Emergency Medicine: Open Access
ISSN: 2165-7548

Alexiou, Emergency Medicine 2012, 2:4
DOI: 10.4172/2165-7548.1000e117

Editorial Open Access

Coagulopathy after Traumatic Brain Injury

George A Alexiou*

Department of Neurosurgery, University Hospital of Ioannina, Ioannina, Greecea

Traumatic brain injury (TBI) is a major public health problem, leading to a considerable number of deaths and disability mainly among young individuals [1]. Approximately 1.7 million people suffer from a traumatic brain injury annually [2].

Coagulopathy is often encountered in patients following TBI. Coagulation disorders can be characterised by a combination of coagulopathy and hypercoagulability and have been considered as a manifestation of the disseminated intravascular coagulation (DIC) disorder [3]. DIC can produce intravascular coagulation-induced cerebral ischaemia that may promote secondary injury [4]. Coagulopathy after TBI depends on the severity of brain injury. Pahatouridis et al. reported that lower GCS scores correlated with increased levels of d-Dimers, prolonged PT and more rapid increase of the PF levels [3]. In a study of patients with severe TBI, coagulopathy was found in 34% and was associated with longer ICU length of stay and an almost 10-fold increased risk of death [4]. Timely recognition of coagulopathy is important in predicting the occurrence of delayed brain injury and preventing possible bleeding disorders. Nevertheless, until now no optimal treatment strategy exists. The administration of fresh frozen plasma proved to have adverse effects, such as increased frequency of delayed hematomas and higher mortality [5]. McQuay et al. in a study of 18 patients reported that administration of recombinant activated factor VII, for the correction of coagulopathy in severe TBI, was safe and effective even among the elderly [6]. Pahatouridis et al. based on the fact that low molecular weight heparin (LMWH) is recommended for the treatment of even asymptomatic DIC, prospectively evaluated the safety of early administration of LMWH at a prophylactic dose in 61 patients with moderate TBI. The authors found that after LMWH administration, no clinical manifestation of DIC or LMWH related side-effects were found, whereas the hemorrhagic lesions that were initially detected on computed tomography did not increase in size [3]. Given the beneficial role for LMWH anticoagulation in the management of DIC, not to mention deep vein thrombosis prophylaxis, a possible positive effect on patients with TBI may also exist. Thus, further studies are urgently needed to determine the role of LMWH in TBI. One major issue that needs also to be resolved is that the definition of coagulopathy varies. This is a major drawback not only for comparing studies but also for identifying patients and monitoring therapy response.

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***Corresponding author:** George Alexiou, Department of Neurosurgery, University Hospital of Ioannina, PO BOX 103, Neohoropoulo, Ioannina, 45500 Greece, Tel: +30 210 6526507; E-mail: alexiougrg@yahoo.gr

Received March 21, 2012; **Accepted** March 21, 2012; **Published** March 23, 2012

Citation: Alexiou GA (2012) Coagulopathy after Traumatic Brain Injury. Emergency Medicine 2:e117. doi:10.4172/2165-7548.1000e117