Pancreas: Cancer versus Inflammation

The pancreas is intrinsically endowed with high signal intensity in T1-weighted images, compared to liver and spleen.

Pancreatic ductal adenocarcinoma accounts for 95% of the malignant tumors of the pancreas. The tumor has a poor prognosis, with a 5-year survival of 5%. Detection of carcinoma is best performed by T1-weighted images, diffusion weighted images and post-gadolinium T1-weighted gradient echo images. Pancreatic adenocarcinoma appears as a definable, circumscribed mass lesion hypointense on T1-weighted images and tumor margins are well delineated from pancreatic tissue, because of the high contrast resolution of MR images. On post-contrast fat-suppressed T1-weighted images, pancreatic adenocarcinoma appears hypointense during the pancreatic phase of the dynamic study, because of its fibrous content. Indirect signs of pancreatic adenocarcinoma are by the stenosis of the main pancreatic duct, without dilatation following the administration of secretin. Downstream pancreatic parenchyma shows normal signal intensity, both on T1- and T2-weighted images, differently from upstream pancreatic parenchyma, which appears hypointense in all pulse sequences.

The extension of the lesion to peri-pancreatic fat and encasement of peri-pancreatic vessels is highly suggestive of pancreatic adenocarcinoma.

Chronic pancreatitis shows similar signal intensity changes of pancreas on non-contrast T1-weighted fat-suppressed and T2-weighted images generally mildly hypointense on T1-weighted images and heterogeneous and mildly hyperintense on T2-weighted images, however the mass effect is less evident compared to pancreatic adenocarcinoma, and if there is mild mass effect usually the pancreatic parenchyma shows preservation of a glandular, feathery, or marbled texture similar to that of remaining pancreas. In severe forms of chronic pancreatitis, the main pancreatic duct may show multiple stenoses, and a beaded appearance, indicative of the diffuse and chronic process.

Pancreatic pseudocyst may occur in patients with chronic pancreatitis with an incidence of 10%, most likely indicating reactivation of the inflammatory process. Pancreatic pseudocysts should be differentiated by retention cysts that can be observed in the upstream pancreas in patients with pancreatic adenocarcinoma.

Rarely, chronic pancreatitis may involve only the focally enlarged portion of pancreas, with the remainder of the pancreas having no inflammatory changes. In these cases, the focus of chronic pancreatitis can simulate the appearance of pancreatic ductal adenocarcinoma. The inflammatory process may be sufficiently destructive that underlying stromal pattern is lost. In these rare cases, diagnosis can be established only by surgical resection and histopathologic examination confirming the absence of malignancy.