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ORIGINAL ARTICLE

Does poorer self-rated health mediate the effect of Roma ethnicity on mortality in patients with coronary artery disease after coronaro-angiography?

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Abstract
Objectives The aim of this prospective cohort study was to assess the effect of Roma ethnicity and self-rated health (SRH) on 9-year all-cause mortality in patients with coronary artery disease (CAD) after coronaro-angiography (CAG), and whether SRH mediates the effect of ethnicity.
Methods 623 patients (103 Roma) were included. We obtained data from medical records and patients interviews. A Cox regression model adjusted for age, gender and education was used to analyze the effect of Roma ethnicity on mortality, as well as potential mediation by SRH.
Results Roma ethnicity and poor SRH were predictors of increased mortality in patients with CAD, with hazard rates (95 % confidence intervals) 2.34 (1.24; 4.42) and 1.81 (1.02; 3.21). Adding education decreased the size of ethnic differences in mortality. The mediating effect of SRH on the association of ethnicity with mortality was not statistically significant; neither modified ethnicity the effect of SRH.
Conclusions Poor SRH does not mediate the higher mortality among Roma patients after CAG even though it indicates an increased risk of mortality. Roma patients with CAD have to be referred for special cardiological care earlier.

Keyword Mortality · Coronary angiography · Coronary artery disease · Roma ethnicity · Self-rated health

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Introduction

Cardiovascular diseases (CVD) are the major cause of premature mortality and disability worldwide (Lozano et al. 2012; Murray et al. 2012). Coronary artery disease (CAD) as one of the two major forms of CVD is the single most common cause of death in Europe, accounting for 1.8 million deaths in Europe each year (Nichols et al. 2013). Death rates from CAD are generally higher in Central and Eastern Europe than in Northern, Southern and Western Europe (Nichols et al. 2013). CAD represents a huge burden on both patients and health care systems in each European country.

The incidence and prognosis of CAD differ by ethnicity within a country, Roma being one of the groups at risk. Over the last decades evidence has increased on ethnic inequalities in Roma population, which forms the biggest minority in Central and Eastern Europe (Masseria et al. 2010; Sudzinova et al. 2013; Babinska et al. 2013; Hajioff and McKee 2000; Janevic et al. 2012; Kohler and Preston 2011; Kolarcik et al. 2009; Kosa et al. 2007; Krajcovicova-Kudlackova et al. 2004; Nesvadbova et al. 2000; Sepkowitz 2006; Skodova et al. 2010; Vozarova de Courten et al. 2003; Zeljko et al. 2008; Zeman et al. 2003; Cook et al. 2013). An estimated 5 to 10 million Roma currently live in the EU, according to European Parliament Resolution of 31 January 2008 (Anonymous 2009). Estimates of the share of the Roma in the Slovak population vary from 2.0 % according to the last Population and house census in

the Slovak Republic in 2011 (The Statistical Office of the Slovak Republic 2012) to 7.2 % (approximately 380,000 Roma) according to the Institute of Informatics and Statistics of the Slovak Republic (Vano 2001). Previous studies found an increased mortality and worse health status in Roma population (Masseria et al. 2010; Sudzinova et al. 2013; Babinska et al. 2013; Kohler and Preston 2011; Kosa et al. 2007; Bogdanovic et al. 2007; Jarcuska et al. 2013; Voko et al. 2009). Only a part of this unfavorable health outcome can be explained by their lower socioeconomic status (Kolarcik et al. 2009).

Self-rated health (SRH) is an independent predictor of increased mortality in patients with known CAD (Bosworth et al. 1999; Keeley and Driscoll 2010; Schenkeveld et al. 2010; Xie et al. 2008). Poor SRH after adjustment for relevant factors was shown to be associated with significantly higher mortality in this group of patients (Bosworth et al. 1999). Roma patients with coronary heart disease showed poorer health-related quality of life when compared to their non-Roma counterparts (Kosa et al. 2007; Skodova et al. 2010; Silarova et al. 2014). However, the association of SRH and Roma ethnicity on all-cause mortality in CAD patients was not yet studied. Therefore, the aim of this study was to assess the effect of Roma ethnicity and SRH on nine-year all-cause mortality in patients with CAD after coronary angiography (CAG), and whether SRH mediates the effect of ethnicity.

Methods

Patients

A total of 1010 consecutive adult Roma and non-Roma patients who underwent routine elective CAG at the East Slovakian Institute for Cardiac and Vascular Diseases in Kosice, Slovakia, in the years 2004–2013 were asked to participate in our study (Fig. 1). The inclusion criteria were being referred for CAG and age under 75 years. We excluded 346 patients based on the exclusion criteria—diagnosis of severe cognitive impairments, psychiatric disorder in the medical history, normal CAG, infectious endocarditis, acute myocardial infarction, severe valve disease, severe atrial or ventricular septal defect. Of the eligible patients, 41 (4.2 %) refused to participate, leading to inclusion of 623 participants in the study (response rate 95.8 %) (see Fig. 1).

The study was approved by the Ethics Committee of the East Slovakian Institute for Cardiac and Vascular Diseases in Kosice in November 2004. All participants were provided with information about the study and signed an informed consent statement prior to the study.

Procedures and measures

Data collection consisted of an interview conducted by a psychologist or trained research assistant with each participant during hospitalization and extraction of data from medical records. Sociodemographic data included age, gender, education and ethnicity. We categorized education into three categories—low (elementary school or secondary school without school leaving exam), middle (secondary school with school leaving exam) and high (university degree). Ethnicity was measured based on each patient's declaration and identification by the doctor. In case of conflicting opinions, an independent third person (a head-nurse) was decisive.

Clinical data were retrieved from the medical records. These included disease history, use of drugs and type of treatment after CAG. Disease history concerned previous myocardial infarction, arterial hypertension, diabetes mellitus, dyslipidaemia and the use of acetylsalicylic acid (ASA), clopidogrel, beta-blockers, statins, nitrates, anxiolytics and non-steroid anti-inflammatory drugs (NSAIDs). We also asked about smoking status (smoker or non-smoker) and alcohol use (alcohol consumption yes or no). Levels of total, low-density lipoprotein (LDL) and high-density lipoprotein (HDL) cholesterol and triglycerides were registered according to laboratory findings. The ejection fraction was measured by ultrasound using either the Simpson or the occulometric method. The type of treatment following the CAG concerned conservative pharmacological treatment, percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG). This was decided on by cardiologists based on the results of CAG and the patient's clinical status, independently of participation in this study. The severity of the CAD was evaluated using the Canadian Cardiovascular Society (CCS) and New York Heart Association (NYHA) classifications. CCS identifies the severity of chest pain in 4 grades, NYHA classifies dyspnea symptoms in 4 grades. In both scales, a higher score represents worse symptomatology.

SRH was measured using the first question of the Short Form Health Survey (SF-36). The answer options 1 (excellent), 2 (very good), 3 (good), 4 (fair) and 5 (poor) were transformed into two categories—good (1 + 2 + 3) and poor (4 + 5). The validity and reliability of the SF-36 scale have been established in patients with CAD (Failde and Ramos 2000, pp 359–365).

Data on patient mortality during 9 years after CAG were obtained from the Central Registry of the Health Care Surveillance Authority of the Slovak Republic.

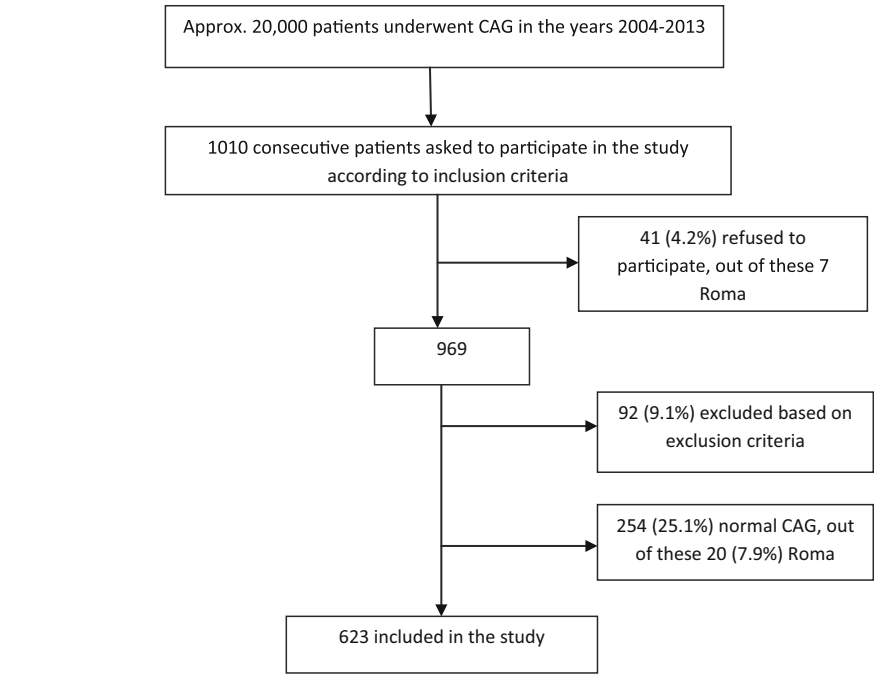

Fig. 1 Flowchart of the study population

CAG – coronary angiography

Statistical analyses

As the first step, we assessed background and clinical characteristics of the Roma and non-Roma patients. Differences were statistically tested using the t test for continuous variables and χ^2 or Fisher exact tests, when appropriate, for categorical variables. Values of $p < 0.05$ were considered statistically significant.

Next, we analyzed the association of Roma ethnicity and SRH on all-cause mortality in CAD patients using a three-step hierarchical Cox regression model. Model 1 tested the effect of age, age^2 (Chen et al. 2007), gender and ethnicity; Model 2 added education; Model 3 all above and SRH and Model 4 all above and smoking. The proportional hazards assumptions were tested using a time varying covariate. Possible mediation between ethnicity and SRH was tested according to Baron and Kenny (Baron and Kenny 1986). All statistical analyses were performed using the statistical software IBM SPSS 20.0 for Windows (IBM company, Chicago, Illinois, USA) and Mplus 7.1 (Muthen&Muthen, Los Angeles, USA).

Results

Baseline characteristics of the study population are given in Table 1. There were 103 (16.5 %) Roma and 520 (83.5 %) non-Roma patients included in our study. Roma patients with CAD were significantly younger, with lower education level and smoked more frequently. No significant difference was found regarding SRH. They had more frequently a positive history of previous myocardial infarction and were more symptomatic according to CCS classes of angina pectoris classification. NYHA classes and ejection fraction were similar in both groups of patients. The Roma patients used significantly more frequently clopidogrel and NSAIDs. Regarding type of treatment after CAG, we found no significant differences between both groups of patients. We did not observe any conflicting opinions in the ethnicity measurements.

During the 9-year follow-up period 82 (13.2 %) patients died, 536 survived (86.0 %) and 5 were lost to follow-up. Cox regression models adjusted for age, age^2 and gender showed that Roma ethnicity significantly predicted an increased 9-year all-cause mortality (hazard ratio (HR) 2.70, 95 % confidence intervals (CI) 1.46; 4.96) (see Table 2). Adding education decreased the size of ethnic differences in mortality (HR 2.37, 95 % CI 1.26; 4.49). Adjustment for SRH did hardly affect the effect of ethnicity and of SRH (HR for Roma ethnicity 2.34, 95 % CI 1.24; 4.42 and HR for SRH 1.81, 95 % CI 1.02; 3.21) (see Table 2). The test of the proportional hazards assumption yielded a p value >0.05 showing that the two mortality curves behave proportionally (see Fig. 2).

Additional analyses on mediation did not show any significant mediating effect of SRH regarding the association of ethnicity and mortality, with no reduction of the hazards for ethnicity due to introduction of SRH.

Table 1 Baseline characteristics of the study population by ethnicity ($N = 623$)

	Number of Roma patients with valid data	Roma ($N = 103$)	Number of non-Roma patients with valid data	Non-Roma ($N = 520$)	Significance of difference
Sociodemographic data					
Age (years, mean ± SD)	103	**53.8 ± 6.8**	520	**58.9 ± 7.3**	***p* < 0.0001**
Age range (years)		35–73		32–75	
Age category distribution					
Up to 45		9 (8.7 %)		22 (4.2 %)	
46–60		77 (74.8 %)		277 (53.3 %)	
61–75		17 (16.5 %)		221 (42.5 %)	
Gender (male)	103	76 (73.8 %)	520	367 (70.6 %)	ns
Education	103		520		***p* < 0.0001**
Low education		**100 (97.1 %)**		**286 (55.0 %)**	
Middle education		**2 (1.9 %)**		**88 (16.9 %)**	
High education		**1 (1.0 %)**		**146 (28.1 %)**	
Self-rated health	76		482		ns
Poor		59 (77.6 %)		323 (67.0 %)	
Good		17 (22.4 %)		159 (33.0 %)	
BMI (kg/m^2)	102	30.1 ± 5.4	507	29.7 ± 12.8	ns
History of disease					
Previous myocardial infarction	103	**68 (66.0 %)**	520	**271 (52.1 %)**	***p* = 0.012**
NYHA III–IV	82	17 (20.7 %)	349	77 (22.1 %)	ns
CCS IV	94	**12 (12.8 %)**	441	**26 (5.9 %)**	***p* = 0.026**
Dyslipoproteinaemia	103	66 (64.1 %)	519	332 (64.0 %)	ns
Arterial hypertension	103	**79 (76.7 %)**	517	**444 (85.9 %)**	***p* = 0.025**
Diabetes mellitus	103	34 (33.0 %)	520	190 (36.5 %)	ns
Smoking	102	**34 (33.3 %)**	519	**55 (10.6 %)**	***p* < 0.0001**
Alcohol	103	**61 (59.2 %)**	520	**367 (70.6 %)**	***p* = 0.027**
Use of drugs					
ASA	101	81 (80.2 %)	518	385 (74.3 %)	ns
Clopidogrel	101	**48 (47.5 %)**	518	**134 (25.9 %)**	***p* < 0.0001**
Beta-blockers	101	82 (81.2 %)	518	435 (84.0 %)	ns
Statins	101	72 (71.3 %)	518	388 (74.9 %)	ns
Nitrates	101	68 (67.3 %)	518	331 (63.9 %)	ns
Anxiolytics	101	9 (8.9 %)	518	25 (4.8 %)	ns
NSAIDs	101	**49 (48.5 %)**	472	**113 (23.9 %)**	***p* < 0.0001**
Laboratory findings (mean ± SD)					
Total cholesterol (mmol/l)	92	4.80 ± 1.31	448	4.93 ± 1.30	ns
HDL cholesterol (mmol/l)	88	1.09 ± 0.79	417	1.26 ± 0.67	ns
LDL cholesterol (mmol/l)	83	2.81 ± 1.09	405	2.91 ± 1.38	ns
Triglycerides (mmol/l)	90	2.36 ± 1.43	439	2.02 ± 1.66	ns
Ejection fraction (%, mean ± SD)	94	49.0 ± 11.2	487	50.2 ± 9.7	ns
Type of treatment					
Pharmacotherapy	103	42 (40.8 %)	520	167 (32.1 %)	ns
PCI/stent	103	34 (33.0 %)	520	196 (37.7 %)	ns
CABG	103	27 (26.2 %)	520	157 (30.2 %)	ns

All figures are numbers and proportions unless otherwise indicated. Kosice, Slovakia, 2014

All statistically significant differences are indicated in bold

ns not statistically significant, *SD* standard deviation, *BMI* body mass index, *ASA* acetylsalicylic acid, *NSAIDs* non-steroid anti-inflammatory drugs, *HDL* high-density lipoprotein, *LDL* low-density lipoprotein, *PCI* percutaneous coronary intervention, *CABG* coronary artery bypass grafting

Table 2 Results of Cox regression analysis of nine-year all-cause mortality adjusted for gender, age, age^2, ethnicity, education, self-rated health (SRH) and smoking in a sample of Roma and non-Roma patients with coronary artery disease: hazard ratio (HR), 95 % confidence intervals (CI) ($N = 551$) and significance of the model change (smc)

	HR	95 % CI	smc
Model 1 ($\chi^2 = 12.58$)$^{p = 0.014}$			
Ethnicity (Roma vs. non-Roma)	**2.70**	**(1.47; 4.96)**$^{p=0.001}$	
Age	0.94	(0.65; 1.38)	
Age^2	1.00	(1.00; 1.00)	
Gender (male vs. female)	1.10	(0.64; 1.88)	
Model 2 ($\chi^2 = 14.37$$^{p = 0.013}$)			$p = 0.159$
Ethnicity (Roma vs. non-Roma)	**2.35**	**(1.25; 4.43)**$^{p=0.008}$	
Age	0.96	(0.66; 1.40)	
Age^2	1.00	(1.00; 1.00)	
Gender (male vs. female)	1.15	(0.66; 1.98)	
Education (low vs. middle and high)	1.44	(0.86; 2.40)	
Model 3 ($\chi^2 = 17.88$$^{p = 0.007}$)			**$p = 0.044$**
Ethnicity (Roma vs. non-Roma)	**2.31**	**(1.23; 4.36)**$^{p=0.01}$	
Age	0.96	(0.65; 1.41)	
Age^2	1.00	(1.00; 1.00)	
Gender (male vs. female)	1.20	(0.69; 2.08)	
Education (low vs. middle and high)	1.38	(0.83; 2.31)	
SRH (poor vs. good)	1.75	(0.99; 3.10)	
Model 4 ($\chi^2 = 21.95$$^{p = 0.003}$)			$p = 0.066$
Ethnicity (Roma vs. non-Roma)	**2.14**	**(1.13; 4.06)**$^{p=0.02}$	
Age	0.96	(0.67; 1.40)	
Age^2	1.00	(1.00; 1.00)	
Gender (male vs. female)	1.14	(0.66; 1.98)	
Education (low vs. middle and high)	1.34	(0.80; 2.25)	
SRH (poor vs. good)	1.72	(0.97; 3.05)	
Smoking (yes vs. no)	1.85	(0.99; 3.46)	

Kosice, Slovakia, 2014

All statistically significant differences are indicated in bold

Model 1 adjusted for age, age^2, gender and ethnicity

Model 2 adjusted for age, age^2, gender, ethnicity and education

Model 3 adjusted for age, age^2, gender, ethnicity, education and SRH

Model 4 adjusted for age, age^2, gender, ethnicity, education, SRH and smoking

HR hazard ratio, *CI* confidence interval for HR, *smc* significance of the model change, *SRH* self-rated health

Discussion

The aim of this study was to assess the effect of Roma ethnicity and SRH on nine-year all-cause mortality in patients with CAD confirmed by CAG and to assess whether SRH mediates the effect of ethnicity. A more than 2 times higher mortality in Roma patients with CAD persisted after adjustment for age, age^2, gender, education, SRH and smoking. SRH by itself was a predictor of mortality in Roma CAD patients, but did not mediate the effect of Roma ethnicity on mortality.

In previous studies, which were rather descriptive, a higher mortality of Roma was shown (Kohler and Preston 2011; Bogdanovic et al. 2007; Rosicova et al. 2011). The current study confirmed that Roma ethnicity was a strong predictor of all-cause mortality, too. Despite the significantly lower educational level in Roma patients with CAD, the ethnic differences in all-cause mortality remained after adjustment for education. This is in contrast with the previously described explanations of the differences in health status and prognosis of Roma patients by the lower SES of the Roma minority (Kohler and Preston 2011; Voko et al. 2009).

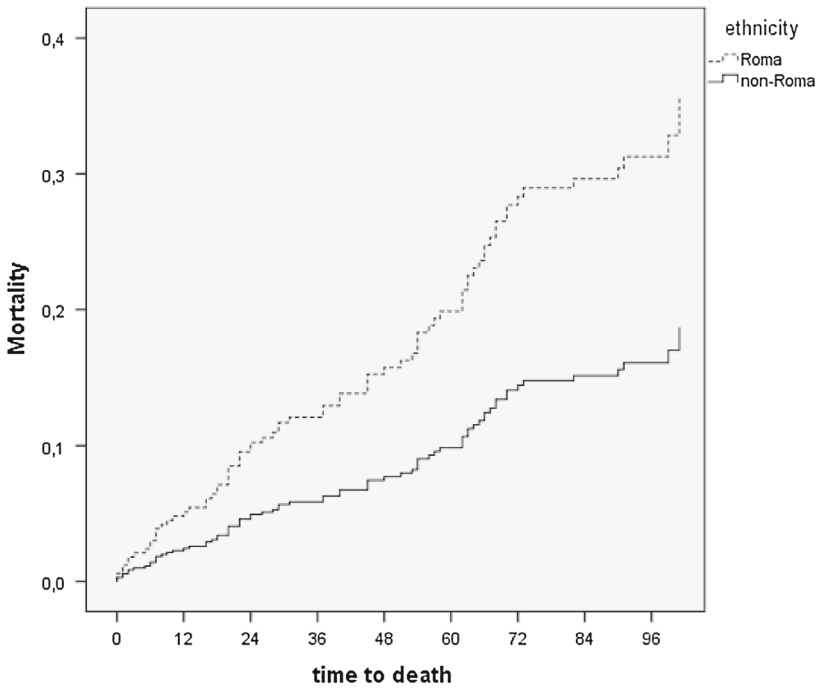

Fig. 2 Kaplan–Meier curve of Roma and non-Roma patients' mortality during the eight-year follow-up

Number of surviving patients	1 year	2 years	3 years	4 years	5 years	6 years	7 years	8 years
Roma (73)	72	68	66	64	63	60	59	59
Non-Roma (478)	462	451	448	442	435	423	422	418

The association of SRH and Roma ethnicity on all-cause mortality in CAD patients was not yet studied before. We assumed that SRH could be a possible mediator of the increased all-cause mortality in Roma patients, but this association was not confirmed in our study. We found a trend towards poorer SRH in Roma, but the difference was not significant, which is in contrast with poorer SRH in Roma described in previous studies (Masseria et al. 2010; Janevic et al. 2012; Skodova et al. 2010; Voko et al. 2009). Regarding studies on Roma ethnicity, mortality and CAD, we did not find any other study exploring the association of SRH, ethnicity and mortality in such a group of patients. The lack of a mediating effect of SRH on the association of Roma ethnicity with mortality indicates that Roma patients with CAD and a poor SRH have an even higher risk of death in long-term outcome.

Our finding of an increased Roma mortality may be related to differences in baseline variation in risk profiles of the Roma and non-Roma with CAD. A worse risk profile regarding CVD was shown in previous studies (Sudzinova et al. 2013; Babinska et al. 2013; Krajcovicova-Kudlackova et al. 2004; Vozarova de Courten et al. 2003; Zeljko et al. 2012). We might consider more severe forms of CAD at the time of CAG, as shown by the difference in CCS IV classes, more frequent history of the previous myocardial infarction and higher use of clopidogrel. In addition, significantly more smokers among Roma existed, which is an important risk factor of CAD. This at average worse general CAD risk profile of Roma may lead to a higher mortality after treatment, too.

On the other hand, this increased mortality among Roma may also be due to delays in their access to adequate health care because they experience barriers in the health care system (Jarcuska et al. 2013). The resulting longer delay between symptom onset and treatment among Roma may decrease their survival. The delay at entry to specialized cardiological care, as shown by worse health status of the Roma patients with more severe symptoms and more frequent positive history of myocardial infarction at younger age when coming for CAG, seems to be the factor that contributes most to the increased mortality. Barriers contributing to poorer access may be the lower educational level of the Roma population and low health literacy. Other barriers that hamper a proper access to care may be the perception of health care professionals that the Roma are aggressive patients, probably due to a low understanding of the health care process, a worse adherence to secondary prevention measures and medical therapy, and less

willingness among Roma to be hospitalized due to their cultural beliefs and discrimination may play an important role as well (Belak 2013). Low-educated Roma patients might also have lower compliance to medical therapy, which could negatively influence their survival.

Strengths and limitations of the study

The strengths of this study are its high response rate (93.8 %), the long observation period and the very low loss to follow-up over the years. It is a longitudinal, single major cardiac center study dealing with unique long-term data on specific Roma ethnicity mortality.

One of the limitations may be the lack of information on the exact cause of death in patients, making it impossible to distinguish between cardiac and non-cardiac mortality. Moreover, SES was determined only by education, which is just one aspect of SES, although one has to consider that Roma score similarly low in household income and occupation (Filadelfiova et al. 2007). Another limitation of this study is a possible bias in the traditional approach in mediation analysis of looking for direct and indirect effects of the given set of mediators by adjusting for the mediator in our regression model. Under certain circumstances this traditional approach may produce flawed conclusions (Richiardi et al. 2013; Tchetgen Tchetgen 2011; VanderWeele 2011). One of the limitations is the relatively small sample size of the Roma group.

Implications

We found that all-cause mortality after CAG is higher among Roma than among non-Roma patients with CAD. Poor SRH does not mediate this higher mortality even though it indicates an increased risk of mortality as well. Roma ethnicity may imply a more aggressive approach to secondary prevention of CAD and its complications like acute myocardial infarction or heart failure. Results must be verified in a larger, multicenter, cohort to allow for generalization. Furthermore, the pathways between psychological, physical and medical determinants associated with Roma ethnicity, SRH and mortality have to be studied in the future. This may offer clues to counteract the increased death rates among Roma.

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Compliance with ethical standards

Conflict of interest The authors declare no conflict of interests.

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