Postconditioning with lactate-enriched blood for cardioprotection in patients with ST-segment elevation myocardial infarction

My colleagues and I recently reported a new approach, postconditioning with lactate-enriched blood (PCLeB), for preventing reperfusion injury in patients with ST-segment elevation myocardial infarction (STEMI). This approach targets reperfusion-induced hypercontracture, which compresses the microvasculature and mechanically disrupts myocardial cell skeletons. PCLeB comprises intermittent reperfusion and timely coronary injections of lactated Ringer’s solution (Figure 1), aiming to achieve controlled reperfusion with tissue oxygenation and minimal lactate washout. This approach was designed based on the results of our previous experimental study. We have reported that abrupt lactate washout during reperfusion after simulated ischemia induced contracture in guinea-pig myocytes despite a substantial decrease in intracellular Ca\(^{2+}\) concentrations ([Ca\(^{2+}\)]\(_i\)), which were elevated during simulated ischemia. This reperfusion-induced contracture developed in association with resensitization of myofilaments to Ca\(^{2+}\). We therefore attempted to create a transition period between ischemia and reperfusion through our new approach. During this transition period, the elevated [Ca\(^{2+}\)]\(_i\) was allowed to resume its normal level safely, while restoration of vigorous myocardial contraction was suspended by keeping tissue lactate concentrations high, which otherwise might lead to hypercontracture. We have treated 76 consecutive patients with STEMI (age, 65.5 ± 14.1 years; 77.6% men; 43.4% anterior STEMI) using percutaneous coronary intervention and PCLeB within 12 h of symptom onset since late 2011. No patient experienced ventricular tachycardia or fibrillation during reperfusion. After PCI, corrected TIMI frame count was 20.4 ± 11.1 (normal value, 21). Peak creatine kinase and creatine kinase-MB levels were 2707 ± 2099 and 264 ± 170 IU/L, respectively. No patient died or experienced worsening/new-onset heart failure at 30 days. Only one patient required oral diuretic therapy at discharge. In conclusion, PCLeB induced augmented microcirculation recovery, abolished reperfusion arrhythmia, and led to zero mortality and no worsening/new-onset heart failure at 30 days in 76 consecutive patients with STEMI.

Related publications


**Biography**

Koyama is currently a vice director of the Saitama Municipal Hospital in Saitama, Japan. He has expertise in research in myocardial reperfusion injury. He has recently developed a new treatment strategy for myocardial reperfusion injury in patients with ST-segment elevation myocardial infarction (STEMI), based on the results of his previous experimental study using guinea-pig myocytes that was published in *Am J Physiol* in 1991. He is basically a clinical cardiologist, performing percutaneous coronary intervention himself. But his experiences not only in STEMI treatment but also in animal experiments inspired him to develop a new treatment strategy for myocardial reperfusion injury, i.e. postconditioning with lactate-enriched blood. He has already published a review paper on this new approach in *IJ Cardiol Heart & Vasculature*, claiming that the new approach may be effective against all four types of myocardial reperfusion injury.

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