WORLD JOURNAL OF PHARMACY AND PHARMACEUTICAL SCIENCES
SJIF Impact Factor 7.421
Volume 7, Issue 12, 494-510 Review Article ISSN 2278 – 4357

NEPHRITIS DISEASE

Ashika L. Madhavi*, Mangala A. Masne, Janhavi V. Lavande, Suchita Gokhale

India.

Article Received on
01 Oct. 2018,

Revised on 22 Oct. 2018,
Accepted on 12 Nov. 2018,

DOI: 10.20959/wjpps201812-12761

***Corresponding Author**
Ashika L. Madhavi
India.

ABSTRACT

Nephritis is the inflammation of the kidney and may involve the glomeruli, tubules or interstitial tissue surrounding the glomeruli and tubules. Nephritis classified in two types Acute nephritis and chronic nephritis. Acute nephritis again classified into Pyelonephritis, Glomerulonephritis, Interstitial nephritis. Also chronic nephritis classified into Chronic glomerulonephritis, IgA nephropathy, Lupus nephritis. This study of Nephritis include information regarding causes, who is at risk factor, symptoms, diagnosis, treatment, medication,prevention, home care and diet. The role of pharmacists is important in improving the medication and recommending better treatment. Additionally pharmacist intervention was defined as any action with the aim of modifying the process of use of drugs; either in patient's activities or medical or health care practitioner's activities.

KEYWORDS: Nephritis, Acute nephritis, Chronic nephritis.

INTRODUCTION

Nephritis is the inflammation of the kidney and may involve the glomeruli, tubules or interstitial tissue surrounding the glomeruli and tubules.[1]

ALL YOU NEED TO KNOW ABOUT NEPHRITIS

To understand kidney problems such as nephritis, its helpful to start with some background on what the kidneys are, and what they do…

KIDNEY

The kidneys are two bean-shaped, fist sized organ just under the ribs on the left and right side of the spine. They remove impurities and extra water from the blood, filtering 120-150 quarts

of blood a day, according to the "National institute of Diabetes and Digestive and Kidney Disease".

Each kidney consists of thousands of structures called nephrons, where the actual blood filtering takes place. In the nephron a two step cleaning process separates what the body needs to keep from what it can get rid off.

A filter called the glomerulus catches the blood cells and proteins, sending water and waste to a second filter called a tubule. The tubules capture minerals. After that what remains leaves the body as urine.

Nephritis describes a condition in which the kidney tubules are nearly inflamed along with the near-by tissues which can lead to kidney damage.

When kidneys are damaged, they don't work properly. Waste builds up and cause serious health problems. If the condition is severe enough, or lasts long enough, it can result in kidney failure.[2]

TYPES OF NEPHRITIS

There are several different types of nephritis:

1. Acute Nephritis

a) Pyelonephritis
b) Glomerulonephritis
c) Interstitial nephritis.

2. Chronic Nephritis

a) Chronic glomerulonephritis
b) IgA nephropathy
c) Lupus nephritis.

1. ACUTE NEPHRITIS

Acute nephritis occurs when your kidney suddenly become inflamed. This condition used to be known as Bright's disease. Acute nephritis has several cause and it can ultimately lead to kidney failure if it left untreated.[2]

TYPES OF ACUTE NEPHRITIS

There are three types of acute nephritis, they are as follows:

a) Pyelonephritis

It is an inflammation of an kidney, usually due to bacterial infection. In the majority of the cases, the infections starts within the bladder and migrates up in the ureter and into the kidney.[3]

b) Glomerulonephritis

This type of acute nephritis produces inflammation in the glomeruli. There are millions of capillaries within each kidney. Glomeruli are the tiny clusters of the capillaries that transport blood and behave as filtering units. Damaged and inflamed glomeruli may not filter the blood properly.[4]

c) Interstitial nephritis

It is inflammation of the spaces between renal tubules. It is also called as "tubulo-interstitial nephritis.[5]

It mainly affects the part of kidney knows as the "interstitium". If patients are quickly taken off the medication causes the problems. A full recovery is possible if a patient follows the medication for few weeks.[2]

CAUSES OF ACUTE NEPHRITIS

Nephritis is often caused by infections and toxins, but it is most commonly caused by autoimmune disorders that affect major organs like kidney.[5]

Each type of nephritis has it's own cause, they are as follows:

a) Pyelonephritis

The majority of pyelonephritis causes results from "Escherichia coli(E.coli)" bacterial infection. This type of bacterium is primarily found in large intestine and is excreted in your stool. The bacteria can travel up from the urethra to the urinary bladder and kidneys, resulting in pyelonephritis.[4]

b) Glomerulonephritis

The main cause of this type of kidney infection is unknown. However some conditions may encourage an infection including; problems in the immune system, a history of cancer, an abscess that breaks and travels to your kidneys through your blood.[4]

c) Interstitial nephritis

It is caused due to the inflammation of the spaces between renal tubules. This type often results from an allergic reaction to a medication or antibiotic. Low potassium in your body blood is another cause of Interstitial nephritis. Taking medications for long period of time may damage the tissues of the kidney and lead to the interstitial nephritis.

WHO IS AT RISK FOR ACUTE NEPHRITIS

Certain people at greater risk for acute nephritis. The risk factor for acute nephritis:

1. A family history of kidney disease and infection.
2. Having an immune system disorders such as lupus.
3. Taking too many antibiotics or pain medications.
4. Recent surgery of urinary tract.[4]
5. High blood pressure.
6. Diabetes.
7. Obesity.
8. Heart diseases.
9. Being 60 years or older.

SYMPTOMS OF ACUTE NEPHRITIS

Symptoms of acute nephritis are usually not severe in the early stages. However to protect the kidneys from permanent damage, it is important to seek medical attention if these symptoms are present:

1. Changes in urinary habits.
2. Swelling anywhere in the body, especially the hands, feet, ankles and face.
3. Change in urine colour.
4. Foamy urine.
5. Blood in the urine (haematuria).

The most common symptoms of all three types of acute nephritis are:

Pain in the pelvis, pain or burning sensation while urination, a frequent need to urinate, cloudy urine, blood or pus in the urine, pain in the kidney area or abdomen, swelling of body, vomiting, fever, high blood pressure, glomerulonephritis.[4]

DIAGNOSIS OF ACUTE NEPHRITIS

- Nephritis may first be detected by routine blood or urine tests.[2] Two important indicators are blood urea nitrogen (BUN) and creatinine. These are waste products that circulate in the blood, where kidneys are responsible for filtering. If there is an increase in these numbers, this can indicate, the kidneys aren't working well. An imaging scan, such as CT-scan or renal ultrasound, can show a blockage or inflammation of kidneys or urinary tract.[4]
- According to the American academy of family physicians (AAFP), a renal biopsy is one of the best ways to diagnose acute nephritis. Because this involves testing an actual tissue sample from the kidney. This test isn't performed on everyone suffering from acute nephritis. If a person isn't responding well to treatment then a doctor must definitely diagnose the condition and a kidney biopsy should be performed.[4]
- Also a blood test that measures a waste product creatinine in the blood also sheds lights on kidney health. The best way to check for acute nephritis is to do a biopsy. For this procedure, doctors may uses a needle to remove of piece of the kidney and study it.[2]
- The diagnosis for acute nephritis depends on the cause of nephritis and blood tests, X-rays an ultrasound can also help ascertain if the individual has the condition.[7]

TREATMENT FOR ACUTE NEPHRITIS

Treatment/management of acute nephritis depends upon what has provoked the inflammation of the kidney.[8] Treatment for glomerulonephritis and interstitial nephritis may require treating the underlying conditions causing the problems. For example: if a medication you're taking is causing kidney problems, your doctor may prescribe an alternate medication.[4]

MEDICATION FOR ACUTE NEPHRITIS

A doctor will typically prescribe antibiotics to treat the kidney infection. If your infection is very serious, may require intravenous (IV) antibiotics within in pill form.[4]

Doctors may prescribe water pills to control both blood pressure and reduce swelling patients have.[2] Infections like pyelonephritis can cause severe pain. Your doctor may prescribe medication to relieve pain as you recover. If your kidneys are very inflamed, then doctor may prescribe corticosteroids.[4]

PREVENTING ACUTE NEPHRITIS

Kidney problems make following these healthy guidelines more importantly than ever;

A healthy diet can help to protect the kidney health.

Maintaining a healthy weight and exercising daily.

Having blood pressure and blood sugar within healthy limits.

Quit smoking, drinking etc.[5]

HOMECARE

When you have nephritis, your body needs time and energy to heal. Your doctor will likely recommend bed rest during your recovery. Your doctor may also advise you to increase your fluid intake to prevent dehydration and keep the kidneys filtering to release waste products. If your consideration affects your kidney function, your doctor may recommend a special diet low in certain electrolytes such as potassium. Many fruits and vegetables are high in potassium, he may instruct you regarding which foods are low in potassium. You can soak some vegetables in water and drain the water before cooking them, this process known as "Leaching" can remove extra potassium. Your doctor may also recommend cutting back on high sodium foods because high sodium in blood holds on water in the kidney which causes high blood pressure. The following are steps you can use to reduce Na^{++} in diet: Use fresh meats and vegetables instead of pre-packaged ones; choose foods labelled low sodium or no sodium; season your food with spices and herbs instead of sodium-blended seasonings or salting.[29]

2. CHRONIC NEPHRITIS

The kidneys contain up to one million nephrons, which are filtering units of the kidneys. Inside each nephron is a tiny network of looping blood vessels called the glomerulus. The glomerulus filters your blood, allowing excess fluid and waste to pass into the tubule and become urine. In a healthy nephron, this filter helps to keep blood cells and proteins in the bloodstream. If the kidney fails to filter waste for extended period of time then it is referred to as chronic nephritis.[9]

TYPES OF CHRONIC NEPHRITIS

There are three types of chronic nephritis, they are as follows;

a) Chronic glomerulonephritis

Chronic glomerulonephritis develops slowly showing very few symptoms in the early stages. This disease can cause serious kidney damage and kidney failure. It may run in the families or develop after a sudden case of disease.[2]

b) IgA nephropathy

One of the most common forms of nephritis, aside from those linked to diseases like diabetes and lupus. It develops when deposits of antibodies are found in the kidney which causes inflammation. It is more common in men than women, it is rarely found in young people because early symptoms are easy to miss. It is often treated with blood pressure medication.[2]

c) Lupus nephritis

It is an auto-immune disease which means it's a condition in which the body's immune system attacks its own tissues. Lupus nephritis is inflammation of the kidney caused by systemic lupus erythematous(SLE) a disease of a immune system.[10]

CAUSES OF CHRONIC NEPHRITIS

Chronic nephritis can caused by some sort of kidney damage. This things can damage kidney:

- High blood pressure and high blood sugar.
- Kidney infections and disease.
- Inherited kidney problems.
- Long term use of certain medications.
- Allergic reactions to medications.
- Streptococcal infections that are left untreated.
- Narrowed or closed off renal artery.

This all can reduce the amount of blood that the kidney receives and needs.[9]

WHO IS AT RISK FOR CHRONIC NEPHRITIS

People who are at higher risk for chronic nephritis can often do something to lower their risk. Some of the risks of chronic nephritis are:

- Diabetes.
- Chronic high blood pressure.
- Blockages.
- Overuse of certain medications such as painkillers.
- Drug abuse.
- Untreated streptococcal infections.
- Having family history of nephritis.
- Premature birth.

- Age.
- Trauma.
- Certain diseases such as AIDS, cancer, sickle cell anaemia, Systemic lupus erythematous (SLE), hepatitis C and congestive heart failure.[10]

SYMPTOMS OF CHRONIC NEPHRITIS

- As the nephritis develops, some of these symptoms may start to show: High blood pressure, drowsiness, swelling, itchy skin, nausea, shortness of breath, abdominal pain, back pain, headache, blood in urine, low urine output.
- Also foamy urine, swelling of the legs, ankles and feet.
- Joint problem, fever etc.[2]

DIAGNOSIS OF CHRONIC NEPHRITIS

If a patient has the symptoms of nephritis a doctor will order a couple different lab tests. He or she will test the urine for an infection or for protein, the blood for urea and creatinine, physical exam like ultrasound, CT-scan and MRIs to look at the kidneys and determine how much blood is getting to the kidneys and see any blockage. Laboratory tests may reveal anaemia or show signs of reduced kidney functioning, including azotaemia.[5]

TREATMENT OF CHRONIC NEPHRITIS

- Diabetes and hypertension are the two most common causes for nephritis. If person is overweight, the doctor may suggest losing weight to help manage the blood pressure or diabetes. Also blood sugar level is checked.[9]
- Regular exercise and a good diet are important parts of treating a chronic nephritis. Abstaining from tobacco, smoking and alcohol are also good ideas to treat it.
- Among the three types of chronic nephritis each behaves differently, the best course of treatment can only be decides after the nephritis has been identified by examining the sample removed by a kidney biopsy.
- Angiotensin converting enzyme (ACE) inhibitors or angiotensin receptor blocker (ARB) have proven to be effective treatment modalities for both reducing the amount of protein in the urine and decreasing the blood pressure.[10]

MEDICATION FOR CHRONIC NEPHRITIS

The pills usually prescribed for blood pressure control when nephritis is a symptoms are thiazide diuretics and beta blockers.

Other medications prescribed are diuretics or anti-inflammatory medications.

If the disease gets to the point where one or both kidneys are not functioning, dialysis may be needed.

PREVENTION

There are many ways to prevent or reduce the risk of most chronic nephritis:

- If you have diabetes, keep a very good watch on your blood sugar.
- If you have high blood pressure, there are certain medications that can be prescribed to keep it lower.
- Sometimes blockages can be opened or repaired, or kidney stones removed if they are the cause of the blockages.
- Cut back on the amount of painkillers you take.
- If your family has a history of kidney problems, have regular check-ups.[30]

HOME CARE

There are many ways to treat nephritis at home.

- Carrot juice
- Banana
- Avocado
- Grapes
- Coconut water.[11]

GLOMERULONEPHRIRIS

Glomerulonephritis(GN),also known as glomerulonephritis is a term used to refer to several kidney diseases(usually affecting both kidneys). Many of the diseases are characterised by inflammation either of the glomeruli or of the small blood vessels in the kidneys, hence the name,[12] but not all diseases necessarily have an inflammatory component. As it is not strictly a single disease, its presentation depends on the specific disease entity; it may present with isolated " Haematuria or Proteinuria " or as a nephrotic syndrome, a nephritic syndrome, acute kidney injury or chronic kidney disease.[28]

They are categorized into several different pathological pattern, which are broadly grouped into NON-PROLIFERATIVE or PROLIFERATIVE types. Diagnosing the pattern of GN is important because the outcome and treatment differs in different types. Primary causes are intrinsic to the kidney. Secondary causes are associated with certain infections(bacterial, viral, or parasitic pathogens), drugs, systemic disorder(SLE) vasculitis or diabetes.[28]

A glomerulus, a function unit that represent the first step in the filtration of blood and generation of urine. Glomerulonephritis refers to an inflammation of the glomerulus, which is the unit involved in the filtration in the kidney. This inflammation typically results in one or both of the " nephrotic" or "nephritic" syndrome.[13]

NEPHROTIC SYNDROME

The nephrotic syndrome is characterised by the finding of edema in a person with increased protein in the blood, with increased fat in the blood. Inflammation that affects the cells surrounding the glomerulus, podocytes, the permeability to proteins, resulting in an increase in excreted proteins. When the amount of protein excreted in the urine exceeds the liver ability to compensate, fewer proteins are detected in the blood-in particular albumin, which makes up the majority of circulating proteins. With decreased proteins in the blood there is a decrease in the "ONCOTIC PRESSURE" of the blood. This results in edema, as the oncotic pressure in tissue remains the same. It should be noted here that although decreased intravascular oncotic (i.e. osmotic pressure) pressure partially explains the patients edema, more recent studies have shown that extensive sodium retention in the distal nephron(usually collecting duct) is the predominant cause of water retention and edema in the nephrotic syndrome.[14] This is worsened by the secretion of the hormone "Aldosterone" by the adrenal gland. Which is secreted in response to the decrease in circulating blood and causes sodium and water retention. "Hyperlipidaemia" is thought to be a result of the increased activity of the liver.

NEPHRITIC SYNDROME

The nephritic syndrome is characterised by blood in the urine(especially Red Blood Cells casts-with dysmorphic red blood cells) and a decrease in the amount of urine in the presence of hypertension. In this syndrome, inflammatory damage to cells lining the glomerulus are thought to result in destruction of the epithelial barrier, leading to blood being found in the urine. At the same time, reactive changes, e.g. proliferation of mesangial cells, may result in a decrease in kidney blood flow, resulting in a decrease in the production of urine. The Renin-

Angiotensin System may be subsequently activated, because of the decrease in perfusion of "Juxtaglomerular Apparatus", which may result in hypertension.[15]

NONPROLFERATIVE

This is characterised by forms of glomerulonephritis in which the number of cells is not changed. These forms usually result in the "nephrotic syndrome".

CAUSES INCLUDE

MINIMAL CHANGE DISEASE

It is characterised as a cause of "nephrotic syndrome" without visible changes in the glomerulus on microscopy. Minimal change disease typically presents with edema, an increase in proteins passed from urine and decrease in blood passed from urine with decrease in blood protein level and increase in circulating lipids(i.e. nephrotic syndrome) and is the most common cause of the nephrotic syndrome in children. Although no change may be visible by light microscopy, changes on electron microscope within the glomerulus may show a fusion of the foot processes of the podocytes. It is typically managed into corticosteroids and does not progress to chronic kidney disease.[13]

FOCAL SEGMENTAL GLOMERULOSCLEROSIS

It is characterised by a Sclerosis of segments of some glomerulus. It is likely to present as a "nephrotic syndrome". This form of glomerulonephritis may be associated with conditions such as "HIV and Heroin abuse, or inherited as Alport syndrome". The cause of about 20-30% of focal segmental glomerulosclerosis is unknown. On microscopy, affected glomerulus may show an increase in hyalin, a pink and homogeneous material, fat cells, an increase in the mesangial material and collagen.

MEMBRANOUS GLOMERULONEPHRITIS

It may cause either nephrotic or nephritic picture. Microscopically, MGN is characterised by a thickened glomerular basement membrane without a hyperproliferation of the glomerular cells. Immunofluorescence demonstrates diffuse granular uptake of IgG. The basement membrane may completely surround the granular deposited, forming a "Spike and Dome" pattern. Tubules also display the symptoms of a typical type III hypersensitivity reaction, which causes the endothelial cells to proliferate, which can be seen under a light microscope with a PAS-stain.[16]

THIN BASEMENT MEMBRANE DISEASE

Thin basement membrane disease is an autosomal dominant inherited disease characterized by thin glomerular basement membranes on electron microscopy. It is a benign condition that causes persistent microscopic haematuria. This also may cause proteinuria which is usually mild and overall prognosis is excellent.[15]

PROLIFERATIVE

Proliferative glomerulonephritis is characterised by an increased amount of cells in the glomerulus. These forms usually present with a triad of blood in the urine, decreased urine production, and hypertension, the nephritic syndrome. These forms usually progress to end-stage kidney failure (ESKF) over weeks to years(depending on types).[15]

IgA NEPHROPATHY

IgA nephropathy, also known as Berger's disease is the most common type of glomerulonephritis, and generally presents with isolated visible or occult haematuria, occasionally combined with low grade proteinuria, and rarely cause a nephritic syndrome characterised by protein in urine. IgA nephropathy is classically described as a self-resolving form in young adults several days after a respiratory infection. It is characterised by deposits of IgA in the space between glomerular capillaries.[2]

Henoch-Schonlein Purpura refers to a form of IgA NEPHROPATHY, Typically affecting children, characterised by a rash of small bruises affecting the buttocks and lower legs, with abdominal pain.[2]

POST INFECTIOUS

Post infectious glomerulonephritis can occur after essentially an infection, but classically occurs after infection with the bacteria Streptococcus Pyogenes. It typically occurs 1-4 weeks after a pharyngeal infection with this bacterium, and is likely to present with malaise, a slight fever, nausea, and a mild nephritic syndrome of moderately increased blood pressure, gross haematuria, and smoky-brown urine. Diagnosis may be made on clinical findings or though Antistreptolysin O antibodies found in the blood. A biopsy is seldom dons, and the disease is likely to self-resolve in children in 1-4 weeks, with a poorer prognosis if adults are affected.[13]

MEMBRANOPROLIFERATIVE

Membranoproliferative GN [MPGN], also known as "Mesangiocapillary glomerulonephritis".[13] Two primary subtypes exist:

- **TYPE 1 MPGN** is caused by circulating immune complexes, typically secondary to systemic iupus erythematosus, hepatitis B and C, other chronic or recurring infections. Circulating immune complexes may activate the complement system, leading to inflammation and an influx if inflammatory cells.[15]
- **TYPE 2 MPGN**, also known as "Dense Deposit Disease" is characterised by an excessive activation of the complement system. The C3 nephritic factor auto-antibody stabilizer C3-convertase, which may lead to an excessive activation of complement.[15]

RAPIDLY PROGRESSIVE GLOMERLONEPHRITIS

Rapidly progressive glomerulonephritis ,also known as crescentic GN ,is characterised by a rapid, progressive deterioration in kidney function. People with rapidly progressive glomerulonephritis may present with a nephritic syndrome.[17] Three main sub types are :-

- **TYPE 1** is a good pasture syndrome an autoimmune disease also affecting the lung. In good pasture syndrome, IgG antibodies directed against the glomerular basement membrane trigger an inflammatory reaction, causing a nephritic syndrome and the coughing up of blood.[15] high dose immunosuppression is required [intravenous methylprednisolone] and cyclophosphamide, plus plasmapheresis. Immunohistochemistry staining of tissue shows linear IgG deposits.
- **TYPE 2:** is characterized by immune-complex-mediated damage, and may be associated with systemic lupus erythematous, post-infective glomerulonephritis, IgA, nephropathy, and IgA vasculitis.[15]
- **TYPE 3:** rapidly progressive glomerulonephritis, also called "Pauciimmune type", is associated with causes of vascular inflammation including glomerulonephritis with polyangiitis (GPA) and microscopic polyangiitis.[27]

Histo-pathologically, the majority of glomeruli present "crescents". Formation of crescents is initiated by passage of into the bowman's space as a result of increased permeability of glomerular basement membrane. Fibrin stimulates the proliferation of endothelial cells of bowman's capsule, and an influx of manocytes. Rapid growing and fibrosis of crescents compresses the capillary loops and decreases the bowman's space, which leads to kidney failure within weeks or months.

DIGNOSIS

Some form s of glomerulonephritis are diagnosed clinically based on findings on history and examination other tests may include:

- Urine examination
- Blood test investigating the cause, including FBC, inflammatory ma markers and special tests including (ALSO, ASLO, ANCA, Anti-GBM, Complement levels, antinuclear antibiotics.)
- Biopsy of the kidney.[17]

LUPUS NEPHRITIS

Lupus nephritis (also known as SLE nephritis)[18] is an inflammation of the kidneys caused by systemic lupus erythematous (SLE), an autoimmune disease. It is type of glomerulonephritis in which said to be secondary and has a different pattern and outcome from conditions with a primary cause originating in the kidney.

SIGNS AND SYMPTOMS

General symptoms of lupus nephritis include Fever, Oedema, High blood pressure, joint pain, muscle pain, molar rash, foamy urine.

CLASS1 DISEASE (MINIMAL MESANGIAL GLOMERULONEPHRITIS)

In it's histology has a normal appearance under a light microscope, but mesangial deposits are visible under an electron microscope. At this stage uranalysis is normal.[6]

CLASS 2 DISEASE (MESANGIAL PROLIFERATIVE GLOMERULONEPHRITIS)

Is noted by mesangial and matrix expansion. Microscope haematuria with or without proteinuria may be a seen. Hypertension, nephrotic syndrome, acute kidney insufficiency are very rare at this stage.

CLASS 3 DISEASE (FOCAL GLOMERULONEPHRITIS)

Is indicated by sclerotic lessons involving less than 50% of the glomeruli, which can be segmental or global, and active or chronic, with endocapillary or extracapillary or extracapillary proliferate lessons. Immunofluroscence reveals positively for IgG, IgA, IgM,c3 and Cg. Clinically, haematuria and proteinuria are present, with or without nephrotic syndrome, hypertension, and elevated serum creatinine.[4]

CLASS 4 DISEASE (DIFFUSED PROLIFERATION NEPHRITIS)

Is both the most severe and the most common subtype. More than 50% of glomeruli are included. Lesions can be segmental or global, and active or chronic with endocapillary or extracapillary proliferative lesions. Clinically it is same as class 3 disease.[6]

CLASS 5 DISEASE (MEMBRANOUS GLOMERULONEPHRITIS)

Is characterized by diffuse thickening of the glomrular capillary wall (segmentally or globally), with diffused membrane thickening, and subepithelial deposits seen under the electron microscope. Clinically it is same as class 3 and class 4 disease but also includes or leads to thrombotic complications such as renel vein thrombosis or pulmonary emboli.[6]

Class 6 disease (Advanced sclerosing lupus nephritis)

It is represented by global by global sclerosis involving more than 90%. Of glomeruli, and represent healing of polar inflammatory injury. It is characterized by slowly progressive kidney dysfunction, with relatively bland urine sediments.[7] It is not diagnostic however, as it exists in other conditions such as HIV infections.[8] It is throughout to be due to chronic Interferon exposure.[9]

CAUSE

The cause of lupus nephritis, a genetic predisposition plays a role in lupus nephritis. Multiple genes many of which are not yet identified, mediate this genetic predisposition.[10]

DIGNOSIS

Dignosis of lupus nephritis depends on the blood test, urinalysis, x-ray, ultrasound, and a kidney biopsy.[11]

TREATMENT

Drug regimens prescribed for lupus nephritis include: Mycophenolate mofetil.[12] Intravenous cyclophosphamide with corticosteroids,[13] and the immune suppressant azathioprine with corticosteroids.[14] This work for maintainance of therapy for individual with high risk of lupus.[15]

ROLE OF PHARMACISTS

Role of pharmacist defines process as "what actually is done in giving and receiving care, including the patient's activities in seeking care and carrying it out, as well as the

practitioner's activities in making a diagnosis and recommending or implementing treatment.[19] and outcomes as changes in health status that can be attributed to that care."[20] Additionally pharmacist intervention was defined as any action with the aim of modifying the process of use of drugs; either in patient's activities or medical or health care practitioner's activities. Over the lasts decades, pharmacist have seen their potential competencies broadened Beyond the traditional dispensing role.[21] The effect of pharmacist delivered service has been reported for heart failure, asthma management, antimthrombotic use and opiod substitutes supply. Furthermore results of pharmacist collaborative practice models in meta-analysis for several physician-pharmacist hypertension and hyperlipidemia. However, to date, there has not been a systematic review describing the impact of interventions carried out by pharmacist in patients with chronic kidney disease. The aim of this review is therefore to address the impact of pharmacist's interventions in patients with chronic kidney disease.[23] Patients with chronic kidney disease have multiple comorbidities and require complicated therapeutic regiments. The role of pharmacists caring for these patients has been documented, but no review of the impact of these interventions has occurred to date.[24] The aim of this work is to assess the impact of pharmacist's interventions in patients with chronic kidney disease.[25]

METHODS

Medicine, Medline international pharmaceutical Abstracts, pharmacy abstracts and the cochrane library were searched for quantitative studies addressing the contribution of pharmacist's interventions in patients with chronic kidney disease. Quality of controlled studies was assessed using the downs and black scale.[26]

CONCLUSION

The evidence of pharmacist's interventions in patients with chronic kidney disease in sparse, of variable quality and with heterogeneous outcomes. On the basis of best available evidence. Pharmacist's interventions may have a positive impact.[24]

REFERENCES

1. Keto Acids-Advances in Research and Application. Edition p.220e,2013
2. Danielle Dresden-Medical New Today, 28 August 2016.
3. Steven Kim, MD-Health line, 28 October 2015.
4. Brindles Lee Macon, Winnie Yu and Rachel Nail-New letter, 2012.
5. Interstitial nephritis, medline plus medical encylopedia 14/06/2015.

6. Www.nim.nih.gov.
7. America Academy, health line, 6 June 1998.
8. Medline plus, drug information, Medline plus.gov.Retrieved 23 July 2016.
9. Chronic nephritis: kidney infection 23 July 2016.
10. Edinburgh, churchill livingstone/Elsevier 1978.
11. Colledge, Nicki R; Walker, Brain R; Ralston, Stuart H, principles and practice of medicine, 21st edition. 12 December 2000.
12. Glomerulonephritis at dorland's medical dictionary.
13. Robert Britton, 21st edition ISBN 970-0-7020
14. Stephan R. Orth, M.D. and Eberhard Ritz, 23 April 1998.
15. Kumar, vinay, ed. Robbins basic pathology 2007.
16. Medlineplus Encyclopaedia, membranous nephropathy.
17. Saunders/Elsevier, Philadelphia 8 November 1995.
18. Ponticelli, C.Moroni, G. Renal transplantation in lupus nephritis 1 January 2005.
19. Lewis, Edmund J; Schwartz, Melvin M. 2010.
20. Practice Essentials, Background, Pathology.
21. Karageorgas TP, Tseronis DD, Distinct clinical pathway 2011.
22. K Foury.H, Biomedicine and Biotechnology journal.2014.
23. Salgado, Alberto, An overview of recent findings 2012.
24. Weening JJ, D. Agati, The clinical pathology, February 2004.
25. Elizabeth D Agabegi, Steven s. Aim of work 6 April 2008.
26. Henderson, L Masson, Medline international pharmaceutical Abstracts, 17 March 2001.
27. Greenberg, Arthur, cheung, Alfred K, Primer on kidney disease 9 June 2004.
28. Appel, Gerald B, Mycophenolate mofetil versus cyclophosphamide 2005.
29. Vanessa E Lupus Research institute PMC 2678035
30. https://en.wikipedia.org/w/index.php