Assaying VLDLs in the Lab

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I was asked by a laboratory representative: "I have a physician who orders a lot of VAPs because it gives him total VLDL. The 2 page NMR only gives Large VLDL and size. He really wants total VLDL, what can I tell him?

Advanced lipid and lipoprotein testing is wonderful because it can better help us understand what is going on in the patient enabling us to do better risk prediction and chose appropriate therapies. VLDLs and how they are evaluated in the lab is not well understood by most providers.

One must as with other lipid values distinguish between lipid and lipoprotein tests. Lipoprotein traffic lipids and there are assays to evaluate each. Total VLDL-C is a free calculation using the Friedewald equation which calculates VLDL-C by dividing TG by 5. This assumes all of the TG are in VLDLs and not other lipoproteins and that the VLDL core composition is 5 times more TG than cholesterol. So one can easily calculate VLDL-C from any fasting lipid profile.

VAP provides on their report form something called VLDL1, VLDL2 and VLDL3. Most lipid novices think VAP is providing VLDL particle or subparticle (large medium and small) concentrations but they are not. What they actually provide is not VLDL-P but rather VLDL1-C, VLDL2-C and VLDL3-C. VAP provides nothing on any lipoprotein concentration. Docs simply do not realize VAP is simply an advanced cholesterol test, not a lipoprotein test. Of course we now know how much cholesterol is within any lipoprotein especially VLDLs often has little meaning related to risk.

LipoScience used to provide small, medium and large VLDL-P and you could add them up to get total VLDL-P. But they now only report the large VLDL-P as well as VLDL size and both of those values should only be used as markers of insulin resistance.

The real reason why is that VLDL-C or even VLDL-P is not that helpful when evaluating atherogenic lipoprotein load is that it does not figure into risk evaluation. In Framingham Offspring Study where Cromwell showed LDL-P (especially when it is discordant) is far more important that LDL-C or non-HDL-C. In that study adding VLDL-P into the adjustment equation had no effect on risk prediction. In other words who cares about VLDL-P because 90-95% of atherogenic particles are LDLs and at best only 5-10% are VLDLs. A normal total VLDL-P is maybe 50-60 nmol/L. A normal total LDL-P is 1000 nmol/L.

There is no relationship of VLDL-C or subparticle VLDL-C to either apoB or VLDL-P because VLDLs have tremendous size variability and have great capacity to carry a lot of lipids per particle: they can become very, very large. So in two persons with the same elevated VLDL-C, the number of VLDL particles could be normal or high. In fact persons with Familial Hypertriglyceridemia who are not at risk for CHD, have very high TG and very high VLDL-C but have normal apoB. They simply have very large VLDL particles. Using the NMR LipoProfile one would see both elevated large VLDL-P, large VLDL size yet unremarkable total LDL-P. Hence there may be risk for pancreatitis but not CHD (because the LDL-P is normal). I do not see how the VAP reported VLDL-C levels would be of help. They would be high and one would probably erroneously treat as if the LDL-P is high.