Journal of Natural Sciences Research www.iiste.org
ISSN 2224-3186 (Paper) ISSN 2225-0921 (Online)
Vol.5, No.1, 2015

Some Microbial and Immunophysiological Parameter in Patients with Renal Failure

Abdalnabi J Abid Rana Jalil
Biology Dept. ,Coll. Of Science For Women ,Babylon Univ. –Iraq
Corresponding author e-mail: dr_almamory59@yahoo.com

Abstract
Background: *Escherichia coli* reported to be the most common bacteria isolate from patients with renal failure, while *Klebsilla pneumoniae*. were found to be the second common isolated, followed by *Staphlococcus aureus*.Also study explained an increased in levels of IL-17,FasL, ,creatinine,urea,.**Objectives:** To isolate microorganisms from patients with renal failure, also to determined Immunphsiological parameter for patients.**Methods:**102 blood sample (61 male (59.8%) and 41female (40.1%)) , 150 urine sample for pateints underogoing hemodialysis at industrial kidney unit in Marjan Teaching Hospital from November 2013 to June 2014.All sample were cultured on different media for full bacteriological identification . In addition blood samples were taken from patients for immunphysiological tests.**Results:**The most common gram negative bacterial isolates were *Escherichiae coli* (38.8%), followed *Klebsiella pneumoniae* (34.32%), *Enterobacter spp* (6.71%) and *Pseudomonas aerogenosa* (4.47%).The gram positive bacteria were *Staphylococcus aureus* (13.43%) and *Staphylococcus epidermidis* (2.24%).Also results explained an increased in levels of IL-17, Fas L, , creatinine , urea,
Keywords :IL17 , Fas-L , Bacteria ,S. creatinine ,B. urea

Introduction

Renal failure is a condition in which the kidneys fail to remove metabolic end-products from the blood and regulate the fluid, electrolyte, and pH balance of the extracellular fluids (Ricci *et al*.,2012). The underlying cause may be renal disease, systemic disease, or urologic defects of no renal origin. Renal failure can occur as an acute or a chronic disorder. Acute renal failure is abrupt in onset and often is reversible if recognized early and treated appropriately. In contrast, chronic renal failure is the end result of irreparable damage to the kidneys, it develops slowly, usually over the course of a number of years (Chue *et al.*, 2010).

The accumulation of nitrogenous wastes is an early sign of renal failure, usually occurring before other symptoms become evident. Urea is one of the first nitrogenous wastes to accumulate in the blood, and the blood urea level becomes increasingly elevated as renal failure progresses (Katzung and Bertram ,2007), raised levels of potassium, decreased levels of calcium, increased levels of phosphate, and in later stages anemia.

Chronic kidney failure (CKD) requires renal replacement therapy, which may involve a form of dialysis, Dialysis is the process of removing waste products and excess fluids from the body. There are two types of dialysis: hemodialysis and peritoneal dialysis. In hemodialysis (HD), blood is removed from the body and pumped by a machine outside the body into a dialyzer (artificial kidney) (Mark, *et al*.,2003). Although true and complete replacement of renal function is not provided by dialysis, this modality removes metabolic wastes and excess body water, and replenishes body buffers in order to sustain life (Rebecca *et al*.,2010).

Study found the most common causative organism is *Escherichia coli*, but, *Pseudomonas, Staphylococcus aureus*, and *Staphylococcus epidermidis* may also cause this infection , other gram-negative rods including *Klebsiella Pneuomoniae* and *Enterobacter spp*. which also causes Kidney failure (Springhouse., 2005).

All aspects of inflammation and immune function may be affected adversely by the high levels of urea and metabolic wastes, including a decrease in granulocyte count, impaired humoral and cell-mediated immunity, and defective phagocyte function (Haroun *et al.*,2003). The acute inflammatory response and delayed-type hypersensitivity response are impaired, the serum levels of cytokines such as IL-17, Fas L are elevated due to general overproduction and inadequate clearance of these substances (Eleftheriadis *et al*.,2007).

Materials & Methods

Microbial samples were isolated from 150 pateints with renal failure Specifically hemodialysis at industrial kidney unit in Marjan Teaching Hospital .Routine diagnostic test were used as a tool for microbial identification, starting by Macroscopical appearance, cultures on differant media included (Nutreint agar, Blood agar, Macconky agar and Manitol agar), to more biochemical tests which included haemolysis on blood agar, Oxidase, Catalase, Coagulase, Indole, Monitol, Methyl red, Urase and Vogas proskauer tests (Collee *et al*.,1996)

Immunological finding included the evaluation of IL-17 and Fas L . Elisa techniques were used in the testing of sera samples, also these tests was conducted in accordance with the manufacturer's instructions

BOSTER .while physiological tests included urea, createnine , these tests was conducted in accordance with the manufacturer's instructions bioMerieux.
Statistical analysis included the use of the specialized statistical software SPSS to determine the values of Least significant differences (LSD) in t-test and data was subjected in Excel to draw the paragraphs (Nizi.,2000).

Results

From 150 isolate of UTI in patients about (134) 89.3% of Sample had positive bacterial culture and (16) 10.6% of them had negative culture as shown in table (1).

Table (1) Number and Percentages of positive sample culture**.**

Urine Culture	Number	Percentage
Culture positive	134	89.3%
Culture negative	16	10.6%
Total	150	100%

Escherichia coli is the most common bacteria 52 (38.3%),followed by *Klabsialla pneumoniae* 46(34.32%) ,*Staphylococcus aureus* 18(13.43%) ,*Enterobacter spp.*9 (6.71%), *Pseudomonas aeruginosa* 6 (4.47%) and *Staphylococcus epidermidis* 3(2.23%) as shown in figure (1).

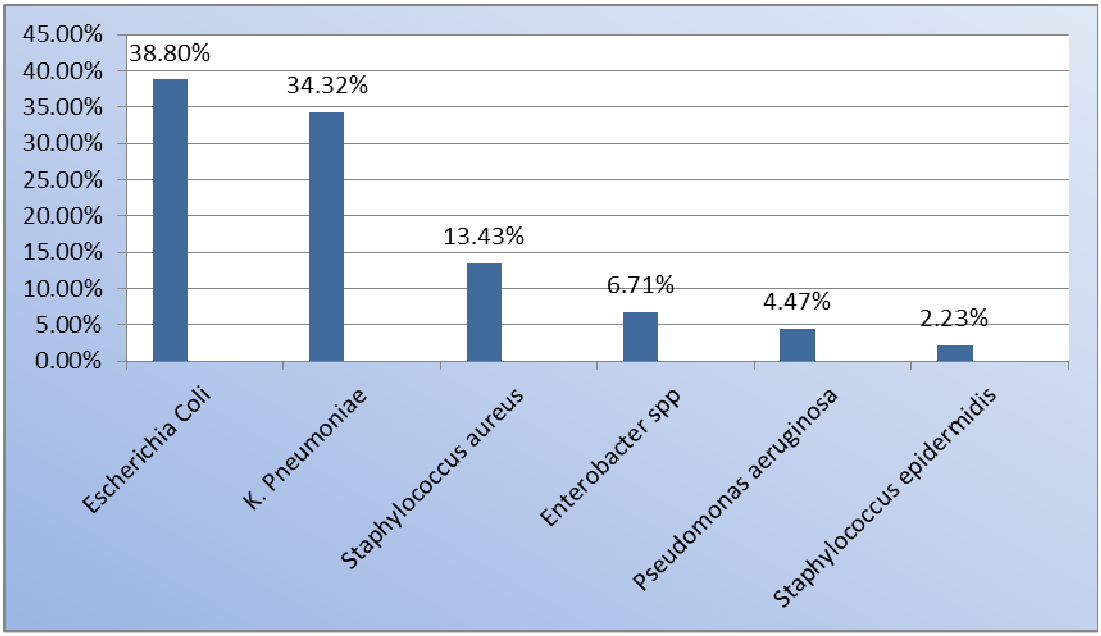

Figure (1) Types and percentage of bacterial isolates

Immunological f indings included the determiation of serum levels of IL-17 and Fas L. The level of IL-17 was significantly raised in all age groups of patients suffering from chronic renal failure in comparison to the control group, especially age group (31-40) accounted (144.33±1.528) while the levels in control group about (49±6.506), ($P<0.05$) as in table (2) .

Table (2) Level of IL-17 in all renal failure patients

Classes	groups	IL-17 Level pg/ml M±SD	L.S.D
10-20	Patients	123.3±28.34	45.4
	Control	58±6	
21-30	Patients	133.3±22.5	36.07
	Control	55.2±0.251	
31-40	Patients	144.33±1.528	2.44
	Control	49±6.506	
41-50	Patients	126±5	8.05
	Control	42.5±0.5	
51-60	Patients	129.3±11.504	18.51
	Control	37±1	
61-70	Patients	57±29.72	16.52
	Control	27.5±2.5	
71-80	Patients	57.3±12.86	20.63
	Control	18.5±0.5	

Fas L was significantly raised in the groups of patients suffering from renal failure in comparison to the control group, the age group (10-20) accounted (121.7±3.51) while the levels in control group was (34±2) ($P<0.05$) as in table (3).

Table (3) Level of FasL in all renal failure patients

Classes	groups	FasL Level pg/ml M±SD	L.S.D
10-20	Patients	121.7±3.51	6.28
	Control	34±2	
21-30	Patients	92.3±31.94	51.21
	Control	24.5±0.5	
31-40	Patients	42.3±15.37	4.82
	Control	19.23±0.257	
41-50	Patients	42.63±15.53	3.43
	Control	17.5±0.5	
51-60	Patients	39.3±11.5	18.44
	Control	15.2±0.251	
61-70	Patients	36.7±20.82	3.206
	Control	13±2	
71-80	Patients	32.3±7.51	12.27
	Control	9.5±1.5	

Urea level a highly significantly $p<0.05$ has been observed in all age groups and the highest concentration was showed in the age group (61-70) years and was (41.8mmol/L) compared with control group (2.3mmol/L) , $p<0.05$ as in figure (2).

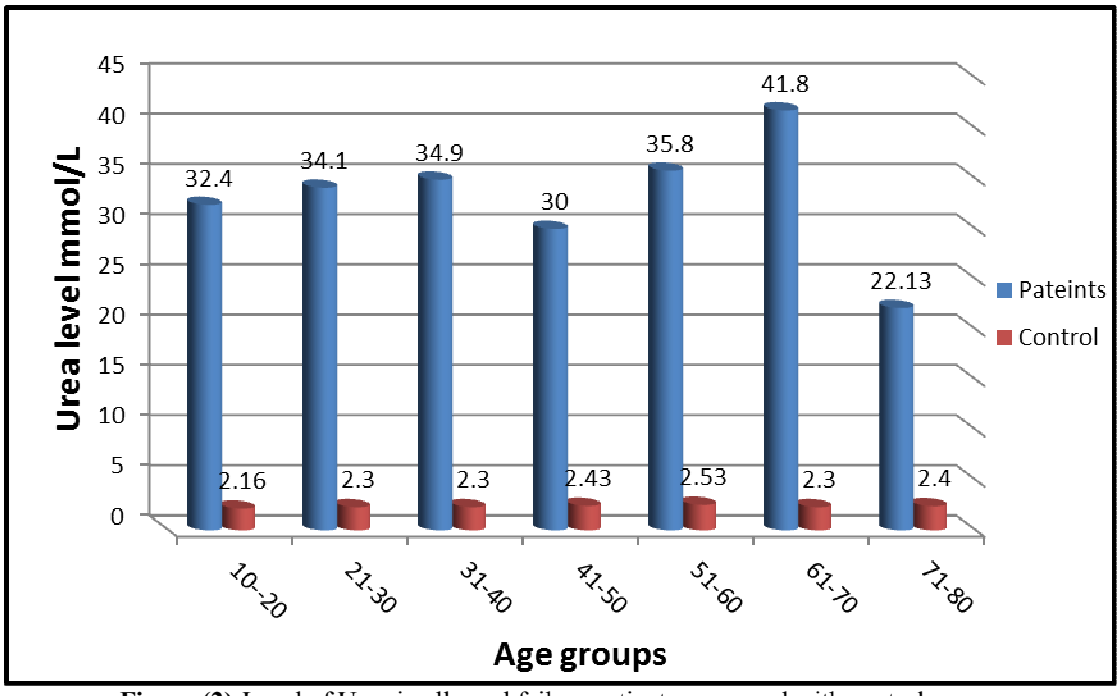

Figure (2): Level of Urea in all renal failure patients compared with control group.

Creatinine were recorded as a higher in age group (21-30) years and range(1033mmol/L) compared with control group (71.93 mmol/L) $p<0.05$ as in figure (3).

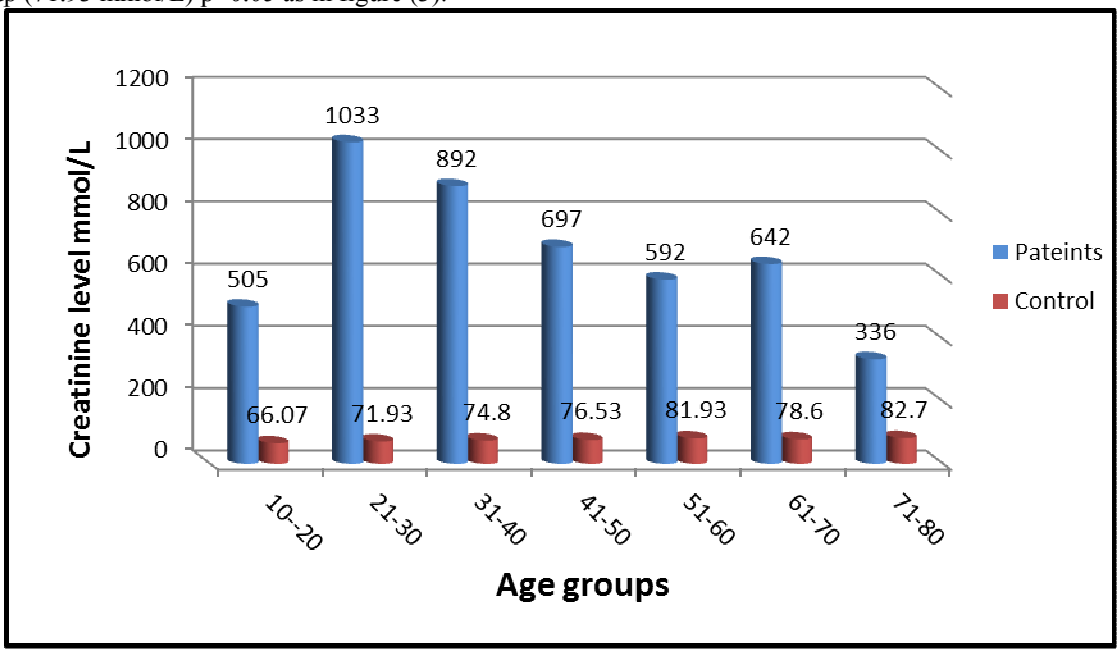

Figure (3): Level of Creatinine in all renal failure patients compared with control group.

Discussion

Patients with kidney failure are more prone to developing urinary tract infections, the mere act of passing urine tends to flush out the urinary tract of infectious agents so they cannot gain a foothold and cause problems (Rajiv Vij ,*et al*.,2009), Once the kidneys fail and the production of urine is decreased, this normal flushing action is gone. UTIs usually are caused by bacteria which normally inhabit the bowel and spread to the urinary tract by local extension may be explained by a greater incidence of urinary obstructions which in turn leads to infections (Ishani *et al* ., 2005).

These results were agreed with other result obtained by (Leblebicioglu and Esen., 2003), had reported that 78.4% of patients with UTI had positive culture and those with negative culture may be associated with Mycoplasmal or Chlamydial infections. Approximately 20% of woman attending to S.T.D clinic have *Mycoplasma* in their urinary tract (Brook *et al*.,2007).

These results were agreed with other result obtained by (AL-Jammaly., 2005), when he studied genitourinary tract infections among Women in Mosul and showed that (33.6%) of UTI in woman were caused by *Escherichia coli*. As well, (Gordon and Jones., 2003), showed that 47% of UTI were caused by *Escherichia coli*,

Generally, *Escherichia coli* is the most common bacteria, because it has some virulence factors, which helps in pathogenesis, as pili, which are responsible for adherence in the urinary tract epithelium. With the intention of the adhesion has important role in the ascending to kidney, (Pallett and Hand.,2010). However, it appeared that *K pneumoniae* is the second causative agent in this study because it isolated from (34.32%) of all isolates. So that ,it was different from(AL-Jammaly., 2005), who showed that only (10.8%) of UTI among woman in Mosul were caused by *K pneumonia*e , (Jones *et al*., 2002), who showed that 11% of UTI were caused by *K pneumonia*e, the difference between recent study and (Jones *et al*., 2002) and (AL-Jammaly, 2005) may reflect the difference in population behavior or personal hygiene, because *K pneumonia*e is present in feces of about 5% of normal individuals (Brook *et al*., 2007), So that, bad hygiene may lead to UTI.

In addition to *E coli* and *K pneumoniae,* also *staphylococcus aureus* was important causative agent that cause in this study, because it represented in 13. 43% of all isolates So that these results were differ from the results obtained by Epidemiologic studies had reported that 40-60% of hemodialysis patients are carriers of Staphylococcus as a normal flora (Ena *et al*.,1994). One of the most cause of UTI in hospitalized patients is Pseudomonas aerugenosa due to its ability to resist different types of antibiotics and other chemicals which used in sterilization processes in hospitals(Dennis, *et al*.,2001; Cruze, *et al*.,2009).

The chronic inflammatory state is considered to be a driving force for Autoimmune Glomerulonephritis (AGN). In recent years the IL-17RA/Th17 axis appears to contribute to the pathogenesis of human AGN (Sinclair and Stevens .,2007). that IL-17 signaling is critical for the development of renal pathology and inflammatory changes in the kidney,Consequently IL-17 enhanced the production of proinflammatory cytokines and chemokines from tubular epithelial cells (Steinman .,2007). which are implicated in the recruitment of innate effector cells in the kidney in addition to the rise in the thyroid gland, which is in the case of excessive increase in chronic renal failure patients as this height increases to stimulate Th cells and thus increase the production of IL -17(McLachlan and Rapoport.,2000).

The Fas L/Fas system regulates immune and inflammatory responses. FasL is expressed by renal cells and its expression increases during kidney damage (Andersen *et al*., 2006). Where the damage is happening in the kidney leads to apoptosis and thus stimulate the FasL Who in turn stimulates Fas receptor which Interfere with FasL and consists FADD compound which passes several pathways to to arrived to target cells in kidney (Ghadimi *et al*.,2002).

Urea level a highly significantly $p<0.05$ has been observed in all age groups and the highest concentration was showed in the age group (61-70) years .The reason for the high concentration of urea in the blood serum of patients with chronic renal failure to the fact that urea basic nitrogen material from metabolic waste, which consists mainly in the liver and exclude from the blood by the urine , In the event of a malfunction, and a shortage of kidney function leads to few exclude of urea and accumulate and concentration its increases in the blood (Patel *et al*.,2007) . Blood urea were increased production due to either to a high protein diet or to excessive destruction of cellular proteins of the body as in ;fever, heavy infections .At nearly all types of kidney disease will scoring in urea retention (Harmon.,2009).

The concentration of creatinine were recorded as a higher in age group (21-30) years In contrast, there was significant highly in creatinine is a waste product of muscle breakdown. The same finding was observed many reviews (Allen.,2012) Healthy kidneys were exclude creatinine from the blood by the urine whereas ,the kidney failure creatinine buils in blood until it is removed by dialysis.that the causes of creatinine levels elevation in serum of kidney failure as End Stage Renal Failure (ESRF) was relative independent of protein ingestion, water intake, and rate of urine production (Rebecca *et al*.,2003). Increased dietary intake of creatine or eating a lot of meat can increase daily creatinine excretion,(Faull., 2007).

References:

Ahmed J. and Weisberg L.S. (2001) Hyperkalemia in dialysis patients " , Sem. Dial 14(5): 356-348.

AL-Jammaly M. H. Modruka ;2005. Genitourinary Tract Infections of Women . M.Sc. thesis. College of Science, Mosul University, Iraq.

Allen PJ (May 2012). "Creatine metabolism and psychiatric disorders: Does creatine supplementation have therapeutic value?". Neurosci Biobehav Rev 36 (5): 144262.

Andersen MH, Schrama D, Thor Straten P, Becker JC (2006). "Cytotoxic T cells". *J. Invest. Dermatol.* 126 (1): 3241.

Bakris,G.;Siomos,M.;Richardson,D.;Janssen,I.a nd others.(2000).ACE inhibition or angiotensin receptor blockade:Impact on potassium in Int.;58:2084-2092 renal failure.kidney

Brooks G.F.; J.S. Butel and S.A. Morse. 2007. Jawetz. Melnick and Adelberg's Medical Microbiology. 24th ed,

McGraw-Hill.
Carmody, JB (Feb 2011). "Urine electrolytes". *Pediatr Rev* 32 (2): 6868.
Chue CD, Townend JN, Steeds RP, Ferro CJ (2010). Arterial stiffness in chronic kidney disease: causes and consequences. Heart 96: 817–823.
Collee J.; A.G. Fraser; B.P. Marmian and S.A. Simmon. 1996. Mackie and McCartney. Practical Medical Microbiology. 14th ed. Churchill and Livingstone, INC.
Costanzo, Linda S. (2007) Lippincott, Williams, & Wilkins pp. 260
Cotran, RS S.; Kumar, Vinay; Fausto, Nelson; Robbins, Stanley L.; Abbas, Abul K. (2005). *Robbins and Cotran pathologic basis of disease*. St. Louis, MO: Elsevier Saunders.ISBN 0-7216-0187.
Edwin, R.; Nicholas, A. and Boon, N. (2000) International editor John, A.A.;Hunter Davidson.Principles and practice.19 th ed.Chrestopher Hasleslt:365.Blood pressure response to changes in sodium and potassium intake: Ametaregression analysis of randomized trials Human. J. Hypertens 17:471-480
Eleftheriadis T, Antoniadi G, Liakopoulos V, Kartsios C, Stefanidis I.(2007). Disturbances of acquired immunity in hemodialysis patients. Semin Dial.20 :440– 451.
Ena, J., J.R. Boelaert, B.A. Boyken, H.W. Van Landuyt, C.A. Godard and L.A. Herwaldt, 1994 Epidemiology of Staphylococcus aureus infections in patients on hemodialysis. Infect. Cont . Hosp Ep., 15: 78-81
Faull R (2007). "Prescribing in renal disease". Australian Prescriber30(1): 17–20.
Ghadimi, Markus Philipp; Sanzenbacher Ralf; Thiede Bernd; Wenzel Jennifer; Jing Qian; Plomann Markus; Borkhardt Arndt; Kabelitz Dieter; Janssen Ottmar (May 2002). "Identification of interaction partners of the cytosolic polyproline region of CD95 ligand (CD178)". *FEBS Lett.* (Netherlands) 519 (1–3): 50–58.
Gordon K.A. and R.N. Jones. 2003. Susceptibility patterns of orally administered antimicrobials among urinary tract infection pathogens from hospitalized patients in North America. Diagn. Micr. Infect. Dis; 45(4): 295-301.
Harmon, W.E. (2009).Glomerular filtration rate in children with chronic kidney disease .Clinical chemistry .; 55 :400-401
Haroun MK, Jaar BG, Hoffman SC, Comstock GW, Klag MJ, Coresh J.(2003). Risk factors for chronic kidney disease: Aprospective study of 23,534 men and women in Washington County, Maryland. *J. Am Soc Nephrol* 14: 2934–2941.
Ishani, A., A.J. Collins and C.A. Herzog, 2005 Septicemea access and Cardiovascular disease in dialysis patients: The USRDS wave 2 study Kidney Int., 68: 311- 318
Jones R.N.; P.R. Rhomberg; D.J. Varnam and D Mathai. 2002. A comparison of the antimicrobial activity of selected broad-spectrum antimicrobials tested against multi drug resistant Gram-negative bacilli including bacteraemic *Salmonella spp*.: initial studies for the MYSTIC programme in India. Int. J Antimicrob Agents; 20(6): 426-31.
Kent, R. and Olson, S.(2004). Poisoning& Drugs overdose. Boston: 491. 4 th ed
Kumar, A.(2004). Anemial physiology. Discovery publishing house
Laragh, J.and Sealey, J. (2001). K epletion and the progression of hypertensive disease or heart failure. Hypertensive; 37:806-810
Le,Tao. *First Aid for the USMLE Step 1* 2013. New York: McGraw-Hill Medical, 2013. Print.
Leblebicioglu H. and S. Esen. 2003. Hospital-acquired urinary tract infections in Turkey: a nationwide multicenter point prevalence study. J .Hosp. Infect; 53(3): 207-10.
Malluche , H.H. ; Sawaya, B.P. ; Hakim , R.M Sayegh , M.H. and Ismail, N. (1999) ," Clinical -Nephrology Dialysis and Transplantation" , Dustri verlag Dr. Karl feistle , USA, pp. 1-10
Mark H. Beers, et al. Kidney and urinary tract disorders. In: The Merck Manual of Medical Information, pp.833-834, Merck Research Laboratories, USA, 2003.
McLachlan SM, Rapoport B: Autoimmune response to the thyroid in humans: thyroid peroxidase—the common autoantigenic denominator. Intern Rev Immunol 2000, 19:587-618 [PubMed]
Nazem AK and Moko J ;The effect of calcitrol on renal anemia in patients in dialysis ,Int urol neph ,2003 ,29(4),p 119-127.
Niazi, A.D.(2000). Statistical analysis in medical research. Republic of Iraq. Al-Nehrien University.
Pallett, A.; Hand, K. (Nov 2010). "Complicated urinary tract infections: practical solutions for the treatment of multiresistant Gram-negative bacteria.". *J Antimicrob Chemother*. 65 Suppl 3: iii25–33.
Patel, P.; Abraham ,G.;Pratap,B.; Ramalakshmi ,R.; Mathew,M.; jeevan, J.M Muralidharan ,T.R.;Moorthy,A. and Leslie ,N. (2007).Clinical and biochemical parameters in chronic Kidney disease with pulmonary hypertension .Indian Journal of Nephrology , 17(1) :46
Pohl, Hanna R.; Wheeler, John S.; Murray, H. Edward (2013). "Chapter 2. Sodium and Potassium in Health and Disease". In Astrid Sigel, Helmut Sigel and Roland K. O. Sigel.*Interrelations between Essential Metal*

Ions and Human Diseases. Metal Ions in Life Sciences 13. Springer. pp. 29–47.

Prahalad AK, Hickey RJ, Huang J, et al. (June 2006). "Serum proteome profiles identifies parathyroid hormone physiologic response". Proteomics 6,(12): 3482-93.

Rajiv Vij, SwethaNataraj, Aldo J. Peixoto Diagnostic utility of urinalysis in detecting urinary tract infection in hemodialysis patients. Nephron ClinPract, 2009;113:c281-c285.

Rebecca J and etal.:Nitric oxide production is low in end stage renal disease patients on peritoneal dialysis ,AMJ Phy Sio,2003,4,p612-625

Rebecca J Schmidt, Jean L Holley, Steve J Schwab, Theodore W Post. Non-access related infections in chronic dialysis patients.Last literature review Up-To-Date version 18.3: September 2010.

Reynolds, R. M; Padfield, PL; Seckl, JR (2006). "Disorders of sodium balance". *BMJ* 332 (7543): 702–5.

Ricci, Zaccaria; Ronco, Claudio (2012). "New insights in acute kidney failure in the critically ill". Swiss Medical Weekly 142: w13662..

Sinclair, D; Stevens, JM (Sep 2007). "Role of antineutrophil cytoplasmic antibodies and glomerular basement membrane antibodies in the diagnosis and monitoring of systemic vasculitides.". *Annals of clinical biochemistry* 44 (Pt 5): 432–42.

Springhouse .2005, Professional Guide to Diseases.8th ed, Lippincott Williams and Wilkins.Isban;1:58255.370.

Steinman L (February 2007). "A brief history of T(H)17, the first major revision in the T(H)1/T(H)2 hypothesis of T cell-mediated tissue damage". *Nat. Med.* 13 (2): 139145.

Wald,H.; Garty,L.G.H. ; Palmar and Popovtzer, M.M. (1998).Differential regulation of ROMK expression in kidney cortex a nd medulla by aldosterone and potassium Am.J.Physiol .; 275: 239-245. [Abstract /freefulltext].

Walter F., PhD. Boron (2004). *Medical Physiology: A Cellular And Molecular Approach*. Elsevier/Saunders. ISBN 1-4160-2328-3.

The IISTE is a pioneer in the Open-Access hosting service and academic event management. The aim of the firm is Accelerating Global Knowledge Sharing.

More information about the firm can be found on the homepage:
http://www.iiste.org

CALL FOR JOURNAL PAPERS

There are more than 30 peer-reviewed academic journals hosted under the hosting platform.

Prospective authors of journals can find the submission instruction on the following page: http://www.iiste.org/journals/ All the journals articles are available online to the readers all over the world without financial, legal, or technical barriers other than those inseparable from gaining access to the internet itself. Paper version of the journals is also available upon request of readers and authors.

MORE RESOURCES

Book publication information: http://www.iiste.org/book/

Academic conference: http://www.iiste.org/conference/upcoming-conferences-call-for-paper/

IISTE Knowledge Sharing Partners

EBSCO, Index Copernicus, Ulrich's Periodicals Directory, JournalTOCS, PKP Open Archives Harvester, Bielefeld Academic Search Engine, Elektronische Zeitschriftenbibliothek EZB, Open J-Gate, OCLC WorldCat, Universe Digtial Library , NewJour, Google Scholar