

2013 American College of Cardiology/ American Heart Association Lipid Guidelines after the 2016 American College of Cardiology Expert Panel Consensus Statement: To err is human; to admit it, divine

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Abstract

The 2016 American College of Cardiology (ACC) Expert Panel Consensus Statement addresses the current gaps in low-density lipoprotein cholesterol (LDL-C) lowering strategies to reduce cardiovascular risk. The goal was to provide practical guidance for clinicians and patients in cases not covered by the 2013 ACC/ American Heart Association lipid guidelines until the next round of guidelines has the opportunity to formally review recent scientific evidence and cardiovascular outcomes trials are completed with new agents for cardiovascular risk reduction. The new aspects in comparison to the 2013 guidelines are mainly two. The Writing Committee suggests specific LDL-C targets (which were absent in the 2013 ACC/ AHA guidelines) rather than LDL-C percent reductions so that we have a bench-



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mark to use statin with non-statin drug combinations, and it encourages the use of ezetimibe and proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors if statin monotherapy is not enough for the attainment of these specific LDL-C targets or in cases of statin intolerance. The Consensus acts in several issues as a bridge between the 2013 guidelines to the next ACC/AHA guidelines as well as to European Society of Cardiology/European Atherosclerosis Society guidelines. Our opinion is that there is still way to go, because we live in an obese world with a pandemic of diabetes mellitus and the related mixed (combined) dyslipidaemia, responsible for the residual cardiovascular risk after statin treatment; this issue has not been addressed at all by the Consensus, which remains LDL-C-oriented.

Key words: 2016 ACC Consensus Statement; 2013 ACC/AHA Guidelines; statins; non-statin treatment

Introduction

The management of dyslipidaemias is a major component of primary and secondary cardiovascular disease (CVD) prevention strategies. In this context, several medical organizations have formulated guidelines for the management of dyslipidaemias. The first lipid guidelines were issued in November of 1985 from the National Cholesterol Education Program (NCEP), a branch of the National Heart, Lung, and Blood Institute (NHLBI) of United States. Europe issued lipid guidelines 4 years later (European Atherosclerosis Society (EAS) 1989) and Greece at present follows the 2014 (updated) Hellenic Atherosclerosis Society guidelines, based on the first Joint EAS/European Society of Cardiology (ESC) lipid guidelines issued in 2011.¹ These guidelines are simple, have one primary objective [the reduction of low density lipoprotein cholesterol (LDL-C)], and 3 levels of CVD risk with specific LDL-C targets of < 115, < 100 and < 70 mg/dl. These guidelines have issued coloured charts showing the total CVD risk according to major CVD risk factors, setting the cut of point (SCORE) at 5% risk of fatal CVD during the next 10 years (**Figure 1**). The guidelines encourage the European countries to issue their own guidelines according to local data, and Greece was the second European country to do that (HellenicSCORE - a Calibration of the ESC SCORE Project, by the group of Profs Stefanadis, Pitsavos and Panagiotakos)². All the above made

HellenicSCORE very easy to implement. It's only disadvantage is that it is based on the risk of CVD mortality and not morbidity and mortality (i.e. it does not predict the risk for non-fatal myocardial infarction and non-fatal stroke).

In 2013, the joint American College of Cardiology (ACC)/American Heart Association (AHA) guidelines on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular (ASCVD) risk in adults were published.³ These guidelines stirred a wave of criticism regarding their accuracy and applicability.^{4,8} The ESC/EAS task force for lipid guidelines published the European evaluation of the 2013 American guidelines and expressed the opinion that the ESC/EAS guidelines from 2011 seem to be the most pragmatic and appropriate choice for European countries.⁸ The criticism on the 2013 ACC/AHA guidelines is summarized below.

1. Summary of the ACC/AHA lipid guidelines³

The guidelines identify four high-risk groups that could benefit from statin treatment:

- Patients with pre-existing CVD
- People with familial heterozygous hypercholesterolaemia (HeFH), as evidenced by an LDL-C \geq 190 mg/dl
- Those aged 40 to 75 years who have type 2 diabetes mellitus (T2DM)
- People 40 to 75 years-old with at least 7.5% risk of developing CVD in the next decade according to a

formula described in the guidelines (Pooled Cohort Risk Equation).

The risk of developing CVD in the next decade was measured by a new risk calculator (<http://tools.acc.org/ASCVD-Risk-Estimator/>).

In contrast to all other guidelines for the management of dyslipidemia, the 2013 ACC/AHA guidelines **do not recommend specific LDL-C targets**. Instead, they propose administering high- or moderate-intensity statin therapy depending on the CVD risk, because the Expert Panel was unable to find randomized controlled trials (RCT) evidence to support the use of specific LDL-C or non-high density lipoprotein cholesterol (HDL-C) treatment targets. The aim of these guidelines was to achieve a > 50% reduction in LDL-C levels in high risk patients, a 30-50% reduction in LDL-C levels in moderate risk patients, and < 30% reduction in low risk patients.³

- High-intensity statin therapy includes atorvastatin 40-80 mg/day and rosuvastatin 20-40 mg/day.
- Moderate-intensity statin therapy includes atorvastatin 10-20 mg/day, rosuvastatin 5-10 mg/day, simvastatin 20-40 mg/day, pravastatin 40-80 mg/day, fluvastatin 40-80 mg/day and pitavastatin 2-4 mg/day.
- According to the ACC/AHA guidelines, patients aged ≤ 75 years with established CVD (coronary heart disease (CHD), stroke or peripheral arterial disease) and subjects with LDL-C levels > 190 mg/dl should be treated with high-intensity statin therapy.
- Patients aged 40-75 years with T2DM and LDL-C 70-189 mg/dl but without CVD should be treated with high-intensity statin therapy only if their estimated 10-year risk for CVD (including CHD death, nonfatal myocardial infarction, fatal and nonfatal stroke) is ≥ 7.5% and with moderate-intensity statin therapy if their estimated 10-year CVD risk is < 7.5%.
- Finally, patients aged 40-75 years with LDL-C 70-189 mg/dl but without T2DM or CVD should be treated with high- to moderate-intensity statin therapy if their estimated 10-year CVD risk is ≥ 7.5%, whereas it is reasonable to administer moderate-intensity statin therapy if their estimated 10-year CVD risk is 5% to 7.5%.
- In other patient groups (i.e. those older than 75 years with or without CVD or T2DM and those

without CVD or T2DM and with 10-year CVD risk < 5%), the use of statins should be individualized based on perceived benefits and risks of statin treatment, potential for drug-drug interactions, and patient's preferences.

2. Potential problems with the ACC/AHA lipid guidelines⁴⁻⁸

1. The guidelines are based solely on epidemiological data and not prospective, randomised, controlled, survival trials.
2. The studies used for the equation formula (pooled cohort risk score)⁹ were conducted in US only. Thus, these guidelines might be applicable in US but not elsewhere (Europe, Asia, South America).⁸
3. The guidelines suggest only statin treatment and practically ignore all other hypolipidaemic agents.⁷
4. The algorithms used to drive the choice of treatment are very complex and might not be understandable by all physicians that have to implement a number of algorithms for various diseases within their speciality.⁷
5. There is no mention about chronic kidney disease (CKD), a CHD risk equivalent.¹⁰
6. The formula for risk calculation (Pooled Cohort Risk Equations) seems to overestimate cardiovascular risk in subjects without overt CVD of diabetes and may substantially increase the number of people that require statin treatment.¹¹⁻¹³ It has been projected that the application of the AHA/ACC guidelines will render eligible for statin treatment more than 1 billion subjects worldwide.⁶
7. The lack of specific LDL-C targets is also an important problem.⁷ The mean LDL-C value in patients with HeFH is 280 mg/dl. According to the ACC/AHA guidelines a 50% reduction is enough. However, could anybody accept that an LDL-C value of 140 mg/dl is acceptable in these patients?
8. These guidelines will be an immense barrier to the use of new hypolipidaemic drugs, such as antibodies against proprotein convertase subtilisin/kexin type 9 (PCSK9) (evolocumab and alirocumab) that are already commercially available.⁵

All the above limitations lead the Task Force of the EAS/ESC, the American Lipid Association, the

American Society of Clinical Endocrinologists, and other scientific societies from South America and Asia to decline to endorse these new cholesterol guidelines and suggest adhering to previous guidelines. In contrast, the 2011 ESC/EAS guidelines appear to be the most pragmatic and appropriate option for European countries.⁸

During the last 2 years there were some publications in different populations mainly within US, which report that the implementation of the 2013 ACC/AHA lipid guidelines is beneficial for the patients, because percent LDL-C reduction added incremental prognostic value over statin dose and attained LDL-C levels.¹⁴⁻²⁰ However, the implementation of these guidelines in US is very low two years after their release. Only 11.5% of 8,762 Medicare beneficiaries with a CVD event received a prescription for a high-intensity statin within one year after hospital discharge.²¹

3. Interesting points of the 2016 ACC Consensus Statement

A few days ago, an ACC Expert Consensus Decision was published.²² The paper argues that at the time of the initial 2013 ACC/AHA guideline publication, the panel could not find any data supporting the routine use of FDA-approved non-statin drugs combined with statin therapy for LDL-C reduction with the objective of further reducing CVD events.²² In addition, no published RCTs that assessed CVD outcomes in statin-intolerant patients were found.²² Therefore, the panel recommended that clinicians treating high-risk patients who have a less than-anticipated response to statins, who are unable to tolerate a less-than-recommended intensity of a statin, or who are completely statin intolerant, may consider the addition of a non-statin cholesterol lowering therapy.²² Below there are some interesting points in agreement with the 2013 ACC/AHA guidelines, but mainly those that differentiate the Consensus from the guidelines:

- The Expert Consensus Writing Committee endorses the evidence-based approaches to cardiovascular risk reduction in adults included in the 2013 ACC/AHA Guideline.
- The treatment algorithms begin with the assumption that the patient is in 1 of the 4 evidence based statin benefit groups identified in the 2013 ACC/AHA guidelines.
- Before initiation of any combination therapy (statin plus a non-statin drug), it is imperative for clinicians and patients to engage in a discussion that addresses the potential net gains, including absolute cardiovascular risk-reduction benefits and potential harms, prescribing considerations, and patient preferences for treatment.
- Assuming adherence to therapy, patients with LDL-C levels above that range may not achieve maximal benefit and might be considered for additional therapy. The Committee therefore judged that it was appropriate to provide levels of LDL-C, or “thresholds”, in terms of both percentage LDL-C reduction from baseline and absolute on-treatment LDL-C levels, which, if not achieved by adherent patients, would serve as factors to consider in decision making regarding additional (non-statin) therapy.
- The Committee endorses use of the Friedewald equation in most cases, given that the majority of RCTs used this equation, it is the most widely available in clinical practice, and tends to cost less.
- Although there is a gap in RCT evidence demonstrating outcome benefits from using combination therapy in patients with stable CVD, the Committee supports consideration of adding ezetimibe as the first non-statin agent, given the benefits on CVD outcomes and demonstrated safety of ezetimibe in patients with acute coronary syndromes (ACS).²³
- If the goals of therapy defined in the clinician-patient discussion have been achieved with the addition of ezetimibe, it is reasonable to continue the statin plus ezetimibe therapy and continue to monitor adherence to medications and lifestyle, and LDL-C response to therapy.
- If CVD patients without comorbidities, who are on maximally tolerated statin-ezetimibe or non-statin combination therapy in the setting of documented statin intolerance, achieve a less than-anticipated response with < 50% reduction in LDL-C (or on-treatment LDL-C \geq levels 100 mg/dL), it is rea-

sonable to engage in a clinician-patient discussion with consideration of the net benefit of alirocumab or evolocumab (in addition to or in place of ezetimibe) as a second step to achieve further LDL-C reduction.

- Referral to a lipid specialist should be strongly considered for patients with LDL-C \geq 190 mg/dL and is definitely recommended for children, adolescents, women during pregnancy, and patients with HoFH or severe HeFH. In patients who have a less-than-anticipated response to maximally tolerated statin therapy with $<$ 50% reduction in LDL-C (or on-treatment LDL-C levels \geq 70mg/dL), the clinician and patient should address statin adherence by assessing the number of missed statin doses per month and evaluating any barriers to adherence. The Committee emphasizes that if an adherent patient has not been administered high-intensity statin, the statin dose should be increased to a high-intensity at this time. Patients who are unable to tolerate even a moderate-intensity statin should be evaluated for statin intolerance and considered for addition of a non-statin medication to the current regimen. Addition of ezetimibe may be considered based upon the improved CVD outcomes. In those patients, PCSK9 inhibitors may be considered as a first step rather than ezetimibe because of greater LDL-C lowering efficacy.^{24,25}
- Available evidence support the continuation of high-intensity statins beyond 75 years of age in persons who are already taking and tolerating these drugs.
- Specialized therapies, such as mipomersen, lomitapide, or LDL apheresis, may be needed to control LDL-C in patients with ASCVD and baseline LDL-C \geq 190 mg/dL who have an inadequate response to statins with or without ezetimibe and PCSK9 inhibitors.²⁶
- Management of FH in children and adolescents was beyond the scope of this Consensus but has been reviewed in detail elsewhere.^{27,28}
- The Committee did not consider therapies (prescription omega-3 fatty acids, fibrates) for severe hypertriglyceridaemia, which is common in pa-

tients with T2DM, since this topic has been addressed elsewhere.^{29,30}

This Consensus Paper tries to incorporate issues raised by the 2013 ACC/AHA Statin Guidelines and improve the acceptance of the (forthcoming) new Lipid ACC/AHA Guidelines, after the publication of the results of the first RCT on CVD event reduction with PCSK9 inhibitors expected later in 2016. This paper seems to act like a transition or a bridge from the 2013 to the (expected) newer ACC/AHA Guidelines, resolving some issues of the past and leaving others to be resolved in the future ACC/AHA Lipid Guidelines.

4. Similarities and differences between the recommendations of the 2016 ACC Consensus and the 2013 ACC/AHA Guidelines

The Consensus considers that it was appropriate to provide levels of LDL-C, or “thresholds”, in terms of both proportional LDL-C reductions from baseline but also absolute on-treatment LDL-C levels. The latter point was the more fundamental difference of the 2013 ACC/AHA Guidelines with all previous American or European Guidelines. Besides, this lack of LDL-C treatment targets was the main point of criticism during the last 3 years.

The first point of interest is related to the LDL-C level point beyond which the atherosclerotic plaques begin to regress. Intensive reduction of LDL-C below specific levels is required to achieve slowing of progression as well as regression of atherosclerosis.³¹⁻³⁴ Thus, there is a need to know these specific LDL-C levels (the Consensus suggests: you may consider LDL-C $<$ 70 mg/dL or non-HDL-C $<$ 100 mg/dL, according to baseline CVD risk) in order to achieve them and induce plaque stabilization or regression in the effort to avoid plaque rupture and cardiovascular events. Plaque regression correlates, among others, with absolute LDL-C levels, but not with LDL-C percent reduction.^{34,35}

The second point of interest is related to the suggestion of the Consensus to use non-statin therapies such as ezetimibe and PCSK9 inhibitors (at present monoclonal antibodies against PCSK9). This is closely related to the first point above, because without

adopting specific treatment LDL-C targets (the Consensus suggests: you may consider LDL-C < 70 mg/dL or non-HDL-C < 100 mg/dL, according to baseline CVD risk) it would be impossible to know when to use non-statin therapies in cases that statin treatment has achieved > 50% reduction, but the baseline LDL-C levels are very high and the post treatment LDL-C values could not be considered acceptable. For example, patients with HeFH have a mean baseline LDL-C value of 280 mg/dL. Even rosuvastatin 40 mg/day (the higher dose of the most potent statin) may reduce it to 125 mg/dL. This is not acceptable for either primary (previous target, < 100 mg/dL) or secondary (previous target, < 70 mg/dL) CVD prevention. In the first case we can use ezetimibe but in the second PCSK9 inhibitor to achieve recommended by the Consensus LDL-C treatment targets. If there is no specific LDL-C treatment target and we only apply a percent reduction in LDL-C, as suggested by the 2013 ACC/AHA Guidelines, it would be impossible to proceed to combination treatment with a statin and a non-statin drug in the effort to optimize hypolipidaemic drug treatment and minimize CVD risk.^{36,37}

The third point of interest is that there is a change of policy in adopting treatments. The 2013 ACC/AHA Guidelines put a lot of emphasis on the fact that these guidelines were based solely on the results of RCTs with clinical outcomes. This is the reason that they did not include any suggestions for the use of other hypolipidaemic drugs besides statins. In contrast, they now state "Although there is a gap in RCT evidence demonstrating outcomes benefits of using combination therapy in stable clinical CVD patients, the Consensus supports consideration of adding ezetimibe 10 mg daily as the first non-statin agent". The basis of suggesting therapies proved as safe and useful only by RCT is abandoned, because it was too narrow to cover all cases. Moreover, the authors state "In the opinion of the Expert Consensus Writing Committee, in a patient with CVD and baseline LDL-C \geq 190 mg/dL with < 50% reduction in LDL-C (and may consider LDL-C \geq 70 mg/dL) it is reasonable to consider a PCSK9 inhibitor as a first step rather than ezetimibe or BAS given PCSK9 inhibitors' great-

er LDL-C lowering efficacy". In the case of ezetimibe there is a positive RCT survival study, even though this included patients with ACS. However, PCSK9 inhibitors have been approved by the FDA and the European Medicines Agency (EMA) but only as hypolipidaemic drugs and there are two post hoc (pre-specified) analyses suggesting substantial clinical benefit^{38,39}; nevertheless there are no results from the large prospective, randomised, double blind, placebo controlled, long term studies and despite this, PCSK9 inhibitors are suggested by the Consensus. The basis of suggesting therapies proved as safe and useful only by RCT is abandoned and even drugs without FDA or EMA approval for CVD prevention are suggested by the Consensus.

In addition, the recognition by the Committee of CKD as a high CVD risk disease state and includes patients with CKD, not on dialysis, with or without overt CVD, in those who may merit special consideration for more intensive LDL-C lowering than the general population even with use of a non-statin medication.

In cases of statin intolerance and LDL-C reduction < 50% and may consider LDL-C \geq 70 mg/dL, addition of a second non-statin agent to achieve further LDL-C reduction is reasonable for patients on maximally tolerated statin-ezetimibe, statin-PCSK9 inhibitor, or non-statin combination therapy in the setting of documented statin intolerance. This is also a helpful recommendation.

Patients with LDL-C \geq 190 mg/dL, probably due to FH, with CVD will probably need treatment with a PCSK9 inhibitor in combination with maximally tolerated statin therapy (LDL-C reduction < 50% and may consider LDL-C level > 70 mg/dL) because they are at a very high CVD risk since birth. However, patients with baseline LDL-C \geq 190 mg/dL and without CVD or comorbidities, achievement of LDL-C reduction \geq 50% and LDL-C < 130 mg/dL is a reasonable therapeutic target not requiring further intensification of therapy. In European guidelines and all FH Consensus, this threshold is < 100 mg/dL.

The suggestion of specialized therapies, such as mipomersen, lomitapide, or LDL apheresis, for HoFH or severe HeFH mainly with CVD as well as the use

of high intensity statins with or without ezetimibe and PCSK9 inhibitors is also very constructive.

5. Issues not addressed by the Consensus Statement

It is not encouraging that the Consensus suggests that in patients with heart failure (HF) New York Heart Association Class II-III, the use of statins should not be considered, with the exception of patients with symptomatic HF due to ischemic aetiology (statin is administered in this case for CHD and not for HF) who have a life expectancy to allow benefit from the statin therapy (3-5 years at least). A recent (2015) large meta-analysis, which analysed unpublished data from 17 major RCT with 132,538 participants (mean follow-up time 4.3 years), showed that statin treatment reduced non-fatal HF hospitalization (risk ratio (RR) 0.90, 95% confidence interval (CI) 0.84-0.97), and the composite outcome (first non-fatal HF hospitalization or HF death) by 8% (95% CI 0.85-0.99).⁴⁰ There was no difference in risk reduction between those who had a history of myocardial infarction and those who did not.⁴⁰ In this case, the authors of the Consensus considered only RTCs like Controlled Rosuvastatin Multinational Trial in Heart Failure (CORONA)⁴¹⁻⁴² and Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto miocardico (GISSI)-HF,⁴³ but not this large meta-analysis⁴⁰ or the n-3 polyunsaturated fatty acids (PUFA) arm of the same GISSI-HF trial,^{44,45} which showed that n-3 PUFA provided a small but significant reduction in all-cause mortality in patients with symptomatic HF of any aetiology, already treated with evidence-based therapies.^{44,45}

The Committee also "did not consider therapies for severe hypertriglyceridaemia (n-3 PUFA or fibric acid derivatives), which have been addressed elsewhere recently".^{46,47} For the same reason, the Consensus did not include RTCs like the GISSI-Prevenzione trial⁴⁸ (n= 11,324) and the Japan EPA Lipid Intervention Study (JELIS) trial⁴⁹ (n= 18,645). The GISSI trial had a primary composite endpoint of cumulative incidence of death, non-fatal myocardial infarction, and stroke. The significant benefit was mainly attributed to sudden cardiac death (44% reduction).⁴⁸ In the JELIS trial, n-3 fatty acid supplementation re-

duced major CHD events by 19% in the secondary prevention subgroup (p= 0.049).⁴⁹ Other studies have not confirmed these findings of the GISSI-Prevenzione and JELIS trials, but these had different target populations, different doses of n-3 fatty acids, and different endpoints. Besides, fibrates and n-3 fatty acids are not used only for the treatment of severe hypertriglyceridaemia, but for the treatment of moderate hypertriglyceridaemia, and for the treatment of mixed (combined) atherogenic dyslipidaemia, which is related to residual CVD risk. Mixed, or combined or atherogenic dyslipidemia is the dyslipidemia of insulin resistance and plays a key role in increasing CVD risk.⁵⁰ Epidemiological data suggest that mixed (atherogenic) dyslipidaemia has a prevalence of 20%⁵¹ in the general population and of 33-45% in statin-treated patients.^{52,53} This raises question on why fibrate treatment was left out of the non-statin Consensus since it is at present (in combination with statins) the main if not the only treatment for mixed dyslipidaemia⁵⁴⁻⁶¹, which is present in a large part of subjects with dyslipidaemia results in increased residual CVD risk despite effective statin treatment and attainment of LDL-C target goals.⁶² Indeed, in the Fenofibrate Intervention and Event Lowering in Diabetes (FIELD) study, which included type 2 diabetes patients (n= 9,795), 19% of the total study population had atherogenic dyslipidaemia. These patients obtained greater benefit from fenofibrate treatment than those without this dyslipidaemia (27% relative reduction in CVD events *vs* 11% in all patients).^{57,64} In addition, in the Action to Control Cardiovascular Risk in Diabetes (ACCORD) Lipid Trial (n= 5,518), a pre-defined subgroup analysis indicated substantial benefit associated with combining fenofibrate with background simvastatin in patients with T2DM and atherogenic dyslipidaemia defined by baseline triglycerides in the upper third of the population and baseline HDL cholesterol levels in the lower third. This group represented about 17% of the overall study population and had a 31% relative reduction in CVD events *vs* no benefit in patients without this dyslipidaemia.^{60,64} Finally, in an Israeli study in patients with ACS, the statin plus fibrate combination yielded a > 50% in CVD mortality

ty during the first 30 days, while the reduction in all CVD events during the subsequent year was 46%.⁶¹

In conclusion, the Consensus improved several aspects of the 2013 ACC/AHA Guidelines that have been a barrier in their implementation. The most important differentiation was the fact that Consensus uses specific treatment goals for LDL-C (< 70 or < 100 mg/dl, according to CVD risk) so that we have a benchmark where to use statin plus non-statin drug combinations. Also, the suggestion to use ezetimibe or/and PCSK9 inhibitors in the cases that specific LDL-C treatment targets cannot be reached with statin monotherapy or in cases of statin intolerance was very constructive. However, the next

ACC/AHA Guidelines will have to deal with fibrates and n-3 PUFA. Alternatively, the Committee might suggest other ways to treat mixed (atherogenic) dyslipidaemia related to insulin resistance in a world with a pandemic of obesity, metabolic syndrome and T2DM. ◊

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Περίληψη

Οι κατευθυντήριες οδηγίες για τη θεραπεία των δυσλιπιδαιμιών του 2013 του Αμερικανικού Κολλεγίου Καρδιολογίας/ Αμερικανικής Καρδιολογικής Εταιρείας μετά τη Συμφωνία Ειδικών του Αμερικανικού Κολλεγίου Καρδιολογίας του 2016: Να σφάλεις είναι ανθρώπινο, να το αναγνωρίζεις θεϊκό

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Το 2016 ομάδα εμπειρογνομόνων του American College of Cardiology (ΑΚΚ) εξέδωσε συμφωνία ειδικών για την αντιμετώπιση των ελλείψεων της θεραπευτικής αντιμετώπισης των υψηλών επιπέδων της χαμηλής πυκνότητας λιποπρωτεϊνικής χοληστερόλης (LDL-C), με σκοπό την μείωση του καρδιαγγειακού κινδύνου. Ο στόχος ήταν η παροχή πρακτικής καθοδήγησης για τους κλινικούς ιατρούς και τους ασθενείς σε περιπτώσεις που δεν καλύπτονται από τις κατευθυντήριες οδηγίες για την θερα-





Περίληψη (συνέχεια)

πεία των δυσλιπιδαιμιών του ΑΚΚ / American Heart Association (ΑΚΕ) του 2013 μέχρι που να εκδοθούν οι επόμενες κατευθυντήριες οδηγίες. Οι νέες πτυχές σε σχέση με τις κατευθυντήριες οδηγίες του 2013 είναι κυρίως δύο. Η Επιτροπή προτείνει συγκεκριμένους στόχους LDL-C (στοιχείο που δεν υπήρχε στις κατευθυντήριες οδηγίες του 2013 ΑΚΚ / ΑΚΕ), πέρα από τις ποσοστιαίες μειώσεις της LDL-C, έτσι ώστε να έχουμε ένα σημείο αναφοράς για χρήση στατινής με συνδυασμούς υπολιπιδαιμικά που δεν είναι στατίνες, και ενθαρρύνει τη χρήση των αναστολέων PCSK9 και της εξετιμίμπη εάν η μονοθεραπεία με στατίνη δεν είναι αρκετή για την επίτευξη αυτών των συγκεκριμένων στόχων της LDL-C ή σε περιπτώσεις δυσανεξίας στις στατίνες. Η συμφωνία ειδικών δρα σε διάφορα ζητήματα ως γέφυρα μεταξύ των κατευθυντήριων γραμμών του 2013 με τις (αναμενόμενες) επόμενες κατευθυντήριες οδηγίες ΑΚΚ / ΑΚΕ, καθώς και με τις Ευρωπαϊκές κατευθυντήριες οδηγίες της Ευρωπαϊκής Καρδιολογικής Εταιρείας / Ευρωπαϊκής Εταιρείας Αθροσκληρώσεως του 2011. Η γνώμη μας είναι ότι υπάρχει ακόμα πολύς δρόμος να διανύσουμε, διότι ζούμε σε ένα παχύσαρκο κόσμο με πανδημία σακχαρώδη διαβήτη που συνδέεται με την μικτή αθηρογόνο δυσλιπιδαιμία, υπεύθυνη για τον υπολειπόμενο καρδιαγγειακό κίνδυνο μετά τη θεραπεία με στατίνες, και η τελευταία δεν έχει αντιμετωπιστεί καθόλου από την συμφωνία ειδικών του ΑΚΚ του 2016, η οποία παραμένει προσανατολισμένη αποκλειστικά στην θεραπεία της υψηλής LDL-C.

Λέξεις ευρητηρίου: 2016 ΑΚΚ συμφωνία ειδικών, 2013 ΑΚΚ/ΑΚΕ κατευθυντήριες οδηγίες, στατίνες, θεραπεία με άλλα υπολιπιδαιμικά πλην στατινών

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