Opportunity for Intervention to Achieve American Heart Association Guidelines for Optimal Lipid Levels in High-Risk Women in a Managed Care Setting

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Background—The American Heart Association (AHA) recently established evidence-based recommendations for cardiovascular disease (CVD) prevention in women, including lipid management. This study evaluated optimal lipid-level attainment and treatment patterns on the basis of these guidelines in high-risk women in a managed care setting.

Methods and Results—We conducted a historical prospective cohort analysis of a 1.1-million-member, integrated, managed-care database. Eligible high-risk women were those with evidence of previous CVD or risk equivalent who had a full lipid panel available between October 1, 1999, and September 30, 2000; were naive to lipid therapy; and had a minimum of 12 months health plan eligibility preindex and postindex lipid panel. Optimal lipid levels were defined as LDL cholesterol (LDL-C) <100 mg/dL, HDL cholesterol (HDL-C) >50 mg/dL, non–HDL-C <130 mg/dL, and triglycerides <150 mg/dL. Laboratory values and lipid pharmacotherapy were assessed longitudinally over the postindex follow-up (up to 36 months). A total of 8353 high-risk women (mean age, 66 ± 14 years) with a mean follow-up of 27 ± 8 months were included. Only 7% attained optimal combined lipid levels initially, and this increased to 12% after 36 months. Lipid-modifying therapy was initiated in 32% of patients, including 35% of women with LDL-C ≥100 mg/dL and 15% with LDL-C <100 mg/dL.

Conclusions—Among high-risk women, few attained the AHA’s standards for all lipid fractions, and only one third received recommended drug therapy, highlighting significant opportunities to apply evidence-based recommendations to manage lipid abnormalities in high-risk women. (Circulation. 2005;111:488-493.)

Key Words: women ▪ lipids ▪ lipoproteins ▪ cardiovascular diseases ▪ cholesterol

Cardiovascular disease (CVD) is the leading cause of death in women in the United States. Despite the advances in treatment, CVD contributes to 500 000 fatalities among women in the United States each year.1 Hypercholesterolemia is one of the main modifiable risk factors of cardiovascular morbidity and mortality.2,3 This is especially true of high-risk women, defined as those with previous CVD, diabetes mellitus (DM), chronic kidney disease (CKD), or a 10-year absolute risk of coronary heart disease (CHD) >20% as calculated by the Framingham equations recommended by the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III).4 Evidence shows that elevated LDL cholesterol (LDL-C) and triglyceride (TG) levels and low HDL cholesterol (HDL-C) levels increase the risk of CVD events in both men and women.5,6 Recent research has shown that high-risk women benefit significantly and to a similar degree as men from lipid-lowering therapy.7 The American Heart Association (AHA) and numerous government and professional organizations have jointly developed evidence-based guidelines to assist with lipid assessment and treatment in high-risk women that take into consideration the quality, quantity, and generalizability of available data to women.8

Abnormal lipid fractions can be managed effectively by both lifestyle and pharmacological interventions. Nonpharmacological interventions, including smoking cessation, increased physical activity, and nutritional therapy, are appropriate for all women with abnormal lipids.4 However, in high-risk women, lifestyle changes alone are often not adequate to achieve optimal lipid levels, and recent data suggest that lipid-lowering therapy may be beneficial, regardless of baseline LDL-C levels.7 To highlight the potential opportu-
Optimal levels of lipids and lipoproteins in women are: LDL-C <100 mg/dL, HDL-C >50 mg/dL, triglycerides <150 mg/dL, and non-HDL-C <130 mg/dL and should be encouraged through lifestyle approaches.

(Diet) In high-risk women: or when LDL-C is elevated, saturated fat intake should be reduced to <7% of calories, cholesterol to <200 mg/d, and trans fatty acid intake should be reduced. (Class I, Level B)* (GI = 1)†

Pharmacotherapy for high-risk women‡

Initiate LDL-C-lowering therapy (preferably a statin) simultaneously with lifestyle therapy in high-risk women with LDL-C >100 mg/dL (Class I, level A)* (GI = 1)† and initiate statin therapy in high-risk women with an LDL-C >100 mg/dL unless contraindicated. (Class I, level B)* (GI = 1)†

Initiate niacin or fibrate therapy when HDL-C is low, or non-HDL-C is elevated in high-risk women. (Class I, level B)* (GI = 1)†

LDL-C indicates LDL cholesterol; HDL-C, HDL cholesterol; and GI, generalizability index.

*Class I, Level A: Intervention is useful and effective, sufficient evidence from multiple randomized trials. Class I, Level B: Intervention is useful and effective, limited evidence from single randomized trial or other nonrandomized studies.

†Generalizability Index 1: Very likely that results generalize to women.

‡High risk in women is defined as CHD or risk equivalent, or 10-year absolute risk >20%.

§Dietary supplement niacin must not be used as a substitute for prescription niacin, and over-the-counter niacin should only be used if approved and monitored by a physician.


In high-risk women, it is important to evaluate benchmark data on goal attainment in high-risk women. The primary objective of this study was to assess the attainment of optimal lipid levels in isolation and in combination and to examine the lipid drug treatment patterns in high-risk women in a managed care setting consistent with the recommendations for lipid management included in the AHA Evidence-Based Guidelines for CVD Prevention in Women (Table 1).

Methods

Data Source

Administrative claims data were obtained from a large southeastern US health plan, which contains approximately 1.1 million patients for whom medical, eligibility, pharmacy, and laboratory results were integrated. Data for these patients were extracted from January 1, 1998, to December 31, 2003, for medical, eligibility, pharmacy, and laboratory records. Laboratory results were provided by a single national laboratory provider. Patient identity was masked throughout in a limited data set format, in accordance with the Health Insurance Portability and Accountability Act.

Study Participants

We performed a historical prospective cohort analysis of administrative claims data and laboratory results. In this study, adult high-risk women (age >18 years) who received a full-panel lipid test between October 1, 1999, and September 30, 2000, were included in the cohort. A full-panel lipid test was defined as the presence of LDL-C, HDL-C, TG, and total cholesterol values for a patient on the same day. The first occurrence of a full-panel lipid test between October 1, 1999, and September 30, 2000, was defined as the “index laboratory date.” High-risk women were defined as those with a history of CVD, DM, and/or CKD on the basis of the AHA guidelines and were identified by the presence of the Internation Classification of Diseases, 9th Revision, Clinical Modification (ICD-9) codes or Current Procedural Terminology, version 4, codes on an inpatient or outpatient claim before the index laboratory date. Women with CVD, DM, and/or CKD were categorized as high-risk women. Women with CVD were identified by the presence of a diagnosis for ischemic heart disease, peripheral vascular disease, cerebrovascular disease (ICD-9: 410.0 to 414, 433.0 to 438, 440 to 441, 443.9). Women with diagnoses for DM (ICD-9: 250) or the presence of prescription claim of a glucose-lowering medication were defined as patients with DM. Finally, diagnosis of CKD was identified on the basis of the presence of a diagnosis claim for CKD (ICD-9: 585). The study sample included only patients who were continuously enrolled in the health plan for a minimum of 12 months before and 12 months after the index laboratory date. The 12-month period before the index laboratory date was referred to as the preindex follow-up, whereas the entire period from the index laboratory date to the end of observation is referred to as the postindex follow-up. To ensure that the patients were lipid-lowering therapy-naïve at the time of the index laboratory claim, patients with a pharmacy claim for lipid-lowering medication in the 6-month period before the index laboratory date were excluded from the analysis.

Lipid Fraction Assessment

Optimal lipid levels were defined as LDL-C <100 mg/dL, HDL-C >50 mg/dL, non–HDL-C <130 mg/dL, and TG <150 mg/dL, on the basis of the AHA guidelines (Table 1). Non–HDL-C was calculated as the difference between total cholesterol and HDL-C levels. Laboratory values were assessed quarterly from index laboratory date and over the follow-up period. After comparing the optimal lipid fraction values with observed lipid values, these results were classified into mutually exclusive groups on the basis of being at optimal value or not at optimal value for each of 3 lipid parameters (LDL-C, HDL-C, TG) alone and in combination.

Lipid-Lowering Therapy

Use of lipid-lowering medication was determined from prescription claims using National Drug Codes for commercially available HMG-CoA reductase inhibitors, bile acid sequestrants, fibrin acid derivatives, nicotinic acid derivatives, and combination products. Lipid-lowering medication was considered to affect a laboratory value if the date of first fill of a lipid-lowering medication preceded the particular full-panel lipid test by at least 28 days.

Outcome Measures

The lipid-level values were evaluated at 90-day intervals. For each quarter, an at-optimum lipid-level status was assigned for each patient on the basis of the corresponding laboratory value. If the women had multiple full-panel lipid tests during a quarter, the last available values were used. For patients lacking a lipid laboratory value during a quarter, we used their laboratory value from the previous quarter, assuming that the value remained static. This approach is also known as the last observation carried forward and it is likely to have minimal impact on our primary and secondary outcome measures. The main outcome measures were attainment of optimal values for LDL-C, HDL-C, TG, non–HDL-C, and combination (LDL-C and HDL-C and TG). Secondary measures included evaluating the association between receipt of lipid-lowering medication with individual and combined lipid fraction target attainment.

Patient Characteristics

Study patients’ ages were obtained from the health plan eligibility file. Because the patients’ observation period was variable, although all patients were followed up for a minimum of 12 months before and after the index date, the duration of preindex and postindex follow-up was ascertained. History of hypertension (ICD-9 CM: 401.90 and prescription fill) was obtained from medical and phar-
TABLE 2. Baseline Characteristics of Study Cohort

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Patients (n=8353)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y (mean±SD)</td>
<td>66±13.9</td>
</tr>
<tr>
<td>Preindex follow-up, mo (mean±SD)</td>
<td>17±3.3</td>
</tr>
<tr>
<td>Postindex follow-up, mo (mean±SD)</td>
<td>27±8.0</td>
</tr>
<tr>
<td>Lipid fraction at index, mean±SD</td>
<td></td>
</tr>
<tr>
<td>LDL-C, mg/dL</td>
<td>133.9±36.3</td>
</tr>
<tr>
<td>HDL-C, mg/dL</td>
<td>55.0±14.9</td>
</tr>
<tr>
<td>TG, mg/dL</td>
<td>152.0±74.2</td>
</tr>
<tr>
<td>Non–HDL-C, mg/dL</td>
<td>164.4±40.6</td>
</tr>
<tr>
<td>Total cholesterol, mg/dL</td>
<td>219.4±40.6</td>
</tr>
<tr>
<td>Significant comorbidities at index, frequency (%)</td>
<td></td>
</tr>
<tr>
<td>CKD</td>
<td>405 (5)</td>
</tr>
<tr>
<td>DM</td>
<td>4177 (50)</td>
</tr>
<tr>
<td>CHD</td>
<td>5058 (61)</td>
</tr>
</tbody>
</table>

CKD indicates chronic kidney disease; DM, diabetes mellitus; CHD, coronary heart disease; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; and TG, triglycerides.

macy claim files. For purposes of this analysis, metabolic syndrome was defined as having either the presence of a medical claim with an ICD-9 code for metabolic syndrome (277.7) or presence of any 3 of the following conditions: codes for obesity, any triglycerides level ≥150 mg/dL, hypertension, fasting blood glucose ≥110 mg/dL, and/or HDL-C <50 mg/dL. The count of distinct prescription medications was determined. A Deyo-Charlson comorbidity score was calculated on the basis of the medical claims in the preindex duration of follow-up in the patients.10

**Statistical Analysis**

Descriptive analyses were used to examine the characteristics of the study sample. In addition, rates of individual and combined optimal lipid value attainment at index and every quarter postindex for the entire duration of observation were evaluated. Univariate logistic regression analyses were conducted to evaluate the odds of individual optimal lipid value attainment over follow-up as predicted by receipt of any lipid-lowering medication. Multivariate analyses consisting of logistic regression analysis were used to examine the odds of combined optimal lipid value attainment over the entire duration of observation. Age, postindex follow-up, Deyo-Charlson comorbidity score, preindex hypertension, preindex DM, preindex CHD, preindex metabolic syndrome, and preindex use of specific pharmacotherapy (such as clopidogrel, β-blockers, and diuretics) were included in the model to assess their impact on goal attainment. The likelihood ratio test was used to compare a model with all possible 2-way interactions and the model without interactions. Because the likelihood ratio test was not significant, we concluded that the interactions did not provide any additional information, and we excluded them from the model. All covariates with insignificant coefficients (P>0.05) were excluded from the model to arrive at the parsimonious model. Model fit was assessed using the Hosmer-Lemeshow goodness-of-fit test; discrimination was measured using the area under the receiver-operated characteristic curve.11 Because inclusion of age as a continuous variable led to a poorly fit model, we categorized age into quartiles to improve model fit. Multiple observations of the same patients and their associated effects on estimates of variance were taken into consideration during hypothesis testing by calculating Huber-White sandwich estimates.12 For all analyses, an a priori 2-tailed level of significance was set at the 0.05 level using SAS software (SAS Institute Inc) version 8.2.

**Results**

**Study Population**

Characteristics of 8353 high-risk women included in the analysis are presented in Table 2. These women were identified with a mean preindex follow-up of 17±3 months and postindex follow-up of 27±8 months. Their mean index LDL-C value was 134±36 mg/dL, and the corresponding non–HDL-C value was 164±41 mg/dL; >50% of the patients were above their target LDL-C and non–HDL-C values at index.

**Optimal Lipid Value Attainment at Index and Over Time**

At index, 17% of women had an LDL-C <100 mg/dL, 19% had a non–HDL-C <130 mg/dL, and 7% attained an LDL-C value ≤100 mg/dL. HDL-C ≥50 mg/dL; ≥100 mg/dL, <130 mg/dL, ≥130 mg/dL; and TG ≤150 mg/dL, >150 mg/dL. Percentage of patients achieving individual and combined optimal lipid values at index and over time. • indicates LDL-C <100 mg/dL; –•, HDL-C ≥50 mg/dL; –•, TG ≤150 mg/dL; –•, non–HDL-C <130 mg/dL; –••, combined LDL-C, HDL-C, and TG.
<100 mg/dL, HDL-C >50 mg/dL, and TG <150 mg/dL (Figure). In addition, during the entire duration of follow-up, attainment of LDL-C and non–HDL-C optimal values exhibited improvement, whereas optimal value attainment in all other lipid fractions either remained static (HDL-C: –1% change) or exhibited only marginal improvement (TG: 3% increase) as shown in the Figure. Transition to combined optimal value attainment was observed to originate from all combined lipid value groups; however, the group not achieving optimal LDL-C but at optimal value for HDL-C and TG accounted for the highest number of transitions (564 of 1265 transitions). A lower proportion of the transitions to the combined optimal lipid value group originated from groups categorized as being at optimal LDL-C compared with those not at optimal LDL-C (23% versus 77%).

Treatment Characteristics

Thirty-two percent of the study cohort received lipiddispersing medication during the entire duration of follow-up. Therapy was initiated in 35% of the women with LDL-C >100 mg/dL and 15% of those with LDL-C <100 mg/dL. Within the subgroup of patients not at combined optimal lipid values at index, only 33% received treatment. Overall, among the patients receiving medication, the mean lag time between index laboratory date and receipt of first prescription fill was 8.9 months. Statin monotherapy was the most commonly prescribed drug in these high-risk women overall (Table 3); however, fibrate therapy was prescribed to the highest percentage of women with LDL-C <100 at index. Statin therapy was initiated in 32% of women with an LDL-C ≥100 mg/dL at index and in 10% of women with an initial LDL-C <100 mg/dL. In addition, medications with a predominant effect on HDL-C and TG, such as niacin or fibrate therapy, were prescribed to 11% of women with an HDL-C <50 mg/dL and/or a non–HDL-C >130 mg/dL.

In subgroups of women with LDL-C >100 mg/dL or TG >150 mg/dL, it was observed that receipt of lipid-lowering medication led to a 4-fold (OR, 4.17; 95% CI, 3.72 to 4.69) and 29% (OR, 1.29; 95% CI, 1.11 to 1.50) increased propensity of achieving these targets, respectively. For women with HDL-C <50 mg/dL, the model exhibited poor fit because of a low number of patients achieving HDL-C >50 mg/dL during the observation period. The likelihood of women having optimal levels of all lipid fractions was strongly associated with receipt of lipid-lowering medication (Table 4). Women receiving any lipid-modifying medication(s) were nearly 4 times more likely to reach combined optimal lipid values during the duration of the study follow-up (OR, 3.60; 95% CI, 3.10 to 4.18), whereas advanced age and diagnosis of metabolic syndrome were associated with a decreased likelihood of attainment.

Discussion

To the best of our knowledge, this is the first study to assess the attainment of the recent lipid benchmarks in high-risk women established by the AHA Evidence-Based Guidelines for Prevention of CVD in Women. Results show that there is substantial opportunity to improve lipid management in high-risk women on the basis of the new guidelines. These data demonstrate the need to develop further dissemination and implementation strategies for the guidelines and serve as baseline data to measure their future impact.

At baseline, attainment of LDL-C <100 mg/dL was low among our high-risk women (17%). Although limited in number, previous studies evaluating LDL-C lipid fraction target attainment have reported similar results. The Third National Health and Nutrition Examination Survey Phase II, conducted from 1991 to 1994, reported that 17.5% of individuals with CHD were at a treatment goal of LDL ≤100 mg/dL. More recently, the Lipid Treatment Assessment Project reported that 18% of CHD patients achieved LDL-C targets (<100 mg/dL).

### TABLE 4. Multivariate Predictors of Combined Optimal Lipid Level Attainment in High-Risk Women

<table>
<thead>
<tr>
<th>Patient Characteristics</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Receipt of lipid-lowering medication</td>
<td>3.60</td>
<td>3.10–4.18</td>
</tr>
<tr>
<td>Age category, y</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;59 (Reference)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>59–69</td>
<td>0.98</td>
<td>0.80–1.21</td>
</tr>
<tr>
<td>70–76</td>
<td>1.21</td>
<td>0.98–1.48</td>
</tr>
<tr>
<td>&gt;77</td>
<td>1.42</td>
<td>1.14–1.76</td>
</tr>
<tr>
<td>Preindex prescription of clopidogrel</td>
<td>0.67</td>
<td>0.46–0.97</td>
</tr>
<tr>
<td>Diagnosis of CHD</td>
<td>1.25</td>
<td>1.03–1.51</td>
</tr>
<tr>
<td>Diagnosis of DM</td>
<td>1.36</td>
<td>1.13–1.64</td>
</tr>
<tr>
<td>Diagnosis of metabolic syndrome</td>
<td>0.40</td>
<td>0.34–0.46</td>
</tr>
</tbody>
</table>

OR indicates odds ratio; CI, confidence interval; CHD, coronary heart disease; and DM, diabetes mellitus.

### TABLE 3. Lipid-Modifying Therapy Initiated During 3-Year Follow-Up

<table>
<thead>
<tr>
<th>Therapy Initiated</th>
<th>Women With LDL-C ≥100 mg/dL (n=6948)</th>
<th>Women With LDL-C &lt;100 mg/dL (n=1405)</th>
<th>All Women (n=8353)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any lipid pharmacotherapy</td>
<td>2432 (35)</td>
<td>209 (15)</td>
<td>2641 (32)</td>
</tr>
<tr>
<td>Statin monotherapy</td>
<td>2230 (92)</td>
<td>134 (64)</td>
<td>2364 (90)</td>
</tr>
<tr>
<td>Prescription niacin monotherapy</td>
<td>28 (1)</td>
<td>5 (2)</td>
<td>33 (1)</td>
</tr>
<tr>
<td>Bile acid sequestrant monotherapy</td>
<td>54 (2)</td>
<td>19 (9)</td>
<td>73 (3)</td>
</tr>
<tr>
<td>Fibric acid monotherapy</td>
<td>109 (4)</td>
<td>51 (24)</td>
<td>160 (6)</td>
</tr>
<tr>
<td>Cholesterol absorption inhibitor monotherapy</td>
<td>2 (&lt;1)</td>
<td>0</td>
<td>2 (&lt;1)</td>
</tr>
<tr>
<td>Any combination therapy</td>
<td>9 (&lt;1)</td>
<td>0</td>
<td>9 (&lt;1)</td>
</tr>
</tbody>
</table>

Values in parentheses are column percentages.
During our study follow-up, the proportion of women attaining an LDL-C <100 mg/dL improved substantially, but still, only approximately one third of high-risk women were at goal at study completion. Conversely, the proportion with HDL-C >50 mg/dL and TG <150 mg/dL was substantially higher at baseline, with little change over 36 months. Overall, combined attainment of optimal values for LDL-C, HDL-C, and TG was exceedingly low at index and increased only marginally over 3 years of follow-up.

Suboptimal lipid value attainment may be the result of multiple factors. In high-risk women, diet and lifestyle changes alone are often not adequate for achievement of optimal lipid levels.\(^8\) Consistent with previous literature, our study reported that only 1 of 3 patients received lipid-lowering medication.\(^15\)–\(^18\) These data suggest that there is a significant treatment gap in high-risk women. Similar to our analyses, previous studies have reported that goal attainment in individual and combined lipid levels are associated with use of lipid-lowering medications.\(^19\)

Although increased pharmacological treatment may improve combined goal attainment in general, it will be more effective if targeted to the specific lipid abnormalities present. Statin monotherapy was the most commonly used pharmacological therapy in this study. Statin monotherapy is effective in altering LDL-C lipid fraction, has a modest effect on TG, and has a minimal effect on HDL-C lipid fraction. In randomized clinical trials, statin monotherapy can lower LDL-C by approximately 25% to 55%, raise HDL-C by 6% to 8%, and lower TG by approximately 10%.\(^4\),\(^7\),\(^20\),\(^21\) Because statin monotherapy was the predominant strategy used in the cohort studied, it follows that there were observed increases in the proportion of women achieving LDL <100 mg/dL and that the bulk of the transitions over time into the category of all optimal lipid values being reached came from those with LDL >100 mg/dL, HDL >50 mg/dL, and TG <150 mg/dL. Only 11% of women meeting the recommendation of the AHA guidelines to use either niacin or fibrate therapy in high-risk women with HDL-C <50 mg/dL and/or non–HDL-C >130 mg/dL received such therapy.

The purpose of this study was to provide benchmark data for opportunities to intervene in high-risk women and should not be interpreted as report card data for adherence to the AHA Evidence-Based Guidelines for CVD Prevention in Women.\(^8\) However, it should be noted that the standard of care at the outset of this study was the NCEP ATP II, which stated that the target level of LDL-C was ≤100 mg/dL for high-risk patients, and the AHA Guide to Preventive Cardiology for Women, published in 1999, also recommended an LDL-C goal ≤100 mg/dL for women with CVD, suggesting that adherence to national standards has been consistently poor.\(^22\),\(^23\)

**Limitations**

Every attempt was made in this analysis to produce unbiased estimates; however, there are limitations that should be considered during interpretation of our data. This study was restricted to data from a southeastern US managed-care database. The database did not have any information on sociodemographic factors such as race, education, or income, which may be related to outcomes. The study results may not be generalizable to settings outside the managed care organization. Moreover, because this was an observational study, our results could potentially be biased because of nonrandom selection of patients and/or nonrandom patient dropout from the study. Because high-risk women with at least 1 full lipid panel were selected nonrandomly, the estimates produced in this study are potentially conservative estimates of individual and combined optimal lipid value attainment; it is likely that women who do not undergo lipid fraction assessment may remain untreated, leading to lower target attainment. However, the baseline lipid values and the proportion of women achieving LDL-C targets in this study are similar to those reported in other national data sets; hence, the estimates from this study appear robust.\(^13\),\(^14\)

**Conclusions**

The findings from this analysis suggest that, on the basis of the recently published AHA Evidence-based Guidelines for CVD Prevention in Women, there is a substantial opportunity to improve lipid management and reduce morbidity and mortality caused by CVD among high-risk women. Health care providers, systems, and patients may jointly develop strategies to promote adherence to the AHA prevention guidelines and thus improve quality outcomes in women.

**Acknowledgments**

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**References**


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