Myocardial remodeling in Kunming mice induced by chronic exposure to Carboxyl-terminal polypeptide of Cardiotrophin-1

CHEN Shu-fen1, ZHANG Wei2, WEI Tao-zhi3, XU Ming-guang4, DONG Zhan-ling1, ZHANG Huì5
1. Central Clinical Laboratory, Affiliated Hospital of Hainan Medical University, Haikou 570102; 2. Department of Cardiology Medicine, People’s Hospital in Hainan Sanya, Sanya 572000; 3. Department of Cardiology Medicine, Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan 430022; 4. Department of Physiology, Hainan Medical University, Haikou 571101, China; 5. Affiliated Hospital of Hainan Medical University, Haikou 570102, China.

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[Author]: CHEN Shu-fen (1966-), Female, Yining Xinjiang, Associate Researcher, M.M., Tel: 13034995295, E-mail: csf_f66@163.com.

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View from specialist: It is creative, and of certain scientific and educational value.

[ABSTRACT] Objective: To study histiocytes characteristics of myocardial remodeling induced by chronic exposure to cardiotrophin-1 in Kunming mice. Methods: Sixteen amino acids from carboxyl-terminal of mouse CT-1 were selected and synthesized to a polypeptide (Carboxyl-terminal polypeptide of Cardiotrophin-1, CT-1-CP), and then were injected intraperitoneally to the Kunming mice for 1 to 4 weeks (4 groups, n = 10, 5 male), the control group (n = 10, 5 male) received intraperitoneal injection of physiological saline for 4 weeks. At every end point of injection the body and heart were weighted, and the hearts tissues samples were isolated and embedded in paraffin for preparing tissue section. The HE and MASSON staining were used for dynamic morphological histological analysis. Results: The increase of body weight at first and second week was higher in the control group than that of the CT-1-CP-injected groups, but was significantly lower than that of the latter at third (t = 4.821, P < 0.01) and forth week (t = 2.019, P < 0.05), and the body weight of the four-week group had exceeded slightly the control group; The heart weight of mice in CT-1-CP-injected groups were higher than that of the control group, but the heart to body weight ratio has no significant difference among them (F = 1.083, P > 0.05). Intraperitoneal injection of CT-1-CP one weeks later, the mice began to appear enlarging of ventricular cavity and thinning of the ventricular wall, and the deranged myofibril and the blurred cross striation complicated with uneven staining of cytoplasm, which were scattered in the ventricular wall, were detected. After 2 weeks, the anatomical change of ventricles became deteriorated and local hypertrophy of ventricular wall appeared, and the focuses of pathological changes of cardiomyocytes increased. 3, 4 weeks later, the lesion was more obvious and the scope of focuses gradually expanded. In focuses with serious pathological change some myofibrils were fragmented and some sarcomeres were partially or wholly lost. Masson staining also shows exaggerated growth of the fibril-connective tissues surrounding myofibrils and in the spaces where the myofibril array disturbed or sarcomere defected. Conclusions: Long-term exposure to CT-1-CP could promote cardiac hypertrophy and lead to cardiac remodeling in Kunming mice as well as the ultrastructure damage of cardiomyocyte in remodeled myocardial tissues, otherwise, induced the hyperplasia of fibril-connective tissues.

[KEY WORDS] Carboxyl-terminal polypeptide of Cardiotrophin-1 (CT-1-CP); Myocardial remodeling; Hyperplasia