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Abstract

To investigate the interaction between immune inflammatory response and sympathetic nervous system in the central regulation of chronic heart failure in rats.Methods: Branch ligation of the left anterior descending the making rat model of chronic heart failure, by AT1 receptor blockade losartan central intervention, intravenous injection of six Ting quaternary amines detected in rat basal sympathetic activity level, supersonic and enchanted figure for the detection of cardiac function, ELISA method for detection of plasma catecholamine and inflammatory factor levels.Results: Compared with the sham operation group, markedly enhanced the heart failure rat sympathetic nerve activity level, peripheral plasma TNF alpha, IL-1 beta and NE levels were significantly increased ($P < 0.05$) and cardiac dysfunction ($P < 0.05$); give AT1 receptor blocker losartan intervention RAS in central nervous system after the excessive enhancement of sympathetic nerve activity levels have decreased significantly, peripheral TNF alpha, IL-1 beta and NE levels were decreased ($P < 0.05$), cardiac function increased significantly ($P < 0.05$).

Index terms— chronic heart failure, sympathetic nervous system, inflammatory cytokines.

1 I. Introduction

mmune inflammatory response and sympathetic nervous system activation plays an important role in the occurrence and development of chronic heart failure (CHF) [1,2] ,But its activation mechanism and the relationship between central and peripheral levels of activation are not clear. There is evidence to show that CHF central renin angiotensin angiotensin system (RAS) over enhancement, central Pro inflammatory cytokines (PIC) increased expression, and promote the activity of the sympathetic nervous system over enhancement, but the second not mediated CHF state week immune inflammatory reaction is uncertain [3,4]. In this study, the CHF model of myocardial ischemia was made by ligation of the left anterior descending branch of the left coronary artery, and the central and peripheral sympathetic activity and immune inflammatory response were observed at different time points after ligation. And through the intervention of the central level of RAS, to observe whether the coronary artery ligation in rats can reduce the level of immune inflammatory reaction and sympathetic nervous system activity, and improve the cardiac function. To provide new ideas and basis for the prevention and treatment of CHF. weeks and plasma catecholamine, PIC levels were compared with the sham operated rats, coronary artery ligation rats plasma NE levels with time prolonged significantly increased, e non significant difference; coronary artery node ligation group plasma pic levels were also increased with time and increased significantly (Figure **??**). 2). 3). IV. Discussion Chronic heart failure (CHF) is a serious hazard to human health, but the treatment effect is not good enough. Urgent need to explore the mechanism of CHF disease progression in order to find a more effective treatment. There is an interaction between sympathetic nervous system and immune system activation in the CHF state. In the past, most of the researches are based on the activation of sympathetic nervous system and immune system, which is an effective method for the treatment of CHF. Less research about the interaction between sympathetic nervous system and immune system in CHF, especially

the change of the peripheral activity of a certain factor. Studies have shown that CHF myocardial ischemia and infarction by autonomic nerve afferent signals reach the central, thereby inducing central pic increased generation [5,8], and central pic and ROS **??**2; 7]; mutual effect of 8] and RAS [9] system in control of sympathetic activity, inhibition of central pic can reduce the CHF of the sympathetic nervous system excitability [6,7].However, it is not clear whether CHF and RAS in the PIC state of cardiovascular central nuclei, such as the nucleus of the hypothalamus (PVN) and the rostral medulla (RVLM), mediate the inflammatory response. In this study, the CHF model of myocardial ischemia was made by ligation of the left anterior descending branch of the left coronary artery, and the central and peripheral sympathetic activity and immune inflammatory response were observed at different time points after ligation. Compared with the sham operated rats, coronary artery ligation rats plasma NE levels with time prolonged significantly increased, e non significant difference was found; coronary artery ligation rats plasma pic levels also with time prolonged increased significantly; coronary artery ligation group rat paraventricular nucleus (PVN), Yin cord rostral ventrolateral (RVLM) TNF alpha and IL-1 beta level from 3 days to 6 weeks were significantly increased, but between each time point without significant difference. Further, we use via mini osmotic pumps to the bilateral lateral ventricle for 6 weeks to give AT1 receptor blocker losartan intervention central RAS, observe whether it can reduce the coronary artery ligated rats the level of peripheral inflammatory reaction and the activity of the sympathetic nervous system, improve heart function; Results suggest that heart failure rat sympathetic nerve activity level was significantly enhanced and peripheral plasma TNF alpha, IL-1 beta and NE levels were significantly increased ($P < 0.05$), and given the AT1 receptor blockade losartan via mini osmotic pumps to the bilateral lateral ventricle administration intervention RAS in central nervous system after the excessive proliferation of strong sympathetic nerve activity level decreased significantly and peripheral TNF alpha, IL-1 beta and NE levels were decreased ($P < 0.05$). That heart failure rat central RAS inhibition can reduce the peripheral excessive inflammatory reaction and the activity of the sympathetic nervous system, also found that the intervention after cardiac function was significantly improved, and the model group were significant differences ($P < 0.05$), suggesting that the cardiac function as with inhibition of Ras in central nervous system, thereby reducing the CHF when the excitement of the sympathetic system and outer peripheral immune inflammation. This study is expected to explain the mechanism of the interaction between the sympathetic nervous system and the immune system, and provide a new idea and basis for the prevention and treatment of CHF. In addition, this study on heart failure rats given AT1 receptor blockade losartan via mini osmotic pumps to the bilateral lateral ventricle administration intervention central Ras levels found the pivot pic levels were also significantly decreased, the interaction between the two and peripheral inflammation, sympathetic nerve activity regulation of network access and the specific mechanism still need further study.

2 II. Materials and Methods

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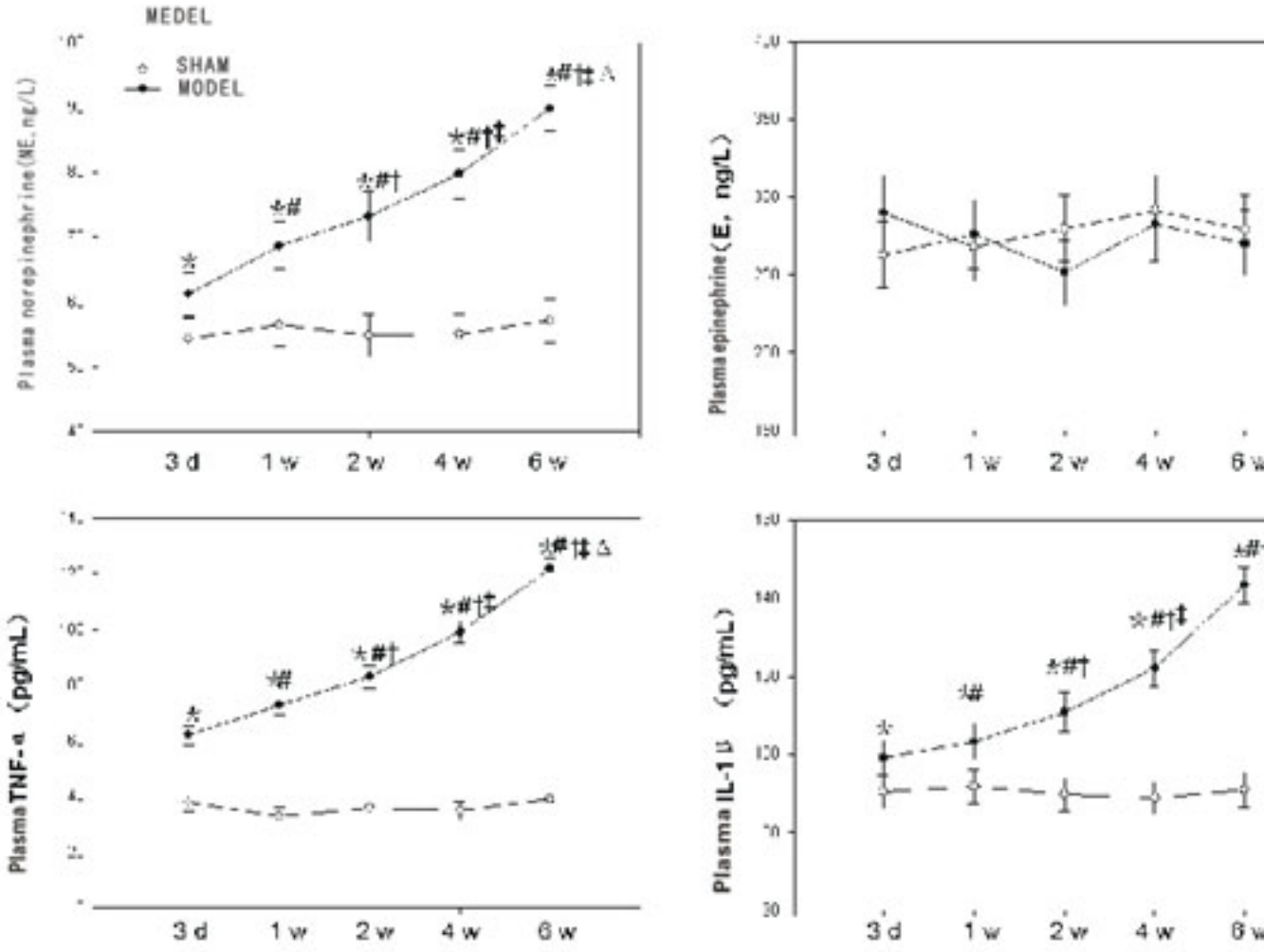

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Figure 1: Fig. 1 : 2)

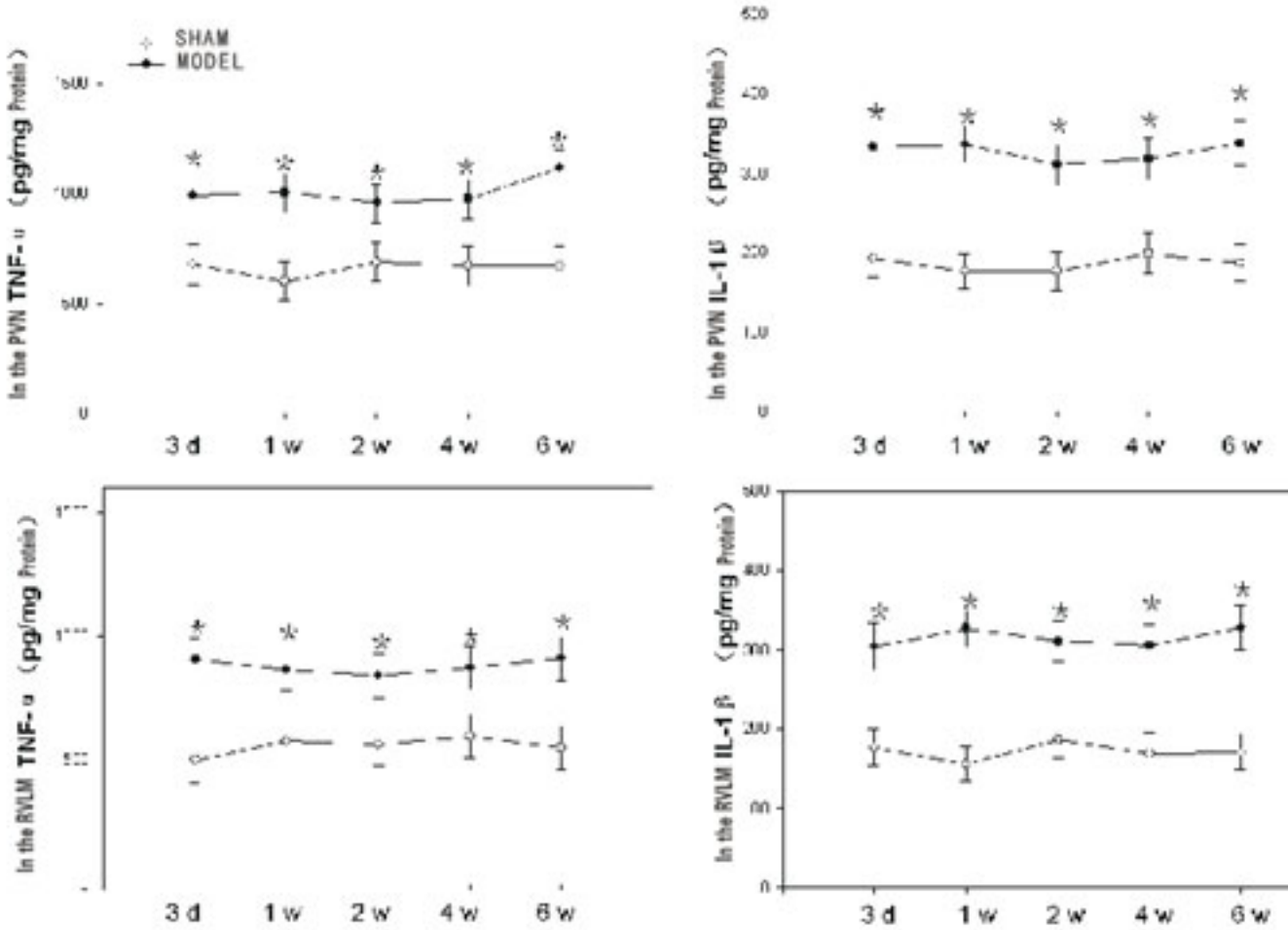

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Figure 2: Figure 2 :

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ligation;

*[Note: a) Animals and groups: 36 rats of SPF grade male Sprague Dawley rats (Chinese Academy of Sciences Shanghai Laboratory Animal Center, weight of 280 to 320 g, were randomly divided into 3 groups: operation group (model), sham operation group (sham) and central intervention group (int), 12 rats in each group; central intervention group and operation group underwent coronary artery ligation surgery. In the sham group only thread Note: compared with Sham group, *P<0.05; compared with Model group, #P<0.05. b) Sympathetic nerve activity level Compared with the sham operation group rats, the rats were subjected to left coronary anterior descending artery ligation]*

Figure 3: Table 1 :

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Figure 4: Table 2 :

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Figure 5: Table 3 :

[Am J Physiol Heart Circ Physiol ()] , *Am J Physiol Heart Circ Physiol* 2004. 287 (2) p. .

[Am J Physiol Heart Circ Physiol ()] , *Am J Physiol Heart Circ Physiol* 2004. 286 (6) p. .

[Circ. Res ()] , *Circ. Res* 2006. 99 (7) p. .

[Hypertension ()] , *Hypertension* 2007. 49 (3) p. .

[Am J Physiol Heart Circ Physiol ()] , *Am J Physiol Heart Circ Physiol* 2007. 293 (1) p. .

[Eur J Heart Fail ()] , *Eur J Heart Fail* 2008. 10 (7) p. .

[Am J Physiol Heart Circ Physiol ()] , *Am J Physiol Heart Circ Physiol* 2008. 295 (1) p. .

[Cardiovasc Res ()] , *Cardiovasc Res* 2008. 79 (4) p. .

[Neuroscience Bulletin ()] , *Neuroscience Bulletin* 2012. 28 (4) p. .

[Francis et al.] *Acute myocardial infarction induces hypothalamic cytokine synthesis*, J Francis , Y Chu , A K Johnson .

[Kang et al.] *Cross-talk between cytokines and renin-angiotensin in hypothalamic paraventricular nucleus in heart failure: role of nuclear factor-kappaB*, Y M Kang , Y Ma , C Elks .

[Guggilam et al.] *Cytokine blockade attenuates sympathoexcitation in heart failure: cross-talk between nNOS, AT-1R and cytokines in the hypothalamic paraventricular nucleus*, A Guggilam , K P Patel , M Haque .

[Yu et al.] *Increased cyclooxygenase-2 expression in hypothalamic paraventricular nucleus in rats with heart failure: role of nuclear factor kappa*, Y Yu , Y M Kang , Z H Zhang .

[Kang et al.] *Inhibition of brain proinflammatory cytokine synthesis reduces hypothalamic excitation in rats with ischemiainduced heart failure*, Y M Kang , Z H Zhang , B Xue .

[Francis et al.] *Neural regulation of the proinflammatory cytokine response to acute myocardial infarction*, J Francis , Z H Zhang , R M Weiss .

[Kang et al.] *Novel effect of mineralocorticoid receptor antagonism to reduce proinflammatory cytokines and hypothalamic activation in rats with ischemiainduced heart failure*, Y M Kang , Z H Zhang , R F Johnson .

[Guggilam et al.] *TNF-alpha blockade decreases oxidative stress in the paraventricular nucleus and attenuates sympathoexcitation in heart failure rats*, A Guggilam , M Haque , E K Kerut .

[Cheng et al.] *Using optogenetics to translate the "inflammatory dialogue" between heart and brain in the context of stress*, Jinbo Cheng , Jie Zhang , Caiyi Lu .