

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

1. Krahulik D, et al. *J Neurosurg*. 2010 Sep;113(3):581-4.
2. Kaulfers AM, et al. *J Pediatr*. 2010 Dec;157(6):894-9.
3. Khadr SN, et al. *Clin Endocrinol (Oxf)*. 2010 Nov;73(5):637-43.
4. Auble BA, et al. *J Neurotrauma*. 2013 Nov 23.

## 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).

COMMENTARY. In a follow-up study of 47 children with ATM at Johns Hopkins, Baltimore, a febrile illness had occurred in 47% and vaccination in 28%. At the nadir of the illness, 89% were unable to walk, required assisted ventilation, or both. At a median of 3.2 years after the acute illness, 43% were unable to walk 30 ft, 68% had urinary urgency, 55% had dysesthesias, and 75% had numbness. Age at onset <3 years was associated with a worse functional outcome. [2]. A longer follow-up period and effect of rehabilitation may explain the better prognosis in the Chinese study group.

### References

1. Transverse Myelitis Consortium Working Group. *Neurology*. 2002 Aug 27;59(4):499-505.
2. Pidcock FS, et al. *Neurology*. 2007 May 1;68(18):1474-80.

## ATTENTION DEFICIT DISORDERS

### DOPAMINERGIC MECHANISMS IN ADHD

Investigators at Addenbrooke's Hospital, University of Cambridge, UK, and centers in Germany and France examined the neural mechanisms underlying attention deficits associated with ADHD and their reversal with a single dose of methylphenidate (MPH). Sixteen adults with ADHD and 16 controls were scanned by PET and MR imaging while performing a computerized sustained attention task after oral MPH (0.5 mg/kg) and placebo, in a double-blind, cross-over design. Patients with ADHD showed significant attention deficits and reduced grey matter volume in fronto-striato-cerebellar and limbic networks. Compared to controls, ADHD patients had equivalent D2/D3 receptor availability and equivalent increases in endogenous dopamine after MPH treatment. Poor attention performers from both the ADHD and control groups had reduced left caudate dopamine activity. MPH significantly increased dopamine levels in all nigrostriatal regions, normalizing dopamine levels in the left caudate in low performers. Behaviorally, MPH improved sustained attention with increased dopamine release in the midbrain. Midbrain dopamine autoreceptor regulation is reduced in low performers, and MPH-induced increases in midbrain dopamine levels are smaller in low compared to high performers. The findings confer midbrain dopamine autoreceptors an important role in the therapeutic effects of MPH in ADHD. (del Campo N, et al. A positron emission tomography study of nigrostriatal dopaminergic mechanisms underlying attention: implications for ADHD and its treatment. **Brain** 2013 Nov;136(Pt 11):3252-70).

COMMENTARY. In a study at the Karolinska Institute of 12 adolescents with ADHD and 10 young adults as controls, attention and motor behavior were investigated with a continuous performance task and motion measurements. In the midbrain, the binding potential values for density of dopamine transporter (DAT) determined by PET were significantly lower in children with ADHD. Dopamine D2 receptor binding (D2R) in the right caudate nucleus correlated significantly with increased motor activity [1][2].

### References

1. Jucaite A, et al. *Biol Psychiatry*. 2005 Feb 1;57(3):229-38.
2. Wong DF, et al. *Int Rev Psychiatry*. 2007 Oct;19(5):541-58.