ORIGINAL ARTICLE

Association of blood groups with prognosis in acute coronary syndrome

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KEY WORDS

ABO status, acute coronary syndrome, D antigen, Rh factor

ABSTRACT

INTRODUCTION Multiple factors that affect the prognosis of acute coronary syndromes (ACS) have been identified. There are scarce data on the effect of the blood group on prognosis in this patient group.

OBJECTIVES We investigated the effect of ABO and Rh (D antigen) status on the prognosis of ACS.

PATIENTS AND METHODS A group of 418 consecutive hospitalized patients with ACS were analyzed. The follow-up period was 2075 ±794 days. The primary endpoint was all-cause mortality. A statistical analysis was performed for the following subgroups: ABO blood group, ABO blood group including Rhesus (Rh) factor (D antigen), Rh-positive vs. Rh-negative blood group, 0 blood group vs. non-0 blood group, blood group with vs. without the B antigen, and blood group with vs. without the A antigen.

RESULTS A total of 348 patients (83.25%) were Rh-positive, while 70 (16.75%) were Rh-negative. The Kaplan–Meier survival plots showed 7-year mortality of 22.7% in patients with blood groups with Rh antigen and of 10% in patients without Rh antigen ($P = 0.014$). Other comparisons were not statistically significant. A multivariable Cox proportional hazards model identified blood group with D antigen as an independent predictor of mortality (hazard ratio, 7.758; 95% confidence interval, 1.748–34.417; $P = 0.007$).

CONCLUSIONS Of all blood groups, only the Rh-positive blood group was an independent predictor of mortality in patients with ACS.

INTRODUCTION An imbalance between pro- and anticoagulant factors combined with atherosclerosis increases the risk of myocardial infarction (MI).[1] There is extensive and constantly increasing amount of data on factors for prognosis after MI. More and more sophisticated factors are correlated with adverse cardiac events after MI.[2] Simultaneously, a considerable effort is made to establish novel variables that can affect long-term outcome after MI.[3] The effect of a blood group on the frequency and prognosis of MI has been studied. Several studies have documented that non-O blood groups are associated with higher risk of cardiovascular diseases while O blood group is not,[4] which has been attributed to the effect of the ABO blood group on the plasma levels of procoagulant von Willebrand factor (VWF).[5] Individuals with non-O groups have higher VWF levels,[6-13] which is associated with a higher risk of thrombosis[14] and, consequently, higher risk of cardiovascular diseases.[15,16] Although there are broad data on an association between a blood group and MI, little is known about the effect of a blood group on prognosis after MI. Therefore, we aimed to assess the effect of ABO and Rhesus (Rh; D antigen) blood groups on the prognosis of patients after acute coronary syndrome (ACS).

PATIENTS AND METHODS The study group consisted of 418 consecutive patients who were diagnosed with ACS at the 1st Department of Cardiology at the Medical University of Warsaw, Warsaw, Poland. Patients were enrolled to the study between January 2002 and January 2004. Informed consent was obtained from all participants. The study protocol was approved by the institutional ethics committee on the Medical University of Warsaw. Primary percutaneous

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coronary intervention (PCI) was performed *ad hoc* when feasible. All patients gave written informed consent to undergo angiography and angioplasty. ACS was diagnosed by chest pain, ST-T changes in electrocardiogram, and elevated cardiac enzymes. ACS includes ST-segment elevation MI (STEMI), non-ST segment elevation MI (NSTEMI), and unstable angina. STEMI was defined as chest pain lasting 12 h or less and ST-segment elevation of 1 mm or higher in at least 2 leads in electrocardiogram on admission with elevated markers of myocardial necrosis. NSTEMI was defined as acute chest pain without persistent ST-segment elevation with positive markers of myocardial necrosis, and unstable angina was defined as chest pain with or without ST abnormalities and negative markers. The ABO blood groups and Rh factor were determined by standard techniques.

The primary endpoint of the study was all-cause mortality. Mortality data were obtained from the National Death Registry of Poland. A statistical analysis was performed for each subgroup of the study: ABO blood groups, ABO blood group including the Rh factor, Rh+ vs. Rh– blood group, O blood group vs. non-O blood group, blood group with vs. without the B antigen, and blood group with vs. without the A antigen.

Continuous variables are presented as mean value ± standard deviation and were compared using the Mann–Whitney *U* test or *t* test. Categorical variables were compared by using either the χ^2 or Fisher exact tests. The log-rank test was performed for Kaplan–Meier probability estimates. The χ^2 test was used for analysis of categorical data. A 2-tailed *P* value of less than 0.05 was considered statistically significant, and confidence intervals were 95%. A multivariable Cox proportional hazards model was performed with the forward and backward method. The model included the variables presented in TABLE 1. SAS 9.2 (SAS Institute Inc, Cary, North Carolina, United States) and Statistica 9 (StatSoft Inc, Tulsa, Oklahoma, United States) were used for all statistical analysis.

RESULTS The study group consisted of 418 patients (63.64% men) aged 61.52 ±11.15 years (range, 26–93 years) with ACS. STEMI was diagnosed in 291 patients (69.61%). Early invasive treatment was performed in 368 patients (87.8%). There were no patients lost to follow-up. During the 2.075 ±794 days of follow-up, 86 patients (20.57%) died. Minimum and maximum follow-up duration was 1945 days and 2743 days for survivors, respectively.

In the entire cohort, the most common blood group was A Rh+ (147 patients, 35.17%), followed by O Rh+ (103 patients, 24.64%) as presented in TABLE 2. The majority of the patients were Rh-positive (348 patients, 83.25%); 70 patients (16.75%) were Rh-negative.

Unadjusted Kaplan–Meier survival plots of cumulative incidence of 7-year mortality in the major ABO blood groups showed no statistical significance (FIGURE 1). Patients with the Rh factor had mortality of 22.7%, while those without the Rh factor had mortality of 10% ($P = 0.014$) (FIGURE 2A). The groups did not differ in terms of baseline clinical characteristics except for prior PCI (3.16% for Rh+ and 8.57% for Rh–, $P = 0.037$). A land-mark analysis (396 patients who were discharged from the hospital) showed similar differences in mortality between the groups with and without the Rh factor (mortality of 18.90% in the Rh+ group vs. 8.82% in the Rh– group, $P = 0.051$). The groups did not differ in terms of short-term mortality (in hospital: 5.75% in the Rh+ group vs. 2.86% in the Rh– group, $P = 0.323$). We did not observe significant differences in major bleeding between the groups with and without the Rh factor (5.17% for Rh+ and 10% for Rh–, $P = 0.161$). Also, there were no significant differences in major bleeding events between the other blood groups.

We also compared other groups: ABO with the Rh factor, blood group with vs. without the A antigen (FIGURE 2B), blood group with vs. without the B (FIGURE 2C), O blood group vs. non-O blood group (FIGURE 2D); however, these comparisons did not achieve statistical significance. Unadjusted Kaplan–Meier curve for all blood groups demonstrated the worst prognosis for Rh-positive patients. Patients with groups B Rh+ and A Rh+ had the highest mortality rate, 26.87% and 23.81%, respectively, compared with the other groups ($P = 0.272$). Patients with group B Rh– had similar mortality rates to those observed in patients with group O Rh+ (19.42%) or AB Rh+ (19.35%).

The comparison of blood groups with and without the B antigen showed a trend for higher mortality in patients with the antigen (23.28% and 19.54%, respectively, $P = 0.209$, FIGURE 2C). There were no significant differences in mortality rates between patients with O and non-O blood groups (17.19% and 22.07%, respectively, $P = 0.265$, FIGURE 2D). The study showed the highest mortality trends for patients with group B, followed by group A, AB, and O (25.00%, 21.26%, 18.75%, and 17.19%, respectively, $P = 0.529$). The differences were not statistically significant (FIGURE 1). Patients with and without the A antigen had almost the same risk of death during a 7-year follow-up (20.87% and 20.28%, respectively, $P = 0.994$) (FIGURE 2B).

We observed significant differences between the characteristics of the patients in the subgroups of survivors and non-survivors (TABLE 1) although there were no differences in the distribution of the main blood groups. Survivors had shorter hospital stay, higher incidence of de-novo angina, and more frequently were smokers. Moreover, they had lower incidence of diabetes, previous MI, vascular disease, and heart failure (class III or IV according to the New York Heart Association Functional Classification). Finally, they had lower C-reactive protein and creatinine levels.

The multivariable Cox proportional hazards model with the backward variable selection method identified Rh-positive status, hypertension, diabetes mellitus, previous PCI, troponin I on

TABLE 1 Baseline clinical characteristics of the patients

Characteristics		Non-survivors (n = 86)	Survivors (n = 319)	*P* value
blood group	A	43.02	41.07	0.57
	B	24.42	19.12	0.57
	AB	6.98	7.52	0.57
	0	25.58	32.29	0.57
	Rh+	91.86	80.56	0.02
STEMI		70.93	69.59	0.81
male sex		67.44	63.01	0.45
de-novo angina		32.56	47.65	0.01
hypertension		58.14	57.99	0.98
current smoker		24.42	42.32	0.003
diabetes mellitus		27.91	13.17	0.001
dyslipidemia		31.40	32.60	0.83
prior myocardial infarction		37.21	18.81	0.0003
prior percutaneous coronary intervention		4.65	4.08	0.82
prior coronary artery bypass grafting		2.33	2.82	0.80
prior stroke		8.14	3.45	0.06
vascular disease		22.09	8.46	0.0004
heart failure, III or IV NYHA functional class		6.98	1.88	0.01
heart rate, beats/min		80.36 ±18.47	77.81 ±17.36	0.22
systolic blood pressure, mmHg		128.23 ±31.93	133.55 ±28.50	0.22
diastolic blood pressure, mmHg		73.63 ±20.19	78.53 ±15.20	0.03
left bundle branch block		4.65	2.82	0.39
right bundle branch block		8.14	1.88	0.004
peak troponin I, µg/l		96.84 ±160.32	97.81 ±130.20	0.02
total cholesterol, mg/dl		188.51 ±43.29	190.35 ±42.32	0.82
low-density lipoprotein cholesterol, mg/dl		112.37 ±39.37	115.43 ±38.92	0.61
high-density lipoprotein cholesterol, mg/dl		45.51 ±14.37	45.45 ±13.55	0.96
triglycerides, mg/dl		152.05 ±86.28	147.64 ±89.63	0.80
C-reactive protein, mg/dl		52.47 ±67.48	24.76 ±37.47	0.0005
creatinine level, mg/dl		1.37 ±1.14	0.97 ±0.27	0.0001
glomerular filtration rate, ml/min/1.73 m^2		66.84 ±30.03	89.67 ±32.03	0.0001
infarct-related artery	LAD	29.07	30.72	0.77
	LCx	12.79	11.91	0.83
	RCA	37.21	39.81	0.66
treatment during hospitalization	ASA	96.51	99.06	0.08
	clopidogrel	84.88	87.46	0.53
	glycoprotein IIb/IIIa inhibitor	55.81	60.19	0.47
	ACEI	80.23	92.48	0.0009
	β-adrenolytic	77.91	95.30	0.0001
	statin	88.37	92.16	0.27
treatment at discharge	ASA	68.60	85.89	0.0002
	clopidogrel	50.00	68.65	0.001
	antithrombotic	8.14	6.27	0.54
	ACEI	69.77	90.28	0.0001
	β-adrenolytic	73.26	93.10	0.0001
	statin	73.26	94.04	0.0001

Data are presented as percentage or mean ± standard deviation.

Conversion factors to SI units are as follows: for glucose – 0.05551, cholesterol – 0.02586, and triglycerides – 0.0114.

Abbreviations: ACEI – angiotensin-converting-enzyme inhibitor, ASA – acetylsalicylic acid, LAD – left anterior descending artery, LCx – left circumflex artery, NYHA – New York Heart Association, RCA – right coronary artery, STEMI – ST-segment elevation myocardial infarction

TABLE 2 Blood group distribution in the study population

Blood group	Rh+, n = 348 (83.25%)	Rh–, n = 70 (16.75%)
A	147 (35.17)	27 (6.46)
B	67 (16.03)	17 (4.07)
AB	31 (7.42)	1 (0.24)
	103 (24.64)	25 (5.98)

Data are presented as number (percentage).

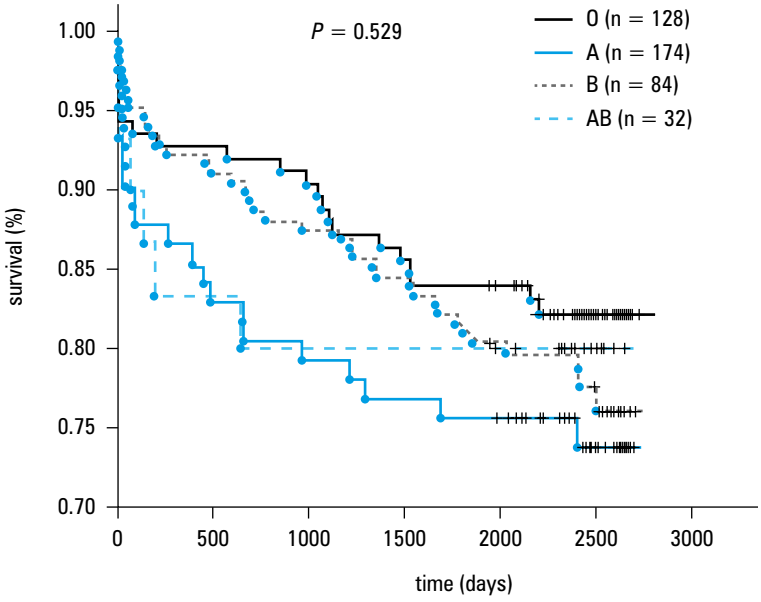

FIGURE 1 Mortality in the main blood groups

admission, peak troponin I, C-reactive protein, glomerular filtration rate, as well as statins and β-blockers prescribed at discharge as independent correlates of 7-year mortality (TABLE 3).

DISCUSSION Our study demonstrated that the negative Rh factor correlates with better long-term prognosis in ACS. Ketch et al.[6] showed that patients with non-O groups had larger myocardial infarcts. In our study, we did not observe higher mortality rates in patients with non-O groups compared with those with the O group (FIGURE 2D). The group of AB– patients was too small to reliably assess mortality. The distribution of the blood groups in our study did not differ significantly from that in the general Polish population.[17]

Although the association between a blood group and cardiovascular diseases has been known for many years, its complex nature has not been fully elucidated yet. The majority of the studies have reported associations between the ABO group and thrombosis, indicating that non-O blood groups carry a higher risk of MI, venous thromboembolism, and cardiovascular diseases than the O blood group. The extent of these associations is still unclear. Most researchers link a non-O blood group with higher VWF levels associated with a higher risk of cardiovascular disease in these patients.[5] A recent study revealed that glycotransferease-deficient enzyme, which encodes group O, reduces the levels of circulating VWF as well as the risk of MI.[18]

The majority of the available studies involved ABO blood group and risk of cardiovascular diseases, while there is lack of data on the effect of Rh factor on the risk and prognosis of MI. Wu et al.[4] published a meta-analysis of ABO blood groups with reference to cardiovascular disease. It included 22 studies on MI, 9 of which reported that individuals with non-O groups had a significantly higher risk of MI and 1 reported a reduced risk in this group of patients.[19] Interestingly, the authors concluded that those differences depended on whether the studies were conducted prospectively or retrospectively. Retrospective studies tended to report a higher risk of MI than prospective ones.

Ketch et al.[6] compared the baseline characteristics, procedural findings, and clinical events during 1-year follow-up between patients with the O blood group and non-O blood group. Patients with the non-O group had significantly lower grades of Thrombolysis In Myocardial Infarction flow and higher amount of visible thrombus on PCI. Moreover, patients with non-O blood group had larger infarct sizes, evidenced by higher median peak troponin, total creatinine kinase (CK), and CK-MB. Unadjusted Kaplan–Meier plots of cumulative incidence of adverse events did not show significant differences between the two

TABLE 3 Multivariate analysis by Cox proportional hazards (only factors that reached statistically significance were shown)

Factor	HR	95% CI	*P* value
Rh+	7.758	1.748–34.417	0.007
hypertension	0.310	0.157–0.609	0.0007
diabetes mellitus	3.752	1.823–7.722	0.0003
prior percutaneous coronary intervention	6.379	1.347–30.203	0.02
troponin I on admission	3.288	1.538–7.028	0.002
peak troponin I	1.002	1.000–1.004	0.05
C-reactive protein	1.012	1.007–1.017	<0.0001
glomerular filtration rate	0.970	0.960–0.980	<0.0001
statin at discharge	0.332	0.129–0.849	0.02
β-adrenolytic at discharge	0.244	0.085–0.699	0.009

Abbreviations: CI – confidence interval, HR – hazard ratio

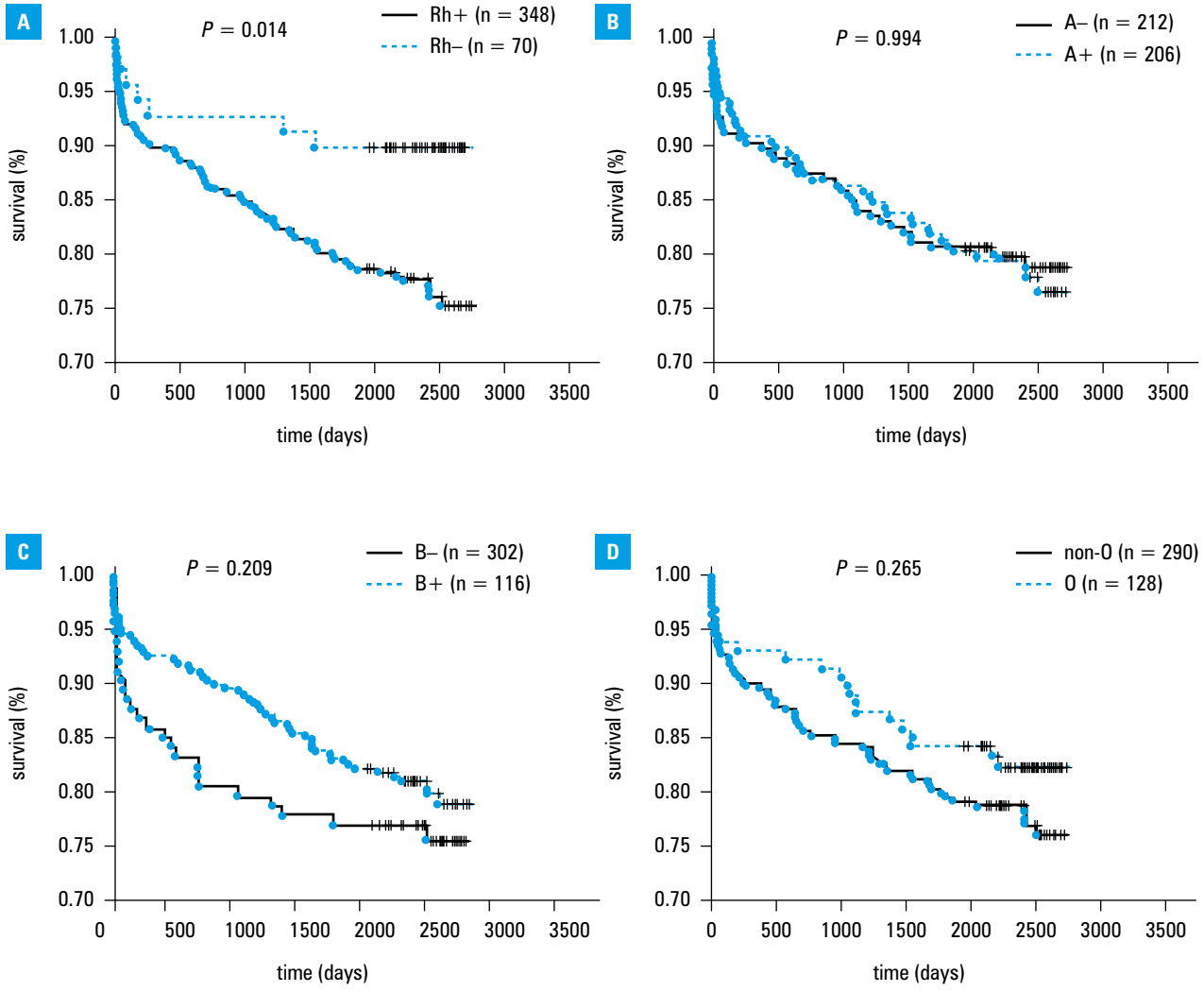

FIGURE 2 **A** – mortality in the groups with and without the D antigen (Rh factor); Rh+ represents the blood groups with and Rh– without the D antigen; **B** – mortality in the groups with and without the A antigen; A+ represents the blood groups with and A– without the A antigen; **C** – mortality in the groups with and without the B antigen; B+ represents the blood groups with and B– without the B antigen; **D** – mortality in patients with the O and non-O blood groups patients; non-O represents blood groups other than the O type and O represents the O blood group

groups. In our study there were no significant differences in baseline characteristics, procedural findings, and clinical events between patients with the non-O and O groups (data not shown).

Dentali et al.[20] conducted a systematic literature review and meta-analysis on the relationship between the ABO blood group and hemorrhage.[20] Interestingly, they found that group O was more frequent in the group of patients with bleeding that in controls (odds ratio, 1.33; 95% confidence interval, 1.25–1.42; P <0.001). They concluded that group O may be one of the important genetic factors for bleeding. In our study, we did not observe similar differences. The effect of Rh factor on mortality may be other than that of increasing the bleeding risk.

The Hoorn Study[21] revealed a 2-fold increase in cardiovascular mortality in patients with non-O group compared with those with O group during a 5-year follow-up. Our study reported all-cause mortality of 22.07% in non-O group compared with that of 17.19% in the O group during a 7-year follow-up, but it was not statistically significant. These discrepancies could possibly be explained by the fact that we evaluated mortality from different causes and applied different follow-up periods.

The Rh-positive group is characterized by the presence of Rh proteins on the erythrocyte membrane. Rh proteins are considered to be the channels for CO_2 and ammonia.[22] They may also be critical for the structure of the erythrocyte membrane. The Rh family of proteins now also includes nonerythroid Rh homologs, which can be found on the epithelial tissues in the kidneys, liver, brain, and skin.[23] Although the knowledge on the function of Rh proteins has developed recently, their complex function is still not fully understood. Some data point to coincidence of certain cardiovascular risk factors with the Rh blood group. Kanbay et al.[24] has described that Rh positivity is an independent risk factor for low levels of high-density lipoprotein cholesterol, a well-established risk factor for cardiovascular diseases. Another study reported an association of the RH genotype with systolic blood pressure and levels

of Apo-A, which remained significant after adjustment for age, sex, weight, and body mass index in Afro-Caribbeans.[25] In our study, hypertension was an independent correlate of 7-year mortality, but the presence of hypertension did not differ significantly between Rh+ and Rh– patients at the time of ACS (P = 0.89).

Our study showed that the Rh-positive factor may be a new potential factor in the global risk assessment in patients with ACS during long-term follow-up. However, the practical implementation of our results and possible treatment modification is an open question. Considering the fact that we confirmed the Rh factor to be a new independent risk factor for ACS, further research and assessment of the predictive value of this marker seems reasonable. At the moment, a treatment strategy depending on the Rh status does not seem entirely feasible and further research is needed to fully investigate this issue. Nonetheless, our results may be used to start a scientific discussion about potential differences in the coagulation system and anticoagulation effects of the used drugs. So far, it is simply a theoretical concept that needs to be verified in trials.

Our study has several limitations. First, we assessed only all-cause mortality, although it was the most important endpoint in our study. Second, the study population included a heterogeneous group of patients with STEMI, NSTEMI, and unstable angina, which, on the other hand, reflected real-life practice. Third, a relatively high percentage of the patients were treated conservatively (51 patients, 12.17%) without PCI. Moreover, the ejection fraction was not calculated. Finally, our study population was relatively small and the results should be confirmed in larger trials.

In conclusion, the presence of the Rh factor is associated with worse long-term prognosis in patients with ACS. To the best of our knowledge, this is the first study describing the effect of the Rh blood group on the prognosis of patients with ACS. However, this association needs to be verified in a multicenter, large-scale, prospective study.

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ARTYKUŁ ORYGINALNY

Związek między grupą krwi a rokowaniem po ostrym zespole wieńcowym

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SŁOWA KLUCZOWE

antygen D, czynnik Rh, ostry zespół wieńcowy, układ grupowy ABO

STRESZCZENIE

WPROWADZENIE Zidentyfikowano wiele czynników wpływających na rokowanie pacjentów po ostrym zespole wieńcowym (OZW). Jest mało danych dotyczących wpływu grupy krwi na rokowanie w tej grupie chorych.

CELE Celem badania była ocena wpływu grupy krwi w układzie ABO i układzie Rh (antygen D) na rokowanie po OZW.

PACJENCI I METODY Przeanalizowano grupę 418 kolejnych pacjentów hospitalizowanych z powodu OZW. Okres obserwacji wynosił 2075 ±794 dni. Pierwszorzędowym punktem końcowym była śmiertelność całkowita. Przeprowadzono analizę statystyczną w obrębie następujących podgrup: układ grupowy ABO, układ grupowy ABO łącznie z czynnikiem Rh (antygen D), pacjenci Rh dodatni *vs* Rh negatywni, pacjenci z grupą krwi 0 *vs* pacjenci z grupą krwi inną niż 0, pacjenci z grupą krwi z antygenem B *vs* pacjenci z grupą krwi bez tego antygenu, pacjenci z grupą krwi z antygenem A *vs* pacjenci z grupą krwi bez tego antygenu.

WYNIKI 348 pacjentów (83,25%) miało grupę krwi Rh-dodatnią, a 70 (16,75%) Rh-ujemną. W analizie przeżyć Kaplana–Meiera wykazano, że 7-letnia śmiertelność w grupie pacjentów z grupami krwi Rh-dodatnimi wyniosła 22,7%, a wśród chorych z grupami krwi Rh-ujemnymi – 10% ($p = 0,014$). Inne porównania nie osiągnęły poziomu istotności statystycznej. W analizie wieloczynnikowej Coxa zidentyfikowano czynnik Rh jako niezależny predyktor śmiertelności ogólnej (HR 7,758; 95% CI: 1,748–34,417; $p = 0,007$).

WNIOSKI Spośród badanych układów grupowych krwi jedynie czynnik Rh był niezależnym predyktorem śmiertelności u pacjentów z OZW.

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