Emergencies in children endocrinology

GLASGOW COMA SCALE (GCS)

Score	Eye Opening	Best Verbal Response	Best Motor Response
6			Obeys verbal command
5		Oriented, converses	Localizes painful stimulus
4	Spontaneous	Disoriented, converses	Flexion withdrawal
3	To speech	Inappropriate words	Flexion abnormal (decorticate)
2	To pain	Incomprehensible sounds	Extension (decerebrate)
1	None	No response	No response

MODIFIED PEDIATRIC GLASGOW COMA SCALE: CHILDREN 4 YEARS OF AGE OR YOUNGER

Score	Eye Opening	Best Verbal Response	Best Motor Response
6			Obeys verbal command
5		Appropriate words or social smile, fixes and follows	Localizes painful stimulus
4	Spontaneous	Cries, but consolable	Flexion withdrawal
3	To speech	Persistently irritable	Flexion abnormal (decorticate)
2	To pain	Restless, agitated	Extension (decerebrate)
1	None	No response	No response

1. Diabetic ketoacidosis/ketoacidemia (DKA) is a potentially life-threatening metabolic disturbance caused by an absolute or relative insulin deficiency with resultant ketone body production and concomitant decrease in the measured total carbon dioxide concentration (TCO2) in serum. Initially, compensatory hyperventilation preserves a normal blood pH (ketoacidosis); without provision of sufficient insulin, ketonemia progresses, and a subnormal blood pH (ketoacidemia) ensues. The presence of even low concentrations of insulin in the portal circulation will usually inhibit the hepatic fatty acyl carnitine cycle, preventing DKA. In the absence of sufficient insulin this cycle is uninhibited, resulting in the production of ketone bodies.

Diabetic Ketoacidosis (DKA) Treatment Protocol

TIME	THERAPY	COMMENTS
1st hour	10–20 mL/kg IV bolus 0.9% NaCl or LR	Quick volume expansion;may be repeated.NPO.Monitor I/O, neurologic status.Use flow she et.Have mannitol at
	Insulin drip at 0.05 to 0.10 μ /kg/hr	bedside;1 g/kg IV push for cerebral edema.
2nd hour until DKA resolution	0.45% NaCl:plus continue insulin drip	
	20 mEq/L KPhos and 20 mEq/L KAc	
	5% glucose if blood sugar <250 mg/dL (14 mmol/L)	
		If K < 3 mEq/L, give 0.5 to 1.0 mEq/kg as oral K solution *OR* increase IV K to 80 mEq/L
Variable	Oral intake with subcutaneous insulin	No emesis;CO_2 ≥ 16 mEq/L;normal electrolytes

Note that the initial IV bolus is considered part of the total fluid allowed in the first 24 hr and is subtracted before calculati ng the IV rate.

MANAGEMENT OF NONKETOTIC HYPEROSMOLAR COMA

Management of NKHC is similar to that of diabetic ketoacidosis with some important additional considerations. The onset of NKHC often is insidious. Patients may have even more profound fluid deficits and hyperosmolality at initial presentation than do those with diabetic ketoacidosis. The hyperosmolar state must be corrected slowly. Normal saline solution is an appropriate choice for initial fluid resuscitation and deficit replacement. In the absence of ketoacidosis, these patients often are more sensitive to insulin, so beginning the insulin drip at 0.05 U/kg/h is prudent. Fluid shifts during therapy can be substantial. As insulin enhances glucose uptake, water follows passively into the cell to maintain osmolality, and an acute decrease in vascular volume may ensue. Fluid requirements must be reassessed frequently during therapy. Repeated small boluses of saline solution may be needed to maintain vascular volume. Some patients need more than 4 L/m^2 /d. Monitoring and management during therapy are otherwise similar to those described for diabetic ketoacidosis

Thyroid Storm

Synonyms

Thyrotoxic crisis

Accelerated hyperthyroidism

Clinical Features of Thyroid Storm

Cardinal presenting features:

(1) Fever (almost invariably present; often severe and in excess of temperature elevation expected from the intercurrent illness)

(2) Tachycardia (out of proportion to the degree of fever)

(3) Acute metabolic encephalopathy (extreme restlessness, agitation, psychosis, delirium, confusion, stupor, or coma)

Other presenting features:

(1) Arrhythmias, palpitations
(2) Profuse sweating (warm moist skin)
(3) Heat intolerance
(4) High-output congestive heart failure
(5) Cardiogenic shock
(6) Goiter
(7) Proptosis, lid retraction
(8) Diarrhea
(9) Abdominal pain
(10) Jaundice
(11) Tremulousness
(12) Nausea and vomiting
(13) Stroke

GOAL	TREATMENT
Inhibition of thyroid hormone formation and secretion	Propylthiouracil (PTU), 400 mg every 8 hr PO or by nasogastric tube Sodium iodide, 1 g IV in 24 hr, or saturated solution of KI, 5 drops every 8 hr
Sympathetic blockade	Propranolol, 20–40 mg every 4–6 hr, or 1 mg IV slowly (repeat doses until heart rate slows); not indicated in patients with asthma or heart failure that is not rate related
Glucocorticoid therapy	Hydrocortisone, 50–100 mg IV every 6 hr
Supportive therapy	Intravenous fluids (depending on indication: glucose, electrolytes, multivitamins) Temperature control (cooling blankets, acetaminophen; avoid salicylates) O2 if required Digitalis for heart failure and to slow ventricular response; pentobarbital for sedation Treatment of precipitating event (e.g., infection)

TREATMENT

Once the diagnosis of pheochromocytoma is established, surgical extirpation is the best treatment. Patients are at high risk of perioperative complications, such as hypertensive crisis, myocardial dysfunction, and shock. The preoperative and intraoperative treatment of patients undergoing surgical removal of pheochromocytoma necessitates careful attention. Preoperative Î±-adrenergic blockade with phenoxybenzamine is the most commonly used approach. Patients initially receive 5 to 10 mg twice a day (0.25â€“2 mg/kg/d), which is gradually increased until the symptoms of hypertension resolve. This generally takes 1 to 2 weeks. A Î²-blocker such as propranolol or atenolol can be added to the regimen in the event that tachycardia or arrhythmia develops during phenoxybenzamine therapy, but only after Î±-adrenergic blockade has been accomplished. This is because the hypertension can worsen if the vasodilating effects of the Î²-blocker are allowed at a time when the Î±-adrenergic receptors remain stimulated. Even with adequate preoperative preparation, careful monitoring is needed during the procedure and in the postoperative period. Pulmonary edema and impaired myocardial function can occur in the postoperative period.