The importance of pancreatic inflammation in endosonographic diagnostics of solid pancreatic masses

Francesco Vitali¹, Deike Strobel¹, Luca Frulloni², Marc Heinrich³, Lukas Pfeifer¹, Ruediger S. Goertz¹, Gheorghe Hundorfean¹, David L. Wachter⁴, Robert Gruetzmann⁵, Thomas Bernatik⁶, Markus F. Neurath¹, Dane Wildner¹

¹Department of Internal Medicine 1, Friedrich-Alexander-University, Erlangen-Nuremberg, Germany, ²Department of Medicine, Pancreas Center, University of Verona, Verona, Italy, ³Department of Radiology, Friedrich-Alexander-University, Erlangen-Nuremberg, Germany, ⁴Institute of Pathology, Friedrich-Alexander-University, Erlangen-Nuremberg, Germany, ⁵Department of General and Abdominal Surgery, Friedrich-Alexander-University, Erlangen-Nuremberg, Germany, ⁶Department of Internal Medicine, Kreisklinik Ebersberg, Germany

Abstract

Aims: Endosonography (EUS) is one of the main diagnostic tools for the differential diagnosis of pancreatic masses. The aim of our study was to describe the value of this technique in the work-up of solid pancreatic lesions, considering the influence of the morphological evidence of pancreatic inflammation in the diagnostic process. Material and methods: Retrospective analysis of prospectively collected data in our tertiary University center. From March 2007 to October 2015, 218 patients underwent EUS for a suspected solid pancreatic neoplasm (based on previous cross-sectional imaging results, idiopathic acute pancreatitis, weight loss, pancreatic hyperenzymemia, painless jaundice or elevated Ca 19-9 values). Results: Malignant lesions were diagnosed in 98 (45%) patients. Sensitivity of EUS for malignancy was 91% and specificity 89.2%. Signs of pancreatic inflammation in the surrounding pancreatic parenchyma around the focal lesion were present in 97 patients (44.4%) (more often in men, smokers and drinkers, and the most common etiology was focal chronic pancreatitis) and in these patients the sensitivity and specificity dropped to 44% and 87.1%, respectively. In patients without signs of pancreatic inflammation, the pancreatic focal lesions were adenocarcinoma, neuroendocrine tumor, ventral/dorsal split, non-pancreatic pathology, pancreatic lipomatosis and autoimmune pancreatitis. Conclusion: Pancreatic inflammation (either focal or involving the whole gland) lowers the diagnostic sensibility of EUS in the work-up of pancreatic masses suspected for cancer, requiring further invasive diagnostic methods. Focal autoimmune pancreatitis and paraduodenal pancreatitis are still confused with pancreatic cancer, even in the absence of pancreatic inflammation.

Keywords: endoscopic ultrasound; pancreatic cancer; benign pancreatic lesion; pancreatitis

Introduction

Endosonography (EUS) is an established technique for the clinical work-up of a newly recognized pancreatic mass. Reported sensitivities range from 93 to 98% for diagnosing pancreatic cancer, especially for lesions smaller than 3.0 cm [1-3]. Cross sectional imaging like computed tomography (CT) and magnetic resonance imaging (MRI) sometimes show abnormal findings that raise the suspicion of pancreatic cancer, resulting in a further work-up [4]. Despite advancements in cross sectional imaging and the use of EUS, published retrospective surgical studies demonstrate that the incidence of pancreatic resections performed for benign lesions mimicking pancreatic malignancy did not change from 1983 to 2014 and is still about 9-10% for pancreatic head lesions [5-19].

New diagnostic modalities such as contrast enhanced-EUS, quantitative contrast enhanced analysis,
fine needle aspiration (FNA) and core biopsy have been added to improve diagnostic performance [20,21]. Recently, real-time elastography and three-dimensional reconstruction have become available, enabling differentiation of typical ductal pancreatic adenocarcinoma from other neoplasms and benign solid lesions with an accuracy of approximately 90% [22-26]. However, despite all these advanced diagnostic tools, the diagnosis is still challenging and radical surgery is still performed [6].

Retrospective data supports evidence that a suspicious pancreatic mass detected upon contrast-enhanced CT should directly undergo surgery without further investigation [27]. Consensus among surgeons recommends primary surgery for pancreatic masses, except in cases highly suspicious of autoimmune pancreatitis (AIP) [28]. On the other hand, without preoperative diagnosis, an unacceptable amount of patients might unnecessarily be exposed to radical pancreatic surgery with considerable morbidity and mortality for pre-malignant or benign lesions [3]. This situation is reported to be in Germany up to 11.1% in very high volume hospitals to 20.2% in very low volume hospitals [29].

AIP, which is a typical mimicry of pancreatic cancer, is increasingly being diagnosed and treated worldwide [30]. In 2011, the International Consensus Diagnostic Criteria (ICDC) for diagnosing AIP was published. Paraduodenal pancreatitis may also present as a mass-forming process and it is increasingly being diagnosed since the publication of histopathological diagnostic criteria [31] and defined clinical and imaging features [32-35].

The aim of this study was to determine the diagnostic sensitivity and specificity of EUS for pancreatic lesions suspicious for malignancy. In addition, we investigated the influence of acute and chronic inflammatory changes of the pancreas on the diagnostic presentation and on the final diagnoses.

**Material and methods**

We conducted a retrospective analysis of prospectively collected data in our tertiary care university hospital. The present study enrolled patients with focal solid pancreatic changes referred from gastroenterological and radiological practices, general practitioners and primary and secondary care centers of our region. Complex cases were mainly referred to our surgical department and discussed in our interdisciplinary board. Some patients had primarily been examined in other departments and were admitted to our center for a second opinion or for a further workup. From March 2007 to October 2015, 2356 EUS examinations were performed. Indications for examination were screened from examining patients’ clinical files in order to select those who underwent EUS for a suspected pancreatic solid lesion (fig 1). EUS was performed by 3 experienced endosonographers (each more than 1000 procedures) of our institution, using Pentax EG-3630UR Radial and EG-3830UT echoendoscopes (PENTAX Europe GmbH, Hamburg) with a Hitachi EUB 6500 ultrasound system (Hitachi Medical Systems GmbH, Wiesbaden). The equipment did not change during the study period. EUS was performed with radial-scanning echoendoscopes. Curvilinear echoendoscopes were used for FNA. Standard EUS for the evaluation of the pancreas was performed. If the EUS findings raised a suspicion that a focal lesion was present, histological specimens were obtained, at the discretion of the endosonographer, with EUS-FNA using a curvilinear array echoendoscope. The needle used was a 19 G needle to achieve histological evaluation. All procedures were performed with the patient under conscious sedation.

Indications for examination were: weight loss of unclear etiology, elevated carbohydrate-associated antigen (CA) 19-9, painless jaundice, acute pancreatitis of unknown etiology, pancreatic hyperenzymemia (increase of either lipase or amylase value or both), “double duct sign” upon imaging (present in transabdominal sonography, MRI or CT), a solid mass suspected upon cross-sectional imaging or transabdominal sonography.

A solid pancreatic lesion was defined as a lesion of the pancreatic parenchyma with predominantly solid tissue seen at standard/grey scale EUS (ie, the solid component constituted at least 90% of the total volume of the lesion). The presence of cystic lesions in other parts of the pancreas was defined as a lesion with predominantly cystic morphology. The study included patients with a focal solid pancreatic lesion suspected by EUS (fig 1). Fig 1. Flow chart of the study group, rate of surgery and inflammatory morphological changes in EUS.
the pancreas was not an exclusion criterion. When an intraductal papillary mucinous neoplasia (IPMN) was not suspected preoperatively and diagnosed only through the histopathological examination, this was reported in the final data.

For the analysis, the patient collective was divided into two groups according to the presence or absence of sign for pancreatitis at EUS in the surrounding pancreatic parenchyma all around the focal lesion: Group 1: healthy pancreas (HP) around the focal lesion and Group 2: inflamed pancreas (IP) around the focal lesion. Patients presenting with normal pancreatic parenchyma upon EUS were assigned to the HP group. Patients presenting with signs of acute [36] or chronic pancreatitis at EUS according to the Rosemont criteria for chronic pancreatitis (i.e. enlarged hypoechoic or inhomogeneous and coarse/lobulated parenchyma, with protein plugs, fibrotic and hyperechoic spots) were assigned to the IP group [22,37-39].

Exclusion criteria were: acute biliary pancreatitis and pre-EUS diagnosis of cholecystolithiasis, predominantly cystic mass, primary cystic lesions without solid component, preoperative diagnosis of IPMN.

For patients presenting with mass-forming chronic pancreatitis, cross-sectional imaging pictures were reviewed by an expert radiologist (M.H.) specialized in abdominal imaging. Criteria of paraduodenal pancreatitis were systematically applied in order to achieve a diagnosis “a posteriori” in accordance to literature [32,33] (thickening of the duodenal wall, fibrous tissue within the pancreaticoduodenal groove showing as hypoenhancement of the duodenal wall after contrast medium application, signs of duodenal stenosis with/without gastric outlet obstruction, duodenal wall cysts, leftward displacement of a normal appearing gastroduodenal artery). The radiologist was blinded to clinical data. Patients’ imaging data were obtained from the computerized archive of the Department of Radiology of the University of Erlangen-Nuremberg.

The diagnostic gold standard was histology from a surgically resected specimen or FNA. Where histology could not be obtained or was not diagnostic, clinical follow-up for at least 6 months was requested in patients without a definitive diagnosis.

**Statistical analysis**

Parametric variables are reported as mean and standard deviation. Student’s t-test was used to compare continuous variables. The χ2 analysis was used for categorical variables. The Fisher exact probability test was used for the 2 x 2 contingency tables, where suitable. A two-tailed distribution was used with p-values <0.05 considered to be statistically significant. Statistics were calculated using the IBM SPSS 20 statistical program (IBM Corporation, Armonk, New York, USA). Regarding diagnostic sensitivity and specificity of EUS predicting malignancy, any neoplastic lesion (e.g., pancreatic neuroendocrine tumor, distal cholangiocarcinoma, ampullary tumor) was considered as a malignancy. The diagnostic gold standard was a surgically resected specimen or diagnostic histology obtained via biopsy.

**Results**

### Patients’ characteristics

Among all patients that underwent EUS in our institution for the suspicion of pancreatic malignancy, 218 patients were included (124 men, 94 women; mean age 60±14 years, range 20-88). The final diagnosis of a benign lesion was made in 120 patients (55.0%) and a malignant lesion was diagnosed in 98 (45%) patients (fig 2). 54 patients (24.8% of all lesions) underwent surgery and 4 patients (1.8%) refused surgery. 14 patients (25.9%) who underwent surgery because of a suspicion of malignancy (no preoperative histology) could not be confirmed in the operative specimen. EUS follow-up was performed in 61 patients (28%), follow-up 16.5±27.3 months. In 18 patients under follow-up, surgery was required. Follow-up was refused by 4 patients. One patient refused both follow-up and surgery. Sensitivity of EUS for malignancy for the whole cohort was 91% and specificity was 89.2%.

**EUS in patients (n=121) with a normal pancreatic parenchyma surrounding the suspicious lesion (HP-group)**

Of all the 218 patients, 121 presented with a not-inflamed, normal pancreatic parenchyma in the surround-

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![Fig 2. Flow chart of the study group divided into malignant and benign lesions](image-url)
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ing tissue of the unclear focal mass (fig 3, fig 4). Clinical characteristics of the patients of the HP-group are shown in table I according to the final diagnosis of malignant and benign lesion. Thirty-three of these patients (28.9%) underwent surgery due to the suspicion of malignancy; 3 patients (2.5%) refused surgery, whereas 21 patients (17.4%) underwent EUS follow-up. The mean follow-up was 11±27.5 (1-125) months after the initial EUS examination.

EUS in the HP group showed sensitivity for malignancy of 91.4% and a specificity of 97.7%. EUS-FNA had sensitivity for malignancy of 70% and a specificity of 80%.

**EUS in patients (n=97) with signs of an inflamed pancreas around the suspicious pancreatic lesion (IP-group)**

Ninety-seven patients showed signs of inflammation in the pancreatic parenchyma upon EUS [69 (71.1%) men; 57.4±14.2 years]. Of these, 14 patients (14.4%) had pancreatic cancer, 36 (37.1%) chronic pancreatitis, 3 (3.1%) AIP (fig 5), 20 (20.6%) acute pancreatitis, 7 (7.2%) necrosis, 7 paraduodenal pancreatitis (7.2%), 3 neuroendocrine tumors (NET) (3.1%) and 4 cholangiocellular carcinoma (CCC) (4.1%). In two patients, no focal lesion was detectable on EUS (one patient showed an atrophic pancreatic parenchyma, the other one had unspecific chronic signs of pancreatitis). The clinical characteristics of patients in the IP-group comparing the subgroups of malignant and benign lesions can be seen in Table II.

In the IP-group, 40 patients (41.2%) underwent EUS follow-up [mean follow-up 19±27.2 (1-123) months after the initial EUS examination].

Table I. Clinical characteristics of patients with normal pancreatic parenchyma surrounding the suspicious lesion (HP group)

| HP group     | Benign n=44 | Malignant n=77 | p     |
|--------------|-------------|----------------|-------|
| Sex M        | 16          | 39             | ns    |
| Mean Age (years) | 60.5±14.4 | 63±13.5        | ns    |
| BMI (kg/m²)  | 26.6±5.2    | 25.5±4.1       | ns    |
| Nicotine abuse | 9         | 17             |       |
| Alcohol abuse | 13         | 23             | ns    |
| Exocrine failure | 3         | 9              |       |
| CA 19-9 elevated | 2       | 34             | 0.001 |
| IgG4 elevated | 5          | 0              | 0.016 |
| Mass on EUS  | 18          | 70             | <0.001|
| Mass on CT   | 18          | 57             | 0.001 |
| Synchronous Neoplasm | 3     | 6              | ns    |
| Extrapancreatic involvement | 2 | 22             | 0.001 |
| Enlarged LN  | 6           | 30             | 0.003 |
| No lesion on EUS | 25    | 6              | <0.001|
| Double duct sign | 2      | 20             | 0.003 |
| MPD dilation | 4           | 37             | <0.001|
| MPD Stenosis | 5           | 18             | ns    |
| Bile duct dilation | 6    | 28             | 0.004 |
| Lipomatosis  | 15          | 9              | 0.003 |
| Jaundice     | 4           | 20             | 0.025 |
| Hyperlipasemia | 4      | 13             | ns    |
| Weight loss  | 8           | 30             | 0.016 |
| Diabetes     | 5           | 22             | 0.04  |
| Surgery      | 3           | 32             | <0.001|

The results are expressed as number or mean±standard deviation. BMI – body mass index; MPD – main pancreatic duct; ns – not significant; FNA fine needle aspiration; TUS – transabdominal ultrasoundography; LN – lymph nodes; CA 19-9: carbohydrate-associated antigen 19-9. Exocrine Failure was defined as pancreatic enzyme supplementation therapy and/or decreased values of pancreatic elastase on stool spot (ELISA assay)
EUS in the IP-group showed sensitivity for malignancy of 44% and a specificity of 87.1%. EUS-FNA presented sensitivity for malignancy of 40% and a specificity of 20%.

In Table III the different etiologies of the pancreatic lesions are presented after proof of the final diagnosis and follow-up, again divided according to HP and IP.

**Comparison between IP-group and HP-group**

Patients in the IP-group showed different clinical features compared to patients in the HP-group (Table IV). Patients in the IP group were more often male with a medical history of nicotine and alcohol abuse, presented more often pancreatic calcification and a pancreatic mass that could not be reproduced in EUS or disappeared during follow-up, indicating the inflammatory genesis of the

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**Table II. Clinical characteristics of patients with signs of pancreatic inflammation (IP group) in the pancreatic parenchyma surrounding the suspicious lesion**

| IP group | Benign n = 76 | Malignant n = 21 | p     |
|----------|---------------|-----------------|-------|
| Sex M    | 56            | 13              | ns    |
| Age (years) | 56.2±14.1    | 61.6±14.3       | ns    |
| BMI (kg/m²) | 25.0±4.5       | 25.6±4.9        | ns    |
| Nicotine abuse | 43          | 9               | ns    |
| Alcohol abuse | 46           | 11              | ns    |
| Exocrine failure | 15         | 3               | ns    |
| CA 19-9 elevation | 9       | 10              | 0.008 |
| IgG4 elevation | 0           | 1               | ns    |
| Mass     | 18            | 70              | <0.001|
| Extrapancreatic involvement | 6         | 5               | 0.056 |
| Synchrone Neoplasm | 5           | 3               | ns    |
| LN       | 35            | 11              | ns    |
| No lesions in EUS  | 41          | 3               | 0.001 |
| Double duct sign | 7            | 8               | 0.001 |
| MPD dilation | 31           | 15              | 0.013 |
| MPD Stenosis | 16           | 8               | ns    |
| Bile duct dilation | 11          | 9               | 0.03  |
| Lipomatosis | 5            | 3               | ns    |
| Jaundice | 9             | 9               | 0.001 |
| Hyperlipasemia | 31          | 6               | ns    |
| Weight loss | 22           | 10              | ns    |
| Acute pancreatitis | 31         | 2               | 0.007 |
| Diabetes | 26            | 5               | ns    |
| Surgery  | 11            | 8               | 0.05  |
| Calcifications | 39          | 10              | ns    |

The results are expressed as number or mean±standard deviation. M – male; BMI – body mass index; MPD – main pancreatic duct; ns – not significant; FNA fine needle aspiration; TUS – transabdominal ultrasonography; LN – lymph nodes; CA 19-9: carbohydrate-associated antigen 19-9. Exocrine Failure was defined as pancreatic enzyme supplementation therapy and/or decreased values of pancreatic elastase on stool spot (ELISA assay).

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**Table III. Etiological distribution of focal pancreatic lesions in consideration of presence and absence of echo-morphological signs of pancreatic inflammation**

| Aetiology                  | Healthy pancreas (HP) n = 121 (55.5%) | Inflamed pancreas (IP) n = 97 (44.5%) | p     |
|----------------------------|----------------------------------------|----------------------------------------|-------|
| Pancreatic cancer          | 46 (21.1)                              | 14 (6%)                                | 0.038 |
| NET                        | 23 (10.5)                              | 3 (0.1%)                               | <0.001|
| Ampullary carcinoma        | 4 (0.1)                                | 0 (0%)                                 | ns    |
| CCC                        | 3 (0.1)                                | 4 (0.1%)                               | ns    |
| IPMN                       | 1 (0.5)                                | 1 (0.5%)                               | ns    |
| Acute pancreatitis         | 0                                      | 20 (9%)                                | <0.001|
| Chronic pancreatitis       | 0                                      | 36 (16.5%)                             | <0.001|
| Pancreatic necrosis        | 0                                      | 7 (3.2%)                               | <0.001|
| Autoimmune pancreatitis    | 8 (3.7)                                | 3 (1.3%)                               | ns    |
| Paraduodenal pancreatitis  | 1 (0.5)                                | 7 (3.2%)                               | 0.01  |
| Choledocholithiasis        | 1 (0.5)                                | 0                                       | ns    |
| Ventral/dorsal split       | 6 (2.7)                                | 0                                       | <0.001|
| Pancreatic lipomatosis      | 3 (1)                                  | 0                                       | ns    |
| Enlarged lymphnodes        | 2 (1)                                  | 0                                       | ns    |
| Microcystic serous mucinous neoplasm | 1 (0.5) | 0 | ns |
| Other*                     | 22 (10)                                | 2 (1)                                  | <0.001|

Results are expressed as number (%). NET – neuroendocrine tumors; CCC – cholangiocellular carcinoma; IPMN – intraductal papillary mucinous neoplasia. *Other rare conditions such as benign choledochus strictures, papillitis stenosans and patients with a pancreatic occupying lesion who refused follow-up.
lesion. In addition, they presented more often acute pancreatitis and hyperlipasemia.

Patients with AIP and paraduodenal pancreatitis were found in both groups. Clinical and imaging features of these patients are shown in supplementary Table I and II, on the journal site. In our case IgG4 elevation presented a sensitivity of 83.3% and a specificity of 77.8% for AIP.

**Discussions**

Our findings describe the influencing factors in the management of solid pancreatic masses undergoing EUS. The accuracy of EUS, with or without FNA, for diagnosing pancreatic cancer in specific cohorts has been described earlier [40,41]. Our data represent a “real-life” scenario from a tertiary referral center in contrast. Additionally, we focused on the characteristics of the surrounding pancreatic parenchyma beside the solid mass with a discrimination of an inflamed or normal pancreatic tissue. In our study, the sensitivity of EUS regarding the diagnosis of malignancy in IP-group was 44% and significantly lower than the 91.4% observed in the HP-group. Sensitivities between 54% and 74% have been reported in previous studies evaluating solid pancreatic masses with EUS-FNA in the setting of chronic pancreatitis [42-44]. Moreover, pancreatic calcifications can create acoustic shadows that may prevent a complete evaluation of the whole gland. Also, dilation of the main pancreatic duct (MPD) may occur years before the detection of a small neoplasm in patients with normal pancreatic parenchyma [45], whereas in the setting of chronic pancreatitis, MPD fibrotic dilation may be present without a cause of obstruction [46].

The differentiation between pancreatic cancer and a solid lesion due to mass forming pancreatitis is still challenging. According to our results, also in the setting of normal pancreatic parenchyma, AIP and paraduodenal pancreatitis can be the causes of focal lesions. Ventral/dorsal split leading to suspicion of cancer, which may appear as a small hypoechoic area in the posterior part of the pancreatic head, represents a differential diagnosis, which can be excluded by experienced endosonographers [47]. In unclear cases, repeated examinations in intervals of at most 4-12 weeks are recommended [48]. Pancreatic lipomatosis, especially if distributed unevenly through the gland, can be confused with pancreatic cancer in cross-sectional imaging [49]. In our series, EUS represents an excellent tool for diagnosis, since no patient with focal lipomatosis underwent pancreatic surgery for suspected malignancy.

The number of FNA procedures in the pancreas is much lower in our center compared to other studies cited in the published literature [50]. The explanation may be that there is a tendency to perform resection in the presence of a pancreatic mass rather than preoperative diagnosis, since the probability of malignancy is high (more
than 90%) [18]. Furthermore, a negative FNA would not necessarily avoid resection due to the possibility of false negative findings. Preoperative tissue acquisition may be considered when an inflammatory mass is suspected on the basis of clinical history (familiarity for pancreatitis, young age, alcohol and smoking habits, previous diagnosis of autoimmune diseases) and of radiological findings suggestive for inflammation and not for cancer. On the other hand, recently a surgical multicentric series from the Netherlands reported results for EUS–FNA similar to ours; i.e. they reported a sensitivity of 63% and specificity of 61.5% for detecting pancreatic malignancy [51] in a “real-life” cohort.

FNA in our series did not assist us in diagnosing benign lesions such as AIP and paraduodenal pancreatitis, since alternative means for tissue diagnosis had to be employed. In unclear cases, clinical and imaging follow-up with more than one modality (usually EUS with CT) or transabdominal core biopsies were necessary for achieving a definitive diagnosis. After exclusion of malignancy with invasive means, as suggested for ICDC in cases suspicious of AIP, a steroid trial of 2 weeks with repeated imaging (MRI) in the short interval may be very helpful for a final diagnosis, despite ambiguous histology findings. In cases of paraduodenal pancreatitis, the history of intense alcohol consumption and smoking may raise suspicions of the disease and define an appropriate treatment, which may also lead to a pancreatoduodenectomy [34]. However, in the case of cancer the operation should be performed within 1 month, whereas in the case of paraduodenal pancreatitis surgery can be delayed.

According to our results, there were clinical differences between the groups. Among the IP-group, patients were more often males, smokers and drinkers. They also presented more often enlarged lymph nodes and diabetes. Among the IP group the pancreatic mass occasionally resolved during follow-up which indicates that pancreatic inflammations (showed also from the increase of lipase in the serum) play a confusing role in the diagnostic process with EUS.

The presence of a pancreatic mass on EUS is not in all cases an indicator of cancer. The presence of enlarged lymph nodes in the absence of pancreatitis is suggestive for locally disseminated cancer. In contrast to the IP-group, a dilation of the pancreatic duct, bile duct or both (double duct sign) and the clinical presence of jaundice, weight loss and worsening diabetes are alarm signs and should lead to aggressive tissue sampling and/or operative evaluation. As already reported by Alston et al we also confirm that particularly in patients of the HP-group, the presence of a mass and weight loss were more often associated with malignancy [52].

This study has some limitations. First, the patients included were a heterogeneous group due to the retrospective design of the study and no effort could be made to quantify the degree of “suspicion” for the presence of pancreatic cancer. However, the patient collective represents a “real-life” cohort of our clinical practice, without selections for high prevalence of malignant neoplasms. As a typical example, the study by Mallery et al had a 92% prevalence of malignancy [53]. Secondly, the patient collective was gathered from our EUS prospective database, so there may be a selection bias for lesions with untypical imaging appearance that requires further investigation before surgery. In our institution, patients are usually discussed in an interdisciplinary conference and are directly referred to surgery if the suspicion of malignancy is strong. Thirdly, not all patients underwent surgical resection, so a radical surgical/pathological diagnosis could not be achieved for all patients.

Conclusions

Sensitivity and specificity of EUS for malignancy is lower in the presence of pancreatitis. In the setting of an inflamed pancreas, an obstructive etiology causing pancreatitis should be expected and clinically further investigated with patient history, imaging and laboratory data (i.e. cancer or chronic inflammation due to smoking, alcohol abuse or autoimmunity). If the pancreas is sonomorphologically normal, a careful evaluation to recognize small pancreatic and extrapancreatic pathology not involving the pancreatic duct (such as anatomic variant or small pancreatic neoplasms) should be performed. Also, in the setting of a normal pancreatic gland, focal mass forming pancreatitis as AIP and paraduodenal pancreatitis should be considered. Unclear cases should thus undergo early follow-up examinations by EUS with additional complementing imaging modalities.

Conflict of interest: none

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