Statin Tolerability: Overcoming the Treatment Gap
IAS-OSLA Course Lipid Metabolism and Cardiovascular Risk
Oman February 9th, 2015

Peter J. Lansberg
Center for Translational Molecular Medicine
Dept. of Vascular Medicine
AMC - Amsterdam Netherlands
'I didn't have time to write a short letter, so I wrote a long one instead'

Mark Twain
History of Cholesterol Lowering

- **1900 – 1920**: Eureka!
- **1920 – 1960**: Nothing
- **1960 – 1970**: Drama
- **1970 – 1980**: Hope
- **1980 – 1990**: Surprise
- **1990 – 2000**: Confidence
- **2000 – 2010**: Inertia – Skepticism
- **2010 – 2020**: ?
Scientific Data

January 31\textsuperscript{th} 2015 PubMed:

- Cholesterol : 232 127
- Chol. Low. Tx : 92 572
- Statin : 34 493

24 Cardiovascular risk guidelines
Scientific Data

Publications in 2014 PubMed:

Cholesterol: 11,345 (31/day)!
Chol. Low. Tx: 4,566 (12/day)!
Statin: 2,467 (7/day)!

24 Cardiovascular risk guidelines
Outline Presentation

1. Statin Safety
2. Statin Tolerability
3. Statin Adherence
Adherence to Statins

- 2234 NSTEMI patients discharged Ator 80 mg
- 12 month follow up
  - Tel. interview in months 1, 6 and 12
- 27.3% of patients stopped after 35 days (21-79)
  - Mild side effects 48%
  - No specific reason 52%
- ↑ risk for discontinuation: elderly and females
- ↓ risk for discontinuation: Diabetics and post PCI

Mortality: 203 patients (9%)
HR: 4.1 (2.1-7.9) P=0.002
Are all statins the same?
LOVASTATIN

SIMVASTATIN

PRAVASTATIN

FLUVASTATIN

ATORVASTATIN

ROSUVASTATIN

PITAVASTATIN
Are all patients the same?
Atherosclerosis
Risk Factors
Are Statins Safe?
### Statin Safety in Perspective

Number needed to treat for 1 year to:

<table>
<thead>
<tr>
<th></th>
<th>Cause a GI Bleed</th>
<th>Cause a Fatal GI Bleed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>248</td>
<td>2066</td>
</tr>
<tr>
<td>Statins</td>
<td>100,000</td>
<td>1,000,000</td>
</tr>
</tbody>
</table>

1. Derry S, Loke YK. 2000
Rhabdomyolysis

In Summary…The Risk is Small!

73 reported cases of fatal rhabdomyolysis

484,273,000 Statin prescriptions

2014: 15 – 20 million statin users

2014 estimation: 3 deaths/year

US population: ± 300 million
Peanut allergy 1.4% of population:
4.2 million affected → 150 fatalities/yr!
Question: Can patients tolerate statins?
Muscle Related Side Effects
A systematic review of statin–induced muscle problems in clinical trials

statin treatment: 12.7% - placebo group: 12.4% (P = .06)
### Musculoskeletal Adverse Events With the 80-mg Dose

<table>
<thead>
<tr>
<th>Reported AE</th>
<th>2006 Safety Analysis</th>
<th>PROVE IT*</th>
<th>IDEAL*</th>
<th>TNT*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Atv 10 (n=7258)</td>
<td>Atv 80 (n=4798)</td>
<td>Atv 80 (n=2063)</td>
<td>Atv 80 (n=2099)</td>
</tr>
<tr>
<td>Myalgia</td>
<td>2.9%</td>
<td>2.7%</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Myopathy</td>
<td>0</td>
<td>0.02%†</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Persistent CPK &gt;10 x ULN</td>
<td>0</td>
<td>0.06%</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td>Rhabdomyolysis</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

NR, not reported

*Studies not included in 2006 safety meta-analysis

†Investigator-reported cases: did not meet criteria for definition of myopathy (persistent CPK elevations >10 x ULN with muscle symptoms)

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Statin Associated Muscle Symptoms SAMS

MYALGIA  →  Pain

MYOPATHY  →  Weakness

MYOSITIS  →  CK > 10X ULN

RHABDOMYOLYSIS  →  CK > 40X ULN
Statin Myalgia Clinical Index Score

- **Probable**
  - 9-11

- **Possible**
  - 7-8

- **Unlikely**
  - <7

**Region**
- Symmetric hip flexors/thigh aches: 3
- Symmetric calf aches: 2
- Symmetric upper proximal myalgia: 2
- Non-specific asymmetric, intermittent: 1

**Time**
- Symptoms onset <4 weeks: 3
- Symptoms onset 4-12 weeks: 2
- Symptoms onset >12 weeks: 1

**De-challenge**
- Improves upon withdrawal (<2 weeks): 2
- Improves upon withdrawal (2-4 weeks): 1
- Does not improve upon withdrawal (>4 weeks): 0

**Challenge**
- Same symptoms reoccur upon rechallenge <4 weeks: 3
- Same symptoms reoccur upon rechallenge 4-12 weeks: 1

**An assessment by the Statin Muscle Safety Task Force: 2014 update**

Robert S. Rosenson, MD, FNLA*, Steven K. Baker, MSc, MD, FRCP(C),
Terry A. Jacobson, MD, FNLA, Stephen L. Kopecky, MD, Beth A. Parker, PhD
STOMP Study

6 months 420 healthy statin naïve subjects Atorvastatin 80 mg vs Placebo

Effect of Statins on Skeletal Muscle Function

Beth A. Parker, PhD; Jeffrey A. Capizzi, MS; Adam S. Grimaldi, BS; Priscilla M. Clarkson, PhD; Stephanie M. Cole, PhD; Justin Keadle, BS; Stuart Chipkin, MD; Linda S. Pescatello, PhD; Kathleen Simpson, MS; C. Michael Ylikorkala, MD; Dr. Paul D. Thompson, MD

Background—Many clinicians believe that statins cause muscle pain, but this has not been observed in clinical trials, and this effect of statins on muscle performance has not been carefully studied.

Methods and Results—In a double-blind, randomized, placebo-controlled trial of 420 healthy, statin-naïve subjects, Atorvastatin 80 mg or placebo was administered for 6 months to 420 healthy, statin-naïve subjects. No individual creatine kinase value exceeded 10 times normal, but mean creatine kinase increased 20.8 ± 141.1 U/L (P=0.01) with atorvastatin. There were no differences in muscle weakness and muscle pain or exercise capacity of atorvastatin 80 mg dose more than placebo, but more atorvastatin than placebo subjects developed myalgia (19 versus 10; P=0.05). Myalgic subjects on atorvastatin or placebo had decreased muscle strength in 3 of 14 and 4 of 14 variables, respectively (P=0.69).

Conclusions—These results indicate that high-dose atorvastatin for 6 months does not decrease average muscle strength or exercise performance in healthy, previously untreated subjects. Nevertheless, this blinded, controlled trial confirms the undocumented impression that statins increase muscle complaints. Atorvastatin also increased average creatine kinase, suggesting that statins produce mild muscle injury even among asymptomatic subjects. This increase in creatine kinase should prompt studies examining the effects of more prolonged, high-dose statin treatment on muscular performance.


Key Words: atorvastatin  ■ exercise test  ■ hydroxymethylglutaryl-CoA reductase inhibitors  ■ muscle strength  ■ myopathy
The Effect of Statins on Skeletal Muscle Function and Performance (STOMP) study

<table>
<thead>
<tr>
<th>atorvastatin 80 mg VS placebo 6 MONTHS n=420</th>
</tr>
</thead>
<tbody>
<tr>
<td>• exercise capacity → No significant changes</td>
</tr>
<tr>
<td>• muscle strength → No significant changes</td>
</tr>
<tr>
<td>• Myalgia                                   → 19 vs 10 (p=0.05)</td>
</tr>
<tr>
<td>• CK ≥10 x ULN                             → none</td>
</tr>
</tbody>
</table>

Myalgic subjects on atorvastatin or placebo had decreased muscle strength in 5 of 14 and 4 of 14 variables, respectively (P=0.69).

Circulation. 2013;127:96-103
The Effect of Statins on Skeletal Muscle Function and Performance (STOMP) study

Circulation. 2013;127:96-103
Discontinuation of Statins in Routine Care Settings
A Cohort Study

Huabing Zhang, MD; Jorge Plutzky, MD; Stephen Skentzos, BA, BS; Fritha Morrison, MPH; Perry Mar, PhD; Maria Shubina, ScD; and Alexander Turchin, MD, MS

Background: Systematic data on discontinuation of statins in routine practice of medicine are limited.

Objective: To investigate the reasons for statin discontinuation and the role of statin-related events (clinical events or symptoms believed to have been caused by statins) in routine care settings.

Design: A retrospective cohort study.

Setting: Practices affiliated with Brigham and Women’s Hospital and Massachusetts General Hospital in Boston.

Patients: Adults who received a statin prescription between 1 January 2000 and 31 December 2008.

Measurements: Information on reasons for statin discontinuations was obtained from a combination of structured electronic medical record entries and analysis of electronic provider notes by validated software.

Results: Statins were discontinued at least temporarily for 57,292 of 107,835 patients. Statin-related events were documented for 18,778 (17.4%) patients. Of these, 11,124 had statins discontinued at least temporarily; 6,579 were rechallenged with a statin over the subsequent 12 months. Most patients who were rechallenged (92.2%) were still taking a statin 12 months after the statin-related event. Among the 2,721 patients who were rechallenged with the same statin to which they had a statin-related event, 1,295 were receiving the same statin 12 months later, and 996 of them were receiving the same or a higher dose.

Limitations: Statin discontinuations and statin-related events were assessed in practices affiliated with 2 academic medical centers. Utilization of secondary data could have led to missing or misinterpreted data. Natural-language-processing tools used to compensate for the low (30%) proportion of reasons for statin discontinuation documented in structured electronic medical record fields are not perfectly accurate.

Conclusion: Statin-related events are commonly reported and often lead to statin discontinuation. However, most patients who are rechallenged can tolerate statins long-term. This suggests that many of the statin-related events may have other causes, are tolerable, or may be specific to individual statins rather than the entire drug class.

Primary Funding Source: National Library of Medicine, Diabetes Action Research and Education Foundation, and Chinese National Key Program of Clinical Science.
Discontinuation of Statins in Routine Care

Statin use 2000 - 2008
Brigham's And Women's hospital Boston

- 107,835 patients
- 57,292 discontinued
- 18,778 documented events
Discontinuation of Statins in Routine Care

Statin use 2000 - 2008
Brigham's And Women's hospital Boston

Re-challenge in 6,579 patients
93% were on statin after 12 months
Lesson Learned
### SEARCH: Myopathy rates by SIMVASTATIN comparison

<table>
<thead>
<tr>
<th>Simvastatin allocation (per 1000 person-years)</th>
<th>80 mg</th>
<th>20 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-1 follow-up</td>
<td>25 (4.2)</td>
<td>1 (0.2)</td>
</tr>
<tr>
<td>2-7 follow-up</td>
<td>28 (0.8)</td>
<td>2 (0.1)</td>
</tr>
<tr>
<td>Total</td>
<td>53</td>
<td>3</td>
</tr>
</tbody>
</table>

Myopathy: New, unexplained muscle pain or weakness plus CK > 10x ULN (7 vs 0 developed rhabdomyolysis)
FDA Warning March 2010 + May 2011

- No Simvastatin with *itraconazole*, *ketoconazole*, *erythromycin*, *clarithromycin*, *telithromycin*, *HIV protease inhibitors*, or *nefazodone*.
- No simvastatin >10 mg with *gemfibrozil*, *cyclosporine*, or *danazol*
- No simvastatin >20 mg with *amiodarone* or *verapamil*
- No simvastatin >40 mg with *diltiazem*
- Patients of **Chinese descent** should not receive **simvastatin 80 mg** with cholesterol-modifying doses of *niacin*-containing products.
- caution when such patients are treated with **simvastatin 40 mg** or less in combination with cholesterol-modifying doses of *niacin*-containing products.
Risk factors for statin-associated muscle symptoms.

- Anthropometric
- Concurrent conditions
- Surgery
- Related history
- Genetics
- Other risk factors
United Network for Organ Sharing (UNOS) Database

- From 1990 – 2002 in the US: 51,741 liver transplants
- three cases of acute liver failure related to statins *
  - 2 → Cerivastatin
  - 1 → Simvastatin

These studies were retrospective analyses, and formal causality assessment of DILI and confirmation that cases were bona-fide cases of DILI from statins were not performed!

Safety and efficacy of long-term statin treatment for cardiovascular events in patients with coronary heart disease and abnormal liver tests in the Greek Atorvastatin and Coronary Heart Disease Evaluation (GREACE) Study: a post-hoc analysis

Vassilis G Androulakis, Konstantinos Tsivrikos, Thomas D Goniakas, Theodore Griva, Panagiota Anerginou, Konstantinos Kastritis, Efstratios D Pagoulatos, Ilir Theodoridou, Antonis Kougianos, Dimitris T Makris, for the GREACE Study Collaborative Group

Summary

Background Long-term statin treatment reduces the frequency of cardiovascular events, but safety and efficacy in patients with abnormal liver tests is unclear. We assessed whether statin therapy is safe and effective for these patients through post-hoc analysis of the Greek Atorvastatin and Coronary Heart Disease Evaluation (GREACE) study population.

Methods GREACE was a prospective, intention-to-treat study that randomly assigned by a computer-generated randomisation list 1600 patients with coronary heart disease (aged < 75 years, with serum concentrations of LDL cholesterol > 2.5 mmol/L and triglycerides < 3.5 mmol/L) at the Hippokration University Hospital, Thessaloniki, Greece to receive statin or usual care, which could include statins. The primary outcome of our post-hoc analysis was risk reduction for first recurrent cardiovascular event in patients treated with a statin who had moderately abnormal liver tests (defined as serum alanine aminotransferase or aspartate aminotransferase concentrations of less than three times the upper limit of normal) compared with patients with abnormal liver tests who did not receive a statin. This risk reduction was compared with that for patients treated (or not) with statin and normal liver tests.

Findings Of 437 patients with moderately abnormal liver tests at baseline, which were possibly associated with non-alcoholic fatty liver disease, 227 who were treated with a statin (mainly atorvastatin 24 mg per day) had substantial improvement in liver tests (p = 0.001) whereas 210 not treated with a statin had further increases of liver enzyme concentrations. Cardiovascular events occurred in 22 (10%) of 227 patients with abnormal liver tests who received statin (3.5 events per 100 patient-years) and 65 (39%) of 210 patients with abnormal liver tests who did not receive statin (10.9 events per 100 patient-years): 68% relative risk reduction, p = 0.001. This cardiovascular disease benefit was greater (p = 0.007) than it was in patients with normal liver tests (90 [54%] events in 653 patients receiving a statin [4.6 per 100 patient-years] vs 117 [23%] in 519 patients not receiving a statin [7.6 per 100 patient-years]; 38% relative risk reduction, p = 0.001). Seven (1%) of 580 participants who received a statin discontinued statin treatment because of liver-related adverse events (transaminase concentrations more than three times the upper limit of normal).

Interpretation Statin treatment is safe and can improve liver tests and reduce cardiovascular morbidity in patients with mild-to-moderately abnormal liver tests that are potentially attributable to non-alcoholic fatty liver disease.

Funding None.

Lancet 2010; 367: 1916-1922
Enzyme activity during 3-year follow-up in patients with raised liver enzymes

Cardiovascular Events in Patients with Normal and Abnormal LFT’s

NAFLD at Baseline and Follow up

Risk reduction of moderate to severe hepatic steatosis: 70%!

Am J Gastroenterol advance online publication, 14 September 2010; doi: 10.1038/ajg.2010.299
Use of Atorvastatin or Pravastatin in Patients with NASH

- 5 patients with NASH 20 mg Pravastatin for 6 months\(^1\)
  - Normalization of liver enzymes in all patients
  - Some improvement in hepatic inflammation and steatosis (liver biopsies)

- 7 patients with NASH 20 mg Atorvastatin 21 months (± 2 months)\(^2\)
  - Well tolerated
  - No increase in liver enzymes
  - Significant improvement of hepatic histology in some patients

Summary of studies using statin therapy for NAFLD

<table>
<thead>
<tr>
<th>References</th>
<th>N</th>
<th>Study design</th>
<th>Diagnosis</th>
<th>Therapy</th>
<th>Dose</th>
<th>Duration (months)</th>
<th>Aminotransferase improvement</th>
<th>Histological improvement</th>
<th>Imaging</th>
</tr>
</thead>
<tbody>
<tr>
<td>Horlander et al. (32)</td>
<td>7</td>
<td>Open label</td>
<td>NASH</td>
<td>Atorvastatin</td>
<td>Varied</td>
<td>12</td>
<td>Improved</td>
<td>Improved IN, S, and F</td>
<td>N/A</td>
</tr>
<tr>
<td>Kiyici et al. (14)</td>
<td>27</td>
<td>Open label</td>
<td>NASH</td>
<td>Atorvastatin</td>
<td>10 mg</td>
<td>6</td>
<td>Improved</td>
<td>N/A</td>
<td>CT improved</td>
</tr>
<tr>
<td>Rallidis et al. (33)</td>
<td>5</td>
<td>Open label</td>
<td>NASH</td>
<td>Pravastatin</td>
<td>20 mg</td>
<td>6</td>
<td>Improved</td>
<td>Improved IN, S, and F, no change in F</td>
<td>N/A</td>
</tr>
<tr>
<td>Hatzitolios et al. (34)</td>
<td>28</td>
<td>Open label</td>
<td>NAFLD</td>
<td>Atorvastatin</td>
<td>20 mg</td>
<td>6</td>
<td>Improved</td>
<td>N/A</td>
<td>US: improved</td>
</tr>
<tr>
<td>Gomez-Dominguez et al. (35)</td>
<td>22</td>
<td>Open label</td>
<td>NAFLD</td>
<td>Atorvastatin</td>
<td>Varied</td>
<td>12</td>
<td>Improved</td>
<td>N/A</td>
<td>US: no change</td>
</tr>
<tr>
<td>Antonopoulos et al. (36)</td>
<td>23</td>
<td>Open label</td>
<td>NAFLD</td>
<td>Rosuvastatin</td>
<td>10 mg</td>
<td>8</td>
<td>Improved</td>
<td>N/A</td>
<td>N/A</td>
</tr>
</tbody>
</table>

F, fibrosis; CT, computed tomography; IN, inflammation; N/A, not applicable; NAFLD, nonalcoholic fatty liver disease; NASH, nonalcoholic steato hepatitis; RCT, randomized control trial; S, steatosis; US, ultrasound.
Statins and Liver Disease

- Decrease the risk of developing hepatocellular carcinoma (HCC) \(^1\)
- Improve portal pressure and risk for bleeding liver cirrhosis\(^2\)
- Double survival in HCC patients\(^3\)
- Inhibit hepatitis C viral replication and improve treatment response rates\(^4,5\)

Original Contribution

An assessment by the Statin Liver Safety Task Force: 2014 update

Harold Bays, MD, FNLA*, David E. Cohen, MD, PhD, Naga Chalasani, MBBS, Stephen A. Harrison, MD, COL, MC

Louisville Metabolic and Atherosclerosis Research Center, 3288 Illinois Avenue, Louisville, KY 40213, USA (Dr Bays); Brigham and Women’s Hospital, Harvard Medical School, Boston, MA, USA (Dr Cohen); Indiana University School of Medicine, Indianapolis, IN, USA (Dr Chalasani); and Brooke Army Medical Center, University of Texas Health Center San Antonio, Uniformed Services University of the Health Sciences, San Antonio, TX, USA (Dr Harrison)

KEYWORDS:
Cholesterol;
National Lipid Association;
Lipid;
Liver;
Statin

Abstract: In the 2006 Report of the National Lipid Association’s Statin Safety Task Force, a panel of experts in hepatology published their findings on specific questions related to the liver blood testing during statin therapy. Among their recommendations was that regulatory agencies reconsider the statin-labeling recommendation at that time, which required post-statin liver enzyme testing. Since then, the Food and Drug Administration altered statin labeling such that unless clinically indicated for other reasons, after a pre-statin therapy baseline evaluation, follow-up liver enzyme testing was not uniformly required after statin initiation. This 2014 report provides an update on interim issues relevant to statins and liver safety. Some of the points discussed include the value of baseline liver enzymes before initiating statin therapy, safety of statin use in patients with nonalcoholic fatty liver disease, potential drug interactions between statins and drugs used to treat hepatitis, the use of statins in liver transplant recipients, and the use of statins in patients with autoimmune liver disease. Finally, this panel provides diagnostic and algorithmic approaches when evaluating statin-treated patients who experience elevations in liver enzymes.

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1. Comprehensive List of Causes for Elevated Liver Enzymes

2. Comprehensive approach to patients with elevated liver blood testing (transaminases, 3 times the upper limits of normal)
Diabetes and Statins
Statins and risk of incident diabetes: a collaborative meta-analysis of randomised statin trials

13 trials 91 140 participants
DM in 4278 (2226 statins/2052 controls)

- 9% ↑ risk for incident diabetes (95% CI 1.02–1.17)
- ↑ risk in older participants
- = baseline body-mass index and change in LDL-cholesterol concentrations

Treatment of 255 (95% CI 150–852) patients with statins for 4 years resulted in one extra case of diabetes.
Statins and risk of incident diabetes: a collaborative meta-analysis of randomised statin trials

Lancet 2010; 375: 735–42
Risk of New-Onset DM2 According to Number of Risk Factors at Baseline

1. Baseline glucose >5.6 mmol/l (100 mg/dl)
2. Fasting triglycerides >1.7 mmol/l (150 mg/dl)
3. BMI >30 kg/m2
4. History of hypertension

J Am Coll Cardiol 2011;57:1535–45
Effect of Pre-Diabetes on Incidence of New-Onset Diabetes (TNT & IDEAL)

![Graph showing the effect of Pre-Diabetes on Incidence of New-Onset Diabetes](image)

- Treatment HR (95% CI) = 1.20 (1.04 to 1.37) p=0.010 (Pre-Diabetic)
- Treatment HR (95% CI) = 1.08 (0.85 to 1.38) p=0.527 (Not Pre-Diabetic)
Diabetes Risk Assessment Form

Circle Answers and add up the points

Age
- 0p. Under 45 years
- 2p. 45-54 years
- 3p. 55-64 years
- 4p. Over 64 years

Body Mass/Waist
- 0p. Lower than 25 kg/m²
- 1p. 25-30 kg/m²
- 2p. Higher than 30 kg/m²

Diet / Exercise
- 0p. No
- 2p. Yes
- 3p. Yes: grandparent, aunt, uncle or first cousin (but no own parent, own child or sibling)

Anti medication
- 0p. No
- 2p. Yes

History of elevated glucose
- 0p. No
- 2p. Yes

Family history
- 0p. No
- 2p. Yes

Total risk score
- Lower than 7 Low: estimated 1 in 100 will develop disease
- 7-11 Slightly elevated: estimated 1 in 25 will develop disease
- 12-14 Moderate: estimated 1 in 6 will develop disease
- 15-20 High: estimated 1 in 3 will develop disease
- Higher Very high: estimated 1 in 2 will develop disease

N.A. Sattar et al. / Atherosclerosis Supplements 15 (2014) 1e15
Major Cardiovascular Events

Event rate was almost the same patients with new onset diabetes compared to patients without diabetes at baseline

HR: 1.02, 95% CI: 0.77 to 1.35
Statin Induced Impairment of Glucose Metabolism

- Inhibition of glucose-induced calcium signaling-dependent insulin secretion
- Decrease Ubiquinone and Mitochondrial transport/ATP production/Reduced Insulin secretion
- Inhibition of the Isoprenylation of GLUT4 transporter
- Beta cell oxidation, oxidative stress and nitric oxide production
- Apoptosis and beta cell destruction from LDL entry

Other Drugs That Affect Glucose

- Atypical antipsychotics
- β-blockers
- Thiazide diuretics
- Protease inhibitors
- Niacin
Statin Effects 9:1 Benefit?

- Of 255 Patients Treated
  +1 case of diabetes

- Of 255 Patients Treated
  -9 cases of CAD/CVA

Lancet 2010; 375: 735–42
Helping your patients stick to their therapy!

Statin Adherence
Patient Adherence Toolkit

The Patient Adherence subcommittee of the NLA developed a patient toolkit to address the importance of patient adherence to medications and lifestyle modifications.

Download the Patient Adherence Toolkit as PDF

The purpose of the patient adherence toolkit is to be a resource to the clinician on how to counsel their patients. The toolkit will challenge the clinician to view adherence like a disease, which requires an understanding of what adherence is, how to “diagnose” it in patients, and what interventions have been successful at improving patient adherence.

Please share your thoughts on this Adherence Toolkit in the comments section below. Was this toolkit helpful to you and your practice?

https://www.lipid.org/practicetools/tools/adherence
<table>
<thead>
<tr>
<th>Interventions to Improve Adherence</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>S</strong> Simplify the regimen</td>
</tr>
<tr>
<td>* Adjust timing, frequency, and dosage</td>
</tr>
<tr>
<td>* Utilize once-daily medications whenever possible</td>
</tr>
<tr>
<td>* Encourage the use of adherence aids (e.g., pillboxes, cell phone alarms)</td>
</tr>
<tr>
<td>* Consider each patient’s activities of daily living (e.g., swing shift workers)</td>
</tr>
<tr>
<td><strong>I</strong> Impart knowledge</td>
</tr>
<tr>
<td>* Patient-provider shared decision making</td>
</tr>
<tr>
<td>* Provide clear instructions and expectations for all prescriptions</td>
</tr>
<tr>
<td>* Involve relatives or caregivers when discussing medications</td>
</tr>
<tr>
<td>* Recommend electronic education formats (e.g., video, websites)</td>
</tr>
<tr>
<td><strong>M</strong> Modify patient beliefs and human behavior</td>
</tr>
<tr>
<td>* Ask patient about their needs and what might help them adhere to therapy</td>
</tr>
<tr>
<td>* Ensure patient understands consequences of non-adherence</td>
</tr>
<tr>
<td>* Addressed perceived barriers of taking the medication</td>
</tr>
<tr>
<td>* Provide rewards for adherence (e.g., praise, coupons, fewer clinic visits)</td>
</tr>
<tr>
<td><strong>P</strong> Provide communication and trust</td>
</tr>
<tr>
<td>* Practice to improve interviewing skills</td>
</tr>
<tr>
<td>* Embrace active listening and provide emotional support</td>
</tr>
<tr>
<td>* Elicit patient’s input when discussing treatment options</td>
</tr>
<tr>
<td>* Allow adequate time for the interaction and encourage patient to ask questions</td>
</tr>
<tr>
<td><strong>L</strong> Leave the bias</td>
</tr>
<tr>
<td>* Foster a greater understanding of health literacy and how it affects patients</td>
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<tr>
<td>* Ensure communication style is patient-centered</td>
</tr>
<tr>
<td>* Take extra time to understand and overcome cultural barriers</td>
</tr>
<tr>
<td>* Tailor education to the patient’s level of understanding</td>
</tr>
<tr>
<td><strong>E</strong> Evaluating adherence</td>
</tr>
<tr>
<td>* Ask patients simply and directly about adherence</td>
</tr>
<tr>
<td>* Engage patients about adherence at every encounter</td>
</tr>
<tr>
<td>* Measure drug levels or efficacy parameters, when applicable</td>
</tr>
<tr>
<td>* Review medication containers, noting last fill date and remaining medicine</td>
</tr>
</tbody>
</table>
How to make people follow our advice
CHANGE Behaviour

1. Own the problem
2. Measure the problem
3. Feed back
4. Trust - Time
5. Transparency
6. Repeat

“What if we don’t change at all ... and something magical just happens?”
Beyond Belief — How People Feel about Taking Medications for Heart Disease

Lisa Rosenbaum, M.D.
Summary of Presentation

1. Statin Safety
2. Statin Tolerability
3. Statin Adherence
Questions?

Lansberg@gmail.com