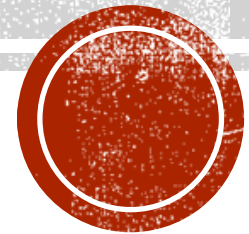


GOUT BEYOND HYPERURICEMIA AND EPISODIC ARTHRITIS: INSIGHT INTO METABOLIC DYSREGULATION AND INTEGRATED COMORBIDITY MANAGEMENT

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DISCLOSERS

■ PI, LOCAL SITE (VCU)

- Phase 3, Multicenter, Double-blind, Randomized Controlled Study Evaluating the Efficacy and Safety of Pegloticase Administered by Subcutaneous Injection Compared With Pegloticase Administered by Intravenous Injection, Both Administered Concurrently With Methotrexate Weekly, in a Participants With Uncontrolled Gout



BACKGROUND

Gout is:

- The most common form of inflammatory arthritis in adults (4% US Population)
- Rapidly increasing in prevalence along with obesity pandemic
- It is caused by hyperuricemia and MSU crystallization in joints and tissues
- Strong associations with CKD, HTN, obesity, and diabetes
- One of the chronic diseases with treatment guidelines and a measurable treatment target

But...

- Many patients never achieve effective disease control or reach urate goal
- Care is often episodic instead of longitudinal
- Not recognized as part of larger metabolic derangement or a chronic disease
- Ownership of management is often unclear



IMPACT OF POORLY CONTROLLED GOUT

- Recurrent ED or Urgent care visits
- Hospitalizations (often misdiagnosed as cellulitis or septic arthritis)
- Delaying hospital discharge and prolonging hospital stay
- Repeated steroid bursts leading to hyperglycemia, delirium, weight gain etc
- Functional impairment leading to poor quality of life
- Many missed workdays
- One uncontrolled gout patient can generate more healthcare utilization than many chronic diseases — **and it is largely preventable**



LEARNING OBJECTIVES:

- Recognize burden of gout as a chronic disease and its association with cardio-renal-metabolic (CKM) comorbidities
- Learn treat-to-target principles in gout management, including appropriate use and titration of urate-lowering therapies and flare prophylaxis
- Identify opportunities to optimize gout outcomes through multidisciplinary care, including coordination with primary care, nephrology, cardiology, and weight management services
- Describe emerging therapeutic and research approaches in gout, including cardiometabolic therapies and biomarker discovery using multi-omic profiling

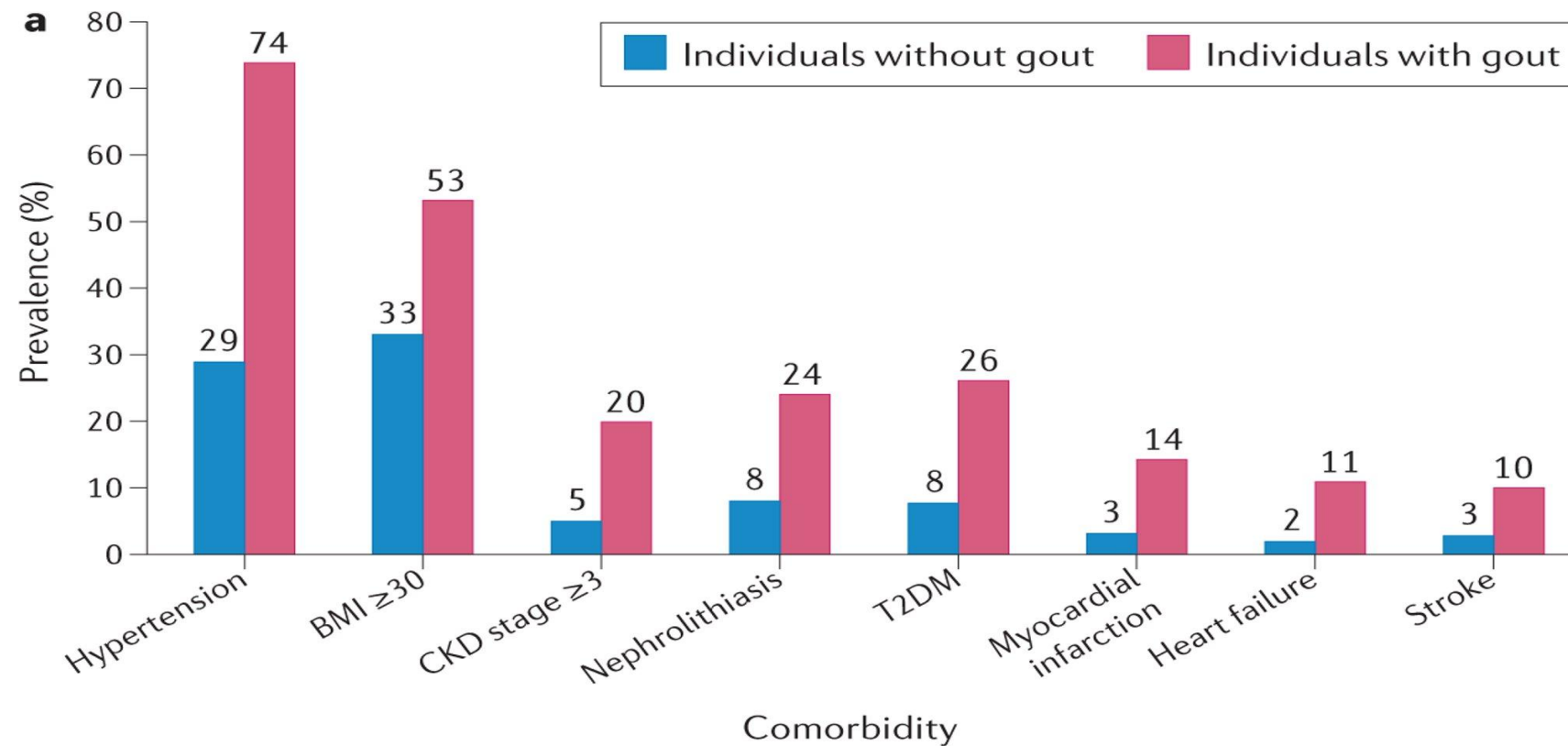


OUTLINE

- The burden of gout and its associated comorbidities
- The bidirectional relationship between gout and CKM comorbidities
- Treat-to-target gout management and flare prophylaxis
- Metabolic therapies and emerging treatments (SGLT2 inhibitors, GLP-1 agonists)
- The VCU Gout Clinic model : co-management and collaboration opportunities
- Step toward precision health, patient-centered gout care



COMORBIDITIES IN US ADULTS WITH AND WITHOUT GOUT (NHANES 2007–2008)



NHANES 2011-2018

- Multimorbidity (≥ 2 CKM comorbidities) 82.5% (OR 2.5 (1.9 to 3.3))
- Polypharmacy (≥ 5 prescription medications) 46.0% (OR 2.8 (2.2-3.6))



BIDIRECTIONAL LINKS BETWEEN GOUT, CARDIOMETABOLIC, AND RENAL DISEASE

CKM Comorbidities and Gout Risk

- CKD (stage ≥ 3): $\sim 2\text{--}3\times$ higher gout prevalence
- Obesity (BMI ≥ 30): $\sim 2\text{--}3\times$ higher incidence
- Metabolic syndrome increases risk of gout by $2\text{--}3\times$
- Metabolic syndrome present in $>60\%$ of gout patients

Gout Predicts Worse CKM Outcomes

- Faster CKD progression (accelerated eGFR decline)
- $\uparrow$ Incident hypertension
- $\uparrow$ Cardiovascular events (MI & stroke $\sim 20\text{--}40\%$ higher risk)
- $\uparrow$ Heart failure hospitalizations
- $\uparrow$ All-cause mortality



EPIDEMIOLOGIC LINK: HYPERURICEMIA/GOUT AND CARDIOVASCULAR/RENAL OUTCOMES

- Observational studies linking hyperuricemia to CKM risk
- Prospective cohorts: hyperuricemia precedes and predicts CKM risk
- **Reverse causality and confounding remains the concern**



CAUSALITY TESTED: MENDELIAN RANDOMIZATION (MR) STUDIES

- MR studies use genetic variants as proxy for an exposure to test **causal relationships** between serum urate and cardiometabolic traits
- Most studies treat **serum urate as the exposure** — asking whether high urate *causes* CMR comorbidities, a few tested it as outcome



KEY FINDINGS FROM BIDIRECTIONAL MR STUDIES

- **Direction of effect appears reversed**

- Genetically higher urate →  *No causal effect on:*
 - Body fat mass
 - Triglycerides
 - Fasting insulin
- Genetically higher fat mass / triglycerides / fasting insulin →  *Increase in serum urate levels.*

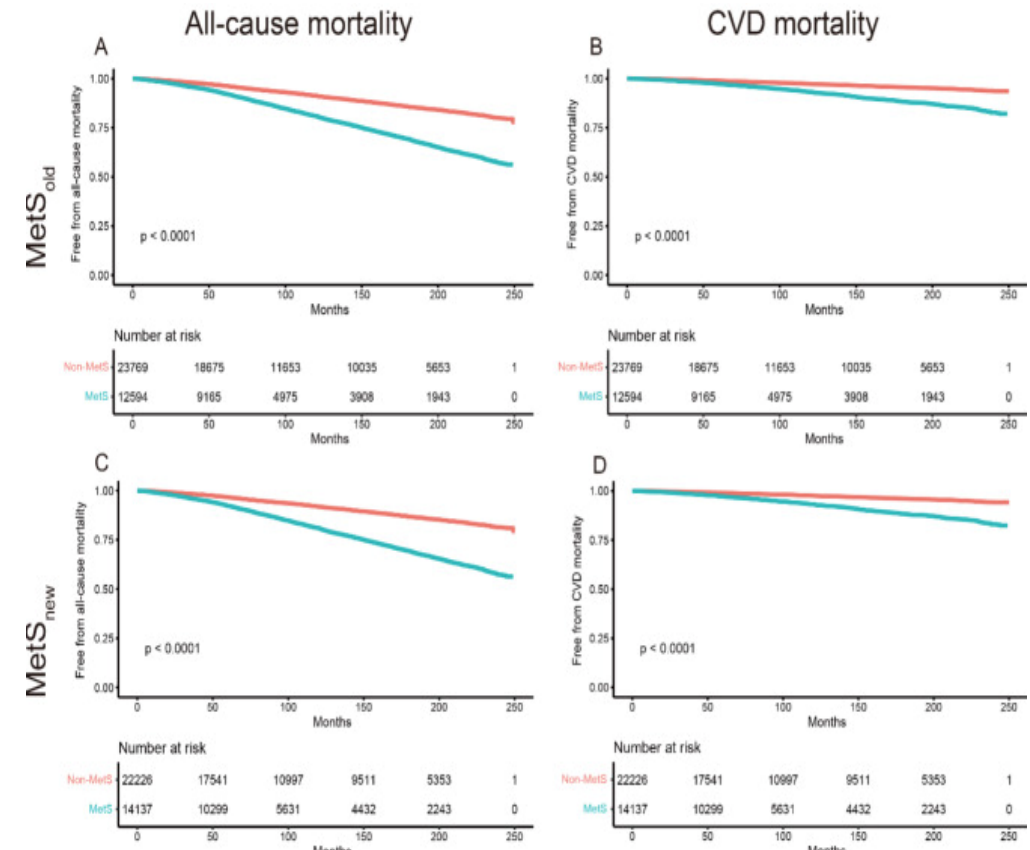
- **Quantitative results-**

- Each SD ↑ in fat mass → +0.3 mg/dL serum urate.
- Each SD ↑ in fasting insulin → +0.6–1.2 mg/dL serum urate



SU AND METABOLIC SYNDROME

- Data for 36,000 American patients extracted from the NHANES(1999–2018)
- Adding hyperuricaemia led to **+4.2%** pts diagnosed with metabolic syndrome
- both definitions were associated with an increased risk of all-cause and CV mortality
- The additional 4.2% pts identified by MetS_{new} exhibited a similar risk of all-cause and CV mortality as those who met MetS_{old} criteria



FREED STUDY

- **Purpose:** To investigate the effect of febuxostat on cerebral, cardiovascular, and renal events in patients aged 65 or older with hyperuricemia (high uric acid).
- **Method:** Patients (n 1070, Japan) were treated with either febuxostat or conventional therapy (lifestyle modification).
- **Key finding:** The febuxostat group showed a **significant reduction in serum uric acid levels and a decreased risk of the primary composite endpoint** (cerebral, cardiovascular, and **renal events**) compared to the conventional therapy group.
- The results were mostly driven by softer renal events than CVD events



RX OF SYMPTOMATIC HYPERURICEMIA

Trial	Population	Intervention	Main Result
FEATHER (2018)	443 patients with CKD stage 3 and asymptomatic hyperuricemia	Febuxostat vs placebo	Lowered uric acid but did not improve eGFR decline overall
PERL (2020)	530 patients with type 1 diabetes, early CKD, hyperuricemia	Allopurinol vs placebo	No difference in GFR decline despite lower uric acid
CKD-FIX (2020)	369 patients with stage 3–4 CKD at high risk of progression	Allopurinol vs placebo	No slowing of CKD progression or eGFR decline



ALL-HEART STUDY

- **Purpose:** To determine if allopurinol could prevent future cardiovascular events in patients with ischemic heart disease.
- **Method:** Patients with ischemic heart disease (n=5217, age >60 yrs, UK) were randomly assigned to receive either allopurinol 600 mg daily or usual care, and their outcomes were tracked. Severe CKD and HF patient were excluded.
- **Baseline uric acid 0.34 mmol/L and e GFR >60 in 90 % of patients**
- **Key finding:** Allopurinol did not significantly reduce the risk of heart attack, stroke, or cardiovascular death compared to usual care in patients with IHD



BEYOND SERUM URATE: CRYSTAL-DRIVEN INFLAMMATION

- Gout involves monosodium urate (**MSU**) **crystals**, which activate the **NLRP3 inflammasome** → release of IL-1 β , IL-18, and other inflammatory mediators
- This crystal-driven inflammation mirrors pathways seen in atherosclerosis:
 - Cholesterol crystals within plaques also activate NLRP3.
 - Creates a self-amplifying inflammatory loop (crystal → inflammasome → cytokines → more crystal deposition).

Supporting Evidence:

- CANTOS trial: IL-1 β inhibitor (canakinumab) in post MI patients with high CRP - ↓ recurrent CV events
 - A subanalysis of the patients with a history of gout (about 8% of participants) had a **52% lower risk of new gout attacks**
- Colchicine trial: colchicine 0.5 mg once daily in patients with chronic coronary A disease - ↓ CV events



PLAQUE & CRYSTALS-HUMAN STUDIES

- **MSU crystals** seen in coronary arteries (6 out of 55 explanted hearts)→suggests possible **local crystal-driven inflammation** in coronaries
- **Coronary Artery Imaging Studies**
 - Dual-energy CT shows MSU deposits in coronary arteries:
 - Found in ~86% of gout patients vs. 15% of non-gout controls.
 - Post-mortem optical studies confirmed MSU crystals in gout patients' coronary plaques (≈ 12 crystals/mm vs. 0.6 in controls).
 - Limitations: Possible CT artifacts (beam-hardening, partial-volume effects).
- **Renal US studies:** MSU deposits in renal medulla → “**gouty nephropathy**” pattern in ~30–40% of severe gout patients



TREAT-TO-TARGET URATE-LOWERING TREATMENT AND CARDIOVASCULAR OUTCOMES IN PATIENTS WITH GOUT

In this new user cohort study of 109 504 patients with gout who initiated UTLs

Exposure: Serum urate levels lower than 6 mg/dL with urate-lowering treatment

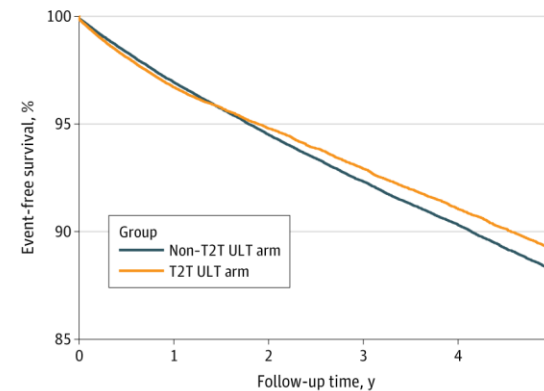
Outcome: Major cardiovascular events

Results: patients achieving the target SU had a significantly lower risk of cardiovascular events than those who did not achieve this urate level.

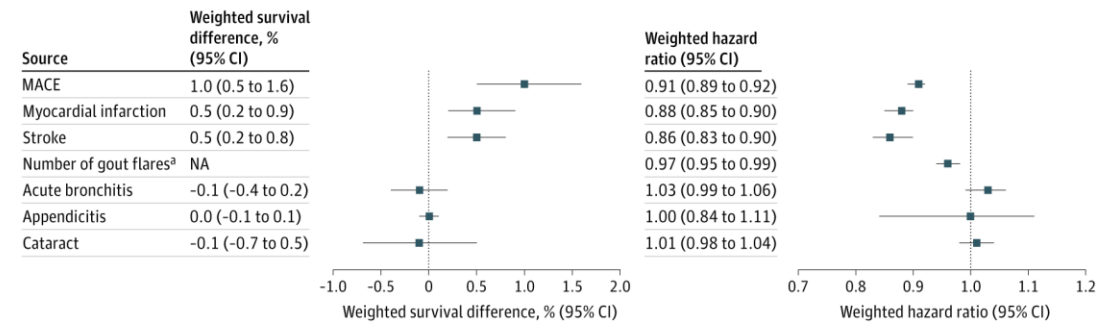
HR: 0.91(CI 0.89 - 0.92)

Meaning Treating gout to target serum urate level (ie, <6 mg/dL) was associated with a reduced risk of cardiovascular events.

A Weighted event-free survival for MACE



B Absolute and relative risk of MACE and control outcomes



RENAL SAFETY AND OUTCOMES OF ULT

Emulated non-inferiority trial of 14,792 patients with gout and chronic kidney disease stage 3 initiating urate-lowering therapy (ULT)

Exposure: serum urate (SU) level <6 mg/dL (0.36 mmol/L).

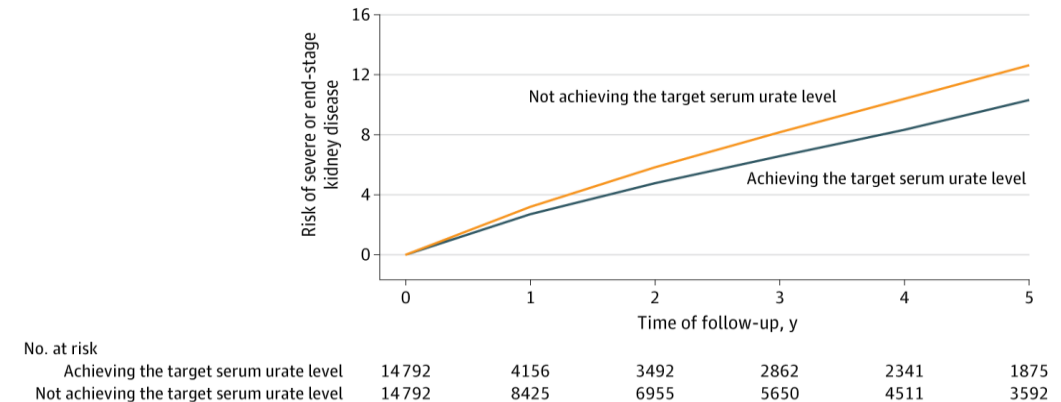
Outcome: progression to chronic kidney disease stage ≥ 4 , kidney transplant, or dialysis.

Results: patients achieving the target SU had a significantly lower 5-year risk of severe chronic kidney disease.

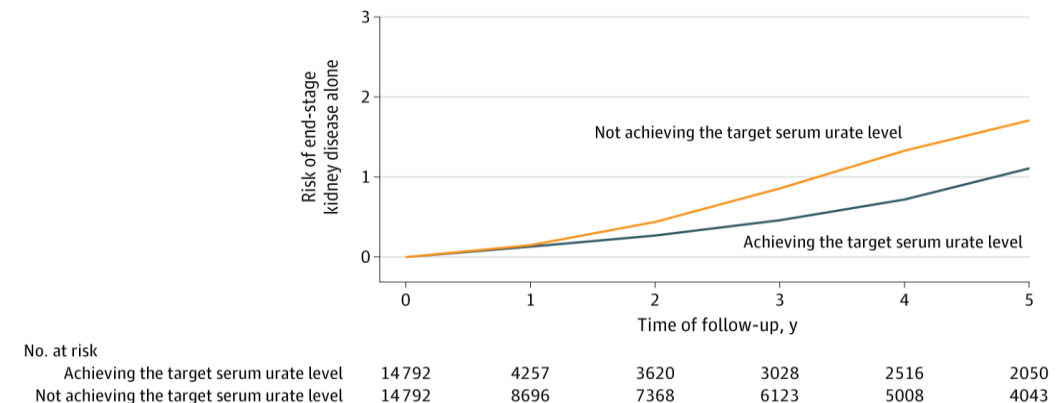
HR 0.89 (95% CI 0.80–0.98)

Meaning Treating gout to target serum urate level (ie, <6 mg/dL) was associated with a reduced risk of progression of CKD

A Severe or end-stage kidney disease

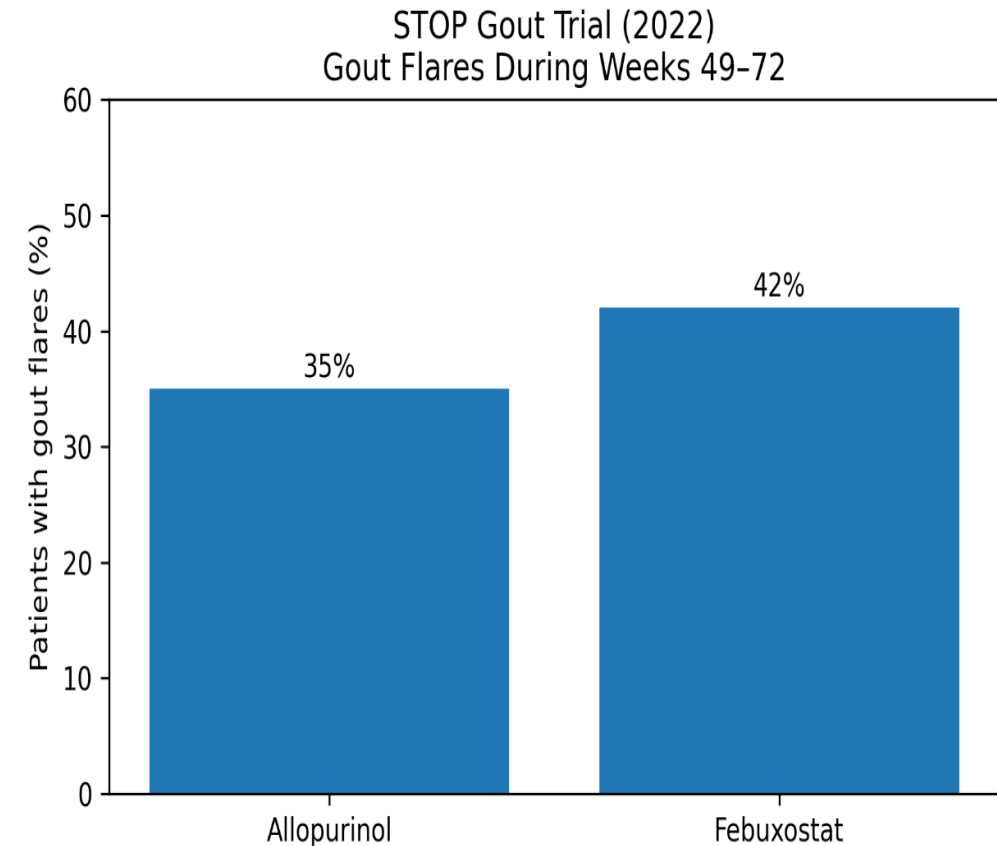


B End-stage kidney disease alone



THE STOP GOUT TRIAL: TREAT-TO-TARGET WORKS

- Multicenter, randomized trial comparing allopurinol versus febuxostat using a treat-to-target (T2T) strategy.
- Dose titration was performed to achieve a serum urate (sUA) target <6 mg/dL.
- **Results:** approximately 80% of participants in both groups achieved the target sUA level.
- During the final observation phase (weeks 49–72), patients treated with ULTs had fewer gout flares:
 - Allopurinol: 35%
 - Febuxostat: 42%
- **Key Message:** a T2T strategy is highly effective regardless of the specific urate-lowering medication used, although allopurinol may be associated with fewer late flares.



CURRENT AMERICAN COLLEGE OF RHEUMATOLOGY (ACR 2020) GUIDELINES FOR GOUT

- Current evidence does **not** support routine ULT solely for asymptomatic hyperuricemia
- Initiate ULT for patients with:
 - ≥ 2 gout flares/year
 - ≥ 1 subcutaneous tophus
 - Radiographic damage attributable to gout
- Consider ULT after a first flare if high-risk features are present:
 - Serum urate ≥ 9 mg/dL
 - CKD stage ≥ 3
 - History of urolithiasis
- Allopurinol is the preferred first-line ULT for all patients, including those with CKD
- Start low and titrate slowly
- Use a treat-to-target strategy with dose escalation to achieve:
 - Serum urate < 6 mg/dL
 - Consider < 5 mg/dL for severe or tophaceous gout



CONT.

- Provide flare prophylaxis when starting ULT for at least 3–6 months:
 - Colchicine
 - NSAID
 - Low-dose prednisone/prednisolone
 - IL-1 inhibitors in selective patients
- Test for HLA-B*58:01 before starting allopurinol in:
 - Southeast Asian ancestry
 - African American
- Avoid stopping ULT during flares; continue and treat the flare concurrently
- Reserve Pegloticase for severe refractory gout
- Conditional recommendation for weight loss for overweight/Obese patients



INTEGRATED TREATMENT STRATEGY

- **Treat-to-Target Urate Lowering:**
 - ULTs: Xanthine oxidase inhibitors, uricosurics, or biologics to dissolve crystals
 - Flare prophylaxis
- **Anti-inflammatory modulation:**
 - Colchicine or IL-1 blockade (e.g., anakinra, canakinumab) - more data needed in gout patients for preventive role for CKM comorbidities
- **Metabolic Interventions:**
 - Lifestyle interventions (diet, exercise, hydration, limiting ETOH use etc.)
 - Weight loss (GLP-1 receptor agonists), insulin sensitization, lipid optimization, and renal protection
 - Use of CKM-friendly urate-lowering agents: SGLT2 inhibitors, losartan, fenofibrate, calcium channel blockers



SGL-2 INHIBITORS

- **Mechanism of Action**

- Block sodium–glucose cotransporter-2 (SGLT-2) in the renal proximal tubule → ↑ urinary glucose excretion.
- Glycosuria increases competition with urate for GLUT9 transporters → ↓ **urate reabsorption** → **lower serum urate levels**.

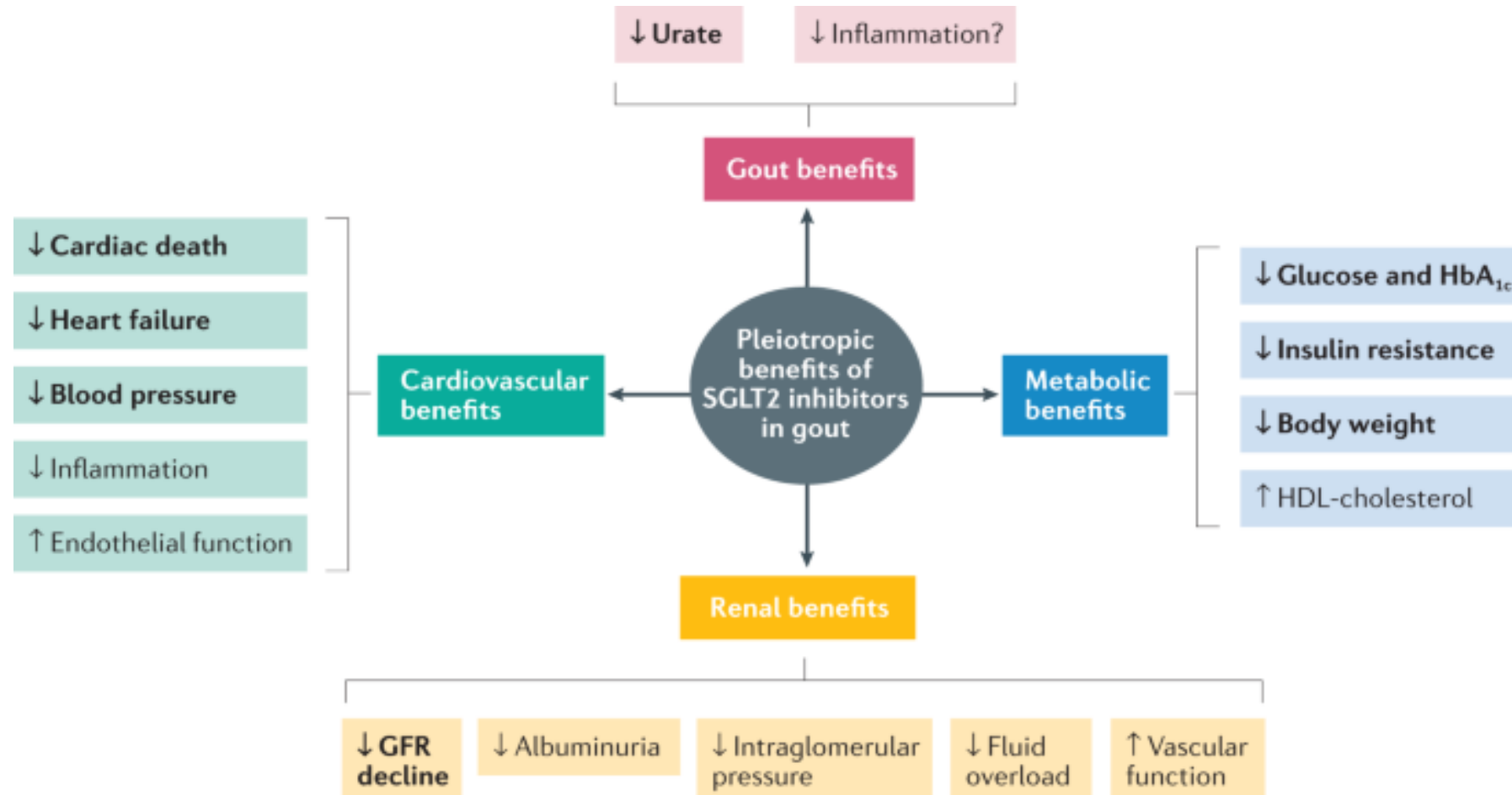
- Additional side benefits: mild diuresis, reduced intraglomerular pressure, improved endothelial and inflammatory profiles

- **Evidence Beyond Diabetes**

- Robust RCT data show benefits independent of diabetes status:
 - ↓ **Heart failure hospitalization** (EMPEROR-Reduced, DAPA-HF).
 - ↓ **CKD progression** and **CV death** (DAPA-CKD, EMPA-Kidney).
- FDA-approved for **heart failure and CKD** even in non-diabetic patients

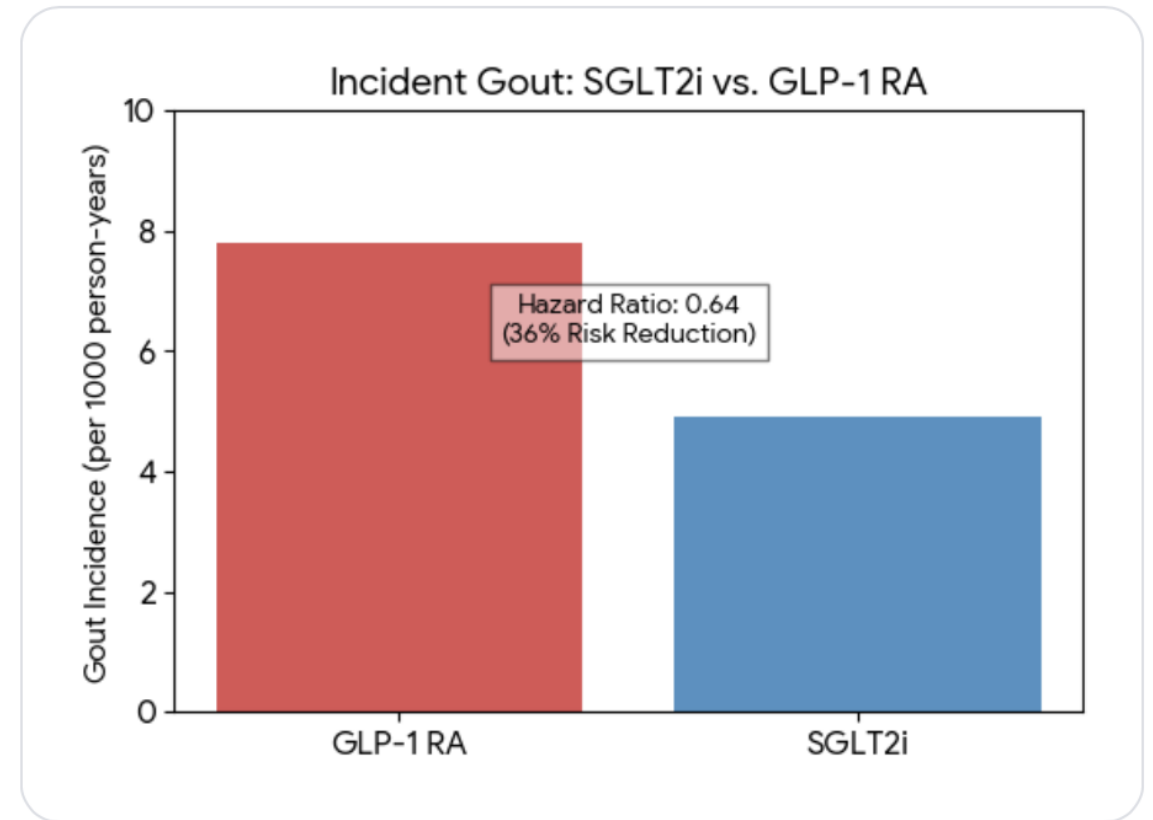


SGL-2 INHIBITORS

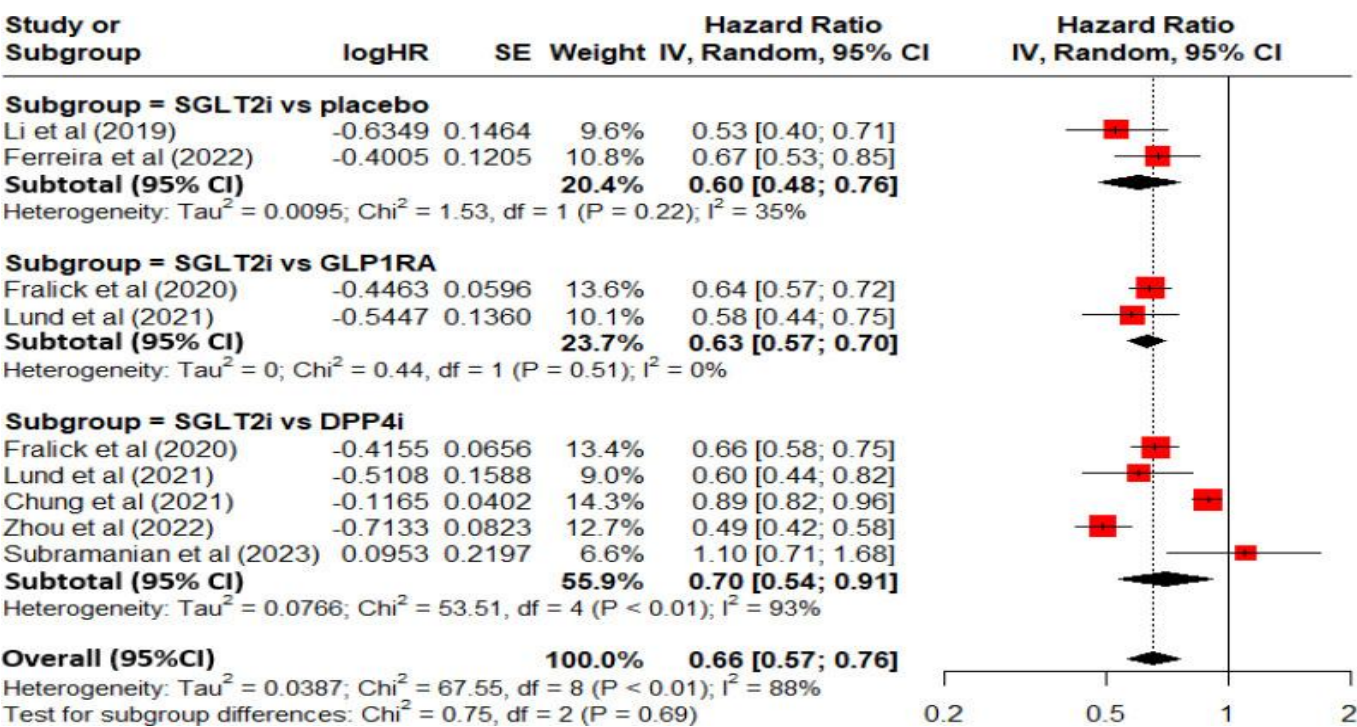


SGL-2 INHIBITORS AND INCIDENT GOUT

- Population: ~300,000 adults with Type 2 Diabetes (T2D) initiating either an SGLT2 inhibitor or a GLP-1 receptor agonist
- **Key Finding:** SGLT2 inhibitor use was associated with a **36% lower risk** of incident gout compared to GLP-1 agonists
- **Impact:** Established SGLT2 inhibitors as a potentially protective class against the development of gout in high-risk diabetic patients



SGL-2 INHIBITORS USE AND THE RISK OF GOUT: A SYSTEMATIC REVIEW AND META-ANALYSIS

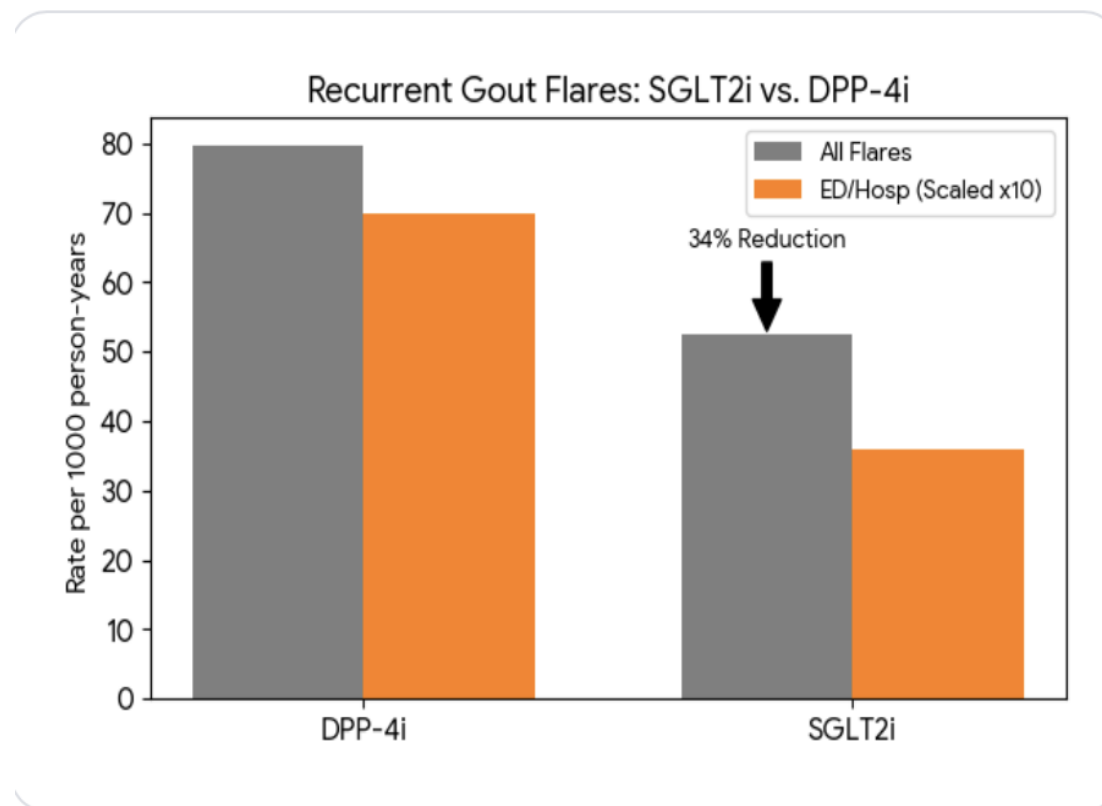


Forest plot for total and subgroup analyses revealing the effect of SGLT2i on the development of gout among patients with T2DM as comparing with placebo or other anti-diabetic drugs.

34% decreased risk of developing gout for SGLT2i use as comparing with non-use of SGLT2i among patients with T2DM

SGLT-2 INHIBITORS AND RECURRENT GOUT FLARES

- **Population:** Patients with **pre-existing gout** and **T2D**. New user cohort study.
- **Key Finding:** SGLT2 inhibitors reduced the rate of recurrent gout flares by **34%** and gout-related emergency department (ED) visits/hospitalizations by **48%** compared to DPP-4 inhibitors.
- **Impact:** Demonstrated that these drugs are effective for secondary prevention (reducing flares) in patients already diagnosed with gout.



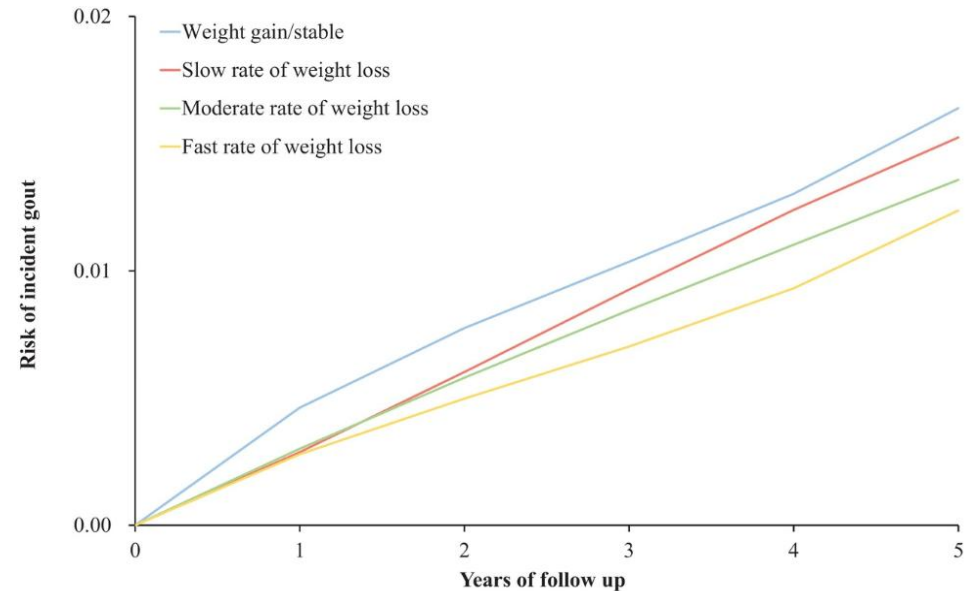
WEIGHT LOSS FOR GOUT

- **Obesity affects >50%** of individuals with gout
- **Strong positive association** between excess adiposity and serum urate (SU)/gout risk
- At the population level, obesity is the **most important modifiable risk factor** for incident gout and hyperuricemia
- **Population attributable risk:** 31% for incident gout and 44% for hyperuricemia
- **2020 ACR Gout Management Guidelines:**
 - “For overweight/obese patients with gout, regardless of disease activity, we *conditionally recommend weight loss.*”
- **Multiple studies** show that interventions with **larger and faster weight loss** produce the **greatest gout-related benefits** (↓ SU levels, ↓ flare frequency)



WEIGHT LOSS PREVENTS GOUT

- Emulated trial of 131,000 overweight/obese adults without gout newly treated with anti-obesity medications (e.g., orlistat).
- **Data source:** primary and secondary care data from the UK Health Improvement Network.
- **Exposure:** amount of weight loss achieved during the first year.
- **Outcome:** incident gout diagnosis (primary outcome) and gout flare rates (secondary outcome)
- **Results:** greater weight loss was associated with progressively lower risk of developing gout



1-Year Weight Loss	Hazard Ratio for Incident Gout
Slow (2–5%)	0.91 (0.81–1.01)
Moderate (5–10%)	0.82 (0.72–0.92)
High (≥10%)	0.73 (0.62–0.86)

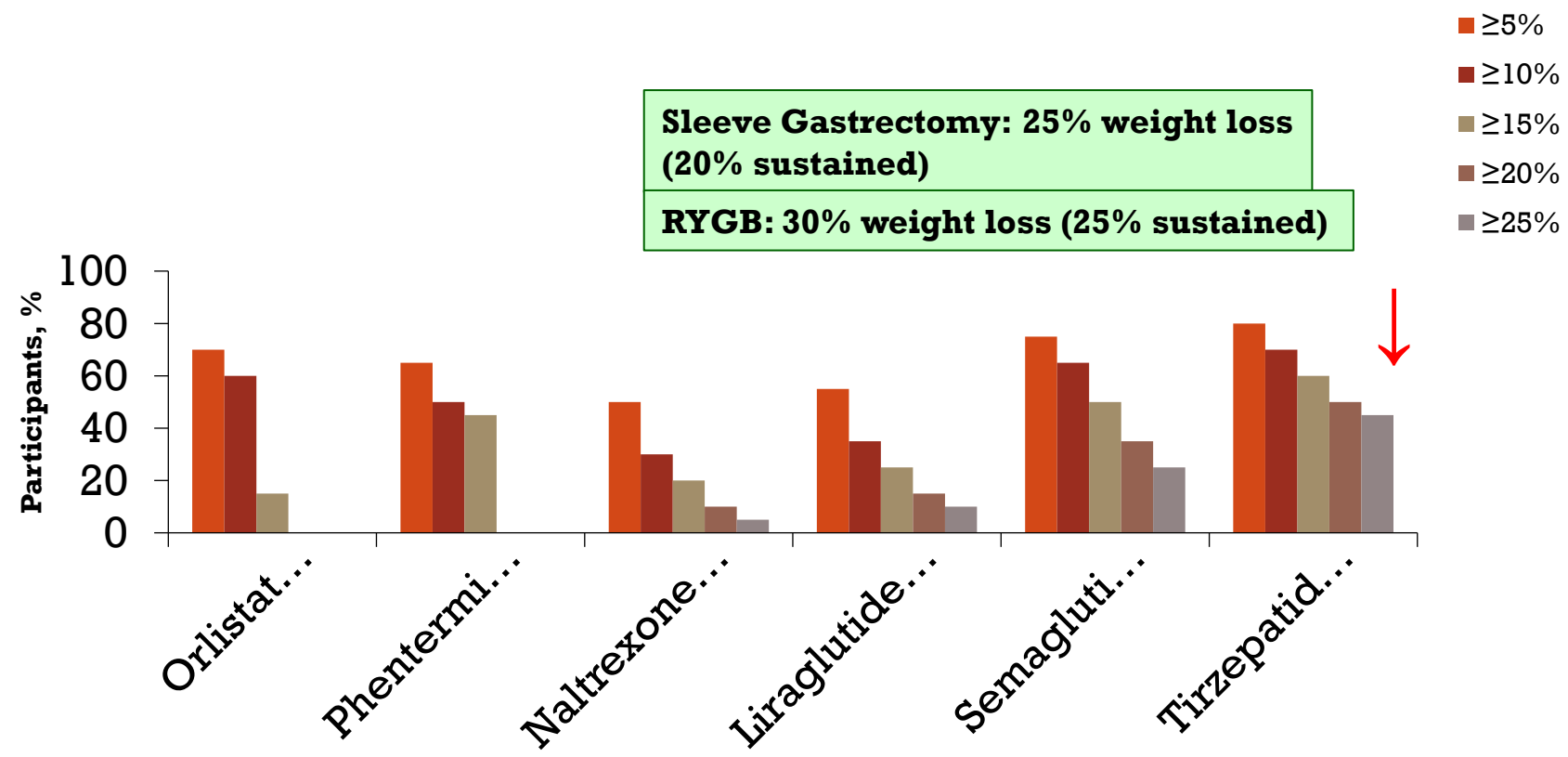


NEWER ANTI-OBESITY MEDICATIONS (AOM)

- **Two main novel AOMs currently on the market**
 - **Semaglutide** – glucagon-like peptide-1 receptor agonist (**GLP-1 RA**)
 - **Tirzepatide** – glucose-dependent insulintropic polypeptide (**GIP**) / GLP-1 RA (“dual agonist”)
 - *Additional agents in the pipeline...*
- **Primary mechanism of action:**
 - Enhance glucose-dependent insulin secretion, inhibit glucagon release, and slow gastric emptying
 - Also act centrally on the **brain–gut axis** to suppress appetite and increase satiety
- **Stronger potency as glucose-lowering medications**
 - Expected **A1c decrease**: 1.5–2.0% with monotherapy
- **Robust RCT evidence for a range of obesity-related conditions**, including:
 - **ASCVD** (atherosclerotic cardiovascular disease)
 - **CKD** – diabetic and nondiabetic
 - **HFpEF** (heart failure with preserved EF)
 - **OSA** (obstructive sleep apnea)
 - **MASLD** (metabolic dysfunction–associated steatotic liver disease)
 - **Osteoarthritis**



MAGNITUDE OF WEIGHT LOSS WITH NOVEL AOM



NOVEL ANTI-OBESITY MEDICATIONS AND GOUT: EMERGING EVIDENCE

- **Data so far are limited:**

- Observational studies comparing *SGLT2 inhibitors* and *GLP-1 receptor agonists* suggest **lower risk of incident and recurrent gout** with SGLT2 inhibitors.
- These analyses were conducted primarily in **patients with type 2 diabetes**, not established gout cohorts

- **Older vs. newer AOMs:**

- *Older agents* (e.g., orlistat, phentermine-topiramate, naltrexone-bupropion) mainly act via caloric restriction or appetite control
- *Newer GLP-1 and dual GIP/GLP-1 agonists* (e.g., semaglutide, tirzepatide) exhibit **broad cardiometabolic and anti-inflammatory actions**

- **Inflammatory modulation:**

- Novel AOMs are reported to **reduce IL-6, TNF- α , and IL-1 β** levels
- Potentially **CV-protective effects independent of weight loss**, though **additional mechanistic studies are needed**



NOVEL ANTI-OBESITY MEDICATIONS AND SERUM URIC ACID IN GOUT

- Obesity affects >50% of gout patients — key modifiable risk factor.
- Retrospective EHR study (Mass General Brigham): 22 gout patients with hyperuricemia (SU ≥ 6 mg/dL) on semaglutide or tirzepatide.
- Average treatment duration ~258 days; mean BMI 39.3 kg/m²; 77% male.
- Each 1 kg weight loss $\rightarrow$ $\downarrow$ serum urate by 0.13 mg/dL; each BMI unit $\downarrow$ $\rightarrow$ 0.41 mg/dL reduction.
- Weight loss effect on SU:
 - 5–10% loss $\rightarrow$ -0.73 mg/dL
 - >10% loss $\rightarrow$ -2.36 mg/dL ($p < 0.001$).
- Conclusion: AOM-induced weight loss significantly lowers serum urate in gout — suggesting potential role as multi-purpose therapy addressing obesity, metabolic disease, and gout



CONCLUSION

- Gout is more than just arthritis
 - Consider it a cardio-renal-metabolic disease risk signal
- Most gout related morbidity is largely preventable
 - Undertreatment and lack of titration are the main problems
 - Treat-to-target urate <6 mg/dL improves gout and CKM outcomes
 - Continue ULTs during flares
- Management is longitudinal, not episodic
 - Structural follow up and patient education is the key
- Newer therapies
 - SGLT2 inhibitors reduces gout risk
 - GLP-1 agonist very effective for weight loss and may have gout benefits
- Multidisciplinary patient centered care
 - Refer difficult-to-control gout to rheumatology
 - Comorbidity optimization should be integral part of gout care
 - Co-management with primary care, nephrology, cardiology, and weight loss services can be beneficial



WHEN SHOULD YOU REFER TO THE RHEUMATOLOGY?

Manage in Primary Care (with Treat-to-Target)

- Typical monoarticular gout
- ≤ 1 flare/year
- Responds to colchicine/NSAID/steroids
- Starting or titrating allopurinol to goal
- No tophi and eGFR ≥ 60

Refer to Gout Clinic

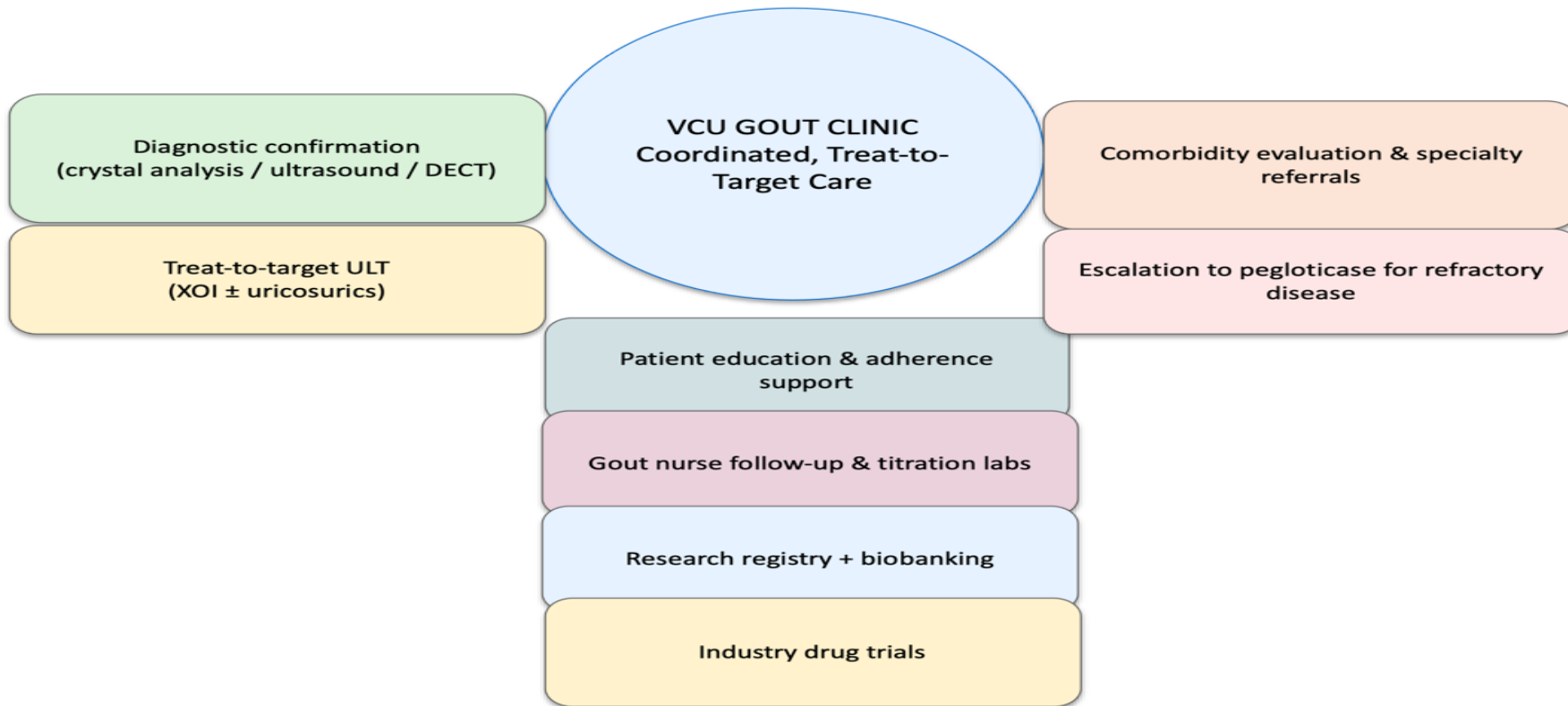
- Uncertain diagnosis or atypical presentation
- ≥ 2 flares/year or polyarticular disease
- Tophi or erosive disease
- Serum urate persistently >9 mg/dL
- CKD stage 3–5 or transplant patients
- Allopurinol intolerance or failure to reach goal
- Considering pegloticase (refractory gout)

How to Refer

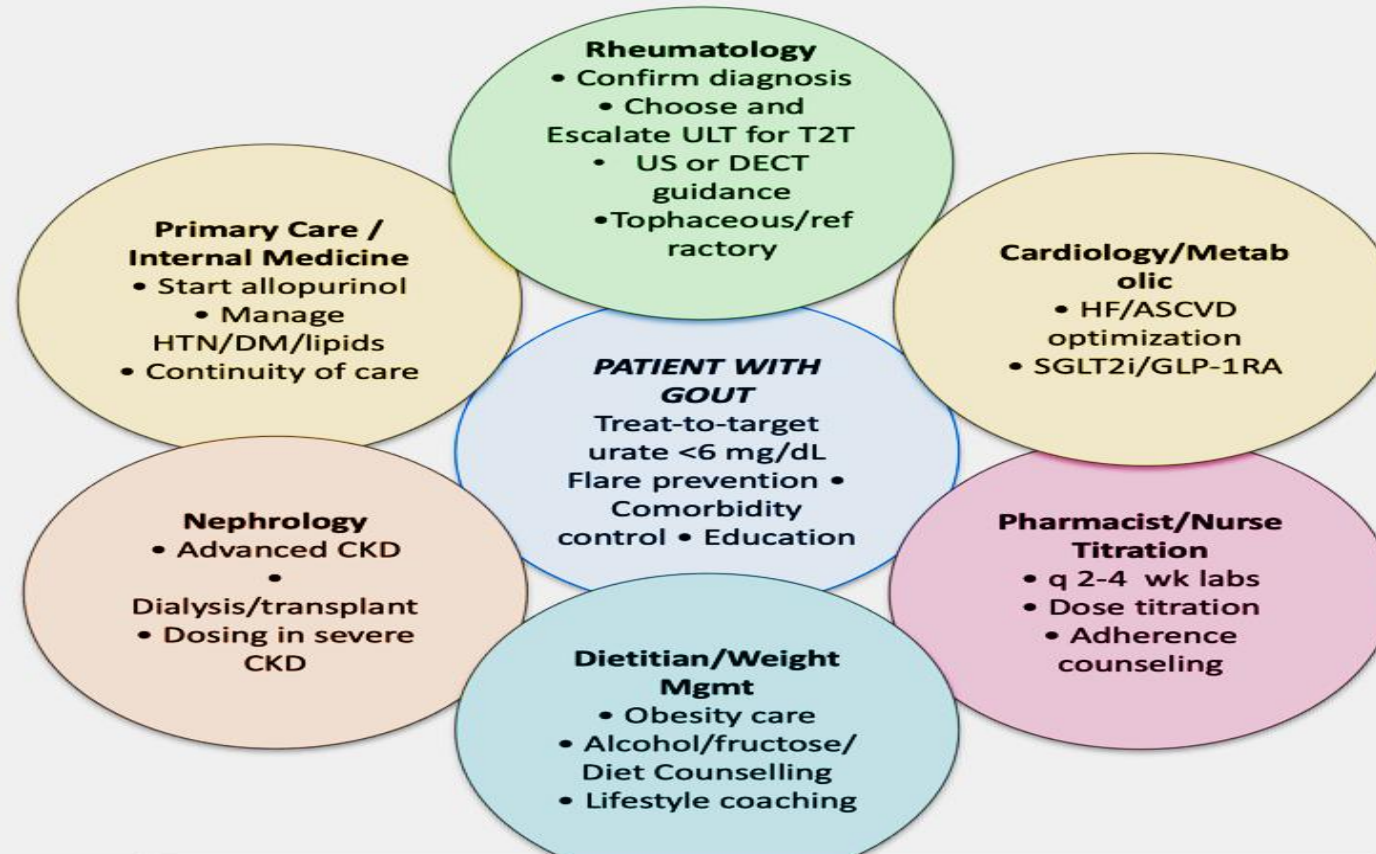
- Place Rheumatology referral: 'Gout Clinic'
- Please order labs (urate, CMP, CBC)
- Do NOT stop allopurinol before visit



VCU SPECIALTY GOUT CLINIC CARE MODEL



MULTI-DISCIPLINARY GOUT CARE MODEL



System Tools: EMR order set • Standing titration protocol • Patient education • Registry tracking



FUTURE VISION-STEP TOWARDS PRECISION HEALTH

- **Prospective clinical registry of gout patients receiving standardized care at VCU**
- **Data Being collected**
 - Demographics
 - Gout activity scores
 - Comorbidities
 - Serum urate trends
 - Medication use and adherence
 - Imaging findings (including ultrasound tophi burden)
 - Renal and CV outcomes
 - Quality of life measures
- **Biobanking** – blood samples collected at base line and annually
- **Why this matters:**
 - Allows quality improvement + real-world outcome tracking
 - Opportunity to assess biomarkers for gout flare and associate CMK risk
 - Use AI to combine clinical phenotyping with multi omic analysis to generate predictive risk models to find high-risk patients for multidisciplinary care



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THANK YOU



Questions ????

