Gout and the Kidneys: A Complicated Cocktail

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PRESENTER DISCLOSURE

- Consulting for
  - Amgen
  - Sobi
- Advisory board chair: UCB
- Advisory board member: Abbvie, J&J, UCB
- Speaker: Novartis

LEARNING OBJECTIVES

- Gout – the clinical basics
- Bidirectional relationship between gout and CKD
- Selecting safe and effective treatments for acute gout flares in CKD
- Initiating and titrating ULT in gout and CKD
- Recognition of medication related risks and drug interactions unique to CKD
- Implement gout flare prophylaxis safely in moderate-advanced CKD

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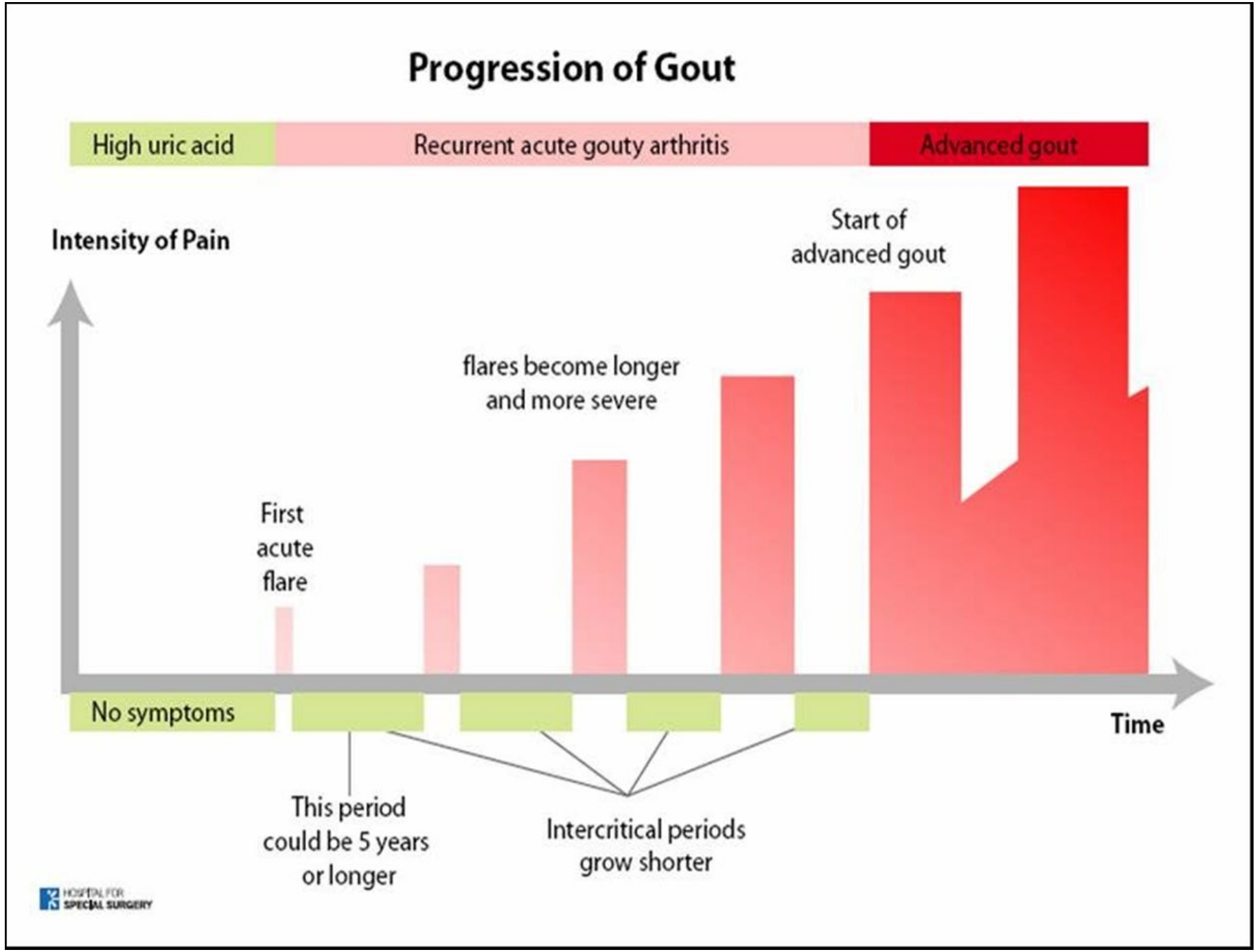
Introduction

- Prevalence of gout is 4% in North America
- Characterised by deposition of MSU crystals, which form in the presence of increased urate concentrations
- Four stages:
  - Hyperuricaemia without evidence of MSU crystal deposition or gout
  - MSU crystal deposition but asymptomatic
  - MSU crystal deposition and acute flares
  - Tophi causing chronic gouty arthritis/ erosions on XR
- Progression from one stage to next is not inevitable

Zhu Y et al. *Arthritis Rheum*. 2011; **63**:3136-3141


Progression of Gout
High uric acid
Recurrent acute gouty arthritis
Advanced gout
Intensity of Pain
Start of advanced gout
flares become longer and more severe
First acute flare
No symptoms
Time
This period could be 5 years or longer
Intercritical periods grow shorter
HOSPITAL FOR SPECIAL SURGERY

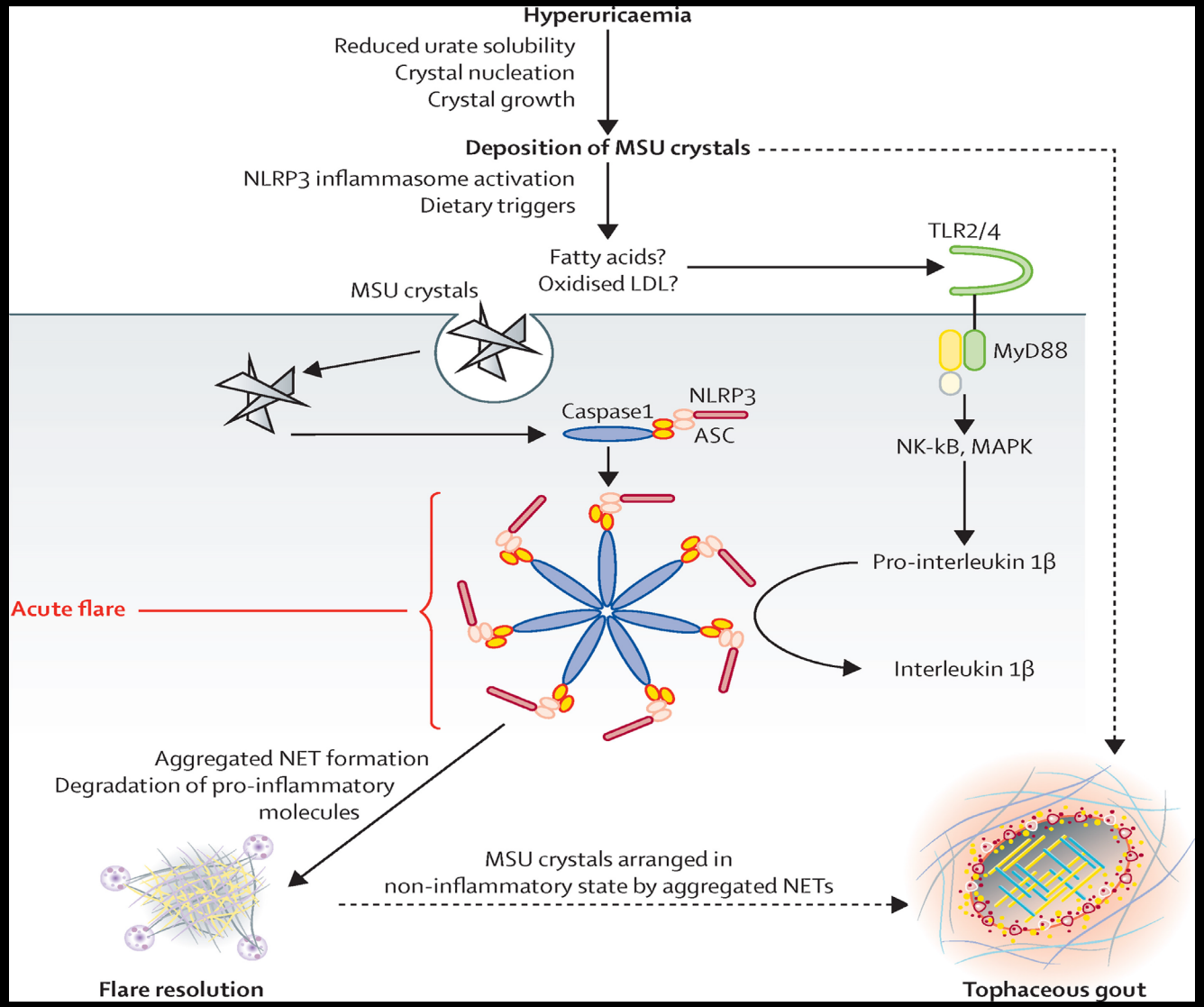
Causes of hyperuricemia

- Genetic mutations causing increased synthesis
- Increased degradation – high cell turn over in psoriasis, haematological malignancy and their treatment
- Increased ingestion – beer, fructose rich beverages, red meat and shellfish
- Decreased fractional excretion via kidneys
- Advancing age (women tend to get raised urate post menopause)

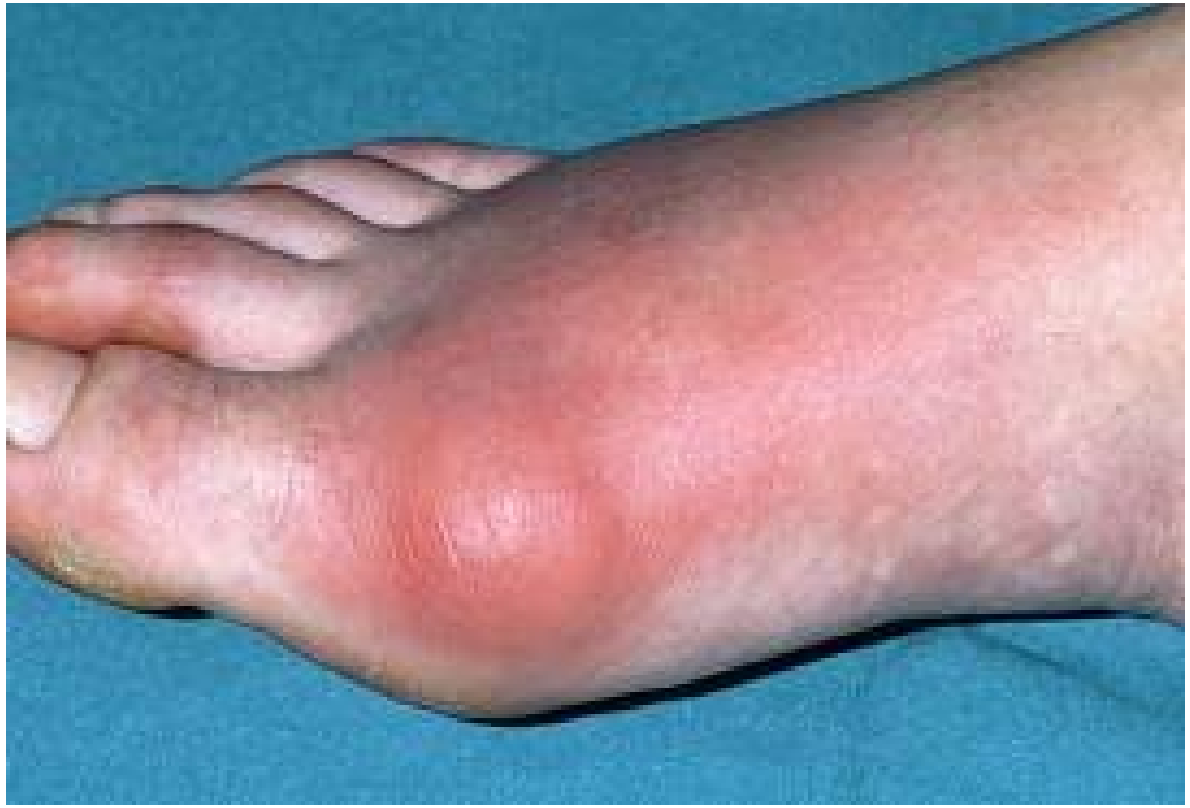
Pathogenesis of gout

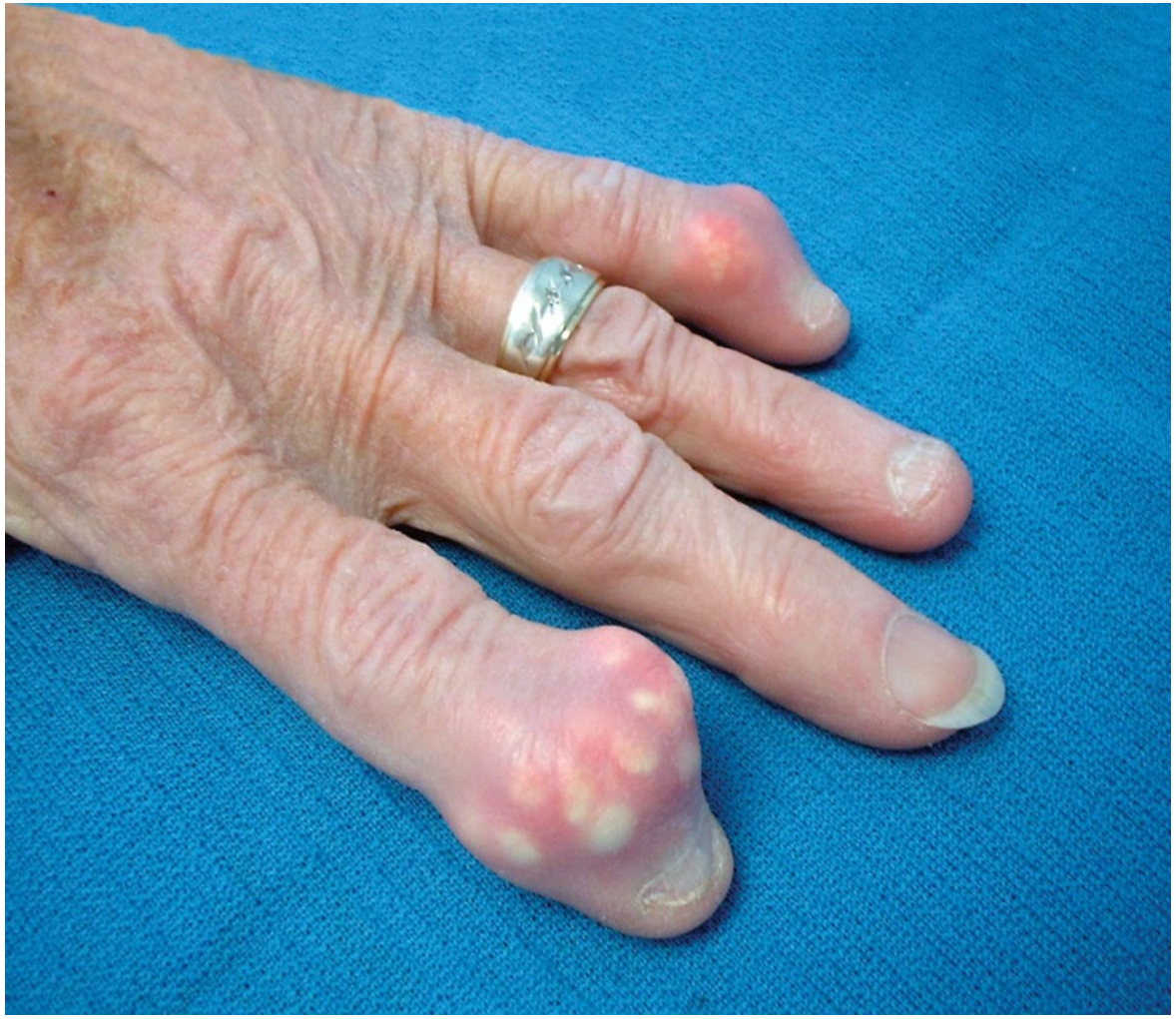
Factors affecting MSU crystal formation:

- Temperature
- Solubility at 360 µmol/L in peripheral joints at 35deg C
- Lower pH
- Increased Salt concentration
- Cartilage matrix components



The Lancet 2016 3882039-2052DOI: (10.1016/S0140-6736(16)00346-9)

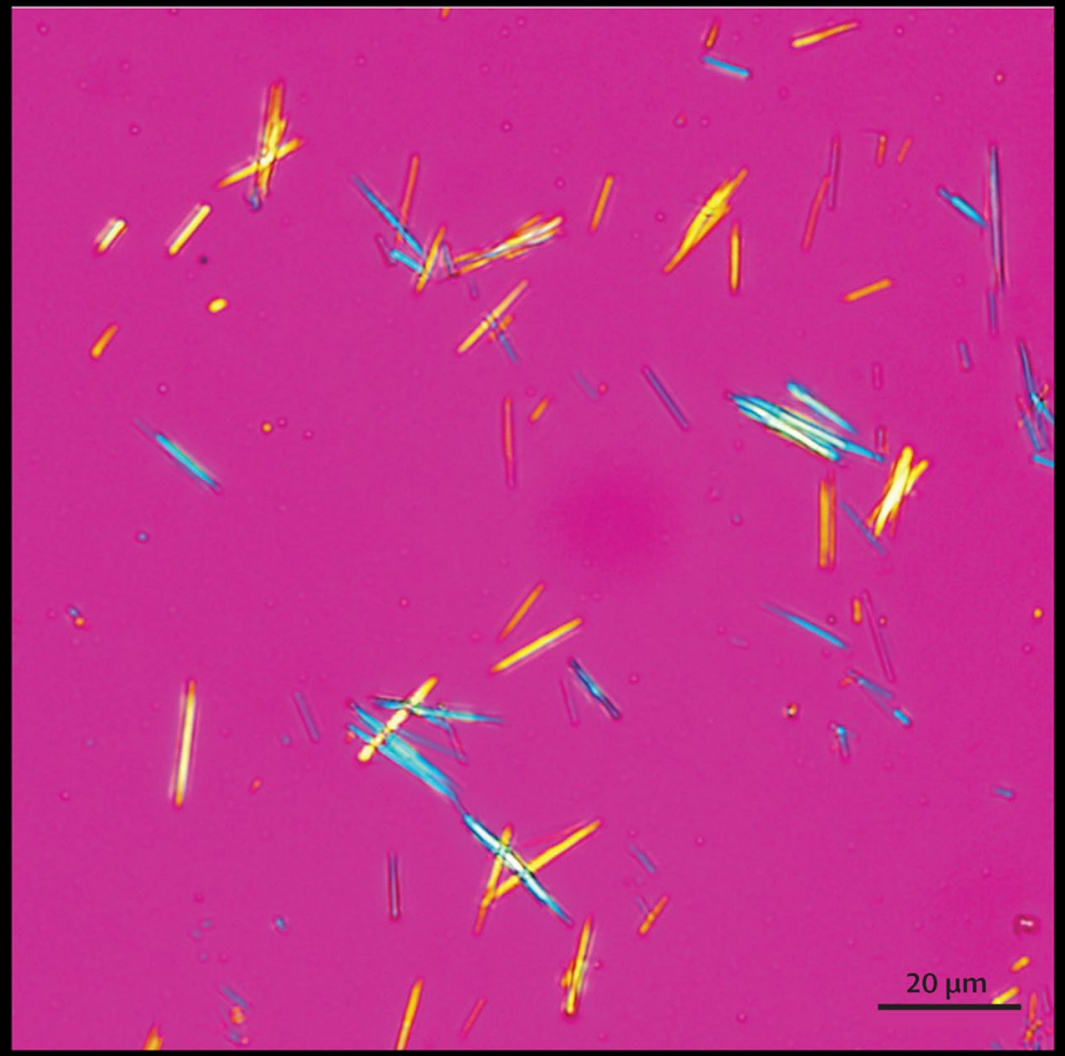




Diagnostic investigations

- Gold standard- arthrocentesis and polarized light microscopy
  - Negatively birefringent needle shaped crystals
  - Synovial fluid during acute flare may appear yellow/ cloudy
  - >50,000 total white cells per mm^3
  - Can get MSU crystals in asymptomatic joints (inter critical period)
- SUA can be high or low during acute flare
- Acute phase reactants CRP/ESR
- Comorbidity screening: lipids, BP, renal function, HbA1c
- XR: punched out periarticular erosions in advanced gout
- US: double contour, starry sky/ snow storm appearance
- DECT: color coded MSU crystal deposits

Gold standard for diagnosis of gout



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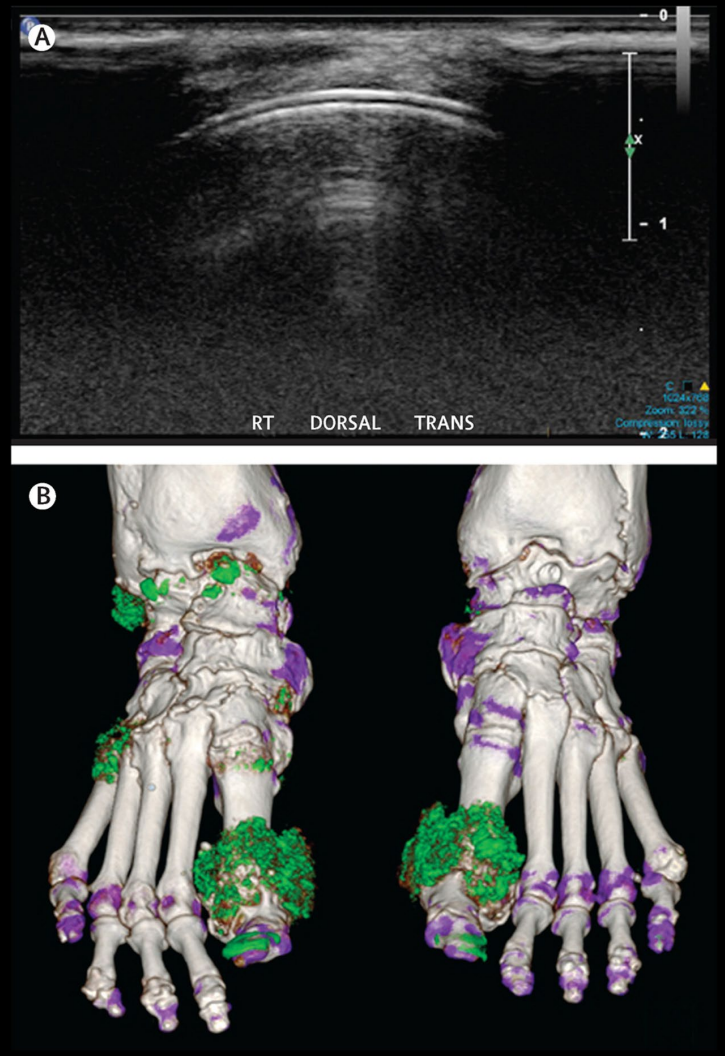
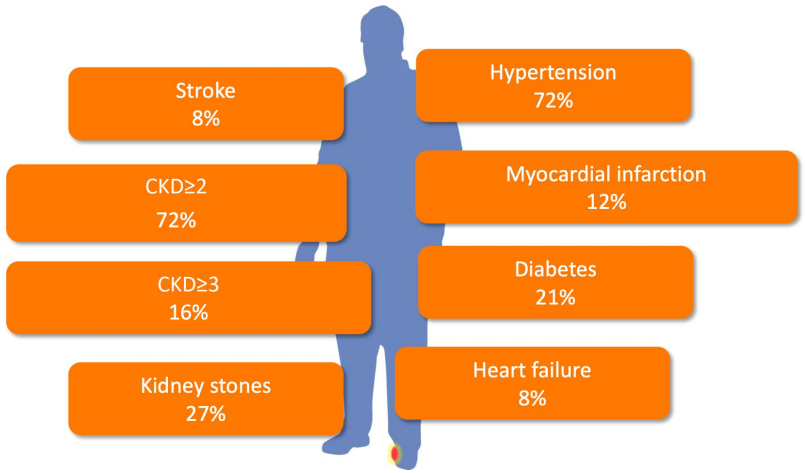
Non invasive methods of diagnosing gout

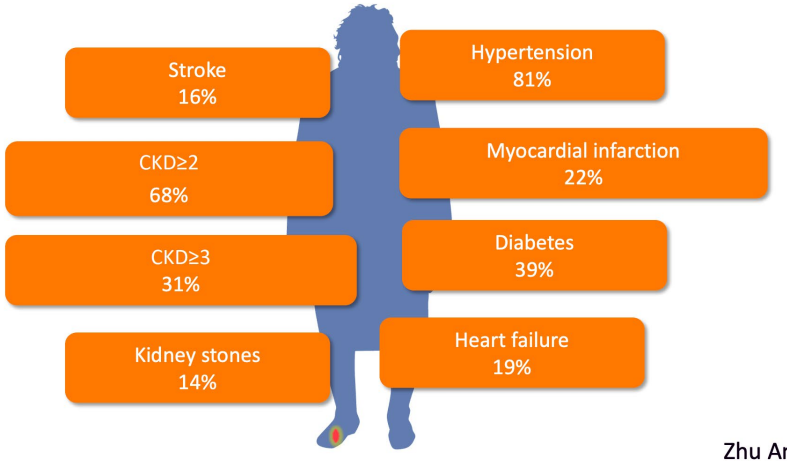


The Lancet 2016 3882039-2052DOI: (10.1016/S0140-6736(16)00346-9)

Men

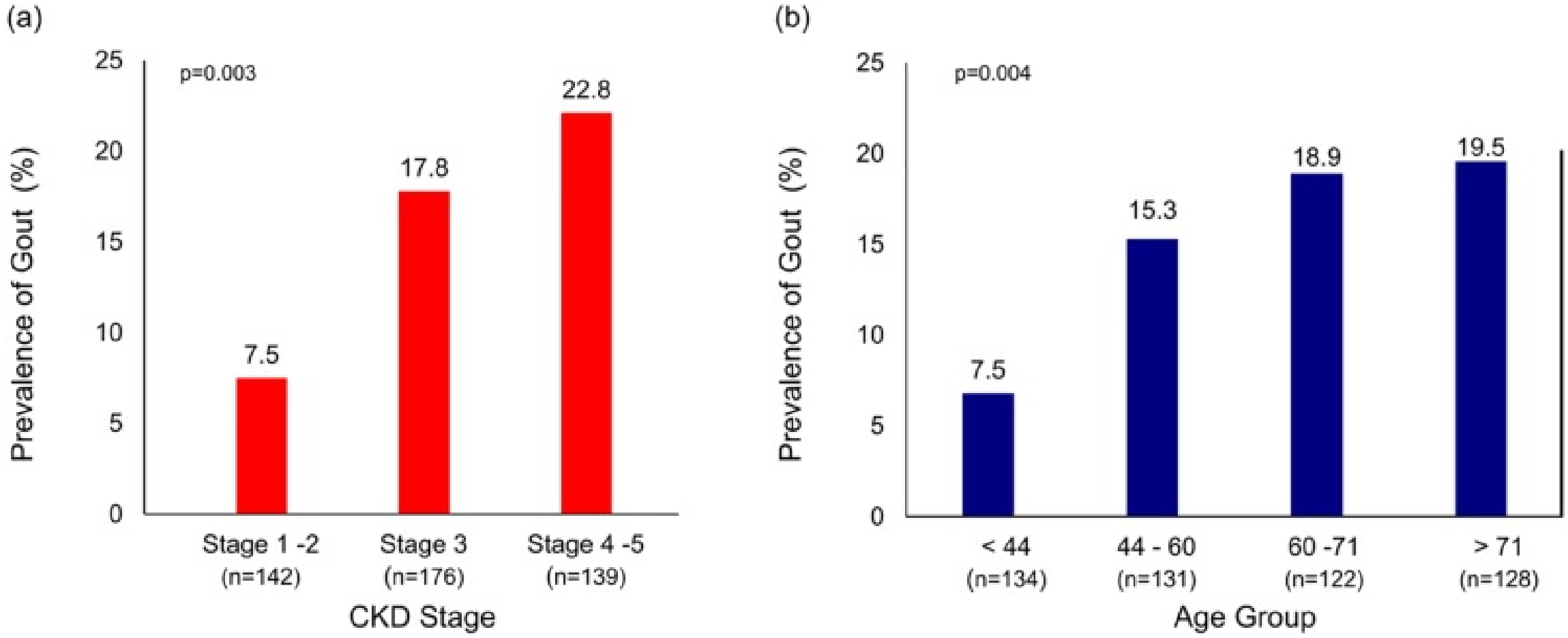


Women



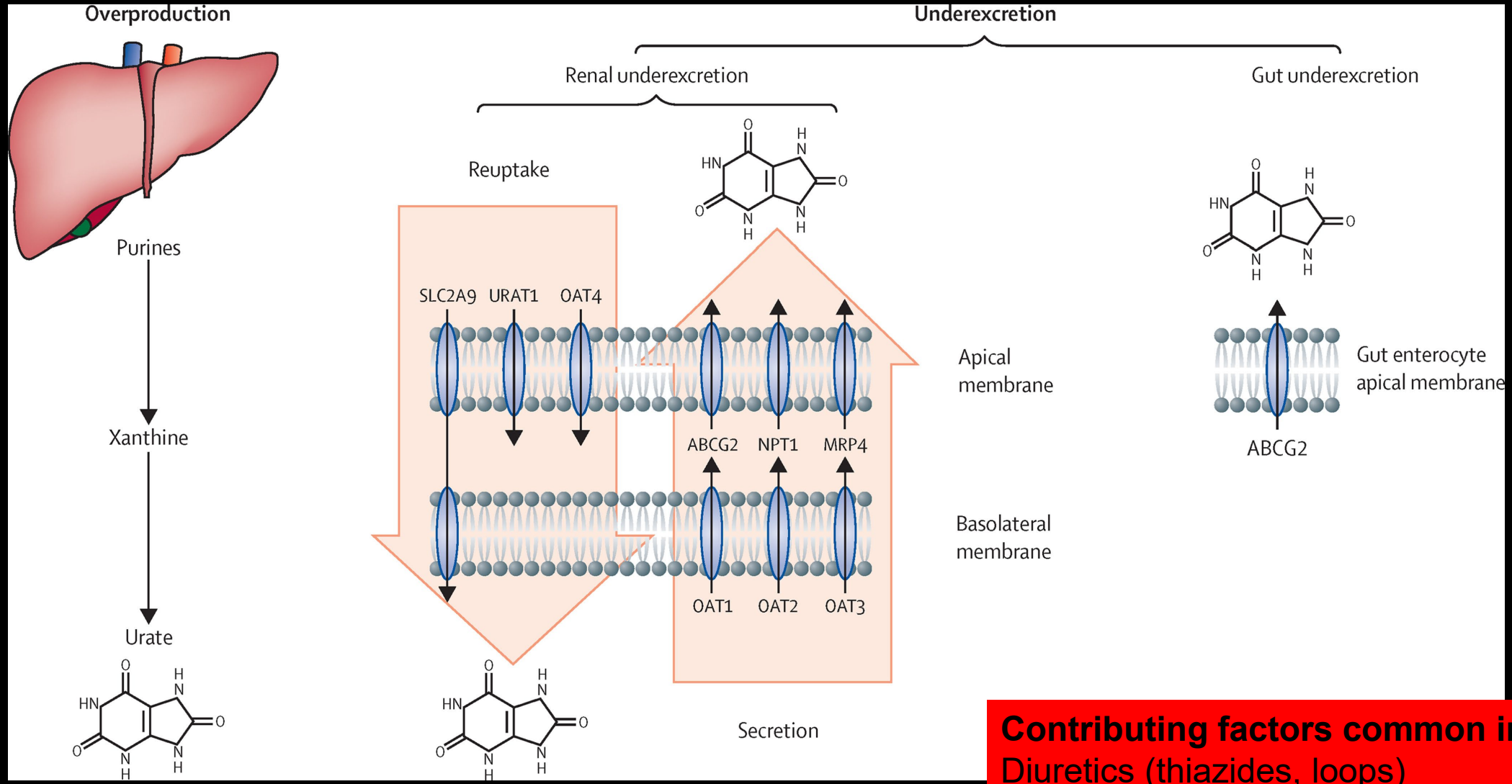
Zhu Am J Med 2012

Prevalence of gout in CKD



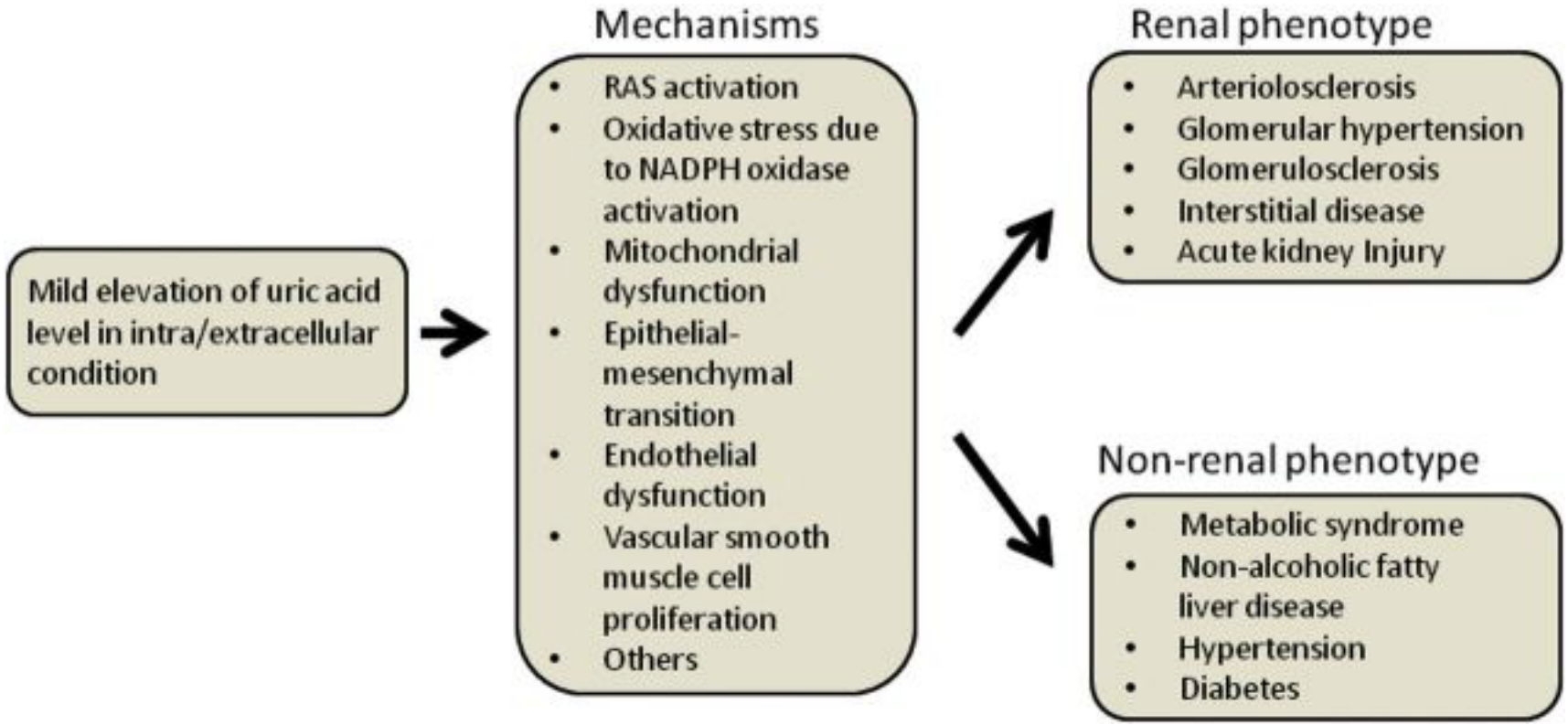
Mohammed E et al. PLoS One. 2019 Jan 25;14(1):e0210487

Renal transport of urate



Contributing factors common in CKD
Diuretics (thiazides, loops)
Insulin resistance / metabolic syndrome
Obesity and hypertension

The Lancet 2016 3882039-2052DOI: (10.1016/S0140-6736(16)00346-9)



Johnson RJ et al. Nephrol Dial Transplant. 2013 Sep;28(9):2221-8.

What makes gout ‘complex’ in the setting of CKD

Chronic recurrent flares

Renal replacement therapy

Renal (or other organ) transplant

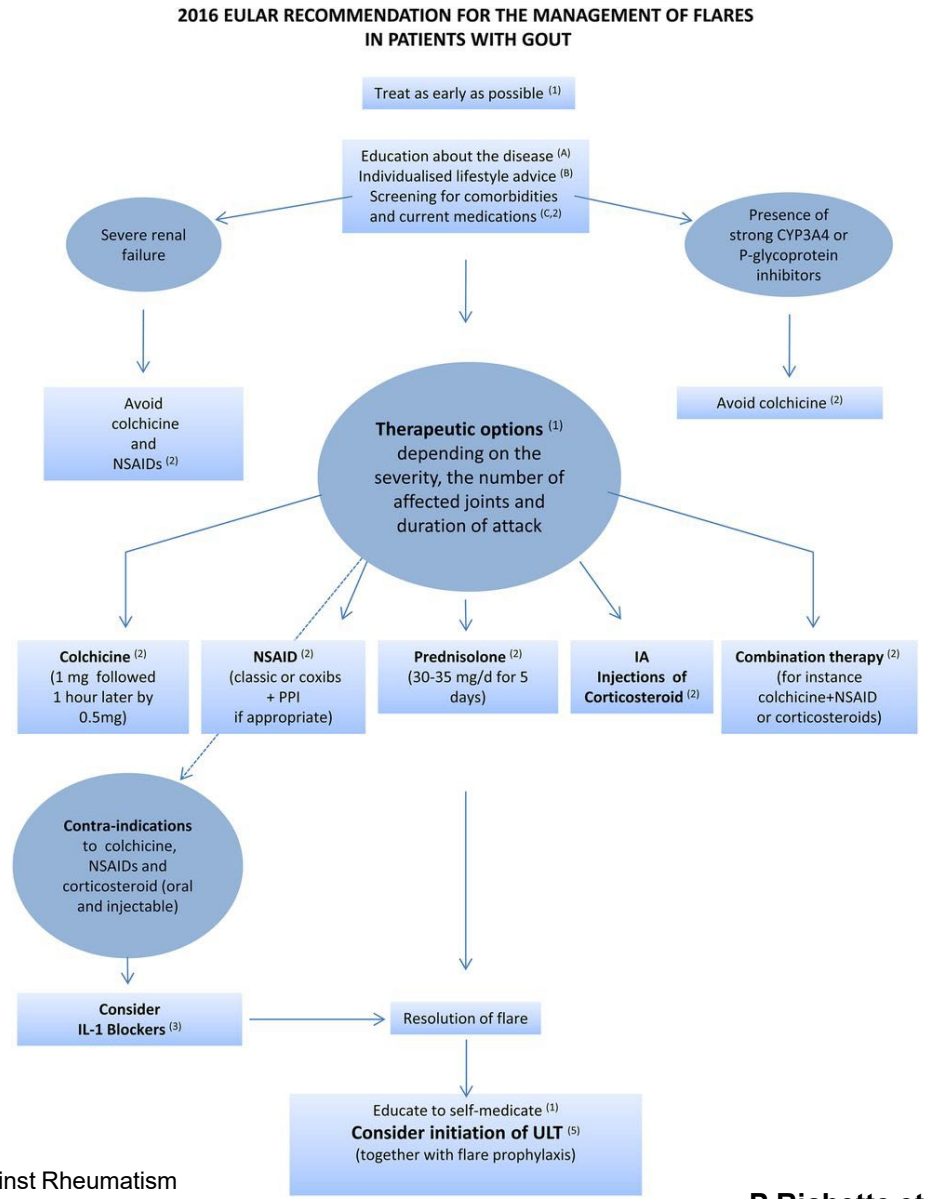
Polypharmacy and drug interactions

Diagnostic uncertainty

Intolerance to standard Urate Lowering Therapy (ULT)

Contraindications to medications for acute gout flares- NSAIDs, colchicine

Management of acute flare according to the European League Against Rheumatism recommendations.



P Richette et al. Ann Rheum Dis 2017;76:29-42

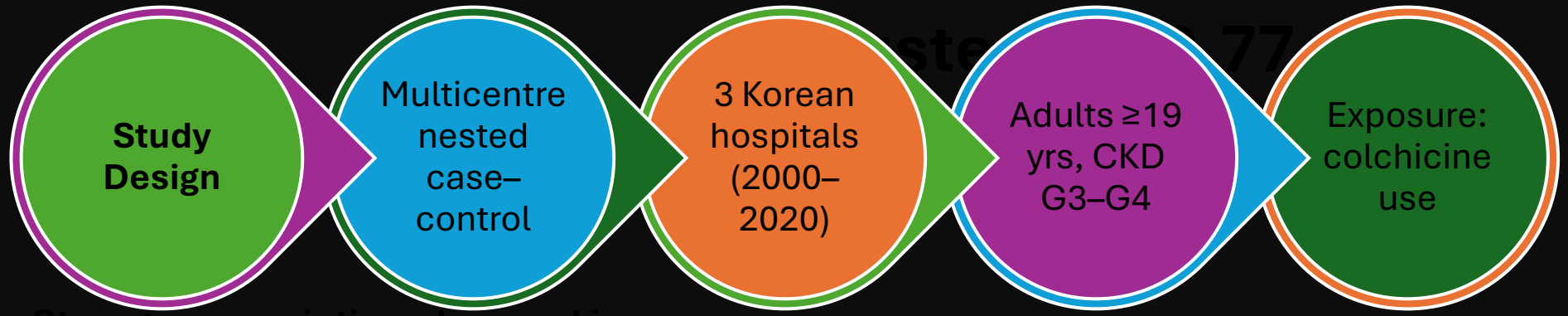
ARD

Colchicine in acute gout treatment in CKD

CKD STAGE	RECOMMENDED COLCHICINE DOSING
eGFR ≥60	~~Standard low-dose: 1.2 mg, then 0.6 mg after 1 hour~~ Colchicine 0.6 mg BID
eGFR 30–59	0.6 mg once; consider no repeat dose
eGFR <30	0.6 mg once only (no repeat)
Dialysis	0.6 mg single dose; do not repeat for ≥14 days

FitzGerald JD et al.2020 American College of Rheumatology Guideline for the Management of Gout.Arthritis Care Res. 2020

Long-term Colchicine Use and CKD Progression



Key Finding

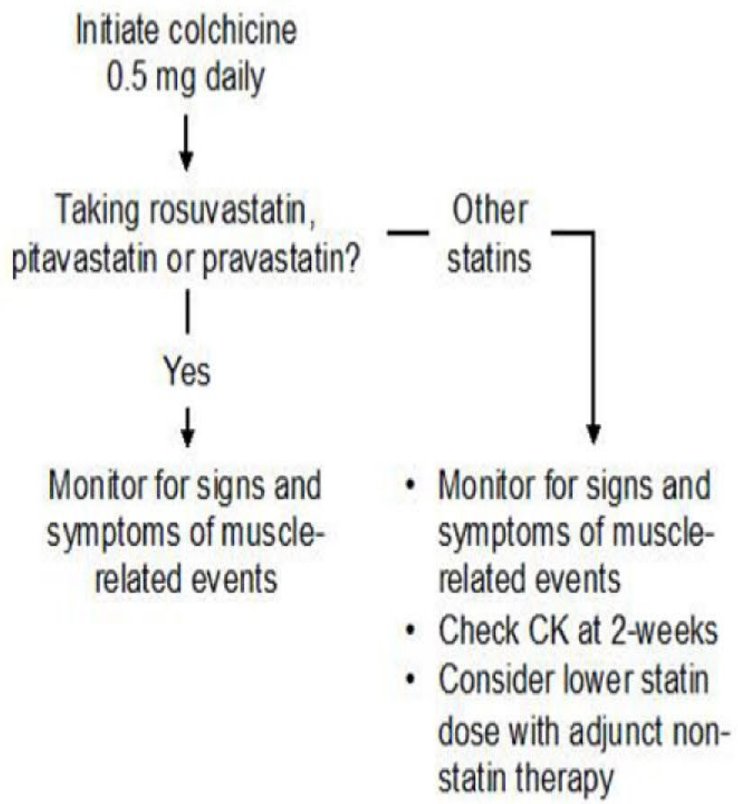
≥90 cumulative colchicine doses

↓ Risk of CKD progression

Adjusted OR 0.77

95% CI 0.61–0.96

Statin-Colchicine Drug-Drug Interaction



Other Colchicine Drug-Drug Interactions

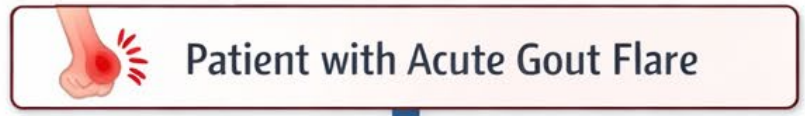
Evaluate*:

- Potential severity and duration of drug-drug interaction
- Potential consequences of stopping colchicine
- Other risk factors for adverse events related to colchicine or interacting drug
- Availability of enhanced monitoring parameters for colchicine and interacting drug

DDI Severity	Duration of Concomitant Therapy: Short (≤14 days)	Duration of Concomitant Therapy: Intermediate-Long (>14 days)
Severe	Hold colchicine	• Stop colchicine and re-evaluate other ASCVD risk reduction options • Consider decreased colchicine dose (off-label, unstudied) or decreased dose of alternative therapy
Minor-Moderate	• Continue colchicine with enhanced monitoring • Can consider holding colchicine	Continue colchicine with enhanced monitoring

Leo F. Buckley. Arteriosclerosis, Thrombosis, and Vascular Biology. Colchicine's Role in Cardiovascular Disease Management, Volume: 44, Issue: 5, Pages: 1031-1041, DOI: (10.1161/ATVBAHA.124.319851)

Acute Gout Flare Management in CKD



Patient with Acute Gout Flare

Assess CKD Stage (eGFR)

CKD 1–2 (eGFR ≥60)

- ✓ **NSAIDs** (standard dose)
- ✓ **Colchicine** 1.2 mg + 0.6 mg
- ✓ **Oral** or **IA** *Corticosteroids*

CKD 3 (eGFR 30–59)

- ✓ **NSAIDs** (cautious use ⚠)
- ✓ **Colchicine** 0.6 mg once
- ✓ **Corticosteroids** (oral or IA)

CKD 4–5 / Dialysis (eGFR <30)

- ✓ **Avoid NSAIDs**
- ✓ **Colchicine** 0.6 mg x 1
  Single dose only
- ✓ **Corticosteroids** (oral or IA)
- ✓ Consider **IL-1 inhibitor**

Supportive Care

- ✓ Rest, joint elevation
- ✓ Acetaminophen (avoid nephrotoxins)
- ✓ Continue Urate-Lowering Therapy

Follow-Up

- Monitor **Renal Function**
- Adjust ULT as Needed

ACR 2020 / EULAR 2016 / KDIGO 2021 / Lancet 2016 / CJASN 2017

Indications for ULT

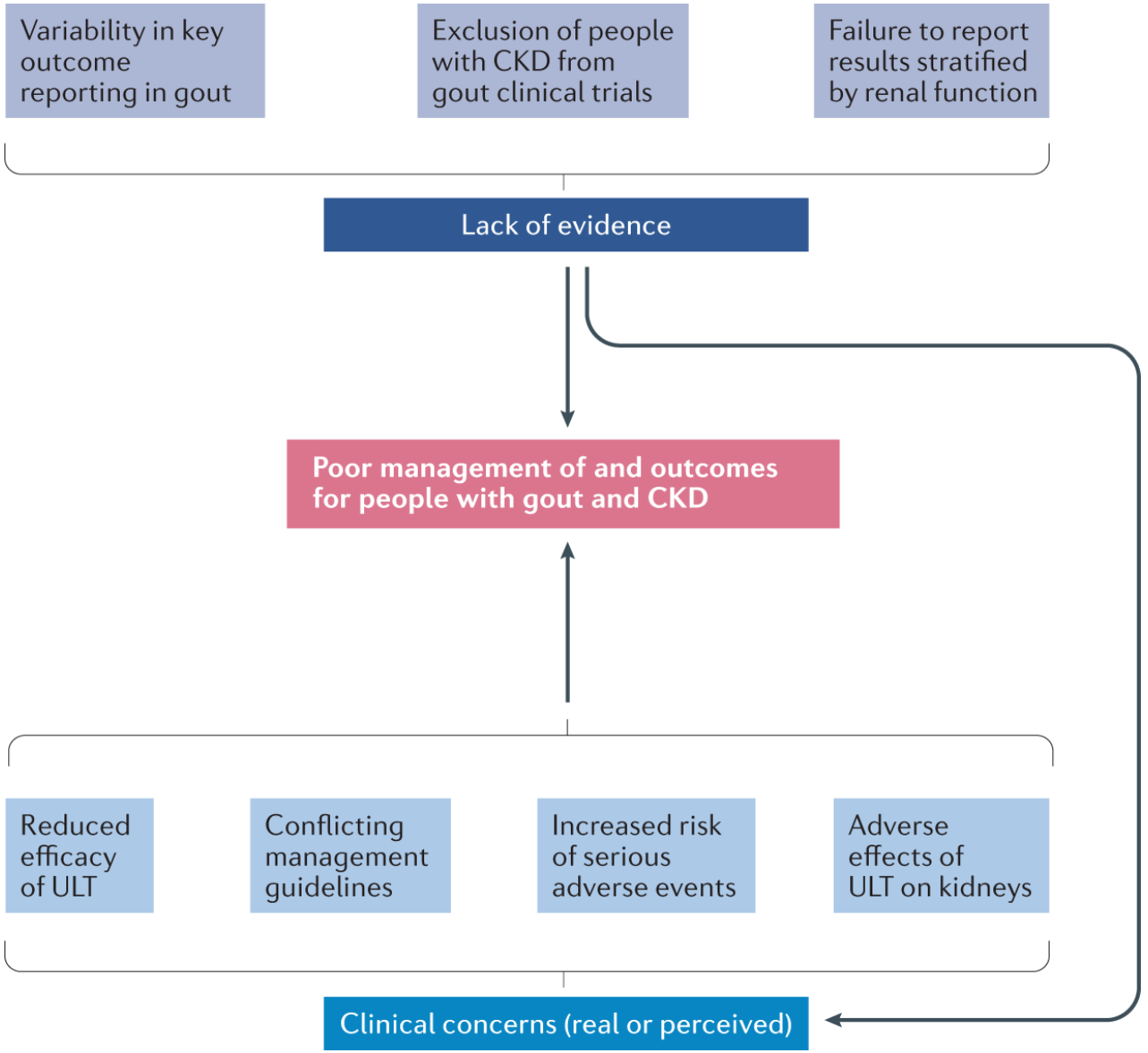
Recommendation
For patients with 1 or more subcutaneous tophi, we strongly recommend initiating ULT over no ULT.
For patients with radiographic damage (any modality) attributable to gout, we strongly recommend initiating ULT over no ULT.
For patients with frequent gout flares (≥2/year), we strongly recommend initiating ULT over no ULT.
For patients who have previously experienced >1 flare but have infrequent flares (<2/year), we conditionally recommend initiating ULT over no ULT.
For patients experiencing their first flare, we conditionally recommend *against* initiating ULT over no ULT, with the following exceptions.
For patients experiencing their first flare and CKD stage ≥3, SU >9 mg/dl, or urolithiasis, we conditionally recommend initiating ULT.
For patients with asymptomatic hyperuricemia (SU >6.8 mg/dl with no prior gout flares or subcutaneous tophi), we conditionally recommend *against* initiating any pharmacologic ULT (allopurinol, febuxostat, probenecid) over initiation of pharmacologic ULT.

Strongly recommend | Conditionally recommend | Strongly recommend against | Conditionally recommend against

Fitzgerald JD et al. 2020 American College of Rheumatology Guideline for the Management of Gout. Arthritis Care Res, 72: 744-760

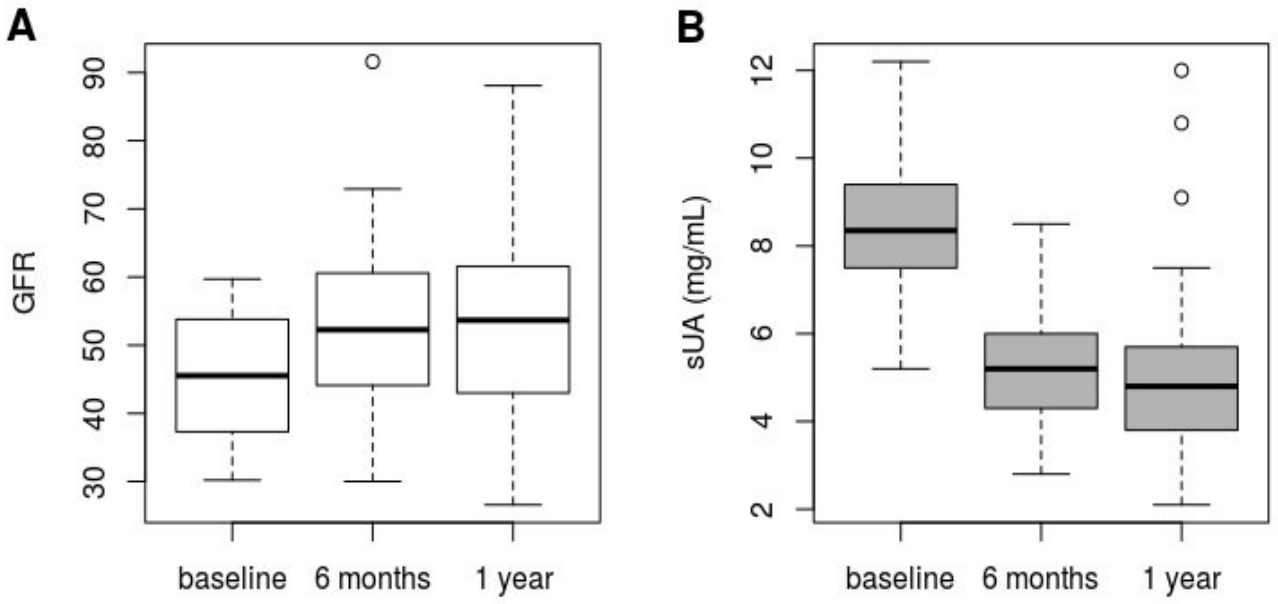
Urate-Lowering Therapies Comparison

Characteristic	Allopurinol	Febuxostat	Probenecid	Benzbromarone	Pegloticase
Mechanism	XOI – prevents urate production	XOI – prevents urate production	↑ renal urate excretion	↑ renal urate excretion	Recombinant uricase → allantoin
Metabolism/excretion	Renal (oxypurinol)	Hepatic (UGT, CYP1A2, 2C8, 2C9)	Renal	Hepatic (biliary)	Renal
Contraindications	Hypersensitivity	Caution: HF/IHD	Blood dyscrasias, stones	Liver disease, porphyria	G6PD deficiency
Drug interactions	Azathioprine, warfarin, diuretics	Azathioprine	Aspirin, methotrexate	Warfarin, sulfonylureas, phenytoin, fluconazole, rifampicin	Other ULTs, PEGylated drugs
Dosing	50–900 mg daily	40–120 mg daily	500–1000 mg BID	50–200 mg daily	8 mg IV q2 weeks
Key side-effects	Flares, AHS	Flares, ↑LFTs	Flares, stones	Flares, hepatotoxicity	Infusion rxns
Monitoring	SU, renal/liver	SU, renal/liver	SU, renal	SU, liver	SU (loss predicts rxn)
Special notes	Titrate even in CKD	Rare cross-reactivity	Hydration, alkalinize	Hydration, alkalinize	Monotherapy only
Anti-inflam. prophylaxis	Yes	Yes	Yes	Yes	Yes



Stamp LK et al. *Nat Rev Rheumatol* **17**, 633–641 (2021)

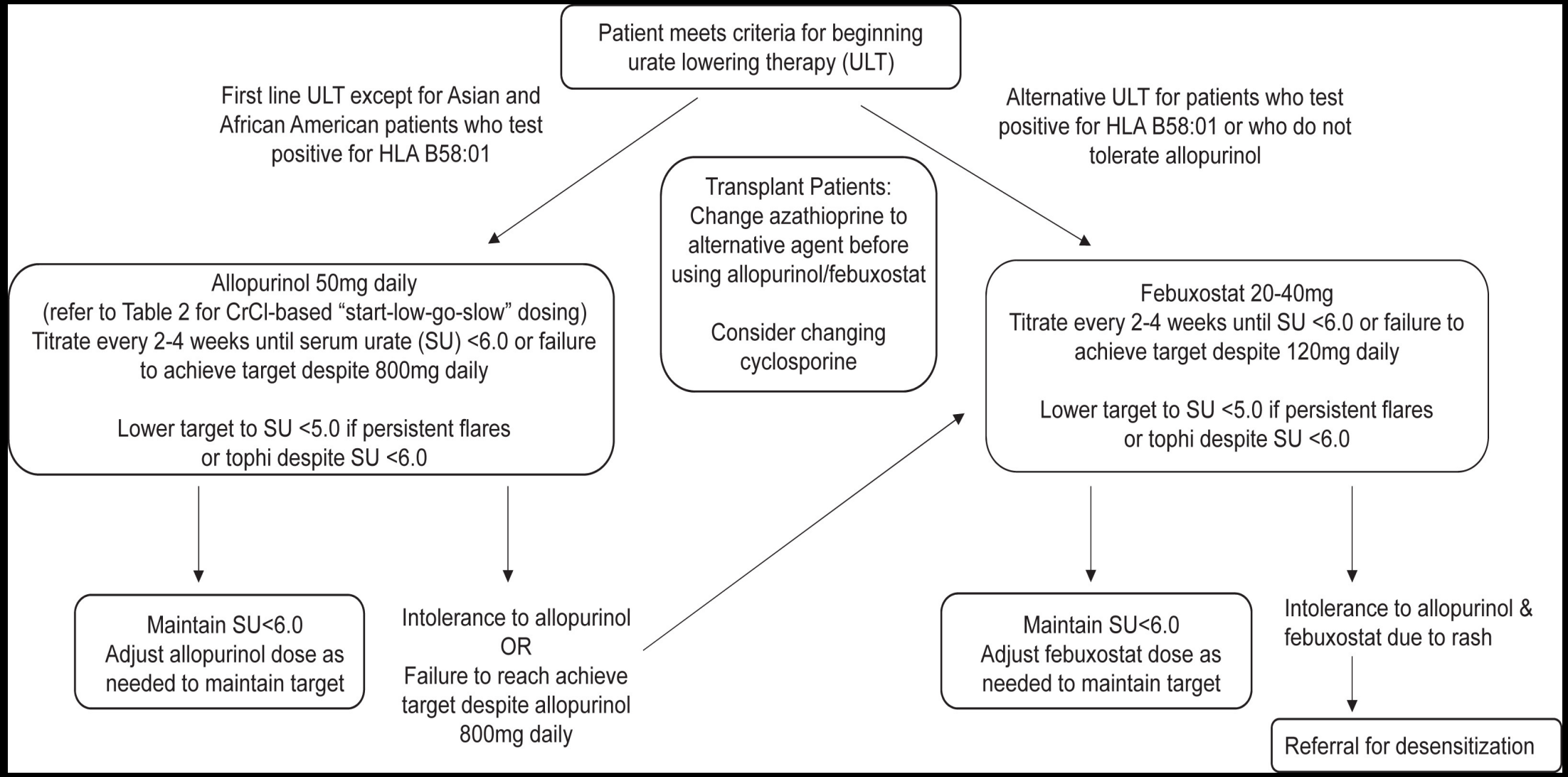
T2T in gout has a reno-protective effect in moderate CKD



- Randomized multi centre obs study 2014-2018
- ACR/EULAR 2015 gout criteria and CKD 3 GFR 30-59, treated with XOI, FU 6 and 12 months
- KT/single kidney excluded
- 50 patients, Mean baseline sUA was 8.55±1.57 mg/dl and mean eGFR 45.52±9.21 ml/min/m2
- Sig improvement in GFR in first 6 months, associated with an inverse relationship with SUA
- Nonsmoker and males had higher GFRs, no difference b/w Febuxostat and Allopurinol

Calvo Aranda E et al. Arthritis Rheumatol. 2019; 71 (suppl 10).

Approach to ULT in CKD



American Journal of Kidney Diseases 2025 86516-524DOI: (10.1053/j.ajkd.2025.04.020)

Starting allopurinol in CKD

- The guidance is to start allopurinol 1.5 mg/ unit of EGFR (mL/min/1.73 m2)
- Cautious approach recommended
- Lifelong – 38.9% of patients experienced a recurrence of gout when ULT is stopped
- Starting at lower doses also may decrease the need for prophylaxis with colchicine (noted for start low and go slow approach with allopurinol and Febuxostat)

Estimated GFR, ml/minute/1.73 m^2	**Allopurinol starting dosage**
<5	50 mg/week
5–15	50 mg twice weekly
16–30	50 mg every 2 days
31–45	50 mg/day
46–60	50 mg and 100 mg on alternate days
61–90	100 mg/day
91–130	150 mg/day
>130	200 mg/day

Stamp LK et al. Arthritis Rheum. 2012 Aug;64(8):2529-36

Perez Ruiz F et al. Arthritis Rheum. 2011; 63:4002-4006

Allopurinol Hypersensitivity Syndrome (AHS)

- **First described:** Hande et al., 1984 (78 patients with CKD)
- **Incidence:** ~0.1%; potentially fatal
- **Clinical features:**
- Severe rash
- Eosinophilia, leukocytosis
- Fever
- Hepatitis
- Acute kidney failure
- **Cutaneous manifestations:**
- Stevens–Johnson syndrome
- Toxic epidermal necrolysis
- Exfoliative dermatitis

RISK FACTORS FOR AHS:

- Age
- Female gender
- Renal function
- Diuretic use
- Starting dose
- Recent initiation
- HLA–B*5801 genotype

Hande K et al. Am J Med. 1984; 76:47-56

CKD based dosing- unintended consequences

- **Strict adherence** often leads to **undertreatment of gout**
- **Serum urate target achieved with allopurinol:**
  - CKD stage 2: **23.3%**
  - CKD stage 3: **20.2%**
  - CKD stage 4: **18.8%**
- **CKD prevalence:**
  - Uncontrolled gout: **49.4%**
  - Controlled gout: **32.4%**

Fuldeore M et al. BMC Nephrol. 2011; 12:36
Francis-Sedlak M et al. Rheumatol Ther. 2021; 8:183-197

T2T in CKD and gout: confusing guidelines?

- EULAR guidelines:
  - recommend restricting allopurinol to doses based on CLcr-based and considering febuxostat as an alternative
- ACR guidelines:
  - gradual escalation of allopurinol above CLcr-based dosing up to the maximum FDA approved doses to achieve target SU
- Most studies have shown patients with CKD tolerated higher doses than CLcr based doses and well tolerated:
  - In a RCT of safety of T2T in CKD, including patients with CLcr <30, 69% with dose escalations achieved target SU

FitzGerald JD et al. Arthritis Care Res. 2020; 72:744-760
Richette P et al. Ann Rheum Dis. 2017; 76:29-42
Stamp LK et al. Ann Rheum Dis. 2017; 76:1522-1528

HLA b5801

- HLA-B*58:01 can present the allopurinol metabolite, oxypurinol, directly to cytotoxic T cells without antigen processing
- T cell mediated cytotoxicity related to allopurinol or oxypurinol is restricted to carriers of HLA-B*58:01
- SCARs: drug rash with eosinophilia and systemic symptoms, Stevens-Johnson syndrome, toxic epidermal necrolysis
- HLA B5801 allele is commoner in Chinese, Thai, and Korean patients
- This allele is 5 x commoner in African Americans compared to white and hispanic population
  - 3 x greater risk of SCAR in AA population compared to the others
- Screening advised in this population group
  - Reduced SCAR
  - Cost effective

- Negative predictive value of HLA-B*58:01 for allopurinol induced SCARs is 100%
- PPV is 2%
- Han Chinese people who carry HLA-B*58:01 have a much higher risk of developing allopurinol induced SCARs than those who do not carry the allele (OR 580.3; 95% confidence interval 34.4 to 9780.9; P<0.001

Ko TM et al. BMJ. 2015 Sep 23;351

Cross reactivity?

- The risk of severe cutaneous adverse reactions:
  - 9.1% of patients who previously had experienced skin reactions on allopurinol also developed skin reactions on febuxostat
- Only 2.5% of patients developed skin reactions on febuxostat who had not done so with allopurinol
- If skin reaction to both allopurinol and Febuxostat (and not a candidate for uricosuric)
  - Allopurinol desensitisation under an allergy specialist

Bardin T et al. Joint Bone Spine. 2016; 83:314-317

Case

- 52 F, Familial hypercholesterolaemia, salivary and renal calculi (recurrent), hypertension, gout, fatty liver
- Multiple ADRs, including allopurinol previously
- DH: Fenofibrate, losartan
- sUA 544µmol/L, eGFR 52, γGT 144, ALT 92
- Freq of attack- once a year
- USS – calcific deposit at achilles tendon –tophi

What is the best treatment option here?

- Allopurinol desensitisation
- Febuxostat
- Probenecid
- Benzbromarone

Febuxostat in CKD

- Febuxostat is safe in GFR ≥ 15
- No dose adjustment needed for CrCl≥30mL/min
- As per FDA, restrict dose to 40 mg daily in for CrCl 15–29mL/min
- Limited data in advanced CKD, HD, transplant

Quilis N et al. Arthritis Rheumatol 2016;68:2563–4.
Saag KG et al. Arthritis Rheumatol 2016;68:2035–43.
Bardin T et al. Joint Bone Spine 2016;83:314–17.

Febuxostat in CVD: to prescribe or not?

- Conditional recommendation to use Febuxostat in those with CVD
- FDA-mandated CARES trial of febuxostat versus allopurinol:
  - there was no difference between the 2 arms in the primary composite CVD end point
  - Febuxostat, however, was associated with a higher risk of CVD-related death and all-cause mortality (driven by CVD deaths) compared with allopurinol
  - but there was no association with the other 3 secondary CVD outcomes (nonfatal myocardial infarction, nonfatal stroke, or urgent revascularization for unstable angina)
  - Difficult to interpret due to high drop out rate and majority of deaths after ULT discontinuation
  - Lack of untreated control group, therefore no measure of absolute CVD risk with Febuxostat
  - FDA black box warning though…
- These results were not replicated in other studies:
  - Large observational study (recruitment not selected for CVD) did not show an increased risk of CVD or all-cause mortality associated with febuxostat compared with allopurinol using methods to address confounding by indication
  - A managed care database study demonstrated lower risk of any major CVD event among febuxostat initiators than allopurinol, though confounding by indication may not have been adequately addressed
  - EMA FAST trial: no signal similar to CARES but majority Caucasian older male population

White WB et al. N Engl J Med 2018;378:1200–10.
Zhang M et al. Circulation 2018;138: 1116–26.
Foody J et al. Am Health Drug Benefits 2017;10:393–401
Mackenzie I et al. The Lancet, Volume 396, Issue 10264, 1745 - 1757

Case

- 57 M polyarticular tophaceous gout with nephrocalcinosis
- PMH: Sarcoidosis, CKD,
- Right elbow swelling with erosions and anterior joint effusion, Rt D2 swelling, left ankle, right wrist swelling. Multiple tophi in the hands, bilateral olecranon bursa
- Intolerance of allopurinol (GI intolerance), last dosed at 100 mg PO daily in July 2025
- Febuxostat 40 mg PO daily Sept 12, 2025- present
- Colchicine 0.3 mg daily

Creatinine		
Ref. Range & Units		62 - 100 umol/L
30/10/25	14:23	340
13/05/24	19:46	344
13/09/23	10:49	384
		384
10/08/22	21:35	269
29/11/21	11:48	257
08/08/19	15:28	289
20/04/18	10:24	256
03/04/18	11:47	254
31/03/17	09:51	298
31/03/17	09:49	301
30/11/16	14:31	296
09/11/16	01:30	389
18/05/16	09:43	317

Urate		
Ref. Range & Units		137 - 452 umol/L
11/07/25	17:27	666
18/02/25	12:46	680
13/12/22	13:51	617
13/12/22	09:12	617
18/07/22	10:21	703
21/06/21	11:12	723
01/06/21	11:50	699

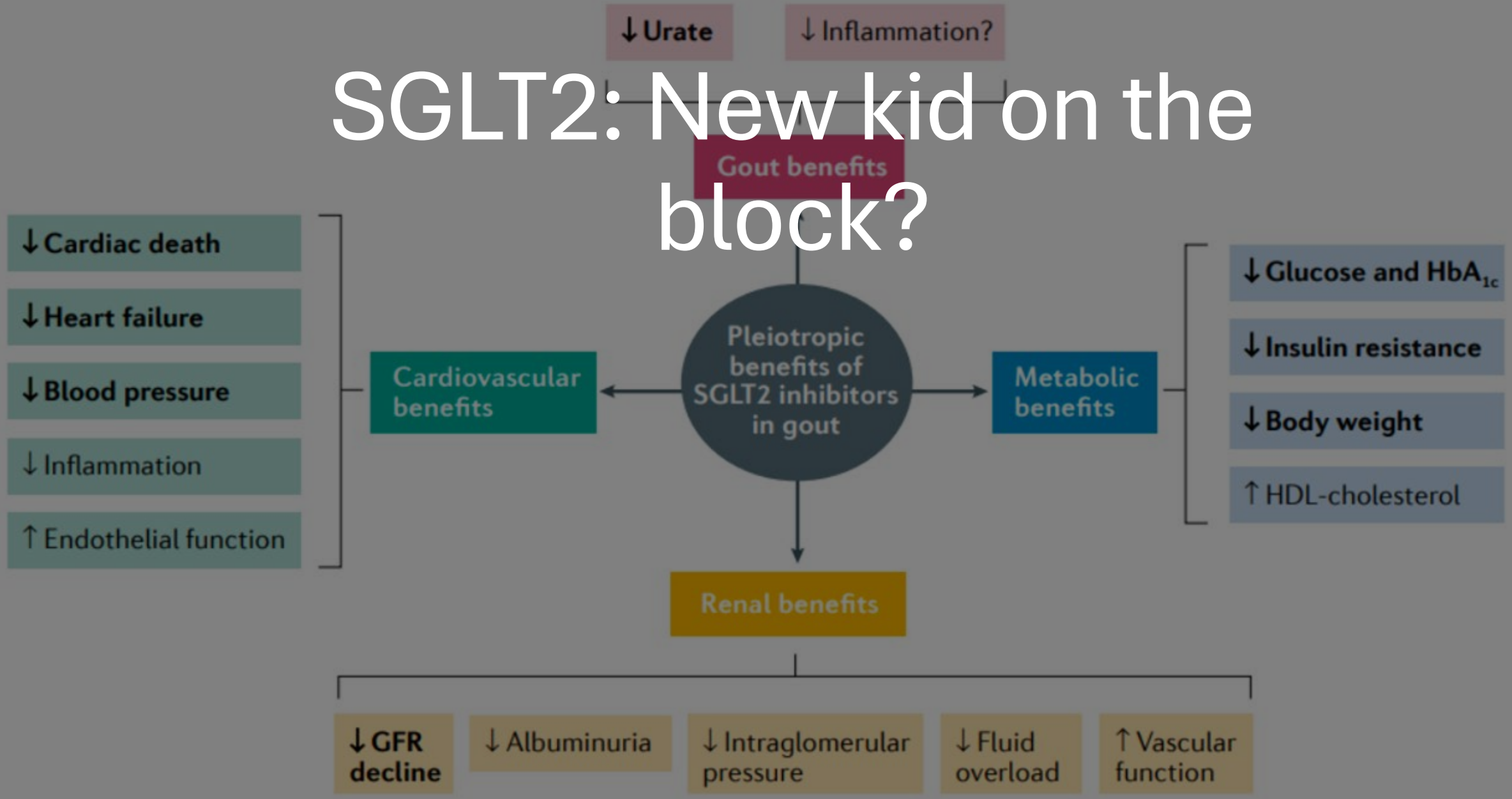
Pegloticase + MTX for chronic refractory gout: the MIRROR trial

- 12 month RCT of PEG (8mg bi weekly) + MTX (15 mg/OW) vs PEG +Placebo
- Inclusion: SUA > 7mg/dL at baseline/ ULT failure or intolerance/ > 2 flares/year, > 1 tophus, gouty arthropathy
- Exclusion: G6PD, MTX C/I, CKD 4-5
- 60% of MTX and 30% of placebo group met primary endpoint of SUA < 6 mg/dL
- 63% placebo and 23% MTX group met SUA discontinuation criteria (2 consecutive pre infusion SUA > 6 mg/dL after week 2)
- Of pts with tophi at baseline, 53.8% vs 31.0% had complete resolution of ≥1 tophus at Wk 52
- Similar safety profile b/w the two groups (with all AEs occurring by wk 24)

Botson J, saag k et al. *Arthritis Rheumatol.* 2022; 74 (suppl 9).

Choi Nat Rev Rheumatol 2022

SGLT2: New kid on the block?



Prophylactic Colchicine Dosing by Kidney & Liver Function: duration 3-6 months

Kidney / Liver Function	Creatinine Clearance	Prophylactic Colchicine Dose
Normal kidney function	>60 mL/min	0.6 mg once or twice daily
CKD Stage 3	30–60 mL/min	0.6 mg once daily
CKD Stage 4	<30 mL/min	0.6 mg every 2–3 days or 0.3 mg once daily
CKD Stage 5 / ESRD	<10 mL/min or hemodialysis	Avoid colchicine
Significant hepatic or hepatobiliary disease	—	Avoid colchicine
Combined hepatic and renal disease	—	Avoid colchicine

Reference: American College of Rheumatology Guideline for the Management of Gout (2020); EULAR recommendations for gout management.
Johnson RJ et al. Kidney Int. 2024; 106:573-582

Gout Management in Kidney Transplant Recipients

Aspect	Key Points
Azathioprine + XO inhibitors	Allopurinol/febuxostat increase risk of severe cytopenias
Dose adjustment	Reducing azathioprine by ≥⅔ lowers but does not eliminate risk
Preferred strategy	Switch azathioprine to mycophenolate mofetil when possible
Cyclosporine	Reduces renal urate clearance → ↑ serum urate and gout risk
Cyclosporine-associated gout	Shared decision making on continuation vs substitution
When to switch cyclosporine	Consider if major contributor to gout
Tacrolimus	Possibly lower gout risk than cyclosporine (data conflicting)
Colchicine interaction	Cyclosporine increases risk of colchicine toxicity

Pharmacokinetics, dosing, and effectiveness of allopurinol in dialysis: a scoping review

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Background

Gout → Treated with → Allopurinol → Oxypurinol → Lowers uric acid

Oxypurinol & uric acid Eliminated by the kidneys

Optimum dose of allopurinol in dialysis

Haemodialysis | Peritoneal dialysis

Aims

Explore:

1. Allopurinol dosing practices
2. Oxypurinol pharmacokinetics

Efficacy

- Urate lowering
- Target attainment
- Gout flares

Methodology

5 databases searched:

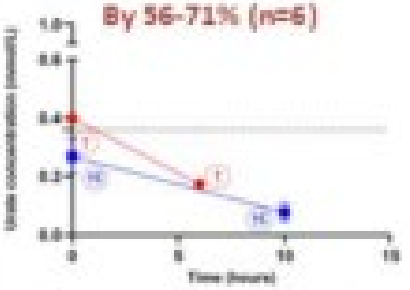
Patients with gout + On allopurinol + Receiving dialysis + and reported on 1 or 2

Results

	Haemodialysis	Peritoneal dialysis
Sample size	274	116
Average dose (mg/day)	100-600	110-125
Administration of allopurinol (n=43)	After haemodialysis session	After dialysate exchange
Oxypurinol dialytic clearance (mL/min)	78-137 (21/274 patients)	3.14 (5/116 patients)
Urate dialytic clearance (mL/min)	80-165 (31/274 patients)	2.7-4 (25/116 patients)
Frequency of gout flares (n=79)	Decreased from 2 to 0.1 p.a. since starting dialysis	
Target urate achievement	61% (20/33 patients)	47% (13/28 patients)

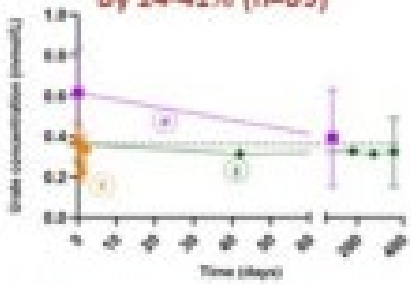
Decrease in urate over a haemodialysis session

By 56-71% (n=6)



Decrease in urate since starting haemodialysis

By 14-41% (n=85)



Decrease in oxypurinol over a haemodialysis session

By 39-57% (n=30)

Conclusions

Dialysis → Post session → Administer allopurinol → Titrate to target urate

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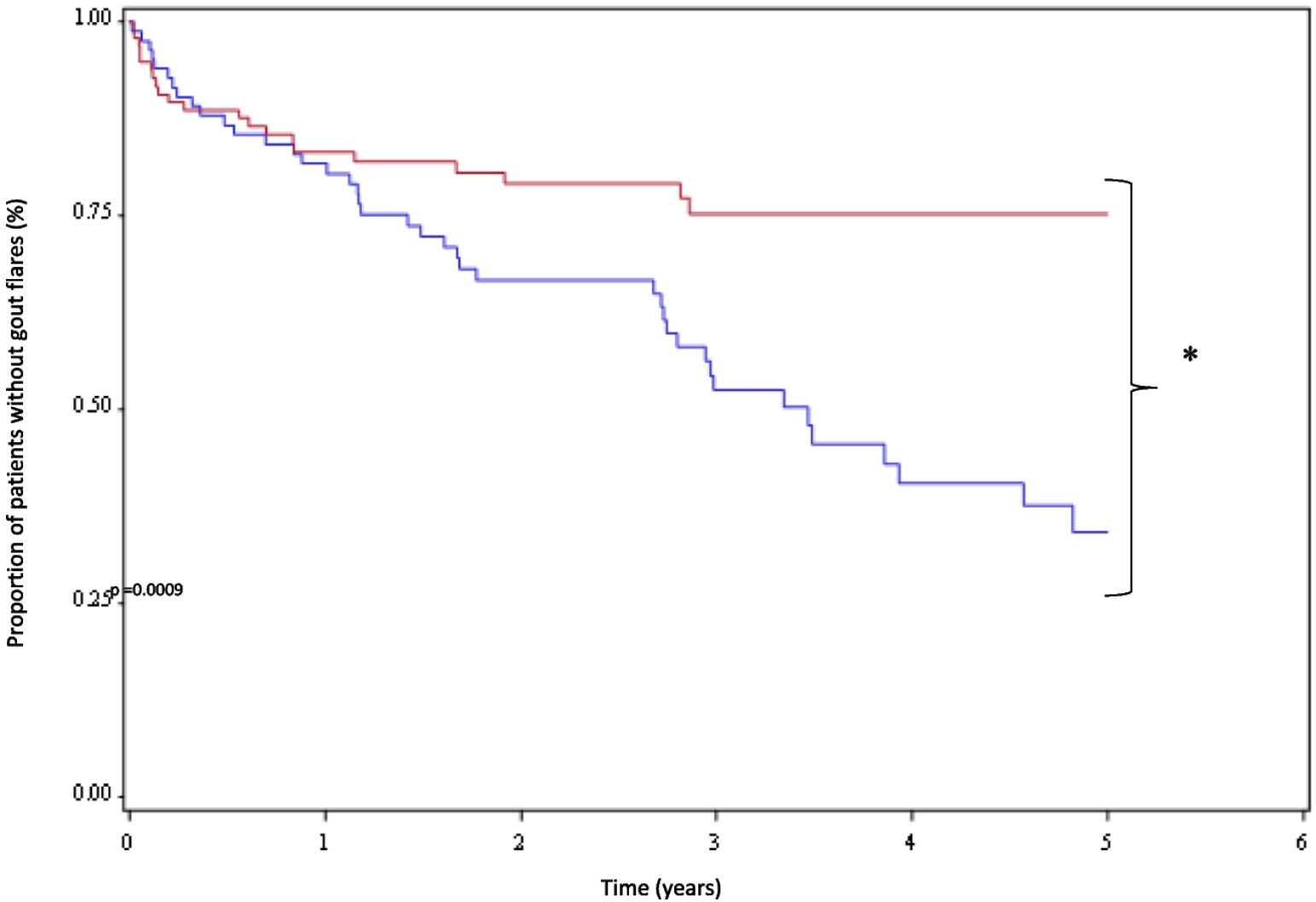
PK of ULT in dialysis

- Urate and oxypurinol were removed at a similar extent in each dialysis modality with higher clearances by HD > PD
- During HD, 39–57% of oxypurinol is cleared, therefore administer allopurinol after HD sessions with up-titration of doses according to T2T approach
- Alternatively, if administered before the HD session, the dose of allopurinol would need to be doubled to account for the oxypurinol clearance during the HD session
- Additional research required to fully understand the impact of dialyser type and dialysis conditions, such as blood and dialysate flow rates, on the pharmacokinetics of oxypurinol and the attainment of the urate target
- Although SUA decreased during a HD session, they increased during the interdialytic period, returning to the levels prior to the dialysis session. Therefore, PRE dialysis SUA monitoring to determine target attainment should be performed regularly
- Fluctuations in SUA are known to precipitate gout flares. Thus, prophylaxis with colchicine (or NSAIDs) is recommended when starting dialysis.

Gout flares in HD: Impact of ULT

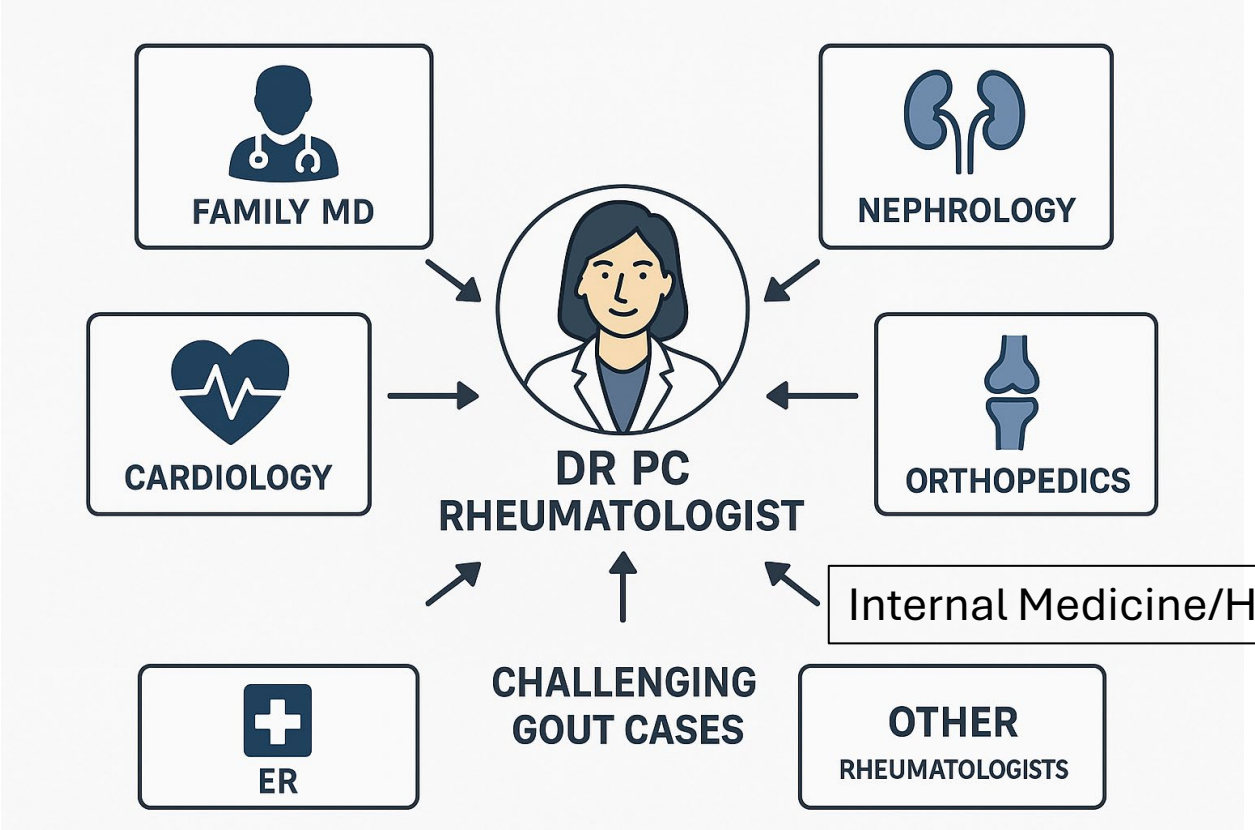


Patients on dialysis without urate lowering therapy

Patients on dialysis with urate lowering therapy

Steelandt, A et al. BMC Nephrol 25, 266 (2024)

Who refers patients to the gout clinic at TOH?



Referral criteria

- Intolerant/ perceived contraindication to allopurinol
- Non-response to moderately-high doses of allopurinol
- CKD stage 3b or greater i.e. eGFR < 45
- Renal replacement therapy
- Renal transplant recipients
- Sickle Cell disease
- Need for a second line ULT
- Need anakinra for management of acute gout flares

Take home messages

Avoid NSAID for prophylactic/acute gout Rx; colchicine needs dose adjustment in CKD, anti IL-1B may be considered
Febuxostat does not require dose adjustment for CrCl>30 ml/min; for CrCl 15-29 ml/min, FDA recommends 40 mg/day
Allopurinol – start low and go slow, can escalate beyond the dose recommended by CrCl
Uricosurics not recommended at low CrCl levels (<30 ml/min for probenecid and <20 ml/min for Benzbromarone)
Pegloticase can be used in advanced CKD, including HD, no need for dose adjustment
Patients on PD/HD can safely receive allopurinol

Thank you

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