Purpose of the study: to analyze the use of inclisiran in real clinical practice for primary and secondary prevention of atherosclerotic cardiovascular diseases in patients with dyslipidemia.

Methods: a retrospective analysis of the treatment data was conducted for the first 20 patients who received two doses of inclisiran. The review covered medical history, physical examination data, additional lipid-lowering therapy, genetic testing, lipid profile data, lipoprotein (a) level, standard biochemical tests at baseline, after 2 and 4 months from the start of inclisiran therapy.

Results: after starting treatment with inclisiran, average total cholesterol (TC) decreased: after 2 months by 41.0% from 5.1 (4.13; 6.47) to 3.01 (2.77; 4.27) mmol/L, after 4 months by 44.7% of the initial level to 2.82 (2.46; 3.44) mmol/L (compared to initial levels $p < 0.001$). The significant decrease of low-density lipoprotein cholesterol (LDL) level was also archived: after 2 months by 47.1% from 3.08 (2.40; 4.73) to 1.63 (1.10; 2.60) mmol/L; after 4 months by 54.5% from initial levels to 1.4 (0.87, 1.90) mmol/L L (compared to initial levels $p < 0.001$). The average triglyceride levels by the 4th month of observation decreased by 27.9%. And a decrease in LP (a) levels by 27.8% was registered after 2 months of observation.

Conclusion: the use of inclisiran in real clinical practice is accompanied by a significant decrease in serum total cholesterol, LDL cholesterol, TG and LP (a) with good tolerability and high efficiency.

Keywords: cardiovascular diseases

Introduction

The main cause of mortality globally is atherosclerotic cardiovascular diseases (CVD) [1–3]. Elevated levels of atherogenic cholesterol fractions are the key risk factor for these diseases, in particular low-density lipoproteins (LDL), non-high-density lipoproteins (non-HDL), triglycerides (TG), as well as lipoprotein (a) (LP(a)) [4]. A proven therapeutic target for primary and secondary prevention of atherosclerotic CVD is LDL cholesterol, the increase in the level of which is lowered by prescribing first-line treatment drugs, i. e. statins, with proven effectiveness and sufficient safety [1]. Over the past 20 years, the target LDL cholesterol levels have been repeatedly revised, setting increasingly lower values, in order to prevent cardiovascular complications [3, 4]. The problem of achieving these values has arisen; therefore, combination lipid-lowering therapy is given special attention in the clinical recommendations to correct lipid metabolism

disorders [1, 5]. The search for strategies to reduce LDL-C concentrations led to the development of inclisiran, a first-in-class small interfering RNA that prevents the translation of proprotein convertase subtilisin/kexin type 9 (PCSK9) in hepatocytes [6]. This increases the recirculation and number of LDL cholesterol receptors on the hepatocyte membrane, which enhances the uptake of LDL cholesterol, reducing their blood concentration [6]. The first phase III double-blind, randomized, controlled trials of inclisiran (ORION-9, ORION-10, and ORION-11) showed a significant reduction in LDL cholesterol levels by approximately 50% compared with placebo. [7–9]. However, a limited number of studies [10, 11] represent the actual experience of inclisiran use. Therefore, we analyzed data from our own clinical experience with this medication.

Purpose of the study:
To analyze the use of inclisiran in real clinical practice for primary and secondary prevention of atherosclerotic CVD in patients with dyslipidemia.

Materials and methods are included as supplementary materials.

Study results
The study included 20 patients who had received the first and second doses of inclisiran. The age of the subjects ranged from 36 to 74 years (average age was 61 (51; 62) years), of which 13 (59%) were men. The main clinical diagnoses and baseline characteristics of patients primary and secondary CVD prevention are presented — in Table 1.

Table 1: Main characteristics of patients primary and secondary CVD prevention.

Sex	Age	Diagnosis	LDL level before inclisiran, mmol/L	Target LDL level, mmol/L	Reason for not achieving the target LDL level	Therapy at the time of inclusion (at least 4 weeks)	FH genotyping results	LDL level 4 months after the start of inclisiran, mmol/L (% compared to baseline)
Primary prevention of CVD in patients without atherosclerotic CVD group (n = 8)								
F	39	Type 1 diabetes, CKD S3a	6.6	< 1.4	Intolerance of max. statin dose	A40+E	No mutations identified	1.9 (↓71%)
F	66	BCA atherosclerosis > 50%, endarterectomy. Type 2 DM	2.62	< 1.4	Lack of efficacy of high-intensity combination therapy	A80+E	No mutations identified	0.84 (↓68%)
F	72	BCA atherosclerosis > 50%, type 2 diabetes, CKD G3b, chronic myeloid leukemia	6.38	< 1.4	Statin intolerance	E	No mutations identified	2.9 (↓55%)
M	57	BCA atherosclerosis > 50%, Hyperlipoproteinemia (a)	6.98	< 1.4	Statin intolerance	E	No mutations identified	2.7 (↓55%)
M	69	BCA atherosclerosis > 50%, type 2 diabetes, CKD G3a	3.63	< 1.4	Intolerance of max. statin dose	A20+E	No mutations identified	1.4 (↓61%)
F	68	BCA atherosclerosis up to 45%, type 2 diabetes, CKD G3b	4.92	< 1.4	Lack of efficacy of high-intensity combination therapy	R40+E	No mutations identified	1.8 (↓63%)
M	36	Heterozygous FH, BCA atherosclerosis 25%	3.08	< 1.4	Intolerance of max. statin dose	A20+E	Pathogenic variant in exon 9 of *LDLR* gene: heterozygous mutation p.V429M (rs28942078)	1.9 (↓38%)
F	71	BCA atherosclerosis > 35%,	4.54	< 1.8	Intolerance of max. statin dose	R20+E	No mutations identified	2.41 (↓47%)
Secondary cardiovascular complications prevention in patients with atherosclerotic CVD group (n = 14)								
F	54	CVA, polyvascular disease, CKD G3b. Hyperlipoproteinemia (a)	2.37	< 1.4	Lack of efficacy of high-intensity combination therapy	R40+E	No mutations identified	0.77 (↓32%)

Sex	Age	Diagnosis	LDL level before inclisiran, mmol/L	Target LDL level, mmol/L	Reason for not achieving the target LDL level	Therapy at the time of inclusion (at least 4 weeks)	FH genotyping results	LDL level 4 months after the start of inclisiran, mmol/L (% compared to baseline)
M	52	IHD: MI, heart attack, PCI + stenting. Atherosclerosis of the aorta and its branches, acute aortic dissection. 2 acute events over the past 2 years. CKD G3b	1.9	< 1.0	Lack of efficacy of high-intensity combination therapy	A80+E	No mutations identified	1.07 (↓44%)
M	59	IHD: MI, heart attack, PCI + stenting. 2 acute events over the past 2 years	2.0	< 1.0	Intolerance of max. statin dose	A20+E	No mutations identified	0.65 (↓67%)
F	59	IHD: exertional angina, CKD G3b	2.4	< 1.4	Lack of efficacy of high-intensity combination therapy	A80+E	No mutations identified	0.61 (↓75%)
F	74	IHD: exertional angina. Polyvascular disease	2.11	< 1.4	Intolerance of max. statin dose	R20+E	No mutations identified	0.9 (↓56%)
F	62	Heterozygous FH. IBS: MI Atherosclerosis obliterans of the arteries of the upper and lower extremities. Stenosing atherosclerosis of BCA. CKD G3b. Two acute events over the past 2 years	5.04	< 1.0	Lack of efficacy of high-intensity combination therapy	R40+E	Pathogenic mutation in the *LDLR* gene: heterozygous variant p.Glu228Ter (rs121908029)	1.2 (↓76%)
M	51	Heterozygous FH IHD: MI, heart attack, PCI + stenting. Type 2 DM	6.4	< 1.4	Lack of efficacy of high-intensity combination therapy	R40+E	Pathogenic variant in exon 26 in the *APOB100* gene: heterozygous variant p.R3527Q (rs5742904)	2.05 ↓68%
M	68	IHD: MI, heart attack, PCI + stenting. Stenosing atherosclerosis of BCA, endarterectomy. CVA. Type 2 DM Hyperlipoproteinemia (a)	2.4	< 1.0	Intolerance of max. statin dose	A40+E	No mutations identified	1.2 (↓50%)
M	66	IHD: heart attack, PCI + stenting. BCA atherosclerosis 45%.	2.55	< 1.4	Statin intolerance	E	No mutations identified	1.9 (↓26%)
M	70	IHD: silent myocardial ischemia. Type 2 diabetes, CKD G3b.	4.32	< 1.4	Intolerance of max. statin dose	R10+E	No mutations identified	1.61 (↓26%)
M	61	IHD: stable angina. CVA.	2.95	< 1.4	Lack of efficacy of high-intensity combination therapy	A80+E	No mutations identified	2.06 (↓63%)
M	61	CVA. Arteritis obliterans of the arteries of the lower	3.08	< 1.4	Intolerance of max. statin dose	A20+E	No mutations identified	1.27 (↓58%) ONMA

Sex	Age	Diagnosis	LDL level before inclisiran, mmol/L	Target LDL level, mmol/L	Reason for not achieving the target LDL level	Therapy at the time of inclusion (at least 4 weeks)	FH genotyping results	LDL level 4 months after the start of inclisiran, mmol/L (% compared to baseline)
		extremities.						

Note. BCA = brachiocephalic artery, CKD = chronic kidney disease, A = atorvastatin, E = ezetimib, MI = myocardial infarction, ACS = acute coronary syndrome, IHD = Ischemic heart disease, PCI (percutaneous coronary intervention) + stenting.

When analyzing the initial characteristics, it was found that 3 (13.6%) patients had complete intolerance to statins, which was manifested by rhabdomyolysis in 2 patients and a clinically significant increase in transaminase levels in 1 patient. 19 (86.4%) patients received statin therapy, of which 9 (45%) showed intolerance to maximum doses of statin in the form of a significant increase in transaminase levels. Women were more likely to receive high-intensity statin therapy than men: 8 (73%) versus 3 (27%) ($p < 0.001$). In this study, only men received moderate-dose statin therapy due to intolerance to high-intensity doses (8 (100%) patients).

20 (100%) patients received ezetimibe therapy as part of both combination lipid-lowering therapy and as a monotherapy (3 patients with statin intolerance: 2 men and 1 woman).

Throughout the entire observation period, all patients adhered to regular therapy with statins and/or ezetimibe, which ensures the validity of the inclisiran therapy effectiveness assessment results.

In the primary CVD prevention group, 8 patients were observed, of whom 7 had a very high cardiovascular risk (CVR) and 1 had a moderate CVR, caused by such conditions as type 1 diabetes, atherosclerotic lesions of the BCA, and HeFH (see Table 1).

In the secondary CVD prevention group, 12 patients were observed, of whom 8 had very high CVR and 4 had extreme CVR, caused by conditions such as coronary heart disease (CHD), acute cerebrovascular accident (CVA), transient ischemic attack (TIA), type 2 diabetes, atherosclerotic lesions of the BCA and arteries of the lower extremities, HeFH (see Table 1).

To assess the lipid profile and serum TG content, a standard photometric method was used, and the data thus obtained were compared with the data obtained by electrophoresis; no significant differences in the results between these two assessment methods were detected, probably due to the absence of patients with severe hypertriglyceridemia. The tables contain data obtained by the standard photometric method.

In the examined group of patients, the initial lipid profile indicators did not correspond to the target values for patients with high and very high cardiovascular risk. However, 2 and 4 months after the start of inclisiran therapy, a significant and persistent decrease in lipid profile parameters was observed (Fig. 1, Table 2), which confirms the known fact that inclisiran reduces LDL concentrations by at least 50% according to the ORION group studies [9].

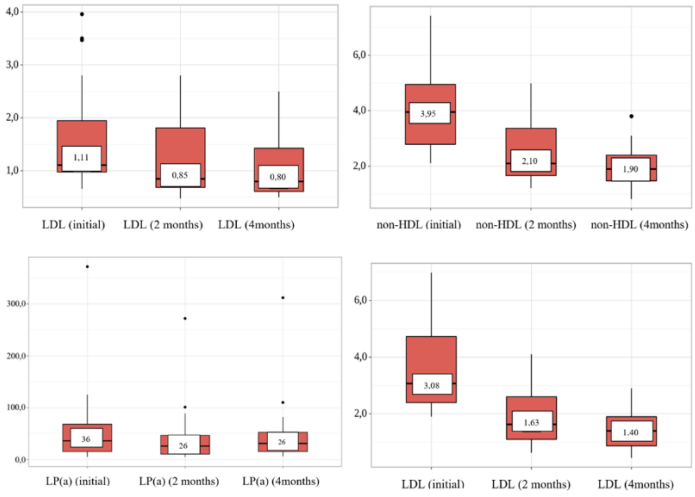

Figure 1: Changes of non-HDL, TG, LP(a) and LDL levels over time in the patients' blood serum during inclisiran therapy. $p < 0.01$ between all subgroups

During inclisiran therapy, a significant decrease was achieved not only in LDL cholesterol, but also in non-HDL cholesterol and TG (Fig. 1).

Table 2. Dynamics of lipid spectrum, TG and LP(a) serum lrvels in patients with dyslipidemia after 2 and 4 months, intensification of treatment with inclisiran (n = 22), Me, (Q_1; Q_3)

Parameter	Initially	After 2 months	After 4 months	probability value (p)
TC, mmol/L	5.10 (4.13; 6.47)	3.01 (2.77; 4.27)	2.82 (2.46; 3.44)	< 0.001 $p_{1-2} = 0.003$ $p_{1-3} < 0.001$ $p_{2-3} = 0.020$
LDL, mmol/L	3.08 (2.40; 4.73)	1.63 (1.10; 2.60)	1.40 (0.87; 1.90)	< 0.001 $p_{1-2} = 0.003$ $p_{1-3} < 0.001$ $p_{2-3} = 0.020$
non-HDL,	3.95 (2.7; 4.95)	2.10 (1.66; 3.36)	1.90 (1.47; 2.40)	< 0.001

Parameter	Initially	After 2 months	After 4 months	probability value (p)
mmol/L				$p_{1-2} = 0.003$ $p_{1-3} < 0.001$ $p_{2-3} = 0.020$
HDL, mmol/L	0.99 (0.86; 1.27)	1.04 (0.99; 1.16)	1.00 (0.90; 1.01)	0.093
TG, mmol/L	1.11 (0.97; 1.94)	0.85 (0.69; 1.81)	0.80 (0.61; 1.43)	< 0.001 $p_{1-2} = 0.040$ $p_{1-2} < 0.001$ $p_{1-2} = 0.040$
LP(a), mg/dL	36.0 (16.0; 68.0)	26.0 (11.0; 46.0)	31.0 (15.0; 53.0)	< 0.001 $p_{1-2} < 0.001$ $p_{1-3} = 0.038$ $p_{2-3} = 0.007$

Individual responses to inclisiran treatment are presented in Table 1, demonstrating a wide LDL-C reduction range, from 26% to 76% of the baseline. The median HDL cholesterol did not change significantly during the observation period (Table 2), and the median TG concentration decreased from 1.11 to 0.61 mmol/L ($p < 0.001$).

Differences in the degree of reduction in LDL cholesterol levels by the 4th month of treatment with inclisiran were examined based on the sex of patients. Thus, in women, the level of LDL-C decreased by 60.3%, and in men — by 52.4% ($p = 0.004$). This finding may be explained by the fact that in this sample of patients, women received high-intensity statin therapy significantly more often than men, as noted above.

The reduction in LDL cholesterol levels after including inclisiran in therapy was significant but depended on concomitant therapy with statins and ezetimibe. Thus, by the 4th month of observation, the greatest reduction in LDL cholesterol levels was achieved in patients receiving high-intensity or maximum doses of statins in combination with ezetimibe 10 mg (reduction by 60.3%); slightly less pronounced, but also a significant reduction of LDL cholesterol was achieved in those patients who initially received medium doses of statins in combination with ezetimibe (reduction by 53.1%); the lowest effect was achieved by those who initially received only ezetimibe due to statin intolerance (reduction by 45.3%). The differences between the groups of patients with three different lipid-lowering therapy strategies were significant at baseline ($p < 0.05$), and the differences in the reduction in LDL cholesterol levels in each group compared to baseline were also significant ($p < 0.05$).

By the 4th month of inclisiran treatment, 15 (75%) patients achieved a reduction in LDL cholesterol levels of 50% or more compared to baseline, and 9 patients (45%) achieved LDL cholesterol levels < 1.4 mmol/L.

Analysis of standard biochemical parameters of liver function assessment (alanine aminotransferase, aspartate aminotransferase, lactate dehydrogenase, alkaline phosphatase) and kidney function (creatinine, urea, glomerular filtration rate (GFR)) and complete blood count did not show significant differences during the observation period.

An adverse event (AE) during the entire observation period was recorded in 1 (5%) of 20 patients. This was a moderate skin hyperemia reaction at the injection site after the first administration of the drug; when it was re-administered in the 3rd month, no such reaction was observed in the same patient. This AE resolved spontaneously within 2 hours.

Special clinical observation groups

Among the special clinically observed cases, the results of inclisiran therapy in patients with heterozygous FH, as well as its effect on the level of LP(a) in the blood serum, should be noted.

Patients with heterozygous FH

The effectiveness of inclisiran therapy in patients with HeFH compared to those examined without proven mutations in lipid metabolism genes was comparable and did not differ significantly, leading to a decrease in LDL cholesterol levels by 60.6% and 58.8%, respectively, by the 4th month of treatment ($p > 0.05$).

Effect of inclisiran on serum LP(a) levels

The LP(a) content was analyzed in all patients initially and over time; in all patients it decreased by 27.8% ($p < 0.001$) by the 2nd month of inclisiran therapy, and when its content was studied 1 month after the second injection (4th month overall) it was slightly less notable, yet still statistically significant — 13.9% ($p = 0.038$) (Fig. 1, 2).

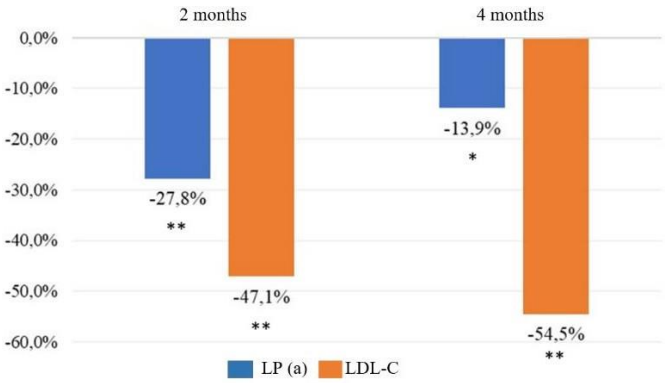

Figure 2. The degree of reduction in the levels of LP(a) and LDL-C in the blood serum of patients on the 2nd and 4th month of inclisiran therapy compared to baseline levels

*p = 0.038; **p < 0.001

Discussion

Insufficient clinical efficacy and adherence to treatment, as well as frequent dose-dependent side effects of statins, represent the main limitations in the treatment of patients with atherogenic

dyslipidemia in real clinical practice [5]. About 50% of patients discontinue statin therapy less than 1 year after initiation [15]. Other lipid-lowering drugs approved for use in Russia, such as ezetimibe and omega-3-polyunsaturated fatty acids, are an addition to the main statin therapy, but in some cases, they are used as the sole drug products. Drugs for the treatment of atherogenic dyslipidemias, such as bempedoic acid, bile acid sequestrants, microsomal triglyceride transfer protein inhibitors (lomitapide), antisense oligonucleotides to the ApoB-100 protein (mipomersen) are not authorized and available in all countries [2, 4, 5]. The emergence of monoclonal antibodies to PCSK9 has expanded the treatment options for such patients. Despite their effectiveness, drugs in this group are not widely available due to their high cost, storage and transportation specifics, and strict acceptance criteria for their use in many countries [16]. Authorization of inclisiran provides an opportunity for a wider choice of drugs for the management of dyslipidemia — not only for those patients who had intolerance or restrictions to the use of humanized monoclonal antibodies to PCSK9, but also for primary use as part of combination therapy with statins and/or ezetimibe, as well as in cases of intolerance to statins in patients with cardiovascular diseases.

The results of our study allowed us to evaluate the experience of using inclisiran in patients with dyslipidemia for the purpose of primary and secondary prevention of CVD in real clinical practice, prescribed on the basis of the national clinical guidelines. The duration of the observation period for the cohort of patients was limited to the use of the first two doses of inclisiran in accordance with the instructions for the drug, according to which patients will continue further treatment once every 6 months. It was found that a double subcutaneous injection of inclisiran reduces the level of LDL cholesterol by 54.5% by the 4th month of treatment [9, 11]. The reduction in LDL-C levels by more than 50% of the baseline level did not depend on gender but was more notable in women than in men, which in our observation may be explained by better tolerability and a higher frequency of prescribing high-intensity statin therapy in women than in men due to the latter being intolerant to high statin doses. The overall results and patterns obtained in this study are consistent with data from the ORION-10 and ORION-11 studies, which showed a reduction in mean LDL cholesterol levels by approximately 50% [8, 9].

Furthermore, in our study, a significantly more pronounced reduction in LDL cholesterol levels was achieved by patients who were prescribed inclisiran in addition to high-intensity statin therapy, which emphasizes the pathogenetic importance of combining two strategies to ensure biological synergy: a decrease in the concentration of free PCSK9, which increases the concentration of LDL receptors, with therapy increasing the transcription of the LDL receptor and accompanied by the inhibition of the enzymatic synthesis of endogenous cholesterol in the liver, with the overall effect being an increase in the clearance of circulating LDL cholesterol [5, 6]. Previous studies have shown that combining PCSK9 inhibitors with high-intensity statin therapy can reduce LDL cholesterol levels by up to 75%–80%[1] [9]. Therefore, it is necessary to emphasize the high clinical importance of maintaining adherence to treatment and patients continuously taking initial lipid-lowering therapy. In our study, all patients remained highly compliant throughout the study period, however, 36.4% of the examined patients had intolerance only to high doses of statins, and 13.6% had absolute intolerance, which is consistent with earlier studies [17]. For many patients, biannual inclisiran injection is a more convenient and less burdensome treatment option that may promote adherence to treatment and overall lead to greater reductions in LDL cholesterol levels in the future [6]. The approval of inclisiran for use in ambulatory care settings may collectively lead to an increase in the number of patients achieving therapeutic goals.

By the 4th month of treatment with inclisiran, a decrease in the levels of total cholesterol, non-HDL cholesterol, TG and LP(a) was achieved. This trend was reported in the analysis of secondary endpoints throughout the follow-up period in the ORION-10 and ORION-11 studies [8, 9]. The similarity of these results indicates their reproducibility in real clinical settings.

An adverse event, hyperemia at the injection site, the likelihood of which was associated with the administration of inclisiran, occurred in 1 patient, and neither met the severity criteria nor required discontinuation of the drug. In the ORION-3, -9, -10, and -11 studies, the incidence of injection site reactions ranged from 3% to 17% [7–9, 18]. In a study by U. Makhmudova et al. [19], who combined the results of 14 lipid clinics in Germany as part of The German Inclisiran Network, a moderate reaction at the injection site was observed in 3% of cases, and no serious AEs were recorded. It is possible that the low incidence of this phenomenon is due to the fact that patients do not report all mild reactions at the injection site. Not a single patient in the presented study discontinued treatment after 2 doses of inclisiran, which contributes to the high treatment efficacy. Large randomized clinical trials and individual observational studies [7, 8, 18] confirmed the favorable safety profile of inclisiran.

It should be noted that inclisiran is highly effective in patients with heterozygous FH, which is consistent with the results of the ORION-9 study [7].

However, the conducted study has several limitations. First, the results represent the lipid clinic experience of only one site. Secondly, the presented sample size is small, so the frequency of side effects may not correspond to previously known ones. Also, the small sample size does not allow the results to be transferred to the population level. In addition, the short observation period limits the assessment of the medicinal product's effectiveness and does not allow us to wait for the therapeutic peak of the effect of inclisiran. Longer follow-up and studies are needed to evaluate the long-term effectiveness and sustainability of the beneficial effects of inclisiran.

Conclusions

1. Double administration of inclisiran in addition to standard combination lipid-lowering therapy in patients with moderate, high, very high, and extreme cardiovascular risk is effective, safe and allows reducing LDL cholesterol levels by more than 50% of initial values, with a high rate of achieving target levels as a secondary, and primary prevention of cardiovascular complications.
2. Double administration of inclisiran in patients with proven absolute intolerance to statins, receiving ezetimibe monotherapy showed high efficacy and safety: the average reduction in LDL cholesterol levels was recorded at 45.3%.

3. Inclisiran is effective and safe for patients with heterozygous FH, which is comparable to patients without this disorder.
4. Additional clinically significant effects of inclisiran were noted, such as a decrease in serum LP(a) and TG concentrations.

References

1. Visseren F.L.J., Mach F., Smulders Y.M. et al. 2021 ESC Guidelines on cardiovascular disease prevention in clinical practice. Eur Heart J. 2021;42(34):3227–3337. DOI: 10.1093/eurheartj/ehab484.
2. Tsao C.W., Aday A.W., Almarzooq Z.I. et al. Heart Disease and Stroke Statistics-2022 Update: A Report from the American Heart Association. Circulation. 2022;145(8): e153–e639. DOI: 10.1161/CIR.0000000000001052.
3. Бойцов С.А., Погосова Н.В., Аншелес А.А. и др. Кардиоваскулярная профилактика 2022. Российские национальные рекомендации. Российский кардиологический журнал. 2023;28(5):5452. [Boytsov S.A., Pogosova N.V., Ansheles A.A. et al. Cardiovascular prevention 2022. Russian national guidelines. Russian Journal of Cardiology. 2023;28(5):5452 (in Russ.)]. DOI: 10.15829/1560-4071-2023-5452.
4. Martin S.S., Sperling L.S., Blaha M.J. et al. Clinician-patient risk discussion for atherosclerotic cardiovascular disease prevention: importance to implementation of the 2013 ACC/AHA Guidelines. J Am Coll Cardiol. 2015;65(13):1361–1368. DOI: 10.1016/j.jacc.2015.01.043.
5. Mach F., Baigent C., Catapano A.L. et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. Eur Heart J. 2020;41(1):111–188. DOI: 10.1093/eurheartj/ehz455.
6. Frampton J.E. Inclisiran: A Review in Hypercholesterolemia. Am J Cardiovasc Drugs. 2023;23(2):219–230. DOI: 10.1007/s40256-023-00568-7.
7. Raal F.J., Kallend D., Ray K.K. et al. Inclisiran for the Treatment of Heterozygous Familial Hypercholesterolemia. N Engl J Med. 2020;382(16):1520–1530. DOI: 10.1056/NEJMoa1913805.
8. Ray K.K., Wright R.S., Kallend D. et al. Two Phase 3 Trials of Inclisiran in Patients with Elevated LDL Cholesterol. N Engl J Med. 2020;382(16):1507–1519. DOI: 10.1056/NEJMoa1912387.
9. Wright R.S., Ray K.K., Raal F.J. et al. Pooled Patient-Level Analysis of Inclisiran Trials in Patients With Familial Hypercholesterolemia or Atherosclerosis. J Am Coll Cardiol. 2021;77(9):1182–1193. DOI: 10.1016/j.jacc.2020.12.058.
10. Черепянский М.С., Пономарева Г.М., Скиба Я.Б. и др. Первый опыт применения препарата инклисиран у пациентов с острым ишемическим инсультом. Кардиология. 2023;63(10):39–46. [Cherepianskii M.S., Ponomareva G.M., Skiba I.B. et al. Inclisiran in patients with acute ischemic stroke: first data. Kardiologiia. 2023;63(10):39–46 (in Russ.)]. DOI: 10.18087/cardio.2023.10.n2560.
11. Banerjee Y., Pantea Stoian A., Cicero A.F.G. et al. Inclisiran: a small interfering RNA strategy targeting PCSK9 to treat hypercholesterolemia. Expert Opin Drug Saf. 2022;21(1):9–20. DOI: 10.1080/14740338.2022.1988568.
12. Интенсивная терапия: национальное руководство. В двух томах. Под ред. Б.Р. Гельфанда, А.И. Салтанова. М.: ГЭОТАР-Медиа; 2011. [Intensive care: national guidelines: in 2 volumes. B.R. Gelfand, A.I. Saltanov, eds. M.: GEOTAR-Media; 2011 (in Russ.)].
13. Fredrickson D.S., Lees R.S. A system for phenotyping hyperlipoproteinemia. Circulation. 1965;31:321–327. DOI: 10.1161/01.cir.31.3.321.
14. Futema M., Bourbon M., Williams M., Humphries S.E. Clinical utility of the polygenic LDL-C SNP score in familial hypercholesterolemia. Atherosclerosis. 2018;277:457–463. DOI: 10.1016/j.atherosclerosis.2018.06.006.
15. Samuel Finnikin, Brian Willis, Rani Khatib, Tim Evans and Tom Marshall. Cardiovascular risk estimation and statin adherence: an historical cohort study protocol. BJGP open 3 12 2024; BJGPO.2024.0258. DOI: 10.3399/BJGPO.2024.0258
16. Schmidt A.F., Carter J.L., Pearce L.S. et al. PCSK9 monoclonal antibodies for the primary and secondary prevention of cardiovascular disease. Cochrane Database Syst Rev. 2020;10(10):CD011748. DOI: 10.1002/14651858.CD011748.
17. Toorani M, Alvi A (February 09, 2024) Recurrence of Statin-Induced Necrotizing Myopathy: A Learning Point. Cureus 16(2): e53945. doi:10.7759/cureus.53945
18. Ray K.K., Troquay R.P.T., Visseren F.L.J. et al. Long-term efficacy and safety of inclisiran in patients with high cardiovascular risk and elevated LDL cholesterol (ORION-3): results from the 4-year open-label extension of the ORION-1 trial. Lancet Diabetes Endocrinol. 2023;11(2):109–119. DOI: 10.1016/S2213-8587(22)00353-9.
19. Makhmudova U., Schatz U., Perakakis N. et al. High interindividual variability in LDL-cholesterol reductions after inclisiran administration in a real-world multicenter setting in Germany. Clin Res Cardiol. 2023;112(11):1639–1649. DOI: 10.1007/s00392-023-02247-8.
20. Costanzo M.R., Dipchand A., Starling R. et al. The International Society of Heart and Lung Transplantation Guidelines for the care of heart transplant recipients. J Heart Lung Transplant. 2010;29(8):914–956. DOI: 10.1016/j.healun.2010.05.034.
21. Chapa J.J., McCollum J.C., Bisono J.Q. et al. PCSK9 Inhibition in Patients After Heart Transplantation: a Retrospective Review and Literature Analysis. Curr Heart Fail Rep. 2023;20(3):168–178. DOI: 10.1007/s11897-023-00604-2.
22. Jennings D.L., Sultan L., Mingov J. et al. PCSK9 inhibitors safely and effectively lower LDL after heart transplantation: a systematic review and meta-analysis. Heart Fail Rev. 2023;28(1):149–156. DOI: 10.1007/s10741-022-10255-5.
23. Wright R.S., Collins M.G., Stoekenbroek R.M. et al. Effects of Renal Impairment on the Pharmacokinetics, Efficacy, and Safety of Inclisiran: An Analysis of the ORION-7 and ORION-1 Studies. Mayo Clin Proc. 2020;95(1):77–89. DOI: 10.1016/j.mayocp.2019.08.021. Visseren F.L.J., Mach F., Smulders Y.M. et al. 2021 ESC Guidelines on cardiovascular disease prevention in clinical practice. Eur Heart J. 2021;42(34):3227–3337. DOI: 10.1093/eurheartj/ehab484.