INTRODUCTION

While the benefit of statins for reducing CV morbidity and mortality in patients with dyslipidaemia is well established, some patients develop SI due to adverse effects (AEs) including muscle aches and weakness, gastrointestinal (GI) symptoms, liver enzyme abnormalities, or other non-specific complaints.1 SII is reported in 5%-10% of subjects in randomized, placebo-controlled trials, and up to 20% in observational studies.1 In patients with dyslipidaemia, SI can lead to statin discontinuation or dose reduction sufficient to prevent patients from reaching target low-density lipoprotein cholesterol (LDL-C) levels. In patients experiencing SI who switch to different statins, symptoms of SI may recur.7

Physician fear of myopathy and other statin-related AEs can also lead to unjustified termination of a statin or use at insufficient doses, depriving patients at high risk for CV events of demonstrated clinical benefits.2 In Canada, comprehensive guidelines on management of SI were published by the Canadian Working Group on Cardiovascular Conference in 2011, and updated in 2013; these recommend trying a different statin, or lower or intermittent statin dosing rather than discontinuation as the main method to manage SI.

Credence, the management of SI in Canadian clinical practice is poorly understood.

Objective

To describe the characteristics and management of SI in patients with dyslipidaemia treated in real-world clinical practice in Ontario, focusing on patients at highest risk for CV events.

METHODS

Study design

This was a non-interventional, observational, retrospective, longitudinal, healthcare database study.

Data source

Analyses were conducted using data from the proprietary Southwestern Ontario (SWO) Primary Care Practice Database (Individual Health Outcomes, Inc.), which contains longitudinal healthcare resource utilization and outcomes data for >33,000 adult patients, contributed by >100 participating physicians at >70 primary practices. All patient records in the SWO database are anonymized to conform to current confidentiality regulations.

Patient selection

The study sample was an open cohort of adult patients diagnosed with hyperglycaemia and initiating statin therapy (i.e., statin index date) between January 1, 2004 and December 31, 2010. Inclusion and exclusion criteria are listed in Table 1. Patients were stratified into three CV risk levels (Table 2).

Outcomes of interest

This presentation focuses on the occurrence and management of SI in the CV high-risk patient group.

RESULTS

Patient characteristics

A total of 41,733 patients who initiated statin therapy were included, of whom 14,607 (35%) were high-risk patients and 12,94 (9% of high-risk patients) had SI.

Patient characteristics at the SI index date are shown in Table 3. At baseline in the high-risk group, the mean (SD) age was 61 (8.5) years, 53% were male, and mean (SD) LDL-C was 2.6 (1.1) mmol/L.

Table 3. Patient characteristics at the statin intolerance index date.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>High-risk patients with SI (N=1294)</th>
<th>All patients (N=41,733)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y), mean (SD)</td>
<td>61 (8.9)</td>
<td>61 (8.5)</td>
</tr>
<tr>
<td>Gender, % male (n male)</td>
<td>53% (684)</td>
<td>52% (36,843)</td>
</tr>
<tr>
<td>LDL-C (mmol/L), mean (SD)</td>
<td>2.78 (1.1)</td>
<td>2.87 (1.1)</td>
</tr>
<tr>
<td>TC/HDL-C, mean (SD)</td>
<td>4.5 (1.0)</td>
<td>4.6 (1.0)</td>
</tr>
<tr>
<td>SBP, mean (SD)</td>
<td>138 (9)</td>
<td>139 (9)</td>
</tr>
<tr>
<td>DBP, mean (SD)</td>
<td>84 (6)</td>
<td>84 (6)</td>
</tr>
</tbody>
</table>

CV risk factors during prior 1-year period, %

Diabetes                        14% (175)
Diabetes + age ≥40 years         8% (105)
Diabetes for >18 years + age ≥30 years 5% (65)
Diabetes + microvascular disease <1% (5)
History of CVD                   10% (128)
Current smokers                 4% (48)
Smoking history                  5% (65)
Male <45 years; n (%)           94% (646)
Female <65 years; n (%)          96% (885)
Family history of premature CVD 9% (114)
Family history of hyperlipidaemia 6% (78)
High-risk hypertension          25% (333)
CKD                             5% (71)
Clinical evidence of atherosclerosis or abdominal aneurysm 5% (68)
Obesity (BMI ≥27 kg/m²)          39% (502)

CONCLUSIONS

SI affects the optimal treatment of dyslipidaemia in this high-risk population, with half of patients with SI not achieving their LDL-C target.