Management of lipids after a cardiovascular event

Welcome to the sixth in a series of webinars as part of the national education programme Tackling Cholesterol Together.

Delivered in partnership by The NHS Accelerated Access Collaborative (AAC), The AHSN Network and the cholesterol charity, HEART UK

The webinar will start at 12 noon

November 2021

All programme content, recordings and next webinar and clinic bookings will be housed in the HEART UK pages. Visit the site for the new e-Learning modules on diet, launching late November https://www.heartuk.org.uk/tackling-cholesterol-together/home
This meeting will be recorded and will be made available in the HEART UK Tackling Cholesterol Together pages.

There will be time to stop and ask questions at the end of the webinar.

Feel free to ask questions or upvote questions in the chat function when it becomes available.

Any questions that we are not able to cover in the Q&A sections today will be addressed following the event.

Any questions you provided during registration will be covered during the session.
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Objectives of today’s webinar

01 Understand the scale of CVD in the UK. Review evidence on the influence of LDL-C on CVD risk and the impact of intensive lipid lowering therapy.

02 After a cardiovascular event, identify the increased risk for another cardiovascular event over time and according to individual age and risk category.

03 Consider the NICE CG181 and ESC targets approach to managing cholesterol in secondary prevention. Review the barriers and find opportunities to optimise, including a review of statin reluctance and intolerance.

04 Referring to NICE CG147, appreciate the clinical presentation of Peripheral Artery Disease as a high risk for cardiovascular events (and the indication for a secondary prevention approach).
Influence of LDL-C on risk after a cardiovascular event

Stephen Wheatcroft
Professor of Cardiometabolic Medicine / Consultant Cardiologist
Cardiovascular disease in the UK - the scale of the problem

Cardiovascular diseases cause a quarter of all deaths in the UK;

>160 000 deaths each year;

~460 deaths each day; one every three minutes

7.6 million people in the UK are living with cardiovascular disease

>100 000 hospital admissions each year with heart attacks

Every 5 minutes, one person has a stroke

>100 000 people in the UK with stroke each year

Every 5 minutes, one person has a stroke

Around 1 in 5 people over the age of 60 have peripheral arterial disease

https://www.bhf.org.uk/what-we-do/our-research/heart-statistics
https://www.stroke.org.uk/what-is-stroke/stroke-statistics
Cardiovascular disease – influence of LDL-cholesterol on CVD risk

Log-linear association per unit change in low-density lipoprotein cholesterol (LDL-C) and the risk of coronary heart disease
Currently available options for lipid lowering

Ezetimibe

Statins

Cholesterol

Degradation

LDL-receptor

Bempedoic acid

siRNA
Block PCSK9 synthesis

PCSK9

PCSK9 inhibiting mAb
Block PCSK9 action

LDL lowering by enhanced receptor-mediated catabolism

Effect on major vascular events of intensive lowering of LDL cholesterol: a meta-analysis of data from 170 000 participants in 26 randomised trials

~ 20% reduction in risk of vascular events per 1mmol/L reduction in LDL-C
Effect of intensive lipid lowering on CV events after myocardial infarction – real world evidence

Data from 40,607 MI patients in the SWEDHEART Registry
Effects of change in LDL-cholesterol between index event and 6-10 week follow up

Data from 40,607 MI patients in the SWEDEHEART Registry
Effects of change in LDL-cholesterol between index event and 6-10 week follow up

Effect of intensive lipid lowering on CV events after myocardial infarction – real world evidence

**A**
- No reduction or increase
- 0 - 50% reduction
- ≥ 50% reduction

**B**
- No reduction or increase
- 0 - 50% reduction
- ≥ 50% reduction

**Event rates by 1000 person-years**
- MACE
- All-cause mortality
- Major vascular event

We know that CV risk is high in the first year after an index CV event.

108,315 patients admitted to hospitals in Sweden with a primary MI.
But remember that CV risk remains high after an index CV event

108,315 patients admitted to hospitals in Sweden with a primary MI – Showing risk of events between 1 year and 4.5 years after event
We are good at initiating therapy after myocardial infarction

MINAP Report 2021
Proportion (%) of patients discharged on all secondary prevention medication for which they are eligible, 2010/11 - 2019/20

Secondary prevention:

- Start statin treatment in people with CVD with atorvastatin 80 mg. Use a lower dose of atorvastatin if any of the following apply: potential drug interactions, high risk of adverse effects, patient preference.
- Do not delay statin treatment in secondary prevention to manage modifiable risk factors.
- If a person has acute coronary syndrome, do not delay statin treatment. Take a lipid sample on admission and about 3 months after the start of treatment.

Monitoring:

- Measure total cholesterol, HDL cholesterol and non-HDL cholesterol in all people who have been started on high-intensity statin treatment at 3 months of treatment and aim for a greater than 40% reduction in non-HDL cholesterol.
- Provide annual medication reviews for people taking statins. Use these reviews to discuss medicines adherence and lifestyle modification and address CVD risk factors. Consider an annual non-fasting blood test to inform the discussion.

‘FIRE AND FORGET’ IS NOT APPROPRIATE

NICE. Cardiovascular disease: risk assessment and reduction, including lipid modification, Clinical guideline [CG181]
We are not good at treating to target after myocardial infarction

Da Vinci Study
5888 people prescribe lipid-lowering therapy across Europe

Attainment of ESC/EAS 2016 LDL-C targets

Attainment of ESC/EAS 2019 LDL-C targets

We are not good at treating to target after myocardial infarction

Profile and treatment of chronic coronary syndromes in European Society of Cardiology member countries: The ESC EORP CICD-LT registry

9174 patients with previous ST-elevation myocardial infarction (STEMI), non-STEMI or coronary revascularisation, or other CCS

“Poorly controlled cardiovascular risk factors were common across all cohorts”

Current smoking (18.5%)

Obesity (33.9%)

Diabetes (25.8%)

Raised low-density lipoprotein cholesterol (73.3%)

Persistent hypertension (24.7%)

We are not good at treating to target after myocardial infarction
Opportunities to optimise lipid management after a CV event – the UK perspective

Cardiovascular event

In hospital discharge cardiac rehab OP appt

Secondary Care

Statin initiation Statin optimisation Ezetimibe initiation Ezetimibe/bempedoic acid initiation

Primary Care

Management of statin intolerance

- LDL-C >1.8mmol/L
- LDL-C >4.0mmol/L (3.5mmol/L if very high risk) Refer for PCSK9i initiation
- LDL-C 2.6mmol/L or greater Inclisiran initiation

Repeat lipid profile assess adherence Lipid profile assess adherence Lipid profile assess adherence
Considerations for Optimising Post CVD Lipids Management

Dr Rani Khatib
Consultant Cardiology Pharmacist
Leeds Teaching Hospitals NHS Trust

Honorary Senior Lecturer, University of Leeds
National Clinical Champion for PCSK9i & Lipid Optimisation
Accelerated Access Collaborative, NHS England
Co-Chair Cardiology Group, UKCPA
Member of Science Committee, European Society of Cardiology, ACNAP

T: @DrRaniKhatib | E: r.khatib@leeds.ac.uk
W: https://medicinehealth.leeds.ac.uk/medicine/staff/507/rani-khatib
Non-modifiable risk factors for MI

- Increasing age
- Being male
- Family history of premature CHD
- Premature menopause
- 40-60% higher risk in South Asian patients compared with other populations

Modifiable risk factors for MI

- Smoking
- Diabetes mellitus (and impaired glucose tolerance)
- Metabolic syndrome
- Hypertension
- Hyperlipidaemia
- Obesity
- Physical inactivity

CHD, coronary heart disease; MI, myocardial infarction.

Patients receive sub-optimal Secondary Prevention Medications (SPM)\(^1,2\)

Cholesterol and blood pressure targets are not achieved\(^1,2\)

Poor medication adherence\(^1,3\)

Post-MI patients who are adherent to SPM are significantly less likely to be readmitted for a cardiovascular-related issue than non-adherent individuals\(^4\). This applies across all classes of post-MI SPM\(^5\).

MI, myocardial infarction; SPM, secondary prevention medications.
Among 500 patients with coronary artery disease in West Yorkshire, 43.5% were found to be non-adherent with at least one SPM.

A number of modifiable barriers to adherence were identified in the 219 non-adherent individuals, including:

- Forgetfulness (84.9%)
- Worry that medicines will do more harm than good (33.8%)
- Feeling hassled about taking medicines (18.7%)
- Feeling worse when taking medicines (14.2%)
- Not being convinced of the benefit of medicines (9.1%)

Statins contributed to 66.7% of overall non-adherence. Aspirin contributed to 61.7% of overall non-adherence, identified by the Single Question tool.

SPM, secondary prevention medications.
12-month review was conducted with 201 post-MI patients
- Patients had ≥10 months of follow up since inpatient treatment for MI between April 2016 and September 2017

Review was in clinic by a cardiologist, consultant pharmacist or advanced clinical pharmacist

Mean time from admission to data extraction was 384 days (range: 308–794 days)

MI, myocardial infarction.
## Patient baseline characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline value (N=210)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sex, n (%)</strong></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>141 (70.1)</td>
</tr>
<tr>
<td>Female</td>
<td>60 (29.9)</td>
</tr>
<tr>
<td><strong>Age, years, mean (range)</strong></td>
<td>66.9 (34–95)</td>
</tr>
<tr>
<td><strong>History of prior MI, n (%)</strong></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>43 (20.9)</td>
</tr>
<tr>
<td>No</td>
<td>158 (78.6)</td>
</tr>
<tr>
<td><strong>History of prior coronary intervention n, (%)</strong></td>
<td></td>
</tr>
<tr>
<td>Yes (any type)</td>
<td>42 (20.9)</td>
</tr>
<tr>
<td>PCI alone</td>
<td>27 (13.4)</td>
</tr>
<tr>
<td>CABG alone</td>
<td>5 (2.49)</td>
</tr>
<tr>
<td>PCI and CABG</td>
<td>10 (5.0)</td>
</tr>
<tr>
<td>No</td>
<td>159 (79.1)</td>
</tr>
<tr>
<td><strong>Risk factors</strong></td>
<td></td>
</tr>
<tr>
<td>Type 1 diabetes</td>
<td>3 (1.5)</td>
</tr>
<tr>
<td>Type 2 diabetes</td>
<td>48 (23.9)</td>
</tr>
<tr>
<td>Current or ex-smoker</td>
<td>99 (49.3)</td>
</tr>
<tr>
<td>eGFR &lt;60 mL/min/1.73 m² on admission</td>
<td>42 (20.9)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline value (N=210)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multi-vessel disease, n (%)</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>104 (51.8)</td>
</tr>
<tr>
<td>No</td>
<td>76 (37.8)</td>
</tr>
<tr>
<td>Unknown</td>
<td>21 (10.4)</td>
</tr>
<tr>
<td><strong>Intervention, n (%)</strong></td>
<td></td>
</tr>
<tr>
<td>PCI</td>
<td>151 (75.1)</td>
</tr>
<tr>
<td>CABG</td>
<td>1 (0.5)</td>
</tr>
<tr>
<td>Medical management</td>
<td>49 (24.4)</td>
</tr>
<tr>
<td><strong>BP recorded at clinic appointment, n (%)</strong></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>121 (60.2)</td>
</tr>
<tr>
<td>No</td>
<td>80 (39.8)</td>
</tr>
<tr>
<td><strong>SBP at clinic appointment, mmHg, mean (range)</strong></td>
<td>125 (88–189)</td>
</tr>
<tr>
<td><strong>DBP at clinic appointment, mmHg, mean (range)</strong></td>
<td>71 (45–99)</td>
</tr>
</tbody>
</table>

BP, blood pressure; CABG, coronary artery bypass graft; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; MI, myocardial infarction; PCI, percutaneous coronary intervention; SBP, systolic blood pressure.

Are we Measuring Lipid Profile?

On Admission

- LTHT Protocols - all patients with MI to have baseline total cholesterol (TC) and low-density lipoprotein (LDL) -cholesterol measured within the first 24 hours.

- 167/201 patients (83.1%) had their baseline TC

- 137/201 (68.2%) had their baseline LDL-cholesterol measured.

Repeat (>3 months post MI)

- 148 patients (73.6%) had TC rechecked

- 93 (46.3%) required LDL-cholesterol testing.

Achieving Targets - Lipid levels

• **Target levels:**
  - Total cholesterol <4.0 mmol/L
  - LDL-cholesterol <1.8 mmol/L

<table>
<thead>
<tr>
<th></th>
<th>Patients (at admission) n/N (%)</th>
<th>Patients (at 12 months) n/N (%)</th>
<th>Absolute change, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC &lt; 4.0 mmol/L</td>
<td>45/167 (26.9)</td>
<td>99/148 (66.9)</td>
<td>40.0</td>
</tr>
<tr>
<td>LDL-C &lt; 1.8 mmol/L</td>
<td>18/137 (13.1)</td>
<td>60/108 (55.6)</td>
<td>42.5</td>
</tr>
</tbody>
</table>

• At 12 months
  - **40% more** patients were achieving the total cholesterol target compared with baseline (admission)
  - **42.5% more** patients were achieving the LDL-cholesterol target at 12 months compared with baseline (admission)

• The majority of patients, whether they achieved targets or not, were on high-intensity statin therapy

LDL, low density lipoprotein.
What do you know about targets for LDL-C?

ESC/EAS recommendations across CV risk categories

- **Low risk**: SCORE <1%
- **Moderate risk**: SCORE 1–4%
- **High risk**: SCORE 5–9%
  - Particularly high single risk factors
  - Particularly single major risk factors
  - Moderate CKD (eGFR 30–59 mL/min)
  - DM without target organ damage or other risk factors, with DM duration <10 years

**CV Risk**

- **Low**
  - Treatment goal for LDL-C
    - 3.0 mmol/L (116 mg/dL)
- **Moderate**
  - 2.6 mmol/L (100 mg/dL)
  - 1.8 mmol/L (70 mg/dL)
- **High**
  - 1.4 mmol/L (55 mg/dL)
- **Very high**
  - Very high risk
    - SCORE ≥10%
    - ASCVD (clinical/imaging)
  - FH with ASCVD or other major risk factor
  - Severe CKD (eGFR <30 mL/min)
  - DM + target organ damage; ≥3 major risk factors or early onset of T2DM with duration >20 years

**Treatment goals based on CV Risk**

- **Primary prevention**
  - Intensify LLT if non-HDL-C reduction from baseline is <40%
  - NICE titration threshold
    - Non-HDL-C <2.5 mmol/L (LDL-C <1.8 mmol/L)
  - JBS3
    - FH
    - Optimise LLT to achieve ≥50% reduction in LDL-C (or Non-HDL-C)
    - If baseline cholesterol is unknown in the setting of secondary prevention, use the JBS3 consensus recommendation. Non-HDL-C = TC – HDL-C; LDL-C = non-HDL-C minus (fasting TG* / 2.2).
    - *valid only when fasting TG <4.5 mmol/L.

**TITRATION THRESHOLD / TARGET**

**Primary prevention**
- Intensify LLT if non-HDL-C reduction from baseline is <40%
- NICE titration threshold
  - Non-HDL-C <2.5 mmol/L (LDL-C <1.8 mmol/L)
- JBS3
  - FH
  - Optimise LLT to achieve ≥50% reduction in LDL-C (or Non-HDL-C)
  - If baseline cholesterol is unknown in the setting of secondary prevention, use the JBS3 consensus recommendation. Non-HDL-C = TC – HDL-C; LDL-C = non-HDL-C minus (fasting TG* / 2.2).
  - *valid only when fasting TG <4.5 mmol/L.

---


- AAC: Accelerated Access Collaborative; ASCVD: atherosclerotic cardiovascular disease; CKD: chronic kidney disease; CV: cardiovascular; DM: diabetes mellitus; EAS: European Atherosclerosis Society; eGFR: estimated glomerular filtration rate; ESC: European Society of Cardiology; HDL-C: high-density lipoprotein cholesterol; FH: familial hypercholesterolaemia; JBS: Joint British Societies; LDL-C: low-density lipoprotein cholesterol; LLT: lipid-lowering therapy; NICE: National Institute for Health and Care Excellence; T1/2DM: type 1/2 diabetes mellitus; TG: triglycerides.
Prescribing Statin and ezetimibe therapy

- The majority of patients were prescribed a statin and a small number of patients were prescribed ezetimibe therapy.

- Atorvastatin was the most frequently prescribed statin, both at discharge and at 12 months.

- The addition of ezetimibe to statin therapy can help in lipid lowering.

- At discharge, 4 patients were prescribed ezetimibe, 3 of whom were not receiving statins.

- At 12 months, 13/201 patients were receiving ezetimibe.
  - 2 of those had LDL-cholesterol < 1.8 mmol/L, 4 had LDL-cholesterol ≥ 1.8 mmol/L, and 7 had no data.

- Atorvastatin was the most frequently prescribed statin, both at discharge and at 12 months.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Discharge, n (%) N=201</th>
<th>12 months, n (%) N=201</th>
</tr>
</thead>
<tbody>
<tr>
<td>Statin</td>
<td>193 (96.0)</td>
<td>188 (93.5)</td>
</tr>
<tr>
<td>Ezetimibe</td>
<td>4 (2.0)</td>
<td>13 (6.5)</td>
</tr>
<tr>
<td>Neither</td>
<td>5 (2.5)</td>
<td>9 (4.5)</td>
</tr>
</tbody>
</table>

Patients were not achieving targets despite being on high intensity statins!

<table>
<thead>
<tr>
<th>Statin therapy</th>
<th>TC ≥ 4.0 mmol/L</th>
<th>LDL-cholesterol ≥ 1.8 mmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N=49</td>
<td>N=47</td>
</tr>
<tr>
<td>High intensity</td>
<td>32 (65.3)</td>
<td>35 (74.5)</td>
</tr>
<tr>
<td>Low intensity</td>
<td>11 (22.4)</td>
<td>9 (19.1)</td>
</tr>
<tr>
<td>None</td>
<td>6 (12.2)</td>
<td>3 (6.4)</td>
</tr>
</tbody>
</table>

Combination Lipid Lowering Therapy

**STATIN INTENSITY TABLE**

<table>
<thead>
<tr>
<th>Statin dose mg/day</th>
<th>Approximate reduction in LDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>21%</td>
</tr>
<tr>
<td>10</td>
<td>24%</td>
</tr>
<tr>
<td>20</td>
<td>27%</td>
</tr>
<tr>
<td>40</td>
<td>32%</td>
</tr>
<tr>
<td>80</td>
<td>37%</td>
</tr>
</tbody>
</table>

- **Fluvastatin**
- **Pravastatin**
- **Simvastatin**
- **Atorvastatin**
- **Rosuvastatin**
- **Atorvastatin + Ezetimibe 10mg**

<table>
<thead>
<tr>
<th>Statin</th>
<th>Approximate reduction in LDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rosuvastatin</td>
<td>38%</td>
</tr>
<tr>
<td>Atorvastatin + Ezetimibe 10mg</td>
<td>52%</td>
</tr>
</tbody>
</table>

- **Low intensity statins** will produce an LDL-C reduction of 20-30%
- **Medium intensity statins** will produce an LDL-C reduction of 31-40%
- **High intensity statins** will produce an LDL-C reduction above 40%
- **Simvastatin** 80mg is not recommended due to risk of muscle toxicity

- **Rosuvastatin** may be used as an alternative to Atorvastatin if compatible with other drug therapy. Lower starting dose maybe needed in some. See BNF.
- Low/medium intensity statins and should only be used in intolerance or drug interactions.
- **Ezetimibe** when combined with any statin is likely to give greater reduction in non-HDL-C/LDL-C than doubling the dose of the statin.
- **PCSK9i** (NICE TA393,384) alone or in combination with statins or Ezetimibe produce an additional LDL-C reduction of approximately 50% (range 25-70%).
- **Bempedoic acid** when combined with ezetimibe (TA694) produces an additional LDL-C reduction of approximately 28% (range 22-33%) but the long-term treatment effect of bempedoic acid is uncertain.

**Intensity of lipid-lowering treatment**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Average LDL-C reduction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate-intensity statin</td>
<td>≈30%</td>
</tr>
<tr>
<td>High-intensity statin</td>
<td>≈50%</td>
</tr>
<tr>
<td>High-intensity statin plus ezetimibe</td>
<td>≈65%</td>
</tr>
<tr>
<td>PCSK9 inhibitor</td>
<td>≈60%</td>
</tr>
<tr>
<td>PCSK9 inhibitor plus high-intensity statin</td>
<td>≈75%</td>
</tr>
<tr>
<td>PCSK9 inhibitor plus high-intensity statin plus ezetimibe</td>
<td>≈85%</td>
</tr>
</tbody>
</table>

**European Heart Journal, Volume 42, Issue 34, 7 September 2021, Pages 3227–3337, [https://doi.org/10.1093/eurheartj/ehab484](https://doi.org/10.1093/eurheartj/ehab484)**
Evolocumab and alirocumab are recommended as options for treating primary hypercholesterolaemia or mixed dyslipidaemia, only if LDL-C concentrations are persistently above the thresholds specified below despite maximal tolerated lipid lowering therapy.

### Patient populations

<table>
<thead>
<tr>
<th>Without CVD</th>
<th>With CVD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary non-FH or mixed dyslipidaemia</strong></td>
<td>Not recommended at any LDL-C concentration</td>
</tr>
<tr>
<td>LDL-C &gt; 5.0 mmol/L</td>
<td>LDL-C &gt; 4.0 mmol/L</td>
</tr>
<tr>
<td><strong>Primary HeFH</strong></td>
<td>LDL-C &gt; 5.0 mmol/L</td>
</tr>
</tbody>
</table>

<sup>*</sup> High risk of CVD is defined as a history of any of the following: ACS (such as MI or UA requiring hospitalisation), coronary or other arterial revascularisation procedures, coronary heart disease, ischaemic stroke or peripheral artery disease.

<sup>†</sup> Very high risk of CVD is defined as recurrent CV events or CV events in more than one vascular bed (that is, polyvascular disease).

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The card reports on medicines in NHS in England which have been positively appraised by NICE.

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### Alirocumab for treating primary hypercholesterolaemia and mixed dyslipidaemia

**Technology appraisal guidance [TA393]**
Published date: 22 June 2016

**Evolocumab for treating primary hypercholesterolaemia and mixed dyslipidaemia**

**Technology appraisal guidance [TA394]**
Published date: 22 June 2016

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What about Inclisiran? The Leeds Lipids Management Pathway

If maximum tolerated dose of statin does not control non-HDL-C/LDL-C well enough after 3 months confirm statin adherence, then consider the following options based on shared decision making with the patient:

- **Injectable therapies are not considered the first line option locally.**
- **Ezetimibe is the locally preferred first line option.**

If non-HDL-C > 2.5mmol/L after 3 months despite maximal tolerated lipid lowering therapy, based on shared-decision making consider suitability for injectable therapies (TA393/394, TA733) (see below)

Consider adding Ezetimibe 10mg OD (NICE TA385). Assess response after 3 months

If recommended statin treatment is contraindicated or not tolerated - follow AAC Statin Intolerance Algorithm for advice regarding adverse effects (click here). Leeds statin intolerance guidance is available here

- If it is confirmed that recommended statin treatment is contraindicated or not tolerated, based on shared-decision making consider the following options:
  - Ezetimibe 10mg monotherapy may be considered. Assess response after 3 months (TA385)
  - Ezetimibe 10mg/Bempedoic acid 180 mg combination may be considered when ezetimibe alone does not control non-HDL-C/LDL-C well enough. (NICE TA694)

- If non-HDL-C > 2.5mmol/L after 3 months despite maximal tolerated lipid lowering therapy, based on shared-decision making consider suitability for injectable therapies (TA393/394, TA733) (see below)

Arrange a fasting blood test for LDL-C measurement to determine if eligibility criteria for injectables are met:
- Consider PCSK9i if eligibility criteria are met (NICE TA393/394; see page 2 ‘Specialist services’).
- Consider inclisiran if LDL-C greater than or equal to 2.6mmol/L (TA733)
- If eligibility criteria are not met, consider remaining options (e.g. ezetimibe if not previously considered)
Overview of the relative prevalence of the main types of adverse effects reported with statin therapy

- **Cataract**: No evidence for increased risk
- **Haemorrhagic stroke**: No increase in risk, although SPARCL suggested a possible increase in risk with prior stroke
- **Muscle symptoms**: Double-blind RCTs: 0.1–0.2%; Non-blinded observational studies: 7–29%
- **Effects on cognition**: No evidence that statins adversely affect cognitive function
- **Effects on liver**: Clinically relevant effects are very rare (~1 per 100,000)
- **Dysglycaemia, new-onset diabetes**: RCTs: ~0.1 per year; individuals with metabolic syndrome or prediabetes are at greater risk
- **Proteinuria**: Low frequency of mild proteinuria; no evidence of clinically significant deterioration of renal function

Adapted from Mach F, et al. 2018.

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- RCT: randomised controlled trial; SPARCL: Stroke Prevention by Aggressive Reduction in Cholesterol Levels trial.
The NECEBO effect
### Statin Intolerance Pathway

**Person at high CVD risk reports potential intolerance to recommended high intensity statin treatment**

1. **Consider other potential side effects for statins**
   - **Be aware of Statin Reluctance** and Nocebo Effect
   - **See ‘Person Centred Care’ box at page 2**

2. **Muscular symptoms not related to statins**
   - **Non SRM: Consider other causes** e.g. PMR, Vit D deficiency.
     - Check bone profile, Vit D, CRP.

3. **Tolerable symptoms**
   - No clinical concern
     - CK < 4x ULN
   - Improvement within 2 weeks
     - Resolved within 6 weeks
     - Patient happy to continue

4. **Intolerable symptoms**
   - and/or clinical concern and/or CK > 4x and < 10x ULN
   - **Stop statin for 4-6 weeks**
     - Document time to symptom onset and time to resolution
   - **Has CK normalised?**
     - Yes
       - **Have symptoms resolved?**
         - Yes
           - **Has the patient been symptom free for at least 2 weeks?**
             - Yes
               - **Consider further options** (For example co-administering ezetimibe or as monotherapy) see page 2 - ‘Statin-based Approaches’
             - No
               - **Consider further options** see page 2 - ‘Statin-based Approaches’
       - No
         - **Recurrence of muscle symptoms**
           - Short time to onset
             - Symptoms intolerable
               - **Consider further options** see page 2 - ‘Statin-based Approaches’
         - **No recurrence of muscle symptoms**
           - Titrated at 8 weeks intervals to achieve appropriate targets

5. **Symptoms tolerable**
   - Treatment goals achieved
     - Patient happy to continue

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**YES**

**NO**

### Other Considerations

- **New onset or worsening of muscle symptoms since starting statins? (pain, tenderness or weakness)**
  - **Symmetrical pain and/or weakness in large proximal muscle groups, worsened by exercise**

- **Non SRM:** Consider other causes e.g. PMR, Vit D deficiency. Check bone profile, Vit D, CRP.

- **Muscular symptoms not related to statins**

- **Symptoms typical for Statin Related Muscle toxicity (SRM)**?
  - **Measure Creatinine Kinase (CK) Assess severity of symptoms +/- repeat baseline assessment**

- **Intolerable symptoms**
  - and/or clinical concern and/or CK > 4x and < 10x ULN
  - **Stop statin for 4-6 weeks**
    - Document time to symptom onset and time to resolution
  - **Has CK normalised?**
    - Yes
      - **Have symptoms resolved?**
        - Yes
          - **Has the patient been symptom free for at least 2 weeks?**
            - Yes
              - **Consider further options** (For example co-administering ezetimibe or as monotherapy) see page 2 - ‘Statin-based Approaches’
            - No
              - **Consider further options** see page 2 - ‘Statin-based Approaches’
        - No
          - **Recurrence of muscle symptoms**
            - Short time to onset
              - Symptoms intolerable
                - **Consider further options** see page 2 - ‘Statin-based Approaches’
          - **No recurrence of muscle symptoms**
            - Titrated at 8 weeks intervals to achieve appropriate targets

- **Symptoms tolerable**
  - Treatment goals achieved
    - Patient happy to continue

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**Symptoms not related to statins**

**Non SRM: Consider other causes** e.g. PMR, Vit D deficiency. Check bone profile, Vit D, CRP.

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**CK = Creatinine Kinase**

**CRP = C-Reactive Protein**

**eGFR = Estimated Glomerular Filtration Rate**

**PMR = Polymyalgia Rheumatica**

**SINAM = Statin Induced Necrotising Autoimmune Myopathy**

**SRM = Statin Related Muscle Toxicity**

**ULN = Upper Limit of Normal Range**

**Vit D = Vitamin D**

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**Please refer to page 2 for more details**

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**This resource relates to NICE guidance:**

- CG181, CG71, TA385, TA393/394, QS100
**Statin**s are the cornerstone for prevention and treatment of cardiovascular (CV) disease with a substantial evidence of reduction of morbidity and mortality. Refer to Lipid Management Pathway and related NICE guidelines (CG181, CG71) for guidance on initiation, titration and monitoring of statin therapy.

In clinical trials, statins were found to be largely well tolerated (often with a similar adverse effect [AE] profile to placebo), however this is not reflected in clinical practice where up to 75% of people started on a statin will discontinue treatment within 2 years.

Stopping statin therapy is associated with an increased risk of major CV events and there is growing concern that clinicians are labelling patients as ‘statin intolerant’ too quickly. Indeed, statin discontinuation is significantly associated with negative media coverage.

**Definition of Statin Intolerance**

Intolerance to initial statin therapy is defined by NICE as the presence of clinically significant adverse effects that represent an unacceptable risk to the patient or that may reduce compliance with therapy.

Other definition: any adverse event (AEs) considered unacceptable by the patient, and/or some laboratory abnormalities, both attributed to statin treatment and leading to its discontinuation.

**Statin-associated muscle symptoms (SAMS)**

SAMS are one of the principal reasons for statin non-adherence and/or discontinuation. However, not all such symptoms should lead to a label of ‘statin intolerance’ as they may not be truly related muscle symptoms. The challenge is to separate these from the spectrum of SRM and reconcile with re-challenge.

**Non-SRM related muscle skeletal abnormalities (Non SRM)**

If patients report symptoms that are not typical of SRM (e.g. asymmetric distribution, failure to resolve with de-challenge despite normal CK) consider other rheumatological disorders, metabolic, degenerative or inflammatory e.g. Vitamin D deficiency, polymyalgia rheumatica. Check Bone profile, VITD, CRP.

**Considerations when starting a statin to reduce risk of SRM**

- Check baseline thyroid, liver and renal function, any potential drug interactions, and avoid the highest doses in at risk groups (See “Risk Factors” below).
- Ask the person if they have had persistent generalised unexplained muscle pain, whether associated or not with previous lipid-lowering therapy. If they have, measure CK, if CK levels are > 4x ULN do not start statin—investigation required. Do not measure CK if person is asymptomatic.
- Warn patients about AEs, specifically muscle symptoms. Advise people who are being treated with a statin to seek medical advice if they develop muscle symptoms (pain, tenderness or weakness). If this occurs, measure CK (see page 1).

**Risk factors for SRM and statin intolerance**

- **Endogenous causes**
  - Female gender
  - Advanced age (> 75yrs)
  - Privately reduced lean body mass
  - History of muscle disorder or high CK
  - Impaired renal or hepatic function
  - Personal or Family history of intolerance to lipid-lowering therapies
  - Hypothyroidism

- **Exogenous Factors**
  - Excessive alcohol intake
  - High intensity exercise
  - Dehydration
  - Liver enzyme abnormalities
  - Irregular eating (or very high intake of alcohol)
  - Clinical conditions causing myositis (SRM5) like reaction, Sleep disturbance, headache, dizziness, fatigue, depression, sexual dysfunction

**Management:** If symptoms appear related, consider de-challenge and re-challenge or change to a different statin (e.g. hydrophilic instead of lipophilic).

Liver enzyme abnormalities - minor increases in liver enzymes (< 2x ULN) may be seen within the first three months of statin therapy; temporary discontinuation and further assessment is warranted if levels exceed 3x ULN. Several studies have confirmed that the cardiovascular benefits of statin treatment in high-risk populations outweigh the rare adverse effects, such as rhabdomyolysis.

**Follow up**

- Follow up on agreed plan and address any value mismatch.
- Advise patients to contact you if they experience muscle symptoms
- Ongoing patient education and regular review helps addressing concerns around medicine safety and underline the importance of adherence.

**Statin-based approaches to manage muscle symptoms**

- Refer to the AAC Lipid Management Algorithm. [click here](#).
- Consider ezetimibe, (NICE TA 393) therapy as per algorithm
- Consider PCSK9 if eligible for treatment according to NICE TA 393, 394

**LDL-C lowering options for patients with genuine statin intolerance**

- **Initial Consultation**
  - Be aware of “nosceo effect” and “statin reluctance”.
  - Reinforce healthy lifestyle habits (e.g. exercise, reducing weight)
  - Listen to the concerns of each patient
  - Explain LDL-C targets and strategies to lower LDL-C/HDL-C
  - Discuss options to reduce LDL-C/HDL-C with (1) Regorafenib (2) Ezetimibe (3) PCSK9 inhibitors
  - Explain the benefits of statins
  - Evaluate & identify any risk factors and address (e.g. drug interactions)
  - Work with patients to identify and agree best options and next steps

- **(1) Nocebo effect** is negative expectations of the patient regarding a treatment leading to reporting more negative effects even if they are prescribed a placebo.
- **(2) Statin reluctance** is an attitudinal state of averse to taking statins (often before prior exposure).

**Classification of statin related muscle toxicity (SRM)**

<table>
<thead>
<tr>
<th>SRM</th>
<th>Phenotype</th>
<th>Incidence</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>SRM 0</td>
<td>CK elevation &lt; 4x ULN</td>
<td>1.5-26%</td>
<td>No muscle symptoms</td>
</tr>
<tr>
<td>SRM 1</td>
<td>No muscle symptoms</td>
<td>190,000,000 Patient-years</td>
<td>Muscle symptoms without CK elevation</td>
</tr>
<tr>
<td>SRM 2</td>
<td>Myopathy, intolerable</td>
<td>0.2-2.1/100,000</td>
<td>Muscle symptoms, CK &lt; 4x ULN, complete resolution on de-challenge</td>
</tr>
<tr>
<td>SRM 3</td>
<td>CK elevation &gt; 4x ULN &lt; 10x ULN</td>
<td>5000 Patient-years</td>
<td>Muscle symptoms, complete resolution on de-challenge</td>
</tr>
<tr>
<td>SRM 4</td>
<td>Severe myopathy</td>
<td>0.11%</td>
<td>CK elevation &gt; 10x ULN &gt; 50x ULN, muscle symptoms, complete resolution on de-challenge</td>
</tr>
<tr>
<td>SRM 5</td>
<td>Rhabdomyolysis</td>
<td>0.1-8.4/100,000</td>
<td>CK elevation &gt; 10x ULN with evidence of renal impairment + muscle symptoms or CK &gt; 50x ULN</td>
</tr>
</tbody>
</table>

**HMGCoA reductase inhibitors (statins)**

- **SRM 0**
  - Detection of HMGCoA reductase antibodies, HMGCoA reductase expression in muscle biopsy showing autoimmune myositis, incomplete resolution on de-challenge

**Initial Consultation**

- Be aware of “nosceo effect” and “statin reluctance”.
- Reinforce healthy lifestyle habits (e.g. exercise, reducing weight)
- Listen to the concerns of each patient
- Explain LDL-C targets and strategies to lower LDL-C/HDL-C
- Discuss options to reduce LDL-C/HDL-C with (1) Regorafenib (2) Ezetimibe (3) PCSK9 inhibitors
- Explain the benefits of statins
- Evaluate & identify any risk factors and address (e.g. drug interactions)
- Work with patients to identify and agree best options and next steps

**Follow up**

- Follow up on agreed plan and address any value mismatch.
- Advise patients to contact you if they experience muscle symptoms
- Ongoing patient education and regular review helps addressing concerns around medicine safety and underline the importance of adherence.

**Statin-based approaches to manage muscle symptoms**

- Adopt person-centred approach as described above
- Therapy by SRM as demonstrated by resolution on de-challenge
- Apply a repetitive “De-Challenge”, “Re-Challenge” approach to establish if symptoms are caused by a (statin) and the best statin regimen for each patient
- Switch to a different statin or re-challenge with the same statin using a lower dose or frequency (intermittent dosing)
- Patients who do not tolerate statins on a daily basis, alternate day or twice-weekly dosing is a good option.
- Rosuvastatin and atorvastatin have longer half lifetimes, permitting use on an-once-daily regimen
- Adding ezetimibe to a lower dose statin may be better tolerated with robust reduction in LDL-C/HDL-C
- Once a new regime is tolerated, dose/frequency can be up-titrated slowly to achieve LDL-C/HDL-C goals with minimal or no muscle complaints

It is important to note that cardiovascular benefits have not been proven for all the above approaches but any reduction of LDL-C/HDL-C is beneficial.

**LDL-C lowering options for patients with genuine statin intolerance**

- Refer to the AAC Lipid Management Algorithm. [click here](#).
- Consider ezetimibe, (NICE TA 393) therapy as per algorithm
- Consider PCSK9 if eligible for treatment according to NICE TA 393, 394

**Non-muscle related statin side effects**

May vary between different statins. In clinical trials some side effects often associated with statins are not statistically different from placebo.

Most commonly reported: gastrointestinal disturbance and asymptomatic increase in hepatic transaminases (ALT or AST), Myalgic efface may affect up to 1 in 10 statin users.

**Rarer side effects include:** Hypo/hyperglycemia, new onset Type 2 Diabetes (benefits outweigh risk, do not stop statin), Renal insufficiency, proteinuria, Neuropsychiatric symptoms

**References**


[2] Intracranial haemorrhage (conflicting evidence, benefit outweigh possible harm). [click here](#).

[3] Intestinal lung disease, Pancreatitis, Skin disorders including alopecia, Lupus-like reaction, Sleep disturbance, headache, dizziness, fatigue, depression, sexual dysfunction.

**Authors:** Dr Ranj Khatib & Dr Dermot Neeley on behalf of the AAC Clinical Subgroup. [click here](#).

**Follow up:**

- Follow up on agreed plan and address any value mismatch.
- Advise patients to contact you if they experience muscle symptoms
- Ongoing patient education and regular review helps addressing concerns around medicine safety and underline the importance of adherence.

**Statin-based approaches to manage muscle symptoms**

- Adopt person-centred approach as described above
- Therapy by SRM as demonstrated by resolution on de-challenge
- Apply a repetitive “De-Challenge”, “Re-Challenge” approach to establish if symptoms are caused by a statin(s) and the best statin regimen for each patient
- Switch to a different statin or re-challenge with the same statin using a lower dose or frequency (intermittent dosing)
- Patients who do not tolerate statins on a daily basis, alternate day or twice-weekly dosing is a good option.
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It is important to note that cardiovascular benefits have not been proven for all the above approaches but any reduction of LDL-C/HDL-C is beneficial.
Lipid Optimisation in Peripheral Arterial Disease

Mr Marc Bailey

PhD MB ChB BSc PGCert MRCS AFHEA RCPathME
Associate Professor of Vascular Medicine
School of Medicine, The University of Leeds
Honorary Senior Clinical Lecturer in Vascular Surgery
The Leeds Vascular Institute, Leeds General Infirmary
Peripheral Arterial Disease

- Reduction in blood flow through large peripheral arteries (typically legs)
- Atherosclerosis
- Thrombo-embolus
- Vasculitis
- External compression
- Rare vascular wall abnormalities (e.g. CAD)
Intermittent Claudication

- Intermittent pain in muscles
- Induced by exercise
- Predictable distance
- Rapidly relieved by rest
- Worse up hill / cold weather
- Limits walking distance / ADLs
- Not limb threatening
- 98% will retain their leg
- 80% stable over 5
Chronic Limb Threatening Ischaemia

- Vascular rest pain
- Typically at night in feet
- Tissue loss (>2/52)
  - Ulceration
  - Gangrene
  - Superadded Infection
- WIFI Score
- High risk of limb loss
- High benefit of Revascularisation
- High mortality (25% at 1 year of diagnosis)
Diagnosis

- **Vascular examination**
- **Physiological bedside tests**
  
  **ABPI***

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<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;1.2</td>
<td>Calcified</td>
</tr>
<tr>
<td>0.9-1.2</td>
<td>Normal</td>
</tr>
<tr>
<td>0.5-0.9</td>
<td>PAD</td>
</tr>
<tr>
<td>&lt;0.5</td>
<td>CLI</td>
</tr>
</tbody>
</table>

  * Toe pressure <60mmHg

* Caution in diabetes or CKD
Peripheral Arterial Disease

- **~237 million** cases worldwide
- ¼ of the global burden of CVD
- Majority are claudicants
- **1-2%** risk of MALE/yr\(^1,2\)
- **5-10%** risk of MACE/yr\(^1,2\)
- Highest risk of future MACE
- High proportion of polyvascular disease
- **75%** of claudicants die from MACE
- Massively medically undertreated\(^3\)

Intermittent Claudication
- Supervised exercise
- Smoking cessation
- Optimal medical therapy
- Vasodilators
- Angioplasty if severely disabling

CLTI
- Urgent assessment by vascular surgery
- Imaging with a view to revascularisation
- MDT review of all patients
Secondary prevention of CVD
- Smoking cessation
- Lipid modification / statins
- Anti-platelet therapy
- Prevention, diagnosis, management
  - Diabetes
  - Hypertension

Lipid targets:
- LDL cholesterol <1.8mmol/L or 40% reduction (NICE)
- LDL cholesterol <1.4mmol/L or 50% reduction (ESC)
- LDL cholesterol >3.5mmol/L on maximal therapy, PCSK9i
Treat to target LDL approach

---

**Check Baseline LDL-C**
- Check LFTs, U&E, HbA1c, eGFR
- Prescribe max intensity statin

**Annual lipid screening**

**LDL-C < 1.4mmol/L?**
- Titrated up to max statin dose
- Add ezetimibe 10mg

**LDL-C > 1.4mmol/L?**
- Recheck LDL-C at 3 months
- Consider PCSK9 inhibitor referral

**LDL-C > 3.5mmol/L?**
- Discuss adherence
- Consider specialist lipid clinic advice

**LDL-C 1.4 to 3.5mmol/L?**
- Recheck LDL-C at 6 months

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**Sucharitkul et al 2021, Annals of Vascular Surgery**

**NICE statin intolerance algorithm**

**NICE statin intensities**
New PAD Referrals
01/2017 – 03/2020
n=376

Fire & Forget Clinic (F&F)
N=224

Treat to Target Clinic (T2T)
N=152

Claudicants only
N=179

Claudicants only
N=109

1 year LDL Fup
N=81

1 year LDL Fup
N=57

CLI N=52

CLI N=43

Sucharitkul et al. unpublished
### Baseline Demographics

<table>
<thead>
<tr>
<th>Characteristics no. (%)</th>
<th>F&amp;F I/II n= 179</th>
<th>T2T I/II n=109</th>
<th>p</th>
<th>adj. p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age yrs mean (SD)</td>
<td>66.8 (±9.8)</td>
<td>68.3 (±10.9)</td>
<td>0.2522</td>
<td>0.7100</td>
</tr>
<tr>
<td>Female</td>
<td>46 (26.7)</td>
<td>32 (29.4)</td>
<td>0.6824</td>
<td>1.0000</td>
</tr>
<tr>
<td>White ethnicity (of given)</td>
<td>148 (86.0)</td>
<td>77 (70.6)</td>
<td>0.7539</td>
<td>1.0000</td>
</tr>
<tr>
<td>BMI kg/m² mean (SD)</td>
<td>27.2 (±4.9)</td>
<td>27.2 (±4.6)</td>
<td>0.8214</td>
<td>1.0000</td>
</tr>
<tr>
<td>Fontaine Staging no. (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>5 (2.9)</td>
<td>1 (0.9)</td>
<td>0.4102</td>
<td>0.9600</td>
</tr>
<tr>
<td>II a/b</td>
<td>167 (97.1)</td>
<td>108 (99.1)</td>
<td>0.4102</td>
<td>0.8200</td>
</tr>
<tr>
<td>Comorbidities no. (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes T1/2</td>
<td>56 (32.6)</td>
<td>37 (33.9)</td>
<td>0.7952</td>
<td>1.0000</td>
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<tr>
<td>IHD</td>
<td>52 (30.2)</td>
<td>33 (30.3)</td>
<td>1.0000</td>
<td>1.0000</td>
</tr>
<tr>
<td>COPD</td>
<td>43 (25.0)</td>
<td>17 (15.6)</td>
<td>0.0730</td>
<td>0.2600</td>
</tr>
<tr>
<td>Hypertension</td>
<td>98 (57.0)</td>
<td>76 (69.7)</td>
<td>0.0331</td>
<td>0.2300</td>
</tr>
<tr>
<td>CKD</td>
<td>96 (55.8)</td>
<td>73 (67.0)</td>
<td>0.0598</td>
<td>0.2800</td>
</tr>
<tr>
<td>Cerebrovascular Hx</td>
<td>20 (11.6)</td>
<td>25 (22.9)</td>
<td>0.0186</td>
<td>0.2600</td>
</tr>
<tr>
<td>Mono PAD</td>
<td>108 (62.8)</td>
<td>67 (61.5)</td>
<td>0.8996</td>
<td>1.0000</td>
</tr>
<tr>
<td>Poly PAD</td>
<td>64 (37.2)</td>
<td>42 (38.5)</td>
<td>0.8996</td>
<td>0.9700</td>
</tr>
</tbody>
</table>

Sucharitkul et al. unpublished
Results: Lipid therapy

Sucharitkul et al. unpublished
Results: LDL-c

Sucharitkul et al. unpublished
• PAD means high cardiovascular risk
• The diagnosis of PAD should be sought out
• All PAD patients need intensive lipid treatment
• A T2T approach to an LDL <1.8mmol/L is advised
• Rapid commencement of highest intensity statin
• Knowledge of the de-challenge/re-challenge statin intolerance approach
• Re-check LDL at 3 months and use Ezetimibe
• Refer to lipid clinic if LDL >3.5mmol/L on maximal treatment or intolerance problems
Conclusion

- People with CV events or with PAD are at high risk of further events
- Effective therapies to reduce risk through intensive lipid lowering are available but are currently under-deployed
- Measure and re-measure full lipids profile appropriately.
- Address statin reluctance & intolerance.
- Optimise Lipid Lowering Therapy and use Combinations.
- Identify and address non-adherence.
Next steps:
Join us for the next webinar:

Weds 8th December 11am-12 noon: ‘Novel Therapies - Now and Future’.

Dr Ameet Bakhai, Professor Kosh Ray and Dr Yassir Javaid will integrate primary and secondary care perspectives, walk us through the novel medicines in the lipid pathway and discuss what is on the horizon.

Dr Ameet Bakhai
Consultant Cardiologist and Research Director. Royal Free London NHS Foundation Trust

Professor Kosh Ray
Professor of Public Heath in the Department of Public Health and Primary Care at Imperial College London as well as Honorary Consultant Cardiologist at the Imperial College NHS Trust.

Dr Yassir Javaid
Cardiovascular Lead Northamptonshire CCG 2013 - present
Primary Care CVD lead East Midlands Clinical Network (2013-2020)

Join us for an informal case based interactive clinic on post CVD event management: Date 1st December 11am-12 noon
All programme content, recordings and next webinar bookings will be housed in the HEART UK pages. Visit the site for the new e-Learning modules on Identifying FH in primary care, Statin Intolerance, and the Lipid Management Pathway
Thank you

This webinar has now finished.

Today’s slides and recording will be available after the webinar on the HEART UK pages. Visit the site for the new e-Learning modules on diet launching in November. Identifying FH in primary care, Statin Intolerance, and the Lipid Management Pathway modules are also available.

All programme content, recordings and next webinar bookings will be housed here: https://www.heartuk.org.uk/tackling-cholesterol-together/home