Improvement in the Management of Dyslipidemia in a Clinical Practice Population at Very High Cardiovascular Risk. The COLIPAR Project

Mejora del tratamiento de la dislipemia para pacientes con muy alto riesgo cardiovascular en la práctica clínica. Proyecto COLIPAR

To the Editor,

Increased low-density lipoprotein cholesterol (LDLc) is associated with symptoms and death due to cardiovascular disease (CVD).[1] Reduction of LDLc with statins decreases the risk of cardiovascular events.[2] The guidelines for the management of dyslipidemia of the European Society of Cardiology, which were in force at the start of the COLIPAR project presented here, recommend LDLc < 70 mg/dL for very high-risk patients.[1]

This goal is, however, attained in only a low percentage of patients: 33% in the DYSIS registry[3] and 26% in the REPAR registry.[4] Furthermore, a very high percentage of patients diagnosed with CVD maintain levels above 70 mg/dL, although clinicians are often unaware that this poor control can negatively affect their patients.

In view of the above, we decided to assess the degree of lipid control in our very high-risk patients and to share the findings with our colleagues in primary and specialist care to determine whether awareness of the degree of lipid control might help to achieve lipid targets.

All patients with documented CVD in our health area were identified with the clinical management system (Outpatient Information System-Abucasis), which covers all diagnoses. For the present study, a patient was considered at high risk if at least 1 of the following conditions was diagnosed: ischemic heart disease, cerebrovascular disease, or peripheral artery disease. All LDLc measurements were retrieved over a 1-year period (September 2012 to September 2013). The percentage of patients with satisfactory control (cLDL < 70 mg/dL) was estimated. A total of 8 face-to-face meetings and 3 online seminars were held during a 1-year period (March 2014 to April 2015). During these seminars, our colleagues in primary and specialist care were informed of the percentage of patients with satisfactory control in their health center or service, and of the benefits of improving lipid control. After 1 year, data were extracted again from the system to assess whether there was any improvement (Figure 1).

Of a total of 311 672 individuals in our health area, we identified 4258 patients with CVD. Of these, only 1820 (43%) had at least 1 laboratory determination that included LDLc during baseline data extraction (September 2012 to September 2013). Initially and in general, 33.6% had adequate LDLc control (mean, 86.7 ± 30.3 mg/dL). One year after the seminars revealing the degree of control to all health centers and specialties and emphasizing the importance of improving control, 1595 of the 1820 patients (88%) had undergone measurement. In these patients, the degree of lipid control improved significantly, to 45.6% of patients with satisfactory control ($P < .0001$). Mean LDLc was 77.9 ± 28.5 mg/dL. As seen in Figure 2, which presents data by individual center, many specialties and health centers improved the percentage of patients with well-controlled LDLc.

In line with all published registries, the present study highlights inadequate lipid control in patients with high cardiovascular risk in our cohort. The novelty of this study, however, is that it demonstrates how a joint effort between specialists and primary care, who were made aware of the percentage of well-controlled patients and the importance of good lipid control, resulted in treatment optimization in these patients and a significant improvement in lipid levels. The most important shortfall detected was the absence of lipid measurements, affecting 57% of patients at baseline and 12% after the program, when clinical practice guidelines clearly recommend annual LDLc measurements in these patients.[1] In contrast, the main achievement detected in this study is that the percentage of patients with LDLc< 70 mg/dL increased from 34% to 46%. This improvement was most evident in centers with lower percentages of patients with good control to start with, and where the message about satisfactory lipid control had the greatest impact (Figure 2).

The baseline figures for failure to attain the lipid goals in patients with very high cardiovascular risk are very similar to those in the DYSIS registry,[3] but they are far below the recommendations of the European Society of Cardiology.[1]

We believe that patients and physicians are both responsible for the failure to attain lipid goals. Often, once the initial stages of the disease have passed, patients may relax and not follow recommendations on diet, exercise, and even medication. For their part,

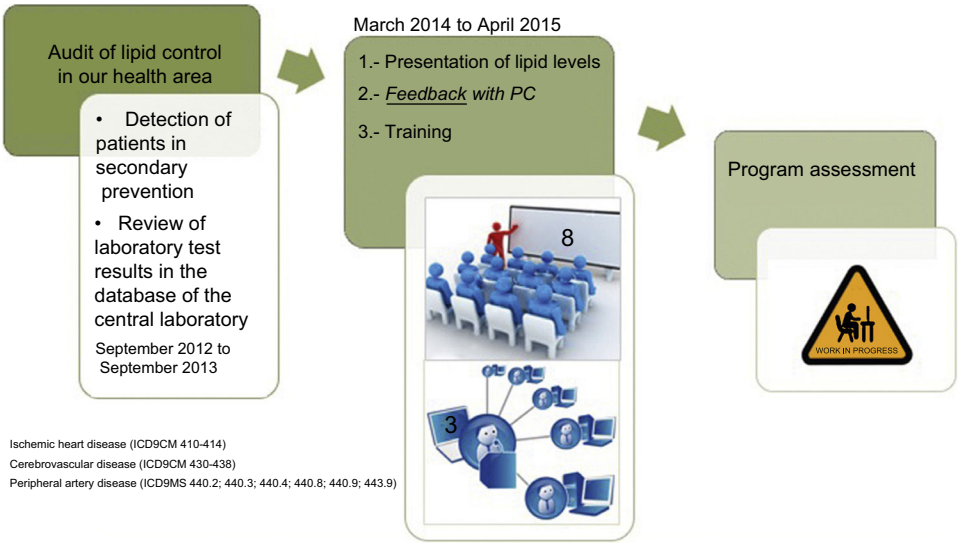

Figure 1. Diagram summarizing the study methodology. ICD9CM, International Classification of Diseases, 9th edition, Clinical Modification; PC, primary care.

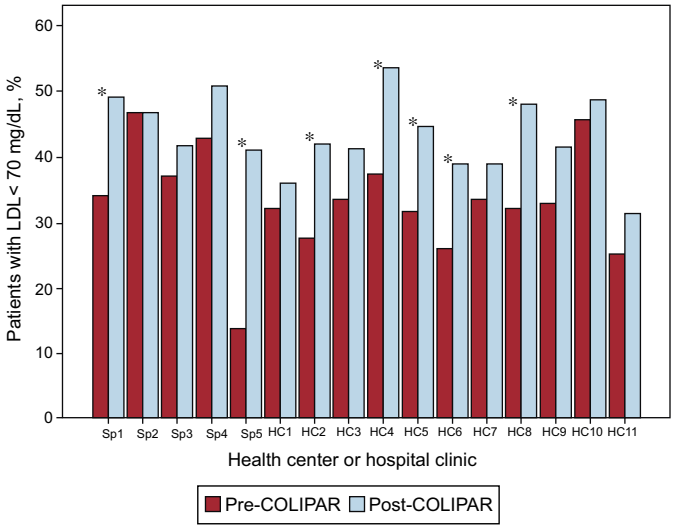

Figure 2. Percentage of patients with LDLc < 70 mg/dL before and after the COLIPAR program. HC, health center; LDLc, low-density lipoprotein cholesterol; Sp, specialty.
*$P < .05$.

physicians suffer from therapeutic inertia due to the lack of time with the patients and lack of awareness of the importance of satisfactory lipid control. These factors prevent them from intensifying treatment to achieve treatment goals. An intervention such as that described in this study, stressing the importance of good control and presenting real data from our patients, has proven useful for improving lipid control.

The main limitation of this study is the lack of information on how long ago the clinical events occurred or on the history or treatments of the patients included.

In summary, lipid control in very high-risk patients in a clinical practice cohort was unsatisfactory, but cooperation with primary care, with appropriate information and heightened awareness, seems essential to improve control.

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Available online 14 October 2017

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http://dx.doi.org/10.1016/j.rec.2017.07.015
1885-5857/

Acute Myocardial Infarction, Stroke and Bilateral Carotid Vasospasm: A Rare Association

Infarto agudo de miocardio, ictus y vasoespasmo carotídeo bilateral: una rara asociación

To the Editor,

A 52-year-old woman was admitted with right ventricular, inferior-posterior wall acute myocardial infarction, Killip class IV. She had a history of active smoking, hypertension, chronic obstructive pulmonary disease, HIV infection on treatment with darunavir and ritonavir, and depressive syndrome treated with citalopram.

She rang the emergency services at 1:15 AM with chest pain that had started at 6:30 PM, and was found to have a blood pressure of 61/30 mmHg, complete atrioventricular block at 20 bpm, narrow QRS interval and ST elevation in inferior and right leads (5 mm), and reciprocal changes in V_2-V_3. At home, the patient was started on an adrenaline intravenous infusion (0.08 mg/h) and was given aspirin 250 mg and unfractionated heparin 4200 IU, both intravenously, and clopidogrel 600 mg orally. She was then transferred to the cardiac catheterization unit at 3:35 AM.

The adrenaline infusion was discontinued. A transfemoral pacing catheter was deployed and coronary angiography was performed via a femoral approach (due to radial vasospasm), showing acute occlusion of the proximal right coronary artery (Thrombolysis in Myocardial Infarction [TIMI] 0, Rentrop 0) (Figure A) and severe vasospasm of the left anterior descending artery (Figure B). A thrombectomy was performed with metal stent implantation. The final TIMI score and blush grade were both 3 at 4:29 AM.

Dobutamine and noradrenaline were then administered for 24 hours postintervention. The pacing catheter was withdrawn 48 hours later. Echocardiography showed moderately depressed biventricular systolic function (left ventricular ejection fraction 35%; tricuspid annular plane systolic excursion [TAPSE] 11 mm) and inferior wall akinesis. The findings were confirmed by cardiac magnetic resonance imaging (MRI).

Despite making good clinical progress, the patient had 2 transient episodes of facial and right arm paresis with motor aphasia, at 72 hours and 96 hours postadmission. Computed tomography showed only severe bilateral stenosis of the extracranial cervical segments of the internal carotid artery. Both arteries were patent (Figure C). These findings were confirmed on carotid Doppler ultrasound and magnetic resonance angiography (Figure D). Carotid dissection and aortic arteritis were ruled out. In the brain MRI we found unexpected multiple subacute ischemic infarcts in different territories (predominantly in the left hemisphere) of possible embolic cause (Figure E) and we therefore prescribed oral anticoagulants.