Radiation encephalopathy is the main complication of cranial radiotherapy. It can cause necrosis of brain tissue and cognitive dysfunction, for which no ideal prevention method has been available until now. As a natural antioxidant, shikonin possesses protective properties against cerebral ischemic injury. Here we investigated the effects of shikonin on radiation brain injury induced by carbon ion beam in mice.

The entire head of each male Kunming mouse was irradiated by a 4.0 Gy (350 MeV/u) $^{12}$C$^{6+}$ ion beam after receiving daily intraperitoneal injections of shikonin for 3 d. 24 h after irradiation, the activities of superoxide dismutase (SOD) and catalase (CAT), the levels of malondialdehyde (MDA), protein carbonyl content (PCO), reduced glutathione (GSH) and oxidized glutathione (GSSG) in brain tissues were measured by the colorimetry methods. The results showed that the carbon ion beam irradiation increased the MDA and protein carbonyl content, whereas the SOD, CAT activities and GSH/GSSG ratio in brain tissue decreased. Pretreatment with shikonin noticeably improved the spatial memory deficits. Shikonin treatment increased the SOD, CAT activities and the ratio of GSH/GSSG; it also reduced the MDA, protein carbonyl contents. All of the results indicate that shikonin mitigated the brain injury through modulation of the oxidative injury induced by the $^{12}$C$^{6+}$ ion beam.

A number of complications induced by radiation have been reported in mammals, including humans. Ionizing radiation can cause cataract formation, retinal degeneration or atrophy, blindness, and abnormal development of lens. The toxic effects of X-ray radiation on eye development was measured using zebrafish as a model organism. Zebrafish embryos at 8 h post-fertilization (hpf) were irradiated by X-rays at doses of 1, 2, 4 and 8 Gy. At 24 and 48 hpf, X-ray radiation induced a significant increase in reactive oxygen species (ROS) content and cell apoptotic signals. Both increases were dose-dependent and there were significant positive relationships between them at 24 hpf. At 48 and 72 hpf, the increase of ROS concentration can be eliminated by increasing activities of superoxide dismutase (SOD) and catalase (CAT). Although the ROS generated by X-ray radiation caused a significant increase in cell apoptosis at 24 and 48 hpf, the cellular layers of the retina and lens formation in the irradiated groups were not significantly disrupted at 144 hpf compared to the control group, with the exception of a heterogeneous distribution of the cells in INL (inner nuclear cell layer) and a significant decrease in the diameters of whole eyes after 8 Gy irradiation. X-ray radiation at later stages of gastrulation may not cause distinct optic complications, however, there is still a risk of microophthalmia at high doses of irradiation.

Reference