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FACULTY OF NURSING

Glumeronephritis

BY:-

Arpit Kamal

Nursing Tutor

MSN Department

Rama College Of Nursing

Glomerulonephritis

- A variety of diseases can affect the glomerular capillaries
- Glomerulonephritis is an inflammation of the glomerular capillaries membranes.
- Caused by an immunologic reaction that result in proliferative and inflammatory change in glomerular structure

- Antigen–antibody complexes form in the blood and become trapped in the glomerular capillaries (the filtering portion of the kidney), inducing an inflammatory response.

- IgG, the major immunoglobulin(antibody) found in the blood, can be detected in the glomerular capillary walls

- The major clinical manifestations of glomerular injury include proteinuria, hematuria, decreased glomerular filtration rate, and alterations in excretion of sodium (leading to edema and hypertension).

- Glomerulonephritis can be acute or chronic
- It is usually manifested by either a nephrotic syndrome or a nephritic syndrome.

Nephrotic syndrome

- Nephrotic syndrome is a sate of clinical manifestations caused by protein wasting secondary to diffuse glomerular damage.
- Manifestations include proteinuria (> 3.5g/day), hypoalbuminemia and edema.

Nephritic syndrome

- Nephritic syndrome refers to a set of clinical manifestations that includes hematuria and at least one of the following:
- Oliguria (urine output < 400ml/ 24 hours)
- Hypertension
- Elevated blood urea nitrogen level (BUN)
- Decreased GFR

- Inflammation of the glomeruli **m**ost often caused by an autoimmune disease, but it can also result from infection.

- If glomerulonephritis occurs on its own, it's known as primary glomerulonephritis.

- If another disease, such as lupus or diabetes, is the cause, it's called secondary glomerulonephritis

- It may present with isolated hematuria and/or proteinuria; or as a nephritic syndrome, acute renal failure, or chronic renal failure.

CLASSIFICATION (based on etiology)

- PRIMARY GLOMERULONEPHRITIS- immune response to pathogens
- Acute glomeruonephritis
- Post-infectious glomeruonephritis
- Post streptococcus glomeruonephritis
- Infectious glomeruonephritis
- Membranoproliferative glomeruonephrits

- Rapidly progressive glomerulonephritis
- Idiopathic membranous glomerulonephritis
- Immune globulin A (IgA) nephropathy
- Chronic glomerulonephritis
- Focal glomerular sclerosis

- SECONDARY GLOMERULONEPHRITIS- related to systemic disease.
- Goodpasture’s syndrome
- Hemolytic uremic syndrome
- Polyarteritis
- Progressive systemic sclerosis
- Systemic Lupus Erythematosis

- Wegener’s granulomatosis
- Thrombocytopenic purpura
- Post partum renal failure

Poststreptococal GN

- Onset- 1 -3 wks after betahemolytic streptococcal infection of throat or skin.
- Diagnosis finding -Elevated antistreptolysin o titer
- Prognosis - Variable complete recovery to end stage renal disease.

Membranoproliferative GN

- Onset-nephrotic syndrome sometime preceded by a streptococcal infection.
- Diagnosis finding- proteinurea, hematuria.
- Prognosis- gradual progressive chronic renal failure.

Rapidly progressive GN

- Onset- nephritic syndrome sudden may follow antigen or infection peak ages 40-60 yr.
- Diagnosis finding- hematuria, edema, hypertension, proteinurea, acidosis
- Prognosis- progress to renal failure with in weeks or month.

Idiopathicmembranous GN

- Onset- peak age 40-70 yrs caused by unknown antigen.
- Diagnosis finding- asymptomatic protein urea.
- Prognosis- 25% have spontaneous remission ,25% have renal failure ,25% have persistent protein urea 25% have deteriorating renal function.

Membrenous glomerulonephritis

onset -Nephrotic syndrome

Commonly secondary to drug therapy toxins or systemic autoimmune disease

Diagnostic findina: heavy proteinuria

Prognosis: variable, 30% have spontenous remission

Types: according duration or nature of illness

- Acute
- Chronic
- Rapid progressive glomerulonephritis

ACUTE GLOMERULONEPHRITIS

- Acute glomerulonephritis is the **inflammation of the glomeruli** which causes the kidneys to malfunction

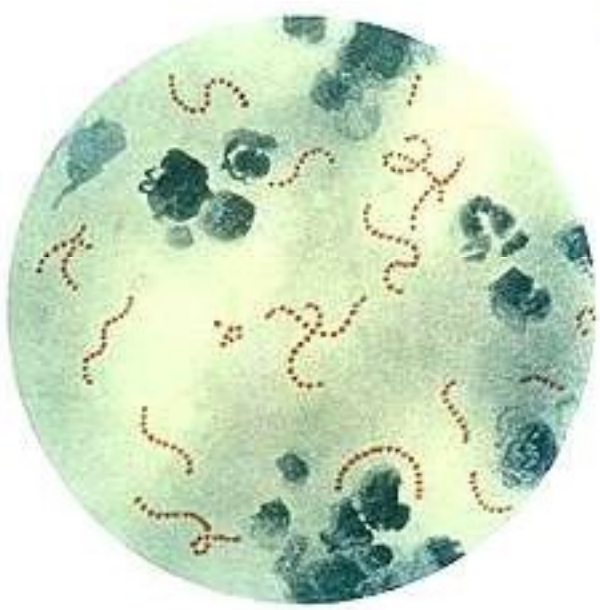
- It is also called Acute Nephritis, Glomerulonephritis and Post-Streptococcal Glomerulonephritis

- Predominantly affects children from ages 2 to 12

- Incubation period is 2 to 3 weeks

Causative factor

- *Streptococcus pyogenes*



- Group A beta hemolytic streptococcal infection of the throat precedes the onset of glomerulonephritis by 2 to 3 weeks
- It may also follow impetigo (infection of the skin)
- Acute viral infections(upper respiratory tract infections, mumps, varicella zoster virus,
- Epstein-Barr virus, hepatitis B, and human immunodeficiency virus infection.

- In some patients, antigens outside the body (eg, medications, foreign serum)
- Initiate the process, resulting inantigen-antibody complexes being deposited in the glomeruli.
- In other patients, the kidney tissue itself serves as the provocative antigen.

Causes

- Primary disease: post infectious glomerulonephritis, membrane proliferative glomerulonephritis, and membranous glomerulonephritis.
- Group A betahemolytic streptococcal infection of throat.
- It may follow impetigo and acute viral infection.
- Antigen outside the body
- Autoimmunity
- Heredity
- Infective/ Sclerotic diseases
- Idiopathic

Pathophysiology

- Antigen
- Circulating antigen- antibody immune complex formed
- That is strapped in the glomerular membrane
- Leading to inflammatory response
- Activation of the immune system
- Release of inflammatory mediator
- Endothelial cell proliferation & glomerular membrane swells
- Becomes permeable to plasma protein and blood cells.
- Normal tissue of glomeruli is replaced by fibrous tissue after inflammation
- Losses of filteration membrane
- Decrease GFR
- Accumulation of metabolic waste products

Clinical Manifestation

- Hematuria (microscopic or macroscopic)
- Edema
- Proteinuria (>3.0g of proteinuria per day)
- Hypoproteinemia
- Reduced glomerular filtration rate/Azotemia
- The urine may apper cola colored because of red blood cells and protein plugs or casts.

- In sever case renal failure and oliguria
- Hypertention
- Hypoalbuminemia, hyperlipidemia, and fatty casts in the urine.
- BUN and serum creatinine levels may increase as urine output decreases.
- May present anaemia

Diagnostic evaluation

- History taking and physical examination: In acute glomerulonephiritis, the kidneys become large edematous, and congested.
- Kidney biopsy: kidney biopsy may be needed for definitive diagnosis.

- Serial determinations of antistreptolysin O or anti-DNase B titers, C - reactive protein are usually elevated in poststreptococcal glomerulonephritis.

- More than half of patients with IgA nephropathy (the most common type of primary glomerulonephritis) have an elevated serum IgA.

Complications

- Hypertensive encephalopathy
- Heart failure
- Pulmonary edema
- ESRD (if not treated)

Management:

- Management consists primarily treating symptoms, attempting to preserve kidney function, and treating complications promptly.

•Pharmacological therapy: depends on the cause

streptococcal infection penicillin is the agent of choice. other antibiotics may be prescribed

- Corticosteroids and immunosuppressant medications may be prescribed for patients with rapidly progressive acute glomerulonephritis

- Dietary protein is restricted when renal insufficiency and nitrogen retention (elevated BUN) developed.
- Potassium and Sodium is restricted when the patient has hypertension, heart failure, and edema.
- Loop diuretics and antihypertensive medications may be prescribed to control hypertension.
- Fluid intake is restricted.
- Carbohydrates are increased liberally to provide energy and reduce catabolism of protein.

Prevention

- Proper hygiene
- Prompt medical assessment for necessary antibiotic therapy should be given when infection is suspected
- Prophylactic immunizations

Chronic Glomerulonephritis

- Chronic glomerulonephritis may be due to repeated episodes of acute glomerulonephritis, hypertensive nephrosclerosis, hyperlipidemia, chronic tubulointerstitial injury or hemodynamically mediated glomerular sclerosis.

- Chronic glomerulonephritis a slowly progressive glomerulonephritis generally leading to irreversible renal failure over a few to as many as 30 years.

- In some people there's no history of kidney disease, so the first indication of chronic glomerulonephritis is chronic kidney failure.

Pathophysiology

- In Chronic glomerulonephritis the kidneys are reduced to as little as one-fifth their normal size (consisting largely of fibrous tissue).

- The cortex shrinks to a layer 1 to 2 mm thick or less
- Bands of scar tissue distort the remaining cortex, making the surface of the kidney rough and irregular.
- Numerous glomeruli and their tubules become scarred, and the branches of the renal artery are thickened.
- The result is severe glomerular damage that results inESRD.

Clinical Manifestations

- The symptoms of chronic glomerulonephritis vary.

- Some patient with severe disease have no symptoms at all for many years
- Hypertension or elevated BUN and serum creatinine
- Sudden severe nosebleed, a stroke, or a seizure
- Feet slightly swollen at night
- Other general symptoms; loss of weight and strength, increasing irritability, nocturia, headache, dizziness, and digestive disturbances are common

- As chronic glomerulonephritis progresses, sign and symptoms of chronic kidney disease and chronic renal failure may develop.
- Poorly nourished, with a yellow-gray pigmentation of the skin and periorbital and peripheral (dependent) edema.
- Blood pressure may be normal or severely elevated.
- Retinal findings include hemorrhage, exudate, narrowed tortuous arterioles, and papilledema
- Mucous membranes are pale because of anemia

- Cardiomegaly, a gallop rhythm, distended neck veins, and other signs and symptoms of heart failure may be present
- Crackles can be heard in the lungs.
- Peripheral neuropathy with diminished deep tendon reflexes and neurosensory changes occur late in the disease.
- The patient becomes confused and demonstrates a limited attention span.
- Late findings evidence of pericarditis with a pericardial friction rub and pulsus paradoxus

- Many patients report that their feet are slightly swollen at night.
- Most patients also have general symptoms, such as loss of weight and strength, increasing irritability, and an increased need to urinate at night (nocturia).
- Headaches, dizziness, and digestive disturbances are common.

- As chronic glomerulonephritis progresses, signs and symptoms of renal insufficiency and chronic renal failure may develop.
- The patient appears poorly nourished, with a yellow-gray pigmentation of the skin and periorbital and peripheral (dependent)
- Edema
- Blood pressure may be normal or severely elevated.

Diagnostic findings

- Urinalysis reveals a fixed specific gravity of about 1.010, variable proteinuria, and urinary casts.
- As renal failure progressses and the GFR falls below 50ml/min, the following changes occur
- Hyperkalemia
- Metabolic acidosis
- Anaemia
- Hyperalbuminemia with edema
- Increased serum phosphorus lev

- decreased serum calcium level
- Mental status changes
- Impaired nerve conduction due to electrolyte abnormalities and uremia.
- Chest x-ray; may show cardiac enlargement and pulmonary edema
- ECG: may be normal or may indicate left ventricular hypertrophy associated with hypertension and signs of electrolyte disturbances, such as tall, tented Twave

Medical management

Symptoms management.

- If hypertention reduction of bloodpressure with sodium and water restriction, antihypertensive agents, or both.
- Monitoring weight daily, and diuretics medications are prescribed to treat fluid overload.
- Providing proteins of high biological value and adequate calories.
- Urinary tract infection must be treated promptly to prevent further renal damage.
- Initiation of dialysis early in the course of the disease.
- Prevent fluid and electrolyte imbalances, and minimize the risk of complications of renal failure

- Adequate calories are also important to spare protein for tissue growth and repair.
- UTIs must be treated promptly to prevent further renal damage.
- Initiation of dialysis is considered early in the course of the disease to keep the patient in optimal physical condition

- prevent fluid and electrolyte imbalances, and minimize the risk of complications of renal failure.
- The course of dialysis is smoother if treatment begins before the patient develops significant complications.

Reduce inflammation

- Plasmopherosis has been used in some research protocol to reduce the number of antigen in certain type of GN.including rapidly progressive GN.
- Large volume of the client's plasma cyclically removed and replace with Fresh frozen plasma.

- Antibiotic therapy such as penicillin for streptococcal GN .

- It is also used prophylactically after streptococcal infection to prevent further damage.

Rapid progressive glomerulonephritis

- Rapid progressive glomerulonephritis is a disease of the kidney characterized clinically by a rapid decrease in the glomerular filtration rate (GFR) of at least 50% over a short period, from a few days to 3 months

Prevention

- proper hygiene
- prompt medical assessment for necessary antibiotic therapy should be sought when infection is suspected
- prophylactic immunizations
- Control the blood sugar to help prevent diabetic nephropathy.
- Control the blood pressure, which lessens the likelihood of damage to the kidneys from hypertension
- Seek prompt treatment of a strep infection causing a sore throat or impetigo.
- To prevent infections, such as HIV and hepatitis that can lead to some forms of glomerulonephritis follow safe-sex guidelines and avoid intravenous drug use

Nursing Management

- **Assessment**: Assess need of the patient through history taking and physical examination
- Obtain medical history: focus on recent infections or symptoms of chronic immunologic disorders (systemic lupus erythmatus, scleroderma).
- Assess urine specimen for blood, protein, color, and amount.
- Perform physical examinitation specifically looking for signs of edema, hypertension, and hypervolemia (engorged neck veins, elevated jugular venous pressure, adventitious lung sounds, and cardiac arrhythmia).
- Evaluate cardiac status and serum laboratory values for electrolyte imbalance.

Nursing Diagnosis and nursing actions

- **Ineffective tissue perfusion related to damage to glomerular function**
- **Excess fluid volume related to compromised renal function**
- ***Altered nutrition; less than required related to anorexia & altered renal function***
- ***Activity intolerance related to fatigue and hematuria***

- ***Altered emotion and coping related to questionable prognosis with prolonged illness***
- ***Skin integrity, high- risk for impaired related to edema***
- ***High risk for infection related to altered immune response secondary to treatment***

Promoting Renal Function

- Monitor vital sign, intake and output, and maintain dietary restrictions during acute phase.
- Encourage rest during the acute phase, as directed, until the urine clears and BUN, creatinine, and BP normalize.
- Administer medications, as ordered, and evaluate patient's response to antihypertensive, diuretics, H2blockers, phosphate binding agents, and antibiotics (if indicated).
- Carbohydrates are given liberally to provide energy and reduce the catabolism of protein.

Improving Fluid Balance

- Carefully monitor fluid balance; replace fluids according to patient's fluid losses (urine, respiration, feces) and daily body weight as prescribed.
- Monitor pulmonary artery pressure and CVP, if indicated, during acute hospitalization.
- Monitor for signs and symptoms of heart failure: distended neck veins, tachycardia, gallop rhythm, enlarged and tender liver, crackles at bases of lungs.
- Fluid intake is usually restricted.
- Thirst may be relieved by sucking on hard candies or lemon slices or by using ice chips rather than a glass of water.
- Assist to plan fluid distribution during day (eg- with meal
- Observe for hypertensive encephalopathy and any evidence of seizure activity.

Improve nutritional status

- It is important to protect kidneys while recovering their function. The diet prescribed by physician is generally high-calorie and low-protein.
- This diet avoids protein catabolism and allows the kidney to rest because it handles few protein molecules & metabolism.
- The degree to which protein restricted depends upon the amount excreted in the urine and the patient's requirements.
- Sodium is also restricted, depending on the amount of edema present.
- Anorexia nausea and vomiting may interfere with adequate intake, requiring creative intervention on a part of nurse.
- Provide antiemetic according to prescription.
- Provide meal frequently in small amount.
- Provide the patient's diet plan with the help of dietician.

Enhance general wellbeing

- Rest is essential- both physical and emotional; there is a direct correlation between activity and amount of hematuria and proteinurea .
- Exercise also increases catabolic activity.
- The allowable amount of activity depends on the report of serial urinalysis.

Enhance coping

- The patient may need assistance in arranging personal matters, such as; family, home, finance and community responsibilities.
- Encourage the patient to talk about fear and concerns, if needed, help the patient to deal with emotional reactions; only after handling these problems will patient be able to rest emotionally.
- Appropriate diversionary activities may help to cope with prolonged illness.

Improve skin integrity

- Edema interferes with cellular nutrition, which makes the patient more susceptible to skin breakdown
- Therefore take precaution to prevent this complication by providing good hygiene, massage and position change.

Prevent infection

- Immunosuppressive and Corticosteroids further reduced host resistance, although isolation is not needed, take care to protect the client from others with obvious infections process.
- General supportive measure to boost the defense mechanism.

Teach appropriate ways to avoid infection:

- use mask
- Treat opportunist infection promptly
- Do not expose self in rush situation
- Take diet high in calorie and vitamins

Thank you