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Significance of Sirt3 expression in acute heart injury induced by paraquat poisoning in rats

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Background: Paraquat (PQ), a common herbicide, has been widely used around the world since its discovery because of its good agricultural efficiency. However, paraquat is highly toxic to human and has a low lethal dose. As it can cause damage to heart, lung, kidney and other organs in human body in a short period of time, paraquat poisoning patients usually die due to multiple organ failure and rapidly progressing pulmonary interstitial fibrosis. Arrhythmia, heart failure or even sudden death may be caused by paraquat poisoning myocardial injury, and there is still no specific antidote. This article aims to study the expression and significance of Sirt3 in myocardial injury induced by paraquat poisoning in rats.

Methods: Male Sprague-Dawley rats (n=60) were randomly divided into natural control group (saline, 1 mL, ip) and paraquat group (PQ 30 mg/kg, ip), and serum and myocardial tissue were collected at 6 h, 12 h, 24 h and 48 h after paraquat exposure. The ATP content of myocardial tissue was detected by biochemical tests. The level of superoxide dismutase (SOD) was measured by colorimetry to estimate oxidative stress. The mitochondrial membrane potential change was checked by JC - 1 probe detection, inflecting the degree of cell apoptosis. HE staining was used to observe the myocardial pathological changes after the poisoning, and Masson staining to observe the degree of myocardial fibrosis. Detection of Sirt3 expression in myocardial tissues was conducted by immunohistochemistry.

Results: Compared with the natural control group, ATP content and mitochondrial membrane potential in the myocardium of rats exposed to paraquat decreased in a time-dependent manner, and SOD activity in serum decreased significantly. HE staining showed clear structure and regular arrangement of cardiac cells and obvious nuclear staining in the natural control group. After 6h of exposure, myocardial cells showed swelling and irregular arrangement, some nuclei were stained pale, and myocardial interstitial edema appeared.12h after poisoning, the myocardial fibers with morphological changes increased, and the intercalary discs were difficult to recognize.24h after poisoning, myocardial interstitial edema and irregular arrangement of cardiac muscle fibers were more obvious, and vacuolar degeneration occurred in myocardial fibers. Partial cell showed disintegration, nuclear dissolution and disappear, and membrane dissolution occurred. Masson staining showed no obvious fibrosis changes in cardiac cells of the natural control group. After paraquat exposure, however, the myocardial cells of rats showed different degrees of fibrosis and the degree of fibrosis increased with the extension of exposure time. By immunohistochemistry, Sirt3 was expressed in a small amount in normal cardiac tissue, and its protein expression increased significantly after paraquat poisoning.

Conclusion: Sirt3 is involved in the process of myocardial injury caused by paraquat and may has an intrinsic protective effect on myocardium. Sirt3 is expected to be a new target for paraquat poisoning treatment and has good clinical application prospects.