CME CREDIT ***EDUCATIONAL OBJECTIVE:*** Readers will treat gout as aggressively in African Americans as in other populations

BHARAT KUMAR, MD
Allergy/Immunology and Rheumatology Fellow, Division of Immunology, University of Iowa Hospitals and Clinics, Iowa City, IA

PETAR LENERT, MD, PhD
Clinical Associate Professor of Internal Medicine, Division of Immunology, University of Iowa Hospitals and Clinics, Iowa City, IA

Gout and African Americans: Reducing disparities

ABSTRACT

African Americans are more likely to suffer from gout and are less likely to receive optimal treatment for it. Physicians should be aware of risk factors for gout and professional guidelines for treating acute attacks and high uric acid levels, and should help develop strategies to reduce disparities in healthcare delivery.

KEY POINTS

Gout is more common in African Americans mainly because of their higher prevalence of risk factors such as obesity, diabetes, chronic kidney disease, and hypertension.

Gout significantly reduces quality of life, economic productivity, and physical function and increases the risks of cardiovascular and renal disease.

Although professional guidelines and effective medications are widely available, studies have found low physician compliance with providing optimal gout treatment, especially for African American patients.

Treatment for gout in African Americans is the same as for all patients. Acute attacks should be treated promptly with anti-inflammatory agents, and uric acid levels should be aggressively lowered with drug therapy and diet modification.

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Despite the historic association of gout with royalty and "rich eating," gout disproportionately affects those of lower socioeconomic status.[1] Risk factors for gout, including obesity, chronic kidney disease, diabetes, and hypertension, are more common in African Americans, resulting in a higher prevalence of the disease. In addition, African Americans are less likely to receive the aggressive treatment for gout advocated by professional societies, such as anti-inflammatory medications during flares and prophylactic urate-lowering therapy.

This article reviews the epidemiology of gout and its pathophysiology, risk factors, and optimal management, focusing on African Americans and strategies to reduce healthcare disparities in this patient population.

GOUT IS COMMON AND SERIOUS

Gout is the most common inflammatory arthritis in the United States today, affecting 4% of the adult US population.[2] It is a chronic disease associated with high levels of uric acid, usually manifesting as intermittent attacks of painful monoarthritis, although multiple joints may be involved.

Despite a popular misconception that gout is merely an episodic nuisance, it is a serious disease that can significantly affect physical function and quality of life.[3] A 2013 systematic review found that quality of life was significantly reduced in patients with gout, particularly those with polyarticular gout, tophi, comorbidities, and radiographic damage.[4]

Economic costs include decreased worker productivity and increased absences from work.[5,6] From 2001 to 2005, an estimated 2

TABLE 1

Dietary factors in gout

Risk factor	Association with gout	Relevance to African Americans
Alcohol	Ethanol metabolism causes adenosine triphosphate consumption, leading to purine degradation and increased serum uric acid	Rates of alcohol use in African Americans are lower than in white Americans, but African Americans are more likely to have negative health consequences from excessive drinking than white Americans
Purine-rich foods	Excess purine is degraded to uric acid, leading to hyperuricemia	Meat consumption is higher among African Americans than white Americans
Sugary drinks	Fructose phosphorylation depletes phosphate, causing accumulation of uric acid	17% of African American adults obtain ≥ 25% of their calories from added sugars, compared with 11% of white adults
Dairy products	Beneficial: uricosuric effect of casein and lactalbumin	African Americans drink less milk than white Americans
Caffeine	Beneficial: uricosuric effect, antioxidant properties may increase insulin sensitivity, may inhibit xanthine oxidase	
Vitamin C	Beneficial: direct uricosuric effect	African Americans eat less fruits and vegetables than white Americans
Cherries or cherry juice	Beneficial: anti-inflammatory effects of polyphenols	

million visits were made to primary care providers due to gout.[7] Because gout frequently coexists with diabetes, hypertension, coronary artery disease, and kidney disease, it is often overlooked during routine clinic visits.[3]

HIGH URIC ACID HAS MANY EFFECTS

Uric acid, a product of purine metabolism, is the key mediator of gouty arthritis. Accumulation of uric acid in joints and other tissues leads to an exuberant inflammatory response manifesting as a gouty attack. When uncontrolled, uric acid may crystallize in joints and other structures, leading to tophi formation, which can cause chronic deforming and erosive arthritis, known as chronic tophaceous gout.[8]

Although gout is considered to be a musculoskeletal disorder, recent evidence indicates that hyperuricemia plays an important role in the development of renal disease and contributes to cardiovascular morbidity and death.[9] Several studies have found that reducing uric acid levels lowers cardiovascular mortality rates and retards the progression of chronic kidney disease.[10]

MEN AND CERTAIN RACIAL GROUPS MOST AFFECTED

Large-scale epidemiologic surveys have established that prevalence varies widely among population groups. Premenopausal women are less likely to be affected than men, presumably due to the effect of female hormones on renal tubular excretion of uric acid.[11]

Certain Asian populations (Filipinos, Taiwanese, Micronesians, the Maoris of New Zealand, Hmong Chinese immigrants in Minnesota) and African Americans have a high prevalence, while people from several sub-Saharan African countries have a very low prevalence. These differences are likely due to a variety of reasons, including genetic predisposition; diet (**Table 1**); risk factors such as obesity, diabetes, and hypertension; less access to healthcare resources; and inappropriate treatment.[12,13]

AFRICAN AMERICANS ARE A DIVERSE GROUP

"African Americans" are a highly heterogeneous group making up about 13% of the US population. Most scholars now consider racial identity largely a product of socioeconomic and political circumstances rather than a scientific concept.[14] The US Census Bureau defines African Americans (or "blacks") as those having origins in the black racial groups of Africa,[15] but even this is problematic, since it combines people as diverse as descendants of 17th, 18th, and 19th century enslaved Africans with recent first-generation African immigrants, as well as with Caribbean- and Latino-Americans of African heritage.

In addition, many African Americans trace their ancestry to other racial and ethnic groups, especially European and Native American. A study of nine European DNA markers among 10 African American populations in the United States and one population of Jamaicans of African descent found a range of frequencies from 7% among Jamaicans to 23% among African Americans living in New Orleans.[16]

Although such diversity argues against generalizing healthcare needs for the entire African American community, some evidence indicates that genetic markers for gene-disease associations may be consistent across traditional racial boundaries.[17]

GENETIC FACTORS CANNOT EXPLAIN GOUT DISPARITIES

Several small studies have found evidence of genetic factors mediating the risk for hyperuricemia and gout. Twin studies indicate that hyperuricemia is highly heritable,[18] although they do not show a concordance in the heritability of gout, suggesting that environmental factors also have an important role in developing clinical manifestations of the disease.[12]

Genome-wide association studies have identified 28 separate loci influencing uric acid levels, including genes for uric acid transporters (eg, *SLC2A9* and *ABCG2*), and metabolic pathway regulators (eg, *PDZK1*, *SCL22A12*, and *PRPSAP1*), but the distribution among African Americans is largely unknown.[19] One important exception is SLC2A9, a renal tubular transporter of uric acid: variants of SLC2A9 were found exclusively in African Americans, but the clinical significance of this association is unclear.[20]

Several epidemiologic studies have looked at gout in African Americans.

In the Coronary Artery Risk Development in Young Adults study,[21] young African Americans had lower levels of uric acid than whites after adjusting for body mass index (BMI), glomerular filtration rate (GFR), diet, and medications. But after up to 20 years of follow-up, the risk of hyperuricemia was 2.3 times higher in African American women than in white women (95% confidence interval [CI] 1.34–3.99). Such differences were not found between African American and white men.

The National Health and Nutritional Examination Survey (NHANES) found that African American adolescents had lower uric acid levels than white adolescents after taking into account sugar intake, GFR, BMI, and onset of puberty.[22]

The Multiple Risk Factor Intervention Trial[23] also found that among those with high cardiovascular risk, the incidence of hyperuricemia and gout was lower in African Americans than in whites.

African Americans are less likely to receive aggressive treatment for gout

COMORBID DISEASES MAY EXPLAIN DISPARITIES

Despite some evidence that African Americans have genetic factors that are protective against gout, they have a much greater frequency of acquired risk factors for gout, including obesity, physical inactivity, hypertension, diabetes mellitus, renal failure, high intake of seafood, elevated blood lead levels, and use of antihypertensive medications (**Table 2**).

Hochberg et al[24] examined the incidence of gout in 352 African American and 571 white physicians and found higher rates in African Americans (9% vs 5%). The authors suggested different rates of hypertension as an explanation, although the use of antihypertensive medications such as diuretics that promote hyperuricemia confounds the strength of this conclusion.

TABLE 2

Modifiable risk factors and comorbidities of gout in African Americans

Risk factor or comorbidity	Association with gout	Relevance to African Americans	Potential interventions
Hyperlipidemia	Increases the risk of hyperuricemia	African Americans actually have lower total cholesterol levels and higher high-density lipoprotein levels than white adults	Fenofibrate lowers serum uric acid levels
Hypertension	40% of patients with gout have hypertension Serum uric acid levels may contribute to the pathogenesis of hypertension	African American adults have a higher prevalence of hypertension (42%) than white adults (28%)	Reconsider prescription of loop and thiazide diuretics, since these increase serum uric acid levels Losartan has mild uricosuric effects
Diabetes mellitus	Incidence of hyperuricemia and gout is increased in patients with prediabetes Glycosuria in advanced diabetes leads to a uricosuric effect, decreasing the incidence of gout	Mean hemoglobin A_{1c} levels are higher in African Americans (6.18%) than in white adults (5.78%) 11.3% of African American adults have diabetes mellitus, compared to 6.8% of white adults	Behavioral modification and pharmacotherapy to control diabetes may improve hyperuricemia as well
Obesity	Obesity produces a proinflammatory state conducive to hyperuricemia and gout	45% of African American adults are obese, compared to 32% of white adults	Weight loss may help to reduce uric acid levels.
Coronary artery disease	Patients with gout have a relative risk of 1.45 (95% confidence interval 1.19–1.75) of coronary artery disease	African Americans have earlier age of onset and higher overall mortality rate from coronary artery disease in all age groups	Aggressive risk factor reduction through counseling and pharmacotherapy
Chronic kidney disease	Renal underexcretion of uric acid is the most common cause of hyperuricemia Hyperuricemia leads to progression of renal disease	0.1% of African American adults have end-stage renal disease vs 0.024% of white adults	Use of allopurinol may help to slow the progression of renal disease Colchicine should be used with extreme caution in chronic kidney disease

Information from references 2, 10, 12, 13, 21, 37, 47, 44, 46

AFRICAN AMERICANS NEED STANDARD TREATMENT …

Unlike for heart failure, in which subgroup analyses of large prospective studies have found different efficacies of medications in African Americans than in whites, no such data exist for gout. Although there is a higher risk of allopurinol hypersensitivity in ethnic groups that express the *HLA-B*5801* polymorphism (eg, Han Chinese, Korean, Thai, Japanese, Portuguese), African Americans are not known to be at greater risk.[25] There are also no special precautions for using febuxostat or probenecid in African Americans.

Absent any compelling reason, gout management should be the same regardless of ethnicity.[26] Patients should be counseled on primary prevention measures such as dietary and behavioral modification and, if necessary, started on aggressive urate-lowering therapy.[27]

… BUT ARE LESS LIKELY TO HAVE GOUT APPROPRIATELY TREATED

African Americans with gout are less likely to receive urate-lowering therapy.[28] According to the 2002 National Ambulatory Medical Care Survey in the United States, African Americans with gout are far less likely to receive allopurinol than whites (42% vs 80%; odds ratio [OR] 0.18; 95% CI 0.04–0.78).[23] Even when therapy is prescribed, rates of nonadherence are greater in African Americans than in whites (OR 1.86, 95% CI 1.52–2.27), though the authors do not speculate why this is so.[29] No studies have compared rates of prescribing febuxostat to African Americans vs whites.

African Americans are also less likely to receive ongoing routine care for their gout. A 2007 study[30] of 663 veterans found that physicians were 1.41 times less likely to adhere to three selected quality indicators when dealing with nonwhite than white patients (95% CI 0.52–3.84). The three quality indicators studied were:

- Lowering of daily allopurinol dose to below 300 mg/day in the presence of renal insufficiency (no longer considered a quality measure)
- Monitoring of serum urate level at least once during the first 6 months of continued use of a xanthine oxidase inhibitor, such as allopurinol
- Monitoring of complete blood cell count and creatinine kinase at least every 6 months in patients with renal impairment receiving long-term prophylactic oral colchicine (> 0.5 mg/day for at least 6 months).

The finding was independent of age, comorbidity index, healthcare access, and utilization characteristics (eg, number of inpatient and outpatient visits, type of physician, most frequently seen physician).

LIFESTYLE MODIFICATION

Dietary modification is a useful initial step toward reducing uric acid levels (**Table 1**).[27] The following measures are recommended:

Reduce alcohol intake. Alcoholic beverages, particularly beer, are strongly linked to hyperuricemia, according to a 2013 meta-analysis.[31] Although alcohol consumption is lower in African Americans than in whites, mortality rates from cirrhosis and other alcohol-related diseases are 10% higher, suggesting metabolic differences that render African Americans more susceptible to the negative health effects of alcohol.[32]

Avoid sugary drinks. Sweetened beverages, especially those rich in fructose, are also implicated in hyperuricemia and gout. NHANES found an increase in serum uric acid of 0.33 mg/dL (95% CI, 0.11–0.73) in those consuming one to three sugar-sweetened drinks per day compared with nonconsumers, adjusting for total energy intake, age, sex, medications, hypertension, and GFR.[33] A prospective study also found a relative risk of 1.85 for those who drink two or more sugar-sweetened beverages per day compared with those who drink fewer than one per month (95% CI 1.08–3.16).[34]

Unfortunately, African Americans consume a disproportionate amount of sugar from all sources: 17% of African Americans are considered heavy consumers vs 9% of whites.[35]

Limit foods rich in purines. Red meat, seafood, and some vegetables, including asparagus, spinach, peas, cauliflower, and mushrooms, are associated with increased serum uric acid levels. NHANES found that greater meat and seafood consumption was associated with increased uric acid levels. Choi et al[36] found that the risk of gout increased by 21% with each additional daily serving of meat; the relative risk of developing gout was 1.41 (95% CI 1.07–1.86) in the fifth quintile of meat intake compared with the first quintile, and 1.51 (95% CI 1.17–1.95) in the fifth quintile of seafood consumption.

Gout can significantly affect physical function and quality of life

Despite these associations with high-purine food consumption and gout, many purine-rich foods may not contribute to hyperuricemia, and therefore a low-purine diet may not be protective. Interestingly, purine-rich vegetable protein intake is not associated with increased gout risk.[37]

Increase dairy consumption. Dairy in the diet is associated with a lower incidence of gout, with a decrease of 0.21 mg/dL (95% CI –0.37 to –0.04) in serum uric acid levels between the highest and lowest quintiles of dairy consumption.[38] A randomized controlled trial found a 10% reduction in serum uric acid levels with milk consumption.[39]

Enjoy coffee. Coffee intake has been inversely correlated with gout. Daily intake of 4 to 5 cups of coffee is associated with a relative risk of 0.60 (95% CI 0.41–0.87) vs no coffee.[40]

Vitamin C and cherry juice[41,42] have also been linked to lower gout risk, but the data are less robust.

Control weight. Primary care providers should advise patients to increase physical activity and maintain a healthy weight.

In a prospective study, Choi et al[43] found that, in men, the risk of gout increased with the BMI. Compared with men with BMIs in the range of 21 to 22.9 kg/m^2, the relative risks were:

- 1.95 (95% CI 1.44–2.65) at BMI 25 to 29.9 kg/m^2
- 2.33 (95% CI 1.62–3.36) at BMI 30 to 34.9 kg/m^2
- 2.97 (95% CI 1.73–5.10) at BMI > 35 kg/m^2.

In addition, those who gained more than 13.6 kg since age 21 had a relative risk of 1.99 (95% CI 1.49–2.66) of developing gout compared with those whose weight remained within 1.8 kg.

For those who cannot achieve weight loss through conservative measures, bariatric surgery has shown promise. In a prospective study of 60 obese patients (BMI > 35 kg/m^2) with gout and type 2 diabetes mellitus, uric acid steadily declined during the first year after bariatric surgery.[44]

African Americans have a greater frequency of risk factors for gout

TREATMENT OF ACUTE ATTACKS

Gout can be effectively managed in most patients. Behavioral and pharmacologic interventions are cheap and effective and have been shown to halt further damage to joints as well as retard the progression of renal disease and reduce cardiovascular morbidity.

During acute attacks, anti-inflammatory medications, principally glucocorticoids, nonsteroidal anti-inflammatory drugs, and colchicine, should be given promptly to reduce the intensity and duration of flares.

Although traditional teaching has been that urate-lowering therapy should not be initiated during an acute gout attack because it could prolong the duration of the attack, guidelines now permit it, based on studies showing that therapy does not prolong attacks.[45,46]

AGGRESSIVE URATE-LOWERING THERAPY FOR PROPHYLAXIS

Long-term treatment of gout is aimed at reducing uric acid levels by mitigating modifiable risk factors and through urate-lowering therapy.[46] For many patients, conservative management with dietary and other behavioral changes is not sufficient to prevent further attacks of gout, necessitating urate-lowering therapy. Comorbid diseases such as obesity, hypertension, chronic kidney disease, and diabetes should also be addressed because they promote hyperuricemia and gouty attacks.[47]

A number of organizations have issued gout management guidelines over the past decade, including the American College of Rheumatology (ACR) in 2012, the European League Against Rheumatism (2006, updated in 2014), and the British Society of Rheumatology (2007). All recommend urate-lowering therapy to prevent gout flares.

The American and European guidelines recommend a target uric acid level below 6 mg/dL, and the British guidelines recommend a target below 5 mg/dL.[48] For patients with palpable and visible tophi, the ACR guidelines state that lowering to below 5 mg/dL may be needed.[46]

First-line agents for urate-lowering therapy are xanthine oxidase inhibitors, which include allopurinol (costing $0.48 per generic 100-mg tablet or $0.92 per 300-mg tablet), and febuxostat ($5.38 per 40-mg or 80-mg tablet). For patients with contraindications or intolerances to allopurinol or febuxostat, probenecid ($1.15 per 500-mg tablet), which functions as a uricosuric agent (ie, increases urinary excretion of uric acid), may be used.

Losartan ($1.68 per 25-mg tablet) and fenofibrate ($1.91 per 48-mg tablet) are also often used to reduce uric acid levels, but they have only modest effects and are not approved for this indication in the United States.[49]

MANAGE CHRONIC CASES WITH CONTINUED THERAPY

ACR guidelines strongly emphasize continuing prophylaxis in case of ongoing gout activity (ie, detection of tophi on examination, recent gout attacks, or chronic gouty arthritis) (**Table 3**). The following durations have been

TABLE 3

Approach to gout management

ESTABLISH THE DIAGNOSIS OF GOUT

Aspirate joint fluid and examine for monosodium urate crystals under polarized light microscopy

Note: European League Against Rheumatism guidelines do not require crystal analysis confirmation to diagnose gout

Evaluate clinical disease activity and burden:
- Disease severity (number of attacks per year)
- Presence of tophi
- Other signs of hyperuricemia (history of kidney stones)

Consider comorbidities related to gout:
- Obesity, dietary factors, excessive alcohol intake
- Metabolic syndrome, diabetes mellitus
- Hypertension, hyperlipidemia, modifiable risk factors for coronary artery disease and stroke
- Serum urate-elevating medications
- History of urolithiasis or chronic kidney disease
- Genetic or acquired causes of uric acid excess
- Lead intoxication

PROMPTLY TREAT INFLAMMATION FROM GOUT

Start anti-inflammatory therapy promptly
- Within the first 36 hours, give 1.2 mg colchicine followed by 0.6 mg 1 hour later, then 0.6 mg once or twice daily (unless dose adjustment is required)
- Nonsteroidal anti-inflammatory drugs (NSAIDs) are an alternate first-line agent
- Prednisone (< 10 mg) may be given if NSAIDs or colchicine cannot be given and intra-articular steroid injection is impractical

If response is inadequate within 24 hours, consider either:
- Switching to another agent
- OR
- Combination therapy

Continue anti-inflammatory gout attack prophylaxis for:
- At least 6 months after the attack
- OR
- 3 months after achieving target serum urate levels (if no evidence of tophi)
- OR
- 6 months after achieving target serum urate levels (if tophi exist)

REDUCE URIC ACID LEVELS TO < 6 MG/DL

Provide patient education on gout, advise modifying diet and lifestyle

Address comorbid conditions

Discontinue nonessential medications that cause hyperuricemia

Pharmacologic urate-lowering therapy is indicated in patients with:
- Tophus or tophi by clinical examination or imaging study
- More than 2 acute gout attacks per year
- Chronic kidney disease stage 2 or worse
- Past urolithiasis

First-line urate-lowering agents:
- Allopurinol and febuxostat
- Allopurinol may be started at 100 mg/day
- In patients with chronic kidney disease stage 4, start at 50 mg/day
- Test for HLA-B*5801 in high-risk populations[a]
- Febuxostat may be started at 40 mg/day
- Second-line urate-lowering agents: probenecid and other uricosuric drugs

KEEP URIC ACID LEVELS < 6 MG/DL

While titrating medications:
- Measure uric acid levels every 2–5 weeks
- Allopurinol can be advanced cautiously in 100-mg increments beyond 300 mg/day, even in chronic kidney disease
- The maximum appropriate dose of allopurinol is 800 mg/day
- In the United States, febuxostat can be titrated up to 80 mg
- A 120-mg dose of febuxostat is available in Europe

While on a stable regimen:
- Measure uric acid levels at least every 6 months
- Monitor for adverse effects of urate-lowering therapy:
- Allopurinol—pruritus, rash, elevated aminotransferases
- Febuxostat—rash, elevated aminotransferases

For difficult-to-control gout:
- Assess adherence to medication regimen
- Consider rheumatology referral

[a] High-risk populations include Thais and Han Chinese, as well as Koreans with chronic kidney disease stage 3.

Based on the 2012 American College of Rheumatology Guidelines, reference 46.

proposed for prophylaxis:

- 6 months after an attack
- 6 months after achieving target uric acid level in patients with evidence of tophi
- 3 months after achieving target uric acid level in patients with resolved tophi.

Two randomized controlled trials support the use of the anti-inflammatory agent colchicine ($4.30 per tablet) for prophylaxis when initiating urate-lowering therapy.[50,51]

Monitor uric acid levels, renal function, adverse effects

The initial dosage of urate-lowering agents depends on the presence of kidney disease (**Table 3**).

Allopurinol is typically started at 50 mg to 100 mg oral daily, and titrated upward in increments of 100 mg depending on uric acid levels. According to the ACR guidelines, uric acid levels should be measured every 2 to 5 weeks.[46] The Febuxostat vs Allopurinol Streamline Trial found that 97% of patients reached target uric acid levels within two titrations.[52]

Especially during the first months of therapy, physicians should be vigilant for adverse effects of allopurinol, including hypersensitivity reaction (rash, fever, Stevens-Johnson syndrome), hepatotoxicity, and myelotoxicity (bone marrow suppression), and for effects of febuxostat, such as rash, diarrhea, elevations in aminotransferase levels, and upper respiratory tract infections.[53] Although the maximum acceptable dose of allopurinol is 800 mg even in chronic kidney disease, regularly monitoring for hypersensitivity reactions and other adverse effects is needed if doses are above 300 mg per day.[46]

Beer and sugary drinks are strongly linked to hyperuricemia

Routine follow-up is essential

Adherence to therapy should be assessed at every visit. Patients should be counseled that gout is a chronic disease and that they should continue on urate-lowering therapy even if they are not having acute attacks. Adverse effects of medications should be monitored and addressed, although for allopurinol and febuxostat these are rare beyond the first few months of initiation and titration. If worsening of gout or uric acid levels occurs, therapy should be augmented and contributors to hyperuricemia reviewed. In refractory cases, rheumatology consultation may be needed; medications such as pegloticase ($16,800/mL) may be deemed necessary for severe tophaceous gout or for patients who need more rapid reduction of urates.[49]

STRATEGIES TO ADDRESS DISPARITIES

Creative approaches are needed to engage African American communities to reduce the burden of gout. No trials have been published evaluating methods for reducing health disparities with gout, but strategies for other chronic conditions may be applicable.

Incorporate guidelines better. Although setting and disseminating guidelines should ensure that care is standardized, studies have found that primary care physicians and rheumatologists frequently do not implement them.[3,54] Reasons cited for poor adherence to gout guidelines include their relatively recent release, poor patient adherence, lack of measurement tools, and inadequate education of primary care physicians. Incorporating the guidelines as "best practice advisories" into electronic medical record systems has been proposed to improve their implementation.[46]

Use a team approach. Some quality improvement projects have used pharmacists and nurses to help implement gout guidelines. In two studies, empowering nurses and pharmacists to better educate patients and implement standardized protocols for titrating urate-lowering medications led to sustained improvements in maintaining serum uric acid levels less than 6 mg/dL.[55–57]

Multidisciplinary involvement by nutritionists, physicians, and community health workers have been found to help improve glycemic control in African Americans.[58] Similar efforts can be undertaken to improve control of uric acid levels through dietary modification and improved compliance.

Address patient concerns. Substantial gaps exist in knowledge about gout between providers and the general population, although large studies specifically focusing on African Americans are lacking.[59] Qualitative studies suggest that patient experience of gout may vary depending on race, with African Americans more likely than whites to rank the following concerns regarding gout as high: dietary restric-

tions, emotional burden, severe pain, the need for canes or crutches during flares, and gout bringing their day to a halt.[60] Another study among African Americans with gout found concerns about the effectiveness of urate-lowering therapy, side effects of medications, polypharmacy, pill size, cost, refill issues, and forgetting to take medications regularly.[61]

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***ADDRESS**: Bharat Kumar, MD, Division of Immunology, University of Iowa Hospitals and Clinics, 200 Hawkins Drive, Iowa City, IA 52242; Bharat-Kumar@UIowa.edu*