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LETTER

Hyperinsulinemia and Poor Coronary Collateral Circulation in Coronary Artery Occlusion: A Novel Finding, Yet of Therapeutic Significance or Not [Letter]

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Dear editor

We are documenting this to express our appreciation for the recent publication of the research article entitled Hyperinsulinemia Impairs coronary collateral circulation in patients with Chronic Total Coronary Occlusion (CTO) by Zou et al.¹ The authors did fantastic work, and the findings of the study have important clinical relevance as they highlight the effects of hyperinsulinemia on the development of collateral blood vessels in patients with CTO.

While the introduction highlights conditions that impede collateral development, such as diabetes and metabolic syndrome, other potential confounding variables or limits that could affect the study's findings in ultimately finding the essential association of hyperinsulinemia and collateral circulation in patients with CTO would be beneficial. Addressing these limitations up front allows readers to grasp the study's potential consequences and generalizability.

The study tells us that higher fasting insulin levels are associated with poor collateral circulation, which will help us in the future in the management of patients with ischemic heart diseases.

We really appreciate the way the author has explained the relationship between hyperinsulinemia and poor collaterals; this can also be used as a prognostic factor in people with ischemic heart disease. Although further studies should be done to investigate the consideration of hyperinsulinemia as a prognostic marker. Adding on, some targeted approaches to reducing insulin levels can provide a direction towards greater therapeutic value.

The study recognises that the sample size is limited. A larger sample size would give significant statistical strength to the study.

The author correlates the two variables in a systemic way, but a potent stimulator, vascular endothelial growth factor (VEGF), is underemphasized. As insulin regulates VEGF gene expression and is an angiogenic mediator that causes vascularization in the myocardium specifically via the insulin receptor and PI3K/Akt pathway.²

The authors investigated the role of hyperinsulinemia on collateral circulation in patients with CTO. However, the available evidence demonstrates that even this type of collateral flow is insufficient to prevent additional ischemia episodes in functional CTOs, pointing to the necessity of revascularization.³ Hence, in the larger context, the prognostic significance of hyperinsulinemia in saving lives is not established.

The study does not explain whether reducing insulin levels or addressing hyperinsulinemia could improve collateral flow.

The research was conducted at a single hospital, which may limit the general representation of the population. If multiple hospital patients were involved in the study, then the impacts would be of greater significance.

Disclosure

The authors report no conflicts of interest in this communication.

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https://doi.org/10.2147/DMSO.S423189