Impaired glucose tolerance worsens exercise capacity and ventilatory response to exercise in patients with chronic heart failure

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Abstract

Background: There is increasing evidence for the importance of peripheral abnormalities in the pathogenesis and progression of heart failure (HF). Recently, glucose and insulin metabolism abnormalities have been intensively investigated in patients with HF.

Aim: To investigate whether coexistence of impaired glucose tolerance (IGT) may decrease exercise tolerance and influence ventilatory response to exercise in patients with systolic HF.

Methods: Maximal cardiopulmonary exercise test with evaluation of peak VO_2 and VE/VCO_2 slope and oral glucose tolerance test were performed in 64 clinically stable patients with HF and LVEF <45%.

Results: Impaired glucose tolerance was diagnosed in 26 (41%) patients and normal glucose tolerance (NGT) in 38 (59%) patients. There were no significant differences in baseline clinical characteristics or LVEF between groups. There were significant differences in peak VO_2 between IGT and NGT (15.4±4.1 vs. 18.7±4.2 ml/kg/min respectively; p=0.003) and VE/VCO_2 slope (35.7±7.3 vs. 31.8±5.7 respectively; p=0.02). The IGT was independently related to peak VO_2 and VE/VCO_2 slope in multivariate regression analysis.

Conclusion: The IGT is associated with worse exercise capacity and ventilatory response to exercise in patients with HF.

Key words: heart failure, impaired glucose tolerance, oxygen consumption, VE/VCO_2 slope

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Introduction

Decreased exercise tolerance is one of the main indicators of heart failure (HF) severity. There is an increasing amount of evidence for the important role of peripheral factors in the pathogenesis and progression of HF symptoms [1, 2]. In recent years, disturbances of glucose and insulin metabolism in patients with HF have been investigated [3-6]. Diabetes mellitus (DM) is an independent risk factor of HF development [7]. According to recent observations, the relationship between DM and HF is complex [6]. Recent reports also confirmed a reciprocal interrelationship between HF and DM and it has been assumed that HF is associated with an increased risk of DM development [8]. Patients with DM constitute about 30% of an increasing number of patients with HF [3, 9]. These patients, compared to patients without DM, present with higher mortality and more advanced HF, estimated according to NYHA class, the result of 6-minute walk test or decreased peak VO_2 [3, 10, 11]. Little is known, however, about patients with HF and other abnormalities of glucose metabolism. In available studies we have not found reports on the influence of impaired glucose tolerance (IGT) on the results of cardiopulmonary stress test in patients with chronic HF.

The aim of the study was to investigate whether coexisting IGT affects exercise capacity and ventilatory response to exercise in patients with chronic HF.

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Methods

Patients

Sixty-four patients with stable HF and left ventricular ejection fraction (LVEF) <45% assessed on echocardiography, referred for diagnostic evaluation, were included in the study. Forty patients were diagnosed with dilated cardiomyopathy (DCM), and 24 (38%) patients with ischaemic cardiomyopathy (ICM) (>50% stenosis of at least one of the main epicardial coronary arteries revealed on coronary angiography). The exclusion criteria included: exertional angina, exercise-induced arrhythmia, DM, significant respiratory disorders, advanced renal failure (creatinine >250 µmol/l) or insufficiency of other organs leading to significant decrease of exercise capacity. Patients were haemodynamically stable and were treated with stable doses of the following medications at least 2 weeks prior to the study: angiotensin-converting enzyme inhibitors in 58 (91%) patients, furosemide in 47 (73%) patients, spironolactone in 36 (56%) patients, beta-blockers in 47 (73%) patients, digitalis in 35 (55%) patients, statins in 25 (39%) patients, and aspirin in 31 (48%) patients.

Biochemical assessments

Impaired glucose tolerance was diagnosed based on oral glucose tolerance test (glucose concentration 2 hours after 75 g glucose load of between 7.8 mmol/l and 11.1 mmol/l).

The results of BNP concentration measurements (RIA kit, SHIONORIA-BNP, CIS Bio International) were available in 23 patients with IGT and in 33 patients with normal glucose tolerance (NGT).

Cardiopulmonary stress test

All patients underwent maximal treadmill test, according to the modified Bruce protocol (by adding 0-3 min stage to standard Bruce protocol, 1.7 km/h, 5% grade), limited by the occurrence of fatigue and/or dyspnoea. None of the patients demonstrated anginal pain or arrhythmia as a reason for test termination. Oxygen uptake (VO_2) and carbon dioxide production (VCO_2) were measured continuously, breath-by-breath technique using Vmax29 Sensor Medics. Before each test, the flowmeter and gas analyser were calibrated with standard gas mixture. Peak oxygen uptake (peak VO_2) was calculated as a mean value from the final 20 seconds of exercise and presented in ml/kg/min and as a percent of age-predicted VO_2 max [12]. Anaerobic ventilation threshold (AT) was defined with V-slope method. Oxygen uptake at anaerobic threshold (VO_2 in AT), oxygen pulse (pulse O_2) and VE/VCO_2 slope were analysed. The VE/VCO_2 slope – an indicator of exercise ventilation – was calculated as a linear regression function of the relation between ventilation and carbon dioxide production during entire exercise.

Ethics

The study was approved by the Karol Marcinkowski University of Medical Sciences Bioethics Committee. All patients gave their written consent to participate in the study.

Statistical analysis

The results are presented as means and standard deviations or as percentages. Statistical significance of differences between groups was assessed with Student's t-test for unpaired variables or Mann-Whitney and χ^2 tests. Multivariate linear regression was used to evaluate the effects of selected factors on exertion tolerance and respiratory function. Differences with a p value of less than 0.05 were regarded as statistically significant. The calculations were performed using Statistica 5.0 software.

Results

The clinical characteristics of investigated groups of patients are presented in Table I. Twenty six (41%) patients were diagnosed with IGT (IGT group); the remaining 38 (59%) patients presented with NGT (NGT group). There were no significant differences between the groups regarding age, BMI and LVEF values, or creatinine, haemoglobin and BNP concentrations. Moreover, the groups did not significantly differ in terms of HF duration, prevalence of coronary artery disease, hypertension, atrial fibrillation, cigarette smoking or the treatment applied.

Peak VO_2 was significantly lower and VE/VCO_2 slope was significantly greater in the IGT group compared to patients with NGT. Heart rate and systolic blood pressure at peak exercise were similar in both groups (Table II).

Multivariate regression analysis in a model containing peak VO_2 as the dependent variable and LVEF, haemoglobin concentration, presence of IGT, atrial fibrillation, BNP concentration and aetiology (ICM, DCM), demonstrated that IGT (p=0.002) and atrial fibrillation (p=0.005) were independently correlated to peak VO_2.

Multivariate regression analysis, including BMI, aetiology (ICM, DCM), LVEF, haemoglobin and BNP concentration and the presence of atrial fibrillation, showed that IGT (p=0.01), LVEF (p=0.000008) and

Table I. Clinical characteristics of patients with HF and impaired (IGT) or normal (NGT) glucose tolerance

Parameter	IGT (n=26)	NGT (n=38)	p
Age [years]	51.8±9.3	49.7±6.7	NS
Gender F/M [%]	100/0	95/5	NS
Cigarette smoking [%]	35	37	NS
BMI [kg/m^2]	29.1±5.0	26.8±5.2	NS
LVEF [%]	26.3±8.6	26.4±8.3	NS
Creatinine [µmol/l]	90.9±20.7	84.4±15.1	NS
BNP [pg/ml] (n=23 vs. 33)	88.6±75	62±67	NS
Haemoglobin [mmol/l]	9.4±0.8	9.2±0.7	NS
Fasting glucose [mmol/l]	5.59±0.8	5.0±0.6	0.001
Glucose after 2 hours [mmol/l]	8.8±1.0	5.7±1.2	<0.0001
Coronary artery disease [%]	46	33	NS
Hypertension [%]	58	50	NS
Atrial fibrillation [%]	12	24	NS
Duration of HF [months]	35.8±33.1	41.9±33.4	NS
NYHA class	2.7±0.7	2.1±0.8	0.009
Beta-blockers [%]	81	68	NS
ACE inhibitors [%]	84	95	NS
Digitalis [%]	54	55	NS
Aspirin [%]	50	47	NS
Statins [%]	38	39	NS
Furosemid [%]	85	66	NS

Abbreviations: BMI – Body Mass Index, LVEF – left ventricular ejection fraction, HF – heart failure, BNP – B-type natriuretic peptide

Table II. The results of cardiopulmonary stress test in patients with HF and impaired (IGT) or normal (NGT) glucose tolerance

Parameter	IGT (n=26)	NGT (n=38)	p
Exercise duration [s]	474±195	545±145	NS
Peak VO_2 [ml/kg/min]	15.4±4.1	18.7±4.2	0.003
VO_2 in AT [ml/kg/min]	11.2±2.3	12.3±2.7	NS
O_2 pulse	10.1±3.7	10.6±2.8	NS
RER peak	1.07±0.06	1.06±0.05	NS
VE/VCO_2 slope	35.7±7.3	31.8±5.7	0.02
HR rest [1/min]	78.6±15	80.8±13	NS
HR max [1/min]	139.5±29.8	144.8±27.3	NS
SBP rest [mm Hg]	116.7±20.4	115.0±14.2	NS
SBP max [mm Hg]	136.2±33.4	136.7±22.2	NS

Abbreviations: AT – anareobic threshold, RER – respiratory exchange ratio, SBP – systolic blood pressure, HR – heart rate

ischaemic aetiology are independently associated with VE/VCO_2 slope.

Discussion

This study provides additional information about the relationship between abnormalities of glucose/insulin metabolism and HF with regard to patients with coexisting HF and IGT. We demonstrated that patients with IGT and HF present with significantly worse exercise capacity and ventilatory response to exercise compared with subjects with HF but without IGT. Moreover, a new observation was made, that IGT is an independent predictor of peak VO_2 and VE/VCO_2 slope in HF.

Available reports indicate that DM, fasting hyperglycaemia and insulin resistance in patients with HF are associated with worse clinical condition, assessed according to NYHA classification, results of 6-minute walk test or decreased peak VO_2 [3, 4, 10, 11].

Insulin resistance is a principal precursor and predominant feature of DM type 2. Hyperglycaemia, leading to hyperinsulinaemia, can further increase insulin resistance. Insulin is an anabolic hormone and controls muscle energetic condition and has been regarded as one of the potential factors contributing to HF pathogenesis [5, 13]. As mentioned above, it has been postulated that HF is associated with insulin resistance (regarded as fasting and stimulated hyperinsulinaemia). Moreover, the more advanced the HF (the lower the peak VO_2), the more pronounced is insulin resistance [13]. Recently it has been revealed that impaired insulin sensitivity is associated with higher mortality rate, independently of body composition and established prognostic factors [14].

In the available literature we have not found any reports on the effects of IGT on exercise capacity of patients with HF. There are many recent reports on IGT and hyperglycaemia. Most of them, however, deal mainly with coronary artery disease [15-17]. In patients with IGT many metabolic abnormalities are found, typical for DM (abnormalities of insulin secretion, insulin resistance). It is relatively easy to establish diagnosis of IGT and diagnostic work-up is postulated to include oral glucose tolerance test in routine evaluation of patients with myocardial infarction [15]. The assessment of insulin sensitivity is not routinely used in evaluation of patients with HF. Moreover, in everyday practice, abnormalities of glucose metabolism are identified in patients with evident DM.

The influence of DM on exercise capacity in patients with HF has been previously evaluated. Patients with HF and DM have worse exercise capacity, assessed according to NYHA classification, 6-minute walk test, or cardiopulmonary stress test (peak VO_2), compared to patients with HF but without DM [3, 4, 10, 11]. Moreover, it has been documented that DM is an independent indicator of a worse result of 6-minute walk test, which is an optional method of functional assessment [10]. Recently published studies of larger groups of patients with HF and DM, including subjects treated with beta-blockers, support the previous observation that peak VO_2 is significantly decreased in patients with HF and coexisting DM, in comparison to patients without DM [11]. It has been demonstrated that DM is an independent indicator of peak VO_2 in subjects with HF [11] and that DM results in cardiovascular and metabolic changes that may lead to a decrease of peak VO_2 [18]. Energetic metabolism of skeletal muscles in patients with DM is deteriorated [19]. Changes in energetic metabolism influence exercise tolerance in patients with HF [20]. Thus, the decrease of exercise capacity in patients with HF and DM most likely results from alterations in energetic metabolism of skeletal muscles, found in both DM and HF.

In the presented study, peak VO_2 was found to be significantly lower in patients with IGT compared to patients with NGT. Moreover, IGT alongside atrial fibrillation was found to be an independent predictor of decreased exercise tolerance, in a model containing the results of routinely performed investigations: LVEF, haemoglobin concentration, BNP concentration and aetiology (ICM vs. DCM). Furthermore, VE/VCO_2 slope, which reflects the ventilatory response to exercise, was found to be substantially higher in subjects with HF and coexisting IGT than in patients without IGT. The IGT and LVEF turned out to be independent predictors of VE/VCO_2 slope. Also patients with HF and DM present with higher VE/VCO_2 slope than patients without DM [4]. Excessive exercise ventilation, intensified in cases of DM, may lead to premature exhaustion of ventilatory reserves and early discontinuation of exercise.

A significant association between VO_2 and VE/VCO_2 slope in patients with HF was demonstrated [21, 22]. The VE/VCO_2 slope is an significant, independent prognostic factor of HF. Moreover, it is considered by some to have a greater prognostic value than peak VO_2 [23-25]. Reasons for increased ventilation during exercise include: increased lung dead space [26], ventilation-perfusion mismatch [27] and disturbances of pulmonary circulation [28]. Excessive activation of chemo- and ergoreceptors in patients with HF, which results from increased sympathetic activity, can lead to increased ventilation that accompanies poor exercise tolerance [29, 30]. Diabetes mellitus is also associated with disturbed autonomic regulation [31]. In patients with HF and DM, a relation between peak VO_2 and

VE/VCO_2 slope and impaired lung function (which might synergistically result from HF and DM) was noted [4].

The mechanism of VE/VCO_2 slope increase in patients with HF and IGT, observed in our study, remains unknown. Diabetes mellitus is known to be a slowly progressive disease. The vascular complications of DM are usually recognised at the time of DM diagnosis. Thus, it might be assumed that vascular disease begins before diagnosis of DM. In such a case, it would be possible to expect the mechanisms of impaired functional condition and increased exercise ventilation also to occur earlier than DM develops, i.e. in patients with IGT.

It seems that abnormalities of glucose metabolism (not only DM, but also IGT) should be included in the assessment of functional capacity of patients with HF. Based on the results of our study, it might be interesting to investigate whether early detection of IGT and initiation of exercise training as a part of rehabilitation would improve the clinical condition and prognosis of patients with HF as well as prevent the development of DM.

Conclusion

The presence of impaired glucose tolerance worsens exercise capacity and exercise ventilation in patients with heart failure.

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Nieprawidłowa tolerancja glukozy pogarsza wydolność i wentylację wysiłkową u chorych z niewydolnością serca

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Streszczenie

Wstęp: Zmniejszona tolerancja wysiłku jest jednym z głównych wskaźników stopnia zaawansowania niewydolności serca (HF). Wzrasta liczba dowodów na istotną rolę czynników obwodowych w patogenezie i progresji objawów tej choroby. W ostatnich latach badane są zaburzenia metabolizmu glukozy i insuliny u chorych z HF. Wśród rosnącej liczby chorych z HF ok. 30% stanowią pacjenci ze współistniejącą cukrzycą (DM). U chorych z DM w porównaniu z chorymi bez DM większa jest umieralność i bardziej zaawansowana HF oceniana wg klasyfikacji NYHA, jako wynik testu 6-minutowego marszu lub obniżony *peak* VO_2. Niewiele wiadomo o chorych z HF i innymi zaburzeniami metabolizmu glukozy.

Cel: Zbadanie, czy współistnienie nieprawidłowej tolerancji glukozy (IGT) może wpływać na obniżenie wydolności i wentylację wysiłkową u chorych z HF.

Metodyka: U 64 chorych z HF i frakcją wyrzutową lewej komory (LVEF) <45% w ocenie echokardiograficznej, wykonano maksymalny test spiroergometryczny na bieżni ruchomej, wg zmodyfikowanego protokołu Bruce'a z oceną *peak* VO_2 i VE/VCO_2 *slope,* oraz doustny test tolerancji glukozy (OGTT). U 40 chorych rozpoznano kardiomiopatię rozstrzeniową (DCM), a u 24 (38%) – kardiomiopatię niedokrwienną (ICM). Kryteriami wyłączenia z badania były: dławica wysiłkowa lub indukowane wysiłkiem zaburzenia rytmu serca, DM, istotne choroby układu oddechowego, ciężka niewydolność nerek (kreatynina >250 μmol/l) lub niesprawność innych narządów, znacząco wpływająca na wydolność fizyczną.

Wyniki: Nieprawidłową tolerancję glukozy rozpoznano u 26 chorych (41%), a prawidłową tolerancję glukozy (NGT) u 38 (59%). Nie stwierdzono istotnych różnic między grupami w odniesieniu do podstawowej charakterystyki klinicznej: wieku, wielkości BMI i LVEF, stężenia kreatyniny, hemoglobiny i BNP. Nie było też istotnych różnic między grupami w czasie trwania objawów HF, częstości występowania choroby wieńcowej, nadciśnienia tętniczego, migotania przedsionków (AF), palenia papierosów oraz stosowanym leczeniu. U chorych z IGT w porównaniu z chorymi z NGT istotnie mniejszy był *peak* VO_2 (15,4±4,1 *vs* 18,7±4,2 ml/kg/min; p=0,003) i istotnie większy VE/VCO_2 *slope* (35,7±7,3 *vs* 31,8±5,7; p=0,02). Czynność serca i skurczowe ciśnienie tętnicze na szczycie wysiłku były podobne w obu grupach. Analiza wieloczynnikowa za pomocą regresji zmiennej zależnej w modelu zawierającym *peak* VO_2 jako zmienną zależną i LVEF, stężenie hemoglobiny, obecność IGT, AF, stężenie BNP i etiologię (ICM, DCM) wykazała, że IGT (p=0,002) i AF (p=0,005) są niezależnie związane z *peak* VO_2. Analiza wieloczynnikowa za pomocą regresji zmiennej zależnej uwzględniająca BMI, etiologię (ICM, DCM), LVEF, stężenie hemoglobiny i BNP, obecność AF i IGT wykazała, że czynnikami niezależnie związanymi z VE/VCO_2 *slope* są IGT (p=0,01), LVEF (p=0,000008) i etiologia niedokrwienna (p=0,01). Wydaje się, że w ocenie wydolności czynnościowej chorych z HF powinno się uwzględniać zaburzenia metabolizmu glukozy – nie tylko cukrzycę, ale i IGT. Na podstawie wyników tego badania interesujące wydaje się sprawdzenie hipotezy, że wczesne wykrycie IGT i interwencja polegająca na włączeniu chorego do programu treningu fizycznego w ramach rehabilitacji może wpłynąć na poprawę stanu klinicznego i rokowanie chorych z HF oraz zapobiegnie rozwojowi DM.

Wniosek: Współistnienie IGT pogarsza wydolność i wentylację wysiłkową u chorych z HF.

Słowa kluczowe: niewydolność serca, nieprawidłowa tolerancja glukozy, zużycie tlenu, VE/VCO_2 *slope*

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