

THE RELATIONSHIP BETWEEN LEVEL OF BLOOD UREA AND AGE IN IRAQI ELDER PEOPLE (MALE AND FEMALE)⁺

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Abstract

Enzymatic colorimetric method is used to determine the level of urea in the blood. Ninety patients were included in this study (45 male, 45 female). The male and female were divided in to three groups. Each subject (15) patients. First group consids age was between (45-54 years). Second subject age between (55-64 years). Third subject age more than (65 years). The percentage of having blood urea was calculated to all subjects. The results are compared with results from other countries. The results show that blood urea increases with increasing age and there is no big difference in sexes, they are approximately same. It is recommended many educational ways be used to reduce blood urea. If any body has blood urea he can reduce the level or make it no more by following this recommendation.

المستخلص :-

تم اخذ تسعون مريضاً على شكل مجموعتين. خمس وأربعون امرأة و خمس وأربعون رجلاً. قسمت كل مجموعة إلى ثلاث فئات كل فئة خمسة عشر شخصاً الفئة الأولى (تتراوح أعمارهم بين خمس واربعين إلى أربع وخمسين سنة) (الفئة الثانية من خمس وخمسين إلى أربع وستين سنة) (الفئة الثالثة فوق الخامسة والستين سنة). تم استخدام الطريقة الفوتومترية لتعيين نسبة اليوريا في الدم. قمنا بحساب النسبة المئوية للإصابة بارتفاع اليوريا في الدم لكل الفئات. أظهرت النتائج إن النسبة المئوية تزداد بازدياد العمر وذكرنا أسباب ذلك. وجدنا إن الفرق طفيف بين معدل الإصابة بين الرجال والنساء. في المناقشة قارنا النتائج التي حصلنا عليها مع نتائج الدول الأخرى ولم يكن هنالك فرقاً كبيراً بينها وبين نتائجنا ثم ذكرنا طرقاً علمية و منهجية للتقليل من الإصابة بارتفاع يوريا الدم وعدم التسبب بارتفاع نسبتها أكثر بالنسبة للأشخاص المصابين بارتفاع نسبتها في الدم.

Introduction

Urea is the small molecule (MW60) with two nitrogen atoms. It is the end product of protein catabolism in mammals. Urea is highly soluble, non toxic compound that enters the blood and is excreted in urine [1, 2]. The urea concentration in glomerular filtration is the same as in the plasma [3]. The biosynthesis of urea from amino nitrogen- derived ammonia is carried out exclusively by hepatic enzymes of the urea cycle. More than 90 percent of urea is excreted through the gastrointestinal tract and skin accounting for most of the remaining minor fraction. Blood urea level is influenced by several factors, including state of hydration (the range from 0.65 during diuresis to 0.35 during antidiuresis), age and dietary protein intake. [4]

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Uremia is clinical syndrome comprised of marked elevation in plasma urea accumulation by acidemia and electrolyte imbalance (especially potassium elevation) of renal failure. [5]. The uremia syndrome consists of symptoms and signs including nausea, vomiting, anemia and variable degrees of altered mutation, that results from toxic effect of elevated levels of nitrogenous and other wastes in the blood. The patient shows sallow coloration of the skin due to accumulation of urochrome pigment and ammonia like Or urine like odor to the breath is rarely seen unless the degree of uremia is severe. The blood urea levels vary in magnitude but usually are in excess of 100mg\dl or approaching 200mg\dl with deep stupor or coma. An increased level of urea in the blood is called azotemia. [6, 7]

Material and Method

This study takes the period between the first of October 2003 to the first of May-2004. Enzymatic colorimetric method was used to determine the level of blood in urea. Ninety samples of blood were included in this study (female and male) they were divided into three groups according to the age. The first one age were between (45-54) years, the second group (55-64) years, third group (65 and up) years.

Normal values of urea

Serum-Plasma 15-40mg\dl
 0.15-40 gm\dl

Urine 2.49-6.66m mol\l
 20-35g\24hrs [8]

Results & Discussion

Table (1) shows the relationship between mean concentration of blood and age in male patients .(P<0.05 significant , P< 0.01 highly significant)

Table (1)			
Age (Year)	No.Of patients	Mean concentration of blood urea mg/dl	P Value
45-54	15	50.8	S
55-64	15	56.7	H.S
65-UP	15	62.5	H.S

Table (2)shows the relationship between mean concentration of blood and age in female patients .(P<0.05 significant , P< 0.01 highly significant)

Table (2)			
Age (Year)	No.Of patients	Mean concentration of blood urea mg/dl	P Value
45-54	15	50.8	S
55-64	15	56.7	H.S
65-UP	15	62.5	H.S

Table (1) and (2) show that the level of blood urea increases with age for both sexes . There is significant difference between the mean concentration value of the first group and the second group (P<0.05) while there is highly significant difference between the mean concentration of the second group and the mean of the third group (P<0.01) and there is also

highly significant difference between the mean concentration value of the third group and the mean of the first group ($P < 0.01$). It is found also there is no big difference between the mean values for each age in both sexes.

The incidence of renal disease increases with age, This is demonstrated in a study from the United Kingdom where the incidence increases from 58 pmp per year in these under the age of 50 years to 588 pmp per year in individuals aged 80 years and over.

In a French study, the increase is most apparent in elderly with an annual incidence of CRF reaching 1124 pmp per year:

The etiology of CRF is also different in the elderly. The rate of progression of CRF and level of blood urea also influenced by age, with elderly patients affected by glomerulonephritis having faster rate of decline of renal function (see table 3)[9].

The blood urea level is influenced by dietary protein intake, hydration status, gastrointestinal bleeding.

Approximately two-thirds of renal function must be lost before a significant rise in blood urea nitrogen becomes evident. Patient's with renal insufficiency may develop extremely high BUN levels that can be partially controlled by decrease in dietary protein [10].

In our project also it is found as in other countries that the level of blood urea increases with increasing the age and its prevalence in elderly people than the younger people and there is no big difference between sex. An elevated BUN may be caused by impaired renal failure (because the kidneys are less able to clear urea from blood stream), congestive heart failures as a result of poor renal perfusion, dehydration (patient who is severely dehydrate may also have a high BUN due to the lack of fluid volume to excrete waste products), excessive protein intake or protein catabolism (because urea is an end product of protein metabolism, a diet high in protein such as high-protein tube feeding, may also cause the BUN to increase), hemorrhage into the gastrointestinal tract (extensive bleeding in the gastrointestinal tract will also cause an elevated BUN because digested blood is a source of urea, for example a hemorrhage of one liter of blood in to the gastrointestinal tract may elevate the BUN up to 40 mg/ml), shock, acute myocardial infarction, are stress.

Because of this important reasons, we have taken the project to compare our results with other countries results.

Table (3) Incidence and prevalence of end stage renal disease in UASA 1996.

Characteristics	Prevalence (pmp)	Incidence
20-44	692	117
45-64	2280	542
65-74	3518	1144

Recommendation

5-1 Diet Therapy (principle of diet therapy)

The basic objective in the dietary treatment of renal disease is to lighten the excretory load of products of metabolism and to help the kidney maintain the normal equilibrium of the body's internal environment at the point in progressive renal failure when clinical symptoms develop, diet modifications become an important aspect of treatment, depending of the type

and course of the kidney disease, progression of renal failure may even be delayed for a period of time by careful dietary measure shown in table (4) .

5-2 Protein

When symptoms of uremia occur or nitrogen retention is evident (BUN over 100mg / dl) or both protein in the diet are restricted to control the amount of nitrogen available for urea syntheses . at the same time the body's need for essential amino acid must be met by proteins of high biologic value .see table (5).

proteins of high biologic value have most of their nitrogen present in their essential amino acid ; the foods that best satisfy these criteria are milk and eggs , meats also satisfy these criteria except that they contain somewhat more nitrogen from nonessential amino acids than do milk and eggs.

5-3 Energy

All patients suffering renal failure, whether acute , chronic or end –stage , require an adequate caloric intake (approximately 35 kcal to 40 kcal per kilogram body weight for adults). Without adequate calories supplied by carbohydrate and fat , amino acids from food and body cells will be deaminized in intermediary metabolism to contribute to energy needs through gluconeogenic pathway . this decreases the amount of protein available to compensate for the loss of protein in the urine (e.g. proteinuria in nephritic syndrome) , or the catabolism of protein for energy increases the amount of nitrogen available for the synthesis of urea , thereby leading to an increase in the amount of the urea to be excreted.

Consideration of this critical relationship between energy and protein in intermediate metabolism is the first step in planning diet therapy for the patient with renal disease and is often not emphasized sufficiently . Patients with chronic renal disease are not often obese. However , for those who are , a moderate restriction of caloric intake of a child must be adequate to support normal growth as as possible . Several studies have reported that growth failure in children with renal disease could be attributed in part to inadequate intakes of energy .

A human subject who consumes 300 gm of carbohydrate , 100gm of fat and 100gm of protein daily excrete about 16.5 of nitrogen each day , 95% in the feces.

Table (4) Recommended dietary intakes for uremic patient .

Component	No dialysis	Hemodialysis or
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		Peritoneal dialysis
Protein	Men:≥40mg/day (0.55-0.60gm/kd/day) (28gm of high biological value) women;small men :≥35gm /day (23-25gm of high biologic value)	HD:1.0 gm /kd/day PH: 1.2-1.5gm/kd/day (>50%of high biologic value)
Calories	≥35kcal/kg/day unless patient is obese	
Vitamins		
Thiamine(mg/day)	1.5	1.5
Riboflavin (mg/day)	1.8	1.8
Pantothenic acid (mg/day)	5	5
Niacin (mg/day)	20	20
Pyridoxine hydrochloride (mg/day)	5	10
Vitamin B12 (mg/day)	3	3
Vitamin C (mg/day)	70-100	100
Folic acid (mg/day)	1	1
Vitamin E (mg/day)	15	15
Minerals		
Sodium (mg/day)	1000-3000	750-1000
Potassium (mEq/day)	40-70	40-70
Phosphorus (mg/day)	600-1200	600-1200
Calcium (mg/day)	1000-2000	1000-1500
Magnesium (mg/day)	200-300	200-300
Trace elements	Unknown	Unknown
Water	Up to 3000ml/day as tolerate	Usually 750-1500 ml/day

Table (5) :Vegetables grouped by their protein content

1 gm	2 gm	3-4 gm
Beans, green or wax	Cauliflower	Asparagus
Beets	Mushroom	Broccoli
Cabbage	Okra	Brussels sprouts
Carrots	Potato ,white or sweet	Corn
Cucumber	Spinach	Mixed vegetable
Egg plant	Squash , winter	Peas
Endive		
Green peppers		
Lettuce		
Radishes		
Squash, summer		
Romaine		
Rutabagas		
Tomatoes		

References

- 1-Shaul G Richard J: *Renal Handling of Organic Compound*, textbook of Nephrology 4thed, printed in USA, pp82., 2001
- 2-Victol Donald B: *Amino Acid Degeneration*, *Book of Biochemistry* 3rd .ed, printed in Egypt, pp459., 1994
- 3-Robert K, Peter A: *Biochemistry and Disease*, Book of Harpers Biochemistry 22nd .ed, printed in Lebanon, PP688-89.
- 4-Robert H: *End Stage Renal Disease*, *Book of Pathology of the Kidney* 4th.ed, Vol. 2, printed in USA...pp720, 1993
- 5-John B: *Metabolism Intermediate*, Inorganic Ions and Biochemical Markers of Bone Metabolism, Book of Clinical Diagnosis and Management by Laboratory Method 20th .ed, Printed in USA, pp3., 1998
- 6-Salah A. Mircea G: *Essentials of Renal Physiology*, Book of Essentials of Basic Science in Urology, printed in USA, pp82., 1991
- 7-John T. Todd S: *Indication of Dialysis*, *Handbook of Dialysis* 2nd, ed., Printed in USA, pp3., 1994
- 8-Mackey, E.N. Ricky LL: *Enzymatic Colorimetric Method Kit from Biomaghreb*. 2001.
- 9-Richard J. Johan F: *Chronic Renal Failure and Uremia Syndrome*, Progression of renal failure, Book of chronic renal failure, Book of comprehensive clinical nephrology, printed in USA, pp671-672., 2000
- 10- Emil A. Jack W: *Urologic Laboratory*. *Book of General Urology* 15th .ed, Printed in Lebanon. pp61., 2000
- 11- Anderson D: *Renal Disease*. Printed in USA, pp542-547., 2002
- 12- Robert K. Dray K: *Catabolism of Proteins and Amino Acid*. *Book of Harpers Biochemistry* 25th .ed. 2000. Printed in USA. pp319.