DOWNLOAD PDF LIPID-LOWERING THERAPY AND THE INTERVENTIONAL CARDIOLOGIST

Chapter 1 : Advanced Lipid Testing | Atherosclerotic Disease | HeartPlace

These findings suggest that lipid-lowering therapy can improve coronary arterial tone, vasomotor response to dynamic exercise, and, potentially, stabilize the coronary atherosclerotic plaque in patients late after PTCA, 21The final explanation for the differences in the angiographic and clinical benefit with lipid-lowering therapy is that.

Diaconu , FESC Peripheral artery disease PAD is a manifestation of systemic atherosclerosis and is associated with high mortality due to cardiovascular events. As lipid-lowering therapy has proven its efficiency in the treatment of patients with PAD, current European guidelines recommend statin treatment for all patients with PAD. Statins are beneficial in preventing cardiovascular events in these patients and, due to their pleiotropic effects, can also increase the functional capacity and lower the risk of adverse limb outcome. Recent data indicate a major paradigm shift: In this paper, we discuss lipid management strategies in patients with PAD. Aortic Disease, Peripheral Vascular Disease, Stroke Introduction Peripheral artery disease PAD is a manifestation of systemic atherosclerosis and is therefore associated with an increased cardiovascular risk [1]. Patients with PAD often have simultaneous coronary artery and cerebrovascular disease, with high cardiovascular morbidity and mortality. Although coronary artery disease and peripheral artery disease share the same risk factors, there are considerably fewer studies concerning risk factor modification in peripheral artery disease than in coronary artery disease. However, risk factor modification is of the outmost importance if we consider the fact that the mortality risk is also increased in patients with PAD without co-existing coronary artery disease and in asymptomatic patients with PAD diagnosed through routine screening [2,3]. These observations should raise awareness about the high prevalence of PAD, the large number of undiagnosed cases of PAD and its importance as an indicator of systemic atherosclerosis. Therefore, routine ankle-brachial index ABI measurements should be used in order to identify patients with high cardiovascular risk and risk factor reduction should be implemented early after the diagnosis has been established. Another major concern regarding patients with PAD is impaired quality of life due to symptoms of claudication, rest pain or risk of limb loss. Although the symptoms in patients with PAD worsen in time, with a decline of functional capacity, severe peripheral complications in these patients are relatively infrequent compared to cardiac complications. Long time symptomatic patients are more likely to receive intense medical treatment and are candidates to lower-extremity peripheral revascularization, which makes critical limb ischemia and amputations rare events. The number of these complications seems to be higher, though, in patients who associate chronic kidney disease or diabetes. For patients with PAD, aggressive risk factor modification is the first approach to be considered, followed by early intensive medical therapy. In clinical practice, the major questions to be addressed regarding lipid-lowering therapy in patients with PAD are: Which are the strategies to be used for lowering the cardiovascular risk? What are the clinical benefits of lipid-lowering treatment in PAD? Which lipid fractions should be targeted? How low is low enough? What should be monitored during statin therapy? Reduction of cardiovascular risk Among cardiovascular risk factors, dyslipidemia especially high LDL cholesterol predicts the risk of cardiovascular events, so an attempt to reduce cardiovascular mortality and morbidity in patients with PAD should include lipid-lowering therapies. Diet is the first step for lowering serum cholesterol, along with statin therapy, which is mandatory. Although other lipid-lowering therapies have been tested, some of them being relatively successful concerning the stabilization of atherosclerotic lesions, the improvement of symptomatology or the reduction of cardiovascular risk, the benefits of statin therapy are multiple, so statins represent, so far, the standard therapy for primary and secondary prevention of cardiovascular disease. Simvastatin treatment reduced the rate of first major vascular events in patients with PAD without pre-existing coronary disease, and also prevented the occurrence of subsequent events. Interestingly, this risk reduction was independent of the severity of pre-existing peripheral arterial disease: Improvement of quality of life Regarding functional capacity, lipid-lowering therapy has also proved to be beneficial, multiple studies showing an improvement of walking performance and claudication.

The same Cochrane meta-analysis mentioned above showed the impact of different lipid-lowering drugs on PAD symptoms [4]. Although there was not a significant change of ABI after lipid-lowering treatment, an improvement in the total walking distance and pain-free walking distance was observed. The results of POSCH Program on the Surgical Control of the Hyperlipidemias showed that cholesterol reduction by partial ileal bypass surgery leads to a reduction in the development of clinically evident PAD and of ABI values, although no changes were observed in the peripheral arteriograms after a mean follow-up period of 10 years[5]. This study demonstrated the beneficial effects of statins in slowing the process of atherosclerosis, no matter its location, by also reducing the incidence of carotid bruits and of new or worsening angina. In addition, simvastatin treatment showed improvement in total walking distances and pain-free walking distances in two other studies [7,8]. Moreover, these effects proved to be significant even at the 3 months evaluation [7]. However, the benefits seem to be higher with long-term treatment. A placebo-controlled study by Aronow et al showed an improvement in the pain-free walking time of 54 sec after 6 months and 95 sec after one year, supporting the long-term intensive statin treatment in patients with PAD [8]. Although simvastatin was the most common used statin in studies of PAD, it appears that atorvastatin can also improve the symptomatology of patients with PAD [9]. As expected, a significant reduction in total cholesterol, LDL-cholesterol and triglycerides levels was noticed, as well as a significant increase in HDL-cholesterol in both arms receiving atorvastatin without a significant difference between these two dosing arms , compared to the placebo group. Pleiotropic effects of statins The beneficial impact of statins in patients with PAD is explained not only by their lipid-lowering properties, but also by their pleiotropic effects. Statins play an important role in stabilization and regression of atherosclerotic lesions, a fact supported by studies that used imaging methods. Although peripheral artery atherosclerosis appears to be a marker of systemic atherosclerosis, the evolution of atherosclerosis in peripheral arteries showed a poor correlation with the evolution of atherosclerosis in coronary arteries. This observation demonstrates the great variability in atherosclerosis not only in different individuals, but also in different locations in the same patient. This rapid effect was attributed to the anti-inflammatory properties of statins. Besides stabilization and regression of atherosclerotic plaques, statins were shown to reduce inflammation reflected in lower levels of hs-CRP, fibrinogen, serum neutrophils which, in patients with PAD, correlates with better survival and event-free survival rates mean follow-up period of 21 months [12]. Therefore, one of the most important mechanism by which statins improve the outcome in atherosclerotic patients may be the reduction of vascular inflammation. Furthermore, statins were shown to improve the endothelial dysfunction and the reduced levels of nitric oxide associated with dyslipidemia, which, in turn, leads to an improvement of blood flow in the microcirculation [13]. However, the impact of statin treatment on vasodilation through increasing the nitric oxide in lower extremity arteries has not been assessed yet in large studies. Non-statin lipid-lowering therapy Although data from the Framingham Study indicated that the lipid profile of patients with PAD is that of metabolic syndrome high level of triglycerides and low level of high-density lipoprotein [14], the impact of lowering the other lipid fractions in patients with PAD has been studied less than the effect of lowering the LDL-cholesterol LDL-C level. A study evaluated the effect of benzafibrate on cardiovascular events in patients with PAD and demonstrated a significant reduction in triglycerides level by However, the clinical benefits were not as satisfactory. Benzafibrate treatment showed a reduction in the incidence of non-fatal coronary events, but failed to prove any benefits regarding coronary heart disease and stroke. Colestipol plus niacin in the Cholesterol Lowering Atherosclerosis Study CLAS also led to a decrease of serum triglycerides and an increase of high-density lipoprotein cholesterol HDL-C , along with a decrease in LDL-C, which correlated with a slower progression of atherosclerosis in femoral arteries, although less marked than expected, considering the previous results in coronary artery disease [16]. One study tried to evaluate the evolution of atherosclerotic plaques in the superficial femoral artery by using magnetic resonance imaging in patients with PAD, treated with statin or statin plus ezetimibe. Statin initiation, with or without ezetimibe, proved to stop the progression of atherosclerosis. These results correlate with those of a different study which compared the

effects of niacin added to statin to ezetimibe added to statin therapy [18]. Although the combination of ezetimibe plus statin led to a greater reduction in LDL-C, the use of niacin in addition to statin led not only to a rise in HDL-C, as expected, but also to a significant reduction in cardiovascular events and regression of carotid intima-media thickness over a follow-up of 14 months. On the contrary, there was a paradoxical increase in the carotid intima-media thickness in patients with lower LDL-C levels among those treated with ezetimibe. Although this study did not assess peripheral arteries, it seems that ezetimibe does not reduce the cardiovascular risk and does not prevent the progress of disease in patients with PAD. Monoclonal antibodies that inhibit proprotein convertase subtilisin-kexin type 9 PCSK9 have emerged in the last years as a promising new class of drugs very effective in lowering LDL-C. A recent meta-analysis that included 24 trials evaluated the effects of PCSK9 antibodies on dyslipidemic patients who had not reached LDL-C goals with statin therapy or who were statin intolerant [19]. More importantly, PCSK9 inhibitors showed a significant reduction in all-cause mortality, cardiovascular mortality and myocardial infarctions. However, although these results are encouraging, larger studies are required in order to better characterize these drugs and to assess their possible role in peripheral atherosclerotic disease. Although a threshold for LDL-C is recommended by the guidelines, there are still questions about the optimal treatment regimen that should be adopted in patients with PAD. How intensive should statin therapy be and what is the threshold at which the possible benefits of statin therapy are outweighed by their adverse effects? A study that included over 1, patients over a period of 15 years showed that higher doses of statins and lower LDL-C levels are both independently associated with improved outcome in patients with PAD [21]. This finding comes to support the benefits of statins beyond their lipid-lowering properties. Concern is raised, however, by the fact that too low cholesterol levels might affect serotonin and steroid hormone production, as well as cell membrane function as cholesterol is an important constituent of cell membranes , with severe health consequences. Although some initial studies suggested that a lower cholesterol level might increase the incidence of non-cardiovascular deaths by violence or suicide , recent trials with statins and PCSK9 inhibitors have weakened such an association. The Justification for the Use of Statins in Prevention: During a median follow-up period of 2 years, the rates of adverse effects were similar in the placebo and rosuvastatin groups, except for muscle symptoms. What should be monitored during lipid-lowering therapy? There is limited evidence regarding the monitoring of lipid-lowering therapy. However, this monitoring interval is arbitrary [23]. Similarly, American College of Cardiology guidelines recommend a fasting lipid panel within weeks after initiation of treatment, followed by other assessments every 3 to 12 months[24]. Considering this evidence, both European and American guidelines on the management of dyslipidemias do not recommend routine measurements of creatine kinase in patients receiving statin therapy [23,24]. After assessment of the baseline CK, further measurements should be reserved for patients who exhibit muscle symptoms, especially for patients at risk elderly patients, on multiple medications, or with liver or renal disease. Severe or moderate muscle symptoms should prompt for discontinuation of statin treatment and evaluation of symptomatology. After the symptoms disappear and no other contraindication exists, the American guidelines recommend the same dose or a lower dose of the same statin [24]. If this is not tolerated, a different statin at a low dose can be used, with further increase of dosage. At the same time, other possible causes for myalgia must be excluded. According to European guidelines, statin therapy should be stopped if CK rises above 5 x ULN with no further indications regarding the period of discontinuation, other possible options being left to the decision of the clinician [23]. Both the European and American guidelines recommend baseline measurement of hepatic aminotransferases levels ALT before initiation of statin treatment. Higher values may prompt for treatment interruption, with the possibility of reintroduction of therapy after the ALT values return to normal. Statins also appear to modestly increase the risk of type 2 diabetes. However, the benefits of statins outweigh this possible side effect and no particular recommendations regarding diabetes screening have been made. Furthermore, patients who develop diabetes during statin treatment are encouraged to continue the statin therapy, in order to reduce the cardiovascular risk. In conclusion, patients with PAD are in the high cardiovascular risk category due to systemic atherosclerosis.

Therefore, the first major step in treating these patients is risk factor modification, dyslipidemia representing one critical point to be addressed. Until further studies assess the effectiveness of other novel lipid-lowering drugs, statins remain the key drugs to be used since they have demonstrated a clear reduction in cardiovascular and cerebrovascular events. Moreover, although other therapies seem to effectively improve the lipid profile, they are yet to be assessed regarding the possible benefits they could have on cardiovascular risk and the symptomatology of patients with PAD. Further studies should also improve our understanding of the role statins play in endothelial dysfunction, microcirculation and inflammation, in order to better use them in clinical practice.

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The present study was conducted to determine the proportion of PCI patients receiving lipid-lowering therapy in the Interventional Cardiology Program of a large, tertiary-care, referral centre (University Health Network, Toronto, Ontario), and to evaluate the factors associated with its use in this population.

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