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Guillain-Barre Syndrome in a Patient With Sickle Cell Anemia

Kunjan Udani, MD

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Introduction

- Sickle cell disease (SCD) can have a myriad of central and peripheral nervous system manifestations.
- Guillain-Barre syndrome (GBS), an acute immune-mediated polyneuropathy, is believed to result from molecular mimicry as an immune response to a preceding infection cross-reacts with peripheral nerves. Symmetrical ascending paralysis and absent deep tendon reflexes are pathognomonic features of GBS.

History

- 24 year old male with sickle cell anemia status post splenectomy, chronic back pain and hypertension was admitted for intractable nausea, vomiting and diarrhea for approximately a week.
- Approximately two weeks after the onset of abdominal symptoms, he developed bilateral lower extremity weakness and paresthesia in the buttocks radiating to the soles associated with numbness as well as bladder & bowel incontinence.

Physical Examination

- Vitals: T 99.9, HR 100, RR 18, BP 153/82, O₂ saturation 93% on room air
- Physical examination:
 - General: awake, alert and oriented x 3
 - CVS: normal heart sounds, regular rate and rhythm, no murmurs
 - Respiratory: clear to auscultation, aerating well
 - Abdomen: soft, nontender, non-distended
 - Neurological: absent Achilles and patellar reflexes, Babinski reflex negative, decreased sensation and proprioception in lower extremities, strength - 4/5 on dorsiflexion bilaterally, 3+/5 on hip extension & flexion and knee extension

Hospital Course

- On admission at an outside hospital, he was diagnosed with acute colitis, sickle cell crisis and acute renal failure, which were treated with intravenous hydration, opiates, antibiotics (clindamycin, levofloxacin) and anti-emetics.
- On day 3 of his admission, he experienced bilateral lower extremity weakness and pain in the buttocks radiating to the soles of the feet with associated numbness and bladder & bowel incontinence.

Hospital Course (continued)

- At this time, he was transferred to our facility for neurosurgery evaluation due to concern for spinal cord compromise. Based on the MRI of the lumbosacral spine, cord compression was ruled out.
- Given the patient's presentation and physical exam, it raised suspicion for GBS. LP was performed and CSF studies revealed cytoalbuminologic dissociation, confirming our doubt.

Imaging

MRI of the lumbosacral spine: epidural lipomatosis and spinal canal stenosis, most notable at L4-5 and L5-S1 with disc bulging and no evidence of spinal cord compromise (red arrows).



CSF Analysis

| Color | Yellow |
|-------------------|--------------------------|
| Appearance | Clear |
| Xanthochromia | Present |
| WBC | <u>12 mm³</u> |
| RBC | 68 mm ³ |
| CSF Total Protein | <u>330 mg/dL</u> |

Cerebrospinal fluid (CSF) analysis consistent with cytoalbuminologic dissociation.

Treatment

- He was treated with a 5-day course of intravenous immunoglobulin with subsequent improvement in symptoms.
- The patient was discharged with instructions to follow up with neurology and neurosurgery outpatient.

Discussion

- Clinically, SCD has been associated with vaso-occlusive crises, increased susceptibility to certain infections and hemolysis. Repeated vaso-occlusive crises lead to multiple complications including chronic pain, ischemic or hemorrhagic strokes, acute chest syndrome, splenic infarction and renal infarction. Neurological complications include chronic headaches, epilepsy and cognitive impairment secondary to anemia, hypoxia and silent infarcts.
- Diagnostic criteria for GBS includes progressive weakness in at least two extremities, areflexia and symptoms progressing for less than 4 weeks. Patients may also complain of sensory deficits and a preceding illness.

Discussion (continued)

- Our patient's presentation posed a diagnostic dilemma. As he was unable to identify if the lower extremity weakness was ascending or symmetrical, it raised a concern for acute sickle cell crisis and resultant spinal cord infarct. Anchoring further affected clinical decision making.
- Continued worsening symptoms with conservative management made us suspicious of other possible etiologies such as GBS, given his antecedent history of gastroenteritis, versus acute inflammatory demyelinating polyradiculoneuropathy. A lumbar puncture was performed and CSF studies revealed a cytoalbuminologic dissociation indicative of GBS.

Conclusion

- GBS should be considered in an appropriate clinical setting, particularly in patients who report an infection preceding the onset of neurologic symptoms such as paralysis and paresthesia.
- Prompt recognition and management is crucial to avoid a catastrophic outcome.
- Anchoring should be avoided and clinical decision making should be questioned frequently in patients with chronic medical conditions and lack of clinical improvement with implemented treatment plans.

References

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Thank you