Investigation of Circulating Apoptotic Factors in Acute Ischemic Stroke Patients

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ABSTRACT
Ischemic stroke is a neurological disorder caused by the cessation of normal blood flow to the brain. The blockage of blood flow to the brain as the outcome of ischemic stroke would initially cause the shortage of nutrients supply to the neuron and glial cells, then eventually the damage and death of the cells. Investigation in animal models has demonstrated that apoptosis could play a role in cerebral ischemia. Its clinical significance in stroke patient has not been well established. In this research, we assayed factors related to apoptosis in peripheral blood of stroke patient. Stroke patients who had MRI study were recruited randomly at Changhua Christian Hospital. Blood samples were taken after informed consent obtained. DNA was extracted from the plasma and quantified PCR. Apoptotic factors were analyzed by western blots with specific antibodies and activities of caspase 3/7 were also investigated. Sixteen samples were taken from 7 stroke patients and 4 control patients. Patients with gray matter infarction showed higher level of soluble TNF-α. Plasma from patients with small white matter infarction also showed lower level of soluble TNF-α and lower caspase 3/7 activities. Soluble TNF-α has greater affinity for TNF-R1 receptor and TNF-R1 receptor has been suggested to be pro-apoptotic. Interestingly the activities of caspase 3/7 are correlated to apoptosis. These results provide apoptotic factors can be detected in the plasma from patients with acute ischemic stroke and might be related to large grey matter infraction.

Keywords : Ischemic stroke、peripheral blood、apoptosis、after、cerebral infarction、tumor necrosis factor (TNF)、membrane TNF-α (mTNF-α)、soluble TNF-α (sTNF-α)
2.9 Tumor Necrosis Factor (TNF)

2.9.1 TNF-α inflammatory response

2.9.2 TNF receptor

2.9.3 TNFR1 and TNFR2

2.9.4 Transmembrane protein (mTNF) and soluble protein (sTNF)

2.9.5 TNF and cell apoptosis

3. Materials and Methods

3.1 Experimental materials

3.2 Experimental methods

3.3 Extraction of blood plasma DNA from stroke patients

3.4 Semi-quantitative PCR for blood plasma DNA quantification

3.4.1 Primer design

3.4.2 Semi-quantitative Polymerase Chain Reaction

3.5 Blood plasma protein quantification

3.6 Western blotting

3.7 Caspase-3/7 assay to test protein activity

3.8 Statistical analysis

4. Results

4.1 Semi-quantitative PCR for blood plasma DNA quantification

4.2 Western blotting comparison of TNF-α protein expression

4.3 Caspase 3/7 assay to test protein activity

4.4 Analysis of cell apoptosis-related factors in all stroke patients

5. Discussion

6. Conclusion

References


Mol Cell. Scheurich P, Wajant H. (2004). NFkappaB activation by Fas is mediated through FADD, Caspase-8, and RIP and is inhibited by FLIP.


Survivin initiates proCaspase 3/p21 complex formation as a result of interaction with Cdk4 to resist Fas- mediated cell death.


Nod1, a CARD protein, enhances pro-interleukin-1β processing through the interaction with pro-caspase-1.


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