Index of Suspicion
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Case 1  Presentation
A 15-year-old boy is admitted to the hospital because of a prolonged generalized seizure after having become progressively lethargic over the past 24 hours. He has a 1-week history of malaise, anorexia, and headaches. His Glasgow Coma Scale (GCS) score is 8/15. Despite termination of abnormal movements following administration of intravenous anticonvulsants, his level of consciousness remains depressed, and endotracheal intubation is performed. He is admitted to the intensive care unit. CT scan of his head shows no focal abnormalities.

He has a mild global developmental delay of unknown cause. He had three febrile convulsions at age 3 years and one generalized seizure when 9 years old. He takes no medication, is fully immunized, and has no family history of similar illness.

The boy fails to awaken after sedation medications are stopped. On physical examination, he remains comatose and has a GCS score of 4/15. His temperature is 100.76°F (38.2°C), pulse is 98 beats/min, and blood pressure is 127/68 mm Hg. Abnormal muscle tone and intermittent extensor posturing are noted. Pupils are equal in size and reactive. No neck stiffness or rash is noted. All other physical findings are normal.

His hemoglobin concentration is 12.3 g/dL (123.0 g/L), WBC count is 8.0×10³/mcL (8.0×10⁹/L), and platelet count is 257.0×10³/mcL (257.0×10⁹/L). Serum electrolytes, C-reactive protein, BUN concentration, creatinine concentration, and coagulation studies are within normal limits. All cultures remain negative. An EEG shows diffuse, severe, nonspecific encephalopathy, but no epileptiform discharges. An additional test result reveals the underlying cause of his illness.

Case 2  Presentation
A 14-year-old boy who has a past history of asthma, allergic rhinitis, sinusitis, and two previous hospitalizations for pneumonia presents to the ED with fewer than 24 hours of severe, worsening respiratory distress and temperature to 103.0°F (39.5°C). His asthma symptoms have been worsening, and he is scheduled to see a pulmonologist soon because bronchodilators and inhaled steroids have not improved his disease. Two maternal cousins also have asthma and recurrent pneumonias.

Physical examination reveals a small, thin boy (weight and height below the 5th percentile for age) who breathes with severe substernal and suprasternal retractions and tachypnea. Breath sounds are equal on both sides, with crackles audible bilaterally. No wheezing is present. Heart sounds are more prominent on the right, but normal first and second heart sounds are audible, and no murmurs are present. The boy has significant digital clubbing. The patient is placed on bilevel positive airway pressure, with resultant improvement in his distress. A chest radiograph elucidates the underlying diagnosis.

Case 3  Presentation
A 9-year-old Amish boy presents with a 3-day history of a sore and stiff neck, jaw pain, drooling, difficulty swallowing, and stiffness of his right leg. Three days ago, he began to complain of jaw stiffness and a sore neck. Yesterday, his neck was canted to the right and his right leg felt sore. Today, he is unable to walk and is brought to the ED. He has received no immunizations.

On physical examination, the boy has severe right torticollis and trismus and is drooling. There is no evidence of respiratory compromise.
He has painful muscle spasms triggered by movement as well as generalized increased muscle tone and brisk-to-hyperactive deep tendon reflexes. His abdomen is tense and firm to palpation. His cognitive abilities are intact. He has a 6.0 × 1.0-cm gaping wound in the left parietal region of his scalp that extends to the galea aponeurotica. The wound bed is moist and red, and there is serious drainage.

Laboratory results reveal a total WBC count of 11.0 × 10^9/mcL (11.0 × 10^9/L) with 82% neutrophils and 7% lymphocytes, Hgb of 14.3 g/dL (143.0 g/L), and platelet count of 327.0 × 10^9/mcL (327.0 × 10^9/L). His creatine kinase concentration is 228 units/L. His electrolyte and liver enzyme values are within normal limits. A clinical diagnosis is made that is confirmed later by a laboratory result.

**Case 1 Discussion**

A metabolic screen was performed and revealed severe hyperammonemia (serum ammonia concentration of 456 mcmol/L; normal is <40 mcmmol/L). A urea cycle disorder (UCD) was suspected, and additional laboratory investigations were ordered to confirm the diagnosis and identify the specific enzyme deficiency. Concentrations of citrulline and argininosuccinic acid were elevated, which suggested the condition argininosuccinate lyase deficiency, a diagnosis confirmed on erythrocyte enzyme analysis.

Continuous high-flow venousous hemofiltration was performed, and within 4 hours, the boy’s serum ammonia concentration fell to the normal range. Concurrent therapy consisted of suspending protein intake, providing calories by intravenous administration of lipids and glucose, administering the nitrogen scavengers sodium benzoate and sodium phenylbutyrate, and providing supplemental arginine. Neurologically, the patient improved to a preadmission mental state within 7 days.

Two months after admission, his ammonia values remain within the normal range on a low-protein diet (approximately 1 g/kg per day) with sodium phenylbutyrate and arginine supplementation. He is back in mainstream school with special educational support.

**Differential Diagnosis**

Acute encephalopathy has a broad differential diagnosis that includes primary structural brain disease (eg, tumor, hemorrhage), trauma, infection (eg, meningoencephalitis), seizures, intoxication, and metabolic disorders. The differential diagnosis can be narrowed by considering age, presentation, relevant history, and physical findings. Cranial ultrasonography in newborns or CT scans in older patients should be performed to rule out structural causes of encephalopathy. The EEG can both confirm global cerebral dysfunction and exclude subclinical seizures, as demonstrated in this patient.

No lumbar puncture was performed in this child due to the possibility of raised intracranial pressure, but he was given antimicrobial therapy because of the potential for meningoencephalitis.

Intoxication is an important diagnostic consideration, and toxicologic screening is mandatory in all patients presenting with acute encephalopathy.

**The Family of Disorders**

Metabolic disorders must be considered in the patient who has encephalopathy. In addition to encephalopathy, other manifestations of inborn errors of metabolism (IEM) include chronic vomiting, developmental delay, psychomotor abnormalities, seizures, failure to thrive, and psychiatric illness. Patients tend to prefer low-protein vegetarian diets. Metabolic disorders also may present as a pattern of episodic acute decompensation triggered by changes in dietary intake, fasting, intercurrent illness, trauma, or childbirth. The specific signs and symptoms depend on the condition and its severity.

Newborns who have an IEM typically appear well after birth and may become symptomatic after feeding has started because milk provides protein and carbohydrate loading. In the neonate, typical presentations include poor feeding, vomiting, lethargy, seizures, and shock. Sepsis usually is suspected initially in a baby who shows these signs, but a metabolic disorder always should be considered if results of the septic evaluation remain negative. Other conditions that can cause a newborn to become acutely ill after a period of stability include duct-dependent heart disease, drug withdrawal, congenital viral infection, and congenital adrenal hyperplasia. Basic laboratory findings suggestive of IEM include hyperammonemia, hypoglycemia, and unexplained acid-base disorders.

Detecting IEM requires a high degree of suspicion, and these disorders can present at any time, even in adulthood. An understanding of the broad clinical manifestations of IEM provides the basis for knowing when to screen for these diseases.

**Diagnostic Steps**

A history and physical examination are essential and should be tailored to the age of presentation. Specific laboratory studies should be undertaken in all patients who have a suggestive history, physical findings, or initial laboratory results. Metabolic samples should be obtained while the patient...
is symptomatic because values can be normal when the patient is well.

Initial evaluation of suspected IEM includes the tests listed in Table 1. Specimens for specialized testing, as listed in Table 2, should be collected and stored in the acute phase and processed when indicated by the initial results and after metabolic specialist input is obtained.

The Specific Condition
The urea cycle is a metabolic pathway that transforms nitrogen derived from protein metabolism to water-soluble urea, which is excreted in the urine. UCDs are IEM characterized by episodic, life-threatening hyperammonemia resulting from partial or complete inactivity of enzymes responsible for eliminating nitrogen waste.

Deficiencies in the first four enzymes of the urea cycle (carbamyl phosphate synthetase I, ornithine transcarbamylase, argininosuccinate synthetase, or argininosuccinate lyase) result in accumulation of ammonia and the precursor metabolites (Fig. 1). Metabolic decompensation resulting in hyperammonemia causes neurologic injury because free ammonia is highly toxic to the CNS.

Plasma quantitative amino acid analysis identifies which precursor metabolites are elevated and can be used to differentiate among the UCDs. The specific UCD, however, must be confirmed by enzyme analysis of tissue samples.

Argininosuccinic aciduria is an autosomal recessive deficiency of the enzyme argininosuccinate lyase. This enzyme catalyzes the conversion of argininosuccinic acid to arginine and fumaric acid (Fig. 1). A deficiency in this enzyme leads to accumulation of argininosuccinic acid and the precursor metabolites citrulline and ammonia as well as a deficiency in arginine, as demonstrated in this case.

The clinical phenotype of UCDs is extremely variable and depends, in part, on the amount of protein intake. The classic presentation in newborns is similar to that of other IEM and includes poor feeding, vomiting, lethargy, and coma due to hyperammonemia.

Patients who have partial enzyme deficiencies may present outside the newborn period. Recurrent vomiting, developmental delay, learning difficulties, seizures, brittle hair in infancy (trichorrhexis nodosa), and protein intolerance are common manifestations, and all were present in this patient on additional inquiry. Failure to thrive and psychomotor delay may warrant suspicion of a UCD. It is of interest that this boy had poor growth as a toddler but now is growing in the normal range, although he is slim. Less severe forms of enzyme deficiency may present in older patients with subtle neurologic abnormalities or psychiatric abnormalities.

Precipitants of acute hyperammonemic encephalopathy include catabolic states due to infection, trauma, or fasting. Medications affecting protein catabolism such as glucocorticoids can induce a metabolic decompensation and should be avoided in patients known to have UCD.

On further questioning, this patient’s mother reported that her son had a “dairy and egg allergy” diagnosed at 2 years of age. Interestingly, the patient had self-selected a low-protein diet; he had refused to eat any meat from a young age and avoided any high-protein food. When he was less able to control his protein intake, as when eating out, he suffered recurrent episodes of vomiting. Furthermore, he has had brittle hair from infancy and failure to thrive, both typical features of a UCD.

General principles for managing hyperammonemic encephalopathy due to decompensation in patients who have UCDs include removing ammonia with hemodialysis or hemofiltration and nitrogen scavengers, decreasing the protein load, minimizing catabolism, and supplementing essential amino acids.

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Table 1. Initial Laboratory Tests for Suspected Inborn Errors of Metabolism

- Complete blood count with differential count
- Arterial blood gas
- Anion gap
- Glucose
- Lactate, pyruvate
- Ammonia
- Electrolytes, blood urea nitrogen, creatinine, uric acid
- Liver function tests: aminotransferase, bilirubin, prothrombin time
- Urinalysis (glucose, ketones, reducing substances)

Table 2. Additional Laboratory Tests for Suspected Inborn Errors of Metabolism

- Quantitative plasma and urine amino acids
- Qualitative urine organic acids
- Cerebrospinal fluid amino and organic acids
- Urine orotic acid
- Acylcarnitine profile
- Very long-chain fatty acids
Lessons for the Clinician
A metabolic disorder should be included in the differential diagnosis of every patient who has encephalopathy of unknown cause regardless of the patient’s age. Inherited metabolic disease can present at any age and requires a high degree of suspicion. This case illustrates the importance of obtaining a past medical history; this patient’s history showed multiple clues of metabolic disease. Early recognition and treatment might have prevented some of his neurologic handicap.

This child’s refusal to eat protein is an example of the unconscious decision-making that can occur without understanding how or why the choice was made. Patients may make such instinctive and effective adjustments to compensate for their own vulnerabilities and, in turn, minimize the manifestations of their diseases.

Case 2 Discussion
The chest radiograph demonstrated situs inversus, with extensive bilateral airspace disease and cystic changes (Fig. 2). A Gram stain of sputum showed many nucleated cells and gram-negative rods (later identified as *Haemophilus influenzae*). Viral direct antigen testing was positive for influenza A.

This clinical spectrum is consistent with the diagnosis of Kartagener syndrome. Although bronchiectasis, multiple pneumonias, and failure to thrive also occur in patients who have cystic fibrosis, the presence of situs inversus is classic for Kartagener syndrome.

Clinical Picture
Clinical manifestations vary, depending on the age when the disease presents. Descriptions have been published of neonates who have situs inversus, rhinorrhea, and radiographic findings consistent with pneumonia or retained lung fluid.
Infants present with chronic otitis, chronic cough, and frequent episodes of bronchiolitis. Older children have the more classic presentation of cough, recurrent sinus disease, and frequent pneumonias.

Decline in pulmonary function and the presence of bronchiectasis tend not to appear until late childhood or early adulthood. Digital clubbing (Fig. 3) is a late finding and indicates severe disease. A misdiagnosis of asthma is common, and many patients are diagnosed as having allergic rhinitis or sinusitis. Occasionally, infertility is the primary presenting complaint, with subsequent elucidation of a history of sinus disease or recurrent pneumonias. Males afflicted with the syndrome have immotile sperm, and females are subfertile but can conceive. Congenital heart lesions are present in approximately 5% to 10% of cases. Various diagnostic tests have been used over the years, but electron micrography of the cilia is the most definitive.

Although the presence of situs inversus is helpful for diagnosis and PCD should be sought in all patients who have situs inversus, it is important to remember that only 50% of patients who have PCD have situs inversus. The presence of situs inversus often leads clinicians to a suspicion of cardiac defects. In reality, very few patients born with true situs inversus have this association.

However, 20% of children born with situs inversus have Kartagener syndrome. Therefore, a patient who has the incidental finding of situs inversus in the absence of a murmur or other cardiac symptoms would be served better by a referral to a pulmonologist than a cardiologist.

Disease progression bears a remarkable similarity to cystic fibrosis, although there is no significant decrease in the lifespan of patients who have PCD if appropriate therapy is obtained. Patients who have PCD tend to follow infectious patterns similar to patients who have cystic fibrosis, with early colonization with *H influenzae* or *Staphylococcus*, and in later life, *Pseudomonas* colonization. Sputum cultures from two previous hospitalizations for this patient were positive for *H influenzae*.

Patients suspected of having PCD should be referred to a pulmonologist, otolaryngologist, and a genetic counselor and males to a urologist to evaluate for infertility. Treatment of PCD involves aggressive lifelong pulmonary hygiene regimens, antibiotics to control tracheobronchial infections and pneumonias, and preventive treatments of sinus and upper airway disease. Optimal nutrition is critical. Unfortunately, surgical intervention for otitis media has not been proven to improve the clinical course, and many patients develop hearing loss requiring hearing aids.

**Lessons for the Clinician**

PCD remains a disease that, although rare, is underdiagnosed. Early diagnosis is critical to prevent decline in pulmonary function, although most patients are of school age or teenagers at the time of diagnosis. PCD should be considered in any patient afflicted with bronchiectasis, in patients suspected of having cystic fibrosis but who have negative sweat test or genetic study results, and in children experiencing multiple pneumonias or chronic otitis media refractory to surgical treatment. The presence of clubbing in any patient should prompt immediate referral to a pulmonologist (or cardiologist if symptoms of heart disease are present). (Michelle Alletag, MD, Craig Huang, MD, University of Texas Southwestern Medical Center at Dallas, Dallas, Tex.)

**Case 3 Discussion**

On further questioning, the boy’s parents recounted that 11 days ago he was hit on his head by a metal part of a wagon. The wound was cleaned at home with hydrogen peroxide, and a cultural home remedy, a poultice of red pepper, was applied. A clinical diagnosis of generalized tetanus was made. The boy received 3,000 units of tetanus immunoglobulin (TIG), one dose of penicillin G, and tetanus toxoid. The head wound was debrided and, thereafter, cleaned twice daily.

His muscle spasms continued to worsen and were treated with intravenous diazepam, with doses titrated upward to 15 mg every 3 hours. He was placed on strict “minimal stimuli” precautions, and the diazepam was changed to “as needed” dosing. He later was placed on baclofen 15 mg orally every 8 hours. His spasms reduced on the baclofen treatment, and the need for frequent diazepam decreased. Intravenous metronidazole was administered at a dosage of 500 mg every 8 hours.

Wound cultures were negative for growth of anaerobic organisms, and there was light-to-moderate growth of *Staphylococcus aureus*, coagulase-negative staphylococci, and *Enterobacter cloacae*.
Three days after admission, the boy began to show improvement in his muscle stiffness. On day 6 of admission, he underwent a bedside swallowing evaluation by the speech therapist, and a modified pureed diet was started. One week after admission, his muscle stiffness was greatly improved, and physical therapy was begun. He was discharged 9 days after admission on oral baclofen, diazepam, and metronidazole.

Polymerase chain reaction analysis performed after discharge revealed *Clostridium tetani* in the red pepper poultice used to treat the scalp laceration.

The Condition

Tetanus in the United States is an infrequent but critical disease that occurs mostly among unvaccinated or inadequately vaccinated individuals. Tetanus is a toxin-mediated infection, caused primarily by tetanospsamin, an exotoxin produced by the anaerobic bacterium *C. tetani*. Tetanospsamin affects the muscle neurons and the CNS to cause skeletal muscle spasms. *C. tetani* is found in animal and human intestines, and its spores are ubiquitous in the soil. The organism multiplies in contaminated wounds. The incubation period of tetanus can range from days to months; most cases occur within 14 days of infection.

Toxin secretion by *C. tetani* is facilitated by necrotic tissue that has a favorable oxidation gradient. Thus, wounds due to trauma (eg, crush injury), surgical wounds, supplicative wounds, and wounds containing a foreign body have been known to develop tetanus infection. Wound contamination with soil or manure makes infection more likely. Wounds inflicted by objects capable of causing deep penetration together with tissue damage (especially wounds tainted with rust, dirt, or foreign debris, including material from socks and shoes) are particularly susceptible to infection by *C. tetani*. Wounds that do not involve tissue injury, such as simple needle sticks, are less prone to develop tetanus. Of note, up to 25% of those who have tetanus do not report recent evidence of a wound.

The clinical picture varies with the different forms of the disease. In localized tetanus, localized muscle spasms occur, usually in areas contiguous to the wound. In generalized tetanus, the spasms often start with trismus, which can progress to “risus sardonicus” (a characteristic grinning facial expression). The muscle spasms then progress to involve the entire body and are triggered by minimal external stimuli. Cephalic tetanus also can occur, is associated with head and neck wounds, is characterized by cranial nerve dysfunction, and may progress to generalized tetanus.

Neonatal tetanus, seen primarily in developing countries, is associated with umbilical stump contamination and manifests with poor suck and opisthotonus. Risk factors for neonatal tetanus in that setting include inadequate immunization of mothers, birth in an unhygienic environment, unclean birthing instruments, and the use of culturally encouraged cow dung poultices or of rags or animal skin dressings (contaminated with mud, manure, animal, or human feces) to cover the umbilical stump.

The differential diagnosis of tetanus includes orofacial infection, dystonic drug reaction, hypocalcemia, hysteria, strychnine poisoning, seizures, rabies, and meningitis. The diagnosis of tetanus remains primarily clinical. Laboratory studies are of little help in diagnosis, with a low yield of positive findings from wound cultures.

Management and Prognosis

Standard management of tetanus includes neutralization of unbound toxin, treatment of infection, supportive care, and active immunization. A single dose of TIG in a dosage of 3,000 to 6,000 units intramuscularly is administered to neutralize unbound tetanospsamin, although some recommend a smaller dose of 500 units. Tetanus antitoxin and intravenous immune globulin can be used if TIG is unavailable. Oral or intravenous metronidazole is the antibiotic of choice; parental penicillin G is an acceptable alternative. Antibiotic treatment for 10 to 14 days is recommended.

Wounds should be debrided, and excision of necrotic tissue may be required. Drugs such as benzodiazepines, phenothiazines, anticonvulsants, magnesium sulfate, and baclofen have been used for supportive care, specifically, muscle relaxation and sedation. In certain cases, therapeutic paralysis and mechanical ventilation are necessary.

The overall case fatality rate for tetanus ranges from 10% to 70%. Shorter incubation periods are associated with a poor prognosis. Recovery in most cases of tetanus is expected to be complete, with return to normal function in most cases. However, there are reported cases of residual physical or psychological effects. Neonatal tetanus may result in developmental disability.

Prevention

Prevention primarily is through active immunization with tetanus toxoid-containing vaccines throughout life (DTaP, Tdap, DT, or Td). Childhood tetanus immunization should consist of five doses of DTaP at 2, 4, 6, and 15 to 18 months and 4 to 6 years. For adolescents 11 or 12 years of age, a single dose of Tdap is recommended. This prep-
Tetanus toxoid efficacy ranges from 80% to 100%, and effective immunity is achieved in most children after three doses have been given. Booster doses are required to provide long-lasting immunity.

Measures to prevent neonatal tetanus include educating pregnant women on the dangers of using cultural poultices and educating midwives on aseptic obstetric practices and immunization of pregnant women. Following a tetanus-prone injury, secondary prevention is attempted with prompt treatment of deep wounds and use of tetanus toxoid or TIG, depending on the nature of the wound and the immunization history (Table 3). Tetanus infection does not produce reliable natural immunity, and an immunization series should be completed after clinical infection.

Tetanus toxoid is very safe, even in immunodeficient patients. Minor reactions may include local pain, erythema, fever, and malaise. Severe adverse events following tetanus toxoid administration are uncommon.

**Lessons for the Clinician**

When dealing with unvaccinated children, a high degree of suspicion must be maintained. Tetanus is a clinical diagnosis, and once the diagnosis is made, immediate treatment is necessary without waiting for laboratory results. Primary prevention by active immunization remains the best method of preventing this disease. (Stella U. Kalu, MD, University of Texas Medical Branch, Galveston, Tex, Bradley J. Sullivan, MD, PhD, Marshfield Clinic, Marshfield, Wisc.)

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**Table 3. Guide to Tetanus Prophylaxis in Routine Wound Management**

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<th>History of Tetanus Toxoid Doses</th>
<th>Clean, Minor Wounds</th>
<th>All other Wounds</th>
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<tr>
<td></td>
<td><strong>Td or Tdap</strong></td>
<td><strong>TIG</strong></td>
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<tr>
<td>&lt;3 or unknown</td>
<td>Yes</td>
<td>No</td>
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<tr>
<td>≥3</td>
<td>No</td>
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</table>

1. Tdap is preferred to Td for adolescents who have never received Tdap. Td is preferred if person received Tdap previously or if Tdap is unavailable.
2. Yes if ≥10 years since last tetanus-containing vaccine.
3. Yes if ≥5 years since last tetanus-containing vaccine.

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