reperfusion therapy, the biphasic response observed in this study can be explained.

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Blood leukocytes in heart failure with preserved ejection fraction: Impact on prognosis

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ARTICLE INFO

Heart failure (HF) with preserved ejection fraction (HFpEF) is an emerging problem of cardiovascular medicine with increasing prevalence and poor prognosis [1,2]. The pathophysiological mechanisms of HFpEF are still poorly understood.

Leukocytes play important role in the pathogenesis and prognosis of HF with reduced ejection fraction (HFrEF) via their contribution to inflammation, extracellular matrix remodelling, and reparatory processes [3]. Relative lymphopenia is a strong predictor of mortality in severe HFrEF [4,5]. In contrast, neutrophils are often increased in HFrEF and pose an increased risk of all-cause and cardiovascular mortality [6]. However, only scarce data are available on the impact of leukocytes on pathophysiology and outcome in HFpEF.

In this study we aimed to compare levels of neutrophiles, lymphocytes and monocytes between patients with HFrEF and HFpEF and to establish their impact on the mortality in HFpEF.

The total number of 1019 HF patients referred the Cardiology Clinic of the Grodno Regional Clinical Hospital in 2008–2009 with either HFpEF ($n=856$, age 55[49–64], 81% males) or HFrEF ($n=163$, age 58[51–65], 56% males) were included into the study. The diagnosis of HFrEF or HFpEF was established according to the current ESC guidelines [7]. Patients with life expectancy <1 year due to noncardiac courses (e.g., cancer) and those with terminal HF (NYHA class IV) were not included.

All subjects underwent transthoracic echocardiography with LVEF measured using the modified Simpson's biplane method and diastolic function assessed by evaluating E/A ratio. The leucocytes were measured using an automatic haematocytometer (Micros-60 Horiba, ABX Diagnostics, France).

The patients were followed for at least 1 year with median (interquartile range) follow-up duration of 17[14–20]months with the end-point of any-cause death registered. The study was approved by the Institutional Research Board of the Grodno State Medical University. The authors of this manuscript have certified that they comply with the Principles of Ethical Publishing in the International Journal of Cardiology [8].

Most patients had mild HF (NYHA I–II class in 98% of HFpEF, 82% of HFrEF) predominantly of ischemic aetiology (74% of HFpEF, 73% of HFrEF). Patients with HFrEF had lower body mass index ($p=0.017$), and proportion of patients with history of hypertension ($p<0.001$), higher proportions of males ($p<0.001$), and subjects with history of previous myocardial infarction ($p=0.002$).

Patients with HFpEF as compared with HFrEF had significantly lower neutrophil count (4140[3498–5293] vs. 3933[3247–4760] per µl, $p=0.004$) and percentage (63[58–68]% vs. 65[59–69]%, $p=0.018$) and higher lymphocyte percentage (30[26–34]% vs. 29[24–33], $p=0.004$). On regression analysis, significant clinical predictors of reduced LVEF in the whole study population were advanced age, male sex, history of hypertension and atrial fibrillation ($p<0.05$). Among leukocytes, high neutrophil count and percentage and low lymphocyte percentage were associated with reduced LVEF and remained so after adjustment for the clinical predictors above ($p<0.05$).

During the follow-up period, 41 deaths (4.02%) occurred (28 in HFpEF and 13 in HFrEF). Using logistic regression analysis, a history of hypertension was the only significant clinical predictor of death in HFpEF. Among leukocytes, high monocyte count was predictive of death in HFpEF before ($p=0.01$) and after ($p=0.012$) adjustment for age, LVEF, and hypertension ($p=0.012$) (Table 1). In HFpEF

Table 1
Predictive value of leukocytes for all cause death in patients with HFpEF.

Parameter	Unadjusted		
	Odds ratio	95% CI	*p*
Neutrophils, per 1000 cells	1.16	0.95–1.41	0.15
Monocytes, per 200 cells	1.42	1.09–1.84	0.01
Lymphocytes, per 1000 cells	1.40	0.78–2.53	0.27
Adjusted for hypertension, age, and ejection fraction			
Monocytes, per 200 cells	1.41	1.08–1.84	0.012

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there was a 41% increase in risk of death per every 200 monocytes/μl increment. Additionally, monocytosis (i.e., monocyte count above 800 cells/μl) was associated with prominent raise in risk of death compared with patients with normal monocyte levels (odds ratio 5.41 [95% confidence interval 1.50–19.6], $p = 0.01$).

The observed impact of monocytes on the outcome of HFpEF is perhaps not unexpected. Monocytes are immune and pro-inflammatory cells with plethora of other biological roles, including the regulation of myocardial extracellular matrix turnover [3,9]. Monocytes orchestrate tissue remodelling by release of various matrix metalloproteinases, cytokines and growth factors [3]. It has been previously shown that high monocyte levels were associated with future myocardial infarction in stable coronary artery disease and in those with myocardial infarction high monocyte numbers were strongly associated with unfavourable outcome [9,10].

In agreement with previous reports, we observed that neutrophils and lymphocytes were associated with LV impairment in HF but not with the outcome in HFpEF [4–6]. This may be due to the predominantly mild HF in the study population in contrast to moderate-to-severe HF in most previous studies.

Among the study limitations is that the leukocytes were measured using a routine haematological analysis, which did not provide information on specific monocyte and lymphocyte subsets nor leukocyte functional activity. Additionally, the death rate was relatively low as the majority of the participants had mild HF and thus, the study results may not be transferable to HF patients with higher NYHA classes.

As a conclusion, there are significant differences in the numbers and percentage of neutrophils and percentage of lymphocytes between HFrEF and HFpEF. High monocyte count is a significant independent predictor of death in patients with HFpEF. The study results advance the knowledge on the significant differences in the pattern of leukocytes between HFpEF and HFrEF and may help to better understand the complex pathophysiology of HFpEF.

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Do surgical procedures affect EuroSCOREs' performance? The role of tricuspid valve surgery

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The EuroSCORE (European System for Cardiac Operative Risk Evaluation) is a tool widely used for predicting hospital mortality after cardiac surgery. Some concerns on its power have recently emerged as estimated risk within specific surgical subpopulations has been reported to be over-predicted [1]. Nonetheless, no data are available on relationship between EuroSCORE performance and surgical procedures and even the recently released updated algorithm categorizes surgeries in general classes. The purpose of this study was to evaluate the impact of surgical procedures on EuroSCOREs' prediction power, modelling the receiver operating characteristic (ROC) curve with a generalized linear model.

Data on 5377 consecutive patients who underwent cardiac surgery from January 2001 to January 2011 were retrieved from our prospective institutional database whose use for research has been approved by the Institutional Review Board. Discriminatory power and calibration were assessed using the c-index (AUC, area under the receiver operating characteristic curve) and the Hosmer–Lemeshow goodness-of-fit test respectively. In order to analyze the effect of type of surgery on discriminatory power, we modeled the ROC curve with a parametric generalized linear model (GLM), using a binormal model [2]. The Institutional Ethical Committee approved the study and the requirement for informed written consent was waived on the condition that subjects' identities were masked. A p value of less than 0.05 was considered

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