Handbook for Dialysis Nurses

2nd Edition

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Handbook For DIALYSIS NURSES

By

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Previous to this appointment he has been the Head of the Department of Nephrology at the Armed Forces Hospital in Riyadh since for 20 years and was the Director of Postgraduate and Academic Affairs for 10 years until 1999.

He obtained a BSc Degree with Honours in Biochemistry from London University in 1970 and qualified as a director with MBBS with Honours from the same university in 1973. He become a Member of the Royal College of Physicians (UK) in 1976 and fellow of the Royal Colleges of London and Edinburgh (FRCP) in 1988 respectively. He obtained the academic degree of MD from the University of London in 1980. He has obtained Masters in Business Administration (MBA in Management Consultancy) from Sheffield Hallam University (UK) in 2001.

Dr Al-Khader has written over 172 publications in Medical Journals on kidney transplantation, dialysis and kidney diseases. He is the Editor-in-Chief of the Saudi Journal of Kidney Diseases and Transplantation and is on the Editorial Boards of a number of Journals including Nephrology Dialysis Transplantation. He is also on the Council of the International Society of Nephrology and is the Vice Chairman of the Middle East subcommittee of the Commission of Global Advancement of Nephrology and is the Chairman of

Global Advancement of Nephrology and is the Chairman of the Scientific Office of the Arab Society of Nephrology and Renal Transplantation. He is also the Chairman of the National committee of kidney Transplantation in Saudi Arabia and is the Director of Planning and Research at the Saudi Center for Organ Transplantation.

He has written 4 other books Handbook for Residents in Nephrology and Conversations with a Nephrologist". The latter written in Arabic and English and Clinico-Pathological corrections-synopsis for the Residents in Nephrology"

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DEDICATIONS

I dedicate this book to my father Ahmed Al-Khader Al-Sayyari and my mother Fatima Al-Merhali and my children Ahmad, Esam and Salah. I cannot thank them enough for all the important and great things that they taught me and for all the joy that they filled my heart with.

Abdulla Ahmed Al-Khader

PREFACE

This is the Second Edition of Handbook for Dialysis Nurses". The first Edition proved to be very popular to dialysis nurses throughout the Arab World in this second Edition we expand the chapter on CAPD bringing up-to-date and add two new chapters related to renal transplantation. We have found that more dialysis nurses in our region get involved with kidney transplantation and many move to a carrier in transplant coordination, One of these chapters is on Social Factors impacting on organ transplantation in Islamic countries and one is a manual for transplant coordination". This later chapter contains some case scenarios for the coordinator.

This book is based on the lectures and tutorials we give to our trainees in the Dialysis Diploma Course" and to our dialysis nurses re-fresher courses. It also reflects in some parts the practice in our unit.

We tried to cover both theoretical and practical aspects of the subject, although it cannot replace practical on the job experience.

We feel that knowledge of causation of disease, renal function and use of laboratory investigations are essential components for a competent dialysis nurse and thereof we set aside chapters on these topics.

Increasingly the dialysis nurses are called on to be part of the management team of transplant patients and patients in the ICU. Often they also rotate through the peritoneal dialysis service and we tried to attend to these topics in this book.

The dialysis nurse should be aware of the ethical and social issues of looking after the patients and this is discussed briefly in one of the chapters.

This book is aimed mainly at dialysis nurses but may be found useful by medical students and doctors looking after dialysis patients,

Abdulla A l- Khader

Mohamed Al - Jondeby

Table of Contents

Chapters

Chapter One

Basic Review of Renal Function

Basic Review of Renal Function

The kidney has three basic functions –regulatory, excretory and productive (of hormones), Its anatomy is perfectly suited to its functions.

For the kidney to perform its functions the blood has to reach it in large amounts. Hence the blood flow to the kidneys is about a liter per minute- a fifth of the cardiac output. This high flow rate is necessary to allow the kidneys to fine tune its regulatory and excretory functions.

The blood reaches the kidney via the renal artery that branches out of the aorta at the level of the kidney. Blood leaves the kidney via the renal vein that joins the inferior vena cava.

The kidneys are situated at the back of the abdomen. Each kidney weighs 160 gms and is 10-15 cms in length and is shaped like a bean with the hilum facing inwards. At the hilum, the renal artery and vein as well as the ureter emerge. The renal artery splits into segmental arteries that split further into interlobar arteries and further still into arcuate arteries. These further become the afferent arterioles. The afferent arteries are very important vessels because they form a tuft of capillaries that form the glomerulus. It is at the glomerulus that the blood is filtered. These capillaries are quite thin walled and their wall is made up of three layers- the endothelial cells layer on the inside, the glomerular basement membrane (GBM) in the middle and the epithelial cell layer on the outside. It is through these layers that the blood is filtered as the first process of eventually forming the urine.

The capillary tuft is formed as 4 to 6 capillary loops supported by specialized cells and matrix called the mesangial cells and mesangial matrix, The capillary tuft (the glomerulus) invaginates the first part of the tubule

that is a long tube that will be described later. The glomerulus with its own tubule forms a single functional unit called "nephron". There are one million nephrons in each kidney and the total length of all the tubules is estimated to be 110 kilometers. The first part of the tubule into which the capillary tuft is invaginated is called the Bowman's capsule or capillary space. The pressure within this space is lower than the pressure inside the capillary itself. This pressure difference or gradient (the transmembranous pressure) facilitates the filtration process. The intracapillary pressure is maintained by the systemic blood pressure (which is transmitted all the way to the afferent arteriole) and also by the fact that the efferent arteriole is more constircted than the afferent arteriole. The joining together of the capillaries of the glomerulus forms the efferent arteriole that emerges out of the glomerulus. See Fig 1). Further down, the efferent arteriole forms the pen-tubular capillaries that receive the water and other substances reabsorbed by the tubules.

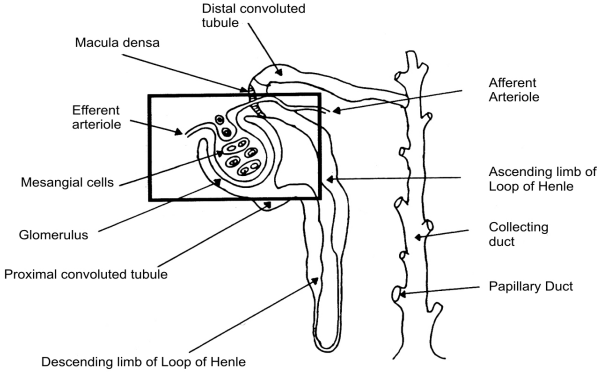

Fig 1

The Glomerulus

Once the ultrafiltnate is formed in the capillary space, it passes through the length of the tubule. The first part of the tubule is the proximal tubule". Then the tubule extends down from the outer part of the kidney (cortex) deep towards the hilum of the kidney (medulla)(see Fig 2). This part is the "descending tubule", It then makes a hairpin curve the loop of Henle) and travels outwards back to the cortex the ascending tubule). Once in the cortex the "distal tubule" begins and is followed by a down turn again back to the medulia. This part is called the "collecting duct"A number of these ducts join together to form 5 to 6 "calyces" that drain the urine to the "renal pelvis" and from there to the ureter down to the bladder. At the junction of the distal tubule and the efferent arteriole a specialized group of cells called "the juxtraglomerular apparatus" could be found. These cells produce renin (see below).

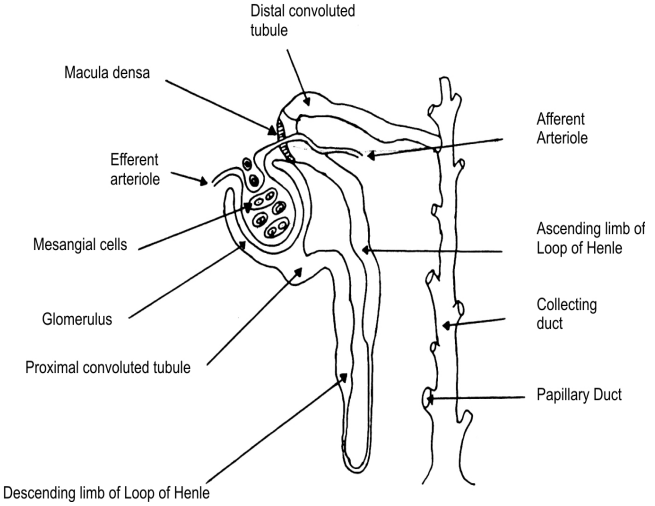

Fig 2

The Nephron

The cortex, thus, contains the glomeruli and the proximal and distal tubules. The medulla contains the rest of the tubules, collecting ducts and calyces.

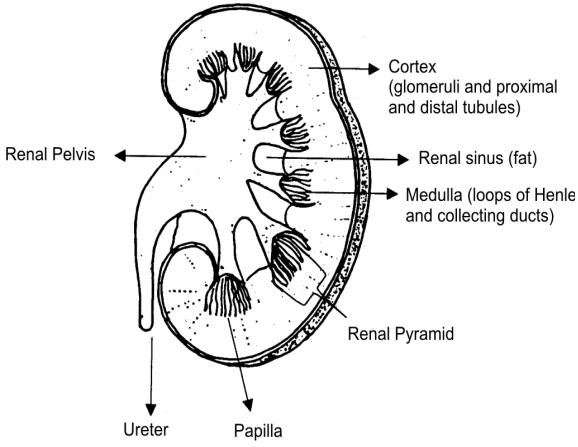

Fig 3

Cross Section of The Kidney

The filtration occurs through the capillary basement membrane and the filtrate formed passes to the capillary space and into the proximal tubules. The amount filtered by the healthy kidneys is 200 liters per day (this is equivalent to 4 times the body water which is 50 liters. In other word the plasma is filtered four times every day). It passes throughout the tubular length and by the time it reaches the ureter, only 2 liters per day are passed out as urine. In other words 99% of the fluid filtered by the glomerulus is reabsorbed back to the blood stream. This re-absorption process involves water, electrolytes, bicarbonate, amino acids, uric acid and phosphate. This is fine tuned to a remarkable degree to keep the internal body environment within narrow physiological limits. At the same time waste products are excreted.

The glomerular basement membrane has pores and slits in it as well as electro-physical properties allowing only small molecules to pass through. Proteins of albumin size or larger as well as cells are not allowed to pass by the healthy glomerulus. Diseased glomeruli, on the other hand, allow them to pass through. Finding proteins and cells in the urine is therefore a hallmark of glomerular disease (glomerulonephritis). (See chapter 3)

Glucose and amino acids are totally re-absorbed by the tubules. 99% to 99.5% of sodium is re-absorbed and this is finely regulated such that the body volume and sodium content is kept remarkably constant. Certain hormones as will be described below influence the degree of tubular re-absorption of water and sodium.

One of the major functions of the kidney is to keep the extra cellular fluid (ECF) volume and composition constant. ECF is the fluid outside the cells, Part of the ECF is within the blood vessels (intravascular fluid) and the rest is outside the blood vessels "bathing" the cells. ECF allows the transport of food, energy and oxygen to the cells and waste products and carbon dioxide out of the cells. It also provides the right physio-chemical environment for the cells to function properly. In a 70-kilogram man, water constitutes 60% i.e. 42 liters. Out of this, 15 liters are in the ECF.

To keep the volume and composition of ECF normal, the kidney has to excrete the waste products delivered to the ECF by the metabolism occurring inside the cells and to maintain its right volume and composition and therefore conserve/excrete the right amount of water and sodium according to the intake. The electrolytes and acid base balance has also to be kept within normal limits. The kidneys are able to do this over a wide environmental variation of temperature, food consumption and water and salt intakes. Thus the kidney is capable to conserve things when they are in short supply and excrete them when they are in excess.

Water regulation is accomplished by a combined mechanism of (a) the increased osmolality (concentration) generated by the loop of Henle (and the veins that are adjacent to it- the vasa recta) and (b) by the effect of the Anti-diuretic Hormone (ADH) acting on the collecting ducts.

As mentioned earlier the tubule dips deep into the medulla forming the loop of Henle. By the movement of chloride and sodium to the interstitium and the vasa recta acting as counter current multiplier, a concentrated environment is created in the medulla. This increases down the depth of the medulla. This allows the re-absorption of water back to the circulation and concentrated urine is thus produced. This is done through the concentration gradient between the fluid in the tubule and in the surrounding interstitiuim. However this only happens in the presence of ADH that acts on the tubules to facilitate the movement of water.

Now when a person is dry, the osmolality of the blood rises. This acts as a powerful stimulus for the production and secretion of ADH from the pituitary gland (at the base of the brain) thus allowing for more water to be re-absorbed by the kidney. This lowers the osmolality of the blood and shuts up the production of ADH. The opposite occurs when there is excess water drunk. Here the plasma osmolality drops, reducing the amount of ADH produced and thus reducing the amount of water reabsorbed and increasing the amount of urine passed.

Sodium regulation on the other hand depends on the stretch sensors at the afferent arteriole wall and on the amount of sodium filtered in the tubules. The stretch is increased by hyper-volaemia that induces more sodium excretion so does the more sodium filtered. Sodium regulation also depends on a hormone called aldestrone (produced by the adrenal gland situated just above the kidney). Aldestrone production is induced when there is a need to conserve sodium as when there is a state of dehydration. Aldestrone causes increased re-absorption of sodium by the

distal tubule. Another factor involved in sodium regulation is the antinatuertic peptide (ANP). The heart produces this hormone and it reduces sodium excretion.

The kidney is also essential in keeping the acid base balance of the blood within narrow acceptable range. It does so by excreting the excess acid (H ion) produced and also by combining hydrogen ions to ammonia forming ammonium in the distal tubule. Ammonium is excreted in the urine.

The kidney also functions as hormone producer. The most important ones are erythropoietin, active vitamin D and renin. These will be described in other chapters in this book (chapters 2,5 & 15).

Chapter Two

Diagnosis and Investigations of Chronic Renal Failure

Diagnosis and Investigations of Chronic Renal Failure

Patients with chronic renal failure often do not have symptoms Until late in their disease (when the function of the kidneys is only 10% of normal). When they eventually develop symptoms, these are nonspecific. Therefore we depend entirely on laboratory and radiological investigations to diagnose chronic renal failure. It may be that the patient is discovered to have renal failure on routine testing of his blood as in routine check up for insurance, employment on prior to surgery. It is possible that the patient is discovered to have it because he complains of tiredness or nausea both of which are nonspecific and occur in many disease processes or even for psychological reasons. Indeed it is not always possible to tell even an apparently healthy person if he does not have some degree of renal impairment without doing the appropriate tests first.

The basic investigations are simple and quick. They consist of measuring plasma urea and creatinine.

Urea is the end product of the breakdown of nitrogenous products such as proteins. Its normal level in the blood is 2.3-to 7.5 mmol/ liter. Its major excretory route is through the kidneys and therefore it rises when there is renal failure and in general the higher it is the more advanced is the renal failure. However it could rise due to other reasons other than renal failure as in the state of dehydration. In situations where there is increase intake of proteins it could also rise although normally not to very high levels. Such situations include high meat content in the diet or bleeding into the gut (the blood acts like high protein intake). Another

situation in which urea level could rise is when there is increase breakdown of body proteins (hyper catabolic state) as occurs in severe infections. Conversely urea level might not rise appropriately to the degree of renal failure in patients with low protein intake (as in malnutrition or in vegetarians) or in patients with severe liver disease since urea is made in the liver.

Despite all these provisos, measurement of blood urea is one of the cornerstones for the diagnosis and follow up of patients with renal failure.

Creatinine is produced from the muscles and is normally excreted largely by the kidneys and so it rises in renal failure. It correlates better with the degree of renal failure than does the urea level. The normal range for its level in blood is 50-115 umol/ liter.

It should be noted that even a small rise in creatinine level might indicate a significant drop in renal function. For example a level of 140 umol/L may indicate a 30% drop in renal function. Moreover a level of only 100 umol/L in a small sized person (with little muscle bulk) or a child may reflect a significant renal impairment. Conversely large muscular patients may normally have raised creatinine levels without any renal impairment. Therefore a more accurate method of measuring renal function is the creatinine clearance. In this test urine is collected over 24 hours and its volume and creatinine are measured as well as the blood creatinine. Creatinine clearance (CrCI) could be calculated by dividing the amount of creatinine excreted (umol) by the plasma concentration of creatinine (umol/ml). Normal range is 90-120 mls per minute. The major drawback with this method is the common inaccuracies of urine collection that could result in significant over or under estimate of the CrCI. Moreover, creatinine is excreted by the tubules and therefore not all the creatinine measured in the urine truly reflects the glomerular filtration rate (GFR) i.e. the CrCI overestimates the GFR For accurate estimation of the renal function the

glomerular filtration rate (GFR) may be measured. One commonly used method is by chromium labeled EDTA, This is injected intravenously and blood samples are obtained from the patient hourly for 6 hours. The isotopic activity in these samples is measured and the rate of its disappearance is used to calculate the GFR since the excretion of this isotope occurs through the kidney glomerulus only.

If the urea and creatinine are raised and the CrCI is reduced, this would indicate that the patient suffers from renal failure. The questions to ask then are:

I. Are we dealing with established chronic renal impairment?

2. Is there a reversible reason to this renal impairment?

3. Is it time to establish vascular access?

   Is the patient more suitable for hemodialysis or peritoneal dialysis (PD)? (See chapter 19))

4. Does he have any symptoms related to his renal impairment? Are any other organ systems affected? (See chapter 5)

5. What could we do to slow the progression of his renal disease?

6. What is the cause of his renal failure? (See chapter 3)

7. You need also to attend to the following very common aspects of the disease:

   I) Avoid any cannulation of veins that could be used for future vascular access (see chapter 10))

   ii) We need to attend to his BR that is often raised (See section on Chronic Complications of Dialysis')

   iii) We attend to his hemoglobin since he is likely to be anemic.

   iv) He is likely to have renal osteodystrophy (See section on Chronic Complications of Dialysis)

   v) Vaccination against Hepatitis B virus

8. Is time for Dialysis at hand? (See section on Indications for Dialysis)
9. Should a renal biopsy be done?
10. Is he a suitable candidate for transplantation? (See chapter 20)

1) Are we dealing with established chronic renal impairment?

This is an important question to answer because if the answer is yes, this implies future progression and warrants implementation of measures that goes with this prognosis (future dialysis access, transplantation, measures to slow down progress etc. (see below), There are a number of ways to ascertain the chronicity of the renal impairment,

a] History of the Disease

You may be able to find this from the records showing evidence of progressively rising of plasma creatinine on falling creatinine clearance. Long standing diabetes mellitus or hypertension or a known history of generalized edema in childhood may indicate chronicity. In a women who develops secondary infertility on early abortion or severe hypertension leading to early delivery in her recent pregnancies the indications are that she has chronic renal impairment.

b) Size of the kidney

It is important in establishing chronicity to find out the size of the kidneys. The best, quickest way is to do an U/S of the kidneys. In the majority of cases of chronicity if will show small sized kidneys that are echogenic. The 2 major exceptions in which the kidney size does not decrease despite chronicity are diabetes mellitus (common) and amyloidosis (uncommon). Incidentally, doing U/S will also help exclude obstruction and may give a

clue about etiology of the renal disease (see below). Intravenous urogram (I.V.U) is not used for this purpose except in a few cases because of the possible (but rare) allergy, because you need big doses in renal impairment and because in some conditions such as diabetes mellitus and dehydration it may cause contrast-associated deterioration in renal function (see section on 'Acute Renal Failure).

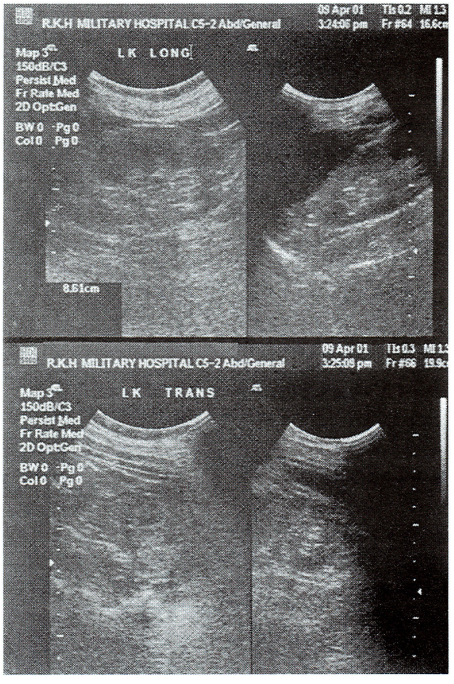

Fig 4

U/S showing small echogenic kidneys

Another method that is very rarely used now, to detect renal size, is tomography of the renal area.

c) Renal Biopsy

The resident should check if the patient has previously had renal biopsy. If it was done in another institution he should make every attempt to get the slides for discussion in the weekly histology meeting. The biopsy is the most important clue for chronicity. The features in the biopsy that will indicate chronicity are glomerulosclerosis. The glomerulosclerosis could be assessed in terms of the percentage of glomeruli that are sclerosed and whether there is partial or global sclerosis in a given glomerulus. Of course, for this assessment to be of use sufficient glomeruli (over 6) should be available for examination in the biopsy. A more important indicator of chronicity is the degree of interstitial fibrosis. This could be seen easily on biopsy especially using Masson's trichrome stain. Here the tubules - that are normally back-to-back - are unduly separated. The degree of fibrosis not only indicates chronicity but also predict progression of the disease.

d) Hemoglobin Level

The vast majority of patients with moderate renal impairment are anemic. This is another indicator of chronicity. But before one determines that the low Hemoglobin is due to the renal failure one should check that there is no other cause for the anemia. The anemia of renal failure is usually normocytic and normochnomic and the cause is multifactorial blood loss through the gut, malnutrition, bone marrow depression due to the uremia and hyperparathyroidsm but the main reason is Erythropoietin deficiency. You need to make sure that there is no B12 and folate deficiency and no source of bleeding.

e) Renal Osteodystrophy

(See section on Chronic Complications of Dialysis))

Finally, another clue one should look for to support chronicity is evidence of renal osteodystrophy. Even when renal impairment is only mild

(creatinine clearance of 70ml/min) a series of changes in bone, calcium and phosphate metabolism occur that lead to osteodystrophy, Changes in the bone take time to be seen radiologically and so when they are present this usually indicates chronicity. (Although these changes could be seen more directly and much earlier by bone biopsy, this is not usually done).

Doing skeletal X-ray could reveal the bone changes but the best places to detect them are the skull X-ray (for pepper pot appearance of hyperparathyroidsm) pelvis and upper part of the femur (for Looser's zones of osteomalacia.) vertebrae (for osteoporosis or osteosclerosis) and hand X-ray (for subperosteal erosions of hyperparathyroidsm) (see chapter 15)

You could see from the above that simple, non-invasive and quick tests could help you determine chronicity namely: U/S of the kidneys, ordinary bone X-rays and hemoglobin measurement as well as properly taken history and study of the patients file.

2) Are there any reversible components to the patient's renal failure?

Even in known cases of established chronicity one should always ask oneself about reversible factors. The most important and commonest factors (most are easy to detect and correct) are:

a) Volume depletion

Volume depletion in patients with pre-existing renal disease should be avoided as it could lead to further deterioration in renal function.

Evidence of volume depletion should be looked for such as history of

bleeding, vomiting, diarrhea, diuretic use, fasting, 3rd space and polyuria as happens with high blood sugar.

Careful look at the patient's records may yield very pertinent and important information. Compared to previous data in the file has the patients weight decreased? Has his BP dropped? Has his hemoglobin and plasma protein concentration risen?

Assessment of skin turgidity is useful but be aware that it could be misleading in elderly patients (who have lack of subcutaneous connective tissue) and in young adults in whom this sign could appear only very late into dehydration. Very useful signs of volume depletion is the jugular vein that couldn't be seen and fills very slowly (on obstructing it by puffing a finger over the vein) and empties rapidly on removing the finger.

But, perhaps, the most reliable signs are related to the BP. Usually it is low and characteristically there is a drop on BP on standing (postural hypotension - usually associated with the patient complaining of dizziness) and the pulse pressure (this is the difference between the systolic and diastolic pressures) is narrow. These signs are particularly useful when the patient's usual BP characteristics are known.

In a patient with volume depletion, on the background of renal impairment, the rise in the urea is much more than the rise in creatinine and this plus the BP changes described above are the most important clues for volume depletion.

b) Nephrotoxic Agents (See chapter 4)

Careful history of drug or herbal intake is very important. Many patients do not consider on counter drugs as drugs and you should query them carefully. It takes only one or two tablets to have nephrotoxic effects.

Vast number of drugs could cause nephrotoxicity but to variable