TRAUMATIC BRAIN INJURY

NEUROLOGICAL DETERIORATION AFTER MILD TBI

Investigators from UMDNJ-New Jersey Medical School, Newark, NJ, studied the cause, course, and outcomes of 757 patients who were admitted over 54 months following mild head injury (MHI) complicated by intracranial hemorrhage (ICH). Of these, 31 (4.1%) experienced delayed neurological deterioration (DND)(Glasgow Coma Scale score decrease >2); 87% deteriorated within 24 hours of admission, 68% had progressive ICH, 32% had medical causes for DND, and 23% died. Factors associated with mortality included age >60 years, coagulopathy, and change in Marshall CT classification. In adolescents and adults the incidence of DND is low but carries significant morbidity and mortality if it results from progressive ICH. (Choudhry OJ, et al. Delayed neurological deterioration after mild head injury: Cause, temporal course, and outcomes. **Neurosurgery** 2013 Nov;73(5):753-60).

COMMENTARY. This study shows that the majority (96%) of adult and adolescent patients with mild head injury plus ICH has a good prognosis and remains stable without neurological decline. In the 4% with delayed neurological deterioration, 87% deteriorated within the first 24 hours, mainly because of a progressive ICH. Coagulopathy is an important risk factor and the diagnosis and correction during transmission to a trauma center improves prognosis [1]. Since age (>60 years) is found to be a risk factor [2], children and adolescents may be expected to carry a low risk of delayed neurological deterioration. The following study, however, emphasizes a residual cognitive disability in TBI children aged 7-18 years.

Residual Cognitive Disability in Children with TBI. On admission to inpatient rehabilitation, patients with TBI had more cognitive disability than those with other injuries, and TBI patients had significant residual cognitive disability on discharge [3].

References

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HYPOPITUITARISM, A SEQUEL TO TBI

Investigators at Cincinnati Children's Hospital, OH, studied the prevalence of hypopituitarism in children with inflicted traumatic brain injury. Of 14 patients evaluated, 86% had at least one endocrine dysfunction, and 50% had 2 or more, a significant increase compared to the general population, estimated to have 2.5% with endocrine abnormality. Elevated prolactin occurred in 64%, abnormal thyroid in 33%, short stature (29%), and low nocturnal growth hormone peak (17%). A child with a history of inflicted TBI should be followed closely for growth velocity and pubertal changes. If growth velocity is slow, prolactin level and full endocrine evaluation are indicated. (Auble BA, Bollepalli S, Makoroff K, et al. Hypopituitarism in pediatric survivors of inflicted traumatic brain injury. **J Neurotrauma** 2013 Nov 23).

COMMENTARY. Hypopituitarism after traumatic brain injury occurs frequently in adults, whereas in children the reported prevalence is variable. In a large study of 89 adults, aged 18-65 years (mean age 36 years), hormonal function evaluated at the time of injury and at 3, 6, and 12 months postinjury showed primary hormonal dysfunction in 19 patients (21%). Major deficits included growth hormone dysfunction, hypogonadism, and diabetes insipidus. MR imaging demonstrated increased frequency of empty sella syndrome in patients with hormonal dysfunction [1].

In children, endocrine dysfunction after TBI is common, but most cases resolve by 1 year. In one study of 31 children, average age 11.6 years, the incidence of endocrine dysfunction was 15% at 1 month, 75% at 6 months, and 29% at 12 months. At 12 months postinjury, 14% had precocious puberty, 9% had hypothyroidism, and 5% had growth hormone deficiency. Endocrine dysfunction does not correlate with severity of injury [2]. In a retrospective study of 33 children with accidental head injury (27 boys), only minor pituitary hormone abnormalities were observed, unrelated to the severity of TBI, and no clinically significant endocrinopathy was identified [3].

Age of occurrence of the TBI appears to be a significant risk factor for postinjury endocrinopathy. In children and adults, endocrine surveillance at 6 and 12 months following moderate or severe TBI is recommended, but in contrast to adults, systematic screening for hormonal dysfunction in children is generally unnecessary [2][3]. A child with a history of inflicted TBI is an exception, and if on follow-up growth velocity is slowed, prolactin level and a full endocrine evaluation should be performed [4].

References

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DEMYELINATING DISORDERS

PROGNOSIS OF ACUTE TRANSVERSE MYELITIS

Investigators at Children's Hospital of Chongqing Medical University, China, reviewed children diagnosed with acute transverse myelitis (ATM) between 1995 and 2008 and selected 39 patients diagnosed according to the new Johns Hopkins Consortium criteria [1]. At a mean follow-up period of 102.7 months, 31 had a good outcome and 8 did poorly. Risks of poor prognosis included secondary infection, increased CSF protein, short time to maximal deficit, long time to peak neurological impairment, and initial duration of treatment. Children with these risk factors were more likely to have residual neurological deficits, resulting in lower qualities of life. Conversion to multiple sclerosis occurred in 2 patients (5.1%). Additional poor prognostic factors included flaccid paraparesis, respiratory failure, age < 6 months, and spinal shock. Good prognostic factors were a plateau shorter than 8 days, supraspinal symptoms, independent walking at <1 month, hyperreflexia and Babinski reflex. (Chen L, et al. Prognostic indicators of acute transverse myelitis in 39 children. **Pediatr Neurol** 2013 Dec;49(6):397-400).