Clinical and Biochemical Study of Acute Renal failure Disease

Zena A.M. Al-Jawadi
Department of Chemistry, College of Science, Mosul University

(NJC)

(Received on 13 /7 /2005) (Accepted for publication on 4/ 3 /2006)

Abstract

A clinical and biochemical study of 60 patients (31 males, 29 females) with renal failure attending the urological out patients clinic in Al-Zahrawi Teaching Hospital and Ibn Sena Hospital in Mosul. Every patients was evaluated clinically and by a chemical laboratory test. The results showed that the period of oliguria : urinary volume is less than (400 ml/24h) is accompanied by a rise in the serum urea and creatinine concentration .As showed in this study incidence of acute renal failure occurs equal in male and female and the incidence occur more often in elderly people than in children. Finally the Chemical investigations help to determine the severity of the disease and to follow its course, but do not help much in determining the cause .

الخلاصة

الدراسة السريرية و الكيموحيوية أجريت لـ 60 مريض (31 ذكور ,29 إناث) مصابين بعجز الكلى الحاد راجعوا قسم المجاري البولية في مستشفى الزهراوي التعليمي في الموصل و مستشفى ابن سينا في الموصل. التقييم السريري للمرضى استند إلى تسجيل التاريخ المرضي و الفحوصات الكيميائية المختبرية . حيث أظهرت النتائج انه في مرحلة قلة الإدرار:حجم الإدرار يكون اقل من (400 مل/24ساعة) وتكون مصاحبة لزيادة تركيز اليوريا و الكرياتنين في مصل الدم . كما أوضحت الدراسة إن نسبة الإصابة تكون متساويةُ في الذكور و الإناث و لكن تكون أكثر حدوثاُ في كبار السن مما في الأطفال. و أخيرا الفحوصات الكيميائية تساعد على تحديد شدة المرض و متابعة مراحله ,لكنها لا تحدد الأسباب بدرجة كبيرة.

Introduction

Renal failure results when the kidneys are unable to remove the body metabolic wastes or perform their regulatory function .The substances normally elimination in the urine accumulate in the body fluids as a result of impaired renal excretion and lead to a disruption in endocrine and metabolic functions as well as fluid, electrolyte, and acid-base disturbances. Renal failure is a systemic disease and is the final common pathway of many different kidney and urinary tract diseases. Each year an estimated 42,000 Americans die of irreversible kidney failure[1,2,3].

Pathophysiology:

Acute renal failure is a sudden and almost complete loss of kidney function caused by failure of the renal circulation or by glomerular or tubular dysfunction. It is manifested by sudden oliguria (less than 400 ml of urine per

day), high urinary output, or anurina (less than 50 ml of urine per day)[4,5,6].

Regardless of the volume of urine excreted, the patient with acute renal failure experiences rising serum creatinine and blood urea level that excreted by the kidney[7,8,9]. Any condition that causes reduction in renal blood flow, such as volume depletion, hypotension, or shock, leads to a reduction in glomerular filtration, renal ischemia, and tubular damage. Renal failure may also result from the adverse effects of burns, crushing injuries and infection as well as from nephrotoxic agents that cause acute tubular necrosis and temporary cessation of renal function[6,10,11,12,13,14]. Although the exact pathogenesis of acute renal failure and oliguria is not always known, various possible mechanisms have been suggested. In many instances, there is a clear-cut underlying disease, mechanical obstruction of the urinary tract by stone or tumor, or renal artery obstruction.A new causative factor in acute renal failure is the use of nonsteroidal anti-inflammatory agents, especially in the elderly. These agents interfere with prostaglandin's that normally protect renal blood flow, and their use impairs this protective mechanism, leading to hypoperfusion of the kidneys[1,9,15].There are three clinical phases of acute renal failure, the period of oliguria, a period of diuresis and a period of recovery [2,16].

Materials and Methods

Sixty patients (31males, 29 females) with renal failure attending the urological out patients clinic in Al-Zahrawi Teaching Hospital and Ibn Sena Hospital in Mosul. Every patients was evaluated clinically and by a biochemical laboratory tests, and compaired with fourty normal subjects (20 males, 20 females).

Serum analysis was done for sodium, potassium using flame photometry . Creatinine, urea, calcium, phosphorus and uric acid using commercial kits (BioMerieux Vitek, Inc., USA), and the volume of 24h urine was noted.

Results and Discussion

Almost every system of the body is affected when there is a failure of normal renal regulatory mechanisms. The patient appears critically ill and lethargic with persistent nausea ,vomiting ,and diarrhea. The skin and mucous membranes are dry from dehydration, and the breath may have the odor of urine. Central nervous system manifestations include drowsiness, headache ,muscle twitching ,and convulsion. The urine output is scanty ,may be bloody and this results agrees with other reports[17,18].Several laboratory tests are useful for assessing the etiology of acute renal failure ,and the findings proper management. These tests include serum and urine biochemical analysis[19]. Table (1) showed high serum potassium, urea , creatinine and uric acid may be in some patients are dangerous ,and need to be determined at least once daily. Plasma sodium is usually low due to combination of factors. In acute renal failure the glomerular filtration rate decrease over days to weeks. As a results ,excretion of nitrogenous waste is reduced, and fluid and electrolyte balances cannot be maintained. Patients with acute renal failure are often asymptomatic, and the condition is diagnosed by observed elevations of blood urea and serum creatinine levels. This results was observed in Table(1), which argrees with the results of previous studies[6,17,20,21,22].

The blood urea correlates poorly with the glomerular filtration rate ,because urea is highly permeable to renal tubules , urea clearance varies with urine flow rate, while serum creatinine provides the physician with accurate and consistent estimation of glomerular filtration rate. Correct interrelation of serum creatinine extends beyond just knowing normal values for the specific laboratory.

Changes in serum creatinine reflect changes in glomerular filtration rate. Rate of change in serum creatinine is an important variable in estimating glomerular filtration rate. Stable changes in serum creatinine with changes in glomerular filtration rate by the following relationships:[22]

Creatinine 1.0 mg/dl - Normal glomerular filtration rate
Creatinine 2.0 mg/dl -50%reduction in glomerular filtration rate
Creatinine 4.0 mg/dl -70-85%reduction in glomerular filtration rate
Creatinine 8.0 mg/dl -90-95%reduction in glomerular filtration rate

As suggested by these data , knowledge of a patients baseline creatinine becomes very important[22,23].And there may be an increase in serum phosphate concentrations while serum calcium concentration is low in response to decreased absorption of calcium from the intestine and in association with an elevation of serum phosphate levels[1,22].

Table(1):Comparison between serum parameters in all patients and control group.

Parameters	Patient	Control
Urea(mg/dl)	58±23.1**	29.71±5.43
Creatinine(mg/dl)	1.88±0.7*	0.77±0.12
Sodium(Meq/l)	155.88±2.6	150.59±4.2
Potassium(Meq/l)	5.63±0.3	4.26±0.6
Calcium(mg/dl)	8.20±0.9	8.96±0.5
Phosphorus(mg/dl)	4.58±1.5*	4.03±0.7
Uric acid(mg/dl)	5.48±1.7*	4.40±1.1

Mean ±SD
*Significant differences at $p \leq 0.05$
** Significant differences at $p < 0.005$

Patients had the following urine studies: dipstick test , total volume of 24h urine, reddish brown or cola-colored urine is present in patients with acute renal failure. The period of oliguria: urinary less than 400ml/24h is accompanied by a rise in the serum concentration of the elements usually excreted by the kidney, which is consistent with the finding of other investigators[6,16,24,25] .

Regardless of the volume of urine excreted, the patient with acute renal failure experiences rising serum creatinine and blood urea levels and retention of other metabolic waste products normally excreted by the kidneys[7,8,9], and this agree with the results indicated in Table(2) in which the concentration of urea, uric acid and creatinine excreted daily was low as compared with control group due to a decreased in the glomerular filtration rate.

Table(2):Comparison between 24h urine parameters in all patients and control group.

Parameters	Patient	Control
Urea mg/24h	154.06±89.67*	218.15±90.71
Uric acid mg/24h	186.25±85.21*	448.18±170.19
Creatinine mg/24h	98.87±29.20*	105.74± 34.93

Mean±SD
*Significant differences at $p \leq 0.05$

Males and females are affected equally and this agree with the results in this study. Men generally have a higher muscle mass per kilogram of body weight and thus a higher serum creatinine than woman[22],and that agree with the results in Table(3) it was showed that higher serum creatinine ,urea and uric acid in males than in females, and the concentration of sodium and potassium it was equally in both sex, because serum potassium adaptation usually produces a normal concentration until advanced failure occur for both sex [25]. While serum sodium concentration is usually low in acute renal failure .On the other hand, urea, creatinine and uric acid excreted per 24h urine was lower in the both sex but more often in males than in females because the glomerular filtration rate decreases over days to weeks. Abnormalities of serum calcium and phosphorus concentration was noted.

Table(3): Comparison between different parameters in serum and urine according to sex.

Parameters	Male(No.=31)	Female(No.=29)
In serum		
Urea(mg/dl)	62.76±29.8*	56±19.7
Creatinine(mg/dl)	2.01±0.9	1.82±0.7
Sodium(Meq/l)	156.4±2.3	154.59±2.7
Potassium(Meq/l)	5.67±0.2	5.54±0.3
Calcium(mg/dl)	8.34±1.0	7.92±1.0
Phosphorus(mg/dl)	4.56±1.6	4.64±1.0
Uric acid(mg/dl)	5.67±1.6	5.05±2.0
In urine		
Urea(mg/24h)	143.04±88.74*	159.37±89.72
Uric acid(mg/24h)	177.01±86.91	204.74±82.94
Creatinine(mg/24h)	97.76±29.25	99.15±28.91

Mean ±SD

*Significant differences at $p \leq 0.05$

Acute renal failure is not a benign disease ,and the patients age has a significant implications for the differential diagnosis of acute renal failure as see in Table(4) in children with age ranged between (10-19) the most common cause of acute renal failure is perennial etiologies and the most common cause of hypovolemia is gastroenteritis and congenital and acquired heart disease are also important causes of decreased renal perfusion in this age group. In adults whose age between (20-40) age group, refer to history for a general discussion of acute renal failure because acute renal failure has such a long differential diagnosis obtain a directed history along the lines of the Pathophysiology of acute renal failure .Patients commonly present with symptoms related to hypovolemia, including thirst, decreased urine output, dizziness, and orthostatic hypotension and patients with advanced cardiac failure. Insensible fluid losses can result in severe hypovolemia in patients with restricted fluid access and should be suspected in the elderly and in comatose or sedated patients. The intrinsic renal failure patients can be divided into those with glomerular and those with tubular etiologies of acute renal failure and pigment-induced acute renal failure should be suspected in patients with possible rabdomyolysis (muscle tenderness, recent coma, seizures, drug abuse, alcohol, excessive exercise, limb ischemia)or haemolysis (recent blood transfusion).

Finally in elderly people (≥40) group age postrenal failure usually occurs in older men with prostatic obstruction and symptoms

of urgency, frequency, and hesitancy. Patients may present with asymptomatic high-grade urinary obstruction because of chronicity of their symptoms. History of prior gynecologic surgery or carcinoma often can be helpful in providing clues to the level of obstruction, flank pain and haematuria should raise a concern about renal stone or papillary necrosis as the source of urinary obstruction . Use of drug due to acute renal failure and hypotension and tachycardia are obvious clues to decreased renal perfusion. This results in Table(4), which argrees with the results of previous studies[14,22]. In humans, renal failure is most often managed by dialysis (haemodialysis or peritoneal dialysis) or renal transplantation[9].

Table(4):Comparison between acute renal failure and age in all patients.

Parameters	Age Groups(year)						
	10-19 (No.=6)	20-29 (No.=8)	30-39 (No.=10)	40-49 (No.=11)	50-59 (No.=7)	60-69 (No.=13)	≥70 (No.=5)
Urea(mg/dl)	65.8±31.6	58.14±18.2	49.18±7.3	52.58±9.4	70.25±42.7	57±16	78.33±36.6
Creatinine(mg/dl)	1.95±0.8	1.94±0.8	1.55±0.2	1.83±0.6	2.18±1.1	1.91±0.6	2.6±1
Sodium(Meq/l)	156.4±2.3	154.7±3.5	156.1±1.4	156.3±2.1	155.3±1.9	156.6±2.6	153.3±2.4
Potassium(Meq/l)	5.65±0.2	5.49±0.2	5.30±1.1	5.68±0.3	5.73±0.2	5.68±0.2	5.46±0.2
Calcium(mg/dl)	8.26±1.2	8.29±0.7	8.30±0.9	8.19±1.2	6.18±1.7	8.17±0.8	8.40±0.3
Phosphorus(mg/dl)	4.71±1.5	4.47±0.6	4.40±0.6	4.40±1.1	5.90±3.7	4.56±0.9	3.43±1.5
Uric acid(mg/dl)	4.810±2.4	5.41±1.7	5.75±1.2	5.63±1.5	5±2	6.08±1.6	4±0.8

Mean ±SD

References

1.Brunner, L.S. and Suddarth, D.S., 1988. Textbook of Medical-Surgical Nursing, 6thed., J.B. Lippincott Company, U.S.A., p1033-1038.

2.Smith,A. F.;Beckett ,G. J. ;Walker, S. W. and P.W.H., 1998. Clinical Biochemistry,6th ed .,U.S.A.,p51-67.

3.Kidny disease ,2002. Kidny disease. Available at: http://www.lbah.com/feline/kidney.htm . Accessed.April 10,2005.

4.Goldstein, M.B.,1983. Acute renal failure, ***Med. Clin. North. Am.***, 1983, **67(6)**, 1325.

5.Baer, C.L., Acute renal failure: Mortality or magic, ***Dimens Crit Care Nurs***, 1983, **2(6)**, 324.

6.Agrawal,M.and Swartz,R., 2000. Acute renal failure , American Family Physician, 61(7).

7.Cameron, J.S., Acute renal failure in the intensive care unit today , ***Intensive Care Med.***, 1986, **12(2)**, 64.

8.Burke, J.F. and Francos, G.C.,1987. Surgery in the patient with acute or chronic renal ailure Med. ***Clin. North. Am.*** , 1987, **71(3)**, 489.

9.New Hope Animal Hospital, 2002. Canine urinary tract.Available at: http://www.nhahonline.com/k9urinarytract.htm#renal . Accessed.March 20,2005.

10.Whittaker , A.A., Acute renal dysfunction: Assessment of patients at risk ,Focus Crit.***Care***,1985, **12(3),** 12.

11.Lazarus , J.M., Acute renal failure, ***Intensive Care Med.***, 1986, **12(2)**, 61.

12.Brady,H.R. and Singer,G.G.,.Acute renal failure , ***lancet***, 1995, **346**, 1533.

13.Thadhani, R.; Pascual, M. and Bonventre,J.V., Acute renal failure ,***N. Eng. J. Med***., 1996, **334**, 1448.
14.Maslansky,R, 2004. Aprisoner with acute renal failure, ***Lancet*** , 2004, 1,**363(9419),** 1474.
15.Coleman , E.A.,1986.When the kidneys fail, R.N., 28-34.
16.Nephrology Channal.htm, 2005. Acute renal failure(ARF). Available at: http://www.nephrologychannel.com/enroll/index,shtml . Accessed.April10,2005.
17.Williams , A.J. and Brown , C.B., Management of acute renal failure , ***Postgraduate Doctor*** , 1988, **11(7)**, 348.
18.Abdulo , J.G.,1995. Diagnosing vascular causes of renal failure , ***Ann. Intern. Med.***, 1995, **123**, 601.
19.Agraharkar, M., 2004. Acute renal failure, e medicine. Available at: http:// www.emedecinine.com/med/topic1595.htm-101k. Accessed.March 20,2005.
20.Becker, G.J.; Whitworth , J.A. and Kincaid-Smith ,P.,1992. Clinical Nephrology in Medical Practice. Blackwell Science , Oxford.
21.Schrier ,R.W. and Gottschalk , C.W., 1993. Diseases of the kidney , 5th ed ., Little, Brown, Boston.
22.Sinert ,R. ,2005. Renal failure ,Acute , e medicine. Available at: http:// www.emedeicine.com/emerg/topic500.htm-111k **.** Accessed.March 20,2005.
23.San ,A.; Selcuk,Y.; Tonbul,Z. and Soypacaci,Z. , Etiology and prognosis in 438 patients with acute renal failure , ***Ren. Fai***. , 1996, **18(4)**, 593.
24.Hricik ,D.E.; Chung-Park ,M. and Sedor ,J.R. , Glomerulonephritis , ***N. Engl. J. Med.*** ,1998, **339**, 888.
25.Merck , 2005. Chronic renal failure . Available at: http://www.merck.com/pubs/mmanual /section17/chapter222/222b .htm .Accessed . April 10,2005.