Med. J. Cairo Univ., Vol. 87, No. 1, March: 561-570, 2019
www.medicaljournalofcairouniversity.net

Role of Magnetic Resonance Imaging in the Diagnosis of Different Pancreatic Lesions

EMAN M. DARWISH, M.Sc.*; FATMA A. EL-SHAARAWY, M.D.*; ASHRAF F. BARAKAT, M.D.** and ABD EL-MONEM N. DARWISH, M.D.*

The Departments of Radiology and Clinical Oncology**, Faculty of Medicine, Tanta University*

Abstract

Background: Pancreatic cancer is one of the most lethal human cancers that requires early diagnosis. Ultrasound, CT and MRI are different imaging modalities used for diagnosis of pancreatic masses. Diffusion-Weighted Imaging (DWI) and Time-signal Intensity Curve (TIC) can add to the diagnosis of pancreatic masses.

Aim of Study: The aim of this study was to evaluate the role of magnetic resonance imaging in the diagnosis of different pancreatic lesions.

Patients and Methods: Twenty-three patients were divided into 4 groups, group A (Acute Pancreatitis) (AP), group B (pancreatic cysts) which subdivided into malignant and benign cysts, group C (adenocarcinoma) and group D (Focal Pancreatitis) (FP), all patients groups were compared to the control group. MRI protocol included MRI-c (T1WI, T1 Fat Suppression (FS), T2WI, T2FS and IV dynamic contrast study) and DWI which performed on (1.5 Tesla) Magnet Unit General Electric (GE). TIC was obtained from the dynamic study and the results were divided into three patterns of curves.

Results: Control group demonstrated type-I TIC, group (A & D) demonstrated type-II and group C demonstrated type-III.

The mean Apparent Diffusion Coefficient (ADC) values were significant lower (p<0.001) in (A & C) groups, statically different (p<0.02) between benign and malignant cysts. Statically different (p<0.001) between control and patients' groups. The sensitivity, specificity, accuracy, positive and negative predictive values of DWI and MRI-c were 92.3%, 90%, 91.3%, 92.3%, 90% and 100%, 90%, 95.7%, 92.9%, 100%, respectively.

Conclusion: DWI and TIC were useful tools in diagnosis, characterization and differentiation between pancreatic lesions.

Key Words: *Pancreatic lesions – DWI imaging – TIC.*

Introduction

ADENOCARCINOMA is the most common malignant swelling of the pancreas which is very violent and lethal, with a five-year survival rate <5% [1]. Resection rates still low at 10-15% due to invasion and distant metastases [2]. Still remains to a trial to distinctive malignant from benign pancreatic soft tissue lesions [3]. "Diffusion-Weighted Imaging (DWI) is based upon the moralities of Brownian motion of small molecules in a tissue" [4].

The benign lesions have stretched extracellular space with freely movement of water molecules that resulting in easier diffusion exhibited as a decreased intensity of signal and increased ADC values, compared to the malignant masses that have large and multi cells which leading to restrict the water movement in between result in decreased ADC value and increased signal intensities [5,6].

The same clinical and image of both cancer pancreas and Chronic pancreatitis were frequently shown whereas a focal pancreatic lesion was seen commonly in the patients with chronic pancreatitis, however the rate of carcinoma is higher in these patients than others, so the detection of pancreatic mass is indicative as a tumor [7-9].

The facility to notice Pancreatic Carcinoma (PC) and pancreatic focal mass that rise from chronic pancreatitis on Magnetic Resonance Imaging (MRI) including T1-weighted gadolinium-enhanced gradient-echo sequences were previously reported [10-13].

There are many categories of pancreatic cysts, including neoplastic and non-neoplastic types with a wide variation of pathologic entities whereas pseudocysts denote about 85-90% of all pancreatic cystic lesions [14-17]. MRI is higher to detect and diagnosis of mild and acute pancreatitis than Computed Tomography (CT), MRI can estimate and evaluate the peri pancreatic inflammation and

Correspondence to: Dr. Eman M. Darwish, The Department of Radiology, Faculty of Medicine, Tanta University

hemorrhage through the use of fluid sensitive sequences such as TSE-short-tau inversion recovery **[18-20]**.

DWI was usefully in recognize and evaluate of pancreatitis, which the DWI showed an increasing of signal intensity with corresponding decreased ADC values compared to normal parenchyma of the pancreas **[21]**.

Patients and Methods

This observational cross-sectional study was approved by the Local Ethics Committee during the period from March 2016 to May 2017.

Inclusion criteria:

1- Patient coming with abdominal pain, abdominal discomfort, jaundice, abdominal mass, significant weight loss and loss of appetite.

2- Patient made ultrasound or CT that reported any pancreatic lesion.

3- Pre-operative patient for detection of tumor staging.

4- Patient with abnormal level of pancreatic enzymes as alkaline phosphatase, amylase and lipase or high level of pancreatic tumor markers such as carbohydrate antigen 19-9 (CA 19-9) and Carcinoembryonic Antigen (CEA).

5- Patient with abnormal level of liver function tests as serum bilirubin, Asparate Aminotransferase (AST) and Alanine Aminotransferase (ALT).

Exclusion criteria:

1- Patient having any metallic implants as pacemaker, aneurysm clips, joint replacement or any other electronic or magnetically activated implant.

2- Patient with renal failure or heart failure.

Patients:

The study included 23 patients (9 males and 14 females); mean age ± SD, 54.4±9.4 years, with clinical symptoms as (jaundice, abdominal pain, vomiting, nausea, fever, dyspepsia and/or weight loss) and any biochemical signs as [increased serum levels of amylase, lipase, alkaline phosphatase and/or carbohydrate antigen (CA 19.9)] suggesting pancreatic disease referred to the Radiology and Imaging Department in Tanta University Hospital from the Department of Internal Medicine, Surgery and Oncology. 23 normal age-matched controls who underwent abdominal MRI in the same period; 10 males and 13 females; the mean age ± SD, 54.4 ±11.9 years.

23 pancreatic lesions were divided into 4 groups; AP (acute pancreatitis) (group A) (n=5), pancreatic cyst (group B) (n=8), adenocarcinoma (group C) (n=9) and FP (focal mass forming pancreatitis) (group D) (n=1), furthermore the cystic lesions were subdivided into benign (n=4) and malignant (n=4) subgroup. They were examined by our standard complete upper abdomen protocol (including T1, T1FS, T2, T2FS and dynamic contrast study) and additional DWI.

Patients were asked to fast for 6 hours before the MRI examination and renal profile was done and reviewed for all patients. All twenty-three patients underwent intravenous MRI contrast; eighteen of them underwent pancreatic surgery or biopsy.

MR protocol and parameters:

1- MTI-c:

All patients were examined with a 1.5-T MR scanner magnet unit General Electric (GE) Healthcare using a phased-array body coil.

All patients were examined by the routine upper abdomen MRI protocol that included non-contrast axial T1-Weighted Images (T1WI) breath hold gradient echo with and without Fat Suppression (FS) (TR/TE, 195/1.5; number of excitation, 1; flip angle, 700), coronal and axial T2 Weighted Images (T2WI) single shot free breathing (TE=28m sec; TR ?4000ms; slice thickness 5mm; slice gap 1-2mm; matrix 200 X 240 with a field of view, 379 X 279) and T2 (FS) sequence (TE=80msec; TR ?4000msec; slice thickness 5mm; slice gap 2mm; matrix 204 X 384 with a field of view, 379 X 279). Then, 0.1mmol/kg of gadopentetate dimeglumine (Magnevist) was injected intravenously followed by flushing with 20ml saline solution.

The dynamic series comprised into 4 individual images, obtained at 25, 54, 08 and 150sec. That related to arterial, pancreatic parenchymal, venous and delayed phase respectively.

2- DWI:

Single-shot spin-echo echo-planar imaging and SPAIR FS pulse sequences were done.

Generalized auto-calibrating was used by integrated parallel imaging techniques and partially parallel acquisitions were used to twofold acceleration, as follows: repetition time/echo time, 5000/80 ms; section thickness, 5mm; gap, 1.7mm; matrix, 156 X 192; field of view, 300-400mm; bandwidth,

1446Hz/pixel; parallel imaging factor of 2; partial Fourier factor, 6/8; averages, 2; free breathing; and b-values of 50, 500 and 1000s/mm2, less than 2min was taken in a typical scan.

The signal intensities of the pancreatic lesions in all DW images were assessed and compared with the signal intensities of the surrounding parenchyma.

3- ADC calculation:

The mean ADCs of the detected lesions and ADCs of the normal pancreatic parenchyma in the control group were obtained through inducement a Region of Interest (ROI) over the lesion then the ADC maps were generated automatically by the MR software, a free-hand ROI was traced with the trying to avoid bile ducts and vessels. The average of three measurements of b factors (50, 500 and 1,000s/mm2) was verified as the final mean ADC.

4- Time Intensity Curve (TIC):

The ROI was drawn at the part of lesion or normal parenchyma in the control group then TICs were obtained. "The pancreatic TIC was generated as a percentage increase in the Signal Intensity (SI), according to the following enhancement formula: (SI post contrast-SI pre-contrast)/SI pre-contrast X 100" **[22]**.

The patterns of TIC in our study were classified into 3 types: Type I; characterized by a rapid rise to a peak (25sec) after administration of contrast material followed by a rapid decline. Type II, characterized by a rapid rise to a peak (45sec) after administration of contrast material followed by a slow decline. Type III, characterized by a slow rise to a peak (80sec) after administration of contrast material followed by a slow decline.

Image analysis:

Each lesion was verified through its morphological pattern including site, shape, margin, size, signal intensity, form of dynamic enhancement then a provisional diagnosis was informed, then we revised the DWI with ADC values and reviewed TIC for final radiological characterization of the pancreatic lesions.

In the control group; ADC values and (TIC) were obtained from their normal pancreatic parenchyma and then we compared its results with patients' results.

Statistical analysis:

Data were evaluated using IBM SPSS advanced statistics Version 22 (SPSS Inc., Chicago, IL). Numerical data were articulated as mean and Standard Deviation (SD) and range. Qualitative data were noted as percentage and frequency.

For normally disseminated quantitative data, comparison between control and patients' groups was done using parametric *t*-test followed by ANOVA test. Calculation of sensitivity, specificity, Positive Predictive Value (PPV), Negative Predictive Value (NPV) and accuracy was done for the two diagnostic techniques (MRI-c and DWI with ADC value) considering results of the histopathology to be the gold standard for 18 focal lesions and clinical assessment with follow up biochemical signs to be the gold standard for 5 AP.

Results

The final diagnosis of 23 pancreatic lesions was 5 lesions of AP and 18 focal lesions by histopathologic examination that was as follows: 13 patients had a malignant lesion and 5 patients had a benign lesion. Malignant lesions included; 9 pancreatic adenocarcinoma, 2 malignant Intraductal Papillary Mucinous Tumour (IPMT), 1 mucinous cystadenocarcinoma, 1 malignant Solid Pseudopapillary Neoplasm (SPN) and benign lesions included; 1 FP, 1 serous cystadenoma, 2 simple pseudo-pancreatic cysts and 1complicated pseudopancreatic cyst.

By using MRI-c; 5 AP, 13 malignant lesions and 4 benign lesions were detected and correctly characterized. One false positive lesion was diagnosed, it was a lesion of Serous Cystadenoma with no false negative lesions. By DWI, 5 AP, 12 malignant lesions and 4 benign lesions were correctly characterized.

One false positive lesion was diagnosed, it was a lesion of complicated pseudo-pancreatic cyst and one false negative lesion, it was a lesion of malignant SPN (Table 1). The sensitivity, specificity, accuracy, positive and negative predictive values of DWI and MRI-c were 92.3%, 90%, 91 .3%, 92.3%, 90% and 100%, 90%, 95.7%, 92.9%, 100%, respectively (Table 2). The mean ADC value ± SD of the cystic lesions (n=8) was (2.80 ± 0.63 X $10^{-3}mm^2$/sec) ranged from 2.10 to 3.87 X $10^{-3}mm^2$ /sec; while the mean ADC of benign cysts (n=4) was 3.24 ± 0.59 X $10^{-3}mm^2$/s ranged from 2.46 to 3 .87 X $10^{-3}mm^2$/s and that of malignant cysts (n=4) was 2.35 ± 0.19 X $10^{-3}mm^2$/s ranged from 2.10 to 2.53 X $10^{-3}mm^2$/s. The mean ADC value ± SD of acute pancreatitis was 1.28 ± 0.08 X $10^{-3}mm^2$/sec, ranged from 1.19 to 1 .36 X $10^{-3}mm^2$ /sec, and that of the pancreatic adenocarcinomas

was 1.12±0.16 X 10^{-3} mm^2/sec, ranged from 0.91 to 1.36 X 10^{-3} mm^2/sec.

While the ADC value of the case with FP lesion, was 1.54 X 10^{-3} mm^2/sec. On the other hand, the mean ADC ± SD of control group was (1.84± 0.12 X 10^{-3} mm^2/s), ranged from 1.65 to 2.12 X 10^{-3} mm^2/s. By TIC; the normal pancreatic tissue in control group demonstrated type I, while in acute pancreatitis, FP demonstrated type II and adenocarcinomas demonstrated type III. The age means difference between patients and control groups were statistically non-significant (F=2.22, p=0.09), while the difference between ADC mean values of our patients' groups and control group were statistically significant (F=84.62 and p <0.001).

The ADC value was also significantly lower for malignant cysts than for benign cysts (2.35±0.19 X 10^{-3} mm^2/s versus 3.24±0.59 X 10^{-3} mm^2/s; p <0.02). The optimal cutoff value of ADC for differentiating malignant cyst from benign cyst was 2.49 X 10^{-3} mm^2/s. The mean ADC values of both acute pancreatitis and adenocarcinoma groups were significantly (p<0.001) lower than those of cystic and control groups Fig. (1).

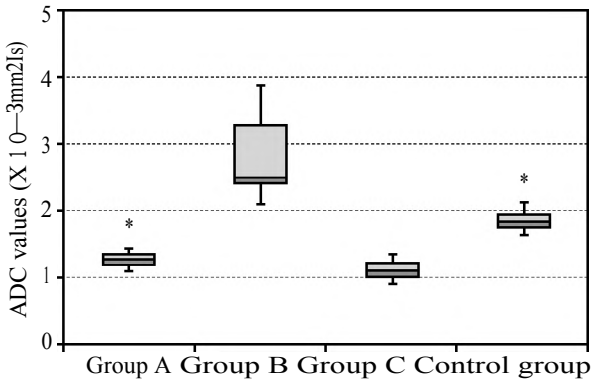

Fig. (1): Box plot of the ADC values of patient groups and control group.

*: p<0.001 vs. control and group B; decompensated lower ADC values and restricted diffusion in both groups (A & C).

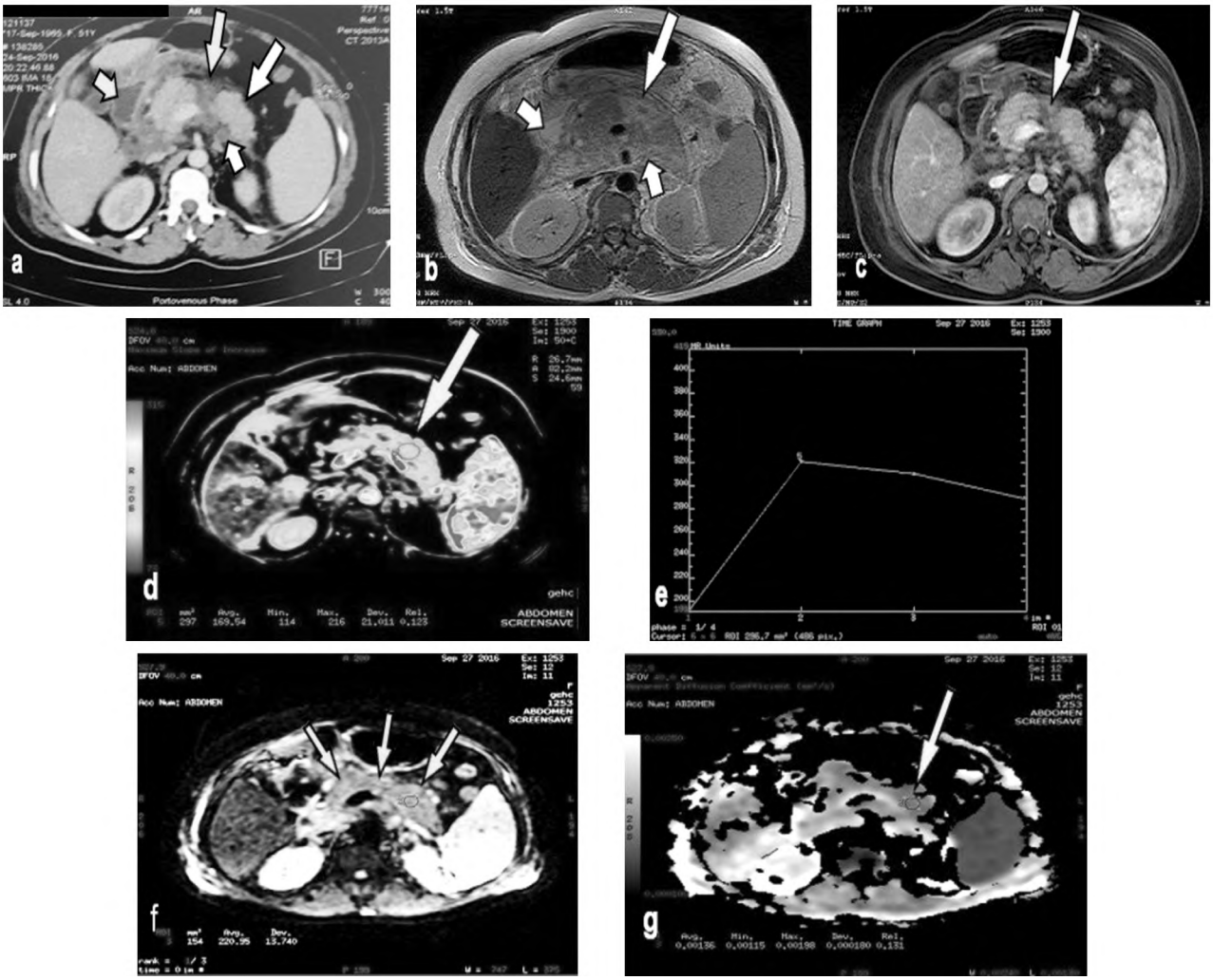

Fig. (2): A 57-year-old female patient complained of acute abdominal pain and back pain. Examination showed acute epigastric pain. Diagnosis of acute pancreatitis.

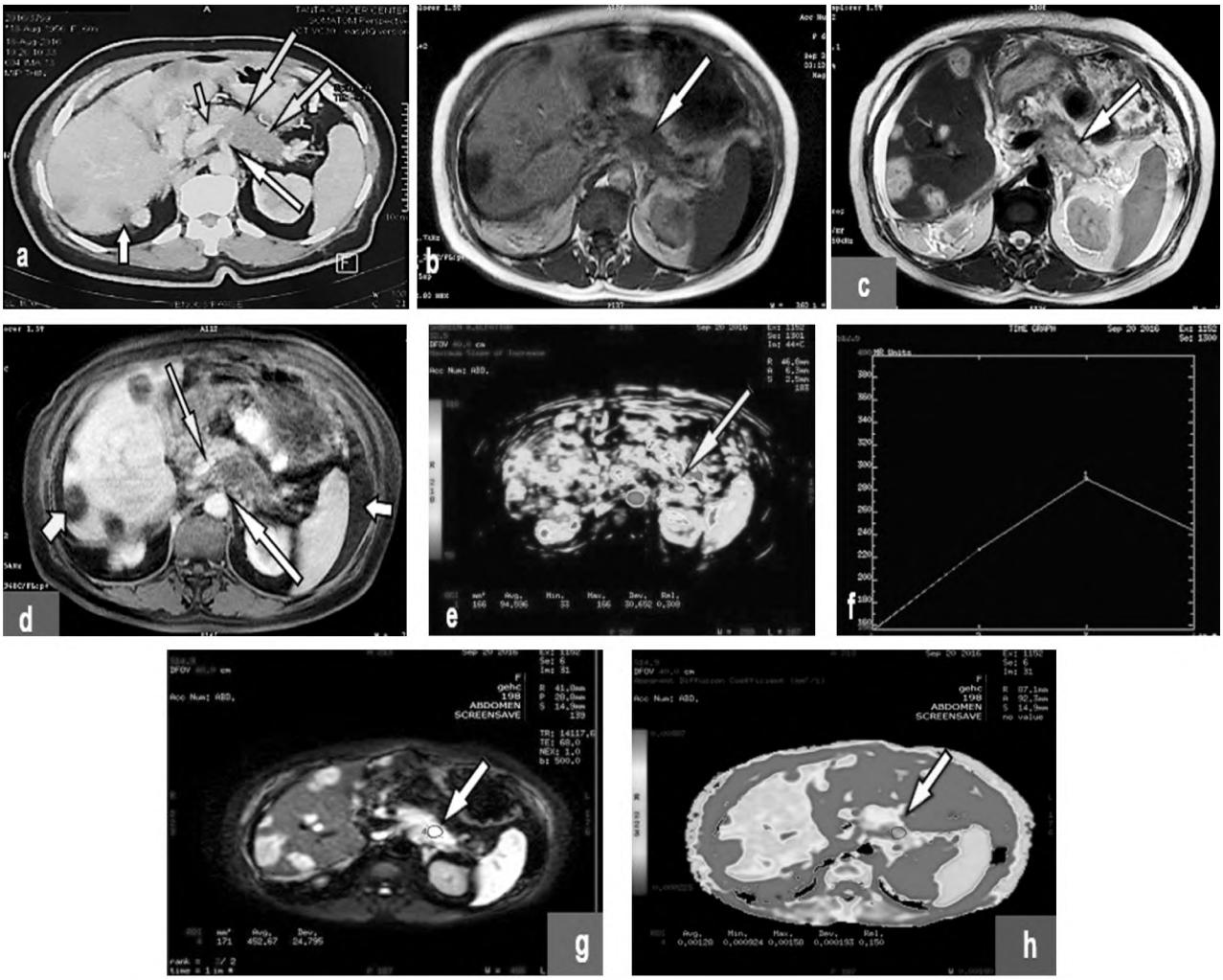

Fig. (3): A 60-year-old diabetic female patient complained of abdominal pain. Examination showed abdominal tenderness over epigastric area. Diagnosis of pancreatic body adenocarcinoma.

Contrast enhanced axial CT showed irregular border of the pancreas (long white arrow) with non-enhanced area of necrosis is seen at the body (about 30%) (long yellow arrow); with strandy density in the fat planes (short white arrow) and fluid collection surrounding it (short yellow arrow) (a). Axial T2WI showed diffuse enlarged pancreas with heterogenous hyperintense signal, strandy density in the fat planes surrounding it (short white arrows) and fluid collection (short yellow arrow) and hyperintense necrotic area is seen at pancreatic body (long yellow arrow) (b). Enhanced T1WI showed non-enhanced area of necrosis at the body (arrow) (c). MIP study of dynamic contrast enhanced MRI with ROI at the body (arrow) (d). TIC obtained by dynamic contrast study showed type II pattern (e). DWI showed hyperintense signal (restricted) all over the pancreas (arrows) (f). ADC map obtained by DWI showed low signal and low selected area ADC value (1.36 X 10^{-3} mm^2/s) (arrow) (g). MIP; (Maximum Intensity Projection).

Contrast enhanced axial CT showed non-enhanced hypodense ill-defined pancreatic body mass (long yellow arrows), showed encasement of celiac trunk (long white arrow) & splenic vein (short yellow arrow) with multiple bilobar hepatic focal lesion (short white arrow) are seen scattered (a). Axial T1WI (b) and axial T2WI (c) showed an ill-defined heterogenous hypointense on T1 and hyperintense on T2 lesion is seen at pancreatic body measures (6 X 4cm) (arrow). Enhanced T1WI showed heterogenous enhancement of mentioned body mass with encasement of celiac trunk (long white arrow) and splenic vein (long yellow arrow) with multiple non-enhanced hepatic focal lesions (short yellow arrow) and ascites (short white arrow) (d). MIP with ROI at the body mass (arrow) (e). TIC obtained by dynamic contrast study showed type III pattern (f). DWI showed (restricted) hyperintense signal at the body mass (arrow) (g). ADC map obtained by DWI showed a low selected area ADC value (1.28 X 10^{-3} mm^2/s) (arrow) (h).

Table (1): True +ve, true –ve, false +ve and false –ve of DWI and MRI-c for pancreatic lesions.

	True +ve	True –ve	False +ve	False –ve
MRI-C[a]	13	9	1	
DWI	12	9	1	1
DWI + MRI-c	13	10	–	–

a: Contrast enhanced MRI. DWI: Diffusion Weighted Image.

Table (2): Qualitative analysis of DWI and MRI-c for pancreatic lesions.

	DWI	MRI-c	DWI + MRI-c
Sensitivity (%)	92.3	100	100
Specificity (%)	90	90	100
PPV (%)	92.3	92.9	100
NPV (%)	90	100	100
Accuracy (%)	91.3	95.7	100

PPV: Positive Predictive Value. NPV: Negative Predictive Value.

Discussion

In our study, we noted that the most of acute pancreatitis lesions (80%) showed restricted diffusion (increase signal intensity on DWI with decrease ADC values) and the mean ADC value in pancreatitis group ($1.28 \pm 0.083 \times 10^{-3}$ mm^2/sec) was significant ($p<0.001$) lower than the mean ADC value of the control group ($1.84 \pm 0.12 \times 10^{-3}$ mm^2/s).

That correlated with Thomas et al., **[23]** and Yencilek et al., **[24]**, who reported restricted diffusion in cases of acute pancreatitis that showed increased intensity of signal on DWI with decreased ADC values, they noted the mean ADC values in pancreatitis was (1.19×10^{-3} mm^2/s$\pm$0.32) lower than normal pancreatic parenchyma (1.78×10^{-3} mm^2/s$\pm$0.29).

In our study, we found that the maximum contrast enhancement in acute pancreatitis was observed at (45sec.) that corresponding to parenchymal phase and showed type II pattern of TIC Fig. (2). Other studies performed by Busireddy et al., **[25]** and Salemi et al., **[26]** had the same conclusion that the supreme enhancement phase of pancreatic parenchyma in acute pancreatitis lesion was detected at the late arterial phase (40sec.), and the TIC showed (rabid increased followed by signal plateau).

In our study, we noted that the mean ADC value of malignant cysts ($2.35 \pm 0.19 \times 10^{-3}$ mm^2/s) was significantly ($p<0.02$) lower than that of benign cysts ($3.24 \pm 0.59 \times 10^{-3}$ mm^2/s) with cutoff value of ADC for differentiate malignant from benign cyst was 2.49×10^{-3} mm^2/s and DWI correctly noted one case of serous cystadenoma, that was falsely characterized as a malignant cyst on MRI-c, which measured high ADC value that was 3.87×10^{-3} mm^2/sec. This correlated with Sandrasegaran et al., **[27]**, who reported that the ADC of malignant cysts were significantly lower than ADC of benign and the cut-off ADC value for differentiating between benign and malignant cystic was 2.85×10^{-3} mm^2/s.

In our study, there was one false positive lesion by DWI; that was a lesion of complicated pseudocyst as it showed diffusion restriction with low ADC value.

Other studies performed by kartalis et al., **[28]**, and Ajaykumar et al., **[29]** had the same conclusion that complicated pseudocysts showed some restriction on DWI that is thought to be due to high viscosity of its content. Many previous studies reported that the neoplastic cysts and complicated pseudocysts have a viscous content and multiseptation that lead to restriction of diffusion and decrease in ADCs values compared to the simple cysts and pseudocysts have a lower viscosity that lead to a higher ADC **[30-32]**.

In our study, we found that the mean ADC value of adenocarcinoma ($1.12 \pm 0.16 \times 10^{-3}$ mm^2/sec): Ranged ($0.91\text{-}1.36 \times 10^{-3}$ mm^2/s) was significantly ($p<0.001$) lower than mean ADC of normal pancreas ($1.84 \pm 0.12 \times 10^{-3}$ mm^2/sec): Ranged ($1.65\text{-}2.12 \times 10^{-3}$ mm^2/sec), findings were consistent with the results of Wang et al., **[33]** and Matsuki et al., **[34]**, they reported that the mean ADC value of PC; ranged ($1.1\text{-}1.4 \times 10^{-3}$ mm^2/s) was significantly lower than that of normal pancreas; ranged ($1.6\text{-}1.9 \times 10^{-3}$ mm^2/s); and correlated with Choi et al., **[35]**, who reported that the mean ADC value of the adenocarcinomas was $1.13 \pm 0.23 \times 10^{-3}$ mm^2/s. DWI correctly characterized one case with FP that was non-restricted (isointense on DWI with isointense signal and ADC value was 1.54×10^{-3} mm^2/sec on ADC maps). That correlated with Zhang et al., **[36]**, who noted that the mean ADC value of masses are lower in PC than FP (1.17 ± 0.23, 1.47 ± 0.18, respectively), and the cutoff value of ADC of adenocarcinoma was 1.3036; and also correlated with Abo Warda et al., **[37]**, who showed that the mass forming pancreatitis did not show high signal intensity on DWI and had benign reading by ADC value.

Previous studies noted that the restricted diffusion in tumor caused by hyper-cellularity and the signal intensities and the ADC values are dependent on the cellularity and amount of tissue fibrosis **[38,39]**.

In our study, we noted that all the pancreatic adenocarcinoma lesions showed delayed heterogenous enhancement in post dynamic contrast study that showed type III pattern of TIC with peak at (80sec.) Fig. (3), while a case of FP that correctly diagnosed by MRI-c showed type II pattern with peak at (45sec.) which related to parenchymal phase. This finding was consistent with Megibow et al., **[40]**, Tajima et al., **[41]** and Zhang et al., **[36]**, who reported that adenocarcinoma displayed delayed enhancement and the peak of FP in the time intensity curve was earlier than the peak of PC; that the peak in FP was at 45 second, while in PC was at 2 or 3 minutes, and the curve of adenocarcinoma demonstrated slower increase to peak followed by slower decline. These were consistent with the previous studies, reporting that pancreatic carcinoma was hypo-vascular tumor character, while FP showed an earlier enhancement followed by a slow decreasing pattern **[42,43]**.

In our study, the mean ADC value of control group (1.84±0.12 X $10^{-3}mm^2/s$), range (1.65-2.12 X $10^{-3}mm^2/s$) and showed type I pattern of TIC with a peak at (25s); that correlated with Thomas et al., **[23]** and Barral et al., **[44]**, they reported that the range of mean ADC value for normal pancreatic group was (1.77±0.32 X $10^{-3}mm^2/s$) and agreed with Kim et al., **[45]**, who noted that the normal pattern of pancreatic parenchymal enhancement showed TIC of type A characterized by rapid peak followed by a rapid decrease. We noted that the mean ADC value of cystic lesions (2.80±0.63) was more than the ADC value of adenocarcinomas (1.12±0.16) and FP (1.54 X $10^{-3}mm^2/s$).

That correlated with Wang et al., **[33]**, who reported that the mean ADC values showed higher values in cystic lesions than in solid lesions.

In our study, we noted that the mean ADC value of adenocarcinoma group (1.12±0.16 X 1 $0^{-3}mm^2$/sec) was lower than the mean ADC value of pancreatitis group (1.28±0.08 X 1 $0^{-3}mm^2$/sec). That correlated with Barral et al., **[44]**, who noted that the mean ADC value of acute & chronic pancreatitis (1 .3 X 10^{-3} & 1.5 X $10^{-3}mm^2/s$) was higher than ductal adenocarcinoma (1.1 X $10^{-3}mm^2/s$) respectively. Finally, our results showed that, MRI-c showed higher sensitivity than DWI. The sensitivity, specificity and NPP of MRI-c were 100%, 90% and 100% while in DWI was 92.3%, 90 and 90% respectively. That correlation with Kartalis et al., **[28]**, who noted that MRI-c showed higher sensitivity than DWI. The sensitivity, specificity and NPP of MRI-c were 100%, 97% and 100%, while in ADC was 92%, 97 and 98% respectively. In our study, the correlation of sensitivity, specificity and NPV between both MRI-c and DWI was 100%, 100% and 100% respectively.

This agreed with the study done by Abo Warda et al., **[37]** that noted both MRI-c and DWI were corresponding to each other; by using two tests, the sensitivity was 100%, specificity was 100% and NPV was 100%.

Conclusion:

MRI plays an important role in the diagnosis of different pancreatic lesions and can assess the neoplastic pancreatic lesions with accurate detection of extension, vascular encasement, nodal involvement and hepatic metastatic lesions.

DWI is an imaging technique that is sensitive to water diffusion in living tissues and ADC measures can differentiate between benign and malignant pancreatic tumors.

Competing interests:

The authors pronounce that they have no competing interests.

References

1- JEMAL A., SIEGEL R., WARD E., HAO Y., XU J., MURRAY T., et al.: Cancer statistics. CA Cancer J. Clin., 58: 71-96, 2008.

2- SCHIMA W., BA-SSALAMAH A., KÖLBLINGER C., KULINNA-COSENTINI C., PUESPOEK A. and GÖTZINGER P.: Pancreatic adenocarcinoma. Eur. Radiol., 17: 638-49, 2007.

3- VAN GULIK T.M., MOOJEN T.M., VAN GEENEN R., RAUWS E.A., OBERTOP H. and GOUMA D.J.: Differential diagnosis of focal pancreatitis and pancreatic cancer. Ann. Oncol., 10: 85-8, 1999.

4- Quoted by: LYNG H., HARALDSETH O. and ROFSTAD E.: Measurement of cell density and necrotic fraction in human melanoma xenografts by diffusion weighted magnetic resonance imaging. Magn. Reson. Med., 43: 828-36, 2000.

5- ROBERTSON R. and GLASIER C.: Diffusion-weighted imaging of the brain in infants and children. Pediatr. Radiol., 37 (8): 749-68, 2007.

6- NONOMURA Y. , YASUMOTO M., YOSHIMURA R., HARAGUCHI K., ITO S., AKASHI T., et al.: Relationship between bone marrow cellularity and apparent diffusion coefficient. J. Magn. Reson. Imaging, 13 (5): 757-60, 2001.

7- STEER M.L., WAXMAN I. and FREEDMAN S.: Chronic pancreatitis. N. Engl. J. Med., 332: 1482-90, 1995.

8- FREENY P.C.: Radiology of the pancreas: Two decades of progress in imaging and intervention. A.J.R. Am. J. Roentgenol., 150: 975-81, 1988.

9- LOWENFELS A.B. and MAISONNEUVE P.: Risk factors for pancreatic cancer. J. Cell Biochem., 95: 649-56, 2005.

10- BIRCHARD K.R., SEMELKA R.C., HYSLOP W.B., et al.: Suspected pancreatic cancer: Evaluation by dynamic gadolinium-enhanced 3D gradientecho MRI. A.J.R. Am. J. Roentgenol., 185: 700-3, 2005.

11- KIM T., MURAKAMI T., TAKAMURA M., et al.: Pancreatic mass due to chronic pancreatitis: Correlation of CT and MR imaging features with pathologic findings. A.J.R. Am. J. Roentgenol., 177: 367-71, 2001.

12- VAN HOE L., GRYSPEERDT S., ECTORS N., et al.: Nonalcoholic duct-destructive chronic pancreatitis: Imaging findings. A.J.R. Am. J. Roentgenol., 170: 643-7, 1998.

13- SEMELKA R.C., SHOENUT J.P., KROEKER M.A. and MICFLIKIER A.B.: Chronic pancreatitis: MR imaging features before and after administration of gadopentetate dimeglumine. J. Magn. Reson. Imaging, 3: 79-82, 1993.

14- ROS P.R., HAMRICK-TURNER J.E., CHIECHI M.V., et al.: Cystic masses of the pancreas. Radio. Graphics, 12: 673-86, 1992.

15- SCHEIMAN J.M.: Management of cystic lesions of the pancreas. J. Gastrointest. Surg., 12: 405-7, 2008.

16- ATTASARANYA S., PAIS S., Le BLANC J., McHENRY L., SHERMAN S. and DeWITT J.M.: Endoscopic ultrasound guided fine needle aspiration and cyst fluid analysis for pancreatic cysts. J.O.P., 8: 553-63, 2007.

17- BUETOW P.C., RAO P. and THOMPSON L.D.: From the Archives of the AFIP. Mucinous cystic neoplasms of the pancreas: Radiologic-pathologic correlation. Radio Graphics., 18: 433-49, 1998.

18- HIROTA M., KIMURA Y., ISHIKO T., BEPPU T., YAMASHITA Y. and OGAWA M.: Visualization of the heterogeneous internal structure of so-called “pancreatic necrosis” by magnetic resonance imaging in acute necrotizing pancreatitis. Pancreas, 25 (1): 63-7, 2002.

19- STIMAC D., MILETIC D., RADIC M., KRZNARIC I., MAZUR-GRBAC M., PERKOVIC D., et al.: The role of nonenhanced magnetic resonance imaging in the early assessment of acute pancreatitis. Am. J. Gastroenterol., 102 (5): 997-1004, 2007.

20- KIM Y.K., KO S.W., KIM C.S. and HWANG S.B.: Effectiveness of MR imaging for diagnosing the mild forms of acute pancreatitis: Comparison with MDCT. J. Magn. Reson. Imaging, 24 (6): 1342-9, 2006.

21- AKISIK M.F., AISEN A.M., SANDRASEGARAN K., JENNINGS S.G., LIN C., SHERMAN S., et al.: Assessment of chronic pancreatitis: Utility of diffusion-weighted MR imaging with secretin enhancement. Radiology, 250 (1): 103-9, 2009.

22- Quoted by: TAJIMA Y., MATSUZAKI S., FURUI J., ISOMOTO I., HAYASHI K. and KANEMATSU T.: Use of the time-signal intensity curve from dynamic magnetic resonance imaging to evaluate remnant pancreatic fibrosis after pancreatico-jejunostomy in patients undergoing pancreaticoduodenectomy. Br. J. Surg., 91: 595-600, 2004.

23- THOMAS S., KAYHAN A., LAKADAMYALI H. and OTO A.: Diffusion MRI of acute pancreatitis and comparison with normal individuals using ADC values. Emerg. Radiol., 19: 5-9, 2012.

24- YENCILEK E., TELLI S., TEKESIN K., OZGUR A., CAK•R O., TURKOGLU O., et al.: The efficacy of diffusion weighted imaging for detection of acute pancreatitis and comparison of subgroups according to Balthazar classification. Turk. J. Gastroenterol., 25: 553-7, 2014.

25- BUSIREDDY K.K., AL-OBAIDY M., RAMALHO M., KALUBOWILA J., BAODONG L., SANTAGOSTINO I., et al.: Pancreatitis-imaging approach. World J. Gastrointest. Pathophysiol., 5 (3): 252-70, 2014.

26- SALEMI S., DONATI F., BORASCHI P., GIGONI R., BARTOLOZZI C. and FALASCHI F.: MR perfusion of pancreatic transplants: Usefulness of time signal intensity curves. Eropian Society of Radiology, 41: 1-12, 2011.

27- SANDRASEGARAN K., AKISIK F.M., PATEL A.A., RYDBERG M., CRAMER H.M., AGARAM N.P., et al.: Diffusion weighted imaging in characterization of cystic pancreatic lesions. Clinical Radiology J., 66: 808-14, 2012.

28- KARTALIS N., LINDHOLM T.L., ASPELIN P., PERMERT J. and ALBIIN N.: Diffusion-weighted magnetic resonance imaging of pancreas tumours: Eur. Radiol. J., 19: 1981-90, 2009.

29- AJAYKUMAR C., KHALED M., PETER S., WILLIAM J., JANIO S. and JONATHAN R.: Abdominal applications of diffusion-weighted magnetic resonance imaging. World J. Radiol., 5 (3): 68-80, 2013.

30- LINDER J.D., GEENEN J.E. and CATALANO M.F.: Cyst fluid analysis obtained by EUS-guided FNA in the evaluation of discrete cystic neoplasms of the pancreas: A prospective single-center experience. Gastrointest. Endosc. J., 64: 703-4, 2006.

31- INAN N., ARSLAN A. and AKANSEL G.: Diffusion weighted imaging in the differential diagnosis of simple and hydatid cysts of the liver. A.J.R., 189: 1031-6, 2007.

32- LEUNG K.K., ROSS W.A. and EVANS D.: Pancreatic cystic neoplasm: The role of cyst morphology, cyst fluid analysis, and expectant management. Ann. Surg. Oncol., 16: 2818-24, 2009.

33- WANG Y., CHEN Z.E. and NIKOLAIDIS P.: Diffusion-weighted magnetic resonance imaging of pancreatic adenocarcinomas: Association with histopathology and tumor grade. J. Magn. Reson. Imaging, 33 (1): 136-42, 2011.

34- MATSUKI M., INADA Y. and NAKAI G.: Diffusion-weighed MR imaging of pancreatic Carcinoma. Abdominal Imaging, 32: 481-3, 2007.

35- CHOI S.Y., KIM S.H., KANG T.W., SONG K.D., PARK H.J. and CHOI Y.H.: Differentiating Mass-Forming Autoimmune Pancreatitis from Pancreatic Ductal Adenocarcinoma on the Basis of Contrast-Enhanced MRI and DWI Findings. A.J.R., 206: 291-300, 2016.

36- ZHANG T.T., WANG L., LIU H.H., ZHANG C., LI X., LU J., et al.: Differentiation of pancreatic carcinoma and mass-forming focal pancreatitis: qualitative and quantitative assessment by dynamic contrast-enhanced MRI combined with diffusion weighted imaging. Onco. Target Impact Journals, 8 (1): 1744-59, 2017.

37- ABO WARDA M., HASAN D. and ELTEEH O.: Differentiation of Pancreatic lesions using Diffusion Weighted MRI. The Egyptian Journal of Radiology and Nuclear Medicine, 46: 563-8, 2015.

38- PADHANI A.R., LIU G., KOH D.M., CHENEVERT T.L., THOENY H.C., TAKAHARA T., et al.: Diffusionweighted

magnetic resonance imaging as a cancer biomarker. Consensus and recommendations. Neoplasia, 11: 102-25, 2009.

39- MURAOKA N., UEMATSU H., KIMURA H., IMAMURA Y., FUJIWARA Y., MURAKAMI M., et al.: Apparent diffusion coefficient in pancreatic cancer: Characterization and histopathological correlations. J. Magn. Reson. Imaging, 27 (1): 302-40, 2008.

40- MEGIBOW A.J.: Are we really closer to predicting the development of pancreatic cancer? Radiology, 254 (3): 642-6, 2010.

41- TAJIMA Y., KUROKI T., TSUTSUMI R., ISOMOTO I., UETANI M. and KANEMATSU T.: Pancreatic carcinoma coexisting with chronic pancreatitis versus tumor-forming pancreatitis: Diagnostic utility of the time-signal intensity curve from dynamic contrast-enhanced MR imaging. World Journal of Gastroenterology, 13 (6): 858-65, 2007.

42- AKISIK M.F., SANDRASEGARAN K., BU G., LIN C., HUTCHINS G.D. and CHIOREAN E.G.: Pancreatic cancer: Utility of dynamic contrast enhanced MR imaging in assessment of antiangiogenic therapy Radiology, 256: 441-9, 2010.

43- PARK M.S., KLOTZ E., KIM M.J., SONG S.Y., PARK S.W., CHA S.W., et al.: Perfusion: Onco target impact journals: CT: Noninvasive surrogate marker for stratification of pancreatic cancer response to concurrent chemo- and radiation therapy. Radiology, 250: 110-7, 2009.

44- BARRAL M., TAOULI B., GUIU B., KOH D.M., LUCIANI A., MANFREDI R., et al.: Diffusion-weighted MR Imaging of the Pancreas: Current Status and Recommendations 1. Radiology, 274: 45-63, 2015.

45- KIM J.H., LEE J.M., PARK J.H., KIM S.C., JOO I., HAN J.K., et al.: Solid Pancreatic Lesions: Characterization by Using Timing Bolus Dynamic Contrast enhanced MR Imaging Assessment- A Preliminary Study 1: Radiology: Number 1-January. Gastrointestinal Imaging: Radial k-Space-weighted Technique for Pancreatic Lesions, 266: 185-96, 2013.

دراسة دور التصوير بالرنين المغناطيسى فى تشخيص إصابات البنكرياس المختلفة

يعتبر سرطان البنكرياس على الرغم من كونه غير مألوف هو من بين الأسباب الرئيسية المرتبطة بالسرطان فى جميع أنحاء العالم. حيث أنه وفقا للجمعيةالأمريكية للسرطان، بأن نسبة البقاء على قيد الحياة سنة واحدة هو ٢٤٪، ونسبة بقائه ٥ أعوام هو ٥٪ مع نتائج مماثلة ذكرت فى جميع أنحاء العالم.

يقدم الإستئصال الجراحى الكامل مع العلاج الكيميائى أفضل النتائج فى علاج هذا السرطان. ومع ذلك، فإن الإكتشاف المتأخر للمريض وتأخر ظهور الأعراض على المريض، قد تكون العملية الجراحية بنية الإستكشاف الجراحى أكثر منها بنية العلاج التى من المحتمل أن تكون غير ناجحة نتيجة لزيادة معدل الإعتلال، لذلك فإن التقييم الدقيق قبل العملية هو المهم على مر السنين.

ولقد أثبت الرنين جدارته وإستخدامه على نطاق واسع فى تشخيص أمراض البنكرياس التعددة حيث أنه تفرد بدقته وحساسيته فى تصوير الأنسجة عن باقى وسائل التصوير المختلفة.

أيضا لقد أثبت الرنين حديثا جدارته فى التشخيص المبكر وتحديد مرحلة ونوع أورام البنكرياس المختلفة بإستخدام وسيلة التصوير بالإنتشار الموزون حيث تقوم فكرته على قياس درجة إنتشار وحركة جزيئات الماء فى الأنسجة المختلفة، وبحساب حركة هذه الجزيئات يتم الحصول على الإنتشار الموزون حيث يتم الإستخراج منه ما يسمى بمعامل الإنتشار الظاهر الذى يتم من خلالهما التفرقة بين أورام البنكرياس الحميدة منها أو الخبيثة.

ولقد تم فى الآونة الأخيرة إستخدام التصوير بالرنين المغناطيسى وإستخدام خاصيته الإنتشار الموزون ومعامل الإنتشار الظاهرى فى تقييم الإلتهاب الحاد والمزمن للبنكرياس والتفرقه بينهما من خلال تلك الفحوصات.

وفى الآونة الأخيرة أيضا أظهرت تقنية منحنى شدة إشارة الوقت للبنكرياس والتى تم الحصول عليها من التصوير الديناميكى بإستخدام الصبغة لتكون تقنية مراقبة موثوقة لتقييم دقيق للتليف البنكرياس.

وتهدف هذه الدراسة إلى تقييم دور التصوير بالرنين المغناطيسى فى تشخيص والتفريق بين إصابات البنكرياس المختلفة.

تم فحص ٢٣ مريضا من المشتبه إصابتهم بأمراض البنكرياس المختلفة تراوحت أعمارهم ما بين ٣٥ إلى ٧٤ عاما قدمو إلى مستشفيات جامعة طنطا يعانون من آلام بالبطن والقيئ وبالفحص الإكلينيكى تبين إشتباه إصابتهم بإلتهاب أو أورام البنكرياس.

تم رصد ٩ حالات لأورام البنكرياس السرطانية و٥ حالات للإلتهابات الحادة والمزمنة للبنكرياس و٨ حالات مصابة بأكياس حميدة وسرطانية.

وتم عمل فحوصات الدم اللازمة والتصوير بالموجات الفوق صوتية والأشعة المقطعية متعددة الحزم وبالتصوير بالرنين المغناطيسى، متضمنة التصوير بإستخدام خاصية الإنتشار الموزون ومعامل الإنتشار والتصوير الدينا ميكى بالصبغة، وخضع ١٨ مريض منهم لأخذ عينة من أفة البنكرياس وقورنت نتائج الفحص بالرنين المغناطيسى بنتائج العينات لهؤلاء المرضى وبالمقارنة بينهم تبين حساسية ودقة الرنين المغناطيسى العالية فى تحديد وتقييم أمراض البنكرياس المختلفة.

وإن الفحص بإستخدام خاصية الإنتشار الموزون أثبت دوره الهام فى تقييم أورام البنكرياس مع تشخيص دقيق لمكان إنتشار الورم، وإلتفافه بالشرايين وإكتشاف إنتشاره للغدد الليمفاوية والكبد.

وأخيرا، يعتبر التصوير بالرنين المغناطيسى من أهم الوسائل التشخيصية التى يمكن أن الإعتماد عليها فى الكشف المبكر عن أورام البنكرياس، ويمكن إستخدامهما كطرق تصويرية أساسية فى الممارسة الإكلينيكية لهذه الحالات.