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Original article

Prevalence of Gout in End-Stage Renal Disease Patients

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Abstract
Gout is a disorder of purine metabolism that results in hyperuricemia, but on the other hand, in ESRD patients, the main cause is decreased glomerular filtration rate (GFR), because of high uric acid in the blood, uric acid builds up in the fluid around joints and soft tissue, it forms tiny crystals called monosodium urate. The association between gout and chronic kidney disease CKD is described as mutual, and CKD is an independent risk factor for gout. This study seeks to determine the prevalence of gout among end-stage renal disease (ESRD) patients. The study was conducted in the State of Libya in Tripoli at the Tripoli Kidney Services Center on patients with renal failure. The number of samples used was 121, The study period was from April to July of 2022. Three analyses were carried out for all patients' urea, creatinine to assess the deterioration of the kidneys, and uric acid to diagnose gout. ESRD patients. All urea and creatinine results were high, and the mean of male urea results (146.984) and female (146.982), the mean of creatinine for females (8.63) was higher than males (8.48). and it was found that uric acid clearance has no relationship during dialysis with urea and creatinine clearance, The prevalence of gout among patients with ESRD in this research was 46.3 % ($p < 0.000$), and the prevalence rate in males is 46 %, while in females it is 47 %, mean age of females was 54.2 and males of 56.4.
Keywords. Gout, End-Stage Renal Disease, Hyperuricemia, Chronic Kidney Disease.

Introduction

The term “gout” from the ancient French word gote and the Latin word gutta, meaning drop, was coined in the 13th century by Randolophus De Bocking [1] due to its links to excessive red meats and alcohol intake. It was dubbed "the illness of kings"[1]. Gout affects 1–4% of the population worldwide; it affects 2.68 people out of 1000 each year. Males are two to six times more affected than females to develop it. Gout is becoming more common around the world as a result of poor eating habits such as junk food, lack of exercise, and an increase in metabolic syndrome and obesity [2].

Gout is caused by disorder purine metabolism, some purines are made from endogenous sources (from the breakdown of nucleic adenosine and guanosine) and others from exogenous sources (from certain food).gout is characterized by hyperuricemia, as a result of high uric acid in the blood uric acid builds up in the fluid around your joints and soft tissue, it forms tiny crystals called monosodium urate, Urate crystals cause gout symptoms, including pain and swelling the most common inflammatory arthritis in the men over the age of 40 years [3], The primary excretion of uric acid occurs through the kidneys, and if weakness occurs, it is compensated by gastrointestinal clearance. When gastrointestinal urate clearance is impaired, thereby increasing the risk for gout [4].

Hyperuricemia is a description of high uric acid in the blood, through which gout can be diagnosed if it is above 7mg/dl, and symptoms may begin. The cause of the increase can be due to an imbalance in the production of uric acid or a weakness in the mechanisms of excretion or both [5].

Chronic kidney disease is defined as the presence of an abnormality in renal structure or function that persists for more than 3 months. The extent of kidney damage is assessed through the 5 stages of chronic kidney disease [6]. The stage is usually determined by 1or more of the following: (1) Glomerular filtration rate (GFR), (2) urinary sediment abnormalities, albuminuria, (3) histology, or imaging suggesting renal damage [6]. It is a major public health problem; in the United States, CKD affects approximately 1 in 6 adults. [7] The prevalence of CKD is higher in the elderly and males [8]. CKD can progress to end-stage renal disease (ESRD), in which a GFR of 15 mL/min or less can lead to early mortality [9].

We review the current literature to discuss the possibility of using uric acid as an indicator of renal impairment [10]. A lot of research has been done for years to prove the relationship between CKD and gout [11]. The association between gout and CKD is described as mutual, and CKD is an independent risk factor for gout [12]. In Chronic kidney disease, elevated plasma uric acid has been explained by a decreased glomerular filtration rate (GFR). Hyperuricemia is one of the most important diagnostic signs of gout [13]. Reducing uric acid is one of the most important therapeutic measures for chronic kidney disease to prevent the development of weakness and cardiovascular events [14]. Two studies were conducted in France to study the prevalence of gout among those suffering from chronic kidney disease, and it was found that the rate does not exceed 1% [15]. In recent research conducted on 18,358 diabetic patients in New Zealand, it was found that decreased kidney function was a major factor in gout [16]. Cross-sectional analyses of data from general practice registers in the United Kingdom suggested that having a diagnosis of chronic renal failure is associated with a 2.5 times higher likelihood of concurrent gout diagnosis [17]. Due to the lack of follow-

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up of patients with chronic kidney disease and the lack of clinical diagnosis, these studies reduced the risk of developing gout [18]. The main objective of this study is to prove the prevalence of gout among end-stage renal disease (ESRD) patients, to study the possibility of using uric acid as an indicator of impaired kidney function, and that chronic kidney disease is one of the risk factors for gout.

Methods

The study was conducted in the State of Libya in Tripoli at the Tripoli Kidney Services Center on patients with renal failure. The number of samples used was 121 to study the extent of gout development in ESRD patients. The patients were randomly selected for different ages. The study period was from April to July 2022. Blood samples were taken from the patients with renal failure at the center.

Three basic analyses were used: the kidney function test, which includes urea and creatinine. This is to prove the deterioration of the kidney condition and that all patients are in the fifth stage of chronic kidney disease, and the level of uric acid in the blood to determine the prevalence of gout among patients. A blood sample is taken from the patients before the washing session and before the heparin injection is administered.

The sample is taken from the veins at 2 mL for the two exams and a plane tube is used and left until the sample clots at room temperature for 10 minutes to ensure the purity of the serum and placed in a centrifuge at a speed of 4000 rpm for 5 minutes, using absorbance photometry method and turbidimetry which are a reader by Cobas Integra 400plus analyzer, the reagent from Roche company was used.

The normal range of uric acid is 2.00-7.00mg/dl. Normal range of urea10-50mg/dl. The normal range of creatinine is 0.3-1.3 mg/dl. Data were calculated through frequencies. The descriptive statistics includes summarizing and organizing data.

Results

The sex distribution of the study participants is shown in Figure 1. The representation of both male and female patients allows for a comparative analysis across genders, highlighting any sex-specific trends in renal function markers.

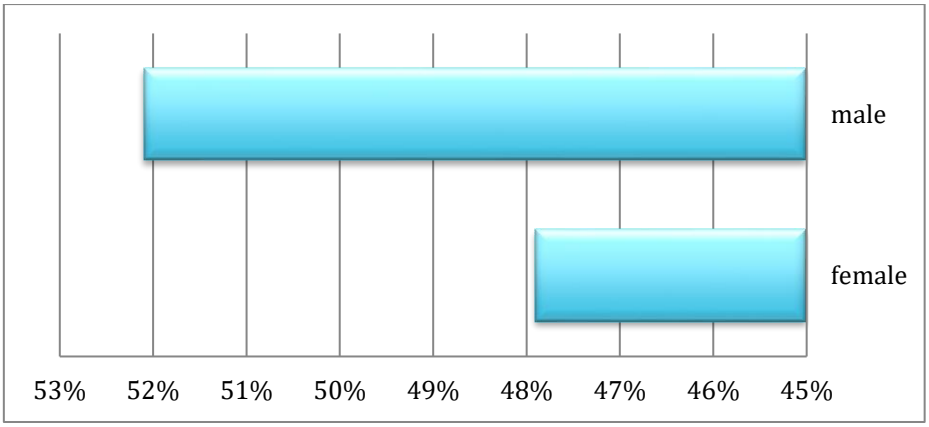

Figure 1. The ratio of male and female ESRD patients in the statistics

Urea status in the female study cases, the percentage of normal urea results among patients is (0%) and the percentage of high results is (100%). As shown in Figure 2-A, all male ESRD patients (100%) exhibited elevated urea levels. This indicates a universal impairment in the kidney's ability to excrete nitrogenous waste among the male cohort. Figure 2-B presents a similar pattern in female patients, with 100% also displaying high urea levels. This finding suggests that regardless of sex, patients with ESRD universally experience severe disruptions in urea clearance.

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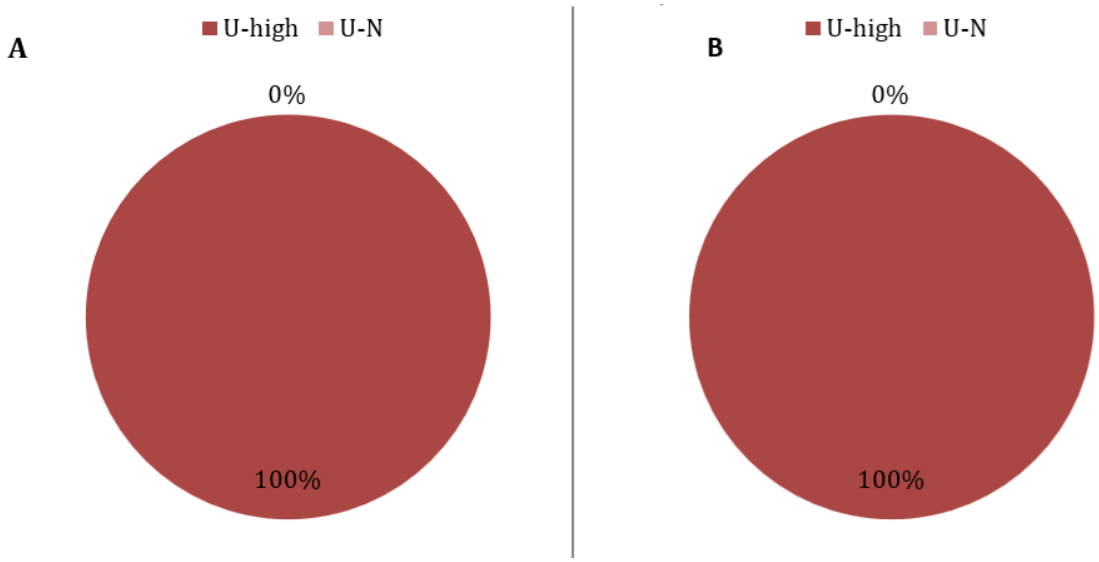

Figure 2. Male and female urea results

Table 1 summarizes all the results of the analyses that include urea, creatinine, and uric acid. Urea status in the male study cases the percentage of normal urea results among patients is (0%) and the percentage of high (100%). These markers are essential in evaluating renal function and are often elevated in ESRD patients due to impaired excretion mechanisms.

Table 1. Overview of Renal Function Markers: Urea, Creatinine, and Uric Acid Levels

Variables		**Test results**					
Gender	Total No. (121)	CR-N	CR-AB	U-N	U-AB	UA-N	UA-AB
Male	63	0	63	0	63	34	29
Female	58	0	58	0	58	31	27

Note: CR-N: creatinine Normal, CR - AB: creatinine abnormal, U - N: urea normal, U- AB: urea abnormal UA -N: uric acid normal, UA- AB: uric acid abnormal

Figure 3 compares urea levels across both sexes and reveals a striking similarity in the severity of uremia. This uniformity underscores the systemic impact of ESRD on nitrogen metabolism, reinforcing that sex does not appear to mitigate or exacerbate urea accumulation in this cohort.

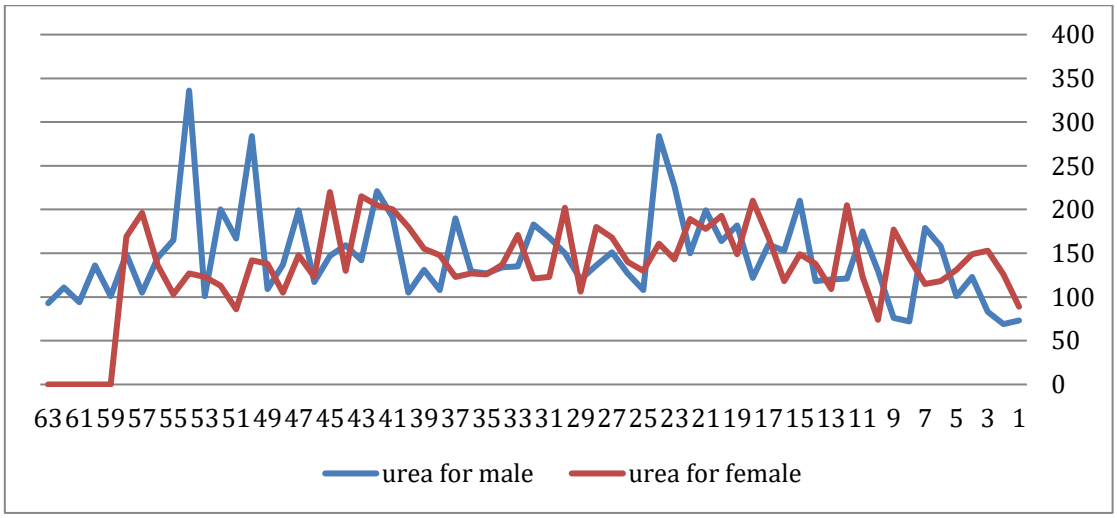

Figure 3. Comparison of urea results between males and females

Creatinine, another critical indicator of renal function, was similarly elevated in all male patients (Figure 4-A) and all female patients (Figure 4-B), with no cases falling within the normal reference range.

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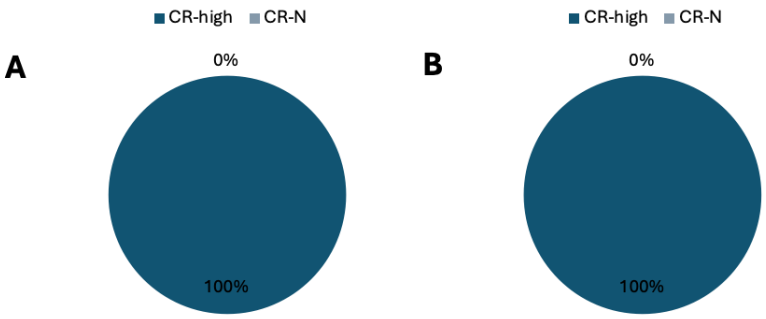

Figure 4. Male (A) and female (B) creatinine results

Figure 5 offers a visual comparison, illustrating equivalent elevations across sexes. Elevated creatinine in all patients is expected in advanced renal failure and is reflective of the drastic reduction in glomerular filtration rate (GFR).

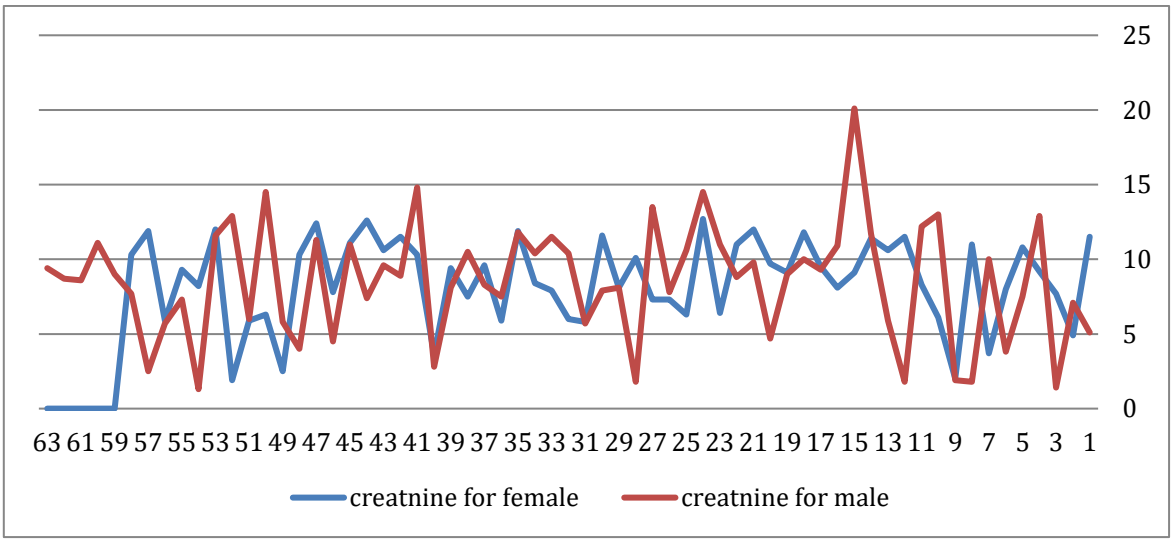

Figure 5. Comparison of creatinine results between males and females

Unlike urea and creatinine, uric acid levels showed more variation. As seen in Figure 6-A, 54% of male patients had normal uric acid levels while 46% had elevated levels. For female patients, Figure 6-B shows a similar distribution, with 53% exhibiting normal uric acid levels and 47% elevated. This near-equal distribution suggests that uric acid levels in ESRD patients are more influenced by factors beyond renal filtration alone, such as diet, metabolic rate, and use of medications like diuretics.

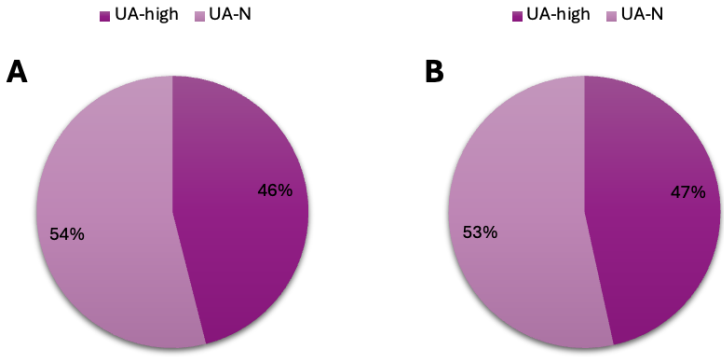

Figure 6. Male (A) and female (B) UA results

Figure 7 compares uric acid levels across sexes and indicates that the proportion of patients with hyperuricemia is nearly equal between males and females. This finding is clinically important as hyperuricemia is a major risk factor for gout, and the data suggest a comparable risk of gout development among both sexes in the ESRD population.

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The overall prevalence of gout in the study population was 46%, as depicted in Figure 7. This suggests that nearly half of all ESRD patients suffer from gout, highlighting the importance of managing uric acid levels even in a population already burdened by renal dysfunction.

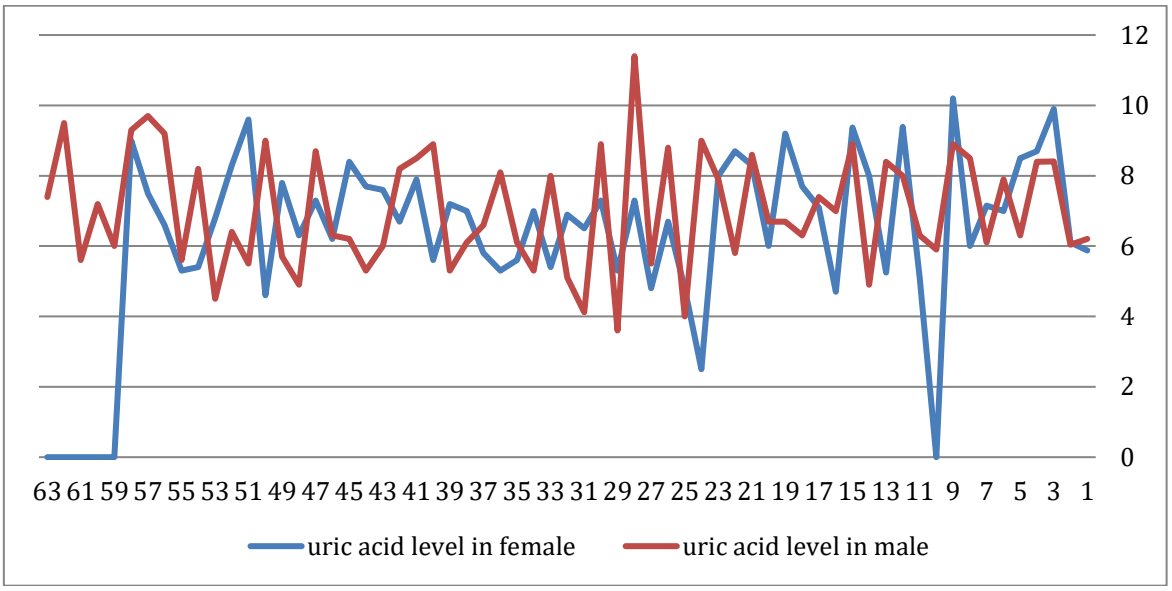

Figure 7. Comparison of uric acid results between males and females

The prevalence of gout among the study cases was 54% who did not suffer from gout, and 46% had gout (Figure 8).

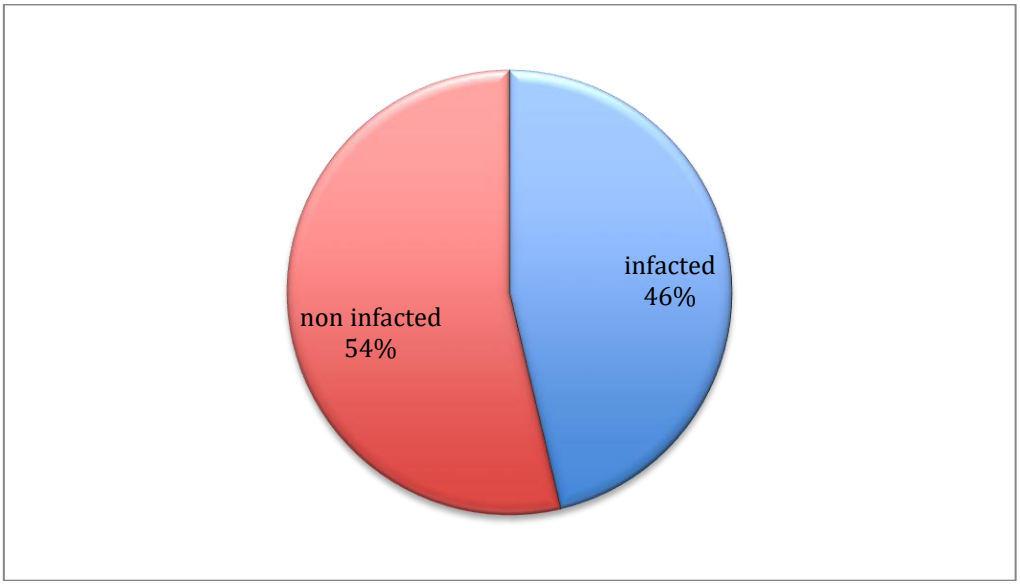

Figure 8. The prevalence of gout among patients with ESRD

Table 2 presents the descriptive statistics for the key variables. The average age of participants was 53.07 years, with a slightly higher mean in males (55.13) compared to females (50.84). The uric acid levels were slightly higher in males (7.04 mg/dL) than in females (6.87 mg/dL), though the difference is not likely clinically significant. Average creatinine levels were high in both sexes—8.48 mg/dL in males and 8.63 mg/dL in females—well above the normal reference range, confirming severe renal impairment. The mean urea level was approximately 147 mg/dL for both sexes, reinforcing the findings of 100% elevation across the study population.

The elevated values of these renal markers in all patients suggest a homogeneously advanced stage of kidney disease. The substantial prevalence of hyperuricemia and gout further emphasizes the need for integrated metabolic management in ESRD care protocols. The comparable findings between males and females indicate that both sexes are equally vulnerable to renal metabolic disturbances, and clinical approaches should therefore be gender-neutral but personalized based on individual biochemical profiles.

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Table 2. Descriptive statistics for each analysis to study the mean and standard deviation for each sex

Variables	N	Minimum	maximum	Male (mean)	Female (mean)	All mean	Standard deviation
Age	121	25	79	55.126	50.844	53.07	14.370
Uric acid	121	2.23	11.40	7.036	6.870	6.95	1.659
creatinine	121	1.30	20.10	8.48	8.63	8.57	3.373
urea	121	69	336	146.984	146.982	146.98	43.897

Discussion

In this study, we estimate the prevalence of gout among patients with ESRD, which is the fifth stage of chronic kidney disease. It is the same stage in which dialysis begins, all patients included in the study are in stage 5 of chronic kidney disease, as it was illustrated in the results of the urea analysis and creatinine in figures (2 and 4) all results were high, since hyperuricemia is one of the most common problems facing patients with kidney failure. It was found that dialysis sessions cannot treat high uric acid in the blood, and other methods, such as medications or special diets, must be applied to reduce gout complications. This is what we also encountered in all patients, as the level of urea and creatinine decreased after dialysis, but uric acid remains constant [19]. In this research, we clarify the levels of both urea and creatinine in males and females to determine which sexes have higher results. It also studies whether there is a relationship between urea, creatinine, and uric acid can be proven. It was found that the mean urea results for males is 146.984 and for females 146.982, where it is very close, and the overall mean is 146.98 (SD 43.8) as shown in Table 2. The results of creatinine, the mean results for males are 8.48, while for females it is 8.63, as the mean of females is slightly higher than males, and the overall mean is 8.57(SD 3.3) (Table 2). We also made curves to compare the results of male and female analysis of creatinine and urea in figures (3 and 5). It was noted that males had the highest results in both analyses, but overall, the results are similar. It has been statistically proven by using a Pearson correlation that there is a direct relationship between urea and creatinine (P=0.000, R=0.335), urea and uric acid (P=0.005, R=0.255). Several studies have shown that hyperuricemia is more likely when the glomerular filtration rate (GFR) decreases [20].

Because the kidneys are responsible for 70% of the excretion of uric acid, hyperuricemia in most cases in CKD patients is the result of the inability of the kidneys to excrete, not because of increased production [21]. Data obtained from NHANES showed that 8.3 million Americans have gout, and 71% have CKD higher than stage 2 (GFR <60) [21]. It is close to another study conducted in the German chronic kidney disease (GCKD) cohort, which included 5,085 patients. It was found that the prevalence of gout among patients with GFR<30ml/min /1.73m² was 35% [22]. Many studies support that the prevalence of gout is between 40%-60% among patients with stage 5 chronic kidney disease. The results of this research are very consistent with the results of our research, where it was found that 46.3% of ESRD patients who undergo dialysis suffer from gout (P <0.000).

Knowing that some patients included in the study take urate-lowering therapy (ULT), which means that the percentage may be higher than stated, to compare its prevalence between males and females, it was found that the prevalence rate in males is 46%, while in females it is 47%. The percentage is very close figures (6 and 8) [23]. Although most studies confirm that its prevalence is higher in males than in females, and that women with hyperuricemia are older and have comorbidities and obesity. On the contrary, in our study, the prevalence of gout was slightly higher in women than in males, and the mean age of females was 54.2 years lower compared to the mean age of males of 56.4 years; this was surprising (Table 2) [24]. In a study evaluating the duration of the onset of gout in ESRD patients, 5% are diagnosed in the first year and 15.4% in the first five years of dialysis, through this research, we estimated the period in which uric acid begins to rise in ESRD patients and correlate it with the duration of the start of dialysis, but the period was not very specific, as it starts from1 year from the beginning of dialysis up to 19 years among the center's patients, but in most cases symptoms begin to appear after the fifth year of dialysis [25].

Conclusion

The study was conducted on patients with ESRD stage 5 of chronic kidney disease to study the prevalence of gout, which was originally a disorder in the metabolism of purine, but in the case of patients with renal failure, the main cause is an impairment in the secretion of uric acid by the kidneys. The other problem is that uric acid does not decrease after a dialysis session, such as urea and creatinine, and it needs special treatments. As a result, the concentration of uric acid in the blood increases (hyperuricemia). It was found that 46 % of ESRD patients in our study suffer from gout, The prevalence rate was slightly higher in females than males, and the average age of women was younger, one of the important relationships that were then proven statistically is the direct relationship between urea, creatinine and urea, uric acid.

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Recommendations

We aspire to link the mutual relationship between gout and chronic kidney diseases for gout sufferers and non-infected people and to study the impact of gout on the kidneys and kidney impairment on the development of gout, on a larger number of cases and over a longer period of time.

Conflicts of Interest

The authors declare no conflicts of interest.

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المستخلص

النقرس هو اضطراب في أيض البيورين يؤدي إلى فرط حمض يوريك الدم، ولكن من ناحية أخرى، في مرضى الفشل الكلوي في مراحله النهائية، فإن السبب الرئيسي هو انخفاض معدل الترشيح الكبيبي(GFR) ، بسبب ارتفاع حمض اليوريك في الدم، ويتراكم حمض اليوريك في السائل المحيط بالمفاصل والأنسجة الرخوة، ويشكل بلورات صغيرة تسمى يورات أحادية الصوديوم. يوصف الارتباط بين النقرس ومرض الكلى المزمن بأنه متبادل، ويُعد مرض الكلى المزمن عامل خطر مستقل للإصابة بالنقرس. تسعى هذه الدراسة إلى تحديد مدى انتشار النقرس بين مرضى الفشل الكلوي في مراحله النهائية (ESRD).أجريت الدراسة في دولة ليبيا في طرابلس في مركز خدمات الكلى بطرابلس على مرضى الفشل الكلوي. بلغ عدد العينات المستخدمة 121 عينة، وكانت فترة الدراسة من أبريل إلى يوليو 2022. أجريت ثلاثة تحاليل لجميع المرضى لليوريا والكرياتينين لتقييم تدهور الكلى وحمض اليوريك لتشخيص النقرس. مرضى الفشل الكلوي في مراحله النهائية. وكانت جميع نتائج اليوريا والكرياتينين مرتفعة، وبلغ متوسط نتائج اليوريا للذكور (146.984) وللإناث (146.982)، وكان متوسط الكرياتينين للإناث (8.63) أعلى من الذكور (8.48). كما وجد أن تصفية حمض البوليك أثناء الغسيل الكلوي لا علاقة لها بتصفية اليوريا والكرياتينين، وقد بلغ معدل انتشار النقرس بين مرضى الفشل الكلوي المزمن في هذه الدراسة 46.3(p < 0.000) %، ومعدل الانتشار عند الذكور 46٪، بينما عند الإناث 47٪، ومتوسط عمر الإناث 54.2 والذكور 56.4.

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