Supplementary Material

Table S1. Diagnostic criteria and possible pathogenic mechanisms of each FLL type of HLP in the present study*

Type	Diagnostic criteria**	Possible pathogenic mechanisms
Type 1 (HLP1)	- ApoB <75 mg/dL - TG ≥150 mg/dL - TG/apoB ratio ≥8.8 - Presence of chylomicrons on LPEP	HLP1 is a very rare disorder with homozygous mutation in *LPL, GHIHBP1, APOC2, APOA5,* or *LMF1* genes and deficiency of lipoprotein lipase (LPL).
Type 2a (HLP2a)	- ApoB ≥120 mg/dL - TG <150 mg/dL - Absence of chylomicrons on LPEP	HLP2a is a common polygenic lipid disorder with hypercholesterolemia and high LDL-C which may be associated with dietary and environmental factors. However, the most severe presentation of HLP2a is familial hypercholesterolemia which is resulted from homozygous or heterozygous mutation in *LDLR, APOB,* or *PCSK9* genes.
Type 2b (HLP2b)	- ApoB ≥120 mg/dL - TG ≥150 mg/dL - Absence of chylomicrons on LPEP	HLP2b is a common polygenic lipid disorder with hypercholesterolemia and mild hypertriglyceridemia (HTG) which is usually associated with dietary and environmental factors.
Type 3 (HLP3)	- ApoB ≥120 mg/dL - TG/apoB ratio <8.8 - TC/apoB ratio ≥2.4 - The presence of broad-β band on LPEP	The cause of HLP3 is a specific presentation (ε2/ε2) of *APOE* gene polymorphism.
Type 4 (HLP4)	- ApoB <120 mg/dL - TG ≥150 mg/dL - TG/apoB ratio <8.8 - TC/apoB ratio <2.4 - Absence of chylomicrons on LPEP	HLP4 is a very common presentation of lipid disorder and mainly caused by metabolic disorders (e.g., obesity, insulin resistance, and diabetes).
Type 5 (HLP5)	- ApoB <120 mg/dL and ≥75 mg/dL - TG ≥150 mg/dL - TG/apoB ratio ≥8.8 - Presence of chylomicrons on LPEP	HLP5 is caused by heterozygous mutation in LPL, GHIHBP1, APOC2, APOA5, or LMF1 genes, or burdens of single nucleotide polymorphisms in multiple TG-associated genes (i.e., *DOCK7, ANGPTL3, GALNT2, KLHL8, AFF1, MAP3K1, ANKRD55, MLXIPL, NAT2, TRIB1, JMJD1C, FADS1, FADS2, FADS3, APOA1, APOC3, APOA4, CAPN3,*

		FRMD5, CETP, SUGP1, PLTP, GCKR, CYP26A1, CILP2) accompanied by secondary metabolic or environmental factors (i.e., obesity, alcohol drinking, or diabetes).

Abbreviations: ApoB = apolipoprotein B; FLL = Fredrickson, Levy, and Lees; HLP = hyperlipoproteinemia; LPEP = lipoprotein electrophoresis; TC = total cholesterol; TG = triglyceride; VLDL = very-low density lipoprotein.

*The unit of each lipid parameter used in the present study was mg/dL.

**Each characteristic should be met.

Table S2. Study variables in the present study	
Variables	**Definition and description**
Age	
Sex	Male or female
Weight	Unit: kilogram
Height	Unit: cm
Smoking habit	Current smoker
Body mass index	Weight in kilograms divided by the square of height in meters
Coronary artery disease (CAD)	defined as those who had > 50% diameter stenosis of major epicardial coronary arteries confirmed by coronary computed tomography (CT) angiography or coronary angiography
Ischemic stroke (IS)	Determined by the neurologist's records with confirmation by brain CT or magnetic resonance imaging
Peripheral arterial disease (PAD)	Confirmed by ankle-brachial index < 0.9 or > 1.4 and/or > 50% diameter stenosis of peripheral arteries observed in CT angiography
Diabetes mellitus (DM)	Determined by medical records and prescribed medications
Hypertension (HTN)	
Pancreatitis	Identified by patients' admission records together with detection of raised circulating pancreatic enzyme and pancreatic CT scans
Prescribed medications	Antiplatelet, beta-blocker, angiotensin con-verting enzyme inhibitor/angiotensin receptor blocker (ACEI/ARB), and lipid-lowering therapy (LLT) including statin, ezetimibe, proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitor, fenofibrate, niacin, and omega-3 fatty acid

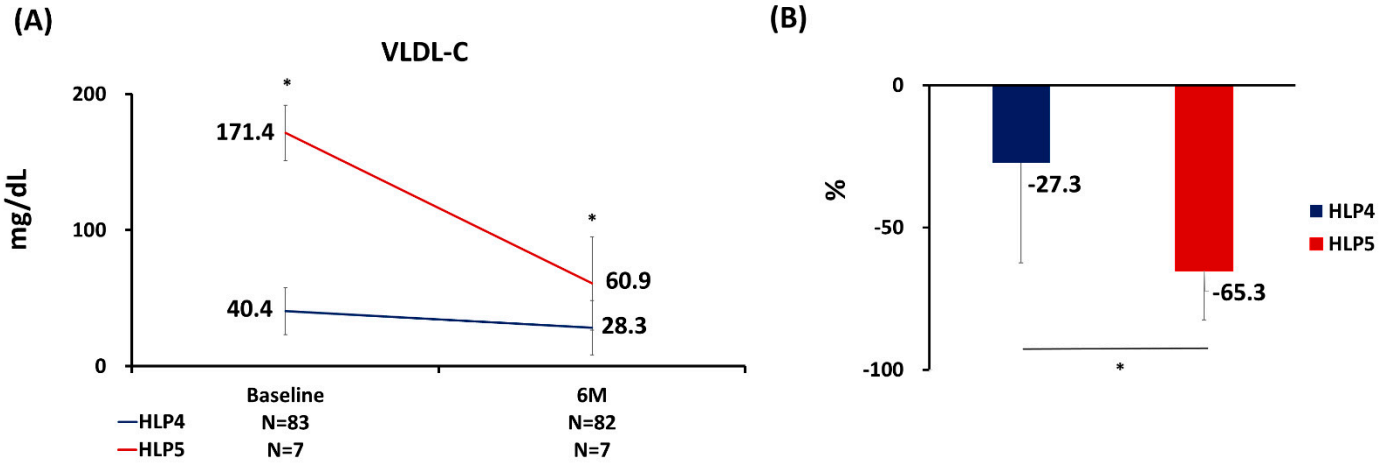

Figure S1. Illustration of plasma VLDL-C and percentages of change in plasma VLDL-C among patients. (A) Plasma VLDL-C. (B) Percentages of change in plasma VLDL-C. HLP4 = type IV hyperlipoproteinemia; HLP5 = type V hyperlipoproteinemia; N = numbers; VLDL-C = very-low-density lipoprotein cholesterol.