Association between intravascular coagulopathy and outcome after traumatic brain injury

It is now well accepted that much of the cerebral damage arising from traumatic brain injury (TBI) actually occurs secondarily at some temporally distant timepoint post insult. While many factors such as hypoxia, hypotension and others may contribute to delayed cerebral vasospasm and ischemia, more recent observations point toward the prominent role for
disseminated intravascular coagulation. Because of the strong association between intravascular microthrombosis and neuronal necrosis in human TBI, intravascular coagulopathy should be considered as a potential cause of ischemia post insult. Multiple studies have found changes in coagulation parameters in the systemic circulation within 24 h after TBI. Changes in systemic blood coagulation probably reflect the cerebral intravascular coagulation process and their study permits the monitoring of central nervous system events post TBI. The present study focuses on the relationship between early changes (within the first 3 h) of systemic hemostatic and fibrinolytic markers after human TBI and indicators of outcome such as the Glasgow Coma scale. This is a novel and non appreciated aspect of this research area. These data suggest that prothrombin time, partial thromboplastin time, fibrin degradation products and D-dimer levels may be useful prognostic indicators in head trauma patients. Such observations may have important implications in patient management.

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References