Role of Apoptosis in CNS Emphasizing Spinal Cord Injuries: A Commentary

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Abstract

Apoptosis is a kind of programmed cell death that is naturally necessary for homeostasis and some processes like organogenesis and implantation of embryo; so it is not pathologic and harmful all the time. The purpose of this commentary was to describe the ways of stimulating and inhibiting the apoptosis process; because rather in the therapeutic protocols we should stimulate or inhibit the process in order to minimize the effects of the traumas and injuries. For using the first results in therapeutic protocols; for example, in order to take action against tumors, we should stimulate apoptosis with chemotherapeutic drugs or other alternatives. On the contrary, in physical traumas of spinal cord, we should inhibit apoptosis with chemical drugs or other alternatives in order to reduce secondary paralysis and other side effects. By collecting the present results and comparing them with our previous original articles, we reached some ideas for the future original research and therapeutic proposals.

Keywords: Apoptosis; Spinal Cord; CNS; Pathology; Neuropathology

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Commentary

Apoptosis is a kind of programmed cell death that is naturally necessary for homeostasis and some processes like organogenesis and implantation of embryo; so it is not pathologic and harmful all the time. Although apoptosis – as presented above – is programmed cell death, the reverse is not true, i.e. all programmed cell deaths are not necessarily apoptosis, for instance, the process senescence as irreversible one to impede cell cycle in damaged cells containing lethal DNA (1).

Apoptosis is one of the main mechanisms associated with spinal cord injury. To reduce the nerve-cell injury secondary to trauma occurrence, inhibition of apoptosis seems as the first protocol (2). Thus the purpose of present commentary is description of the ways of stimulating or inhibiting apoptosis, because rather in the therapeutic protocols we should stimulate or inhibit the process in order to minimize the effects of the traumas.

The most common methods for studying apoptosis are cytotoxicity, morphological changing, laddering model of DNA, TUNEL method and flow cytometry. Some methods can't distinguish apoptosis from necrosis, because they just show the percentage of cell death in comparison to control group (3). It seems that the most specific method is assaying expression of Bax and BCl-2 with the help of reverse transcriptase polymerase chain reaction (RT-PCR) and immunohistochemistry (4).

Trauma and injury in central nerve system can induce apoptosis in neighbour intact nerve-cells resulting in different levels of nervous paralysis. To prevent these damages, the best way accepted right now is to inhibit the apoptosis.

Ischemia-reperfusion and neutrophils with secreting factors like reactive oxygen species (ROS) can induce apoptosis through the extrinsic pathway (5,6). The first results were the usage of apoptosis in the therapeutic protocols, for example, in order to fight against tumors, and we should stimulate apoptosis with chemotherapeutic drugs or other alternatives such as natural honey (7). On the contrary, in the physical traumas of spinal cord, we should inhibit apoptosis with chemical drugs or other alternatives such as green tea (2) in order to reduce secondary paralysis and other side effects.

Neuroprotective effects of herbal medicines are proposed as a hot topic nowadays, because of their anti-oxidant (6), anti-inflammatory (8) and anti-apoptotic effects. Green tea has neuroprotective effects such as anti-inflammatory ones as well as decreasing apoptosis (2). Rhodiola plant (through increasing ATP) can protect the nerve system from necrosis and apoptosis (9). Oleuropein – an anti-oxidant in olive oil– plays anti-apoptotic role in CNS through induction of BCl-2 and inhibition of Bax (10-12).

Collecting the present results and comparing them with our previous original articles, we reached some ideas for the future original research and therapeutic proposals.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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