Cardiac markers in Black, Asian and minority ethnic (BAME) patients with COVID-19

The novel coronavirus, SARS-CoV-2, which causes COVID-19, has rapidly progressed to a global health emergency.[1] COVID-19 predominantly presents with respiratory complications and associated mortality; however, emerging reports show that patients can present with cardiac involvement.[1] Cardiovascular manifestations are not unique to COVID-19 as cardiac involvement have been reported with SARS-CoV and Middle East respiratory syndrome-CoV in the past.[2] The pandemic in the UK has highlighted the higher risk to the Black, Asian and minority ethnic (BAME) population of adverse clinical outcomes compared with patients of white British ethnicity.[3] Furthermore, BAME men aged over 50 years are noted to be at an even higher risk of severe COVID-19.[4]

We retrospectively reviewed the cardiac markers Troponin T, N-terminal pro B-type natriuretic peptide (NT-proBNP) and the inflammation marker C reactive protein (CRP) requested in patients with suspected COVID-19, admitted to a tertiary London hospital during the peak of the pandemic (April 2020) in the UK. Only patients who had a Troponin measurement (with or without NT-proBNP) were included and we assessed the difference in biomarkers on initial presentation to emergency department (ED) between the BAME and white populations.

Troponin T and CRP were measured by Roche (Roche c-702 and e-801, Burgess Hill, UK) and NT-proBNP by Immulite (Siemens Healthineers, Frimley, UK) platforms, respectively. Data were compared using Mann-Whitney U test, χ^2 test and Kruskal-Wallis analysis of variance (ANOVA) with Bonferroni correction as relevant, using Analyse-IT (V.5.2). Data are presented as median (IQR) and a p value <0.05 was considered significant.

There were 167 patients with cardiac markers and a positive COVID-19 serology, out of which 122 (73%) were BAME. Compared with the white population, BAME patients were younger ($p<0.001$) table 1. A total of 47 (28%) were admitted to intensive therapy unit (ITU) table 1. In both groups, this was predominantly men (74% for BAME and 75% for white).

All whites in ITU were aged ≥50 years while 77% of BAME were aged ≥50 years. All patients subsequently admitted to ITU had higher CRP (172.6 (93.3–251.1) mg/L vs 92.5 (42.6–146.8) mg/L) and Troponin (27 (15–61) ng/L vs 15 (2–29) ng/L) on presentation to ED compared with non-ITU admissions (both $p<0.01$). However, NT-proBNP was similar between ITU, 219 (88–863) pg/mL and non-ITU, 242 (72–882) pg/mL ($p=0.68$) admissions. There were no differences in CRP, Troponin or NT-proBNP between the BAME and white ITU admissions. There were also no differences in CRP, Troponin or NT-pro BNP in those aged less than or more than 50 years in both groups.

Table 1 Demographics and biochemical data in BAME and white British populations

	BAME (n=122)	White (n=45)	P value
Age (years)	58 (53–64)	73 (64–81)	<0.0001
Male n (%)	84 (69%)	29 (64%)	NS
C reactive protein (mg/L)	114.5 (69.3–191.4)	62 .4 (23.7–185.2)	NS
Troponin T (ng/L)	19.0 (8.2–52.2)	35.0 (16.0–74.0)	0.02
NT-pro BNP (pg/mL)*	221.5 (97.5–880.5)	427 (133.5–2797)	NS
ITU admissions n (%)	35 (29%)	12 (27%)	NS

*BAME (n=32) and White (n=11).
BAME, Black, Asian and minority ethnic; ITU, intensive therapy unit; NS, not significant; NT-proBNP, N-terminal pro B-type natriuretic peptide.

In our cohort, those of BAME ethnicities presented with relatively higher inflammatory response but with lower Troponin and similar NT-proBNP compared with those of white ethnicity. However, out of the 55 patients who had a Troponin >40 ng/L, 65% were of BAME ethnicities, reflective of the higher percentage of BAME being admitted to our hospital. Compared with whites, younger BAME patients required ITU admissions.

The pathophysiological mechanisms underlying myocardial injury due to COVID-19 are not fully elucidated, but multiple studies have indicated higher Troponin concentrations in patients with severe COVID-19.[5] If COVID-19 is manifesting via vascular endothelial dysfunction, individuals with pre-existing cardiovascular risk factors, as seen in BAME populations, will be at a much higher risk of acute coronary syndrome.[6] However, a study of 1326 cases from the UK Biobank showed that the greater risk of severe COVID-19 in BAME populations is not explained by cardiometabolic, socioeconomic or behavioural factors.[4] Our data are in keeping with this as we did not observe higher cardiac markers in BAME compared with the white population.

Limitations of our data are that it was only laboratory based. There was also variation in requesting patterns and a lack of pre-existing comorbidity details, clinical management and clinical outcome for these patients. Nonetheless, our aim was to assess cardiac involvement on presentation to ED based on baseline biochemistry.

A recent study on a German cohort has demonstrated cardiac involvement in 78% and ongoing myocardial inflammation in 60% patients, independent of pre-existing comorbidities, severity and overall course of the acute illness as well as time from the initial presentation with COVID-19.[7] This further highlights the need for larger studies to better understand the pathophysiology of COVID-19-related cardiovascular disease including in different ethnicities.

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