**GLUCOCORTICOID AMELIORATED EARLY CARDIAC DYSFUNCTION AFTER CORONARY MICROEMBOLIZATION BY SUPPRESSING TGF-BETA1/SMAD3 AND CTGF EXPRESSION**

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Objectives: This study was designed to evidence the protective effect of glucocorticoid therapy on cardiac dysfunction after coronary microembolization (CME), and clarified its mechanism with the expression of TGF-beta1/Smad3 and CTGF.

Methods: Fourteen mini-pigs were subjected to this study: Sham-operation group (n=4), CME group (n=6) and Glucocorticoid therapy group (n=4, received methylprednisolone 25mg/kg intravenously 30 minutes before CME). Cardiac magnetic resonance imaging was performed to evaluate cardiac function at baseline, 6th hour and 1 week after CME. Serum TGF-beta1, CTGF and troponin T were detected after CME. Myocardial expressions of TGF-beta1, CTGF, Smad3 and total collagen expression were also detected.

Results: Compared with Sham-operation group, the values of left ventricular end-systolic volume and left ventricular end-diastolic volume in CME group were increased at 6th hour after CME, while left ventricular ejection fraction (LVEF) was decreased (62.3±2.8% vs. 52.3±2.8%, P<0.05). Methylprednisolone greatly improved LVEF after CME, especially at 6th hour after CME (6th hour after CME: 58.6±1.9% vs. 52.3±2.8%, P<0.05). Furthermore, methylprednisolone not only decreased serum TGF-beta1, CTGF and troponin T, but also reduced myocardial expressions of TGF-beta1, CTGF and Smad3 after CME. In addition, collagen volume fraction in glucocorticoid therapy group was markedly lower than that in CME group.

Conclusions: Glucocorticoid therapy could improve early cardiac function after CME, and its mechanism could be associated with TGF-beta1/Smad3 and CTGF suppression.