Abstract:

The heart and brain form an essential battery where each organ relies on the support of the other for homeostatic function. But what happens when one component of this network is damaged, for example after ischemic injury? Does damage to one organ predispose or even precipitate damage to distant and reciprocal organs? After acute myocardial infarction, an organized and balanced inflammatory response mediates tissue repair. Overzealous or imbalanced immune activity can result in infarct expansion, and worse prognosis culminating in heart failure. In addition to leukocyte invasion of the damaged myocardium, a systemic immune response is initiated which contributes to parallel neuroinflammation. This initial priming of microglia in the central nervous system may predispose the brain post-myocardial infarction to gradual cognitive impairment. Modulation of the cardiac inflammatory response affects the intensity of microglial activation, suggesting then opportunity to influence neurologic outcome. These processes can be effectively monitored using total body PET imaging of the mitochondrial 18kDa translocator protein TSPO, allowing simultaneous assessment of cardiac and neuroinflammation after ischemic injury. The expansion of total body imaging capabilities via multi position imaging and long bore PET cameras affords the opportunity to translate these approaches to clinical application. Collectively, these techniques provide mechanistic insights into the unique connection between heart and brain and provide fundamental basis for image guided therapy to improve long term outcome for both organs.