



The Effect of Hemodialysis on Hemoglobin Concentration, Platelets count and White Blood Cells Count in End Stage Renal Failure

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ABSTRACT

To evaluate the effect of hemodialysis machine in complete blood count with focus on hemoglobin, platelets and total white blood cells count for patients of end stage renal disease, to evaluate the effect of dialysis on hemoglobin, platelets and white blood count, to estimate the values of change session of dialysis, to clarify the major cause of End Stage Renal Failure among the study group. 3 ml of blood were collected from 199 patients, aseptically by standard phlebotomy technique by trained phlebotomist from each patient and dispensed in to tri-potassium Ethylenediamine tetra-acetic acid(K3 EDTA) anticoagulant containers about 10-15 minutes after the hemodialysis. The study revealed that (83,9%) of patients with higher decrease range reach to 4.3g, about.(14.1%) have stable concentration, and only(2%) their Hb increased after dialysis, 83.9% of patients have noticeable increase in , 14.1% of patients show decrease in TWBCs and 2% have stable count, there is decrease in platelets count in (99.5%) of patients almost in and only one patient showed stable count after dialysis (0.5%), The study revealed that a significant number of low hemoglobin concentration , low platelets count and high white blood count.

Key words: Hemoglobin (Hb), White blood cells, Platelets.

INTRODUCTION

Chronic renal disease (CRD) is a patho-physiologic process with multiple etiologies, resulting in the inexorable attrition of Nephron number and function and frequently leading to end-stage renal disease (ESRD). In turn, ESRD represents a clinical state or condition in which there has been an irreversible loss of endogenous renal function, of a degree sufficient to render the patient permanently dependent upon renal replacement therapy (dialysis of transplantation) in order to avoid life – threatening uremia [32]. Wide geographical variations in the incidence of disorders causing CRD exist [40]. The high incidence of chronic kidney disease among Black Americans has been demonstrated in several studies. Unfortunately, lack of functioning registries in most of sub-Saharan Africa has resulted in a lack of reliable statistics. However, there is a general impression that it is at least 3–4 times more frequent than in more developed countries; uremia was reported to account for 1.0%–1.5% of total annual deaths among Egyptians, both in the pre-dialysis era and for 2 decades thereafter [5]. Chronic kidney disease affects mainly young adults aged 20–50 years in sub-Saharan Africa and is primarily due to hypertension and glomerular diseases, unlike developed countries where chronic kidney disease presents in middle-aged and elderly patients and is predominantly due to diabetes mellitus and hypertension [3]. Hypertension is a cause of chronic kidney failure in Africa, especially in Black patients. Hypertension affects <25% of the adult population and is the cause of chronic kidney failure in 21% of patients on renal replacement therapy in the South African Registry [66], hypertension was the most common cause of end-stage renal disease (ESRD) in Black South Africans and accounted for 34.6% of

ESRD in that racial group. In contrast, hypertension was reported to be the cause of ESRD in 4.3% of Whites, 13.8% of Indians, and 20.9% of people of mixed ancestry. In a study to determine the pathologic basis of ESRD in Black South Africans, essential malignant hypertension was the single most common cause of ESRD, occurring in 49%^[24]. The estimated increase in diabetes mellitus in Africa is anticipated to be 12.7 million, an increase of 140% by 2025^[70]. Diabetes mellitus may present one of the most daunting challenges in the future and affects 9.4 million people in Africa^[48]. Screening programs are in their infancy in most of Africa, with a few programs in South Africa, Nigeria, Ghana, and Kenya. Screening for kidney disease in high-risk populations (e.g., patients with hypertension and diabetes mellitus and a family history of kidney disease) should probably be instituted as the first step in kidney disease prevention in developing countries such as Africa^[34]. Education of patients and health care workers regarding hypertension, diabetes, obesity, and protein urea and health promotion (e.g., prudent diet and exercise) is essential. COMGAN programs and World Kidney Day have heightened awareness of kidney disease in medical professionals and the public. Efforts should be made to optimize the therapy of hypertension, diabetes mellitus, and renal disease. Implementation of recommended targets for control of hypertension and diabetes is essential. In areas where there are insufficient numbers of physicians, nurses and other health workers could be trained to manage these conditions at a local level, with clearly defined criteria for referral of patients^[62].

Thrombocytopenia is a known potential side effect of hemodialysis^[33]. In pre-dialysis patients, as well as in hemodialysis patients, platelet number tends to be reduced^[21].

Platelets have been known to interact with dialysis membranes since the 1970's; dialysis membranes have been shown to cause platelet adhesion, aggregation, and activation^[43].

General objectives

To evaluate the effect of hemodialysis in complete blood count with focus on hemoglobin, platelets and total white blood cells count for patients of end stage renal disease.

Specific objective

1. To evaluate the effect of dialysis on hemoglobin, platelets and total white blood count.
2. To estimate the values of change/ session of dialysis.
3. To clarify the major cause of End Stage Renal Failure among the study group.

The Blood:

The blood is composed of cells, cell fragments and an aqueous solution (plasma). Blood makes up about 8% of the human body weight. It contains erythrocytes, leucocytes, thrombocytes (platelets) and plasma. The volume percentage of all blood cells in the whole blood is about 45% in adults (hematocrit). The rest consists of liquid plasma (e.g. water, plasma proteins, electrolytes, etc)^[29].

Red blood cells:

Red blood cells have a unique structure. Their flexible disc shape helps increase the surface area-to-volume ratio of these extremely small cells. This enables oxygen and carbon dioxide to diffuse across the red blood cells plasma membrane more readily. Red blood cells contain enormous amounts of a protein called hemoglobin. This iron containing molecule binds oxygen as oxygen molecules enter blood vessels in the lungs. Unlike other cells of the body, mature red blood cells do not contain a nucleus, mitochondria, or ribosomes. The absence of these cell structures leaves room for the hundreds of millions of hemoglobin molecules found in red blood cells. The shape of red blood cells provides it ability to maneuver through tiny blood vessels to deliver oxygen to organs and tissues. Red blood cells are also important in determining human blood type. Blood type is determined by the presence or absence of certain identifiers on the surface of red blood cells. These identifiers, also called antigens, help the body's immune system to recognize its own red blood cell type^[29].

The white blood cells:

Also called leukocytes. They originate from bone marrow stem cells and circulate in blood and lymph fluid. Leukocytes are able to leave blood vessels to migrate to body tissues. White blood cells are categorized by the apparent presence or absence of granules (sacs containing digestive enzymes or other chemical substances) in their cytoplasm.

A white blood cell is considered to be a granulocyte or an agranulocyte^[59]. There are three types of granulocytes: neutrophils, eosinophils, and basophils.

As seen under a microscope, the granules in these white blood cells are apparent when stained. Neutrophils cells have a single nucleus that appears to have multiple lobes. Neutrophils are the most abundant granulocyte in blood circulation. They are chemically drawn to bacteria and migrate through tissue to the site of infection.

Neutrophils are phagocytic in that they engulf the target cell (bacterium, diseased or dead cell, etc) and destroy it. When released, neutrophil granules act as lysosomes to digest cellular macromolecules^[59].

Eosinophils nucleus is double lobed and often appears U-shaped in blood smears. Eosinophils are often found in connective tissues of the stomach and intestines. Eosinophils are phagocytic and primarily target antigen-antibody complexes. These complexes are formed when antibodies bind to antigens to identify them as substances to be destroyed. Eosinophils become increasingly active during parasitic infections and allergic reactions. Basophils are the least numerous of the white blood cells. They have a multi-lobed nucleus, and their granules contain substances such as histamine and heparin. Heparin thins blood and inhibits blood clot formation. Histamine dilates blood vessels, increases the permeability of capillaries, and increases blood flow, which helps to transport leukocytes to infected areas. Basophils are responsible for the body's allergic response^[59]. There are two types of agranulocytes: lymphocytes and monocytes. These white blood cells appear to have no obvious granules. Agranulocytes typically have a large nucleus due to the lack of noticeable cytoplasmic granules. After neutrophils, lymphocytes are the most common type of white blood cell. These cells are spherical in shape with large nuclei and very little cytoplasm. There are three main types of lymphocytes: T cells, B cells, and natural killer cells. T cells and B cells are critical for specific immune responses. Natural killer cells provide nonspecific immunity. Monocyte cells are the largest of the white blood cells. They have a large, single nucleus that can have various shapes. The nucleus often appears to be kidney-shaped. Monocytes migrate from blood to tissues and develop into macrophages and dendritic cells. Macrophages are large cells present in nearly all tissues. They actively perform phagocytic functions. Dendritic cells are commonly found in tissue located in areas that come in contact with antigens from the external environment. They are found in the skin, internally in the nose, lungs, and gastrointestinal tract. Dendritic cells function primarily to present antigenic information to lymphocytes in lymph nodes and lymph organs. This aids in the development of antigen immunity. Dendritic cells are so named because they have projections that are similar in appearance to the dendrites of neurons^[59].

Cells fragment (the platelets):

Platelets are extremely small and discoid 3,0x0,5 micrometer in diameter. With mean volume 7-11 fimtoliter (Lucile, 2015) they are the smallest of the three major types of blood cells are only about 20% of the diameter of red blood cells^[18]. Platelets are produced in the bone marrow by fragmentation of cytoplasm of megakaryocytes, one of the largest cells in the body. The precursor of the megakaryocytes – the megakaryoblast – arises by the process of differentiation from the haemopoietic stem cells^[46].

The dominant hormone controlling megakaryocytes development is thrombopoietin. Platelets are actually not true cells but merely circulating fragments of cells. But even though platelets are merely cell fragments, they contain many structures that are critical to stop bleeding. They contain proteins on their surface that allow them to stick to breaks in the blood vessel wall and also to stick to each other. They contain granules that can secrete other proteins required for creating a firm plug to seal blood vessel breaks. Also platelets contain proteins similar to muscle proteins that allow them to change shape when they become sticky^[18]. The main function of platelets is formation of mechanical plugs during normal haemostatic response to vascular injury. In the absence of platelets, spontaneous leakage of blood through small vessels may occur. The immobilization of platelets at the sites of vascular injury require specific platelets- vessel wall (adhesion) and platelets – platelets (aggregation) interaction^[27].

The plasma:

Plasma is the forgotten component of the blood. White blood cells, red blood cells and platelets are essential to the body function, but plasma also plays a crucial, and mostly unrecognized, job, carrying these blood components throughout the body as the fluid in which they travel. Plasma is largest component of the blood making about 55% of its overall contents. When it is isolated on its own, blood plasma is light yellow liquid, similar to the color of straw. Along with water, plasma carries salts and enzymes.

The important constituents include electrolytes such as sodium, potassium, chlorine, bicarbonate, magnesium, and calcium, in addition, there are trace amounts of other substances, including amino acids, vitamins, organic acids, pigments, and enzymes. Hormones such as insulin, corticosteroids, and thyroxin are secreted into the blood by the

endocrine system. Plasma concentration of hormones must be carefully regulate for blood health. Nitrogenous wastes (e.g., urea and creatinine) transported to the kidney for excretion increase markedly with renal failure^[10].

Urinary system:

Also known as the renal system produces stores and eliminates urine, the fluid waste excreted by the kidneys. The urinary system consists of the kidneys, ureters, urinary bladder, and urethra. The kidneys filter the blood to remove wastes and produce urine. The ureters, urinary bladder, and urethra together form the urinary tract which acts as plumbing system to drain urine from the kidneys, store it, and then release it during urination^[36].

The kidneys:

Kidneys are primary organs of the renal system; they perform the function of the renal system. The paired kidneys are located between the twelfth thoracic and third lumbar vertebrae, one on each side of the vertebral column. The right kidney is usually lower than the left because the liver displaces it downward. In the adult each kidney is approximately 3cm thick, 6cm wide and 12cm long. It is roughly bean shaped with an indentation, called hilum, on the medial side. The hilum leads to a large cavity called the renal sinus, within the kidney. The ureters and renal vein leave the kidney, and renal artery enters the kidney at the hilum. The outer reddish, next to the capsule, is the renal cortex.

This surrounds a darker reddish brown called the renal medulla consists of a series of renal pyramids, which striated because they contain tubular structure and blood vessels. The wide bases of the pyramids are adjacent to the cortex and the pointed ends, called renal papillae. The cortex and medulla make up the parenchyma or functional tissue, of the kidney^[69]. The central region of the kidney contains the renal pelvis, which is located in the renal sinus and continuous with the ureters. The renal pelvis is a large cavity that collects the urine as it is produce. Each kidney contains over million functional units called nephrons, in the parenchyma (cortex and medulla). A nephrons has two parts: renal corpuscle and a renal tubules^[69]. The renal corpuscle consist of a cluster of capillaries, called glomerulus, surrounded by a double layered epithelial cup, called the glomerular capsule. The juxtaglomerular apparatus, which monitors blood pressure and secret renin enzyme, is formed from modified cells in the afferent arterioles and the ascending limb of the nephrons loop^[69].

Physiology of the kidney:

- Excretion of waste products such as urea, uric acid.
- Control of ECF volume (by excretion of more or less water in urine).
- Control of ECF electrolytes (by regulation of electrolytes excretion in urine).
- Control of ECF osmolarity(by regulation of sodium and water excretion).
- Control of blood pressure (long term effect).
- Control of pH (acid base balance).
- Endocrine function:-
 - I. Synthesis and secretion of erythropoietin.
 - II. Activation of vitamin D.
 - III. Release of renin enzyme in to the blood^[26].

The ureters:

From the kidneys, urine travels down two thin tubes, called ureters, to the bladder. The ureters are about 8 to 10 inches long (20 to 25 centimeters). Muscles in the ureter walls continuously tighten and relax to force urine away from the kidneys, a backup of urine can cause a kidney infection. Small amounts of urine are emptied into the bladder from the ureters about every 10 to 15 seconds^[36].

The bladder and urethra:

The bladder is a hollow, balloon-shaped organ that is located in the pelvis. It is held in place by ligaments attached to other organs and the pelvic bones, according to the Kidney and Urology Foundation of America. The bladder stores urine until the brain signals the bladder that the person is ready to empty it. A normal, healthy bladder can hold up to 16 ounces (almost half a liter) of urine comfortably for two to five hours.

To prevent leakage, circular muscles called sphincters close tightly around the opening of the bladder into the urethra, the tube that allows urine to pass outside the body. The only difference between the female and male urinary system is the length of the urethra, according to Merck Manuals. In females, the urethra is about 1.5 inches (3.8 cm)

to 2 inches (5.1 cm) long and sits between the clitoris and the vagina. In males, it runs the length of the penis, is about 8 inches (20 cm) long and opens at the end of the penis. The male urethra is used to eliminate urine as well as semen during ejaculation^[36].

Renal diseases

Urinary tract infections (UTIs) occur when bacteria enters the urinary tract and can affect the urethra, bladder or even the kidneys. While UTIs are more common in women, they can occur in men. Incontinence is another common disease of the urinary system. It can come in the form of a pelvic prolapse, which can result in leakage and can be the result of a vaginal delivery. Then there is the overactive bladder, “which we see a lot and is not related to having children or trauma, Dr. Oscar Aguirre, urogynecologist in Denver said. A third condition involves overflow, in which the bladder does not completely empty. Some common treatments involve medications, physical therapy and pelvic mesh surgery, Aguirre noted. Vaginal laser surgery is also becoming a viable treatment option, he explained. In another 10 to 15 years, vaginal laser surgery will be another common option for the treatment of urinary conditions^[36]. Interstitial cystitis (IC), also called painful bladder syndrome, is a chronic bladder condition, primarily in women, that causes bladder pressure and pain and, sometimes, pelvic pain to varying degrees, according to the Mayo Clinic. It can cause bladder scarring, and can make the bladder less elastic. While the cause isn’t known, many people with the condition also have a defect in their epithelium, the protective lining of the bladder. Prostatitis is a swelling of the prostate gland and, therefore, can only occur in men. Often caused by advanced age, symptoms include urinary urgency and frequency, pelvic pain and pain during urination, the Mayo Clinic noted^[36]. Kidney stones are clumps of calcium oxalate that can be found anywhere in the urinary tract. Kidney stones form when chemicals in the urine become concentrated enough to form a solid mass, according to the Cleveland Clinic. They can cause pain in the back and sides, as well as blood in the urine. Many kidney stones can be treated with minimally invasive therapy, such as extracorporeal shock wave lithotripsy, which disintegrates the kidney stones with shock waves. Bladder cancer is diagnosed in about 75,000 Americans each year and is more frequent in men and the elderly according to the American Cancer Society. The symptoms, including back or pelvic pain, difficulty urinating and urgent/and or frequent urination, mimic other diseases or disorders of the urinary system. Kidney failure, also called renal failure and chronic kidney disease, can be a temporary (often acute) condition or can become a chronic condition resulting in the inability of the kidneys to filter waste from the blood. Other conditions, such as diabetes and hypertension, can cause chronic kidney disease, according to the Mayo Clinic. Acute cases may be caused by trauma or other damage, and may improve over time with treatment. However, renal disease may lead to chronic kidney failure, which may require dialysis treatments or even a kidney transplant^[36]. Chronic renal failure (CRF) is defined as an irreversible and progressive reduction in the glomerular filtration rate (GFR) to below 25% of the normal level (decline of 30 ml/min/1.73 m²) for at least three months.

Creatinine clearance (Ccr) is a good indicator of the GFR and is helpful in monitoring renal function of patients in various age groups. CRF staged according to residual renal function into: Impaired renal function: 40-80%; Chronic renal insufficiency: 25 – 50%; Chronic renal failure: <30%; ESRD: < 10%. CRF is characterized by progressive destruction of renal mass with irreversible sclerosis and loss of nephrons over a period of at least months to many years, depending on the underlying etiology^[39]. Regardless of the cause of kidney damage, once a critical level of renal functional deterioration is reached, progression to end stage renal failure is inevitable^[52]. The term impaired renal function refers to an individual who is asymptomatic and has renal function of 40% to 80% of normal. The term CRI is associated with residual renal function of 25% to 50% of normal. At this level of renal function, distinct biochemical abnormalities may present only when the patient is stressed. The term CRF is used to describe a patient who has residual renal function of less than 30%. The patient with CRF exhibits biochemical abnormalities even when not stressed. ESRD is a term reserved, when dialysis or transplantation is required; uremia is a symptom complex that includes anorexia, nausea, itching, neuropathy and malaise^[52].

End stage renal disease

End-stage renal disease is a condition in which the kidneys no longer function normally. "Renal" describes anything having to do with the kidneys. Nearly everyone is born with two kidneys. They both need to fail for end-stage renal disease to develop. Kidneys eliminate poisons from the body, and keep a normal balance of fluid and certain minerals in the body. When the kidneys can no longer perform this function, a person becomes very ill and ultimately dies. In end-stage renal disease, the kidneys function at a fraction of their normal capacity.

When this occurs, there are only two options: replace the job the kidneys are supposed to do by using a machine, instead (kidney dialysis) or transplant a new, healthy kidney. A single new kidney can do the work of the two

kidneys. Diabetes is the leading cause of end-stage renal disease. Kidney disease can result from type 1 or type 2 diabetes. With either type, poor control of blood sugar increases the risk of end-stage renal disease ^[14].

Other common causes of end-stage renal disease are:

- High blood pressure
- Atherosclerosis
- Autoimmune diseases like systemic lupus erythematosus (lupus)
- Genetic disorders, such as polycystic kidney disease
- Exposure to toxic drugs, including:
 - o certain antibiotics
 - o chemotherapy
 - o contrast dyes
 - o Pain relievers ^[14].

Treatment

The two treatments for end-stage renal diseases are dialysis and kidney transplant.

Dialysis

Clinically, dialysis is a technique in which substances move from the blood through a semi permeable membrane and into a dialysis solution (dialysate). It is used to correct fluid and electrolyte imbalances and to remove waste products in renal failure.

Dialysis is the movement of fluid and molecules across a semi permeable membrane from one compartment to another ^[37]. There are two types of dialysis:

1. Hemodialysis. During hemodialysis, blood is removed from a vein. It is run through filters to remove waste products. The blood is then returned to the body. Hemodialysis usually is done at a dialysis center. The treatments are done three times a week, in three- to four-hour sessions.
2. Peritoneal dialysis. During peritoneal dialysis, sterile fluid is infused into the abdomen. Waste products gradually accumulate in the fluid, which is drained several hours later. Peritoneal dialysis is done at home. It takes longer than hemodialysis and must be done four to five times a day. It can be automated to occur during sleep ^[37]

General principles of hemodialysis:

- a. Hemodialysis diffusion: is the movement of solutes from an area of greater concentration to an area of lesser concentration.
- b. Ultra filtration: (water and fluid removal) results when there is an osmotic gradient or pressure gradient across the membrane.
- c. Dialysis solution: Dialysis solutions are available commercially in 1 or 2 liters (sometimes large or small) with glucose concentration of 1.5%, 2.5%, and 4.25%. The electrolyte composition is similar to that of plasma.

Peritoneal dialysis system:

Three types of peritoneal dialysis currently being used are:

1. Conventional dialysis.
2. Automated peritoneal dialysis.
3. Continuous ambulatory peritoneal dialysis

Automated peritoneal dialysis:

An automated device called a cycler is used to deliver the dialysate for APD. The automated cycler times and controls the fills, dwell and drain phases. The machine cycles 4 or more exchanges per night with 1-2 hours per exchange.

Continuous ambulatory peritoneal dialysis:

Instillation of 2 liters of dialysate fluid into peritoneal cavity Leave for equilibration period Drain the dialysate fluid back from peritoneal cavity ^[37].

Hemodialysis:

Hemodialysis removes toxic wastes and other impurities from the blood of a patient. In hemodialysis, blood is removed from the patient's body through a surgically created access site, pumped through a filtration unit to remove toxins, and then returned to the body.

Procedure: Prepare access. Flush the lines with NS. Add heparin to blood. Counter flow - blood flow rate 200-500 ml/min - dialysate flow rate 300-900 ml/min. Return of blood to patient [28].

Dialysis machine:

Is a medical machine that removes excess water and waste materials from a patient by filtering the blood of the patient. Dialysis is an alternative for many duties of the human kidneys. Therefore, a dialysis machine can be referred to as an artificial kidney. A dialysis machine consists mainly of three components or parts. It has plastic tubing whose role is to carry the blood removed from the patient to the second part called the dialyzer. This is a collection of open-ended fibers forming a semi-permeable kind of a membrane which filters out the nitrogenous impurities from the body. The dialyzer diffuses the blood by the use of a salty solution; dialysate which is in turn diffused with the patient's removed blood. The patient receives back cleansed blood after the filtration process is over. A patient with kidney problems can use the dialysis machine at a dialysis clinic or use a peritoneal dialysis at home [28].

Dialysis machine benefits:

There are a number of dialysis machine benefits include; a patient will enjoy four dialysis-free days in a week. This is because the dialysis is done once in a week unlike the peritoneal dialysis. It is also possible to have a dialysis machine installed in the patient's home thereby saving him/her from having to make regular dialysis unit visits. However, there are criteria to be met before considering a home hemodialysis and these include: the patient's physical and mental capability to operate the dialysis equipment, whether or not previous home hemodialysis has stabilized the patient's symptoms and whether you have a caregiver who has made a solid decision to help you with the dialysis among other criteria [28]. Another dialysis machine benefit is that a patient can get attention and medical supervision of the unit staff which is assuring and comforting. Using a dialysis machine at a clinical unit also has the benefit of giving the patient a contact with other kidney patients and staff. Another dialysis machine benefit is the quick medical availability in case of an emergency because the patient is near a medical unit.

On the other hand, there are a number of dialysis machine drawbacks. Which include a permanent access usually in your arm is required. This may bulge as more and more needles are inserted. During each treatment, there is insertion of two needles. This may create needle phobia with the patient. It also calls for a restricted diet often with limited fluid intake. There are risks of infection and possible cases of nausea, tiredness, leg cramps or even headache discomfort [28].

Dialysis access:

Before treatments can begin, the doctor will need to create a site where the blood can flow in and out of the body during the dialysis sessions. This is called the dialysis access. The type of dialysis access will depend in part on how quickly the patient needs to begin hemodialysis. There are different types of access for hemodialysis:

a. Fistula: A fistula is created by connecting one of the arteries to one of the veins in the lower arm. A fistula allows repeated access for each dialysis session. It may take several months for the fistula to form. A fistula may not clot as easily as other dialysis access methods. A fistula is the most effective dialysis access and the most durable. Complications include infection at the site of access and clot formation (thrombosis).

b. Graft: A vascular access that uses a synthetic tube implanted under the skin in the arm (graft) may be used if you have very small veins. The tube becomes an artificial vein that can be used repeatedly for needle placement and blood access during hemodialysis. A graft does not need to develop as a fistula does, so a graft can sometimes be used as soon as 1 week after placement. Compared with fistulas, grafts tend to have more problems with clotting or infection and need to be replaced sooner. A poly tetra fluoro ethylene (PTFE or Gore-Tex) graft is the most common type used for hemodialysis.

c. Venous catheter: A tube, or catheter, may be used temporarily if the patient has not had time to get a permanent access. The catheter is usually placed in a vein in the neck, chest, or groin. Because it can clog and become infected, this type of catheter is not routinely used for permanent access. But if you need to start

hemodialysis right away, a catheter may be used until your permanent access is ready. Hemodialysis for acute kidney injury may be done daily until kidney function returns ^[2].

Hematological Complications of chronic renal failure:

Anemia:

Anemia commonly occurs in people with chronic kidney disease (CKD) the permanent, partial loss of kidney function. Anemia might begin to develop in the early stages of CKD, when someone has 20 to 50 percent of normal kidney function. Anemia tends to worsen as CKD progresses. Most people, who have total loss of kidney function, or kidney failure, have anemia. A person has kidney failure when he or she needs a kidney transplant or dialysis ^[68]. Healthy kidneys produce a hormone called erythropoietin (EPO). A hormone is a chemical produced by the body and released into the blood to help trigger or regulate particular body functions. EPO prompts the bone marrow to make red blood cells, which then carry oxygen throughout the body ^[12].

Normally 90% of the hormone is produced in peritubular interstitial cells of the kidney and 10% in the liver and elsewhere, there are no performed stores and the stimulus to erythropoietin production is then oxygen tension in the tissues of the kidney, erythropoietin production there for increase in anemia, when hemoglobin for some reason is unable to give up oxygen normally, when atmospheric oxygen is low or when destructive cardiac or pulmonary function or damage to the renal circulating affect oxygen delivery to the kidney. Recombinant erythropoietin is of great value in treating anemia resulting from renal disease or from various other causes. It is given intravenously or subcutaneously either 3-7 times weekly or once every 1-2 weeks depending on indication and on the preparation used (erythropoietin alpha or beta or darbepoetin alpha, a heavily glycosylated longer acting form). The main indication is end stage renal disease (with or without dialysis) other include pre-autologous blood transfusion, the anemia of chronic disorders, and some cases of myelodysplasia or myeloma, in these conditions higher dose are used ^[27]. Hemoglobin levels in individuals with chronic kidney disease fluctuate frequently above or below the recommended target levels within short periods of time even though the calculated mean hemoglobin remains within the target range of 11 to 12 g/dl ^[31]. Correction of anemia and maintenance of stable hemoglobin levels using erythropoiesis-stimulating agents (ESA) is an important aspect of disease management ^[68]. In clinical studies, moderate increase in hemoglobin concentration is associated with relief from symptoms of anemia, improved quality of life, and decreased blood transfusion rate ^[51]. The National Kidney Foundation Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines recommend target hemoglobin levels in the range 11 to 12 g/dl, whereas hemoglobin >13 g/dl should be avoided ^[35]. Several recent randomized clinical trials showed targeting hemoglobin levels >13 g/dl to "normalize" hemoglobin in CKD may be associated with poor clinical outcomes ^[9]. and recent expert review by the Food and Drug Administration has left the target range between 10 and 12 g/dl unchanged ^[22]. A 2-yr study by Ofsthun ^[53]. showed that of 41,919 dialysis patients, >50% spent >1.2 to 6.0 mo at hemoglobin levels <11 g/dl. A longitudinal analysis that was conducted by Lacson ^[41]. and involved >65,000 dialysis patients showed only approximately 38% had hemoglobin levels within the range of 11 to 12 g/dl. Despite a mean hemoglobin level of 11.5 g/dl, the average individual patient had a ± 1.4 -g/dl fluctuation in hemoglobin during the course of 1 yr on the basis of 3-mo rolling average values ^[41]. A 15-mo retrospective study of standard clinical practice conditions demonstrated substantial hemoglobin variability in 987 epoetin-treated hemodialysis patients ^[7]. The range of mean hemoglobin values (10.9 to 11.2 g/dl) that included the middle 50, 80, and 90% of values from a single month were within 1.7, 3.3, and 4.4 g/dl, respectively. One-month hemoglobin values exhibited the greatest degree of variability, with longer rolling intervals being associated with narrower hemoglobin ranges; however, even when a 6-mo rolling average was applied, <50% of hemodialysis patients had hemoglobin values within the KDOQI-recommended 11- to 12-g/dl range.

Hemoglobin levels >12 g/dl were predicted to occur approximately 21% of the time ^[7]. In addition to associations of blood hemoglobin with mortality and hospitalizations in the CKD population, anemia is associated with fatigue, weakness, shortness of breath, and a decreased health-related quality of life ^[47].

Furthermore, hemoglobin overshoot may be associated with various safety concerns, including the development of elevated BP with risk for hypertensive encephalopathy ^[15]. iron deficiency, ^[16]. high platelet count ^[6]. thrombotic events ^[38]. and accelerated left ventricular dysfunction and hypertrophy ^[55].

Thrombocytopenia

Thrombocytopenia is a known potential side effect of hemodialysis, however, it is rarely seen in patients who undergo hemodialysis using biocompatible membranes ^[33].

Platelet number, survival, and function in chronic kidney disease:

In pre-dialysis patients, as well as in hemodialysis patients, platelet number tends to be reduced^[21], in the range of 175–180,000/mm³ compared with 250,000/mm³ in healthy controls. In continuous ambulatory peritoneal dialysis patients, platelet counts have been reported to be closer to the normal range^[45]. Platelet survival in hemodialysis patients is thought to be of normal duration, although the only paper examining this was published in 1967^[64]. The megakaryocyte number in bone marrow is normal^[21], but the reticulated platelet count, a measure of thrombopoiesis, is reduced, despite elevated thrombopoietin levels^[45],^[1]. Platelets have been known to interact with dialysis membranes since the 1970's; dialysis membranes have been shown to cause platelet adhesion, aggregation, and activation^[43]. Platelet activation has been demonstrated by elevated levels of platelet factor 4^[11,13], as well as thromboxane^[72], following hemodialysis. Accordingly, thrombocytopenia is also a well-known complication of hemodialysis treatment.

Hakim and Schafer suggested that thrombocytopenic episodes occurring with hemodialysis were associated with complement activation, specifically C3a, in addition to activation of platelets themselves^[25]. Complement activation occurred specifically in the setting of cuprophane membranes, and thrombocytopenia was only observed in the presence of complement activation^[72].

Thrombocytopenia in dialysis patients:

In addition to HIT, dialysis patients, especially those in the intensive care unit, but even those treated as outpatients in dialysis units, often can be affected by other medical conditions associated with thrombocytopenia. In acutely ill patients with sepsis, thrombocytopenia is common, with or without disseminated intravascular coagulation^[20]. Platelet consumption due to thrombotic thrombocytopenic purpura or idiopathic thrombocytopenic purpura, due either to immunologic diseases, especially various forms of vasculitis, or drugs, is not uncommon in renal patients. Physical destruction of platelets may occur because of intravascular catheters^[43]. Thrombocytopenia and platelet dysfunction are commonly seen in liver disease, paraproteinemia, myeloproliferative disorders, and myelodysplastic syndrome^[52]. Thrombocytopenia may be found in patients receiving nicotinamide for treatment of cholesterol abnormalities or hyperphosphatemia^[63], and many drugs sometimes taken by hemodialysis patients, including clopidogrel and other antiplatelet agents, as well as quinine, for example, can cause drug-induced thrombocytopenia. Finally, in actively bleeding patients, dilutional thrombocytopenia can occur when transfusing packed red blood cells (RBCs) only, as functional platelets are not present in packed RBC transfusions^[23].

MATERIALS AND METHODS

Study population:

The study conducted on patients attending the Gezira Hospital for Renal Diseases and Surgery, at Wad Medani City, the capital of Gezira State. There were 6 of Renal Diseases and Surgery centers at Gezira state; 5 governmental and one private, about 960 patients on regular hemodialysis. The Gezira center established in 2003, a center for referral of patients with chronic kidney diseases, now it contain 23 machines (1 for HBV + ve patients, 1 for HCV + ve patients, 1 for emergency not booking patients and 20 for patients on regular hemodialysis(HD)).

Study subjects:

Since the international prevalence of end stage renal disease(ESRD) is very small and there is no prevalence from nearby country, a total coverage sample has been taken. This included all patients with ESRD presenting to the Gezira center and fulfilling the inclusion criteria, from July–September 2015. Patients were asked to participate in the study when they came to the Center for dialysis. The purpose of the study was explained to each patient and then the patient chose to accept or decline to participate. Those patients who did not meet the inclusion criteria were thanked for their time and not included in the study. The total number was 199 patients during the period of study.

Inclusion criteria:

All patients with ESRD were included, after taking verbal consent from them.

Exclusion criteria:

Patients with infection, inflammation, dehydration and on station therapy.

Sampling and Biochemical Measurements:

About 3 ml of blood were collected aseptically by standard phlebotomy technique by trained phlebotomist from each patient and dispensed in to tri-potassium Ethylenediamine tetra-acetic acid(K3 EDTA) anticoagulant contained containers before and about 10-15 minutes after the hemodialysis and processed by SYSMEX KX 21N in the Laboratory of Renal Center. The remain of pre-dialysis samples were tested to measure serum albumin, cholesterol, urea and creatinine values.

Clinical data includes:

Personal data(age, sex, occupation, education level, causes of ESRD, vascular access, duration of HD and sessions.

Statistics:

The data was entered into the computer (the Statistical Package for Social Studies; SPSS) which used for analysis.

RESULTS AND DISCUSSION**The statistics of the study population samples:**

199 patients were included in the study. The majority of patients are males (132) represents (66, 3%) with most frequent age group (45-54) about (18%) which is the largest percentage among all gender groups and 67 females (33, 7%).

The mean (+ SD) age of the study group was 46.2 years (range: 47-45.4 years). The age distribution of patients in the study group is shown in (Figure, 4.2). Almost about two third of the patients were married(69%) and (31%) single (Figure, 4.3).

More than the half of the patients (53.8%) was reached primary school, (23.6%) illiterate, (19%) secondary school and only (3.5%) university (Figure, 4.4). The majority of the patients (73.9%) were unemployed and (14.6%) free workers (Figure, 4.5).

AV fistula is the main venous access method used (99%) and only (1%) undergo permanent catheter access (Figure, 4.6). Most of the patients (97.5%) had two HD sessions per week (Figure, 4.7). 41.2% of the patients, hypertension causes their ESRD,(18, 1%) GN and (18, 6%) others causes (Figure, 4.8).

(34,2%) of patients start dialysis more than 48 weeks, (25,6%) their starting duration range from 12 -24 weeks.

The effect of hemodialysis on platelets count:

The study found that there is decrease in platelets count in (99.5%) of patients almost in all age intervals with only one patient showed stable count after dialysis (0.5%), Considering the count (140-400,000/mm³) is the standard control count, Table (4.1) .The higher decrease range present among the age group (45-54) ranging from 1.0-101,000/mm³, followed by group (75-84) with decrease count rang 2.0 - 94,000/mm³.

Thrombocytopenia is a known potential side effect of hemodialysis ^[32]. Dialysis membranes have been shown to cause platelet adhesion, aggregation, and activation ^[43]. Platelet activation has been demonstrated by elevated levels of platelet factor 4 ^[11,13], as well as thromboxane ^[72], following hemodialysis. Accordingly, thrombocytopenia is also a well-known complication of hemodialysis treatment ^[26].In pre-dialysis patients, as well as in hemodialysis patients, platelet number tends to be reduced ^[21], in the range of 175–180,000/mm³ compared with 250,000/mm³ in healthy controls.

In continuous ambulatory peritoneal dialysis patients, platelet counts have been reported to be closer to the normal range ^[45]. Platelet survival in hemodialysis patients is thought to be of normal duration, although the only paper examining this was published in 1967 ^[64]. The megakaryocyte number in bone marrow is normal,1 but the reticulated platelet count, a measure of thrombopoiesis, is reduced, despite elevated thrombopoietin levels ^[1,45]. Despite the many possible causes for thrombocytopenia in dialysis patients, a number of reports have been published, in which there is no obvious explanation other than the dialysis procedure ^[67,71,58,57,54].

The effect of hemodialysis on hemoglobin concentration:

The study found that there were normocytic normochromic anemia in (87,4%) of patients and the rest (12,6%) have hemoglobin concentration greater than 11g/dl. The concentration of (11-17g/dl) considered as target hemoglobin level of normal control individuals, decrease ratio on hemoglobin concentration/ session of dialysis demonstrated in (83,9%) of patients with higher decrease range reach to 4.3gabout.(14.1%) have stable concentration, and only(2%) their Hb increased after dialysis. Hemoglobin levels in individuals with chronic kidney disease fluctuate frequently above or below the recommended target levels within short periods of time even though the calculated mean hemoglobin remains within the target range of 11 to 12 g/dl^[31]. Anemia commonly occurs in people with chronic kidney disease (CKD) the permanent, partial loss of kidney function. Anemia might begin to develop in the early stages of CKD, when someone has 20 to 50 percent of normal kidney function. Anemia tends to worsen as CKD progresses. Most people, who have total loss of kidney function, or kidney failure, have anemia. A person has kidney failure when he or she needs a kidney transplant or dialysis^[68,12].

The effect of hemodialysis on white blood cells:

With TWBCs count ranging between 3,8-10,8x10³mm³ as control count of normal individuals the study found that 83.9% of patients have noticeable increase in TWBCs count with maximum value reach to 2.400 cell /mm³ and minimum value 100 cell/mm³. About 14.1% of patients show decrease in TWBCs count range between 100-1600 cell/mm³, 2% have stable count .

CONCLUSIONS**Conclusions:**

1. 99,5% of patients have reduced platelets count after hemodialysis, while 0.5% have stable count of platelets.
2. 98,5% of patient have reduced hemoglobin concentration after hemodialysis.
3. 83,9% show increase in total white blood cells count after hemodialysis.
4. 14,1% have stable white blood cells count and only 2% their count decrease from pre-dialysis count.

REFERENCES

- [1] Ando, M.; Iwamoto, Y. and Suda, A. (2001). New insights into the thrombopoietic status of patients on dialysis through the evaluation of megakaryocytopoiesis in bone marrow and of endogenous thrombopoietin levels. *Blood*, 97: 915–921.
- [2] Anne, C. and Poinier, M. D.(2014). Internal Medicine Specialist Medical Reviewer Tushar J. Vachharajani, MD, FASN, FACP – Nephrology Current as of November 14, 2014
- [3] Arogundade, F. A. and Barsoum, R. S. (2008). CKD prevention in sub-Saharan Africa: a call for governmental, nongovernmental and community support. *Am. J. Kidney Dis.*, 51:515–523.
- [4] Aronoff, G. R.; Duff, D. R.; Sloan, R. S.; Brier, M. E.; Maurice, B.; Erickson, B. and Golper, T. A. (1990). The treatment of anemia with low-dose recombinant human erythropoietin. *Am. J. Nephrol.*, 10[Suppl 2] : 40– 43.
- [5] BarsoumRS, Rihan ZE, Ibrahim AS, Lebstein A.(1974). Long term intermittent haemodialysis in Egypt. *Bull World Health Organ.* 1974;51:647–654.
- [6] Beguin, Y.; Loo, M.; R'Zik, S.; Sautois, B.; Lejeune, F.; Rorive, G. and Fillet, G. (1994). Effect of recombinant human erythropoietin on platelets in patients with anemia of renal failure: Correlation of platelet count with erythropoietic activity and iron parameters. *Nippon Jinzo Gakkai Shi.*,36 :1288– 1295.
- [7] Berns, J. S.; Elzein, H.; Lynn, R. I.; Fishbane, S.; Meisels, I. S. and Deoreo, P. B. (2003). Hemoglobin variability in epoetin-treated hemodialysis patients. *Kidney Int.*,64 :1514– 1521.
- [8] Besarab A, Mc Crea JB.(1993). Anemia in ESRD, In: Nissenson MR, Fine RN, (eds). *Dialysis therapy*. 2nd ed. Philadelphia: Hanley Inc; 1993. pp 223- 225.
- [9] Besarob, A.; Bolton, W. K.; Browne, J. K.; Egrie, J. C.; Nissenson, A. R.; Okamoto, D. M.; Schwab, S. J. and Goodkin, D. A. (1998). The effects of normal as compared with low hematocrit values in patients with cardiac disease who are receiving hemodialysis and epoetin. *N. Engl. J. Med.*, 339 : 584– 590.
- [10] Bowers, N.; RN, BSN, M. P. H. (2015).Topic (What is plasma.) Available at: <https://www.urmc.rochester.edu/encyclopedia/.2015>
- Brandt, P.; Jespersen, J. and Sorensen, L. H. (1981). “Antithrombin-III and platelets in hemodialysis patients,” *Nephron*, 28(1): 1–3.
- [11] Brugnara, C. and Eckardt, K. U. (2011). Hematologic aspects of kidney disease. In: Taal MW, ed. *Brenner and Rector's The Kidney*. 9th ed. Philadelphia: Saunders; 2011: 2081–2120.

- [12] Bucciati, G.; Pogliani, E. and Miradoli, R. (1982). Reduction of plasma levels of beta-thromboglobulin and platelet factor 4 during hemodialysis: a possible role for a short acting inhibitor of platelet aggregation. *Clinical Nephrology*, 18(4): 204–208.
- [13] Cerner, M.; Wolters, K. (2015). *End-Stage Renal Disease Guide Causes, Symptoms and Treatment Options*. Copyright © 2000-2015 Drugs.com. All rights reserved.
- [14] Chen, J.; Gul, A. and Sarnak, M. J. (2006). Management of intradialytic hypertension: The ongoing challenge. *Semin. Dial.*, 19 :141– 145.
- [15] Coyne, D. (2006). Challenging the boundaries of anemia management: A balanced approach to i.v. iron and EPO therapy. *Kidney Int. Suppl.*, S1– S3.
- [16] Dahl, N. V.; Henry, D. H. and Coyne, D. W. (2008). Thrombosis with erythropoietic stimulating agents-does iron-deficient erythropoiesis play a role? *Semin. Dial.*, 21 : 210– 211.
- [17] David, H. and Yawn, M. D. (2015). *The plasma*. Encyclopedia Britannica, Inc.
- [18] Dodds A. Nicholls M. (1983). *Haematological aspects of renal disease*. Pub Med – index for medline. 1983; 11(4): 361-68.
- [19] Drews RE, Weinberger SE. (2000). Thrombocytopenic disorders in critically ill patients. *Am J Respir Crit Care Med* 2000; 162: 347–351.
- [20] Gafter, U.; Bessler, H. and Malachi, T. (1987). Platelet count and thrombopoietic activity in patients with chronic renal failure. *Nephron*, 45: 207–210.
- [21] Gaithersburg, M. D. (2008). FDA Panel Votes against Anemia Drug Restrictions. *Renal Business*, 2007. Available at: <http://www.renalbusiness.com/hotnews/79h14962958311.html>. Accessed September 5, 2008
- [22] George, J. N. and Aster, R. H. (2009). Drug-induced thrombocytopenia: pathogenesis, evaluation, and management. *Am. Soc. Hematol. Educ. Program*, 2009: 153–158.
- [23] Gold CH, Isaacson C, Levin J. (1982). The pathological basis of end stage renal disease in Blacks. *S Afr Med J*. 1982;20:263–265.
- [24] Hakim, R. M. and Schafer, A. I. (1985). Hemodialysis-associated platelet activation and thrombocytopenia. *American Journal of Medicine*, 78(4): 575–580.
- [25] Hakim, T. (2008). *The core of medical physiology (volume 2)*, 1st edition 2. Khartoum: Ruaa printing; 2008. ISBN:978-99942-884-6-5. Pg 73.
- [26] Hoffbrand, A. V.; Moss, P. A. H. and Pettit, J. E. (2006). *Essential haematology*, fifth edition, 2006, publish British library, pg 12, 14.
- [27] James, Cawker. (2015). Topic (hemodialysis machine, hemodialysis machine benefits). <http://dialysis-stlucia.com/06/05/201508/06/2015>
- [28] Janice, H. (2013). *Learn About Blood*, American Red Cross. Accessed 9 August 2013 (<http://www.redcrossblood.org/learn-about-blood>)
- [29] John, T.; Daugirdas, A. and Bernardo, A. (2012). Hemodialysis effect on platelet count and function and hemodialysis-associated thrombocytopenia. *Kidney International* (2012) 82, 147–157.
- [30] Kamyar, K.; Zadeh, A. and George, R. A. (2009). Hemoglobin Variability in Anemia of Chronic Kidney Disease. *JASN*, 20(3): 479-487.
- [31] Kasper, D.L.; Braunwald, E.; Fauci, A.S.; Hauser, S.L.; Longo, D.L. and Jameson, J.L. (editor) (2005). *Harrison's principles of internal medicine*. 16th edition, McGraw Hill Companies, Inc., USA, 2005, pp. 1430, 1653-1654, 2286-2295.
- [32] Kathryn, B.; Muir, A. and Clifford, D. P. (2012). Thrombocytopenia in the Setting of Hemodialysis Using Biocompatible Membranes Volume 2012 (2012), Article ID 358024, 4 pages doi:10.1155/2012/358024
- [33] Katz I. (2005). Kidney and kidney related chronic diseases in South Africa and chronic disease intervention program experiences. *Adv Chronic Kidney Dis*. 2005;12:14–21.
- [34] KDOQI (2007). Clinical practice guideline and clinical practice recommendations for anemia in chronic kidney disease—2007 update of hemoglobin target. *Am. J. Kidney Dis.*, 50 : 471– 530.
- [35] Kim, A. Z. (2015). *Urinary System Facts, Functions & Diseases*. www.livescience.com , junaury.15.2015.
- [36] Kiran, R. (2015). Army collage of nursing. Hemodialysis and peritoneal dialysis presented by <http://www.authorstream.com/Presentation/randhawakiran231122516>
- [37] Kooistra, M. P.; van Es, A.; Marx, J. J.; Hertsig, M. L. and Struyvenberg, A. (1994). Low-dose aspirin does not prevent thrombovascular accidents in low-risk haemodialysis patients during treatment with recombinant human erythropoietin. *Nephrol Dial. Transplant*, 9 :1115– 1120.
- [38] Krause, R. S. (2004). *Renal failure: chronic and dialysis complications*. www.emedicine.com. 2004.
- [39] Kumar, P. and Clark, M. (editors) (2005). *Clinical medicine*, 6th edition, Elsevier Saunders, 2005, pp. 605-689, 7998-800, 1137.

- [40] Lacson, E. J.; Ofsthun, N. and Lazarus, J. M. (2003). Effect of variability in anemia management on hemoglobin outcomes in ESRD. *Am. J. Kidney Dis.*, 41 :111– 124.
- [41] Lefebvre, P.; Vekeman, F.; Sarokhan, B.; Enny, C.; Provenzano, R. and Cremieux, P. Y. (2006). Relationship between hemoglobin level and quality of life in anemic patients with chronic kidney disease receiving epoetin alfa. *Curr. Med. Res. Opin.*, 22 : 1929– 1937.
- [42] Lindsay, R. M. and Clark, W. F. (1982). Platelet destruction in renal disease. *Semin. Thromb. Hemost.*, 8: 138– 155.
- [43] Lindsay, R. M.; Prentice, C. R.; Burton, J. A.; Ferguson, D. and Kennedy, A. C. (1973). “The role of the platelet-dialysis membrane interaction in thrombus formation and blood loss during hemodialysis. *Transactions American Society for Artificial Internal Organs*, 19:487–491.
- [44] Linthorst, G. E.; Folman, C. C. van Olden, R. W. (2002). Plasma thrombopoietin levels in patients with chronic renal failure. *Hematol. J.*, 3: 38–42.
- [45] Lucile, P. (2015). anatomy of the urinary system. Available at: www.Stanfordchildrens.Org/en/topic/,
- [46] Macdougall, I. C. (1998). Quality of life and anemia: the nephrology experience. *Semin. Oncol.*, 25: 39– 42.
- [47] McLigeyo SO, Kayima JK.(1993). Evolution of nephrology in East Africa in the last seventy years—studies and practice. *East Afr Med J.* 1993;70:362–368.
- [48] Mohammed Alhafiz, E.Elham G E (2009). Causes of end stage renal disease in Sudan: single -center experience. *Saudi Journal of Kidney Diseases And Transplantation*. Volume 22. Issue:2. Page 373-376. 2011.
- [49] Moreno, F.; Aracil, F. J.; Perez, R. and Valderrabano, F. (1996). Controlled study on the improvement of quality of life in elderly hemodialysis patients after correcting end-stage renal disease-related anemia with erythropoietin. *Am. J. Kidney Dis.*, 27: 548– 556.
- [50] Moreno, F.; Sanz-Guajardo, D.; Lopez-Gomez, J. M.; Jofre, R. and Valderrabano, F. (2000). Increasing the hematocrit has a beneficial effect on quality of life and is safe in selected hemodialysis patients. Spanish Cooperative Renal Patients Quality of Life Study Group of the Spanish Society of Nephrology. *J. Am. Soc. Nephrol.*, 11: 335– 342.
- [51] National kidney foundation (2002). Clinical practice guidelines for chronic kidney disease: evaluation, classification and stratification. *Am. J. Kidney Dis.*, 2002 (Suppl): S1-S66.
- [52] Ofsthun, N. J.; LaBrecque, J.; Keen, M.; Youngson, H. I.; Krishnan, M. and Lazarus, J. M. (2005). The association of mortality and hospitalization with hemoglobin (Hb) and missed dialysis treatments in stage 5 chronic kidney disease (CKD) patients with and without cardiac comorbidities. *Nephrol Dial Transplant*, 20:v268, 2005.
- [53] Olafiranye F, Kyaw W, Olafiranye O.(2011). Resolution of dialyzer membrane associated thrombocytopenia with use of cellulose triacetate membrane: a case report. *Case Rep. Med.*, 2011; 2011: 134295.
- [54] Parfrey, P. S.; Foley, R. N.; Wittreich, B. H.; Sullivan, D. J.; Zagari, M. J. and Frei, D. (2005). Double-blind comparison of full and partial anemia correction in incident hemodialysis patients without symptomatic heart disease. *J. Am. Soc. Nephrol.*, 16 : 2180– 2189.
- [55] Phrommintikul, A.; Haas, S. J.; Elsik, M. and Krum, H. (2007). Mortality and target haemoglobin concentrations in anaemic patients with chronic kidney disease treated with erythropoietin: A meta-analysis. *Lancet*, 369 : 381– 388.
- [56] Posadas MA, Hahn D, Schleuter W. (2011). Thrombocytopenia associated with dialysis treatments. *Hemodial. Int.*, 2011; 15: 416–423.
- [57] Post JB(2010). Thrombocytopenia associated with use of a biocompatible hemodialysis membrane: a case report. *Am J Kidney Dis* 2010; 55:e25–e28.
- [58] Regina, B. (2013). Learn About Blood, American Red Cross. Accessed 9 August 2013 (<http://www.redcrossblood.org/learn-about-blood>)
- [59] Remuzzi G, Rossi EC.(1995). Hematologic consequences of renal failure, In: Brenner BM, (eds). *The kidney*. 5th ed. Philadelphia: WB Saunders Co 1995; pp 2170-2185.
- [60] Rice L, Alfrey CP, Driscoll T, Whitley CE, Hachey DL, Suki W.(1999). Neocytolysis contributes to the anemia of renal disease. *Am J Kidney Dis* 1999;33:59-62.
- [61] Saraladevi, N.(2009). END-STAGE RENAL DISEASE IN SUB-SAHARAN AFRICA.Ethnicity& Disease, Volume 19, Spring 2009
- [62] Shahbazian, H.; Zafar, M. A. and Ghorbani, A. (2011). Oral nicotinamide reduces serum phosphorus, increases HDL, and induces thrombocytopenia in hemodialysis patients: a double-blind randomized clinical trial. *Nefrologia*, 31: 58–65.
- [63] Stewart, J. H. (1967). Platelet numbers and life span in acute and chronic renal failure. *Thromb. Diath. Haemorrh.*, 17: 532–542.

- [64] Strippoli GF, Craig JC, Manno C, Schena FP.(2004). Hemoglobin targets for the anemia of chronic kidney disease: A meta-analysis of randomized, controlled trials. *J Am Soc Nephrol.* 2004; 15(12):3154-65
- [65] Veriava Y, Du Toit E, Lawley CG, et al.(1990). Hypertension as a cause of end stage renal failure in South Africa. *J Hypertens.* 1990;4: 379–383.
- [66] Vicks SL, Gross ML, Schmitt GW.(1983). Massive hemorrhage due to hemodialysis-associated thrombocytopenia. *Am J Nephrol* 1983; 3: 30–33.
- [67] Weiss, G.; Goodnough, L. T. (2005). Anemia of chronic disease. *N. Engl. J. Med.*, 352 : 1011– 1023.
- [68] William, M. (2015). *Human anatomy and physiology*, scribed publications 2015, pages, 102-103.
- [69] World Health Organisation Report, 1998.
- [70] Yang RC, Lindsay RM.(2005). Dialyzer reactions in a patient switching from peritoneal dialysis to hemodialysis. *HemodialInt* 2005; 9: 120–126.
- [71] Ylikorkala, O.; Huttunen, K.; Jarvi, J. and Viinikka, L. (1982). Prostacyclin and thromboxane in chronic uremia: effect of hemodialysis. *Clinical Nephrology*, 18(2): 83–87.