Observations on echocardiographic findings in patients with COVID-19

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Keywords: coronavirus, COVID-19, cardiac complications, echocardiography, tricuspid regurgitation, pulmonary hypertension

DOI: https://doi.org/10.21203/rs.3.rs-58076/v1

Abstract

Background The novel coronavirus (SARS-CoV-2) has created global havoc by causing Coronavirus Disease 2019 (COVID-19). Cardiovascular involvement in COVID-19 varies from troponin rise or arrhythmia/myocarditis to fulminant cardiogenic shock. There is limited data on echocardiographic findings in such patients. We aimed to assess abnormal echocardiographic findings and contributory factors in patients with COVID-19.

Methods We performed retrospective analysis of COVID-19 positive patients who underwent a transthoracic echocardiogram (TTE) at Sandwell and West Birmingham (SWBH) NHS Trust between March 2020 and May 2020. Patients were compared based on TTE changes and divided into two groups (abnormal TTE and normal TTE).

Results 66 out of 463 patients with COVID-19 had a TTE. 46 patients (69.7%) had abnormal findings on their TTE. Tricuspid regurgitation was the most common abnormality observed (26 (56.5%) patients), followed by aortic regurgitation (13 (28.3%) patients) and mitral regurgitation (12 (26.1%) patients). Haemoglobin and LDH were predictors of abnormal TTE (Hb OR: 0.97, p = 0.049, LDH, OR: 1.00, p = 0.03). Significantly more patients in the abnormal TTE group died during their inpatient stay compared to normal TTE (p = 0.01). Having an abnormal TTE was an independent predictor of death on regression analysis (OR: 0.229, p = 0.034).

Conclusions This is the first detailed observational study looking at echocardiographic changes in admitted COVID-19 patients irrespective of disease severity. The most common abnormality was valve regurgitation. Patients with abnormal TTE were more likely to die in hospital.

Introduction

Viral pneumonias have recently emerged as a great threat to global public health (1). Most recently, the novel severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), which is known to cause severe pneumonia has reaped global havoc affecting even the most substantial health care systems in the world. As with most viral pneumonias, a strong relationship between cardiovascular disease and COVID-19 has been noted (1). More specifically, COVID-19 caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), a single-stranded RNA virus, has its deleterious effect by binding to the human angiotensin-converting enzyme 2 (ACE2) receptor expressed in alveolar cells, vascular endothelium, intestinal epithelial cells, kidneys and the heart (2–4). This possibly explains the multi-organ involvement/injury seen with COVID-19 infection. ACE2 counteracts the effects of the renin-angiotensin system, which is hyperactivated in states such as hypertension and heart failure (5). Furthermore, high levels of ACE2 have been noted in patients with diabetes and/or hypertension treated with ACE inhibitors (6, 7). Published data demonstrates that between 12 to 22% of COVID-19 patients admitted to hospital have diabetes and up to 30% have hypertension (8–10).

It has been previously well characterised that myocardial injury (demonstrated by elevated high-sensitivity cardiac troponin I) is seen in patients with COVID-19 infection (5). In fact, cardiac injury was observed to confer a higher risk of mortality for such inpatients (11). Although, the mechanistic awareness of how SARS-CoV-2 causes myocardial damage is still unknown; hypoxaemia induced pulmonary hypertension, systemic inflammation resulting in increased afterload, worsening cardiac function and direct invasion of the virus into the myocardium resulting in myocarditis are possible mechanisms of injury (1, 12).

There is at present insufficient data to determine the cause or consequence of cardiac injury. Some have stipulated various consequences and molecular mechanisms, nothing of which is substantial or causal (5, 12). Although many studies have examined the potential of troponin I as a marker of severity, there are very few studies, to our knowledge, that have examined the echocardiography findings in COVID-19 patients (13–15). Complicating this is the difficulty of performing echocardiography at bedside whilst using personal protective equipment and the risk of infection to staff from enhanced exposure to this virus. We aimed to assess abnormal echocardiographic findings and contributory factors in patients with COVID-19.

Methods

The data underlying this article are available in Harvard Dataverse, at https://doi.org/10.7910/DVN/FTBAH6 (16).

We carried out a retrospective, observational study of COVID-19 positive patients admitted to Sandwell and West Birmingham (SWBH) Hospitals NHS Trust from March 2020 to May 2020 who underwent a transthoracic echocardiogram (TTE). Patients included were either admitted with symptoms of COVID-19 infection or had a positive swab result whilst already an in-patient with an alternative diagnosis. The TTE was requested by an experienced doctor based on clinical symptoms and signs. The study was approved by the local audit department (reference number: 1176) and followed the principles of the Declaration of Helsinki. The study also adhered to “Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement: guidelines for reporting observational studies” as well as the “Transparent reporting of a multivariable prediction model for individual prognosis or diagnosis (TRIPOD)” guidelines (17–19).

Data was extracted from the directorate’s daily COVID-19 report database by members of the Cardiology Department. The following patients were excluded from the analysis: age < 18 years old, outpatients who were COVID-19 positive (in the community), patients admitted to other hospitals, and patients who did not have an echocardiogram. All TTE scans were performed by experienced, British Society of Echocardiography (BSE) accredited sonographers following consent from the patients where possible. Abnormal echocardiogram was defined as any one of the following:

- impaired left ventricular or right ventricular function

- atrial dilatation
- valvular impairment (any regurgitation or stenosis)
- presence of pericardial effusion

Patients were divided into two groups, those with an abnormal echocardiogram and those with a normal echocardiogram. In addition to the echocardiographic data, information on baseline clinical characteristics including age, sex, ethnicity, height, weight, body mass index (BMI), co-morbidities and smoking status was also collected. Biomarkers associated with poor prognosis in COVID-19 patients were also included in the analysis, namely full blood count (FBC) including haemoglobin (Hb), estimated Glomerular Filtrate Rate (eGFR), troponin I, D-Dimer, Lactate Dehydrogenase (LDH), ferritin, magnesium and procalcitonin. Patients' 12 lead ECG on admission was also reviewed. Information on patients' outcome including death was also collected.

Statistical Analysis

Descriptive statistics are presented as mean ± standard deviation (SD) or median with interquartile range, as appropriate for continuous variables. Categorical variables are expressed as numbers and percentages. Statistical analysis was performed using SPSS software, version 26 (SPSS Inc., Chicago, Illinois). Continuous variables were tested for normality using the Shapiro-Wilk test. Non-normally distributed data were logarithmically transformed and the distribution re-checked with a Shapiro-Wilk test. If passed, data was analyzed using an independent Student's t-test. Data that were still not normally distributed were analyzed with the Mann-Whitney U test using original non-log transformed data. A p value of < 0.05 was considered statistically significant.

Univariate analysis compared independent variables between patients with abnormal and normal echocardiograms. Significant variables identified on univariate analysis were then included in binary regression analysis to identify predictors of abnormal echocardiography and the model was assessed by c-statistic. Univariate analysis was also performed to identify significant variables between survivors and non-survivors. Significant variables were then included in a further binary regression analysis to identify predictors on in-hospital death.

Results

463 patients in our trust between March and May 2020 with COVID-19 were screened of whom 66 had a TTE. 32 of these patients were critically unwell of which 25 were invasively ventilated (2 requiring tracheostomies) at the time of TTE. 46 patients (69.7%) had abnormal findings on their TTE. Patients in the two groups, abnormal TTE and normal TTE were well matched for age, sex, ethnicity and clinical characteristics (Table 1). Shortness of breath, cough and fever were the most common presenting symptoms in our patients (Table 1).

Table 1
– Demographics and clinical characteristics

Variable	Abnormal TTE (n = 46)	Normal TTE (n = 20)	Significance p value
Age (mean ± SD)	63 ± 14	60 ± 18	0.38
Sex	26 (56.5%)	12 (60%)	0.79
Male, n (%)	20 (43.5%)	8 (40%)	
Female, n (%)			
Ethnicity	18 (39.1%)	9 (45%)	-
Caucasians, n (%)	12 (26.1%)	6 (30%)	
Asians, n (%)	2 (4.3%)	2 (10%)	
Blacks, n (%)	1 (2.2%)	0 (0%)	
Mixed, n (%)	13 (28.3%)	3 (15%)	
Unknown, n (%)			
Clinical characteristics			
Height (m)	1.67 ± 0.10	1.68 ± 0.09	0.75
Weight (kg)	79.2 ± 1.2	84.3 ± 1.3	0.33
BMI (kg/m^2)	29.05 ± 5.97	31.49 ± 7.60	0.21
Hypertension, n (%)	21 (45.7%)	12 (60%)	0.28
Diabetes Mellitus, n (%)	18 (39.1%)	7 (35%)	0.75
COPD, n (%)	6 (13%)	2 (10%)	1.00
Smoker, n (%)	10 (21.7%)	2 (10%)	0.71
AF, n (%)	6 (13.0%)	0 (0%)	0.17
Hypercholesterolemia, n (%)	8 (17.4%)	4 (20%)	1.00

Descriptive data are presented as numbers (with percentages). Normally distributed data are expressed as mean ± standard deviation (SD). Non-normally distributed data are displayed as median with interquartile ranges. Normality test was performed using Shapiro-Wilk test. Statistical differences were tested using an independent t-test for normally distributed data and Mann-Whitney U test for non-normally distributed data. Categorical data was compared using Chi-square test. Where Chi-square test was not valid, Fisher's Exact Test was used. Significance $p \leq 0.05$. – = unable to calculate p value as sample size too small/statistical test not valid. AF = atrial fibrillation; AV = atrioventricular; BMI = Body Mass Index; bpm = beats per minute; CABG = Coronary Artery Bypass Graft; COPD = Chronic Obstructive Pulmonary Disease; eGFR = estimated Glomerular Filtration Rate; IHD = Ischaemic Heart Disease; LDH = Lactate Dehydrogenase; MI = Myocardial Infarction; PCI = Primary Coronary Intervention; PVD = Peripheral vascular disease; TTE = Transthoracic echocardiogram

Variable	Abnormal TTE (n = 46)	Normal TTE (n = 20)	Significance p value
Previous IHD, n (%)	5 (10.9%)	2 (10%)	1.00
Previous MI, n (%)	1 (2.2%)	1 (5%)	0.52
Previous PCI, n (%)	1 (2.2%)	1 (5%)	0.52
Previous CABG, n (%)	0 (0%)	0 (0%)	-
PVD, n (%)	3 (6.5%)	1 (5%)	1.00
Haemoglobin (g/L)	115 ± 24	129 ± 26	**0.03**
White cell count (10*9/L)	10.5 ± 1.6	9.4 ± 1.6	0.42
Lymphocytes (10*9/L)	1.1 ± 1.9	1.2 ± 1.7	0.45
eGFR (ml/min/1.73 m^2)	60 [40–83]	74 [49–90]	0.22
Hs Troponin I (ng/L)	35 [10–203]	16 [8–66]	0.37
Highest Hs Troponin I (ng/L)	313 ± 7	640 ± 19	0.66
D-Dimer (ugFEU/ml)	8.3 ± 6	2.5 ± 3	0.21
LDH (U/L)	544 ± 1	359 ± 2	**0.004**
Ferritin (µg/L)	751 ± 3	618 ± 3	0.60
Magnesium (mmol/L)	0.86 ± 1.21	0.86 ± 1.30	1.00
Procalcitonin	0.89 ± 3.96	0.58 ± 4.59	0.54

Descriptive data are presented as numbers (with percentages). Normally distributed data are expressed as mean ± standard deviation (SD). Non-normally distributed data are displayed as median with interquartile ranges. Normality test was performed using Shapiro-Wilk test. Statistical differences were tested using an independent t-test for normally distributed data and Mann-Whitney U test for non-normally distributed data. Categorical data was compared using Chi-square test. Where Chi-square test was not valid, Fisher's Exact Test was used. Significance $p \leq 0.05$. – = unable to calculate p value as sample size too small/statistical test not valid. AF = atrial fibrillation; AV = atrioventricular; BMI = Body Mass Index; bpm = beats per minute; CABG = Coronary Artery Bypass Graft; COPD = Chronic Obstructive Pulmonary Disease; eGFR = estimated Glomerular Filtration Rate; IHD = Ischaemic Heart Disease; LDH = Lactate Dehydrogenase; MI = Myocardial Infarction; PCI = Primary Coronary Intervention; PVD = Peripheral vascular disease; TTE = Transthoracic echocardiogram

Variable	Abnormal TTE (n = 46)	Normal TTE (n = 20)	Significance p value
ECG Rhythm	20 (43.5%)	10 (50%)	-
Normal sinus rhythm	4 (8.7%)	0 (0%)	
AF/Atrial flutter	11 (23.9%)	9 (45%)	
Sinus tachycardia	4 (8.7%)	0 (0%)	
Bundle Branch Block	2 (4.3%)	0 (0%)	
AV block	2 (4.3%)	0 (0%)	
ST elevation	2 (4.3%)	1 (5%)	
ST depression	9 (19.6%)	5 (25%)	
T-wave inversion			
ECG Rate (bpm)	95 ± 19	98 ± 17	0.61
Critically ill	25 (54%)	7 (35%)	0.15
On ventilator	20 (43%)	5 (25%)	0.16
Outcome			
Death	20 (43.5%)	2 (10%)	**0.01**

Descriptive data are presented as numbers (with percentages). Normally distributed data are expressed as mean ± standard deviation (SD). Non-normally distributed data are displayed as median with interquartile ranges. Normality test was performed using Shapiro-Wilk test. Statistical differences were tested using an independent t-test for normally distributed data and Mann-Whitney U test for non-normally distributed data. Categorical data was compared using Chi-square test. Where Chi-square test was not valid, Fisher's Exact Test was used. Significance $p \leq 0.05$. – = unable to calculate p value as sample size too small/statistical test not valid. AF = atrial fibrillation; AV = atrioventricular; BMI = Body Mass Index; bpm = beats per minute; CABG = Coronary Artery Bypass Graft; COPD = Chronic Obstructive Pulmonary Disease; eGFR = estimated Glomerular Filtration Rate; IHD = Ischaemic Heart Disease; LDH = Lactate Dehydrogenase; MI = Myocardial Infarction; PCI = Primary Coronary Intervention; PVD = Peripheral vascular disease; TTE = Transthoracic echocardiogram

The echocardiogram findings of patients in the abnormal TTE group are detailed in Table 2. Majority of these patients had evidence of valvular heart disease but no other pathology. Tricuspid regurgitation (TR) was the most common abnormality, seen in 26 (56.5%) patients, followed by aortic regurgitation (13 (28.3%) patients) and mitral regurgitation (12 (26.1%) patients). Figure 1 describes the severity of each valvular lesion. 12 patients (26.1%) had impaired right ventricle (RV) systolic dysfunction and 14 patients (30.4%) had any atrial dilatation (Table 2). The mean pulmonary artery systolic pressure (PASP) was found to be 47 ± 12 mmHg with the majority of patients having mild pulmonary hypertension. 22 out of 66 patients had previous TTE of whom 11 had worse changes on their recent TTE.

Table 2
– Echocardiogram findings in abnormal TTE group

Variable	Parameters	Number of patients
LV systolic function	Normal	34
	Mildly impaired	2
	Moderately impaired	2
	Severely impaired	4
	Hyperdynamic	4
LV Regional Wall Motion Abnormalities (RWMAs)	Present	4
	Absent	42
RV systolic function	Normal	34
	Impaired	12
Atrial size	Normal	32
	Dilated LA	4
	Dilated RA	4
	Bilateral dilatation	6
Valvular disease	Present	37
	Absent	9
Pericardial effusion	Present	4
	Absent	42
Pulmonary Artery Systolic Pressure (PASP)	No TR so unable to measure	42
	Mean PASP	47 ± 12
Pulmonary hypertension	None (no TR)	41
	Incomplete TR jet	1
	< 35 mmHg (none)	3
	35–49 mmHg (mild)	12
	50–59 mmHg (moderate)	5
	> 60 mmHg (severe)	4

Patients in the abnormal TTE group had significantly lower mean Hb compared to the normal TTE group (115 ± 24 g/L vs 129 ± 26 g/L, p = 0.03). There were no significant differences in white cell count, lymphocytes, renal function, admission and highest troponins between the two groups. Mean LDH was noted to be significantly higher in the abnormal TTE group compared to normal TTE group (544 ± 1 U/L vs 359 ± 2 U/L, p = 0.004). D-Dimer, ferritin, magnesium and procalcitonin levels were similar between the two groups. Low Hb and raised LDH were predictors of abnormal TTE (Hb OR: 0.97, p = 0.049, LDH, OR: 1.00, p = 0.03). The c-statistic for this model was 0.819 (95% confidence interval (CI): 0.681–0.957, p = 0.001, Fig. 2).

Most patients in both groups were in normal sinus rhythm on their 12 lead electrocardiograms (ECGs) on admission. 4 patients (8.7%) in the abnormal TTE group were found to be in atrial fibrillation/flutter compared to 0 patients in the normal TTE group. Similarly, there were 4 patients with bundle branch block, 2 with first degree AV block, 2 with ST elevation in abnormal TTE group compared to 0 in the normal TTE group. There were no significant differences in the mean heart rate on the 12 lead ECG between the two groups.

Significantly more patients in the abnormal TTE group died during their hospital admission compared to normal TTE (20 patients (43.5%) abnormal TTE and 3 patients (15%) normal TTE, p = 0.028). Having an abnormal TTE and elevated PASP on echocardiogram were found to be independent predictors of death on univariate analysis (TTE p = 0.028 and PASP p = 0.022) but on binomial logistic regression analysis only abnormal TTE was found to be a significant predictor (OR: 0.229, p = 0.034).

Discussion

We present one of the first detailed analyses of echocardiographic changes in patients with COVID-19. Valvular regurgitation was the commonest abnormality observed. Patients with an abnormal TTE were more likely to die in hospital.

Our data is consistent with many previous studies suggesting that patients with abnormal TTE were more likely to die in hospital (13, 14, 20–22). Peng *et al*. have shown that abnormal echocardiography features were linked to the severity of disease and consequent cardiovascular sequelae (23).

Ours is one of the first studies to assess echocardiographic findings in admitted COVID-19 patients, irrespective of severity and its relationship to several biomarkers. Previous studies have detailed cardiac injury associated with COVID-19 based on troponin levels, ECG findings and echocardiogram features (13–15). The aetiology of cardiac dysfunction as it relates to abnormal TTE is likely to be multifactorial as shown in our study with no relation between troponin and abnormal TTE. Although direct invasion of the virus into the myocardium is one reason; it is likely that in the majority of the cases, myocardial infarction (Type II mainly) due to reduced oxygen perfusion and respiratory failure, microangiopathy secondary to cytokine storm and stress cardiomyopathy are potential causes (8, 24–30).

Participants in our groups were well-matched for age, sex and comorbidities. The commonest echocardiogram abnormality noted in our study was TR, similar to findings from another study (31), and under one third of our patients demonstrated RV dysfunction. The latter could be secondary to the acute respiratory distress syndrome seen in critically ill COVID-19 patients, which results in RV systolic dysfunction (8, 32, 33). Despite not being overly sensitive for a pulmonary embolism (PE), echocardiographic features of right heart strain and right heart dysfunction can indicate the presence of a pulmonary embolus (PE) or Pulmonary Intravascular Coagulation (PIC) which has been hypothesised to be one of the causative mechanisms leading to respiratory distress in COVID-19 patients (34).

Although RV dysfunction relates to poor outcomes, RV longitudinal strain is more highly predictive of mortality in COVID-19 patients (32, 35, 36). Despite the advantages of RV longitudinal strain, time constraints and increased risk of exposure to the SARS-CoV-2 virus for the sonographer limits its usage in the COVID-19 positive patients. Patients with disproportionately enlarged RV and reduced left ventricle (LV) systolic function need to be monitored closely with early goal-directed therapies as this has been shown to guide management of the critically ill (23, 37–39).

Our data also showed that elevated PASP on echocardiogram was an independent predictor of death. Our study corroborates with other literature which suggests that elevated PASP was related to morbidity (32). A case series also noted elevated PASP in their patients, which could be explained by pulmonary hypertension or recurrent pulmonary-embolic disease (37, 40). High PASP relates to pulmonary hypertension, whether underlying or as a consequent result of SARS-CoV-2-related lung injury, pulmonary hypercoagulable state or cardiomyopathy, confers significant mortality and morbidity (41, 42).

Anecdotal evidence has demonstrated that elevation of PASP and then a sudden depreciation in COVID-19 fulminant myocarditis was likely linked to right heart functional decline secondary to sustained overload (43). In concordance with other studies, elevated PASP and right heart enlargement was significantly predictive of mortality (31). In fact, taking into consideration the crucial role of right heart function, the German Society of Cardiology concluded that optimum RV functioning was essential for COVID-19 patients' prognosis (44).

We also found that LDH was significantly raised in COVID-19 patients with an abnormal TTE. It is well known that raised LDH, although non-specific, correlates well with cardiac dysfunction (45, 46). Furthermore, we noted that mean haemoglobin in patients with abnormal TTE was significantly lower than in patients with normal TTE and predicted abnormal TTE. There appears to be no data that shows a relationship between SARS-CoV-2 and anaemia. However, one can hypothesise that as anaemia results in reduced oxygen perfusion, the consequent compensatory cardiovascular response can induce diastolic dysfunction and left ventricular remodelling observed on TTE.

Strengths and limitations

Our study does have several limitations. Our study is a retrospective observational study over a limited period of time from a single centre and with modest sample sizes. The latter raises the potential for a

type II error, which may contribute to the small number of independent associations seen in our analysis. Also, we included patients who had already been hospitalised prior to their diagnosis of COVID-19 and therefore this may have been a confounding factor as their underlying primary diagnosis could have influenced their echocardiographic changes. Furthermore, the decision to perform the echocardiogram was likely due to specific symptoms or concerns and thus, we cannot completely exclude the possibility of selection bias. A few of the patients tested were critically unwell, and some requiring ventilator support at the time of the TTE; this could have influenced the TTE findings. Therefore, we cannot conclude that abnormal TTE findings were directly attributed to COVID-19, even though we did not find any significant differences between the two groups. For definite conclusions, a larger cohort or multicentre analysis is required. We suggest that biomarkers such as Hb and LDH can be used as a screening tool in COVID-19 patients to perform an echocardiogram.

Conclusions

Patients with COVID-19 do have abnormal findings on TTE with valvular regurgitation being the most common. Patients with abnormal TTE and elevated PASP were more likely to die in hospital and such patients require close surveillance.

Abbreviations

ACE2
angiotensin-converting enzyme 2
SARS-CoV-2
severe acute respiratory syndrome (SARS) coronavirus
TTE
transthoracic echocardiogram
BMI
body mass index
Hb
Haemoglobin
LDH
lactate dehydrogenase
PASP
pulmonary artery systolic pressure
TR
tricuspid regurgitation
RV/LV
right ventricle/left ventricle

Declarations

Funding/Support and Role of Sponsor

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Disclosures

No conflicts of interest, financial or otherwise, are declared by the authors.

Author contributions

A.A.K., S.A. and V.S. conceived and designed research; C.V. provided a list of patients and gained audit approval; M.Y., L.W., N.H., M.R., A.A.K. and V.S. collected data; A.A.K. analyzed data; A.A.K., S.J. and V.S. interpreted results of experiments; A.A.K, S.J. and V.S. drafted manuscript; A.A.K., S.J., N.H. and V.S. edited and revised manuscript; A.A.K., S.J., M.Y., L.W., N.H., M.R., C.V., S.A., and V.S. approved final version of manuscript.

Ethical Approval and Consent to participate

Approved by local audit department (see methodology)

Consent for publication

Not applicable

Availability of data and materials

The data underlying this article are available in Harvard Dataverse, at https://doi.org/10.7910/DVN/FTBAH6 (16).

Competing interests/funding

No conflicts of interest, financial or otherwise, are declared by the authors.

Acknowledgements

Not applicable

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Figures

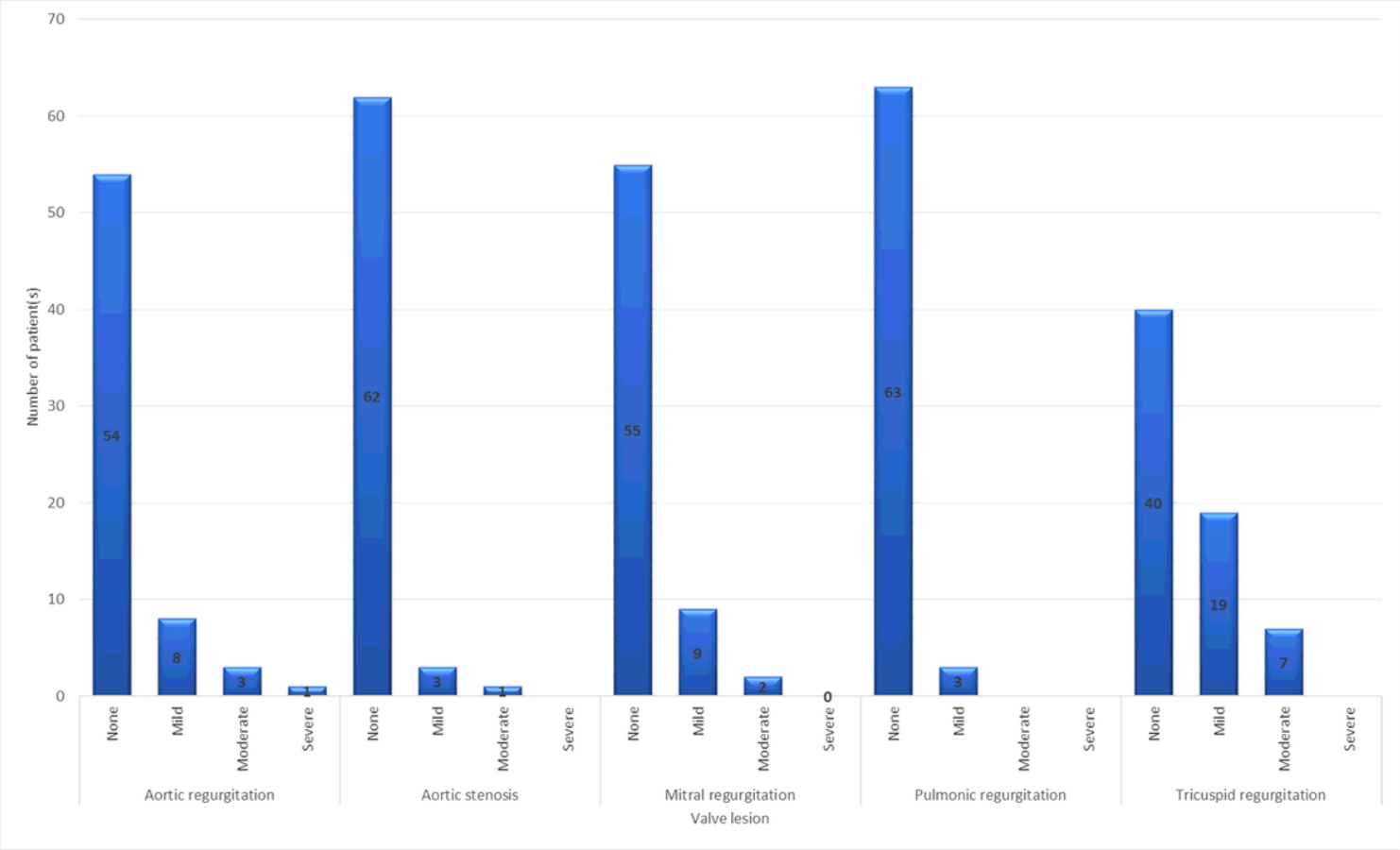

Figure 1

Details of valvular abnormalities noted on echocardiogram performed on patients with COVID-19 at our trust. Valve abnormalities divided between stenosis or regurgitation and graded as none, mild, moderate and severe. Only valve lesions found on the echocardiogram are presented. For example, if mitral stenosis was not seen on any of patients, it is not presented in the figure.

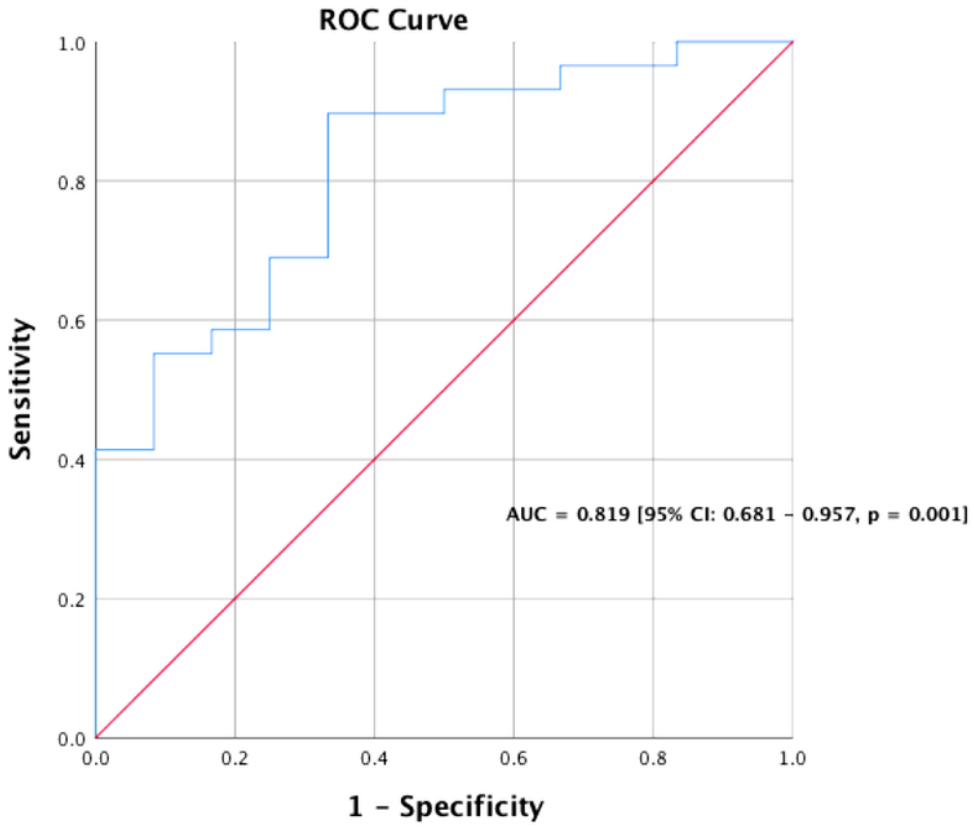

Figure 2 - ROC curve for abnormal TTE

Figure 2

Receiver Operating Characteristic (ROC) curve for abnormal echocardiogram model with haemoglobin (Hb) and lactate dehydrogenase (LDH) as predictors. Produced using SPSS software, version 26 (SPSS Inc., Chicago, Illinois)