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Microvascular dysfunction in diabetic patients after cardiac surgery

Diabetes mellitus (DM) is associated with severe autonomic dysfunction and vasomotor dysregulation. DM has been associated with increased morbidity and mortality in patients undergoing any cardiac surgical procedures and following coronary artery bypass grafting (CABG) specifically. In particular, these changes are more profound in patients with poorly controlled diabetes. Diabetes is associated with vascular dysfunction in all tissues, including the microvasculature. DM is associated with significant changes in vascular reactivity of coronary/peripheral microcirculation, vascular permeability, gene/protein expression, and programmed cell death, as well as with increased morbidity and mortality after surgical procedures. Many of the microvascular and macrovascular complications of diabetes are related to increased oxidative/nitrosative stress, hyperglycemia, and changes in vascular signaling. Recently, we reported differential microvascular regulation before and after CP/CPB, correlating to the extent of serum glucose control. Alterations in vasomotor regulation can lead to vasoplegia, a common complication of CP/CPB seen in up to 25% of patients. Vasoplegia manifests with decreased systemic vascular resistance and hypotension. These patients are at increased risk of morbidity and mortality following cardiac surgery and CP/CPB. The incidence of postoperative vasodilatory shock is higher in patients with diabetes for a number of reasons. Vasoplegia has traditionally been treated with vasopressors, such as phenylephrine, and vasopressin. These medications must be administered carefully to avoid potentially dangerous side effects, including peripheral ischemia of the extremities and mesenteric ischemia, leading to tissue necrosis, mucosal injury and metabolic acidosis. In addition, peripheral vascular responses to vasoactive agents such as phenylephrine may affect the coronary circulation in a differential manner from the rest of the body by increasing systemic blood pressure suddenly while reducing coronary artery blood flow. A better understanding of the regulation of the microvasculature may lead to improved outcomes in the patients with and without diabetes.

Biography

Jun Feng is currently an Associate Professor of Surgery (Research) at Warren Alpert Medical School of Brown University and at Department of Surgery, Cardiovascular Research Center, Rhode Island Hospital. He is also the Director and Senior Research Scientist of Cardiothoracic Surgery Research Laboratory at Rhode Island Hospital. He serves as Principal Investigator on grants funded by National Institute of Health (2 active R01s and 2-NIH-COBRE-pilot projects), American Heart Association (Grant-In-Aid, active), and Rhode Island Foundation. He also serves as Co-investigator on a number of grants funded by the National Institute of Health and other research-funding organizations. He has published more than 120 peer-reviewed/editorial articles/book chapters and 140 abstracts as correspondent author, first author and co-author. He has served as an Editorial Member, Editorial Commentator and Peer Reviewer for several scientific journals in Cardiovascular Research and Medicine.

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