CARDIOLOGY UPDATES NEWSLETTER

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IN THIS ISSUE

Dear Doctor,

Greetings from Micro Labs Limited. We are happy to bring you "Cardiology updates" which contains latest and interesting news in the field of Cardiology

Impact of statin therapy and long-term cardiovascular risks on socioeconomic disparities: microsimulation model

INTRODUCTION

Cardiovascular disease

Globally, cardiovascular disease (CVD) is the primary cause of illness and mortality. Disease policy models estimate the health status of people and groups by modelling important disease phases and linkages using epidemiological data. Unlike CVD risk equations, like QRISK or SCORE, which calculate the risk of a specific CVD outcome over a predetermined time frame (usually 5 or 10 years), policy models are able to project long-term risks, taking competing risks into account and enabling the estimation of long-term costs and quality-of-life-adjusted survival, which can be useful in making decisions about clinical care and policy.[1] American College of Cardiology/American Heart Association new cholesterol treatment guidelines suggest statin medication for a greater proportion of the US population to avoid atherosclerotic cardiovascular disease (ASCVD).[2] The aim of this study was to present a novel model of cardiovascular disease (CVD) and assess health outcomes and the effects of guideline directed statin therapy, across UK quintiles of socioeconomic distress.

METHODS

A total of 49,878 individuals with a history of CVD and 68,018 participants without any history were followed for an average of 3.9 and 4.6 years in Cholesterol Treatment Trialists' (CTT) studies. In UK Biobank (UKB), the average follow-up period was 7.9 years for 57,278 people with a history of CVD and 8.1 years for 444,576 participants without CVD history. CVD microsimulation model was developed using risk equations for myocardial infarction, stroke, coronary revascularization, cancer, and vascular and nonvascular mortality that were derived using trial data. This study also used the UKB cohort to calibrate and enhance this model, adding new features and a diabetes risk equation, and validated the model using the UKB and Whitehall II cohorts. Across quintiles of socioeconomic disadvantage, this study employed the model to predict the incidence of CVD, life expectancy, quality-adjusted life years (QALYs), and the effect of statin therapy as directed by UK guidelines.

RESULTS

The current study results showed that the main risk factors for CVD were age, sex, socioeconomic disadvantage, smoking, hypertension, diabetes, and cardiovascular events. The baseline characteristic of the participants was shown in Table 1. For all participant categories, the observed rates and the model-predicted incident rates matched rather well. A significant

factor affecting quality of life was CVD. A decrease in EuroQoL-5 Dimension (EQ-5D) utility of 0.10 (95%CI 0.03-0.16) in the year of the occurrence and 0.07 (0.04-0.10) in the followed years, that was linked to MI. A decline of 0.09 (0.04-0.13) in the year of the occurrence and 0.13 (0.11-0.16) in the followed years that was linked to stroke. A decline of 0.04 (0.03-0.06) in the first 10 years following diagnosis and 0.08 (0.06-0.10) in the followed years that was linked to diabetes. Daily life-threatening cancer was linked to a 0.13 (0.11–0.14) decline in quality of life. Across the quintiles of socioeconomic deficiency, there were moderate gradients at 10 years in the predicted years of life and QALYs. The model predicted significant gradients in residual life expectancy, with disparities between the quintiles with the lowest and highest levels of socioeconomic deprivation ranging from 4 to 5 years (5 to 8 less QALYs). Treatment with statins, as advised by guidelines, was predicted to enhance QALYs, with bigger increases in quintiles of higher deprivation.

	CTT COLLABORATION		UK BIOBANK	
	Without CVD history	With CVD history	Without CVD history	With CVD History
N	68,018	49,878	444,576	57,278
LDL CHOLESTEROL(mmol/L)	3.5(0.89)	3.8(0.85)	3.6(0.82)	3.1(0.87)
HDL Cholesterol	1.3(0.38)	1.1 (0.31)	1.5(0.37)	1.3(0.36)
Creatinine (umol/L)	91 (24)	98(23)	71(15)	77(19)
Systolic blood pressure(mmHg)	142(20)	139(22)	138(19)	139(19)
Diastolic blood pressure(mmHg)	83(11)	81(11)	82(10)	81(10)
On hypertension treatment	35,478(52%)	25,472(51%)	71,930(16%)	26,184(46%)

Table 1. Baseline characteristics of study participants

DISCUSSION

The current study showed that statin therapy, which is advised by guidelines, has the potential to lessen inequality in socioeconomic status.[1] Similar to this, Wu R et al. showed that the more socioeconomically deprived groups did not have a poorer statin treatment rate.[3]

CONCLUSION

The current study concludes that statin therapy, as advised by guidelines, has the ability to lessen socioeconomic inequalities. For individualized long-term forecasts of health outcomes and the impact of CVD therapies, this CVD model is an innovative tool.

Treatment with statins, as advised by guidelines, offers a good chance of reducing health inequalities across socioeconomic status.

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Association between Mean Platelet Volume and mortality among Peritoneal Dialysis patients

INTRODUCTION

Platelets are important in haemostasis and thrombosis, and their function is influenced by a variety of factors, including platelet size. Mean platelet volume (MPV) is a platelet size metric that may be easily measured in a complete blood count test.[1] In comparison to hemodialysis (HD), peritoneal dialysis (PD) is a significant therapy option for individuals with chronic kidney disease (CKD) that may offer benefits in terms of cost, logistics, and clinical outcomes. Approximately 50% of these individuals' deaths and 33% of their hospital stays are attributable to cardiovascular disease (CVD).[2] In the general population, elevated MPV has been linked to an increased risk of significant adverse cardiovascular events and mortality in patients with acute coronary syndrome, as well as ischemic stroke and hypertension. Increased MPV in diabetic individuals has been associated with poor glycaemic control and a higher risk of diabetic complications. Cardiovascular morbidity and death are more likely to occur in hemodialysis patients, and MPV has been identified as a promising biomarker for predicting difficulties in these individuals.[1] This study aimed to assess the relationship between MPV and cardiovascular and overall mortality in Peritoneal Dialysis (PD) patients.

METHODS

A total of 1322 patients were recruited in this study. Age, gender, and previous comorbidities including diabetes and premorbid CVD were recorded under the patients' demographic data. The end-stage renal disease (ESRD) etiology, body mass index (BMI), Charlson Comorbidity Index (CCI), hypertension, estimated glomerular filtration rate (eGFR), white blood cell count, platelet count, neutrophils, lymphocytes, haemoglobin, creatine, urea nitrogen, albumin, total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), phosphate, intact parathyroid hormone (iPTH), and antiplatelet drugs were among the principal clinical and biological parameters evaluated. Before PD therapy started, baseline data were gathered. Mortality from all causes and

cardiovascular disease were the main outcomes. According to the cut-off value, which was established using maximally selected rank statistics, patients were separated into two groups. The Cox regression models were used to evaluate the mortality hazard ratio.

RESULTS

The current study results showed that the average age of the 1322 PD patients who were enrolled was 49.3 ± 14.5 years, 57.6% of them were men, and 18.8% of them had diabetes. Chronic glomerulonephritis, which affected 66.1% of this group, was the primary cause to ESRD, followed by diabetic nephropathy (14.7%) and hypertension (12.0%). 360 individuals passed away during a median follow-up of 50 months (IQR: 30-80); 167 of these deaths were related to cardiovascular illnesses. According to correlation analyses, MPV had a negative correlation with the NLR ($r=-0.072$, $p=0.009$), cholesterol ($r=-0.098$, $p<0.001$), platelet count ($r=-0.266$, $p<0.001$), and LDL-C ($r=-0.072$, $p<0.001$). All-cause and cardiovascular death rates were lower in the higher-MPV group than in the lower-MPV group, according to a survival analysis ($p<0.001$ and $p<0.001$, respectively) (Figure 1). A larger MPV was substantially linked to a hazard ratio of 0.77 for all-cause death and 0.75 for cardiovascular mortality after full adjustment (95% CI: 0.60-0.98, $p=0.036$). Age and MPV had a significant interaction, according to subgroup analysis ($p<0.001$). Only in patients under 60 years old had decreased MPV, linked to a higher death risk.

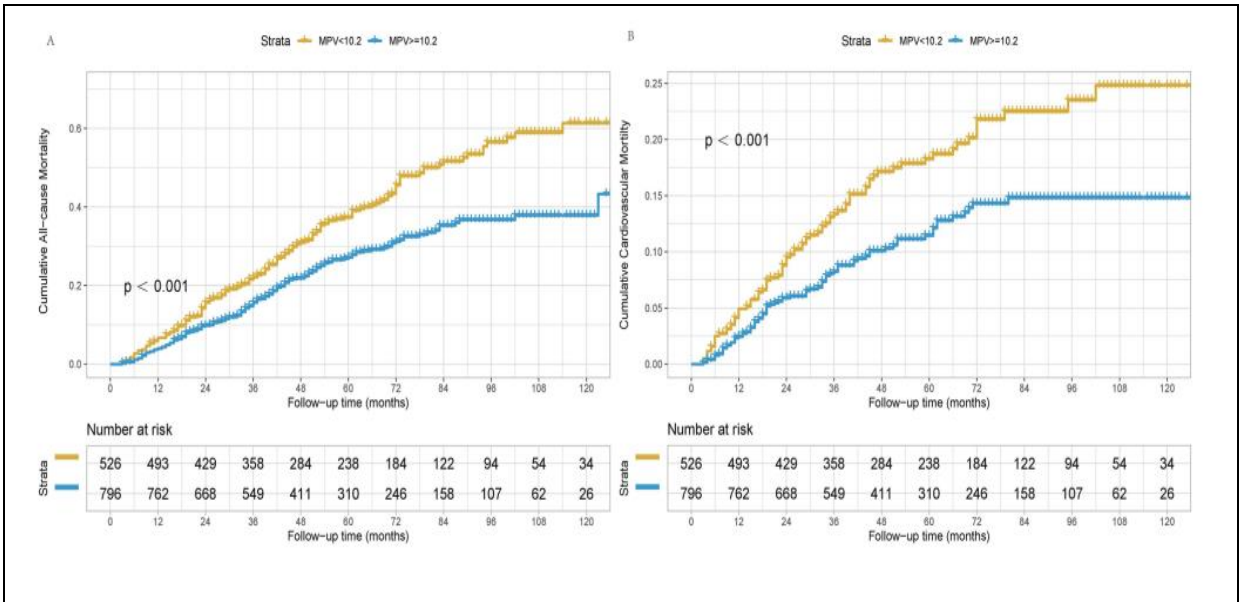

Figure 1. Patient survival curves Stratified by MPV (A) Cumulative all-cause mortality (B) Cumulative cardiovascular mortality

DISCUSSION

The current study showed that PD patients with greater MPV levels had a considerably reduced risk of cardiovascular and all-cause death than PD patients with lower MPV levels. Similar to this, Chen J et al. showed that in PD patients less than 60 years old, decreased MPV/PC level

was an independent risk factor for all-cause and CV death.[3] Zhu Y et al. showed that increased MPV or platelet count linked to both cardiovascular death and all-cause death. [4]

CONCLUSION

This study concludes that PD patients who have high MPV have a lower risk of cardiovascular and all-cause mortality. This study finding add to the body of knowledge regarding the connection between MPV and cardiovascular and all-cause mortality, and they may be crucial for managing and treating PD patients.

Peritoneal dialysis patients who have high Mean Platelet Volume have a lower risk of cardiovascular and all-cause mortality.

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Correlation between the atherogenic index of plasma and adverse long-term prognosis among chronic coronary syndrome patients

INTRODUCTION

Coronary artery disease (CAD) is the leading cause of death from cardiovascular diseases, accounting for 244.11 million individuals worldwide. It also poses a large public health burden. According to the American College of Cardiovascular Diseases' 2016 data, it is predicted that by the year 2030, about 18% of adults would have chronic coronary syndromes（CCS), raising serious concerns for people's general wellbeing. In order to prevent or manage the condition, it is essential to develop significant biomarkers linked to CCS for early detection, risk assessment, and specific interventions. A recently discovered biomarker known as the Atherogenic Index of Plasma (AIP) that is connected to lipid metabolism has shown to have strong predictive potential in people with cardiovascular disease.[1] The ratio between the

amount of triglyceride (TG) and high-density lipoprotein cholesterol (HDL-C) is used to construct the atherogenic index of plasma (AIP), which is a measure of the features and severity of abnormal lipid metabolism. As an alternative for small, dense low-density lipoprotein (sdLDL) in determining plasma atherogenicity, it has also been frequently employed.[2] Its effect on chronic coronary syndrome (CCS) have not yet been fully studied. The objective of the current study was to investigate any potential links between patient AIP levels and long-term clinical outcomes in diagnosed CCS patients.

METHODS

This was a single-center retrospective observational study and included 404 participants with a diagnosis of CCS who had coronary angiography. High-density lipoprotein cholesterol and triglycerides were used to calculate the AIP index. AIP is calculated mathematically from log (TG/HDL-C) and is based on the base 10 logarithms of the ratio of the TG level to HDL-C level in molar concentration (mmol/L). The AIP values of each patient were then used to create major adverse cardiovascular events (MACE) and non-MACE groups as well as quartile groups (Q1: < -0.064; Q2: -0.064–0.130; Q3: 0.130–0.328; Q4: > 0.328). The main clinical outcomes in this study were major adverse cardiovascular events (MACE), which refers to cardiovascular death, ischemia-driven revascularization, nonfatal myocardial infarction (MI), heart failure, and nonfatal stroke. MACE were monitored during each patient's follow-up period. To investigate the connection between AIP and MACE, Cox regression analysis and Kaplan-Meier curve analysis were used. Additionally, the ideal AIP cut-off value for clinical MACE prediction was determined using ROC analysis.

RESULTS

The current study showed that 144 patients (35.6%) out of 404 had diabetic mellitus (DM). A total of 88 patients (21.8%) experienced MACE after a median follow-up of 35 months, including 7 (1.7%) cardiovascular deaths, 29 (7.2%) Ischemia-driven revascularizations, 5 (1.2%) nonfatal myocardial infarction (MI), 27 (6.7%) heart failure, and 20 (5.0%) nonfatal strokes. Patients in the Q4 group who had higher AIP levels also tended to be more hyperlipidemic, diabetic, and had higher BMIs. The Q4 group's blood parameters, such as TG, total cholesterol (TC), remnant-C, non-HDL, haemoglobin A1c (Hb1AC), haemoglobin (Hb), alanine aminotransferase (ALT), and fasting blood glucose (FBG), were considerably greater than those of the Q1, Q2, and Q3 groups, but HDL-C decreased as AIP values increased. Notably, patients in the Q4 group had a substantially greater incidence of MACE than patients in the Q1, Q2, and Q3 groups who had lower AIP values (31.7% vs. 16.8%, 15.7%, and 23.0%, respectively; P=0.023). According to the Kaplan Meier curves, patients in the Q4 group had a higher probability of developing MACE than those in the other groups (log-rank P=0.014). According to the results of the multivariate Cox regression analysis, people in the Q4 group had a 7.892-fold higher risk of MACE than people in the Q1 group (adjusted HR, 7.892; 95% CI 1.818-34.269; P=0.006). The findings demonstrated a greater correlation between the risk of MACE and AIP in younger, male, hypertensive, high-sensitivity C-reactive protein (hs-CRP<10), and non-diabetic CCS individuals. Additionally, AIP has strong correlations with age ($r = -0.193$; $p = 0.021$), FBG ($r = 0.206$; $p = 0.015$), TC ($r = 0.288$; $p<0.001$), low-density

lipoprotein cholesterol (LDL-C) (r = 0.220; p = 0.009), HDL-C (r = -0.640; p<0.001), and TG in CCS patients with DM, (r=0.948; p<0.001). The ROC curve study also identified an ideal AIP cut-off value of 0.24 for clinical MACE prediction in CCS patients (Figure 1).

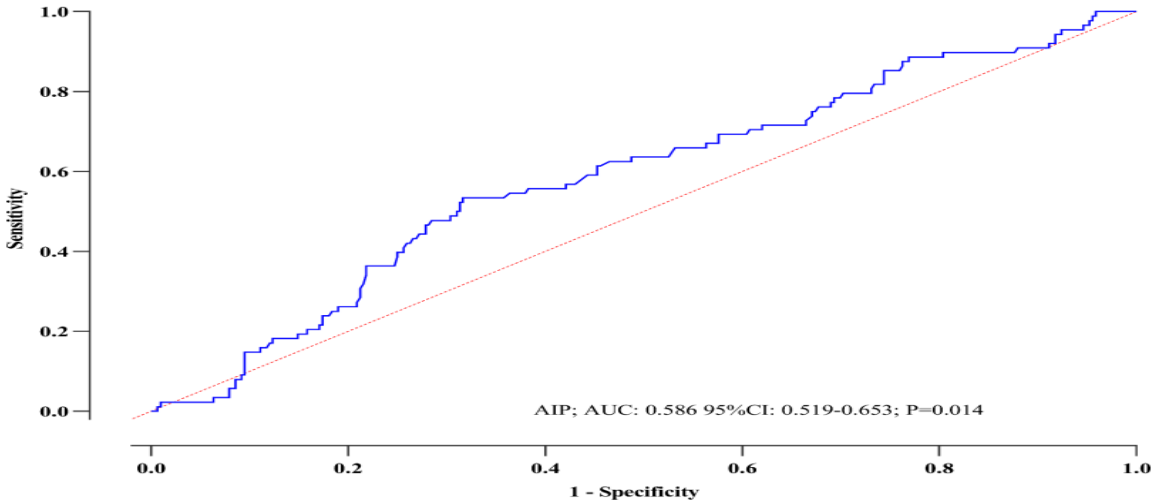

Figure 1. Analysis of the receiver operating characteristics for the AIP's capability of predicting MACE in the study population. **AUC** area under the curve, **AIP** atherogenic index of plasma, **CI** confidence interval

DISCUSSION

The current study showed that in individuals with CCS, elevated AIP level was observed to be independently related with a higher risk of MACE.[1] Similar to this, Hang F et.al showed that among elderly non-diabetic hypertensive patients, an independent and favorable correlation between high AIP and risk of MACEs.[2] The prognosis of diabetic individuals with high AIP levels included higher MACEs and was unaffected by LDL-C levels.[3] For CCS patients, AIP has become a substantial independent risk indicator for predicting the likelihood of MACE.[1] Among older persons with non-diabetic hypertension, the value of AIP was a reliable indicator of cardiovascular prognosis.[2]

CONCLUSION

This study concludes that increased AIP is closely associated with higher risk in CCS patients. The ideal cut-off value for AIP, an independent predictor of MACE in CCS, is 0.24. This study data also demonstrated the critical therapeutic importance of AIP in the context of CCS early risk prediction and preventive interventions.

Increased atherogenic index of plasma (AIP) is closely associated with higher risk in chronic coronary syndromes (CCS) patients.

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Case report on the Vicious Cycle of BRASH Syndrome

INTRODUCTION

BRASH syndrome is a medical disorder defined by a combination of bradycardia (slow heart rate) and shock in people with renal failure, a history of taking atrioventricular (AV) nodal blocking drugs, and hyperkalemia (high potassium levels in the blood).[1] Synergism between hyperkalemia and AV nodal blockage is the source of the pathophysiology of BRASH syndrome.[2] This syndrome is more common in elderly people with impaired kidney function and a history of taking AV nodal blocking drugs on a frequent basis. Hypovolemia and decreasing renal function are major risk factors for BRASH syndrome. Early diagnosis of BRASH syndrome is essential because it can lower mortality rates and avoid delays in getting a diagnosis.[1] This study presents a case on Vicious Cycle of BRASH Syndrome in a 66- year old male patient.

CASE PRESENTATION

A 66-year-old male patient arrived with repeated episodes of vomiting, generalized fatigue, vomiting, and multiple episode of syncope. The patient presented with a medical history that included high blood pressure, stage 3 chronic renal disease, coronary artery disease, heart failure with a poor ejection fraction (25%), and type 2 diabetes. His prescribed drugs, which comprised sacubitril/valsartan 25 mg twice daily, furosemide 40 mg once daily (OD), bisoprolol 2.5 mg OD, and spironolactone 12.5 mg OD, had all been taken as directed. According to the initial medical evaluation, the patient was conscious of time, location, and others, although being somewhat sleepy. When he arrived, his Glasgow Coma Scale score was

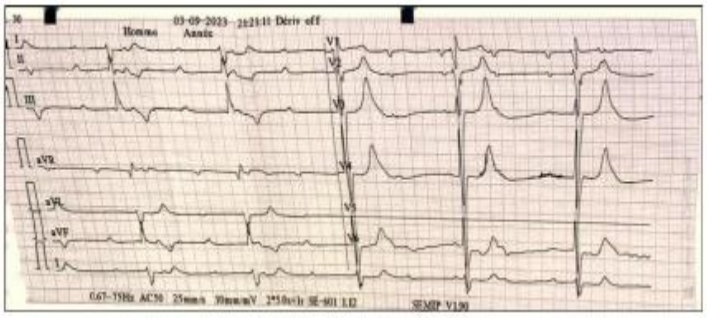

Figure 1. An Electrocardiogram showed Junctional rhythm bradycardia.

15/15. He appeared dehydrated and his heart rate was 25 beats per minute, and blood pressure was 76/55. He underwent a full physical examination, and nothing odd was found. An electrocardiogram (ECG) revealed junctional rhythm bradycardia (Figure 1). Regular biochemistry blood tests revealed metabolic acidosis, abrupt deterioration of chronic renal failure (creatinine at 35 g/L, GFR of 17 ml/min, declining from a baseline of 40 ml/min), and severe hyperkalemia at 6 mEq/L. His blood tests for biochemistry resulted other values that were within acceptable limits. Vasopressor agent-assisted fluid resuscitation was part of the immediate intervention. Salbutamol (10 mg) inhalation, regular insulin (5 units) with dextrose, and the rapid delivery of calcium gluconate (1 gram) were the necessary treatments for hyperkalemia. The patient's blood pressure and heart rate improved as his condition became better. Bradycardia continued, prompting the placement of a temporary transvenous pacemaker to give initial stabilization due to the patient's refractory hyperkalemia (5.4 mmol/L).

DISCUSSION

This case report showed that an electrocardiogram (ECG) revealed junctional rhythm bradycardia in a 66-year-old male patient.[1] Similar to this, Saini T et al. showed that the EKG revealed ventricular escape complexes and total heart block along with bradycardia at a rate of thirty.[2]

CONCLUSION

This study concludes that the unclear symptoms of BRASH syndrome make it difficult to detect and treat this as a life-threatening illness, which is routinely underdiagnosed. In order to effectively manage the condition, practitioners must be more aware of the need of early identification and addressing contributing variables. Correcting hyperkalemia, treating underlying reasons to improve renal function, and restricting the use of AV nodal blocking drugs are the main strategies for treating BRASH syndrome.

Correcting hyperkalemia, treating underlying reasons to improve renal function, and stopping the use of AV nodal blocking drugs are the main strategies for treating BRASH syndrome.

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