



# RhAPP

RHEUMATOLOGY ADVANCED  
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**RhAPP**

RHEUMATOLOGY ADVANCED  
PRACTICE PROVIDERS

# Giant Cell Arteritis Roundtable

Monica Richey, MSN, ANP-BC

Betsy Kirchner, DNP

# Disclaimer

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# Faculty Disclosures

**Monica Richey, MSN, ANP-BS**

Consultant: AstraZeneca, GSK

**Betsy Kirchner, DNP**

Consultant: Janssen, Novartis

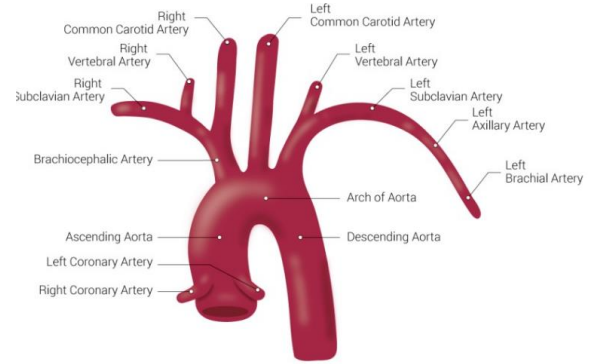
Speaker's Training: Sanofi Genzyme

# Outline

- What is GCA?
- Treatment issues
- Open Discussion

# What Is GCA?

- Systemic vasculitis
  - Most common
  - Most symptoms stem from inflammation of cranial branches of arteries that originate from the aortic arch
- Typically older, white patients
  - Almost never seen in < 50 years old
  - Incidence peaks in 70s

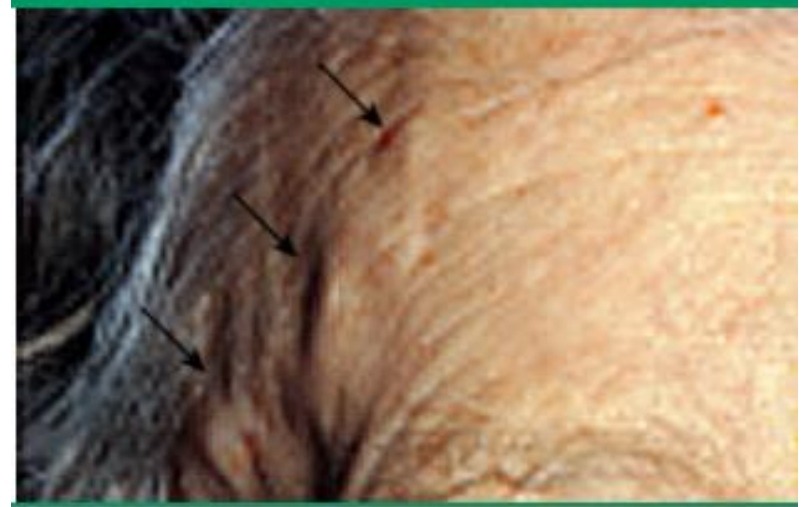


# Classic Clinical Presentation

- New headaches
- Abrupt onset of visual disturbances
- Jaw claudication
- Unexplained fever, anemia, or other constitutional symptoms
- High ESR and/or high CRP
- A current or prior diagnosis of polymyalgia rheumatica (PMR).
  - 40-50% of GCA have PMR
  - 10% of PMR have GCA

# Diagnosis

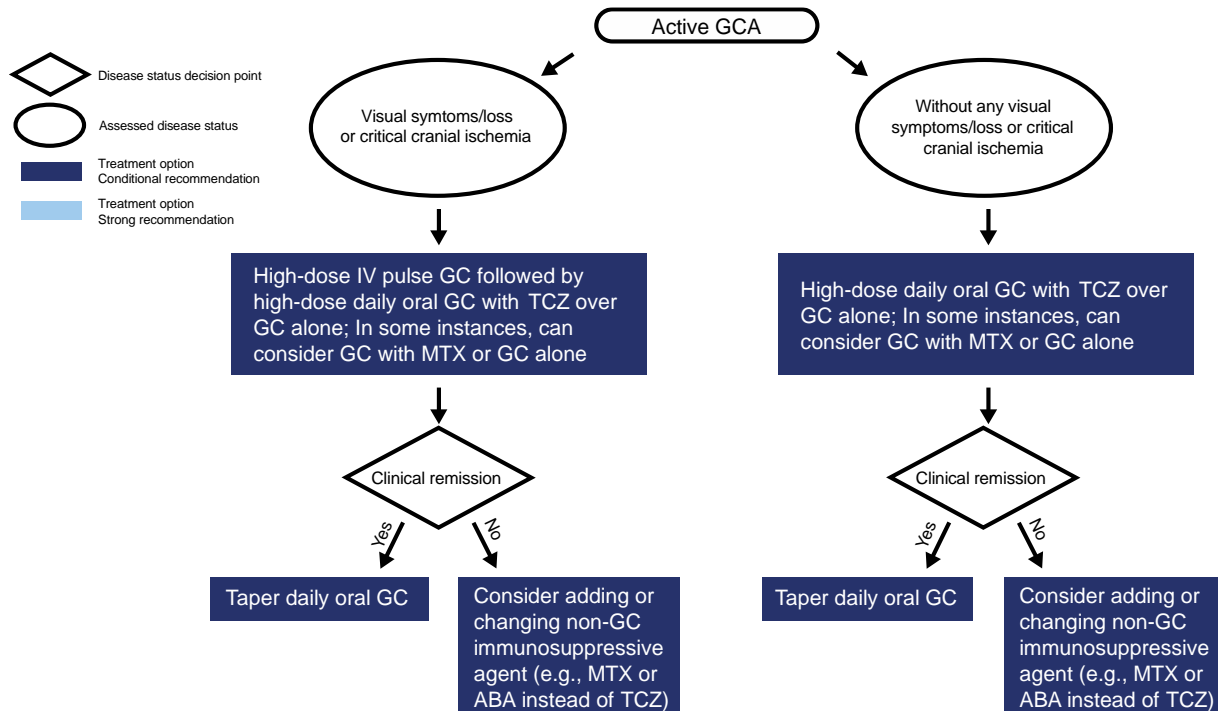
- History
- Physical
- Labs
- Imaging
- Temporal artery biopsy





# 2021 ACR – GCA Treatment Guidelines

## Overview of Treatment of Giant Cell Arteritis (GCA)



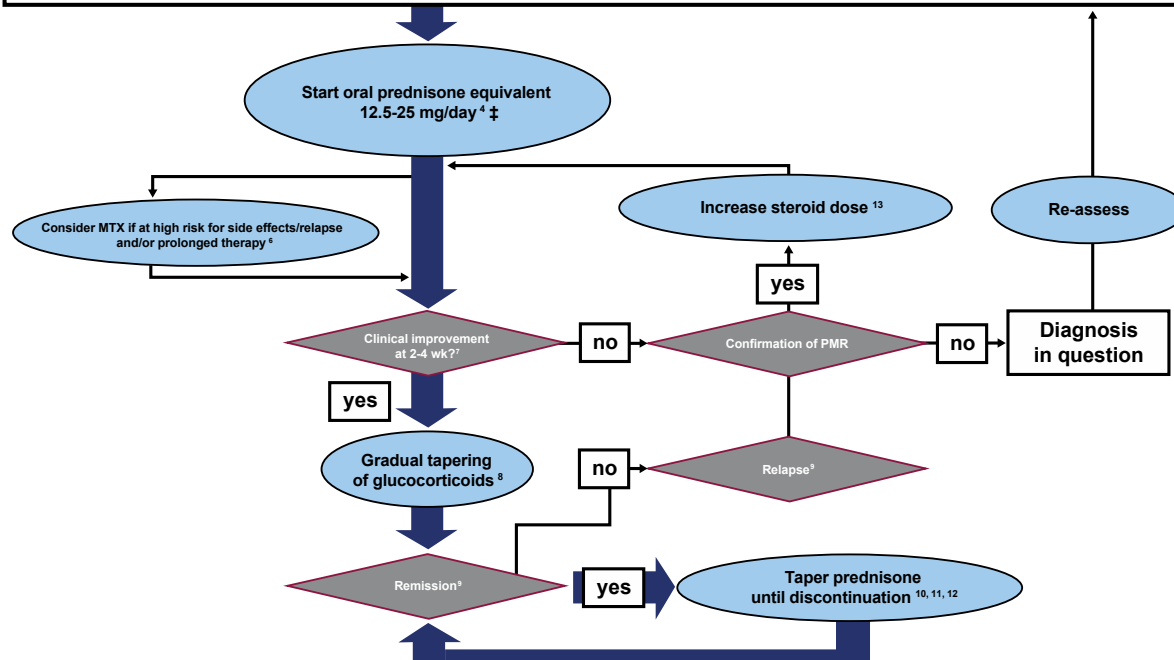
ABA = abatacept, AZA = azathioprine, GC = glucocorticoids, IV = intravenous, MTX = methotrexate, TCZ = tocilizumab.

Figure 1. Overview of treatment of giant cell arteritis.

# 2015 ACR – PMR Treatment Guidelines

## Patient fulfilling PMR case definition (primary or secondary ca re)

1. Assess comorbidities<sup>1</sup>, other relevant medications and other risk factors for steroid related side effects<sup>2</sup>
2. Assess possible risk factors for relapse/prolonged therapy<sup>3</sup>
3. Consider specialist referral (experience or risk of side-effects, relapse/prolonged therapy and/or atypical presentation)
4. Document minimal clinical and laboratory dataset



‡Consider i.m. methylprednisolone as an alternative to oral prednisone<sup>5</sup>

# Small Arsenal for GCA

- GCA is caused by vasculogenic T-cell and macrophages
- Corticosteroids:
  - target innate cytokines, such as IL-1 $\beta$ , IL-12, and IL-6, but have little effect on tissue-residing T cells.
- IL-6
  - secreted by T-cells
  - Antibody production by B cells
  - Serum concentration of IL-6 increase in active disease
  - Secondary inhibition of JAK

# Unanswered Questions

- Length of therapy
- Relapse
- Laboratory monitoring
  - Patient X inflammatory markers
- Is there a role for JAKs?
- Can we use it in PMR

# References

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3. Choy EH, De Benedetti F, Takeuchi T, Hashizume M, John MR, & Kishimoto T. Translating IL-6 biology into effective treatments. *Nature Reviews Rheumatology*. 2020; 16(6): 335-345.