RESEARCH ARTICLE

Is endoscopy mandatory in cases mimicking acute pancreatitis?

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Abstract

Objective: To detect the utility of nonspecific rising of pancreatic enzymes in patients with stomach discomfort, and to explore the possibility of hyperamylasemia as a differential diagnosis.

Method: The cross-sectional study was conducted from April 2020 to April 2021 at two large tertiary care centres in Kafrelsheikh and Zagazig governorates in northern Egypt, and comprised patients complaining of dull aching abdominal pain. They were classified into two groups. Patients with nonspecific rise in pancreatic enzymes less than threefold in the absence of acute pancreatitis were in group I, while those having abdominal pain without rise in pancreatic enzymes were in group II. All patients were subjected to detailed history and clinical examination followed by laboratory assessment, imaging studies and upper endoscopy. Data was analysed using SPSS 20.

Results: Of the 270 patients, 170(63%) were in group I; 120(70.5%) males and 50(29.5%) females with mean age 51±6.58 years, There were 100(37%) patients in group II; 65(65%) males and 35(35%) females with mean age 53±8.96 years ($p>0.05$). Amylase, lipase, serum glutamic-oxaloacetic transaminase, serum glutamic-pyruvic transaminase, C-reactive protein and helicobacter pylori values were significantly different between the groups ($p<0.05$).

Conclusions: Elevation of pancreatic enzymes with a level less than three-fold in patients with abdominal pain reflected mucosal injury of the gastrointestinal tract and may raise the necessity for diagnostic upper endoscopy.

Keywords: Alanine transaminase, Hyperamylasemia, Diabetic ketoacidosis, Amylases, Lipase, Pancreatitis, Chronic, Pancreatic ducts, Liver diseases, Peptic ulcer, Metaplasia, Aspartate aminotransferases.

DOI: 10.47391/JPMA.EGY-S4-17

Introduction

Pancreatic diseases are related to amylase and lipase levels in the blood. The presence of changes in the pancreas and a threefold or more increase in blood amylase is usually a sign of acute pancreatitis. Extrapancreatic diseases, such as gastrointestinal (GI) diseases or chronic viral liver diseases, may be associated with this increase if it is not greater than threefold.[1-7] Head trauma, human immunodeficiency virus (HIV) infection, mumps, ectopic pregnancy are all related to this condition.[8] The chronic pancreatic non-pathological hyperenzymemia syndrome was identified in 1996, and was defined by the absence of pancreatic diseases and an asymptomatic state with elevated blood levels of pancreatic enzymes.[9-11] This elevation has no evident underlying cause. Some suggested that it could be produced by a defect in the acinar cells' basolateral area [12], causing an increase in enzyme transit into the bloodstream, or by a defect in the Wirsung duct caused by secretin stimulation[11], which may increase the need of endoscopic examination in a patient with the nonspecific increase in pancreatic enzymes.

The current study was planned to detect the utility of nonspecific rising of pancreatic enzymes in patients with stomach discomfort, and to explore the possibility of hyperamylasaemia as a differential diagnosis.

Patients and Methods

The non-randomised cross-sectional study was conducted from April 2020 to April 2021 at the Department of Hepatology, Gastroenterology and Infectious Diseases, Kafrelsheikh University Hospital, and the Department of Tropical Medicine, Zagazig University Hospital, Egypt. The sample was raised from among those visiting the outpatient departments (OPDs). Those included were adult patients of either gender complaining of dull aching abdominal pain with or without nonspecific rise in the level of pancreatic enzymes. Patients with typical symptoms of acute pancreatitis, or those who were either pregnant or refused endoscopic assessment were excluded.

After taking written informed consent from all the subjects, they were classified into two groups. Patients with nonspecific rise in pancreatic enzymes less than threefold in the absence of acute pancreatitis were in group I, while those having abdominal pain without rise in pancreatic enzymes were in group II.

All patients were subjected to detailed history and clinical examination, followed by laboratory assessment, imaging studies and upper GI endoscopy.

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Blood samples were tested for serum amylase, lipase and complete blood count (CBC)done on automated cell counter (Pentra XL80, Horiba, France), and C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), urea, creatinine, triglycerides (TG) and lactate dehydrogenase (LDH) using the colorimetric method on automated chemistry analyser (Pentra C 400-Horiba, France) using its commercially available reagents. Helicobacter (H.) pylori antigen and occult blood were checked in the stool. Imaging studies included ultrasound (US) and computed tomography (CT) of abdomen.

The upper endoscopy was performed under minimal sedation, with the patient breathing spontaneously with an oxygen mask. Upper endoscopy (Pentax EG3890) was performed by the same endoscopist at the same endoscopy unit, with a full examination up to the second portion of the duodenum. Biopsies were obtained from the suspicious lesions in order to conduct a detailed histopathological evaluation.

The endoscopic examination was done without knowledge of the pancreatic enzymes and other laboratory parameters.

Mucosal biopsies were randomly taken from the suspected lesions (oesophageal, stomach, or duodenum), targetting the area affected macroscopically with the significant mucosal disease. Two pathologists with specialisation in gastroenterology evaluated the biopsies and reported using a standardized checklist for histopathology that included histologically normal, gastritis, mucosal erosions, peptic ulcer, others (gastric metaplasia or malignancy, etc). Chronic gastritis was defined histologically as having characteristics of chronic mucosal illness, such as chronic inflammatory cells. A gastric erosion was a superficial mucosal lesion that did not invade the submucosa, unlike a stomach ulcer. Intestinal metaplasia was a potentially reversible transformation in which a fully differentiated cell type was changed with another differentiated cell type.

Permission and official approval to carry out the study was obtained from the concerned authorities. All patients signed a written informed consent prior to the study and the institutional ethical committee in Kafrelshiekh and Zagazig University Faculty of Medicine approved the study.

Data was analysed using SPSS 20. Data was expressed as mean and standard deviation or as frequencies and percentages, as appropriate. Chi-square test and t test comparisons were done, as appropriate. P<0.05 was considered statistically significant.

Results

Of the 270 patients, 150(55.5%) were at the Kafrelsheikh University Hospital, and 120(44.4%) were at the Zagazig University Hospital. Of the total, 170(63%) were in group I; 120(70.5%) males and 50(%) females with mean age 51±6.58 years. There were 100(37%) patients in group II; 65(65%) males and 35(35%) females with mean age 53±8.96 years (p>0.05) (Table 1).

Amylase, lipase, SGOT and SGPT, CRP and H. pylori values were significantly different between the groups (Table 2).

Intergroup differences were also significant in values associated with gallbladder (GB) (Table 3).

Gastritis was significantly associated with abdominal pain in group II, but erosion, peptic ulcer and intestinal metaplasia were significantly associated with group I (Figure).

Table-1: Demographic data and smoking status.

	Abdominal pain with raising of pancreatic enzyme (n=170)	Abdominal pain without raising of pancreatic enzyme (n=100)	*p-value*
Age	51 ±6.58	53 ±8.96	0.14
Gender			
Male	120 (70.5)	65 (65.0)	0.33
Female	50 (29.5)	35 35.0%	
Smoker	88 (51.7)	52 (52.0)	
Non-Smoker	82 (48.3)	48 (48.0)	0.97

Table-2: Pathological parameters.

	Abdominal pain with raising of pancreatic enzyme (n=170)	Abdominal pain without raising of pancreatic enzyme (n=100)	*p-value*
Amylase	315.36±35.63	56.36±15.85	0.00**
Lipase	385.32±27.88	43.25±12.63	0.00**
SGOT	64.23±21.63	37.71±11.87	0.00**
SGPT	58.97±14.36	35.97±10.51	0.00**
Creatinine	1.03±0.14	0.99±0.11	0.098
Urea	29.63±5.36	27.98±7.25	0.421
RBG	132.36±10.36	129.68±12.36	0.345
Hb	12.58±2.32	12.87±1.89	0.789
WBCs	6.25±2.07	5.87±1.45	0.298
CRP			
-VE	120 (70.5)	93 (93.0)	0.00001**
+VE	50 (29.5)	7 (7.0)	
ESR			
Low	170 (100.0)	100 (100.0)	1.0
High	0 (0.0)	0 (0.0)	
Ca	8.95±1.05	8.92±0.89	0.81
Mg	2.42±0.34	2.39±0.41	0.48

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Table-2: *continued from previous page*

	Abdominal pain with raising of pancreatic enzyme (n=170)	Abdominal pain without raising of pancreatic enzyme (n=100)	*p-value*
NA	139.9±3.25	140.04±4.02	0.085
K	4.87±0.43	4.92±0.37	0.24
LDH			
-VE	160 (94.1)	98 (98.0)	0.13
+VE	10 (5.9)	2 (2.0)	
TG			
Low	140 (82.3)	90 (90.0)	0.087
High	30 (17.7)	10 (10.0)	
H, pylori			
-VE	125 (73.6)	85 (85.0)	0.02*
+VE	45 (26.4)	15 (15.0)	
Occult blood			
-VE	158 (93.0)	97 (97.0)	0.25
+VE	12 (7.0)	3 (3.0)	

SGOT: Serum glutamic-oxaloacetic transaminase, SGPT: Serum glutamic-pyruvic transaminase, RBG: Random blood glucose, Hb: Haemoglobin, WBCs: White blood cell counts, ESR: Erythrocyte sedimentation rate, Ca: Calcium, K: Potassium,
Mg: Magnesium, CRP: C-reactive protein, LDH: Lactate dehydrogenase, TG: Triglycerides, H. pylori: Helicobacter pylori.

Table-3: Radiological findings.

	Abdominal pain with high pancreatic enzyme (n=170)	Abdominal pain without high pancreatic enzyme (n=100)	*p-value*
US			
Normal	170 (100.0)%	90 (90.0)%	0.0001**
GB	0 (0.0)%	10 (10.0)%	
CT			
Normal	170 (100.0)%	90 (90.0)%	0.0001**
GB	0 (0.0)%	10 (10.0)%	

US: Ultrasound, GB: Gallbladder, CT: Computed tomography.

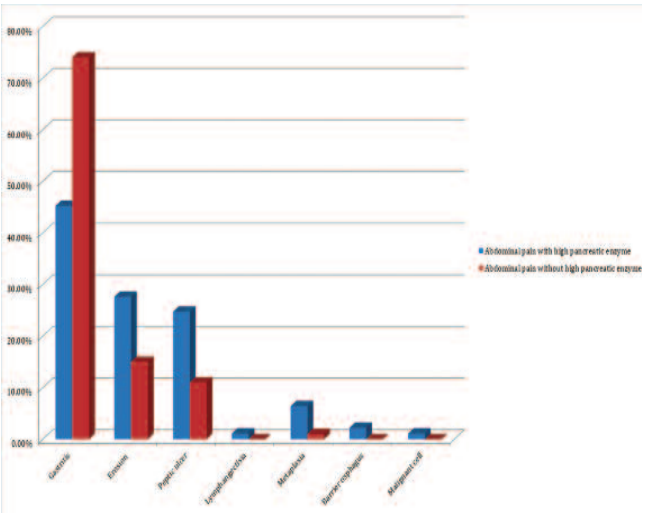

Figure: Histopathological results of endoscopic findings.

Discussion

The current study explored if it it really necessary to use the level of pancreatic enzymes as a biomarker for patients presenting with abdominal pain even when symptoms of typical pancreatitis are not apparent. It also explored if the nonspecific elevation of pancreatic enzyme level can be taken as a reliable indicator of ongoing mucosal injury in the GI tract.

Egypt has limited health insurance coverage, and a large percentage of patients, particularly in rural areas, lack convenient and effective communication with endoscopists and gastroenterologists. As a result, a large number of patients are diverted to primary healthcare (PHC), which is generally available and accessible. In majority cases, the staff in PHC centres are junior caregivers, general practitioners, or family physicians who lack experience and facilities in managing patients with abdominal pain, whether or not their pancreatic enzymes are elevated. In some cases, they may mistakenly diagnose patients as having pancreatitis with nonspecific symptoms. Acknowledging the ground realities, the current study tried to identify simple and reliable biomarkers and non-invasive tests for a clear and simple aetiological assessment of stomach discomfort.[13]

Multiple studies and systematic reviews have identified a wide range of pancreatic enzyme elevations due to other intra-abdominal diseases resulting from the stomach, oesophagus, intestine and hepatobiliary tract, as well as from neoplastic disease[14-17], which is consistent with the current findings.

The current study noticed that some biomarkers other than pancreatic enzymes, like C-RP, ESR, LDH and TG levels in patients presenting with abdominal pain reflected the value of these nonspecific markers in assessing the need for endoscopy. An earlier study reported that LDH was an intracellular cytoplasmic enzyme found in all tissues of the human body.[18]

Conclusion

Increased pancreatic enzymes less than threefold in patients with abdominal pain usually reflected mucosal injury of the GI tract, highlighting the need for diagnostic upper endoscopy.

Limitation: The sample size for the study was not calculated. All patients attending the department of Hepatology, Gastroenterology and Infectious Diseases, in Kafrelsheikh University Hospital, and the department of Tropical Medicine, Zagazig University Hospital, Egypt, from April 2020 to April 2021 were included.

Disclaimer: None.

Conflict of Interest: None.

Source of Funding: None.

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