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Special Issue

Exosomes and Delayed Encephalopathy After Acute Carbon Monoxide Poisoning (DEACMP)

Acute CO poisoning is the most common type of acute poisoning worldwide, and about 40 % of surviving patients develop delayed encephalopathy after acute carbon monoxide poisoning (DEACMP). The symptoms of DEACMP include impaired judgment, poor concentration and memory, cognitive dissonance, personality changes, psychosis, and even Parkinsonism symptoms. To date, the mechanisms of DEACMP remain unclear.

Exosomes are a heterogeneous group of nano-sized natural membrane vesicle released from various cells and exist in body fluids. A growing amount of evidence shows that exosomes involve in pathogenesis, diagnosis and treatment modalities of some brain disease, such as ischemic stroke, Alzheimer's disease, Parkinson disease, multiple sclerosis, brain cancers, and so on. In the light of exosomes can overcome the blood-brain barrier (BBB), exosomes as therapeutic drug carrier for brain drug delivery, have received extensive attention.

Previous studies suggested that DEACMP may be the outcome of acute CO poisoning induced cerebral cellular hypoxia, postischemic reperfusion injury, free radical damage, immune damage, and apoptosis. The secretion of exosomes by cerebral cells is mediated by various physical, chemical, and biological stimuli such as hypoxia, low pH, extracellular ATP, increased intracellular Ca2+, and DNA damage. Therefore, the potential roles of exosomes in pathogenesis and prognosis of DEACMP is worth deep exploration.

In this issue we welcome original research articles and reviews on this topic to provide recent findings and explore diverse functions of exosomes in DEACMP.

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