



LIPOPROTEIN(a) IN CARDIOVASCULAR DISEASE

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LIPOPROTEIN(a)

Molecular Architecture: The Weaponized LDL

Standard LDL Particle

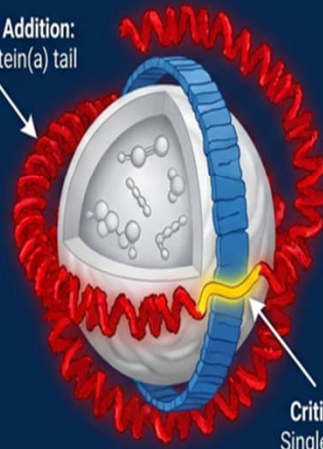
Cholesteryl esters,
phospholipids, and
triglycerides



Apolipoprotein B-100
(ApoB-100)

Lipoprotein(a) Particle

The Lethal Addition:
Apolipoprotein(a) tail



Critical Link:
Single covalent
disulfide bridge

Clinical Insight: The presence of the Apo(a) chain transforms a standard LDL particle into a highly toxic, multi-weaponized vascular threat.

THE GENETIC ABSOLUTE

GENETIC DETERMINATION

Levels are 70% - 90% determined by the LPA gene on chromosome 6.



LIFELONG STABILITY

Levels lock in and remain permanently stable over the patient's lifetime beginning at age 5.



CLINICAL STATUS

An independent, causal, and progressive risk factor for ASCVD.



Independent

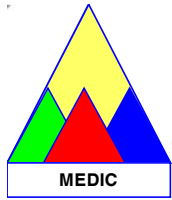


Causal

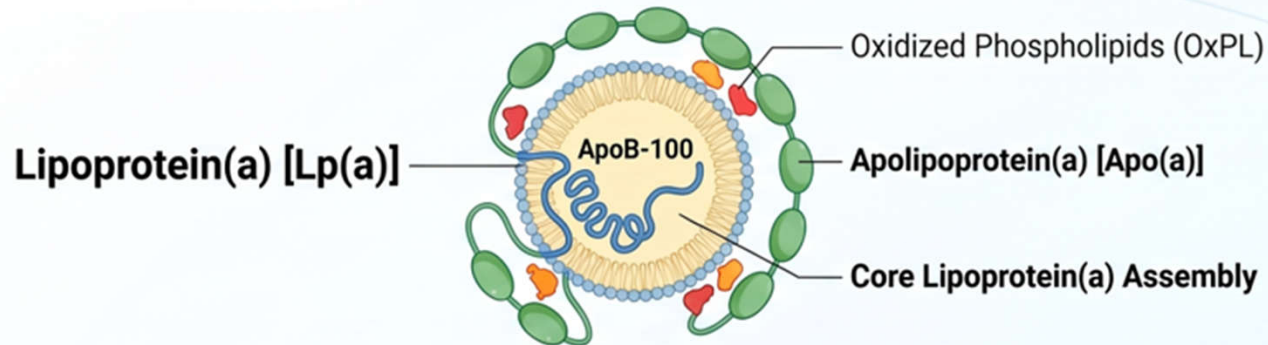


Progressive

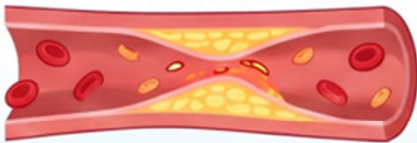
ASCVD : Atherosclerotic CardioVascular Disease



Lipoprotein(a): Pathogenic Mechanisms, and Clinical Impact



1. Atherogenesis (The Artery)



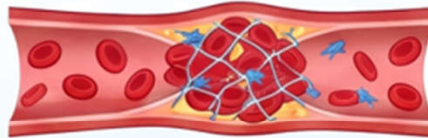
Promotes accelerated arterial atherosclerosis, leading to heart attack, stroke, and peripheral artery disease (PAD).

Clinical Outcomes



Heart Attack, Stroke, PAD

2. Thrombosis (The Clot)



Decreases fibrinolysis via Apo(a), impairing clot breakdown and increasing ischemic risk.

Clinical Outcomes



Ischemic Risk, Clot Formation

3. Calcification (The Valve)



OxPL transforms valvular interstitial cells into osteogenic-like cells, causing aortic stenosis and requiring surgical replacement (AVR).

Clinical Outcomes



Aortic Stenosis, AVR



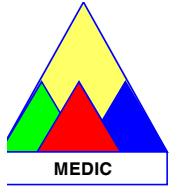
An estimated
20% to 30%
of people
worldwide have
high levels
of plasma
lipoprotein(a)



The Hidden Culprit of Residual Risk

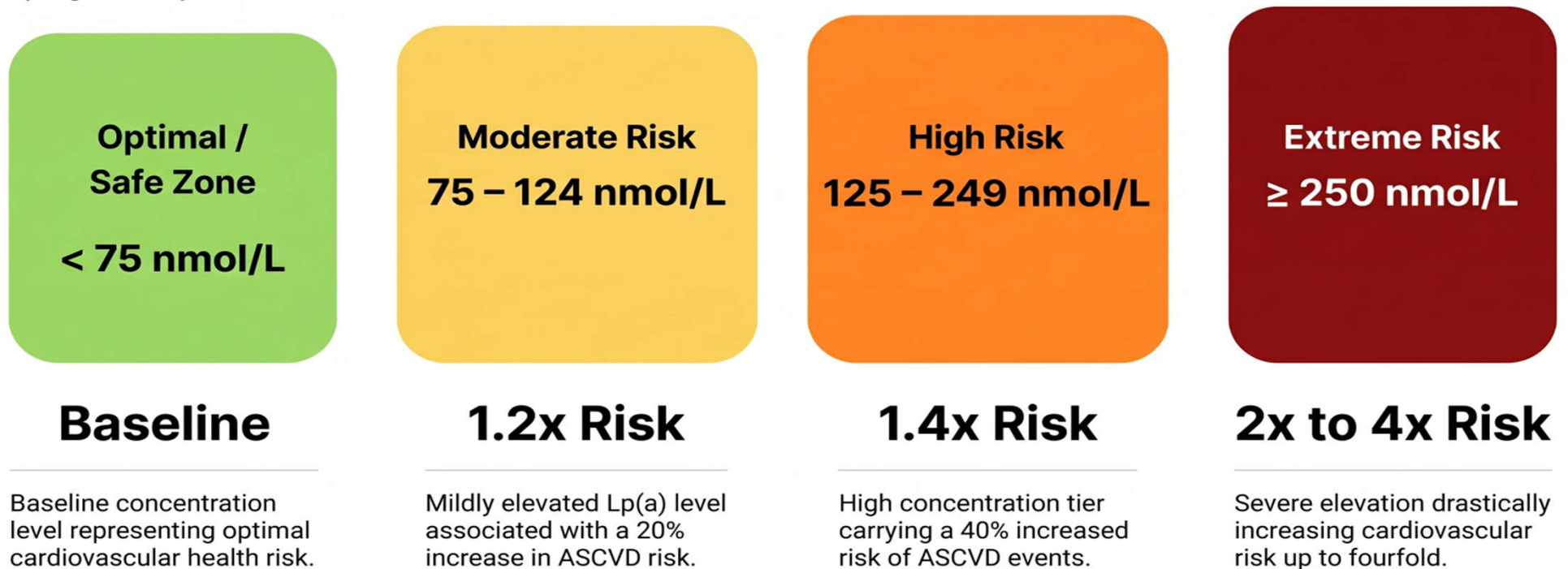
LDL-C (Controlled)

**Lp(a): Silent Inflammation
& Thrombosis**



ACC Guidelines 2026: Lp(a) Concentration & ASCVD Risk Stratification

The 2026 ACC Guidelines stratify Lipoprotein(a) [Lp(a)] blood concentration into four distinct tiers. As Lp(a) levels increase from baseline to extreme concentrations, the relative risk of developing Atherosclerotic Cardiovascular Disease (ASCVD) scales up significantly.



ACC/AHA Multisociety Guideline on the Management of Dyslipidemia. Journal of the American College of Cardiology (JACC), Vol. 87, No. 11, 2026



Non-Genetic Influences on Lipoprotein(a) Levels

Increase

Renal Diseases Drive Highest Increases



Nephrotic syndrome increases Lp(a) levels by **3–5x**

Peritoneal dialysis causes a **2x** increase



Hormonal Changes & Pregnancy Double Levels



Growth hormone therapy causes a **2x** increase in concentration



Pregnancy causes a **2x** increase in concentration

Decrease

Targeted Therapies & Diets Reduce Levels



Tocilizumab reduces levels by **30%–40%**

Postmenopausal HRT reduces levels by **~25%**



No / Minimal Change

Fasting & Exercise Show Minimal Impact



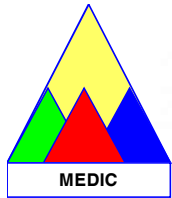
Fasting exerts no or minimal effect



Physical activity exerts no or minimal effect



Endogenous sex hormones and **Menopause** exert no or minimal effect



Lp(a) Screening Indications: ESC 2019 vs. ACC 2026 Guidelines

Clinical guidelines for Lipoprotein(a) [Lp(a)] screening have evolved from conditional consideration in specific high-risk adults to strong, universal adult screening and family cascade testing recommendations.

ESC 2019 Guidelines



Targeted Lifetime Risk Screening

Lp(a) Measurement



Measurement should be considered at least once in adults to detect inherited levels >180 mg/dL.

Targeted Lifetime Risk Screening



Identifies adults with ASCVD risk equivalent to heterozygous familial hypercholesterolaemia.

ACC 2026 Guidelines



Universal Adult Screening

Lp(a) Measurement



Measurement of Lp(a) concentration is recommended at least once in all adults for ASCVD risk assessment.

Family Cascade Testing



Cascade testing of first-degree relatives recommended for individuals with FH, premature ASCVD, or high Lp(a).

Pediatric Lipoprotein(a) Screening & Risk Guide

CLINICAL INDICATIONS



Suspected or Confirmed Familial Hypercholesterolemia (FH)

Recommended for youth with clinically suspected or genetically confirmed FH.



Family History of Premature ASCVD

Indicated if 1st-degree relatives had ASCVD at <55 years (men) or <65 years (women).



High Family Lp(a) or Unexplained Ischemic Stroke

Applies to cascade screening (parent/sibling with elevated Lp(a)) or unexplained stroke.

OPTIMAL AGE WINDOWS FOR TESTING



Level Stabilization at Age 5

Lp(a) concentrations reach adult-equivalent stability starting at age 5.



Golden Window: Ages 9–11 Years

Optimal time to test high-risk children alongside universal pediatric lipid screening.

Lp(a) RISK CONCENTRATION LEVELS



LOW RISK THRESHOLD

Concentration under 75 nmol/L (< 30 mg/dL).



INTERMEDIATE RISK THRESHOLD

Concentration between 75 and 125 nmol/L (30–50 mg/dL).



HIGH RISK THRESHOLD

Concentration at or above 125 nmol/L (≥ 50 mg/dL).



LP(a) 80 YEARS CVD RISK (ESC 2025)

http://www.eas-society.org/LPA_risk_and_benefit_algorithm

Enter your health information below

Cholesterol units:
☒ mmol/L ☐ mg/dL

Sex

Age (ages 30-75)

Cholesterol
Total Cholesterol (mmol/L) (range 3.5 - 8.0)

LDL Cholesterol (mmol/L) (range 2.0 - 5.0)

HDL Cholesterol (mmol/L) (range 0.6 - 2.8)

Systolic Blood Pressure (mmHg) (range 90 - 200)

Are you taking a medicine to lower blood pressure?

Height units:
☒ cm ☐ in

Weight units:
☒ kg ☐ lbs

Height (cm)

Weight (kg)

Your BMI is calculated as:
BMI:

Do you have diabetes?

Do you currently smoke?

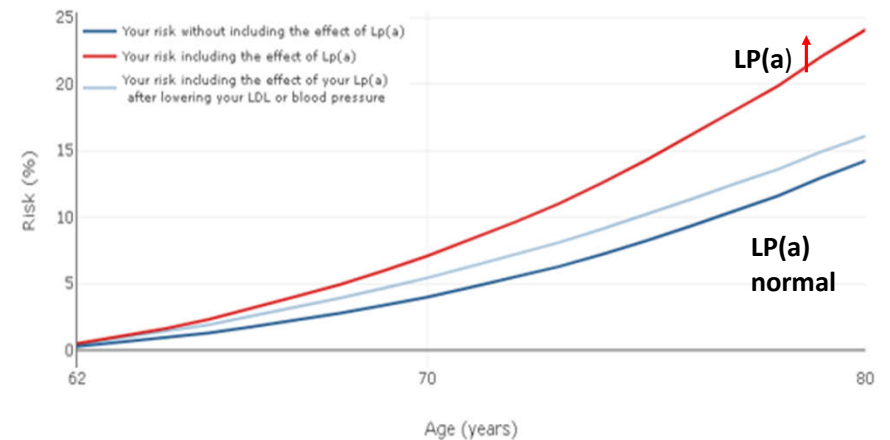
Have you ever smoked?

Has anyone in your family had a heart attack or stroke?

Lp(a) units:
☒ nmol/L ☐ mg/dL

Enter your Lp(a) level below to see how much it increases your risk of heart attack and stroke.

Your risk of having a heart attack or stroke



With an Lp(a) level of 213 nmol/L, your estimated risk of having a heart attack or stroke up to age 80 changes from 14.2% to:

24.1%

With an Lp(a) of 213 nmol/L and an estimated risk of 24.1%, lowering your LDL by 2 mmol/L beginning at age 62 will reduce your risk of having a heart attack or stroke to:

16.1%

How much should I lower my LDL? - 2



How much should I lower my SBP? - 0





Lp(a) Therapy: Comparative Impact of Lipid-Lowering Treatments

Differential efficacy of standard vs. advanced therapies on LDL-C and Lipoprotein(a) [Lp(a)] levels, specifically highlighting targeted drug options.

Conventional Lipid-Lowering Therapies



Standard therapies offer 0% Lp(a) reduction



Lifestyle modification



High-intensity statins



Ezetimibe

0%
Lp(a)
Reduction



Show zero effect on Lp(a).

Targeted & Novel Lp(a) Therapies

Advanced treatments for significant Lp(a) reduction



PCSK9 Inhibitors

PCSK9 Inhibitors yield moderate Lp(a) reduction



Massive
LDL-C Drop

-20% to -30%

Lp(a) Reduction

Specific Drug Names: **Alirocumab, Evolocumab**



Lipoprotein Apheresis

Lipoprotein Apheresis yields transient reduction



-60% to -80%

Lp(a) Reduction (Transient)



RNA Therapies (ASO/siRNA)

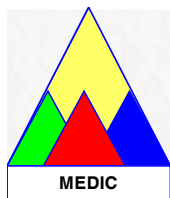
RNA Therapies provide maximum sustained Lp(a) drop

Neutral / Slight
LDL-C Drop

-70% to -95%

Sustained Lp(a) Reduction

Specific Drug Names: **Pelacarsen, Olpasiran**



4-Step Clinical Workflow for Managing Elevated Lp(a)

Outline the clinical management protocol for patients with elevated Lipoprotein(a) based on international guidelines (ACC/AHA, ESC/EAS, CCS).

Step 1: Re-Stratify Risk Tier

Upgrade baseline risk tier using **PREVENT** or **SCORE2** models with Lp(a) as a risk enhancer.



	Risk Stratum	Target LDL-C Goal (Post-Restratification)
1	High Risk	< 70 mg/dL (1.8 mmol/L)
2	Very High Risk	< 55 mg/dL (1.4 mmol/L)
3	Extreme Risk	< 40 mg/dL (1.0 mmol/L)

Step 2: Combine Lipid-Lowering Therapies

Maximize **Statin + Ezetimibe + PCSK9 inhibitor** (lowers Lp(a) by 20–30%).



STATIN



EZETIMIBE



PCSK9 INHIBITOR

LOWERS Lp(a) by 20–30%



Step 3: Manage Thrombotic Risk

Consider **low-dose Aspirin** (75–100 mg/day) to mitigate Lp(a)-driven clot risks.



MITIGATE Lp(a)-DRIVEN CLOT RISKS

Step 3: Optimize Comorbidities

Strictly control blood pressure (<130 mmHg), blood sugar (HbA1c), and active lifestyle.



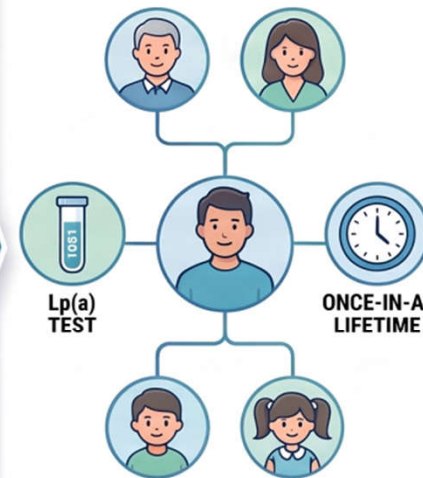
<130 mmHg

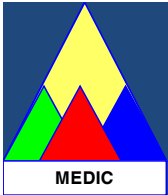


ACTIVE LIFESTYLE

Step 4: Conduct Family Cascade Screening

Order a **once-in-a-lifetime Lp(a) test** for all first-degree relatives.





CASE 1

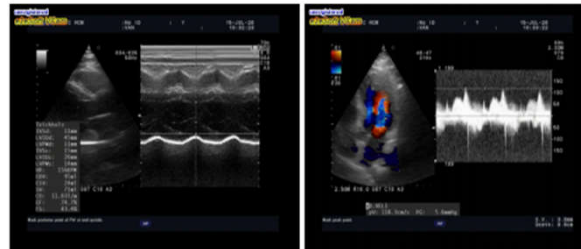
- 44F
- Chief complaint: Intermittent dyspnea.
- Past medical history: Hypertension
- BP 160/100 mmHg, P 85 bpm

VÙNG KHẢO SÁT : SIÊU ÂM TIM MÀU

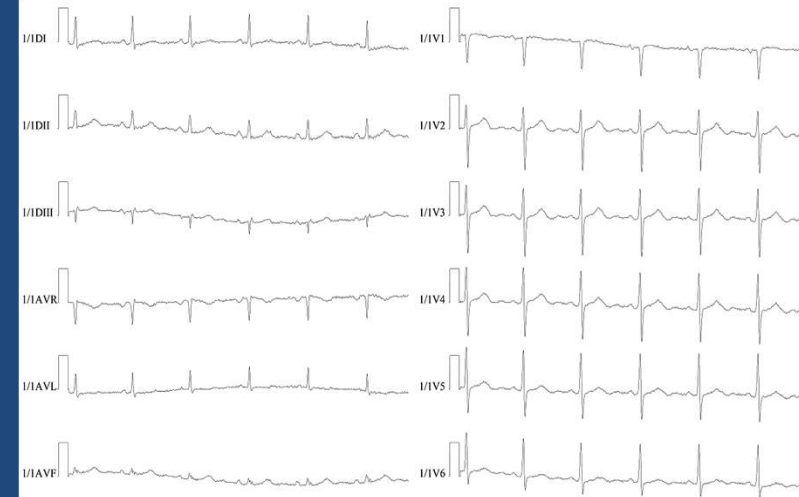
1-Thất trái dày nhẹ. Các buồng tim khác trong giới hạn bình thường LVDD= 45mm
Không rối loạn vận động khu trú. Chức năng tâm thu thất trái tốt EF= 74%(Teichholz)
Chức năng tâm thu thất phải bình thường TAPSE= 22mm

2-Van 2 lá dày, hở 1/4. Van động mạch chủ dày, hở 1/4
Không tăng áp động mạch phổi PAPs=28 mmHg
Động mạch chủ ngực kích thước bình thường

3- Không tràn dịch màng ngoài tim.



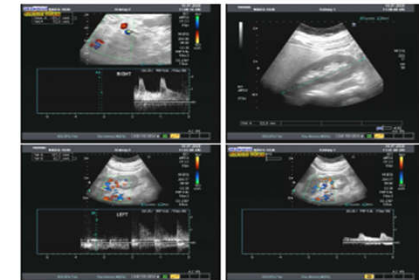
KẾT LUẬN: DẤY THẤT TRÁI NHẸ CÓ THỂ DO CAO HUYẾT ÁP
HỞ VAN 2 LÁ 1/4, HỞ VAN ĐỘNG MẠCH CHỦ 1/4



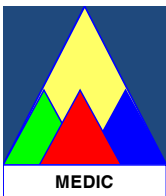
KẾT LUẬN : THEO DÕI TĂNG GÁNH THẤT TRÁI

VÙNG KHẢO SÁT : SIÊU ÂM DOPPLER MẠCH MÁU THẬN

- GAN: bờ đều, cấu trúc phản âm dày, giảm âm vùng sâu, xóa mờ mạch máu và đường mật trong gan, mờ vòm hoành, đồng dạng, không sang thương khu trú.
- MẬT: túi mật không sỏi. - Đường mật trong gan không dẫn.
- Ống mật chủ không sỏi, không dẫn.
- TỤY: Bình thường. LÁCH: không to.
- THẬN P- THẬN T: kích thước dài d=112-122 mm, tùy - vô phản biệt, không sỏi, không ứ nước, Động mạch thận (P) PSV=195 cm/s, Động mạch thận (T) PSV=177 cm/s, RI trong giới hạn bình thường.
- BÀNG QUANG: không sỏi, không bươu.
- Động mạch chủ bụng không phình.
- tử cung: nội mạc 8mm, thành sau có nhân xơ d=21x33 mm, buồng trứng không u.
- Ascites (-). Không hạch bệnh lý. - Không tràn dịch màng phổi.



KẾT LUẬN: Hẹp nhẹ động mạch thận (P>T)
Gan nhiễm mỡ, nhân xơ tử cung



Lp(a) 188 nmol/l, LDL-C 5.6 mmol/l

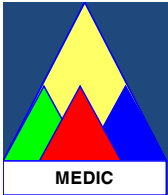
TÊN XÉT NGHIỆM	KẾT QUẢ	KHOẢNG THAM CHIẾU	MÃ QT
Calcium oxalate dihydrate	0	(0 - 6)	
Amor.Phosphate	0	(0 - 6)	
Uric acid	0	(0 - 6)	
Casts	1	(0 - 6)	
Epithelial Cells	33 H	(0 - 10)	
Bacteria	60	(0 - 130)	
III. SINH HOÁ - BIOCHEMISTRY			
HbA1C (HPLC) ¹ :	*		QTSH012
HbA1c (IFCC)	46.23	(20 - 38.80 mmol/mol)	
HbA1c (NGSP)	6.38	(4.0 - 5.70 %A1c) Quyết định 5481/QĐ-BYT 2020	
Glucose (mmol/L) (FPG) ¹	5.57	(3.90 - 5.60 mmol/L)	QTSH001
Glucose (mg/dL)	100.3	(70.2 - 100.8 mg/dL) Quyết định 5481/QĐ-BYT 2020	
IONOGRAMME ² :	*		QTSH067
Na	136.9	(130 - 145 mmol/L)	
K	3.96	(3.40 - 5.1 mmol/L)	
Ca	2.30	(2.1 - 2.80 mmol/L)	
Cl	99.64	(96 - 108 mmol/L)	
Uric Acid/Serum ¹	8.29 H	(Nam: 3.4 - 7.0; Nữ: 2.4 - 5.7 mg/dL)	QTSH014
*Độ Lọc Cầu Thận (CKD-EPI)	*		
Creatinin/Serum ²	0.689	(Nam: 0.6 - 1.3; Nữ: 0.5 - 1.1 mg/dL)	QTSH027
eGFR (CKD-EPI)	110	(≥ 90 mL/min/1.73 m ²)	
LDL Cholesterol ²	5.60 H	(<2.59; Ngưỡng: 2.59-4.13 mmol/L) (Cao: 4.14 - 4.91; Rất cao ≥ 4.92)	QTSH093
HDL Cholesterol ²	1.69	(≥1.55; Ngưỡng: 1.04-1.54 mmol/L) (Thấp: < 1.04)	QTSH084
Triglycerides ¹	1.81	(<1.70; Ngưỡng: 1.70-2.25 mmol/L) (Cao: 2.26 - 5.64; Rất cao ≥ 5.65)	QTSH015
LIPOPROTEIN_APO-B	168.0	(Nữ: 53.0 - 182.0 mg/dL)	QTSH097
Lipoprotein (a) (nmol/L)	188.0 H	(≤ 105 nmol/L)	
Lipoprotein (a) (mg/dL)	87.99 H	(≤ 50 mg/dL)	
GGT ¹	58.50 H	(Nam < 55 U/L, Nữ < 36 U/L)	QTSH004
SGOT (AST) ¹	37.68 H	(< 35 U/L)	QTSH005
SGPT (ALT) ¹	43.33 H	(< 30 U/L)	QTSH013
IV. MIỄN DỊCH - IMMUNOLOGY			
TSH u.sensitive (3rd G) ¹	1.34	(0.32 - 5 µIU/mL)	QTMD009



XQ Lồng Ngực Thẳng [Film]

Thành ngực	: Không có ảnh bất thường
Màng phổi	: Không có ảnh bất thường
Trung thất	: Không có ảnh bất thường
Tim	: Không có ảnh bất thường
Động mạch chủ	: bụng rộng
Huyết phế quản	: Không có ảnh bất thường
Phổi	: Không có ảnh bất thường
Cơ hoành	: Không có ảnh bất thường

Cảm nghĩ : BỆNH TIM MẠCH DO XƠ ĐỘNG MẠCH VÀ/HAY CAO HUYẾT ÁP
Đề nghị : kết hợp siêu âm tim, ECG



TREATMENT GOAL FOR LDL-C

LDL CHOLESTEROL 5.6MMOL/L

- SCORE2/SCORE2-OP $\geq 10\%$ and $< 20\%$
- Markedly elevated single risk factors, in particular TC > 8 mmol/L (310 mg/dL) or LDL-C > 4.9 mmol/L (190 mg/dL) or BP $\geq 180/110$ mmHg
- FH without other major risk factors
- Moderate CKD (eGFR 30–59 mL/min/1.73 m²)
- DM w/o target organ damage, with DM duration ≥ 10 years or other additional risk factor

**HIGH
RISK**

+

**Lp(a)
188 nmol/l**

**VERY HIGH
RISK**

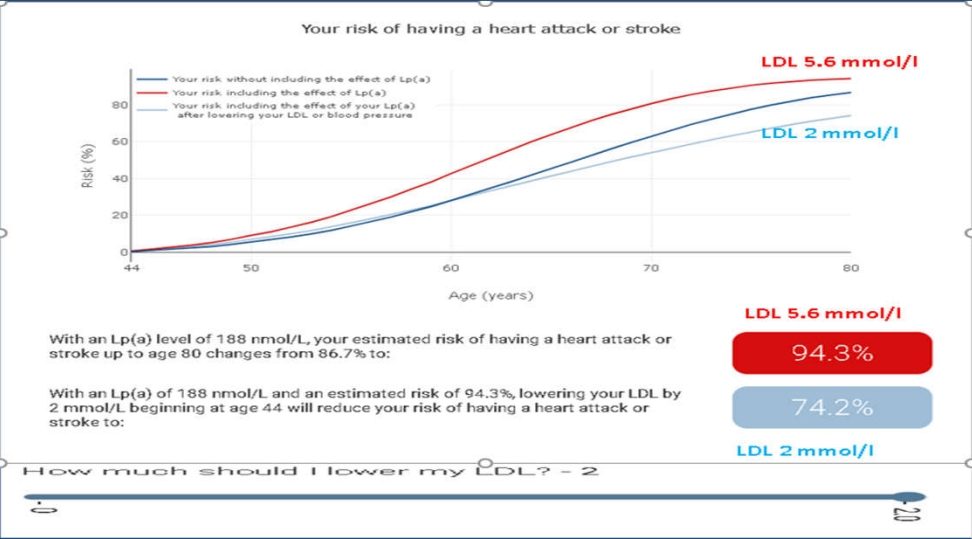
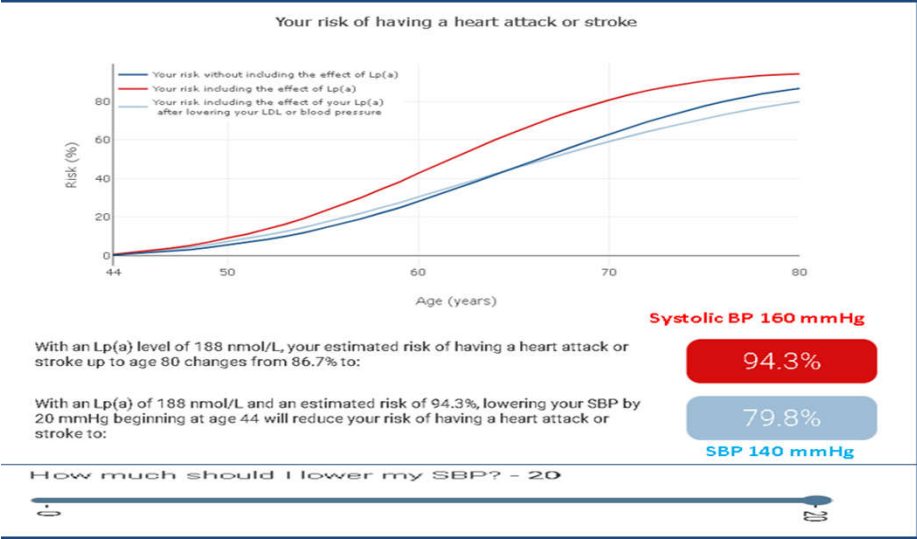
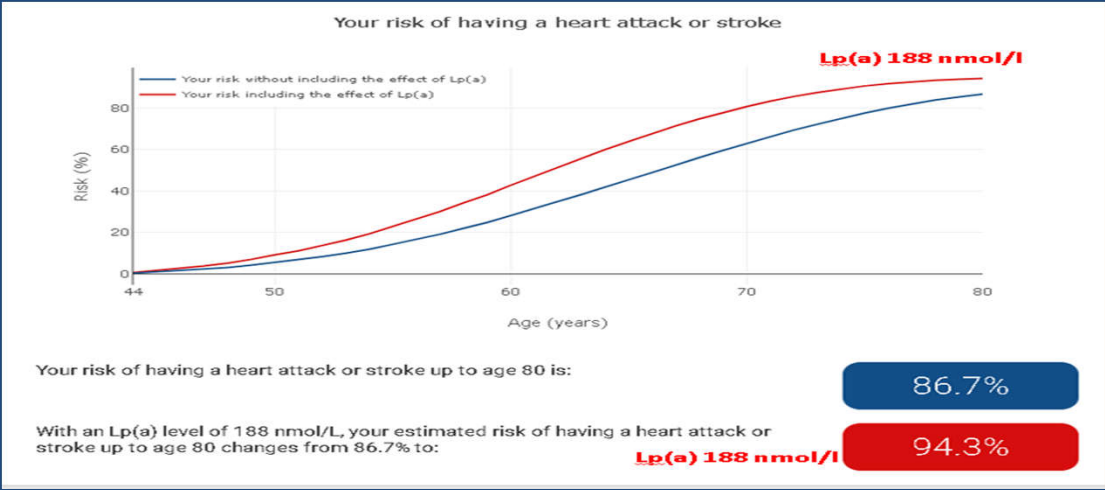
**< 1.4 mmol/L
(< 55 mg/dL)**

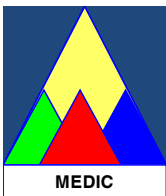
Treatment goal
for LDL-C

ESC 2025 GUIDELINE



LP(a) 80 YEARS CVD RISK (ESC 2025)





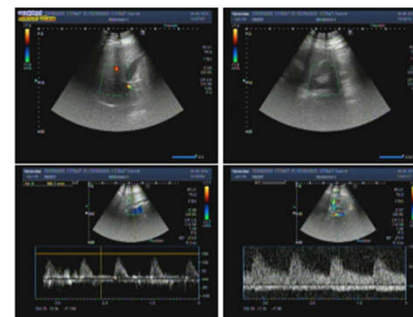
FAMILY SCREENING (F1: 22 F)

TÊN XÉT NGHIỆM	KẾT QUẢ	KHOẢNG THAM CHIẾU	MÃ QT
Epithelial Cells	10	(0 - 10)	
Bacteria	38	(0 - 130)	
III. SINH HOA - BIOCHEMISTRY			
HbA1C (HPLC) ¹ :	*		QTSH012
HbA1c (IFCC)	41.75	(20 - 38.80 mmol/mol)	
HbA1c (NGSP)	5.97	(4.0 - 5.70 %A1c) Quyết định 5481/QĐ-BYT 2020	
Glucose (mmol/L) (FPG) ¹	4.80	(3.90 - 5.60 mmol/L)	QTSH001
Glucose (mg/dL)	86.40	(70.2 - 100.8 mg/dL) Quyết định 5481/QĐ-BYT 2020	
hs CRP	1.72	(≤ 3 mg/L)	QTSH028
*Độ Lọc Cầu Thận (CKD-EPI)	*		
Creatinin/Serum ²	0.690	(Nam: 0.6 - 1.3; Nữ:0.5 - 1.1 mg/dL)	QTSH027
eGFR (CKD-EPI)	126	(≥ 90 mL/min/1.73 m ²)	
LDL Cholesterol ²	3.42	(<2.59; Ngưỡng: 2.59-4.13 mmol/L)	QTSH093
	.	(Cao: 4.14 - 4.91; Rất cao ≥ 4.92)	
HDL Cholesterol ²	1.27	(≥1.55; Ngưỡng: 1.04-1.54 mmol/L)	QTSH084
	.	(Thấp: < 1.04)	
Triglycerides ¹	3.03 H	(<1.70; Ngưỡng: 1.70-2.25 mmol/L)	QTSH015
	.	(Cao: 2.26 - 5.64; Rất cao ≥ 5.65)	
Cholesterol, Total ¹	5.43	(<5.18; Ngưỡng: 5.18-6.21 mmol/L)	QTSH003
	.	(Cao: ≥ 6.22)	
LIPOPROTEIN_APO-B	103.5	(Nữ: 53.0 - 182.0 mg/dL)	QTSH097
Lipoprotein (a) (nmol/L)	168.0 H	(≤ 105 nmol/L)	
Lipoprotein (a) (mg/dL)	78.82 H	(≤ 50 mg/dL)	
SGOT (AST) ¹	60.62 H	(< 35 U/L)	QTSH005
SGPT (ALT) ¹	104.1 H	(< 30 U/L)	QTSH013

Lp(a) 168 nmol/l

VÙNG KHẢO SÁT : SIÊU ÂM DOPPLER MẠCH MÁU THẬN

- GAN: bờ đều, cấu trúc GAN THẨM MỖ NẶNG , đồng dạng, không sang thương khu trú.
- MẬT: túi mật không sỏi. - Đường mật trong gan không giãn.
- Ống mật chủ không sỏi, không giãn.
- TỤY: Bình thường. LÁCH: không to.
- Không u thượng thận .THẬN P-T: không sỏi, không ú nước. DOPPLER ĐỘNG MẠCH THẬN HAI BÊN KHÔNG HẸP
- BÀNG QUANG: không sỏi, không bướu.
- Động mạch chủ bụng không phình.
- TC - BT không u.
- Ascites (-). Không hạch bệnh lý.



KẾT LUẬN: GAN THẨM MỖ. (ĐỘ 3)
DOPPLER ĐỘNG MẠCH THẬN HAI BÊN KHÔNG HẸP

VÙNG KHẢO SÁT : SIÊU ÂM TIM MÀU

- 1- Các buồng tim trong giới hạn bình thường LVDD=53 mm
Không rối loạn vận động khu trú.Chức năng tâm thu thất trái tốt EF=80 %(Teichholz)
Chức năng tâm thu thất phải bình thường TAPSE= 22mm
- 2- Van 2 lá dày, hở <1/4. Van động mạch chủ dày, hở <1/4
Không tăng áp động mạch phổi PAPs=28 mmHg
Động mạch chủ ngực kích thước bình thường
- 3- Không tràn dịch màng ngoài tim.

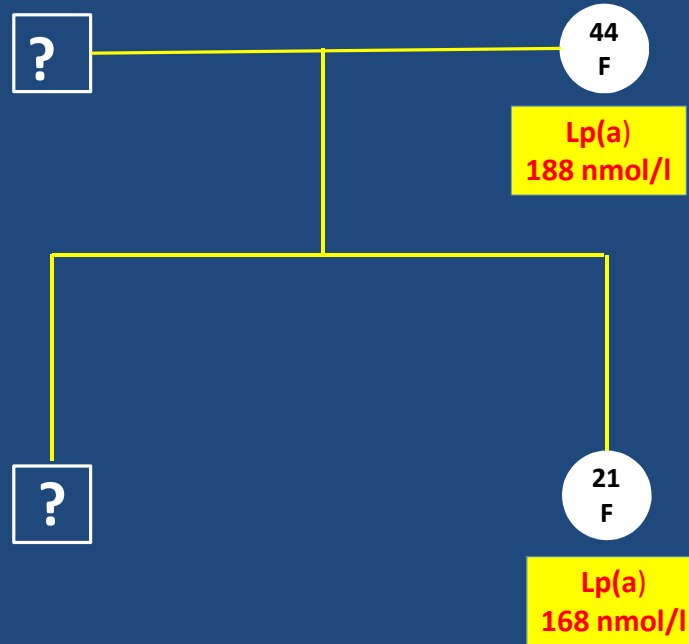


KẾT LUẬN: HỞ VAN 2 LÁ <1/4, HỞ VAN ĐỘNG MẠCH CHỦ <1/4

Đề nghị:



CASE 1





CASE 2

- 43M
- Medical check up
- PMH: Asthma, HBP, Gout
- BP:144/108 mmHg
- P: 78bpm

VÙNG KHẢO SÁT : SIÊU ÂM TIM MÀU

1- Dây thất trái nhẹ. Các buồng tim khác bình thường LVDd= 41mm
Không rối loạn vận động khu trú. Chức năng tâm thu thất trái tốt EF= 67%(Teichholz)
Chức năng tâm thu thất phải bình thường TAPSE= 22mm

2- Van hai lá dây hở 1/4; Van ĐM chủ 3 mảnh , dây hở 1/4
Không tăng áp phổi PAPS=25mmHg
Động mạch chủ ngực giãn đoạn lên d=38mm.

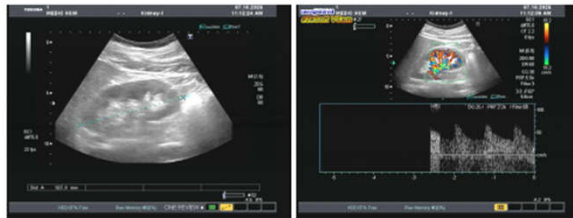
3- Không tràn dịch màng ngoài tim.



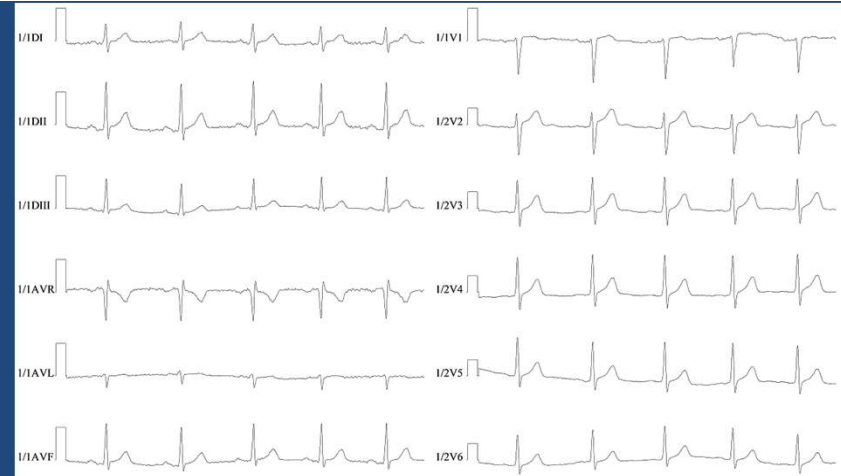
KẾT LUẬN : DÂY THẤT TRÁI NHẸ CÓ THỂ DO CAO HUYẾT ÁP
HỞ VAN HAI LÁ 1/4, HỞ VAN ĐM CHỦ 1/4

VÙNG KHẢO SÁT : SIÊU ÂM DOPPLER MẠCH MÁU THẬN

- GAN: bờ đều, cấu trúc phần âm dày nhẹ, đồng dạng, không sang thương khu trú.
- MẬT: túi mật có polyp 3mm - Đường mật trong gan không giãn.
- Ống mật chủ không sỏi, không giãn.
- TỤY: Bình thường. LÁCH: không to.
- THẬN P- THẬN T: kích thước dài d= 105-108mm, tùy - vỏ phân biệt, không sỏi, không ứ nước, Động mạch thận (P) PSV= 220cm/s, Động mạch thận (T) PSV=130 cm/s, RI trong giới hạn bình thường.
- BANG QUANG: không sỏi, không bướu.
- Động mạch chủ bụng không phình.
- Vùng chậu không ứ.
- Ascites (-). Không hạch bệnh lý. - Không tràn dịch màng phổi.



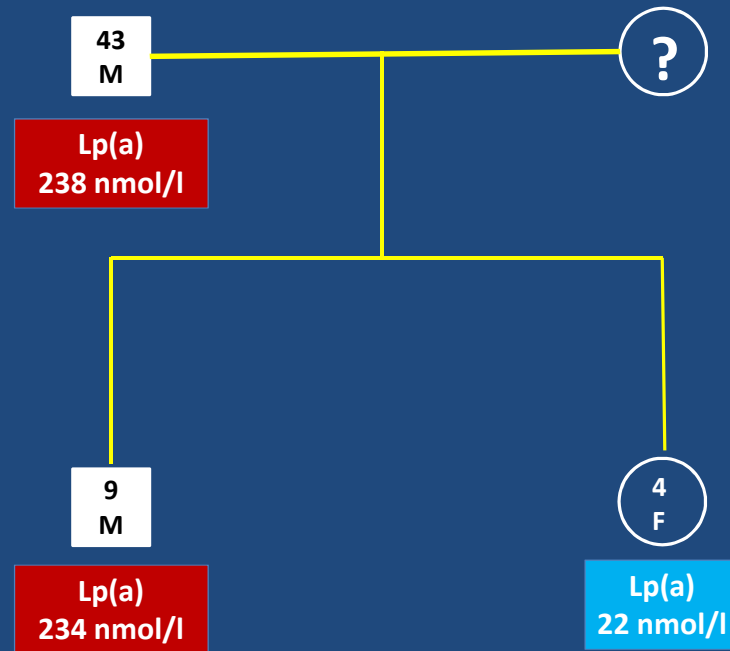
KẾT LUẬN : Xơ vữa, hẹp nhẹ động mạch thận phải.
Gan nhiễm mỡ nhẹ, polyp túi mật.



TÊN XÉT NGHIỆM	KẾT QUẢ	KHOẢNG THAM CHIẾU	MÃ QT
Bacteria	9	(0 - 130)	
III. SINH HOÁ - BIOCHEMISTRY			
HbA1c (HPLC) ¹ :	*		QTSH012
HbA1c (IFCC)	42.19	(20 - 38.80 mmol/mol)	
HbA1c (NGSP)	6.01	(4.0 - 5.70 %A1c) Quyết định 5481/QĐ-BYT 2020	
Glucose (mmol/L) (FPG) ¹	5.40	(3.90 - 5.60 mmol/L)	QTSH001
Glucose (mg/dL)	97.20	(70.2 - 100.8 mg/dL) Quyết định 5481/QĐ-BYT 2020	
IONOGRAMME ² :	*		QTSH067
Na	138.8	(130 - 145 mmol/L)	
K	4.13	(3.40 - 5.1 mmol/L)	
Ca	2.40	(2.1 - 2.80 mmol/L)	
Cl	102.4	(96 - 108 mmol/L)	
MICRO.ALB/Urine (Quantitative):	*		QTSH099
Micro Albumin/Creat	6.45	(< 30 mg/g)	
Uric Acid/Serum ¹	11.76 H	(Nam: 3.4 - 7.0; Nữ: 2.4 - 5.7 mg/dL)	QTSH014
*Độ Lọc Cầu Thận (CKD-EPI)	*		
Creatinin/Serum ²	1.08	(Nam: 0.6 - 1.3; Nữ:0.5 - 1.1 mg/dL)	QTSH027
eGFR (CKD-EPI)	87	(≥ 90 mL/min/1.73 m²)	
LDL Cholesterol ²	3.52	(<2.59; Ngưỡng: 2.59-4.13 mmol/L)	QTSH093
	*	(Cao: 4.14 - 4.91; Rất cao ≥ 4.92)	
HDL Cholesterol ²	0.910 L	(≥1.55; Ngưỡng: 1.04-1.54 mmol/L)	QTSH084
	*	(Thấp: < 1.04)	
Triglycerides ¹	1.52	(<1.70; Ngưỡng: 1.70-2.25 mmol/L)	QTSH015
	*	(Cao: 2.26 - 5.64; Rất cao ≥ 5.65)	
Lipoprotein (a) (nmol/L)	238.0 H	(≤ 105 nmol/L)	
Lipoprotein (a) (mg/dL)	238.0 H	(≤ 30 mg/dL)	
GGT ¹	22.53	(Nam < 55 U/L; Nữ < 36 U/L)	QTSH004
SGOT (AST) ¹	22.37	(< 35 U/L)	QTSH005
SGPT (ALT) ¹	16.02	(< 30 U/L)	QTSH013



CASE 2





CONCLUSION



RESPECT THE GENETIC ABSOLUTE

Recognize Lp(a) as a causal driver of atherogenesis, thrombosis, and valve calcification that is entirely immune to lifestyle interventions.

Atherogenesis,
Thrombosis,
Calcification



Immune to
Lifestyle



OFFSET THE RISK [3]

In the absence of RNA therapies today, the immediate clinical response to high Lp(a) is dramatic risk reclassification and aggressive LDL-C lowering (Statin + Ezetimibe + PCSK9i).

High
Lp(a)

Risk
Reclassification



LDL-C



TEST ONCE IN A LIFETIME [1, 2]

Universal screening is now a Class I (ACC) / Class IIa (ESC) mandate for all adults. Do not guess; measure directly in nmol/L.

Universal Screening

Fasting is not required for Lp(a) testing.



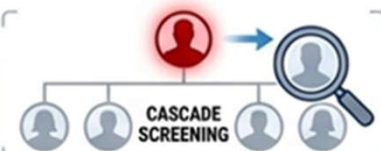
PROTECT THE FAMILY [1]

Immediately execute cascade screening for all first-degree relatives.



Golden Window: Ages 9–11 Years

Optimal time to test
high-risk children
alongside universal
pediatric lipid screening.





REFERENCES

- [1] *2026 ACC/AHA Multisociety Guideline on the Management of Dyslipidemia*. Journal of the American College of Cardiology (JACC), Vol. 87, No. 11, March 2026.
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- [3] *Impact of Lipoprotein(a) on Residual Cardiovascular Risk after Acute Coronary Syndrome*. PubMed Central (PMC) / Insights in Cardiology, Vol. 14, June 2026
- [4] *The Lipoprotein(a) Implementation Gap: Bridging Evidence and Emerging RNA-Targeted Therapeutics*. PMC Cardiovascular Medicine, Vol. 19, Art. 45, June 2026.
- [5] *Lp(a) and Baseline ASCVD Status* , Berman et al , J A C C V O L . 8 3 , N O . 9 , 2 0 2 4 ; 8 7 3 – 8 8 6

