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Tumor-forming pancreatitis

Inflammatory pancreatic masses are often hard to distinguish from ductal adenocarcinomas, especially if not accompanied by calcifications or a dilated pancreatic duct (4). Pancreatitis can produce a mass in all parts of the pancreas, mainly in the head or in the groove between the duodenum and the head of the pancreas (9). Such pancreatitis has been called "mass forming" pancreatitis in Japan and pseudotumorous pancreatitis in Western countries (7, 9).

The aim of the study was to evaluate the possibilities of imaging procedures in identification inflammatory pancreatic masses and review a clinical and radiological experience of mass-forming pancreatitis and pancreatic cancer to identify possible findings which can be used to differentiate between these two conditions.

MATERIAL AND METHODS

A total of 21 patients with a pathological pancreatic mass visualised during a computed tomography (CT) examination was included into the study. A mass was defined as a tumor-like lesion with focal enlargement of the pancreas. Enlargement of the entire pancreas or focal enlargement with calcifications was excluded from the series. The surgical resection was achieved in all cases with suspicion of pancreatic cancer and the histopathological examination of the resected specimens prove no malignancy in these patients.

RESULTS

In most patients the mass was localized within the head of the pancreas – 92%, 5% in the body and 3% in the tail. The mean diameter of the mass was 5.6 cm, with 46% masses below 5 cm. Almost all patients have solid masses, with only 23% of mixed solid-cystic lesions, due to pathological fluid formations or pseudocyst. This second feature was characteristic of pancreatitis. Only 15% of patients have diminished intensity of tumors on post contrast-enhanced images. In the rest of 85% the mass was isovascular. This is also the sign of importance in differential diagnosis with pancreatic cancer. The shape of the mass was irregular in 66% of patients and the borders of the mass were irregularly separated from the surrounding pancreas in 87%. The dilated pancreatic duct was observed in 54% of patients with chronic inflammation. Smooth limits of the duct were present in 24%, irregular limits in 76%. The dilated duct was visible inside the pancreatic mass in 26%. The common bile duct was dilated in 54%. In 65% of patients CT or ultrasound (US) examination prove pathological changes in gallbladder, with gallstone, hydrops or resection. 19% of changes infiltrated the surrounding fatty tissue, the sign usually characteristic of pancreatic cancer.

DISCUSSION

Patients with chronic pancreatitis often have areas of focal enlargement in the gland. In the absence of calcifications or duct strictures and dilations the distinction between inflammatory and malignant enlargement is problematic (4, 6). Focal pancreatic enlargement is generally indicative of a neoplasm, even if lymphadenopathy or hepatic metastases are not evident, but chronic pancreatitis may also cause a focal pancreatic mass (5). Calcifications and a dilated pancreatic duct strongly suggest an inflammatory pancreatic mass (5).

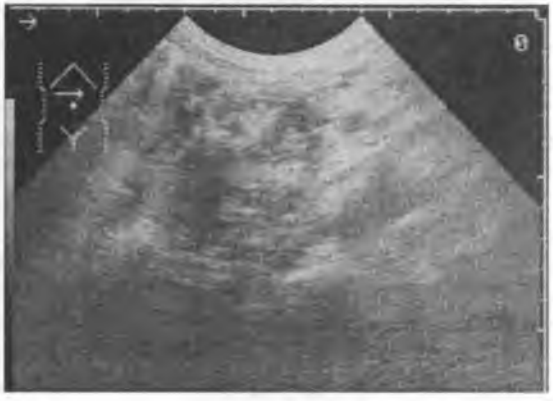

Fig. 1. US. Chronic pancreatitis. Heterogeneous inflammatory tumor with hyperechoic regions

US is a useful tool for screening patients who are suspected of having a pancreatic carcinoma (1). Characteristic findings of pancreatitis by US are pancreatic stones, hyperechoic region with heterogeneous internal structures (Fig. 1) and cystic lesions communicating with pancreatic ducts. The last sign was present in 13% of patients from this study. On the contrary, a low echoic mass with obstruction of the pancreatic duct is highly suggestive of pancreatic carcinoma (1). On contrast-enhanced sonography, 95% of inflammatory pancreatic masses showed isovascularity and 91% of ductal carcinomas revealed hypovascularity compared with the pancreatic parenchyma (4). CT is the most important tool for the demonstration of the mass lesion of the pancreas. On unenhanced CT both inflammatory masses and small carcinomas are usually iso-intense with normal pancreatic tissue (Fig. 2). Both lesions, when localized in the head of the pancreas, can cause upstream dilation of the duct and atrophy of the tail. Carcinoma is typically hypovascular and shows much less contrast enhacement than normal pancreatic tissue (6). The reasons for hypovascularity of ductal carcinomas are desmoplastic changes and vascular encasement, causing arterial stenosis or obstruction (4). An inflammatory pancreatic mass, which is the focal swelling of the pancreas, consist of inflammatory changes such as interlobular fibrosis and chronic inflammatory infiltrate around lobules and ducts (4). Those inflammatory changes result in hyper or isovascularity, but if severe fibrosis replace pancreatic cells the mass may be hypovascular with reduced contrast enhacement, which was present in 15% of patients from this study.

A dilation of main pancreatic duct, larger in diameter, with smooth, linear contours is found in cancer, whereas in chronic inflammation a smaller dilation with irregular contours is observed (3). The fibrous tissue replaces pancreatic lobules causing narrowing of ducts and a dilation and congestion above, resulting in atrophy of the normal pancreatic tissue and forming concrements. Narrowing of the main pancreatic duct and its branches can be caused by an inflammatory, local or

diffuse mass. In cancer the walls of main pancreatic duct are usually parallel, resembling an image of a smooth, hypodense column. Sudden and important dilation of main pancreatic or/and common bile duct is characteristic of malignancy (8).

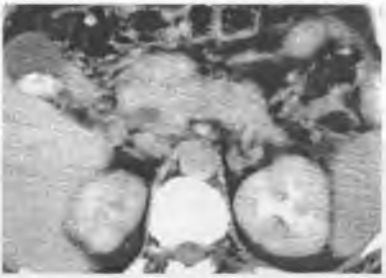

Fig. 2. CT. Chronic pancreatitis. Isodense mass in the head of the pancreas, with gallstone and dilation of common bile duct

Recently the concept of autoimmune pancreatitis has been proposed. This newly identified clinical entity is characterized by irregular narrowing of the main pancreatic duct with diffuse enlargement of the pancreas (2, 7), but these changes in contrary to pancreatic cancer or chronic pancreatitis are reversible. Patients with focal autoimmune pancreatitis have frequently been treated surgically for suspected carcinoma, because this variant form also shows a focal mass in the pancreas, usually with a localized stenosis or obstruction of the main pancreatic duct. Most focal autoimmune pancreatitis cases have been diagnosed only after surgery or after swelling has become diffuse (7). In patients with focal autoimmune pancreatitis, dynamic CT frequently demonstrates low attenuated areas in the focal lesions in early phase images. In delayed phase images the lesions are well enhanced up to the surrounding normal parenchyma. These findings are thought to be characteristic of autoimmune pancreatitis. Another test suggesting this entity is the presence of a serum autoimmune phenomenon with hypergammaglobulinemia or positive autoantibodies, a peculiar pancreatic histopathology showing severe fibrosis with marked lymphocytic or plasmocytic infiltration, and a dramatic response to corticosteroid therapy (7). Because autoimmune pancreatitis is a reversible disease, recognition of these new findings strengthen the need for early differentiation of sources of pancreatic enlargement to avoid unnecessary resection. Even in advanced inflammatory disease, the attenuation of abnormal pancreatic tissue remains normal on CT, and the early changes of the disease are difficult to recognise (6). At present time no single parameter or imaging procedure is sufficient to differentiate between tumor-forming chronic or autoimmune panreatitis and pancreatic cancer. The challenges for imaging are to detect early stages of chronic pancreatitis and to differentiate between carcinoma and focal inflammatory masses.

CONCLUSIONS

Inflammatory pancreatic masses usually show the same vascularity as the normal parenchyma of pancreas, sometimes with coexisting cystic lesions. Chronic pancreatitis can cause a dilation of the main pancreatic duct, smaller than pancreatic cancer, with irregular contours of the duct. In case of pancreatic mass ductal adenocarcinoma cannot be completely excluded. Autoimmune pancreatitis should also be considered.

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SUMMARY

The aim of the study was evaluation of the imaging findings in patients with inflammatory pancreatic mass. A total of 21 patients with a pathological pancreatic mass visualised during a CT examination were included into the study. A mass was defined as a tumor-like lesion with focal enlargement of the pancreas. Enlargement of the entire pancreas or focal enlargement with calcifications was excluded from the series. The surgical resection was achieved in all cases with suspicion of pancreatic cancer and the histopathological examination of the resected specimens prove no malignancy in this patients. Inflammatory pancreatic masses usually show the same vascularity as the normal parenchyma of pancreas, sometimes with coexisting cystic lesions. Chronic pancreatitis can cause a dilation of the main pancreatic duct, smaller than pancreatic cancer, with irregular contours of the duct. Nonetheless in case of pancreatic mass ductal adenocarcinoma cannot be completely excluded. Autoimmune pancreatitis should also be considered.

Guzowate zapalenie trzustki

Celem pracy była ocena zmian w badaniach obrazowych u chorych z zapalnym guzem trzustki. Do badań włączono grupę 21 chorych z patologiczną masą trzustkową stwierdzoną w badaniu tomografii komputerowej. Masę zdefiniowano jako zmianę guzopodobną z ogniskowym powiększeniem trzustki. Przypadki z powiększeniem całej trzustki lub z obecnością zwapnień w trzustce wyłączono z badań. U wszystkich chorych wykonano chirurgiczną resekcję zmiany z oceną histopatologiczną, nie stwierdzając zmian złośliwych. Guzy zapalne trzustki zwykle są izodensyjne w stosunku do pozostałego miąższu trzustki, czasami ze współistniejącymi zmianami torbielowatymi. Przewlekłe zapalenie trzustki może powodować poszerzenie głównego przewodu trzustkowego, z nieregularnym obrysem ścian przewodu. Mimo to w przypadku obecności masy trzustkowej nie można wykluczyć przewodowego raka trzustki. Należy rozważyć także możliwość autoimmunologicznego zapalenia trzustki.