Nontraumatic Coma in Children and Adolescents: Diagnosis and Management

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KEYWORDS

• Nontraumatic • Coma • Children • Adolescents • Causes
• Management • Increased intracranial pressure

Coma, defined as an impairment of arousal, is an important clinical emergency in pediatric practice. Despite the advances in diagnostic techniques since one of us wrote on this subject in 1977,[1] clinical acumen is still central to diagnosis, management, and prognostication, and the approach of Plum and Posner[2] remains relevant.

The focus of our review is nontraumatic coma (NTC) in children (the term includes adolescents, ie, those <20 years of age). Nonaccidental head injury (NAHI) is addressed in so far as it enters into the differential diagnosis of NTC. Coma of less than 1 hour in duration is excluded from discussion.

DIFFERENTIAL DIAGNOSIS

The assessment of consciousness is dependent on motor responses. Hence, a child with total generalized loss of motor function, as may happen with severe myasthenia

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gravis, botulinum poisoning, or Guillain Barré syndrome, may be erroneously labeled as comatose; the pupils are normally reactive in these situations despite presumed deep coma and absent oculomotor reflexes.

Apparent coma may be presentations of conversion or factitious disorders and malingering: resistance to eyelid opening with a normal Bell phenomenon, normal responses to cold caloric testing (including nystagmus), and otherwise normal neurologic examination should make one suspect these possibilities.

The electroencephalogram (EEG) is normal for age and reactive to stimuli in these situations.

EPIDEMIOLOGY

Relative incidences vary by country, region, season, especially for infective causes, and period of data collection.[3] Ethnicity is relevant for several inborn errors of metabolism (IEM) that produce coma.[4,5] The age-specific incidence of NTC was 160 per 100,000 children per year for those less than 1 year of age, and less than 40 per 100,000 children in those aged 2 years to 16 years for the period July 1994 to December 1995, in a population-based study in England.[6] NTC has also been commoner in children less than 6 years than in older children in hospital-based studies.[1,7–9] The higher incidence in early childhood is likely because of greater susceptibility to infective causes. Anemia, malnutrition, and adverse socioeconomic factors predispose to infectious encephalopathy, children being most vulnerable.

GENETIC SUSCEPTIBILITY?

Kobayashi and colleagues'[10] data suggest that polymorphisms in the sodium channel α 1 subunit may predispose to some acute encephalopathies. Variations in the carnitine palmitoyl transferase II (CPT II) gene, apparently common in East Asians (eg, Japanese), may be a risk factor for a variety of acute encephalopathies among them.[11–13]

CAUSES

We have used the concepts of Plum and Posner[2] to classify the causes of NTC in children[3]; the distinction between (1) conditions associated with structural changes in the brain and (2) those in which metabolic or toxic disturbances predominate (often with no or little structural change), and between those with focal signs and diffuse signs may still have practical merit (**Box 1**). However, the differentiation is often indistinct in clinical practice. Clinical features may evolve from focal to diffuse, and conditions listed under the structural category may produce metabolic dysfunction, and vice versa. Clinical, laboratory, EEG, and neuroimaging data should be considered collectively when attempting to arrive at a specific diagnosis. Some causes are discussed in further detail. Because of travel and migration, physicians, especially in Western countries, should be familiar with important global causes, particularly infective and genetic.

Infective Causes

A variety of organisms (bacterial, viral, protozoal, and fungal) can cause sepsis or invade the nervous system (meningoencephalitis) and produce coma. Many occur exclusively or more frequently in some geographic regions than others, and several are seasonal.

The incidence of bacterial meningoencephalitis has been greatly reduced in many advanced economies with the introduction of conjugate vaccines, but vaccines are