Research Square

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Apolipoprotein-B Levels May Have Role In Predicting The Risk For Acute Pancreatitis in Patients With Hypertriglyceridemia

Ceyda Dincer Yazan

dincer.ceyda@gmail.com

Marmara University School of medicine

Dilek Gogas Yavuz

Marmara University School of medicine

Research Article

Keywords: pancreatitis, hypertriglyceridemia

Posted Date: September 20th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10541675/v1

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Additional Declarations: No competing interests reported.

Abstract

Background: Hypertriglyceridemia is associated with serious complications, including acute pancreatitis and cardiovascular disease. This study aimed to evaluate the clinical characteristics and laboratory parameters associated with the risk of acute pancreatitis in patients referred to our endocrinology clinic for hypertriglyceridemia.

Methods: Patients with serum triglyceride levels >500 mg/dL who were admitted to the endocrinology outpatient clinic between 2015 and 2020 were retrospectively evaluated. Clinical characteristics and laboratory data of patients with and without a history of acute pancreatitis were obtained from medical records.

Results: A total of 82 patients with hypertriglyceridemia (35 females and 47 males; mean age, 45.4 ± 11.2 years) were included. Thirty-one patients (37.8%) had experienced at least one episode of acute pancreatitis. Fifty patients had triglyceride levels between 500 and 1,000 mg/dL, whereas 32 had severe hypertriglyceridemia. Non-high-density lipoprotein cholesterol (non-HDL-C) levels were significantly higher in patients with severe hypertriglyceridemia than in those with triglyceride levels between 500 and 1,000 mg/dL ($p = 0.001$). The median triglyceride level was significantly higher in patients with a history of acute pancreatitis than in those without ($p = 0.004$). Four of the five patients classified as having familial chylomicronemia syndrome (FCS) and four of the six patients classified as having either FCS or partial lipoprotein lipase deficiency had a history of acute pancreatitis. Receiver operating characteristic (ROC) analysis identified a triglyceride cut-off value of 980 mg/dL (AUC = 0.68) and a TG/ApoB ratio cut-off value of 8.5 (AUC = 0.73) for predicting acute pancreatitis. At least one secondary factor contributing to hypertriglyceridemia was identified in 59 patients (71.9%).

Discussion and conclusion: Triglyceride levels ≥1,000 mg/dL were associated with an increased risk of acute pancreatitis, consistent with previous reports. Classification of the dyslipoproteinemia phenotype may help identify patients at higher risk for acute pancreatitis. Measurement of apolipoprotein B (ApoB) and the TG/ApoB ratio may provide additional value in predicting pancreatitis risk in patients with hypertriglyceridemia. Further prospective studies with larger cohorts are needed to validate these findings.

Introduction

Severe hypertriglyceridemia, defined as a serum triglyceride level ≥ 1,000 mg/dL, is a well-recognized cause of acute pancreatitis (1, 2). It is closely associated with obesity, type 2 diabetes mellitus, and metabolic syndrome and is also linked to an increased risk of cardiovascular disease. Cardiovascular risk appears to increase progressively with rising triglyceride levels up to approximately 1,000 mg/dL (3, 4).

Hypertriglyceridemia may result from excessive alcohol consumption, medications (including thiazide diuretics, β-blockers, estrogens, antiretroviral agents, and immunosuppressive drugs), or secondary

conditions such as diabetes mellitus, chronic kidney disease, liver disease, and pregnancy (5). It may also have a genetic basis, including familial hypertriglyceridemia (Fredrickson type IV), familial chylomicronemia syndrome (Fredrickson type I), familial hypoalphalipoproteinemia, familial combined hyperlipidemia (Fredrickson type V), and dysbetalipoproteinemia (Fredrickson type III) (6–9). Patients with Fredrickson type I, IV, and V dyslipoproteinemia are at increased risk of acute pancreatitis (4).

There are no specific evidence-based guidelines for the management of hypertriglyceridemia-associated acute pancreatitis. Initial treatment consists of fasting, aggressive intravenous fluid resuscitation, and glycemic control, including insulin infusion in patients with diabetes mellitus (3). Therapeutic plasma exchange (plasmapheresis) may be considered in selected patients with very severe hypertriglyceridemia who do not respond adequately to conventional medical therapy during an acute pancreatitis episode. However, it is not recommended as first-line treatment because its beneficial effects on mortality and morbidity have not been consistently demonstrated (10).

Hypertriglyceridemia is a well-established cause of acute pancreatitis, accounting for up to 10% of cases (11). Approximately 10–20% of patients with severe hypertriglyceridemia experience at least one episode of acute pancreatitis (12, 13). Although the triglyceride threshold associated with an increased risk of acute pancreatitis remains controversial, several studies have shown that the risk begins to increase at serum triglyceride levels above 500 mg/dL (3, 4). In one study, the median triglyceride level among patients with hypertriglyceridemia-associated acute pancreatitis was 2,622 mg/dL (14). The clinical presentation of hypertriglyceridemia-associated acute pancreatitis is generally similar to that of pancreatitis due to other etiologies; however, several studies suggest that it is associated with greater disease severity (11, 12). Novel therapeutic agents targeting severe hereditary forms of hypertriglyceridemia are currently under clinical development, particularly for patients with recurrent episodes of acute pancreatitis (3, 4). Therefore, identifying patients at high risk of pancreatitis who are most likely to benefit from these emerging therapies is of considerable clinical importance.

Recent studies have evaluated lipid subfractions in patients with hypertriglyceridemia. Apolipoprotein B (ApoB) has been suggested as a potential marker for identifying patients at increased risk of acute pancreatitis (15). In addition, another study reported that lower serum apolipoprotein A1 (ApoA1) levels were associated with greater severity of acute pancreatitis (16). Furthermore, measurement of triglyceride (TG), total cholesterol (TC), and the TG/ApoB ratio may facilitate the classification of dyslipoproteinemia phenotypes and help identify patients at higher risk of cardiovascular disease or acute pancreatitis (17).

The present study aimed to evaluate the clinical characteristics of patients with hypertriglyceridemia followed at our endocrinology clinic and to investigate whether lipid subfraction parameters, particularly ApoB and the TG/ApoB ratio, provide additional predictive value for identifying patients at increased risk of acute pancreatitis.

Material and Method

Eighty-two patients with moderate to severe hypertriglyceridemia who were followed at a tertiary clinic between 2015 and 2020 were retrospectively evaluated. Patients with serum triglyceride levels ≥ 500 mg/dL were eligible for inclusion.

Demographic and clinical data, including a history of acute pancreatitis, cardiovascular disease, comorbidities, medication use, and body mass index (BMI), were obtained from the medical records. The following laboratory parameters were recorded, when available: fasting plasma glucose, glycated hemoglobin (HbA1c), thyroid-stimulating hormone (TSH), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides (TG), apolipoprotein A1 (ApoA1), and apolipoprotein B (ApoB).

Patients were classified into dyslipoproteinemia phenotypes according to the algorithm proposed by Sniderman *et al.* based on TG, ApoB, the TG/ApoB ratio, and the TC/ApoB ratio (17).

Serum glucose levels were automatically measured by the hexokinase method (AU5800, Beckman Coulter, USA). Hba1c percentages were calculated in EDTA whole blood samples by boranetafinite chromatography (TrinityBiotech Ireland). TSH was automatically studied by the paramagnetic particle chemiluminescence immunoassay method in serum samples (DX1800, Beckman Coulter, USA). HDL, Triglyceride, and total cholesterol were analyzed using the spectrophotometric method (AU680, Beckman Coulter, USA). Non-HDL cholesterol was calculated as total cholesterol minus HDLc. APO-A and APO-B were reviewed by nephelometry (Beckman Coulter Image, USA). The presence of chylomicrons was determined by centrifugation after blood sampling. After overnight storage at 4oC, the presence of chylomicrowasaswas determined by photographing the tubes in front of a white paper for the creamy supernatant layer (13).

The statistical evaluation was done with the SPSS statistic program. Descriptive analysis, and patient number, frequencies, mean, median, standard deviation, and interquartile range (IQR) were calculated. Continuous variables with normal distribution were shown as mean,±standard deviation and compared using Student's test-test. Continuous variables without normal distribution were established as the median and interquartile range (25%-75%) and compared with the Mann-Whitney U test. All p values under 0.05 were considered statistically significant.

The study protocol was approved by the Marmara University Faculty of medicine local ethics committee with a number of 09.2021.80.

Results

A total of 82 patients with hypertriglyceridemia (35 females and 47 males; mean age, 45.4 ± 11.2 years) were included in the study. The clinical characteristics and laboratory findings of the study population are summarized in Table 1. Thirty-one patients (37.8%) had experienced at least one episode of acute pancreatitis, whereas 51 (62.2%) had no history of acute pancreatitis. The mean body mass index (BMI) was 27.8 ± 4.74 kg/m².

Table 1
Clinical and laboratory parameters of patients according to serum triglyceride levels

Age (years)	**Total (n:82)**	**TG 500–1000 mg/dl (n:50)**	**TG > 1000 mg/dl (n:32)**	**P value**
	45.4±11.2	**45.2±11.5**	**45.1±10.6**	**0.706**
Sex (F/M)	35/47 (42.6%/57.3%)	22/28 (44%/56%)	13/19 (40.6%/59.3%)	0.472
BMI (kg/m^2)	27.8±4.74	28.8±5.2	26.7±3.9	0.193
History of Pancreatitis (n)	31 (37.8%)	12 (24%)	19 (59.3%)	**0.001**
CVD (n)	11 (13.4%)	7 (14%)	4 (12.5%)	0.418
Type 2 Diabetes (n)	41 (50%)	26 (52%)	14 (43.75%)	0.308
Obesity (n)	23 (28%)	17 (34%)	6 (18.75%)	0.110
Glucose (mg/dl) (mean±SD)	142.5±77.6	145.5±74.8	137±82.8	0.666
Hba1c (%) (mean±SD)	7.2±2.38	7.45±2.55	6.95±2.09	0.476
APO A (mg/dl) (median)	116 (IQR:103–134)	119.8 (IQR:107–134)	108.9 (IQR:96–150)	0.389
APO B (mg/dl) (median)	115 (IQR:99.5-139.5)	114.5 (IQR:102.2–134)	127 (75–150)	0.921
TSH mU/L (median)	1.84 (IQR:1.3–2.92)	2 (IQR:1.43–2.97)	1.51 (IQR:1.04–2.82)	0.169
Non-HDLc (mg/dl) (median)	247 (IQR:193–337)	221.5 (IQR:170.7-260.5)	370.5 (IQR:248–612)	**0.001**
HDLc (mg/dl) (median)	40 (IQR:30-53.5)	39.5 (IQR:31–45)	44 (IQR:24–131)	**0.471**

Secondary factors contributing to hypertriglyceridemia were identified from the medical records. Six patients had a history of cholelithiasis, six reported excessive alcohol consumption, seven were receiving thiazide diuretics, and five were using β-blockers. Overall, 23 patients had no identifiable secondary cause after evaluation for diabetes mellitus, alcohol use, medication use, hypothyroidism, and obesity.

Among the study population, 26 patients had hypertension, 41 had type 2 diabetes mellitus, 23 had obesity, and 11 had coronary artery disease as comorbid conditions associated with hypertriglyceridemia.

Regarding lipid-lowering therapy, 20 patients were receiving fenofibrate monotherapy, 17 were receiving combination therapy with fenofibrate and eicosapentaenoic acid/docosahexaenoic acid (EPA/DHA), four were receiving statin therapy, and two were treated with gemfibrozil.

Fifty patients had serum triglyceride levels between 500 and 1,000 mg/dL, 19 had levels between 1,000 and 2,000 mg/dL, and 13 had levels > 2,000 mg/dL. Patients were subsequently categorized into two groups according to triglyceride levels (< 1,000 mg/dL and ≥ 1,000 mg/dL). Among the laboratory parameters evaluated, only non-high-density lipoprotein cholesterol (non-HDL-C) levels differed significantly between the two groups, with significantly higher non-HDL-C levels observed in patients with triglyceride levels ≥ 1,000 mg/dL ($p = 0.001$). The clinical characteristics and laboratory findings of patients with moderate (< 1,000 mg/dL) and severe (≥ 1,000 mg/dL) hypertriglyceridemia are summarized in Table 1.

According to the dyslipoproteinemia classification algorithm proposed by Sniderman *et al.* (17), 30 patients were classified as having familial combined hyperlipidemia. Eleven patients were classified as having familial hypertriglyceridemia and/or secondary hypertriglyceridemia associated with metabolic syndrome, obesity, diabetes mellitus, or partial lipoprotein lipase (LPL) deficiency. Eight patients were classified as having familial dysbetalipoproteinemia (Fredrickson type III), hepatic lipase deficiency, or secondary dyslipoproteinemia associated with diabetes mellitus, obesity, or metabolic syndrome. Five patients were classified as having complete lipoprotein lipase deficiency (familial chylomicronemia syndrome) or apolipoprotein C-II deficiency. Six patients were classified as having either familial chylomicronemia syndrome or partial lipoprotein lipase deficiency in the presence of secondary contributing factors. Other 22 patients could not be classified because of insufficient data.

Patients were stratified according to their history of acute pancreatitis, and their clinical characteristics and laboratory findings are summarized in Table 2. The median serum triglyceride level was significantly higher in patients with a history of acute pancreatitis than in those without ($p = 0.004$). Despite lipid-lowering treatment, 61.2% of patients with a history of acute pancreatitis continued to have severe or very severe hypertriglyceridemia during follow-up.

Table 2
Clinical and laboratory parameters of patients with pancreatitis and without pancreatitis history

Age (years)	**Patients with pancreatitis history (n:31)**	**Patients without pancreatitis history (n:51)**	**P value**
	44.2±9.52	**46.1±12.2**	**0.490**
Sex (n)	F/M: 12/19	F/M: 23/28	0.571
BMI (kg/m^2)	26.9±3.93	28.4±5.19	0.342
Triglyceride (mg/dl) (median)	1137 (IQR:656–1870)	671 (IQR:557–1341)	**0.004**
TG 500–999 mg/dl	12 (38.7%)	38 (74.5%)	-
TG 1000–2000 mg/dl	12 (38.7%)	7 (13.7%)	-
TG≥2000 mg/dl	7 (22.5%)	6 (11.7%)	-
Pancreatitis attack number	2.8±2.3 (mean)	-	-
Plasmapheresis (n)	13 (41.9%)	-	-
Diabetes (n)	15 (48.4%)	25 (48.1%)	0.956
CVD (n)	7 (22.5%)	4 (7.8%)	-
Obesity (n)	8 (25.8%)	15 (28.8%)	0.862
Glucose (mg/dl) (mean±SD)	147.9±93.2 mg/dl	139.1±67.1 mg/dl	0.625
Hba1c (%) (mean±SD)	7.42±2.49%	7.2±2.36%	0.749
Non-HDLc (mg/dl) (median)	267 (IQR:222–482)	233 (IQR:187–294)	0.068
HDLc (mg/dl) (median)	37.5 (IQR:29-81.5)	41 (IQR:31–50)	0.371
APO A (mg/dl) (median)	108.9 (IQR:98–129)	121 (IQR:106.5-137.5)	0.173
APO B (mg/dl) (median)	111 (IQR:101–138)	121.5 (IQR:95.2-141.5)	0.679
TSH mu/L(median)	1.79 (IQR:1.25–3.36)	1.85 (IQR:1.27–2.79)	0.817

Among the 31 patients with a history of acute pancreatitis, 21 experienced recurrent episodes, with a total of 93 pancreatitis episodes recorded. Receiver operating characteristic (ROC) curve analysis was performed to evaluate the predictive value of lipid subfraction parameters for acute pancreatitis. The ApoB/ApoA1 ratio showed no significant predictive value, with an optimal cut-off value of 1.52 (AUC = 0.56, p = 0.43). In contrast, serum triglyceride levels and the TG/ApoB ratio demonstrated significant

predictive performance. A triglyceride cut-off value of 980 mg/dL yielded an AUC of 0.68, with a sensitivity of 61% and a specificity of 74% (p = 0.0027). The TG/ApoB ratio showed better discriminatory performance, with a cut-off value of 8.5 corresponding to an AUC of 0.736, a sensitivity of 64%, and a specificity of 75% (p = 0.0004) (Fig. 1).

Among the 31 patients with a history of acute pancreatitis, 11 were classified as having familial combined hyperlipidemia (including four with type 2 diabetes mellitus and two with obesity), five as familial hypertriglyceridemia, four as familial chylomicronemia syndrome (FCS), four as either familial chylomicronemia syndrome or partial lipoprotein lipase (LPL) deficiency, and two as dysbetalipoproteinemia.

Among the 21 patients with recurrent episodes of acute pancreatitis, six were classified as having familial combined hyperlipidemia, five as familial hypertriglyceridemia, and four as familial chylomicronemia syndrome.

Eleven patients had coronary artery disease, of whom five were classified as having familial combined hyperlipidemia.

The presence of chylomicronemia was assessed in 58 patients by visual inspection of serum after centrifugation. A creamy supernatant was observed in 17 patients, a turbid appearance in 12 patients, and a clear serum appearance in 29 patients. Among patients with a history of acute pancreatitis, 12 had a creamy supernatant, six had a turbid appearance, and seven had clear serum after centrifugation.

The mean serum triglyceride level was 2,328 ± 1,987 mg/dL in patients with a creamy supernatant, 1,956 ± 1,655 mg/dL in those with a turbid appearance, and 721 ± 288 mg/dL in those with clear serum. All five patients classified as having familial chylomicronemia syndrome (FCS) exhibited a creamy supernatant after centrifugation.

Eighteen patients were admitted to our emergency department with acute pancreatitis and had complete laboratory and radiological data available during the acute episode. The mean serum triglyceride level at presentation was 2,348 ± 1,746 mg/dL. Computed tomography findings were consistent with acute pancreatitis in all patients.

Seven patients underwent therapeutic plasma exchange (plasmapheresis) during the acute episode. All of these patients were classified as having severe acute pancreatitis according to the Ranson criteria. Therapeutic plasma exchange was completed without procedure-related complications in all cases. Chylomicronemia was present in eight patients during the acute pancreatitis episode. All 18 patients recovered without major complications and were discharged from the hospital.

Discussion

In the present study, we evaluated patients referred to our endocrinology clinic for hypertriglyceridemia and found that 37.8% had experienced at least one episode of acute pancreatitis. Among these patients,

35.5% had serum triglyceride levels > 2,000 mg/dL at presentation to the emergency department. Only three patients had moderate hypertriglyceridemia at the time of the acute pancreatitis episode, whereas the remaining patients had severe or very severe hypertriglyceridemia, consistent with previous reports (10). Furthermore, 21 patients experienced recurrent episodes of acute pancreatitis.

Previous studies have reported that approximately 20% of patients with severe hypertriglyceridemia develop acute pancreatitis (10, 11). Although the risk of acute pancreatitis appears to increase at triglyceride levels above 500 mg/dL, patients with moderate hypertriglyceridemia should be carefully evaluated for secondary etiologies, as the causal relationship between moderate hypertriglyceridemia and acute pancreatitis is less well established than that observed with severe hypertriglyceridemia (10, 18–21).

In our cohort, the optimal triglyceride cut-off value for predicting acute pancreatitis was 980 mg/dL, which is consistent with previous studies. Moreover, our findings suggest that ApoB provides additional predictive value beyond serum triglyceride levels alone. The TG/ApoB ratio demonstrated better discriminatory performance for predicting acute pancreatitis than triglyceride levels alone, as reflected by a higher AUC. This finding is consistent with the only previous study evaluating the role of ApoB in predicting pancreatitis risk in patients with hypertriglyceridemia (15).

Unlike most previous studies, our patients were classified according to the dyslipoproteinemia algorithm proposed by Sniderman *et al.*, which incorporates serum TG, total cholesterol, ApoB, the TG/ApoB ratio, and the TC/ApoB ratio to define dyslipoproteinemia phenotypes (17). Among the five patients classified as having FCS, four had a history of acute pancreatitis and recurrent episodes. Similarly, four of the six patients classified as having either FCS or partial LPL deficiency had experienced acute pancreatitis. In contrast, among the 11 patients with coronary artery disease, five were classified as having familial combined hyperlipidemia, which is considered one of the most atherogenic dyslipoproteinemia phenotypes (17).

These findings suggest that dyslipoproteinemia classification based on TG, TC, and ApoB measurements may represent a practical approach for identifying patients at increased risk of acute pancreatitis or cardiovascular disease and may facilitate a more individualized management strategy according to the underlying dyslipoproteinemia phenotype (17). However, only 60 of the 82 patients in our cohort could be classified because complete lipid subfraction data were not available for all participants. Therefore, larger prospective studies are warranted to further clarify the role of ApoB and dyslipoproteinemia phenotyping in predicting the risk of acute pancreatitis and guiding clinical management.

In the present study, 71.9% (n = 59) of the patients had at least one identifiable secondary factor contributing to hypertriglyceridemia. This finding is consistent with the results of a nationwide survey conducted in Japan, in which secondary causes were identified in 74% of patients with hypertriglyceridemia (22).

Hypertriglyceridemia is known to be closely associated with metabolic disorders, particularly type 2 diabetes mellitus and obesity (3). In our cohort, approximately half of the patients had type 2 diabetes mellitus, and 28% were obese. Previous studies have shown that patients with hypertriglyceridemia typically have reduced HDL-C levels. Although low HDL-C was previously considered a major contributor to the increased cardiovascular risk associated with hypertriglyceridemia, accumulating evidence suggests that non-HDL-C and ApoB, which better reflect the burden of atherogenic lipoproteins, are more informative predictors of cardiovascular risk (3).

Because the number of patients with established cardiovascular disease in our cohort was relatively small, we were unable to adequately evaluate the associations of ApoB and non-HDL-C levels with cardiovascular outcomes. Further studies with larger cohorts are needed to clarify the prognostic value of these lipid parameters in patients with hypertriglyceridemia.

Conclusion

Triglyceride levels ≥ 1,000 mg/dL are associated with an increased risk of acute pancreatitis. Patients with hypertriglyceridemia should be carefully evaluated for secondary causes, and modifiable risk factors should be identified and appropriately managed. Although current evidence has not consistently demonstrated increased mortality in hypertriglyceridemia-associated acute pancreatitis, several studies suggest that these patients experience more severe disease than those with acute pancreatitis of other etiologies. Dyslipoproteinemia phenotyping may help identify patients at increased risk of acute pancreatitis and facilitate risk stratification. Furthermore, ApoB measurement, particularly in combination with the TG/ApoB ratio, may provide additional predictive value for identifying patients at higher risk of acute pancreatitis. Larger prospective studies are needed to validate these findings and determine the clinical utility of ApoB-based risk assessment.

Declarations

Consent to participate

The requirement for informed consent was waived due to the retrospective nature of the study.

Conflict of interest:

The authors clarify that there is no conflict of interest to declare

Funding:

There is no funding to declare.

Author Contribution

Author contribution: The study was designed by Ceyda Dincer Yazan and Dilek Gogas Yavuz. The data was recorded by Ceyda Dincer Yazan and statistics and evaluation were done by Ceyda Dincer Yazan and Dilek Gogas Yavuz. All authors read and approved the final version of the manuscript.

Data availability:

The data of this study can be reached upon request from corresponding author.

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Figures

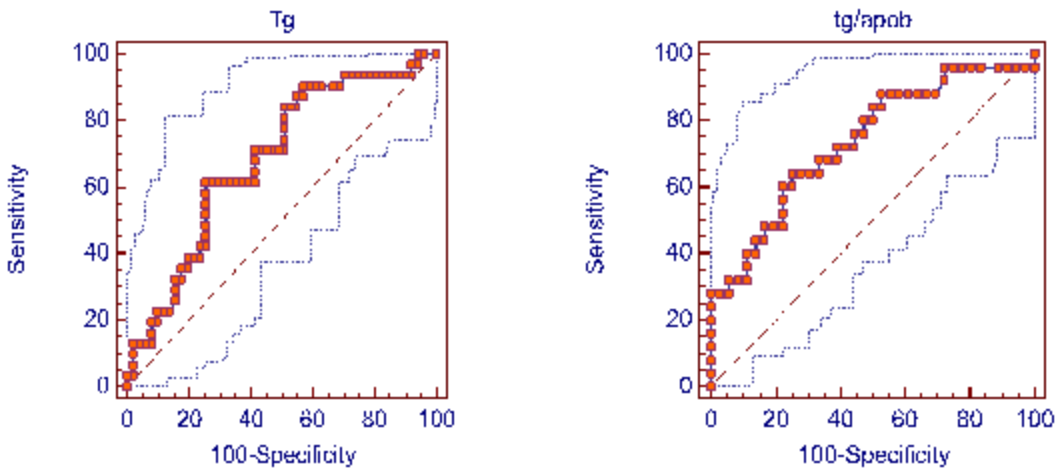


Figure 1

Specificity and sesitivity of tg and tg/apob for pancreatitis