Web Training Modules

Module 15: Advanced Abdominal Imaging, Part II

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Kidneys

Anatomy

The kidneys are the waste filtering and disposal system of the body. As much as 20-33% of all blood leaving the heart passes into the kidneys to be filtered before flowing to the rest of the body’s tissues. Our kidneys are vital organs—while a person can live with only one kidney, the loss of both kidneys would lead to a rapid accumulation of wastes, and death within a few days’ time.

The kidneys are paired retroperitoneal structures, normally located between the transverse processes of the T12-L3 vertebrae. The left kidney is typically somewhat more superior in position than the right, due to the large liver on the right side of the body. The kidneys are bean-shaped, with the convex side of each organ located laterally, and the concave side medially. Their upper poles are normally oriented more medially and posteriorly than the lower poles. They are each approximately 4 ½” in length, and 2 ½” in width. The ribs and muscles of the back protect the kidneys from external damage. Adipose tissue known as perirenal fat surrounds the kidneys and acts as protective padding.

A thin layer of fibrous connective tissue forms the renal capsule surrounding each kidney, providing a stiff outer shell to maintain the shape of the soft inner tissues (Figure 1). Deep to the renal capsule is the soft, dense, vascular renal cortex. Seven cone-shaped renal pyramids form the renal medulla deep to the renal cortex. These renal pyramids are aligned with their bases facing outward toward the renal cortex, and their apices pointing inward toward the center of the kidney. Each apex connects to a minor calyx, which is a small hollow tube that collects urine. The minor calyces merge to form 3 larger major calyces, which then merge to form the hollow renal pelvis at the center of the kidney. The renal pelvis exits the kidney at the renal hilus, where urine drains into the ureter. The renal hilus is also the point of entry for the renal artery and renal vein.

![Figure 1 Renal anatomy and renal fascia](image-url)
The renal arteries branch directly from the abdominal aorta, entering the kidneys through the renal hilus, just posterior to the renal vein. The first branch from the renal artery is the inferior suprarenal artery (Figure 2). The renal artery then branches off into 5 segmental branches. These arteries branch into interlobar arteries, which travel in a parallel fashion in between the major calyces. The interlobar arteries branch further into arcuate arteries that run within the cortex, across the bases of the renal pyramids. The arcuate arteries radiate into interlobular arteries, which extend into the cortex of the kidney, to finally become afferent arterioles. (Some of the terminal branches of the interlobular arteries become perforating radiate arteries, which supply the renal capsule). Each afferent arteriole carries blood into the renal cortex, where it separates into a bundle of capillaries known as a glomerulus. From the glomerulus, the blood recollects into smaller efferent arterioles that descend into the renal medulla. The efferent arterioles separate into the peritubular capillaries that surround the renal tubules. The peritubular capillaries merge to form veins, which merge again to form the large renal vein. The renal vein exits the kidney and joins with the inferior vena cava, which carries blood back to the heart.

![Figure 2 Intrarenal arteries](image)

The renal vein is generally anterior to the renal artery at the hilum. The left renal vein is longer than the right, as it crosses the midline to reach the inferior vena cava. Typically, the left gonadal vein drains into the left renal vein inferiorly, while the left suprarenal vein drains into the superior aspect of the left renal vein at approximately the same level. Posteriorly, the left second lumbar vein typically drains into the left renal vein as well. The left renal vein then crosses under the origin of the superior mesenteric artery to reach the inferior vena cava. The right renal vein and gonadal vein drain separately and directly into the inferior vena cava.
Each kidney contains approximately 1 million individual nephrons, which are the microscopic functional units of the kidney that filter blood to produce urine. The nephron has two main parts: the renal corpuscle and the renal tubule (Figure 3). The renal corpuscle is responsible for filtering the blood, and is formed by the capillaries of the glomerulus and the glomerular capsule (or Bowman’s capsule). The glomerulus is a bundled network of capillaries that increases the surface area of blood that is in contact with the blood vessel walls. The glomerular capsule surrounds the glomerulus. This capsule is a cup-shaped double layer of simple squamous epithelium with a hollow space between the layers. Special epithelial cells known as podocytes form the layer of the glomerular capsule that surrounds the capillaries of the glomerulus. Podocytes work with the endothelium of the capillaries to form a thin filter to separate urine from the blood that is passing through the glomerulus. The outer layer of the glomerular capsule holds the urine separated from the blood within the capsule. At the far end of the glomerular capsule, opposite the glomerulus, is the mouth of the renal tubule.

![Figure 3 Microanatomy of the nephron](image)

The renal tubule is actually a series of tubes that concentrate urine and recover non-waste solutes from the urine. The renal tubule carries urine from the glomerular capsule to the renal pelvis. The first section of the renal tubule is the curvy proximal convoluted tubule. The cells that line this tubule reabsorb much of the water and nutrients that are initially filtered into the urine. Urine next passes through the loop of Henle, which is a long, straight tubule that carries urine into the renal medulla before making a hairpin turn and returning to the renal cortex. The distal convoluted tubule follows the loop of Henle. Urine from the distal tubules of several nephrons enters the collecting duct, which carries the concentrated urine through the renal medulla, and into the renal pelvis. From the renal pelvis, urine from many collecting ducts combines and flows out of the kidneys and into the ureters.
Physiology

Every minute, 1300 ml of blood enters the kidneys, and 1299 ml leave the kidney, with 1 ml leaving as urine. The kidneys are the major organs that maintain homeostasis in the body, and help control blood pressure. They maintain balance in electrolytes, acid-base, and fluid in the blood. The kidneys remove nitrogenous waste from the body (creatinine, urea, ammonia) and keep essential substances the body needs to function as it should. The kidneys also produce the hormone erythropoietin, which stimulates the production of red blood cells and enzymes.

Excretion of Wastes

The primary function of the kidneys is the excretion of waste products resulting from protein metabolism and muscle contraction. The liver metabolizes dietary proteins to produce energy, and produces toxic ammonia as a waste product. The liver is able to convert most of this ammonia into uric acid and urea, which are less toxic to the body. Meanwhile, our muscles use creatine as an energy source and, in the process, produce the waste product creatinine. Ammonia, uric acid, urea, and creatinine all accumulate in the body over time, and need to be removed from circulation to maintain homeostasis. The glomeruli in the kidneys filter all four of these waste products out of the bloodstream, and they are excreted from our bodies in urine. Approximately 50% of the urea found in the blood is reabsorbed by the tubule cells of the nephron and returned to the blood supply. Urea in the blood helps to concentrate other more toxic waste products in urine by maintaining the osmotic balance between urine and blood in the renal medulla.

Filtration, Reabsorption, and Secretion

Blood containing urea and metabolic waste products enters the kidneys from the liver. The blood is mechanically filtered to remove fluids, wastes, electrolytes, acids and bases into the tubular system, while leaving blood cells, proteins, and chemicals in the bloodstream. The nephrons also reabsorb and secrete ions that control fluids and electrolyte balance.

Blood enters the kidney and goes to the glomerulus. Pressure forces fluid out of the blood through membrane filtration slits, creating a cell-free fluid of water and small molecules that enter into the renal tubule, while large cells and proteins stay in the blood. This tubular filtrate moves to the proximal convoluted tubule, which has permeable cell membranes that reabsorb glucose, amino acids, metabolites, and electrolytes into nearby capillaries, and allows for circulation of water. The filtrate runs down into the medulla, at which point water content has been reduced by 70%, and it contains high levels of salts. It moves into the loop of Henle, where more water is removed, which further concentrates the filtrate. It then moves to the distal convoluted tubule, where most of the salts are reabsorbed. The remaining filtrate is further modified until it becomes concentrated urine, which contains urea and other waste products. The filtrate ends its journey through the distal convoluted tubule at the collecting duct, which is formed where numerous tubules join together. Numerous collecting ducts combine their concentrated urine at the renal pelvis, where it flows out of the kidney into the ureters for eventual excretion.
Glomeruli, which are the collection of capillaries in the nephron, are responsible for the filtration portion of the kidneys’ work. As the blood is filtered through the glomeruli, a filtrate is formed. The tubules in the nephrons perform the reabsorption, as they reabsorb the filtered blood in nearby blood vessels. Secretion occurs as the filtrate passes through the tubules to the collecting ducts, and on to the bladder.

The glomerular filtration rate (GFR), a value that is typically examined before the injection of gadolinium contrast to assess kidney function, measures the rate at which the glomeruli filter the blood, or the flow rate of filtered fluid through the kidney. A suggested normal value is approximately 120 ml/minute. The most accurate measure of the GFR is done by measuring creatinine clearance, which is the complete removal of creatinine from the blood. Creatinine is a good measure to use, as it is filtered by the blood but not reabsorbed by the tubules.

**Water Homeostasis**

The kidneys control the volume of water in the body by changing the reabsorption of water by the tubules of the nephron. Under normal conditions, the tubule cells of the nephron tubules reabsorb nearly all of the water that is filtered into urine by the glomerulus. Water reabsorption leads to very concentrated urine and the conservation of water in the body. The hormones aldosterone and anti-diuretic hormone both increase the reabsorption of water until almost 100% of the water filtered by the nephron is returned to the blood. In situations where there is too much water in the blood, the heart secretes the hormone atrial natriuretic peptide, in order to increase the excretion of Na⁺ and Cl⁻ ions (salt). An increased concentration of these ions in urine draws more water into the urine, which increases the volume of urine production.

**Electrolyte Homeostasis**

The kidneys maintain the homeostasis of important electrolytes by controlling their excretion into urine, including:

- **Sodium**- Vital electrolyte for muscle function, neuron function, blood pressure regulation, and blood volume regulation; over 99% of the sodium ions passing through the kidneys are reabsorbed into the blood from tubular filtrate
- **Potassium**- Vital for muscle function, neuron function, and blood volume regulation; 60-80% of the potassium ions passing through the kidneys are reabsorbed
- **Chloride**- The most important anion (negatively charged ion) in the body; vital to the regulation of pH and cellular fluid balance; helps to establish the electrical potential of neurons and muscle cells; approximately 90% of chloride ions filtered by the kidneys are reabsorbed
- **Calcium**- Essential electrolyte for contraction of muscle tissue, release of neurotransmitters by neurons, and stimulation of cardiac muscle tissue in the heart; parathyroid hormone increases the reabsorption of calcium in the kidneys when blood calcium levels become too low
- **Magnesium**- Essential electrolyte for the proper function of enzymes that work with phosphate compounds like ATP, DNA, and RNA
**Blood Pressure Homeostasis**

The kidneys control blood pressure in the body by regulating the excretion of sodium ions and water, and by producing the enzyme renin. Since blood is made mostly of water, an increase in the volume of water in the body results in an increase in the volume of blood in the blood vessels. Increased blood volume means the heart has to pump harder to push blood into vessels that are crowded with excess blood, thereby increasing blood pressure. If the body is dehydrated, the volume of blood and blood pressure decrease. The kidneys are able to control blood pressure by either reabsorbing water to maintain blood pressure (reabsorb additional sodium ions), or by allowing more water than usual to be excreted into urine to reduce blood volume and pressure (excrete extra sodium ions).

The kidneys produce the enzyme renin to prevent the body’s blood pressure from becoming too low. When renin is released into the blood, it begins a complex process that ultimately causes the release of the hormone aldosterone by the adrenal glands. Aldosterone stimulates the cells of the kidneys to increase their reabsorption of sodium and water in order to maintain blood volume and pressure.

**Hormones**

The kidneys produce the hormones calcitriol and erythropoietin. Calcitriol is the active form of vitamin D in the body. Tubule cells of the proximal convoluted tubule produce calcitriol from inactive vitamin D molecules. It travels through the bloodstream to the intestines, where it increases the absorption of calcium from food in the intestinal lumen. Erythropoietin is a hormone that is produced by cells of the peritubular capillaries in response to hypoxia, which is a low level of oxygen in the blood. It stimulates the cells of red bone marrow to increase their output of red blood cells. Oxygen levels in the blood increase as more red blood cells mature and enter the bloodstream. Once oxygen levels return to normal, the cells of the peritubular capillaries stop erythropoietin production.

**MR Imaging of Kidney Pathology**

MRI has become a powerful tool in the detection and characterization of renal lesions, due in no small part to the superior soft-tissue contrast that it offers. Fast imaging techniques are essential, due to respiratory motion of the kidneys. Scans should be performed within one breath-hold when possible, or respiratory gating can be used. Imaging should be performed during expiration, as the kidney position is more constant in expiration rather than inspiration.

The main goal in the evaluation of renal lesions is to differentiate surgical lesions from those that are nonsurgical. Most simple cysts are easily recognized and do not need further analysis. Complicated or multiloculated cysts need more attention in order to differentiate them from cystic carcinomas. With most solid renal lesions, neither CT nor MRI is able to reliably distinguish benign from malignant. Some solid lesions, such as angiomyolipomas, can be identified as benign with high confidence. In general, if a lesion cannot be characterized as benign or malignant, it should be considered malignant.
Malignant Renal Lesions

Renal cell carcinomas account for 3% of all malignancies in adults, with almost 50% being detected incidentally. Nearly 85% of suspicious renal lesions are diagnosed as malignant. Features that indicate potential malignancy of a renal lesion include size, presence of calcifications, the distribution of the calcifications within the lesion, wall thickness and the presence of septa in cases of cystic lesions, inhomogeneity of the lesion, extension of the tumor beyond the renal fascia, and enhancement after contrast administration (Figure 4). Research performed on 186 renal tumors found that all tumors greater than 7 cm were malignant. Approximately 80% of tumors smaller than 3 cm were malignant. The differential diagnosis of solid renal lesions smaller than 7 cm consists of oncocytoma, angiomyolipoma, hemangioma, leiomyoma, and focal xanthogranulomatous pyelonephritis. Of these lesions, only cysts and angiomyolipomas can often be positively identified as benign lesions. Less than 5% of renal cell carcinomas are cystic.

![Image of renal lesions](image.png)

Figure 4 Gradient echo images with intravenous gadolinium at baseline (a), 8 months later (b) and 15 months later (c); complicated cortical cyst (arrow) in left kidney progresses into a frank renal cell carcinoma with multiple enhancing internal septations (c); several simple cysts are visible. Images courtesy of Roy S. Dwarkasing
MRI is generally performed only after a renal lesion has been detected by ultrasound or CT. The main MRI feature indicating potential malignancy of a renal tumor is enhancement after intravenous gadolinium administration, differentiating the lesion from a cyst (Figure 5). Enhancement on MRI cannot be “measured” in the same manner as on CT. Subjective assessment of enhancement has been shown to be accurate in detecting renal cell carcinomas. In cystic lesions with only a small solid component, subtraction images may be used to better assess the presence of enhancement. Renal cell carcinomas may be hypovascular, and may display lower enhancement than the surrounding renal parenchyma. Hypovascular lesions, as well as lesions that are hyperintense on T1-weighted imaging, may benefit from the use of subtracted images for diagnosis, rather than subjective assessment alone. Investigators have performed quantitative assessment of enhancement by calculating relative enhancement, which is defined as the signal intensity increase after contrast administration compared to the signal intensity before contrast administration. Relative signal intensity enhancement was used to differentiate cysts from malignant lesions. One study used a threshold of 15% relative signal intensity enhancement after administration of IV gadolinium for 74 patients with renal lesions. Their results demonstrated a sensitivity of 100% (probability that a person with a disease will have a positive test) and specificity of 94% (probability that a person without a disease will have a negative test) in the detection of renal cell carcinoma. The relative enhancement peak was at its maximum between 2 and 4 minutes after gadolinium contrast injection. Investigators also noted that cysts showed a mean enhancement change of up to 5%, which may be attributed to motion artifacts and volume averaging.

Several subtypes of renal cell carcinoma are recognized, including clear cell carcinoma, papillary carcinoma, and chromophobe carcinoma. The most frequent subtype is conventional or clear cell carcinoma, which comprises 88% of all renal cell carcinomas. Papillary carcinomas account for approximately 10% of the subtypes, with chromophobe carcinomas at approximately 2%. A few studies have used MRI to attempt to differentiate renal cell carcinoma subtypes. Clear cell carcinomas may show loss of signal intensity on out-of-phase images compared to in-phase images, due to intracellular lipid. However, this distinction is of little use clinically, as tumors that do not show signal loss can still be clear cell carcinomas.

Renal cell carcinomas may contain macroscopic fat, although this is rare. Intralesional fat has long been considered diagnostic for angiomyolipoma, so its presence may cause confusion. Most of the reported cases of fat-containing renal cell carcinomas have also contained intratumoral calcifications, which are rare in angiomyolipomas. The presence of calcifications in a lesion with macroscopic fat should be a warning that the lesion may be a carcinoma.
Staging of renal cell carcinomas is based on the degree of tumor spread beyond the kidney (Figure 6). Staging is usually performed using CT, but similar accuracy has been found with MRI. Determination of the extent of the tumor into the renal vein and the inferior vena cava is important for the surgical approach. Involvement of the IVC is reported to occur in 4-10% of renal cell carcinoma patients. Gadolinium contrast is indicated for use in differentiation between bland thrombus and tumor thrombus. Enhancement of the thrombus indicates a tumor, while the lack of enhancement indicates a clot.

For the detection of abdominal and pelvic metastases, MRI with ferumoxtran has significantly improved diagnostic precision compared to MRI without this contrast. Ferumoxtran is one of the ultra-small superparamagnetic iron oxide particles used as a negative contrast agent for the detection of small lymph node metastasis. These particles are injected through an IV, and accumulate in healthy lymph nodes. They are ingested by macrophages through phagocytosis, and cause a decrease in signal intensity on T2 and T2*-weighted images. Gradient echo sequences are the most sensitive for these susceptibility effects. Lymph node metastases displace the macrophages in the lymph node, so the loss in signal intensity seen in normal lymph nodes is not seen.
The number of small, incidentally detected renal tumors is increasing, along with the use of MRI for patients being considered for nephron-sparing surgery (Figure 7). Partial nephrectomy preserves more function, and survival after partial nephrectomy for small renal cell carcinomas is comparable to total nephrectomy. The ideal tumor for partial nephrectomy is smaller than 3 cm, is confined to the parenchyma of the kidney, and has a peripheral location. Presence of a pseudocapsule around a renal tumor is a sign of lack of perinephric fat invasion, which is a favorable sign for partial nephrectomy. A pseudocapsule consists of compressed renal tissue and fibrous tissue, and is fairly well depicted on MRI. It presents as a hypointense rim around the tumor on both T1- and T2-weighted images, but is best seen on T2-weighted images, and gadolinium-enhanced gradient echo images.

![Figure 7 T1-weighted gradient echo images before (a) and after (b) gadolinium administration; a mass in the renal pelvis (arrow) shows moderate enhancement after gadolinium administration; a transitional cell carcinoma was suspected, which was confirmed after nephrectomy](image-url)
Benign Renal Lesions

Oncocytomas are benign and usually asymptomatic renal tumors. They represent 2-12% of renal masses. On MRI, oncocytomas show variable low signal intensity on T1-weighted images and heterogeneous high signal intensity on T2-weighted images. In 33-54%, a central scar with low signal intensity on T1- and T2-weighted images is visible (Figure 8). After contrast enhancement, a spoke-wheel pattern may be observed. However, these features are not specific for oncocytomas, as both a central scar and spoke-wheel pattern may also be seen in renal cell carcinoma. Oncocytomas may show a pseudocapsule, which consists of compressed renal parenchyma and fibrous tissue. Again, a pseudocapsule is often seen in renal cell carcinomas. Since the characteristics of oncocytomas and renal cell carcinomas show considerable overlap, the therapy for a suspected oncocyto is usually surgical.

Angiomyolipomas are benign hamartomatous tumors, consisting of fat, smooth muscle, and blood vessels. They are the only solid renal tumors that can be positively characterized using MRI. Angiomyolipomas are identified by demonstrating macroscopic fat in the lesion (Figure 9). Differentiation of these benign tumors is especially urgent in patients with tuberous sclerosis, as angiomyolipomas develop in about 80% of these patients (Figure 10). At the same time, these patients are at an increased risk of developing renal cell carcinomas. Macroscopic fat in a renal lesion can be detected on MRI by using fat suppression techniques. On out-of-phase gradient echo images, macroscopic fat is demonstrated by a hypointense rim surrounding the fat. If the amount of intralesional fat is small, the differentiation between angiomyolipoma and renal cell carcinoma may be difficult. The signal loss displayed by angiomyolipomas on out-of-phase images can also make them difficult to distinguish from clear cell carcinoma, which shows the same signal loss, due to intracellular lipid. In-phase gradient echo images may be helpful, as the fatty portion of angiomyolipomas will be hyperintense, and renal clear cell carcinomas are generally hypo- or isointense. Clear cell carcinomas are also hyperintense on T1-weighted images, so spectral fat-suppression may be needed to prove the presence of macroscopic fat in the angiomyolipoma. Carcinomas may also contain hemorrhage, causing high signal intensity on in-phase T1-weighted images. In these cases, out-of-phase images and spectral fat suppression will not show a drop in signal intensity.
Figure 9 T1-weighted gradient echo image (a), post-contrast fat-suppressed T1-weighted gradient echo image (b), and T2-weighted SSFSE image of left kidney; hyperintense parts of tumor in lower pole (black arrow) on T1-weighted image show drop in signal intensity on post-contrast fat-suppressed T1-weighted image, proving presence of fat, while hypointense parts of tumor enhance after gadolinium; fatty portions are hyperintense on T2-weighted sequence, but not as high as cyst (white arrow) in midportion of kidney; MRI characteristics of tumor in lower pole are consistent with angiomyolipoma.

Figure 10 Lesion in upper pole of right kidney in patient with tuberous sclerosis (arrow); lesion shows low signal intensity on T2-weighted FSE images (a) and intermediate signal intensity on in-phase T1-weighted gradient echo images (b); no signal loss is observed on out-of-phase T1-weighted gradient image (c); lesion shows moderate enhancement after intravenous gadolinium administration (d); pathologic examination after resection of lesion showed an angiomyolipoma; this was an unusual case, as no macroscopic fat was detected on MRI.
Xanthogranulomatous pyelonephritis is a rare chronic pyelonephritis, which may result in severe renal impairment. It is most common in middle-aged women, but may occur in children. It is often accompanied by calculi, calyx obstruction, and parenchymal abscesses. In xanthogranulomatous pyelonephritis, the affected renal parenchyma is replaced by lipid-laden macrophages. This condition can involve the whole kidney, or it may be focal, in which case it may be mistaken for a renal carcinoma. The solid component of the lesion may be isointense or hyperintense on T1-weighted images, which can be attributed to the fatty component. On T2-weighted images, the signal intensity of the solid component is isointense to slightly hypointense. Parenchymal cavities that are filled with fluid and pus show high signal intensity on T2-weighted images, and low signal intensity on T1-weighted images, varying according to the protein concentration in the cavity. In addition, the perirenal fascia may be thickened and show enhancement after gadolinium administration. The absence of hyperintense signal on T2-weighted images of the solid components may be helpful in the differentiation between xanthogranulomatous pyelonephritis and renal tumor.

**MR Urography**

In MR urography, the pyelocalyceal system and the ureters are best visualized using heavily T2-weighted images or T1-weighted images with gadolinium contrast. On the heavily T2-weighted images, the urine in the pyelocalyceal system and ureters is hyperintense, due to its long T2 relaxation time, while the surrounding tissue is hypointense. It is essential that the urinary tract is sufficiently filled with urine for T2-weighted MR urography, which means a diuretic may be necessary. Single shot Fast Spin Echo sequences are suitable for MR urography, as they are fast, and maintain sufficient in-plane resolution. Thin sections can be used for detailed evaluation, and MIPs are useful for an overview of the urinary tract.

When performing contrast-enhanced MR urography, intravenous gadolinium can be combined with a T1-weighted 3D gradient echo sequence. A 3D gradient echo EPI sequence can be used if the patient is unable to hold their breath, as it offers the additional advantage of reduced ghost artifacts from ureteral peristalsis. However, conventional gradient echo images provide higher resolution images than EPI. In contrast-enhanced MR urography, images should be acquired in the excretory phase, typically 5-8 minutes after the intravenous gadolinium injection. Additional use of a diuretic is advisable to increase excretion and to dilute the excreted contrast. If the excreted gadolinium is too concentrated the T2* effect may cause signal loss.

The accuracy of MR urography in assessing renal obstruction is similar to CT urography. However, MR urography is better able to detect perirenal edema as a secondary sign of obstruction. Studies have shown that gadolinium-enhanced MR urography shows renal calculi with considerably higher sensitivity than T2-weighted MR urography. Calculus appears as a signal void on MR urography, which is nonspecific. Blood clots, gas, sloughed papilla, and tumors may also appear as a low signal within the bright signal of urine. If a signal void is not clearly detached from the wall of the pelvis or ureter, additional T1-weighted and contrast-enhanced images are necessary to further characterize the lesion.
MRI of Potential Donor Kidneys

The importance of living kidney donors is increasing, due to the demand for donor kidneys, and the relative shortage of cadaver kidneys. Nephrectomy is increasingly performed by laparoscopic surgery to keep the burden for the donor as low as possible. A thorough pre-operative evaluation of the donor kidney is essential in order to also keep the risks as low as possible. It is important for the surgeon to be informed about the arterial and venous vasculature of the kidney, the presence of accessory vessels, and abnormal vessel locations, such as extrahilar branching and retrocaval vessel positions (Figure 11). The surgeon must also be informed about the presence of an abnormal collecting system, as well as any cysts or tumors.

In addition to the standard parenchymal imaging of the kidney, special attention should be focused on arterial and venous imaging. For the arterial MR angiography, a 3D fast gradient echo with intravenous gadolinium can be used, with thin-section coronal reconstruction. A large flip angle (up to 40°) can be used to minimize background signal around the high signal of the renal arteries. The venous angiography sequence should immediately follow the arterial angiography. The concentration of gadolinium in the renal veins is lower than in the renal arteries, due to the excretion of gadolinium by the kidneys, causing lower contrast of the veins compared to the background. A lower flip angle (15°) can be used to compensate for the lower gadolinium concentration, at the expense of more background signal. The 3D data set can be used for the reconstruction of thin 2D sections for detailed evaluation, as well as for MIP reconstructions of the vessels.

The time-of-flight technique can be used to evaluate renal arteries, but it is not recommended for the detection of accessory renal arteries of small caliber. It can be used to clarify intraluminal filling defects potentially caused by flow artifacts on gadolinium-enhanced MR angiography. Phase contrast imaging can be used for detection and grading of renal artery stenosis, but has limitations in evaluating potential kidney donors.
MRI for Renal Function

Progress continues to be made in the use of MRI for the evaluation of renal function. Renal function is often impaired by renal disease, so measurement of renal function can be used as an indicator of severity of disease and can direct therapy. Serum creatinine and creatinine clearance tests are the simplest renal function tests, but they do not provide information about the function of each individual kidney. This information can be important in case of a living renal kidney donor, prior to nephrectomy, or in case of renal artery stenosis. MRI has the potential to combine the functional and anatomic information about each kidney individually.

Measurement of renal perfusion may be a tool to assess the significance of renal artery stenosis, to assess ischemic nephropathy, and in renal transplant assessment. Arterial spin labeling has also been used to assess renal perfusion. Techniques are also being developed to measure glomerular filtration rate through the use of MRI.

Diffusion Weighted Imaging (DWI)

In renal MRI, DWI has been applied in patients with solid renal masses, pyelonephritis, as well as those in renal failure. ADC (Apparent Diffusion Coefficient) mapping has shown differences between lesions and normal tissue, and research continues on the use of DWI in characterizing different abnormalities. DWI has been applied to differentiate between hydronephrosis and pyonephrosis. In a limited group of patients, DWI showed a hypointense pyelocalyceal system in hydronephrosis, and a hyperintense pyelocalyceal system in pyonephrosis. The hyperintensity in pyonephrosis is thought to be due to the high viscosity of the pus, whereas the free-moving molecules in hydronephrosis cause low signal intensity. Diffusion in the kidney has been shown to be anisotropic, due to the radial orientation of the tubules in the pyramids, and the blood vessels in the renal cortex (Figure 12).

![Figure 12 Diffusion tensor image of right kidney on a 3T system; renal pyramids show lower signal intensity than surrounding parenchyma because of radial orientation of tubules in the pyramids, restricting Brownian motion of water molecules to one direction](image-url)
Adrenal Glands

Anatomy

The adrenal glands, or suprarenal glands, are paired retroperitoneal endocrine organs. They are thin, inverted Y-or V-shaped soft tissue structures, with flat or concave margins (Figures 13, 14). The right and left adrenals lie superior and anteromedial to their respective kidneys. Each of the adrenals is approximately 2 inches in length, and 1 inch in width. Adrenal size is larger in neonates and infants, being almost one-third of the size of the kidney.

![Figure 13 Anterior view of adrenal glands](image13.png)  
![Figure 14 Y-shape of adrenal gland (white arrow)](image14.png)

Each adrenal gland is composed of an outer cortex and an inner medulla (Figure 15). The gland is covered by a collagenous capsule. The cortex is the largest part of the gland, and is subdivided into three zones:

1. **Zona glomerulosa** - outer zone; responsible for the production of mineralocorticoids, mainly aldosterone, which regulates blood pressure and electrolyte balance
2. **Zona fasciculate** - middle zone; responsible for the production of glucocorticoids, predominantly cortisol, which increases blood sugar levels via gluconeogenesis, suppresses the immune system, and aids in metabolism; secretes cortisol both at a basal level and as a response to the release of adrenocorticotropic hormone from the pituitary gland
3. **Zona reticularis** - inner zone; produces gonadocorticoids, and is responsible for administering these hormones to the reproductive regions of the body; most of the hormones released by this layer are androgens, with the main androgen being dehydroepiandrosterone, which is the most abundant hormone in the body; this is starting material for other important hormones produced by adrenal gland, such as estrogen, progesterone, testosterone, and cortisol
The suprarenal medulla is composed of special cells called chromaffin cells, which are organized in clusters around blood vessels. These cells produce epinephrine and norepinephrine. These two hormones prepare the body for the fight or flight response by increasing the heart rate, constricting blood vessels, increasing the metabolic rate, heightening cognitive awareness, and increasing the respiratory rate.

Figure 15 Transverse and microscopic sections of adrenals
The adrenal glands require a large supply of blood, and release hormones directly into the bloodstream. They are among the most extensively vascularized organs in the body. Three sources of arteries maintain blood supply to the suprarenal glands (Figure 16). The superior adrenal arteries are multiple small branches from the inferior phrenic artery. The middle suprarenal artery is a direct branch from the abdominal aorta. An inferior adrenal artery, sometimes multiple, arises from the renal artery on each side. On the left side, venous drainage involves the blood draining through the suprarenal vein to the left renal vein. On the right side, blood drains from the right adrenal vein directly to the inferior vena cava.

![Figure 16 Adrenal arteries and veins](image-url)

**Physiology**

The adrenal glands are responsible for the release of hormones that regulate metabolism, immune system function, the salt-water balance in the bloodstream, and aiding in the body’s response to stress.

**Adrenal Cortex**

The adrenal cortex secretes the following three types of hormones:

1. Mineralocorticoids- most important of these is aldosterone; secreted by the zona glomerulosa
2. Glucocorticoids- predominantly cortisol; secreted by the zona fasciculate and, to a lesser extent, zona reticularis
3. Adrenal androgen- mainly dehydroepiandrosterone; predominantly secreted by the zona reticularis, with small quantities released from the zona fasciculate

All adrenocortical hormones are steroid compounds derived from cholesterol. Cortisol binds to proteins in the blood, and more than 90% of cortisol is transported in the blood in this bound form. Only 50% of aldosterone is bound to protein in the blood. All adrenocortical steroids are degraded in the liver, and predominantly conjugate to glucuronides, with lesser amounts of sulfates formed. About 75% of these degradation products are excreted in the urine, with the remainder excreted in the stool by means of the bile.
Mineralocorticoids

Aldosterone accounts for 90% of mineralocorticoid activity, with some activity contributed by deoxycorticosterone, corticosterone, and cortisol. Aldosterone promotes sodium reabsorption and potassium excretion by the renal tubular epithelial cells of the collecting and distal tubules. As sodium is reabsorbed, water follows passively, leading to an increase in the extracellular fluid volume with little change in the plasma sodium concentration. Persistently elevated extracellular fluid volumes cause hypertension. A phenomenon known as “aldosterone escape” helps minimize further increases in extracellular fluid volume by causing a pressure diuresis in the kidney, which means there is an increase in urinary excretion of water due to an increase of arterial pressure. Without aldosterone, the kidney loses excessive amounts of sodium and, consequently, water, leading to severe dehydration.

As sodium is actively reabsorbed, potassium is excreted. Imbalances in aldosterone thus lead to hypokalemia and muscle weakness if levels are increased, and to hyperkalemia with cardiac toxicity if levels are decreased. Aldosterone also stimulates sodium chloride reabsorption and potassium secretion in the excretory ducts (sweat and salivary glands), which helps prevent excessive salivation and conserve body salt in hot climates. It affects sodium absorption in the intestine, especially the colon, where a deficiency of aldosterone may cause a watery diarrhea from the unabsorbed sodium and water.

Glucocorticoids

Approximately 95% of glucocorticoid activity comes from cortisol, with corticosterone, a glucocorticoid less potent than cortisol, making up the remainder. Cortisol release is almost entirely controlled by the secretion of adrenocorticotropic hormone by the anterior pituitary gland, which is controlled by corticotropin-releasing hormone secreted by the hypothalamus. Normally, these hormones and cortisol secretory rates demonstrate a circadian rhythm. Various stresses stimulate increased adrenocorticotropic hormone and, thus, cortisol secretion. A negative feedback effect of cortisol on the anterior pituitary and the hypothalamus help control these increases and regulate plasma cortisol concentrations. Cortisol has many effects on the body, including:

- Stimulates gluconeogenesis in the liver, and simultaneously decreases glucose use by extrahepatic cells in the body; overall result is an increase in serum glucose (i.e., adrenal diabetes) and increased glycogen stores in the liver
- Decreases protein stores in the body, except in the liver
- Has clinically significant anti-inflammatory effects; blocks the early stages of inflammation and induces rapid resolution of inflammation that is already in progress
- Adversely affects immunity; eosinophil and lymphocyte counts in the blood decrease with atrophy of lymphoid tissue
Adrenal Androgens

The adrenal cortex continually secretes several male sex hormones, including dehydroepiandrosterone and androstenedione, with small quantities of the female sex hormones progesterone and estrogen. Most of the effects result from extra-adrenal conversion of the androgens to testosterone. They likely play a role in early development of the male sex organs in childhood, and they have an important role in women during pubarche, which is the first appearance of pubic hair in a child. Adrenocorticotropic hormone has a definite stimulatory effect on androgen release by the adrenal, so secretion of these hormones parallels that of cortisol.

Adrenal Medulla

The adrenal medulla is quite a different entity from the adrenal cortex. Epinephrine (80%) and norepinephrine (20%), with minimal amounts of dopamine, are secreted into the bloodstream due to direct stimulation by acetylcholine release from sympathetic nerves. These hormones are responsible for an increase in cardiac output and vascular resistance, and for all the physiologic characteristics of the stress response.

Endocrine Diseases

Systemic disease tends to affect the whole of the adrenal glands bilaterally.

Cushing’s Syndrome

Cushing’s syndrome is a multisystem disorder that results from chronic exposure to elevated concentrations of free-circulating glucocorticoids. It can be adrenocorticotropic hormone (ACTH)-dependent or ACTH-independent. When ACTH-dependent, Cushing’s syndrome is caused by a pituitary corticotroph adenoma (80-85% of cases), or an ectopic ACTH-secreting tumor (15-20% of cases). ACTH hyperstimulation results in adrenal hyperplasia that is typically bilateral. It may be either diffuse, which is most common, or nodular. An apparently normal MRI appearance of the adrenal glands, observed in 30% of cases, should not exclude suspicion of histopathological hyperplasia. Diffuse hyperplasia is characterized by diffuse, uniform thickening of the adrenal glands, which maintain a smooth contour. Nodularity is thought to follow long-standing adrenal stimulation. The nodules may be microscopic (micronodular), or visible (macronodular). One or more discrete nodules that are isointense to normal liver on T2-weighted MRI will be seen in macronodular hyperplasia. The internodular cortex is always hyperplastic in such cases.

ACTH-independent Cushing’s syndrome is caused by an autonomous primary adrenal pathology that produces cortisol. Adrenal adenomas and carcinomas account for 95% of cases. Bilateral disease, including primary pigmented nodular adrenal dysplasia (PPNAD) and ACTH-independent macronodular adrenal hyperplasia (AIMAH) is responsible for most of the remainder. PPNAD is a rare cause of Cushing’s syndrome, typically affecting young females. Adrenal glands may be normal or minimally hyperplastic with multiple unilateral or bilateral benign cortical nodules on MRI. The nodules do not normally exceed 0.5 cm, but may be up to 1 or 2 cm in older patients. Nodules are hyperintense on both T1- and T2-weighted imaging, and are macroscopically pigmented. The internodular cortex is atrophic.
AIMAH is another rare cause of Cushing’s syndrome, occurring more frequently in men in their 40’s. It is characterized by massive bilateral adrenal enlargement, together with the appearance of multiple macronodules that measure up to 5 cm in diameter. T1-weighted MRI shows the nodules as being isointense relative to muscle, while on T2-weighted MRI they are hyperintense relative to normal liver. Signal intensity may be lost on in- and out-of-phase imaging.

**Conn’s Syndrome**

Conn’s syndrome, or primary aldosteronism, results from excessive adrenal production of aldosterone. The majority of cases (95%) are due to an autonomous cortical adenoma (aldosteronoma). Most of the remaining cases result from primary idiopathic bilateral hyperplasia. Adrenocortical carcinomas only rarely excrete aldosterone. Identification of the underlying cause of primary aldosteronism has important therapeutic implications, as adenomas are treated surgically, while hyperplasia is managed with drug therapy. Aldosteronomas usually range in size from 0.5 to 3.5 cm. Primary adrenal hyperplasia may be micronodular or macronodular. In macronodular hyperplasia, the presence of a dominant nodule or of a concomitant non-hyperfunctioning adenoma may lead to a false-positive diagnosis of aldosteronoma.

**Addison’s Disease**

Addison’s disease, or primary adrenocortical insufficiency, results from the destruction of at least 90% of the adrenal cortex. The adrenal glands do not produce enough of the hormone cortisol, and often the hormone aldosterone. The acute form is most frequently caused by bilateral adrenal hemorrhage. The subacute form (less than 2 years) is usually attributable to infectious-inflammatory and granulomatous processes. The chronic form of Addison’s disease (more than 2 years) is most often due to idiopathic adrenocortical atrophy (autoimmune process- the immune system may attack the gland). Additional common causes of Addison’s disease include previous granulomatous disease, leaving small glands that are partly or completely calcified; remote adrenal hemorrhage, leaving very dense calcifications with no soft tissue; hemochromatosis, leaving normal or small glands with increase attenuation values on CT. Although less likely, damage to the suprarenal cortex can also occur from a tumor, or through certain infections, such as HIV infection or tuberculosis.

**Primary Adrenogenital Syndrome**

Primary adrenogenital syndrome is a rare inherited disorder based on a congenital enzyme defect in steroid synthesis. This results in absent or deficient cortisol and/or aldosterone production. ACTH stimulation by the pituitary gland is increased, leading to excessive androgen secretion. Imaging studies show adrenal hyperplasia. Enlargement is bilateral, coarse, and tumorous, though the glands retain their normal shape.

**Suprarenal Fatigue**

Suprarenal fatigue can occur when prolonged stress and malnutrition weaken the suprarenal glands. When stress occurs over a prolonged period of time, the adrenal glands can either diminish in size or enlarge. If exposed to an extended period of stress, the suprarenal glands can overproduce hormones that suppress the immune system and create and imbalance in the body’s normal blood sugar levels.
MR Imaging of Adrenal Gland Pathology

Fat-Containing Adrenal Masses

These masses can be classified into 2 main types—those that contain intracellular fat (adenoma) and those with macroscopic fat (myelolipoma). Adrenal masses that contain intracellular fat have been shown to lose signal intensity on out-of-phase images compared with in-phase images, due to the presence of intracellular lipid. Adrenal lesions that contain macroscopic fat demonstrate a loss of signal intensity on fat-saturated images. In addition, a loss of signal intensity on in-and out-of-phase imaging can be seen at fat-water interfaces, typically at the borders of such lesions.

Adrenal Adenoma

Adrenal adenomas are the most common adrenal lesions. They are typically round or oval homogeneous masses with well-defined margins, and are usually smaller than 3 cm. Their most important characteristic feature is the presence of intracellular lipid. Adenomas typically reveal homogeneous signal intensity on all MR pulse sequences, with relative isointensity to the liver on T2-weighted images. The amount of intracellular lipid corresponds to the amount of signal intensity lost on out-of-phase images (Figure 17). A decrease in signal intensity of more than 20% is considered diagnostic of an adenoma. Uniform enhancement on immediate contrast-enhanced images is also typical of adenomas. Small, rounded foci of altered signal intensity may be seen within an adenoma owing to cystic changes, hemorrhage, or variation in vascularity. MRI findings do not usually allow the differentiation of functioning from non-functioning adenomas.

Figures 17 Axial in-phase image of an adrenal adenoma on left, and out-of-phase image on right (white arrows); out-of-phase image exhibits typical decrease in signal intensity
Rarely, adrenal adenomas may contain foci of hemorrhage (Figure 18). The appearance of blood products on MRI varies with their stage of evolution. A lesion that loses a substantial amount of signal intensity on in-phase images compared with out-of-phase images obtained with a shorter echo time may contain blood products.

Figure 18 Axial T1-weighted out-of-phase image shows an adrenal adenoma (black arrow) with a focal area of high signal intensity hemorrhage (white arrow)
Myelolipoma

Myelolipomas are uncommon benign tumors composed of mature adipose tissue and hematopoietic tissue. Most of these lesions are discovered incidentally. The presence of macroscopic fat is critical to the diagnosis because virtually no other adrenal lesion contains fat. The fatty component of this tumor is hyperintense on non-fat-suppressed T1-weighted images (Figure 19). The use of fat suppression can help confirm the diagnosis by demonstrating a loss of signal intensity within the fatty component. Myelolipomas can be categorized into 3 main groups on the basis of their MR imaging features:

1. Homogeneous hyperintense masses on T1-weighted images with intermediate signal intensity on T2-weighted images; these findings are suggestive of lesions that are predominantly composed of fat
2. Heterogeneous masses containing foci with the same signal intensity as that of fat intermixed with focal high-signal-intensity areas on T2-weighted images and contrast-enhanced T1-weighted images; these findings are indicative of mixed fatty and myeloid elements
3. Nodules that are hypointense relative to liver on T1-weighted images and hyperintense relative to liver on T2-weighted images, and that enhance after gadolinium contrast; results in an appearance of focal mass-like areas primarily composed of myeloid cell

Myelolipomas can be large and symptomatic secondary to spontaneous hemorrhage. Large myelolipomas can be confused with other retroperitoneal lipomatous tumors such as liposarcoma.

Cystic Masses

Adrenal cysts are rare, and are often detected incidentally during radiologic investigation or at autopsy. Although they are usually asymptomatic, physical manifestations may consist of pain or palpable mass, especially with larger lesions. Acute symptoms can also occur with cyst hemorrhage, rupture, or infection.
Simple Cysts

Endothelialized cysts are the most common pathologic subtype of adrenal cyst (45%). Simple cysts are hypointense on T1-weighted images and hyperintense on T2-weighted images, with no soft-tissue component and no internal enhancement (Figure 20).

![Figure 20](image)

Figure 20 Coronal T1-weighted image on left, and T2-weighted image on right show an oval, well-circumscribed right adrenal cyst (arrow on right), with a thin wall (arrowhead on right); cyst shows typical appearance of low signal intensity on T1 and high signal intensity on T2

Pseudocysts

Pseudocysts are the second most common cystic lesions of the adrenal gland, accounting for approximately 39% of adrenal cysts. They are more likely to be symptomatic compared to simple cysts. Pseudocysts typically arise after an episode of adrenal hemorrhage, and do not have an epithelial lining. Adrenal pseudocysts have a complicated appearance on MR images, manifesting with septations, blood products, or a soft-tissue component secondary to hemorrhage or hyalinized thrombus (Figure 21).

![Figure 21](image)

Figure 21 Axial T2-weighted MR image obtained with inversion recovery on left shows a left adrenal pseudocyst; note the soft-tissue component in the wall, and the posteriorly located calcification (arrow); coronal T2-weighted image in center, and axial contrast-enhanced image on right show left adrenal mass with areas of signal intensity similar to that of blood; specimen showed hemorrhagic complicated adrenal cyst with hyalinized material
Lymphangioma

Cystic lymphangioma of the adrenal gland is rare and asymptomatic. They can be visualized as thin-walled cystic lesions with low signal intensity on T1-weighted imaging, and high signal intensity on T2-weighted imaging (Figure 22). They do not exhibit substantial internal enhancement.

Hypervascular Lesions

Pheochromocytomas

Pheochromocytomas are tumors that arise from pheochromocytes, the predominant cells of the adrenal medulla. This diagnosis can be established biochemically by the detection of elevated levels of catecholamines and their metabolites in blood plasma and urine. Pheochromocytomas do not contain a substantial amount of cytoplasmic lipid, and therefore they maintain their signal intensity on out-of-phase gradient echo images. Most of these tumors demonstrate high signal intensity on T2-weighted images (light bulb sign), and low signal intensity on T1-weighted images, then show intense enhancement after the administration of contrast material (Figure 23). Pheochromocytomas are called the “10% tumor”, because approximately 10% are bilateral, 10% are extraadrenal, 10% occur in children, and 10% are malignant.
Adreniform Adrenal Masses

Adrenal Cortical Hyperplasia

Adrenal cortical hyperplasia is often seen in patients with Cushing syndrome, which results from hyperproduction of cortisol. The hyperplasia may be diffuse or nodular, and is typically bilateral. The signal intensity of hyperplastic adrenal glands is typically similar to normal adrenal glands, but signal intensity may decrease on out-of-phase images, especially in patients with adenomatous cortical nodules (Figure 24). Bilateral cortical hyperplasia is seen in 45% of patients with Cushing syndrome, whereas nodular cortical hyperplasia is seen in only 3% of these patients.

Figure 24 Coronal in-phase image on left, out-of-phase image on right show bilateral large adreniform masses (black arrows), which represent adrenal cortical hyperplasia
Adrenal Hemorrhage

Adrenal hemorrhage can occur in the setting of trauma, adrenal vein thrombosis, stress, hypotension, and various blood disorders. Adrenal insufficiency (Addison disease) can be a secondary effect of bilateral adrenal hemorrhage. Because MR imaging features vary according to the age of the hematoma, it is the most sensitive and specific modality for diagnosing adrenal hemorrhage. Acute hemorrhage (days 1-6) will be rich in deoxyhemoglobin, which is isointense relative to muscle on T1-weighted images, and has low signal intensity on T2-weighted images. Subacute blood (1-5 weeks after onset) in the form of methemoglobin is hyperintense on T1-weighted images (Figure 25). Initially, methemoglobin is intracellular and has low signal intensity on T2-weighted images. As the red cells lyse and the methemoglobin becomes extracellular, it has high signal intensity on T2-weighted images. Chronic hemorrhage (after 5 weeks) has low signal intensity on both T1- and T2-weighted images due to the presence of hemosiderin. T1-weighted fat-saturated images are quite sensitive in the detection of methemoglobin. Gradient echo images can magnify the susceptibility effects of decreased signal intensity seen with hemosiderin and deoxyhemoglobin, thereby increasing their conspicuity. Calcifications may develop a few months after adrenal hemorrhage.

Figure 25 Axial unenhanced T1-weighted 3D image demonstrates a right adrenal gland with a high signal intensity rim (white arrows), a finding that is consistent with subacute hematoma
Malignant Neoplasms

Adrenocortical Carcinoma

Primary carcinoma of the adrenal gland is a rare tumor, with peak prevalence in patients 30-70 years of age. The tumor is generally large at diagnosis, with diameters of more than 6 cm, and up to 20 cm. Adrenocortical carcinoma can manifest as a hyperfunctioning mass causing Cushing syndrome or Conn syndrome (also known as primary aldosteronism), and as an abdominal mass or abdominal pain. These tumors appear heterogeneous on both T1- and T2-weighted MR images, owing to the presence of internal hemorrhage and necrosis. Hemorrhagic byproducts, principally methemoglobin, can result in areas of high signal intensity within the lesion on T1-weighted images. Areas of necrosis have high signal intensity on T2-weighted images (Figure 26). Adrenocortical carcinomas can contain foci of intracytoplasmic lipid, which results in a loss of signal intensity on out-of-phase images. Large tumors of this type tend to invade the adrenal vein and inferior vena cava.

Figure 26 Sagittal T1-weighted 3D image on left, and coronal T2-weighted image on right show a large mass involving the right adrenal gland; the mass exhibits heterogeneous low signal intensity on the T1-weighted image on the left, and high signal intensity with areas of necrosis (arrow in right image) in the T2-weighted image on the right, indicating adrenocortical carcinoma
Adrenal Lymphoma

Primary adrenal lymphoma is rare; however, secondary involvement of adrenal glands by lymphoma is reported in 1-4% of cases. Non-Hodgkins lymphoma is more common than Hodgkin’s disease at this site. Bilateral involvement is seen in 50% of patients, who may present with Addison’s disease (Figure 27). Lymphoma is usually characterized as an area of low signal intensity on T1-weighted images, and as an area of heterogeneous high signal intensity on T2-weighted images, with minimal progressive enhancement after administration of contrast material.

Figure 27 Axial T1-weighted in-phase image on left, and out-of-phase image on right show bilateral lymphomatous deposits; deposits have low signal intensity, which does not decrease on out-of-phase image; diffuse large cell lymphoma that manifested as an adrenal mass
Metastases

Metastases are the most common malignant lesions involving the adrenal gland, and are found in as many as 25% of patients with known primary lesions. Common primary sites of tumors that metastasize to the adrenal glands include the lung, bowel, breast, and pancreas. Renal cell carcinomas and melanomas may also metastasize to the adrenals. Adrenal metastases are usually bilateral. They typically exhibit low signal intensity on T1-weighted images, and high signal intensity on T2-weighted images, with progressive enhancement after contrast administration (Figure 28). The most important diagnostic feature is the lack of signal loss on out-of-phase images, in contrast to what is seen with adrenal adenomas.

![Figure 28 Axial T2-weighted inversion recovery image on left, and contrast-enhanced T1-weighted image on right show metastasis from renal cell carcinoma, which has a central area of necrosis](image)

Pediatric Neoplasms

Neuroblastoma is the most common extracranial solid tumor in children. Other adrenal tumors, such as pheochromocytoma, adrenocortical carcinoma, and lymphoma are rare in children.
Neuroblastoma

Neuroblastoma is the second most common pediatric abdominal mass, representing approximately 5-15% of all malignant tumors in children. It arises from the neural crest in the adrenal medulla or along the sympathetic chain. In general, neuroblastoma is clinically silent until it invades or compresses adjacent organs, metastasizes, or causes paraneoplastic syndromes. Neuroblastoma usually demonstrates heterogeneous low signal intensity on T1-weighted images, high signal intensity on T2-weighted images, and enhancement after administration of contrast. Calcification is present in 80-90% of the lesions, but may be difficult to discern on MRI. Areas of intratumoral hemorrhage typically have high signal intensity on T1-weighted images (Figure 29). Cystic changes have high signal intensity on T2-weighted images. MRI has been found to be more sensitive than CT in the diagnosis of these tumors, owing to its higher soft tissue contrast resolution and multiplanar capabilities, allowing for excellent detection of the tumor origin.

Figure 29 Coronal unenhanced T1-weighted image on left, and axial T2-weighted inversion recovery image on right show a right adrenal tumor; tumor is predominantly hypointense on T1-weighted image, with areas of high signal intensity hemorrhage (arrow); tumor is hyperintense on T2-weighted image
Ganglioneuroblastoma

The malignant potential of ganglioneuroblastoma is intermediate - between that of neuroblastoma and ganglioneuroma. Similar to neuroblastoma, ganglioblastomas also arise from the neural crest. They tend to be smaller and more well-defined than neuroblastoma at diagnosis. They usually demonstrate intermediate signal intensity on T1-weighted images, and heterogeneously high signal intensity on T2-weighted images, with heterogeneous moderate enhancement after administration of contrast (Figure 30).

MRI is an excellent imaging tool in the diagnosis, evaluation, and characterization of various adrenal lesions and abnormalities. Certain imaging features can be helpful for suggesting a diagnosis. Adenomas and metastases are common, and a decrease in signal intensity on out-of-phase images can be used to differentiate between them. Carcinoma is a possible diagnosis if that decrease in signal intensity is heterogeneous. Benign disease is diagnosed if macroscopic fat or homogeneous cyst-like lesion is seen.

Figure 30 Axial in-phase T1-weighted image shows a heterogeneous mass with intermediate signal intensity involving the right adrenal gland; lobulated ganglioneuroblastoma with areas of necrosis and compression of adjacent kidney
Appendix

Anatomy

Located in the right iliac region of the abdomen, the appendix is a narrow pouch of tissue extending from the inferior end of the large intestine’s cecum, where the small intestine empties its contents. It does not elongate as rapidly as the rest of the colon, thus forming a worm-like structure that inspired the name “vermiform” (or worm-like) appendix (Figure 31). The average length of the appendix is 8-10 cm (3-4 inches), but its length can range from 2-20 cm. It is typically less than ½ inch in width. The cavity of the appendix is much narrower where it joins the cecum than at its closed end. The base of the appendix is consistently located at the posteromedial wall of the cecum, approximately 2.5 cm below the ileocecal valve. While the appendiceal base is in a constant location, the position of the tip of the appendix varies widely. In 65% of patients, the tip is located in a retrocecal position; in 30%, it is located at the brim or in the true pelvis; in 5%, it is extraperitoneal, situated behind the cecum, ascending colon, or distal ileum. The location of the tip of the appendix determines early signs and symptoms of appendicitis.

Figure 31 Location of appendix (courtesy of Encyclopaedia Britannica, Inc.)
Like the rest of the digestive tract, the appendix is made of an inner layer of mucosa with submucosa, muscularis, and serosa layers surrounding it. Unlike the rest of the large intestine, however, the submucosa of the appendix contains many masses of lymphoid follicles. These follicles enlarge, peak between ages 12 and 20, then decrease, correlating with the incidence of appendicitis. This suggests that the appendix may play a role in the immune system, in addition to the digestive system. The muscular walls of the appendix are ordinarily capable of expelling into the cecum the mucous secretions of the appendiceal walls, or any of the intestinal contents that have worked their way into the structure. If anything blocks the opening of the appendix, or prevents it from expelling its contents into the cecum, appendicitis may occur (Figure 32).

![Figure 32 Appendix showing inflamed and damaged tissue due to acute appendicitis (courtesy of Ed Uthman, M.D.)](image)

Blood supply to the appendix is mainly from the appendicular artery, a branch of the ileocolic artery, which is from the superior mesenteric artery. The appendicular artery courses posteriorly to the terminal ileum in the free wall of the mesoappendix, which is the fold of peritoneum around the appendix. An accessory appendicular artery can branch from the posterior cecal artery. Damage to this artery can lead to significant intraoperative and postoperative hemorrhage.

### Physiology

The appendix is not a vital organ, and medical researchers still debate its exact function. One hypothesis suggests that it is a non-functioning remnant of a once larger cecum, which would have been used by our vegetarian ancestors to digest cellulose from plants. Another hypothesis suggests that it houses and cultivates beneficial gut flora that can help a person recover more rapidly from illness by enabling the bacteria to re-colonize the intestines after the illness has passed. Another thought is that the appendix provides a site for the production of endocrine cells in the fetus that produce molecules important in regulating homeostasis. It may also play a possible role in immune function during the first 3 decades of life by exposing leukocytes (white blood cells) to antigens in the gastrointestinal tract. This action would stimulate antibody production that may help modulate immune responses in the gut. While the specific functions of the appendix remain unclear, there is general agreement among scientists that the appendix is gradually disappearing from the human species over evolutionary time.
Pathology

Appendicitis

Acute appendicitis remains one of the most common surgical diseases encountered by physicians. When appendicitis manifests in its “classic” form, it is easily diagnosed and treated. Classic symptoms include pain and tenderness near the navel that grows sharper and spreads downward into the lower right abdomen; nausea, vomiting, and fever; “rebound tenderness”, in which the application of pressure to the area causes pain that sharpens after the pressure is released. Unfortunately, these classic symptoms occur in just over half of patients with acute appendicitis. If the appendix is in an abnormal position, the pain from an appendicitis attack may be in a different or misleading location. Symptoms become difficult to distinguish from the abdominal pain caused by a variety of other diseases. Atypical presentations of the appendix often lead to a delay in diagnosis, perforations, prolonged hospitalization, and increased morbidity.

Appendicitis typically progresses through the following four stages:

1. Acute or focal appendicitis
2. Suppurative appendicitis
3. Gangrenous appendicitis
4. Perforated appendicitis

The basic pathophysiology of appendicitis is obstruction of the lumen of the appendix followed by infection. Obstruction caused by hyperplasia of the submucosal follicles occurs in 60% of patients. This is mainly observed in children, and is known as catarrhal appendicitis. A fecalith or fecal stasis causes luminal obstruction 35% of the time, and is usually observed in adults. Obstruction can also be caused by foreign bodies (4%) and tumors (1%). Following obstruction, an increase in mucus production occurs, leading to increased intraluminal pressure. Because of the increased pressure and stasis from obstruction, bacterial overgrowth occurs. The mucus then turns into pus, which causes a further increase in luminal pressure. This leads to distention of the appendix and visceral pain, typically located in the epigastric or periumbilical region. As the luminal pressure continues to increase, lymphatic obstruction occurs, leading to edema in the appendiceal wall. The overlying parietal peritoneum becomes irritated, causing the pain to localize to the right lower quadrant. This series of events results in the classic migrating abdominal pain described in patients with appendicitis. This stage is known as acute or focal appendicitis (Figure 33).

Figure 33 Axial (on left) and coronal (on right) contrast-enhanced T1-weighted images show the enlarged appendix (arrow) with a diameter of 8mm, and an enhancing wall.
Further increases in pressure lead to venous obstruction, causing edema and ischemia of the appendix. The ensuing bacterial invasion of the wall of the appendix is known as acute suppurative appendicitis (Figure 34). Finally, with continued increases in pressure, venous thrombosis and arterial compromise occur, leading to gangrene and perforation, which are the third and fourth stages of appendicitis (Figures 35, 36).

Figure 34 Contrast-enhanced, fat-suppressed, T1-weighted coronal image of acute suppurative appendicitis; markedly enhanced and thickened inflamed appendix (arrows) with pericecal enhancement due to the extent of inflammation

Figure 35 Gangrenous appendix in a pregnant patient; axial T2-weighted image shows a dilated, fluid-filled appendix (arrow) to the right of the uterus, with appendiceal wall thickening to 5mm

Figure 36 Perforating appendicitis, with image on left at location superior to image on right; both are contrast-enhanced T1-weighted images, showing extensive peritoneal enhancement in the right lower pelvis surrounding the perforated appendix; appendix appears as an enhancing tubular structure on the left (arrows), and has a thickened and enhancing wall on the right (arrow)
If the appendix ruptures, fecal matter leaks out of the cecum. Left untreated, the bacteria-laden fecal matter spreads throughout the abdominal cavity, where the bacteria begin to digest the peritoneum that lines the cavity. The infection and inflammation of the peritoneum, known as peritonitis, is a severely painful and potentially fatal consequence of appendicitis. Fortunately, peritonitis is usually prevented by the protective mechanisms of the body. The omentum, which is an abdominal sheet of fatty tissue, often wraps itself around the inflamed appendix, and an exudate that normally develops in the areas of inflammation behaves like glue and seals off the appendix from the surrounding peritoneal cavity. Once the perforation is sealed off, the pain may improve. However, underlying symptoms may not completely resolve, and diffuse peritonitis may develop.

The outcome following appendectomy for acute or suppurative appendicitis is excellent. Prolonged hospitalization and additional diagnostic and therapeutic procedures may be required when perforated appendicitis is encountered.

Stump appendicitis is an uncommon late complication of appendectomy. It is residual or progressive acute inflammation in the remaining stump of the appendix after surgery. It is a relatively infrequent occurrence, but a high level of suspicion should be maintained for patients who have signs and symptoms of appendicitis and have had a prior appendectomy. Findings that include a distended appendicular stump, fecalith, pericecal fat stranding, or abscess help to confirm the diagnosis. Consequences of a delayed diagnosis include stump necrosis, gangrene, and perforation, which occur in as many as 40% of patients.

A clinical review of cases of periappendicitis showed that there were significant clinical differences, including longer duration of pain, localization less often in the right lower quadrant, and fewer peritoneal signs, compared with patients with classic acute appendicitis. Suspected periappendicitis has been attributable to a variety of processes, including gonococcal and chlamydial salpingitis, yersiniosis, Meckel diverticulitis and associated intraperitoneal abscess, urologic disorders, colonic neoplasms, infectious colitis, abdominal aortic aneurysm, bacterial peritonitis, and GI perforation.

Chronic appendicitis is an umbrella diagnosis that encompasses any type of potentially chronic inflammatory condition of the appendix other than classic acute appendicitis. This terminology has been used to describe fibrous replacement of the appendiceal wall after severe or recurrent bouts of acute appendicitis, which has also been referred to clinically as subacute appendicitis. Causes of chronic appendicitis include inflammatory bowel disease, interval appendicitis, diverticular disease, sarcoidosis, granulomatous appendicitis, infections, and cystic fibrosis.

**Carcinoid Tumors**

Carcinoid tumors are slow growing tumors that can develop anywhere in the gastrointestinal tract. They occur when there is an overgrowth of enterochromaffin cells, which are the cells in the body that produce hormones. Carcinoid tumors excrete a large amount of serotonin, histamine, dopamine or prostaglandins, which dilate the blood vessels and cause facial flushing, diarrhea, wheezing, low blood pressure and heart palpitations. Benign carcinoid tumors often go undiagnosed, while malignant carcinoid tumors may spread to other parts of the body through the blood stream or lymphatic system. Carcinoid tumors can cause carcinoid syndrome, which is a rare, but debilitating condition in which the symptoms are often more uncomfortable than the actual tumor.
Appendiceal Carcinoma

Appendiceal carcinoma is a rare form of colorectal cancer that strikes the appendix. The growth of cancer in the appendix can lead to a blockage resulting in appendicitis. Appendiceal carcinoma can also spread to the stomach. The initial diagnosis of appendiceal carcinoma is often delayed because its symptoms are similar to appendicitis.

Adenomas

Adenomas are benign tumors that may develop in and around the appendix. They are the result of an overgrowth of epithelial cells. They occasionally turn malignant, but the main concern with adenomas is that their growth will compress the appendix and lead to appendicitis. Adenomas can develop in other areas of the body, such as on the pituitary or thyroid gland. Because, they develop on glandular areas, they secrete hormones.

Diverticular Disease

Diverticular disease of the appendix is rare, with the congenital form of this disease more rare than the acquired disease. It is seen most often in older males, as well as among patients with cystic fibrosis, as their mucosal secretions are markedly thickened. Diverticular disease may be asymptomatic, or may manifest with acute or chronic pain, mimicking acute appendicitis. During the acute phase of illness, the incidence of perforation is approximately three times higher than that for patients with classic appendicitis (33% vs. 10%). Acquired diverticula lack a muscular wall, so perforation is not surprising.

Ulcerative Colitis and Crohn’s Disease

Ulcerative appendicitis, which represents appendiceal involvement in patients with ulcerative colitis, is typically seen in patients with pancolitis, but it may also occur as a skip lesion in patients with subtotal, left-sided, or rectal-only disease. It has an overall incidence of 50% among patients with ulcerative colitis. Appendectomy is a protective factor in ulcerative colitis.

Most appendices removed from patients with Crohn’s disease of the small and large intestine are histologically normal. Patients with appendiceal involvement in Crohn’s usually have extensive ileocolonic involvement as well (Figure 37). Appendiceal involvement in Crohn’s disease is approximately 20%.

Figure 37 Contrast-enhanced T1-weighted image shows intense enhancement (circled area) of the terminal ileal wall and the adjacent small bowel mesentery, findings suggestive of active Crohn disease
Pseudomyxoma Peritonei

Pseudomyxoma peritonei is a rare malignant growth characterized by the progressive accumulation of mucus-secreting (mucinous) tumor cells within the abdomen and pelvis. This disorder develops after a small polyp located within the appendix bursts through the wall of the appendix, and spreads mucus-producing tumor cells throughout the surrounding surfaces (e.g., the abdominal peritoneum). As mucinous tumor cells accumulate, the abdominal area becomes swollen and gastrointestinal function becomes impaired. Pseudomyxoma peritonei develops at a variable rate, but may grow at a slower rate than other malignancies within the abdomen. Similar disorders are referred to as mucinous adenocarcinoma of the appendix, mucinous neoplasms, or mucoceles of the appendix.

MR Imaging of Appendix Pathology

General Appendicitis

Acute appendicitis is a common clinical condition that affects both children and adults, and is the most common pathology that employs MRI of the appendix. The frequency of this diagnosis is increasing, with a life time risk as high as 9%. Only half of patients present with the classic symptoms of periumbilical pain migrating to the right lower quadrant, and nausea with vomiting. Localization of pain may occur anywhere in the abdomen, particularly in children and pregnant women, and many other acute abdominal diseases present with similar signs and symptoms. Early surgery based on clinical findings alone leads to a significantly higher negative laparotomy rate than when cross-sectional imaging is used to evaluate the patient.

Imaging has played a central role in the diagnosis of appendicitis. Ultrasound is rapid, inexpensive, and has no known safety-related contraindications. It is particularly well suited for children and pregnant patients. However, it is also highly operator, system, and patient dependent, and the anatomic location of the appendix can limit its utility. The high number of ultrasound’s “inconclusive” cases frequently necessitates a second imaging study for many patients. Ultrasound is also limited for the diagnosis of alternative conditions, which are more frequent than appendicitis. CT has become the modality of choice for the workup of most patients with suspected appendicitis. At many institutions, CT is the first-line imaging modality. It is highly effective for the evaluation of alternative diagnoses, but CT requires the use of ionizing radiation, which raises concerns about its use in children and pregnant patients. In addition, most institutions use IV iodinated contrast agents, which carry a low but finite risk of nephrotoxicity and allergic reactions. Historically, ultrasound has been the preferred initial examination for children. For pregnant women, recent data support the use of MRI after an equivocal or inconclusive ultrasound.

The increasing prevalence of and access to MRI have allowed emergency departments to shift toward it as a primary cross-sectional imaging tool for appendicitis. Higher temporal and spatial resolutions can be achieved, and motion-related artifacts have been reduced. The safety profile of gadolinium-based contrast agents exceeds that of iodinated contrast agents. The overall cost of MRI has decreased and now approaches that of CT, or combined CT and ultrasound, in many institutions. MRI combines the advantages of ultrasound (i.e., it is noninvasive and lacks ionizing radiation) with the high-resolution 3D cross-sectional information of CT. MRI has become increasingly attractive as a safe and effective imaging alternative for suspected appendicitis, especially for children, young adults, and pregnant women, and potentially also in the future for the general adult population.
Some institutions are performing contrast-enhanced MRI on patients with nontraumatic abdominal pain and suspected appendicitis (Figure 38). Recommended anatomic coverage includes the peritoneal cavity, kidneys, and gallbladder, where most abnormalities that cause acute abdominal pain are found. In certain circumstances extended coverage that includes the entire perineum or liver may be needed. Breath-holding, at end-tidal volume, or respiratory gating should be used to minimize motion-related artifacts. The use of gadolinium-based contrast agents is helpful in the confirmation of alternative diagnoses. However, contrast is contraindicated for pregnant patients, and for patients with renal failure, due to concerns for nephrogenic systemic fibrosis. Unenhanced MRI is accurate for the diagnosis of equivocal appendicitis in nonsedated pediatric patients.

Figure 38 Appendicitis (arrow) seen on T1-weighted contrast-enhanced MR image
T2-weighted single shot FSE sequences in at least two orthogonal planes are recommended for MRI of the appendix, as they provide an excellent motion-insensitive overview of the abdomen and appendix. These sequences depict abnormal thickening, mucosal edema, and periappendiceal inflammation as hyperintensities. Multiple imaging planes are essential, as there are multiple variations in the position and length of the appendix. Multiple planes are often complementary in displaying the appendix in both the short and long axes. A Balanced Steady state Acquisition with Rewound Gradient Echo (Balanced SARGE or BASG) sequence can be used as an effective method for depiction of appendiceal edema and extraintestinal findings, such as mesenteric lymphadenopathy, inflammatory changes, and abscess formation. It is relatively insensitive to motion, so it can be used as a primary sequence in patients with reduced compliance, such as pediatric patients and those in severe pain. Fat suppression should be added to at least one of the single shot FSE sequences for the depiction of submucosal edema, per-appendicular fluid collections, and periappendiceal inflammatory changes. Fat suppression leads to a low signal intensity appearance of fat, with clear delineation of adjacent edema or even abscess formation next to the appendix (Figure 39). The use of fat suppression also plays a critical role in the identification and characterization of other pathologic abnormalities, including the presence of intraperitoneal blood, which can have the same signal intensity as fat on sequences that are not fat suppressed.

Figure 39 Appendicitis on T2-weighted images without fat sat on left, and with fat sat on right; bowel wall edema can be nicely depicted on T2 fat sat image
The dynamic contrast-enhanced acquisition is typically performed as an axial T1-weighted fat-saturated Reverse Steady state Acquisition with Rewound Gradient Echo (Reverse SARGE or RSSG). This unenhanced sequence is important for characterizing tissues of intrinsic high T1 signal, such as blood products and other structures or fluid with high protein content. It can be used to characterize abnormalities such as hemorrhagic ovarian cysts, which are a common diagnostic consideration in premenopausal female patients with acute abdominal pain. When performed post-contrast, the RSSG sequence helps to show abnormal enhancement of an inflamed appendix, as well as depiction of mucosal discontinuity with appendiceal rupture. Research has shown that peak small bowel enhancement occurs at approximately 50 seconds, so T1-weighted post-contrast images should be acquired at that time, with additional acquisitions over 2-3 minutes. Coronal contrast-enhanced sequences are invaluable for evaluation of the arteries and veins of the abdomen and pelvis, to evaluate for aortoiliac pathologic abnormalities, and for deep venous thrombosis. The use of fat suppression makes these sequences ideal for visualization of mesenteric and paraaortic lymphadenopathy, as well as providing visualization of the gastrointestinal tract and other organs in a manner that is complementary to the T2-weighted single shot FSE acquisitions. In general, inflammatory changes and abnormal mucosal enhancement seen with appendicitis are best visualized at the later time points.

Diffusion Weighted Imaging (DWI) has been used for small bowel imaging to improve the diagnostic confidence for identifying acute changes seen with inflammatory bowel disease. Active inflammatory processes, such as the swelling or edematous changes that may be seen in the appendix, lead to a reduction in the normal diffusivity of water in tissue. DWI methods that use echo-planar techniques are fat-suppressed and T2-weighted. Acute pathologic abnormalities, such as acute appendicitis, have high signal intensity on fat-suppressed DWI due to the combination of restricted diffusion and edema (Figure 40). A “b value” of zero has been effective for visualizing periappendiceal fluid, and a “b value” of 500 has been found to improve the conspicuity for detection of acute appendicitis.

Figure 40 Appendicitis on DWI (arrow)
In patients with acute nontraumatic abdominal pain in whom appendicitis is suspected, the frequency of proven appendicitis is typically 25-30%. There are a wide variety of causes that can mimic appendicitis that can be diagnosed with MRI, including inflammation and infection of the remaining gastrointestinal tract (enteritis, colitis, or diverticulitis). Other bowel pathologic abnormalities, including malignancy, can be found on MRI as well. A variety of genitourinary abnormalities can also mimic the clinical presentation of appendicitis, such as ovarian abnormalities (Figure 41) and obstructing urolithiasis (Figure 42). Acute calculous cholecystitis, a fairly common cause of nontraumatic abdominal pain, may be detected on MRI due to the extended coverage of anatomy that it offers (Figure 43). MRI is emerging as a viable, safe, and effective imaging alternative for the diagnosis of appendicitis, and the accurate differentiation of acute appendicitis from other causes of abdominal or pelvic pain.

Figure 41 On left, fluid-fluid level seen in ovarian cyst on T2-weighted image with fat sat; on right, unenhanced T1-weighted image shows multiple ovarian cysts

Figure 42 Coronal SS-FSE image without fat sat on left shows right hydronephrosis and perirenal edema due to obstructive ureteral stone; axial SS-FSE image without fat sat on right demonstrates normal tip of appendix (arrow)
Pediatric Appendicitis

Acute appendicitis is the most common surgical emergency in children. CT is a favored imaging modality, but concern about the potential risks associated with the inherent ionizing radiation exposure has increased the use of alternative modalities in children. Ultrasound is favored by many physicians, but it can be limited by operator dependence, patient body habitus, and sonographically obscure anatomic locations (e.g., retrocecal and deep pelvic regions). A recent study found that ultrasound was indeterminate in 28% of acute appendicitis cases in children. These limitations of CT and ultrasound have led to the introduction of MRI for the assessment of appendicitis in children.
Diagnostic performance and clinical outcomes validate the efficacy of an abbreviated protocol that does not involve contrast or patient sedation for emergent evaluation of children with suspected appendicitis. One institution has found success with a rapid four-sequence protocol consisting of axial and coronal T2 single shot FSE (Fast Spin Echo), and axial and coronal T2 single shot FSE with fat saturation (Figure 44). While gadolinium-enhanced T1-weighted sequences may increase the radiologist’s degree of confidence in interpretation, the diagnostic performance is statistically similar, and added value beyond the non-contrast technique has not been confirmed. An important aspect of a successful program is avoiding sedation. Most patients 5 years and older, and selected younger patients, may undergo evaluation with the above-mentioned protocol without sedation.

Figure 44 Acute appendicitis in 14-year old girl; coronal SS-FSE on left and axial SS-FSE with fat sat on right demonstrate an enlarged, fluid-filled appendix with surrounding inflammatory changes (arrows), which is the typical appearance of an acute appendicitis.
The normal appendix on MR, as on CT, is visible as a blind-ending tubular structure arising from the cecum (Figure 45). It may be filled with air (low T2 signal in a non-dependent position) or fluid. The normal appendix will be visualized in 70-80% of cases. Inherent non-visualization of the appendix due to adjacent bowel or lack of intraperitoneal fat implies that the appendix is not inflamed or enlarged. An inflamed appendix will almost certainly be visible on MR. A small amount of pelvic peritoneal free fluid is a nonspecific finding in children with abdominal pain, and is not diagnostic of appendicitis. However, this finding does merit an additional higher level of scrutiny.

Figure 45 Normal appendix in 10-year old girl; coronal SS-FSE image on left, and axial SS-FSE image on right show the appendix as a non-distended tubular structure arising from the cecum (arrows); there are no inflammatory changes in the periappendiceal soft tissues.
Focal periappendiceal inflammation is a hallmark of acute appendicitis, and is readily detectable on the T2-weighted fluid sensitive sequences as high signal in the soft tissues adjacent to the appendix. In addition, fluid fills the lumen of the obstructed appendix. It should be noted that a normal appendix may occasionally have a small amount of intraluminal fluid. Appendicoliths are often visible as intraluminal foci of signal dropout, and can be helpful in making the diagnosis, particularly when found obstructing the appendiceal lumen (Figure 46). In tip appendicitis, the proximal appendix may appear normal; thus, it is important to assess the entire length of the appendix. Appendiceal enlargement is often present, but is not an absolute criterion to diagnose appendicitis in the absence of surrounding inflammation. A ruptured appendix with abscess formation will be evident if an adjacent fluid collection is present (Figure 47).
An additional benefit of MR is that it can be used to identify alternative diagnoses as the cause of abdominal pain. These alternative findings may be visible on CT, but would not necessarily be evident on a directed ultrasound examination. The most common alternative diagnoses include adnexal cysts, and enteritis/colicitis. Other relatively common and alternative diagnoses include pyelonephritis, hydronephrosis, and ovarian torsion (Figure 48).

Figure 48 9-year old male with nephrotic syndrome; axial SS-FSE image on left shows normal appendix (arrowhead) which is not enlarged and has no intraluminal fluid; axial image and coronal SS-FSE with fat sat image on right both show multiple small-bowel intussusceptions (arrows)
Appendicitis in Pregnancy

Acute appendicitis complicates approximately one in 766 pregnancies, and is the most common non-obstetric surgical emergency in pregnancy. It has been associated with premature labor and fetal and maternal death, particularly when perforation with peritonitis occurs. These patients rarely present with classic symptoms such as anorexia, fever, nausea, vomiting, and periumbilical pain localizing to the right lower abdominal quadrant. Laboratory findings, such as leukocytosis or elevated erythrocyte sedimentation rates are unreliable parameters during pregnancy. Clinical symptoms can be difficult to interpret due to the alteration of the position of intraabdominal contents by the pregnant uterus (Figure 49). A progressive cranial displacement of the appendix and cecum has been noted during pregnancy. The appendix is usually located caudal to the level of the iliac crest during the first trimester and cranial to it during the third trimester. The appendix can be displaced superiorly and elevate the anterior peritoneum, which hampers a proper physical examination; as a result, preterm labor and delivery can occur before an acute abdomen presents. Laparotomy can cause surgical complications and lead to preterm delivery. A correct diagnosis allows for the use of a special small incision to detect and localize the inflamed appendix.

CT and ultrasound are widely used for the preoperative diagnosis of acute appendicitis in adults. Although ultrasound is the first-line investigation for suspected appendicitis in a pregnant patient, MRI is more appropriate than CT as the second-line imaging modality when ultrasound results are non-diagnostic or equivocal. CT involves the use of ionizing radiation, which is a major concern for the fetus. Although the safety of MR imaging to the fetus has not been definitively proven, no proven human teratogenic or carcinogenic effects of MRI have been described in the literature. Pregnant patients may undergo MRI at any stage of pregnancy, if the radiologist determines that it is warranted by the risk-benefit ratio. The radiologist should document that the following three criteria have been satisfied:

1. The information could not be obtained with Ultrasound
2. The information to be obtained with MRI will likely affect the care of the patient, the fetus, or both
3. It is not prudent to postpone imaging until the patient is no longer pregnant
Extra caution is urged during the first trimester of pregnancy, as it is a time of organogenesis, when the fetus might be more vulnerable to any unknown effects of MRI. However, the risk of exposing the developing fetus to any diagnostic imaging technique that uses ionizing radiation is probably greater than the theoretic risk of MRI.

In a study performed over a 3-year period, ultrasound was performed on 284 patients with clinically suspected appendicitis. Of this group, 12 were pregnant; they became the study group to evaluate whether MRI could also be used to accurately diagnose and exclude appendicitis in pregnant patients with clinically suspected appendicitis. MRI sequences performed included T1- and T2-weighted FSE, and T2-weighted FSE with fat suppression, all with breath-holds. The MRI criteria for appendicitis included an enlarged appendix with a diameter of more than 6 mm, and signs of periappendiceal inflammatory changes. MRI exclusion of appendicitis was based on a normal appendix of less than 6 mm, or an appendix with a diameter of more than 6 mm with no evidence of periappendicitis. The final treatment of the patient was based on clinical, sonographic, and MRI results.
Sonography did not detect the appendix in 11 of the 12 patients in the study, nor did it detect inflammatory changes in the region where the appendix was expected to be located. The MRI studies showed a normal appendix in 7 patients, with results for 3 patients suggestive of appendicitis. The gestational ages of the fetuses of the 3 “positive” patients were 11, 13, and 16 weeks. The appendix was not seen on MRI in 2 patients. Neither the sonography nor the MRI studies showed any complications such as abscesses, signs of perforation, or ileus. In the 3 patients with positive MRI findings, the lumen of the appendix had high signal intensity on the T2-weighted and fat-suppressed images, and low signal intensity on the T1-weighted images. The appendiceal wall was hypointense on the T1-weighted images, and slightly hyperintense on the T2-weighted images (Figure 50). The surrounding fatty tissue had high signal intensity on the T2-weighted and fat-suppressed images, and low signal intensity on the T1-weighted images, representing the inflammatory changes in the surrounding fatty tissue (Figure 51).
When the appendix was seen in its short axis, a typical target sign was observed. All 3 patients underwent emergency laparotomy, and the diagnosis of appendicitis was confirmed at the time of surgery, and later with pathologic investigations.

Of the 9 patients in whom MRI did not show an inflamed appendix, four had signs of hydronephrosis and hydroureter on the right side; in two of these patients, the appendix was not seen on MRI. MRI clearly showed that the uterus was compressing the right ureter where it crosses the right iliac vessels, causing the hydronephrosis and hydroureter. This is a known phenomenon in pregnancy, and normally requires no treatment. In this study, MRI proved to be a valuable and safe technique for the evaluation of pregnant patients clinically suspected of having acute appendicitis.

**Conclusion**

This concludes the Advanced Abdominal Imaging, Part II: Kidneys, Adrenal Glands, Appendix module. You must complete the post-test for this activity with a score of 75% or better in order to receive Continuing Education credits.
Appendix A: References for Advanced Abdominal Imaging, Part II Module

- Robertson, Dr. Sarah, Bashir, Dr. Omar et al. (n.d.). *Adrenal gland.* Retrieved from https://radiopaedia.org/articles/adrenal-gland


Appendix B: References for Pictures for Advanced Abdominal Imaging, Part II Module

- Fig 1 – https://emedicine.medscape.com/article/1948775-overview#a2
- Fig 2 – https://emedicine.medscape.com/article/1948775-overview#a2
- Fig 3 – https://emedicine.medscape.com/article/1948775-overview#a2
- Fig 4 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2039780/figure/Fig1/
- Fig 5 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2039780/figure/Fig2/
- Fig 6 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2039780/figure/Fig3/
- Fig 7 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2039780/figure/Fig4/
- Fig 8 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2039780/figure/Fig5/
- Fig 9 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2039780/figure/Fig6/
- Fig 10 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2039780/figure/Fig7/
- Fig 11 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2039780/figure/Fig8/
- Fig 12 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2039780/figure/Fig9/
- Fig 13 – https://emedicine.medscape.com/article/1898785-overview#a2
- Fig 14 http://images.rsna.org/index.html?doi=10.1148/rg.24si045514&fig=F3
- Fig 15 – https://emedicine.medscape.com/article/1898785-overview#a2
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- Fig 25 – http://images.rsna.org/index.html?doi=10.1148/rg.24si045514&fig=F13
• Fig 30 – http://images.rsna.org/index.html?doi=10.1148/rg.24si045514&fig=F18A
• Fig 31, 32 – https://www.britannica.com/print/article/30542
• Fig 34 – https://emedicine.medscape.com/article/363818-overview
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• Fig 37 – http://images.rsna.org/index.html?doi=10.1148/rg.275065021&fig=F14
• Fig 38 – https://www.ajronline.org/doi/full/10.2214/AJR.15.15948, Fig 2B
• Fig 39 – https://www.ajronline.org/doi/full/10.2214/AJR.15.15948 Fig 2A on left, https://www.ajronline.org/doi/full/10.2214/AJR.15.15948 Fig 2D on right
• Fig 40 – https://www.ajronline.org/doi/full/10.2214/AJR.15.15948 Fig 2E
• Fig 41 – https://www.ajronline.org/doi/full/10.2214/AJR.15.15948 Fig 6E, https://www.ajronline.org/doi/full/10.2214/AJR.15.15948 Fig 6D
• Fig 42 – https://www.ajronline.org/doi/full/10.2214/AJR.15.15948 Fig 7A, https://www.ajronline.org/doi/full/10.2214/AJR.15.15948 Fig 7C
• Fig 43 – https://www.ajronline.org/doi/full/10.2214/AJR.15.15948 Fig 8A, https://www.ajronline.org/doi/full/10.2214/AJR.15.15948 Fig 8C
• Fig 44 – https://appliedradiology.com/articles/mri-for-appendicitis-in-pediatric-patients Fig 1A, https://appliedradiology.com/articles/mri-for-appendicitis-in-pediatric-patients Fig 1B
• Fig 45 – https://appliedradiology.com/articles/mri-for-appendicitis-in-pediatric-patients Fig 6A, https://appliedradiology.com/articles/mri-for-appendicitis-in-pediatric-patients Fig 6B
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• Fig 47 – https://appliedradiology.com/articles/mri-for-appendicitis-in-pediatric-patients Fig 5
• Fig 48 – https://appliedradiology.com/articles/mri-for-appendicitis-in-pediatric-patients Fig 10A, https://appliedradiology.com/articles/mri-for-appendicitis-in-pediatric-patients Fig 10B
Fig 51 –