

The cell death phenomenon, an abstract

The cell death phenomenon, apart from being an important feature in the development of the nervous system. Many neurological diseases are characterized by the gradual loss of specific sets of neurons and result in disorders of movements and CNS functions, such as diseases are hypoxic ischemic encephalopathy. The mechanism of apoptotic neurodegeneration in thalamus and other brain regions after neonatal hypoxic ischemia are completely unknown. However, death receptor-activated pathways, altered mitochondrial function and changes in expression of mitochondrial-related bcl-2 family proteins are likely important effectors of programmed cell death. Fas/Apo-1 (CD95) is an important one of mitochondrial-related bcl-2 family proteins. Fas death receptor (CD95) expression increased in hippocampus bilaterally during 24 hours immediately after neonatal hypoxic ischemic encephalopathy. This study was designed to evaluate the apoptotic process in CNS tissue which occurred in neonatal hypoxic ischemic encephalopathy by measuring the levels of Fas in serum and CSF in neonatal hypoxic ischemic encephalopathy. Material and Methods: The study was done on 80 neonates, 60 neonates with hypoxic ischemic encephalopathy were admitted in premature and IC units in AL-minya university hospital and 20 healthy neonates as control group. All neonates were subjected to careful history of HIE. Clinically delaminated with assessment of severity of HIE by using Apgar and Thompson score. Laboratory investigations were done on all neonates included: complete blood picture, C-reactive protein, urine analyses and culture, CSF by lumbar puncture. Fas/Apo-1 (CD95) was measured in serum and CSF by a solid-phase sandwich, two-site, enzyme-linked immunoassay (ELISA). Results: Serum soluble Fas levels in HIE neonates were significantly elevated (15.81 ± 7.13 ng/dl) in comparison with control group (4.09 ± 1.15 ng/dl) ($p < 0.0001$). Significantly increased in soluble Fas levels in CSF of HIE neonates (3.93 ± 3.01 ng/dl) when compared with healthy control neonates (0.33 ± 0.27 ng/dl), ($p < 0.0001$). Significant positive role of prematurity on Fas in serum and CSF, where a statistically significantly increased in serum and CSF Fas levels in HIE premature neonates (210.9 ± 5.59 ng/dl) (6.62 ± 2.73 ng/dl) respectively, compared to those of HIE full-term neonates (12.76 ± 5.76 ng/dl) (2.58 ± 2.11 ng/dl) ($p < 0.0001$). There was a significant correlation between the levels of Fas in serum and CSF with the severity of HIE according to Thompson score. Conclusion: we concluded that an increase in Fas in serum and CSF of HIE in neonates explains the apoptotic process in brain tissue damage in HIE. Also by measuring Fas levels in serum and CSF of HIE neonates we can use it as a marker of apoptosis and can detect the severity of HIE.

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