Chapter 32. Disorders of the Kidneys

Jack W. McAninch, MD, FACS, FRCS(E)(Hon)

Congenital Anomalies of the Kidneys

Congenital anomalies occur more frequently in the kidney than in any other organ. Some cause no difficulty, but many (eg, hypoplasia, polycystic kidneys) cause impairment of renal function. It has been noted that children with a gross deformity of an external ear associated with ipsilateral maldevelopment of the facial bones are apt to have a congenital abnormality of the kidney (eg, ectopy, hypoplasia) on the same side as the visible deformity. Lateral displacement of the nipples has been observed in association with bilateral renal hypoplasia.

A significant incidence of renal agenesis, ectopy, malrotation, and duplication has been observed in association with congenital scoliosis and kyphosis. Unilateral agenesis, hypoplasia, and dysplasia are often seen in association with supralevator imperforate anus. For a better understanding of these congenital abnormalities, see the discussion of the embryology and development of the kidney in Chapter 2.

Agenesis

Bilateral renal agenesis is extremely rare; no more than 400 cases have been reported. The children do not survive. The condition does not appear to have any predisposing factors. Prenatal suspicion of the anomaly exists when oligohydramnios is present on fetal ultrasound examination. Pulmonary hypoplasia and facial deformities (Potter facies) are usually present. Abdominal ultrasound examination usually establishes the diagnosis.

One kidney may be absent (estimated incidence: 1 in 450–1000 births). In some cases, this may be because the ureteral bud (from the Wolffian duct) failed to develop or, if it did develop, did not reach the metanephros (adult kidney). Without a drainage system, the metanephric mass undergoes atrophy. The ureter is absent on the side of the unformed kidney in 50% of cases, although a blind ureteral duct may be found (see Chapter 2).

Renal agenesis causes no symptoms; it is usually found by accident on abdominal or renal imaging. It is not an easy diagnosis to establish even though on inspection of the bladder, the ureteral ridge is absent and no orifice is visualized, for the kidney could be present but be drained by a ureter whose opening is ectopic (into the urethra, seminal vesicle, or vagina). If definitive diagnosis seems essential, isotope studies, ultrasonography, and computed tomography (CT) should establish the diagnosis.

There appears to be an increased incidence of infection, hydronephrosis, and stones in the contralateral organ. Other congenital anomalies associated with this defect include cardiac, vertebral column, and anal anomalies, as well as anomalies of the long bones, hands, and genitalia.

Hypoplasia

Hypoplasia implies a small kidney. The total renal mass may be divided in an unequal manner, in which case one kidney is small and the other correspondingly larger than normal. Some of these congenitally small kidneys prove, on pathologic examination, to be dysplastic. Unilateral or bilateral hypoplasia has been observed in infants with fetal alcohol syndrome, and renal anomalies have been reported in infants with in utero cocaine exposure.

Differentiation from acquired atrophy is difficult. Atrophic pyelonephritis usually reveals typical distortion of the calyces. Vesicoureteral reflux in infants may cause a dwarfed kidney even in the absence of infection. Stenosis of the renal artery leads to shrinkage of the kidney.

Such kidneys have small renal arteries and branches and are associated with hypertension, which is relieved by nephrectomy. Selective renal venography is helpful in differentiating between a congenitally absent kidney and one that is small and nonvisualized.

Supernumerary Kidneys

The presence of a third kidney is very rare; the presence of four separate kidneys in one individual has been reported only once. The anomaly must not be confused with duplication (or triplication) of the renal pelvis in one kidney, which is not uncommon.

Dysplasia and Multicystic Kidney

Renal dysplasia has protean manifestations. Multicystic kidney of the newborn is usually unilateral, nonhereditary, and characterized by an irregularly lobulated mass of cysts; the ureter is usually absent or atretic. It may develop because of faulty union of the nephron and the collecting system. At most, only a few embryonic glomeruli and tubules are observed. The only finding is the discovery of an irregular mass in the flank. Nothing is shown on urography, but in an occasional case, some radiopaque fluid may be noted. If the cystic kidney is large, its mate is usually normal. However, when the cystic organ is small, the contralateral kidney is apt to be abnormal. The cystic nature of the lesion may be revealed by sonography, and the diagnosis can be established in utero. If the physician feels that the proper diagnosis has been made, no treatment is necessary. If there is doubt about the diagnosis, nephrectomy is considered the procedure of choice. Neoplastic changes in multicystic renal dysplasia have been noted, but this is accepted as a benign condition.

Multicystic kidney is often associated with contralateral renal and ureteral abnormalities. Contralateral ureteropelvic junction obstruction is one of the common problems noted. Diagnostic evaluation of both kidneys is required to establish the overall status of anomalous development.

Dysplasia of the renal parenchyma is also seen in association with ureteral obstruction or reflux that was probably present early in pregnancy. It is relatively common as a segmental renal lesion involving the upper pole of a duplicated kidney whose ureter is obstructed by a congenital ureterocele. It may also be found in urinary tracts severely obstructed by posterior urethral valves; in this instance, the lesion may be bilateral.

Microscopically, the renal parenchyma is “disorganized.” Tubular and glomerular cysts may be noted; these elements are fetal in type. Islands of metaplastic cartilage are often seen. The common denominator seems to be fetal obstruction.

Adult Polycystic Kidney Disease

Adult polycystic kidney disease is an autosomal dominant hereditary condition and almost always bilateral (95% of cases). The disease encountered in infants is different from that seen in adults, although the literature reports a small number of infants with the adult type. The former is an autosomal recessive disease in which life expectancy is short, whereas that diagnosed in adulthood is autosomal dominant; symptoms ordinarily do not appear until after age 40. Cysts of the liver, spleen, and pancreas may be noted in association with both forms. The kidneys are larger than normal and are studded with cysts of various sizes.

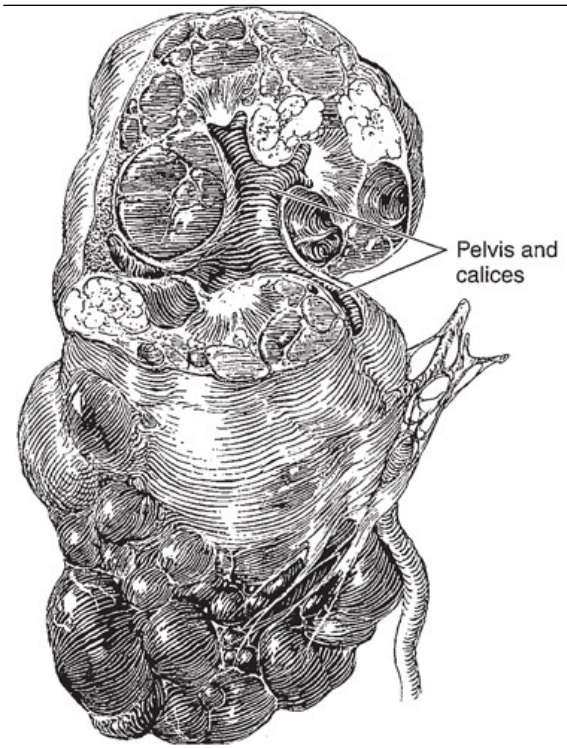
Etiology and Pathogenesis

The evidence suggests that the cysts occur because of defects in the development of the collecting and uriniferous tubules and in the mechanism of their joining. Blind secretory tubules that are connected to functioning glomeruli become cystic. As the cysts enlarge, they compress adjacent parenchyma, destroy it by ischemia, and occlude normal tubules. The result is progressive functional impairment.

Pathology

Grossly, the kidneys are usually much enlarged. Their surfaces are studded with cysts of various sizes (Figure 32–1). On section, the cysts are found to be scattered throughout the parenchyma. Calcification is rare. The fluid in the cyst is usually amber colored but may be hemorrhagic.

Figure 32–1.



Source: McAninch JW, Lue TF: *Smith & Tanagho's General Urology*, 18th Edition: www.accessmedicine.com

Polycystic kidney. Multiple cysts deep in the parenchyma and on the surface. Note distortion of the calyces by the cysts.

Microscopically, the lining of the cysts consists of a single layer of cells. The renal parenchyma may show peritubular fibrosis and evidence of secondary infection. There appears to be a reduction in the number of glomeruli, some of which may be hyalinized. Renal arteriolar thickening is a prominent finding in adults.

Clinical Findings

Symptoms

Pain over one or both kidneys may occur because of the drag on the vascular pedicles by the heavy kidneys, from obstruction or infection, or from hemorrhage into a cyst. Gross or microscopic total hematuria is not uncommon and may be severe; the cause for this is not clear. Colic may occur if blood clots or stones are passed. The patient may notice an abdominal mass.

Infection (chills, fever, renal pain) commonly complicates polycystic disease. Symptoms of vesical irritability may be the first complaint. When renal insufficiency ensues, headache, nausea and vomiting, weakness, and loss of weight occur.

Signs

One or both kidneys are usually palpable. They may feel nodular. If infected, they may be tender. Hypertension is found in 60–70% of these patients. Evidence of cardiac enlargement is then noted.

Fever may be present if pyelonephritis exists or if cysts have become infected. In the stage of uremia, anemia and loss of weight may be evident. Ophthalmoscopic examination may show changes typical of moderate or severe hypertension.

Laboratory Findings

Anemia may be noted, caused either by chronic loss of blood or, more commonly, by the hematopoietic depression accompanying uremia. Proteinuria

and microscopic (if not gross) hematuria are the rule. Pyuria and bacteriuria are common.

Progressive loss of concentrating power occurs. Renal clearance tests show varying degrees of renal impairment. About one-third of patients with polycystic kidney disease are uremic when first seen.

X-Ray Findings

Both renal shadows are usually enlarged on a plain film of the abdomen, even as much as five times normal size. Kidneys >16 cm in length are suspect.

The renal masses are usually enlarged and the calyceal pattern is quite bizarre (spider deformity). The calyces are broadened and flattened, enlarged, and often curved, as they tend to hug the periphery of adjacent cysts. Often, the changes are only slight or may even be absent on one side, leading to the erroneous diagnosis of tumor of the other kidney. If cysts are infected, perinephritis may obscure the renal and even the psoas shadows.

CT Scanning

CT is an excellent noninvasive technique used to establish the diagnosis of polycystic disease. The multiple thin-walled cysts filled with fluid and the large renal size make this imaging method extremely accurate (95%) for diagnosis.

Isotope Studies

Photoscans reveal multiple “cold” avascular spots in large renal shadows.

Ultrasonography

Sonography appears to be superior to both excretory urography and isotope scanning in diagnosis of polycystic disorders.

Instrumental Examination

Cystoscopy may show evidence of cystitis, in which case the urine will contain abnormal elements. Bleeding from a ureteral orifice may be noted. Ureteral catheterization and retrograde urograms are rarely indicated.

Differential Diagnosis

Bilateral hydronephrosis (on the basis of congenital or acquired ureteral obstruction) may present bilateral flank masses and signs of impairment of renal function, but ultrasonography shows changes quite different from those of the polycystic kidney.

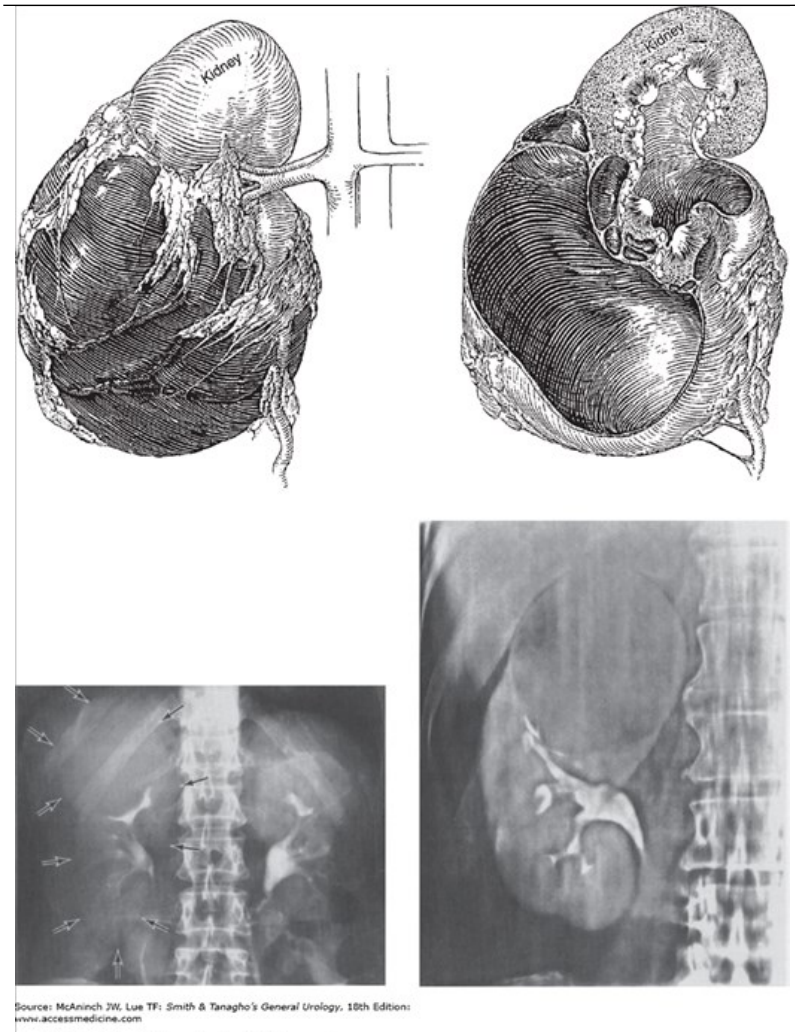
Bilateral renal tumor is rare but may mimic polycystic kidney disease perfectly on urography. Tumors are usually localized to one portion of the kidney, whereas cysts are quite diffusely distributed. The total renal function should be normal with unilateral tumor but is usually depressed in patients with polycystic kidney disease. CT scan may be needed at times to differentiate between the two conditions.

In **von Hippel-Lindau disease** (angiomatous cerebellar cyst, angiomatosis of the retina, and tumors or cysts of the pancreas), multiple bilateral cysts or adenocarcinomas of both kidneys may develop. The presence of other stigmas should make the diagnosis. CT, angiography, sonography, or scintiphotography should be definitive.

Tuberous sclerosis (convulsive seizures, mental retardation, and adenoma sebaceum) is typified by hamartomatous tumors often involving the skin, brain, retinas, bones, liver, heart, and kidneys (see Chapter 21). The renal lesions are usually multiple and bilateral and microscopically are angiomyolipomas. The presence of other stigmas and use of CT or sonography should make the differentiation.

A **simple cyst** (see section following) is usually unilateral and single; total renal function should be normal. Urograms usually show a single lesion (Figure 32–2), whereas polycystic kidney disease is bilateral and has multiple filling defects.

Figure 32–2.



Simple cyst. ***Upper left:*** Large cyst displacing lower pole laterally. ***Upper right:*** Section of kidney showing one large and a few small cysts. ***Lower left:*** Excretory urogram showing soft-tissue mass in upper pole of right kidney. Elongation and distortion of upper calyces by cyst. ***Lower right:*** Infusion nephrotomogram showing large cyst in upper renal pole distorting upper calyces and dislocating upper portion of kidney laterally.

Complications

For reasons that are not clear, pyelonephritis is a common complication of polycystic kidney disease. It may be asymptomatic; pus cells in the urine may be few or absent. Stained smears or quantitative cultures make the diagnosis. A gallium-67 citrate scan will definitely reveal the sites of infection, including abscess.

Infection of cysts is associated with pain and tenderness over the kidney and a febrile response. The differential diagnosis between infection of cysts and pyelonephritis may be difficult, but here again a gallium scan will prove helpful. In rare instances, gross hematuria may be so brisk and persistent as to endanger life.

Treatment

Except for unusual complications, the treatment is conservative and supportive.

General Measures

The patient should be put on a low-protein diet (0.5–0.75 g/kg/day of protein) and fluids forced to 3000 mL or more per day. Physical activity may be

permitted within reason, but strenuous exercise is contraindicated. When the patient is in the state of absolute renal insufficiency, one should treat as for uremia from any cause. Hypertension should be controlled. Hemodialysis may be indicated.

Surgery

There is no evidence that excision or decompression of cysts improves renal function. If a large cyst is found to be compressing the upper ureter, causing obstruction and further embarrassing renal function, it should be resected or aspirated. When the degree of renal insufficiency becomes life threatening, chronic dialysis or renal transplantation should be considered.

Treatment of Complications

Pyelonephritis must be rigorously treated to prevent further renal damage. Infection of cysts requires surgical drainage. If bleeding from one kidney is so severe that exsanguination is possible, nephrectomy or embolization of the renal or, preferably, the segmental artery must be considered as a life-saving measure. Concomitant diseases (eg, tumor, obstructing stone) may require definitive surgical treatment.

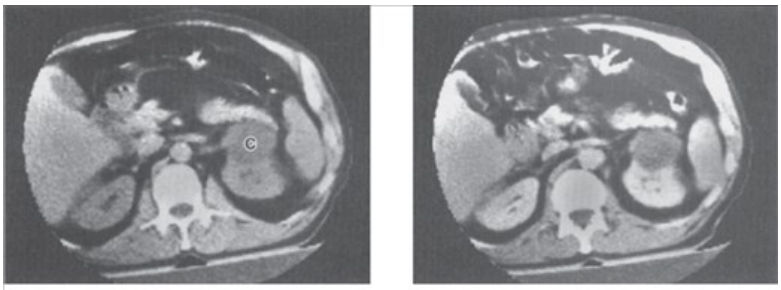
Prognosis

When the disease affects children, it has a very poor prognosis. The large group presenting clinical signs and symptoms after age 35–40 has a somewhat more favorable prognosis. Although there is wide variation, these patients usually do not live longer than 5 or 10 years after the diagnosis is made, unless dialysis is made available or renal transplantation is done.

Simple (Solitary) Cyst

Simple cyst (Figures 32–2 and 32–3) of the kidney is usually unilateral and single but may be multiple and multilocular and, more rarely, bilateral. It differs from polycystic kidneys both clinically and pathologically.

Figure 32–3.



Left renal cyst. ***Left:*** Computed tomography (CT) scan shows a homogeneous low-density mass (C) arising from anterior border of left kidney just posterior to tail of the pancreas. The CT attenuation value was similar to that of water, indicating a simple renal cyst. ***Right:*** After intravenous injection of contrast material, the mass did not increase in attenuation value, adding further confirmatory evidence of its benign cystic nature.

Etiology and Pathogenesis

Whether simple cyst is congenital or acquired is not clear. Its origin may be similar to that of polycystic kidneys, that is, the difference may be merely one of degree. On the other hand, simple cysts have been produced in animals by causing tubular obstruction and local ischemia; this suggests that the lesion can be acquired.

As a simple cyst grows, it compresses and thereby may destroy renal parenchyma, but rarely does it destroy so much renal tissue that renal function is impaired. A solitary cyst may be placed in such a position as to compress the ureter, causing progressive hydronephrosis. Infection may complicate the picture.

Acquired cystic disease of the kidney can arise as an effect of chronic dialysis. The spontaneous regression of cysts has occasionally been noted.

Pathology

Simple cysts usually involve the lower pole of the kidney. Those that produce symptoms average about 10 cm in diameter, but a few are large enough to fill the entire flank. They usually contain a clear amber fluid. Their walls are quite thin, and the cysts are "blue-domed" in appearance. Calcification of the sac is occasionally seen. About 5% contain hemorrhagic fluid, and possibly one-half of these have papillary cancers on their walls.

Simple cysts are usually superficial but may be deeply situated. When a cyst is situated deep in the kidney, the cyst wall is adjacent to the epithelial lining of the pelvis or calyces, from which it may be separated only with great difficulty. Cysts do not communicate with the renal pelvis (Figure 32–2). Microscopic examination of the cyst wall shows heavy fibrosis and hyalinization; areas of calcification may be seen. The adjacent renal tissue is compressed and fibrosed. A number of cases of simple cysts have been reported in children. However, large cysts are rare in children; the presence of cancer must therefore be ruled out.

Multilocular renal cysts may be confused with tumor on urography. Sonography usually makes the diagnosis. Occasionally, CT and magnetic resonance imaging (MRI) may be necessary.

The Bosniak classification of simple renal cysts is an aid to determining the chance of malignancy based on imaging criteria. Type I cysts are simple and smooth walled, with clear fluid; Type II cysts are also benign, but may have minimal septations and a small fine rim of calcification; Type III cysts are more complex, with more calcification, increasing septations, and a thick cyst wall; Type IV cysts have a thickened irregular wall, often with calcifications, and a mass may be noted inside the cyst, suggesting carcinoma. Numerous variations of the findings are used as a guide in the diagnosis of renal cancer.

Clinical Findings

Symptoms

Pain in the flank or back, usually intermittent and dull, is not uncommon. If bleeding suddenly distends the cyst wall, pain may come on abruptly and be severe. Gastrointestinal symptoms are occasionally noted and may suggest peptic ulcer or gallbladder disease. The patient may discover a mass in the abdomen, although cysts of this size are unusual. If the cyst becomes infected, the patient usually complains of pain in the flank, malaise, and fever.

Signs

Physical examination is usually normal, although occasionally a mass in the region of the kidney may be palpated or percussed. Tenderness in the flank may be noted if the cyst becomes infected.

Laboratory Findings

Urinalysis is usually normal. Microscopic hematuria is rare. Renal function tests are normal unless the cysts are multiple and bilateral (rare). Even in the face of extensive destruction of one kidney, compensatory hypertrophy of the other kidney will maintain normal total function.

CT Scanning

CT scan appears to be the most accurate means of differentiating renal cyst and tumor (Figure 32–3). Cysts have an attenuation approximating that of water, whereas the density of tumors is similar to that of normal parenchyma. Parenchyma is made more dense with the intravenous injection of radiopaque fluid, but a cyst remains unaffected. The wall of a cyst is sharply demarcated from the renal parenchyma; a tumor is not. The wall of a cyst is thin, and that of a tumor is not. CT scan may well supplant cyst puncture in the differentiation of cyst and tumor in many cases.

Renal Ultrasonography

Renal ultrasonography is a noninvasive diagnostic technique that in a high percentage of cases differentiates between a cyst and a solid mass. If findings on ultrasonography are also compatible with a cyst, a needle can be introduced into the cyst under ultrasonographic control and the cyst can

be aspirated.

Isotope Scanning

A rectilinear scan clearly delineates the mass but does not differentiate cyst from tumor. The technetium scan, made with the camera, reveals that the mass is indeed avascular.

Percutaneous Cyst Aspiration with Cystography

If the studies listed leave some doubt about the differentiation between cyst and tumor, aspiration may be done (see Section "Treatment," given later).

Differential Diagnosis

Carcinoma of the kidney also occupies space but tends to lie more deeply in the organ and therefore causes more distortion of the calyces. Hematuria is common with tumor, rare with cyst. If a solid tumor overlies the psoas muscle, the edge of the muscle is obliterated on the plain film; it can be seen through a cyst, however. Evidence of metastases (ie, loss of weight and strength, palpable supraclavicular nodes, chest film showing metastatic nodules), erythrocytosis, hypercalcemia, and increased sedimentation rate suggest cancer. It must be remembered, however, that the walls of a simple cyst may undergo cancerous degeneration. Sonography, CT scan, or MRI should be almost definitive in differential diagnosis. It is wise to assume that all space-occupying lesions of the kidneys are cancers until proved otherwise.

Polycystic kidney disease is almost always bilateral. Diffuse calyceal and pelvic distortion is the rule. Simple cyst is usually solitary and unilateral. Polycystic kidney disease is usually accompanied by impaired renal function and hypertension; simple cyst is not.

Renal cortical abscess is rare. A history of skin infection a few weeks before the onset of fever and local pain may be obtained. CT scan of the kidney will usually show signs of the abscess. The kidney may be fixed; this can be demonstrated by comparing the position of the kidney when the patient is supine and upright. Angiography demonstrates an avascular lesion. A gallium-67 scan demonstrates the inflammatory nature of the lesion, but an infected simple cyst might have a similar appearance.

Hydronephrosis may present the same symptoms and signs as simple cyst, but the urograms are quite different. Cyst causes calyceal distortion; with hydronephrosis, dilatation of the calyces and pelvis due to an obstruction is present. Acute or subacute hydronephrosis usually produces more local pain because of increased intrapelvic pressure and is more apt to be complicated by infection.

Extrarenal tumor (eg, adrenal, mixed retroperitoneal sarcoma) may displace a kidney, but rarely does it invade it and distort its calyces. If an echinococcal cyst of the kidney does not communicate with the renal pelvis, it may be difficult to differentiate from solitary cyst, for no scoleces or hooklets will be present in the urine. The wall of a hydatid cyst often reveals calcification on x-ray examination (see Figure 14–5). A skin sensitivity test (Casoni) for hydatid disease may prove helpful.

Complications (Rare)

Spontaneous infection in a simple cyst is rare, but when it occurs, it is difficult to differentiate from carbuncle. Hemorrhage into the cyst sometimes occurs. If sudden, it causes severe pain. The bleeding may come from a complicating carcinoma arising on the wall of the cyst.

Hydronephrosis may develop if a cyst of the lower pole impinges on the ureter. This in itself may cause pain from back pressure of urine in the renal pelvis. This obstruction may lead to renal infection.

Treatment

Specific Measures

1. If renal sonography, CT, or MRI does not lead to a definitive diagnosis, renal angiography or needle aspiration of the cyst may be necessary. Should aspiration be necessary, it can be done under sonographic guidance. The recovery of clear fluid is characteristic of a benign cyst, which should be confirmed by cytologic evaluation. In some centers, contrast radiopaque fluid is injected into the cyst after aspiration for a more thorough

evaluation of the cyst wall. A smooth cyst wall, free of irregularities, supports the presence of a benign cyst. If the aspirate contains blood, surgical exploration should be considered, because the chances are great that the growth is cancerous.

2. If the diagnosis can be clearly established, one should consider leaving the cyst alone, since it is rare for a cyst to harm the kidney. Sonography is useful in follow-up of patients with cysts.

Treatment of Complications

If the cyst becomes infected, intensive antimicrobial therapy should be instituted; antimicrobial drugs have been found to attain very low concentrations in the cyst fluid. Therefore, percutaneous drainage is often required. Surgical excision of the extrarenal portion of the cyst wall and drainage are curative when percutaneous drainage fails.

If hydronephrosis is present, excision of the obstructing cyst will relieve the ureteral obstruction. Pyelonephritis in the involved kidney should suggest urinary stasis secondary to impaired ureteral drainage. Removal of the cyst and consequent relief of urinary back pressure make antimicrobial therapy more effective.

Prognosis

Simple cysts can be diagnosed with great accuracy using sonography and CT scan. Yearly sonography is recommended as a method of following the cyst for changes in size, configuration, and internal consistency. CT scan may be done if changes suggest carcinoma, and aspiration may then be performed if necessary to establish a diagnosis. Most cysts cause little difficulty.

Renal Fusion

About 1 in 1000 individuals has some type of renal fusion, the most common being the horseshoe kidney. The fused renal mass almost always contains two excretory systems and therefore two ureters. The renal tissue may be divided equally between the two flanks, or the entire mass may be on one side. Even in the latter case, the two ureters open at their proper places in the bladder.

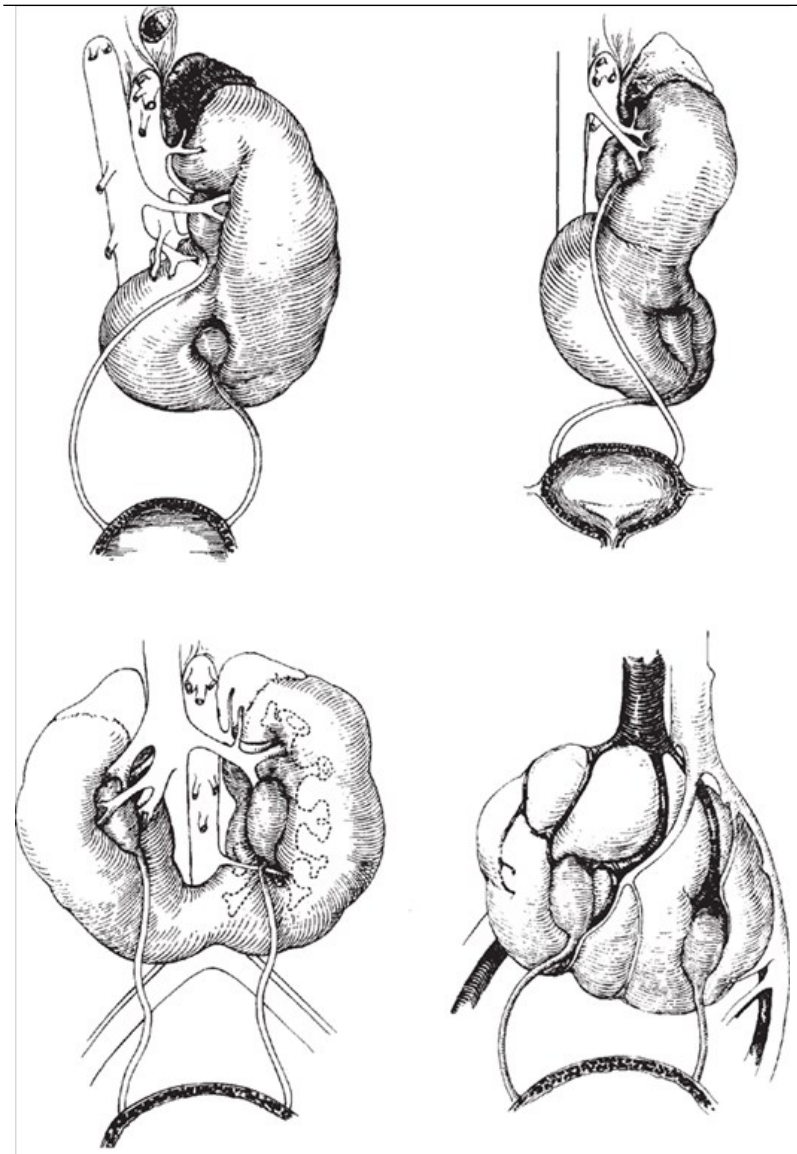
Etiology and Pathogenesis

It appears that this fusion of the two metanephroi occurs early in embryologic life, when the kidneys lie low in the pelvis. For this reason, they seldom ascend to the high position that normal kidneys assume. They may even remain in the true pelvis. Under these circumstances, such a kidney may derive its blood supply from many vessels in the area (eg, aorta, iliacs). In patients with both ectopia and fusion, 78% have extraurologic anomalies and 65% exhibit other genitourinary defects.

Pathology (Figure 32–4)

Because the renal masses fuse early, normal rotation cannot occur; therefore, each pelvis lies on the anterior surface of its organ. Thus, the ureter must ride over the isthmus of a horseshoe kidney or traverse the anterior surface of the fused kidney. Some degree of ureteral compression may arise from this or from obstruction by one or more aberrant blood vessels. The incidence of hydronephrosis and, therefore, infection is high. Vesicoureteral reflux has frequently been noted in association with fusion.

Figure 32–4.



Renal fusion. ***Upper left:*** Crossed renal ectopy with fusion. The renal mass lies in the left flank. The right ureter must cross over the midline. ***Upper right:*** Example of "sigmoid" kidney. ***Lower left:*** Horseshoe kidney. Pelves are anterior. Note the aberrant artery obstructing the left ureter and the low position of renal mass. ***Lower right:*** Pelvic kidney. Pelves are placed anteriorly. Note the aberrant blood supply.

In horseshoe kidney, the isthmus usually joins the lower poles of each kidney; each renal mass lies lower than normal. The axes of these masses are vertical, whereas the axes of normal kidneys are oblique to the spine, because they lie along the edges of the psoas muscles. On rare occasions, the two nephric masses are fused into one mass containing two pelves and two ureters. The mass may lie in the midline in order to open into the bladder at the proper point (crossed renal ectopy with fusion).

Clinical Findings

Symptoms

Most patients with fused kidneys have no symptoms. Some, however, develop ureteral obstruction. Gastrointestinal symptoms (renodigestive reflex) mimicking peptic ulcer, cholelithiasis, or appendicitis may be noted. Infection is apt to occur if ureteral obstruction and hydronephrosis or calculus develops.

Signs

Physical examination results are usually negative unless the abnormally placed renal mass can be felt. With horseshoe kidney, it may be possible to palpate a mass over the lower lumbar spine (the isthmus). In the case of crossed ectopy, a mass may be felt in the flank or lower abdomen.

Laboratory Findings

Urinalysis is normal unless there is infection. Renal function is normal unless disease coexists in each of the fused renal masses.

X-Ray Findings

In the case of horseshoe kidney, the axes of the two kidneys, if visible on a plain film, are parallel to the spine. At times, the isthmus can be identified. The plain film may also reveal a large soft-tissue mass in one flank, yet not show a renal shadow on the other side. Excretory urograms establish the diagnosis if the renal parenchyma has been maintained. The increased density of the renal tissue may make the position or configuration of the kidney more distinct. Urograms also visualize the pelvis and ureters.

1. With horseshoe kidney, the renal pelves lie on the anterior surfaces of their kidney masses, whereas the normal kidney has its pelvis lying mesial to it. The most valuable clue to the diagnosis of horseshoe kidney is the presence of calyces in the region of the lower pole that point medially and lie medial to the ureter (Figure 32–4).

2. Crossed renal ectopy with fusion shows two pelves and two ureters. One ureter must cross the midline in order to empty into the bladder at the proper point (Figure 32–4).

3. A cake or lump kidney may lie in the pelvis (fused pelvis kidney), but again its ureters and pelves will be shown (Figure 32–4). It may compress the dome of the bladder.

CT scan clearly outlines the renal mass but is seldom necessary for diagnosis. With pelvic fused kidney or one lying in the flank, the plain film taken with ureteral catheters in place gives the first hint of the diagnosis. Retrograde urograms show the position of the pelves and demonstrate changes compatible with infection or obstruction. Renal scanning delineates the renal mass and its contour, as does sonography.

Differential Diagnosis

Separate kidneys that fail to undergo normal rotation may be confused with horseshoe kidney. They lie along the edges of the psoas muscles, whereas the poles of a horseshoe kidney lie parallel to the spine and the lower poles are placed on the psoas muscles. The calyces in the region of the isthmus of a horseshoe kidney point medially and lie close to the spine.

The diagnosis of fused or lump kidney may be missed on excretory urograms if one of the ureters is markedly obstructed, so that a portion of the kidney, pelvis, and ureter fails to visualize. Infusion urograms or retrograde urograms demonstrate both excretory tracts in the renal mass.

Complications

Fused kidneys are prone to ureteral obstruction because of a high incidence of aberrant renal vessels and the necessity for one or both ureters to arch around or over the renal tissue. Hydronephrosis, stone, and infection, therefore, are common. A large fused kidney occupying the concavity of the sacrum may cause dystocia.

Treatment

No treatment is necessary unless obstruction or infection is present. Drainage of a horseshoe kidney may be improved by dividing its isthmus. If one pole of a horseshoe is badly damaged, it may require surgical resection.

Prognosis

In most cases, the outlook is excellent. If ureteral obstruction and infection occur, renal drainage must be improved by surgical means so that antimicrobial therapy will be effective.

Ectopic Kidney

Congenital ectopic kidney usually causes no symptoms unless complications such as ureteral obstruction or infection develop.

Simple Ectopy

Simple congenital ectopy usually refers to a low kidney on the proper side that failed to ascend normally. It may lie over the pelvic brim or in the pelvis. Rarely, it may be found in the chest. It takes its blood supply from adjacent vessels, and its ureter is short. It is prone to ureteral obstruction and infection, which may lead to pain or fever. At times, such a kidney may be palpable, leading to an erroneous presumptive diagnosis (eg, cancer of the bowel, appendiceal abscess).

Excretory urograms reveal the true position of the kidney. Hydronephrosis, if present, is evident. There is no redundancy of the ureter, as is the case with nephroptosis or acquired ectopy (eg, displacement by large suprarenal tumor). Obstruction and infection may complicate simple ectopy and should be treated by appropriate means.

Crossed Ectopy Without Fusion

In crossed ectopy without fusion, the kidney lies on the opposite side of the body but is not attached to its normally placed mate. Unless two distinct renal shadows can be seen, it may be difficult to differentiate this condition from crossed ectopy with fusion (Figure 32–4). Sonography, angiography, or CT should make the distinction.

Abnormal Rotation

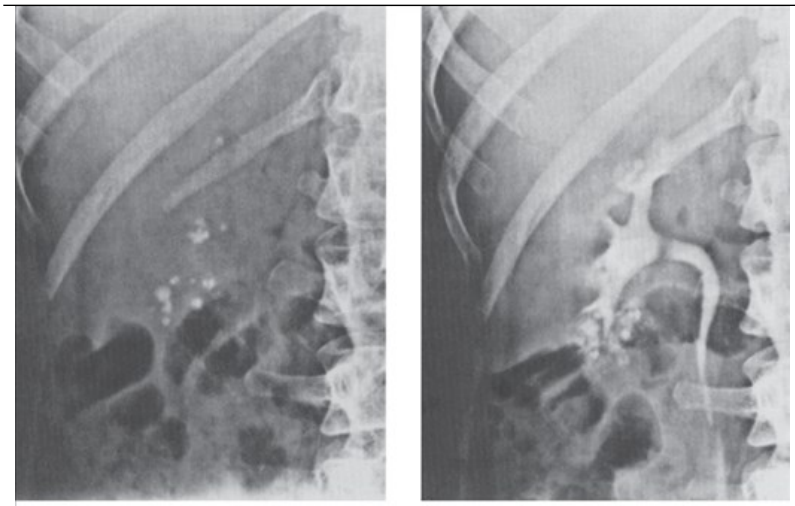
Normally, when the kidney ascends to the lumbar region, the pelvis lies on its anterior surface. Later, the pelvis comes to lie mesially. Such rotation may fail to occur, although this seldom leads to renal disease. Urography demonstrates the abnormal position.

Medullary Sponge Kidney (Cystic Dilatation of the Renal Collecting Tubules)

Medullary sponge kidney is a congenital autosomal recessive defect characterized by widening of the distal collecting tubules. It is usually bilateral, affecting all of the papillae, but it may be unilateral. At times, only one papilla is involved. Cystic dilatation of the tubules is also often present. Infection and calculi are occasionally seen as a result of urinary stasis in the tubules. It is believed that medullary sponge kidney is related to polycystic kidney disease. Its occasional association with hemihypertrophy of the body has been noted.

The only symptoms are those arising from infection and stone formation. The diagnosis is made on the basis of excretory urograms or contrast-enhanced CT scan (Figure 32–5). The pelvis and calyces are normal, but dilated (streaked) tubules are seen just lateral to them; many of the dilated tubules contain round masses of radiopaque material (the cystic dilatation). If stones are present, a plain film will reveal small, round calculi in the pyramidal regions just beyond the calyces.

Figure 32–5.



Medullary sponge kidneys. ***Left:*** Plain film of right kidney showing multiple small stones in its mid portion. ***Right:*** Excretory urogram showing relationship of calculi to calyces. Typically, the calyces are large; the stones are located in the dilated collecting tubules.

The differential diagnosis includes tuberculosis, healed papillary necrosis, and nephrocalcinosis. Tuberculosis is usually unilateral, and urography shows ulceration of calyces; tubercle bacilli are found on bacteriologic study. Papillary necrosis may be complicated by calcification in the healed stage but may be distinguished by its typical calyceal deformity, the presence of infection, and, usually, impaired renal function. The tubular and parenchymal calcification seen in nephrocalcinosis is more diffuse than that seen with sponge kidney (see Figure 16–3); the symptoms and signs of primary hyperparathyroidism or renal tubular acidosis may be found.

There is no treatment for medullary sponge kidney. Therapy is directed toward the complications (eg, pyelonephritis and renal calculi). Only a small percentage of people with sponge kidney develop complications. The overall prognosis is good. A few patients may pass small stones occasionally.

Abnormalities of Renal Vessels

A single renal artery is noted in 75–85% of individuals and a single renal vein in an even higher percentage. Aberrant veins and, especially, arteries occur. An aberrant artery passing to the lower pole of the kidney or crossing an infundibulum can cause obstruction and hydronephrosis. These causes of obstruction can be diagnosed on angiography or spiral CT.

Acquired Lesions of the Kidneys

Aneurysm of the Renal Artery

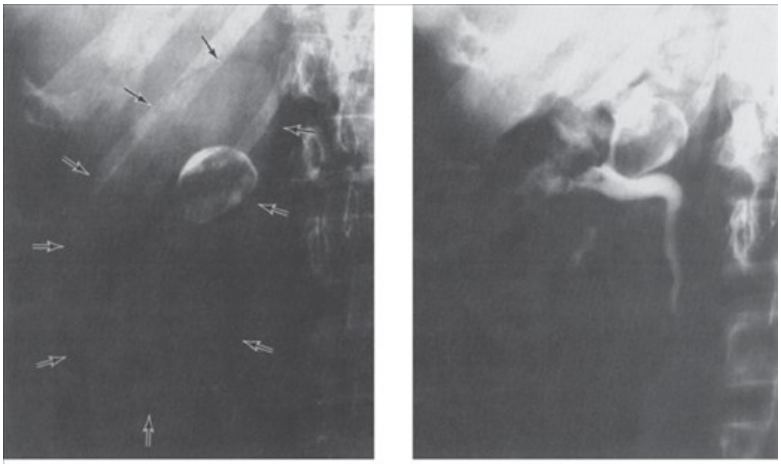
Aneurysm of the renal artery usually results from degenerative arterial disease that weakens the wall of the artery so that intravascular pressure may balloon it out. It is most commonly caused by arteriosclerosis or polyarteritis nodosa, but it may develop secondary to trauma or syphilis. Well over 300 cases have been reported. Congenital aneurysm has been recorded. Most cases represent an incidental finding on angiography.

Aneurysmal dilatation has no deleterious effect on the kidney unless the mass compresses the renal artery, in which case some renal ischemia and, therefore, atrophy are to be expected. A true aneurysm may rupture, producing a false aneurysm. This is especially likely to occur during pregnancy. The extravasated blood in the retroperitoneal space finally becomes encapsulated by a fibrous covering as organization occurs. An aneurysm may involve a small artery within the renal parenchyma. It may rupture into the renal pelvis or a calyx.

Most aneurysms cause no symptoms unless they rupture, in which case, there may be severe flank pain and even shock. If an aneurysm ruptures into the renal pelvis, marked hematuria occurs. The common cause of death is severe hemorrhage from rupture of the aneurysm. Hypertension is not usually present. A bruit should be sought over the costovertebral angle or over the renal artery anteriorly. If spontaneous or traumatic rupture has occurred, a mass may be palpated in the flank.

A plain film of the abdomen may show an intrarenal or extrarenal ringlike calcification (Figure 32–6). Urograms may be normal or reveal renal atrophy. Some impairment of renal function may be noted if compression or partial obstruction of the renal artery has developed. Aortography delineates the aneurysm. Sonography and CT scanning may prove helpful.

Figure 32–6.



Intrarenal aneurysm of renal artery. ***Left:*** Plain film showing calcified structure over right renal shadow. ***Right:*** Excretory urogram relating calcific mass to pelvis and upper calyx. (Courtesy of CD King.)

The differential diagnosis of rupture of an aneurysm and injury to the kidney is difficult unless a history or evidence of trauma is obtained. A hydronephrotic kidney may present a mass, but renal imaging clarifies the issue.

Because a significant number of noncalcified and large calcified aneurysms rupture spontaneously, the presence of such a lesion is an indication for operation, particularly during pregnancy. The repair of extrarenal aneurysms may be considered, but complications (eg, thrombosis) are not uncommon. If an intrarenal aneurysm is situated in one pole, heminephrectomy may be feasible. If it is in the center of the organ, however, nephrectomy is required. Therapeutic occlusion of an aneurysm by intra-arterial injection of autologous muscle tissue has been reported. Those few patients with hypertension may become normotensive following definitive surgery.

Renal Infarcts

Renal infarcts are caused by arterial occlusion. The major causes are subacute infective endocarditis, atrial or ventricular thrombi, arteriosclerosis, polyarteritis nodosa, and trauma. A thrombotic process in the abdominal aorta may gradually extend upward to occlude the renal artery. Renal infarcts may be unilateral or bilateral.

If smaller arteries or arterioles become obstructed, the tissue receiving blood from such a vessel will first become swollen and then undergo necrosis and fibrosis. Multiple infarcts are the rule. If the main renal artery becomes occluded, the entire kidney will react in kind. The kidney may become functionless and atrophic, therefore, as it undergoes necrosis and fibrosis.

Partial renal infarction is a silent disease, but it can result in flank pain and microscopic or gross hematuria. Sudden and complete infarction may cause renal or chest pain and at times gross or microscopic hematuria. Proteinuria and leukocytosis are found. “Epitheluria,” representing sloughing of renal tubular cells, has been noted. Tenderness over the flank may be elicited. The kidney is not significantly enlarged by arterial occlusion.

CT scan may fail to have contrast enhancement in a portion of the kidney with partial infarction; with complete infarction, none of the radiopaque fluid is excreted. If complete renal infarction is suspected, a radioisotope renogram should be performed. A completely infarcted kidney shows little or no radioactivity. A similar picture is seen on CT scans performed after injection of radiopaque contrast medium. Even though complete loss of measurable function has occurred, renal circulation may be restored spontaneously in rare instances. Renal angiography or CT makes the definitive diagnosis. A

dynamic technetium scan will reveal no perfusion of the affected renal vasculature.

During the acute phase, infarction may mimic ureteral stone. With stone, the excretory urogram may also show lack of renal function, but even so, there is usually enough medium in the tubules for a "nephrogram" to be obtained (see Figure 16–3). This will not occur with complete infarction. Evidence of a cardiac or vascular lesion is helpful in arriving at a proper diagnosis.

The complications are related to those arising from the primary cardiovascular disease, including emboli to other organs. In a few cases, hypertension may develop a few days or weeks after the infarction. It may later subside.

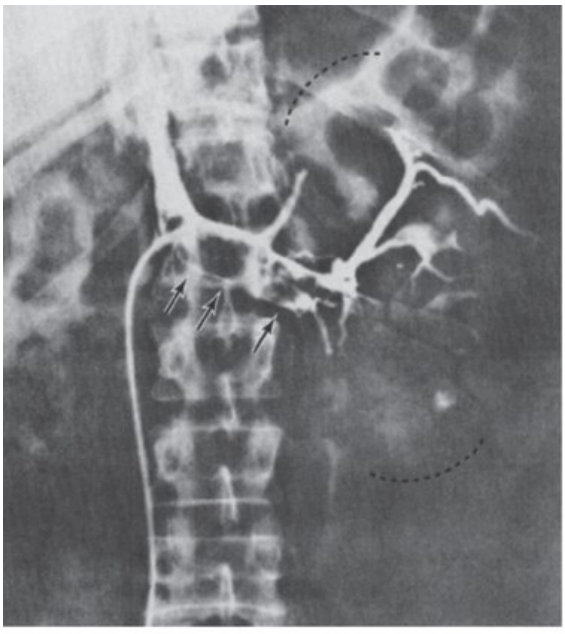
Although emergency surgical intervention has been done, it has become clear that anticoagulation therapy is the treatment of choice. It has been shown that an infusion of streptokinase may dissolve the embolus. Renal function returns in most cases.

Thrombosis of the Renal Vein

Thrombosis of the renal vein is rare in adults. It is frequently unilateral and usually associated with membranous glomerulonephritis and nephrotic syndrome. Invasion of the renal vein by tumor or retroperitoneal disease can be the cause. Thrombosis of the renal vein may occur as a complication of severe dehydration and hemoconcentration in children with severe diarrhea from ileocolitis. The thrombosis may extend from the vena cava into the peripheral venules or may originate in the peripheral veins and propagate to the main renal vein. The severe passive congestion that develops causes the kidney to swell and become engorged. Degeneration of the nephrons ensues. There is usually flank pain, and hematuria may be noted. A large, tender mass is often felt in the flank. Thrombocytopenia may be noted. The urine contains albumin and red cells. In the acute stage, urograms show poor or absent secretion of the radiopaque material in a large kidney. Stretching and thinning of the calyceal infundibula may be noted. Clots in the pelvis may cause filling defects. Later, the kidney may undergo atrophy.

Ultrasonography shows the thrombus in the vena cava in 50% of cases. The involved organ is enlarged. CT scan is also a valuable diagnostic tool; visualization of the thrombus can be noted in a high percentage of cases. Recently, MRI has proved to be a very sensitive diagnostic tool. Renal angiography reveals stretching and bowing of small arterioles. In the nephrographic phase, the pyramids may become quite dense. Late films may show venous collaterals. Venacavography or, preferably, selective renal venography demonstrates the thrombus in the renal vein (Figure 32–7) and, at times, in the vena cava.

Figure 32–7.



Source: McAninch JW, Lue TF: *Smith & Tanagho's General Urology*, 18th Edition: www.accessmedicine.com

Thrombosis of renal vein. Selective left renal venogram showing almost complete occlusion of vein. Veins to lower pole failed to fill. Note the large size of kidney.

The symptoms and signs resemble obstruction from a ureteral calculus. The presence of a stone in the ureter should be obvious; some degree of dilatation of the ureter and pelvis also should be expected. Clot obstruction in the ureter must be differentiated from an obstructing calculus.

While thrombectomy and even nephrectomy have been recommended in the past, it has become increasingly clear that medical treatment is usually efficacious. The use of heparin anticoagulation in the acute phase and warfarin chronically offers satisfactory resolution of the problems in most patients. In infants and children, it is essential to correct fluid and electrolyte problems and administer anticoagulants. Fibrinolytic therapy has also been successful. Renal function is usually fully recovered.

Arteriovenous Fistula

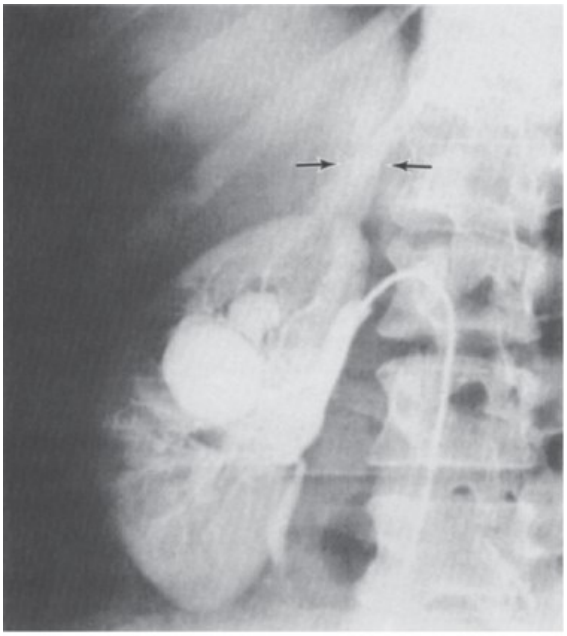
Arteriovenous fistula may be congenital (25%) or acquired. A number of these fistulas have been reported following needle biopsy of the kidney or trauma to the kidney. A few have occurred following nephrectomy secondary to suture or ligature occlusion of the pedicle. These require surgical repair. A few have been recognized in association with adenocarcinoma of the kidney.

A thrill can often be palpated and a murmur heard both anteriorly and posteriorly. In cases with a wide communication, the systolic blood pressure is elevated and a widened pulse pressure is noted. Renal angiography or isotopic scan establishes the diagnosis. CT scan, sonography, and, recently, duplex ultrasound with color flow are particularly helpful. Arteriovenous fistula involving the renal artery and vein requires surgical repair or nephrectomy. Most, however, can be occluded by embolization, balloon, or steel coil. Those that develop secondary to renal biopsy tend to heal spontaneously.

Arteriovenous Aneurysm

About 100 instances of this lesion have been reported (Figure 32–8). Most follow trauma. Hypertension is to be expected and is associated with high-output cardiac failure. A bruit is usually present. Nephrectomy is usually indicated.

Figure 32–8.



Source: McAninch JW, Lue TF: *Smith & Tanagho's General Urology*, 18th Edition: www.accessmedicine.com

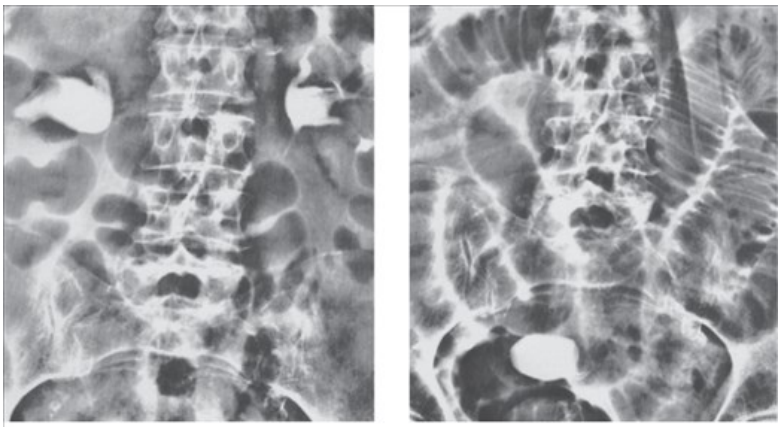
Arteriovenous aneurysm. Selective renal angiogram. Note the aneurysm in center of kidney, with prompt filling of the vena cava (shown by ***arrows***).

Renoalimentary Fistula

Over 100 instances of renoalimentary fistula have been reported. They usually involve the stomach, duodenum, or adjacent colon, although fistula formation with the esophagus, small bowel, [appx], and rectum has been reported.

The underlying cause is usually a pyonephrotic kidney or renal cell carcinoma that becomes adherent to a portion of the alimentary tract and then ruptures spontaneously, thus creating a fistula (Figure 32–9). A few cases following trauma have been reported. The patient is apt to have symptoms and signs of acute pyelonephritis. Urography may show radiopaque material escaping into the gastrointestinal tract. Gastrointestinal series may also reveal the connection with the kidney. The treatment is nephrectomy with closure of the opening into the gut.

Figure 32–9.



Nephroduodenal fistula and small-bowel obstruction from renal staghorn calculus. ***Left:*** Excretory urogram showing nonfunction of right kidney; staghorn stone. ***Right:*** Patient presented with symptoms and signs of bowel obstruction 4 years later. Plain film showing dilated loops of small bowel down to a point just proximal to ileocecal valve. Obstruction due to stone extruded into duodenum. (Courtesy of CD King.)

Renobronchial Fistula

Renobronchial fistulas are rare. They are caused by rupture of an infected, calculous kidney through the diaphragm.

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