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Serum erythrocyte sedimentation rate is higher among ethnic South Asian compared to ethnic Chinese ischemic stroke patients. Is this attributable to metabolic syndrome or central obesity?

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ABSTRACT

Inflammation, a vascular risk factor, is more pronounced among ethnic South Asians compared to ethnic Chinese in the general population. We compared serum erythrocyte sedimentation rate (ESR) levels between ethnic South Asian and Chinese acute ischemic stroke patients, and further investigated if metabolic syndrome or central obesity could account for any difference detected. We prospectively recruited consecutive ischemic stroke patients within seven days of onset. Measurement of serum ESR was performed within two days of admission. Median serum ESR was higher among the 55 ethnic South Asian (16 mm/h IQR 3–35) compared to the 165 ethnic Chinese patients (9 mm/h IQR 4–19), $p=0.004$). Serum ESR was correlated with age. Higher serum ESR was associated with female gender, non-smokers, patients with central obesity and low high-density lipoprotein (HDL) cholesterol. Using regression analysis, South Asian ethnicity remained significantly associated with serum ESR, independent of age, gender, smoking status, metabolic syndrome, central obesity and low HDL. Ethnic South Asian ischemic stroke patients have a higher inflammatory state compared to ethnic Chinese patients. As the higher inflammatory state is independent of demographic and risk factors, we propose an underlying genetic or cultural basis for the ethnic difference.

1. Introduction

South Asians are the most prevalent ethnicity in the world [1]. Ethnic South Asians are responsible for 17% of the world's vascular disease burden, with a higher prevalence of vascular disease compared to many other ethnicities [2,3]. In multi-ethnic Singapore, ethnic South Asians have a higher incidence of atherosclerotic vascular disease including young onset of myocardial infarction and stroke, compared to their ethic Chinese counterparts [4–7]. Inflammation is a known risk factor for vascular disease including ischemic stroke [8]. Serum erythrocyte sedimentation rate (ESR) is an inflammatory marker that is commonly measured in clinical practice.

Inflammatory markers have been shown to be higher among ethnic South Asians compared to other ethnicities. In the Canadian general population, another inflammatory marker, C-reactive protein (CRP), was higher among ethnic South Asians, compared to both ethnic Chinese and ethnic Europeans [9]. Chambers et al. reported that serum CRP was higher among ethnic South Asians compared to ethnic Europeans in a United Kingdom population study [10]. Among patients admitted for evaluation of chest pain in Singapore, a higher proportion of ethnic South Asians had raised CRP compared to ethnic Chinese [11].

Higher levels of inflammatory markers among ethnic South Asians have been postulated to be related to greater central obesity and insulin resistance. In the study by Chambers, higher serum CRP among ethnic South Asians compared to ethnic Europeans was attributable to greater central obesity and insulin resistance [10]. Our group has shown that metabolic syndrome (which is associated with insulin resistance) was more prevalent among ethnic South Asian ischemic stroke patients compared to matched ethnic Chinese counterparts [12].

Inflammatory marker levels have not been compared between ethnic South Asian stroke patients with any other ethnic group. We hypothesized that serum ESR is higher among ethnic South Asian compared to ethnic Chinese ischemic stroke patients. In addition, we investigated if metabolic syndrome or central obesity played a role in any ethnic difference in inflammatory state.

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2. Subjects and methods

2.1. Study population

In this prospective observational study, we recruited consecutive ethnic South Asian and ethnic Chinese acute ischemic stroke patients within seven days of onset who were admitted to the Singapore General Hospital. Eligibility for inclusion in this study was serum ESR measurement within two days of admission and anthropometric measurements during the acute admission. The study was approved by the Singapore General Hospital's Institutional Review Board and informed written consent obtained from participants.

2.2. Evaluation

Demographic and risk factor data were collated. We employed the Trial of Org 10172 in Acute Stroke Treatment (TOAST) classification to subtype stroke [13]. For the purpose of this study, small vessel stroke was used as a marker of small infarct size. Venous blood samples were obtained on the first or second day of admission. Serum ESR measurement was performed in the central laboratory of the Singapore General Hospital using the Westergen method with a Sediplast autozero ESR system (Polymedco, Cortlandt, New York).

2.3. Definitions

Metabolic syndrome was defined by the National Cholesterol Education Program Adult Treatment Panel criteria, [14] using the Asian Pacific World Health Organisation (WHO) modification [15]. A patient was considered to have metabolic syndrome if 3 or more of the following criteria were satisfied: (1) central obesity with waist circumference 90 cm or greater for men and 80 cm or greater for women; (2) abnormal glycemia with fasting glucose level of 6.1 mmol/L or greater or known treatment for diabetes; (3) low high-density lipoprotein (HDL) cholesterol less than or equal to 1.0 mmol/L in men and less than or equal to 1.3 mmol/L in women; (4) high triglyceride level 1.7 mmol/L or greater; and (5) high blood pressure 130/85 mm Hg or greater or known treatment for hypertension. Cholesterol measurements were done by colorimetric methods, at the earliest time after hospital admission once the patient had fasted for 10 h. If a patient was taking a statin, we reviewed previous lipid profiles before initiation of the statin and used these to determine if the low HDL and high triglyceride components of metabolic syndrome were fulfilled. Blood pressure readings were taken manually using a sphygmomanometer. For the purpose of fulfilling the criterion of high blood pressure, we took into consideration at least two blood pressure readings after 48 h of stroke onset. A patient was considered a smoker if there was a history of smoking in the prior 3 years.

2.4. Data analysis

We performed statistical analyses with SPSS version 16.0. Univariate analysis for associations with serum ESR was performed using the Wilcoxon test for nominal data and Spearman's correlation for continuous data. Multivariate linear regression analysis was performed to ascertain independent associations of serum ESR.

3. Results

3.1. Baseline characteristics

From February 2006 to April 2007, we recruited 248 ischemic stroke patients of South Asian or Chinese ethnicity. Serum ESR was not measured for 10% (25) of subjects recruited and another 3 patients did not have anthropometric measurements performed. Of the 220 eligible patients, 165 were ethnic Chinese and 55 ethnic South Asian. The median age was 64 years (IQR 56–72). There was a coronary artery disease (CAD) prevalence of 79% and 66% were male. The distribution of serum ESR was not normal, with a positive skew. Median serum ESR was 10 mm/h (IQR 4–22). Median waist circumference was 84 cm (IQR 80–92).

3.2. Comparison between ethnic South Asian and Chinese cohorts

There was no difference in age ($p=0.817$) and gender ($p=0.621$) distribution between the two ethnic groups. Among ethnic South Asians, the CAD prevalence was higher (40% vs 15%, $p<0.001$) and median waist circumference larger (87 cm IQR 82–97 vs 84 cm IQR 79–92, $p=0.040$). The prevalence of smokers was 32% among ethnic Chinese and 22% among ethnic South Asian patients ($p=0.170$). There was no statistically significant difference in stroke subtype distribution between the two ethnic groups. Among ethnic South Asians, 35% had large vessel stroke, 14% cardioembolic stroke, 39% small vessel stroke, 4% stroke due to other aetiology and 8% due to undetermined aetiology. Among ethnic Chinese, 27% had large vessel stroke, 6% cardioembolic stroke, 50% small vessel stroke, 3% stroke due to other aetiology and 13% due to undetermined aetiology. There was also no difference in the prevalence of small vessel stroke between ethnic South Asians and Chinese ($p=0.173$).

3.3. Correlations and associations of serum ESR

Ethnic South Asians had a significantly higher median serum ESR compared to ethnic Chinese patients (16 mm/h IQR 3–35 vs 9 mm/h IQR 4–19, $p=0.004$) (Table 1). Median serum ESR was also significantly higher among females ($p<0.001$), patients categorised with central obesity ($p=0.013$) and low HDL ($p=0.013$), as well as non-smokers ($p=0.023$). Serum ESR did not differ significantly for patient categorised with metabolic syndrome ($p=0.111$), high blood pressure ($p=0.181$), abnormal glycemia ($p=0.116$), raised triglycerides ($p=0.112$) and CAD ($p=0.535$). There was a significant correlation between serum ESR and age (Spearman's coefficient 0.293, $p<0.001$). Serum ESR did not

Table 1
Associations and correlations of serum erythrocyte sedimentation rate (ESR)

	Median serum ESR (IQR)	*p* value
Male ($n=146$)	7 (3–17)	<0.001
Female ($n=74$)	17 (9–28)	
Ethnic South Asian ($n=55$)	16 (5–35)	0.004
Ethnic Chinese ($n=165$)	9 (4–19)	
Concomitant CAD ($n=47$)	13 (2–31)	0.535
No concomitant CAD ($n=173$)	10 (5–20)	
Metabolic syndrome ($n=132$)	12 (5–23)	0.111
No metabolic syndrome ($n=88$)	9 (4–20)	
High blood pressure ($n=171$)	10 (5–22)	0.181
No high blood pressure ($n=49$)	9 (4–17)	
Abnormal glycemia ($n=99$)	12 (5–23)	0.116
No abnormal glycemia ($n=121$)	9 (4–20)	
Central obesity ($n=97$)	13 (5–23)	0.013
No central obesity ($n=123$)	8 (3–18)	
Raised triglycerides ($n=66$)	8 (3–21)	0.112
No raised triglycerides ($n=154$)	10 (5–22)	
Low HDL ($n=55$)	15 (6–28)	0.013
No low HDL ($n=165$)	9 (4–20)	
Smoker	6 (2–23)	0.023
Non-smoker	14 (5–21)	
Small vessel stroke ($n=104$)	10 (4–22)	0.605
Other TOAST subtypes ($n=116$)	11 (5–22)	
	Spearman correlation coefficient	*p* value
Age	0.293	<0.001
Waist circumference	0.060	0.313
Serum HDL	−0.012	0.220

ESR: erythrocyte sedimentation rate.
CAD: coronary artery disease.
HDL: high-density lipoprotein cholesterol.