N- Acetyl Cysteine: A Possible Treatment for Diabetic Cardiomyopathy

Sir,

Diabetic cardiomyopathy is one of the major complications in patients diagnosed with chronic diabetes mellitus. Cardiomyopathy is the degeneration of cardiomyocytes, leading to the inability of the myocardium to contract. It is a complication caused by two interconnected pathological processes – cardiac hypertrophy and fibrosis – which lead to irreversible damage of the cardiomyocytes. It is a well-established fact that diabetes increases oxygen stress, which plays a causative role in cardiac dysfunction. N-Acetyl Cysteine (NAC), an antioxidant and glutathione precursor, has been used as a form of treatment, to protect the cardiomyocytes and endothelium from cell death. NAC prevents cardiac fibrosis and ventricular functional remodelling; however, it is unable to reverse the pathological process. NAC also blocks hyperglycemia, inhibits cardiac fibroblast proliferation and myofibroblast differentiation, via inhibition of Reactive Oxygen Species (ROS). ROS has been seen to cause apoptosis in both endothelial cells and cardiomyocytes, leading to fibrosis.

Just recently, a study was carried out in which Liu and his associates discovered that the use of NAC had favourable results, indicating it as a possible treatment for diabetic cardiomyopathy. In this clinical trial, 35 mice were divided into 7 groups, with 5 in each. Mice with a blood glucose level above 13.5 mmol/L were considered diabetic. Twenty-five mice were injected intraperitoneally with streptozotocin (STZ) to induce diabetes, dissolved in a mixture of citrate buffer after overnight fast. One of the groups was not treated with NAC; whereas, the others received NAC at varying time intervals. Of the two groups of non-diabetic mellitus (non-DM) mice, one received NAC while the other did not. An echocardiogram was taken along with some heart tissues, to observe the heart and perform Hematoxylin and Eosin (H&E) and ROS staining, respectively. A quantitative Polymerase Chain Reaction (PCR) analysis was also performed to observe fibrotic gene expression and cell proliferation. NAC did not alter the body weight and blood glucose level. However, it did improve the cardiac systolic and diastolic function in diabetic mice, by inhibiting ROS production and cardiac fibrosis. Abdollahi et al. has shown that earlier the NAC treatment was started, the better results obtained.

The use of NAC has proved to improve in cardiac function immensely by either preventing diabetic cardiomyopathy or by improving the role of the cardiomyocytes. NAC can further be used to prevent diastolic and systolic heart failure in longstanding type-2 diabetic patients in the local population. Nevertheless, further research studies need to be conducted to establish the efficacy of this technique.

REFERENCES