

The Importance of the “Obesity Paradox” in Cardiac Surgery

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Obesity Paradox in Cardiac Surgery

Obesity is an increasingly chronic health condition, predisposing to coronary artery disease (CAD) and adverse cardiovascular events, having independent effects on hemodynamics and cardiovascular structures, contributing to the derangement of adipose tissue function and inflammatory pathways [1,2]. Obesity promotes endothelial dysfunction favouring atherosclerotic processes, reduces insulin sensitivity, and enhances free fatty acid turnover, thrombogenicity and leptin resistance [3]. In addition, obesity is a cluster of related risk factors including hypertension, diabetes, dyslipidaemia and renal dysfunction, and exacerbating the ongoing cardiovascular disease epidemic [1-3].

Therefore, the reported association between obesity and mortality is not unexpected, despite several studies in both general and disease specific populations have surprisingly reported paradoxical survival benefit in obese patients [4,5]. This inverse relationship between body fat composition and all-cause mortality is commonly labelled as “obesity paradox” [6]. However, prior attempts to firmly demonstrate this paradox and to identify its pathophysiologic mechanisms yielded conflicting results [1-3]. Plausible explanations include increased lean body mass, protective peripheral body fat, reduce inflammatory response, reverse epidemiology, and genetics [4-7].

This should not be certainly taken as an argument to encourage body weight excess, and the ample evidences of the correlation between obesity and death, especially in the long-term period, are indisputable [1,2]. However, why is so important the obesity paradox in cardiac surgery?

In an experimental swine model, high fat diet promoting obesity and renal inflammation prevented post-CPB acute kidney injury [7]. Consonant data have been observed in a high-fat feeding mice model inducing coronary atherosclerosis with related metabolic anaerobic stress changes [8]. During global ischemia, heart with induced coronary disease were markedly resistant to ischemic-reperfusion injury than non-disease heart, suggesting a protective preconditioning

metabolic pathway [8]. Similarly, pigs fed a high-fat diet developing hyperlipidaemia in absence of significant weight gain were protected against myocardial ischemic-reperfusion injury [9].

Therefore, the complete understanding of underlying protective mechanisms behind the “obesity paradox” could improve clinical outcomes after cardiac surgery in the early perioperative period.

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