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EFFECTS OF RENAL SYMPATHETIC DENERVATION SODIUM-WATER HOMOEOSTASIS OF HEART FAILURE AFTER CARDIAL INFARCTION IN RATS

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Objectives Heart failure (HF) is the common end stage in the development of a variety of structural heart disease. The excess activation of the renin—angiotensin—aldosterone (RASS) system played an important role in the development of HF. Recently, it has been shown that renal sympathetic denervation (RD) effectively inhibited the excessive activation of the RASS system and improved the refractory hypertension. However, whether we could achieve the purpose of prevention and treatment of heart failure through the inhibition of the RASS system in patients after myocardial infarction (MI) remains to be further studied. The purpose of the present study was to investigate the effects of RD at different time points on RASS system related hormone levels and water sodium excretion in the progress of HF due to MI.

Methods 60 Male Wistar rats, weighing 300±20 g, were divided into six groups randomly: rats with RD 1 day after MI (MI_1+RD, n=10), rats with RD 1 week after MI (MI_7+RD, n=10), rats with RD 1 month after MI (MI_{30}+RD, n=10), rats with MI and renal innervations (MI+INN, n=10), rats with RD but without MI (Sham+RD, n=10), rats without MI and with renal innervation (INN+Sham, n=10). MI was induced by ligation of the left anterior descending coronary atery. The hallmark of a successful MI model is S-T segment raising significantly 30 min after MI compared with before ligation through ECG. EF<50% is the sign of HF. Bilateral RD was coursed by stripping the adventitia of renal arteries. Four-weeks after treatment, measured urine output, urinary sodium content, and the content of rennin, angiotensin II and aldosterone in plasma respectively.

Results

1. Sham+RD: Compared with INN+Sham group, there were no significant difference ($p>0.05$) in the urine output, urine sodium content and plasma renin, angiotensin II and aldosterone content.
2. MI+INN: The incidence of HF was 92%, compared with Sham +INN group, the urine output decreased obviously $p<0.05$), urinary sodium content did not change significantly, plasma renin, angiotensin II and aldosterone content were increased visibly $p<0.05$).
3. MI_1+RD and MI_7+RD: Compared with the MI group, the incidence of HF were much lower in MI_1+RD and MI_7+RD group, urine output was significantly increased $p<0.05$), plasma renin, angiotensin II and aldosterone content were obviously decreased $p<0.05$), and all the indicators above closed to normal. There was no significant difference in these indicators between these two groups.
4. MI30+RD: Compared with MI+INN group, except for plasma angiotensin II concentration decreased significantly lower, the other indexes had no visible improvement ($p>0.05$).

Conclusions

1. RD has little impact on salt and water metabolism at normal level, so it is a therapy with high security.
2. RD operated within 1 week after MI, can effectively inhibit the excessive activation of the RASS system after MI, improve retention of sodium and fluid, prevention and treatment HF. Its efficacy is similar, no matter what time RD operated, as long as in the first week after MI. And the efficacy maintained at least 4 weeks.