HỘI NGHỊ KHOA HỌC THƯỜNG NIÊN NĂM 2024

Bài báo cáo

Các nền tảng của liệu pháp điều trị hạ lipid máu và dự phòng biến cố tim mạch

BS. CKII. Trần Thị Thanh Trúc
Bệnh viện Đa Khoa Tâm Anh

VN-LIPI-2024-00011

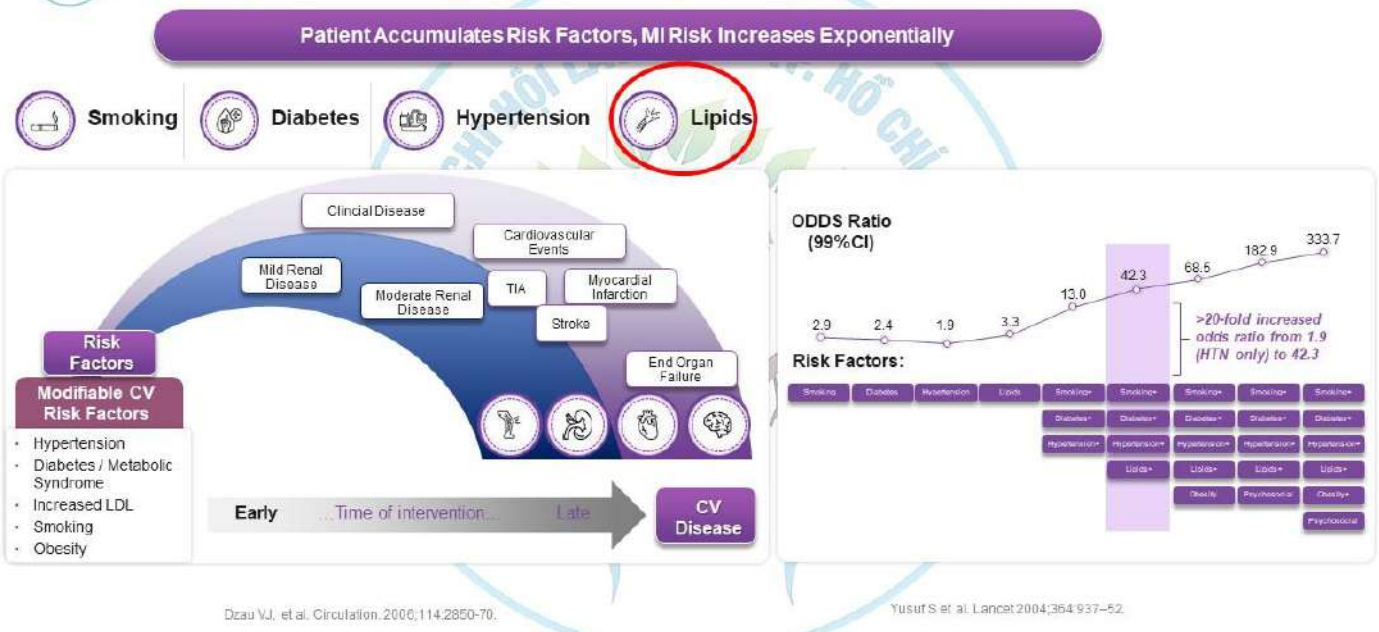
Lưu ý

- Nội dung trình bày chỉ thể hiện quan điểm và kinh nghiệm của báo cáo viên và không nhất thiết thể hiện quan điểm hay khuyến nghị của Viatris Việt Nam dưới bất kỳ hình thức nào.
- Hình ảnh/nội dung trích dẫn trong bài báo cáo thuộc về báo cáo viên hoặc sử dụng bởi báo cáo viên.
- Viatris Việt Nam đã kiểm tra nội dung để đảm bảo thỏa một số tiêu chuẩn cụ thể nhưng không đảm bảo sự chính xác trong trích dẫn tài liệu, và bản quyền hình ảnh và nội dung trích dẫn. Viatris Việt Nam, các công ty con hoặc công ty liên kết không chịu trách nhiệm dưới bất kỳ hình thức nào cho tính chính xác của nội dung bài báo cáo.

NỘI DUNG CHÍNH

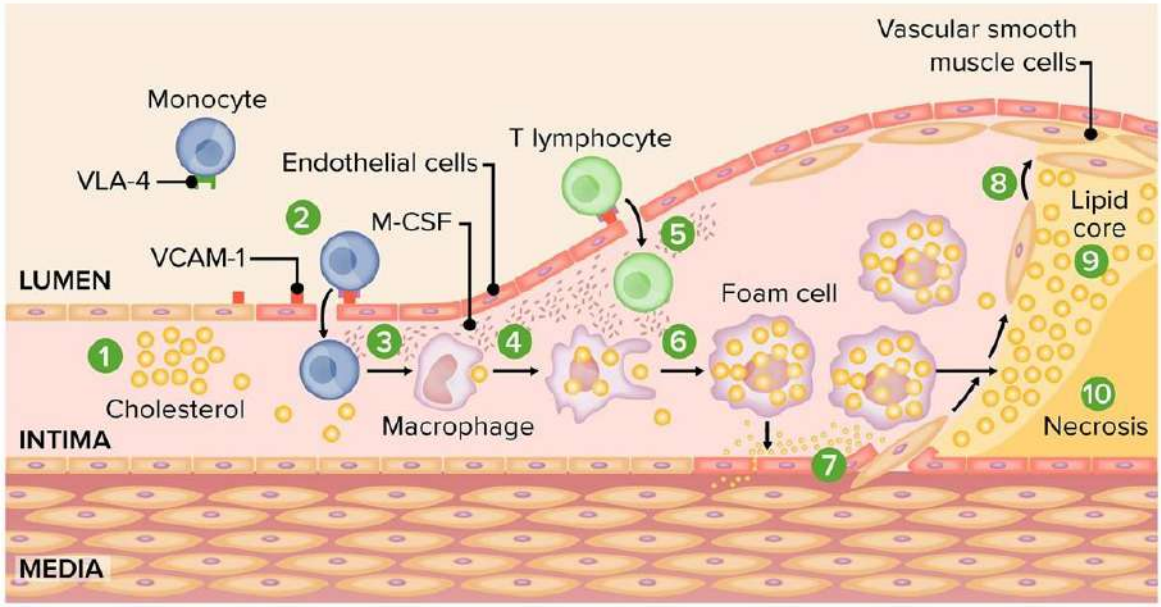
1. **TỔNG QUAN VỀ CÁC YẾU TỐ NGUY CƠ BỆNH LÝ TIM MẠCH TIẾN TRIỂN**
2. VAI TRÒ STATIN TRONG PHÒNG NGỪA NGUYÊN PHÁT VÀ THỨ PHÁT BỆNH LÝ TIM MẠCH XƠ VỮA
   - —Nghiên cứu nền tảng về vai trò statin trong phòng ngừa nguyên phát và thứ phát bệnh lý mạch máu.
   - —Từ nghiên cứu đến khuyến cáo thực hành
   - —Lợi ích của statin trong các bệnh lý thận và gan nhiễm mỡ.
3. KẾT LUẬN

Các yếu tố nguy cơ bệnh lý tim mạch



Dzau VJ, et al. Circulation. 2006;114:2850-70.

Yusuf S et al. Lancet 2004;364:937–52.

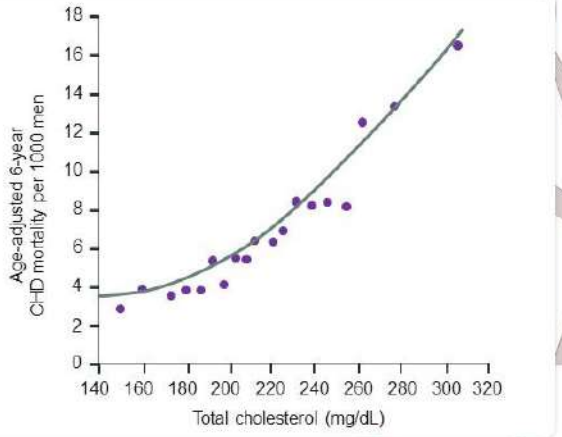


Cơ chế hình thành mảng xơ vữa

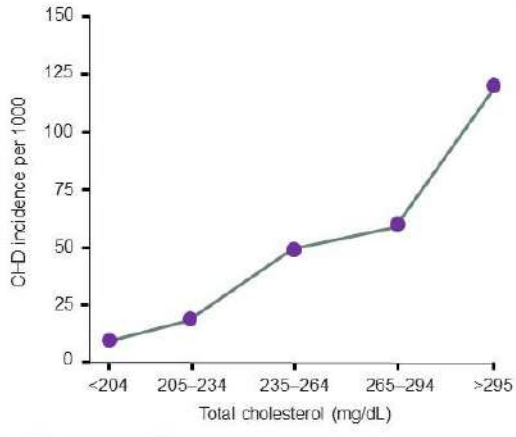
https://www.powerofparticles.com/nanoparticle-based-therapies-treatment-atherosclerosis. Accessed in Jan 2024

Cholesterol toàn phần tăng tỉ lệ với nguy cơ bệnh lý mạch vành

Multiple risk factor intervention trial (MRFIT) (n=361,662)[1]

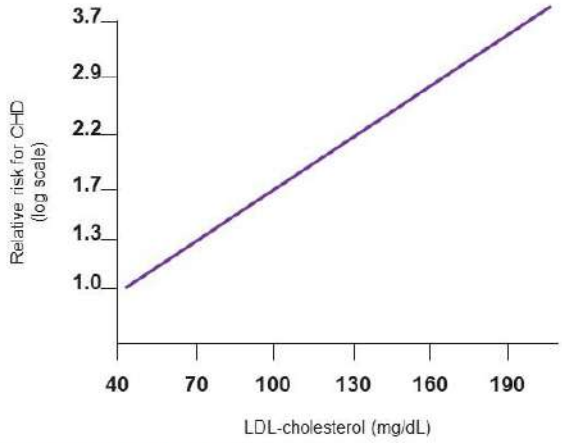


Framingham study (n=5209)[2]



CHD, coronary heart disease
1. Martin MJ, et al. Lancet. 1986;329:933–936; 2. Castelli WP. Am J Med. 1994;76:4–12

Tăng nồng độ LDL-c tỉ lệ thuận với tăng nguy cơ bệnh lý mạch máu xơ vữa



LDL= Total Cholesterol - HDL - TG/5

- Serum total LDL-cholesterol (LDL-C) levels are continuously correlated with CHD risk
- CHD risk rises more steeply with increasing LDL-C
- On a log scale the relationship between LDL-C levels and CHD risk is linear
- **For every 30 mg/dL (~0.8 mmol/L) increase in LDL-C, the relative risk for CHD increases by about 30%**
- Elevated LDL-C or total cholesterol increase the risk of CHD, independent of other risk factors

CHD, coronary heart disease
Grundy SM, et al. Circulation 2004;110:227–239. Reproduced with permission from Wolters Kluwer Health

NỘI DUNG CHÍNH

1. TỔNG QUAN VỀ CÁC YẾU TỐ NGUY CƠ BỆNH LÝ TIM MẠCH TIẾN TRIỂN

2. VAI TRÒ STATIN TRONG PHÒNG NGỪA NGUYÊN PHÁT VÀ THỨ PHÁT BỆNH LÝ TIM MẠCH XƠ VỮA

- Nghiên cứu nền tảng về vai trò statin trong phòng ngừa nguyên phát và thứ phát bệnh lý mạch máu.
- Từ nghiên cứu đến khuyến cáo thực hành
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3. KẾT LUẬN

Cơ chế tác động giảm Cholesterol máu của nhóm Statin

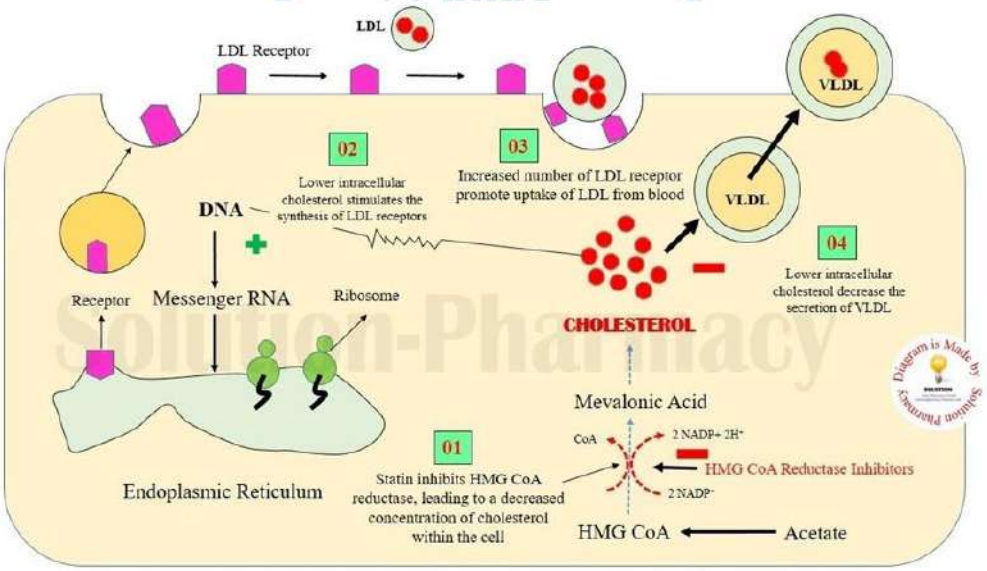


Diagram- Mechanism of Action of HMG CoA Reductase Inhibitors (Reference- Lippincott)

https://pubmed.ncbi.nlm.nih.gov/12067471/. Accessed Jan 2024

Hiệu quả của Statin trong phòng ngừa tiên phát đột quỵ

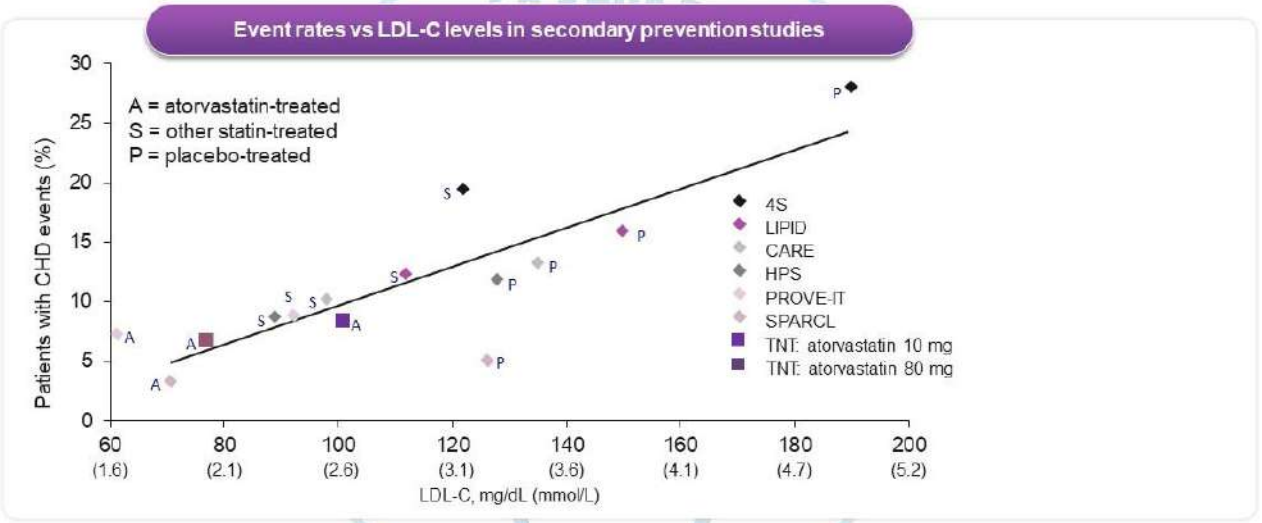
Incidence of ALL strokes reduced by 18% (95% CI, 13%–23%; p<0.0001)

	Active group, %	Control group, %	RR (95% CI)	RR (95% CI)
Primary prevention of stroke	**Meta-analysis: 24 statin trials, 165,792 patients**			
SEARCH	4.2	4.6		0.91 (0.77-1.08)
JUPITER	0.4	0.7		0.52 (0.34-0.78)
ASPEN	2.8	3.2		0.88 (0.58-1.40)
MEGA	1.3	1.6		0.83 (0.57-1.20)
IDEAL	3.4	3.9		0.87 (0.70-1.08)
TNT	2.3	3.1		0.76 (0.60-0.96)
ALLIANCE	2.9	3.2		0.90 (0.58-1.42)
CARDS	1.5	2.8		0.53 (0.31-0.90)
PROVE-IT	1.0	0.9		1.09 (0.59-2.01)
A to Z	1.2	1.6		0.79 (0.48-1.29)
ASCOT-LLT	1.7	2.4		0.73 (0.56-0.96)
ALLHAT-LLT	4.0	4.5		0.91 (0.76-1.09)
GREACE	1.2	2.1		0.53 (0.24-1.18)
HPS (with no prior CVD)	3.2	4.8		0.67 (0.57-0.77)
PROSPER	4.7	4.5		1.04 (0.82-1.31)
MIRACL	0.8	1.6		0.50 (0.25-1.00)
GISSI	0.9	0.9		1.05 (0.56-1.96)
AFCAPS-TexCAPS	0.4	0.5		0.82 (0.41-1.67)
LIPID (with no prior CVD)	3.3	3.9		0.84 (0.67-1.05)
Post-CABG	3.6	2.4		1.12 (0.58-2.18)
CARE (with no prior CVD)	1.9	2.8		0.67 (0.44-1.01)
WOSCOPS	1.4	1.5		0.90 (0.61-1.34)
SSSS	2.5	3.5		0.72 (0.51-1.01)
Subtotal: p<0.0001				**0.81 (0.75-0.87)**

Log scale 0.1 0.2 0.5 1 2 5 10

Amarenco P, Labreuche J. Lancet Neurol. 2009;8:453–463. CVD, cerebrovascular disease

Giảm biến cố bệnh mạch vành có ý nghĩa khi giảm nồng độ LDL-c với statin



A secondary to MI/ACS, except for SPARCL (secondary to stroke or TIA).ACS, acute coronary syndrome; MI, myocardial infarction.
LaRosa JC, et al. N Engl J Med 2005;352:1425–1435; Cannon CP, et al. N Engl J Med 2004;350:1495–504; Amarenco P, et al. N Engl J Med 2006;355:549–59

ASCOT-LLA (Anglo-Scandinavian Cardiac Outcomes Trial—Lipid Lowering Arm)

Patient population

- 10 305 patients with hypertension and mild-to-moderate elevations in total-C
- No history of CHD
- ≥3 other CHD risk factors*
- BP ≥160/100 mmHg
- Aged 40–79 years

4-week run-in period

Only patients with non-fasting total-C of ≤ 250 mg/dL (6.5 mmol/L) not receiving statin or fibrate treatment were randomized to receive treatment

Double-blind period

- Atorvastatin 10 mg (n=5168)
- Placebo (n=5137)

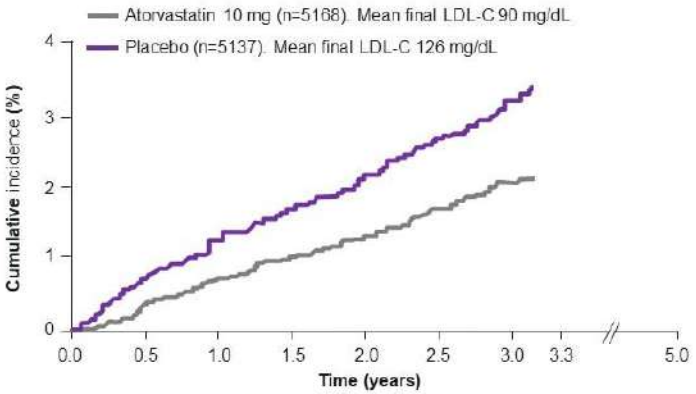
3.3-year median follow-up

Primary endpoint: composite of nonfatal MI and fatal CHD

Sever PS, et al. Lancet 2003;361:1149–1158.
*Left-ventricular hypertrophy, other specified abnormalities on electrocardiogram, type 2 diabetes, peripheral arterial disease, previous stroke or transient ischemic attack, male sex, age ≥55 years, microalbuminuria or proteinuria, smoking, ratio of plasma Total-C to HDL-C ≥6 or higher, or premature family history of CHD.BP, blood pressure; MI, myocardial infarction; total-C, total cholesterol

ASCOT-LLA: Atorvastatin giảm biến cố tim mạch ở bệnh nhân nguy cơ cao có kèm tăng huyết áp

Incidence of nonfatal MI and fatal CHD over 3.3 years[1]



ASCOT-LLA was stopped 2 years early due to significant CV benefits with atorvastatin

	Nonfatal MI[2]	Revascularization[2]
36% RRR	HR 0.64 95% CI 0.50–0.83 (p=0.0005) ARR=1.1% NNT 91 over 3.3 years	
45% RRR	HR 0.55 95% CI 0.40–0.75 (p=0.0002)	
42% RRR		(p=0.0002)

ASCOT-LLA, Anglo-Scandinavian Cardiac Outcomes Trial–Lipid-Lowering Arm
CI, confidence interval; HR, hazard ratio; NNT, number needed to treat; RRR, relative risk reduction

1. Sever PS, et al. Lancet 2003;361:1149–1158.
2. Data on file

CARDS : Collaborative Atorvastatin Diabetes Study

CARDS was a multicenter, randomized, double-blind study

Patient population

- Type 2 diabetes, no clinically evident CHD
- ≥1 other CHD risk factor (smoking, hypertension, albuminuria, retinopathy)
- LDL-C ≤160 mg/dL
- TG ≤600 mg/dL
- Aged 40–75 years

n=2838

4.0-year median follow-up

- Atorvastatin 10 mg
- Placebo

3.9-year median follow-up

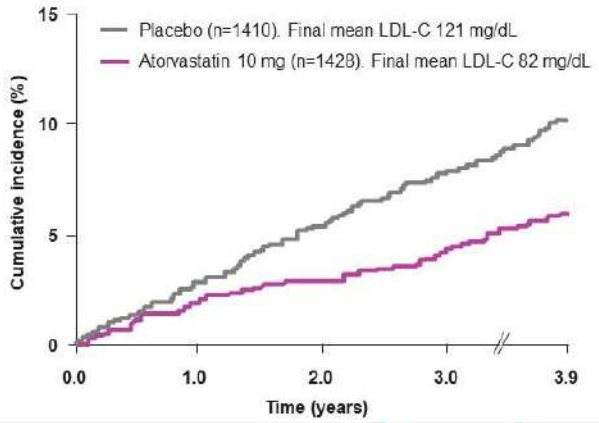
Primary endpoint: Time to first occurrence of acute CHD events (ie MI including silent MI, unstable angina, acute fatal CHD, resuscitated cardiac arrest), coronary revascularization, or stroke

CARDS, Collaborative AtoRvastatin Diabetes Study; TG, triglyceride
Colhoun HM, et al. Lancet 2004;364:685–696.

CARDS: Atorvastatin giảm có ý nghĩa nguy cơ tim mạch ở bệnh nhân đái tháo đường

CARDS was stopped ~2 years early due to significant CV benefits with atorvastatin

Incidence of major CV events*



*Primary endpoint

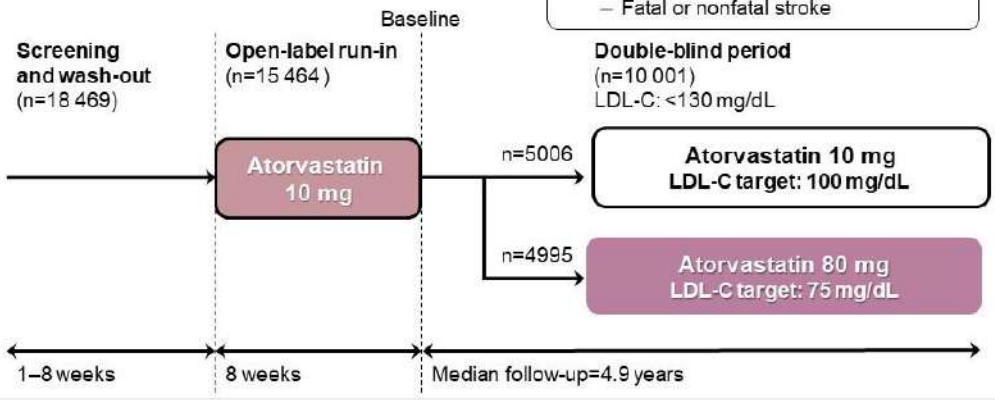
	Stroke	Fatal/ nonfatal MI
	37% RRR	**48%** RRR	**42%** RRR
	95% CI 17%–52% (p=0.001)1	HR=0.52 95% CI 0.31–0.89 (p=0.016)2	HR=0.58 95% CI 0.39–0.86 (p=0.007)3
	ARR=3.2% NNT 31 over 2 years	ARR=1.3%	ARR=1.9%

1. Colhoun HM, et al. Lancet 2004;364:685–696;
2. Hitman GA, et al. Diabetes Med 2007;24:1313–1321;
3. Lipitor Highlights of US Prescribing Information, 2013.

TNT (Treating to New Targets) so sánh atorvastatin cường độ cao và trung bình

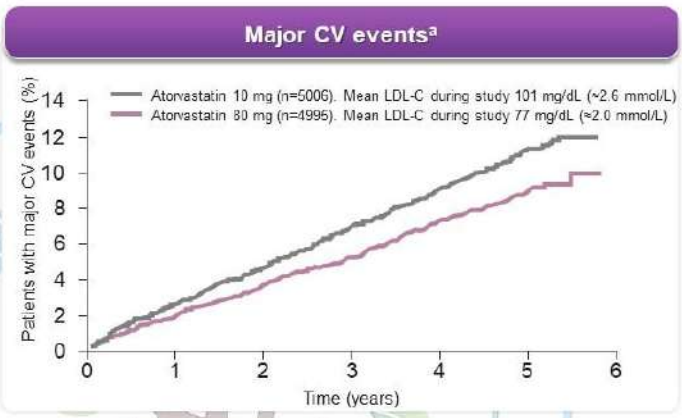
Patient population:
- CHD
- LDL-C: 130–250 mg/dL
- Triglycerides ≤600 mg/dL

Primary efficacy outcome measure:
- Occurrence of a major CV event:
  - CHD death
  - Nonfatal, non-procedure-related MI
  - Resuscitated cardiac arrest
  - Fatal or nonfatal stroke

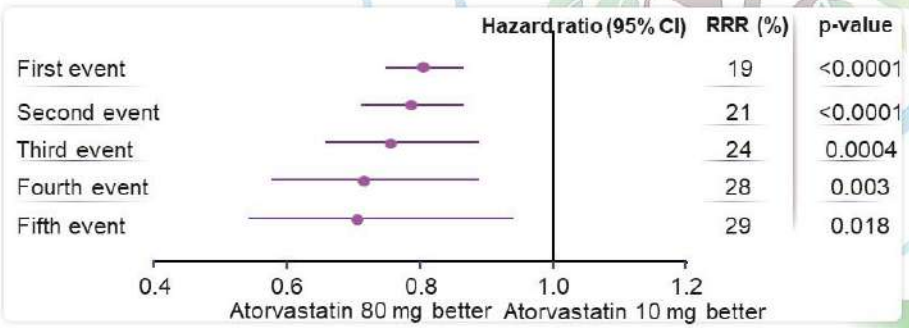


LaRosa JC, et al. N Engl J Med 2005;352:1425–1435

TNT (Treating to New Targets)



22%
RRR
HR=0.78
95% CI
0.69–0.89
(p<0.001)
ARR=2.2%
NNT 45
over 4.9 years



Effect of high- vs moderate-intensity atorvastatin on first and subsequent CV events

LaRosa JC, et al. N Engl J Med 2005;352:1425–1435

TNT: An toàn của atorvastatin ở bệnh nhân có bệnh lý mạch vành

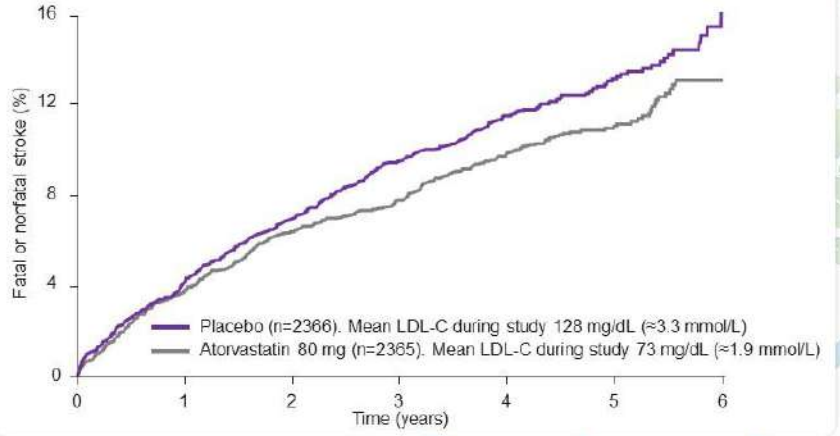
Patients (%)[1]	Atorvastatin 10 mg (n=5006)	Atorvastatin 80 mg (n=4995)	
Treatment-related AEs	5.8	8.1	p<0.001
Discontinuation due to treatment-related AEs	5.3	7.2	p<0.001
Treatment-related myalgia	4.7	4.8	p=0.72
Rhabdomyolysis	0.06	0.04	NA
Persistent elevation in ALT and/or AST[a]	0.2	1.2	p<0.001

TNT subgroup analysis in patients with diabetes (n=1501)2 and diabetes + CKD (n=546)3 also reported that atorvastatin 10 mg or 80 mg was generally well tolerated

aTwo consecutive measurements obtained 4–10 days apart that were >3 ×upper limit of normal.AE, adverse event; ALT, alanine aminotransferase; AST, aspartate aminotransferase. 1. LaRosa JC, et al. N Engl J Med 2005;352:1425–1435; 2. Shepherd J, et al. Diabetes Care 2006;29:1220–1226; 3. Shepherd J, et al. Mayo Clin Proc 2008;83:670–679

SPARCL: The Stroke Prevention by Aggressive Reduction of Cholesterol Levels

Fatal or nonfatal stroke[1]



Major coronary events[2]

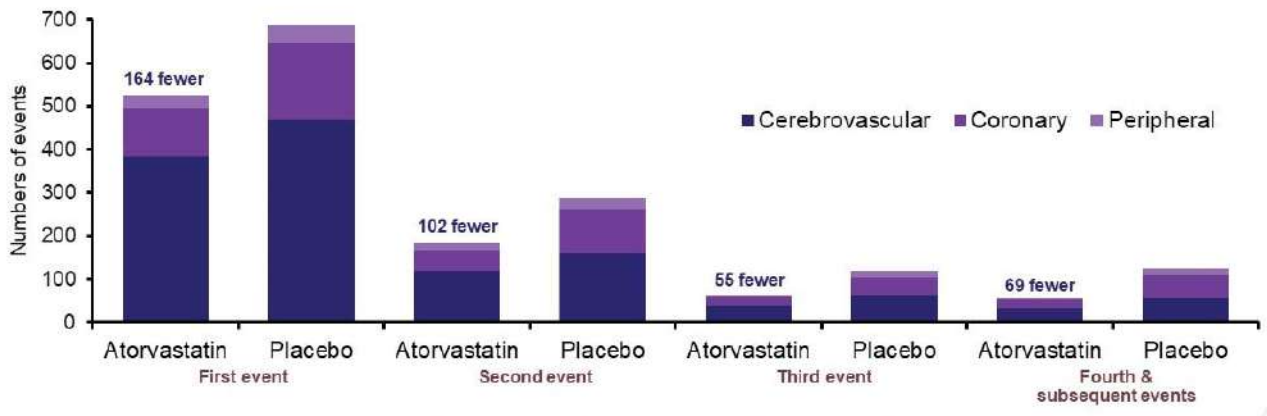
16%
RRR
HR=0.84
95% CI
0.71–0.99
(p=0.03)
ARR=2.2%
NNT 45
over 5 years

35%
RRR
HR=0.65
95% CI
0.49–0.87
(p=0.003)
ARR=1.7%
NNT 58
over 5 years

1. Amarenco P, et al. N Engl J Med 2006;355:549–559
2. Amarenco P et al. Stroke. 2010;41:126–130

SPARCL: Giảm có ý nghĩa các biến cố mạch máu với Atorvastatin liều cao

Reductions in events with atorvastatin were evident in each vascular territory



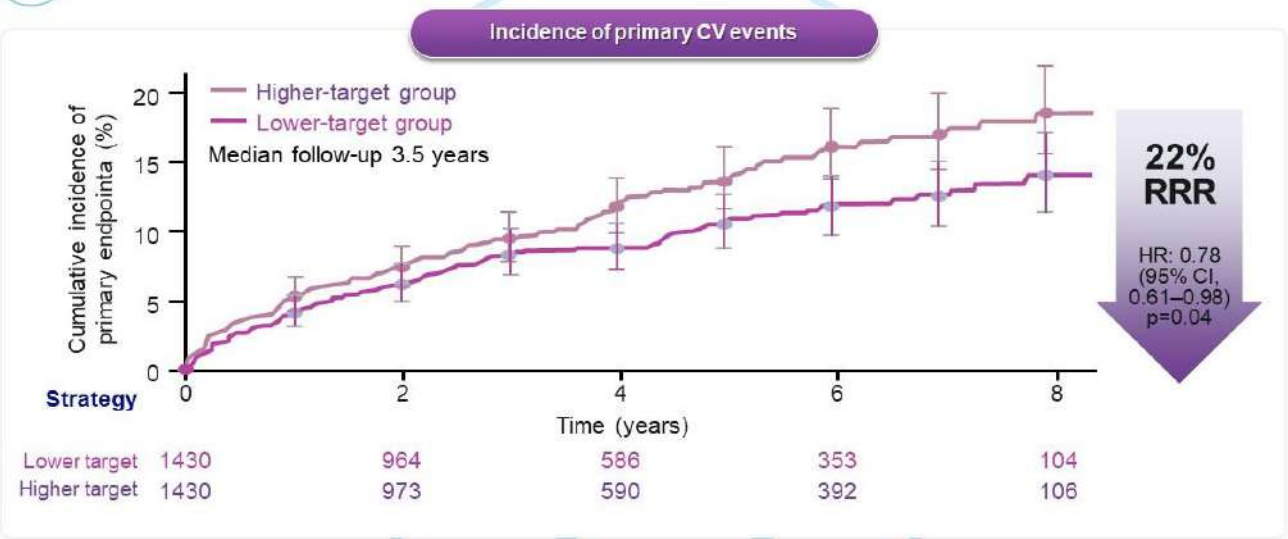
Szarek M et al. J Am Coll Cardiol 2020;75(17):2110–2118

SPARCL: An toàn của atorvastatin liều cao trên bệnh nhân nguy cơ cao đột quỵ/ TIA

Patients, n/N (%)	Placebo	Atorvastatin
Musculoskeletal AEs		
Myalgia	141/2366 (6.0)	129/2365 (5.5)
Myopathy	7/2366 (0.3)	7/2365 (0.3)
Rhabdomyolysis	3/2366 (0.1)	2/2365 (0.1)
ALT or AST >3 × ULN[a]	11/2366 (0.5)	51/2365 (2.2)[b]
CPK >10 × ULN[a]	0/2366 (0.0)	2/2365 (0.1)

aPersistent = 2 consecutive measurements bp<0.001 vs. placebo ULN, upper limit of normal
Amarenco P, et al. N Engl J Med 2006;355:549–559

Treat Stroke to Target (TST) trial



aPrimary endpoint is a composite of: Nonfatal cerebral infarction or stroke of undetermined origin, Nonfatal myocardial infarction, Hospitalization for unstable angina followed by urgent coronary-artery revascularization, TIA treated with urgent carotid revascularization Cardiovascular death, including unexplained sudden death
The I bars indicate 95% confidence intervals
TIA, transient ischemic attack

Amarenco P et al. N Engl J Med 2020;382:9–19

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- 3. KẾT LUẬN

Statins remain first-line for CVD prevention

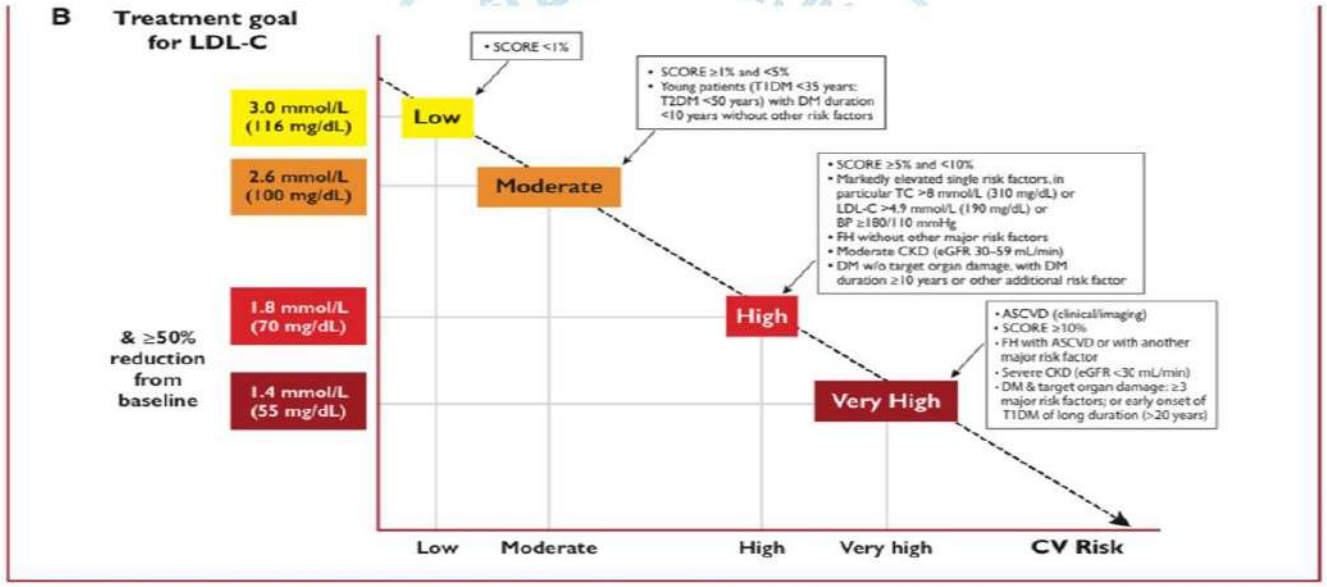
Lifestyle is the Foundation of ASCVD Risk Reduction

Clinical ASCVD LDL-C ≥190 mg/dL Higher Risk Diabetes Primary prevention ≥20% 10-year ASCVD risk	High intensity statin (Titrated up to highest tolerated dose to reach LDL-C goals for specific levels of risk)
Lower Risk Diabetes Primary prevention ≥7.5 to <20% 10-year ASCVD risk[a,b]	Moderate intensity statin
Primary prevention 5 to <7.5% 10-year ASCVD risk[a]	Moderate intensity statin

aFavors statin if risk enhancers present, but if reluctant to start statin, consider CAC. ASCVD, atherosclerotic cardiovascular disease; CAC, coronary artery calcium
Grundy SM et al. Circulation. 2019;139:e1082–e1143

ESC guideline

ESC/EAS guidelines recommend statins as the first-line treatment of dyslipidemia



https://www.escardio.org/Guidelines/Clinical-Practice-Guidelines/Dyslipidaemias-Management-of Accessed in 2024

Recommendations for pharmacological low-density lipoprotein cholesterol lowering

Recommendations	Class[a]	Level[b]
It is recommended that a high-intensity statin is prescribed up to the highest tolerated dose to reach the goals set for the specific level of risk.[32,34,38]	I	A
If the goals[c] are not achieved with the maximum tolerated dose of a statin, combination with ezetimibe is recommended.[33]	I	B
For primary prevention patients at very-high risk, but without FH, if the LDL-C goal is not achieved on a maximum tolerated dose of a statin and ezetimibe, a combination with a PCSK9 inhibitor may be considered.	IIb	C
For secondary prevention, patients at very-high risk not achieving their goal[c] on a maximum tolerated dose of a statin and ezetimibe, a combination with a PCSK9 inhibitor is recommended.[119,120]	I	A
For very-high-risk FH patients (that is, with ASCVD or with another major risk factor) who do not achieve their goal[c] on a maximum tolerated dose of a statin and ezetimibe, a combination with a PCSK9 inhibitor is recommended.	I	C
If a statin-based regimen is not tolerated at any dosage (even after rechallenge), ezetimibe should be considered.[197,265,353]	IIa	C
If a statin-based regimen is not tolerated at any dosage (even after rechallenge), a PCSK9 inhibitor added to ezetimibe may also be considered.[197,265,353]	IIb	C
If the goal[c] is not achieved, statin combination with a bile acid sequestrant may be considered.	IIb	C

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ASCVD = atherosclerotic cardiovascular disease; FH = familial hypercholesterolaemia; LDL-C = low-density lipoprotein cholesterol; PCSK9 = proprotein convertase subtilisin/kexin type 9.
[a]Class of recommendation.
[b]Level of evidence.
[c]For definitions see *Table 7*.

https://www.escardio.org/Guidelines/Clinical-Practice-Guidelines/Dyslipidaemias-Management-of Accessed Jan 2024

Other CVD prevention guidelines recommend high-intensity statin for specific patient groups

Guideline	Patient groups	Recommendation, LDL-C target
ESC/EAS[1]	Established CVD or very high risk High risk	Statin → <55 mg/dL (≈1.4 mmol/L) Statin → <70 mg/dL (≈1.8 mmol/L)
ADA[2]	CVD (any age) No CVD, Age 50–70 years OR ≥1 other risk factor No CVD, 40–75 years No CVD, 20–<40 years + ≥1 other risk factor	High-intensity[a] statin High-intensity[a] statin Moderate-intensity[b] statin Consider moderate-intensity[b] statin
JBS3[3]	Statin according to age and other risk factors Post-MI T2DM + CVD, proteinuria, or CKD (eGFR 30–60 mL/min)	Statin → <70 mg/dL Intensive statin
NICE[4]	Established CVD T1D T2DM + 10-year risk ≥10%	Atorvastatin 80 mg Consider atorvastatin 20 mg Atorvastatin 20 mg
KDIGO[5]	Primary or secondary prevention	Statin

a. Expected to reduce LDL-C by ≈≥50%. bExpected to reduce LDL-C by ≈30–<50%
ADA, American Diabetes Association; ESC/EAS, European Society of Cardiology/European Atherosclerosis Society; CKD, chronic kidney disease; JBS3, Joint British Societies; KDIGO, Kidney Disease: Improving Global Outcomes; NICE, National Institute for Health and Care Excellence (UK)
1. Mach F et al. Eur Heart J 2020;41:111–188; 2. American Diabetes Association. Diabetes Care 2020;43(Suppl. 1):S111–S134; 3. Joint British Societies. Heart 2014;100 Ii1–Ii167 DOI 10.1136/heartjnl-2014-305693; 4. NICE Lipid modification September 2016. Available at: http://www.nice.org.uk/Guidance/CG181 (last accessed October 2020); 5. KDIGO. Kidney Int Suppl 2013;3:271–279

Từ kết quả SPARCL & TST, AHA/ASA Guidelines 2021 khuyến cáo

Secondary prevention of stroke in very high-risk patients

Ischemic stroke + very high risk[a]

↓

Taking maximally tolerated statin + ezetimibe

↓

+ LDL-C >70 mg/dL (≈1.8 mmol/L)

↓

PCSK9 inhibitor (Class 2a, Level B-NR)

Secondary prevention of stroke in patients with ASCVD

Ischemic stroke or TIA + Atherosclerotic disease[a]

↓

Statin (Class 1, Level A)

↓

+ ezetimibe if needed (Class 1, Level A)

↓

LDL-C <70 mg/dL (≈1.8 mmol/L) (Class 1, Level A)

Secondary prevention of stroke in patients without CHD

Ischemic stroke
No known CHD
No cardiac sources of embolism
LDL-C >100 mg/dL (≈2.6 mmol/L)

↓

Atorvastatin 80 mg/day (Class 1, Level A)

Kleindorfer DO et al. Stroke. 2021;52:e364–e467 Please refer to the guideline for further information
CHD, coronary heart disease; LDL-C, low-density lipoprotein cholesterol; aintracranial, carotid, aortic, or coronary disease TIA, transient ischemic attack
aStroke + another major ASCVD or stroke plus multiple high-risk conditions
PCSK9, proprotein convertase subtilisin/kexin type 9; TIA, transient ischemic attack
Please refer to the guideline for further information

NỘI DUNG CHÍNH

1. TỔNG QUAN VỀ CÁC YẾU TỐ NGUY CƠ BỆNH LÝ TIM MẠCH TIẾN TRIỂN
2. VAI TRÒ STATIN TRONG PHÒNG NGỪA NGUYÊN PHÁT VÀ THỨ PHÁT BỆNH LÝ TIM MẠCH XƠ VỮA
   - Nghiên cứu nền tảng về vai trò statin trong phòng ngừa nguyên phát và thứ phát bệnh lý mạch máu.
   - Từ nghiên cứu đến khuyến cáo thực hành
   - **Lợi ích của statin trong các bệnh lý thận và gan nhiễm mỡ.**
- 3. KẾT LUẬN

PLANET I and II : Tác động của atorvastatin và rosuvastatin lên chức năng thận ở bệnh nhân mắc bệnh thận mạn có và không có đái tháo đường kèm theo.

PLANET I and II were multicenter, randomized, double-blind studies

Patient population

PLANET I
- Type I or II diabetes

PLANET II
- No diabetes

Both studies
- Moderate proteinureab
- Hypercholesterolemiac
- ACEis or ARBs for ≥3 months prior to screening

PLANET I: n=325[a]
PLANET II: n=220[a]

52 weeks follow-up

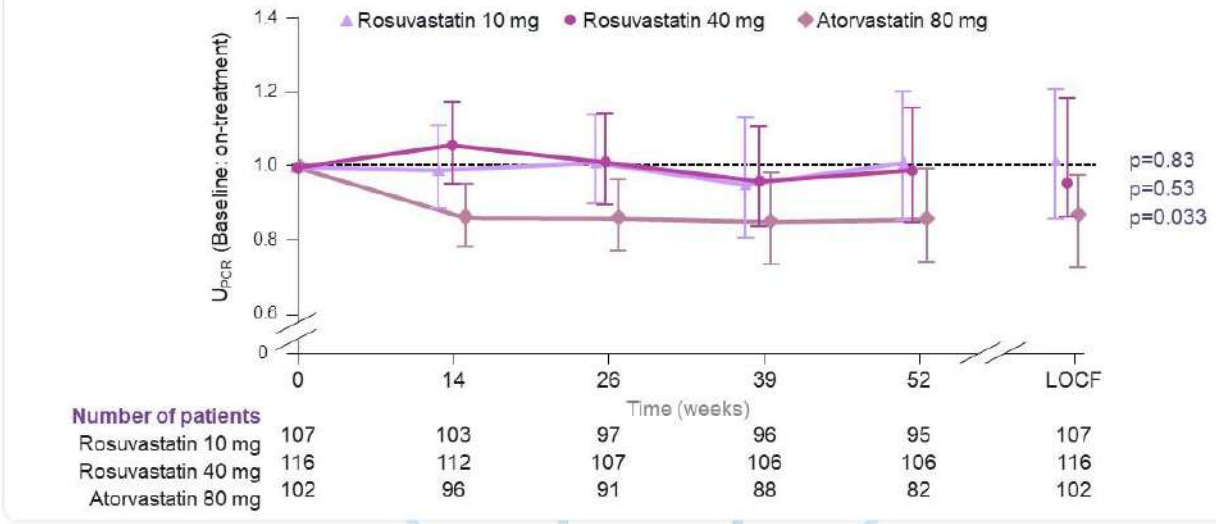
- Rosuvastatin 40 mg/day
- Rosuvastatin 10 mg/day
- Atorvastatin 80 mg/day

Primary endpoint: Within-group change in urinary protein/creatinine ratio (U_{PCR}) from baseline to Week 52 or last on-treatment observation carried forward (Week 52 LOCF)

- a)Intention-to-treat (ITT) populations b Urinary protein/creatinine ratio 500–5,000 mg/g cFasting LDL-C ≥90 mg/dL (2.33 mmol/L)
- ACEi, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker

- De Zeeuw D, et al. Lancet 2015;3:181–190

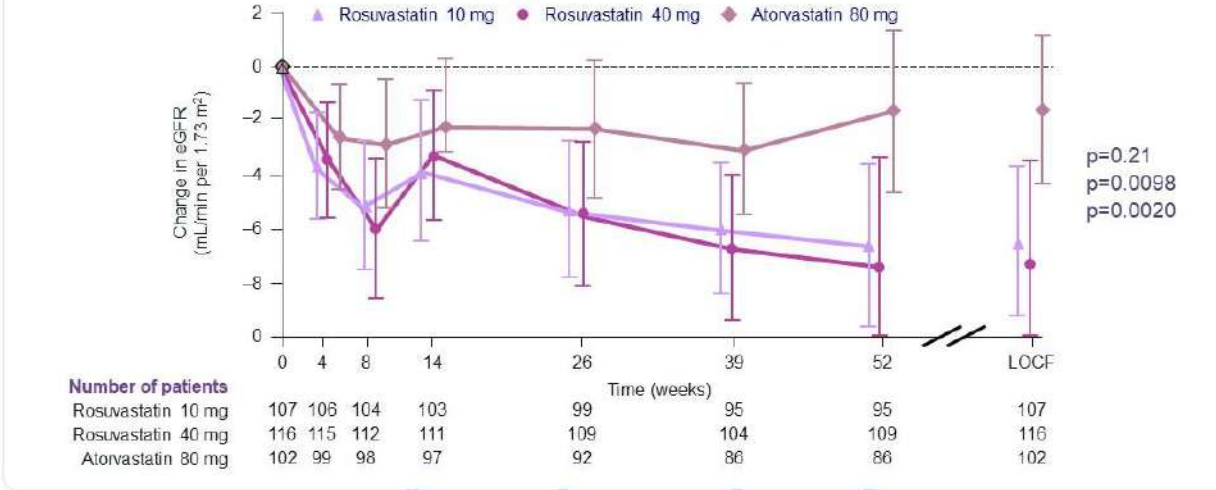
PLANET I: Tác động của Atorvastatin và Rosuvastatin trên giảm tỉ lệ Protein/creatinine niệu



- Data are mean baseline on-treatment ratios. Error bars are 95% CIs. LOCF marks 52-week data accounting for all patients in ITT population
- ITT, intent-to-treat; LOCF, last observation carried forward; UPCR, urinary protein/creatinine ratio
- p values are vs baseline

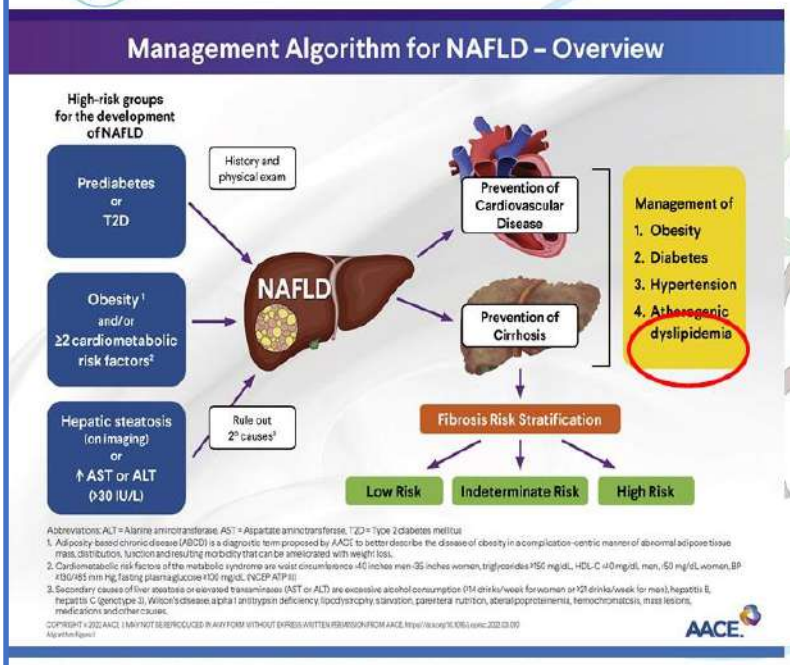
De Zeeuw D, et al. Lancet 2015;3:181–190

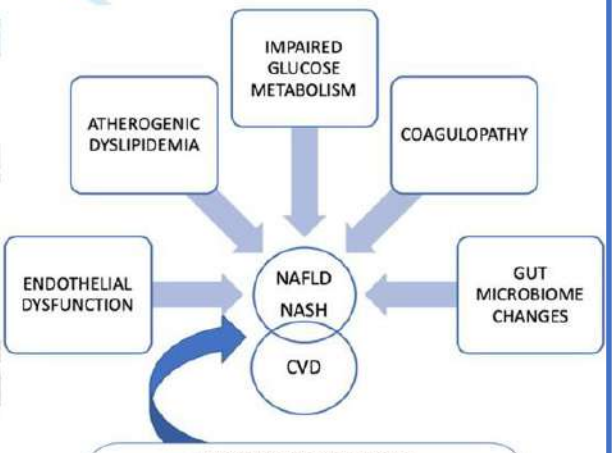
PLANET I: Tác động của Atorvastatin và Rosuvastatin trên thay đổi eGFR



- Data are mean baseline on-treatment ratios. Error bars are 95% CIs. LOCF marks 52-week data accounting for all patients in ITT population
- p values are vs baseline
- De Zeeuw D, et al. Lancet 2015;3:181–190

Lợi ích của Statin trong bệnh cảnh gan nhiễm mỡ



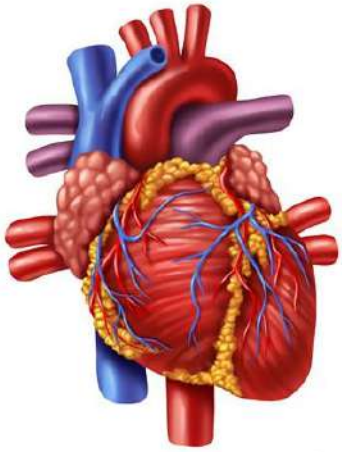


STATINS MODULATE:

-Small Guanine Triphospate Binding Proteins
- Kupffer Cell Crown-Like Structures
-Paraoxonase 1
-Stellate Cells
-Liver Endothelial Cells
-Proinflammatory factors

https://doi.org/10.1016/j.eprac.2022.03.010 Accessed Jan 2024

KẾT LUẬN



Atherosclerosis is caused by the trapping and retention of apoB-containing lipoproteins within the artery wall

- As more particles are trapped in the artery wall over time – atherosclerotic plaque develops slowly and progresses

Therefore, LDL and other apoB-containing lipoproteins have both a causal and a cumulative effect on the risk of atherosclerosis

- Cumulative exposure determines plaque burden and corresponding ASCVD risk

Thus, the risk of atherosclerosis and the benefit of lipid lowering therapies is determined by BOTH the magnitude and duration of exposure to LDL and other apoB-containing lipoproteins

"Lower is better, Earlier is better, and Longer is better"

BS. CKII. Trần Thị Thanh Trúc - Bệnh viện Đa Khoa Tâm Anh

HỘI NGHỊ KHOA HỌC THƯỜNG NIÊN NĂM 2024 34