

Medullary sponge kidney

Symptoms usually appear only as a result of complications and are seldom present before adulthood. Complications include formation of calcium oxylate stones, which lodge in the dilated cystic collecting ducts or pass through a ureter, and infection secondary to dilation of the ducts. These complications, which occur in about 30% of patients, are likely to produce severe colic, hematuria, lower urinary tract infection ([UTI]; burning on urination, urgency, frequency), and pyelonephritis. Secondary impairment of renal function from obstruction and infection occurs in only about 10% of patients.

Polycystic kidney disease

Adult polycystic kidney disease is commonly asymptomatic through the patient's 40s, but may induce nonspecific symptoms, such as hypertension, polyuria, and recurrent UTIs. Later, the patient develops overt symptoms related to the enlarging kidney mass, such as lumbar pain, widening girth, and swollen or tender abdomen. Abdominal pain is usually worsened by exertion and relieved by lying down. In advanced stages, this disease may cause recurrent hematuria, life-threatening retroperitoneal bleeding resulting from cyst rupture, proteinuria, and colicky abdominal pain from the ureteral passage of clots or calculi. Generally, about 10 years after symptoms appear, progressive compression of kidney structures by the enlarging mass produces renal failure and uremia. Hypertension is found in about 20% to 30% of children and up to 75% of adults due to intrarenal ischemia, which activates the renin-angiotensin system.

Acute renal failure

Acute renal failure is a critical illness. Its early signs are oliguria, azotemia and, rarely, anuria. Electrolyte imbalance, metabolic acidosis, and other severe effects follow, as the patient becomes increasingly uremic and renal dysfunction disrupts other body systems: GI: anorexia, nausea, vomiting, diarrhea or constipation, stomatitis, bleeding, hematemesis, dry mucous membranes, uremic breath P Central nervous system (CNS): headache, drowsiness, irritability, confusion, peripheral neuropathy, seizures, coma Cutaneous: dryness, pruritus, pallor, purpura and, rarely, uremic frost Cardiovascular: early in the disease, hypotension; later, hypertension, arrhythmias, fluid overload, heart failure, systemic edema, anemia, altered clotting mechanisms Respiratory: pulmonary edema, Kussmaul's respirations. Fever and chills indicate infection, a common complication.

Acute pyelonephritis

Typical clinical features include urgency, frequency, burning during urination, dysuria, nocturia, and hematuria (usually microscopic but may be gross). Urine may appear cloudy and have an ammonia-like or fishy odor. Other common symptoms include a temperature of 102° F (38.9° C) or higher, shaking chills, flank pain, anorexia, and general fatigue. These symptoms characteristically develop rapidly over a few hours or a few days. Although these symptoms may disappear within days, even without treatment, residual bacterial infection is likely and may cause symptoms to recur later.

Acute poststreptococcal glomerulonephritis

APSGN begins within 1 to 3 weeks after untreated pharyngitis. Symptoms include mild to moderate edema, oliguria (less than 400 ml/24 hours), proteinuria, azotemia, hematuria, and fatigue. Mild to severe hypertension may result from either sodium or water retention (due to decreased GFR) or inappropriate renin release. Heart failure from hypervolemia leads to pulmonary edema.

Acute tubular necrosis

Nephrotoxic injury causes multiple symptoms similar to those of renal failure, particularly azotemia, anemia, acidosis, overhydration, and hypertension. Some patients may also experience fever, rash, and eosinophilia. However, ATN is usually difficult to recognize in its early stages because effects of the critically ill patient's primary disease may mask the symptoms of ATN. The first recognizable effect may be decreased urine output. Generally, hyperkalemia and the characteristic uremic syndrome soon follow, with oliguria (or, rarely, anuria) and confusion, which may progress to uremic coma. Other possible complications may include heart failure, uremic pericarditis, pulmonary edema, uremic lung, anemia, anorexia, intractable vomiting, and poor wound healing due to debilitation.

Renal infarction

Although renal infarction may be asymptomatic, typical symptoms include severe upper abdominal pain or gnawing flank pain and tenderness, costovertebral tenderness, fever, anorexia, nausea, and vomiting. Gross hematuria may be present. When arterial occlusion causes infarction, the affected kidney is small and not palpable. Renovascular hypertension, a frequent complication that may occur several days after infarction, results from reduced blood flow, which stimulates the renin-angiotensin mechanism.

Renal calculi

Clinical effects vary with size, location, and etiology of the calculi. Pain, the key symptom, usually results from obstruction; large, rough calculi occlude the opening to the ureter and increase the frequency and force of peristaltic contractions. The pain of classic renal colic travels from the costovertebral angle to the flank, to the suprapubic region and external genitalia. The intensity of this pain fluctuates and may be excruciating at its peak. If calculi are in the renal pelvis and calyces, pain may be more constant and dull. Back pain (from calculi that produce an obstruction within a kidney) and severe abdominal pain (from calculi traveling down a ureter) may also occur. (See Types of renal calculi.) Nausea and vomiting usually accompany severe pain.

Renal vein thrombosis

Clinical features of renal vein thrombosis vary with speed of onset. Rapid onset of venous obstruction produces severe lumbar pain and tenderness in the epigastric region and the costovertebral angle. Other characteristic features include fever, leukocytosis, pallor,

hematuria, proteinuria, peripheral edema and, when the obstruction is bilateral, oliguria and other uremic signs. The kidneys enlarge and become easily palpable. Hypertension is unusual but may develop. Gradual onset causes symptoms of nephrotic syndrome. Peripheral edema is possible but pain is generally absent. Other clinical signs include proteinuria, hypoalbuminemia, and hyperlipidemia. Infants with this disease have enlarged kidneys, oliguria, and renal insufficiency that may progress to acute or chronic renal failure.

Nephrotic syndrome

The dominant clinical feature of nephrotic syndrome is mild to severe dependent edema of the ankles or sacrum, or periorbital edema, especially in children. Edema may lead to ascites, pleural effusion, and swollen external genitalia. Accompanying symptoms may include orthostatic hypotension, lethargy, anorexia, depression, and pallor. Major complications are malnutrition, infection, coagulation disorders, thromboembolic vascular occlusion, and accelerated atherosclerosis.

Chronic glomerulonephritis

Chronic glomerulonephritis typically develops insidiously and asymptotically, usually over many years. At any time, however, it may suddenly become progressive, producing nephrotic syndrome, hypertension, proteinuria, and hematuria. In late stages of progressive chronic glomerulonephritis, it may accelerate to uremic symptoms, such as azotemia, nausea, vomiting, pruritus, dyspnea, malaise, and fatigability. Mild to severe edema and anemia may accompany these symptoms. Severe hypertension may cause cardiac hypertrophy, leading to heart failure, and may accelerate the development of advanced renal failure, eventually necessitating dialysis or transplantation.

Renovascular hypertension

In addition to elevated systemic blood pressure, renovascular hypertension usually produces symptoms common to hypertensive states, such as headache, palpitations, tachycardia, anxiety, lightheadedness, decreased tolerance of temperature extremes, retinopathy, and mental sluggishness. Significant complications include heart failure, myocardial infarction, stroke and, occasionally, renal failure.

Hydronephrosis

Clinical features of hydronephrosis vary with the cause of the obstruction. In some patients, hydronephrosis produces no symptoms or only mild pain and slightly decreased urinary flow; in others, it may produce severe, colicky renal pain or dull flank pain that may radiate to the groin, and gross urinary abnormalities, such as hematuria, pyuria, dysuria, alternating oliguria and polyuria, or complete anuria. Other symptoms of hydronephrosis include nausea, vomiting, abdominal fullness, pain on urination, dribbling, or hesitancy. Unilateral obstruction may cause pain on only one side, usually in the flank area. The most common complication of an obstructed kidney is infection (pyelonephritis) due to stasis that exacerbates renal damage and may create a life-threatening crisis. Paralytic ileus frequently accompanies acute obstructive uropathy.

Renal tubular acidosis

In children and adults, RTA may lead to urinary tract infection, rickets, and growth problems. Possible complications of RTA include nephrocalcinosis and pyelonephritis.

Chronic renal failure

Chronic renal failure produces major changes in all body systems: Renal and urologic: Initially, salt-wasting and consequent hyponatremia produce hypotension, dry mouth, loss of skin turgor, listlessness, fatigue, and nausea; later, somnolence and confusion develop. As the number of functioning nephrons decreases, so does the kidneys' capacity to excrete sodium, resulting in salt retention and overload. Accumulation of potassium causes muscle irritability, then muscle weakness as the potassium level continues to rise. Fluid overload and metabolic acidosis also occur. Urinary output decreases; urine is very dilute and contains casts and crystals. Cardiovascular: Renal failure leads to hypertension, arrhythmias (including life-threatening ventricular tachycardia or fibrillation), cardiomyopathy, uremic pericarditis, pericardial effusion with possible cardiac tamponade, heart failure, and periorbital and peripheral edema. Respiratory: Pulmonary changes include reduced pulmonary macrophage activity with increased susceptibility to infection, pulmonary edema, pleuritic pain, pleural friction rub and effusions, crackles, thick sputum, uremic pleuritis and uremic lung (or uremic pneumonitis), dyspnea due to heart failure, and Kussmaul's respirations as a result of acidosis. GI: Inflammation and ulceration of GI mucosa cause stomatitis, gum ulceration and bleeding and, possibly, parotitis, esophagitis, gastritis, duodenal ulcers, lesions on the small and large bowel, uremic colitis, pancreatitis, and proctitis. Other GI symptoms include a metallic taste in the mouth, uremic fetor (ammonia smell to breath), anorexia, nausea, and vomiting. Cutaneous: Typically, the skin is pallid, yellowish bronze, dry, and scaly. Other cutaneous symptoms include severe itching; purpura; ecchymoses; petechiae; uremic frost (most often in critically ill or terminal patients); thin, brittle fingernails with characteristic lines; and dry, brittle hair that may change color and fall out easily. Neurologic: Restless leg syndrome, one of the first signs of peripheral neuropathy, causes pain, burning, and itching in the legs and feet, which may be relieved by voluntarily shaking, moving, or rocking them. Eventually, this condition progresses to paresthesia and motor nerve dysfunction (usually bilateral footdrop) unless dialysis is initiated. Other signs and symptoms include muscle cramping and twitching, shortened memory and attention span, apathy, drowsiness, irritability, confusion, coma, and seizures. EEG changes indicate metabolic encephalopathy. Endocrine: Common endocrine abnormalities include stunted growth patterns in children (even with elevated growth hormone levels), infertility and decreased libido in both sexes, amenorrhea and cessation of menses in females, and impotence, decreased sperm production, and testicular atrophy in males. Increased aldosterone secretion (related to increased renin production) and impaired carbohydrate metabolism (increased blood glucose levels similar to diabetes mellitus) may also occur. Hematopoietic: Anemia, decreased red blood cell (RBC) survival time, blood loss from dialysis and GI bleeding, mild thrombocytopenia, and platelet defects occur. Other problems include increased bleeding and clotting disorders, demonstrated by purpura, hemorrhage

from body orifices, easy bruising, ecchymoses, and petechiae. Skeletal: Calcium-phosphorus imbalance and consequent parathyroid hormone imbalances cause muscle and bone pain, skeletal demineralization, pathologic fractures, and calcifications in the brain, eyes, gums, joints, myocardium, and blood vessels. Arterial calcification may produce coronary artery disease. In children, renal osteodystrophy (renal rickets) may develop.

Lower urinary tract infection

Lower UTI usually produces urgency, frequency, dysuria, cramps or spasms of the bladder, itching, a feeling of warmth during urination, nocturia, and possibly urethral discharge in males. Inflammation of the bladder wall also causes hematuria and fever. Other common features include low back pain, malaise, nausea, vomiting, abdominal pain or tenderness over the bladder area, chills, and flank pain.

Vesicoureteral reflux

Vesicoureteral reflux typically manifests itself as the signs and symptoms of UTI: frequency, urgency, burning on urination, hematuria, foul- P smelling urine and, in infants, dark, concentrated urine. With upper urinary tract involvement, signs and symptoms usually include high fever, chills, flank pain, vomiting, and malaise.

Neurogenic bladder

Neurogenic bladder produces a wide range of clinical effects, depending on the underlying cause and its effect on the structural integrity of the bladder. Usually, this disorder causes some degree of incontinence, changes in initiation or interruption of micturition, and the inability to empty the bladder completely. Other effects of neurogenic bladder include vesicoureteral reflux, deterioration or infection in the upper urinary tract, and hydronephrosis. Depending on the site and extent of the spinal cord lesion, spastic neurogenic bladder may produce involuntary or frequent scanty urination, without a feeling of bladder fullness, and possibly spontaneous spasms of the arms and legs. Anal sphincter tone may be increased. Tactile stimulation of the abdomen, thighs, or genitalia may precipitate voiding and spontaneous contractions of the arms and legs. With cord lesions in the upper thoracic (cervical) level, bladder distention can trigger hyperactive autonomic reflexes, resulting in severe hypertension, bradycardia, and headaches. Flaccid neurogenic bladder may be associated with overflow incontinence, diminished anal sphincter tone, and a greatly distended bladder (evident on percussion or palpation), but without the accompanying feeling of bladder fullness due to sensory impairment.

Prostatitis

Acute prostatitis begins with fever, chills, low back pain, myalgia, perineal fullness, and arthralgia. Urination is frequent and urgent. Dysuria, nocturia, and urinary obstruction may also occur. The urine may appear cloudy. When palpated rectally, the prostate is tender, indurated, swollen, firm, and warm. Chronic bacterial prostatitis sometimes produces no symptoms but usually elicits the same urinary symptoms as the acute form but to a lesser

degree. UTI is a common complication. Other possible signs include painful ejaculation, hemospermia, persistent urethral discharge, and sexual dysfunction.

Epididymitis

The key symptoms are pain, extreme tenderness, and swelling in the groin and scrotum with erythema, high fever, malaise, and a characteristic waddle—an attempt to protect the groin and scrotum during walking. An acute hydrocele may also result from inflammation.

Benign prostatic hyperplasia

Clinical features of BPH depend on the extent of prostatic enlargement and the lobes affected. Characteristically, the condition starts with a group of symptoms known as prostatism: reduced urine stream caliber and force, urinary hesitancy, and difficulty starting micturition (resulting in straining, feeling of incomplete voiding, and an interrupted stream). As the obstruction increases, it causes frequent urination with nocturia, dribbling, urine retention, incontinence, and possibly hematuria. Physical examination indicates a visible midline mass above the symphysis pubis that represents an incompletely emptied bladder; rectal palpation discloses an enlarged prostate. Examination may detect secondary anemia and, possibly, renal insufficiency secondary to obstruction. P As BPH worsens, complete urinary obstruction may follow infection or use of decongestants, tranquilizers, alcohol, anti-depressants, or anticholinergics. Complications include infection, renal insufficiency, hemorrhage, and shock.