Web Training Modules

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Liver

Anatomy

The liver is the body’s largest gland, as well as the largest internal organ, typically weighing in at approximately 3 pounds. It is considered a gland- an organ that secretes chemicals- because it produces bile, a substance needed to digest fats. The liver supports nearly every other organ in the body in some facet, and keeps the body pure of toxins and harmful substances. It has an incredible capacity for regeneration, and can tolerate major liver resections involving up to 70-75% of liver parenchyma. The liver is capable of growing as quickly as a cancerous tumor to restore its normal size and function.

The liver is a roughly triangular organ that extends across the entire abdominal cavity just inferior to the diaphragm. It is made up of soft, pinkish-brown tissues encapsulated by a connective tissue capsule. This capsule is further covered and reinforced by the peritoneum of the abdominal cavity, which protects the liver and holds it in place within the abdomen. The peritoneum connects the liver in 4 locations: the coronary ligament, the left and right triangular ligaments, and the falciform ligament. These are not true ligaments in the anatomical sense; rather, they are condensed regions of peritoneal membrane that support the liver. The coronary ligament and the left and right triangular ligaments all connect superior portions of the liver to the diaphragm. The falciform ligament runs from the diaphragm across the anterior edge of the liver to its inferior border, where it forms the round ligament of the liver and connects it to the umbilicus. The round ligament is a remnant of the umbilical vein that carries blood into the body during fetal development.
The liver is separated into 4 distinct lobes— the right, left, caudate and quadrate lobes. The right and left lobes are the largest, with the right lobe approximately 5-6 times larger than the tapered left lobe, and they are separated by the falciform ligament. The small caudate lobe extends from the posterior side of the right lobe and wraps around the inferior vena cava. The small quadrate lobe is inferior to the caudate lobe, extending from the posterior side of the right lobe and wrapping around the gall bladder. The internal structure of the liver is made up of approximately 100,000 small hexagonal functional units known as lobules. Each lobule is made up of numerous liver cells, called hepatocytes, which are lined up in radiating rows (Figure 1). Each lobule also has a central vein surrounded by 6 hepatic portal veins and 6 hepatic arteries. The blood vessels are connected by many capillary-like tubes called sinusoids, which are between the rows of hepatocytes. The sinusoids extend from the portal veins and arteries to meet the central vein, like spokes on a wheel. Each sinusoid passes through liver tissue containing 2 main cell types: Kupffer cells and hepatocytes. Kupffer cells capture and break down old, worn out red blood cells passing through the sinusoids. Hepatocytes line the sinusoids and make up the majority of cells in the liver. They perform most of the liver’s functions—metabolism, storage, digestion, and bile production. Tiny bile collection vessels called bile canaliculi run parallel to the sinusoids on the other side of the hepatocytes. The bile canaliculi join together to form larger bile ducts, found throughout the liver.
The network of bile ducts connect to form the large left and right hepatic ducts, which carry bile from the left and right lobes of the liver (Figure 2). The left and right hepatic ducts join to form the common hepatic duct, which drains all bile away from the liver. The common hepatic duct joins with the cystic duct from the gallbladder to form the common bile duct, which carries bile to the duodenum of the small intestine. Most of the bile produced by the liver is pushed back up the cystic duct by peristalsis to arrive in the gallbladder for storage, until it is needed for digestion.

![Figure 2 Ducts of liver and gallbladder](image)

The liver has a unique dual blood supply, holding approximately 13% of the body’s total blood supply at any given moment. This amount is usually near 1500mL/min, with 20-40% from the hepatic artery, and 60-80% from the hepatic portal vein system. The hepatic portal vein is formed by the union of the superior mesenteric vein and the splenic vein. Nutrient-rich blood traveling to the spleen, stomach, pancreas, gallbladder, and intestines passes through capillaries in those organs and is collected into the hepatic portal vein, which is the liver’s primary blood source. The hepatic portal vein delivers this blood to the tissues of the liver, where the contents of the blood are divided up into smaller vessels and processed, before being passed on to the rest of the body. Blood leaving the tissues of the liver collects in a central vein in each lobule, moves into the 3 hepatic veins, on to the inferior vena cava, and finally back to the heart. The hepatic artery is the liver’s secondary blood source, delivering oxygen-rich blood from the heart to the liver. Like other organs, the liver has its own system of arteries and arterioles.

**Physiology**

The liver is involved in many essential functions related to digestion, metabolism, immunity, and the storage of nutrients within the body. These functions make the liver a vital organ, without which the tissues of the body would quickly die from lack of energy and nutrients.
Digestion

Hepatocytes in the liver produce bile, which is a mixture of water, bile salts, cholesterol and the pigment bilirubin. It passes through the bile ducts of the liver to be stored in the gallbladder. The gallbladder releases bile into the duodenum to emulsify large masses of fat for easier digestion.

Bilirubin present in bile is a product of the liver’s digestion of worn out red blood cells. Kupffer cells in the liver catch and destroy old, worn out red blood cells, and pass their components on to hepatocytes. Hepatocytes metabolize hemoglobin (the red oxygen-carrying pigment of red blood cells) into the components heme and globin. Globin protein is further broken down and used as an energy source for the body. The iron-containing heme group cannot be recycled by the body, and is converted into the pigment bilirubin and added to bile to be excreted. Bilirubin gives bile its distinctive greenish color. Intestinal bacteria further convert bilirubin into the brown pigment stercobilin, which gives feces their brown color.

Metabolism

The hepatocytes of the liver are tasked with many of the important metabolic jobs that support the cells of the body. Since all of the blood leaving the digestive system passes through the hepatic portal vein, the liver is responsible for metabolizing carbohydrates, lipids, and proteins into biologically useful materials.

Our digestive system breaks down carbohydrates into glucose, which cells use as a primary energy source. Blood entering the liver through the hepatic portal vein is extremely rich in glucose from digested food. Hepatocytes absorb much of this glucose, and store it as glycogen. They can pack away large amounts of glucose, but still quickly release it between meals. This absorption and release of glucose by the hepatocytes helps to maintain homeostasis and protects the rest of the body from dangerous spikes and drops in the blood glucose level.

Hepatocytes can also produce lipids like cholesterol, phospholipids, and lipoproteins that are used by other cells throughout the body. Much of the cholesterol produced by hepatocytes gets excreted from the body as a component of bile.

The digestive system breaks down dietary proteins into their component amino acids before passing them on to the hepatic portal vein. Amino acids entering the liver require metabolic processing before they can be used as an energy source. Hepatocytes first remove the amine groups of the amino acids, and convert them into ammonia, and eventually urea. Urea is less toxic than ammonia and can be excreted in urine as a waste product of digestion.

Detoxification

As blood from the digestive organs passes through the hepatic portal circulation, the hepatocytes of the liver monitor the contents of the blood, removing many potentially toxic substances before they can reach the rest of the body. Enzymes in hepatocytes metabolize many of these toxins, such as alcohol and drugs, into their inactive metabolites. In order to keep hormone levels within homeostatic limits, the liver also metabolizes, and removes from circulation, hormones produced by the body’s own glands.
Storage

The liver provides storage of many essential nutrients, vitamins, and minerals obtained from blood passing through the hepatic portal system. Glucose is transported into hepatocytes and stored as glycogen. Hepatocytes also absorb and store fatty acids from digested triglycerides. The storage of these nutrients allows the liver to maintain the homeostasis of blood glucose. The liver also stores vitamins A, D, E, K, and B12, and the minerals iron and copper, in order to provide a constant supply of these essential substances to the tissues of the body.

Production

The liver is responsible for the production of several vital protein components of blood plasma, including prothrombin, fibrinogen, and albumins. Prothrombin and fibrinogen proteins are coagulation factors involved in the formation of blood clots. Albumins are proteins that maintain the isotonic environment of the blood so that cells of the body do not gain or lose water in the presence of body fluids.

Immunity

The liver functions as an organ of the immune system through the function of the Kupffer cells that line the sinusoids. Kupffer cells capture and digest bacteria, fungi, parasites, worn-out blood cells, and cellular debris. There is a large volume of blood passing through the hepatic portal system and the liver, and the Kupffer cells can clean this large volume very quickly.

MR Imaging of Liver Pathology

Imaging of the liver is undertaken for the detection and characterization of suspected primary or secondary neoplasms, prior to planning a surgery or chemotherapy pump placement, for assessing treatment response, for evaluating biliary pathology, and for screening for liver neoplasms in high-risk groups. Primary hepatic neoplasms are common, especially in the presence of diffuse liver disease such as cirrhosis, hemochromatosis, and steatohepatitis. The liver is also the most common site of metastasis from gastrointestinal tumors. High blood flow (about 25% of cardiac output), a favorable microscopic anatomy, and a rich biochemical environment favor the rapid growth of metastatic deposits in the liver. In patients with liver tumors, it is crucial to detect and stage the tumors at an early stage, in order to select patients who will benefit from curative liver resection and avoid unnecessary surgery. An optimal preoperative evaluation of the liver is necessary, and a contrast-enhanced MRI is widely considered the state-of-the-art method.

MRI has more advantages than ultrasound, computed tomography, positron emission tomography, or any other imaging modality in diagnosing focal hepatic masses. Most liver lesions can be adequately diagnosed with a combination of T1- and T2-weighted sequences, Diffusion Weighted Imaging (DWI), and hepatobiliary contrast agents, which includes MultiHance® and Eovist®. Benign lesions (e.g., cysts, hemangiomas, focal nodular hyperplasias, or adenomas) can be distinguished from malignant lesions. In a non-cirrhotic liver, the most common malignant lesions are metastases, which may be hypovascular or hypervascular. In the cirrhotic liver, hepatocellular carcinoma is of considerable importance, as well as intrahepatic cholangiocarcinoma and other less common malignancies that must be assessed.
Non-Contrast Enhanced Liver MRI

Non-enhanced T1- and T2-weighted sequences are decisive in the characterization of focal liver diseases in cirrhotic as well as non-cirrhotic livers (Figure 3). High signal intensity on T1-weighted images can be caused by bleeding, fat, or deposition of copper or glycogen. This can be seen in dysplastic nodules, as well as hepatocellular carcinoma. On T2-weighted images, most benign tumors are bright, whereas malignancies are slightly hyperintense. On T1-weighted out-of-phase imaging, abnormal fat accumulation in the liver will be hypointense. This sequence is more accurate for the diagnosis of steatosis, focal fatty infiltration, and focal fatty sparing of the liver. In-phase and out-of-phase images are valuable in the diagnosis of iron storage diseases, as iron-containing liver parenchyma is hypointense on in-phase and isointense on out-of-phase images.

![Figure 3 A- Multiple hypointense lesions on T1 in portovenous phase after gadolinium injection; B- Lesions are not visible on T1 in-phase; C- Lesions are hypointense on T1 out-of-phase; typical for focal fatty infiltration or steatosis](image)
Diffusion Weighted Imaging (DWI) has been shown to be a reliable method to detect liver metastases (Figure 4). Claims have been made that the Apparent Diffusion Coefficient (ADC) measurement has the potential to discriminate between benign and malignant focal hepatic lesions. However, reproducibility is somewhat limited, due to different imaging techniques (including the choice of different b-values), and differences in scanner technology. Cystic or necrotic metastases showing relatively high ADC values may be false negative, while atypical hemangiomas with uncommonly low ADC values may be false positive. One large study showed that cysts have significantly higher values compared to hemangiomas, which have significantly higher ADC than solid lesions. However, using the ADC values, solid lesions could not be distinguished from one another. Although DWI is an excellent diagnostic tool for the detection of even sub-centimeter liver lesions, a complementary contrast-enhanced imaging study should be performed when further characterization is needed. This combination would probably be the most efficient preoperative evaluation. DWI is also of value to identify lymph nodes and other extrahepatic lesions.

Figure 4 Large liver metastasis with 3 cysts; A, B- On DWI, cysts (arrows) have benign pattern with hyperintensity on b=50 (A) and isointensity on b=500 (B) compared to metastasis (arrow heads), which has malignant pattern with hyperintensity also on b=500; C- In hepatobiliary phase, 2 hours after gadolinium injection, cysts are more hypointense compared to metastasis

Contrast-Enhanced Liver MRI

Liver MRI is very dependent on the administration of contrast agents, especially when detection and characterization of focal lesions are the issues. Liver MRI combined with MRCP is useful to evaluate patients with hepatic and biliary disease.
Gadolinium Contrast Agents

There are several gadolinium based extracellular contrast agents that have been extensively used for dynamic MRI of the liver. Solid and vascularized lesions enhance, whereas cystic and necrotic lesions do not. Hypervascularized lesions enhance intensely and early, in contrast to hypovascularized tumors, which enhance less and later. Gadolinium based extracellular agents diffuse rapidly to the extravascular space of tissues which are being cleared by glomerular filtration at the kidney. These characteristics can be problematic when imaging a large organ with a huge interstitial space, such as the liver. These contrast agents provide a small temporal window for imaging- a matter of seconds. They will then begin to diffuse to the interstitial space of not only healthy liver cells, but also that of lesions. This action reduces the contrast gradient necessary for easy lesion detection. Dynamic MRI with multiple phases (arterial, portal and late phase) after IV gadolinium based extracellular agents provides additional information.

Hepatobiliary Contrast Agents

An advantage of MRI is the availability of liver-specific, or hepatobiliary contrast agents. These contrast agents can be used to detect metastases, characterize liver lesions (with or without hepatocyte function/uptake), and to evaluate biliary excretion. The two main groups are gadolinium based agents, and superparamagnetic iron oxide particles. The hepatobiliary specific gadolinium agents include two high relaxivity agents: gadobenate dimeglumine, marketed as MultiHance®, and gadoxetate disodium, marketed as Eovist®. Both of these agents are linear ionic molecules, and show higher T1 relaxivity as compared to the other gadolinium chelates. These agents initially act like non-specific extracellular gadolinium chelates post bolus injection, showing three primary phases of vascular and tissue enhancement (arterial, blood pool, and extracellular phases). However, in the delayed (hepatobiliary) phase, they are taken up by the liver as their excretion is not only through the renal route, but the hepatic route as well. These contrasts are absorbed by hepatocytes via “transporters”. The enhancement of lesions in the hepatobiliary phase depends on the expression and activity of these “transporters”, which depend on the presence or absence of functioning hepatocytes. During the hepatobiliary phase, they selectively increase the liver signal intensity, and aid in the detection of small tumors. The hepatobiliary phase is prolonged with the use of hepatobiliary-specific MR contrast agents, so precisely timed imaging is not required in this phase. High spatial resolution sequences in separate breath holds can complete imaging, and longer imaging time can be used if studying bile leak or contrast agent washout. In addition, the biliary excretion enables biliary ductal mapping (post-contrast MRCP/functional MRCP) using 3D T1-weighted fat-saturated gradient echo images.

These two hepatobiliary contrasts, MultiHance® and Eovist®, may work in similar ways, but they do have different profiles. MultiHance® has weak and transient protein bonding (<5%) and only 2-4% of it is taken up by the liver cells. Its delayed imaging time is between 90 to 120 minutes. MultiHance® also has higher relaxivity values and signal intensity changes than the non-liver specific gadolinium based contrast agents, but maintains a similar safety profile. Eovist® has protein bonding of <10%. It has almost equal biliary and renal excretion (=50% each), so it offers more intense liver parenchymal enhancement compared to MultiHance®. However, the vascular enhancement with Eovist® is less pronounced and has a short duration. The low dynamic enhancement of Eovist® may be increased with a slower injection rate. The delayed imaging time for Eovist® is more convenient at 10-20 minutes. Some facilities opt to use MultiHance® when the dynamic enhancement in different phases is more important than the hepatobiliary function, and choose Eovist® when the hepatobiliary function is more crucial than the dynamic imaging. Eovist® is not recommended for use with T2-weighted MRCP sequences, as the
rapid biliary excretion of this contrast agent decreases the signal intensity of the biliary structures on T2. If an MRCP is performed both before and after an injection of Eovist®, a decrease or disappearance of high signal intensity can be interpreted as evidence that bile secretion works. DWI may be performed after Eovist® without compromising contrast to noise ratio or ADC of focal hepatic lesions.

Superparamagnetic Contrast Agents

Two of the superparamagnetic iron oxide particles that have been approved for MR imaging are magnetite and maghemite. They both have crystalline cores of iron oxide crystals, which are water insoluble, and they are coated with dextran or a biodegradable polysaccharide, which prevents particle aggregation. This modifies their biological behavior and makes the total size of the iron oxide particle substantially larger. These particles do not leak into the interstitium. Instead, they act as intravascular contrast agents or blood pool agents, as long as the vessel endothelium is intact and unaltered by any pathological process. Their elimination from the blood is by uptake into the reticuloendothelial system cells in the liver (Kupffer cells), spleen, bone marrow and lymph nodes. They are phagocytosed by macrophages throughout the body.

Superparamagnetic iron oxide particles agents are preferentially entrapped by the Kupffer cells in the liver and spleen, and reduce their T2 relaxation times, causing normal liver to appear dark on T2-weighted images. Most liver tumors, which are usually deficient in Kupffer cells, do not exhibit superparamagnetic iron oxide particles uptake, and appear relatively hyperintense. However, well-differentiated tumors which have not lost all their Kupffer cells will take up superparamagnetic iron oxide particles agents and exhibit reduced signal intensity. A minimum delay of approximately 10 minutes between injection (or infusion) and MR imaging is recommended. Cross-section flow void in narrow blood vessels may impede the differentiation from small liver lesions. In addition, aortic pulsation artifacts may become more pronounced.

Two of the superparamagnetic contrast agents that have been approved for MR imaging are Ferumoxide, marketed as Endorem or Feridex, and Ferucarbotran, marketed as Resovist or Cliavist. Ferumoxide has a particle size of 50-180 nm, with a thin, incomplete dextran coating that causes individual particles to form polycrystalline aggregates. These aggregates behave in solutions or within cells as large particles. Endorem and Feridex are used for the detection of liver lesions that are associated with an alteration in the reticuloendothelial system. Ferumoxide agents distribute relatively rapidly to organs with reticuloendothelial cells, primarily the liver, spleen, and bone marrow, which will all have decreased signal intensity. These agents are taken up by macrophages, found in healthy liver cells, but not tumors. This results in an increased contrast to noise ratio between tumor and normal liver. Hepatocellular lesions, such as adenoma or focal nodular hyperplasia, contain reticuloendothelial cells, so they will behave similar to the liver, with decreased signal on T2-weighted images. There is typically some circulating contrast agent, and blood vessels show increased signal intensity on T1-weighted images. Ferumoxide (Endorem or Feridex) is typically administered as a slow IV infusion over 30-60 minutes, with imaging typically performed 1-4 hours after infusion. The liver appears darkest on T2*/T2-weighted images in the first 24 hours post-infusion. The liver signal intensity returns to normal within 7-14 days in most cases; however, complete metabolism requires 14-28 days. The most common complication is acute severe low back pain, which can be minimized with a slow infusion.

Ferucarbotran (Resovist, Cliavist) is used for the detection and characterization of especially small focal liver lesions. It has a particle size of approximately 60 nm, and is coated with low molecular weight carboxydextran. These nanoparticles are accumulated by phagocytosis in cells of the reticuloendothelial...
system of the liver. Their uptake results in a decrease of the signal intensity of normal liver parenchyma on both T2- and T1-weighted images. Most malignant liver tumors do not contain reticuloendothelial cells, and therefore do not uptake the iron particles. The resulting image effect is improved contrast between the tumor (bright) and the surrounding tissue (dark). Resovist is provided as a ready to use formulation and is administered as a rapid bolus. It can be used with both dynamic and delayed imaging. The most commonly reported adverse events were vasodilation and paresthesias.

**Benign Focal Liver Disease**

Hepatic cysts are found in 2-7% of the population. They can range in size from a few mm to several cm. The diagnostic accuracy of MRI for hepatic cysts is approximately 97%. They typically show the same signal intensity as CSF in all MR pulse sequences. Enhancement indicates infection, inflammation or a neoplasm.

**Hemangiomas**

Hemangiomas are the most common benign tumors of the liver, with cavernous hemangiomas diagnosed at an incidence of 20%. Hemangiomas are found more commonly in women, and may appear singly or as one of many tumors. They can range in size from less than 1 cm to 10 cm. Larger tumors may have different imaging features due to internal hemorrhage and fibrosis. Hemangiomas are typically well-delineated and follow the signal intensity of blood- hypointense on T1-weighted images, and hyperintense on T2 (Figure 5). Heavy T2-weighted imaging is generally used to differentiate hepatic hemangiomas from malignant lesions, as the hemangiomas retain their higher signal. Hemangiomas have a distinctive enhancement pattern in MRI, described as “peripheral globular enhancement with progressive fill-in”. Smaller hemangiomas (less than 2 cm) typically show homogeneous enhancement in the late arterial phase, resembling both hepatocellular cancer and hypervascular metastasis.

![Figure 5 A- Hemangioma with high signal on T2; B- Hemangioma with low signal on T1; C- Nodular enhancement in arteriportal phase after gadolinium contrast; D- Five minutes after injection, filling in during late phase](image-url)
Focal Nodular Hyperplasia

Focal nodular hyperplasia is an incidental finding in approximately 90% of cases, with patients usually asymptomatic. It is commonly found in young women, especially those on oral contraceptives. Focal nodular hyperplasia is typically isointense on T1-weighted imaging, and isointense to mildly hyperintense on T2, with no tumor capsule noted (Figure 6). Focal nodular hyperplasia may be isointense pre-contrast and in the venous phase, and hyperintense in the arterial phase. There may be a loss of signal if ferumoxide contrast is used, due to the presence of Kupffer cells. MRI has a 95% sensitivity and specificity for the diagnosis of focal nodular hyperplasia. There is typically no treatment given, unless this condition is causing pain.

Figure 6 Typical focal nodular hyperplasia (large arrows) in coronal (A) and transverse planes (B-E); slightly hyperintense to the liver on T2 (B) and enhances richly on T1 in arterial phase (A, C), followed by isointensity in delayed phases (D,E); small arrow indicates central scar
Hepatic Adenomas

Hepatic adenomas are less common than focal nodular hyperplasia, but are also found in young women on oral contraceptives. They are also seen in men who are taking anabolic steroids. Hepatic adenomas may cause acute abdominal pain and hepatomegaly, and may require surgery if bleeding, which can be due to intratumoral hemorrhage. They are typically 5-10 cm in size, with a well-defined capsule. The presence of a capsule, and the presence of intralesional fat on out-of-phase T1 imaging, makes hepatic adenomas easier to identify on MRI. They are considered pre-malignant, but malignancy is rare unless the hepatic adenoma is larger than 10 cm. Hepatic adenomas typically have heterogeneous signal intensity on both T1- and T2-weighted images, due to hemorrhage, necrosis, and/or steatosis (Figure 7). After gadolinium contrast, they show signal enhancement in the arterial phase, as they are supplied by the hepatic artery, and will be hypointense in the hepatobiliary phase.

Hepatic adenomatosis typically involves 10-50 adenomas. This is a progressive and symptomatic disease, and is more likely to lead to impaired liver function, hemorrhage, and malignant degeneration as compared to hepatic adenoma. Imaging characteristics are similar to those listed for hepatic adenomas.

Benign Lipomatous Tumors

Benign lipomatous tumors include lipomas, myelolipomas, and angiomyelolipomas. Their imaging features depend on the amount of fat in the lesion. MRI demonstrates fat in a simple lipoma, which does not enhance on a contrast study. Angiomyelolipomas show enhancing soft tissue in addition to fat, which should be interpreted with caution if significant soft-tissue components are present, as hepatocellular carcinoma can also contain fat.
Malignant Hepatic Lesions

Hepatocellular Carcinomas

Hepatocellular carcinomas are the most common primary malignancy of the liver, and the third most common cause of cancer-related deaths, after lung and stomach cancers. If caught early, patients could benefit from surgical resection or transplant. Risk groups with cirrhosis (chronic viral hepatitis B/C, alcoholism and/or non-alcoholic steatohepatitis) should be in surveillance programs, receiving ultrasounds for surveillance purposes. The chance to find a small hepatocellular cancer is very small. Hepatocellular carcinomas can be focal, multifocal, or diffuse. They are typically hypointense to the liver on T1, and moderately hyperintense on T2 (Figure 8). The critical sequence is a dynamic gadolinium-enhanced sequence. Hepatocellular carcinoma is supplied by the hepatic artery, and will demonstrate signal enhancement in the arterial phase, and rapid washout on portal venous phase images. When ferumoxide contrast is used, these carcinomas are hyperintense to the normal liver. If cirrhosis is present, ferumoxide is a better contrast choice for identifying small hepatocellular carcinomas. The accuracy of the imaging diagnosis of hepatocellular carcinoma in liver cirrhosis depends on the degree of arterial hypervascularization, which increases with tumor size and grade of malignancy. Larger hepatocellular carcinomas are heterogeneous, and may invade the portal vein. In a cirrhotic liver, a solid lesion that is hyperintense on T2 is suspected to be hepatocellular carcinoma. This disease is a bit of a chameleon, and can mimic hemangioma, adenoma, focal nodular hyperplasia and hyper vascular metastasis in the cirrhotic liver. Hepatocellular carcinoma may display as isointense in the arterial phase, and lack wash-out in the venous phase. Hepatobiliary MRI may produce better characterization of hypervascular nodules that are larger than 2 cm. Ninety-five percent of hepatocellular carcinomas will be hypointense on T1, while ninety-four percent of pseudolesions will be isointense. On DWI, hepatocellular carcinoma is typically hyperintense on images with a high B value, and dark on ADC images, indicating restricted diffusion.

Figure 8 Hypervascular lesion in cirrhotic liver; A- Lesion is hyperintense in T1 arterial phase; B-Hypointense washout in portovenous phase; C- Lesion is slightly hyperintense on T2; D- Hyperintense lesion on DWI; characteristic findings for hepatocellular carcinoma
Cholangiocarcinomas

Amongst cholangiocarcinomas, 60-70% occur at the bifurcation of the hepatic duct, with 20-30% in the distal common bile duct, or within the liver (5-15%). These lesions are hypointense on T1, hyperintense on T2, and show delayed enhancement on contrast studies (Figure 9). Ferrumoxide is helpful as a contrast agent for detecting small lesions. Intrahepatic cholangiocarcinoma is rare, and associated with repeated/chronic cholangitis and cystic disease of the liver. It is typically hyperintense on T2, and often obstructs vessels and bile ducts, with upstream ductal dilatation. After contrast, enhancement is delayed, starting in the periphery similar to hemangioma. However, enhancement of cholangiocarcinoma is not isointense to vessels; it may display homogeneous arterial contrast enhancement.

![Figure 9 A- Cholangiocarcinoma that is hyperintense on T2; B- Cholangiocarcinoma that is hypointense on T1; note dilated bile ducts in periphery of tumor (arrows)]](image)

Fibrolamellar Hepatocellular Carcinomas

Fibrolamellar hepatocellular carcinoma is more common in women and patients with no existing diffuse liver disease. It typically presents as a large tumor that is hypointense on T1 and hyperintense on T2. This type of carcinoma is rare, and is seen without any underlying cirrhosis. These tumors are usually larger than 12 cm, and have metastases in 66% of cases.
Metastases

Metastases are the most common malignant liver neoplasms, and are rare in cirrhotic livers. These tumors are typically hypovascular or hypervascular, and are best diagnosed with dynamic gadolinium-enhanced imaging. Most hepatic metastases are multiple, hypointense on T1, hyperintense on T2, and hypovascular in dynamic contrast imaging (Figure 10). Cystic metastases may be intensely bright on T2 and resemble cysts, abscesses, and hemangiomas. After hepatobiliary contrast, metastases are hypointense on T1 in a bright liver. Hepatobiliary contrast agents may further increase the sensitivity of MRI for the diagnosis of colorectal cancer liver metastases especially after neoadjuvant chemotherapy. Metastases may become difficult to diagnose due to changed tumor vascularity and liver steatosis. DWI is more sensitive than T2-weighted MR for the detection of hepatic metastases. They show higher signal intensity on DWI that on T2-weighted Spin Echo images, whereas the signal from vessels and cysts are suppressed with DWI.

Hypervascular metastases are often multiple, and do not follow the signal intensity of vessels. On T1, they are typically hypointense, and their signal intensity is usually moderately elevated on T2. Melanoma is an exception, where melanin accumulation may result in hyperintensity on T1. Hypervascular liver metastases from renal cell carcinoma, neuroendocrine tumors, sarcoma, malignant melanoma, thyroid carcinoma, some breast and colorectal cancer are hyperintense in late arterial phase imaging. There are also common benign hypervascular liver lesions, hemangioma, focal nodular hyperplasia, and some tumor-like conditions. On DWI, metastases with a marked hypervascularity can attain high ADC values and be false negative.

![Figure 10 Carcinoid liver metastasis (arrows): A- hyperintense and readily detected on DWI, intensity less pronounced on b=0; B- Intensity more pronounced on b=800; C- Lesion is dark with lox ADC value, hard to detect and delineate due to small size; D- Tumor is hyperintense due to hypervascularity post contrast injection on T1 arterioporal phase; E- T1 hepatobiliary phase shows lesion as hypointense](image-url)
Lymphoma

Primary liver lymphoma is rare and typically involves solitary lesions, while secondary liver lymphoma involves multiple lesions. Compared to the surrounding liver tissue, liver lymphoma is isointense to the spleen, hypointense on T1, and hyperintense on T2. After contrast enhancement, lymphoma is hypointense centrally, with a peripheral enhancement. In the hepatobiliary phase, the lesions are hypointense, and may have a target-like appearance.

Diffuse Liver Disease

There are many different pathologic conditions that comprise diffuse liver disease, including abnormalities of metabolism, infections, chronic injury due to toxins, and malignant diseases. Many of the diffuse liver diseases predispose one to the development of primary hepatic neoplasms. The aim of imaging in these cases is to characterize some of these conditions, detect early hepatic neoplasms, and differentiate benign lesions from malignancies.

MRI is very suitable for imaging diffuse liver disease due to its ability to characterize tissue based on T1- and T2-weighted image characteristics. Based on its distribution, and the presence of abnormal signals, diffuse liver disease can be divided into four imaging patterns:

- Diffuse homogeneous distribution- observed in hemochromatosis, steatohepatitis, and glycogen storage diseases
- Segmental distribution- seen with focal fatty infiltration and subacute hepatitis
- Diffuse nodular distribution- seen with post-viral cirrhosis, Wilson’s disease, and sarcoidosis
- Perivascular abnormalities- seen with congested liver and Schistosomiasis japonica infection
Liver Abscesses

The majority of liver abscesses are pyogenic, and portal or biliary in origin. They are usually a cluster of small abscesses that coalesce into a large cavity with an air or fluid level, which is then surrounded by an enhancing capsule. Smaller liver abscesses (less than 1 cm) may enhance and mimic hemangioma (Figure 11). Metastases, which become necrotic after treatment, may be indistinguishable from an abscess. DWI can be used to discriminate between an abscess and a cystic or necrotic tumor, as an abscess has lower ADC values when compared with necrotic portions of tumors.

Figure 11 Candidiasis with multiple microabscesses (arrows) in both liver and spleen; abscesses are in portal-venous phase, hypointense on T1
Hemochromatosis

Primary hemochromatosis is due to genetic mutation in the HFE (High Iron FE) gene, while secondary hemochromatosis may result from excessive transfusions. Iron overload can be diagnosed using MRI, as the liver appears dark on T2-weighted images (Figure 12). It is better appreciated on a Gradient Echo T2* sequence due to its inherent high magnetic susceptibility. The presence of hepatocellular carcinoma can be detected with high confidence, as it does not contain iron, and appears bright against the dark background of the normal liver on T2-weighted images.

Steatohepatitis

Steatohepatitis demonstrates varying pathologic degrees of steatosis (fatty liver), mixed cellular inflammatory infiltrate across the lobule, the presence of hepatocyte injury, and fibrosis. This condition is unrelated to alcohol abuse. Radiologically, steatohepatitis is difficult to distinguish from other causes of fatty liver. However, MRI is better than other modalities, such as CT, in detecting microscopic fat. Chemical shift imaging techniques such as in- and out-of-phase T1 gradient-recalled echo techniques are useful, as the liver loses signal on out-of-phase imaging in the presence of intracellular fat.

Fatty Liver

Fatty liver occurs in patients with diabetes, hyperalimentation, alcohol abuse, pregnancy, chemical toxicity, and transplanted liver. Imaging features are similar to those of steatohepatitis, except for localized segmental distribution, resulting from regional differences in perfusion.
Cirrhosis

Cirrhosis is the end result of chronic liver injury, secondary to viral infections, alcohol abuse, and toxins. Pathologically, the liver demonstrates varying degrees of fibrosis and regenerative nodules. Additional secondary changes follow, including portal hypertension and ascites. Imaging’s role in cirrhosis is the early detection of hepatocellular carcinoma, and the differentiation of regenerative nodules from dysplastic nodules and hepatocellular carcinoma. Regenerating nodules appear hypointense on T2-weighted images, while hepatocellular carcinoma appears hyperintense. Dysplastic nodules and hepatocellular carcinomas demonstrate arterial enhancement, while regenerative nodules are non-enhancing. Ferumoxide-enhanced MRI is useful in demonstrating hepatocellular carcinomas in cirrhosis, as they don’t contain functioning Kupffer cells, and appear bright on T2-weighted post-contrast images. In one study, the sensitivity of gadolinium-enhanced MRI for detecting hepatocellular carcinomas in cirrhosis increased with an increase in the size of the tumor.

Passive Venous Congestion

Passive venous congestion of the liver occurs with any cause, resulting in right heart failure, constrictive pericarditis, and hepatic venous outflow obstruction. MRI may demonstrate periportal hyperintensity secondary to perivascular lymphedema on T2-weighted images.
Pancreas

Anatomy

The pancreas is an abdominal glandular organ, with both digestive and hormonal functions. It is elongated and tapered, approximately 6-10 inches in length. The pancreas lies transversely, and somewhat obliquely, on the posterior abdominal wall behind the stomach, at the level of the L1-L2 lumbar vertebrae. Aside from its tail, the pancreas is a retroperitoneal structure.

The pancreas is arbitrarily divided into five parts (Figure 13). These include:

1. Head- widest part of the organ, makes up its right side, located near the center of the abdomen; constitutes approximately 50% of the parenchymal mass of the pancreas; lies within the C-shaped curve created by the duodenum, and is connected to it by connective tissue; anterior to the inferior vena cava and the left renal vein
2. Uncinate process- projection arising from lower part of the head that extends medially to lie beneath the body of the pancreas; lies posterior to the superior mesenteric vessels (vein on right, artery on left), and anterior to the aorta
3. Neck- located between the head and the body of the pancreas; overlies superior mesenteric vessels, which form a groove in its posterior aspect
4. Body- centrally located, crossing the midline of the body to lie behind the stomach and to the left of the superior mesenteric vessels
5. Tail- along with the body, constitutes remaining 50% of pancreatic parenchymal mass; thin end of the pancreas that runs obliquely upward and to the left, anterior to the aorta and left kidney; lies within close proximity to the hilum of the spleen; only part of the pancreas that is intraperitoneal

Figure 13 Sections of the pancreas
The pancreatic body and tail are supplied by the multiple pancreatic branches of the splenic artery (Figure 14). The head is supplied by the superior mesenteric artery, and the superior and inferior pancreaticoduodenal arteries, which are branches of the gastroduodenal artery from the celiac trunk. Because of the rich blood supply from the celiac trunk and the superior mesenteric artery, these vessels should be evaluated when angiography is performed for bleeding as a complication of acute or chronic pancreatitis, or pancreatoduodenectomy. Venous drainage of the head of the pancreas is into the superior mesenteric branches of the hepatic portal vein. Numerous small, fragile veins drain directly from the pancreatic body and tail into the splenic vein. The splenic vein arises in the splenic hilum behind the tail of the pancreas, and runs from left to right on the posterior surface of the pancreatic body. The union of the “horizontal” splenic vein and the “vertical” superior mesenteric vein forms the portal vein behind the neck of the pancreas. The portal venous system, which includes the splenic, superior mesenteric, and portal veins, has no valves.

The pancreas is drained by lymphatic vessels that follow the arterial supply. The head of the pancreas drains into pancreaticoduodenal lymph nodes, and lymph nodes in the hepatoduodenal ligament and pyloric nodes. The pancreatic body and tail drain into mesocolic lymph nodes (around the middle colic artery), and lymph nodes along the hepatic and splenic arteries. Final drainage occurs into celiac, superior mesenteric, and para-aortic and aortocaval lymph nodes.

The pancreas is a composite gland that contains both exocrine and endocrine components. Most of the pancreas (95%) consists of exocrine tissue that produces pancreatic enzymes for digestion. The remaining tissue consists of endocrine cells that produce hormones that regulate blood sugar and pancreatic secretions.
The exocrine portion of the pancreas is classified as a serous gland, producing enzymes that are important to digestion. These enzymes include trypsin and chymotrypsin to digest proteins; amylase for the digestion of carbohydrates; and lipase to break down fats. The exocrine component is composed of a million “berry-like” clusters of cells called acini, which are arranged in lobules. Each lobule has its own ductule, and many ductules join to form intralobular ducts. Intralobular ducts then form interlobular ducts, which drain into branches of the main pancreatic duct. When food enters the stomach, these pancreatic enzymes are released into the system of ducts in an inactive form. The pancreatic juices make their way to the main pancreatic duct, which runs the length of the pancreas. The pancreatic duct joins the common bile duct, which originates in the liver and gall bladder and produces the important digestive juice called bile (Figure 15). Once united, the pancreatic and common bile ducts form the ampulla of Vater, which opens into the duodenum. When the pancreatic enzymes enter the duodenum, they are activated to aid in digestion. The pancreas’ exocrine tissue also secretes bicarbonate to neutralize stomach acid in the duodenum. A smooth muscle sphincter, called the sphincter of Oddi, is present around the common channel of the pancreatic duct and the common bile duct. This sphincter prevents reflux of duodenal juices into the pancreatic and common bile ducts. Additional smooth muscle sphincters are present around the terminal part of the main pancreatic duct before it joins the common bile duct (to prevent reflux of bile into the pancreatic duct), and around the lower part of the common bile duct (to prevent reflux of pancreatic juices into the common bile duct).

Figure 15 Pancreatic duct and common bile duct
The endocrine component of the pancreas consists of clusters of islet cells, formerly known as the islets of Langerhans, which secrete the hormones glucagon, insulin, somatostatin, and pancreatic polypeptide. Islets constitute only about 2% of the pancreatic parenchyma. The pancreatic islets each contain four varieties of cells:

1. Alpha cell- produces the hormone glucagon, which plays an important role in blood glucose regulation; low blood glucose levels stimulate its release; makes up approximately 20% of each islet
2. Beta cell- produces the hormone insulin; elevated blood glucose levels stimulate the release of insulin to lower blood sugar; makes up approximately 75% of each islet
3. Delta cell- secretes the peptide hormone somatostatin, which is also released by the hypothalamus, the stomach, and the intestines; pancreatic somatostatin inhibits the release of both glucagon and insulin; makes up approximately 4% of each islet
4. PP cell- secretes the pancreatic polypeptide hormone, which is thought to play a role in appetite, as well as the regulation of pancreatic exocrine and endocrine secretions; may reduce further food consumption when released following a meal, but is also released in response to fasting; makes up approximately 1% of each islet

Pancreatic hormones are released directly into the bloodstream. Their work in maintaining proper blood sugar levels is crucial to the functioning of key organs, including the brain, liver, and kidney.

**Physiology**

**Regulation of Blood Glucose Levels**

Glucose is required for cellular respiration and is the preferred fuel for all body cells. It is derived from the breakdown of the carbohydrate-containing foods and drinks we consume. Glucose not immediately taken up by cells for fuel can be stored by the liver and muscles as glycogen, or converted to triglycerides and stored in the adipose tissue. Hormones regulate both the storage and the utilization of glucose as required. Receptors located in the pancreas sense blood glucose levels, and subsequently the pancreatic cells secrete glucagon or insulin to maintain normal levels.
Glucagon Regulation

During periods of fasting or during prolonged labor or exercise, receptors in the pancreas sense the decline in blood glucose levels. The alpha cells of the pancreas secrete the hormone glucagon, which has several effects:

1. It stimulates the liver to convert its stores of glycogen back into glucose, which is released into the circulation for use by cells of the body. This response is known as glycogenolysis.
2. It stimulates the liver to take up amino acids from the blood and convert them into glucose, which is called gluconeogenesis.
3. It stimulates lipolysis, which is the breakdown of stored triglycerides into free fatty acids and glycerol. Some of the free glycerol released into the bloodstream travels to the liver, which converts it into glucose. This is also a form of gluconeogenesis.

Together, these actions increase blood glucose levels. The activity of glucagon is regulated through a negative feedback mechanism; rising blood glucose levels inhibit further glucagon production and secretion.

Insulin Regulation

The primary function of insulin is to facilitate the uptake of glucose into body cells. Red blood cells, cells of the brain, liver, and kidneys, as well as the lining of the small intestine, do not have insulin receptors on their cell membranes, and do not require insulin for glucose uptake. All other body cells do require insulin if they are to take glucose from the bloodstream, with skeletal muscle cells and adipose cells being the primary targets of insulin.

The presence of food in the intestine triggers the release of gastrointestinal tract hormones, such as glucose-dependent insulinotropic peptide. This is the initial trigger for insulin production and secretion by the beta cells of the pancreas. Once nutrient absorption occurs, the resulting surge in blood glucose levels further stimulates insulin secretion. Insulin triggers the rapid movement of a pool of glucose transporter vesicles to the cell membrane, where they fuse and expose the glucose transporters to the extracellular fluid. The transporters then move glucose into the cell interior by facilitated diffusion.

Insulin also reduces blood glucose levels by stimulating glycolysis, which is the metabolism of glucose for generation of ATP (adenosine triphosphate). It stimulates the liver to convert excess glucose into glycogen for storage, and inhibits enzymes involved in glycogenolysis and gluconeogenesis. Insulin also promotes triglyceride and protein synthesis. The secretion of insulin is regulated through a negative feedback mechanism; as blood glucose levels decrease, further insulin release is inhibited.
MR Imaging of Pancreas Pathology

The most important issues in pancreatic imaging are the detection and staging of pancreatic cancer, the differentiation between cancer and focal pancreatitis, the characterization of cystic lesions, and the search for neuroendocrine tumors. MR can be used to provide a definitive diagnosis in patients with equivocal findings from CT or ultrasound, as to whether or not a tumor is present. The use of MR in cancer staging aids in identifying patients with advanced tumors that are unresectable. Cystic lesions found in CT may be better characterized through the use of MR.

T1-weighted gradient echo images performed in- and out-of-phase provide good anatomic detail of the pancreas. The normal pancreas has the highest signal intensity of the intra-abdominal organs on fat-suppressed T1-weighted images (Figure 16). This is attributed to the larger amount of aqueous protein within the glandular elements, an abundance of endoplasmatic reticulum of the protein-producing acinar cells, and the high content of paramagnetic ions, such as manganese, within the pancreas. In elderly patients, this high T1 signal intensity may be reduced in comparison with the signal intensity of the liver, most likely due to age-related fibrosis. The most common pancreatic diseases, which are pancreatitis and pancreatic adenocarcinoma, have longer T1 times due to increased free water protons. These diseases then have lower signal intensity compared with that of the surrounding normal pancreatic parenchyma. The conspicuity of pancreatic tumors and the delineation of the pancreas from surrounding fat are best established on fat-suppressed T1-weighted images.

Figure 16 Pancreas displays high signal intensity on T1 fat suppressed image
Normal pancreatic parenchyma has a shorter T2 than most abdominal organs, and exhibits low-to-intermediate signal on T2-weighted images (Figure 17). The liver, and especially the spleen, have a longer T2 than the pancreas, and demonstrate normally higher signal intensity than the pancreas on T2-weighted images. Single shot Fast Spin Echo (FSE) sequences with breath-holds also produce T2-weighted images, but may have a lower signal-to-noise ratio and blurring of tissues due to their higher echo factors. T2-weighted FSE images with and without fat suppression provide good delineation of organ contour, and the presence of peripancreatic inflammation, but less lesion contrast is obtained.

![Figure 17 T2-weighted pancreas exhibit low to intermediate signal](image)

Administration of a gadolinium chelate contrast agent increases the differences in signal intensity between normal pancreatic parenchyma and the usually less vascular neoplastic tissue. Gadolinium-enhanced MR is useful in evaluating acute pancreatitis, and differentiating hypervascular pancreatic masses that may simulate cystic lesions on non-contrast images. Dynamic T1-weighted gradient echo images with fat saturation are often performed post-contrast, with imaging performed in different phases of perfusion (Figure 18).

![Figure 18 T1 post gadolinium fat suppressed images in different phases of perfusion](image)

A liver-specific contrast agent called Mn-DPDP (manganese dipyridoxyl diphosphate, also known as mangafodipir trisodium) has found use in pancreatic MRI. Normal pancreatic tissue becomes hyperintense on T1-weighted images following intravenous administration of Mn-DPDP, allowing for easier delineation of unenhanced pancreatic ductal adenocarcinoma from normal pancreas. This is especially useful in diagnosing smaller tumors, and in differentiating pancreatic carcinomas from chronic pancreatitis.
MR pancreatography or cholangio-pancreatography (MRCP) has several advantages over endoscopic retrograde pancreatography. When performed using MR, this study is noninvasive, requires no anesthesia, and does not use ionizing radiation. There is no increased risk of acute pancreatitis, and it can be performed on patients with altered pancreaticobiliary anatomy following surgery, as well as in patients with complete obstruction of the ducts. MRCP can also demonstrate the upstream anatomy, as well as periductal abnormalities. Visualization of the duct is based on the signal features in the pancreatic secretions, so the duct can be visualized before and after an obstructing lesion occurs. The duct fluid can actually provide additional image contrast with which to visualize a non-gland-deforming tumor. MRCP images are typically heavily T2-weighted and are especially sensitive to fluid (Figure 19). High signal intensity is produced not only for the cholangiographic tract, but also for the intra-gastrointestinal tract liquid component. A gastrointestinal T2-shortening negative contrast agent may be useful in minimizing this problem.

![Figure 19 Magnetic resonance pancreatography results in heavily T2-weighted image of pancreatic duct with hyperintense pancreatic juices](image)

3D gadolinium-enhanced magnetic resonance angiography (3D Gd-MRA) is the best technique to evaluate the peripancreatic vessels using MR, especially in patients with pancreatic neoplasm. This method can also be used to exclude arterial or venous invasion, and to diagnose anatomical vascular abnormalities or variants. The signal obtained is determined by the local concentration of gadolinium in the vessels; therefore, image acquisitions following an optimal time delay is required. Subtraction images can also be acquired to highlight venous anatomy selectively, to remove disturbing high-intensity background tissue, or to evaluate flow patterns or organ perfusion.

Functional MR of the pancreas has been investigated, in which MR images are obtained before and after the administration of IV secretin. After slow IV administration, secretin will stimulate the secretion of fluid and bicarbonate by the exocrine pancreas, and increase the tonus of the sphincter of Oddi, thereby improving the visualization of the pancreatic duct and its side branches. The volume of effluent into the duodenal lumen can be graded, allowing a relative estimation of the exocrine reserve. The main indications for functional imaging are the characterization of filling defects, the assessment of exocrine function in the presence of parenchyma atrophy, and a better evaluation of the papillary region.
Ductal Pancreatic Adenocarcinoma

Ductal pancreatic adenocarcinoma accounts for approximately 90% of all malignant pancreatic neoplasms. Approximately 80% of the tumors occur in patients 60-80 years of age, affecting men about twice as often as women. In up to 70% of the cases, the tumor is located within the pancreatic head. Due to the close relationship of the pancreatic head to the common bile duct and duodenum, pancreatic adenocarcinoma in the head generally presents at an earlier stage than tumors in the pancreatic body or tail. At the time of clinical presentation, two thirds of patients have an advanced tumor stage, with metastatic disease present in 85% of cases, which may preclude surgical resection. The pancreas is not confined by a distinct capsule, so the invasion of surrounding structures is common, especially the peripancreatic vessels.

On T1-weighted unenhanced fat-suppressed images, pancreatic cancer appears as a low-signal intensity mass, with even small tumors clearly separated from the normally hyperintense pancreatic tissue (Figure 20). On T1-weighted post-gadolinium images, pancreatic ductal adenocarcinoma tends to be hypointense, as it is typically a hypovascular lesion. Delineation of tumors is difficult on T2-weighted images, as they may appear iso- or only mildly hyperintense.

![Figure 20 Ductal pancreatic adenocarcinoma; image on left shows fat suppressed pancreatic cancer appearing as low signal intensity mass; image in center shows low signal intensity mass on T1-weighted images post contrast during pancreatic phase; image on right shows secondary finding of pancreatic duct dilatation](image-url)
Secondary findings associated with pancreatic adenocarcinoma include pancreatic duct dilatation, atrophy of the tail of the pancreas, and dilated collateral veins, which suggest venous invasion (Figure 21). Post-gad gradient images are important for the detection of hepatic metastases, specifically during the portal venous phase.

![Image](image.png)

**Figure 21** Gadolinium-enhanced T1-weighted gradient echo image shows a hypointense mass in the pancreatic head, which abuts the superior mesenteric artery; superior mesenteric vein is not visible, indicative of tumor compression

### Cystic Epithelial Pancreatic Neoplasms

MRI has great potential to accurately assess cystic neoplasms, due to its heightened sensitivity to fluid. Fluid within the cyst improves contrast resolution within the mass, often rendering subtle irregularities of the cyst wall that aid in differential diagnosis between benign and malignant neoplasms and pseudocysts.

### Serous Cystadenoma

A serous cystadenoma is generally considered to be a benign neoplasm, with only sporadic reports of malignant degeneration. It represents 2% of all pancreatic neoplasms, occurring most frequently in elderly patients, more commonly in women (70%), with a propensity for occurring in the pancreatic head. Serous cystadenomas can be microcystic or macrocystic, with those that are microcystic measuring 6-10 cm in diameter, consisting of multiple smaller cysts (2 cm in diameter) divided by thin septations. This well-circumscribed tumor is often lobulated, and contains a central, stellate, calcified scar. The presence of multiple small cysts within this mass is suggestive of serous cystic rather than mucinous cystic neoplasm. This tumor is typically not associated with pancreatic duct dilatation or atrophy of the tail of the pancreas.
MR images show the well-delineated contour of serous cystadenomas, the thin septations, and their clear fluid cystic components. The fluid in the cysts is glycogen-rich, with a notable absence of mucin. These tumors are hypervascular, secondary to their rich subepithelial capillary network, and have a propensity for hemorrhage. T1-weighted images show these tumors to be of low signal intensity, except in cases in which the tumors have hemorrhaged. Areas of hemorrhage will appear hyperintense on T1-weighted images. Serous cystic tumors are usually markedly hyperintense on T2-weighted images (Figure 22). They may display some central areas of low signal intensity, caused by the presence of a fibrous scar or calcification.

Figure 22 Serous cystadenoma displays as hyperintense on T2-weighted image; central area of low signal intensity from fibrous scar or calcification
Mucinous Cystic Tumors

Mucinous cystic tumors range from tumors with malignant potential to frankly malignant mucinous cystadenocarcinoma. The mucinous macrocystic adenoma is a benign lesion that usually presents in the fifth decade, and is more common in women (90%). Approximately 85% of these neoplasms arise in the tail or body of the pancreas. Typically, these solitary, large hypovascular tumors are multilocular and contain smaller individual cysts. The cysts contain mucin, a class of glycoproteins found in saliva, gastric juices, etc. that act as lubricants or protectants. Mucinous tumors have a thick wall, internal septations, solid papillary excretions, and may have peripheral calcifications. MRI shows the unilocular or multilocular nature of this mass; however, if it is unilocular and without septations on imaging, its differentiation from a pseudocyst may not be possible. MR images show the content of these cystic masses to be variable in signal intensity, which is related to either hemorrhage or the proteinaceous nature of the fluid. Images obtained after the intravenous injection of contrast material may demonstrate enhancement of the septations and peripheral wall (Figures 23, 24).

Figure 23 Solitary large mass in image on left is a mucinous cystic tumor; same type of tumor on right displays peripheral wall enhancement after contrast

Figure 24 MRCP image on left shows a macrocystic mass; Gadolinium-enhanced T1 gradient echo image on right shows septations, but no solid nodules, which indicates malignancy
Solid Pseudopapillary Tumors

Solid pseudopapillary tumors are benign or low-grade malignancies that represent 1-2% of all pancreatic tumors. They typically present in young women (average age 30 years), and are often found incidentally, or in a work-up for abdominal discomfort. Metastases are rare, but local recurrence is possible. After resection of the tumor, the prognosis is excellent, with a cure rate of 95%. Solid pseudopapillary tumors are large (average diameter of 10 cm), well-demarcated, solitary masses occurring in every portion of the pancreas. They are encapsulated by a thick, fibrous capsule, so invasion into adjacent organs is very rare. The tumor can be entirely solid; however, with increasing tumor size, solid and cystic components can be found side by side due to hemorrhage and necrosis. The solid parts of the tumor are well vascularized, and tend to have calcifications. High T1 and low T2 signal intensities are classic MR features if internal hemorrhage has occurred (Figure 25). These tumors appear as large heterogeneous masses with a thick, solid capsule that enhances after contrast injection.

Figure 25 Image on left is solid pseudopapillary tumor, displaying internal hemorrhage with high signal intensity; image on right displays low T2 signal intensity; these tumors typically have thick solid capsules
Intrapapillary Mucinous Neoplasm

A relatively recent and increasingly reported entity is the intrapapillary mucinous neoplasm (IPMN). This mucin-producing tumor is thought to originate in the main pancreatic duct or its side branches. When arising from a side branch, they are most commonly located in the uncinate process. They have either papillary hyperplasia, atypical, or malignant epithelium that can cause local and vascular invasion and distant metastases. They present at an average age of 65, and are slightly more common in men. An IPMN will typically appear on MRI as a uni- or multilocular cystic lesion combined with a dilated main pancreatic duct due to marked mucin secretion. Communication between the main pancreatic duct and the cystic lesion may be depicted. The most specific predictive signs of a malignant IPMN tumor are the presence of a solid mass, main pancreatic duct dilation, diffuse or multifocal involvement, and attenuating or calcified intraluminal content (Figures 26,27).

![Image 1](image1.png) ![Image 2](image2.png)

**Figure 26** Image on left demonstrates an intrapapillary mucinous neoplasm with a multilocular cystic lesion (arrow); image on right demonstrates communication between the main pancreatic duct and a cystic lesion (arrow)

![Image 3](image3.png)

**Figure 27** MRCP with intraductal papillary mucinous tumor displaying severely dilated pancreatic duct

Other epithelial exocrine pancreatic tumors, such as acinar cell carcinoma and pancreatoblastoma, are rare; however, pancreatoblastoma is the most common pancreatic tumor in children. Both of these tumors present as a large mass, and are more prevalent in males. Acinar cell carcinomas presents at an average age of 65, while pancreatoblastoma presents at an average age of 4.
Endocrine Pancreatic Tumors

Endocrine pancreatic tumors, formally known as islet cell tumors, are benign or malignant neoplasms with endocrine cell differentiation. Most are classified as hyperfunctioning (insulinoma, gastrinoma, glucagonoma, vipoma, and somatostatinoma) or nonhyperfunctioning, and can be located in the pancreas or peripancreatic tissue. These are uncommon tumors, representing only 1-2% of all pancreatic tumors, and found most often in patients in their fifties.

Endocrine tumors typically become clinically apparent due to their hormone release and associated symptoms, or because of effects related to their size, especially with the nonhyperfunctional tumors. Malignancy amongst these tumors can be difficult to assess microscopically, as vascular invasion is the only reliable marker. Macroscopic markers of malignancy are direct invasion by the tumor into adjacent organs, and metastases. The likelihood of malignancy increases along with tumor size, heterogeneity, multiplicity, and presence of calcifications.

Hyperfunctioning Tumors

Insulinomas are the most common endocrine tumors of the pancreas. Their clinical onset is typically associated with what is called the Whipple triad (starvation attack, hypoglycemia after a fasting period, and relief by IV dextrose). At the time of diagnosis, these tumors are usually small, solitary intrapancreatic lesions. Local invasion is found in 25% of cases, and distant metastases in 10%. Malignant insulinomas typically present with a larger diameter. Multiple insulinomas are found in 5-10% of cases, with a slightly higher incidence in the body and tail. The classic findings of insulinoma are a solid, homogeneous, hypervascular mass.

Gastrinomas are the second most common endocrine tumor of the pancreas. Their clinical presentation is determined by excessive gastrin production, which induces hypersecretion of gastric acid, followed by peptic ulcerations and diarrhea. In up to 90% of cases, this typically small tumor occurs within the “gastrinoma triangle” - superior margin is the junction of the cystic and common bile duct, inferior margin is the second and third part of the duodenum, and medial margin is the junction between the head and body of the pancreas. Forty percent of gastrinomas are located outside the pancreas, with distant metastases detected in 25% of cases at the time of diagnosis. They usually present as solid, homogeneous, hypervascular lesions. Twenty percent of the masses will have associated calcification.

Glucagonomas are uncommon endocrine tumors, found predominantly in the pancreatic body and tail. Their syndrome includes diabetes mellitus anemia, weight loss, and hypercoagulability. Glucagonomas are associated with local invasion in 40% of cases, and distant metastases in 55% of cases. They tend to be larger than insulinomas or gastrinomas, with the majority presenting as solitary, heterogeneous cystic lesions.

Only about 8% of hyperfunctional pancreatic tumors are vipomas, but 60% of vipomas are malignant. These tumors are characterized by the secretion of vasoactive intestinal peptide, which produces a syndrome that includes watery diarrhea, and hypokalemia. These tumors are typically found in the body and tail of the pancreas, with 4-10 cm diameters.

Somatostatinomas are the rarest endocrine pancreatic tumors, with 75% being malignant at the time of diagnosis. The clinical symptoms are related to the complex effects of the suppression of growth
hormone, thyroid-stimulating hormone, insulin, glucagon, gastric acid, pepsin, and secretin. Patients present with diabetes, mellitus, gallstones, low gastric output, and weight loss.

**Nonhyperfunctioning Endocrine Tumors**

As a group, these endocrine tumors are the third most common islet cell neoplasms, accounting for approximately 15% of all endocrine pancreatic tumors. They typically cause no symptoms until patients present with stomach or biliary tract obstruction. At that time, 90% of the tumors are malignant, with distant metastases in 25%, and local invasion in 50%. These tumors are usually larger in size, and tend to necrose and hemorrhage.

**MR Imaging of Hyperfunctioning and Nonhyperfunctioning Endocrine Tumors**

Insulinomas and gastrinomas appear as lesions with low signal intensity on T1-weighted images, and high signal intensity on T2-weighted images. Fat suppression sequences are useful in emphasizing the signal intensity differences between tumor and normal hyperintense pancreatic tissue. The use of IV gadolinium is helpful, especially in the detection of islet cell tumors, as they are hypervascular (Figure 28). Ring-like enhancement in the periphery of the tumor is frequently seen, while the center may remain hypointense, secondary to fibrosis. The more uncommon, but usually larger hyperfunctioning (glucagonoma, vipoma, somatostatinoma) and nonhyperfunctioning tumors present with slightly lower signal intensity on T1-weighted images, and moderate signal intensity on T2-weighted images. Contrast administration reveals an enhancement pattern similar to that of the smaller insulinomas or gastrinomas. Hemorrhage or necrosis may occur in large tumors, with each being easily detected on MR.

![Figure 28 Insulinoma; T2-weighted FSE image on left shows a round mass in the head, which is minimally hyperintense (arrow); hypervascular tumor is clearer on gadolinium-enhanced T1 image on right (arrow)](image)

**Additional Endocrine Tumors**

Non-epithelial tumors such as lymphoma, teratoma, lymphangioma, lipoma, and neural tumors can occur in the pancreas. Lymphoma can cause multiple or solitary lesions that are associated with adenopathy and advanced systemic involvement.

Metastatic disease can also spread to the pancreas, usually from primary renal, lung, or breast cancer, as well as melanoma. Metastases typically occur in the presence of advanced metastatic disease.
Pancreatitis

Imaging in cases of suspected pancreatitis is used to help confirm the diagnosis, to establish the cause of the disease, to assess the severity, and to detect complications.

Acute Pancreatitis

Acute pancreatitis is an acute inflammatory process of the pancreas, with variable involvement of other regional tissues or remote organ systems. The three main causes include cholelithiasis (38%), alcoholism (35%), and idiopathic etiologies. Acute pancreatitis can be classified as mild, which involves an edematous gland and/or spotty peripancreatic fatty tissue necrosis, or severe, which involves alternating chalky white fat necrosis and hemorrhage and/or pancreatic necrosis.

In assessing acute pancreatitis, MRI can evenly depict the presence and extent of necrosis and peripancreatic fluid collections, and has been found to be superior to CT in the detection of mild acute pancreatitis. For staging of acute pancreatitis and reaching a prognosis, a recommended protocol should include a fat-suppressed T1-weighted sequence, a T2-weighted sequence, and T1-weighted gradient echo sequences prior to and immediately following gadolinium administration.

For severe acute pancreatitis, parenchymal edema is best shown on an unenhanced T1-weighted sequence. T2-weighted sequences are the most sensitive in revealing fluid collections. Gadolinium-enhanced sequences are useful for the assessment of pancreatic parenchymal perfusion and the presence of necrosis. Pancreatic enhancement is maximal within 20-40 seconds after gadolinium administration. The extent of parenchymal necrosis is well demonstrated on sequential, multi-slice acquisitions obtained during the first 1-2 minutes after the gadolinium injection. The additional use of MRCP or MRA allows the accurate diagnosis of underlying etiologies, such as choledocholithiasis (gallstones in common bile duct) or pancreas divisum (congenital anomaly involving dorsal and ventral pancreatic ducts, rather than a single duct), as well as vascular complications.

Chronic Pancreatitis

Chronic pancreatitis is an irreversible clinical disorder that includes irregular fibrosis and cellular infiltration, ductal abnormalities, and loss of exocrine and endocrine pancreatic function. MRI should include T1- and T2-weighted sequences, and MRCP. Chronic pancreatitis presents with decreased T1 and variable T2 signal intensity. Enhancement after gadolinium administration is reduced and delayed. MRCP can be helpful in assessing the extent of morphologic ductal changes, and the severity of the disease.

Autoimmune pancreatitis is a special form of chronic pancreatitis that is unique in its effective clinical response to steroid therapy. On MRI, it presents as a diffuse or focal pancreatic enlargement associated with narrowing of the main pancreatic duct, with decreased signal intensity on T1-weighted images and increased signal intensity on T2-weighted images. Contrast-enhanced imaging may show a peripancreatic rim of decreased enhancement surrounding the pancreas and the common bile duct on the arterial phase images.
Focal Pancreatitis

Imaging modalities have difficulty differentiating between focal pancreatitis and pancreatic adenocarcinoma. Both types of lesions occur most often in the pancreatic head, may cause bile duct obstruction, and may present in the absence of calcification and duct strictures or dilatation. Patients with an inflammatory mass are likely to be younger at the time of presentation, and have a history of alcoholism and previous episodes of acute or chronic pancreatitis. MRI can help differentiate these two pathologies, especially with respect to focal or diffuse changes in signal intensity and contrast enhancement. On fat-suppressed T1-weighted images, areas of chronic pancreatitis show diminished signal intensity compared with the normal pancreas, but the degree of signal reduction is usually less than that associated with carcinoma. Inflammatory masses in chronic pancreatitis may result in signal intensities comparable with those of the liver, whereas carcinoma often shows signal intensities hypointense to the liver. The “duct penetrating sign” on MRCP images may contribute to characterization: a tumor mass does not contain pancreatic ductal structures, whereas focal pancreatitis may display numerous dilated side branches traversing the mass. The most reliable sign with gadolinium-enhanced MRI is the detection of a focal and defined mass.
Spleen

Anatomy

The spleen is a spongy oval-shaped organ that is roughly the size of a person’s fist. It is located in the left upper quadrant of the abdomen, just under the left ribcage, but is not palpable when healthy. The spleen has many important functions in the body involving the filtration and storage of blood, and immune activity, but it is not a vital organ. It can be surgically removed without significantly impairing the quality of life, although a person may be more prone to infections. Red bone marrow, the liver, and lymph nodes can complete the filtration and blood recycling functions of the spleen in its absence. Damage to the spleen is almost always treated by its complete removal. Because it is so spongy and vascular, untreated damage to the spleen can quickly lead to massive internal hemorrhaging and eventual death.

The approximate dimensions of the spleen are 5 inches in length, 3 inches in width, and 1 inch thick. Its weight averages 5-7 ounces, but it can expand up to 3 times its size, which significantly increases its weight. If enlarged, the spleen is palpable on the anterolateral abdominal wall, below the left costal margin. It sits against the diaphragm and the posterior abdominal wall, in close relation to the left 9th-11th ribs. This location is helpful in protecting the spleen, which is a fragile organ, but the protective feature of the ribs can be detrimental. A fractured rib can pierce the spleen and cause it to rupture.

The spleen lies behind the stomach, above the left colic flexure, and to the lateral side of the left kidney (Figure 29). It is surrounded by peritoneum (except at the splenic hilum), and is suspended by ligaments. The gastroplenic ligament connects the spleen to the greater curvature of the stomach. The splenorenal ligament connects the spleen to the left kidney, and contains the tail of the pancreas. These ligaments contain the splenic vessels, and are attached to the medial aspect, or visceral surface, of the spleen.

The two surfaces of the spleen are the diaphragmatic and the visceral surfaces. The diaphragmatic surface is smooth and convex; the diaphragm separates the spleen from the pleura and the lung. The visceral surface is on the medial aspect of the spleen, and is irregular and concave, with “impressions” for surrounding anatomy that contacts the spleen. The gastric impression is for the fundus of the stomach, which is the largest and most concave impression on the spleen. The renal impression is for the anterior surface of the left kidney. The colic impression is for the splenic flexure of the colon, and the pancreatic impression is for the tail of the pancreas.
A tough, connective fibroelastic tissue capsule surrounds the soft inner tissue of the spleen. The spongy inner tissue contains many tiny blood vessels and hollow sinuses that store blood. The spleen can release its stored blood into circulation to replace blood lost during a traumatic injury. Many platelets are also stored with the blood in the spleen to help form blood clots to prevent further blood loss. Around the vessels and sinuses of the spleen are the parenchymal tissue regions of pulp, which consist of red pulp, and smaller nodules known as white pulp, with a marginal zone in between. The red pulp regions contain many net-like reticular fibers that filter worn-out red blood cells from the blood flowing through the spleen. Captured red blood cells are digested to recycle the iron and protein components of hemoglobin. The marginal zone between the red and white pulp acts as a filter to capture pathogens in the blood and pass them on to the white pulp. White pulp regions are made of lymphatic tissue containing macrophages, T lymphocytes, and B lymphocytes that destroy pathogens in the blood and produce antibodies. Arteries that enter the parenchymal pulp are surrounded by the white pulp nodules, which contain large amounts of white blood cells that trap any invading pathogens. The blood then filters through the sinuses of the red pulp, and exits the spleen via the veins. The spleen may enlarge during certain infections due to an increase in the number of white blood cells, captured pathogens, and antibodies inside the spleen.

The splenic hilum is on the medial aspect of the spleen, and is the only splenic area not surrounded by visceral peritoneum. This is the site where the many branches of the splenic artery enter the spleen, and the splenic vein exits. The splenic artery is the largest branch of the celiac trunk, which is a major branch of the abdominal aorta. The splenic artery reaches the hilum by passing through the splenorenal ligament. Before entering the spleen proper, it divides into five or more branches. Splenic circulation is adapted for the separation and storage of the red blood cells. The spleen has superior and inferior vascular segments based on the blood supply. The segments are separated by an avascular plane. The splenic vein arises from several tributaries within the spleen to provide the principle venous drainage of the spleen. It is joined by the inferior mesenteric vein initially, and later with the superior mesenteric vein to form the hepatic portal vein.

Lymphatic fluid from the spleen empties into the lymph nodes at the splenic hilum. The lymph then passes through the pancreaticosplenic lymph nodes, and empties into the celiac nodes. The splenic nerve supply is derived from the celiac plexus.

**Physiology**

The functions of the spleen include immune responses, phagocytosis, hematopoiesis, and storage of red blood cells. Phagocytosis is the process whereby living cells ingest other cells. Hematopoiesis involves the formation, development and differentiation of blood cells.

**Immune Responses**

The spleen is the largest lymphatic organ in the body. It helps white blood cells to proliferate, and initiates the appropriate immune response when necessary. Any microorganisms in the bloodstream are quickly trapped by the lymphocytes of the white pulp upon entering the spleen via the arteries. The lymphocytes can also exit the spleen and enter the general circulation to fight an infection at a distant site. Lymphocytes are the main cells in lymph, and one of five types of white blood cells.
Phagocytosis

Phagocytosis, which involves living cells ingesting and destroying unwanted foreign material, is the spleen’s most important function. The spleen is a component of the reticuloendothelial system, which also includes the blood, connective tissue, liver, lungs, bone marrow, and lymph nodes. The cells and tissues in this system are concerned with blood cell formation and destruction, storage of fatty materials, and metabolism of iron and pigment. They also play a role in inflammation and immunity. Splenic phagocytes include reticular cells and free macrophages of the red pulp. Phagocytes in the spleen filter the blood by removing debris, old and ineffectual red blood cells, other blood cells, and microorganisms. The spleen also recycles the iron and globin component of the hemoglobin molecule. Phagocytosis of circulating antigens initiates the immune response.

Hematopoiesis

The spleen is an important hematopoietic organ during fetal life, meaning it is involved in the formation, development, and differentiation of blood cells. However, after birth, the spleen’s role is to destroy worn out red blood cells and platelets. In the adult spleen, hematopoiesis may restart in certain diseases, such as chronic myeloid leukemia and myelosclerosis. The lymphopoiesis process, which is the generation of lymphocytes, continues throughout life.

Storage of Red Blood Cells

The red pulp of the spleen is also a blood reservoir, and stores red blood cells, platelets, and monocytes until they are needed in the circulation. Approximately 8% of the circulating red blood cells are present within the spleen. The elastic splenic capsule has smooth muscle within it; when required, it can contract and “squeeze” a substantial volume of blood into the circulation.
MR Imaging of Spleen Pathology

MRI permits the characterization of the most common splenic lesions, such as cysts, small hemangiomas, and hamartomas, as well as improvement in the detection of malignant diseases such as lymphoma and metastases.

The normal signal intensity of the spleen on T1-weighted images is lower than that of hepatic tissue. On T2-weighted images, the spleen shows higher signal intensity, appearing brighter than the liver (Figure 30). The distinctive microscopic anatomy of the spleen may be reflected on diffusion-weighted images and ADC maps. Studies have shown significant differences in the mean ADC’s between the spleen and other abdominal organs. Immediately after contrast administration, the spleen demonstrates a heterogeneous serpentine or arciform pattern enhancement, which is secondary to the differences in flow between the red pulp and white pulp (Figure 31). This pattern should appear homogeneous in venous and interstitial phases, which occur 60-90 seconds after injection. Any heterogeneity after this period is considered pathologic.

Figure 30 Normal spleen showing coffee bean configuration; axial T2 FSE with fat suppression image on left, with normal spleen showing higher signal intensity than hepatic tissue; axial T1 out-of-phase gradient echo image on right showing signal intensity of normal spleen lower than that of hepatic tissue

Figure 31 Pattern of enhancement of normal spleen in post-contrast axial T1 3D gradient echo with fat suppression; arterial phase on left with spleen showing a heterogeneous pattern of enhancement, secondary to differences in flow between red and white pulps; venous phase on right showing homogeneous pattern
Normal Variants and Congenital Diseases

The congenital absence of a spleen is known as asplenia, and the presence of one or more spleens is known as polysplenia. Both conditions are very rare, and usually associated with other congenital abnormalities. Polysplenia is more common in females, and often seen in conjunction with abdominal situs and cardiovascular anomalies. Classically, numerous small splenic masses are seen in the right or left hypochondrium.

Accessory spleens are found in 10% of individuals, and are usually less than 4 cm in diameter. They are typically located near the splenic hilum or near the pancreatic tail (Figure 32). The presence of accessory splenules may arise within the substance of solid organs, notably the pancreas. A well-margined rounded mass located within 3 cm of the distal tail of the pancreas with signal intensity features of the spleen on all MR sequences suggests the diagnosis of intrapancreatic accessory spleen. DWI and superparamagnetic iron oxide- enhanced MRI can be used to characterize the lesion and to establish the definite diagnosis, as other entities may mimic the signal intensity and post-gadolinium enhancement features of an intrapancreatic accessory spleen.

An additional normal variant is the upside-down spleen, which occurs due to an abnormal splenic rotation. The hilum will be superiorly located and the convex border will be medial and adjacent to the left kidney.
Trauma

The spleen is the most commonly ruptured intra-abdominal organ due to trauma. It is particularly susceptible to injury after blunt trauma due to its complex ligamentous attachments and spongy parenchymal consistency. Trauma to the spleen can cause the development of splenosis, in which splenic tissue is seeded within the abdomen or pelvis, causing confusion in diagnosis (Figures 33, 34). MR imaging characteristics of splenic hematomas follow those of heme and heme products, with evolution like hematomas in other parts of the body. Acute hematomas demonstrate prolonged T2 times, when compared to splenic signal intensity.

Figure 33 Pelvic splenosis; axial T2 SS-FSE on left with multiple nodular masses in left hypochondrium (arrows) consistent with splenosis; axial T2 FSE on right displays multiple well-defined nodules in pelvis demonstrating high signal intensity on T2 (Arrow)

Figure 34 Post-contrast axial T1 3D gradient with fat suppression in arterial phase on left, and venous phase on right; heterogeneous enhancement immediately after contrast administration on left (arrow), becoming homogeneous in venous phase (arrow), consistent with splenosis
Inflammation

Splenic abscesses were previously considered to be uncommon lesions with high mortality rates, due to delayed detection and treatment. Their frequency has increased due to the higher number of immunocompromised patients, such as those with hematologic disorders (e.g., leukemia), those with recreational intravenous drug abuse, and those with acquired immunodeficiency syndrome (AIDS). These lesions can be solitary, multiple, or multilocular, with irregular and undefined margins. Splenic abscesses are hypointense on T1-weighted images, and display moderate to high signal intensity on T2-weighted images (Figure 35). Following the administration of intravenous contrast, peripheral enhancement may be seen, although it is less intense when compared to a liver abscess.

Candidiasis

Candidiasis is the opportunistic infection that most frequently affects the liver and spleen in immunocompromised patients. MR has been found to be superior to CT for the detection and characterization of splenic microabscesses secondary to candidiasis. These lesions appear as multiple hyperintense lesions on T2-weighted images, and hypointense ring-enhancing lesions on gadolinium-enhanced images (Figure 36).
Histoplasmosis

The prevalence of histoplasmosis is greater in immunocompromised patients, but can also be seen in those with competent immune systems. In the acute and subacute phases of this disease, it is demonstrated on MR as scattered hypointense lesions on both T1- and T2-weighted images (Figure 37). Old granulomas can be calcified, causing characteristic signal intensity changes with blooming artifacts, best appreciated on T1-weighted gradient echo images with long echo times.

![Figure 37 Axial contrast-enhanced 3D gradient on left, and T2-weighted inversion recovery on right show scattered low-signal intensity lesions, which represent infection of the spleen with Histoplasma capsulatum](image)

Sarcoidosis

Sarcoidosis is a granulomatous systemic disease of unknown etiology that may involve several organs. In patients with systemic sarcoidosis, 24-59% had biopsy-documented splenic sarcoidosis. Nodular sarcoidosis has been reported to demonstrate low signal intensity in all MR sequences (Figure 38). The lesions are most conspicuous on T2-weighted fat-suppressed or early phase contrast-enhanced images. Sarcoid lesions enhance minimally on delayed images.

![Figure 38 Sarcoidosis; axial T2 FSE with fat suppression on left, post-contrast axial T1 3D gradient with fat suppression in arterial (center) and venous (right) phases; arrows on left depict low signal intensity nodules in spleen on T2; lesions are most conspicuous on fat suppressed T2 or early phase contrast-enhanced images; note progressive enhancement on venous image](image)
Vascular Disorders

Infarction

Spleenic infarcts may result from venous or arterial blood supply interruption. Vascular occlusions can be the result of a thromboembolic process caused by any type of hemolytic anemia, endocarditis or chronic valvular diseases, hematologic malignancies, vascular collagen diseases, or portal hypertension. On MR, a splenic infarct typically appears as a triangular wedge-shaped area at the periphery of the spleen, with decreased signal intensity on both T1- and T2-weighted images (Figure 39). However, signal intensity may vary according to the age of the infarct. Spleenic infarcts do not enhance after gadolinium injection, and are best depicted in late vascular phases, when the spleen is homogeneously enhanced.

![Figure 39: Splenic infarct; axial T2 FSE on left, axial post-contrast fat suppressed 3D gradient at venous phase on right; small triangular wedge-shaped area at periphery of the spleen is noted (arrow on left) with hypointensity signal on both T1 and T2, with no enhancement on post-contrast images](image)
Hematoma

Splenic hematomas are usually secondary to trauma. Like splenic infarcts, their MR appearance is variable, depending on the age of the lesion. In acute (1 to 2 days) and early subacute phases (2 to 7 days), hematomas show low signal intensity on T2-weighted image, and intermediate and increasingly higher signal intensity on T1-weighted images. In late subacute phase (7 to 14-28 days), hematomas show hyperintensity on both T1- and T2-weighted images. After 3 weeks (chronic), the hematoma may have a cystic appearance, regarded as a hyperintensity lesion on T2-weighted sequences, with low signal intensity on T1-weighted images (Figure 40). Older hematomas appear hypointense on both T1- and T2-weighted images, due to their fibrotic component.

![Hematoma; axial T2 SS-FSE on left, post-contrast axial T1 3D gradient with fat suppression in arterial phase on right; chronic hematoma is depicted with a cystic appearance with moderate hyperintensity on T2 and hypointensity on T1 with no perceptible enhancement](image)

Splenic Artery Aneurysm

Splenic artery aneurysms are secondary to multiple causes, such as portal hypertension, congenital causes, fibromuscular and pseudoaneurysms from trauma and pancreatitis. 3D gradient echo sequences or 3D MRA sequences are best for evaluating these lesions (Figure 41).

![Figure 41 Axial contrast-enhanced 3D gradient echo images show aneurysmal dilatation of distal end of splenic artery (arrows)](image)
Splenic Vein Thrombosis

Splenic vein thrombosis is most commonly secondary to pancreatitis, which causes compression and fibrosis. It can also be caused by erosion of a pseudocyst into the splenic vein. Splenic vein thrombosis may result in esophageal, gastric, or colonic varices. It is usually visualized as an intraluminal filling defect after the administration of intravenous contrast material (Figure 42).

![Figure 42 Axial venous phase gadolinium-enhanced 3D gradient echo image shows a thrombus filling the splenic vein (arrowheads); thrombus appears as an area of signal void](image)

Arteriovenous Malformation

Arteriovenous malformations are somewhat rare in the spleen, but can occur anywhere in the human body. On MR imaging of the spleen, they are demonstrated as multiple signal voids on all non-enhanced pulse sequences. After the intravenous injection of gadolinium contrast material, arteriovenous malformations demonstrate serpentine enhancement (Figure 43).

![Figure 43 Axial T2 inversion recovery on left, and contrast-enhanced 3D gradient echo images on right show a splenic lesion that appears as an area of signal void (arrows); the lesion demonstrates serpentine enhancement on the contrast-enhanced image and represents an arteriovenous malformation](image)
Hematologic Disorders

Sickle Cell Disease and Hemosiderosis

Sickle cell disease is common in the Afro-descendant population, with a prevalence of 0.2% displaying the homozygous form, and 8-10% demonstrating the heterozygous form. The spleen is the most commonly involved organ, appearing as an area nearly void of signal, due to iron deposition from blood transfusions. Autosplenectomy, in which the spleen is shrunken and non-functional, is often found in patients with homozygous sickle cell disease (Figure 44).

![Figure 44](image)

Figure 44 Autosplenectomy in a patient with homozygous sickle cell disease; axial T2 SS-FSE on left, coronal image in center, and post-contrast axial 3D gradient T1 with fat suppression at arterial phase on right; note small remnant of spleen, and diffuse diminished signal intensity of hepatic parenchyma on both T1 and T2 as a result of iron deposition

Hemosiderosis is an iron overload, which can result from hemosiderin deposition. Hemosiderin is an intracellular iron storage complex. When the spleen is involved, MR shows diffuse diminished signal intensity of the organ on both T1- and T2-weighted images, relative to musculature, as a result of hemosiderin deposition (Figure 45).

![Figure 45](image)

Figure 45 Paroxysmal nocturnal hemoglobinuria; coronal and axial T2 SS-FSE on left and center, and axial T2* image on right; diffuse diminished signal intensity of liver and spleen on T2 as a result of hemosiderin deposition; image on right shows iron accumulation on renal cortex
Extramedullary Hematopoiesis

Extramedullary hematopoiesis is a compensatory response to failure of the bone marrow cells. The liver and spleen are mainly the affected organs, and may show a focal mass-like involvement. On MR, the appearance of the nodular lesions depends on the evolution of the hematopoiesis. Active lesions reveal intermediate signal intensity on T1-weighted images, high signal intensity on T2-weighted images, and moderate enhancement after administration of intravenous gadolinium. Older lesions are hypointense on T1- and T2-weighted images, and do not show any enhancement. These lesions usually demonstrate low signal intensity on in-phase T1-weighted gradient echo images compared with that on out-of-phase images due to the presence of iron (Figure 46).

Figure 46 Axial in-phase on left and out-of-phase on right show a splenic area of extramedullary hematopoiesis (arrow); the lesion has reduced signal intensity on the in-phase image compared with that on the out-of-phase image (on right), secondary to iron deposition
Benign Neoplasms or Cysts

Splenic Cyst

Cysts are the most common benign focal splenic masses. Approximately 25% of splenic cysts are epithelial or true cysts, which are lined with epithelium, and include epidermoid and parasitic cysts. True cysts commonly have cyst wall trabeculations or peripheral septations. The remaining 75% of splenic cysts are pseudocysts, which can be posttraumatic or secondary to pancreatitis. Pseudocysts have fibrotic walls, and lack a true cellular lining. Posttraumatic pseudocysts are thought to represent the final stage in the evolution of a splenic hematoma, although they might also be secondary to infarct or infection. Pancreatic pseudocysts arising in the tail of the pancreas may involve the spleen by extending either beneath the splenic capsule, or into the splenic parenchyma proper. Patients with pancreatic pseudocysts usually have a history of acute pancreatitis. MR imaging characteristics of splenic cysts follow those of cysts in other organs of the body, with a well-defined round mass, lack of tissue architecture, and high water content. They demonstrate longer T1 and T2 times relative to normal splenic tissue. Splenic cysts are homogeneous and hypointense on T1-weighted images, hyperintense on T2-weighted images, and do not enhance following the administration of gadolinium contrast material (Figure 47). It is often impossible to distinguish between true and false cysts, but clinical presentation and patient history may help to narrow the differential diagnoses.

Figure 47 Splenic cyst; axial T2 FSE with fat suppression on left, post-contrast axial 3D T1 gradient with fat suppression at interstitial phase on right; note thin-walled and well-defined nodule, homogeneously hyperintense on T2, with no enhancement on post-contrast image, characteristic of cysts.
Hemangiomas

Splenic hemangiomas are the most common benign solid tumors of the spleen. They are more frequently found in males, and are believed to be congenital in origin. Most are less than 2 cm in diameter; however, once large, they can spontaneously rupture, causing intra-abdominal hemorrhage. Splenic hemangiomas are composed of epithelial-lined blood-filled spaces of varying size, and can be characterized by the size of these spaces as capillary or cavernous lesions. Most hemangiomas are well-defined and homogeneous. When compared to splenic parenchyma, they are hypo- to isointense on T1-weighted images, and hyperintense on T2-weighted images (Figure 48). On dynamic contrast-enhanced studies, they usually show peripheral enhancement with centripetal, delayed progression. Late phase images are important for a correct diagnosis as, similar to liver hemangiomas, splenic lesions may undergo sclerosis.

Figure 48 Splenic hemangioma; axial T2 FSE with fat suppression on left, post-contrast axial 3D T1 gradient with fat suppression at arterial (center) and interstitial (right) phases; hemangioma is depicted as a well-defined homogeneous and hyperintense lesion on T2 (arrow), with a peripheral enhancement with centripetal and delayed progression on post-contrast images (center and right); note hepatic hemangiomas on same imaging plane
Hamartomas

Hamartomas are benign, usually asymptomatic, tumors of the spleen. They are non-neoplastic tumors, composed of a mixture of normal elements of splenic red and white pulp components. Splenic hamartomas are considered to be congenital in origin, but are associated with tuberous sclerosis, and possibly previous trauma. Splenic hamartomas are sharply defined, rounded, and typically single solid lesions. They are usually isointense on T1-weighted MR images, and heterogeneously hyperintense on T2-weighted images (Figure 49). After intravenous gadolinium administration, there is usually diffuse heterogeneous enhancement, which may be useful in distinguishing this lesion from the typical peripheral enhancement noted in hemangiomas (Figure 50). Prolonged enhancement may be appreciated, which has been attributed to stagnant contrast material within the sinusoids of the red pulp of splenic tissue. Persistent areas of hypointensity may be seen, which correspond to areas of necrosis within the lesion.

Figure 49 Splenic hamartoma; axial T2 SS-FSE on left, pre-contrast axial 3D T1 gradient with fat suppression on right; multiple rounded lesions are seen on T2 and T1

Figure 50 Splenic hamartoma; post-contrast axial 3D T1 gradient with fat suppression in arterial phase on left, and venous phase on right; lesions demonstrate hyperenhancing characteristics on arterial phase, progressing to isointensity on venous phase
Hydatid Cysts

Hydatid cysts usually involve the liver or lungs, but occasionally they may involve the spleen. On MRI, these cysts share imaging characteristics to those located in the liver. They are hyperintense on T2-weighted images, and hypointense on T1-weighted images (Figure 51). Hydatid cysts can be unilocular, or contain daughter cysts. They may be distributed peripherally, or throughout the lesion, giving them a multilocular appearance. A “serpent” or “snake” sign is sometimes noted, which represents collapsed parasitic membranes within the cyst. A continuous and irregular 4-5 mm-thick, low signal intensity rim surrounding the cyst may be seen, which corresponds to the dense fibrous capsule encasing the parasitic membranes. Typically, there is no enhancement following IV contrast administration.

Figure 51 Hydatid cyst; axial T2 SS-FSE with fat suppression image on left displays splenic hydatid cyst as multilocular lesion with moderate hyperintensity (arrow); axial post-contrast 3D T1 gradient with fat suppression in venous phase in center also displays hydatid cyst in liver; axial post-contrast 3D T1 gradient with fat suppression in venous phase on right shows no enhancement following IV contrast
Littoral Cell Angioma

Littoral cell angioma is a newer, rare benign tumor of the spleen that develops from the lining cells of the red-pulp sinuses, which are the so-called “littoral cells”. These angiomas are of variable size and commonly multinodular. They are composed of anastomosing vascular channels with irregular lumina, featuring cyst-like spaces. On MR, littoral cell angiomas are slightly hypointense on unenhanced T1-weighted images, and inhomogeneously hyperintense on T2-weighted images, with signal intensity similar to that of hemangiomas. They may show low signal intensity on all sequences due to hemosiderin accumulation within neoplastic littoral cells (Figure 52). Dynamic post-contrast T1-weighted images show delayed contrast enhancement, suggestive of a vascular lesion with contrast media pooling (Figure 53).

Figure 52 Littoral cell angioma; axial T2 FSE with fat suppression on left displays a hypointense nodular lesion (arrow); note anteriorly adjacent splenic cyst; pre-contrast axial 3D T1 gradient with fat suppression on right

Figure 53 Littoral cell angioma; post-contrast axial 3D T1 gradient with fat suppression at venous phase on left and 10 minutes post contrast on right; hypovascular nodules show subtle peripheral enhancement with progressive slow centripetal accumulation of contrast, indicated by arrow on right
Lymphangioma

Lymphangioma is a rare benign vascular lesion filled with lymph rather than red blood cells, as seen in hemangioma. It is usually diagnosed in childhood, appearing as solitary or multiple splenic lesions. This condition is also termed lymphangiomatosis when it appears as diffuse involvement replacing most of the splenic parenchyma. The most frequent type of lymphangioma is cystic, characterized by a honeycomb of large and small thin-walled cysts containing lymph-like clear fluid. On MRI, they usually present as well-defined multilocular cystic lesions with thin septations. Lymphangiomas are usually hyperintense on T2-weighted images. Some cysts may also be hyperintense on T1-weighted images due to protein or hemorrhagic content.

Malignant Neoplasms

Angiosarcoma

Although they are exceedingly rare, angiosarcomas are the most common primary malignant nonlymphoid tumors of the spleen. These tumors are highly aggressive, and manifest with wide-spread metastatic disease or splenic rupture. On MR imaging, splenic angiosarcomas typically have low signal intensity on T1-weighted images, and heterogeneous high signal intensity on T2-weighted images (Figure 54). However, signal intensities for both T1- and T2-weighted images can vary, due to the presence of hemorrhage with different ages, siderotic nodules, and areas of necrosis. Following the intravenous administration of gadolinium, these lesions demonstrate heterogeneous enhancement, with multiple hyperintense nodular foci and hypointense regions. The overall precise assessment and staging offered by MRI for this type of tumor is particularly valuable for a timely diagnosis of this rapidly fatal disease.

Figure 54 Coronal contrast enhanced 3D T1 gradient image on left and T2-weighted image on right; splenic mass with low signal intensity on T1 and high signal intensity on T2, with heterogeneous enhancement represents a splenic angiosarcoma
Lymphoma

Lymphoma is the most common splenic malignancy. Both Hodgkin’s and non-Hodgkin’s lymphoma may present in the spleen as the primary site, or as part of systemic involvement. Lymphoma in the spleen may produce homogeneous enlargement (the most common finding), multiple small nodules, a single solitary mass, or a combination of these appearances. Lymphomatous deposits are typically isointense to normal splenic parenchyma on T1- and T2-weighted images (Figure 55). They may present some hypointensity on T2-weighted images, which may help to distinguish them from metastatic lesions. On post-contrast images, lymphomatous lesions are usually hypovascular with lower signal intensity relative to normal spleen, which increases their conspicuity (Figure 56). Diffuse involvement may be seen as large irregularly enhancing regions. Multifocal disease is also common, and may be seen as multiple focal lesions that are hypointense relative to the uniformly or arciform enhancing spleen.

Figure 55 Lymphoma; axial T2 SS-FSE on left with pre-contrast axial 3D T1 gradient with fat suppression on right; spleen is enlarged; lymphomatous nodules are isointense to splenic parenchyma on both T2- and T1-weighted images; T2 image on left displays one moderately hypointense nodule (arrow); this feature aids in distinction against metastatic lesion, which are commonly hyperintense

Figure 56 Lymphoma; post-contrast 3D T1 gradient with fat suppression in arterial phase on left and venous phase in center; coronal fat suppressed 3D T1 gradient in interstitial phase on right; lymphomatous lesions demonstrate hypovascular nature with lower signal intensity relative to normal spleen on post-contrast images, thereby increasing conspicuity
Metastases

Although the spleen is the most vascular organ in the body, it is an infrequent site for metastatic disease. Splenic metastases are mainly due to melanoma and breast cancers, and, to a lesser extent, cancers of the lung, colon, stomach, ovary, endometrium, and prostate. They may be solitary, multiple, or diffuse, and differ in number and size from a few millimeters to several centimeters. On MR imaging, metastases typically appear as hypo- to isointense masses on T1-weighted images, and hyperintense masses on T2-weighted images (Figure 57). The presence of blood products from hemorrhage, or other paramagnetic substances, may result in high signal intensity on T1-weighted images. Central tumor necrosis is seen as regions of hyperintensity on T2-weighted images. The degree and characteristics of enhancement post-contrast depend on the nature and type of the underlying primary neoplasm. Contrast enhancement is typically inhomogeneous, and usually displays a peripheral ring-like pattern.

![Figure 57 Splenic metastasis in patient with small-cell lung carcinoma; axial T2 SS-FSE on left, post-contrast axial 3D T1 gradient with fat suppression at arterial phase in center, and venous phase on right; note nodular lesion depicted as a hyperintense nodule on T2 with peripheral ring-like enhancement](image-url)
Perisplenic Neoplasms Infiltrating the Spleen

Implants on the serosal surface of the spleen are seen in patients with peritoneal carcinomatosis, commonly from ovarian or gastrointestinal primary neoplasms. These implants may cause indentation and scalloping of the surface of the spleen. Direct tumor invasion of the spleen is uncommon, but can be seen in tumors originating from the pancreas, stomach, colon, left kidney, and retroperitoneum (Figures 58, 59).

![Image 1](image1.jpg)

Figure 58 Pancreatic tail clear-cell renal cell carcinoma metastases with infiltration of the spleen through the splenic hilum; axial T2 SS-FSE without fat suppression on left, axial T2 SS-FSE with fat suppression on right; large heterogeneous mass is seen in pancreatic tail infiltrating the spleen

![Image 2](image2.jpg)

Figure 59 Pancreatic tail clear-cell renal cell carcinoma metastases with infiltration of the spleen through the splenic hilum; axial post-contrast 3D T1 gradient with fat suppression in arterial phase on left and venous phase on right; large heterogeneous mass is seen in pancreatic tail infiltrating the spleen
Diffuse Diseases

Splenomegaly

Splenomegaly is a radiologic and clinical sign, classically described when the craniocaudal splenic length is greater than 12 cm (Figure 60). This condition can result from congestion (portal hypertension, splenic vein occlusion, or thrombosis), infiltrative disease (Gaucher disease or histiocytosis), hematologic disorders (polycythemia vera, myelofibrosis), inflammatory/infectious diseases (HIV, mononucleosis, amyloidosis), cysts, or tumors (leukemia, lymphoma, or metastases).

Figure 60 Splenomegaly; coronal T2 SS-FSE on left, post-contrast axial 3D T1 gradient with fat suppression in interstitial phase on right; homogeneous splenomegaly resulting from congestion (portal hypertension) is seen
Portal Hypertension

Portal hypertension is considered to be the most common cause of splenomegaly in the United States. With splenomegaly that is secondary to portal hypertension, MR imaging often reveals associated signs of hepatic cirrhosis with or without change in the liver size, depending on the stage of hypertension. On images obtained immediately post-contrast, uniform enhancement is usually seen. Dilated collateral veins may also be demonstrated at the splenic hilum (Figure 61). Foci of hemosiderin deposition are seen in 9-12% of patients with portal hypertension. These foci are called Gamna-Gandy bodies. On MRI, they demonstrate low signal intensity with all pulse sequences. These foci exhibit “blooming” artifact on gradient echo sequences, secondary to iron deposition.

Figure 61 Axial contrast-enhanced T1 gradient on left, and coronal T2 image on right show hepatosplenomegaly secondary to portal hypertension; note diffuse enhancement of splenic parenchyma on left, and collateral veins at the hilum (arrows on left)
**Gaucher Disease**

Gaucher disease is a disorder secondary to a lack of the enzyme glucocerebrosidase. This enzyme causes the breakdown of glucocerebroside, which is a fat molecule known as a glycolipid that is used in the manufacture of blood cells. When the enzyme glucocerebrosidase is missing, glucocerebroside accumulates inside body organs, causing enlargement of the spleen and liver. On T1-weighted images, signal intensity is low relative to the normal spleen, secondary to the glucocerebroside. Nodal clusters of Gaucher cells appear isointense to the spleen on these images. On T2-weighted images, signal intensity is intermediate, except for the nodal clusters of Gaucher cells, which appear hypointense on T2 (Figure 62). Splenic infarcts and fibrosis associated with Gaucher disease may exhibit a multifocal pattern.

![Image of T2-weighted images showing splenomegaly with Gaucher lesions](image)

*Figure 62 Coronal T2 image on left, axial T2 inversion recovery in center, and axial contrast-enhanced 3D T1 gradient on right showing splenomegaly with Gaucher lesions, which are hypointense on both T1- and T2-weighted images*

**Conclusion**

This concludes the Advanced Abdominal Imaging, Part I: Liver, Pancreas, Spleen 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 I Module


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

- Figure 1 – https://en.wikipedia.org/wiki/Lobules_of_liver
- Figure 2 – https://www.hopkinsmedicine.org/healthlibrary/conditions/liver_biliary_and_pancreatic_disorders/biliary_system_anatomy_and_functions_85,P00659
- Figure 3 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3462338/figure/F1/
- Figure 4 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3462338/figure/F2/
- Figure 5 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3462338/figure/F3/
- Figure 6 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3462338/figure/F4/
- Figure 7 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3462338/figure/F5/
- Figure 8 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3462338/figure/F6/
- Figure 9 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3462338/figure/F7/
- Figure 10 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3462338/figure/F8/
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- Figure 12 – http://theoncologist.alphamedpress.org/content/9/4/385/F11.expansion.html
- Figure 16 – Figure 1a http://appliedradiology.com/articles/magnetic-resonance-imaging-of-the-pancreas
- Figure 17 – Figure 1b http://appliedradiology.com/articles/magnetic-resonance-imaging-of-the-pancreas
- Figure 18 – Figures 1c, 1d, 1e http://appliedradiology.com/articles/magnetic-resonance-imaging-of-the-pancreas
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- Figure 21 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1766565/figure/F1/
- Figure 22 – Figure 5b http://appliedradiology.com/articles/magnetic-resonance-imaging-of-the-pancreas
- Figure 23 – Figures 6a, 6b http://appliedradiology.com/articles/magnetic-resonance-imaging-of-the-pancreas
- Figure 24 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1766565/figure/F3/
- Figure 25 – Figures 7a, 7b http://appliedradiology.com/articles/magnetic-resonance-imaging-of-the-pancreas
- Figure 26 – Figures 8a, 8b http://appliedradiology.com/articles/magnetic-resonance-imaging-of-the-pancreas
- Figure 27 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1766565/figure/F4/
- Figure 28 – https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1766565/figure/F2/
• Figure 29 – Picture 56 https://www.emedicinehealth.com/image-gallery/spleen_picture/images.htm
• Figure 30 – https://www.hindawi.com/journals/rrp/2013/219297/fig1/
• Figure 31 – https://www.hindawi.com/journals/rrp/2013/219297/fig2/
• Figure 32 – https://www.hindawi.com/journals/rrp/2013/219297/fig3/
• Figures 33, 34 – https://www.hindawi.com/journals/rrp/2013/219297/fig4/
• Figure 35 – https://www.hindawi.com/journals/rrp/2013/219297/fig6/
• Figure 36 – https://www.hindawi.com/journals/rrp/2013/219297/fig7/
• Figure 38 – https://www.hindawi.com/journals/rrp/2013/219297/fig8/
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• Figures 49, 50 – https://www.hindawi.com/journals/rrp/2013/219297/fig14/
• Figure 51 – https://www.hindawi.com/journals/rrp/2013/219297/fig12/
• Figures 52, 53 – https://www.hindawi.com/journals/rrp/2013/219297/fig15/
• Figures 55, 56 – https://www.hindawi.com/journals/rrp/2013/219297/fig16/
• Figure 57 – https://www.hindawi.com/journals/rrp/2013/219297/fig17/
• Figures 58, 59 – https://www.hindawi.com/journals/rrp/2013/219297/fig18/
• Figure 60 – https://www.hindawi.com/journals/rrp/2013/219297/fig19/