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18 CHAPTER

Disorders of Kidney and Urinary Tract

CHAPTER HIGHLIGHTS

- Congenital Abnormalities of the Kidney and Urinary Tract
- Wilms' Tumor (Nephroblastoma)
- Nursing Management of the Child with Urologic Surgery
- Acute Glomerulonephritis
- Chronic Glomerulonephritis
- Urinary Tract Infections
- Nephrotic Syndrome
- Acute Renal Failure
- Chronic Renal Failure
- Care of the Child Who Undergoes Dialysis

INTRODUCTION

Disorders of kidney and urinary tract are commonly seen in pediatric units as medical and surgical problems. Congenital malformations, neoplasms, infections, inflammations and progressive impairment of renal functions are common conditions found in children. The important clinical features are abnormal micturition, hematuria, oliguria, dysuria, ureteric colic, enuresis, polyuria and polydipsia. Other features are edema, proteinuria, anemia, hypertension, growth failure, abdominal mass, etc.

Diagnostic evaluation for these problems should include details history of illness, clinical examination, urine examination, blood tests to assess renal functions (creatinine, urea), serum albumin, cholesterol, antistreptolysin O (ASO) titer, glomerular filtration rate (GFR), urinary concentration test and imaging of urinary tract by plain X-ray, intravenous pyelogram (IVP), micturating cystourethrogram (MCU), ultrasonography, radionuclide imaging and renal biopsy, etc.

CONGENITAL ABNORMALITIES OF THE KIDNEY AND URINARY TRACT

Congenital malformations of the kidneys and urinary tract are commonly found in the pediatric surgery units. These problems usually required surgical correction. Some of them are producing no clinical symptoms, but approximately about 25% cases of chronic renal failure are due to congenital anomalies. These problems may be associated with other congenital anomalies like trisomy-21, neural tube defects and structural abnormalities of lower limbs.

Congenital anomalies of urinary tract are found in about 8 to 10% of children. Common anomalies are mentioned below:

- **Kidney and ureter:** Renal agenesis, renal hypoplasia, horse-shoe kidney, polycystic renal disease, ectopic kidney, duplication of renal pelvis and ureter, congenital renal neoplasm (nephroblastomatosis), hydronephrosis, pelviureteric obstruction, hydroureter or megaureter, ureterocele, ectopic ureteral insertion, etc.
- **Bladder and urethra:** Ectopia vesicae, patent urachus, bladder neck obstruction, posterior urethral valves, neurogenic bladder, hypospadias, epispadias, phimosis, urethral stenosis, meatal stenosis, etc.

Renal Agenesis

Renal agenesis is the absence of kidney due to failure of ureteric bud formation or mesenchymal blastema differentiation of final mesenchymal condensation. It may be unilateral or bilateral and associated with various congenital anomalies. Bilateral renal agenesis with associated malformations is known as Potter's syndrome.

Renal Hypoplasia and Dysplasia

Renal hypoplasia occurs due to reduction of renal mass affecting the nephron. It may be unilateral or bilateral. It is sometimes difficult to differentiate from renal dysplasia which is characterized by disorganization of renal parenchyma with immature nephron and ductal elements resulting in a large or small kidney. Renal hypoplasia may be simple, segmental or oligomeganephric type. Renal dysplasia may be multicystic and sometimes hypoplastic or aplastic. It can be segmental or total and may be associated with urinary tract obstruction.

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Horse-shoe Kidney

Horse-shoe kidney develops when lower poles of the kidneys are fused in the midline due to fusion of ureteric buds during fetal development. These kidneys are more vulnerable to develop Wilms' tumor than in general. The child presents with midline firm mass with associated pyuria, albuminuria and vomiting. Diagnosis is confirmed by IVP which shows a single mass with malposition of pelvis and a bridge of tissue. Surgery is indicated when uncontrolled urinary infections result in pyelonephritis.

Polycystic Kidneys

Polycystic kidney is one of the most common congenital anomalies as inherited autosomal disease. It is a complex syndrome resulting from progressive dilatation of specific portion of the nephron which may arise during development and after completion of development of the nephron due to an adverse hereditary metabolic environment.

There are two distinct types of polycystic kidney, e.g.,

i. infantile type (autosomal recessive inheritance) and
ii. adult type (autosomal dominant inheritance).

Infantile type presents with palpable bilateral nodular cystic masses with hypertension and progressive renal failure. There may be associated anomalies of liver, lungs, central nervous system (CNS) and chorionic villus sampling (CVS).

Adult type presents with anemia, polyuria, osteodystrophy, hypertension and bilateral palpable nodular renal masses with irregular distribution. This type may not present even before 40 years of age.

Diagnosis of the disease is confirmed by IVP and renal angiography. Management is purely symptomatic. Prognosis is worst in early onset.

Obstructive Lesions of the Urinary Tract

Obstructive uropathy is mainly caused by congenital abnormalities like pelviureteric junction obstruction and posterior urethral valves which may lead to irreversible renal damage. Other congenital causes of obstructive lesions are duplex renal system, neuromuscular bladder dysfunction, meatal stenosis, phimosis, etc. Acquired conditions like renal calculi, trauma, tumor, tuberculosis also may produce the obstruction.

Pelviureteric Junction Stenosis

Pelviureteric junction (PUJ) obstruction is commonly found as unilateral or bilateral stenosis of PUJ causing hydronephrosis. It can be associated with ectopic and horse-shoe kidney. PUJ stenosis may present as a flank mass without any symptoms or with urinary tract infection (UTI) and upper abdominal pain. The diagnosis is confirmed by ultrasound sonography (USG), IVP or renal scan and renal function test. Surgery is indicated for removal of obstruction and to prevent complications.

Hydronephrosis

Hydronephrosis is the dilatation of renal pelvis which may be found as unilateral or bilateral. It may be due to obstruction of urine flow in the distal urinary tract or reflux of urine up the ipsilateral ureter or due to bladder neck obstruction or urethral obstruction. This condition is more common in males than females and more frequently found in left-sided kidney.

The patient with hydronephrosis may present with urinary infections or with large abdominal mass. Other features are abdominal pain, failure to thrive, anemia, hypertension, hematuria and renal failure. Diagnosis is confirmed by USG, IVP, MCU, diuretic isotope renography, etc. Management is done by surgical removal of obstruction or pyeloplasty. Percutaneous nephrostomy or nephrectomy may be indicated in complicated cases.

Posterior Urethral Valve

Posterior urethral valves (PUVs) almost invariably occur in the male child and most frequent cause of distal urinary tract obstruction. The child present with dribbling of urine, abnormal urine stream, palpable bladder, recurrent UTIs, vomiting and failure to thrive. In severe and prolonged obstruction, renal dysplasia and anuria may develop.

Diagnosis of this condition is confirmed by MCU, USG and endoscopy. The valves are found usually at the point of junction of posterior urethra with anterior urethra. There is dilated posterior urethra and enlargement of bladder with vesicoureteral reflux (VUR).

Initially *management* is done with urinary catheterization following aseptic precautions. Definitive management is performed by transurethral destruction of the valvular leaflet by balloon catheter or endoscopic fulguration of the valves, as early as possible after confirmation of diagnosis. Other operative interventions may be indicated in some cases as temporary urinary diversion by cutaneous vesicostomy or bilateral ureterostomies or pyelostomy. Presence of associated complications should be detected and appropriate management to be arranged.

Meatal Stenosis

Meatal stenosis may occur as congenital abnormality. It may be found due to meatal ulcer and scaring, following infection or circumcision and improper hygiene. It can cause lower urinary tract obstruction. Meatal dilatation or meatoplasty may be needed to relieve the obstruction.

Exstrophy of Bladder (Ectopia Vesicae)

Exstrophy of bladder is a congenital malformation in which the lower portion of the abdominal wall and the anterior wall of the bladder are missing, so that the bladder is everted

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through the opening and may found on the lower abdomen just above the symphysis pubis with continuous passage of urine to the outside. It is also termed as 'ectopia vesicae', e.g., malposition or displacement of urinary bladder from its normal position in the pelvis. Exstrophy of bladder usually associated with numbers of congenital anomalies, related to urogenital tract, musculoskeletal system and sometimes of GI system. Male children are more commonly affected.

Clinical Manifestations

This condition is diagnosed on inspection at birth. The child is manifested with constant urinary dribbling through the defect, skin excoriation, infection and ulceration of the bladder mucosa.

The child may have ambiguous genitalia and waddling unsteady gait. The condition may be complicated with UTI and growth failure. Cystoscopic examination, X-ray, USG, IVP and urodynamic testing can be done to detect the extent of anomaly and other associated problems.

Management

Exstrophy of bladder can be managed by surgical closure of the bladder within 48 hours. Urinary diversion may be needed in some cases before reconstructive surgery. Complete correction of the malformation can be done in stages by reconstruction of the defect. Orthopedic surgery may be needed in some cases with associated musculoskeletal problems. These interventions should be done by school age.

Supportive nursing care is important before and after reconstructive surgery to prevent complications.

In preoperative period, special attention should be given for protection of bladder area from infections and trauma. These can be obtained by avoiding irritating clothing and linen over the exposed bladder, positioning the infant on back or side, humidifying the exposed bladder by covering with wet gauze, maintaining aseptic precautions and general hygienic measures along with other routine care. Preparation of parents and child for planned reconstructive surgery is important aspect of nursing care.

Postoperative care should include close monitoring of child's condition, vital signs, features of infections, intake/output, etc. Special attention should be given for care of urinary catheter, its position, drainage and aseptic precautions. The catheter may be placed urethral and/or ureteral. Instruction should be given to parents about necessary precautions for prevention of infection, dislodgement or leakage. Routine postoperative care should be provided to promote early recovery. Necessary information and demonstration must be given to the parents regarding home-based care, follow-up and next probable data of operative interventions, if needed. Teaching about signs of complications and their prevention are important.

Epispadias

Epispadias is the congenital abnormal urethral opening on the dorsal aspect of the penis. Urethra is displaced dorsally due to abnormal development of the infraumbilical wall and upper wall of the urethra. It is usually associated with exstrophy of bladder and ambiguous genitalia. Rarely, it may found in female infants.

Classification

Epispadias in male child can be classified as:

- Anterior epispadias with normal continence:
  - Glandular
  - Balanitis or penile.
- Posterior epispadias—associated with incomplete bladder neck and incontinence of urine:
  - Penopubic
  - Subsymphyseal

The *male infants* with epispadias are having short and broad penis with dorsal curvature.

In females, a cleft extends along the roof or entire urethra, involving the bladder neck. Urethra is short and patulous.

Female epispadias can be classified as:

- Bifid clitoris with no incontinence of urine
- Subsymphyseal with incontinence of urine.

In both male and female, pubic bones are usually found separated, along with epispadias. There is low incidence of associated anomalies. The condition is usually diagnosed at birth. IVP and MCU are performed to detect abnormalities of upper urinary tract, vesicoureteric reflux (VUR) and bladder capacity.

Management

Management of epispadias is done by the surgical correction usually in three stages. First stage operation is done in about 1.5 to 2 years of age for penile lengthening, elongation of urethral strip and chordee correction. Second stage operation is done at least 6 months after first stage for urethral reconstruction. Third stage operation is done about 3 to 4 years of age for bladder neck reconstruction and correction of VUR. Cystoplasty can be done to enhance the bladder capacity after 2 to 3 years of 3rd stage operation.

Supportive nursing care should emphasize on prevention of infections, emotional support for long-term management schedule and routine preoperative and postoperative management. Health maintenance and promotion of growth and development should be emphasized by balanced diet, immunization, hygienic measures and parental guidance.

Hypospadias

Hypospadias is the congenital abnormal urethral opening on the ventral aspect (under surface) of the penis. It is one of the most common malformations of male children.

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Undescended testes or inguinal hernia or upper urinary tract anomalies may be associated with hypospadias. It may found in females as urethral opening in the vagina with dribbling of urine.

Classification

Hypospadias can be classified depending upon the site of the urethral meatus.

- **Anterior hypospadias (65–70%):** It may be found as glandular or coronal or on distal penile shaft.
- **Middle (10–15%):** Penile shaft hypospadias.
- **Posterior hypospadias (20%):** It may be found on proximal penile shaft or as penoscrotal, scrotal or perineal type.

Problems Related to Hypospadias

A child with hypospadias may have following problems:

- Presence of painful downward curvature of the penis during erection as chordee.
- Due to chordee, there is deflected stream of urine and the child wets his thigh during urination.
- Inability to void urine while standing, in case of peno-scrotal, scrotal and perineal hypospadias. It also may found with the penis in the normal elevated position.
- If appropriate management is not done or left untreated, the condition, in later life, interferes during sexual intercourse with difficulty in penetration due to the presence of chordee. Severe form of hypospadias interferes with reproductive ability as the sperms are deposited outside the vagina due to proximal situation of meatus.
- There can be meatal stenosis, fistula, urethral stricture or stenosis or diverticulum.

Management

Management of hypospadias is done by surgical reconstruction to obtain straight penis at erection, to form urethral tube and urethral meatus at the tip of glans penis.

Meatotomy is done at any age after birth. Chordee correction and advancement of prepuce can be done at the age of 2 to 3 years. Urethroplasty is done 3 to 4 months after chordee correction. The surgical repair should be completed before admission to the school. Operation can be performed as multistage or single stage repair.

Phimosis and Paraphimosis

Phimosis

Phimosis refers to the narrow opening of the prepuce that prevents it being drawn back over the glans penis. The inability to retract the prepuce after the age of 3 years, should be considered as true phimosis. Forcible retraction of the prepuce should be avoided as it may tear and can cause scarring and persistent phimosis.

Phimosis can be congenital or may be acquired due to inflammation of glans or prepuce. Phimosis can predispose UTI. Children with phimosis and recurrent UTI should be evaluated for obstruction of urinary tract or vesicoureteral reflux (VUR).

Circumcision, e.g., excision of the foreskin of the glans penis, is the choice of operative intervention done to treat phimosis. Other measure is the use of betamethasone cream to the narrowed preputial skin for two times daily for 4 weeks. This treatment usually becomes successful as the foreskin becomes soft, elastic and can be retracted gently and gradually.

Paraphimosis

Paraphimosis may develop in phimotic child whom also need for surgical management by circumcision or reduction with application of lubricant under deep sedation. Paraphimosis is the retraction of a phimotic foreskin, behind coronal sulcus. It cannot be reduced spontaneously. It results in edematous swelling due to venous stasis causing severe pain. It needs urgent interventions.

Postoperative care after circumcision should include control of bleeding from operated site and prevention of infections. This operation is done as day-care surgery and does not require hospitalization. Parents should be explained about the home-based care and features of complications.

WILMS' TUMOR (NEPHROBLASTOMA)

Max Wilms, German surgeon described this most common renal tumor of childhood. It is associated with chromosomal deletions, especially from chromosomes 11 and 16. Wilms' tumor is a rapidly developing highly malignant embryonal tumor usually diagnosed within 3 years of age. It is generally unilateral and can be familial in some cases. It may be associated with other congenital anomalies, like hemihypertrophy of the vertebrae, genitourinary anomalies, aniridia, ambiguous genitalia, etc.

This tumor develops within the kidney parenchyma, distorting it and invading the surrounding tissues. The tumor tends to grow in a concentric fashion invading the adjacent renal parenchyma. Characteristically a well-developed capsule is present. Hemorrhage, necrosis and calcification may occur rarely. Metastatic spread occurs by lymphatics to the renal hilar, periaortic and pericaval lymph nodes. Distant blood-borne metastasis occurs most frequently in the lungs, bone and liver.

Clinical Manifestations

Majority of affected children present with an asymptomatic abdominal mass, detected during routine examination or by the mother during daily bath or undressing the child. Increasing abdominal girth is important finding.

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Microscopic hematuria, pain abdomen, fever, pallor, hypertension and superficial venous engorgement may present. Gross hematuria indicates advanced Stage III or IV tumor. On examination large, smooth, fixed, firmed, nontender palpable mass detected in the flank.

Clinical Staging of Wilms' Tumor

This staging helps to determine prognosis and survival of the patients.

- **Stage I:** Tumor limited to kidney and can be fully excised. Renal capsule is intact. Tumor not ruptured and no residual tumor after excision.
- **Stage II:** Tumor extends beyond kidney but can be completely excised. There is regional extension of the tumor by penetration through renal capsule. Vessels outside kidney are either infiltrated or contain tumor thrombus. No residual tumor is apparent beyond the margins of resection.
- **Stage III:** Residual nonhematogenous extension of the tumor confined to the abdomen following surgery. Involvement of hilar, periaortic or other lymph nodes may present. The tumor cannot be completely resectable because of local infiltration into the vital structure.
- **Stage IV:** Hematogenous metastasis to distant organs, e.g., lungs, liver, brain, etc.
- **Stage V:** Bilateral renal involvement which occurs in 5 to 8% of the cases.

Diagnostic Evaluation

Clinical suspicion should be supported by history of illness and physical examination. The diagnosis can be confirmed by X-ray abdomen and chest, IVP, USG, CT scan, MRI, renal function test and urine analysis. Liver function test, bone marrow study and other diagnostic measures help to detect the metastatic spread.

Management

Management of Wilms' tumor depends upon histological findings, clinical staging and metastasis.

Stages I and II Wilms' tumors with favorable histology are usually managed with nephrectomy and chemotherapy for 18 weeks. Stages III, IV and V tumors are treated with nephrectomy, abdominal radiotherapy and chemotherapy for 24 weeks. Preoperative radiotherapy and chemotherapy are indicated in massive tumors. Chemotherapy is usually administered with vincristine, dactinomycin, actinomycin, adriamycin, doxorubicin and cyclophosphamide. Radiotherapy is not given in children below 1 year of age.

Nursing management should include special care during nephrectomy, radiotherapy and chemotherapy. Reducing anxiety of the parents, by explanation, involvement in childcare and teaching long-term home-based care are important nursing interventions to help the family to cope with the situation.

Prognosis

Prognosis of Wilms' tumor depends upon clinical staging, age of the child, size of the mass and histological findings. The disease-free survival rates are in Stage I—95%, Stages II and III—85%, Stage IV—70 to 80% and with unfavorable histology 25 to 50%. Recurrence of tumor carries bad prognosis.

NURSING MANAGEMENT OF THE CHILD WITH UROLOGIC SURGERY

Basic preoperative and postoperative care to be provided with special attention for the following:

- Promoting understanding of the parents about the planned surgical interventions by explanation according to level of understanding.
- Preparing for necessary diagnostic procedures.
- Involving the parents in childcare.
- Promoting normal urinary functions by monitoring intake and output, care of urinary catheter and drainage tube, (if present), encouraging adequate fluid intake and hygienic measures and observing signs of urinary infections.
- Preventing infections by aseptic precautions, hand washing practices, taking care of wound, administering prescribed antibiotics and other medications, IV fluid therapy, monitoring features of infections and vital signs, etc.
- Providing comfort by rest, sleep, comfortable positioning of operated side with support, administering analgesics, antispasmodics and organizing diversions and recreations by play and toys.
- Providing adequate nutrition to promote healing and prevent infections along with adequate fluid to promote urinary functions.
- Teaching the parents about related care, prevention of infections and complications, importance of fluid intake, diet, follow-up, available support services and facilities.

ACUTE GLOMERULONEPHRITIS

Acute glomerulonephritis (AGN) is an immune-mediated inflammatory disease of the capillary loops in the renal glomeruli. The antigen-antibody complex deposition within the glomeruli results in glomerular injury which is manifested as hematuria, oliguria, edema and hypertension. Onset of the disease process is relatively abrupt and commonly seen in preschool or in early school age group of male children.

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Etiology

There is initial infection of upper respiratory tract (throat) or skin, usually 1 to 3 weeks before the onset of symptoms. The most frequent causative microorganism is nephritogenic strains of group-A, beta-*Streptococcus hemolyticus* (type-12). Acute poststreptococcal glomerulonephritis (APSGN) is the most common.

Pathophysiology

Streptococcal infection produces an antigen antibody complex which trapped in the glomerular loops and causes inflammatory reaction due to activation of complement. There is proliferation and swelling of the endothelial cells. The changes in the glomerular capillaries diminish the amount of the glomerular filtrate and allow the passage of blood cells and protein into the filtrate. Sodium and water are retained. The damage of glomerular basement membrane may lead to progressive renal failure.

Clinical Manifestations

The clinical presentations may be found in the mild or severe form with rapid onset of symptoms. History of sore throat or pyoderma or scabies or impetigo usually presents in most of the patients.

The child may present with decreased urine output with presence of blood or brown color urine. Edema is manifested as periorbital puffiness, found in the morning. Some patients may have pedal edema and generalized edema. Rapid weight gain usually found due to presence of massive edema. The child may have fever, headache, nausea, vomiting, anorexia, abdominal pain and malaise. Hypertension may present in more than 50% cases with sudden onset and may appear during 4 to 5 days of illness.

Clinical features of AGN may present in two phases. In Phase-I, child presents with edema and oliguria which persists for 5 to 10 days. In Phase-II, edema reduces and urinary output increases.

Complications

AGN can be complicated with congestive cardiac failure (CCF), acute renal failure, hypertensive encephalopathy, persistent hypertension, anemia, growth failure and chronic glomerulonephritis.

Diagnostic Evaluation

History of illness and physical examination help in clinical diagnosis. The confirmation of diagnosis is done by the followings:

Urine Examination

It shows increased specific gravity, smoky dirty brown color urine with reduced total amount in 24 hours. Mild to moderate or severe albuminuria is detected. Microscopic examination reveals presence of red cells, WBCs, pus cells, epithelial cells and granular cast.

Blood Examination

Blood examination demonstrates increased level of urea, creatinine, erythrocyte sedimentation rate (ESR), ASO titer and anti-DNAse 'B'. There is decreased level of Hb%, serum complement and albumin in blood. Hyponatremia and hyperkalemia may occur in persistent oliguria.

Throat Swab Culture

Throat swab culture may show presence of beta-hemolyticus *Streptococcus* in some children.

Chest X-ray

It may show pulmonary congestion.

Management

AGN with impaired renal function as severe oliguria and azotemia needs hospitalization for special attention. Mild oliguric patients with normal blood pressure can be managed at home with OPD-based treatment.

The *management* of AGN should include the followings:

- Bed rest for few weeks to be provided, till urine is free from RBC.
- Diet should be arranged with restriction of protein, salt and fluid intake, till oliguria and increased blood urea level persist. Carbohydrate containing foods need to be allowed freely. Overhydration and sodium retention may increase blood pressure and lead to left ventricular failure. Fluid intake should be allowed in a calculated amount (e.g., total amount of previous day urine output in 24 hours plus insensible losses to be allowed to drink on that day). Daily weight recording is important to assess the increase and decrease of edema.
- Administration of antibiotics (preferably penicillin) is needed for 7 to 10 days to treat pharyngitis or pyoderma (if present). Other symptomatic management includes antihypertensive drugs (nifedipine, atenolol) to control blood pressure and its consequences. Sedative-tranquilizer (diazepam) may be needed in case of hypertension, convulsions and encephalopathy. Diuretic is not usually indicated except in association with pulmonary edema and left ventricular failure.
- Dialysis may be needed in renal failure and severe electrolyte imbalance.
- Management of complications like CCF, hypertensive encephalopathy, etc., should be done promptly to prevent life-threatening outcome. Dopamine infusion, steroid therapy and respiratory support may be required for some patients.

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Nursing Management

Nursing Assessment

Nursing assessment should include special attention for history of sore throat or skin infection along with other history of illness. Physical assessment should be done with emphasize on blood pressure and daily body weight recording, calculation of fluid intake and urine output, assessment of edema, skin conditions and urine testing along with other investigations.

Nursing Diagnoses

Important nursing diagnoses in case of AGN include the followings:

- Impaired urinary elimination related to glomerular dysfunction.
- Fluid volume excess related to altered renal function.
- Activity intolerance related to edema.
- Altered skin integrity related to edema.
- Altered nutrition, less than body requirement, related to albuminuria and GI disturbances.
- Fear and anxiety related to disease processes.
- Knowledge deficit regarding care of the child with renal disease and continuation of care at home.

Nursing Interventions

- Monitoring urinary pattern by maintaining intake/output and testing urine at regular interval.
- Reducing excess fluid volume by restriction of salt and fluid intake. Recording of daily weight should be done to assess the level of fluid retention. Everyday there should be approximate loss of 0.5% body weight. Gain in weight indicates increased edema and fluid retention. Diuretics to be given as prescribed. Antihypertensive drugs need to be administered after checking of blood pressure.
- Administering antibiotics other drugs, as prescribed.
- Promoting rest, sleep and comfortable position with gentle approach during nursing procedures. Allowing self-care activities to the children is important when acute stage is over and as per capability of the child.
- Providing skin care with special attention to the edematous part by preventing drying, scratching and injury. Maintaining cleanliness and general hygienic measures must be emphasized.
- Promoting intake of nutritious diet by allowing carbohydrate foods with restriction of protein and salt in oliguric phase. Normal protein intake can be allowed when urine output becomes normal. Arranging small frequent easily digestible diet considering child's likes and dislikes. Spicy food to be avoided.
- Providing play material for diversion and recreation especially when acute stage is over. Allow mother to involve in childcare.
- Providing emotional support to parents and family members.
- Teaching the parents about continuation of care and follow-up.

CHRONIC GLOMERULONEPHRITIS

Chronic glomerulonephritis is an advanced, irreversible impairment of renal function with or without symptoms. It may develop as a primary glomerular disease or may occur in SLE, drug-induced nephropathies and polyarteritis nodosa.

Pathological changes are found as diffuse thickening of glomerular basement membrane or focal segmental glomerulosclerosis with variable deposition of immunoglobin, complement and fibrin. Glomerular filtration rate is reduced. Mesangial proliferation may occur. In late stages, most glomeruli become sclerosed with tubular, interstitial and vascular changes.

Clinical Manifestations

The condition may remain asymptomatic and may be diagnosed during routine urine examination. The symptomatic patients present with edema, severe hypertension, hematuria, nocturia and persistent anemia. Bone pain, bony deformities and failure to thrive are usually present.

Diagnosis is confirmed by urine and blood examination. Urine analysis shows presence of protein, RBC and cast. Blood examination shows increased urea and creatinine. USG is helpful to diagnose the condition.

Management

Management of chronic glomerulonephritis can be done with steroid and other immunosuppressive drugs as there is no specific treatment for the condition. Antihypertensive drugs and antibiotics are useful as symptomatic measures. The condition may be complicated with chronic renal failure that should be treated accordingly. Symptomatic and supportive care promotes the prognosis.

URINARY TRACT INFECTIONS

Urinary tract infections (UTIs) are the presence of infective agents (usually bacteria) that exists anywhere between the renal cortex and the urethral meatus. It is sometimes difficult to determine the exact location of the infections. It may cause irreversible renal damage, especially in association with urinary tract anomalies and vesicoureteral reflux (VUR).

The most common causative organism is *E. coli*. Other organisms are *Streptococcus, Staphylococcus, Pseudomonas*, etc. The most common route of entry is the ascending

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infections from the urethra or maybe hematogenous spread. This infection is more common is girls.

The contributing causes of UTI are urinary stasis, congenital anomalies or obstruction of urinary tract (PUJ, bladder-neck obstruction, PUV), VUR, poor perineal hygiene, short female urethra, urinary catheterization, or instrumentation, local inflammation, chronic constipation or infection anywhere in the body.

Pathophysiology

Most commonly UTI are found in lower urinary tract. It may occur as cystitis or pyelonephritis. Inflammation results in urinary retention and stasis. Backflow of urine (reflux) into the kidney may occur though ureter. Inflamed part become edematous, thickened and fibrosed. Ureter and renal pelvis may be dilated. If left untreated, kidney may become small and tissue may be destroyed resulting disturbances of renal functions.

Clinical Manifestations

The onset of symptoms may be abrupt or gradual, even the condition may have no symptoms. The patient usually have moderate to high fever with chills, rigors, convulsions, anorexia, malaise, irritability and vomiting. Urinary symptoms may be found as frequency and urgency, dysuria, dribbling and bed wetting. Urine may be foul-smelling. The child may complain abdominal or flank or suprapubic pain. It may present as acute abdomen. Neonates with UTI may have lethargy, diarrhea, jaundice, poor weight gain and features of sepsis.

Diagnosis

The diagnosis of UTI is confirmed by urine culture and urine analysis (routine and microscopic) by clean catch or sterile urine specimen. Associated congenital anomalies or obstructive uropathies should be detected by USG, MCU, IVP, renal scan, etc. Blood examination shows increased total leukocyte count (TLC) and ESR with decreased Hb%.

Management

Management of UTI is mainly done with antibiotic therapy. Specific symptomatic and supportive treatment is essential. Associated congenital anomalies should be detected early for prompt management.

The antibiotic therapy is given with combination of ampicillin and gentamicin or amikacin for 7 to 10 days. Other antibiotics also can be used, e.g., co-trimoxazole, ceftriaxone, cefotaxime, cephalexin, nitrofurantoin, etc. Supportive measures include management of fever by rest, tepid sponge and/or antipyretics with large amount of fluid intake. Improvement of personal hygiene (perineal hygiene) is important preventive measure. Frequent emptying of bladder should be encouraged to prevent stasis of urine.

Repeat urine culture to be done to discontinue the treatment. The UTI may be complicated with recurrent infections, renal damage and severe VUR.

NEPHROTIC SYNDROME

Nephrotic syndrome is one of the common cause of hospitalization among children. Incidence of the condition is 2 to 7 per 1000 children. It is more common in male child. Mean age of occurrence is 2 to 5 years.

Nephrotic syndrome is a symptom complex manifested by massive edema, hypoalbuminemia, marked albuminuria and hyperlipidemia (hypercholesterolemia). It can be classified as congenital, idiopathic or primary and secondary types.

Types

Congenital Nephrotic Syndrome

It is rare but a serious and fatal problem usually associated with other congenital anomalies of kidney. It is inherited as autosomal recessive disease. Severe renal insufficiency and urinary infections along with this condition result is poor prognosis.

Primary or Idiopathic Nephrotic Syndrome

It is the most common type (about 90%) and regarded as autoimmune phenomenon, as it responds to immunosuppressive therapy. The subgroups of this type are: (a) minimal change nephrotic syndrome (85%), (b) mesangial proliferative nephrotic syndrome (5%), and (c) focal sclerosis nephrotic syndrome (10%).

Secondary Nephrotic Syndrome

It occurs in children about 10% of all cases. This condition may occur due to some form of chronic glomerulonephritis, or due to diabetes mellitus, systemic lupus erythematosus (SLE), malaria, malignant hypertension, hepatitis B, infective endocarditis, HIV/AIDS, drug toxicity, lymphomas, syphilis, etc.

Pathophysiology

The pathological changes in nephrotic syndrome may be due to loss of charge selectivity and thickening of the foot plate of the glomerular basement membrane. These result in increased glomerular permeability, which permits the negatively charged protein, mainly albumin to pass through the capillary walls into the urine. Excess loss of albumin results in decrease in serum albumin (hypoalbuminemia). Heavy proteinuria more than 1 g/m^2 body surface area per day leads to serum albumin level below 2.5 g/dL. As a result of hypoalbuminemia, there is reduction in plasma oncotic pressure. Thus fluid flows from the capillaries into the interstitial spaces and produced edema.

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The shift of fluid from the plasma to the interstitial spaces reduces the intravascular fluid volume resulting hypovolemia, which stimulates the renin-angiotensin axis and volume receptors to secrete aldosterone and antidiuretic hormone.

These lead to increased reabsorption of sodium and water in distal tubules resulting edema.

Loss of protein and immunoglobins predisposes infections in the child. Diminished oncotic pressure leads to increased hepatic lipoprotein synthesis, which results in hyperlipidemia. low-density lipoprotein (LDL) and cholesterol level increased initially, then very low-density lipoprotein (VLDL) and triglyceride are also elevated.

Clinical Manifestations

The onset of the disease is usually gradual or it may be acute. The child may present with periorbital puffiness. Edema may be minimal or massive. Profound weight gain within a short period of days or week is found. Dependent edema develops in the ankle, feet, genitalia (scrotum) and hands. The edematous part is soft and pits easily on pressure. Striae may appear on the skin due to overstretching by edema. Fluid accumulates in the body space, resulting ascites, pleural effusion with respiratory distress and generalized edema (anasarca) (**Fig. 18.1**). Urine output is reduced in the edematous phase. Urine becomes concentrated with frothy appearance.

GI disturbances usually found as vomiting, loss of appetite and diarrhea. Other features include fatigue, lethargy, pallor and irritability. Hypertension, hematuria, hepatomegaly and wasting of muscle may found in some cases.



Fig. 18.1: A child with nephrotic syndrome (body weight 15 kg, age 3 years).

Complications

A child with nephrotic syndrome may be complicated with ascites, pleural effusion, generalized edema, coagulation disorders, thrombosis, recurrent infections of different systems and renal failure. Other complications are growth retardation, calcium and vitamin D deficiency, protein-energy malnutrition and relapse of nephrotic syndrome.

Diagnostic Evaluation

History of illness and physical examination is significant to exclude clinical features help to diagnose the condition clinically. *Laboratory investigations* to confirm the diagnosis may include the followings:

- Urine examination shows gross proteinuria (2–20 g/day), presence of cast, slight hematuria and increased specific gravity.
- Blood examination demonstrates reduced total protein, albumin <2.5 g/dL and cholesterol >200 mg/dL. Lipoproteins and BUN (blood urea nitrogen) are increased. Serum albumin and globulin ratio is reversed. There is hypogammaglobulinemia, hypomagnesemia and low-creatinine level.
- Renal biopsy is indicated in case of poor response to steroid therapy.
- Other investigations show low ASO titer and IgM, raised IgG and IgE, serum complement (C_3 and C_4) is normal.

Management

- *Bed rest* and *high-protein diet* with restriction of fluid intake are important aspects of management.
- *Steroid therapy* with oral prednisolone is the most significant aspect of management of nephrotic syndrome. It is given 2 mg/kg/day in 2 to 3 divided doses for at least 4 to 6 weeks and then gradually tapered off or abruptly stopped, after another 4 to 6 weeks. Antacid is given along with prednisolone to prevent gastric complications.
- *Antibiotic therapy* is indicated in the presence of any infection. Prophylactic use of antibiotic is not recommended.
- *Diuretics* are prescribed in the presence of severe edema and massive ascites. Frusemide 1 to 3 mg/kg/day in 2 divided doses alone or with spironolactone 2 to 3 mg/kg/day in 2 divided doses is given. Rapid fluid loss should not be attempted in 8 to 12 hours. Potassium supplementation must be given along with diuretics.
- *Albumin infusion* (1 g/kg/day) may be given in case of massive edema and ascites. It helps to shift the fluid from interstitial space into the vascular system. Diuretic

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therapy is given in combination of albumin infusion. Plasma or blood transfusion may be given is some cases to treat hypoalbuminemia.

- *Immunosuppressive drugs* (levamisole, methotrexate, cyclophosphamide, cyclosporine, chlorambucil) may be administered along with prednisolone in case of frequent (4 or more per year) relapses and in steroid dependent cases.
- *Renal transplantation* is indicated in end-stage renal failure due to steroid resistant glomerulosclerosis (focal and segmental).
- Nursing management (*see* the nursing process below).

Prognosis

In early diagnosis and appropriate treatment with steroid therapy and supportive care, about 80% children with nephrotic syndrome, recover. About 10 to 15% children become complicated with chronic renal failure, and only 2 to 4% of cases may have fatal outcome due to infections and coagulation disorders.

Sample nursing process of a child with nephrotic syndrome is given below.

Ramu, a 4-year-old boy, admitted in pediatric medical unit with edema all over the body, less urine output, loss of appetite and lethargy (*see* below for details).

ACUTE RENAL FAILURE

Acute renal failure (ARF) is a severe deterioration of renal function, manifested as sudden reduction of urine excretion (<1 mL/kg/h) as oliguria or anuria leading to fluid and electrolyte imbalance and accumulation of metabolic nitrogenous wastes and other biochemical products. It is a life-threatening situation.

Etiology

The causes of ARF can be divided into three groups—(i) prerenal, (ii) intrarenal, and (iii) postrenal causes.

Prerenal Causes

These conditions are related to the problems occur in blood supply to the kidney either due to systemic hypovolemia or due to renal hypoperfusion. The important causes are hypovolemia due to diarrheal dehydration, shock, burns, diabetic acidosis, trauma hemorrhage, etc., and CCF.

Intrarenal Causes

These intrinsic renal conditions are related to problems within the kidneys and their functions, causing reduction of GFR, renal ischemia and tubular damage. The important causes are glomerulonephritis, hemolytic uremic syndrome (HUS), renal vein thrombosis, acute tubular necrosis, (due to fluid loss, hemorrhage, shock, nephrotoxic drugs and toxins), DIC, sepsis, interstitial nephritis (due to infections, drugs), etc.

Postrenal Causes

These conditions are related to the problems of the ureter or lower urinary system causing obstruction of urine flow due to obstructive uropathies. The causes include renal calculus, PUV, bladder-neck obstruction, congenital lesion or pus collection in urinary tract or following drug therapy (sulfonamide).

Phases of ARF

Four phases of ARF are identified in children depending upon the course of illness.

Nursing process of a child with nephrotic syndrome

- Identification data — Date and Time
- History of illness in details
- Findings of physical examination and investigations

Assessment day-1	*Nursing diagnosis*	*Planning*	*Implementation*	*Evaluation day-2*
• Edema around eyes, feet and genitalia • Urine output in 24 hours—200 mL • Body weight—17 kg • Fluid intake—250 mL in 24 hours	Fluid volume excess related to fluid accumulation in tissue	To reduce excess amount of fluid accumulated in tissues	• Providing rest, comfortable position and frequent change of position • Allowing diet with low salt and high protein (egg, fish, pulse) • Administering prescribed medications (prednisolone, lasix) orally • Offering potassium containing food (orange juice, banana) • Restricting fluid intake • Maintaining intake-output and body weight chart • Explaining and reassuring about the treatment plan and involving mother in childcare with necessary instruction • Urine testing for albumin	• Edema reduced slightly • Body weight 18 kg • Urine output 400 mL in 24 hours • Fluid intake 600 mL in 24 hours

Contd...

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Contd...

Assessment day-1	*Nursing diagnosis*	*Planning*	*Implementation*	*Evaluation day-2*
• Edematous skin • Protein loss in urine (+ +)	Risk for infection	To protect the child from infection	• Examining the child for any signs of infections and recording TPR • Monitoring blood count (TLC, DLC) • Providing skin care, keeping the skin dry and applying body powder for soothing • Keeping the nails short • Maintaining general cleanliness and hygienic measures • Preventing any injury of edematous skin • Avoiding the contact of two edematous skin (thigh) surfaces to prevent friction • Avoiding invasive procedure as possible • Teaching the mother about skin care and signs of infections and involving the mother during care of the child	• No signs of infection • No skin breakdown • Hygiene maintained • TPR normal
• Loss of appetite • Proteinuria • Lethargy	Altered nutrition less than body requirement	To improve nutritional status	• Providing small frequent feeding with protein and carbohydrate, considering dietary restriction and child's likes and dislikes • Providing nutritional supplementation as needed • Encouraging child to take food	Dietary intake increased
• Anxiety of the parents • Fear of the child	Altered family process	To provide emotional support	• Allowing parental involvement in childcare • Allowing play and self-care as tolerated by the child • Encouraging interaction with other child having chronic illness • Answering the questions asked by the parents and allowing to express frustration	Parents and child express their concern about the illness
Inability to take care of the child by the parents	Knowledge deficit	To improve knowledge about childcare by health teaching	• Discussing about the care after discharge from hospital, regarding rest, diet, hygiene, continuation of medication, need for medical help and follow-up • Teaching about features of infections, signs of relapse and precautions to prevent complications	Mother listen the information and ensure to follow them

Initiating Phase

This phase lasts from hours to days with features of renal function impairment.

Oliguric Phase

This phase lasts from 5 to 15 days but can be prolonged for weeks. It is shorter in infants and children (3–5 days) and longer in older children (10–14 days). More than 3 weeks duration of oliguric phase indicates irreversible renal damage. It depends upon severity and duration of initial stage causing acute vasospastic nephropathy.

Diuretic Phase

This phase lasts for few days and highly variable with mild to severe clinical features.

Recovery Phase

This phase marks the final resumption of normal urine osmolarity, constituents and biochemical alterations in the blood.

Pathophysiology

The cause of renal failure may be different but the end result is renal hypoperfusion and renal outflow obstruction

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which leads to impairment of renal functions and renal failure.

There is marked reduction of glomerular filtration rate and renal blood flow due to renal vasoconstriction resulting sodium and fluid retention which leads to edema.

Hypertension may develop due to renin-angiotensin mechanism caused by arteriolar constriction, increased circulatory overload and sodium retention.

Metabolic acidosis may develop as a result of failure of excretion of hydrogen ions due to reduced functions of nephron and hypercatabolic states, which lead to increased production of acids.

Hyperkalemia may develop due to disturbed sodium and potassium exchange in the distal tubules and increased extracellular potassium level resulting from damage tissue cells, RBCs and hypercatabolic states.

Clinical Manifestations

Clinical presentations of ARF depend upon the underlying cause and duration of the problem. The child may present with severe oliguria or anuria. In some cases, urine output may not be reduced. The child may look remarkably well or extremely sick. These features of underlying cause are usually prominent. Other signs and symptoms include nausea, vomiting, lethargy, dehydration, acidotic breathing, alteration of level of consciousness, irregularities in cardiac rate and rhythm and edema.

Diagnostic Evaluation

The following investigations may help to confirm the diagnosis along with history of illness and clinical findings:

- Blood examination shows raised serum creatinine level as important criteria for diagnosis. Blood urea, electrolytes, pH, bicarbonate and complete blood count are also useful.
- Urine examination reveals proteinuria, hematuria and presence of casts.
- Ultrasonography helps to detect the structural abnormalities, calculi, etc.
- IVP can be done to detect acute tubular necrosis.
- Radionuclide studies can be done to evaluate GFR and renal blood flow distribution.
- Specific investigations to be performed to determine the suspected underlying cause and complications related to ARF.

Management

Management of ARF should be planned on the basis of underlying cause. Correction of dehydration, treatment of shock and hyperkalemia are the immediate concern.

Fluid and electrolyte balance to be maintained promptly. Fluid requirement to be calculated on the basis of previous day urine output in 24 hours and insensible losses (approximately 300 mL/m^2) with other losses (vomiting, loose motion, etc.). Excessive fluid intake is risky. Fluid can be given orally or parenterally depending upon the condition of the patient. About 5% dextrose is given and no potassium is usually administered or very little can be given depending upon the blood K^+ level.

Diet should be planned with low sodium, low potassium, low phosphate and moderate protein (0.6 to 1 g/kg). The recommended calorie requirement is 50 to 60 cal/kg. Liberal amount of carbohydrates and fats can be given along with vitamin and mineral supplementation.

Use of diuretics like mannitol and furosemide is recommended by some authority. Steroids can also be used. In case of presence of complications like anemia, hypertension and CCF, these should be adequately controlled and managed.

Continuous monitoring of patient's condition and identification of complications are important. Complications like fluid-electrolyte imbalance, hyperkalemia, metabolic acidosis, hyponatremia, convulsions, etc., should be detected early for prompt management to prevent fatal outcome.

Dialysis (peritoneal or hemodialysis) is indicated in life-threatening complications and should be arranged in following conditions:

- Persistent hyperkalemia, serum potassium level more than 7 mEq/L.
- Congestive cardiac failure.
- Pulmonary edema.
- Severe acidosis, pH <7.2, TCO_2 <10 to 12 mEq/L.
- Neurological problems due to uremia and hyponatremia.
- Hyperphosphatemia.

Supportive care during dialysis and pre- or postdialysis period are very important. The patient with ARF should be managed by intensive care approach with specialized nursing interventions.

Nursing Management

Nursing Assessment

Assessment should include details history of illness, recent and past illness or injury, and drug therapy or allergy or exposure to toxic substances. Thorough physical examination should be done especially hydration status, cardiorespiratory monitoring, daily weight recording and strict monitoring of intake and output. Necessary laboratory investigations should be arranged to assess the underlying problems.

Nursing Diagnoses

The important nursing diagnoses are:

- Risk of fluid-electrolyte imbalance related to impaired renal functions.
- Risk for infection related to alteration of host defense.

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- Activity intolerance related to acute illness.
- Altered thought process related to CNS problem.
- Altered nutrition less than body requirement related to GI disturbances.
- Fear and anxiety related to life-threatening illness.
- Knowledge deficit related to management of acute renal failure and its consequences.

Nursing Interventions

- Maintaining fluid and electrolyte balance with prescribed IV infusion and maintaining intake and output chart. Monitoring serum electrolytes and features of heart failure are very significant.
- Administering prescribed medications with necessary precautions.
- Providing diet with high carbohydrate food, low potassium and low sodium in frequent small amount feeding.
- Providing rest, comfort, change of position, skin care, care of urine drainage (urinary drainage using condom may be given to the male child) and other hygienic care.
- Preventing infections by aseptic precautions, avoiding urinary catheterization and teaching the parents about infection control measures. Prophylactic antibiotics are not recommended. Presence of infection to be detected early by continuous monitoring and should be treated accordingly.
- Preventing injury during convulsions and assessing neurological status.
- Providing care for dialysis (*see* page 369).
- Providing emotional support to the parents, child and family members.
- Teaching about care of the child after discharge at home especially about diet, prevention of infection and follow-up.

Prognosis

Mortality rate of ARF is about 20 to 40% which is influenced by the cause and duration of renal failure with severity of pathological changes. Poor prognosis is related to associated sepsis, hemolytic uremic syndrome (HUS), prolonged anemia, cardiac failure, hepatic failure and respiratory failure with delayed initiation of treatment.

CHRONIC RENAL FAILURE

Chronic renal failure (CRF) is a permanent irreversible destruction of nephron leading to severe deterioration of renal function, finally resulting to end-stage renal disease (ESRD).

Etiology

The common causes of chronic renal failure below 5 years of age are the congenital renal anomalies and urinary tract malformations. The most common causes in 5 to 15 years age group include glomerular diseases, hereditary renal disorders and renal vascular diseases.

The important causes of CRF can be listed as follows:

- **Glomerular diseases:** Glomerulonephritis, SLE, HUS, familial nephropathy, Henoch-Schönlein purpura, amyloidosis.
- **Congenital anomalies:** Bilateral renal hypoplasia or dysplasia, polycystic kidney.
- **Obstructive uropathy:** PUJ, renal stones, PUV.
- **Miscellaneous:** Bilateral Wilms' tumor, renal vein thrombosis, renal cortical necrosis, renal tuberculosis, reflux nephropathy.

Pathophysiology

Reduction in renal functions leads to metabolic, endocrinal and hematological disturbances including disturbances of internal homeostatic equilibrium.

Damage of nephron results in hypertrophy and hyperplasia of remaining nephron leads to reduced functions of nephrons to excrete effectively thus resulting azotemia and clinical uremia.

Impaired renal function leads to hyperphosphatemia resulting disturbance of calcium absorption (hypocalcemia) due to inability to vitamin D synthesis. These disturbances produce renal ricket and renal osteodystrophy.

Anemia develops due to disturbance of erythropoietin synthesis. Mild hemolysis and GI bleed also contribute to produce anemia. Excretion of nitrogenous wastes through sweat causes pruritus. Fluid overload leads to edema and hypertension.

Severity of CRF is indicated by GFR. The lower the GFR, the greater the loss of renal function. End-stage renal disease may develop by decline of renal function to an extent that life cannot be continued without dialysis or renal transplantation.

Clinical Manifestations

The clinical features of CRF are variable and not found in chronological sequence. The child may present with initial polyuria, or frequent passage of urine, later oliguria, or anuria develops. Other clinical presentation include increased thirst, decreased appetite, weakness, low energy level, bone pain or joint pain, dryness and itching of skin. The child may have progressive anemia, hypertension and growth retardation.

In late stage, the child manifested with acidotic breathing, hiccough, nausea, vomiting, diarrhea, peripheral neuropathy and convulsions. Purpura, cardiomyopathy, pericarditis and superadded infections may found. The patient may present with unconsciousness or complications like CCF, pulmonary edema, hypernatremia, hyperkalemia and intercurrent infections.

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Complications

The complications of CRF include azotemia, metabolic acidosis, electrolyte imbalance, CCF, hypertension, severe anemia, growth retardation and delayed or absent sexual maturation.

Diagnostic Evaluation

The exact cause of CRF should be detected by detail history of illness, clinical examination and laboratory or radiological investigations.

- Blood examination shows decreased level of hematocrit, Hb%. Na^+, Ca^{++}, HCO^- and increased level of K^+ and phosphorus.
- Renal function test shows gradual increase of BUN, uric acid and creatinine values.
- Urinalysis shows variation in specific gravity increased creatinine level in urine and change in total amount of urine output.
- X-ray chest, hands, knees, pelvis, spine, etc., help to detect bony involvement.
- ECG, IVP, MCU, radionuclide imaging help to detect the extent of complications.

Management

At the initial stage, the management of CRF is planned to retard the progression of the disease by rest, diet, supportive care and symptomatic relief. Later, the treatment of complications, dialysis and renal transplantation can be provided as per need.

Diet should be planned with special attention on maintenance of calorie as per normal requirements. Diet should contain high polyunsaturated fat and complex carbohydrates. Protein intake should be adequate (0.8–1 g/kg/day) with food items of high biologic value (egg, milk, meat, fish).

Sodium intake needs to be allowed depending upon the level of impairment of sodium reabsorption, presence of edema, hypertension and azotemia. Potassium balance must be maintained by avoiding potassium containing food. Dairy milk containing high phosphate need to be avoided but calcium supplementation is required. Vitamin B_1, B_2, folic acid, B_6 and B_{12} supplementation should be given. Water restriction is usually not essential except in ESRD and fluid overload.

Correction of acidosis should be done with sodium bicarbonate. Hypertension must be managed with antihypertensive drugs. Infection should be managed with least toxic antibiotics. Antihistamines is given to relief from pruritus.

Correction of anemia can be done with iron-folic acid supplementation, blood transfusion or by human erythropoietin or synthetic erythropoietin. Renal osteodystrophy is managed with low phosphate in diet, antacid therapy (calcium carbonate) and vitamin D supplementation. Correction of calcium and phosphorus imbalance is essential. Growth hormone may be needed to correct growth retardation.

Dialysis (peritoneal or hemodialysis) and renal transplantation are indicated in ESRD.

Nursing Management

Chronic renal failure leads to multisystem physiologic crisis. Thorough assessment of all systems are essential to detect the problems and for planning of care. All the nursing diagnoses may be applicable during nursing interventions. Special care should be provided in relation to renal transplant, and dialysis. Routine care should emphasize on maintenance of fluid-electrolyte balance, skin integrity, nutritious diet, ensuring safety from infections and injury, assisting to cope with long-term illness and teaching for continuation of care.

CARE OF THE CHILD WHO UNDERGOES DIALYSIS

Dialysis is the diffusion of solute molecules through a semipermeable membrane, passing from higher concentration to that of lower concentration.

The *purpose of dialysis* is to remove endogenous and exogenous toxins and to maintain fluid-electrolyte and acid-base balance till the renal function recovers. It is a substitute for some excretory functions of kidneys but does not replace the endocrine and metabolic functions.

The *indications of dialysis* in renal failure are as follows:

- Uremic symptoms with neurologic abnormalities
- Persistent hyperkalemia, above 6.5 mEq/L
- Blood urea level >150 mg/dL
- Severe acidosis, pH <7.2, TCO_2 <10 to 12 mEq/L
- Hyperphosphatemia
- Pulmonary edema and CCF.

Methods of Dialysis

- Peritoneal dialysis—it can be done as:
  - Intermittent peritoneal dialysis or
  - Continuous ambulatory peritoneal dialysis or
  - Continuous cycling peritoneal dialysis.
- Hemodialysis
- Continuous renal replacement therapy with special procedures like:
  - Continuous arteriovenous hemofiltration
  - Continuous venovenous hemodialysis
  - Continuous arteriovenous ultrafiltration
  - Slow continuous ultrafiltration.

Both hemodialysis and peritoneal dialysis are effective in the management of renal failure. Peritoneal dialysis does not require vascular access and sophisticated equipment. It is easy to perform this procedure.

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The following principles should be followed by the nurses during dialysis of pediatric patients.

- **Peritoneal dialysis:** The volume of dialysate should be 30 to 50 mL/kg body weight or 1000 to 1200 mL/m^2. The child should be able to cooperate during the procedure and to perform self-care.
- **Hemodialysis:**
  - Vascular access should be done on suitable peripheral blood vessels depending upon the patient's size.
  - Blood pressure cuff and tourniquets should not be applied to the limb with fistula.
  - Subcutaneous or intramuscular injections should be avoided because the child is receiving anticoagulant therapy (heparin).
  - Extracorporeal blood volume should be as small as possible. It should not exceed 8 to 10% of the child's total blood volume.
  - Efficiency or adequacy of the dialyzer should be noted and blood pump speed to be adjusted accordingly.
  - External catheter should be secured to prevent trauma.
  - Special care for the child during dialysis should be provided by specially trained personnel who is very familiar with the protocol of dialysis and the equipment and machinery being used.

Nursing Interventions

- Preparing the child for the dialysis by explanation and encouraging to express feeling. Allowing to talk to other child who have undergone dialysis.
- Explanation and reassurance to the parents and family members to relief their anxiety so that they can provide support to the child.
- Obtaining informed consent for the procedure.
- Ensuring that all blood examination reports are available with the child before dialysis.
- Protecting the child from infections by strict aseptic technique, hygienic measures, adequate diet with vitamin supplementation, care of wound (if present) and immunization (hepatitis B).
- Maintaining appropriate fluid and nutritional intake by small frequent feeding in regular diet. Fluid and sodium restriction may be needed to prevent fluid overload especially in presence of oliguria and hypertension. Protein should not be restricted as it is important to maintain normal growth and development.
- Assisting during dialysis and following specialized procedures and instructions. Keeping the unit ready for the procedure and preparing the child on the day of dialysis according to the specific protocol.
- Maintaining careful record of temperature, pulse, respiration, blood pressure, intake and output and laboratory investigation reports. Recording body weight before and after each dialysis and continuous monitoring of general condition of the child must be done. All these records are valuable to assess the effectiveness of the therapy and to detect complications.
- Symptomatic and supportive nursing measures before and after dialysis to be provided along with promotion of self-care.
- Teaching the parents and family members regarding dialysis schedule, dietary restriction, medications, prevention of infections, possible complications, available support services, emergency measures, regular follow-up and home-based continued care.

Complications Related to Hemodialysis

- **Complications of vascular access:** Infections, central venous thrombosis or stricture, ischemia of the hand, aneurysm or pseudoaneurysm
- Arteriosclerotic cardiovascular disease, coronary heart disease, CHF, stroke, hypertension
- Disturbances in lipid metabolism
- Intercurrent infections
- Anemia
- Gastric ulcer
- Psychological problems like depression, regression.

REVIEW QUESTIONS AND MCQs

1. Discuss about pathophysiology and clinical manifestations of nephrotic syndrome in children.
2. Enlist the causes of acute renal failure in children.
3. Prepare a nursing process for a child with acute glomerulonephritis.
4. Describe about the nursing management of a child with hemodialysis.
5. Write about 'Exstrophy of urinary bladder' and its management.
6. **Select most appropriate answer of the following questions:**
   i. The dilatation of renal pelvis due to obstruction of urine flow in distal urinary tract is termed as:
      a. Urinary tract obstruction
      b. Hydronephrosis
      c. Pyelonephritis
      d. Polycystic kidney

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ii. Wilm's tumor is a malignant condition of:
   a. Kidney
   b. Adrenal gland
   c. Urinary bladder
   d. Liver and gallbladder

iii. Abnormal urethral opening on ventral aspect or under surface of penis is termed as:
   a. Epispadias
   b. Hypospadias
   c. Phimosis
   d. Paraphimosis

iv. Indications of dialysis in renal failure include all, ***except:***
   a. Persistent hyperkalemia more than 6.5 mEq/L
   b. Blood urea level more than 150 mg/dL
   c. Pulmonary edema and CCF
   d. Severe alkalosis

v. Circumcision operation is done as surgical treatment in case of:
   a. Hypospadias
   b. Epispadias
   c. Phimosis
   d. None of the above

Answers

i. b	ii. a	iii. b	iv. d	v. c

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