Review Article

Triglycerides fasting or non-fasting? Current knowledge in diagnostic values

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Abstract

Triglyceride levels are increased for six to eight hours after a standard meal. If patient has consumed a very high fat content meal prior to testing. Fasting Triglyceride and non fasting Triglyceride levels were discussed in the present review article. Triglyceride storage and circulation in blood is kept under control by several enzymes, which are regulated by many genes and they reflect the nutritional needs of organism including fasting and non fasting status. In the majority of patients, non-fasting blood samples for lipids is the preferred specimen, providing similar clinical value to fasting samples for screening for cardiovascular disease (CVD) risk, and in most patients for monitoring. Triglycerides increases step wise after diet and reach a highest level after 5 hours post pandrially. Previous studies suggest that estimation of fasting Triglyceride may be carried only for cardiovascular patients, but it is very difficult for the patients to know whether they are in high risk of cardiovascular until they diagnosis properly.

Keywords: Cholesterol, triglyceride, lipid profile, cardiovascular disease, chylomicrons

Introduction

Basically fasting state is essential for triglycerides estimation because as mentioned above it remains high for several hours after meal and the Friedewald equation, used for calculation of LDL cholesterol (LDL cholesterol= total cholesterol - HDL cholesterol - [triglycerides/5]), uses fasting triglycerides value. If non-fasting triglycerides value is used in this equation the LDL cholesterol, the primary target of lipid lowering therapy, will be underestimated. However, this problem can be overcome to some extent by using direct LDL cholesterol estimation as this can be done in non-fasting specimen.

As a fast-food meal consisting of e.g. a burger, a shake, and fries might be considered a fat tolerance test, in areas where fast-food consumption is especially high patients may be advised to avoid high-fat, fast-food meals on the day of lipid profile testing. Also, as LDL cholesterol is often calculated by the Friedewald equation, which includes the triglyceride concentration, calculated LDL cholesterol has been thought to be affected substantially by food intake; however, directly measured and calculated LDL cholesterol values are similar using both fasting and non-fasting lipid profiles (Tanno et al., 2010; Mora et al., 2009).

Fasting Triglyceride and non fasting Triglyceride levels

If this Friedewald equation is employed, there may be some underestimation of LDL cholesterol when chylomicrons are present, which may even be circumvented if a modification of this equation is used (Hegele et al., 2014). Lipid-lowering trials have used fasting lipid measurements and, in order to follow evidence-based practice, fasting blood sampling has often been the standard in everyday risk assessment. There is evidence that the non-fasting condition may marginally lower plasma LDL cholesterol concentrations owing to liberal intake of fluids, and therefore lead to a potential minor misclassification of cardiovascular risk, as well as to error in initiating or altering lipid-lowering medication; although not all studies agree, this risk is small and may chiefly apply to diabetic subjects (Martin et al., 2013; Watts et al., 2011; Langsted et al., 2011). While a non-fasting sample is sufficient to diagnose an isolated hypercholesterolemia, such

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as familial hypercholesterolemia, or elevated Lp (a), it can possibly confuse the distinction between familial hypercholesterolemia and genetic forms of high triglycerides. Since non-fasting may therefore impair the accuracy in diagnosing some forms of hyperlipidaemia, we recommend that laboratories should also offer measurement of fasting triglycerides according to clinical context and indications, as in the case of very high non-fasting triglyceride concentration.

Life-threatening or extremely abnormal test results deserve special attention and reactions of the clinical biochemical laboratory. In this regard, the following extreme hyperlipidaemias should be noted: triglycerides.10 mmol/L (880 mg/dl) because of risk of acute pancreatitis, (Klop, 2011; Sidhu et al., 2012) LDL cholesterol .5 mmol/L (190 mg/dl) in adults or.4 mmol/L (155 mg/dL) in children and particularly (Sidhu et al., 2012). mmol/L (500 mg/dl) because of suspicious heterozygous and homozygous familial hypercholesterolaemia,(Steiner et al., 2011; Lund et al., 2011; Wyler von ballmoos et al., 2015) respectively, and Lp (a).150 mg/dL (99th percentile) for very high risk of myocardial infarction and aortic valve stenosis (Nordestgaard et al., 2013; Cuchel et al., 2014; Wiegman et al., 2015). It is also important to refer patients with very low concentrations of LDL cholesterol, apolipoprotein B, HDL cholesterol, or apolipoproteinA1 to a specialist lipid clinic for further evaluation of a major monogenic disorder of lipid metabolism Each country, state, and/or province in individual countries should adopt strategies for implementing routine use of non-fasting rather than fasting lipid profiles as well as flagging of abnormal values based on desirable concentration.

At present, the majority of guidelines recommend a fasting serum lipid test. This recommendation is based on achieving consistency between patients and over multiple tests by ensuring a relatively standardized metabolic state. It is also because the majority of research has been performed using fasting lipids; therefore it was assumed that making comparisons and analyzing risk would be less precise if using non-fasting tests.

Triglyceride storage and circulation in blood is kept under control by several enzymes, which are regulated by many genes and they reflect the nutritional needs of organism including fasting and non fasting status (Nordestgaard et al., 2010; Thanassoulis et al., 2013). In addition, triglycerides are closely associated with many other lipid factors. They are positively associated with atherogenic LDL cholesterol, presence of small LDL particles, remnant lipoproteins, apolipoprotein C-III (Apo C-III) and negatively with athero protective HDL cholesterol and apolipoprotein C-II (Apo C-II). This implicates that reliable assessment of the real and independent role of circulating triglycerides in the process of atherosclerosis and other diseases in epidemiological and clinical studies is difficult and that robustness of their association with cardiovascular disease depends more on the extent of statistical standardization for other risk and lipid factors than on their real biological role.

Factors affecting Triglyceride levels and related risk

Triglyceride levels are increased for six to eight hours after a standard meal (Kamstrup et al., 2014; White et al., 2010). If a patient has consumed a very high fat content meal prior to testing, or if they have slow lipid particle clearance after food (post-pandrial dyslipidaemia), triglyceride levels could be increased more than the estimated 0.3 mmol/l variance, and misrepresent clinical significance. Measuring non-fasting triglyceride levels may provide additional information for determining cardiovascular risk. Peak non-fasting triglyceride levels, four hours after a meal, are reported to be a strong predictor of cardiovascular events and insulin resistance, and risk equations may be developed based on these levels in the future (Kamstrup et al., 2014; White et al., 2010). Light to moderate drinking, e.g. one to three standard drinks per day for males or one to two standard drinks per day for females, has very little acute effect on triglyceride levels (Patel et al., 2004).However, excessive alcohol intake may cause an increase in triglyceride levels immediately following intake and after fasting (Patel et al., 2004).When alcohol consumption is accompanied by a meal containing fat it has a significant additive effect on the resultant triglyceride increase (Patel et al., 2004).

The initiation of lipid-lowering treatment, e.g. a statin, is based on CVD risk, which can be calculated using a non fasting sample. A non-fasting lipid test can also be used to monitor response to treatment (unless high triglycerides are being treated) (Sidhu et al., 2012).Lowering of LDL is currently the primary indicator of lipid management. A direct measure of LDL can be arranged in such circumstances from most community laboratories (this is not measured as part of the standard lipid panel in India). In the majority of patients, non-fasting blood samples for lipids is the preferred specimen, providing similar clinical value to fasting samples for screening for CVD risk, and in most patients for monitoring. Screening for diabetes, using HbA1c is also performed on a non-fasting sample. Screening for cardiovascular risk assessment by the Framingham formula (or related calculations) uses the Total Cholesterol/HDL ratio. This ratio is not affected by food intake. In most patients, the increase in triglycerides after food intake is small, about 0.3 mmol/l. Patients with high triglyceride levels have poor lipid clearance and may be at increased CVD risk. Monitoring lipid lowering

therapy is based on LDL levels. LDL is calculated from the other lipid results. The calculation is not valid when triglycerides are > 4.5 mmol/l, these patients will need a follow-up fasting sample. In high risk patients, treated to very low LDL levels, a fasting sample is preferable.

Background information

Total Cholesterol and LDL decrease by as much as 0.2 mmol/l and HDL around 0.1 mmol/l for about 3 – 4 hours after a meal. The Total Cholesterol / HDL ratio does not change. The main reason for using fasting lipid samples is historic. Most population studies and statin trials have used fasting values; fasting values became "the norm". A meta-analysis of 20 trials showed no difference in predictive power for CVD events using non-fasting samples.

LDL is calculated using the Friedewald equation.

LDL = Total Cholesterol – HDL – (Triglycerides / 2.2).

Following food intake, triglycerides increase and the calculated LDL decreases. In most patients triglyceride increase by no more than 0.3 mmol/l and the resulting decrease in calculated LDL is insignificant. When triglyceride level is 2.5 mmol/l, the error is about 0.5 mmol/l. While this error is still fairly small, in patients on intensive statin therapy targeting low LDL levels, fasting samples may be preferable. A minority of patients clears their lipids slowly; they typically have low HDL and elevated triglycerides while fasting, with further increase in triglycerides after meals. In these patients, non-fasting triglyceride levels may be even better predictors of future CVD events.

Biological variation, i.e., the normal day-to-day variation of total cholesterol, LDL cholesterol, and HDL cholesterol, is on the order of 3-5%. Many studies have also documented a seasonal variation. Although there is discordance between the studies, cholesterol levels tend to be higher in the winter months and lower in the summer months independent of the country of origin, ethnicity, age, sex, and baseline lipids. The seasonal variation has been reported to be as high as 12%.

Variation in lipid values

Age and gender – Cholesterol levels vary with age and sex. Under age 20, females have higher cholesterol levels than males. Adult males between 20 and 45 years of age generally have higher levels than females of the same age. Total cholesterol, LDL, and triglycerides increase with age for both sexes. Peak lipid levels for men generally occur between the ages of 40 and 60 and for women, between the ages of 60 and 80. Several factors that occur before or during blood collection, during storage, or shipping to the laboratory may affect the lipid results. It is important to understand and control these factors as much as possible in order to get accurate results.

Patient preparation and blood collection procedures should be standardized according to these guidelines

(1). Make note of whether the patient has fasted for at least 12 hours or has engaged in physical activity within the past 24 hours for any analysis other than total cholesterol.

(2). If only total cholesterol and HDL cholesterol are to be measured, either fasting or non-fasting samples can be used. However, the variability of cholesterol fractions may be increased postpandrially; thus, if triglycerides and lipoproteins are to be measured, the patient should be instructed to take nothing by mouth (other than water and prescribed medications) for at least 12

hours before the blood sample is taken.

(3). The patient should sit quietly for about five minutes before venipuncture. If the sitting position is not possible, the same position should be used each time the patient is sampled.

(4). Prolonged venous occlusion should be avoided. If a tourniquet is used, the sample should be obtained within one minute of tourniquet application. Release the tourniquet as soon as possible during venipuncture. If difficulties are encountered, use the other arm, or, if this is not feasible, release the tourniquet for a few minutes before attempting a second venipuncture.

The Friedewald formula was subsequently introduced to allow calculation of a low density lipoprotein cholesterol (LDL-C) level using fasting data (Langsted et al., 2008). The guidelines appropriately emphasize the general utility of a fasting lipid panel, they also indicate that obtaining lipids in the fasting state is preferred, rather than mandatory, depending on the clinical scenario (Mora et al., 2008). The definition of LDL-C estimated by the traditional Friedewald formula includes cholesterol contained in biological low density lipoprotein (LDL), intermediate-density lipoprotein, and lipoprotein (a). Patients with significantly elevated triglycerides may also have a genetic disorder, such as familial combined hyperlipidemia, familial hypertriglyceridemia, or familial type III dyslipoproteinemia. Fasting lipids, especially if combined with an apolipoprotein B (apo B) level, may help to distinguish among these conditions and define the risk for pancreatitis (NIH Publication, 1995). Patients with pancreatitis should have fasting lipids checked to assess whether hypertriglyceridemia (500 mg/dl) is the cause. After initiating treatment, a repeat fasting lipid profile should be rechecked to ensure that recurrent pancreatitis is unlikely.

Although screening the general population for elevated

fasting triglycerides to identify those at risk for pancreatitis is neither practical nor warranted, certain subpopulations of patients may benefit from testing. Such patients at increased risk include those:

(1) With human immunodeficiency virus (HIV) treated with highly active antiretroviral therapy,

(2) Treated with medications that can greatly elevate triglycerides, such as long-term high-dose steroids or retinoic acid derivatives;

(3) With visceral adiposity or those with a family history of dyslipidemia before starting oral contraceptive or hormone replacement therapy; or

(4) Women planning to get pregnant with known dyslipidemia or a family history of a lipid disorder. The greatest elevations in triglyceride levels often occur in these scenarios when genetically predisposed patients are exposed to an unhealthy diet, drugs that raise triglycerides, diseases associated with hypertriglyceridemia, or certain metabolic disorders.

Hyper triglyceridemia is accordingly to recent recommendations categorized as mild, moderate and extreme (Friedewald et al., 1972). Mild to moderate values are most often associated with cardiovascular events and non-alcoholic steatohepatitis while extremely elevated values are most often associated with pancreatitis and lipemia retinalis. On the contrary, low levels especially of non fasting triglycerides associated with relatively common mutations in lipoprotein lipase (LPL) have positive effect on mortality as found in recent work, in which stepwise reduced risk of mortality by decreasing concentrations of non fasting plasma triglycerides was detected.

Lifelong low triglycerides resulting from genetic polymorphisms are associated with a decreased burden of atherosclerosis expressed as coronary artery calcification (Friedewald et al., 1972). Mutation carriers had lower fasting and postprandial serum triglycerides, higher levels of HDL cholesterol and lower levels of LDL-cholesterol. Triglyceride levels less than 3.39 mmol/l the multivariate model adjusted for age, family history, fasting glucose, high-density lipoprotein cholesterol, blood pressure, body mass index, and changes between time 1 and time 2 in body mass index previous studies mentioned strong epidemiological evidence for the association between fasting and even more for non fasting triglycerides and cardiovascular disease. The robustness of this association depends on design of the study and statistical standardization. Moderately elevated plasma/serum triglycerides signalize increased risk for cardiovascular disease; extremely elevated triglycerides signalize increased risk for pancreatitis and lipemia retinalis. Several genetic and non-genetic factors are included in regulation of triglycerides levels. Extremely elevated triglycerides (concentration more than 10 mmol/l) not provoked by dietary factors, especially by high alcohol intake are more likely to have a monogenic cause. On the contrary, mildly to moderately elevated triglycerides (concentration 2 to 10 mmol/l) have mostly polygenic origin and often coexist with other metabolic disturbances, mainly with central obesity, insulin resistance and diabetes mellitus.

Risk in disease conditions

However, changes of circulating triglycerides are rather marker for changes in several metabolic pathways, some of them definitely associated with so called cardio metabolic risk. Most recently discussed is the role of Apo C-III which inhibits triglyceride hydrolysis and has been implicated in coronary artery disease (Stone et al., 2014; Sniderman et al., 2007).The most frequently discussed Triglycerides associated with several diseases are listed below. First, increased triglycerides in fasting and even more in non fasting status could be marker for increased levels of remnant lipoprotein particles, which could be directly atherogenic, they could penetrate into the vessel wall and cause inflammation. In contrast to LDL particles, they do not need to be modified / oxidized to become atherogenic (Pollin et al., 2008). They could be more important in women (Borén et al., 2015) especially in women changing reproductive status from premenopause to menopause (Hegele et al., 2014). Now a day's using so called non HDL/non LDL cholesterol obtained mainly from non fasting blood and calculated as total cholesterol minus LDL cholesterol minus HDL cholesterol. This parameter could rather reliably reflect cholesterol content in remnant lipoprotein particles and is strongly associated with cardiovascular events (Valdivielso et al., 2014). Second and already mentioned is the inappropriate impact of Apo C-III on metabolism of lipids, which often leads to hyper triglyceridemia. Third, increased triglycerides could also indicate malfunctioning reverse cholesterol transport, namely malfunctioning of fraction cholesterol esterification rate of HDL as a causative factor of increased cardiovascular risk. It is assessed by simple calculation from standard lipid profile as a logarithmically transformed ratio of molar concentrations of triglycerides to HDL-cholesterol. This parameter was named atherogenic index of plasma (AIP) (Bansal et al., 2007).

The close association of fraction esterification rate of HDL with AIP can be explained by triglyceride participation in the production of large VLDL and small dense LDLs and have also been proposed to be the major determinants of cholesterol esterification /transfer and HDL remodeling in particles that regulate the esterification rate. The strong correlation of AIP with HDL lipoprotein particle size may also explain its high predictive value. It was proved, that

AIP values significantly increase with increasing cardiovascular risk. AIP was the best predictor of positive findings (Piťha et al., 2015). AIP was also a highly sensitive marker of differences in lipoprotein profiles in families of patients with premature myocardial infarction and in control families. As a marker of lipoprotein particle size it adds predictive value beyond that of the individual lipids including total/HDL cholesterol ratio. However, the changes of these risk biomarkers with different therapies and their relation to treatment outcomes have not been studied. Another recently discussed entity is microvascular disease in patients with diabetes mellitus type 2 and its reduction by treatment by fenofibrate, drug reducing triglycerides (Varbo et al., 2014). In this causative factor might be stimulation of transforming growth factor beta by triglyceride-rich lipoproteins and inducing the production of reactive oxygen species causing damage to the glomeruli and glomerular glycocalyx and it could be speculated that these processes could also affect basal membranes in small vessels not only in kidneys. Excess amounts of a triglyceride-rich lipoproteins worsens diabetes-associated microvascular and macrovascular disease, increases glomerular injury, increases tubule interstitial fibrosis, and accelerates the progression of diabetic nephropathy (Dobiásová et al., 2006). Extreme hyper triglyceridemia could be also strong risk factors of pancreatitis. Excess of triglycerides hydrolyzed by high levels of pancreatic lipase released in the vascular bed of the pancreas. Subsequently, free fatty acids are formed in high concentrations, which overwhelm the binding capacity of albumin and they self-aggregate to micellar structures with detergent properties and then acinar cell and pancreatic capillary injury is promoted.

The resultant ischemia creates an acidic environment, which further triggers free fatty acids toxicity. Additionally, the elevated levels of chylomicrons increase the viscosity of blood and therefore impair the blood flow in the pancreas causing ischemia and acidosis within the pancreas. Furthermore, endoplasmic reticulum stress is and also causative factor (Dobiásová et al., 2011; Rajamani et al., 2009). Rather rare consequence of hyper triglyceridemia is lipemia retinalis which is a rare and asymptomatic condition which occurs when high levels of triglycerides and chylomicrons are present in blood. The main task in clinical practice is to assess if hyper triglyceridemia is atherogenic. Hyper triglyceridemia is rather marker than causative factor for several disease entities, but mainly for cardiovascular disease.

A recent AHA statement on hypertriglyceridemia and coronary heart disease suggests that clinicians can use a non fasting triglyceride level of >200 mg/dlto identify hypertriglyceridemic states (Rutledge etal., 2010). Among normo triglyceridemic subjects (i.e., fasting triglyceride levels <150 mg/dl), consumption of a low-fat breakfast (typically <15 g of fat) before blood sampling should not induce an increase in postprandial triglyceride levels by more than 20%, and is unlikely to cause levels to exceed 200 mg/dl (Thomsen et al., 2014). Additional more recent data has suggested that a non fasting triglyceride level of 175 mg/dl could also be reasonable (Valdivielso et al., 2014). Follow-up fasting triglyceride testing in these cases is not needed, but this should not dissuade further discussion of lifestyle measures. In contrast, among those with a non fasting triglyceride level 200 mg/dl, a follow-up fasting lipid panel in 2 to 4 weeks is helpful. Of note, if the non fasting triglyceride level is >1,000 mg/dl, a diagnosis of severe hypertriglyceridemia is present, and the heightened risk of hyperlipidemic pancreatitis should be addressed promptly.

During the last few years efforts have been made to simplify blood sampling by replacing fasting lipid profile with non-fasting lipid profile as it has been found that lipids, lipoproteins and apolipoproteins were not much different in fasting and non-fasting state with the exception of triglycerides which were higher in non-fasting state and all these were associated with cardiovascular risk prediction (American Heart Association 2011). However, a fasting sample is preferred if cardiovascular disease (CVD) risk assessment is based on total cholesterol, LDL cholesterol or non-HDL cholesterol but HDL cholesterol, triglycerides, total/HDL cholesterol ratio and apolipoprotein A-1 predict CVD when measured non fasting (Dubois et al., 1998). The most interesting part is that non-fasting triglycerides levels may be even better predictor of cardiovascular risk as compared to fasting triglycerides (White et al., 2015; Nordestgaard et al., 2009). Although the terms non-fasting and postprandial can be considered synonyms but there is some difference as non fasting sample means blood draw at any time without knowledge of the time of previous meal while post pandrial implies a sample at a fixed time after a standard meal. Moreover, triglycerides increase step wise after fat diet, therefore, non-fasting triglycerides would vary depending on time after meal with highest levels 4–5 h post prandially (De Backer et al., 2003). Further, the cut off levels of non-fasting triglycerides for cardiovascular risk have not yet been defined. It is important to compare serum lipid profile in fasting and at different time interval after a representative meal in terms of prediction of cardiovascular risk. As is true for fasting triglycerides, postprandial lipemia can be affected by ethnicity, alcohol consumption, and menopausal status, and thus these factors should be considered in clinical practice (Bansal et al., 2007). Thus, a lot has yet to be done in this area and then if the use of non-fasting lipid profile could be included in recommended guidelines then the sampling for lipid profile would be

simplified and this will improve the compliance for lipid lowering treatment. Till then we have to believe in fasting lipid profile for assessment and management of cardiovascular risk.

In addition to fasting/non-fasting state there are other factors (pre-analytical) which may affect lipid components:

(1). A change from an upright to a supine position due to dilutional effect can reduce the cholesterol levels by10% and triglycerides by 12% (Nordestgaard et al., 2007). (2). The disease conditions like nephrotic syndrome increase total cholesterol, LDL cholesterol and VLDL cholesterol (NCEP 2002) and hypothyroidism increases LDL cholesterol and total cholesterol. Infection and inflammation may decrease total cholesterol and HDL cholesterol and increase triglycerides. Lipids alter following myocardial infarction (Ridker et al., 2008; Narayana et al., 1996) and these changes may persist for several weeks. That is why it is better to do lipid profile in such patients within 24 h of myocardial infarction. The study (Joven et al., 1990) showed that all individual values of the lipid profile in patients admitted with acute illness vary significantly during and after hospital stay, whereas the ratio of total cholesterol to HDL remains relatively stable. It is important that all these factors should be kept in mind while interpreting the lipid profile.

In general, a non-fasting lipid test would be appropriate in the following clinical scenarios

CVD risk assessment, Initial investigation of lipid levels (unless the patient has a history of familial hyperlipidaemia), Monitoring lipid levels over time, Monitoring response to lipid-lowering treatment (unless the patient has high triglycerides), Testing for any reason in patients who are “hard to reach” or have low motivation for undergoing a fasting test.

Most evidence on calculating cardiovascular risk is based on fasting lipid test results, however, results from non-fasting lipid tests have also been shown to be strongly predictive of adverse cardiovascular events (Ryder et al., 1984; Nigam et al., 2004; Nawaz et al., 2006). During a CVD risk assessment, specific values are entered into a risk calculator, such as the Framingham equation. The Framingham equation uses total and HDL cholesterol values, which have the lowest variation between fasting and non fasting samples, and a range of other factors, such as blood pressure, to calculate risk (Kelly et al., 2005). The extra precision gained from a fasting result is therefore unnecessary (Greenland et al., 1990). Patients with a low cardiovascular risk and a non-fasting lipid-profile within the ideal range will not require further testing for five to ten years provided there are no significant changes to lifestyle or diet, or no significant new information arises, e.g. significant family history or relevant new personal history (Myers et al., 1989). A non-fasting lipid test can be used as an initial investigation of hyperlipidaemia. A non-fasting sample is appropriate for subsequent tests, unless very high triglycerides have been identified.

Conclusion

There are two inherent sources of variability in cholesterol and triglycerides measurements: biological and analytical, Biological variation is <5% for cholesterol, LDL cholesterol, and HDL cholesterol and 20 to 30% for triglycerides, Considerable variation can occur from one assay to another between clinical laboratories, for patient care, it is important to know if the LDL is calculated or is measured directly. In order to compare results from different laboratories, it is important to know which assay method is utilized. If patient is non-fasting, a direct LDL test is recommended. Sudden changes in lipid values may indicate a change in diet, medications, or onset of a new disease state.

We have shown that there are a number of clinical scenarios in which fasting lipids offer valuable clinical information, but that in others, non fasting lipids will suffice. To assess the initial risk of CVD in an untreated patient, fasting or non fasting total cholesterol and HDL-C levels provide all that is needed. Understanding a patient's metabolic burden can provide a useful baseline for lifestyle counseling. Although a diagnosis of metabolic syndrome requires a tally of metabolic risk factors measured in the fasting state, it can be approximated for practical purposes by non fasting results. Among those with a non fasting triglyceride level 200 mg/dl, a follow-up fasting lipid panel should be performed. Those who present with secondary causes of hyperlipidemia (due to diet, drugs, diseases, or disorders of metabolism) should have a fasting lipid panel performed. Indeed, it may be important for those about to initiate therapy with estrogenic hormones, steroids, retinoic acid, or certain anti neoplastic agents to understand their propensity for severe hypertriglyceridemia and subsequent risk of pancreatitis.

When assessing a patient's response to lipid therapy, the 2013 guidelines note that fasting lipids and calculation of the change in LDL-C allow estimation of therapeutic response and adherence to therapy. In other scenarios, however, including the one described in the introductory case, non fasting lipid scan provide requisite information without further inconvenience. Therefore, when attempting to answer whether fasting or non fasting lipids are most appropriate, it is important to first think carefully about the clinical scenario and consider what question is to be answered with the results.

Triglyceride one of the lipid profile, is a marker of cardiovascular disease as well as metabolic syndrome (MS) particularly in obese patients. Few studies have reported

that simplify the blood sampling by replacing fasting lipid profile with non-fasting lipid profile, since not much difference in fasting and non-fasting state. But triglycerides increases step wise after diet and reach a highest level after 5 hours post prandially. Previous studies suggest that estimation of fasting Triglyceride may be carried only for cardiovascular patients, but it is very difficult for the patients to know whether they are in high risk of cardiovascular until they diagnosis properly.

Conflicts of interest

There is no conflict of interest in the present study.

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