Review

Ethnicity and peripheral artery disease

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Summary

Peripheral arterial disease (PAD) is an important healthcare problem and is an indicator of widespread atherosclerosis in other vascular territories, such as the cerebral and coronary circulations. PAD is associated with considerable morbidity and mortality. Most population-based studies investigating PAD prevalence and risk factors for its development and progression have been based on predominantly White ethnic groups. Much less is known about the characteristics of this disease in other ethnic groups. Understanding the epidemiology of PAD amongst ethnic minority groups is relevant, given that the population of minority ethnic groups in countries such as the United Kingdom rose by 53% between 1991 and 2001 and is expected to rise further in the future. This article aims to provide an overview of possible pathophysiological differences between ethnic groups for PAD, focussing predominantly on South Asians (people originating from India, Bangladesh and Pakistan) and Blacks (people of Black Caribbean and Black African descent) as these groups comprise the majority of all ethnic minorities in the United Kingdom.

Introduction

Peripheral artery disease (PAD) is an important healthcare problem in developed nations and is associated with considerable morbidity and mortality. PAD is the disease process resulting from obstruction of large peripheral arteries, exclusive of the coronary and intracranial cerebrovascular system, most commonly due to atherosclerosis. Most typically, it is referred to in relation to the lower limbs. PAD is an indicator of widespread atherosclerosis in other vascular territories, such as the cerebral and coronary circulations.¹ There is also considerable overlap between PAD, cerebrovascular disease (CBVD) and coronary artery disease (CAD),²,³ with the presence of PAD being associated with and increased risk of CBVD and CAD and their consequences.⁴⁻⁶ People with PAD have 4–5 times greater risk of dying from a cardiovascular disease event compared to those without, and a 2–3 times greater all-cause mortality.⁷,⁸

Several population-based studies⁹⁻¹¹ based on predominantly white European populations have found the prevalence of PAD to be between 6% and 18% over the age of 55 years. The prevalence rises with age and has been found to be approximately 20% in people over 70 years of age¹² and up to 60% in the over 85 age group.¹⁰ There has, however, been very little research into the prevalence of PAD in non-Caucasian populations, although previous population-based studies have shown variations in the prevalence of this disease amongst different ethnic groups (Table 1).¹³⁻¹⁹
Ethnic minority groups make up 7.9% of the general population of the United Kingdom. The largest of these being Asian/Asian British (50.2%) and Black/Black British (24.8%). The aim of this article is to provide an overview of possible pathophysiological differences between ethnic groups for PAD, focussing in particular on South Asians (that is, people originating from India, Bangladesh and Pakistan) and Blacks (people of Black Caribbean and Black African descent) populations, as these groups comprise the majority of all ethnic minorities in the United Kingdom.

Risk factors for the development of PAD are very similar to those of CAD and CBVD. Classic risk factors include tobacco smoking, diabetes mellitus (DM), dyslipidaemias, hypertension, advancing age and male gender. DM and smoking are the strongest modifiable risk factors for PAD in predominantly Caucasian populations. Age and sex are the most important non-modifiable risk factors in all ethnic groups, with the prevalence of PAD increasing by up to 2-fold per decade of life. Negative risk factors include regular physical activity and moderate alcohol intake.

In recent years, several new plausible risk factors for atherosclerosis have been discovered, which include homocysteine, C-reactive protein (CRP), fibrinogen, lipoprotein(a), increased platelet activity and hypercoagulability. The relationship between PAD and these cardiovascular risk factors have not been fully understood and require further investigation.
elucidated, although some may explain the ethnic variations in susceptibility to PAD.42

Ethnic differences in PAD

There are well-recognized differences in vascular disease in different ethnic groups. South Asians living in the United Kingdom have a higher mortality for ischaemic heart disease than Europeans45 and Blacks have a higher CBVD mortality in both sexes.45 However studies both in the United Kingdom and India suggest PAD prevalence is lower in South Asians than in Caucasians in general population, diabetic and CAD cohorts.13,15–17 Traditional risk factors could not account for the observed difference in PAD in a recent UK study17 as Indians are exposed to a number of risk factors which should increase their risk of PAD, such as dyslipidaemia and diabetes.46

Several US studies (Table 1) found the prevalence of PAD in African Americans to be higher than in Non-Hispanic Whites (NHW). Compared to NHW, African Americans also had a significantly worse risk factor profile, with the exception of dyslipidaemia.22 However, differences in prevalence were only partially attributable to higher rates of DM and hypertension in this ethnic group.14 In the National health and Nutrition Examination Survey (NHANES), when adjusting for all traditional risk factors the odds ratio (OR) for PAD in African Americans over NHW was 1.67. This OR reduced when further adjusting for novel risk factors suggesting that novel risk factors play some role at least in their susceptibility to PAD.22

Whilst the prevalence of risk factors is variable amongst different ethnic groups it is thought that the underlying pathophysiology of arterial disease is affected by ethnicity and race and that some ethnic variations in risk factors may be genetically based.

Pathophysiology

Tobacco smoking

Smoking tobacco is a major risk factor in the development of PAD31,35 as a sequel of atherosclerosis.47 It can result in a 7-fold increase in PAD48 and its impact persists with advanced age.49 Smoking has been related to elevated CRP, fibrinogen and homocysteine, novel risk factors, in both white and non-white ethnic groups.50

The Edinburgh Artery Study reported a dose-dependent relationship between smoking and PAD,46 a finding supported by other, more recent studies.51,52 NHANES found PAD prevalence increased sequentially from never smokers to ex-smokers to current smokers across all ethnic groups.22 In patients with PAD, smoking is associated with increased progression of disease and increased risk of amputation.53 It also reduces the effectiveness of anti-platelet medication in such patients.54 Cessation is associated with reduced disease progression and increased survival.55

The prevalence of smoking varies among ethnic groups and gender. Even within the South Asian group there is considerable variation; Bangladesh men being the heaviest and Indians the lowest of the male smokers and low rates of smoking amongst all of the female South Asians.46 In the United States, Asian Indians were much less likely to be current smokers than NHW.56

There is limited evidence investigating smoking and PAD in ethnic groups. A population-based study in South India13 found that smokers had 2.7 times higher risk for PAD, although the result did not reach statistical significance. A recent UK study on Indian Asians and Europeans with equivalent rates of CAD found the prevalence of PAD to be much lower in the former group,17 with smoking rates also lower in Indians. Adjustment for pack years reduced the significance of ethnicity considerably but could not account for the ethnic differences in PAD observed.17

South Asian smokers have four times greater risk than never smokers for developing CAD, a similar elevation in risk to Europeans. Indeed only 1–5% of Asian females smoke yet they have double the risk of suffering CAD than European females.46 You would expect that with the overlap that exists with chronic vascular disease at different sites in predominantly Caucasian populations, there would be a similar relationship in ethnic minority groups. Whereas South Asians are at increased risk of premature death from CAD57,58 as compared to European populations, limited data suggests that the prevalence of PAD in this group is much lower than in Europeans.13 Another hypothesis suggests that either South Asians are not living long enough to develop symptomatic PAD or other—as yet undetermined—factors are responsible for the development of atherosclerosis in different vascular territories.

Evidence investigating the relationship between Black ethnicity, smoking and PAD is conflicting. A UK study15 found that PAD was less frequent in Blacks than Europeans and that the proportion of never smokers was greater in the former group. African Americans on the other hand, were found to have a higher prevalence of PAD compared to NHW yet had a lower prevalence of smoking in another study,59 yet in another60 the percentage of current smokers was higher in African Americans with
sub-clinical vascular disease than in NHW. With methodological differences between these studies it is difficult to elucidate whether smoking confers a greater risk for PAD in Blacks than in Caucasians.

**DM and impaired glucose tolerance**

DM is a major cardiovascular risk factor, and the incidence of type 2 diabetes is rising in developed nations as a consequence of lifestyle changes resulting in increased obesity.

DM is associated with an increased risk of stroke and with a 2–4-fold increased risk of developing CHD and PAD. DM and smoking are together the most important risk factors for the development and progression of PAD. In diabetic patients, the risk of PAD is increased by age, duration of DM, blood-glucose control and peripheral neuropathy. Approximately 5–10% of PAD patients have type 1 and 90–95% have type 2 DM. Blood-glucose control seems to be an important factor for PAD and it has been estimated that with each 1% increase in glycosylated haemoglobin level comes a 28% increased risk of incident PAD. In the Framingham Heart Study, 20% of symptomatic PAD patients had DM. Other studies have found the prevalence of PAD in diabetics to be 20% in people over 40 years old and 29% in people aged over 50 years. IC and Critical leg ischaemia are also more common in patients with DM; the risk of IC being 2-fold higher.

The prevalence of type 2 DM in the United Kingdom is much higher in African Caribbean and South Asian groups compared to the general population. Various studies have shown that 16–17% of middle-aged African Caribbeans and 20% of South Asians have type 2 DM compared to 3–5% of Europeans. After standardizing for age, South Asians have a 3–6-fold and African Caribbeans a 2.5-fold greater risk of DM than the general population. By contrast, the prevalence of DM in rural India is 2–3% and ~8% in urban areas.

Interestingly, a migration study found fasting glucose to be much higher in South Asians living in the United Kingdom than siblings living in India. This difference infers that there could be a genetic susceptibility to the development of DM, which is exacerbated by lifestyle differences and environmental factors which arise from living in developed countries. However, this was not supported by another migration study in Gujuratis, which found the diabetes prevalence to be similarly high in both native Indians and UK migrants.

A population-based study in South India reported a prevalence of PAD of 6.3% amongst diabetics compared to 3.2% in the whole population. This contrasts with a population-based study from the United States which reported the PAD prevalence to be 22% in its diabetic cohort as compared to 3% in people with normal glucose tolerance. When compared to diabetic Caucasians in other studies, the prevalence of PAD in diabetic South Asians was found to be much lower. Chaturvedi et al. found Indian Asians to have a lower prevalence of PAD than Europeans with equivalent CAD levels despite Indians having a higher prevalence of insulin resistance and a higher HbA1c.

African Americans with diabetes were found to have a higher prevalence of PAD than NHW in the United States but no difference in prevalence between Blacks and Caucasians were apparent in a UK study. Interestingly amongst patients with PAD in the United States a similar prevalence of DM was found between African Americans and NHW. As with smoking, the methodology was different for each of these studies so it is difficult to establish a link between diabetes and PAD which accounts for the apparent increase in prevalence of this disease in Blacks.

**Dyslipidaemia**

Abnormalities in the components of the lipid profile are associated with PAD. These do not appear to carry the same importance as smoking and DM in its development. The ratio of Total Cholesterol (TC): High Density Lipoprotein Cholesterol (HDL-C) is the strongest lipid predictor of PAD risk. The most frequent dyslipidaemia associated with PAD is elevated triglycerides and low HDL-C. The association between Low Density Lipoprotein Cholesterol (LDL-C) and PAD appears to be weaker than for HDL-C and triglycerides (TG), and furthermore weaker than its association with the development of CAD.

In the United Kingdom no significant differences in TC were found between ethnic groups, with the exception of African Caribbean females. In the United States, the TC:HDL-C ratio has also been found to be quite similar in NHW, African Americans and Asians. South Asians appear to have a less favourable lipid profile than other ethnic groups with a lower HDL-C and higher TG than Caucasians. This finding is apparent even in children. These abnormalities in HDL-C and TG become more pronounced on migration from the Indian subcontinent, suggesting both environmental and genetic factors are at play. On the other hand Blacks have a more favourable lipid profile than Caucasians with higher HDL-C levels. Interestingly significant disease/race
interactions for LDL-C levels have been found, showing a stronger association in Blacks (especially in women).60

There are limited data linking PAD to dyslipidaemia in ethnic groups. A population-based study in India linked hypercholesterolaemia and LDL-C to the development of PAD (ORs 1.4 and 1.5, respectively),13 although the findings were not significant. A South African study found Blacks with PAD had lower TC, TG and HDL levels than Caucasians with PAD.86 The discordance between lipid profile and PAD risk in South Asians and Blacks suggests that dyslipidaemia does not play as important a role in its development as in the pathogenesis of CAD.

**Hypertension**

Hypertension is a major risk factor for all vascular disorders and is associated with a 2- to 3-fold risk for PAD.1 Several components of blood pressure, including pulse pressure, systolic blood pressure (SBP) and diastolic blood pressure have been shown to be independent cardiovascular risk factors.87 SBP in particular is a major risk factor for CAD and stroke. In PAD, hypertension is a risk factor for both symptomatic and asymptomatic disease40,66 and the degree of hypertension is also closely linked to the development of PAD.66

PAD and hypertension are associated diseases with 35–55% of patients with PAD also having hypertension at presentation.88-90 The prevalence of PAD in patients with DM increases with the presence of systolic hypertension in all racial groups. If hypertension in these patients is controlled, the progression of PAD can be slowed.91

The prevalence of hypertension is well-recognized as being higher in Blacks compared to Caucasians.92 Black people have been found to develop hypertension at an earlier age,93 have more severe disease,94 have worse BP control, have a different distribution of target organ damage93 and a raised mortality from hypertension; perhaps 1.5 times higher than national average.92 In some studies, the prevalence of hypertension in the South Asian immigrant population in the United Kingdom is also significantly higher.95,96 However, the mean population blood pressure in these ethnic groups is similar to that of white European populations.69

A meta-analysis of hypertension in South Asians in United Kingdom found that they had lower mean systolic but higher diastolic blood pressures in both males and females compared to whites.97 The prevalence of hypertension was higher in South Asian males but results were inconclusive in females.97

The researchers commented that mean BP and prevalence were different amongst South Asian subgroups but they were usually combined into one homogenous group.97 This is an important finding given that the classic risk factor profile of these subgroups differs, which may affect any analyses associating them with PAD. A meta-analysis in India found that hypertension is rising in general, and significantly more so in urban than rural populations,98 suggesting that changes in lifestyle and environmental factors play an important role. This is supported by a recent UK migrant study, which found a higher prevalence of hypertension in migrants than in Indigenous Indians.74

While being a risk factor for PAD, hypertension appears to have a stronger association with CAD and both haemorrhagic and ischaemic stroke. The association appears to be greater for ischaemic stroke.99 Treating hypertension reduces the incidence of both haemorrhagic and ischaemic stroke.99 This finding may explain why CBVD is more common in African Caribbeans.100,101 It may also explain the higher risk of and mortality from stroke in South Asians compared to Caucasians in the United Kingdom.102

There is a paucity of data regarding the association between hypertension and PAD in different ethnic groups. The US studies have shown that among patients with PAD, the prevalence of hypertension was higher in African Americans than NHW14,59 and lower in Asians.14 A population-based study in India found that PAD patients had a significantly higher mean SBP than those without PAD13 and the OR for hypertension and PAD was 2.7.13 However, more research needs to be undertaken to explain the variation in PAD with hypertension in different ethnic minority groups.

**Novel risk factors and/or associations for PAD**

Several novel risk factors have been investigated in recent years in an attempt to establish further associations with PAD. While there are considerable interactions between established and novel risk factors, it is thought that the novel risk factors have independent associations with the development of PAD. Indeed after adjusting for traditional risk factors some novel risk factors still remain significantly associated with PAD.22,42 Although most of the research has been carried out on predominantly Caucasian populations, limited research suggests that novel risk factors play some role at least in the ethnic susceptibility to PAD.22
Inflammation

Inflammation plays a major role in all stages of atherosclerotic vascular disease. Indeed, inflammation occurs in response to a variety of stimuli and is associated with classic cardiovascular risk factors, including smoking, dyslipidaemias, hypertension, DM, obesity and infection. Inflammatory markers such as IL-6, CRP and fibrinogen have been investigated in numerous studies and are associated with PAD development, progression and its severity.

Elevated CRP levels have been shown to predict the development of type 2 DM and have also been associated with a higher risk of future coronary events and symptomatic PAD. Furthermore, CRP has been found to be inversely associated with ABPI as well as with endothelial dysfunction and PAD disease severity.

It appears that different ethnic groups have a different susceptibility to raised CRP. In the United Kingdom highest levels were seen in Pakistani women and much lower levels in Black Caribbeans. Limited research has found the association between CRP and PAD is stronger in Blacks than in Caucasians. However the researchers in this study commented that inflammation was only associated with PAD in higher levels of CRP suggesting a threshold effect for inflammation. Therefore the association between inflammation and PAD may have been stronger in Blacks because a greater number may have reached the threshold levels of inflammation.

A similar but non-significant difference across ethnic groups was also present for fibrinogen.

Lp(a) has been associated with atherosclerosis and thrombogenesis and has been shown to be an independent risk factor for CAD and PAD. Levels have also been found to correlate with more severe forms of PAD. Lp(a) levels increase steadily from absence of PAD to mild and severe PAD.

Lp(a) is thought to be an important molecule because its levels are mainly genetically determined and are only weakly associated with environmental factors, including the classical vascular risk factors. This suggests that any effects of Lp(a) in the development of PAD are unlikely to be due to confounding from classical vascular risk factors. There appears to be variation in Lp(a) levels among populations with South Asians having higher serum concentrations of Lp(a) when compared to the general population in the United States and the United Kingdom. This increase, found in indigenous Indians, is not affected by migration. However, we could find no studies specifically investigating Lp(a) and PAD in South Asians.

Black people have substantially higher Lp(a) levels than do Caucasians and Asians and this difference is not explained by differences in apo(a) size distribution, or so far by other genetic factors. A recent study found that as well as African Americans having higher Lp(a) levels than Caucasians, higher levels of log Lp(a) were independently associated with a lower ABPI in this group.

Studies investigating Lp(a) and CAD suggest this molecule does not explain the susceptibility of different ethnic groups to CAD, as both South
Asians and Blacks have high Lp(a) but only South Asians have an elevated risk of CAD.47,74 Indeed Black people have a lower CAD mortality than Caucasians in the United Kingdom.45 Most of the research investigating Lp(a) and PAD has been on predominantly Caucasian populations. However, the differences between Black and non-Black populations regarding Lp(a) levels might provide a possibility to assess any relationship between PAD risk and Lp(a) even though findings in CAD do not support this.

**Homocysteine**

Clarke et al.127 were the first to report that hyperhomocystinaemia could be an independent risk factor for atherosclerosis. Homocysteine has been thought to be associated with thrombotic events although the precise mechanisms by which elevated homocysteine levels contribute to the pathogenesis of vascular disease are unknown. Homocysteine is associated with male sex, smoking, renal failure and deficiencies of enzymes involved in homocysteine metabolism128,129 as well as being negatively correlated to dietary factors.130,131 Importantly Homocysteine has been associated with an increased risk of PAD and lower ABPI measurements in several studies25,42,132 although this finding has been disputed by others.133 As homocysteine levels increase following an acute thrombotic event, it is difficult to interpret whether increased thrombosis is due to elevated homocysteine or vice versa and may be the reason why a link between hyperhomocysteinaemia and PAD has not been confirmed conclusively.

The UK studies found that fruit and vegetable consumption is higher in South Asians and Blacks than in the general population46,69 but there was considerable variability within the South Asian group. Interestingly, one UK study found homocysteine levels to be raised in South Asian Hindus but not Muslims; with levels being lowest in Blacks.134 Levels also appear to be lower on migration to the United Kingdom from the Indian subcontinent.74 Although a previous US study found elevated levels of plasma homocysteine in Asians,135 these findings suggest that both genetic and environmental factors could play a role in determining its levels.

Recently homocysteine levels were found to be similar in Blacks and Caucasians42 but this was not supported by a previous finding which found elevated levels in the former group.42 The general lack of research in this area makes it difficult to elicit any real difference in homocysteine level contributing to the ethnic variation in PAD.

**The metabolic syndrome**

Many studies have identified a pattern of interrelated metabolic disturbances that are associated with cardiovascular risk, which are referred to as the so-called ‘metabolic syndrome’. The current definition of metabolic syndrome requires the presence of three or more of the following: increased waist circumference, reduced HDL cholesterol, increased triglycerides, hypertension, impaired fasting glucose or DM.136 Metabolic syndrome varies by ethnic group and is especially prevalent amongst South Asians.79 Even though African Caribbeans have a tendency for insulin resistance and an increased risk of DM, they appear to have a more favourable lipid profile than South Asian85 and thus do not fit the typical definition of metabolic syndrome.137 Metabolic syndrome has been associated with the development and progression of and more advanced PAD.138-140 However, with the components of metabolic syndrome being associated with low ABPI, defining the syndrome does not add any more per se to the patient’s classical risk factor status when assessing the risk of suffering from asymptomatic PAD.140 While this syndrome has been associated with PAD, it seems clear that ethnicity/race play a greater role in conferring susceptibility to this disease.

**Chronic kidney disease**

While not being a novel risk factor per se, it is becoming increasingly apparent that CKD is a significant risk factor for all vascular diseases, with these patients experiencing up to a 10-fold increased risk of cardiovascular mortality than the general population.141 A recent study reported patients with CKD and PAD had a much higher mortality than patients with either CKD or PAD alone, or no disease.142 Various measurements of CKD severity, including albuminuria, estimated glomerular filtration rate (eGFR) and serum creatinine or presence of end stage renal disease (ESRD),143-145 have been associated with PAD. ESRD patients having an increased risk and poorer prognosis, including less successful revascularization procedures and amputation,145 than those without. Whilst no studies have investigated all of the risk factors for PAD specifically in patients with CKD,146 CKD and PAD share similar risk factors, including DM, obesity, hypertension, dyslipidaemia and CRP.143,147 Homocysteine levels, which are often elevated in dialysis patients, have been associated with PAD in this group.148
The prevalence of CKD is reported as being higher among Blacks and Asians with diabetes than in Whites.\textsuperscript{149–152} Blacks develop ESRD a decade earlier than Whites,\textsuperscript{153} are more likely to have ESRD attributable to hypertension than Whites and are less likely to have renal artery stenosis than Whites.\textsuperscript{154,155} However, once dialysis is commenced, Whites have a higher mortality than either Blacks or Asians.\textsuperscript{156,157} Interestingly a recent study reported Whites more likely to develop incident or recurrent PAD events (Amputation, AAA or iliofemoral bypass) than Blacks, in patients on dialysis, after adjusting for traditional cardiovascular and dialysis-related risk factors.\textsuperscript{158} Further research is required to ascertain whether ethnic differences in CKD are responsible for the apparent ethnic difference in PAD prevalence, although clearly the two are associated diseases that share many of the same risk factors.

**PAD distribution in ethnic groups**

As well as the variations that exist in vascular risk factors, the anatomical distribution of vascular disease appears to differ between ethnic groups. Black people have an increased incidence of CBVD and ESRD than Caucasians and a lower incidence of CHD, whereas South Asians appear to have a higher incidence of CHD.\textsuperscript{57} Caucasians, on the other hand, appear to have a higher rate of abdominal aortic aneurysm (AAA).\textsuperscript{159} However, there are no significant population studies estimating the prevalence of AAA in minority ethnic groups. Interestingly when AAA are found in Blacks, they present at a significantly younger age than Caucasians and atherosclerosis is less likely to be the causative factor.\textsuperscript{160,161} In people with strokes, Black people appear to have more cerebral small vessel and less large vessel atherosclerotic disease than Caucasians.\textsuperscript{162} They also have a greater mortality from this disease than Caucasians.\textsuperscript{45}

Previous studies have indicated that African Caribbeans and South Asians seemed to report more distal PAD than the general population.\textsuperscript{18,85,159,160,163} In Indians, *thromboangiitis obliterans* appears to contribute to this increase.\textsuperscript{164} In a previous hospital-based study in India, this disease reflects the younger age of presentation of patients with distal PAD.\textsuperscript{164} The recent study by Chaturvedi et al.\textsuperscript{17} found that Indian Asians had less lower limb atherosclerosis than Europeans, while common carotid intima-medial thickness (CIMT) did not differ and disease distribution was similar. This finding is consistent with other studies reporting lower rates of limb amputation in Indian Asians compared to Europeans.\textsuperscript{13,16,77}

It is not known whether distal PAD is due to genetic differences in disease distribution or the fact that the incidence of DM is higher in South Asians and Blacks than in Caucasians and DM has been shown to be associated with distal PAD. There is evidence to suggest that there are regional differences in atherosclerosis risk susceptibility to specific risk factors.\textsuperscript{17} DM usually affects smaller arteries and therefore is associated with femoro-popliteal and tibial PAD.\textsuperscript{37,165} This pattern differs from other risk factors such as smoking and hypertension which are associated with more proximal disease in the aortoiliac vessels.\textsuperscript{37} However, the finding of distal PAD in South Asians and Blacks is apparent even after adjusting for the increased prevalence of DM.\textsuperscript{166}

**Conclusion**

PAD is an important healthcare problem worldwide due to morbidity and mortality associated with this condition. Most population-based studies investigating PAD prevalence and risk factors for its development and progression have been based on predominantly Caucasian groups. Much less is known about the characteristics of this disease in ethnic minority groups. As a consequence, decisions regarding management of PAD, including risk factor modification, have been made on the basis of majority Caucasian populations. Understanding the epidemiology of PAD amongst ethnic groups is relevant, given that the proportion of minority ethnic groups in the United Kingdom rose by 53\% between 1991 and 2001\textsuperscript{31} and it is likely to rise further in the next census of 2011. It is also evident that there is considerable heterogeneity in risk factors within the South Asian ethnic group, which may affect any conclusions made linking PAD and ethnicity to traditional vascular risk factors.

Although South Asians have an apparently worse risk factor profile than the general population and a greater risk of CAD, they appear to have a lower prevalence of PAD. This could be a reflection of the higher premature death rate from CAD in South Asians compared to the general population.\textsuperscript{45} Susceptible South Asians may not live long enough to develop symptoms of PAD. Given that PAD prevalence risk increases with age the apparent finding of lower prevalence in South Asians may be genuine; however, communication difficulties in describing symptoms of PAD which may result in its under diagnosis in this group. Amongst people of Black ethnicity, there is a discrepancy as to whether or not they have a higher prevalence of PAD.
Given the lack of concordance between risk of CAD, CBVD and PAD in South Asian and Black ethnic groups it would appear likely that the development and distribution of chronic vascular diseases have some genetic basis. With little data it is hard to make firm conclusions regarding the importance of risk factors in the development of PAD in different minority ethnic groups.

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