The beneficial effect and mechanism of QiShenYiQi Pills on ischemia/reperfusion induced myocardial injury and fibrosis in rats

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Thrombolytic therapy and/or percutaneous coronary intervention may lead to myocardial ischemia and reperfusion (I/R) injury, which further develops to myocardial fibrosis, and finally heart failure. Myocardial I/R injury, resulted from recanalization of the occluded vessels, is a complicated pathological condition including abnormal energy metabolism, oxidative stress, inflammatory factors and apoptosis. Ribosomal protein S19 (RP S19), released after myocardial injury, induces monocyte infiltration and macrophage polarization towards M2. M2 macrophages secrete transforming growth factor β 1 (TGF β 1), which acts on fibroblast TGF β receptor II (TGFR β R II), activates Smad system, and leads to collagen deposition and myocardial fibrosis. However, at present, drugs recommended by the guidelines are not able to block myocardial I/R injury and fibrosis.

QiShenYiQi Pills (QSYQ), consisting of Radix Astragalus, Salvia miltiorrhiza Bunge, Panax notoginseng and Dalbergia odorifera, is a compound Chinese medicine (Registration NO.: Z20030139), which is adopted by expert consensus on the prevention and treatment of chronic heart failure by combining traditional Chinese medicine with western medicine. Our work demonstrated that pre-treatment with QSYQ improved myocardial energy metabolism and ameliorated myocardial injury during the ischemia period. It also reduced myocardial infarction area and apoptosis and improved myocardial blood flow. On the other hand, post-treatment with QSYQ inhibited myocardial fibrosis, improved cardiac function and increased myocardial blood flow after I/R, which was related to inhibiting RP S19 release, monocyte infiltration, macrophage polarization towards M2, TGFβ1 release, Smad activation and collagen deposition. Our in vitro study demonstrated that QSYQ reduced C5aR protein expression and C5aR-RP S19 co-localization in monocytes and TGF β R II expression and P-Smad3 translocation from cytoplasm to nuclei in fibroblasts. In the seminar, the speaker will systematically introduce the mechanism underlying myocardial injury and myocardial fibrosis induced by I/R, as well as the beneficial effect and possible mechanism of QSYQ on myocardial injury and fibrosis.