An Interesting Presentation of Shoulder Weakness

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Disclosures

I have no actual or potential conflict of interest in relation to this program/presentation.
M.M. is a 53 yo healthy male lifeguard who presents for evaluation of acute onset right shoulder girdle pain and weakness after attempting to pull a 600 pound jet ski off of a trailer the previous day. Deep aching pain in the shoulder and weakness with overhead press and side planking on the right at the gym the morning after the injury. Denies neck pain. Denies previous injury to the shoulder.
History cont’d

• ROS: Negative outside of right shoulder pain. Denied recent illness.
• PMHx: Negative
• PSHx: Negative
• FamHx: Non-contributory
• SocHx: Non smoker, rare Etoh use.
• Works as lifeguard. Very physically active-running, lifting, swimming daily.
• Medications: None.
• Vaccinations: UTD. Influenza vaccine 2 wks prior.
Physical Exam

- **General:** Well appearing in NAD
- **Right Shoulder:** Normal muscle bulk, no skin changes
- AROM 140 flex/abduction-**painful arc** >90, Normal IR/ER
- No ttp over cervical trigger points, AC, biceps tendon
- **5-/5 Strength w/shoulder abduction**
- Negative impingement signs
- Negative Speeds, Yergason’s
- Negative O’Briens
- Negative apprehension, negative instability
- **++Medial scapular winging and weakness with wall push up**

**IMAGING**
- **C-Spine XR:** Mild multi level DDD
- **Shoulder XR:** Normal
Working diagnosis

- Traction injury brachial neuritis long thoracic nerve neurapraxia serratus anterior palsy medial scapular winging.

- Treated with prednisone taper
- MRI C-spine ordered
- Placed off work
- Close follow up arranged
Interval History

Over the next **5 weeks** shoulder girdle pain and weakness progressed to include night pain limiting sleep and difficulty with lifting the arm overhead to shampoo his hair.

MRI of the right shoulder done during this time demonstrated **small SLAP tear**.

**4 weeks** after initial presentation reported pins and needles sensation starting on soles of bilateral feet and ascending to the level of the Achilles.

XR and MRI of the lumbar spine demonstrated **mild DDD w/o stenosis**.

Patient was **referred to neurology** for further evaluation of paresthesias.

**5 weeks** following presentation noted similar pins and needles sensation in 4/5th digits of bilateral hands.

**6 weeks** after initial presentation presents to the ER for evaluation of progressive bilateral upper and lower extremity paresthesias, weakness and gait disturbance.
Physical Exam

VS: Afebrile  BP: 126/78  HR: 64  RR:16  SpO2 99% RA

Gen: Anxious appearing, non toxic, normal speech, no respiratory distress

MSK: R Shoulder AROM 100 flexion and abduction with painful arc
• 4+/5 strength bilateral shoulder abduction with R medial scapular winging noted
• 4+/5 bilateral grip strength
• Impaired heel and toe walking

Neuro: CN 2-12 normal
• Negative Romberg
• Hoffman negative.
• Down going great toes bilaterally
• Cerebellar signs negative.
• Sensation grossly intact to light touch & temp
• Wide based gait

Reflexes:
• Biceps/Triceps/Brachioradialis: 0+ b/l
• Quadriceps: 1+ b/l
• Achilles 0+ b/l
Differential Dx

- Multiple Sclerosis
- Peripheral polyneuropathy
- Guillain-Barré Syndrome
- Amyotrophic Lateral Sclerosis
- B12 deficiency
- Transverse Myelitis
- Idiopathic Inflammatory Myopathy
ER Work Up - Labs/Imaging

- CXR: Normal
- MRI Brian: *Nonspecific white matter hyperintensities*
- MRI C/S: Mild multilevel DDD w/o stenosis
- **CMP:** ALT 43 otherwise wnl
- CBC: wnl
- **ESR:** 26  B12: wnl
- CPK: wnl
- ANA: Negative
- RPR: Negative
- LP: *Albuminocytologic Dissociation*
- Negative oligoclonal bands
- B6 wnl
- Negative Gram stain and Culture
Guillain-Barré syndrome
Admitted to ICU under Neurology Service

5 days of plasmapheresis followed by 5 days IV immunoglobulin (IVIG)

Cessation of ascending paresthesia by completion of plasmapheresis and ambulatory with PT in the halls following completion of IVIG.

D/c’d from the hospital after 17 day inpatient stay with plans for outpatient PT and close neurology follow up.
Following discharge patient was actively participating in aggressive outpatient PT and making strength gains in BUE/BLE, still with right scapular winging.

Stocking glove paresthesia improving up until **3 weeks post discharge** (10 weeks from sx onset) when he developed a recurrence of ascending paresthesia, increased weakness and gait disturbance.

Subsequently treated with **5 day outpatient course of IVIG** with improvement in fatigue, paresthesia and weakness.

NCS of BLE demonstrated **SEVERE SENSORIMOTOR PERIPHERAL NEUROPATHY WITH MIXED AXONAL AND DEMYELINATING FEATURES OF LOWER EXTREMITIES.**
Outcome & Follow Up

- Preliminary NCS of BUE demonstrated **RIGHT SUPRASCAPULAR MOTOR DEMYELINATIVE NEUROPATHY. NO ELECTROPHYSIOLOGICAL EVIDENCE OF BRACHIAL PLEXOPATHY**

- **ABNORMAL BLINK TEST** (subclinical trigeminal and facial nerve neuropathy w/stimulation of supraorbital nerve)

- Currently awaiting EMG of BLE and RUE along with brachial plexus MRI.

- Consult with orthopedics re: SLAP tear but deferred pending further neurology w/u

- Has not returned to work but approved to enroll in EMT recertification course
Current Working Diagnosis

Based on sx relapse > 8 weeks, severe demyelination demonstrated on NCS and abnormal blink response.
Discussion

Guillain Barre’ Syndrome (GBS)- An acute inflammatory demyelinating disorder of the PNS that presents with symmetric progressive ascending flaccid paralysis, paresthesias and sensory changes.

- **EPIDEMIOLOGY**
  - Incidence: 1-2/100,000 annually
  - M>F (Approximately 1.1-1.7:1)
  - Most commonly presents 5-6th decade
  - Unclear etiology: 2/3 have preceding viral illness 1-4 weeks prior to sx onset.
    - *Campylobacter* 20%, EBV, CMV, Mycoplasma, HIV
    - Single case report in the literature of GBS following upper extremity traction injury.
  - Typical course<4 weeks with 50% patients reaching nadir in sx by week 2 and 90% by week 4
**DIAGNOSIS**
- Clinical diagnosis supported by LP demonstrating elevated protein without pleocytosis and EDS
- NCS: *nerve conduction slowing or block c/w demyelination*. Can help delineate subtypes of GBS and rule out alternative diagnosis.

**TREATMENT**
- 1/3 of those affected will require ventilatory support due to phrenic nerve involvement
- Treatment includes supportive care, IVIG, plasmapheresis.
- **Steroids not effective** in acute GBS and in fact may worsen symptoms.

**OUTCOME**
- Most begin to recover at 4 weeks with *mean time to complete recovery being 6 months* in 80% of cases.
- 10-15% of patients are left with permanent neurological impairments
- **3-5% of patients with GBS will die** from complications
- Relapse rate of 5% mostly within first 8 weeks of sx onset.
- **Approximately 2% of patients dx with GBS will progress to diagnosis of CIDP.**
## Discussion

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<tr>
<th>GBS</th>
<th>CIDP</th>
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<tr>
<td><strong>Incidence</strong> 1-2/100,000 annually</td>
<td>1.6/100,000 annually</td>
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<td><strong>M:F</strong> 1.1-1.7/1</td>
<td><strong>M:F</strong> 1:2</td>
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<td><strong>Peak incidence</strong> 5th &amp; 6th decades</td>
<td>Increased risk &gt;50 yo</td>
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<td><strong>Rapid onset with quick progression. Course stable by week 4 (90%).</strong></td>
<td><em>Slowly progressive course &gt;4-8 weeks. Increased frequency of relapse.</em></td>
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<td><strong>Preceding hx of URI/GI illness, ?vaccination</strong></td>
<td><strong>Hx of preceding URI/GI illness rare (&lt;10%)</strong></td>
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<td>+Albuminocytologic dissociation</td>
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<td><strong>Respiratory failure more common (up to 1/3)</strong></td>
<td><strong>Respiratory failure less common</strong></td>
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<td><strong>More common to see cranial nerve involvement</strong></td>
<td><strong>Posterior column sensory signs more common</strong></td>
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<td>Not steroid responsive. IVIG, plasmapheresis.</td>
<td><strong>Steroid responsive.</strong> IVIG, plasmapheresis, immune modulators</td>
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**Take Home Points**

*Things are not always as they seem.* Dx is difficult and presentation can be varied from case to case. Must keep in mind that **GBS/CIDP is a spectrum of disease.** Additionally, there may be concurrent diagnoses confounding the picture.

*Dx can be messy.* Overall a poorly understood disease process. Etiology is not well established. **Association** with preceding URI/GI illness, recent surgery, **trauma,** post vaccination.

**GBS is a medical emergency.** Rapidly progressive disease course that can result in respiratory failure if not addressed in a timely manner. **5% mortality associated with dx.**

**Think outside the box.** Dx of CIDP should be considered when a patient thought to have GBS *deteriorates again after 8 weeks from onset* or when deterioration occurs 2x or more.

**Don’t put all your eggs in one basket.** While NCV/EMG can help guide us in identifying subtypes of GBS and r/o alternative diagnoses they do not always provide a complete picture of disease process. **Can be negative in early disease.** Must use clinical correlation.
Thank you for your attention!
References

• Donofrio, P.D. Guillain Barre Syndrome Continuum (Minneap Minn) 2017;23(5):1295–1309

