Abstract

Iron cations are an important metal ions required to number of essential cell functions. On the other hand, ferrous iron can be very toxic as well. When surplus iron is present in cells, it can catalyze the formation of reactive oxygen species (especially hydroxyl radicals) by Fenton reaction. Iron homeostasis is predominantly regulated by very strict mechanisms on the level of iron uptake into the body. Moreover, iron absorption, transport and storage within the body can be also regulated using complex mechanisms which differ on the level of individual cells and on the level of whole organism. Deregulation of iron homeostasis causing an iron overload and generation of reactive oxygen radicals can evoke serious cell damage leading up to apoptotic cell death. Excess iron storage and subsequent development of oxidative stress can affect lot of different tissues in the body. The organ damages such as fibrosis, cirrhosis, hepatocellular carcinoma, heart failure, loss of β cells and glucose intolerance or diabetes mellitus in patients with iron overload are very often seen. Nevertheless, the apoptosis induced by iron overload has not been well elucidated yet. There are no complex informations about the precise mechanism by which oxidative stress affects different cell types or whether there are other unknown factors involved in apoptosis induction. Thus, the better understanding of apoptosis causation due to the body iron overload may be useful in the treatment of diseases associated with iron organ damage.