Table causes HTG

Secondary, non-genetic hyperlipoproteinemias and factors influencing lipid metabolism.

Underlying disease	LDL-C	Triglycerides	HDL-C
Endocrinology and metabolism			
Hypothyroidism	↑ ↑	⟷, ↑	⟷
Diabetes mellitus	⟷	↑ ↑	↓
Acromegaly	⟷	↑	↑
Hypercortisolism (Cushing's disease)	↑	↑	⟷
Adipositas	⟷	↑	↓
Pregnancy	↑ ↑	↑	↑
Kidney disease			
Nephrotic syndrome	↑ ↑	↑	⟷
Renal insufficiency	⟷, ↑	↑ ↑	⟷, ↑
Kidney transplant	↑ ↑	↑	⟷
Liver disease			
Non-alcoholic steatosis	⟷	↑ ↑	↓
Cholestasis	↑	↑	↓
Hepatitis	⟷, ↑	↑	↓
Other diseases			
Alcoholism	⟷	↑	↑
HIV	↑	↑	↓
Autoimmune diseases	↑	↑	↓ ↓
Medication			
Estrogens	↓	↑ ↑	↑
Tamoxifen	↓	↑ ↑	(↑)
Androgens	↑	↑	↓ ↓
Corticosteroids	↑	↑	↑ ↑
Cyclosporin A	↑	↑	↑ ↑
Tacrolimus	⟷	↑	↑ ↑
Retinoids	↑	↑ ↑	↓
Antihypertensive (β-blocker, Thiazide)	↑	↑ ↑	⟷
Antipsychotics	⟷	↑	⟷
HIV-Proteaseinhibitor	↑	↑ ↑	↓ ↓

Arrow explanation: Elevated (↑), high elevated (↑ ↑), no change (⟷), decreased (↓), strongly decreased (↓ ↓).

References: März W et al. Labordiagnostik von Fettstoffwechselstörungen. Dtsch Med Wochenschr 2023;148: e120-e145

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Table causes HTG

Primary genetic hyperlipoproteinemias with elevated TGs

Disease	Genes	Frequency	Clinic
Disorders predominantly in the metabolism of LDL			
Familial combined hyperlipoproteinemia (FCHL)	Polymorphism in the genes with effect on LDL-C and TG	1:100	Autosomal dominant, different lipoprotein phenotypes (IIa, IIb and IV), LDL-C and/or TG elevated, atherogenic lipoprotein phenotype, features of the metabolic syndrome, xanthomas make the diagnosis unlikely, 10% of patients with myocardiac infarction.
Deficiency of lysosomal acid lipase	LIPA	1:1000 000	Autosomal recessive; a) infantile form: Wolman disease, malabsorption, failure to thrive, hepatomegaly; b adult course form: cholesterol ester storage disease, hepatomegaly, high cholesterol LDL-C, high TG, low HDL-C, altherosclerosis
Disorders predominantly in the metabolism of the remnants of triglyceride-rich lipoproteins			
Type III hyperlipoproteinemia	APOE	1:2000	autosomal recessive, xanthomas of the hand lines, accumulation of Chylomicron remnants, greatly increased risk of atherosclerosis
Definciency of hepatic lipase	LIPC	Rare	Autosomal recessive, xanthomas possible, high concentration of Intermediate density lipoproteins (IDL)
Disorders predominantly in the metabolism of triglyceride-rich lipoproteins			
Monogenic hypertriglyceridemia (familial chylomicronemia)	LPL, APOC2, APOA5, LMF1, GPIHBP1, CREB3L3, GPD1, GCKR	1:000000	Mainly autosomal recessive; eruptive xanthomas, *lipaemia retinalis*, precurrent pancreatitis, risk of atherosclerosis not increased
Polygenic (”multifactorial”) hypertriglyceridemia	Polymorphisms with effect of the metabolism of TG-rich lipoproteins		Autosomal-dominant; TG between 200 and 500 mg/dl (2.3 mmol/l and 5.7mmol/l), expressivity variable, coincidence with diabetes melitus, hyperuricemia and hypertension
Lipodystrophies	At least 21 candidates genes	Rarely	Deficiency of adipose tissue, either generalized or partial, genetic or acquired, diabetes mellitus, hypertriglyceridemia, fatty liver, hepatocellular carcinoma, pancreatitis, heart disease (cardiomyopathies), renal failure

Abbrivations:
APOA5, Apolipoprotein A5; APOE, Apolipoprotein E; APOc2, apolipoprotein C2; C, Cholesterol; CREB3L3, CAMP-Responsive Element-Binding Protein-3-Like-3; GCKR, Glucokinase regulatory protein; GPD1, glycerol-3-phosphate dehydrogenase-1; GPIHBP1, Glycosylphosphatidylinositol-anchored HDL-binding protein 1; HDL, High density lipoprotein; LDL, Low density lipoproteins; LIPA, lysosomal acid lipase; LMF, Lipase maturation factor; LIPC, Lipase Member C (Hepatic Lipase); LPL, Lipoprotein lipase; TG, Triglycerides

References:
März W et al. Labordiagnostik von Fettstoffwechselstörungen. Dtsch Med Wochenschr 2023;148: e120-e145.
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