**Introduction**

Copper deficiency is a rare complication of peptic ulcer or bariatric surgeries, malabsorptive disorders such as celiac disease, and excess zinc ingestion. A mimicker of subacute combined degeneration seen in cobalamin (vitamin B12) deficiency, copper deficiency myelopathy most commonly presents with sensory ataxia, paresthesia, anemia or neutropenia, and dorsal columnar enhancement on contrast imaging.

**Clinical Timeline**

- **-6 mo**
- **-4 mo**
- **-2 mo**
- **-2 wks**
- **-1 wk**
- **Hospitalization**
- **+3 d**
- **+6 wks**
- **+12 wks**

- **-6 months from presentation:** *First fall* thought to be mechanical.
- **-4 months:** *Second fall* occurred while walking down stairs.
- **-2 months:** *Third fall* triggered by vertigo.
- **-1 week:** Establishes care with PCP. Positive orthostatic vitals. Several centrally acting medications are discontinued.
- **Timepoint 0:** Hospitalization 3 weeks later for ascending bilateral foot and hand paresthesias and unsteady gait.
- **+3 days:** Neurology initiates treatment for copper deficiency myelopathy with IV cupric chloride 8 mg daily for 7 days followed by oral cupric chloride 4 mg twice daily.
- **+6 weeks:** At PCP follow-up copper and ceruloplasmin levels normalize (174 ug/ml and 42 ml/dl respectively), paresthesias and proprioceptive sensation improve, distal reflexes and strength recover.
- **+12 weeks:** Follow-up with PCP demonstrates persistent vibratory sensation loss and atactic gait.

**Discussion**

- Copper absorption occurs via P-type ATPase in the duodenum and stomach. Sensory ataxia in copper deficiency involves both central myelopathy and length dependent peripheral neuropathy of axonal large fibers.
- Differential for posterior columnar enhancement on contrast spinal imaging:
  - Diminished pinprick sensation in stocking and glove distribution
  - Spastic gait
  - Diminished hip flexor strength (4/5)
  - Absent patellar and ankle deep tendon reflexes
  - Absent vibratory sensation in bilateral lower extremities to the knees
  - Upward Babinski
  - Positive Romberg

**Laboratory Findings**

<table>
<thead>
<tr>
<th>ABNORMAL</th>
<th>NORMAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic hyponatremia 124 mEQ/l</td>
<td>Remainder of BMP, Ca²⁺</td>
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<tr>
<td>Normocytic anemia Hb 10.2 g/dl</td>
<td>ACTH stim testing</td>
</tr>
<tr>
<td>B6 low 10 ng/ml</td>
<td>B12, Folate, MMA</td>
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<tr>
<td>Copper low 47 ug/ml (norm 80-155)</td>
<td>Zinc</td>
</tr>
<tr>
<td>Ceruloplasmin low 17 ml/dl (norm 20-60)</td>
<td>Niacin</td>
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<tr>
<td>Peripheral smear: hypochromic RBC</td>
<td>TSH</td>
</tr>
<tr>
<td>Ferritin low 12.6 ng/ml</td>
<td>RPR, SPEP, UPEP</td>
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<tr>
<td>Iron 31 ug/ml</td>
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<tr>
<td>Albumin low 3.4 g/dl</td>
<td>AST, ALT, TB, TP</td>
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<tr>
<td>Platelets elevated 650 x 10⁹/L</td>
<td>Urine studies:</td>
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<td></td>
<td>Na 31, Osm 432</td>
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</tbody>
</table>

**Imaging**

**Figures 2 and 3:** Sagittal and axial views of T2 weighted MRI total spine demonstrating hyperintensity of the dorsal columns of the cervical spinal cord from C3 to C6.

**Key Points**

- Unexplained neuropathy, anemia, and sensory ataxia in a patient with a history of gastric surgery or malabsorptive disorder should cue physicians to evaluate for copper deficiency, particularly if repletion of B complex vitamins fails to improve symptoms.
- Repletion with 2-4 mg/day of oral or intravenous then oral therapy has been reported to prevent further neurologic deterioration even in cases of suspected malabsorption. 5,11
- Repletion of copper stores and reversal of the underlying etiology (cessation of exogenous zinc, bypass revision) can result in rapid resolution of the myelopathy and hematologic insults. However, complete recovery of the axonal predominant peripheral neuropathy is unlikely.

**References**