Young Investigator’s Award: Induction of Apoptosis Following Traumatic Head Injury in Humans

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Abstract

Apoptosis or programmed cell death plays an important role in many developmental and pathological processes of the central nervous system. In head injury, apoptosis has been recently implicated in many studies on animal brain samples the phenomenon of apoptotic gene expression (bax and bcl-2).

Twenty specimens of contused brain tissue (temporal and frontal lobe) from 20 patients who underwent emergency craniotomy and removal of mass lesions were obtained from May to October 1997. The samples collected were immediately snap frozen in liquid nitrogen and stored at -80°C. Immunohistochemical analysis was performed to detect the expression of bcl-2, bax and p53 using standard avidin-biotin complex second antibody conjugate methodology utilising commercially available primary and secondary antibodies.

The average age of cohort was 46.24 ± 22.17 years, the average Glasgow Coma Scale on admission was 9.19 ± 4.72, and the average duration from injury to collection of the sample was 20.62 ± 40.57 hours. There was documented hypoxia and hypotension seen in 5 of the 20 patients (25%). Significant levels of bax protein expression were noted in all samples, and p53 expression in 30% of samples. No bcl-2 expression was observed.

Our study showed that for the first time the strong expression of the pro-apoptotic gene (bax) and low levels of the anti-apoptotic gene (bcl-2), thus implicating the mechanism of apoptosis in brain injury following trauma. The use of agents to inhibit apoptosis may be beneficial in head injury patients.

Key words: Bax, Bcl-2, Brain contusion, p53, Programmed cell death


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