# Familial Hypercholesterolemia presenting with Acute Coronary Syndrome (NSTEMI)

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**Abstract**

Familial hypercholesterolemia (FH) is the most common metabolic disorder and is inherited in an autosomal dominant fashion. FH presents with xanthomas and early coronary artery disease, owing to relatively high levels of low-density lipid-cholesterol (LDL-C) compared to hypercholesterolemia without a genetic predisposition. The first clinical manifestation of this condition could be an acute coronary syndrome in undiagnosed and unsuspecting patient. Various clinical criteria are available to make a diagnosis of FH and a clinician should use the one which he is familiar with. Owing to very high LDL-C levels patients often need multiple oral lipid lowering agents to achieve the desired LDL-C goal. There are multiple novel parenteral lipid lowering agents in offing and some are in pipeline with advantage of high potency and weekly or monthly dosing. This case report emphasizes the importance of FH screening and aggressive treatment to decrease morbidity and mortality in the general population.

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**Abstract**

Familial hypercholesterolemia (FH) is the most common metabolic disorder and is inherited in an autosomal dominant fashion. FH presents with xanthomas and early coronary artery disease, owing to relatively high levels of low-density lipid-cholesterol (LDL-C) compared to hypercholesterolemia without a genetic predisposition. The first clinical manifestation of this condition could be an acute coronary syndrome in undiagnosed and unsuspecting patient. Various clinical criteria are available to make a diagnosis of FH and a clinician should use the one which he is familiar with. Owing to very high LDL-C levels patients often need multiple oral lipid lowering agents to achieve the desired LDL-C goal. There are multiple novel parenteral lipid lowering agents in offing and some are in pipeline with advantage of high potency and weekly or monthly dosing. This case report emphasizes the importance of FH screening and aggressive treatment to decrease morbidity and mortality in the general population.

**Key Words-** Low density lipoprotein, Dutch lipid clinic network score, Xanthomas, Myocardial Infarction.

**Introduction** Familial hypercholesterolemia (FH) is the most common metabolic disorder and is inherited in an autosomal dominant fashion. FH presents with xanthomas and early coronary artery disease, owing to relatively high levels of low-density lipid-cholesterol (LDL-C) compared to hypercholesterolemia without a genetic predisposition (1,2). As a result of the rapid progression of arteriosclerosis due to the high LDL-C levels and early-onset coronary atherosclerosis, vigorous lipid-lowering therapy is required. However, it can be challenging for people with FH to use oral medication alone to reduce their LDL-C levels to the goal range recommended by guidelines (3).

**Case Presentation**

A 20-year-old male presented to the cardiology outpatient department (OPD) with complaints of acute onset chest pain for the last three days, which had been aggravated, and was at peak since 4 am in the morning of the OPD visit. This episode of pain on the day of the visit was associated with nausea and vomiting. There was no previous history of chest pain on exertion, chronic kidney disease, chronic liver disease, diabetes, or bleeding diathesis. On examination, he had a heart rate of 78 beats per minute and blood pressure of 124/76 mm of Hg, S1 and S2 sounds were heard normally with no rub or murmur, and the chest was bilaterally clear with equal air entry. Routine investigations were ordered and the significant findings have been summarized in**Figure 1** . An Electrocardiogram (ECG) on admission showed ST segment coving and T wave inversion in precordial leads V2-V4. (**Figure 1** ) The two dimensional echocardiography revealed regional wall motion abnormality in the left anterior descending artery (LAD) territory and a left ventricular ejection fraction of 44%.An diagnosis of Acute coronary syndrome / Non ST elevation MI involving LAD territory was entertained and patient was taken up for cardiac catheterization. The coronary angiography depicted a right dominant system with a 30% stenosis in the distal left main coronary artery, 95% stenosis in the proximal left anterior descending artery, and 100% chronic total occlusion in the right coronary artery. (**Figure 2** ) The distal right coronary could be seen filling retrogradely from the left coronary system via collaterals vessels. Without complications, the patient underwent percutaneous coronary stenting to LAD with an everolimus eluting stent - Xience Xpedition (Abott Vascular Inc.,Abott park,Illinios,USA)

of size 2.5mm x 48mm. Because of high LDL-C levels and premature CAD, a suspicion of Familial Hypercholesterolemia was there and a through physical examination was done. An ocular examination revealed arcus senilis in upper quadrants of both corneas.(**Figure 3** ) There were also xanthomas on the external aspects of Achille's tendon, ankle ,elbow and triceps tendon. (**Figure 4** ) According to the Simon Broome Criteria, since the total serum cholesterol and serum LDL-C were elevated to more than 7.5 mmol/L and 4.9 mmol/L respectively with tendinous xanthomas in the patient a definitive diagnosis of FH was made (4). According to the Dutch Criteria, our patient had a total score of 17 hence again providing a definitive diagnosis of FH (Definite FH needs Score >8). (5) The hospital course was uncomplicated and the patient was discharged in a hemodynamically stable condition. On discharge, the following drugs were advised to the patient- dual antiplatelet therapy (aspirin plus clopidogrel), Beta blocker, ACE inhibitor, Aldosterone and Furosemide. Lipid lowering therapy consisted of High intensity statin therapy - atorvastatin in a dose of 80 mg. Subsequently, on follow up ezetimibe and bempedoic acid were added to lipid lowering regimen but despite triple oral lipid lowering therapy LDL was still not at goal. (**Figure 5** ) He had been advised parenteral lipid lowering therapy- Evolocumab (a PCSK-9 inhibitor) but due to cost consideration he could afford it.

**Discussion**

FH is a genetic disorder in which LDL-C levels are significantly elevated in the blood. It is an underdiagnosed autosomal dominant condition presenting with arcus cornealis, xanthomas, and premature coronary artery disease. All these findings directly result from the abnormally elevated LDL-C levels in the plasma. (6) The genes that encode LDL receptors are apolipoprotein B (ApoB), proprotein convertase subtilisin kexin-9 (PCSK9), and low-density lipoprotein receptor adaptor protein 1 (LDLRAP1), and these are the main genes which are affected in FH. (1) It is inherited in an autosomal dominant pattern, so there is a 50% chance of inheriting the mutated gene. Some forms of FH are inherited in an autosomal recessive pattern, due to mutations in the LDL receptor adaptor protein 1 ( LDLRAP1) gene, but it is very rare. (7) The mutation

in the LDL receptor gene causes various clinical features like tendon xanthoma, premature coronary artery disease, and aortic valve involvement. They present early in childhood in cases of homozygous disease variants. (8) The high levels of LDL present in patients of FH lead to progressive atherosclerosis from a young age, which results in a high risk of coronary artery disease in the future (up to 20-fold). (9,10)

The US MedPed Programme, the Dutch Lipid Clinic Network, and the Simon Broome Register Group in the United Kingdom are the three groups that have produced clinical diagnostic methods for familial hypercholesterolemia. (**Figure 6**; 4,5,11) The Simon-Broome Criteria offers advantages in ease of remembrance and economic viability for diagnosing familial hypercholesterolemia (FH) in lipid clinics, being widely used by physicians. Its reliance on conventional methods avoids expensive DNA testing, making it suitable for healthcare centers. However, a major disadvantage is its inability to differentiate between FH types and non-FH conditions, potentially overlooking patients with mild phenotypes or in the pediatric population. It lacks consideration for genotype-phenotype correlations, hindering its ability to discern responses to specific therapies. Overall, while advantageous in simplicity and cost-effectiveness, its limitations in precision and scope should be acknowledged. The Simon-Broome criteria have been modified to become the Dutch criteria for the diagnosis of FH. The primary motivation behind the creation of the Dutch criteria was the fact that the Simon-Broome criteria uses the patient's medical history, physical examination, and test results to diagnose FH. However, the Simon-Broome criteria ignores the fundamental molecular defect of FH. The Dutch criteria took into account the molecular flaw of FH and proposed a point system to solve this deficit. Age- and relative-specific criteria for total cholesterol are used in the US MEDPED criteria. Despite being easy to apply, the criteria ignores FH-associated gene alterations and clinical features. It does not take into account factors influencing cholesterol levels, as demonstrated by a study on seasonal fluctuations in lipid levels, although it does urge cascade screening after diagnosis. Increased temperature and physical activity were associated with changes in plasma volume, particularly relative hypervolemia during the summer, which was associated with higher variations in women and hypercholesterolemic people. (12,13)

Cessation of tobacco use and treating hypertension and diabetes are important parts of managing cardiovascular complications in FH.(14) But LDL lowering remains the mainstay of FH management. Statin therapy remains the cornerstone of lipid lowering in the scenario of FH. High dose statins reduce LDL-C by almost 50% and moderate dose statins by 30%. (15) Based on data from clinical trials over past 2 decades, the 2013 ACC/AHA cholesterol guidelines define Atorvastatin 40-80 mg or Rosuvastatin 20-40 mg as high intensity statins. The guidelines define - Atorvastatin 10-20 mg , Rosuvastatin 5-10 mg ,Pitavastatin 2-4 mg and simvastatin 20-40 mg as moderate intensity statins. Long term studies have demonstrated their role in retarding the progression of atherosclerosis and ameliorating risk of cardiovascular events.(16,17) However, because of high baseline LDL-C levels many FH patients will need combination therapy to attain LDL-C levels. Ezetimibe and Bempedoic acid are two additional oral lipid lowering drug who have shown to reduce LDL-C by about 15-20% when added to statins and also improvement in cardiovascular outcomes.(18,19) Bile acid sequestrants can reduce LDL-C by 20% but gastrointestinal side effects limit their use in clinical practice. Most FH patients will need dual or triple lipid lowering therapy as in our case. But despite the use of combination of oral lipid lowering therapy a substantial proportion of FH will need parenteral lipid lowering therapies which are more potent and longer acting.

Antibodies against proprotein convertase subtilisin-kexin type 9 (PCSK9) have a notable effect on decreasing LDL-C, and it is anticipated that they will be helpful in lipid-lowering therapy for FH, which is poorly controlled by conventional oral lipid lowering treatment.(20,21) Evolocumab the PCSK-9 approved by US FDA can be given every 2 weeks or 4 weeks and produces up to 60% LDL-C reduction. More recently, small interfering RNA against PCSK-9 (Inclisiran) and monoclonal antibody against ANGPTL3 (Evinacumab) have been shown to reduce LDL-C effectively in FH patients.(22) Inclisiran has the advantage of administration every 6 months. In a pooled analysis of phase 3 trials in FH patients, the twice yearly dose of Inclisiran has been shown to reduce LDL -C by 50% (over maximally tolerated statin) and is also safe barring minor injection site issues.(23) Because LDL-C receptors (LDL-R) are severely depleted in Homozygous FH, the efficacy of many lipid lowering therapies (LDL-R dependent - statin, PCSK-9 I , Bempedoic acid and ezetimibe) is attenuated. Evinacumab inhibits ANGPTL3 and reduces LDL-C independent of LDL-R in patients

with FH. In the ELIPSE-HoFH study, the drug given every 4 weeks , achieved 50% LDL-C reduction at 6 months.(24) Gene editing technology has been utilized to silence PCSK-9 gene in liver and consequently decrease LDL-C levels. VERVE 101 is a CRISPER based gene editing technique given as a single infusion and has been shown to reduce LDL-C by 39%-55% levels in 10 FH patients in the phase1B Heart-1 trial (**NCT05398029** ).(25)The effect on LDL-C lowering were durable up to 6 months. **Figure 7** details the expected LDL lowering with currently available agents.

The HeFH phenotype is identified using the Dutch Lipid Clinic Network criterion, which is based on LDL-C level, family history of FH, the existence of tendon xanthoma, and arcus cornealis. The early detection of FH by a family member of the index case and the prevention of CAD-related morbidity and mortality through dietary changes and prudent use of hypolipidemic medications are both made possible by the early diagnosis of FH (6).Still, FH remains widely underdiagnosed, undertreated, and understudied, especially in pediatric patients. (26)

The most effective way to perform cascade testing for FH is to do a genotype analysis of the relatives of the index patients with a known mutation, however, this practice is only in its infancy in middle-income nations like India. This is a more precise way than doing plasma cholesterol screening on the family members. (27)

*Conclusion*

The underdiagnosis of FH is a well-recognized problem, especially in the low to middle-income world. The first clinical manifestation of this condition can be an acute coronary syndrome in undiagnosed and unsuspecting patient. This case report emphasizes the importance of FH screening and aggressive therapy to decrease morbidity and mortality in the this population.

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*Figures*

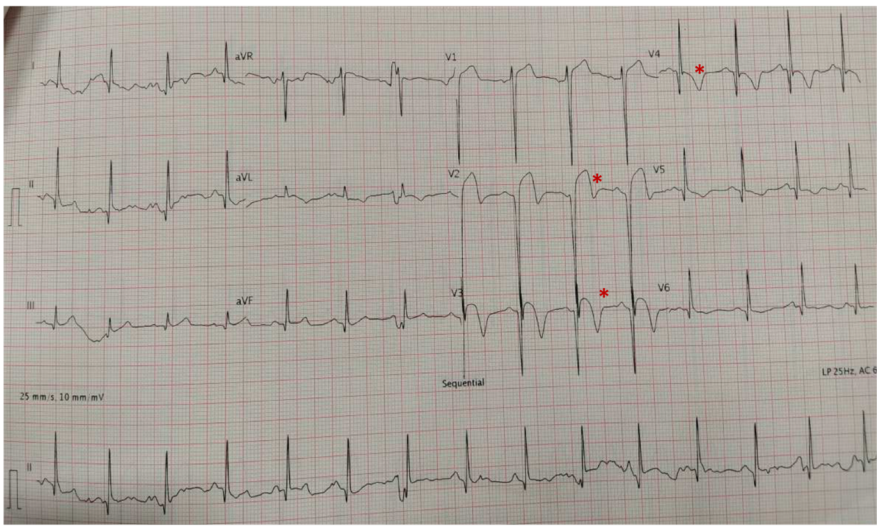


**Figure 1.** A 12 lead electrocardiogram depicting ST coving with T wave inversion (*) in precordial leads V2-V4 suggestive of LAD territory ischemia/infarct.

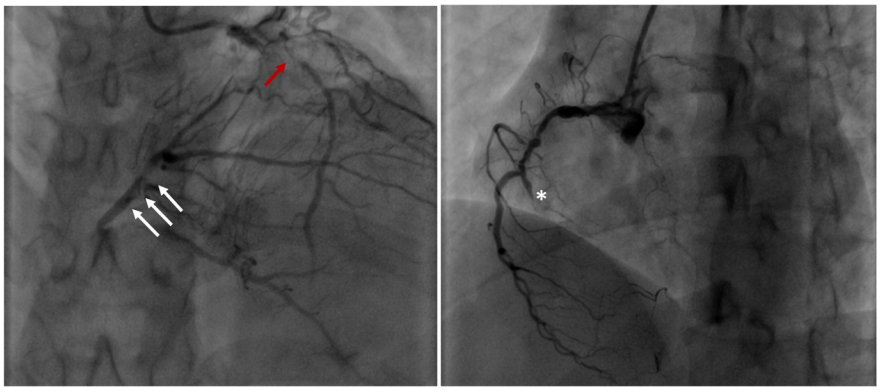

**Figure 2.** Coronary angiography of the patient showing severe obstructive disease. The left panel depicts severe 95% stenosis in Proximal LAD (red arrow) and distal RCA filling retrogradely (white arrows) via collaterals vessels from left system. The right panel depicts total occlusion in the mid RCA (*) after the origin of right ventricular branch.

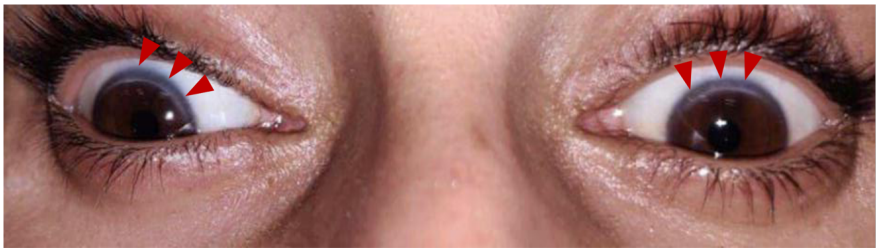

**Figure 3.** Grayish depositions in upper quadrants of both corneas suggestive of Arcus Senilis.

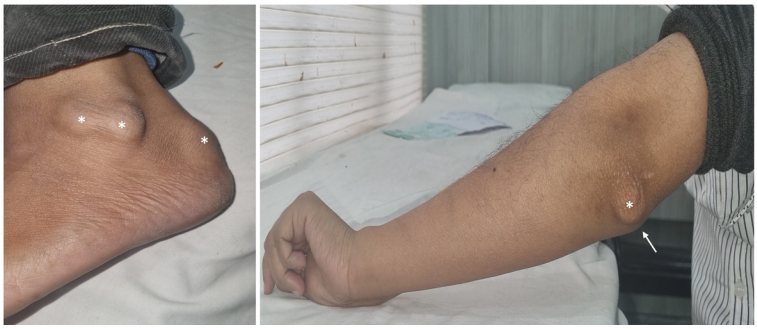

Figure 4. Tendinous Xanthoma in the Achilles Tendon , ankle joint (asterixis-left panel), elbow & Triceps Tendon (asterixis & white arrow -right panel).

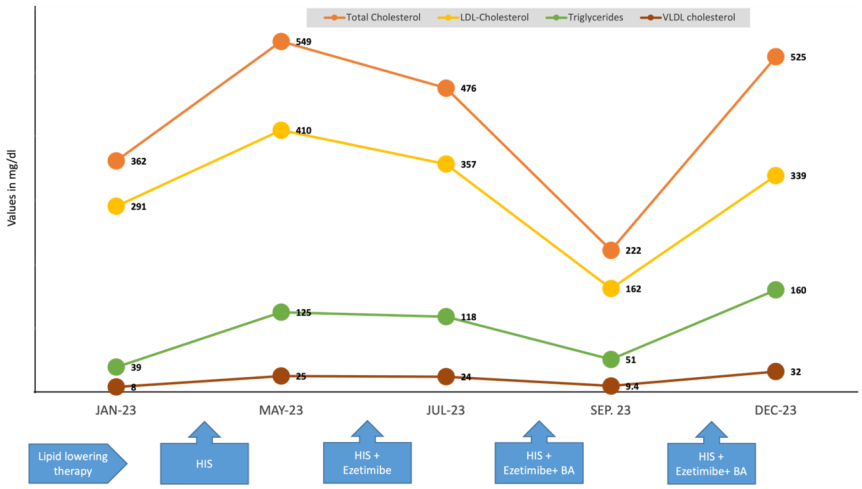


Figure 5. Temporal changes in lipid profile with escalating oral lipid lowering therapy. High Intensity statin therapy is defined as Atorvastatin 40-80 mg or Rosuvastatin 20-40 mg by 2013 ACC/AHA guidelines for cholesterol control.

[LDL- Low density lipoprotein ; VLDL- Very low density lipoprotein; HIS- High intensity statin ; BA – Bempedoic acid]

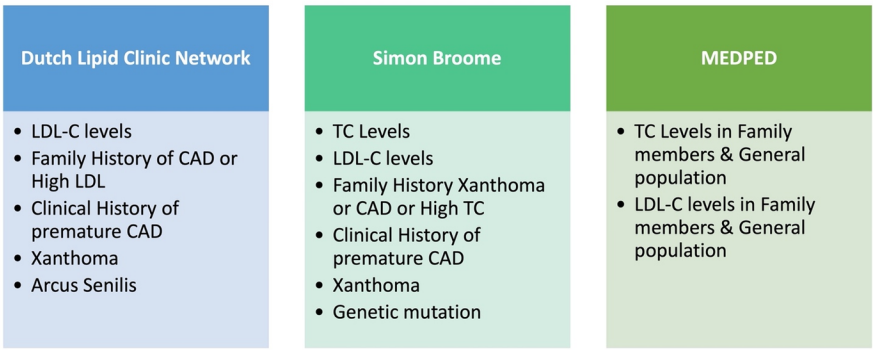


Figure 6. Comparison of the parameters utilized in three commonly used scores used to diagnose FH.

[LDL-C- Low density lipoprotein cholesterol ; TC- Total Cholesterol; CAD- Coronary artery disease ]

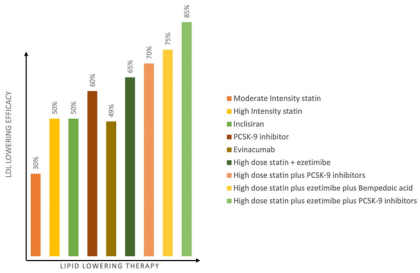


Figure 7. The efficacy of various lipid lowering agents for LDL Lowering. The ACC/AHA 2013 cholesterol guidelines define High intensity statin as Atorvastatin 40-80 mg or Rosuvastatin 20-40 mg. The guidelines define moderate intensity statin as- Atorvastatin 10-20 mg , Rosuvastatin 5-10 mg ,Pitavastatin 2-4 mg and simvastatin 20-40 mg.