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Performance of Intracystic Glucose Measurement for the Characterization of Pancreatic Cystic Lesions

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ABSTRACT

Background & Aims: Endoscopic ultrasound (EUS)-guided fine-needle aspiration (FNA) is essential for the classification of pancreatic cystic lesions (PCLs). Recently, intracystic glucose has been suggested as an alternative to carcinoembryonic antigen (CEA) level as a predictor of mucinous cystic lesions (M-PCLs). This study aims to evaluate the diagnostic performance of intra-cystic glucose in distinguishing between M-PCLs and non M-PCLs (NM-PCLs) and to analyze the possibility of on-site glucose measurement with a standard glucometer.

Methods: Patients with PCLs submitted to EUS-FNA with simultaneous intracystic glucose measurement between 2017 and 2022 were included. The diagnostic performance of glucose versus CEA for the differentiation between M-PCLs and NM-PCLs was compared to a final diagnosis based on the analysis of surgical specimen, intracystic biopsy or, if this data was unavailable, multidisciplinary evaluation. A cut-off of <50 mg/dL was used for the diagnosis of MCLs. Additionally, the agreement between on-site glucose determination with a standard glucometer and laboratory glucose measurement was assessed.

Results: Mucinous lesions accounted for 56% of all PCLs. The median values of glucose and CEA for M-PCLs were 18 mg/dL and 286 ng/mL, respectively. Intracystic glucose had a sensitivity and specificity of 93.2% and 76.5%, respectively, for the diagnosis of MCLs (versus 55.6% and 87.5%, respectively, for CEA). The area under the curve was 0.870 for on-site glucose (versus 0.806 for CEA). An excellent correlation was observed between on-site and laboratory glucose measurement (ρ=0.919).

Conclusions: The measurement of intracystic glucose showed superior performance compared with CEA in distinguishing between M-PCLs and NM-PCLs, with excellent correlation between on-site and conventional lab glucose measurement. Thus, on-site intracystic glucose appears to be an excellent biomarker for the characterization of PCLs due to its low cost, high availability, and the need for a minimal cyst fluid volume for its determination.

Key words: pancreatic cystic lesions – glucose – carcinoembryonic antigen – endoscopic ultrasound.

Abbreviations: AUROC: area under the receiver operating curve; CEA: carcinoembryonic antigen; EUS: endoscopic ultrasound; FNA: fine-needle aspiration; M-PCL: mucinous pancreatic cystic lesion; NM-PCL: non mucinous pancreatic cystic lesion; NPV: negative predictive value; PCL: pancreatic cystic lesion; PPV: positive predictive value; TTNB: through-the-needle biopsy.

INTRODUCTION

Pancreatic cystic lesions (PCLs) are increasingly prevalent lesions due to the widespread use of cross-sectional abdominal imaging, and most are discovered incidentally [1]. This group of lesions encompasses a large array of entities, ranging from benign lesions with negligible malignant potential to premalignant and malignant lesions. An accurate differential diagnosis between lesions with no malignant potential and premalignant or malignant lesions is paramount as it prevents morbidity resulting from overtreatment of benign lesions and identifies patients requiring active surveillance or surgical treatment [2, 3]. Distinguishing between mucinous PCLs (M-PCLs) and non-mucinous PCLs (NM-PCLs) represents the main challenge, as most premalignant PCLs are mucinous.

Current recommendations indicate endoscopic ultrasound (EUS) as the subsequent step for presumed M-PCLs

presenting with worrisome features on cross-sectional imaging. Endoscopic ultrasound evaluation should include guided fine needle aspiration (FNA) for cyst fluid analysis combining cytology and carcinoembryonic antigen (CEA) levels [4, 5]. Cyst fluid cytology presents a low sensitivity, and the distinction between M-PCLs and NM-PCLs is frequently based on CEA levels [6]. Nevertheless, this biomarker has shown a suboptimal performance for differentiating M-PCLs, with sensitivities and specificity ranging from 52-78% and 63-91%, respectively, when using the historical cut-off of ≥192 ng/mL [5, 7]. Furthermore, lab assessment of CEA levels requires significant cystic fluid volumes (>200 μL) and has reproducibility and interpretability issues with varying cut-off values according to different laboratory assays [8]. Over the last decade, molecular analyses for cystic fluid and tissue sampling techniques have been developed, but their application to clinical practice has been hampered by their limited availability and high costs [9-11].

More recently, intracystic glucose has emerged as a potential biomarker for the distinction between M-PCLs and NM-PCLs. Indeed, M-PCLs appear to have a lower intracystic glucose concentration, probably reflecting a higher glucose uptake of premalignant PCLs [12]. Glucose is widely available and inexpensive biomarker and several studies have shown superiority over intracystic CEA. Nevertheless, there is a paucity of studies reporting on the use of glucose for the classification of PCLs and evidence comparing on-site glucometry and laboratorial glucose measurement remains scarce. Our study aimed to assess the performance of intracystic glucose for the differentiation between M-PCLs and NM-PCLs, assessing the correlation between on-site glucometer and lab-based glucose measurement, and ultimately compare it to that of standard of care biochemical assessment of intracystic CEA.

METHODS

Population and Study Design

This was a retrospective study performed at a single center (Centro Hospitalar Universitário de São João, Porto, Portugal). Patients undergoing EUS-FNA due to PCLs, for whom a glucose determination, either using on-site glucometer or lab measurement.

All collected data was introduced into an electronic database. Patients' demographics and clinical features (gender, age, symptoms, history of pancreatitis, indication for EUS, smoking habits, and family history of pancreatic disease), along with data regarding the EUS features of cysts (location, size and morphology, wall thickness and presence of mural nodules/solid components) and histopathological findings were collected from individual electronic clinical records.

EUS Procedures and Pancreatic Cystic Fluid Analysis

All EUS procedures were performed by two experienced endosonographers (F.V.B. and P.M.R.), each possessing more than a decade of experience in EUS practice and having completed over 1000 procedures. The procedures were carried out using Olympus® GF-UCT180 and Olympus® GF-UC140 curvilinear echoendoscopes, coupled with the Olympus® EU-ME2 ultrasound processor. All interventions were performed under anesthesiologist-guided for sedation.

Cystic lesions were punctured using 19-gauge or 22-gauge FNA needles (Expect™ Slimline, Boston Scientific Corp., Marlborough, Massachusetts, USA) either through the stomach for lesions situated in the body or tail, or via the duodenum for lesions located in the head of the pancreas. For patients with more than one cystic lesion, only the larger was considered for analysis.

Glucose was measured using both an on-site and a laboratory approach. On-site glucose measurement was performed using a conventional glucometer (GlucoMen® Aero 2K, A. Menarini, Firenze, Italy), with a range between 20-600 mg/dL. All samples with glucose levels < 10 mg/dL were recorded and analyzed as 19 mg/dL. In patients with an appropriate cyst fluid volume, the values of CEA levels were determined.

Outcomes

The final diagnosis of cystic lesions as M-PCLs or NM-PCLs was based on the surgical specimen for patients undergoing resection, a conclusive EUS-guided through-the-needle-biopsy (Moray® microforceps, STERIS, Mentor, Ohio, USA) or a global evaluation after multidisciplinary discussion, including imaging features, combined with cyst fluid cytology, CEA and glucose levels.

Primary outcome measures include the sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), accuracy, and area under the receiver operating curve (AUROC). A cut-off for intracystic glucose of <50 mg/dL was used as a threshold for the diagnosis of M-PCLs. For the estimation of these performance marks, we used any available glucose measurement, either at the lab or on-site. When both values were available, on-site glucose measurement was chosen. A CEA value >192 ng/mL was considered for the diagnosis of M-PCLs.

Statistical Analysis

Categorical variables are reported as frequencies and percentages. Comparisons of these variables using the chi-square or the Fisher exact tests. Continuous variables are described as median and interquartile range (IQR). The correlation between on-site and lab glucose measurements was assessed using Spearman's Rank Correlation Coefficient. The discriminating performance of glucose and CEA was assessed by the analysis of the respective area under the receiver operating characteristic curves (AUROC). These curves were compared using the DeLong test. A two-sided p value of 0.05 was considered for statistical significance.

Statistical analysis was performed using SPSS Statistics version 29 (Armonk, NY, USA), and MedCalc version 22.014 (MedCalc Software, Ostend, Belgium).

RESULTS

Patient Characteristics

We included a total of 78 patients, of whom 44 (56%) had a final diagnosis of M-PCL. The final diagnosis was achieved by global evaluation in 39 patients (50%), surgical specimen in 28 patients (36%) and EUS guided through-the-needle biopsy (TTNB) in 11 patients (14%). For patients ultimately diagnosed

with M-PCLs, most were diagnosed with intraductal papillary mucinous neoplasm (IPMN, n=39), whereas the most common final diagnosis in patients with NM-PCLs were serous cystic neoplasms (n=16, 47%) and pseudocysts (n=14, 41%). Included patients were predominantly female individuals (n=48, 62%) and had a median age at diagnosis of 64 years (IQR 52 – 72). The most frequent cyst location was the pancreatic head (n=25, 32%), followed by the body and tail (each n=18, 23%). The cysts had a median size of 31 mm (IQR 26 – 43). Demographics and clinical data of the patients are summarized in Table I.

Table I. Population demographics and pancreatic cysts characteristics

	M-PCLs (n=44)	NM-PCLs (n=34)	p
Female, n (%)	30 (68)	18 (53)	0.170
Age, median (IQR)	67 (58 – 76)	54 (46 – 66)	<0.001
Cyst diameter, mm (IQR)	29 (24 – 35)	37 (30 – 62)	<0.001
Cyst location			0.701
Head	14 (32)	11 (32)	
Neck	6 (14)	7 (21)	
Body	12 (27)	6 (18)	
Tail	9 (21)	9 (27)	
Uncinate	3 (7)	1 (3)	
Final diagnosis			0.294
Surgical specimen, n (%)	19 (43)	9 (27)	
EUS-TTNB, n (%)	6 (14)	5 (15)	
Global evaluation, n (%)	19 (43)	20 (59)	
Cyst histologic type			
IPMN, n (%)	39 (89)	-	
SCN, n (%)	-	16 (47)	
Pseudocyst, n (%)	-	14 (41)	
MCN, n (%)	5 (11)	-	
SPN, n (%)	-	2 (6)	
NET, n (%)	-	1 (3)	
Foregut ciliated cyst, n (%)	-	1 (3)	

EUS-TTNB: endoscopic ultrasound-guided through the needle biopsy; IQR: interquartile range; IPMN: intraductal papillary mucinous neoplasm; PCL: pancreatic cystic lesion; M-PCL: mucinous PCL; MCN: mucinous cystic neoplasm; NET: neuroendocrine tumor; NM-PCL: non-mucinous PCL; SCN: serous cystic neoplasm; SPN: solid pseudopapillary neoplasm.

Correlation between Laboratorial and On-site Glucose Measurement

All included patients had at least one glucose measurement. An on-site glucose measurement was available for 54 patients (69%), while a laboratorial assessment was performed for 64 patients (82%). Forty patients had both evaluations performed simultaneously (51%). A strong correlation existed between on-site and lab glucose measurements (ρ=0.919, p<0.001). Fig. 1 shows the differences in glucose determinations. For patients who had both glucose determinations, the median difference was 9 (IQR 0 – 10; range -47 - 98). On-site glucose provided higher values than lab glucose determination in 30 cases (75%). In only one case (2.5%), with an ultimate diagnosis of a pseudocyst, glucose determinations resulted in conflicting classifications (lab 66 mg/dL vs. on-site 19 mg/dL).

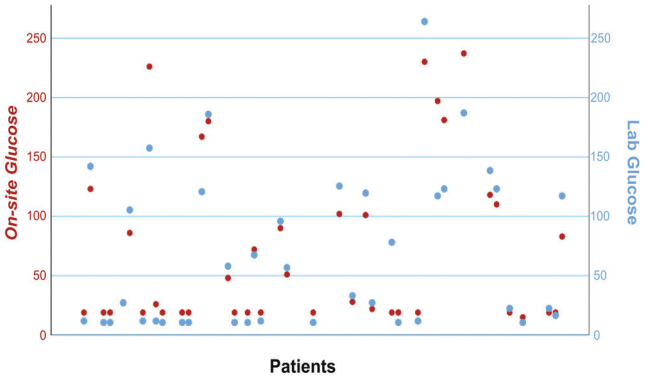

Fig. 1. Difference in glucose values for patients with simultaneous laboratory and on-site glucose determinations. Glucose levels are presented in mg/dL.

Performance of Glucose and CEA in the Differentiation of PCLs

The median glucose concentration was 18 mg/dL (IQR 9 – 19) for M-PCLs, whereas this value was 98 mg/dL for NM-PCLs (IQR 59 – 119) (Table II). The median CEA value was 286 (IQR 28 – 1736) for M-PCLs and 3 (IQR 1 – 42) for NM-PCLs. A glucose concentration <50 mg/dL had a sensitivity of 93.2%, specificity of 76.5%, PPV of 83.7%, NPV of 76.5%, and an overall accuracy of 85.9% for the identification of M-PCLs. Oppositely, a CEA value > 192 ng/mL differentiated M-PCLs from NM-PCLs with a sensitivity of 55.6%, a specificity of 87.5%, a PPV of 83.3%, a NPV of 64.6%, and an overall accuracy of 70.6%. The AUROC for the differentiation of M-PCLs versus NM-PCLs was 0.870 and 0.912 for on-site glucose and lab glucose, respectively (Fig. 2). No significant differences were observed between the AUROC lab and on-site glucose determinations (p=0.13). CEA measurement showed a significantly lower AUROC compared to any glucose determination (AUROC 0.806, p=0.01) (Fig. 3).

Table II. Median glucose values by type of measurement

	M-PCLs (n=44)	NM-PCLs (n=34)	p
On-site glucose, mg/dL, median (IQR)	19 (19 – 19)	110 (75 – 164)	<0.001
Lab glucose, mg/dL, median (IQR)	9 (9 – 10)	89 (53 – 107)	<0.001

For abbreviations see Table I.

DISCUSSION

The establishment of diagnostic and accurate risk stratification remain a significant challenge for the management of patients with PCLs [13]. Differentiating between M-PCLs and NM-PCLs represents the most important step in the clinical management of these patients. Nevertheless, to this date, the differentiation between both groups is sustained upon EUS morphological characterization, limited by a significant interobserver variability, as well as biochemical and cytological analyses, which have shown to have limited sensitivity and high associated costs. More recently, a large interest has been devoted to new sophisticated diagnostic methods to provide an

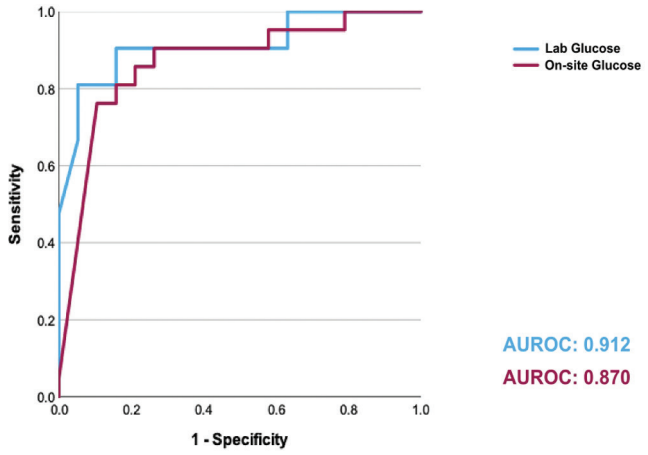

Fig. 2. Receiver operating characteristic curve analysis for both laboratory and on-site glucose determinations. AUROC: area under the receiver operating characteristic curve.

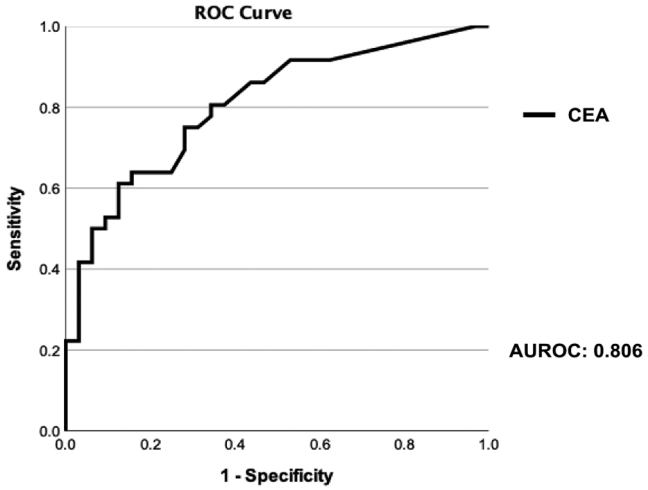

Fig. 3. Receiver operating characteristic curve analysis for carcinoembryonic antigen. For abbreviation see Fig. 2.

answer to this question, particularly with the development of EUS-TTNB and artificial intelligence algorithms. Nevertheless, these methods are not widely available and, particularly for the latter, real-life clinical validation studies have not yet been performed.

In this study, using a cut-off of 50 mg/dL, intracystic glucose allowed an accurate classification of PCLs, with an overall accuracy of 89% and AUROCs ranging from 0.87 and 0.91. Moreover, the correlation between lab glucose and on-site glucose was determined for the first time, and a strong correlation between both determinations was established (ρ=0.919, p<0.001).

The findings of our study are in line with the evidence of recent studies on the role of intracystic glucose for the evaluation of pancreatic cystic lesions. A recent meta-analysis of studies comparing intracystic glucose and CEA for the identification of M-PCLs demonstrated a higher pooled sensitivity and overall accuracy for intracystic glucose (91% vs. 56% and 94% vs. 85%, respectively, both p<0.001), while preserving similar specificity values [14]. Faias et al. [15] have assessed frozen PCL fluid samples from 82 patients and identified significant differences in intracystic glucose levels between M-PCLs and NM-PCLs (19 vs. 105 mg/dL, respectively, p<0.0001). A cut-off of <50 mg/dL allowed the identification of M-PCLs with a sensitivity of 89% and a specificity of 86%, compared with a sensitivity and specificity of 72% and 96%, respectively, for CEA. That study was performed using frozen samples, rather than fresh PCL fluid, from patients with PCLs with a definite histologic result from surgical specimens or a conclusive cytology. While this ground truth is optimal for the assessment of the diagnostic performance of intracystic glucose, the clinical relevance of these results is hampered by the low number of patients with PCLs who ultimately undergo surgery and the poor sensitivity of EUS-FNA cytology. Moreover, the impact of freezing in intracystic glucose and the final classification of PCLs based on its values remains unexplored. More recently, in a cohort of 93 patients with PCLs with definite histologic diagnosis who have had intracystic glucose determined by lab glucometry, a threshold of ≤ 40 mg/dL showed a sensitivity, specificity and accuracy of 95%, 82% and 90%, respectively [16].

The biochemical characterization of PCLs submitted to FNA has relied on the determination of CEA values on PCL fluid. Nevertheless, the use of this biomarker has several pitfalls, most notably its limited sensitivity and accuracy for the identification of M-PCLs and the significant heterogeneity in cut-off values used, thus limiting its reproducibility and interpretation [8]. Moreover, CEA dosing requires significant PCL fluid volumes (>200 L), which can difficult to obtain, and has a high cost per procedure (over 100$) [17]. The use of intracystic glucose measurement as a biomarker for differentiation of PCLs can overcome many of these limitations. Indeed, recent studies have showed a superior performance of PCL fluid glucose compared to CEA for the identification of M-PCLs [14, 16]. Furthermore, the measurement of glucose levels requires significantly lower PCF volumes, which can be particularly helpful for PCLs with scant PCL fluid volume. In this study, on-site glucometry required as little as 0.5 L, as per glucometer manufacturer specifications, to report glucose levels [18]. Nevertheless, despite this technical vantage over CEA dosing, there are some feasibility issues, especially in patients with higher PCF viscosity, for whom valid readings may not be possible [15]. Indeed, reading errors occurred in up to 22% due to high PCF viscosity, which, nevertheless, constitutes a feature of a mucinous phenotype [15, 19]. In this study, we did not observe on-site intracystic measurement errors using the chosen glucometer. Additionally, we observed a strong correlation between on-site and lab glucometry (0.919, p<0.001). These results are in line with those reported by Noia et al. [19], which calculated an intraclass correlation coefficient of 0.98 [19].

Our results, in line with previous evidence, may have a significant clinical impact. Indeed, glucose has some advantages over CEA as a biomarker for the characterization of PCLs. First, M-PCLs have significant lower glucose levels comparing with NM-PCLs, and glucose has been shown to perform superiorly and be more reproducible than CEA dosing [14, 16]. A notable exception are pseudocysts, which have been demonstrated to have lower glucose levels compared to other NM-PCLs [15]. In our study, 7 PCLs were misclassified as M-PCLs based on PCF glucose levels, 4 of which were ultimately diagnosed as

pseudocysts, and the others as a serous cystic neoplasm, a solid pseudopapillary neoplasm and a foregut ciliated cyst (each n=1). Therefore, classification of these lesions must integrate other findings, including aspects from clinical presentation, imaging, as well as intracystic CEA levels. Second, glucose measurement, and particularly on-site glucometry is highly available, has a low cost. Moreover, the strong correlation between both types of glucose determination, ensures that on-site determination provides valid results, thus streamlining subsequent clinical management of patients with PCLs.

This study has several limitations. Firstly, this was a unicentric study, including a limited number of patients with PCLs. Secondly, the definitive diagnosis for each included PCL was not based on histology results (either from through-the-needle biopsy or surgical specimens). Despite this limitation, this approach follows current standards of clinical practice, as only a small percentage of patients are submitted to EUS-TTNB or pancreatic surgery. Thirdly, both glucose determinations were not performed for all patients. Therefore, the correlation between both determinations was established in a smaller subset of patients. Finally, most included lesions were IPMNs (n=39), serous cystic neoplasm (n=16) and pseudocysts (n=14) and, thus, the intracystic glucose profile of less prevalent lesions could not be fully determined.

CONCLUSIONS

In our study, we observed that, comparing to the current standard of care (CEA measurement), intracystic glucose determination had a superior diagnostic performance for the differentiation between M-PCLs and NM-PCLs. Moreover, we verified that on-site glucose measurement provides reproducible results compared to lab glucometry, thus making glucose an accurate, highly available and low-cost biomarker for PCLs.

Conflicts of interest: None to declare.

Author's contribution: T.R. and G.M. conceived the study. T.R., S.L., P.M.R., G.M. and F.V.B. designed the study. S.L., P.M.R and F.V.B performed the endoscopic ultrasound procedures. T.R. and F.V.B collected the data. T.R drafted the manuscript. S.L., P.M.R., G.M. and F.V.B critically revised the manuscript. All the authors approved the final version and agree to be accountable for all aspects of the work.

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