

## Failure to Thrive in a Foreign Adoptee

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You are examining a patient new to your practice, he is a 5-year-old boy who recently was adopted from a former Soviet state. He is small for his age and not well developed. In addition, he has had fatigue, headaches, and a low-grade fever for several weeks. His past medical history is incomplete. His adoptive mother says that he tires easily and that he squats when he is very tired. She explains that his former pediatrician prescribed ibuprofen for the headaches, which are intermittent. She adds that his left face and arm seem to twitch at night. She offers a written report from a doctor in his country of origin but it is written in Russian, but you note that the accompanying EKG is remarkable for right ventricular hypertrophy. The chest radiography report notes dark, clear lung fields. On physical examination, the boy is very irritable, his temperature is 101.5°F (38.6°C), heart rate is 100 beats/min, respiratory rate is 24 breaths/min, and blood pressure is 118/78 mm Hg. You note perioral cyanosis and a III/VI systolic murmur loudest along the left sternal border. He has no surgical scars on his chest, no hepatomegaly, no peripheral edema, and no nuchal rigidity. Neurologic evaluation reveals a weak left hand grip, weak leg strength on his left side and a 1- to 2-minute staring episode with facial twitching. You begin to think back to your PICU rotation during residency.

2) Of the following, the evaluation that is MOST likely to lead to an immediate diagnosis for this child's neurologic status is

- A cerebral angiography
- B computed tomography scan of the head with contrast
- C electroencephalography
- D lumbar puncture
- E magnetic resonance imaging of the neck

Answer on page 20

1) You suspect an underlying cardiac disease, given this child's exam what is the most likely:

- A: Ventricular septum defect
- B: L-Transposition of the Great Arteries
- C: Tetralogy of Fallot
- D: Epstein's Anomaly
- E: Hypoplastic left heart syndrome.

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(Continued)**

Answers are C and B;

The boy described in the vignette has uncorrected tetralogy of Fallot (TOF) (overriding aorta, right ventricular hypertrophy, pulmonary stenosis, and ventricular septal defect) and insidiously developing neurologic signs and symptoms suggestive of brain abscess. Brain abscesses are rare before age 2 years because they develop on previous small infarctions. Congenital cyanotic heart disease (CCHD) is the most common cause of brain abscesses in children, and TOF accounts for approximately 50% of cases. The incidence of this complication is significant in developing countries where most children who have CCHD do not undergo surgical correction.

A brain abscess is a focal collection within the brain parenchyma that may arise as a complication of a variety of infections, trauma, or surgery. It begins as a localized area of cerebritis and develops into a collection of pus surrounded by a well-vascularized capsule. The final common pathway for the development of an abscess is a compromised area of the brain. The hypoxia, cyanosis, and polycythemia that may be associated with CCHD may lead to an increase in blood viscosity and a reduction in brain capillary flow. Such changes may produce microinfarction, which predisposes patients to cerebral abscess, particularly during episodes of bacteremia, dehydration, or cardiac dysfunction. If bacteremia develops, the organisms or septic emboli may escape the normal defenses of the lung capillary filter and seed the brain directly via right-to-left shunting. Brain abscesses associated with bacteremia result in multiple abscesses in the distribution of the middle cerebral artery. Abscesses occur at the grey-white junction and may damage the blood-brain barrier. Once infection is established in the brain, acute inflammatory cells are recruited, and local vascular permeability is altered.

Early cerebritis occurs during days 1 to 3 and leads to a perivascular inflammatory response surrounding the developing necrotic center and profound edema in the surrounding white matter. The necrotic center reaches its maximum size on days 4 through 9. Fibroblasts appear, capsule formation begins, neovascularity increases, and the new capillaries leak proteinaceous fluid that produces persistent white matter edema. The capsule forms fully on days 10 through 13, with a well-developed layer of fibroblasts; after day 14, the capsule continues to thicken and become encased with collagen. Capsule formation is usually more complete on the cortical side compared with the ventricular side, allowing for possible rupture into the ventricular system rather than into the cerebral cortex. Encapsulation is less extensive in abscesses arising from direct spread compared with those developing from hematogenous spread.

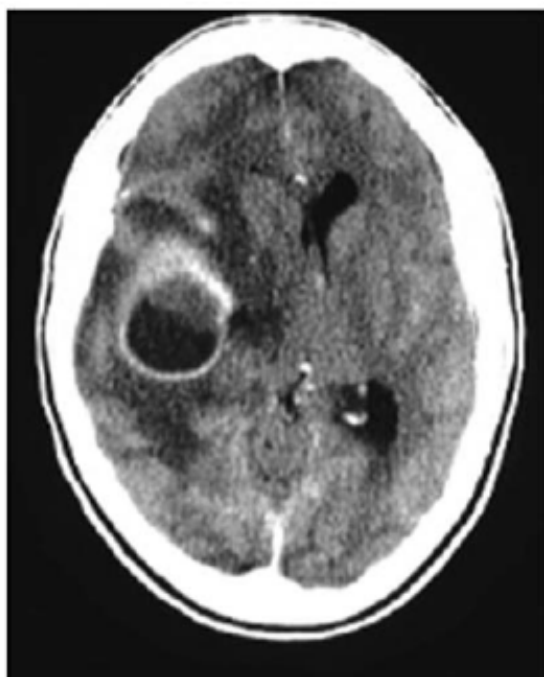
Cerebral abscesses arising from direct spread include those in the inferior temporal lobe and cerebellum (subacute and chronic otitis and mastoiditis) and those of the frontal lobes from either the frontal or ethmoid sinuses or a dental infection. Direct extension may occur through areas of associated osteitis or osteomyelitis or retrograde thrombophlebotic spread via diploic or emissary veins into the central nervous system.

The annual incidence of brain abscess is estimated to be 0.3 to 1.3/100,000 people. The risk is increased in patients who have CCHD, acute or chronic sinusitis, chronic otitis media, penetrating cranio-

cerebral trauma, craniotomy, pulmonary arteriovenous malformations, or endocarditis; those who have received solid organ transplants or bone marrow transplants; and those who have human immunodeficiency virus infection.

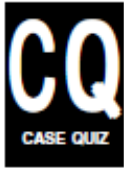
The clinical presentation of brain abscess depends on the site involved, number of lesions, and any other central nervous system-associated injury. Symptoms commonly include headache, vomiting, fever, seizures, listlessness, disorientation, and neck pain. Among the neurologic findings are lateralizing signs, hemiparesis, increased deep tendon reflexes, stupor, neck stiffness, and aphasia.

Major complications associated with cerebral abscesses include intraventricular rupture, cerebral edema, elevated intracranial pressure, seizures, and hydrocephalus. Management involves attention to the airway, breathing and circulation, and immediate neurosurgical consultation is suggested. The imaging study of choice is computed tomography (CT) of the head with intravenous contrast. The CT can be performed quickly compared with magnetic resonance imaging, which may not be immediately available, takes longer to perform, and requires sedation for the child. Cranial CT with contrast also can allow differentiation of infarction from abscess. An acute infarction usually does not appear on the initial CT scan, and an abscess has characteristic ring enhancement with surrounding cerebral edema following the administration of contrast (Figure).



**Figure:** Head computed tomography scan obtained with contrast. The large cerebral abscess on the right has large area of surrounding cerebral edema.

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Electroencephalography does not provide useful information about a brain abscess, and MRI of the neck is not helpful because the pathology is in the cortex. Cerebrospinal fluid findings may include an elevated protein concentration and a variable pleocytosis, but rarely is an organism isolated. Lumbar puncture also presents a significant risk of cerebral herniation in patients who have large abscesses and increased intracranial pressure. Angiography is an invasive approach that is not helpful in diagnosis or management of cerebral abscesses.

Prompt institution of antibiotics and consideration of neurosurgical drainage are paramount. Gram-positive organisms, particularly streptococcal and staphylococcal species (*Streptococcus milleri* and *Staphylococcus aureus*), account for most brain abscess isolates. Anaerobic *Bacteroides* sp and fusobacteria are common in those who have CCHD. Empiric antibiotic therapy should include a third-generation cephalosporin, metronidazole, and an antistaphylococcal penicillin, with the regimen modified after an organism is identified. The usual length of treatment is 6 to 8 weeks. In the early phase of cerebritis, the abscess may respond to antibiotic therapy alone without surgical drainage. Abscesses greater than 2 cm in diameter or located deep in the parietal-occipital regions should be aspirated immediately using CT scan guidance.

Adjunctive therapy with corticosteroids should be restricted to patients experiencing progressive neurologic deterioration or impending cerebral herniation and radiologic evidence that the abscess is causing significant cerebral edema and mass effect. Overall mortality for brain abscesses is approximately 10%, with sequelae including seizures and motor and sensory dysfunction.

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