Editorial

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New findings on hypertriglyceridemia during pregnancy

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Severe hypertriglyceridemia, characterized by extremely high levels of triglycerides in the blood, is a significant risk factor for the development of acute pancreatitis, a severe and potentially life-threatening condition [1]. Acute pancreatitis occurs when the pancreas becomes inflamed, often leading to symptoms such as severe abdominal pain, nausea, vomiting, and, in some cases, systemic complications like organ failure. The presence of severe hypertriglyceridemia can exacerbate these effects by contributing to the formation of free fatty acids through the breakdown of triglycerides by pancreatic lipase. These free fatty acids can damage pancreatic cells and contribute to the inflammatory process. During pregnancy, the risk associated with severe hypertriglyceridemia is heightened due to physiological changes, such as increased insulin resistance and hormonal shifts, which can exacerbate lipid imbalances. When acute pancreatitis develops in this context, it can have catastrophic consequences, including preterm labor, severe maternal complications such as multi-organ failure, and even fetal loss or stillbirth [2]. Despite these severe outcomes, the precise mechanisms by which hypertriglyceridemia triggers acute pancreatitis-especially in pregnant women-remain poorly understood. Previous researches suggest that factors such as genetic predisposition, hormonal modulation of lipid metabolism, and inflammation might play crucial roles, but further investigation is required to clarify these pathways. This study by Sadakata and colleagues is elucidating the mechanism of abnormal lipid metabolism during pregnancy. The researchers investigated the involvement of glycosylphosphatidylinositol-anchored high-density lipoprotein-binding protein 1 (GPIHBP1), an anchor protein for lipoprotein lipase (LPL) – a key enzyme in lipid metabolism – by measuring its blood levels during different pregnancy stages. GPIHBP1 has recently attracted attention as a potential cause of severe hypertriglyceridemia. It plays a crucial role not only in the transport of LPL to the subendothelium of blood vessels but also in facilitating the binding of LPL to the surface of vascular endothelial cells, making it a vital component in LPL metabolism [3]. Cases of hyperchylomicronemia, thought to be caused by GPIHBP1 deficiency or GPIHBPI autoantibodies, have been reported [3,4]. In this study, during different pregnancy stages, blood concentrations of GPIHBP1 and LPL were observed to decrease transiently, returning to nonpregnant levels after delivery. When adjusted for albumin concentration, the decrease in GPIHBP1 levels was negated. However, LPL levels continued to show a transient decrease during late pregnancy, even after adjusting for albumin levels. These results suggest that during the course of normal pregnancy, the transient decrease in GPIHBP1 levels may be attributed to physiological dilution. In contrast, the transient decrease in blood LPL levels is likely due to inhibited lipolysis. This study has several limitations, including the reliability of the blood sample data and the influence of insulin resistance. To confirm these findings, further research is needed to elucidate the underlying mechanisms.

Refference

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