Relationship of Socioeconomic Status to Arterial Stiffness: Comparison Between Medical Aid Beneficiaries and National Health Insurance Beneficiaries

Hack-Lyoung Kim,[1] Jin Yong Lee,[2] Woo-Hyun Lim,[1] Jae-Bin Seo,[1] Sang-Hyun Kim,[1] Joo-He Zo,[1] and Myung-A Kim[1]

BACKGROUND
There is no general agreement on underlying pathophysiology explaining the high burden of cardiovascular disease on people at low socioeconomic status (SES). This study was conducted to investigate the association between healthcare systems and arterial stiffness.

METHODS
A total of 8,929 subjects (60 years old and 55% were male) who underwent brachial-ankle pulse wave velocity (baPWV) measurement were retrospectively analyzed. There were 8,237 National Health Insurance (NHI) beneficiaries (92.2%) and 692 medical aid (MA) beneficiaries (7.8%). The median value of baPWV was 1,540 cm/s.

RESULTS
Subjects with higher baPWV values (≥1,540 cm/s) were older, and more frequently had cardiovascular risk factors and unfavorable laboratory findings than those with lower values baPWV (<1,540 cm/s). The baPWV values were significantly higher in MA beneficiaries than in NHI beneficiaries (1,966 ± 495 vs. 1,582 ± 346 cm/s, $P < 0.001$). The proportion of MA beneficiaries was significantly higher in subjects with higher baPWV than those with lower baPWV (13.1% vs. 2.3%, $P < 0.001$). In multivariable analysis, MA beneficiaries were significantly associated with higher baPWV values even after controlling for potential confounders (odds ratio, 5.41; 95% confidence intervals, 4.02–7.27; $P < 0.001$).

CONCLUSIONS
The baPWV values were significantly higher in MA beneficiaries than in NHI beneficiaries. The result of this study provides additional evidence on the association between low SES and arterial stiffening.

Keywords: arterial stiffness; blood pressure; healthcare system; hypertension; medical aid; National Health Insurance; socioeconomic status

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Cardiovascular disease (CVD) is the leading cause of death worldwide.[1] In spite of the decline in mortality in recent years, the burden of CVD on our society is still huge.[2,3] In order to improve clinical outcome associated with CVD, risk factors such as hypertension, diabetes mellitus, dyslipidemia, smoking, and obesity have been identified, and widely used as a guidance in risk stratification and management.[4,5] However, these traditional risk factors do not account for all cardiovascular risks.[4] Many studies have suggested that low socioeconomic status (SES) is associated with increased CVD risk.[6–8] However, there is no general agreement on underlying pathophysiology explaining the high burden of CVD in people with low SES. Elucidating the exact mechanism is very important, because it could facilitate the effort to develop better prevention measures.

Arterial stiffening is a marker for high cardiovascular risk, independent of traditional risk factors.[9–13] To date, there has been limited evidence on how arterial stiffness is affected by SES. We hypothesized that arterial stiffness would play an important role in mediating the relationship between low SES and high CVD risk. Therefore, this study was conducted to investigate the association between SES and arterial stiffness.

METHODS

Study subjects

This single center study was performed at a general hospital in Seoul, South Korea. Between January 2010 and December 2016, consecutive subjects between the ages of 18 and 90 who visited our hospital and underwent the measurement of brachial-ankle pulse wave velocity (baPWV) were eligible for this study. The baPWV is measured as a

Correspondence: Hack-Lyoung Kim (khl2876@gmail.com).

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[1]Division of Cardiology, Department of Internal Medicine, Boramae Medical Center, Seoul National University College of Medicine, Seoul, Republic of Korea; [2]Department of Public Health and Community Medicine, Boramae Medical Center, Seoul National University College of Medicine, Seoul, Republic of Korea

part of the routine cardiovascular examinations for stable patients at the study hospital. To ensure the reliability of baPWV results, subjects with the following conditions were excluded: (i) unstable vital signs; (ii) recently aggravated chest pain, dyspnea, and palpitation; (iii) low left ventricular ejection fraction <50%; (iv) regional wall motion abnormality of left ventricle; (v) significant valvular stenosis or regurgitation more than mild degree; (vi) pericardial effusion; (vii) atrial fibrillation; and (viii) ankle-brachial index <0.9 or >1.4. This study was performed in accordance with the Helsinki Declaration. Approval for the study protocol was obtained from the Institutional Review Board (IRB) of Boramae Medical Center (Seoul, South Korea) (IRB number, 10-2019-40). Informed consent was waived by IRB due to the routine nature of the information collected and retrospective study design.

Healthcare systems in South Korea

In South Korea, the National Health Insurance (NHI) system covers almost all Koreans since 1989.[14] There is also the medical aid (MA) system, a public assistance program that provides healthcare benefits to low-income families, such as those who were qualified through the National Basic Livelihood Security System.[15] NHI operates on NHI premiums collected from beneficiaries and government subsidies. MA is an option for Koreans who are unable to pay NHI premiums, very similar to Medicaid in the United States. MA beneficiaries had low levels of education (≤elementary: 60.9% vs. 39.9%) and household income (low ordinary income: 94.8% vs. 33.6%), and the proportion of those with no economic activity was significantly higher (81.7% vs. 57.1%), compared with NHI beneficiaries.[16] The MA system is tax operated. Recipients are exempt from premiums and use medical institutions free of charge or as a reduction in medical costs. Korea has secondary or private insurance, but its role is very limited. NHI and MA cover most of the healthcare costs, and private insurance only serves to reduce deductibles or out-of-pocket costs or services not covered by NHI.[17] In 2019, MA beneficiaries accounted for 2.9% of all Koreans in 2017, while other Koreans (97.1%) were NHI beneficiaries.[18,19] Since the hospital that conducted this study was a metropolitan government hospital, the proportion of MA beneficiaries in this study was about 7.8%, much higher than the average.

Clinical data collections

Medical records of the enrolled patients were retrospectively reviewed. Body mass index was calculated as weight in kilogram divided by height in square meters (kg/m^2). Information on history of cardiovascular risk factors, such as hypertension, diabetes mellitus, and dyslipidemia, was obtained. Hypertension was defined as systolic blood pressure ≥140 mm Hg and/or diastolic blood pressure ≥90 mm Hg or use of antihypertensive medications at the time of admission. Diabetes mellitus was defined as serum fasting glucose ≥126 mg/dl in repeated measurements or use of oral hypoglycemic agents or insulin at the time of admission. Dyslipidemia was defined as having low-density lipoprotein (LDL) ≥160 mg/dl or taking any lipid-lowering medication, or previously diagnosed. Patients with a history of smoking within the past 12 months were considered smokers. All blood samples were collected after overnight fasting. Blood levels of white blood cell count, hemoglobin, glucose, glycated hemoglobin, creatinine, total cholesterol, LDL cholesterol, high-density lipoprotein cholesterol, triglyceride, and C-reactive protein were assessed. Estimated glomerular filtration rate was calculated using 4-component Modification of Diet in Renal Disease (MDRD) Study equation in incorporating age, race, sex, and serum creatinine level. Information on cardiovascular medications, including calcium channel blocker, beta-blocker, renin–angiotensin system (RAS) blocker, and statin, was also obtained.

baPWV measurement

The methods for baPWV measurement were previously described.[20,21] The baPWV was measured using the volume-plethysmography (VP-1000; Colins, Komaki, Japan). Cigarette smoking, and drinking of alcohol and caffeine containing beverage were restricted, but regular medications were allowed at the day of examination. Before the test, in a quiet and isolated space, the patient lay in bed and rested for >5 minutes. Blood pressure cuffs were wrapped around both the upper arms and ankles, blood pressures, heart rates, pulse volume waveforms, and phonograms were measured simultaneously. The baPWV value was calculated by estimating the differences in pulse wave time between the brachial and posterior tibial arteries. The average value of right and left baPWV was used in this study analysis. The baPWV measurement was performed by 1 experienced operator. The coefficient of variation in baPWV measurement for intraobserver variability was 5.1% in our laboratory.[22]

Statistical analysis

Continuous variables are expressed as mean ± SD, and categorical variables are expressed as percentages. In the comparisons between the 2 groups, Student's *t* test was used for continuous variables, and chi-square test was used for categorical variables. Correlations between age and baPWV values were assessed using Pearson's correlation method. Multivariable logistic regression analysis was performed to find out factors associated with higher baPWV values. A *P* value of <0.05 was considered statistically significant. All statistical analyses were performed using SPSS 22.0 (IBM, Armonk, NY).

RESULTS

A total of 8,929 subjects were analyzed in this study. Of these patients, 8,237 (92.2%) were NHI beneficiaries and 692 (7.8%) were MA beneficiaries. The mean age of total population was 60.3 ± 12.8 years and 54.9% were male.

The clinical characteristics of the study subjects are shown in Table 1. MA beneficiaries were older, and more frequently had cardiovascular risk factors including hypertension and

Table 1. Clinical characteristics of NHI and MA beneficiaries

Characteristic`	NHI (*n* = 8,237)	MA (*n* = 692)	*P* value
Age, years	60.0 ± 12.7	64.4 ± 12.4	<0.001
Female sex	3,745 (45.1)	318 (45.2)	0.947
Body mass index, kg/m^2	24.8 ± 3.4	24.6 ± 3.9	0.091
Systolic blood pressure, mm Hg	130 ± 18	132 ± 20	0.008
Diastolic blood pressure, mm Hg	77.3 ± 12.7	76.1 ± 14.9	0.028
Heart rate, per minute	70.0 ± 12.4	72.7 ± 13.7	<0.001
Cardiovascular risk factors			
Hypertension	3,605 (43.4)	443 (63.0)	<0.001
Diabetes mellitus	1,749 (21.1)	304 (43.2)	<0.001
Cigarette smoking	903 (10.9)	152 (21.6)	<0.001
Coronary artery disease	182 (2.2)	21 (3.0)	0.173
Stroke	56 (0.7)	6 (0.9)	0.851
Laboratory findings			
White blood cell count, per μl	6,801 ± 3,120	7,679 ± 4,576	<0.001
Hemoglobin, g/dl	13.6 ± 1.8	12.5 ± 2.0	<0.001
Fasting glucose, mg/dl	119 ± 36	132 ± 54	<0.001
HbA1c, %	6.33 ± 1.11	6.70 ± 1.28	<0.001
eGFR, ml/min/1.73 m^2	86.0 ± 26.3	73.4 ± 35.7	<0.001
Total cholesterol, mg/dl	161 ± 39	149 ± 38	<0.001
LDL cholesterol, mg/dl	93.0 ± 32.9	82.4 ± 30.4	<0.001
HDL cholesterol, mg/dl	50.0 ± 12.8	47.6 ± 14.2	<0.001
Triglyceride, mg/dl	126 ± 74	131 ± 69	0.116
C-reactive protein, mg/dl	0.87 ± 2.90	1.94 ± 4.57	<0.001
Cardiovascular medications			
Calcium channel blocker	1,894 (22.8)	126 (17.9)	0.003
Beta-blocker	1,479 (17.8)	157 (22.3)	0.003
RAS blocker	2,150 (25.9)	140 (19.9)	<0.001
Statin	3,333 (40.1)	275 (39.1)	0.595

Numbers are expressed as mean ± SD or *n* (%). Abbreviations: eGFR, estimated glomerular filtration rate; HDL, high-density lipoprotein; LDL, low-density lipoprotein; MA, medical aid; NHI, national health insurance; RAS, renin–angiotensin system.

diabetes mellitus. In laboratory findings, blood levels of white blood cell count, fasting glucose, glycated hemoglobin, and C-reactive protein were significantly higher, and blood levels of estimated glomerular filtration rate, total cholesterol, LDL cholesterol, and high-density lipoprotein cholesterol were significantly lower in MA beneficiaries than in NHI beneficiaries. Calcium channel blocker and RAS blocker were more frequently prescribed in NIH beneficiaries, and beta-blocker was more frequently prescribed in MA beneficiaries.

The distribution of baPWV of this study population is shown in Figure 1. The mean value of baPWV was 1,611 ± 373 cm/s, median value was 1,540 cm/s, and interquartile range was 1,365–1,777 cm/s. As previously presented,[23] baPWV correlated well with age (r = 0.492, P < 0.001) (Figure 2). Table 2 demonstrates the clinical characteristics of the study subjects according to baPWV values. Since the baPWV cutoff value applicable to our study population is not well known, the median value of baPWV (n = 1,540 cm/s) was used to divide it into 2 groups. Subjects with higher baPWV values (≥1,540 cm/s) were older and more frequently female than those with lower baPWV values (<1,540 cm/s). Subjects with higher baPWV more frequently had cardiovascular risk factors including hypertension and diabetes mellitus. In laboratory findings, blood levels of white blood cell count, fasting glucose, glycated hemoglobin, and C-reactive protein were significantly higher, and blood levels of hemoglobin, estimated glomerular filtration rate, total cholesterol, LDL cholesterol, and high-density lipoprotein cholesterol were significantly lower in subjects with higher baPWV values than those with lower baPWV values. Cardiovascular medications, including calcium channel blocker, beta-blocker, RAS blocker, and statin, were more frequently prescribed in subjects with higher baPWV values than those with lower baPWV values.

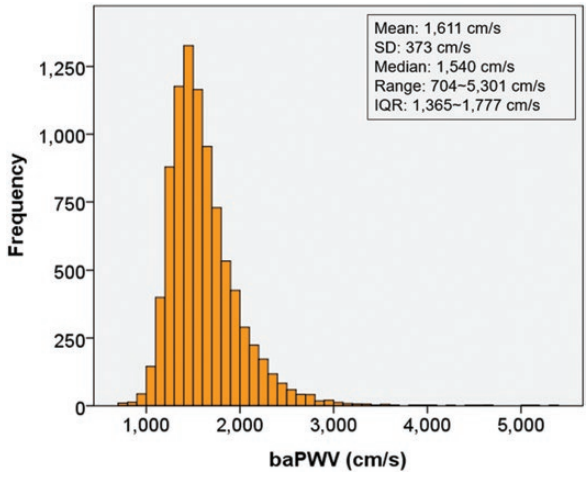

Figure 1. Distribution of baPWV values. Abbreviations: baPWV, brachial-ankle pulse wave velocity; SD, standard deviation; IQR, interquartile range.

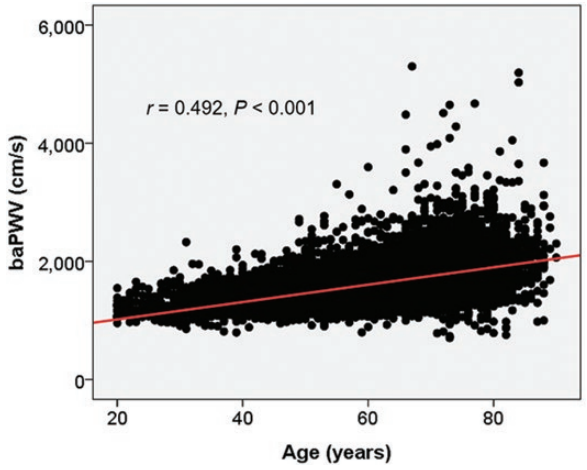

Figure 2. Correlation between age and baPWV. Abbreviation: baPWV, brachial-ankle pulse wave velocity.

The baPWV values were significantly higher in MA beneficiaries than in NHI beneficiaries (1,966 ± 495 vs. 1,582 ± 346 cm/s, $P < 0.001$) (Figure 3). The proportion of MA beneficiaries was significantly higher in subjects with higher baPWV values than in those with lower baPWV values (13.1% vs. 2.3%, $P < 0.001$) (Figure 4). Independent predictors for higher baPWV are shown in Table 3. MA beneficiaries are significantly associated with higher baPWV even after controlling for potential confounders including, age, sex, blood pressure, heart rate, diabetes mellitus, cigarette smoking, estimated glomerular filtration rate, LDL cholesterol, C-reactive protein, and the use of cardiovascular medications (odds ratio, 5.41; 95% confidence intervals, 4.02–7.27; $P < 0.001$).

DISCUSSION

The main findings of our study were: (i) MA beneficiaries were older, and more frequently had cardiovascular risk factors and unfavorable laboratory findings than those NHI beneficiaries; (ii) the baPWV values were significantly higher in MA beneficiaries; (iii) the proportion of MA was significantly higher in patients with higher baPWV values; and (iv) MA was independently associated with higher baPWV.

A few studies have addressed the association between SES and arterial stiffness. Din-Dzietham *et al.*[24] investigated 268 African Americans and 2,459 whites, and showed that the β stiffness index of the carotid artery was 9% higher in African Americans than in whites. They suggested that higher prevalence of cardiovascular risk factors and lower SES in African Americans might be attributed to the ethnic difference in arterial stiffness between the groups.[24] Similarly, another study reported that African Americans and lower SES adolescents had a greater arterial stiffness compared with Caucasians and higher SES adolescents.[25] In a study of 7,803 adults in France, lower levels of education and occupation were adversely related to carotid stiffness, independently of traditional risk factors.[26] A Japanese study showed that the SES gradient, especially the educational level, was significantly associated with baPWV in men.[27] In a longitudinal study, aortic pulse wave velocity (PWV) increase over 5 years was higher among participants with lower employment grade, household income, and education.[28] Puolakka *et al.*[29] demonstrated that higher family SES in childhood was associated with lower arterial stiffness in adulthood. In line with these studies, our studies also showed that compared with NHI beneficiaries, MA beneficiaries were independently associated with higher baPWV values.

Although the underlying pathophysiology of why people with low SES have high arterial stiffness is not well known, several hypotheses can be proposed. As shown in our study, MA beneficiaries are older and more have cardiovascular risk factors, which are factors that increase arterial stiffness.[30] There was also a possibility that MA beneficiaries received less adequate medical treatment to control risk factors than NHI beneficiaries. Indeed, the treatment rate for high blood pressure was significantly lower in MA beneficiaries than in NHI beneficiaries in our study (43.8% vs. 52.8%, $P = 0.012$), which is in line with previous study.[31] However, MA itself was independently associated with higher baPWV even after controlling for these traditional risk factors in our study. This implies that there might be some other independent mechanisms mediating the interplay between low SES and arterial stiffening. Chronic inflammatory reactions are known to increase in people with low SES,[32] which is also an important risk factor for increasing arterial stiffness.[30,33] Our results also showed increased levels of inflammatory markers, including white blood cell count and C-reactive protein, in MA beneficiaries and in subjects with higher baPWV values. Another possibility is that people with low SES are exposed to more stress,[34] which activates the sympathetic nervous system, and eventually increases arterial stiffness.[32,35]

There are several methods for measuring arterial stiffness.[36] Among them, PWV is the most widely used. Carotid–femoral PWV, representing stiffness of the aorta and its first branches, has been validated in many clinical studies, and is considered as the gold standard measurement of arterial

Table 2. Clinical characteristics of study subjects according to median value of baPWV

Characteristic	baPWV <1,540 cm/s (*n* = 4,466)	baPWV ≥1,540 cm/s (*n* = 4,463)	*P* value
Age, years	54.6 ± 12.4	66.0 ± 10.4	<0.001
Female sex	1,879 (42.1)	2,145 (48.1)	<0.001
Body mass index, kg/m^2	24.9 ± 3.4	24.8 ± 3.3	0.164
Systolic blood pressure, mm Hg	122 ± 14	138 ± 18	<0.001
Diastolic blood pressure, mm Hg	74.4 ± 11.6	80.1 ± 13.4	<0.001
Heart rate, per minute	68.0 ± 11.5	72.4 ± 13.1	<0.001
Cardiovascular risk factors			
Hypertension	1,589 (35.6)	2,414 (54.1)	<0.001
Diabetes mellitus	697 (15.6)	1,336 (29.9)	<0.001
Cigarette smoking	520 (11.6)	525 (11.8)	0.560
Coronary artery disease	100 (2.2)	102 (2.3)	0.883
Stroke	25 (0.6)	37 (0.8)	0.126
Laboratory findings			
White blood cell count, per μl	6,666 ± 2,910	7,050 ± 3,514	<0.001
Hemoglobin, g/dl	13.8 ± 1.8	13.2 ± 1.9	<0.001
Fasting glucose, mg/dl	115 ± 36	124 ± 45	<0.001
HbA1c, %	6.20 ± 1.06	6.52 ± 1.18	<0.001
eGFR, ml/min/1.73 m^2	90.2 ± 24.9	80.0 ± 28.5	<0.001
Total cholesterol, mg/dl	165 ± 39	156 ± 38	<0.001
LDL cholesterol, mg/dl	95.6 ± 32.8	88.9 ± 32.6	<0.001
HDL cholesterol, mg/dl	50.6 ± 12.7	49.1 ± 13.1	<0.001
Triglyceride, mg/dl	125 ± 74	127 ± 74	0.405
C-reactive protein, mg/dl	0.68 ± 2.60	1.17 ± 3.36	<0.001
Cardiovascular medications			
Calcium channel blocker	879 (19.7)	1,125 (25.2)	<0.001
Beta-blocker	722 (16.2)	892 (20.0)	<0.001
RAS blocker	992 (22.2)	1,281 (28.7)	<0.001
Statin	1,674 (37.5)	1,909 (42.8)	<0.001

Numbers are expressed as mean ± SD or *n* (%). Abbreviations: baPWV, brachial-ankle pulse wave velocity; eGFR, estimated glomerular filtration rate; HDL, high-density lipoprotein; LDL, low-density lipoprotein; RAS, renin–angiotensin system.

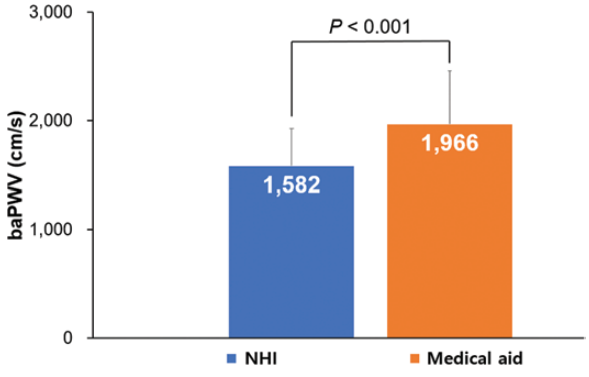

Figure 3. The values of baPWV according to the types of healthcare systems. Abbreviations: baPWV, brachial-ankle pulse wave velocity; NIH, National Health Insurance.

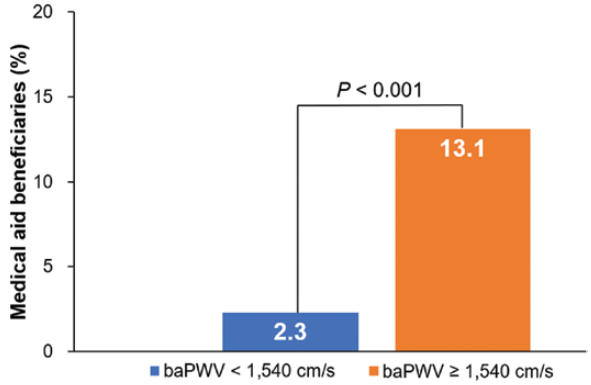

Figure 4. Proportion of medical aid beneficiaries according to baPWV values. Abbreviation: baPWV, brachial-ankle pulse wave velocity.

Table 3. Independent predictors for higher baPWV

Variable	OR (95% CI)	*P* value
Age ≥65 years	5.63 (4.91–6.46)	<0.001
Female sex	1.16 (1.02–1.33)	0.023
Systolic blood pressure ≥140 mm Hg	5.98 (5.10–7.00)	<0.001
Heart rate ≥69 beats per minute (= median value)	1.78 (1.56–2.03)	<0.001
Diabetes mellitus	1.70 (1.45–2.01)	<0.001
Cigarette smoking	0.91 (0.74–1.11)	0.363
eGFR <60 ml/min/1.73 m^2	1.23 (0.98–1.53)	0.065
LDL cholesterol ≥160 mg/dl	0.85 (0.58–1.23)	0.391
CRP ≥0.09 mg/dl (= median value)	1.06 (0.93–1.21)	0.380
Calcium channel blocker	1.03 (0.87–1.21)	0.723
Beta-blocker	1.00 (0.84–1.18)	0.999
RAS blocker	1.25 (1.06–1.47)	0.006
Statin	0.99 (0.86–1.15)	0.979
MA beneficiaries	5.41 (4.02–7.27)	<0.001

Abbreviations: baPWV, brachial-ankle pulse wave velocity; CI, confidence interval; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate; LDL, low-density lipoprotein; MA, medical aid; OR, odds ratio; RAS, renin–angiotensin system.

stiffness.[30] However, the measurement of carotid–femoral PWV needs technical skills for finding the proper position of the carotid and femoral arteries, which can cause discomfort to the patient.[37,38] The baPWV measurement is a more recently developed measure of arterial stiffness by just rapping blood pressure cuffs to both upper arms and ankles, which is a simpler way to measure than carotid–femoral PWV.[37,38] There are many clinical data showing the usefulness of baPWV.[10,19,20,27,38] Therefore, baPWV is especially useful for mass screening.

Our result showed that the use of RAS blockers was independently associated with increased baPWV value in multivariable model. This may imply that the more individuals at high risk, the more likely they were prescribed RAS blockers with good cardiovascular protection.

Our study has several clinical implications. Although it is well known that low SES is associated with high CVD risk,[6–8] the exact mechanism for this is not well understood. Our study with a larger number of patients provides more strong evidence that arterial stiffness plays an important role as a mediator linking low SES with poor cardiovascular outcomes. In addition, given that increased arterial stiffness is an independent risk factor for the development of CVD and mortality,[9–13] our result showing an increased arterial stiffness in MA beneficiaries indicates that they are exposed to high cardiovascular risk. Risk factors should be more aggressively controlled in MA beneficiaries. Additionally, considering the simplicity of baPWV measurement with many clinical data showing good risk predictive performance,[10,19,20,27,38] baPWV could be used as a marker for predicting cardiovascular risk and for monitoring management in MA beneficiaries. Furthermore, our study suggests that research should be conducted to observe if treatment aimed at improving arterial stiffness can improve clinical outcomes.

Study limitations

Our study has several limitations. First, this cross-sectional design could not confirm the causal relationship between low SES and increased arterial stiffness. Second, there was a possibility of selection bias: the proportion of MA beneficiaries in our hospital was about 10%, but it was 7.8% in our study. Some MA beneficiaries might have rejected baPWV measurement due to economic problem. Lastly, our study population was restricted to all Koreans, which may make it difficult to generalize our results to other ethnic groups.

The baPWV values were significantly higher in MA beneficiaries than in NHI beneficiaries. The result of this study provides additional evidence on the association between low SES and arterial stiffening. More careful monitoring and aggressive preventive strategies should be applied to this high-risk group of people.

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DISCLOSURE

The authors declared no conflict of interest.

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