Introduction

Hemodialysis (HD) is one of the three renal replacement therapies for Chronic Kidney Disease (CKD) (the other two being renal transplantion; peritoneal dialysis). It is a medical procedure that uses a special machine (a dialysis machine) to filter waste products from the blood and to restore normal constituents to it. This shuffling of multiple substances is accomplished by virtue of the differences in the rates of their diffusion through a semi-permeable membrane (a dialysis membrane). Although HD may be done for acute renal failure, it is more often employed for chronic renal disease. HD is frequently done to treat. End-Stage Renal Disease (ESRD). Under such circumstances, kidney dialysis is typically administered using a fixed schedule of three times per week ***(Graham, 2004).***

Patients with CKD have higher nutrient requirements, in addition, HD is a catabolic process, thus, nutritional status is an important predictor of the clinical outcome in chronic HD patients. Poor nutritional status and muscle wasting are common in patients with ESRD and are associated with increased hospitalization and death. Among different causes that are associated with altered nutritional status in ESRD, the HD procedure has been associated clearly with net whole-body protein and skeletal muscle protein loss. This catabolic process may be reversed acutely by administration of intradialytic oral nutrition (IDON) ***(Pupim et al., 2004).***

Although intradialytic parenteral nutrition (IDPN) has anabolic effects, its administration is costly, and patient eligibility for this type of nutrition support is widely restricted. In addition, the anabolic effects of IDPN seem to be limited to the period of administration, with no evidence of persistent anabolism once its infusion is shut off. IDON is a promising anabolic intervention in chronic HD patients because of its physiologic and affordable characteristics. Despite its potential benefits, only limited studies have evaluated the effects of IDON on protein metabolism in chronic HD patients ***(Pupim et al., 2006).***

Aim of The Work

1. Evaluating the nutritional status of the patients with CRF on regular HD.
2. Calculating the amount of daily proteins and caloric supply which are given to the patient at home and/or at our hospital, as well as, to evaluate whether these amounts are fulfilling the daily requirements of the patients or not.
3. Evaluating the role of IDON in patients with deranged nutritional status.

Chronic Kidney Disease

Chronic kidney disease (CKD) could be defined as a structural or functional abnormality of the kidney that leads to impaired kidney functions that persist for 3 months or more with or without decreased glomerular filtration rate (GFR). It is manifested by pathologic abnormalities noted on renal biopsy specimens or abnormalities revealed by blood, imaging or urine tests. Utilizing this definition, CKD is present if the GFR is less than 60 mL/min per 1.73m2. In addition, CKD is also present if the GFR is greater than or equal to 60 mL/min per i.73 m2 if other evidence of kidney damage exists ***(Perazella et al., 2003).***

Staging of CKD: CKD is classified into stages of severity according to the GFR and not to the levels of serum creatinine **(Table 1).**

Table (1): Stages of CKD:

Stage	Definition	Description	GFR (mL/min/1.73 m2)
1	Kidney damage with normal or increased GFR	Functional or structural damage of the kidney with GFR initially normal and decreases over time.	>90
2	Kidney damage with mild decrease in GFR	Patients may have hypertension and/or laboratory abnormalities indicating dysfunction in other organ systems, but most patients are asymptomatic. If the serum creatinine level is elevated, it may be more than borderline and of equivocal significance.	60-89
3	Moderate decrease in GFR	At this stage azotemia is present, defined by the accumulation of the end products of nitrogen metabolism in the blood and expressed by elevated serum creatinine and blood urea nitrogen (BUN) concentrations. Erythropoeitin production decreases and laboratory abnormalities. Kidney function may be reduced as much as 70%	30-59
4	Severe decrease in GFR	This is a severe stage of CKD, in which the worsening of azotemia, anemia and other laboratory abnormalities occurs. However, patients usually have mild symptoms	15-29
5	Kidney failure	This level of kidney dysfunction is " accompanied symptoms and laboratory abnormalities of several organ systems, which are collectively referred to as “uremia”. Initiation of kidney replacement therapy (dialysis or transplantation) is typically required for treatment of co-morbid conditions or complications of decreased GFR.	<15 or dialysis

- GFR= Glomerular Filtration Rate.
- ESRD=End Stage Renal Disease.

(Warady et al., 2006) , (Obrador et al., 2002).

The normal level of the GFR in particular reference to children varies with age, gender and body size and increases with maturation from infancy, approaching adult mean values at approximately 2 years of age (Table 2). In turn, GFR ranges that define the five CKD stages apply only to children 2 years of age and older ***(Warady et al., 2006***).

Table 2: Normal GFR in children and adolescents:

Age	**Mean GFR+/- SD (mL/min/1.73m2)**	**Normal S. creatinine (mg/dL)**
1 Week (males & females)	41+/-15	0.6-1.2
2-8 Weeks (males & females)	66+/-25	0.3-1
>8 Weeks (males &females)	96+/-22	0.2-0.4
2-12 Years (males & females)	133+/-27	0.3-0.7
13-21 Years (males)	140+/-30	0.5-1.0
13-21 Years (females)	126+/-22	0.5-1.0

(Warady et al., 2006), (Nicholson and Pesce, 2004)

Pathogenesis of CRF:

In CRF renal injury may develop despite the removal of the original disease and although the precise mechanisms of the progression of the disease are unclear, putative factors include:

- ***Hyperfilteration injury***: structural and functional hypertrophy of the remaining nephrons is a compensatory mechanism that temporarily preserves the total renal function but it results in increased glomerular blood flow and hyperfilteration that cause progressive damage by a

direct effect by the elevated hydrostatic pressure on the integrity of the capillary wall.

- ***Proteinuria:*** Proteins that traverse the glomerular capillary wall may exert a toxic effect and recruit monocytes and macrophages enhancing the process of glomerular sclerosis and tubulointerstitial fibrosis.

- ***Uncontrolled Hypertension:*** May exacerbate disease progression by causing arteriolar nephrosclerosis as well as by increasing hyperfilteration.

- ***Hyperphosphatemia:*** Leading to more Calcium-Phosphate deposition in the renal interstitium and blood vessels.

- ***Hyperlipidemia:*** A common condition in CRF patients which affects glomerular function through oxidant-mediated injury ***(Vogt et al., 2004).***

Etiology of CRF:

The most common causes of ESRD in pediatric patients were documented are obstructive uropathies, aplasia/ hypoplasia/ dysplasia and reflux nephropathy whereas, structural causes predominates in elder children ***(Warady et al., 2006).***

Hereditary renal disorders, such as polycystic kidney disease, primary hyperoxaluria and congenital nephrotic syndrome, also contribute with high prevalence especially where consanguinity is common (***Warady et al., 2006).***

The chief causes of ESRD in pediatric patients include the following:

- Obstructive uropathy.
- Hypoplastic or dysplastic kidneys
- Reflux nephropathy.
- Focal segmental glomerulosclerosis as a variant of childhood nephritic syndrome.
- Polycystic kidney disease, both autosomal-dominant and autosomal- recessive variants ***(Gulati, 2009)***

Etiology of ESRD in patients on regular HD in the Pediatric Dialysis Unit (PDU) in Ain Shams University in the year 2001 are mentioned in (Table 3), Chronic glomerulo-nephritis is the most common cause ***(Farid, 2001).***

Table (3): Etiology of ESRD in patients on Regular HD in Ain Shams University Pediatric Dialysis Unit in the year 2001.

Pathology	Percent
Chronic glomerulonephritis	52%
Obstructive uropathy	16%
Systemic lupus erythomatosis	8%
Hypoplastic kidney	8%
Renal stones	3%
Unknown	13%

(Farid, 2001)

Diagnosis of a case with CRF:

1. Clinical picture:

CRF usually produces symptoms when renal function - which is measured as the glomerular filtration rate (GFR)- falls below 30 milliliters per minute (< 30 mL/min).This is approximately 30% of the normal values and that is when signs of uremia (high blood level of protein by-products, such as urea) may become noticeable. When the GFR falls below 15 mL/min most people become increasingly symptomatic ***(Swierzweski, 2007).***

The clinical picture of CRF is quite and varied and dependant on the underlying renal disease. Children with CRF from Glomerulonephritis and Nephrotic syndrome may present with: edema, hypertension, proteinuria and hematuria. Infants and children with congenital disorders as Dysplasia or obstructive uropathy may present with: failure to thrive, dehydration or recurrent urinary tract infections. Children with familial nephronephthisis may have a very subtle presentation with non specific complaints such as headache, fatigue, lethargy, anorexia, polyuria, polydepsia and growth failure over a number of years ***(Vogt et al., 2004).***

Uremic symptoms can affect every organ system, most noticeably the following:

1. Neurological system–cognitive impairment, personality change, asterixis (motor disturbance that affects groups of muscles), seizures (rare).

2. Gastrointestinal system–nausea, vomiting, food distaste (often described as bland, metallic, "like cardboard").

3. Blood-forming system–anemia due to erythropoetin deficiency, easy bruising and bleeding due to abnormal platelet function.

4. Pulmonary system–fluid in the lungs, with breathing difficulties.

5. Cardiovascular system –chest pain due to inflammation of the sac surrounding the heart (pericarditis) and pericardial effusion (fluid accumulation around the heart).

6. Skin–generalized itching and earthy look. ***(Swierzweski, 2007).***

Table (4): The physical examination and the pathophysiology of CRF:

Manifestation	Mechanism
Accumulation of waste products	Decreased GFR
Acidosis	Impaired bicarbonate reabsorption Excessive renin production
Sodium retention	Oliguria Solute dieresis
Sodium wasting	Tubular damage
Urinary concentrating defect	Solute dieresis Decreased GFR
Hyperkalemia	Metabolic acidosis Hyporeninemic hypoaldosteronism Excessive potassium intake
Renal Osteodystrophy	Hyperphosphatemia, Hypocalcemia Inadequate oral intake Secondary hyperparathyroidism
Growth retardation	Renal osteodystrophy Anemia Metabolic acidosis Growth hormone resistance
Anemia	Defective erythropoietin synthesis Iron, Folate, Vitamin B12 defeciency Decreased erythrocyte survival.
Bleeding tendency	Defective platelet function
Infection	Defective granulocyte function Impaired cellular immunity function Indwelling dialysis catheters.
Neurological manifestations	Uremic factors, Hypertension Aluminum toxicity.
Gastrointestinal manifestations	Gastroesophygeal reflux Decreased intestinal motility.
Hypertension	Volume overload Excessive renin production.
Hyperlipidemia	Decreased plasma lipoprotein lipase activity.
Pericarditis/cardiomyopathy	Uremic factor, Hypertension Fluid overload.
Glucose intolerance	Tissue insulin resistance.

(Vogt et al., 2004).

2. Laboratory findings:

The diagnosis of renal failure is made by documenting elevations of the BUN and serum creatinine concentrations. Further evaluation is needed to differentiate between acute and CRF. Evidence of previously elevated BUN and creatinine, abnormal prior urinalyses, and stable but abnormal serum creatinine on successive days is most consistent with a chronic process ***(Kamis., 2008).***

Anemia, metabolic acidosis, hyperphosphatemia , hypocalcemia, and hyperkalemia are also evident ***(Kamis., 2008).***

The urinalysis shows proteinuria, isosthenuria, erythrocytes, and leukocytes depending on the eitiology. The urinary sediment can show broad waxy casts as a result of dilated, hypertrophic nephrons. Urine specific gravity becomes fixed at 1010 ***(Kowalak, 2001).***

3. Imaging:

Pelvic-abdominal ultrasound, ascending cysto-urethrogram (ACUG), C.T. Scan and nuclear imaging are often helpful to determine the suspected cause of CRF, Ultrasound may reveal increased echogenicity, loss of corticomedullary differentiation and small shrunken kidneys ***(Marcdante, 2006).***

Radiologic evidence of renal Osteodystrophy is another helpful finding, since x-ray changes of secondary

hyperparathyroidism do not appear unless parathyroid levels have been elevated for at least 1 year. Evidence of subperiosteal reabsorption along the radial sides of the digital bones of the hand confirms hyperparathyroidism ***(Kamis, 2008).***

4. Renal biopsy:

May be indicated if the presentation is atypical, to assess the severity of systemic disease involvement, guide therapy or establish a prognosis. Usually done percutaneously, light microscope should be augmented by special studies such as Immunofluorescence and electron microscope ***(Marcdante: 2006).***

Management of CRF:

1. Identifying the underlying renal disease.
2. Prevent further renal damage.
3. Control the reversible factors which are worsening the renal functions as:
    - Hypertension.
    - Reduced renal perfusion: Renal artery stenosis, Hypotension, Sodium and water depletion, Poor cardiac function.
    - Anemia.
    - Gastritis.
4. Attempt to limit the adverse effects of the loss of the renal function.
5. Institute renal replacement therapy (Dialysis or transplantation).

Hemodialysis

What is HD?

Dialysis is a procedure whereby the solute composition of a solution , A, is altered by exposing solution A to a second solution, B, through a semi permeable membrane which is a sheet perforated by pores through which water molecules and low molecular weight solutes can pass through the pores of the membrane and intermingle together but larger solutes such as proteins can not pass through the semi permeable barrier and the quantities of high molecular weight solutes on either sides of the membrane will remain unchanged. Solutes that pass through the membrane pores are transported by Diffusion and Ultra filtration ***(Daugirdas, 2001).***

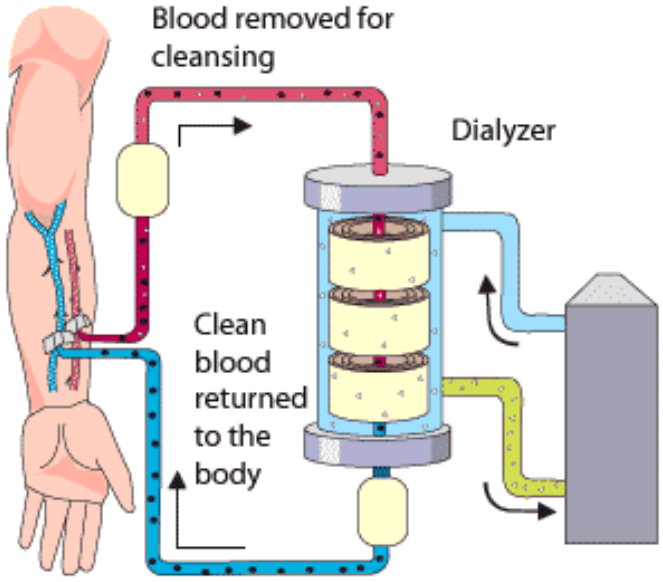

Fig. (1): Circuit of blood in HD ***(Gralapp, 2008)***.

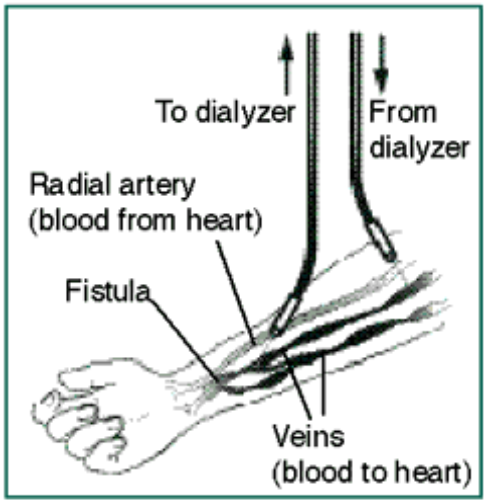

Fig. (2): Arterio-Venous shunt ***(Gralapp, 2008)***.

When to start renal replacement therapy (RRT)?

Determination of the optimal time to start RRT for children is seldom straight forward except in extreme circumstances such as bilateral nephrectomies. Both clinical and laboratory parameters contribute to the decision to start renal replacement therapy. According to the National Kidney Foundation Kidney Disease Outcomes Quality Initiative (NKF K-DOQI) guidelines, initiation of dialysis should be considered when the GFR falls below 14ml/min/1.73m2 body surface area (BSA) and is strongly recommended when the GFR is <8ml/min/1.73m2 ***(Schroder et al., 2008)***.

HD is the main RRT considered after renal transplantation and its main role is very evident in the treatment of ESRD. There are many complications that may prompt initiation of HD other than measuring the GFR as: