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Short Communication

After the Meal Surge: Rethinking Cardiovascular Risk Through Postprandial Triglycerides

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Abstract

While fasting lipid profiles have long been the cornerstone of cardiovascular risk assessment, growing evidence suggests that postprandial triglycerides (PPTs) may offer a more dynamic and clinically relevant measure of metabolic health. Following a meal, triglyceride-rich lipoproteins surge in circulation, reflecting the body's capacity to process dietary fats. Impaired clearance of these lipids contributes to endothelial dysfunction,

inflammation, and atherogenesis. This article explores the physiology of postprandial lipid metabolism, the pathophysiological consequences of elevated PPTs, and their emerging role as a predictor of cardiovascular disease. It also highlights diagnostic challenges and therapeutic strategies aimed at mitigating postprandial lipemia, ultimately advocating for a shift toward non-fasting lipid evaluation in clinical practice

Introduction

Traditionally, lipid testing has focused on fasting levels to standardize measurements and reduce variability. However, humans spend the majority of their day in a postprandial state, rendering fasting measurements somewhat limited in capturing real-world metabolic responses. Postprandial triglycerides (PPTs) represent the transient rise in blood triglyceride levels after food intake, particularly meals rich in fats and carbohydrates. Increasingly, research indicates that exaggerated or prolonged postprandial lipemia is a significant contributor to cardiovascular disease (CVD).

PhysiologyofPostprandialLipidMetabolism

After ingestion of a meal, dietary fats are absorbed in the intestine and packaged into chylomicrons—large triglyceride-rich lipoproteins. These particles enter the bloodstream via the lymphatic system and deliver triglycerides to peripheral tissues with the help of lipoprotein lipase (LPL). The remnants are then cleared by the liver. Simultaneously, the liver produces very-low-density lipoproteins (VLDL), which also contribute to circulating triglycerides.

In healthy individuals, this process is efficient, and triglyceride levels return to baseline within a few hours. However, in individuals with metabolic dysfunction—such as insulin resistance, obesity, or type 2 diabetes—this clearance is delayed, leading to prolonged elevation of triglycerides.

PathophysiologicalImplications

Elevated PPTs are associated with several atherogenic mechanisms:

- **Endothelial Dysfunction:** Triglyceride-rich lipoproteins impair nitric oxide availability, reducing vascular relaxation.
- **Inflammation:** Postprandial lipemia triggers inflammatory cytokine release, contributing to vascular injury.
- **Oxidative Stress:** Lipoprotein remnants are prone to oxidation, enhancing their atherogenic potential.
- **Formation of Small Dense LDL:** Elevated triglycerides promote the formation of more atherogenic LDL particles.

These processes collectively accelerate plaque formation and increase the risk of cardiovascular events.

ClinicalSignificance

Recent studies suggest that non-fasting triglyceride levels may be superior predictors of cardiovascular risk compared to fasting levels. Elevated PPTs have been linked to increased incidence of myocardial infarction, stroke, and overall mortality.

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Moreover, postprandial measurements may uncover hidden dyslipidemia in individuals with normal fasting lipid profiles. This has led to a paradigm shift in some clinical guidelines, which now accept non- fasting lipid testing as a valid alternative.

DiagnosticChallenges

Despite their clinical relevance, PPTs are not routinely measured due to several challenges

- **Lack of Standardization:** There is no universally accepted protocol for timing or composition of test meals.
- **Variability:** Individual responses to meals vary based on genetics, diet, and metabolic status.
- **Practicality:** Serial blood sampling over several hours is cumbersome in routine clinical settings.

To address these issues, simplified approaches such as single non-fasting triglyceride measurements or standardized fat tolerance tests are being explored.

ManagementStrategies

Reducing postprandial triglyceride levels involves both lifestyle and pharmacological interventions:

- **Dietary Modifications:** Limiting saturated fats and simple sugars, and increasing fiber intake can improve lipid metabolism.
- **Physical Activity:** Exercise enhances LPL activity and accelerates triglyceride clearance.
- **Weight Management:** Reducing adiposity improves insulin sensitivity and lipid handling.
- **Medications:** Fibrates, omega-3 fatty acids, and certain statins can reduce triglyceride levels and improve postprandial responses.

Emerging therapies targeting specific pathways in lipid metabolism are also under investigation.

Conclusion

Postprandial triglycerides offer a valuable window into the body's metabolic response to dietary fat and are increasingly recognized as important markers of cardiovascular risk. As our understanding of lipid metabolism evolves, integrating postprandial measurements into routine practice may enhance early detection and prevention of cardiovascular disease. Future research should focus on standardizing assessment methods and refining therapeutic approaches to better manage postprandial lipemia.

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