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Reperfusion therapy – what's with the obstructed, leaky and broken capillaries?

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Abstract

Microvascular dysfunction is well established as an early and rate-determining factor in the injury response of tissues to ischemia and reperfusion (I/R). Severe endothelial cell dysfunction, which can develop without obvious morphological cell injury, is a major underlying cause of the microvascular abnormalities that accompany I/R. While I/R-induced microvascular dysfunction is manifested in different ways, two responses that have received much attention in both the experimental and clinical setting are impaired capillary perfusion (no-reflow) and endothelial barrier failure with a transition to hemorrhage. These responses are emerging as potentially important determinants of the severity of the tissue injury response, and there is growing clinical evidence that they are predictive of clinical outcome following reperfusion therapy. This review provides a summary of animal studies that have focused on the mechanisms that may underlie the genesis of no-reflow and hemorrhage following reperfusion of ischemic tissues, and addresses the clinical evidence that implicates these vascular events in the responses of the ischemic brain (stroke) and heart (myocardial infarction) to reperfusion therapy. Inasmuch as reactive oxygen species (ROS) and matrix metalloproteinases (MMP) are frequently invoked as triggers of the