International Journal of Biology and Life Sciences
ISSN: 2957-9511 | Vol. 3, No. 3, 2023

Clinical Significance of Serum Creatinine, Urea Nitrogen and Uric Acid Levels in Patients with Chronic Renal Failure

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Abstract: Chronic renal failure (CRF) is a clinical syndrome characterized by chronic progressive renal parenchymal damage caused by various reasons, resulting in obvious atrophy of the kidney and inability to maintain basic functions. Metabolite retention, imbalance of water, electrolyte and acid-base balance, and systemic involvement of various systems are the main manifestations in clinic. Due to its complicated metabolic disorder, once it develops to the stage of renal failure or even the end stage of uremia, it is often more difficult to treat other renal diseases. Therefore, early detection, early diagnosis and early treatment of chronic renal failure are of great significance to improve the therapeutic effect of patients. In this project, 150 subjects were divided into chronic renal failure group (75 cases) and healthy group (75 cases). By analyzing the data of renal function indexes (blood creatinine, urea nitrogen, uric acid) of patients with chronic renal failure and healthy control group, the clinical significance of blood creatinine, urea nitrogen and uric acid level detection for chronic renal failure was discussed, and the importance of blood creatinine, urea nitrogen and uric acid monitoring for chronic renal failure was explained, providing scientific basis for timely prevention or delay of chronic renal failure. Biochemical tests were carried out on 150 serum samples from the combined healthy group of chronic renal failure. the values of uric acid, urea nitrogen and serum creatinine detected by the two groups were counted and treated with statistical methods. the results showed that the values of urea nitrogen, uric acid and serum creatinine in the chronic renal failure group were significantly higher than those in the healthy group, and the same indexes in the two groups were compared, $P<0.05$. statistics showed that the difference of the same data between the two groups was significant. Serum uric acid, urea nitrogen and creatinine are easy to detect and the test results are relatively accurate. The results can be used for auxiliary diagnosis of patients with chronic renal failure and have clinical significance for diagnosis of patients with chronic renal failure.

Keywords: Chronic Renal Failure; Serum Creatinine; Urea Nitrogen; Uric Acid.

1. Introduction

Chronic kidney failure (CRF) is not a separate disease, and is sometimes referred to as uremia. But due to the combination of various causes caused by kidney damage and development of deterioration, once when chronic kidney failure progress to the end stage, chronic kidney failure patients with kidney function close to the normal kidney function of 10% to 15%, will lead to a series of clinical syndromes. The glomerular filtration rate is reduced, and the reabsorption of urea nitrogen, water, and sodium by the renal tubules is relatively increased. At this time, the patient's serum urea nitrogen and blood creatinine are increased, and the uric acid detection level is increased.

Urea nitrogen is the nitrogen in urea, which is usually used in our country to express the concentration of urea nitrogen with urea. Amino acids are metabolized in the liver to produce toxic ammonia, which can be hydrolyzed to non-toxic UN, which is also one of the manifestations of liver detoxification function, and urea nitrogen (BUN) in serum is usually filtered through the glomeruli and discharged from the body [7]. The reference value ranges from 2.9 to 7.5mmol/L (8 to 21mg/dl). An increase in this level is commonly seen in renal insufficiency.

Domestic and foreign scholars and medical personnel believe that blood creatinine is endogenous blood creatinine, and muscle generates endogenous creatinine after a series of metabolic reactions. Creatine is produced in muscles through a series of reactions, and creatine is produced into blood creatinine after a series of irreversible non-enzymatic dehydration reactions in muscles, which is released into the blood and then eliminated from the body through urine [10]. Therefore, the relationship between creatinine production and muscle mass is positively correlated, and the production of creatinine is not easily affected by diet. Because creatinine is a small molecule, it can be directly filtered out by the glomeruli, so the blood creatinine is not reabsorbed by the renal tubules. No matter how much urine is produced daily, the amount of serum creatinine ultimately excreted in urine will not be affected.

Birds and reptiles in the body after a series of metabolic reactions to produce the product of uric acid, in the normal human urine, most of the components are urea, uric acid content is relatively small. Purines undergo a series of metabolic reactions in the body to produce uric acid as the final product. The uric acid produced does not remain in the body, but is filtered by the kidneys and excreted in the urine.

When the condition of chronic renal failure progresses to the stage of renal failure, or even uremia, the treatment is often more difficult than other kidney diseases, so the early detection, early diagnosis and early treatment of chronic renal failure are of great significance to improve the treatment effect of patients. Therefore, it is necessary to investigate the clinical significance of the changes of serum urea nitrogen, uric acid and creatinine in the diagnosis of chronic renal failure.

2. Research Background

2.1. Overview of Chronic Renal Failure

Chronic kidney failure (CRF) is not a separate disease, and is sometimes referred to as uremia. But due to the combination of various causes caused by kidney damage and development of deterioration, once when chronic kidney failure progress to the end stage, chronic kidney failure patients with kidney function close to the normal kidney function of 10% to 15%, will lead to a series of clinical syndromes. Due to the above reasons, metabolic disorders appear in the various systems of patients with chronic kidney failure, and once they develop to the stage of renal failure, even uremia is generally more difficult to treat than his kidney disease. Therefore, it is of great significance for the early detection and diagnosis of chronic renal failure, and for improving the treatment effect of patients and the possibility of cure. There are multiple causes of chronic renal failure such as chronic pyelonephritis, diabetic nephropathy and so on.

2.2. Clinical Presentation and Laboratory Examination of Patients with Chronic Renal Failure

2.2.1. Clinical Presentation

First, the digestive system

Patients with chronic kidney failure have symptoms in the digestive system, such as gastrointestinal bleeding and ulcers. The digestive system is the first and most common symptom in patients with chronic renal failure.

Second, the blood system

1, uremia patients will show symptoms of anemia. The severity of patients with uremia (kidney function) is proportional to the severity of their anemia, and decreased erythropoietin (EPO) [2] is the main cause of anemia.

2. Bleeding tendency: The reason is caused by elevated toxins, which can be corrected by dialysis.

3, abnormal white blood cells: abnormal white blood cells are usually manifested as leukopenia, and patients are more prone to infection due to some of the weakened abilities of white blood cells, such as phagocytosis and bactericidal ability. Patients can improve after dialysis.

Third, cardiovascular system

1. Hypertension: Hypertension of varying degrees will occur in most patients with chronic renal failure. When hypertension occurs in patients with renal failure, some symptoms will appear, such as left ventricular hypertrophy, arteriosclerosis and other symptoms [12].

2. Heart failure: Patients with chronic renal failure often show symptoms of cardiomyopathy, which is caused by water and sodium retention and high blood pressure [15].

Fourth, the nervous and muscular system

1. Early stage: The main manifestations of the early nervous and muscular system are fatigue, poor sleep quality, and insufficient concentration.

2. Advanced stage: Peripheral neuropathy is the main manifestation of advanced nervous and muscular system, and sensory nerve is more significant than motor nerve [19].

Five. Respiratory system

1. When patients have acidosis, their respiratory system is manifested as deep and long breathing [18].

2, some patients will also have uremic bronchitis, pneumonia (butterfly wings), pleurisy and so on.

Six. Endocrine system performance

Insufficient erythropoietin and excessive renin-angiotensin II in the kidney are the main manifestations of endocrine dysfunction of the kidney itself [2].

Seven. Concurrent infection

Pulmonary infection was the most common co-infection. Fever during infection may sometimes be less pronounced than normal.

Eight. Skeletal changes

Uremia, which causes bone changes, is also known as renal bone disease.

Nine. Skin symptoms

Skin symptoms can be characterized by extremely itchy skin, uremic cream deposition, and uremic face are the main symptoms of skin symptoms, and generally dialysis cannot improve this symptom.

2.2.2. Laboratory Examination

1. Commonly used laboratory tests

In clinical practice, urine routine, renal function and even imaging are usually used as laboratory tests for patients with chronic renal failure

2. Imaging examination

The commonly used imaging methods include renal CT, magnetic resonance and so on.

2.3. Common Blood Biochemical Indicators

2.3.1. Urea Nitrogen

Urea nitrogen is the nitrogen in urea, urea or urea nitrogen can be used to express the concentration of urea nitrogen, and it is usually used to express the concentration of urea nitrogen with the latter in China. Amino acids are metabolized in the liver to produce toxic ammonia, which can be hydrolyzed to non-toxic UN, which is also one of the manifestations of liver detoxification function. Urea nitrogen (BUN) in serum is usually filtered through the glomeruli and discharged from the body [7]. The reference value ranges from 2.9 to 7.5mmol/L (8 to 21mg/dl). Uric acid levels above this range are also known as azotemia and are commonly associated with renal insufficiency. The increase of urea nitrogen is usually seen when the glomerular filtration rate (GFR) is as low as 50%, and the sensitivity to urea nitrogen is poor. The causes of elevated BUN are usually extra-renal factors such as wasting diseases, gastrointestinal bleeding, and dehydration, and elevated urea nitrogen levels can also be caused by a high-protein diet, so urea nitrogen is not the best indicator to evaluate GFR. Urea nitrogen levels can usually be determined by enzymatic methods, and kits are generally available for determination [6].

Most of the protein metabolized by the human body is ultimately produced as urea nitrogen. Amino acids undergo a series of reactions in the body to produce NH3 and CO2, which then react in the liver to produce urea, and one gram of protein in the body through a series of reactions can produce 0.3 grams of urea. The content of nitrogen in urea is about 50%. The generated urea is excreted out of the body through the kidney, and the filtered urea is reabsorbed in the renal tubules, and the reabsorption of urea by the renal tubules is easily affected by the urine flow rate and is inversely proportional to it [6].

The indexes of urea nitrogen and serum creatinine have similar changes in the early stage of renal injury, and serum urea nitrogen nitrogen will be higher than the normal range in the early stage of renal injury. Serum urea nitrogen concentrations rise sharply when renal function is impaired to less than 50% of normal value. Under normal circumstances, the ratio of serum urea nitrogen to blood creatinine is about

10:1, when the following conditions such as high-protein diet, high catabolic state, etc., will cause these ratios to rise rapidly, sometimes reaching 20 to 30.

In summary, serum urea nitrogen is not only easily affected by diet, but also affected by renal blood flow.

2.3.2. Serum Creatini (Scr)

Domestic and foreign scholars and medical personnel believe that blood creatinine is endogenous blood creatinine, and muscle generates endogenous creatinine after a series of metabolic reactions. Creatine produced in muscle does not exist in the form of creatine, but is produced into blood creatinine after a series of irreversible non-enzymatic dehydration reactions in muscle, and then released into the blood and finally eliminated from the body through urine [10]. Therefore, the relationship between creatinine production and muscle mass is positively correlated, and the production of creatinine is not easily affected by diet. Because creatinine is a small molecule, it can be directly filtered out by the glomeruli, so the blood creatinine is not reabsorbed by the renal tubules. Because of the above properties of serum creatinine, it can be used as a good indicator to evaluate kidney function. The normal value of serum creatinine (SCr) varied between the sexes: 53 to 106 micromo/L (0.6 to 1.2 mg/dL) for males; For women, it was 44-97 micromo/L (0.5-1.1 mg/dL); In children, it was 24.9~69.7μmol/L.

As previously discussed, serum creatinine is mainly filtered through the glomerulus, so the amount of serum creatinine is closely related to the filtration capacity of the glomerulus. Therefore, when the filtration capacity of the glomerulus is lower than the normal value, the serum creatinine measurement value will also increase. Therefore, when the serum creatinine value is higher than the normal value, it can reflect the damage of kidney function. However, although serum creatinine can be used as an indicator of renal impairment, it is not a sensitive indicator. The reason is that only when the glomerular filtration rate is less than one-third of the normal glomerular filtration rate, the blood creatinine will appear more obvious rise. In summary, in the early stage of renal function injury, the status of renal function cannot be determined by measuring serum creatinine. Only when the glomerular filtration rate drops to a certain extent, can the renal function injury be determined by blood creatinine. Therefore, in the early days of kidney injury, patients will not feel obvious discomfort. When the kidney injury is severe, patients will show symptoms such as nausea and vertigo, and the serum creatinine value will increase significantly [14].

We should also note that when the glomerular filtration rate does not fall below 50% of the normal glomerular filtration rate, serum creatinine will be in the normal range [19].

In addition, we should also note that it is not necessary to increase serum creatinine only when the glomerular filtration rate is decreased, but also when symptoms such as hyperthyroidism and acromegaly are present. Therefore, when we find a significant increase in the serum creatinine value of patients, we should also do some related kidney tests [20].

When testing the serum creatinine value, the serum creatinine value at different times will not be fixed, but due to fluctuations. When the serum creatinine value increases significantly due to the injury of renal function, the reason is that the kidney has been invaded by some causes. The phenotype of the damaged kidney cells will be transformed in the first step, followed by pathological changes, which will lead to the conversion of fibroblasts into myofibroblasts in the kidney. At the same time, It also invades other normal kidney tissues, and its lesions are similar to those described above, eventually leading to the damage of the kidney from point to surface, from local to whole, and finally to the whole kidney [16]. When the kidney occurs comprehensive damage, due to the excretory function of the disorder, will lead to the accumulation of toxins in the body, at the time of examination, there will be a rise in serum creatinine, urea, uric acid levels. Patients with chronic renal failure also have edema and high blood pressure in terms of physical symptoms [7].

We mentioned earlier that serum creatinine is produced by creatine in muscle through a series of reactions. According to the relevant personnel, 1mg of creatinine can be produced by 20g muscle through a series of reactions. Creatinine is then filtered as a small molecule by the glomeruli. We define endogenous creatinine as serum creatinine, and some meat foods can also generate exogenous creatinine after reaction in the body. Here we only study endogenous creatinine, so we will not go into details. When the amount of meat consumed by the human body is stable, the production of endogenous creatinine produced by the body will also be stable [14].

2.3.3. Uric Acid

Birds and reptiles undergo a series of metabolic reactions in the body to produce uric acid. In normal human urine, the vast majority of the components are urea, the content of uric acid is relatively small. Purine substances after a series of metabolic reactions in the body to produce the final product is uric acid, its essence is trioxy purine, chemically its alcohol form is weak acid. Uric acid, which is produced by the complex metabolic reaction of purine substances in the body, is filtered by the kidneys and excreted in urine. Clinically, we define hyperuricemia when the uric acid content in the blood is higher than 420μmol [18].

Under normal circumstances, the uric acid content of normal human body is about 1200mg, of which the uric acid generated by normal human body through complex metabolic reaction is about 600mg every day, at the same time, the amount of uric acid excreted by normal human body is about 600mg every day, so in normal human body, the amount of uric acid excreted by normal human body is about 600mg. The new uric acid produced each day is in balance with the uric acid to be excreted in the urine that day. On the contrary, when the uric acid produced in the body is greater than the uric acid excreted in urine or the excretion mechanism of the human body deteriorates, the concentration of uric acid in the blood in the body will rise, and once the concentration exceeds 7 mg/dl, the body fluid will become acidic, which will affect the function of normal somatic cells [18]. If the concentration of uric acid in the body is higher than normal for a long time, it may lead to the occurrence of gout, but gout is not necessarily caused by uric acid, but can also be caused by other factors.

Unlike serum creatinine, uric acid is not a small molecule. In the kidney, uric acid is filtered through the glomerulus first, and changes in uric acid content in body fluids can be used to fully reflect the immune function, metabolism and other functions of the human body [5].

The main reasons for the formation of uric acid are as follows: First, excessive intake of purines: As we discussed earlier, purines in meat foods eventually produce uric acid after complex metabolic reactions in the human body, so the content of uric acid is positively correlated with the number of purines ingested by the human body. 2. Abnormal renal processing of urine.

2.3.4. Changes in Other Indicators

In the late stage of renal failure, a large number of renal parenchyma is destroyed, the glomerular filtration rate is decreased, the blood NaHCO3 is decreased, and other organic acid retention is observed, so patients have different degrees of acidosis.

3. Materials and Methods

3.1. Experimental Materials

3.1.1. Object of Study

Seventy-five patients with chronic renal failure and 75 normal persons (150 in total) hospitalized in the Department of Endocrinology and nephrology of the First Affiliated Hospital of Henan University of Science and Technology from 2017 to 2019 were selected as the study subjects. The patient's medical history (gender, age) and laboratory test results (serum creatinine, serum urea nitrogen, serum uric acid) were recorded.

3.1.2. Main Instruments

Figure 1. AVDIA 2400 automatic biochemical analyzer

BAYER company launched AVDIA 2400 full automatic biochemical analyzer in recent years, overturns the traditional way of adding sample and sampling cycle, the characteristics of simple operation, less sample and reagent dosage and flexible analysis program etc., also can directly and automated sample preparation system link, and BAYEY immunity, Blood corpuscle analyzer subject combination can realize the so-called full laboratory linkage (TAL), which can help improve laboratory work process, make the medical workers can easily deal with the increasing amount of specimen, meet the demand for speed up the turnaround time in clinic. Increase productivity. Bayer Diagnostics, in line with the attitude of "promoting human health as its own responsibility", provides more advanced and high-quality laboratory optimization programs for laboratories. ADVIA 2400 integrates today's advanced science and technology to achieve the urgent needs of laboratory innovation in clinical practice. The unique "site forward" function of ADVIA 2400, in the determination of high concentration of samples, especially in enzymatic detection, due to the high concentration of enzyme in the sample, the substrate will be depleted quickly in the process of reaction, so that the reaction curve is no longer in accordance with the standard reaction curve, and the data obtained from the original reading window is not linear. This eventually leads to errors in outcome measurement. If an alert is behind the tip. The operator needs to re-dilute the sample and reexamine it. ADVIA 2400 automatically changes the reading window on the standard curve through the software. According to the preset limit, the reading window will automatically move forward to search for a linear section of the reaction curve (because the Bayer biochemical instrument adopts the design of the whole reading point). When the instrument reads 6 or more valid detection points, the instrument can detect the sample result. Thus, the standard curve is optimized and the most accurate reading point determination. "Read forward" increases the linear range of the assay and reduces dilution and retesting rates. Reduce the cost. The ADVIA 2400 also features a unique and accurate dosing system and pre-dilution function, unique microtechnology, accurate optical reaction system, and a high-tech electrolyte module (ISE) system.

In this experiment, the constant temperature and high-speed centrifugation method was used to ensure that the obtained serum was well free of fibrin clots, and the activity and substance content of serum hormones at constant temperature were stable.

3.1.3. Major Reagents

One, Creatinine determination kit (Sarcosine oxidase method)

1, the test principle

R1 was added to the sample to first remove the creatinine reaction in the sample, and R2 was added to initiate the creatinine reaction.

Creatinine + H_2O creatine

Creatine + H_2O sarcosine + urea

Sarcosine + H_2O glycine + formaldehyde + H_2O_2

H_2O_2 + 4 amino anti - than Lin + TOOS benzene quinone imine + H_2O

The color of the resulting benzoquinone imine is proportional to the creatinine concentration.

2, the main composition

R1:3 - [N] - morpholine generation tendency for 2 L, propane sulfonic acid buffer PH 7.4. Creatine hydrolase 27KU/L; Sarcosine oxidase 7.5KU/L; Vitamin C oxidase 4.7 KU/L; N-ethyl-n (2-hydroxy-3-propyl sulfonate) -3-methylaniline 0.14g/L; Preservative (Proclin300) 0.01%.

R2:3 -[N-morpholine] propane sulfonate buffer, 100mmol/L, PH 7.3; Sarcosine hydrolase 300KU/L; Peroxidase 10 ku/L; 4-aminoantipyrine 0.6g/L; Preservatives (Proclin300) 0.01%.

Note: different batches kits in the corresponding component are not interchangeable.

3. Sample requirements

Serum or plasma: stored at 2-8 ° C for seven days, frozen for long-term stability.

4. Result calculation

CERA (umol/L) = (measured A-blank A)/ (calibrated A-blank A) × calibrator concentration

(2-5)

5. Reference range

Serum (OAR) adult: male 59-104u mol/L; Female: 45-85u mol/L

This reference range is derived from references and it is recommended that each laboratory establish its own reference range.

6. Limitations of test methods

"For diagnostic purposes, CREA results should be evaluated in conjunction with history, clinical examination, and other findings." When bilirubin >171umol/L, hemoglobin >466umol/L, TG>22.8mmol/L, the results were

obviously interfered.

Two, Urea nitrogen determination kit (Urea nitase-glutamate dehydrogenase)

1. Inspection principle

Urease catalyzes the hydrolysis of urea nitrogen to ammonia and carbon dioxide. Under the catalysis of glutamate dehydrogenase, the resultant ammonia reacts with α-ketoglutaric acid and reduced coenzyme I to form oxidized coenzyme I. The production of oxidized coenzyme I is proportional to the amount of urea nitrogen in the sample.

2. Main components

Table 1. Main components of urea nitrogen assay kit

Components of	Ingredients	concentration	Ingredients	concentration
R1	Trimethylol aminomethane	120mmol/L	Sodium azide	1g/L
	Adenosine diphosphate	0.8mmol/L	Glutamate dehydrogenase	2KU/L
	Alpha-ketoglutaric acid			10mmol/L
R2	Reduced coenzyme I	0.5mmol/L	urease	5KU/L

3. Sample requirements

Specimens for fasting serum.

4. Reference values (reference range)

2.86-8.20 mmol/L, the data refer to the "National Clinical Laboratory Operation procedures", and experimental verification on 200 healthy individuals, for reference only. It is recommended that laboratories develop their own reference ranges.

Three, Uric acid determination kit (uric acid enzymatic method)

1. Inspection principle

Uric acid by the catalytic oxidation of allantoin and uric acid oxidase, hydrogen peroxide, after oxidation of hydrogen under the action of hydrogen peroxide enzyme and phenol derivatives and 4 - amino anti than Lin imide reaction generated after red quinone pigments, the color intensity is proportional to the content of uric acid, uric acid levels can be calculated.

2. Main components

Table 2. Main components of uric acid determination kit

Components of	Ingredients	concentration	Ingredients	concentration
R1	phosphate	100mmol/L	Sodium azide	1g/L
	2,4,6, -triiodine-3-hydroxybenzoic acid			5mmol/L
R2	Uric acid enzyme	3KU/L	4-aminoantipyrine	4.5mmol/L
	peroxidase	40KU/L	Sodium azide	1g/L

Calibrator: uric acid, the value can be traced to the international reference material SRM909c. The components in different batch kits are not interchangeable.

3. Calculation

The calibration curve was drawn by taking the change value of the absorbance of the calibrator ΔA (A calibration-A blank) as the ordinate, and its corresponding concentration C calibration as the abscissa. According to the sample of Δ A (A determination - A blank) to find the corresponding sample concentration on the calibration curve.

4. Reference values

The normal distribution method was used to calculate the reference range of 200 healthy individuals in body side, and the reference range was male: 210-430 umol/L; Female: 150-360 umol/L, for reference only, it is recommended that laboratories develop their own reference ranges.

3.2. Research Methods

Take all of the patients admitted to hospital to get up early the next day fasting venous blood (or check-up crowd fasting venous blood), with 3 to 5 ml, coagulant vacuum acquisition in low temperature centrifuge 2500 r/min, 15 min after the use of disposable suction drain upper serum put in clean sterile dry tube, again with centrifuge to 2500 r/min, 10 min, A second separation of serum was obtained. Blood urea nitrogen, uric acid, creatinine determination by Siemens AVDIA 2400 fully automatic biochemical analyzer.

3.3. Statistical Methods

All data were expressed as mean ± SD. The t test was used to compare the means of multiple groups, the t test was used to compare the means of the experimental group and the control group, and the comparison between the means of the two samples.

3.4. Quality Control of the Trial

First, in the detection process, we must ensure the accuracy of the experimental data measured, in order to ensure this accuracy, we must carry out strict quality control before the experiment.

Second, for patients with suspected chronic renal failure, before drawing the patient's blood, it is important to inform the patient to eat a light diet and not eat irritating food before preparing the blood.

Third, when some patients suffer from platelet diseases, blood diseases, etc., do not use large needles to draw blood when drawing blood, should use small needles to draw blood, and when drawing blood, at least massage the patient's blood for ten minutes.

4. After the specimen collection is completed, the time of blood submission and temperature should be controlled, and the time between the completion of blood collection and the specimen detection should not exceed two hours. Otherwise, the accuracy of the final result of the test will be affected.

5. When the blood sample is centrifuged, it is necessary to carefully check the balance. When the centrifugation is completed and the specimen is taken out, do not let the specimen oscillate, so as not to affect the serum.

4. Results and Discussion

4.1. Results

In 75 cases of patients with chronic renal failure (25 patients with 50 patients with male, the female) and 75 (44

patients with male and female patients with 31 cases) cases of healthy group of centrifugal blood samples for testing, to ensure specimens from the collected samples are not yet received damage and test results are accurate, the levels of urea nitrogen, serum creatinine and uric acid between the two groups were analyzed by statistical methods. In the results of the two groups of tested serum, the mean ± standard deviation of urea nitrogen in the healthy group was 5.604± 1.10854mmol/L, the mean ± standard deviation of serum creatinine was 60.251±10.526μmol/L, and the mean ± standard deviation of serum creatinine was 60.251 ± 10.526 μmol/L. The mean ± standard deviation of uric acid was 300.720±86.215μmol/L, urea nitrogen was 29.567± 11.885mmol/L, creatinine was 922.760±336.094μmol/L, uric acid was 502.640±402.415 μmol/L in chronic renal failure group. The mean value and variance of urea nitrogen, serum creatinine and uric acid in patients with chronic renal failure and healthy group were analyzed by statistical methods. Finally, P<0.05 was obtained, which showed that there were significant differences in serum creatinine, urea nitrogen and uric acid levels between chronic renal failure group and healthy group.

Table 3. Comparison of serum uric acid, urea nitrogen and creatinine levels between healthy group and chronic renal failure group

Group of groups	Uric acid (umol/L)	Urea nitrogen (mmol/L)	Creatinine (umol/L)
The healthy group	300.72±86.215	5.045±1.109	60.252±10.526
Chronic renal failure group	502.64±402.416*	29.568±11.885*	922.760±336.094*

Note: * indicates that the chronic renal failure group compared with the healthy group, P<0.05, indicating that the difference is statistically significant.

In ensuring specimens from the collected samples are not yet received damage and test results are accurate, we from Table 3 can see very clear, health and chronic renal failure group of uric acid, urea nitrogen, serum creatinine level is comparatively large difference.

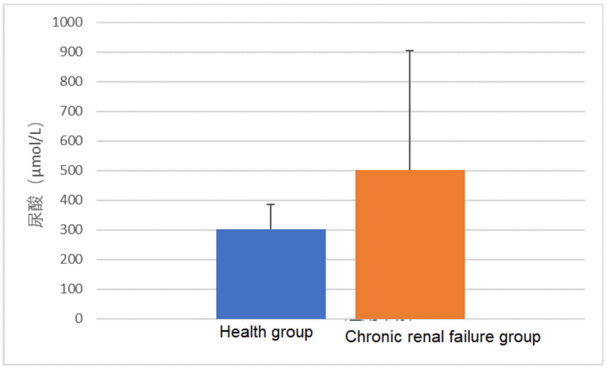

Figure 2. Comparison of uric acid level between healthy group and chronic renal failure group

As can be seen from Figure 2, there is a relatively large difference in uric acid level between the healthy group and the chronic renal failure group. When the occurrence of chronic renal failure leads to the decline of renal function, the excretion of uric acid will decrease, which will lead to the increase of uric acid level.

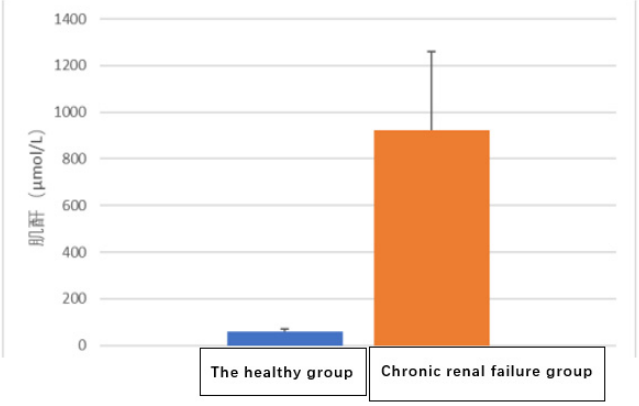

Figure 3. Healthy group and the comparison of chronic renal failure group serum creatinine levels

By figure 3 can see out, health and chronic renal failure group the levels of serum creatinine are large, these are indirect proof when kidney function decline to a certain conclusion serum creatinine will appear obvious rise.

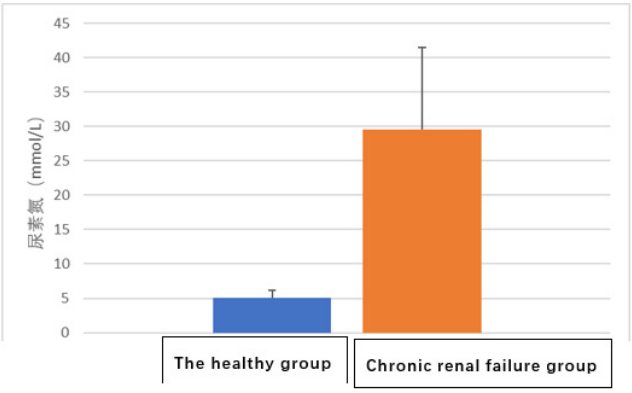

Figure 4. Comparison of urea nitrogen levels between healthy and chronic renal failure groups

According to figure 4 we can clearly see that chronic renal failure group and healthy group of urea nitrogen level difference is bigger, and it is working with the described earlier "when it became apparent when it comes to chronic renal failure, kidney function, urea nitrogen levels rise significantly".

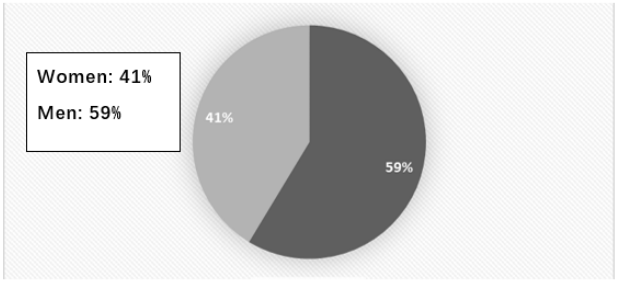

Figure 5. Gender ratio of healthy group

Figure 5. shows the gender ratio of 75 patients in the healthy group, among which 44 were males and 31 were females.

Figure 6 shows the ratio of gender of 75 patients in the chronic renal failure group, including 50 males and 25 females.

The gender of the chronic renal failure group and the healthy group were statistically analyzed by statistical method,

P>0.05, therefore, the gender difference between the chronic renal failure group and the healthy group was not statistically significant.

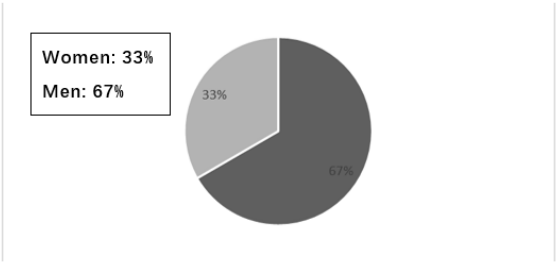

Figure 6. Chronic renal failure group sex ratio

4.2. Discussion

Urea nitrogen or the nitrogen in urea, urea and urea nitrogen available to represent the concentration of urea nitrogen, in our domestic is usually used for the concentration of urea nitrogen in urea. Amino acids are metabolized in the liver to produce toxic ammonia, which can be hydrolyzed to non-toxic UN, which is also one of the manifestations of liver detoxification function. Blood urea nitrogen (BUN) in serum is usually filtered through the glomeruli and excreted from the body [7]. The reference range is 2.9-7.5 mmol/L (8-21 mg/dl). Uric acid levels above this range are also known as azotemia and are commonly seen in renal insufficiency.

Most of man's final production is urea nitrogen. Amino acids in the body undergo a series of reverse finally produce NH3 and CO2, and then the two react in the liver to produce urea. One gram of protein in the body through a series of reactive protein metabolism can produce 0.3g of urea. The nitrogen content in urea of about 50%. Generated after renal excretion of urea in vitro, after filtering, the urea in the renal tubules was again.

Urea nitrogen and serum creatinine in early renal damage, its index similar happens, serum urea nitrogen and nitrogen in renal injury will be higher than the normal range. Serum urea nitrogen concentration rises sharply when renal function is damaged to less than 50% of normal value.

According to the experimental results, when chronic renal failure occurs, the level of serum urea nitrogen will be significantly increased, and its increasing trend is obvious. And will be healthy and chronic renal failure group compared with statistical method, $P < 0.05$, significant differences in statistics. Therefore, it can be used to speculate on the occurrence of chronic renal failure.

Scholars at home and abroad and the medical staff think serum creatinine, serum creatinine is the endogenous muscles after a series of metabolic reactions to generate endogenous creatinine. Creatine generated in the muscle does not exist in the form of creatine in the muscle, but creatine undergoes a series of complex reactions in the muscle to produce blood creatinine, and then the generated blood creatinine is released into the blood, filtered through the glomerular, and excreted with urine [10]. "Therefore, the relationship between serum creatinine production and muscle mass is positively correlated, and serum creatinine production is not easily affected by diet." Serum creatinine is no longer reabsorbed by the renal tubules. Due to the nature of serum creatinine above, so it can be used as evaluation of a good indicator of renal function.

When the glomerular filtration rate is lower than one third of the normal glomerular filtration rate, serum creatinine will appear more obvious rise. In conclusion, in the early stage of renal function injury, renal function cannot be determined by observing serum creatinine, and only when the glomerular filtration rate decreases to a certain extent, renal function injury can be determined by serum creatinine. Therefore, in the early stage of renal function injury, patients will not feel obvious discomfort. When the kidney injury is serious, patients will have nausea, vertigo and other symptoms, at which time there will be a significant increase in the value of serum creatinine [14].

As mentioned above, serum creatinine is produced by a series of reactions of creatine in muscle, and it has been determined by relevant personnel that 1mg of creatinine can be produced by 20g of muscle through a series of reactions. Creatinine is then filtered by the glomerulus as a small molecular substance. We define the endogenous creatinine is the serum creatinine, some meat food after reaction in the body can also generate exogenous creatinine, endogenous creatinine, here we only study is no longer here. When the amount of meat intake is stable, the generation of endogenous creatinine generated in the body will also be relatively stable [14].

The results show that when chronic renal failure occurs, the level of serum creatinine will be significantly increased, and it will progress with the progress of the patient's condition. The healthy group and chronic renal failure group were compared by statistical methods, $P<0.05$, there was a significant difference in statistics. Therefore, can be used to speculate that the occurrence of chronic renal failure.

Uric acid is produced by a series of metabolic reactions in birds and reptiles. In the urine produced by the normal human body, the vast majority of components are urea, and the content of uric acid is relatively small. Uric acid is the final product of purine substances after a series of metabolic reactions in the body. Its essence is called trioxypurine in chemistry, and its alcohol form is weak acid in chemistry. Purine substances undergo a series of complex metabolic reactions in the body to produce uric acid, which is then filtered by the kidneys and excreted in the urine. Clinically, we define hyperuricemia when the content of uric acid in the blood is higher than 420μmol [18].

Under normal circumstances, the content of uric acid in the normal body is about 1200mg, of which the normal body produces about 600mg of uric acid every day through complex metabolic reactions. At the same time, the amount of uric acid excreted by the normal body through urine is about 600mg every day, so in the normal body, every new generation of uric acid in urine and day to drain off the uric acid out of equilibrium. On the contrary, when the uric acid produced in the body on the same day is greater than the uric acid excreted in the urine or the human excretion mechanism is degraded, the blood uric acid concentration in the body will increase. Once the concentration exceeds 7 mg/dL, the body fluid will become acidic, which will affect the function of normal human body cells [18].

Uric acid and serum creatinine is different, it is not a small molecule, in kidney, uric acid also should pass glomerular filtration in the first place, and then by renal tubular reabsorption [5]. When occurrence problem of kidney function, the determination of uric acid values also can appear to rise.

The experimental results show that when chronic renal failure occurs, the level of uric acid will increase significantly due to the decrease in excretion of uric acid, showing a

progressive trend. After excluding the factors such as tumor and leukemia, the healthy group was compared with the chronic renal failure group by statistical methods, $P<0.05$, there was a statistically significant difference. Therefore, it can be used to speculate on the occurrence of chronic renal failure.

5. Conclusion

Through the above analysis, chronic renal failure group of serum creatinine, blood urea nitrogen, uric acid level is significantly higher than healthy group with statistical significance. Therefore, serum creatinine, uric acid and urea nitrogen can be used as auxiliary diagnostic indicators for patients with chronic renal failure.

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