Diagnosing Giants

Guiset Carvajal Bedoya, M.D.
Internal Medicine Resident PYG-2
Billings Clinic
Disclosure

I have nothing to disclose
Case: Clinical Presentation

68 yo M found to have an asymptomatic elevated BP of 255 / 117 during a PM&R visit.

Complains: Unilateral shoulder pain - Parsonage Turner Syndrome.

Past medical history: Severe pityriasis rosea in 2004, former smoker.

No surgical history.

Medication history: Acetaminophen PRN.

Social history: Occasional alcohol use, no tobacco or illegal drug use, married, retired.
Case: Clinical Presentation

Review of systems
(-) Constitutional symptoms
(-) Rash
(-) Visual symptoms, unusual pain in the face, throat or tongue, jaw or arm claudication, temporal headaches
(-) CP, SOB, palpitations, orthopnea, syncope, edema
(-) Abd pain, melena, hematemesis
(-) Hematuria, dysuria
(-) Weakness, shoulder or pelvic girdle stiffness
(-) Paralysis
Case: Physical Examination

BP 255/117 HR 82, RR 18, Sat 96%
BP and pulses were symmetric

Well developed, in no distress

Moist mucous membranes, PERRLA, EOMI, no temporal artery tenderness

No thyroid enlargement or nodularity, no carotid bruits

RRR, CTA, PMI non displaced, no peripheral edema

No abdominal bruits or megaly

No LAD, rashes or other skin lesions
Case: Initial Management

- He was started on two different antihypertensive medications and followed up 1 week later.

- His BP continued to be elevated at 236/100. He was then started on a third, and later a four antihypertensive w/o achieving BP control.

- Evaluation for secondary causes of hypertension was initiated.
# Case: Laboratory Data

<table>
<thead>
<tr>
<th>Creatinine of 1.0 (baseline), K 3.8</th>
</tr>
</thead>
<tbody>
<tr>
<td>UA without active urinary sediment</td>
</tr>
<tr>
<td>Plasma renin activity and am cortisol WNL</td>
</tr>
<tr>
<td>Aldosterone was low, metanephrines and normetanephrine’s were not elevated</td>
</tr>
<tr>
<td>ANA panel, ENA Ab panel and ANCA screen negative, thyroid peroxidase antibody was normal</td>
</tr>
</tbody>
</table>

Normal anti CCP, IgG4, RF

ESR 64, CRP 4.6
Case: Imaging

- **TTE** EF 55-59%, normal systolic function. No pulmonary hypertension.
- **Carotid and abdominal - renal US** were w/o abnormalities.
Case: Imaging
Case: Imaging
What is the diagnosis?
Vasculitis - Overview

Giant Cell Arteritis (GCA)
Giant Cell Arteritis

- It is the most common systemic vasculitis. (1)

- Higher incidence among individuals of Northern European ancestry. 3:1 Female to male. (1)

- Age – greatest risk factor, >50y – Peaks 70-79y.

- PMR occurs in 40-50%.

- Etiology and pathogenesis are unknown – HLA-DR4.

# Giant Cell Arteritis

## Clinical Features

<table>
<thead>
<tr>
<th>Large vessel GCA</th>
<th>Cranial Arteritis GCA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Younger Patient (66)</td>
<td>Older onset (72)</td>
</tr>
<tr>
<td>- Constitutional symptoms</td>
<td>- Constitutional symptoms</td>
</tr>
<tr>
<td>- Less likely to have HA</td>
<td>- New Headache</td>
</tr>
<tr>
<td>- Arm claudication</td>
<td>- Jaw claudication</td>
</tr>
<tr>
<td>- Musculoskeletal symptoms</td>
<td>- Amaurosis fugax</td>
</tr>
<tr>
<td>- Aorta and its major proximal branches</td>
<td>- Permanent visual loss</td>
</tr>
<tr>
<td>* Atypical Features</td>
<td>- Musculoskeletal symptoms</td>
</tr>
<tr>
<td></td>
<td>- Cranial branches of the arteries originating from aortic arch</td>
</tr>
</tbody>
</table>
Giant Cell Arteritis

Physical exam findings

- Decrease pulses
- Discrepant BP
- Temporal artery tenderness
- Bruits: Carotid, supraclavicular, axillary, brachial, femoral, orbits, abdominal
Giant Cell Arteritis

Laboratory findings

- CBC – Normochromic anemia, reactive thrombocytosis, normal WBC
- Elevated ESR vs CRP
- ?IL-6
Giant Cell Arteritis

Diagnosis

- Symptoms + Histopathology (Temporal artery biopsy)

- Symptoms + Imaging (Color Doppler US, CT, CTA, MRI, MRA, PET, PET CT)

- ESR, CRP can aid the diagnosis – but non specific
Giant Cell Arteritis

Treatment

- Prednisone – Methylprednisone
- ASA
- Steroid Taper (9 – 12 months)
- Tocilizumab
- Other: Vit D, Calcium, Bisphosphonates
Steroids were started.

After one month of treatment his BP improved, but he continued to need antihypertensive medications.

His ESR and CRP normalized.
Back to the case

• Steroids were tapered in combination with tocilizumab, an interleukin 6 receptor blocker that was initiated to be continued as monotherapy thereafter.
Trial of Tocilizumab in Giant-Cell Arteritis


ABSTRACT

BACKGROUND
Giant-cell arteritis commonly relapses when glucocorticoids are tapered, and the prolonged use of glucocorticoids is associated with side effects. The effect of the interleukin-6 receptor alpha inhibitor tocilizumab on the rates of relapse during glucocorticoid tapering was studied in patients with giant-cell arteritis.

METHODS
In this 1-year trial, we randomly assigned 251 patients, in a 2:1:1:1 ratio, to receive subcutaneous tocilizumab (at a dose of 162 mg) weekly or every other week, combined with a 26-week prednisone taper, or placebo combined with a prednisone taper over 12 weeks. Among the 125 patients in the tocilizumab group, 61 received prednisone tapering every other week, and 64 received weekly tocilizumab; among the 126 placebo recipients, 62 received prednisone tapering every other week, and 64 received weekly placebo.
Questions
Key Points

• Large vessel vs Cranial arteritis

• Diagnosis: Symptoms + histopathology or symptoms + Imaging

• Steroid taper – Tocilizumab as monotherapy
Thank You

Billings Clinic
Internal Medicine Residency

Class of 2019