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News & Comments

Bisac Bisacurone: A Crucial Agent for the Therapeutic of Congestive Heart Failure

Muhammad Sajid

According to one study, aortic stenosis (AS) is one of the most common causes of CHF (Congestive Heart Failure), affecting about 3% of people over the age of 65, with an overall survival rate of 2-3 years in symptomatic patients. Every year, it is estimated that more than 4 million Chinese people are affected by CHF, with a 30% death rate during three years. Even though CHF is a rapidly developing cardiovascular illness, it is associated with a significant economic burden, with treatment costs of $126.819 per patient.

Various pharmaceutical therapies, such as angiotensin-II receptor blockers (ARBs), $-blockers, RAS inhibitors, ACE inhibitors, antifibrogenic drugs, and aldosterone antagonists, are now used to treat CHF. Cardiac hypertrophy caused by aortic constriction is a widely used, well-established, and highly reproducible experimental animal model that has been utilized by several researchers to assess the efficacy of various therapeutic strategies to prevent LV remodelling.

Bisacurone, a bioactive terpenoids derived from C. longa, is widely known for its antioxidant and anti-inflammatory activities, which are achieved by inhibiting the release of proinflammatory cytokines including tumour necrosis factor-alpha (TNF-"). Bisacurone has also been shown to have hepatoprotective properties in both preclinical and clinical studies.

The experiment was performed in the Pharmacology laboratory of the Department of Cardiology, Pidu District People's Hospital Chengdu, China. Adult male Sprague-Dawley rats (200-250 g, 7-8 weeks, n = 110) were procured from the Third Affiliated Hospital of Chengdu Medical College's experimental animal centre. MP Biomedicals India Private Limited, India, provided the total ribonucleic acid (RNA) extraction kit and the one-step qualitative reverse transcriptase-polymerase chain reaction (RT-PCR) kit. The AS rats were randomly allocated into the following experimental groups (n = 15) one week after surgery. Bisacurone was newly made in three distinct dosages (25, 50, and 100 g kg^{-1})and given orally to all groups once daily for 28 days at a pre-determined time. Rats were anaesthetized with urethane injection (1.25 g kg^{-1} i.p.) at the end of the treatment period, and blood was taken by a retro-orbital puncture.

After that, all animals were slaughtered, and the heart was immediately isolated on the 29th day. The extracted tissue was cut into small pieces and stored for 24 hours in 10% formalin. Specimens were cut into 3-5 m thick pieces with a microtome and stained with hematoxylin and eosin. Disterene Phthalate

Xylene was used to mount the samples (DPX). The results were presented as Mean Standard Error Mean (SEM). The software Graph Pad Prism 5.0 was used to analyse the data (Graph Pad, San Diego, CA, USA).

In patients with hypertension, left ventricular hypertrophy has been proposed as an independent cardiovascular risk factor. Activation of the Renin-Angiotensin-Aldosterone System (RAAS) has been implicated in controlling vascular remodelling-induced hypertension and thus cardiac and renal functioning in several studies.

Pressure overload generated cardiac remodelling and hypertension, which led to heart failure when the abdominal aorta was constricted above the renal arteries. In the current investigation, changes in cardiovascular parameters enhanced myocardial apoptosis and oxidative stress, implying that constriction of the abdominal aorta caused cardiac hypertrophy. LVEDP (Left Ventricular end-diastolic Pressure) is a method of determining the total volume and pressure present in the left ventricle. The current investigation's findings are consistent with those of prior investigations.

During the pathogenesis of pressure overload cardiotoxicity, researchers have thoroughly documented the role of excessive intracellular calcium influx, activation of intracellular proteolysis, inflammatory infiltration, and endothelial dysfunction. In conclusion, constriction of the abdominal aorta caused changes in electrocardiographic, hemodynamic, and left ventricle contractile functions, as well as enhanced cardiac oxidative stress and apoptosis, implying induction of cardiac hypertrophy.

Nonetheless, bisacurone therapy reduced the progression of pressure overload-induced cardiovascular dysfunction and heart hypertrophy.

JOURNAL REFERENCE

Zeng, X., H. Gong, L. Zhang, Y. Lan, S. Yang and F. Xu, 2022. Bisacurone ameliorated pressure overload-induced cardiac hypertrophy in experimental rats through inhibition of oxidative stress and Bax/Caspase-3 pathway. Int. J. Pharmacol., 18: 415-427.

KEYWORDS

Bisacurone, heart diseases