Increased ethnicity and socioeconomic data collection required in stroke associated with COVID-19

For over 4 months, we have been in the privileged position of witnessing real-time trends in the literature via our weekly curation of The Neurology and Neuropsychiatry of COVID-19 blog.1 There is an emerging recognition of the need to report the severity and outcomes of neurologically complications of SARS-CoV-2 infection. Stroke is a rare but well-documented complication, with most prevalence estimates varying slightly around 0.8%.2 It is therefore important to elucidate both those at risk of developing stroke and stratifying those at risk of more severe outcomes when stroke occurs.

We were greatly interested to read the letter by Dmytriw et al,3 which highlighted potential racial disparities in outcomes from ischaemic stroke after COVID-19. Their novel study built on the hypothesis that black and minority ethnic (BAME) individuals have poorer outcomes from COVID-19 in general.4 An editorial commentary by Renieri5 further highlighted the need to elucidate mechanisms that may drive an interaction between stroke and COVID-19 to explain increased mortality in ethnic minorities. Previously also, Katz et al reported that patients with stroke with COVID-19 were more likely to be from a BAME population than patients without COVID-19. The potential over-representation of BAME individuals in COVID-19 stroke cohorts raises many questions. Could it be due to increased prevalence of COVID-19 in BAME groups?2 Or are BAME populations at relatively greater risk of severe outcomes, including stroke, after COVID-19?4

Through curation of our blog, we have noted that many studies of stroke in the context of COVID-19 infection are published each week. However, reviewing our database of studies to date (8th September 2020), we found that only 16/64 (25%) primary research studies, 2/42 (4.8%) case reports and 6/35 (17.1%) secondary review articles referred to ‘ethnicity’ or ‘race’ in the context of the explicit coincidence of stroke and COVID-19. These proportions suggest to us that opportunities are being missed to increase our understanding (or refutation) of potential racial disparities.

We therefore echo the recommendation of Public Health England to ‘mandate comprehensive and quality ethnicity data collection and recording’.4 The findings from Dmytriw et al’s study lead us to question further why there may be an increased stroke risk and mortality in African–Americans. They found for instance that significantly more African–American patients with stroke with COVID-19 had diabetes (63% vs 28.6%) and higher levels of low-density lipoproteins (37.7% greater) in general. Diabetes is more common in people from black backgrounds than in the general population, and it is well recognised that comorbidities confer higher risk of stroke and mortality. Accordingly, one possibility is that an increase in stroke mortality rates in BAME groups may simply reflect an increased prevalence of comorbidities such as diabetes or higher cardiovascular risk.

A second possibility is that race and ethnicity may interact with comorbidity and lower socioeconomic background to limit access to care. Both Dmytriw et al and Katz et al proposed social determinants of health, access to care and geographical differences as potential mediators of the increased incidence and mortality of COVID-19-related stroke. Stakeholders interviewed by Public Health England4 argue further that COVID-19 has exacerbated existing health inequalities and highlight the lack of targeted programmes for chronic disease prevention in these groups. Although explanatory mechanisms remain unclear, we noted in passing the lower prevalence of thrombectomy and use of tissue plasminogen activator in African–Americans in Dmytriw et al’s cohort.

Great caution is required in interpreting racial discrepancies in outcomes from COVID-19. The influence of ethnicity is likely to be complex and multifactorial. Renieri drew on broader COVID-19 research on increased mortality associated with black race to suggest that after adjusting for various socioeconomic factors, black race with COVID-19 was not associated with increased in-hospital mortality. Although this finding goes some way to exploring potential underlying mechanisms driving racial disparities, it may yet miss mediators of mortality associated with stroke in COVID-19 specifically or influences on higher viral transmission among black individuals.

In future, for instance, studies might hypothesise an interaction between these outcomes and overcrowded housing, greater reliance on public transport, living in dense population centres, occupational risks and/or socioeconomic inequalities.4 Katz et al proposed further that differences in risk factor modification, diet and genetic predisposition may also contribute to health outcome inequality. We commend the authors of these studies for highlighting these issues and the potential reasons behind any racial disparity. It is notable, however, that both Katz et al’s and Dmytriw et al’s studies collected data from North America. Differing ethnic profiles, healthcare systems and the impacts of racial discrimination between North America and other countries may it difficult to generalise any social and structural effects on racial disparities in COVID-19 infection and stroke but are useful in highlighting a gap in the global literature.

Therefore, we wish to propose that alongside collecting and recording comprehensive ethnicity data, prospective stroke registers and studies in COVID-19 broaden globally beyond simply measuring race. Socioeconomic background, occupation, lifestyle, housing circumstances, access and attitudes to care, and feelings of discrimination may all help shed light on mechanisms for potential racial disparity. This added granularity of detail will help build high-quality evidence to unpick the complex effect of race on health inequalities driving differential racial outcomes in stroke after COVID-19.
Competing interests None declared.

Patient consent for publication Not required.

Provenance and peer review Not commissioned; internally peer reviewed.

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Received 8 September 2020
Accepted 9 September 2020
Published Online First 18 September 2020

doi:10.1136/jnnp-2020-325057

ORCID ID
Danish Hafeez http://orcid.org/0000-0003-3712-136X

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