Case Presentation #1: "Fever and Not Acting Right"

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A mother brings in her 12-month old boy because he has had fever, vomiting, and is not acting right. On examination, he appears listless and does not react to your exam.

Instructor Information

Begin discussion of assessment and management of a patient in shock.

The PAT is as follows

- *Appearance*: Abnormal. The boy is listless and lying supine on the gurney – although his eyes are open, he does not turn toward his mother's voice or you as you approach.
- *Work of Breathing*: Normal. The boy's respirations seem to be at a normal rate and volume. There are no retractions.
- *Circulation to the Skin*: Normal. The patient has normal skin color and a fine maculopapular rash on the trunk and legs. You see no petechiae.

Vital signs include

- Heart rate: 130 bpm
- Respiratory rate: 28 breaths/min
- Blood pressure: 80/palp
- Temperature: 38.9°C
- Weight: 11 kg

Initial assessment

- A: Open, no stridor
- B: Breath sounds clear
- C: Color normal, skin warm and dry, tachycardia, brachial pulse strong
- D: Tone normal
- E: No sign of injury, rash as described

Focused History

- S: Child began with irritability and fever for 2 days and today developed nausea and vomiting and a "drunken walk." Mother thought this might be related to fever, so she gave him acetaminophen but she became concerned when he became listless and did not respond to her normally.
- A: No allergies, breastfed
- M: None
- P: Born full-term NSVD
- L: Breakfast - 6 hours ago
- E: Normal feeding until today when vomiting began

Detailed Physical Exam

- Skin: Maculopapular diffuse rash on trunk and extending onto upper legs and arms; no petechiae.
- Head: No signs of trauma, fontanelle closed.
- Chest: Clear, without rales, rub, or wheeze.
- Heart: Without murmur, regular rhythm.
- Abdomen: Soft without hepatosplenomegaly.
- Neurologic examination: No meningismus, but poor response to environmental stimuli with a brief turn of the head; no vocalization; minimal response of withdrawal to pain.

Key Questions

What is your general impression of this patient?

Ask the audience to characterize the patient's condition as one of the following:

- Stable
- Respiratory Distress
- Respiratory Failure
- Shock
- Primary CNS/Metabolic Dysfunction
- Cardiopulmonary Failure/Arrest

Core Knowledge Points – General Impression

- Patient has abnormal appearance and neurological evaluation but normal work of breathing and perfusion.

Key Questions

What are your initial management priorities?

Critical Actions

Management priorities should be geared initially toward evaluation of the etiology of fever and altered mental status.

- Place the patient on a cardiorespiratory monitor.
- Obtain IV access and obtain rapid bedside glucose and other lab studies.
- Fever guidelines indicate that ≥39°C is a significant fever in a non-toxic child; however, in this case the patient appears toxic (he does not respond normally to caregiver or environmental stimuli) so a full sepsis work-up is indicated to locate source of infection. This will include complete blood count; urinalysis and urine culture; toxicology screen; chest radiograph; blood culture; cerebral spinal fluid analysis and culture.
- Electrolytes, liver function tests, renal function tests, calcium, ammonia level, alcohol level, a toxicology screen, and an ECG would also be performed to evaluate the altered level of consciousness (ALOC).

Case Development

- The rapid glucose is 90 mg/dL.
- The bedside hemoglobin is 10 g/dL (low).
- All laboratories are normal except serum sodium which is low at 130 mEq/L and the CSF which shows an elevated protein of 160 mg/dL and a CSF white blood cell count of 40/mm. CSF gram stain is negative. 85% of the WBC's in the CSF are lymphocytes.
- CT scan of the head is normal as is the urinalysis, chest radiograph, and ECG.

Case Development

Differential Diagnosis:

- Consider the differential for ALOC which can be remembered by the mnemonic AEIOU TIPS:
  - A – Alcohol/acidosis/ammonia (metabolic disease)
  - E – Epilepsy
  - I – Infection
  - O – Opiates
  - U – Uremia
  - T – Trauma
  - I – Insulin/Hypoglycemia
  - P – Poisoning/Psychogenic
  - S – Shock/Sepsis
- Given the fever, infection or sepsis is likely.

Key Question

What do these results indicate?

- These results indicate possible central nervous system (CNS) infection — encephalitis.

Core Knowledge Points — Encephalitis

- Encephalitis is an inflammation of the brain.
- Encephalopathy is suggested when the patient demonstrates the clinical manifestations of encephalitis, but inflammation of the brain has not occurred (eg, Reye's syndrome, hepatic encephalopathy).
- The majority of cases occur in the late summer or early fall and may be epidemic in pattern.
- This pattern reflects the etiologic agents most often responsible for encephalitis, namely, arboviruses, and enteroviruses. (Causes of encephalitis are listed in the table below).

Core Knowledge Points — VIRAL AGENTS

I	*Spread by man to man*
	Mumps
	Measles
	Enterovirus
	Rubella
	Herpes virus (herpes simplex 1 and 2)
	Varicella zoster
	Cytomegalovirus
	Epstein-Barr
II	*Arthropod-borne agents*
	Eastern equine
	California
	Japanese B
	Western equine
	Powassan
	West Nile
	St. Louis
	Venezuelan equine
	Dengue
III	*Spread by warm-blooded animals*
	Rabies
	Herpes virus simiae
	Lymphocytic choriomeningitis (rodent excreta)

Critical Actions — Encephalitis

- Management is supportive.
- CSF should be sent for special testing (call Pediatric ID specialist if possible).
- ABCs; ECG and pulse oximetry monitoring.
- Increased ICP may need to be aggressively treated with ET intubation, hyperventilation, and mannitol (0.5 to 1 g/kg).
- Corticosteriods are controversial in this setting.
- Antiviral therapy should be initiated.
- Administer acyclovir at an initial dose of 20 mg/kg IV.
- Pediatric ICU care is indicated so patient may require transfer.

Core Knowledge Points — West Nile Virus (WNV)

West Nile Virus first appeared in the Americas in summer 1999, when it became a New York City epidemic. It is an arbovirus in the Flaviviridae family which was first isolated in 1937 in a woman with a febrile illness in Uganda. It is transmitted by mosquitoes of the Culex family; with birds as natural hosts. Humans become infected but they are dead end hosts — meaning if a mosquito bites an infected person; that human will not directly transmit the disease.

Most WNV infections are generally asymptomatic with approximately 120 to160 asymptomatic infections per one symptomatic patient. Significant WNV infections are rare in children.

Severe neurological disease due to WNV infection has occurred in patients of all ages although older patients are at higher risks. WNV should be considered in all persons with unexplained encephalitis and meningitis.

- Most WNV infections are mild and often clinically unapparent.
  - Approximately 20% of those infected develop a mild illness (West Nile fever).
  - The incubation period is thought to range from 3 to 14 days.
  - Symptoms generally last 3 to 6 days.
  - Nonspecific signs and symptoms initially; usually fever and abrupt onset of symptoms:
    - Malaise
    - Periocular pain
    - Lymphadenopathy
    - Mylagia
    - Headache
    - Nausea
    - Vomiting

- A minority of patients with severe disease developed a maculopapular or morbilliform rash involving the neck, trunk, arms, or legs.
- Neurological presentations include: altered mental status, ataxia and extrapyramidal signs, cranial nerve abnormalities, myelitis, optic neuritis, polyradiculitis, and seizures.
- Several patients experience severe muscle weakness and flaccid paralysis.
- Rare symptoms include: myocarditis, pancreatitis, and hepatitis.
- Treatment at this time is supportive.

Critical Actions – West Nile Virus

- Total leukocyte counts in peripheral blood are often normal or elevated, with lymphopenia and anemia also occurring.
- Hyponatremia is sometimes present, particularly among patients with encephalitis.
- CSF shows a pleocytosis with a predominance of lymphocytes and moderately elevated protein.
- CSF glucose is generally normal.
- Test of choice is West Nile Virus ELISA (blood or CSF); may see false positives with other viral infections.
- WNV testing for patients with encephalitis or meningitis can be obtained through local or state health departments: http://www.cdc.gov/ncidod/dvbid/westnile/city_states.htm
    - The most efficient diagnostic method is detection of IgM antibody to WNV in serum or CSF collected within 8 days of illness onset using the IgM antibody capture enzyme-linked immunosorbent assay (MAC-ELISA).
    - Since IgM antibody does not cross the blood-brain barrier, IgM antibody in CSF strongly suggests central nervous system infection.
    - Patients who have been recently vaccinated against or recently infected with related flaviviruses (e.g., yellow fever, Japanese encephalitis, dengue) may have positive WNV MAC-ELISA results.

Management – West Nile Virus

- Supportive care including monitoring, intravenous fluid resuscitation and control of secondary infection.
- Ribavirin in high doses and interferon alpha-2b were found to have some activity against WNV in vitro, but no controlled studies have been completed on the use of these or other medications, including steroids, antiseizure drugs, or osmotic agents, in the management of WNV encephalitis (CDC WNV fact sheet).

Core Knowledge Points - Reporting Suspected WNV Infection

Refer to local and state health department reporting requirements:
www.cdc.gov/ncidod/dvbid/westnile/city_states.htm

- WNV encephalitis is on the list of designated nationally notifiable arboviral encephalitides.
- Aseptic meningitis is reportable in some jurisdictions.

The timely identification of persons with acute WNV or other arboviral infection may have significant public health implications and will likely augment the public health response to reduce the risk of additional human infections.

Case Development

- PCR of CSF for herpes simplex infection was negative.
- West Nile Virus ELISA positive.
- Patient discharged 14 days after admission with mild neurologic sequelae.

References

Peterson LR, Hughes JM: West Nile encephalitis. *N Engl J Med* 2002;347:1225-1226.

Yim R, Posfay-Barbe KM, Nolt D, Fatula G, Wald ER. Spectrum of clinical manifestations of West Nile virus infection in children. *Pediatrics* 2004;114(6):1673-5.

www.cdc.gov **(accessed 09-21-05)**

Further reading

APLS: The Pediatric Emergency Medicine Resource Encephalitis pp159-162; ALOC 148-153.