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Research Article

Serum Pseudocholinesterase as a Biomarker in the Differentiation between Gastric Cancer and Benign Gastric Diseases

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ABSTRACT

Background: Worldwide gastric cancer is the fifth most common cancer with poor prognosis. In early stages, it is hard to distinguish gastric cancer from benign gastric diseases, resulting in delayed diagnosis. There is a need to develop a biomarker for differentiating between gastric cancer and benign gastric diseases. Serum cholinesterase is synthesized in liver and released into plasma, and it has an important role in oncogenesis.

Objectives: To determine the correlation between serum cholinesterase activity and gastric cancer, in comparison to benign gastric diseases.

Subjects and Methods: A case control study carried out at Medical City Directorate\ Gastroenterology, Hepatology Hospital, and at Oncology Teaching Hospital from April 2022 to September 2022. It involved 25 patients with gastric cancer and age matched 25 patients with benign gastric diseases. Serum cholinesterase activity was determined by a colorimetric method..

Results: There was a significant difference in the mean level of serum cholinesterase between gastric cancer group (5339.28 U/L±1816) and benign gastric diseases group (7516.92 U/L±2351) with (P value<0.001). Significant association between low levels of serum cholinesterase and early cancer stages and grades (P value<0.001). Serum cholinesterase showed 60% sensitivity and 80% specificity in differentiating between gastric cancer and benign gastric diseases with optimal cutoff value of 5568U\L.

Conclusions: Serum cholinesterase can be considered as a potential rapid and non-invasive biomarker for differentiating between gastric cancer and benign gastric diseases.

Introduction

Gastric cancer (GC) is one of the most common malignancies worldwide and it is the fourth leading cause of cancer-related death [1]. Due to the lack of typical symptoms of early GC, most patients with GC were diagnosed in the late stage of the disease with extensive regional lymph nodes involvement and/or invasion of adjacent structures. However, the prognosis for patients with advanced GC remains poor, with median survival times of 10–13 months [2]. The 5-year survival rate for GC is 31% in the United States. Average survival rates reflect the fact that most cases diagnosed are already metastatic. The 5-year survival rate for pre-metastatic diagnosis is 67%. Survival is highly variable based on stage during surgical intervention [3]. In most countries, where

screening programs for early detection of gastric cancer are neither in use nor practical because of low incidence, diagnosis is often made late in the disease course. Approximately 50% of patients present with advanced disease at diagnosis and will likely have a poor outcome [4]. Additionally, nearly 80% of patients have involvement of the regional lymph nodes and the number of positive lymph nodes has a profound influence on survival. In patients with localized resectable disease, outcome depends on the surgical stage of the disease [5]. Several factors have been noted to have a significant impact on the increased risk of developing GC, like family history, diet, alcohol consumption, smoking, Helicobacter pylori (H. pylori) infection and Epstein–Barr virus (EBV) infection [6]. Benign gastric diseases (BGDs) (gastric ulcer, gastritis and gastric polyp) are sharing GC in their symptoms, so it is urgent to discover novel non-invasive, sufficiently sensitive and specific biomarker for differentiating between GC and BGDs. [7]. Recently, the Kyoto classification of gastritis was developed based on the endoscopic characteristics of H. pylori infection-associated gastritis to diagnose H. pylori infection accurately and identify the risk factors of gastric carcinoma. Some endoscopic findings of gastritis including mucosal atrophy, intestinal metaplasia, enlarged folds, nodularity, and diffuse redness were recommended to be evaluated during oesophagogastroduodenoscopy (OGD) [8]. Previous studies have shown that the autonomic nervous system plays a role in the occurrence and development of GC. Currently, most of the studies on the autonomic nervous system and GC focus on a single species nerve fiber or transmitter [7]. The contribution of nerves to the pathogenesis of malignancies has emerged as an important component of the tumor microenvironment [9]. Nerves are traditionally regarded as transmission structures for electrical and chemical signals. However, increasing evidence has shown that they play an important role in tissue repair, regeneration and tumorigenesis. The interaction between nerves and cancer has been established in several tumor types [10]. The vagus nerve promotes the growth of GC through the high distribution of nerve fibers and the secretion of acetylcholine transmitters [7]. Pseudocholinesterase (ChE) is one of the serum proteins, also known as plasma cholinesterase or butyrylcholinesterase (BChE), is an alpha-glycoprotein, serine hydrolase present in almost all mammalian tissues with the highest levels detected in plasma and liver. ChE hydrolyzes acetylcholine and all chemicals containing ester bonds, of sufficient size to fit into the active site pocket. Moreover, ChE in the plasma serves as the first line of defense against toxic compounds reaching the blood stream that might inhibit acetylcholinesterase (AChE) activity. ChE sequesters (irreversibly binds) acetylcholinesterase inhibitors acting at the neuromuscular junction such as organophosphates, warfare nerve agents, and pesticides, thus protecting the nervous system before the poisons reach their target, i.e., synaptic AChE, and exert effects. From a clinical point of view, ChE is a perfect indicator of pesticide poisoning and nerve agent exposure because lowered ChE plasma activity is the first sign of the toxic effect of these compounds that is easy and convenient to monitor. The ChE is present in human plasma mainly as glycosylated soluble tetramers [11]. ChE is secreted into plasma immediately after synthesis in the liver [12]. The aim of this study is to determine the correlation between serum cholinesterase (pseudocholinesterase) activity and gastric cancer, in comparison to benign gastric diseases.

Subjects and Methods

Analytical case control study had been performed at Medical City Directorate\Gastroenterology and Hepatology Hospital and at Oncology Teaching Hospital between April 2022 and September 2022. The study analyzed the data of 50 patients, divided into two groups: 25 patients diagnosed with gastric adenocarcinoma and 25 patients diagnosed with benign gastric diseases (Gastritis, gastric polyp, gastric ulcer). Both groups were matched in their ages. Patient characteristics collected for analysis included age, gender (present pregnancy and contraceptives in females), history of resected gastric tumor, history of possible organophosphorus poisoning, history of chronic illness (liver cirrhosis, gammopathy), history of acute illness, history of myocardial infarction, past surgical history & medical history, number of chemotherapy cycles in patients with gastric cancer, date of first chemotherapy cycle, clinical staging and laboratory data (CEA, CA19-9 and AFP). All patients were informed about the nature of the study, and all who agreed to participate provided verbal informed consent and an accurate interview with each participant was conducted during their visit. The study was approved by the ethics committee of the institutions from which patients samples collected, stored and analyzed (Medical City Directorate\Gastroenterology and Hepatology Hospital, Oncology Teaching Hospital and The National Poisoning Inquisition and Poisoning Treatment Center laboratory at Ghazi Al-Hariri Surgical Specialties Hospital in Medical City Directorate).

Inclusion criteria: Involved patients either diagnosed by radiological and histological examinations with gastric adenocarcinomas and referred to Oncology Teaching Hospital in Medical City Directorate to receive treatment or complained from alarm features of dyspepsia (Unintentional weight loss, anaemia, persistent vomiting, haematemesis and/or melaena, dysphagia and palpable abdominal mass) who were diagnosed later by an endoscopic and histological examination as benign gastric diseases at Medical City Directorate\Gastroenterology and Hepatology Hospital. Age range for involved patients was 17-75 years, of them were 20 males and 30 females.

Exclusion criteria: Pediatrics age groups, patients with history of possible insecticide poisoning, patients already diagnosed with hepatitis, liver cirrhosis, myocardial infarction, Waldenstroms macroglobulinemia, atypical phenotypes of cholinesterase enzyme and any type of cancer other than GC.

Oesophago-Gastro-Duodenoscopy (OGD) was performed by one expert (gastroenterology specialist) to reduce inter-observer variation using the Olympus GIF-1100 and Pintax A-120109 scope to confirm the diagnosis of benign gastric diseases macroscopically according to findings of Kyoto classification and biopsy taken when necessary for further histological examination in association with patient clinical examination.

Five milliliters of non-fasting patient's peripheral venous blood were aspirated from the median cubital vein to measure serum cholinesterase (pseudocholinesterase) of each participant. The blood

samples were collected into disposable gel tubes and allowed to clot at room temperature for 30 minutes before separation of serum by centrifugation. Separated serum then stored in disposable plain tubes at ≤ -20 °C (according to manufacturer's instruction) at Oncology Teaching Hospital, the time from serum separation to storage did not exceeded two hours. At the end of the collection period, all samples were transferred simultaneously to the National Poisoning Inquisition and Poisoning Treatment Center laboratory at Ghazi Al-Hariri Surgical Specialities Hospital in Medical City Directorate for analysis of pseudocholinesterase by colorimetry based Kit on a biochemistry autoanalyzer. Centrifugation of samples containing precipitates before performing the assay was done according to the manufacturer's standard operating procedure (SOP).

Pseudocholinesterase (ChE) activity was determined by an assay based on the method published by Schmidt et al. using a kit provided by Roche diagnostics (Cholinesterase Gen.2), an in vitro test for the quantitative determination of pseudocholinesterase in human serum and plasma on Roche/Hitachi cobas c 311 system. The activity of pseudocholinesterase was measured by an autoanalyzer with wavelength (sub/main) 700/415 nm. Cholinesterase catalyzes the hydrolysis of butyrylthiocholine to thiocholine and butyrate. Thiocholine instantaneously reduces the yellow hexacyanoferrate (III) to the almost colorless hexacyanoferrate (II). This decrease in color can be measured photometrically.

Statistical Package for Social Sciences (SPSS) version 26 for Windows 10 (IBM, USA, 2019) and Microsoft Office Excel (2010) were used to analyze the data from this study. The continuous variable results were presented in form of mean ± standard deviation (m±SD). Frequencies and percentages were used to express categorical variables. Kolmogorov-Smirnov and Shapiro-Wilk tests are used to determine the normality distribution of data. Data analysis was done using chi-square test for homogeneity of variables. Levene's test was used for checking Equality of Variances between groups. Comparison of continuous variables was analyzed using Independent Samples t-test for normally distributed data. One Way Analysis Of Variance (ANOVA) is used to assay difference among studied subgroups. At the level of ($p \leq 0.01$) and ($p \leq 0.05$), the statistical significances were considered. Receiver operating curve (ROC) analysis was performed to calculate area under the curve (AUC), cut-off value, sensitivity and specificity. Positive predictive value (PPV), negative predictive value (NPV), and accuracy for the target marker were assessed by applying the marker specific cutoff value and then calculating them from a 2*2 truth table in the usual way that these values are defined.

Results

Analytical case control study was done to evaluate the utility of serum ChE as a biomarker for differentiation between GC and BGDs. It enrolled 50 patients (25 patients with gastric adenocarcinoma and 25 patients with BGDs). The age range of male patients involved in study was 40-75 years, mean ± SD of male was (57.45±10.49), and age range of female patients involved in study was17-71 years , mean ± SD of female was 45.67±16.94). Chi-square test was used to check the difference in gender between cancer and benign groups. No significant difference was found in gender proportion ($p = 0.083$).

Independent samples t-test was used to find the difference of serum ChE level in GC and BGDs. In the GC group, there were 12 females and 13 males aged from 24 to 75 years old (the mean age, 56.20 ± 12.37). In the BGDs group, there were 7 males and 18 females aged from 17 to 75 years old (the mean age, 44.56 ± 16.71). The mean value of serum ChE level was 5339.28 U/L in the GC group, whereas in the BGDs group the mean value of serum ChE was 7516.92 U/L, with a statistically significant difference between the two groups ($p < 0.001$) as presented in Table 1.

Table 1: The difference of serum ChE level in GC and BGDs. *

Group	Number of cases	Serum ChE level (U/L) (mean±SD)	p- value
Gastric cancer	25	5339.28 ± 1815.68**	
Benign gastric diseases	25	7516.92 ± 2351.12	0.000

*Independent samples t-test was used

**P ≤ 0.001 is statistically significant compared with the BGDs group. SD (standard deviation)

To further investigate whether there is a difference of ChE level between early grade II GC and BGDs, comparison of ChE level in patients with grade II GC and patients with BGDs was done. Gastric cancer group was subdivided according to tumor grade into three groups, grade I could not be obtained in this study. Six patients (24%) were grade II, fourteen patients (56%) were grade III and five patients (20%) were grade IV. The mean± SD of serum ChE level in grade II GC group was 4676.17± 1547.04 U/L, and it was 7516.92± 2351.12 U/L in the BGDs group, which was higher than the former with a statistically significant difference ($p \leq 0.01$) as presented in Table 2.

Table 2: The difference of ChE level in grade II GC and BGDs. *

Group	Number of cases	Serum ChE level (U/L) (mean±SD)	p- value
Gastric cancer (Grade II)	6	4676.17 ±1547.04	0.009**
Benign gastric diseases	25	7516.92 ±2351.12	

* Independent samples t-test was used.

**-Correlation is significant at P≤ 0.01 level (2-tailed)

To further investigate whether there is a difference of ChE level between early stage I GC and BGDs, comparison of ChE level in patients with Stage I GC and patients with BGDs. was done. Gastric cancer group was subdivided according to tumor stage into four groups. Four patients (16%) were stage I, nine patients (36%) were stage II, six patients (24%) were stage III and six patients (24%) were stage IV. The mean of serum ChE level in Stage I GC group was 4763.00± 1226.58 U/L, and it was 7516.92± 2351.12 U/L in the BGDs group, which was higher than the former, with significant statistical difference between groups ($P<0.05$) Table 3.

Table 3: The difference of ChE level in early stage I GC and BGDs.*

Group	Number of cases	Serum ChE level (U/L)(mean±SD)	p- value
Gastric cancer (Stage I)	4	4763.00±1226.58	
Benign gastric diseases	25	7516.92±2351.12	0.03**

* Independent samples t-test was used.
** Correlation is significant at P ≤ 0.05 level.

Mean ± SD of number of chemotherapy cycles taken by patients with gastric adenocarcinoma were (8.08±4.96). As ChE level was found to be normally distributed, One-Way Analysis Of Variance (ANOVA) test was used to compare the difference in serum ChE among the GC subgroups (I, II, III, IV) divided according to chemotherapy cycles number, with no significant statistical difference between subgroups (p=0.49) Table 4.

Table 4: Gastric cancer subgroups according to number of chemotherapy cycles received and the difference in serum ChE level. *

Gastric cancer subgroups	Number of chemotherapy cycles received	Patients number and percentage	Serum ChE level (U/L) (mean±SD)	F	p-value
I	0-4	7 (28%)	5675.86±1561.11		
II	5-9	7 (28%)	5202.86±1481.81		
III	10-14	8 (32%)	4713.00±2324.45	0.83	0.49
IV	15-19	3 (12%)	6542.33±1596.74		

* One Way Analysis Of Variance (ANOVA) test was used.

The diagnostic value of ChE in differentiating between GC and BGDs is analyzed by applying Receiver Operating Curve (ROC) analysis. Area under the curve (AUC) was 0.756 (95% CI 0.624 – 0.888, p = 0.002). ROC curve analysis suggested that with the optimal cutoff value of 5568 U/L, ChE could produce 60% sensitivity and 80% specificity in differentiating between GC and BGDs. Positive predictive value [PPV] = 75%, negative predictive value [NPV] = 66.6%) and test accuracy =70% as is shown in Figure1.

Discussion

ChE is an enzyme which hydrolyses acetylcholine. There are two types of cholinesterase in the mammalian system, the true cholinesterase or acetylcholinesterase (AChE) and pseudocholinesterase (ChE). ChE is also known as serum cholinesterase or butrylcholinesterase (BChE) [13]. True cholinesterase is found in the central nervous system, muscles, and in erythrocytes. Pseudocholinesterase is an alpha-glycoprotein found in the central and peripheral nervous system, in most tissues, and in liver [14]. Serum ChE level is decreased in several conditions such as acute and chronic liver damage, cirrhosis, inflammation and organophosphate exposure [13]. Therefore, liver diseases, severe infection, and organophosphorus poisoning were excluded in this study. There is growing evidence suggesting that ChE is involved in the regulation of cell proliferation, apoptosis, differentiation, and cell-cell interaction [15]. The non-classical roles of ChE have been implicated in modulating cancer growth. Reduced circulating levels of ChE as compared with normal controls were observed in many malignancies, such as colorectal, pancreatic, bladder and prostate cancers [13,16,17]. Although the involvement of ChE in tumorigenesis remains unclear, findings indicated that high ChE was capable of reducing cell differentiation and inhibiting signal transduction.

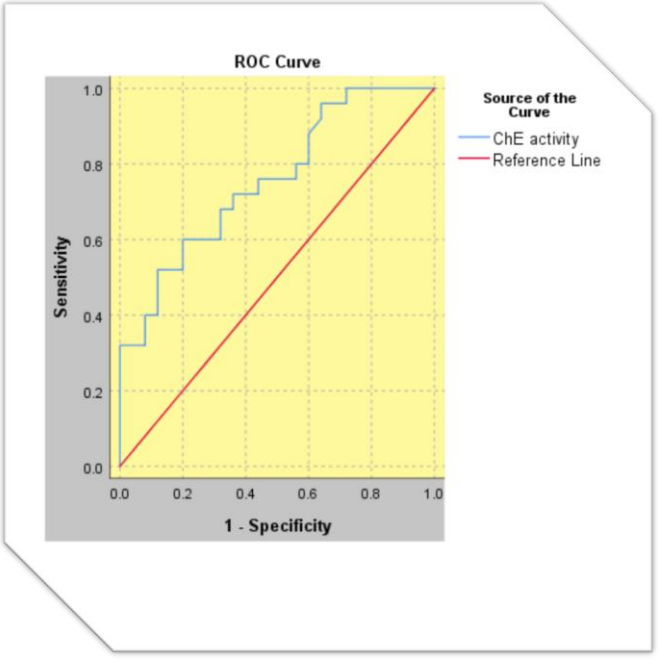

Figure 1: ROC curve for ChE in differentiating between GC and BGDs. AUC for ChE was 0.756 (95% CI 0.624 – 0.888 , p = 0.002)

Another study indicated that decreased expression of the ChE protein might contribute to tumorigenesis [13]. This study showed a statistically significant difference in serum ChE between gastric cancer patients and benign gastric diseases group (p-value< 0.001), which is consistent with the findings of Gao et al. [13] who found statistically significant difference in serum ChE between the cancer and benign groups (p<0.001). Also they compared ChE levels between stage I GC and benign gastric diseases and found significant differences between means and this agreed with present findings (p<0.05). Also significant statistical difference found in this study when compare grade II GC (Grade I GC could not be obtained in this study) with benign gastric diseases (p<0.01). Another authors Gu et al. [18] who found that the serum ChE activity in the GC group was significantly lower than that in the non-malignant tumor group (p=0,0003). However, this study is different from theirs in some aspects. First, the control group in this study includes patients with benign gastric diseases (gastritis, gastric ulcers and gastric polyp), while the control group in their study includes patients with non-malignant tumors; Second, the GC group in this study was subdivided into subgroups according to different TNM stages and grades while the GC group in their study was not. Furthermore, comparison TNM Stage I and grade II GC with benign gastric diseases was done and found that ChE level of Stage I and grade II GC groups was significantly lower than that of benign diseases group, indicating that ChE might differentiate between benign gastric diseases and the early stages of GC. Third, this study focused on the role of ChE in differentiating between gastric cancer and benign gastric diseases. Moreover, a study by Chougule et al. [19] observed that the ChE levels were lower (31-49% of normal value)

in all patients with malignancies. And the patients with no detectable/visible disease activity at 6 months follow-up showed ChE values in the normal range. They also found that the serum ChE levels start increasing as radiotherapy progresses. These findings are in line with those obtained by this study to some extent which showed highest ChE level in group IV GC as chemotherapy cycles progress to more than 15 cycles. A possible explanation is that the low levels of ChE in gastric cancer patients come from the high distribution of nerve fibers and the secretion of acetylcholine transmitters that is promoted by the vagus nerve [13]. Another possible mechanism responsible for low ChE activity in cancer patients could be secondary to anorexia accompanying malignancy due to chronic underfeeding [20]. To further evaluate the diagnostic value of ChE in differentiating between GC and benign gastric diseases, ROC curve for ChE was generated. Results showed that ChE had the AUC (0.756). With the optimal cutoff value of 5568 U/L, ChE produced 60% sensitivity, 80% specificity, indicating that ChE might become a suitable biomarker for differentiating between GC and benign gastric diseases. No statistical difference in gender proportion between groups that observed in this study lead to decrease limitations caused by gender variation.

Conclusion

Serum cholinesterase can be regarded as a potential simple, rapid, convenient, non-invasive and inexpensive biomarker for differentiating between gastric cancer and benign gastric diseases. Measurement of serum cholinesterase will help physicians to predict the probability of gastric cancer and to determine whether further invasive examinations should be performed.

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Conflict of Interest

Authors declare no conflict of interest.

Data availability

Data are available upon reasonable request.

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