



## How to encounter the child in coma

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ARTICLE INFO	ABSTRACT
<p><b>Article type</b> Review article</p> <p><b>Article history</b> Received: 20 Dec 2013 Revised: 24 Dec 2013 Accepted: 7 Jan 2014</p> <p><b>Keywords</b> Coma Glasgow Coma Scale Pediatric</p>	<p>Non-traumatic coma is a medical emergency and should be evaluated as soon as possible. Pediatric coma is more serious because of patient's capacity of pathological stressor tolerance is limited especially in neonates. Several etiologies could be listed for loss of consciousness (LOC) and coma in childhood. According to the epidemiological studies, causes of coma are different all around the world. Glasgow Coma Scale has been used for coma scaling. In this review, we focused on some highlight causes of coma in pediatric medicine.</p>

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### Introduction

Comatose child almost always refers to the pediatric emergency unit (1). Many reports showed that encounter with these situations need good level of critical care knowledge and good level of professional clinical practice (2,3).

Level of consciousness in children is a very important question with difficult answer (4). Coma refers to a state of complete unawareness and unresponsiveness (5). One of the most important scales for the

assessment of neurologic conditions is the Glasgow Coma Scale (Table 1) (6). Some etiologies and diagnosis of acute childhood comatose are listed in Table 2 and 3 (5).

Differential diagnosis for children presenting with coma or altered level of consciousness is very important for any pediatrician (7-9).

### Pathophysiology of coma

Brainstem and pons have important role in

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our consciousness; they modulate cortical signals for other downstream targets of central nervous system (CNS) (10).

Ascending reticular activating system (ARAS) is responsible for awareness. Good function of the ARAS and brain hemispheres depends on many factors, including the presence of substrates needed for energy production, adequate blood flow to deliver these substrates, maintenance of normal body temperature, absence of abnormal serum concentrations of metabolic wastes elements or exogenous toxins, and the absence of CNS infection or seizure with abnormal neuronal activity (11).

**Table1.** Glasgow Coma Scale in Pediatric Medicine

Eye Opening		score
Spontaneous		4
To speech		3
To pain		2
None		1
Best Motor Response		
Obeys verbal command		6
Localizes to painful stimulus		5
Flexion withdrawal		4
Flexion decorticate		3
Extension decerebrate		2
No response		1
Best Verbal Response		
year 5<	year 5 >	
<b>Oriented, converses</b>	Alert, babbles, coos, words or sentences normal	5
<b>Disoriented, converses</b>	Less than usual ability, irritable cry	4
<b>Inappropriate words</b>	Cries to pain	3
<b>Incomprehensible sounds</b>	Moans to pain	2
<b>No response</b>	No response to pain	1
<b>Preverbal children should receive full verbal score for crying with stimulation.</b>		

**Table2.** Etiology and diagnosis of acute childhood comatose (Central Nervous System Diseases)

<b>Trauma</b>	Intracranial hematoma (subdural, epidural, intraparenchymal) Cerebral contusion Cerebral edema Concussion
<b>Seizures</b>	Status epilepticus (convulsive, nonconvulsive) Postictal state
<b>Infection</b>	Meningitis Encephalitis Focal infections (brain abscess, subdural empyema, epidural abscess)
<b>Neoplasm</b>	Tumor (edema, hemorrhage)
<b>Vascular disease</b>	Cerebral infarct (thrombotic, hemorrhagic, embolic) Central venous thrombosis Subarachnoid hemorrhage Vascular malformation/aneurysm
<b>Hydrocephalus</b>	Obstructive (from tumor or other cause) Cerebrospinal fluid shunt malfunction

### *Non-traumatic Coma CNS infection*

Meningitis, encephalitis, or other types of CNS infection may cause coma in a child (12,13). Inflammation of the brain might be due to infections and this condition should be seriously considered because of unexpected results (14). Despite the fact that Haemophilus influenza and Streptococcus pneumoniae vaccines are available, bacterial infection is still a common cause of loss of consciousness (LOC), but other microorganisms can involve in LOC (15). Enteroviruses and Herpes viruses could cause viral encephalitis. Most of the time, fungal infections or parasitic infections (toxoplasmosis, etc.) have a slower onset of symptoms (16). Focal infections (brain

abscess, subdural empyema, epidural abscess) could induce focal seizure and may lead to LOC (17).

### **Malignancy**

Direct invasion of the ARAS by the malignancies such as hematologic malignancy with CNS involvement may cause LOC (18) which may be due to increase in intracranial pressure, seizure (19) or brain hemorrhage. Lethargy and vomiting are some of the signs and symptoms in brain malignancy involvement (20).

### **Vascular cause**

Cerebrovascular origin of coma is important for patient's management. Three types of etiology interrupt cerebral blood flow including brain hemorrhage, thrombosis and embolism (21). Arteriovenous malformation (AVM), aneurysm, and cavernous hemangioma are structural abnormalities which cause brain hemorrhage (22,23). They may lead to the spontaneous intracranial hemorrhage and LOC (23).

**Table 3.** Etiology and diagnosis of acute childhood comatose (Conditions Affecting the Brain)

<b>Vital sign abnormalities</b>	Hypotension, hypertension Hypothermia, hyperthermia
<b>Hypoxia</b>	Pulmonary disease Severe anemia Methemoglobinemia Carbon monoxide Posthypoxic encephalopathy
<b>Intoxications</b>	Sedative drugs: antihistamines, barbiturates, benzodiazepines, ethanol, gamma-hydroxybutyrate (GHB) and analogs, narcotics, phenothiazines Tricyclic antidepressants Anticonvulsants Salicylates
<b>Metabolic abnormalities</b>	(Hypoglycemia (sepsis, insulin overdose, ethanol intoxication) Hyperglycemia (diabetic ketoacidosis, hyperglycemic hyperosmolar syndrome) Metabolic acidosis Metabolic alkalosis Hyponatremia, hypernatremia Hypocalcemia, hypercalcemia Hypomagnesemia, hypermagnesemia Hypophosphatemia (Uremia (kidney failure) Liver failure (Acute toxic encephalopathy (Reye's syndrome) Inherited metabolic disorders
<b>Others</b>	Intussusception Hemolytic uremic syndrome Dehydration Sepsis (Rheumatologic conditions (SLE, Behçet's Psychiatric conditions

Ischemic stroke occurs due to thrombosis or embolism. Stroke usually may cause focal neurological deficit, not coma (24).

Brain hemorrhage and parenchymal swelling could lead to raised ICP (25) and make ARAS blood flow deficiency.

Consequently, this deficiency leads to LOC (26).

### **Toxic Ingestions**

Pediatric toxic ingestion or accidental poisoning is a very common chief complaint in pediatric emergency unit (27,28). Toxin ingestion could be unintentional or intentional (29). Body packing is one of the child abuses and may be a cause of coma that should be considered (30). Special epidemiological surveillance in any region is important and necessary (28). Some toxin may cause coma or LOC due to several pathophysiology.

### **Serum metabolites and substrate**

Abnormal serum levels of substrates or metabolites could lead to the LOC and coma. Hypoglycemia, metabolic acidosis or alkalosis, abnormal serum electrolyte levels (Na, K, Ca, P and Mg) are some of the main causes of serum substrate abnormalities which may lead to LOC (31,32). Renal and hepatic failure may result in progressive apathy, confusion and coma (33).

Urea cycle deficiency could present with ALOC and hyperammonemia in neonates or young children. Hyperammonemic coma is a result of total enzyme insufficiency (34).

Reye's syndrome is very rare but could induce liver failure and could predispose the patient to delirium that progresses to coma (35).

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### **Conflict of Interest**

The authors declare no conflict of interest.

### **References**

1. Kirkham FJ, Newton CR, Whitehouse W. Paediatric coma scales. *Dev Med Child Neurol*. 2008;50:267-274.
2. Le Roux PD, Jardine DS, Kanev PM, et al. Pediatric intracranial pressure monitoring in hypoxic and nonhypoxic brain injury. *Childs Nerv Syst*. 1991;7:34-39.
3. Kirkham FJ. Non-traumatic coma in children. *Arch Dis Child*. 2001;85:303-312.
4. Dean JM, Kaufman ND. Prognostic indicators in pediatric near-drowning: the Glasgow coma scale. *Crit Care Med*. 1981;9:536-539.
5. Fleisher GR, Ludwig S. *Textbook of pediatric emergency medicine*. 6 ed. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins Health; 2010.
6. Kliegman R. *Nelson Textbook of Pediatrics*. 19 ed. Philadelphia: Elsevier/Saunders; 2011.
7. Pollard AJ, Britto J, Nadel S, et al. Emergency management of meningococcal disease. *Arch Dis Child*. 1999;80:290-296.
8. Trubel HK, Novotny E, Lister G. Outcome of coma in children. *Curr Opin Pediatr*. 2003;15:283-287.
9. Thimann DA, Huang CJ, Goto CS, et al. Benzonatate toxicity in a teenager resulting in coma, seizures, and severe metabolic acidosis. *J Pediatr Pharmacol Ther*. 2012;17:270-273.
10. Parvizi J, Damasio AR. Neuroanatomical correlates of brainstem coma. *Brain*. 2003;126:1524-1536.
11. Brown EN, Lydic R, Schiff ND. General anesthesia, sleep, and coma. *N Engl J Med*. 2010;363:2638-2650.
12. Gualberto FA, de Oliveira MI, Alves VA, et al. Fulminant encephalitis associated with a vaccine strain of rubella virus. *J Clin Virol*. 2013;8:737-740.
13. Tellez de Meneses M, Vila MT, et al. Viral encephalitis in children. *Medicina (B Aires)*. 2013;73:83-92.
14. Johnson RT. Acute encephalitis. *Clin Infect Dis*. 1996;23:219-224.
15. Kulik DM, Uleryk EM, Maguire JL. Does this child have bacterial meningitis? A systematic review of clinical prediction rules for children with suspected bacterial meningitis. *J Emerg Med*. 2013;45:508-519.
16. Nickerson JP, Richner B, Santy K, et al. Neuroimaging of pediatric intracranial infection--part 1: techniques and bacterial

- infections. *J Neuroimaging*. 2012;22:e42-51.
17. Caplivski D, Scheld WM. Consultations in infectious disease: A case based approach to diagnosis and management. Oxford University Press; 2012.
  18. Legriel S, Marijon H, Darmon M, et al. Central neurological complications in critically ill patients with malignancies. *Intensive Care Med*. 2010;36:232-240.
  19. Shein SL, Reynolds TQ, Gedela S, et al. Therapeutic hypothermia for refractory status epilepticus in a child with malignant migrating partial seizures of infancy and mutation: A case report. *Ther Hypothermia Temp Manag*. 2012;2:144-149.
  20. Vagace JM, de la Maya MD, Caceres-Marzal C, et al. Central nervous system chemotoxicity during treatment of pediatric acute lymphoblastic leukemia/lymphoma. *Crit Rev Oncol Hematol*. 2012;84:274-286.
  21. Gregorakos L, Sakayianni K, Hroni D, et al. Prolonged coma due to cerebral fat embolism: report of two cases. *J Accid Emerg Med*. 2000;17:144-146.
  22. Takashima S, Becker LE. Neuropathology of cerebral arteriovenous malformations in children. *J Neurol Neurosurg Psychiatry*. 1980;43:380-385.
  23. de Ribaupierre S, Rilliet B, Cotting J, et al. A 10-year experience in paediatric spontaneous cerebral hemorrhage: which children with headache need more than a clinical examination? *Swiss Med Wkly*. 2008;138:59-69.
  24. Rivkin MJ, Volpe JJ. Strokes in children. *Pediatr Rev*. 1996;17:265-278.
  25. Na DG, Kim EY, Ryoo JW, et al. CT sign of brain swelling without concomitant parenchymal hypoattenuation: comparison with diffusion- and perfusion-weighted MR imaging. *Radiology*. 2005;235:992-948.
  26. Freundlich CL, Cervantes-Arslanian AM, Dorfman DH. Pediatric stroke. *Emerg Med Clin North Am*. 2012;30:805-828.
  27. Daugherty LE, Maffei F. Toxicology for the pediatric intensivist. In: Lucking SE, Maffei FA, Tamburro RF, et al. editors. *Pediatric critical care study guide*; 2012 p. 912-932.
  28. Andiran N, Sarikayalar F. Pattern of acute poisonings in childhood in Ankara: what has changed in twenty years? *Turk J Pediatr*. 2004;46:147-152.
  29. Dinis-Oliveira RJ, Magalhaes T. Children intoxications: what is abuse and what is not abuse. *Trauma Violence Abuse*. 2013;14:113-132.
  30. Traub SJ, Kohn GL, Hoffman RS, et al. Pediatric "body packing". *Arch Pediatr Adolesc Med*. 2003;157:174-177.
  31. Zuckerman GB, Uy CC. Shock, Metabolic acidosis, and coma following ibuprofen overdose in a child. *Ann Pharmacother*. 1995;29:869-871.
  32. Roberts KE. Pediatric fluid and electrolyte balance: critical care case studies. *Crit Care Nurs Clin North Am*. 2005;17:361-373.
  33. Kasapkara ÇS, Akar M, Yürük Yildirim ZN, et al. Severe renal failure and hyperammonemia in a newborn with propionic acidemia: effects of treatment on the clinical course. *Renal Failure*. 2013. [Epub ahead of print].
  34. Krivitzky L, Babikian T, Lee HS, et al. Intellectual, adaptive, and behavioral functioning in children with urea cycle disorders. *Pediatr Res*. 2009;66:96-101.
  35. Duncan CC, Ment LR, Shaywitz BA. Evaluation of level of consciousness by the Glasgow coma scale in children with Reye's syndrome. *Neurosurgery*. 1983;13:650-653.