Dyslipidemia break out session. Leftover questions…

Lp(a)

Essentially well recognized as a prognostic risk factor. But no clear therapeutics nor targets currently. PCSK-9i have lowered Lp(a) levels, but has not translated to robust CV outcome improvement. Thus most useful if elevated CV risk changes management. Eg, deciding timing of statin for primary CV prevention or deciding on more aggressive LDL targets in secondary prevention. European Society of Cardiology is starting to incorporate it more, American and Canadian Cardiovascular Society has not as aggressively incorporated yet.

Keto diets / low carb-high fat

Some general principles when discussing keto diets: 1) few people can sustain a true ketogenic diet (<20g carb/d), 2) weight loss does not automatically equate to CV health, 3) keto diet/low carb does not have to be high fat.

Often people state they are on keto diets, but in reality, they're not restricting carbs enough to cause ketosis. Thus many variants out there. Would recommend to be cautious about yo-yo weight loss due to difficulty maintaining such a restrictive diet. Low carb/keto diets do improve glycemic control. No strong evidence of keto diets to improve CV outcomes. Of note lowering a1c levels have not been shown to improve macrovacular events (a1c reduction mostly benefits microvascular events). Most interpretation of keto diet is other side of plate is high fat, with subsequent negative effects on lipid profile. But can do low carb/keto with healthy proteins/veggies which likely mitigates the impact on lipid profile.

Intermittent fasting

Probably mild impact on lipid profile compared to usual portion controlled, balanced diet and weight loss. Caution re "rebound" eating. But if adhered to healthily, likely improved TG's and some mild LDL improvement. No known robust CV outcomes evidence.

Cancer risk (with pravastatin)

This is essentially phenomenon of epidemiology. Any therapy that has robust benefit in CV mortality will likely have higher rates of cancer. Two main pathologies as we age are CV disease and cancer, thus lowering CV disease rates, increases lifespan to have a better chance of getting cancer. In current era, no evidence of statins as a causative agent of malignancies. (As a hypothetical exercise, imagine if we invented a drug that completely cured CV disease, what would then impact your patient's mortality? Another historical analogy would be blaming antibiotics, clean water, vaccines etc as a "cause" of cardiovascular disease. Without TB, measles, dysentery, plagues etc, the rise of CV disease in the population came about.)

Statin and elevated liver enzymes/fatty liver disease

Roughly 5-8 yrs ago, the FDA recommended to physicians to stop monitoring liver enzymes as part of statin surveillance. ~30 yrs of post market data failed to show any significant concern for liver injury. Fulminant hepatic failure is extremely rare and occurs at same rate as non-statin users. There was actually a large RCT in using statins to treat fatty liver disease. (It didn't work, but no harm either, further demonstrating safety). Summary, no need for monitoring of liver enzymes, and liver disease is not considered a contraindication for statin use.

Managing pts with both high HDL and high LDL (assuming question of being less aggressive with LDL management in setting of high HDL)

LDL management (starting statins and targeting levels) would be independent of HDL levels. High HDL should not be viewed as a "1:1" impact on CV risk, and should not be viewed as sufficient to counter negative impact of LDL.

Lipid lowering in pregnancy

No proven lipid lowering medications safe during pregnancy or breast feeding. If patient has strong indication for lipid lowering meds during child bearing age (ie Familial Hypercholesterolemia), they are instructed to stop meds as soon as they are trying or become pregnant. Unfortunately, pregnancy often worsens lipid profiles. Patients in this scenario ideally should be referred to specialist.

Statin use in kids/teens

Pediatric lipidologists may be using statins for Familial Hypercholesterolemia patients as young as 8 yrs old. If you have a pediatric patient that you think needs statins, these FH patients should be referred to specialty clinic which would have a multi-disciplinary approach to discuss risk/benefits of starting pharmacotherapy.

LDL targets in relation to FRS (or tailoring LDL targets to CV risk)

Currently, in Canada, the level of CV risk dictates the timing of statin initiation. But once decision to start meds made, one target LDL is recommended, regardless of risk level. This was mostly driven by idea of a simple target to manage. (In other words, do not confuse levels that determine Threshold to Treat as synonymous as Target Level). However, as we gain more data on lower LDL targets, these would need to be justified in patients with higher CV risk. Very low LDL targets may not be justifiable in for example moderate risk primary prevention population. My personal opinion is that the CCS (Canadian Cardiovascular Society) will eventually break their one target strategy as early as this fall. This would be consistent with the European approach.