GCA can occur in people of colour: an international, multicentre perspective.

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Giant cell arteritis (GCA) is the most common form of systemic vasculitis in individuals aged 50 years or older. GCA is considered a medical emergency. Urgent diagnosis and initiation of treatment are critical to prevent devastating potential sequalae of permanent visual loss and vascular complications. GCA may present with non-specific symptoms and whilst fast-track investigative pathways may improve patient outcomes, cognitive biases may confound patient selection and interpretation of results, especially where there is diagnostic ambiguity [1, 2]. Commonly-heard heuristics in teaching include "GCA is rare in ethnic minorities", which in our experience often extends to "GCA only occurs in white people". Given the importance of pre-test probability in diagnosis, even with modern imaging tests, such myths can have devastating consequences for people of colour.

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Epidemiological studies consistently demonstrate the highest incidence of GCA in Northern European populations[3]. Genetics are predictors of disease incidence and genetic traits tend to cluster geographically. Whilst ethnicity-related differences can be a useful prompt, over-simplification can lead to premature diagnostic closure and false attribution of the significance of race to biological causative mechanisms. Although it is widely accepted that GCA has variable global incidence, skin colour should not be used as a surrogate marker to define populations. It has been proposed that race is a socio-political construct, not a 'natural' biological variable. Defining populations on arbitrary features, such as skin pigmentation without genetic or phenotypic uniformity, promotes societal and institutional racism and may drive inequity[4,5]. Although the

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recording and reporting of ethnicity currently confers limited meaning, we acknowledge that these data are still important and may have potential future research implications.

Despite sufficient evidence that GCA can occur in people of colour, there is resistance to dismiss the popular contrary dogma. In a seminal study of 586 patients who underwent temporal artery biopsy, population-adjusted age-and sex-standardized incidence rates per 100,000 patients were 3.1 for black patients and 3.6 for white patients, suggesting near parity [6]. Our literature review (table 1) demonstrates that large observational datasets corroborate this finding, including recent case series. Furthermore, the true incidence of GCA among non-white groups is likely under-reported in currently available real-world data for the following reasons. Firstly, inclusion in epidemiological research is subject to ascertainment bias and varies greatly by geography, with most vasculitis registries recruiting patients in affluent, resource-rich, predominantly white regions. The collection of ethnic and racial data in some countries (such as Germany, France, Sweden, Denmark) is also legally prohibited to avoid potential discriminatory practice[7].Secondly, despite initiatives promoting patient and public involvement in healthcare access, policy and research, minority ethnic participation and recruitment is suboptimal [8]. To tackle this, it is imperative to examine and dismantle structural, language and cultural barriers and address historical mistrust. Finally, ethnic group classification is problematic and difficult to establish on self-reported categories. Even the 'white' category is heterogeneous and may not originate from North Europe, as exemplified by a Brazilian cohort study of GCA patients where the 'white' group included people of Portuguese, Italian and Spanish descent[9].

Our understanding of the aetiopathogenesis of GCA is evolving. It is difficult to unravel the true contribution of genetic and environmental factors, such as seasonality and infective precipitants. The north-south gradient of incidence of GCA (increased at higher latitudes) observed in European studies[3] follows a similar pattern to population prevalence of the main genetic risk factor, HLA-DRB1*04[10] but this is far from an actionable understanding of genetic predisposition in GCA. The role of other genetic polymorphisms in explaining geographic differences is yet to be defined. Although contemporary genetic ancestry tests are widely available, their reliability and clinical relevance is unclear.

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Timely recognition and management of GCA is paramount and misdiagnosis can be devastating. The popular rhetoric that GCA is only a disease of North European ancestry is not supported by observational data. There is high probability that such data are under-reporting the incidence of GCA among people of colour. We propose that race should not influence diagnostic decision-making in GCA. Risk assessment should focus on clinical presentation, rather than ethnic profiling. Rheumatologists have a responsibility to remain open and mindful to avoid inadvertently further reinforcing inequity. Careful communication within and cross-speciality, and with patients (including appropriate patient information resources) is critical to avoid racial stereotyping and contributing to worse health outcomes for people of colour. The research agenda should be relevant and representative for all patients. To achieve this will require commitment, collaboration and funding. Re-examining cognitive biases to align with inclusion and diversity principles will reduce health care inequalities and improve outcomes for all patient populations, including people of colour[1].

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Author contributions

TG and PM drafted the manuscript. AS, SH, MP contributed real-world data. All authors contributed to discussions, revisions and approved the final version of the manuscript.

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