Western and TCM treatment of VMC Cull[4]demonstrated the role of interferon in the treatment of myocarditis, and the experiments show that interferon I transgene expression can alter the cytotoxicity of cytomegalovirus infection. At present, 1,6 diphosphate fructose and antioxidant vitamin C and energy mixture in the treatment of VMC is also more common, and the effect is also recognized[5].

VMC is a modern medical term, according to its different clinical symptoms can be attributed to the motherland medicine “palpitations” “palpitation” “chest” and “virtual” and other areas. The national standard “Chinese medicine clinical diagnosis and treatment terminology” named it as “heart attack”. Qing Lin[6] divided the disease into 4 types: toxic heat ,pathogenic factor demage the heart-yin,Deficiency of Qi and YIN, Deficiency of Yin and Yang. Zhigancao Decoction which comes from the”Treatise on Febrile Diseases”. It is commonly used in the treatment of VMC in clinic and held a hight cure rate. Animal experimental studies have shown that, it can effectively inhibit the myocardium inflammation and connective tissue proliferation of the mice infected with CVB3[7].

Thinking and Prospect Recently, compared with modern medicine treatment on symptomatic, TCM has got remarkable achievements.However, prescription is a complex giant system and it is not clear that the mechanism of traditional Chinese medicine treatment of VMC. As early as the beginning of twentieth Century, Xijun Wang[8] has put forward the concept and systematic method of “Chinmedomics”, which has being perfected and achieved great breakthrough after many years of practice. Wang’s team is currently working on VMC . It is believed that in the near future, the mechanism of traditional Chinese medicine in the treatment of VMC will be clarified, and explore the pharmacological basis of the prescription for it, so that, the Traditional Chinese Medicine can be more accepted by the world and promoted to be internationalization and modernization.

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RESEARCH PROGRESS OF THE VIRAL MYOCARDITIS PATHOGENESIS IN RECENT YEARS

Hui Shi, Guangli Yan, Hongwei Song, Hui Sun, Aihua Zhang, Xijun Wang*

Sino-America Chinmedomics Technology Collaboration Center, National TCM Key Laboratory of Serum Pharmacochemistry, Chinmedomics Research Center of TCM State Administration, Metabolomics Laboratory, Department of Pharmaceutical Analysis, Heilongjiang University of Chinese Medicine, Heping Road 24, Harbin 150040, China. E-mail:xijunwangls@126.com.

Abstract Viral myocarditis is the focal or diffuse myocardial cell degeneration and necrosis, accompanied inflammatory cell infiltration by virus, which leads to the myocardial injury, cardiac dysfunction and arrhythmia. Most patients can recover, but some patients will develop chronic inflammation or dilated cardiomyopathy. Review the pathogenesis of viral myocarditis has not been fully elucidated; the direct effects of the virus and the immune response of the body are the main pathogenesis of viral myocarditis. This review summarizes the current progress about the pathogenesis of viral myocarditis in direct damage of the virus, immune response and so on.

Key words Viral myocarditis; Pathogenesis; Virus; Immune response; Dilated cardiomyopathy.

Introduction Myocarditis is common cause for inflammatory heart diseases. There are three distinct phases in the pathogenesis of viral myocarditis. The first stage is the replication phase of the virus and its direct damage to the cardiomyocytes [1]. The second phases, myocardial injury induced by immune response [2]. The third stage is dilated cardiomyopathy [3]. This paper reviews the research progress on the pathogenesis of viral myocarditis.

Virus The viruses which cause myocarditis have Coxsackie viruses B (CVB) [4], human herpes virus 6 and parvovirus B19 and so on [5-6]. The virus enters the cardiac myocardial cell, in a short period of time to inhibit the physiological functions of cells, leading to cell rupture caused by increased cell membrane permeability. Direct destruction of cardiomyopathy occurs by virus mediated lysis, causing degradation of cell structures, which in turn facilitates entry of the virus into the cells with consequential myocyte injury and cardiac dilatation. This initial

phase frequently passes unnoticed since the initial damage is often prevented by the innate immune response.

Immunity Cell-mediated immunity plays an important role in the pathogenesis of viral myocarditis. T lymphocytes are mainly caused by the effect of myocardial cell injury immune cells. T responses in the pathogenesis of myocarditis has included T helper (Th) 1 [7], Th17 [8] and Th22 response [9]. Recent data indicate that elevated Th2 and Th17 responses during acute CVB3 myocarditis are critical for the progression from myocarditis to DCM and heart failure because of their ability to induce cardiac remodeling.

Dilated cardiomyopathy Part of the viral myocarditis (VMC) delayed healing eventually develop dilated cardiomyopathy (DCM), which is a kind of composite cardiomyopathy etiology, left ventricular and right ventricular or double heart enlargement, cause cardiac dysfunction such as characteristic. The results showed that the B virus (CVB) and DCM were most closely related to viral infection, especially Coxsackie virus [10].

Conclusion In summary, viral myocarditis is a complex process of interaction virus direct injury, immune response and so on. It is can control the occurrence and development of viral myocarditis through study the pathogenesis and mechanism of viral myocarditis. With the continuous development of modern immunology and molecular biology, for the study of the pathogenesis of VMC provides an important method. The study of the immune mechanism inhibits viral invasion of myocardium and its immune responses will for the therapy of myocarditis open a new way.

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STUDY ON THE MODEL OF COMMONLY USED RAT FEVER

Jialin WU,Yongji Li*

(Research Institute of College of Pharmacy, Heilongjiang University of Chinese Medicine, Harbin 150040,China)

Abstract Objective: To explore the process and characteristics of fever induced by dry yeast, 2,4-dinitrophenol,lipopolysaccharide (LPS) in SD rats, and to compare theinfluence of different types of exogenous pyrogens on the fever process.

Methods: To establish rat fever models induced by ten percent of dry yeast (10ml/kg), and 2 ,4 -dinitrophenol(20mg/kg) , LPS(20μg/kg),record the fever values at different time points, and compare their fever characteristics.

Results: In the febrile rats induced by subcutaneous injection of dry yeast suspension, first the temperature to drop, temperature began to rise after 2 hour to 3 hour, reached to the peak value after 5 hour to 7 hour, and lasted for 21 hour. In the fibrile-rats induced by subcutaneous injection of 2,4 -dinitrophenol solution,the temperature began to rise after 20min, reached to the peak value after 1-1.5 hour, and lasted for 4 hour to 5 hour. In the febrile rats induced by intraperitoneal injection of LPS,temperature began to rise after 0.5 hour, then the fever curves were biphasic or triphasic, and lasted for 6 hour to 8 hour.

Conclusions: Different exogenous pyrogens at different concentrations cause different fever process and characteristics in SD rats. In antipyretic experiments,suitable fever models should be appropriately selected based on the nasture of the tested drug and according to the types of fever process and characteristics of the used animal models.

Key words: Febrile rats model; Fever process; Fever characteristics; Drug research

1. Materials and methods

1.1 Materials