July 2017 Critical Care Case of the Month

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History of Present Illness

A 62-year-old man was brought to the Emergency Department with an altered mental status after a neighbor found him unresponsive. Medications the paramedics found in his home were cyclobenzaprine, duloxetine, gabapentin, levothyroxine, ibuprofen, and tramadol.

Past Medical History, Social History and Family History

He had a past medical history of neck and back pain and hypothyroidism. He lived alone. There was a history of a C3-4 anterior cervical discectomy in 2010. Other history including family history was unobtainable.

Physical Examination

- Vital Signs: HR 61 beats/min, BP 86/50 mm Hg, RR 8 breaths/min, T 32.2° C
- General: arousable but did not answer questions. He had multiple tattoos. No needle track marks are identified.
- HEENT: pupils were small but reacted to light.
- Lungs: clear to auscultation.
- Heart: regular rhythm without murmur.
- Abdomen: soft without organomegaly or masses.
- Neurology: he moved all 4 extremities but minimally. Plantar reflexes were downgoing.

Which of the following should be done **immediately**?

1. Administer naloxone
2. CT scan of the head
3. Obtain a blood glucose
4. 1 and 3
5. All of the above

Correct!
4. 1 and 3

The cause of his altered mental status was unclear. A mnemonic for possible causes of altered mental state was AEIOU TIPS (Table 1).

Table 1. Causes of an altered mental state

- A — Alcohol/Acidosis
- E — Endocrine or Encephalopathy
- I — Infection
- O — Opiates, Overdose
- U — Uremia
- T — Trauma
- I — Insulin (hypo or hyperglycemia)
- P — Poisoning/Psychosis
- S — Stroke/Seizure/syncope

The patient has back pain and his pupils were small. These are sufficient to warrant administration of naloxone for possible narcotic overdose. He had a dramatic response to naloxone and becomes alert after naloxone administration. He states he may have taken some barbiturates. Initial laboratory values include:

- CBC: 13.8 K/mm³, hemoglobin 14.5 g/dL, hematocrit 44, platelets 240K/mm³
- Na⁺ 141 mmol/L, K⁺ 4.0 mmol/L, glucose 118 mg/dL, creatinine 1.7 mg/dL
- Lactate 1.0 mmol/L
- Aspartate aminotransferase (AST) 137 IU/L

He has no focal neurological signs and a head CT was not indicated at this time.

What additional laboratory **testing should be done?**

1. Creatine phosphokinase (CPK)
2. Thyroid function testing
3. Urine drug screen
4. 1 and 3
5. All of the above

Correct!
5. All of the above

He was mildly hypothermic which could be explained by hypothyroidism. He was apparently found unresponsive for an unknown length of time and rhabdomyolysis was possible. Although he responded dramatically to naloxone, drugs other than opiates could be causing his altered mental state.

Table 2. Additional laboratory obtained.

- Thyroid function studies: freeT4: 0.88 ng/dL (normal 0.7-1.9 ng/dl), TSH 13.3 mIU/L (normal 0.4-4.0)
- CPK 6,536 IU/L (normal 22-198)
- Urine drug screen: amphetamine, cannabis, heroin, opiates, oxycodone

A portable chest x-ray was also performed (Figure 1).

Figure 1. Admission portable AP chest radiography.

He was admitted for observation and given fluids because of his hypotension. External warming was initiated. Empirical antibiotics were started for possible sepsis although no focal infection was apparent.

Early the next morning he suffered a pulseless electrical activity (PEA) arrest. The nurse reports that he complained of difficulty breathing and just “bradied down”. Cardiopulmonary resuscitation (CPR) was initiated and he was intubated and given atropine. He was transferred to the ICU on mechanical ventilation with propofol that had been initiated on the floor. Initial vital signs in the ICU were a BP 57/38 mm Hg, pulse 72 beats/min, temp 37° C.

What should be **done at this time?**

1. Begin norepinephrine administration
2. Repeat his portable chest x-ray
3. Stop the propofol and perform a careful neurological examination
4. 1 and 3
5. All of the above

Correct!
5. All of the above

He had received fluid administration overnight and vasopressor administration was appropriate for his hypotension. Norepinephrine (Levophed) is a commonly used vasopressor. His blood pressure quickly rose to 190/78 with norepinephrine administration. His propofol was stopped and his neurological examination showed he moved his eyes, head, neck, and chewed his endotracheal tube. The only deep tendon reflex elicited was his ankles which were only 1+. He did not move his arms or legs against gravity, but seemed to feel pain with a weak withdrawal of his right leg and arms. Babinsky's sign was not seen. A repeat portable chest x-ray is shown in Figure 2.

Figure 2. Portable AP chest radiograph after arrest.

What are the **possible causes** of his neurological state and dysautonomia?

1. Guillain-Barré syndrome
2. Wound botulism from injecting black tar heroin
3. Spinal cord compression
4. 1 and 3
5. All of the above

Correct!
4. 1 and 3

The cause of his neurological picture is not entirely clear. He appears to have an acute quadraparesis with dysautonomia and hyporeflexia. This syndrome is classic for acute demyelinating radiculopathy or Guillain-Barré syndrome (1,2). His history suggests the possibility of wound botulism, but this usually affects muscles of the head and neck, and was not associated with dysautonomia. Also, no focal skin infection or skin-popping sites were apparent. Spinal cord compression was typically associated with hyperreflexia and Babinsky's sign, but hyporeflexia and dysautonomia can be seen in acute high cervical lesions. Review of the emergency medical system records revealed that the patient had initially complained of being unable to move for the 24 hours prior to his admission, but this history was initially lost early in the admission when attention was focused on his altered mental status. His chest x-ray showed increased consolidation in his right lung most likely secondary to aspiration pneumonia – possibly related to respiratory failure due to neuromuscular weakness of the bellows.

What testing **should** be performed?

1. Magnetic resonance imaging (MRI) of the cervical spine
2. Electrophysiological studies (nerve conduction and electromyography)
3. Spinal tap
4. 1 and 3
5. All of the above

Correct!
5. All the above

A spinal tap was performed which showed a normal opening pressure and pink clearing fluid. In tube 1 the white blood cell count was 1/HPF and protein 138 mg/dL (normal 15 to 45 mg/dL) MRI of the cervical spine was shown in Figure 3.

Figure 3. Representative view of the cervical MRI.

The neurology consultant was asked to perform electrophysiological studies, but declined. The consultant felt the patient was most likely suffering from acute spinal cord compression related to chronic spinal stenosis. The Intensivist felt that Guillain-Barré syndrome could not be entirely ruled out since prior MRI scans were not available, and hyporeflexia and dysautonomia are less common in acute spinal cord compression. Abuminocytological dissociation seen in the CSF was seen in about 70% of patients with GBS after the first 7 days of illness, but was non-specific. Guillain-Barré syndrome (GBS) was a rapid-onset autoimmune disease of the peripheral nervous system causing muscle weakness (1). During the acute phase, the disorder can be life-threatening with about 15% developing weakness of the breathing muscles requiring mechanical ventilation and some are affected by severe dysautonomia – a common cause of PEA arrest. Because of the autoimmune nature of GBS, treatment usually consists of intravenous immunoglobulin (IVIG) or plasmapheresis in addition to supportive therapy (1). Simultaneous GBS and cervical myelopathy have been previously reported (3). The patient was initially treated with IVIG for GBS, but after the MRI was obtained, this was discontinued and a decompressive laminectomy was performed the next day.

References

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3. Al-Harbi T, Abdulmanaa S, Bunyan R. Coincidence of Guillain-Barré syndrome in a patient with cervical spondylotic myelopathy, case report. Journal of Neurological Sciences. 2015; 357:e328. [\[CrossRef\]](#)