




## High sensitivity troponin and COVID-19 outcomes

Nikolaos Papageorgiou<sup>a,b</sup>, Catrin Sohrabi<sup>a</sup> , David Prieto Merino<sup>c,d</sup> , Angelos Tyrllis<sup>a</sup>, Abed Elfattah Atieh<sup>a</sup>, Bunny Saberwal<sup>a</sup>, Wei-Yao Lim<sup>a</sup>, Antonio Creta<sup>a</sup>, Mohammed Khanji<sup>a</sup>, Reni Rusinova<sup>a</sup>, Bashistraj Chooneea<sup>a</sup>, Raj Khiani<sup>d,e</sup>, Nadeev Wijesuriya<sup>e,f</sup>, Anna Chow<sup>e,f</sup>, Haroun Butt<sup>e,f</sup>, Stefan Browne<sup>e,f</sup>, Nikhil Joshi<sup>e,f</sup>, Jamie Kay<sup>e,f</sup>, Syed Ahsan<sup>a</sup> and Rui Providencia<sup>a,g</sup> 

<sup>a</sup>St. Bartholomew's Hospital, Barts Health NHS Trust, London, UK; <sup>b</sup>Institute of Cardiovascular Science, University College London, London, UK; <sup>c</sup>Department of Non-communicable Disease Epidemiology, Faculty of Epidemiology and Population Health, London School of Hygiene & Tropical Medicine, London, UK; <sup>d</sup>Applied Statistics in Medical Research Group, Catholic University of Murcia (UCAM), Murcia, Spain; <sup>e</sup>Barnett Hospital, London, UK; <sup>f</sup>Royal Free Hospital, London, UK; <sup>g</sup>Institute of Health Informatics Research, University College of London, London, UK

### ABSTRACT

**Background:** Recent reports have demonstrated high troponin levels in patients affected with COVID-19. In the present study, we aimed to determine the association between admission and peak troponin levels and COVID-19 outcomes.

**Methods:** This was an observational multi-ethnic multi-centre study in a UK cohort of 434 patients admitted and diagnosed COVID-19 positive, across six hospitals in London, UK during the second half of March 2020.

**Results:** Myocardial injury, defined as positive troponin during admission was observed in 288 (66.4%) patients. Age (OR: 1.68 [1.49–1.88],  $p < .001$ ), hypertension (OR: 1.81 [1.10–2.99],  $p = .020$ ) and moderate chronic kidney disease (OR: 9.12 [95% CI: 4.24–19.64],  $p < .001$ ) independently predicted myocardial injury. After adjustment, patients with positive peak troponin were more likely to need non-invasive and mechanical ventilation (OR: 2.40 [95% CI: 1.27–4.56],  $p = .007$ , and OR: 6.81 [95% CI: 3.40–13.62],  $p < .001$ , respectively) and urgent renal replacement therapy (OR: 4.14 [95% CI: 1.34–12.78],  $p = .013$ ). With regards to events, and after adjustment, positive peak troponin levels were independently associated with acute kidney injury (OR: 6.76 [95% CI: 3.40–13.47],  $p < .001$ ), venous thromboembolism (OR: 11.99 [95% CI: 3.20–44.88],  $p < .001$ ), development of atrial fibrillation (OR: 10.66 [95% CI: 1.33–85.32],  $p = .026$ ) and death during admission (OR: 2.40 [95% CI: 1.34–4.29],  $p = .003$ ). Similar associations were observed for admission troponin. In addition, median length of stay in days was shorter for patients with negative troponin levels: 8 (5–13) negative, 14 (7–23) low-positive levels and 16 (10–23) high-positive ( $p < .001$ ).

**Conclusions:** Admission and peak troponin appear to be predictors for cardiovascular and non-cardiovascular events and outcomes in COVID-19 patients, and their utilisation may have an impact on patient management.

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## Introduction

Coronavirus disease 2019 (COVID-19) caused by SARS-CoV-2 is a global pandemic [1]. So far, it has affected more than 100 million people worldwide, and is associated with multi-organ dysfunction and high mortality rates [1].

Studies suggest that some patients present to hospital and have a relatively benign course, being discharged within a few days. However, for other patients the disease course is more aggressive, requiring multiple interventions, while they experience higher mortality and longer in-hospital stay [2]. It

would be therefore of importance to have a biomarker which could help in distinguishing between these two groups of patients, not only for prognosis, but also, potentially, for treatment decisions.

Troponin is a marker of myocardial injury, but it is also found to be raised in several conditions. Recent reports demonstrated high troponin levels in patients affected by COVID-19. These were found to have higher mortality rates during the initial outbreak in Wuhan cohorts [3,4]. In addition, higher mortality rates have been observed in the UK, as an older and multi-ethnic population with more comorbidities was affected by the disease [5].

**CONTACT** Rui Providencia  [r.providencia@ucl.ac.uk](mailto:r.providencia@ucl.ac.uk) Barts Heart Center, St. Bartholomew's Hospital, West Smithfield, London EC1A 7BE, UK

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It is still unknown which patients are more likely to develop myocardial injury in the setting of COVID-19, and whether or not, after adjustment for confounders associated with rise in troponin, myocardial injury can be used as an independent predictor of the disease in Western multi-ethnic populations. Yet, it remains to be determined if admission troponin can be used as a predictor, and if the magnitude of troponin rise translates into different outcome rates (i.e. whether patients with higher rise in troponin levels experience a more severe disease progression than those with negative or mildly increase in troponin levels).

We aimed to assess: (1) the risk factors for myocardial injury; (2) the impact of myocardial injury on different COVID-19 associated outcomes and (3) whether admission troponin, peak troponin and magnitude of troponin rise have a similar prognostic capacity.

## Methods

In this multi-centre study, we assessed the association between high-sensitivity troponin (hsTrop) and COVID-19 intra-hospital clinical trajectory (comprising mortality, utilisation of procedures, and cardiovascular and non-cardiovascular outcomes) in a UK cohort of 434 patients admitted and diagnosed positive, across six hospitals in London, UK. All patients admitted to the participant hospitals from the 16th to the 30th of March 2020 with a diagnosis of COVID-19 and having at least one troponin measurement were considered eligible for analysis. This observational study was approved by the Clinical Effectiveness Unit at Barts Health NHS Trust (*Project ID: 11103; Title: COVID-19 and cardiovascular disease (CVD) outcomes*) and by the Quality Governance Department at Royal Free London NHS Trust (*Cardiovascular Implications of Outcomes of Patients With COVID-19; 22/04/2020*).

Patient demographic characteristics, laboratory results, procedures, comorbidities, procedures and outcomes were extracted from the electronic records and paper notes. In order for patients to be included in the study, these should be diagnosed COVID-19 positive, as confirmed by polymerase chain reaction (PCR) swab. Patients with two positive swabs and older than 16 years were included in the study. Troponin T levels were measured with a high-sensitivity assay on and during admission as per Trusts' protocols.

## Measurement of blood parameters

Routine bloods were obtained from patients on and during admission. Routine hospital laboratory methods

were used for the analysis. These were available on the electronic systems and included: full blood count parameters, high-sensitivity troponin T, C-reactive protein (CRP), lactate dehydrogenase (LDH), N-terminal pro B-type natriuretic peptide (NT-proBNP), international normalised ratio (INR), creatine kinase (CK), D-Dimers, activated partial thromboplastin time (APTT), fibrinogen, thrombin time and creatinine. Glomerular filtration rate (eGFR) was estimated using the Modification of Diet in Renal Disease (MDRD) Study equation. Patients were classified as having moderate CKD when eGFR was less than 60 ml/min.

Troponin measurements were performed based on clinical indication or based on local protocols for risk stratification of COVID-19 patients.

The term "*myocardial injury*" was used for patients with positive troponin levels (defined as  $\geq 15$  ng/mL – the 99th percentile in the normal population according to our lab). Peak troponin for each patient was measured and based on these values, three troponin levels were defined, utilising tertiles to define the cut-offs, as: "negative" if  $< 15$  ng/mL (no myocardial injury according to our definition above), "low-positive" if levels  $\geq 15$  ng/mL and  $< 47$  ng/mL and "high-positive" level if  $\geq 47$  ng/mL.

## Study endpoints

The study primary endpoint was defined as all-cause mortality. Secondary outcomes were: (i) pneumonia, (ii) acute kidney injury (defined as a 50% increase in creatinine compared to chronic/baseline levels), (iii) myocardial injury (defined as high-sensitivity troponin above the 99th percentile of normal), (iv) acute heart failure, (v) acute atrial fibrillation episode, (vi) stroke, (vii) venous thromboembolic disease (including pulmonary embolism and/or deep vein thrombosis) and (viii) utilisation of procedures (non-invasive ventilation, mechanical ventilation, ECMO and renal replacement therapy).

## Statistical analysis

Descriptive statistics are presented as proportions for binary variables and median and inter-quartile for continuous variables. Parametric (Student's *T*) or equivalent non-parametric tests (Mann–Whitney) were used where appropriate for comparisons of continuous variables among groups. Proportions were compared with a Chi<sup>2</sup> test.

Binary logistic regression was used to assess for predictors of myocardial injury using the forward

likelihood ratio (LR) method, with probability for step-wise .05. Significant predictors identified through univariate analysis are added in the multivariable model through a stepwise process with predictors entering at each step as long as they significantly improve the predictive capacity of the model.

The association of troponin levels with each predefined procedure and clinical outcome was assessed with a separate logistic regression. Each model was adjusted for the clinical variables that showed significant association with myocardial injury at baseline (age, hypertension, dyslipidaemia, diabetes, previous myocardial infarction/ischaemic heart disease, asthma, chronic obstructive pulmonary disease – COPD – and eGFR). Duration of hospital stay (median and inter-quartile range) was also compared for the three different troponin levels.

Results with  $p < .05$  were considered as significant. PASW Statistics (SPSS Inc., Chicago, IL) version 18.0 was used for statistical analysis.

## Results

During the study period, 629 patients were admitted and diagnosed with COVID-19. Among those, troponin measurements were performed during admission in 434 patients (Figure 1). Demographic characteristics of the patients enrolled in this study are presented in Table 1. The median age was 66 (56–80) years, and 62.9% of patients were men. A positive troponin was detected at least once during admission in nearly two thirds of patients. Seventeen patients (3.9%) were diagnosed with myocardial infarction during the admission. Median left ventricular ejection fraction was significantly lower for troponin positive patients as compared to troponin negative patients ( $p = .015$ ).

Patients with positive troponin levels were significantly older ( $p < .001$ ), and more frequently had risk factors such as hypertension ( $p < .001$ ), hyperlipidaemia ( $p < .001$ ), asthma ( $p = .031$ ) or COPD ( $p = .026$ ) and were less likely to be of Asian ethnicity as compared to troponin negative patients (Table 1).

Troponin positive patients had significantly higher white blood cell count, lower haemoglobin levels, higher creatinine levels, lower eGFR, higher D-Dimer levels and higher NTpro-BNP levels (Table 2).

## Factors associated with myocardial injury

Our analysis showed that age, Asian ethnicity (as compared to Caucasian), hypertension, diabetes, asthma, COPD and moderate CKD were associated with myocardial injury on univariate analysis (Tables 1 and 3). However, after adjusting for baseline differences only age, hypertension and moderate CKD independently predicted myocardial injury on multivariate analysis (i.e. after correcting for confounders, the probability of myocardial injury was found to increase by 1.68 per every 10 additional years of age, and was 1.81 and 9.12 times higher when in the presence of hypertension and/or moderate CKD, respectively).

## Myocardial injury and cardiovascular & non-cardiovascular events, and procedures

Median length of in-hospital stay was 12 (6–20) days. This was longer for troponin positive patients [15 (9–23) versus 8 (5–13) in negative troponin patients,  $p < .001$ ].

Among patients with troponin measurements, 109 patients had two troponin measurements, 33 patients had three measurements and 61 patients had four or more troponin measurements. During these repeat measurements, an additional 24 patients who initially tested negative presented with positive troponin results.

Seven patients stayed in hospital for less than 24 h: four were discharged and three died within the first day. All the patients who died within 24 h had a positive troponin in the higher level, and those discharged on the same day had a negative troponin.

After adjustment for baseline differences (age, hypertension, diabetes, previous myocardial infarction/ischaemic heart disease, asthma, COPD and eGFR),



Figure 1. Flowchart illustrating cohort selection.

**Table 1.** Demographic characteristics of the study population on admission and association with myocardial injury.

	All (n = 434)	Negative hsTrop <15 ng/L (n = 146)	hsTrop ≥15 ng/L (n = 288)	p
<i>Demographics &amp; risk factors</i>				
Age	66 (56–80)	59 (51–64)	72 (58–81)	<.001
Men	62.9% (273)	58.9% (86)	64.9% (187)	.219
BMI	26.6 (23.7–31.1)	26.2 (22.9–30.1)	26.0 (22.7–30.7)	.680
Ethnicity: Caucasian	42.9% (186)	34.2% (50)	47.2% (136)	.002
Asian	37.1% (161)	48.6% (71)	31.3% (90)	
Afro-Caribbean	20.0% (87)	17.1% (25)	21.5% (62)	
Hypertension	48.6% (211)	31.5% (46)	57.3% (165)	<.001
Type2 DM	33.9% (147)	25.7% (37)	38.3% (110)	.007
Dyslipidaemia	38.7% (168)	24.0% (35)	46.2% (133)	<.001
Smokers	3.2% (14)	4.1% (6)	2.8% (8)	.415
Ex-smokers	20.3% (88)	17.1% (25)	21.9% (63)	
IHD	14.1% (61)	6.2% (9)	18.2% (52)	.001
Moderate CKD	33.6% (146)	6.2% (9)	47.4% (137)	<.001
Asthma	11.8% (51)	16.4% (24)	9.4% (27)	.031
COPD	10.8% (47)	6.2% (9)	13.2% (38)	.026
<i>Chronic medication</i>				
ACE-i	21.4% (93)	11.0% (16)	26.7% (77)	<.001
ARB	12.9% (56)	12.3% (18)	13.2% (38)	.799
Statin	48.0% (208)	29.5% (43)	57.5% (165)	<.001
<i>Disease presentation</i>				
Symptom onset before admission (days)	7 (3–10)	7 (2–9)	4 (1–7)	.003
Symptom at onset				
Cough	27.4% (119)	15.8% (23)	33.3% (96)	<.001
Fever	17.1% (74)	19.9% (29)	15.6% (45)	
Both	36.4% (158)	52.7% (77)	28.1% (81)	
Other	19.1% (83)	11.6% (17)	22.9% (66)	

hsTrop: high-sensitivity Troponin-T on admission; BMI: body-mass index; DM: diabetes mellitus; IHD: ischaemic heart disease; CKD: chronic kidney disease; COPD: chronic obstructive pulmonary disease; ACE-I: angiotensin-converting enzyme inhibitors; ARB: angiotensin receptor blockers. Bold values are statistically significant at  $p < .05$ .

**Table 2.** Laboratory test results on admission and association with myocardial injury.

	All (n = 434)	Negative hsTrop <15 ng/L (n = 146)	hsTrop ≥15 ng/L (n = 288)	p
<i>Laboratory data – admission</i>				
WBC ( $10^9/L$ )	7.4 (5.4–10.5)	6.3 (4.8–8.2)	8.4 (5.8–11.3)	<.001
Lymphocytes ( $10^9/L$ )	0.9 (0.7–1.3)	0.9 (0.7–1.2)	0.9 (0.6–1.3)	.404
Haemoglobin (g/L)	12.7 (11.2–13.9)	13.0 (12.2–14.4)	12.2 (10.7–13.8)	<.001
Platelets ( $10^9/L$ )	217 (166–285)	214 (177–265)	217 (167–304)	.785
CRP (mg/L)	97 (43–181)	102 (46–185)	111 (62–194)	.834
Creatinine (umol/L)	91 (72–125)	77 (67–92)	102 (78–151)	<.001
eGFR mL/min MDRD	72 (50–93)	87 (74–110)	65 (42–85)	<.001
D-Dimers (mg/mL)	1.18 (0.55–2.49)	0.63 (0.38–1.32)	1.60 (0.85–3.62)	<.001
NTproBNP (pg/mL)	537 (176–2329)	153 (48–330)	1219 (346–3563)	<.001
aPTT (s)	27.0 (24.0–29.0)	26.7 (24.9–28.5)	27.0 (24.0–30.7)	.196
INR (ratio)	1.1 (1.0–1.2)	1.1 (1.0–1.1)	1.1 (1.1–1.2)	<.001
Thrombin time (s)	16 (15–18)	15 (14–16)	18 (15–20)	.002

hsTrop: high-sensitivity Troponin-T; WBC: white blood cell count; CRP: C-reactive protein; eGFR: estimated glomerular filtration rate; aPTT: activated partial thromboplastin time; INR: international normalised ratio. Bold values are statistically significant at  $p < .05$ .

patients with at least one positive troponin result during admission (i.e. a positive peak troponin level) were more likely to need both non-invasive and mechanical ventilation (2.40 and 6.81 significantly higher odds, respectively) and significantly more likely to require urgent renal replacement therapy – RRT (4.14 higher odds of RRT) (Table 4).

With regards to events, after adjustment to the previously mentioned confounders, positive troponin during admission was significantly associated with acute

kidney injury (6.76 higher odds), thromboembolic events (11.99 higher odds) and presence of atrial fibrillation (10.66 higher odds). Importantly, raised troponin levels were significantly and independently associated with death during admission (2.40 higher likelihood of dying when a positive troponin was detected) (Table 4).

Patients in whom no troponin measurement was performed during admission had comparable hospitalisation length and mortality rate when compared to

**Table 3.** Predictors of myocardial injury (positive troponin).

Variables	Univariate		Multivariate	
	OR	<i>p</i>	OR	<i>p</i>
	95% CI		95% CI	
Age (per 10 years)	1.80	<b>&lt;.001</b>	1.68	<b>&lt;.001</b>
	1.62–1.99		1.49–1.88	
Men	1.29	0.220	–	–
	0.86–1.94			
BMI	1.00	.538	–	–
	0.99–1.01			
<i>Ethnicity</i>				
Asians vs. Caucasians	0.47	<b>.001</b>	–	–
	0.30–0.73			
Afro-Caribbean vs. Caucasians	0.91	.749	–	–
	0.52–1.61			
Hypertension	2.92	<b>&lt;.001</b>	1.81	<b>.020</b>
	1.92–4.44		1.10–2.99	
Type2 DM	1.83	<b>.007</b>	–	–
	1.18–2.85			
Dyslipidaemia	2.72	<b>&lt;.001</b>	–	–
	1.74–4.25			
Smokers	0.71	.529	–	–
	0.24–2.09			
Ex-smokers	1.34	.271	–	–
	0.80–2.24			
IHD	3.38	<b>.001</b>	–	–
	1.62–7.08			
Asthma	0.56	<b>.033</b>	–	–
	0.29–0.95			
COPD	2.31	<b>.030</b>	–	–
	1.09–4.93			
Moderate CKD (eGFR < 60 ml/min)	13.81	<b>&lt;.001</b>	9.12	<b>&lt;.001</b>
	6.77–28.18		4.24–19.64	

BMI: body-mass index; IHD: ischaemic heart disease; DM: diabetes mellitus; COPD: chronic obstructive pulmonary disease; CKD: chronic kidney disease; eGFR: estimated glomerular filtration rate. Note: Method forward LR: probability for stepwise .05.

Bold values are statistically significant at  $p < .05$ .

patients with measured troponin ( $14.6 \pm 11.5$  vs.  $15.3 \pm 12.9$  days,  $p = .475$  &  $32.3\%$  vs.  $33.0\%$ ,  $p = .856$ , respectively).

### Peak troponin levels and admission troponin vs. outcomes

Median length of in-hospital stay (in days) was longer for patients with positive peak troponin levels: 8 (IQR 5–13) for negative troponin, 14 (IQR 7–23) for low-positive, and 16 (IQR 10–23) for high-positive peak troponin ( $p < .001$  vs. negative troponin, but  $p = NS$  when comparing low and high-positive peak troponin groups).

All the significant associations previously reported for myocardial injury (observed anytime throughout the admission) were also observed for admission troponin levels, except for non-invasive ventilation (Supplementary Table 1 and Figure 2).

Cardiovascular complication rates were comparable in the low and high-positive troponin groups. However, need for invasive ventilation, and acute

kidney injury were significantly more frequent in the high-positive troponin group (Supplementary Table 2).

### Discussion

In the present study, we report our experience on the association between troponin levels and COVID-19 outcomes. We have found that nearly two-thirds of admitted patients are troponin positive. These patients have a longer in-hospital stay, which appears to be associated with magnitude of troponin rise. Troponin positive patients were more likely to need both non-invasive and mechanical ventilation as well as urgent RRT. Raised troponin levels were associated with acute kidney injury, thromboembolic events and presence of atrial fibrillation.

Higher levels of troponin were associated with further increase in the risk of acute kidney injury and need for mechanical ventilation. Admission troponin levels also had good predictive value, being associated with most of the assessed cardiovascular and non-cardiovascular outcomes. Importantly, positive troponin levels were significantly associated with death during admission.

Early experience from China, Italy and USA suggests that COVID-19 can be a mild condition in most individuals [6–8]. People who will more often require intensive care unit admission are the elderly and those with several other comorbidities including CVD.

In a cohort of 416 positive patients, Shi et al. [3] reported that 86 patients had evidence of myocardial damage as indicated by increased in troponin levels. Those patients with higher troponin levels had also increased in-hospital mortality. Similar results were reported by Guo et al. [4], with highest mortality rates in those with elevated troponin levels and underlying CVD. Interestingly, our cohort displayed a threefold higher frequency of positive troponin levels in COVID-19 patients admitted to hospital (60% as compared to 20% in previous populations) from the region of Wuhan.

In the aforementioned study by Guo et al. [4], troponin levels were associated with CRP and NT-proBNP levels correlating myocardial injury and ventricular dysfunction. We did not observe the same association for CRP levels, but higher white blood cell count, D-Dimers and lower haemoglobin levels suggested that positive troponin can occur as a result of an inflammatory process. Importantly, we found that NT-proBNP levels were significantly higher in the troponin positive patients, who also had higher in-hospital mortality. This is in agreement with previous

**Table 4.** Association of positive peak troponin with outcomes.

	All (n = 434)	Negative hsTrop <15 ng/L (n = 146)	hsTrop ≥15 ng/L (n = 288)	Adjusted OR 95% CI	p
<i>Procedures</i>					
Non-invasive ventilation	18.4% (80)	15.8% (23)	19.8% (57)	2.40 1.27–4.56	<b>.007</b>
Mechanical ventilation	22.8% (99)	13.0% (19)	27.8% (80)	6.81 3.40–13.62	<b>&lt;.001</b>
ECMO	0.9% (4)	0.7% (1)	1.0% (3)	5.06 0.32–79.56	.323
Urgent RRT	8.3% (35)	3.4% (5)	10.8% (30)	4.14 1.34–12.78	<b>.013</b>
Cardiac pacing	0.5% (2)	0% (0)	0.7% (2)	N/A	N/A
PCI	0.2% (1)	0% (0)	0.3% (1)	N/A	N/A
<i>Outcomes</i>					
Pneumonia	80.2% (348)	82.2% (120)	79.2% (228)	1.18 0.63–2.21	.597
Acute kidney injury	36.3% (154)	10.3% (15)	50.0% (139)	6.76 3.40–13.47	<b>&lt;.001</b>
Acute HF	7.6% (33)	2.1% (3)	10.4% (30)	2.76 0.73–10.48	.136
Ischaemic stroke	3.7% (16)	2.7% (4)	4.2% (12)	1.31 0.34–5.09	.693
Venous thromboembolic disease	7.4% (32)	2.1% (3)	10.1% (29)	11.99 3.20–44.88	<b>&lt;.001</b>
AF episode	6.9% (30)	0.7% (1)	10.1% (29)	10.66 1.33–85.32	<b>.026</b>
Ventricular tachycardia	0.7% (3)	0% (0)	1.0% (3)	N/A	N/A
Death during admission	33.1% (140)	16.4% (24)	41.9% (116)	2.40 1.34–4.29	<b>.003</b>

PCI: percutaneous coronary intervention; HF: heart failure; hsTrop: high-sensitivity Troponin-T; ECMO: extracorporeal membrane oxygenation; AF: atrial fibrillation.

Adjustment for clinical baseline differences: age, ethnicity, hypertension, diabetes, dyslipidemia, previous MI/ischaemic heart disease, asthma, COPD and eGFR (Method: Enter).

Bold values are statistically significant at  $p < .05$ .

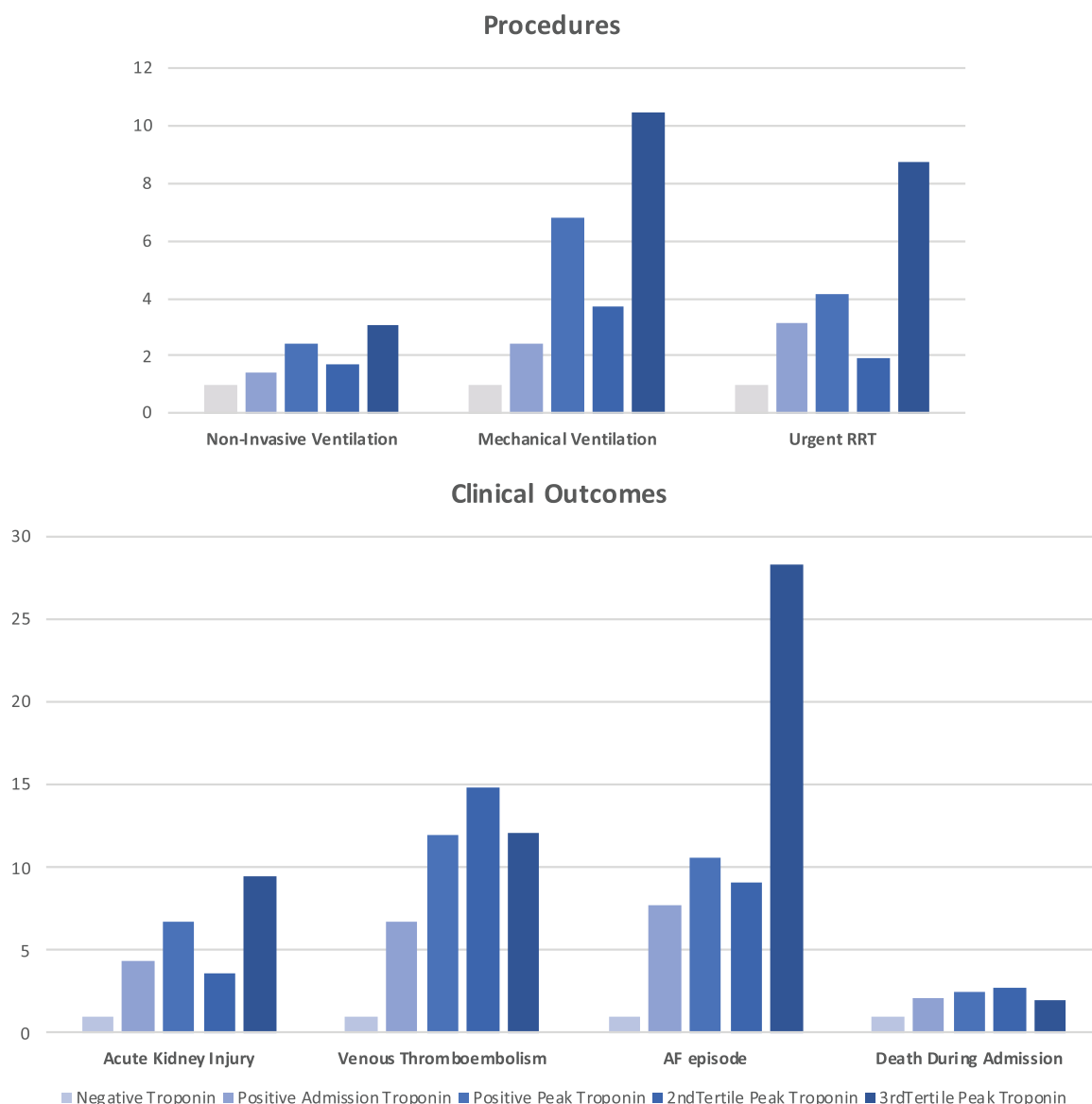
studies suggesting that NT-proBNP might be an independent risk factor for in-hospital death in patients with severe COVID-19 [9]. The observed changes in INR and D-dimers also suggest that positive troponin can translate some of the coagulation changes observed in this group. Finally, the higher creatinine and lower eGFR levels in these patients may be signalling the presence of more frequent micro- and macro-vascular disease. Further research underpinning the association of positive troponin with changes in these pathways is required for further understanding of the pathophysiology of COVID-19 and the associated cytokine storm.

In addition to myocardial injury, arrhythmias are also known to occur in COVID-19. In a study [10] including 138 patients with COVID-19 there was a 16.7% incidence of arrhythmias. This was much higher in patients requiring admission to intensive care compared to those who did not. We did not observe high prevalence of ventricular arrhythmias as described in previous cohorts of COVID-19 positive patients [4,10]. In our cohort, combining AF and ventricular tachycardia, sustained arrhythmias occurred in only 7% of patients, half to a third of what was reported in Wuhan.

We have shown that increased troponin levels occurred in patients with CVD risk factors, as well as in those with COPD and those in angiotensin-converting enzyme inhibitors (ACE-I) and statin therapy. Of the parameters we examined, age, hypertension and moderate CKD were independent predictors of myocardial injury.

It is known that troponin levels can be increased in patients with renal failure due to the high prevalence of coronary artery disease [11]. However, changes of baseline level with a distinct rise and fall supports the presence of myocardial injury. This is illustrated in our findings, in the setting of COVID-19, with the presence of moderate CKD, measured through estimated glomerular filtration rate <60 mL/min, using the MDRD equation, also being independently associated with a ninefold increase in myocardial injury after adjustment (Table 3).

Our results raise several hypotheses. Bed management and hospital capacity is one of the concerns during the COVID-19 pandemic. We wonder if utilising troponin levels (single or repeated measurements), alongside with other clinical and laboratory variables commonly used for assessing if a patient is fit for hospital discharge (time of disease progression,



**Figure 2.** Association (adjusted OR) of the different troponin-derived variables with procedures and clinical events.

acceptable O<sub>2</sub> saturation, and improved respiratory symptoms, improvement of blood markers of infection, etc.) [12], can be used for deciding which patients can be treated in the ambulatory care. Furthermore, the presence of myocardial injury can potentially be a good criterion for inclusion in a clinical decision rule (alongside other risk factors and biomarkers) in selecting patients for aggressive treatment with drugs aiming at reducing viral load or blocking the cytokine storm phase. However, these hypotheses need to be assessed in a randomised trial. Optimal number and frequency of troponin measurements during admission, or in patients receiving ambulatory care, remains to be determined.

We acknowledge that the present study has limitations. The use of cardiac magnetic resonance or

echocardiography were limited due to safety and logistic issues, and hence we were not able to assess for specific features of acute myocardial injury. In addition, we cannot prove a direct effect of COVID-19 on myocardium or involvement of inflammation in the observed outcomes. However, our multi-ethnic cohort is significantly larger and more applicable to other ethnicity groups when compared to the very early Wuhan cohorts which were limited to the Chinese population.

## Conclusions

Patients with CKD, hypertension and more advanced age are more likely to present with myocardial injury during COVID-19 hospitalisation. Admission and peak

troponin appear to be predictors for cardiovascular and non-cardiovascular events and outcomes in COVID-19 patients. Further research is needed on the potential usefulness of troponin for the management and treatment decisions in COVID-19 patients.

## Disclosure statement

The authors report no conflicts of interest.

## ORCID

Catrin Sohrabi  <http://orcid.org/0000-0003-2773-5659>

David Prieto Merino  <http://orcid.org/0000-0001-5001-0061>

Rui Providencia  <http://orcid.org/0000-0001-9141-9883>

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