ORIGINAL ARTICLE

Does esophageal dysfunction affect the course of treadmill stress test in patients with recurrent angina-like chest pain?

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KEY WORDS

coronary artery disease, esophageal manometry, pH-metry, recurrent chest pain, treadmill stress test

ABSTRACT

INTRODUCTION Cardioesophageal reflex may increase severity of chest pain and signs of myocardial ischemia on electrocardiogram (ECG), both in patients with and without significant coronary artery stenosis.

OBJECTIVES The aim of the study was to evaluate the relationships between esophageal pH and pressure and clinical and electrocardiographic signs of myocardial ischemia.

PATIENTS AND METHODS In 129 consecutive patients with recurrent chest pain, 77 without significant coronary artery lesions in coronary angiography and 52 with myocardial ischemia, panendoscopy, pH-metry, manometry, and treadmill stress test were performed.

RESULTS The prevalence of esophageal disorders was similar in patients with and without significant coronary artery narrowing. Subjects with significant ST interval depression in the stress test had a higher rate of simultaneous esophageal contractions. There were no differences in the results of the treadmill test between patients with and without esophageal disorders. Forty percent of patients with significant coronary artery lesions, who had to stop the test because of chest pain, did not present significant ST interval depression on ECG; however, such depression was observed in 60% of patients with normal coronary angiography. Patients with exercise-provoked chest pain had more pronounced abnormalities in esophageal pH, together with the amplitude and coordination of esophageal contractions. Demographic and clinical factors associated with chest pain and changes in exercise ECG were not evaluated.

CONCLUSIONS Esophageal disorders are an important cause of chest pain, potentially affecting the results of the treadmill stress test. However, further research is needed to determine the predictors of the cardioesophageal loop activity.

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INTRODUCTION The association between esophageal and cardiac function has been known since the 18th century.[1] It results from intrathoracic location, common risk factors, and the activity of vagal reflexes. The latter neural mechanism may cause impairment of myocardial perfusion after esophageal mucosa exposition to acid and a decrease in lower esophageal sphincter (LES) pressure in response to myocardial receptor activation by the products of anaerobic metabolism (vicious circle mechanism).[1-3] The above associations have been observed in about 56% of subjects with and without obstructive lesions in coronary angiogram.[2] The clinical symptoms include noncardiac chest pain mimicking angina pectoris (angina-like chest pain), cardiac chest pain due to impairment of myocardial perfusion, and cardiac arrhythmia.[1,4,5] Treatment of esophageal dysfunction may reduce severity of these symptoms.[4-6]

The effect of esophageal acidification on electrocardiogram (ECG)[1,6] and the results of the treadmill stress test[4,5] has been studied previously.

Makk et al.[7] showed the presence of esophageal motility disorders during coronary angioplasty; Stec et al.[8] revealed esophageal dysmotility induced by premature ventricular beats; and Krysiak et al.[9] observed hiccups as a symptom of myocardial ischemia. Their observations could be explained by the activation of cardioesophageal reflex. However, clinical associations between the results of the treadmill stress test and esophageal motility have not been investigated so far. To my knowledge, the present study has been the first to assess these associations.

PATIENTS AND METHODS The analysis was performed in 129 consecutive patients admitted to hospital due to recurrent angina-like chest pain (defined as precordial symptoms induced by exercise and receding after rest or administration of nitroglycerin) in order to undergo a series of diagnostic tests. A cardiologist described the recurrent symptoms as noncardiac because they were resistant to treatment aimed at coronary reserve improvement and empirical therapy with proton pump inhibitors (PPI); patients were referred to a gastroenterologist. An elective coronary angiogram was performed for each patient prior to gastroenterological procedures. The group comprised 52 patients (40%) who had significant angiographic changes with more than 50% of narrowed coronary vessels but not requiring revascularization using invasive procedures, and 77 patients (60%) with a normal coronary angiogram and no obstructive lesions. Patients with coronary artery disease (CAD) and those without significant coronary artery stenosis had similar demographic and clinical characteristics, except for those concernig the course of CAD (TABLE 1).

The inclusion criteria were as follows: age between 40 and 70 years, prior coronary angiography, angina-like chest pain (class II or III according to the Canadian Cardiovascular Society), and persistent symptoms despite adequate anti-angina treatment and therapy for at least 1 month with a double dose of omeprazole (2 × 20 mg). The exclusion criteria were as follows: changes in ECG that preclude evaluation of ischemia signs (e.g., left bundle branch block or pre-excitation syndrome) and use of medications that might have affected gastric acid secretion or digestive tract motility up to 2 weeks prior to the study, with the exception of the ad hoc administration of nitroglycerin tablets. All patients with CAD took their previously recommended cardiovascular drugs, including acetylsalicylic acid, β-blockers, angiotensin-converting enzyme inhibitors, and statins.

Gastroenterological examinations at baseline Medical history, physical examination, 24-hour esophageal pH-metry and manometry, and panendoscopy with gastric and esophageal biopsy were performed in all patients. Ambulatory evaluation of esophageal function was conducted with a multi-use antimony probe (Synetics Medical AB, Sweden) and a manometry catheter (Synectics, Medtronic) with 3 pressure sensors and a Synectic Digitrapper. An esophageal pH-metric sensor was positioned 5 cm above the manometrically determined LES. A pressure probe was placed via the other nostril at 3, 8, and 13 cm above the LES. On the next day, during continuous esophageal pH-metry and manometry monitoring, a treadmill stress test was performed at approximately 7 a.m. using a device manufactured by Schiller, Switzerland, according to the Bruce protocol (the speed and gradient of the running track were increased every 3 minutes by 2.7, 4, 5.5, 6.8 km/h and by 10, 12, 14, and 16%, respectively). The test was defined as ECG-positive if horizontal or down-sloping ST interval depression by ≥1mm (0.1mV) was observed.

After the stress test, the data obtained during esophageal function monitoring were analyzed using the GASTROSOFT software (Gastrosoft Inc, United States). Gastroesophageal reflux (GER) was diagnosed if the time percentage with esophageal pH <4 was lower than 4.5%. Diffuse esophageal spasm (DES) was diagnosed if ≥30% of contractions during a meal were simultaneous. Nutcracker esophagus (NTC) was diagnosed if more than 10% of contractions had amplitude above 180 mm Hg.[10,11] Exercise-provoked GER (EpGER) was defined as a decrease in esophageal pH<4 for more than 10 seconds during the treadmill stress test. Exercise-provoked esophageal spasm (EpES) was defined as an esophageal motility disorder with more than 55% of simultaneous contractions.[12,13] In patients with the diagnosis of hypertensive esophageal motility disorders (e.g., DES, NTC), a PPI (omeprazole 1 × 20 mg) and a calcium channel antagonist (amlodipine 1 × 10 mg or diltiazem of prolonged release 2 × 120–180 mg, depending on the patient's resting heart rate) were recommended.

Ethics The study protocol was approved by the local Bioethics Committee of the Nicolaus Copernicus University in Toruń and Ludwik Rydygier Collegium Medicum in Bydgoszcz, Poland. All subjects gave their informed consent prior to the study. All procedures were conducted in compliance with the Declaration of Helsinki.

Statistics Statistical analysis was performed using a licensed version of the statistical software STATISTICA PL 9.0 for Windows. The results were mainly presented as the mean ± standard deviation, median; range, or number and percentage. The statistical significance of differences between patients with and without CAD as well as between the other subgroups was tested using the unpaired t-Student test, χ^2 or Fisher's exact test, or the Mann-Whitney U test, depending on the distribution of variables. The Spearman's correlations were tested.

RESULTS Patients with and without CAD did not differ in relation to their endoscopic, pH-

TABLE 1 Demographic and clinical features of patients divided according to the presence of significant coronary artery stenosis (n = 129)

Feature	Patients without coronary artery lesions (n = 77)	Patients with significant coronary artery narrowing (n = 52)
sex, male, n (%)	35 (45)	35 (67)
age, y	53.8 ±7.9	55.0 ±8.8
BMI, kg/m^2	28.7 ±4.2	27.8 ±3.9
smoking, n (%)	13 (17)	11 (21)
coronary angiogram, n (%)	without significant stenosis	significant narrowing in: 1 vessel, 27 (52) 2 vessels, 12 (23) 3 vessels,13 (25)
history of PCI, n (%)	0	24 (46)[a]
history of CABG, n (%)	0	10 (19)[a]
history of myocardial infarction, n (%)	0	21 (40)
hypertension, n (%)	24 (31)	22 (52)
diabetes, n (%)	4 (5)	7 (13)
total cholesterol, mg/dl	195.7 ±28.1	218.9 ±48.3[a]
LDL-C, mg/dl	122.7 ±25.9	136.1 ±39.6
HDL-C, mg/dl	52.1 ±11.9	48.5 ±12.9
triglycerides, mg/dl	100.1 ±33.0	166.1 ±84.5[a]
glucose, mg/dl	100.2 ±15.8	95.0 ±17.7
chest pain within stress test, n (%)	21 (27)	10 (19)
ST interval depression by ≤1 mm, n (%)	41 (53)	27 (52)
location of ST interval depression (inferior wall), n (%)	30 (73)	17 (63)
esophagitis, n (%)	16 (21)	17 (33)
hiatal hernia, n (%)	24 (31)	13 (25)
pathological GER criteria, n (%)	19 (25)	16 (31)
NTC, n (%)	3 (4)	4 (8)
DES, n (%)	24 (31)	9 (17)
nonspecific esophageal disorders, n (%)	10 (13)	6 (12)
EpES, n (%)	14 (18)	9 (17)
normal esophageal manometry, n (%)	26 (34)	24 (46)

a $P < 0.05$

Abbreviations: BMI – body mass index, CABG – coronary artery bypass graft, DES – diffuse esophageal spasm, EpES – exercise-provoked esophageal spasm, GER – gastroesophageal reflux, HDL-C – high-density lipoprotein cholesterol, LDL-C – low-density lipoprotein cholesterol, NTC – nutcracker esophagus, PCI – percutaneous coronary intervention

-metric and manometric parameters of esophageal function, both during a 24-hour monitoring (TABLE 1) and within the treadmill stress test. The horizontal or down-sloping ST interval depression by ≥1mm was observed in 69 individuals (52%). Of these, 42 subjects (54%) had a normal coronary angiogram and 27 (52%) had significant coronary artery stenosis (P = 0.82). Patients with significant ST interval depression during the treadmill stress test (n = 69) were usually women, had a history of hypertension more often, and presented signs of uncoordinated esophageal motility (a significantly lower percentage of peristaltic contractions and a greater percentage of simultaneous contractions during the treadmill stress test) compared with patients with normal ECG (n = 60) (TABLE 2).

Because subjects with ST interval depression had more pronounced esophageal motility disorders, it was supposed that the observed ischemic-like ECG changes, at least in patients with a normal coronary angiogram (n = 27), might have also resulted from hyperactivity of the cardio-esophageal neural loop. Exercise-provoked esophageal disorders (EpGER and EpES) were found in 17/42 patients (41%) with and in 8/35 patients (23%) without significant ST interval depression (P = 0.094). In addition, there were no significant differences in demographic and clinical characteristics between the groups. On the other hand, patients with esophagitis (n = 33, 26%) reached greater load during the treadmill stress test than patients without esophagitis (11.6 ±2.8 vs. 9.7 ±2.9 METs (metabolic equivalents); P = 0.004).

TABLE 2 Parameters of 24-hour esophageal manometry in patients with and without significant ST interval depression

Parameter	Significant ST interval depression (n = 68)	Without significant ST interval depression (n = 61)	*P*
age, y	54.5 ±9.3	51.9 ±7.9	0.29
sex, male, n (%)	29 (42)	41 (68)	0.003
chest pain during stress test, n (%)	18 (26)	13 (21)	0.50
significant coronary artery narrowing, n (%)	27 (39)	25 (42)	0.76
history of hypertension, n (%)	31 (46)	15 (25)	0.02
peristaltic contractions during exercise, %			
13 cm above LES	56.4 ±18.5	66.7 ±19.0	0.01
8 cm above LES	55.8 ±19.3	65.4 ±19.6	0.02
3 cm above LES	59.5 ±19.2	58.9 ±23.0	0.89
simultaneous contractions during exercise, %			
13 cm above LES	35.8 ±22.4	25.8 ±18.8	0.023
8 cm above LES	38.7 ±21.4	27.3 ±20.2	0.01
3 cm above LES	32.7 ±20.2	32.4 ±22.2	0.93

Abbreviations: LES – lower esophageal sphincter

In subjects with endoscopic signs of hiatal hernia (n = 37, 29%), chest pain that required to stop the test occurred earlier than in the remaining patients (200; 180–240 s). However, I found no differences in the results of the stress test, signs of myocardial ischemia, and demographic and clinical characteristics between patients with (n = 63, 49%) and without *Helicobacter pylori* infection, with (n = 35, 27%) and without pH-metric criteria of gastroesophageal reflux disease (GERD), as well as with (n = 40, 31%) and without hypertensive esophageal motility disorders (NTC and DES). Only a few significant but weak correlations between the values of the treadmill stress test and endoscopy, pH-metry, and manometry were observed; for example, duration of the stress test with esophageal pH <4 correlated with the maximal ST interval depression (R = 0.23, *P* = 0.048). Patients with esophagitis had greater esophageal exposure to acid, but no associations between the manometric parameters and the results of endoscopy and pH-metry were found.

The next part of the analysis was designed to determine the factors that caused chest pain during the treadmill stress test. It was observed in 21/77 individuals (27%) with normal coronary angiography and in 10/52 patients (19%) with CAD (*P* = 0.29). It was associated with significant ST interval depression in 12/21 subjects (57%) with normal coronary angiography and in 6/10 patients (60%) with CAD (*P* = 0.82). Subjects who ended the treadmill stress test because of chest pain showed a higher rate of simultaneous esophageal contractions than asymptomatic patients, both in the whole study group and in the subgroups of patients with and without significant coronary artery narrowing. There was no association between the parameters of esophageal pH-metry and manometry (including the prevalence of GERD, DES, and NTC) and chest pain. The analysis conducted in patients with CAD and significant ST interval depression showed that symptomatic patients (n = 6) had greater esophageal exposure to acid during the 24-hour esophageal pH-metry (TABLE 3).

DISCUSSION The present study focused on the associations between esophageal disorders and clinical and electrocardiographic signs of myocardial ischemia, in patients with and without significant coronary artery stenosis in coronary angiography. The main findings are: 1) patients with recurrent chest pain, with and without significant coronary artery narrowing, showed no significant differences in the prevalence of esophageal disorders and ECG changes suggesting myocardial ischemia (TABLE 1); 2) a false-positive result of the treadmill stress was associated with female sex, hypertension, lower waist-to-hip ratio, and the presence of uncoordinated esophageal peristalsis in esophageal manometry performed during the test (TABLE 2); 3) no demographic and clinical factors were identified that might help define subjects in whom ST interval depression would result from esophageal dysmotility and hyperactive cardioesophageal reflex; 4) patients diagnosed with the above espophageal disorders had similar results of the treadmill stress test to those without such diagnosis; 5) exercise-provoked chest pain was not associated with significant ST interval depression in about 40% of subjects with significant coronary artery stenosis, but significant ST was observed in 60% of patients with normal coronary angiogram.

The current results are consistent with those of other authors. A similar prevalence of esophageal disorders in patients with recurrent chest pain, with and without significant coronary artery

TABLE 3 Parameters of 24-hour esophageal pH-metry in patients with coronary artery disease and symptomatic and "silent" ST interval depression during the treadmill stress test

Parameter	Symptomatic myocardial ischemia (n = 6)	"Silent" ischemia (n = 15)	*P*
total number of acid refluxes	164.4 ±43.3	28.1 ±57.3	0.06
number of refluxes >5 min	3.2 ±2.7	0.9 ±1.5	0.025
duration of the longest reflux, min	48.9 ±41.5	7.3 ±9.9	0.002
time with pH <4, %	12.8 ±14.1	2.6 ±3.9	0.02
time with pH <4 in supine position, %	17.3 ±15.8	2.6 ±4.2	0.004
time with pH <4 in horizontal position, %	8.9 ±4.9	2.3 ±3.5	0.12
DeMeester score	52.3 ±60.1	10.7 ±15.9	0.024
EpGER, n (%)	3 (50)	0	0.003

Abbreviations: EpGER – exercise-provoked GER, others – see TABLE 1

narrowing was reported,[14,15] although other authors observed a higher prevalence of these abnormalities in patients with normal coronary angiography.[16-18] The association between cardiac and esophageal disorders might be explained by their common risk factors, the effect of neural[2,7] and hormonal[3] reflexes, and the adverse effects of drugs used for the diseases of both systems. The clinical implication of these results is that the diagnosis of chest pain should be based on clinical symptoms and their response to the recommended therapy. First, life-threatening causes of chest pain should be excluded, particularly those associated with CAD. However, if therapy does not reduce the recurrent symptoms, gastroenterologic causes of chest pain should be tested, regardless of the presence of coronary artery lesions. This means that in patients with CAD, the presence of both cardiac and esophageal causes of chest pain should be taken into account.[4-6] However, such a possibility should also be considered in patients with normal coronary angiography, especially women, because of the relatively high prevalence of small coronary vessel disease.[19]

Since the publication by Chauhan et al.,[2] it has been known that the association between esophageal pH and pressure on coronary perfusion observed in 56% of subjects results from a cardioesophageal reflex. This observation has been confirmed during subsequent years by a number of authors[7,20]; however, only a few[4-6] found associations between esopahegal pH and ECG changes. Unfortunately, clinical characteristics of patients with cardioesophageal reflex have not been determined either in this study or those by Chauhan et al.,[2] Schofield et al.,[21] Dobrzycki et al.,[6] Makk et al.,[7] and Rosztóczy et al.[20] Only Cuomo et al.[22] observed that a decrease in idiopathic cardiac dysrhythmia after therapy with PPI was more frequent in subjects with a correlation between esophageal pH and a ratio of low-to-high frequency components of the power spectrum in heart rate variability analysis. Their observations confirmed the role of neural reflexes in the pathogenesis of cardioesophageal associations but with little use in everyday clinical practice.

I did not confirm the clinically important association between gastroenterologic diagnosis and the result of the treadmill stress test, even if the analysis was restricted to the subgroup of patients with significant ST interval depression and diagnosis of esophageal disorders. This probably resulted from the fact that cardioesophageal reflex was present only in a half of the patients and comparisons of the mean values may cover the potential differences. However, because a recent study has shown[23] that diagnostic yield of elective coronary angiography (about 20% of all procedures) was as low as 38%, and almost 70% of the patients undergoing elective coronary angiography had positive findings on noninvasive examination, further research concerning demographic and clinical predictors of activity of cardioesophageal reflex is needed.

The present study showed that 40% of exercise-provoked chest pain episodes were not associated with ischemic ST interval changes either in patients with or without significant coronary artery stenosis, which confirmed that the esophagus is one of the most important causes of precordial symptoms in these patients. Esophageal causes of chest pain were also observed in patients with significant ST interval depression (TABLES 2 and 3). These causes were expressed by EpGER (in subjects with CAD) and by exercise-provoked simultaneous contractions. In the other study, diagnosis of EpES was a significant favorable prognostic factor in a 2.7-year follow-up.[13] Moreover, lack of angina-like symptoms during the treadmill stress test showed that esophageal disorders are not very likely to be the cause of recurrent chest pain.[12] This might be explained by the results of studies on the associations between gastroesophageal pH or pressure and chest pain threshold.[24-26] It is possible that everyday esophageal exposure to low pH may cause chest pain sensation, even in subjects with similar signs of myocardial ischemia on ECG and similar lesions in coronary angiography (TABLE 3). This observation is corroborated by the favorable effect of PPI in patients with CAD.[4-6] However, a number of previous studies have shown that reduction in the occurrence of chest pain and improved

results of treadmill stress test after therapy with PPI may be related to other potential mechanisms, such as an increase in β-endorphin[27] and nitric oxide[28] secretion.

Conclusions In patients with recurrent chest pain, both cardiac and esophageal causes of the symptoms should be considered. Forty percent of exercise-provoked chest pain episodes in patients with and without significant coronary artery stenosis were not related to significant ST interval depression but were associated with esophageal disorders. Further research should be conducted to determine factors associated with hyperactive cardioesophageal reflexes and the potential effect of esophageal dysfunction on false-positive results of the treadmill stress test.

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ARTYKUŁ ORYGINALNY

Czy dysfunkcja przełyku wpływa na przebieg testu wysiłkowego u pacjentów z nawracającym bólem podobnym do dławicowego?

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SŁOWA KLUCZOWE

ból w klatce piersiowej, choroba wieńcowa, manometria przełykowa, pH-metria, test wysiłkowy

STRESZCZENIE

WPROWADZENIE Neurogenny odruch przełykowo-sercowy może być powodem nasilenia dolegliwości dławicowych i elektrokardiograficznych (EKG) wykładników niedokrwienia mięśnia sercowego, zarówno u pacjentów z istotnymi zwężeniami naczyń wieńcowych, jak i u pacjentów bez istotnych zwężeń.

CELE Ocena związku między pH i ciśnieniem przełykowym a klinicznymi i elektrokardiograficznymi wykładnikami niedokrwienia mięśnia sercowego.

PACJENCI I METODY U 129 pacjentów z nawracającymi bólami typu dławicowego, 77 bez istotnych zmian w koronarografii i 52 z chorobą wieńcową wykonano panendoskopię, pH-metrię, manometrię przełykową oraz test wysiłkowy na bieżni.

WYNIKI Pacjenci z istotnymi zmianami w koronarografii nie różnili się pod względem częstości występowania zaburzeń czynności przełyku od pacjentów bez takich zmian. U chorych ze znamiennym obniżeniem odcinka ST podczas testu wysiłkowego zaobserwowano m.in. zwiększoną częstość przełykowych skurczów jednoczesnych. Pacjenci z nieprawidłowościami przełykowymi nie różnili się istotnie pod względem przebiegu testu wysiłkowego od pacjentów bez tych nieprawidłowości. U 40% pacjentów z istotnymi zwężeniami naczyń wieńcowych kończących test wysiłkowy z powodu bólu dławicowego nie wystąpiło obniżenie odcinka ST w EKG, obserwowano je natomiast u 60% pacjentów z prawidłowym wynikiem koronarografii. U chorych z bólem zaobserwowano większe nieprawidłowości pH przełykowego oraz amplitudy i koordynacji skurczów przełykowych niż u chorych bez bólu. Nie określono demograficznych i klinicznych czynników związanych z wystąpieniem bólu w klatce piersiowej ani zmian w wysiłkowym EKG.

WNIOSKI Zaburzenia czynności przełyku stanowią istotną przyczynę bólu w klatce piersiowej i potencjalnie mogą mieć wpływ na wynik testu wysiłkowego. Konieczne są dalsze badania w celu określenia klinicznych predyktorów aktywności pętli sercowo-przełykowej zarówno u pacjentów z istotnymi zwężeniami w naczyniach wieńcowych, jak i u pacjentów bez tych zwężeń.

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