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Prevalence and characteristics of metabolic syndrome in gout patients in a hospital setting in sub-Saharan Africa

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ABSTRACT

Background: Evidence from epidemiological studies suggests an important association between gout and the metabolic syndrome (MetS). However, to the best of our knowledge, prevalence of metabolic syndrome in gout has not been reported in sub-Saharan African (SSA) settings.
Objectives: The aim of this study was to determine the prevalence and characteristics of MetS in gout in a SSA population.
Method: After prior ethical clearance, we carried out a cross-sectional study involving gout patients in a referral hospital in Douala-Cameroon. Metabolic syndrome was defined using International Diabetes Foundation criteria. Associations between variables were assessed using logistic regression.$p < 0.05$ was considered significant.
Results: On 174 gout patients (48.3% females) who consented to participate in the study, the median (IQR) age was 55.00 (14.25) years, and the median (IQR) duration of gout was 7.5 (10.0) years. Prevalence of metabolic syndrome was 54.6% (95% CI: 47.9%–62.8%). One hundred and forty-seven (84.5%) participants had central obesity, 62 (35.6%) raised triglycerides, 79 (45.4%) reduced HDL-C, 129 (74.1%) raised blood pressure, and 85 (48.9%) had raised fasting plasma glucose. On logistic regression analyses, gout patients with metabolic syndrome significantly had a higher body mass index (OR: 1.09, 95% CI: 1.02–1.17), and higher levels of serum uric acid (OR: 1.02, 95% CI: 1.01–1.04).
Conclusions: About 1 out of every 2 gout patients in this population have metabolic syndrome. These gout patients with metabolic syndrome significantly have a higher body mass index, and higher levels of serum uric acid. Cohort studies are required to clearly establish the direction of the relationship between gout and metabolic syndrome.

1. Background

Gout is the most prevalent form of inflammatory arthritis, with increasing prevalence and incidence in many parts of the world [1–3]. It is characterised by chronic deposition of monosodium urate (MSU) crystals in tissues, and is associated with reduced quality of life [2,4]. Gout typically presents with acute, very painful, self-limiting episodes of peripheral joint synovitis, and can eventually evolve into joint damage and deformity, chronic usage-related pain, and subcutaneous tophus deposition [2,3]. While risk factors for development of gout include hyperuricemia, genetic factors, dietary factors and alcohol consumption, evidence from epidemiological studies suggest an important association between gout and metabolic syndrome [2,4–6].

Metabolic syndrome (MetS) is a cluster of cardiovascular risk factors in an individual, which confer increased risks of atherosclerotic cardiovascular diseases, diabetes mellitus and mortality from all-causes [7,8]. The International Diabetes Federation (IDF) estimates that about a quarter of the world's adult population have MetS [8]. Although the underlying cause of MetS is not well understood, insulin resistance and central obesity are considered significant factors [8]. Raised serum uric acid (SUA) appears to be the key factor in the observed association between gout and

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metabolic syndrome. SUA has been reported as an independent predictor of MetS [9,10]. Also, higher insulin resistance and higher leptin production decrease renal excretion of uric acid, thereby increasing serum concentrations of uric acid [11,12]. Choi et al. in The Third National Health and Nutrition Examination Survey on 8669 adults reported that the prevalence of MetS increases substantially with increasing levels of serum uric acid [13]. In addition, the individual elements of MetS including hypertension, obesity, dyslipidaemia, hyperglycaemia, and insulin resistance, are positively correlated with serum levels of uric acid [14]. Jung et al. reported a significantly higher mean level of SUA in gout patients with metabolic syndrome, compared to gout patients without metabolic syndrome [15].

High prevalence of MetS in gout has been reported in Asian and American populations, ranging from 30.1% to 62.8% [5,15–19]. However, to the best of our knowledge, prevalence of MetS in gout has not been reported in sub-Saharan African settings. The aim of this study was to determine the prevalence and characteristics of MetS in gout in a sub-Saharan African population.

2. Materials and methods

2.1. Study design and setting

We carried out a cross sectional study in the Rheumatology unit of the Douala General Hospital (DGH) over a period of 6 months (December 2016–May 2017). DGH is referral hospital in Cameroon. After obtaining ethical clearance from the Douala General Hospital Research and Scientific Committee, we targeted patients with gout based on American College of Rheumatology endorsed guidelines [20]. We calculated a minimum sample size of 169 participants, using previously reported prevalence of MetS in the general population in Cameron [21]. We used consecutive, non-probability sampling method to select eligible study participants. We excluded pregnant women, patients with chronic kidney disease and patients on cancer chemotherapy, as reported in their health records. All study participants gave consent to participate.

2.2. Data sources and measurement

Socio-demographic data, medical history details and clinical history of gout were obtained from the patient's health record, alongside an interview by study investigators. All study participants were physically examined by the principal investigator. A stadiometer and automatic weighing scale were used to measure height to the nearest 0.1 cm and weight to the nearest 0.1 kg respectively. Following World Health Organisation guidelines, a measuring tape and automatic blood pressure machine (MAGNIEN B1, BP 3BM1-3 P) were used to measure waist circumference (WC) and blood pressure (BP) respectively [22,23].

After a fasting period of 12 h, 5 ml of venous blood was drawn from any of the patient's upper limbs using a 10 ml syringe. Fasting blood sugar (FBS) was assessed using a glucometer (OneTouch® Ultra). Serum triglycerides (TG), serum high-density lipoprotein-cholesterol (HDL-C) and SUA were assessed using a biochemistry analyser (Roche Hitachi Cobas® 6000). Serum TG, serum HDL-C and SUA were calculated using the enzymatic method, direct method, and direct kinetic uricase method respectively [24–26].

2.3. Variables

Residence was defined as urban or rural, occupation defined as employed or unemployed (including students and retirees) and marital status defined as married or single (including divorced and

Table 1
Baseline characteristics of study participants.

Parameter	Total, N = 174	MetS, N = 95	No MetS, N = 79	[a]*p* value
Age (years), median (IQR)	55.00 (14.25)	55.00 (11.00)	55.00 (20.00)	0.090
Gender (Female), n (%)	84 (48.3)	50 (52.6)	34 (43.0)	0.207
Residence (Urban), n (%)	145 (83.3)	81 (85.3)	64 (81.0)	0.454
Occupation (Unemployed), n (%)	49 (28.2)	32 (33.7)	17 (21.5)	0.076
Marital status (Married), n (%)	135 (77.6)	72 (75.8)	63 (79.7)	0.533
Hypertension, n (%)	62 (35.6)	35 (36.8)	27 (34.2)	0.715
Diabetes, n (%)	23 (13.2)	18 (18.9)	5 (6.3)	**0.014**
Family history of Obesity, n (%)	43 (24.7)	21 (22.1)	22 (27.8)	0.382
Smoking, n (%)	26 (14.9)	14 (14.7)	12 (15.2)	0.933
Alcohol consumption, n (%)	117 (67.2)	60 (63.2)	57 (72.2)	0.208
Medications, n (%)				
Anti-hypertensives	55 (31.6)	32 (33.7)	23 (29.1)	0.519
Anti-diabetics	16 (9.2)	15 (15.8)	1 (1.3)	**0.001**
Lipid-lowering drugs	3 (1.7)	3 (3.2)	0 (0.0)	0.111
Diagnosis of Gout, n (%)				
MSU crystals in joint fluid	53 (30.5)	25 (26.3)	28 (35.4)	0.193
Tophus	61 (35.1)	31 (32.6)	30 (38.0)	0.462
Six or more [b]ARA criteria for gout	158 (90.8)	88 (92.6)	70 (88.6)	0.360
Duration of Gout (years), median (IQR)	7.5 (10.0)	7.5 (10.0)	2.5 (10.0)	**0.035**
BMI (kg/m^2), median (IQR)	32.50 (10.00)	34.00 (5.00)	30.00 (10.00)	**0.001**
Abdominal circumference (cm), median (IQR)	100.00 (17.25)	102.00 (13.00)	94.00 (19.00)	<**0.001**
Systolic BP (mm Hg), mean (SD)	139.50 (22.49)	146.24 (23.83)	131.39 (17.76)	<**0.001**
Diastolic BP (mm Hg), mean (SD)	89.29 (12.39)	93.75 (12.81)	85.41 (10.22)	<**0.001**
FBS (mg/dl), median (IQR)	96.00 (16.00)	105.00 (24.00)	90.00 (17.00)	<**0.001**
Triglycerides (mg/dl), median (IQR)	118.00 (71.50)	150.00 (88.00)	103.00 (48.00)	<**0.001**
HDL-C (mg/dl), median (IQR)	46.00 (25.00)	37.00 (16.00)	55.00 (21.00)	<**0.001**
Serum uric acid (mg/l), median (IQR)	80.00 (22.25)	81.00 (23.00)	76.00 (26.00)	**0.026**

MetS: metabolic syndrome, MSU: monosodium urate crystals, ARA: American Rheumatism Association, BMI: body mass index, BP: blood pressure, FBS: fasting blood glucose, HDL-C: high density lipoprotein cholesterol, IQR: interquartile range, SD: standard deviation.

[a] Pearson's chi-squared test and Fisher's exact test (where appropriate) for categorical variables, Independent student's *t*-test and Mann-Whitney U test (where appropriate) for continuous variables.

[b] ARA criteria for gout: more than one attack of acute arthritis, maximum inflammation developed within one day, monoarthritis attack, redness observed over joints, first metatarsophalangeal joint painful or swollen, unilateral first metatarsophalangeal joint attack, unilateral tarsal joint attack, tophus (suspected or proven), hyperuricemia, asymmetric swelling within a joint on x-ray, subcortical cysts without erosions on x-ray, joint fluid negative for organisms during attack.

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