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CLINICAL & BASIC RESEARCH

Characteristics and Outcomes of Critically Ill Patients with Raised Cardiac Troponins Admitted in the Intensive Care Unit

A single centre experience from Oman

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abstract: ***Objectives:*** Critically ill patients have raised troponins. This study aimed to assess the incidence of myocardial injury in the intensive care unit (ICU) at a tertiary care hospital and assess the management and prognosis. ***Methods:*** This retrospective study included adult patients who were admitted to the ICU of Sultan Qaboos University Hospital, Muscat, Oman, between January and December 2019 and had undergone a high-sensitive cardiac troponin (hs-cTn) assay. Patients admitted with a primary diagnosis of myocardial infarction were excluded. ***Results:*** A total of 264 patients had their hs-cTn measured during the study period. Of these, 128 (64.3 ± 17.1 years; 58.6% male) had elevated levels, giving an incidence rate of approximately 48.5%. Those with raised troponin were older and had more co-morbidities. These patients were also more critically ill with lower blood pressure, higher heart rates and increased hypotensive episodes. Of these, 47 were treated for acute coronary syndrome, 32 underwent coronary angiography and only three required stenting. Patients with raised troponin had a poor outcome with only 45 (35.2%) surviving to discharge compared to 101 (74.3%) with normal troponin. Patients with raised troponin had shorter hospital stays than those with normal troponin (16 versus 19 days; P = 0.017). ***Conclusion:*** A high proportion of critically ill patients showed evidence of myocardial injury without significant coronary artery disease, which is associated with a poor prognosis. Further prospective studies are required to ascertain the best course of treatment for these patients.

Keywords: Troponin; Biomarkers; Intensive Care; Myocardial Infarction; Oman

Advances in Knowledge

- *Results show that the incidence of type 2 myocardial infarction (MI) in critically ill patients is relatively high and that of coronary artery disease is low.*
- *This is the first study of its kind from Oman and describes the incidence of type 2 MI in the intensive care setting at a single tertiary care centre in Oman.*

Application to Patient Care

- *The study shows that the probability of occurrence of type 1 MI in critically ill patients is low.*
- *These patients can be managed conservatively without the need for invasive tests until they are stable enough for further cardiac investigations.*

The use of high-sensitive cardiac troponin (hs-cTn) assays has made it possible to identify previously unrecognised patients with myocardial injury in a variety of clinical settings.[1,2] The heart, like any other organ, can be affected by systemic illnesses and although hs-cTn is highly specific to cardiac muscle, it does not differentiate between the aetiologically diverse types of myocardial infarction (MI) or non-MI-related myocardial injury.[2,3] Therefore, the Fourth Universal Definition of MI includes the conditions of myocardial injury without infarction and states criteria to help differentiate between infarction and injury.[4] Indeed, in the definition, five subtypes have been introduced to account for the various pathophysiological processes involved.

Type 1 MI is the subtype commonly referred to as a 'heart attack' or an atherothrombotic MI due to plaque rupture. Conversely, type 2 MI is associated with a demand–supply mismatch and is commonly found in critically ill patients admitted into the intensive care unit (ICU) and in patients with other severe systemic illnesses. The other types of MI are those occurring in special circumstances, such as after an angioplasty, a coronary artery bypass grafting or a cardiac arrest.

In critically ill patients, especially those who have had multi-organ failure and are hypotensive or hypoxic, the myocardium, along with other organs, is affected by the associated decreased perfusion. This leads to a demand–supply mismatch, which is exacerbated in the presence of coronary atheroma.[5]

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In milder forms, there could be a hypoxic myocardial injury, but in severe cases, the decreased perfusion could lead to muscle necrosis and infarction even in the absence of plaque rupture, ultimately leading to type 2 MI.

Previous studies have demonstrated a poorer prognosis in critically ill patients with raised troponin, either with or without features of type 2 MI.[6,7] There are no clear guidelines on the management of the conditions of these patients; the management often varies from institution to institution and, indeed, sometimes even within an institution. This study aimed to assess the clinical features, treatment and prognosis of critically ill patients admitted to the ICU with non-cardiac issues and raised troponin levels. In addition, this study sought to assess the angiographic results of these patients to evaluate the true incidence of coronary artery disease (CAD) in this patient population.

Methods

This retrospective study was conducted at the Sultan Qaboos University Hospital, Muscat, Oman, from January to December 2019 and included patients who were admitted to the ICU within the one-year study period. All patients above the age of 18 years at the time of admission to the ICU during this period were eligible for inclusion. Those who were admitted primarily with an MI (either with or without ST-segment elevation on their electrocardiogram [ECG]) and those who did not have a troponin test done during their stay in the ICU were excluded. Electronic case records of eligible patients were reviewed for data retrieval.

According to the Fourth Universal Definition of MI, a patient is diagnosed as having a MI if their troponin levels increase by more than 20% from the baseline value (with at least one value above the 99th percentile) and the patient has chest pain, new ECG changes or new regional wall motion abnormality on echocardiogram.[4] During the current study, if a patient exhibited a significant troponin rise with no other associated feature, a diagnosis of myocardial injury was made.

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS), Version 22.0 (IBM Corp., Chicago, Illinois, USA). All data were presented as number (percentage) and mean ± standard deviation if normally distributed or median (interquartile range [IQR]) if not normally distributed. Analysis was done using Student's t-test, Mann-Whitney U test or Chi-square test, as appropriate. Logistic regression was performed with inpatient death as the outcome variable and the various comorbid onditions, blood investigations and clinical parameters as the input variables. P <0.05 was considered to be statistically significant.

Ethical approval was obtained from the hospital under concern prior to the commencement of the study (SQUH 1461). As this was a retrospective study, patient consent was not required.

Results

A total of 352 patients above the age of 18 were admitted to the ICU at the Sultan Qaboos University Hospital during the study period. Of these patients, 264 had a troponin test done. The results of 128 patients showed raised troponin levels, resulting in a prevalence rate of approximately 48.5%. No gender-based difference was

Table 1: Demographic and clinical characteristics of patients who underwent a high-sensitive cardiac troponin assay and were admitted to the intensive care unit at Sultan Qaboos University Hospital, Muscat, Oman, between January and December 2019 (N = 264)

Demographic characteristic	n (%)		*P* value*
	Troponin not raised (n = 136)	Troponin raised (n = 128)	
Mean age in years ± SD	54.6 ± 19.3	64.3 ± 17.1	<0.001
Gender			0.87
Male	81 (59.6)	75 (58.6)	
Female	55 (40.4)	53 (41.4)	
Clinical characteristic			
Hypertensive	51 (37.5)	83 (64.8)	<0.001
Diabetic	35 (25.7)	55 (43.0)	0.003
Dyslipidaemia	19 (14.0)	37 (28.9)	0.003
Smoker	14 (10.3)	4 (3.1)	0.02
Known IHD	9 (6.6)	22 (17.2)	0.008
Known CKD	36 (26.5)	56 (43.8)	0.003
Mean SBP on admission in mmHg ± SD	123.1 ± 22.6	115.7 ± 28.4	0.014
Mean DBP on admission in mmHg ± SD	74.8 ± 13.2	65.3 ± 16.1	<0.001
Mean HR on admission in bpm ± SD	92.4 ± 17.3	96.2 ± 23.0	0.07
Hypotensive episodes	46 (33.8)	92 (71.9)	<0.001

SD = standard deviation; IHD = ischaemic heart disease; CKD = chronic kidney disease; SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate; bpm =beats per minute.

**Using Student t-test and Chi-square test, as appropriate.*

Table 2: Laboratory results of patients on admission to the intensive care unit who were tested for cardiac troponin levels (N = 264)

	n (%)		*P* value*
	Troponin not raised (n = 136)	Troponin raised (n = 128)	
ECG changes			<0.001
Not done	11 (8.1)	0 (0.0)	
Normal	66 (48.5)	43 (33.6)	
Sinus tachycardia	37 (27.2)	47 (36.7)	
BBB	21 (15.4)	19 (14.8)	
ST depression	1 (0.7)	26 (20.3)	
T wave inversion	0 (0.0)	20 (15.6)	
Frequent PVC†	0 (0.0)	10 (7.8)	
Atrial fibrillation	0 (0.0)	13 (10.2)	
ST elevation	0 (0.0)	6 (4.7)	
Heart block	0 (0.0)	1 (0.8)	
Other findings			
Ejection fraction	55 ± 9	48 ± 15	<0.001
Admission Hb in g/dL	10.8 ± 1.5	10.3 ± 1.9	0.013
Lowest Hb in g/dL	9.3 ± 1.8	8.2 ± 1.7	<0.001
Median admission creatinine in µmol/L (IQR)	65 (48–88)	76 (57–120)	0.004
Median peak creatinine in µmol/L (IQR)	84 (68–104)	124 (76–250)	<0.001
Median GFR on admission to ICU in mL/min (IQR)	90 (65–90)	81 (46–90)	<0.001
Median lowest GFR in mL/min (IQR)	75 (55–90)	45 (21–82)	<0.001
Median first troponin in ng/L (IQR)	17 (13–24)	50 (21–130)	<0.001
Median peak troponin in ng/L (IQR)	17 (12–22)	274 (99–756)	<0.001
Median CRP in mg/L (IQR)	110 (54–238)	179 (99–290)	0.004
Median D-dimer µg/LFEU (IQR)	2.5 (1.1–6.2)	4.5 (1.2–10.2)	0.21
Mean total cholesterol in mmol/L ± SD	3.9 ± 1.2	3.9 ± 1.7	0.9
Mean LDL in mmol/L ± SD	2.2 ± 1.05	1.7 (1.3–2.7)	0.28
Mean HDL in mmol/L ± SD	0.99 ± 0.38	1.1 ± 0.4	0.3
Median triglycerides in mmol/L (IQR)	1.4 (0.97–2.11)	1.2 (0.8–1.5)	0.37
Median LDH in U/L (IQR)	439 (223–942)	405 (226–569)	0.43
Median NT-proBNP in pg/mL (IQR)	1,270 (280–3,379)	3,855 (1,479–11,571)	0.003

ECG = electrocardiogram; BBB = bundle branch block; PVC = premature ventricular contractions; Hb = haemoglobin; IQR = interquartile range; GFR = glomerular filtration rate; ICU = intensive care unit; CRP = C-reactive protein; FEU = fibrinogen equivalent units; SD = standard deviation; LDL = low-density lipoprotein; HDL = high-density lipoprotein; LDH = lactic dehydrogenase; NT-proBNP = N-terminal pro-brain-type natriuretic peptide.

**Using Student's t-test, Chi-square test or Mann–Whitney U test, as appropriate.*

†Frequent PVCs were defined as more than 10 PVCs per minute.

found between the patients with raised troponin and those with normal troponin. However, those who had a raised troponin level were significantly older (64.3 ± 17.1 versus 54.6 ± 19.3 years; *P* <0.001) and had more co-morbidities compared to those whose with normal troponin levels. In addition, those who had a raised troponin level had lower systolic (115.7 ± 28.4 versus 123.1 ± 22.6; *P* = 0.014) and diastolic blood pressure (65.3 ± 16.1 versus 74.8 ± 13.2; *P* <0.001) at the time of admission to the ICU compared to those with normal troponin levels. Furthermore, patients with raised troponin levels were more likely to have at least one hypotensive episodes (defined as one sustained episode of mean arterial pressure of <60 mmHg that lasts for 30 minutes; 71.9% versus 33.8%; *P* <0.001) [Table 1].

Those with normal troponins were more likely to have normal ECG or sinus tachycardia (75.7%), while those with raised troponins were more likely to have abnormalities such as ST elevation or depression (25.0%), T wave inversions (15.6%) and bundle branch block (14.8%). The ejection fraction, as measured by echocardiography, was lower in patients with raised troponins; however, none had been diagnosed with stress-induced cardiomyopathy. Patients with raised troponins also had lower haemoglobin and glomerular filtration rates, along with higher serum creatinine, C-reactive protein, N-terminal pro-brain type natriuretic peptide and troponins [Table 2].

Of the patients with raised troponins, 110 were referred to the cardiology department for specialist's opinion. The treatment for acute coronary syndrome (ACS)—dual antiplatelet and therapeutic anticoagulation therapy—was initiated for 47 of these. ACS management was not started in the remaining patients for the following reasons: the rise was estimated to not be related to a cardiac condition in 44 patients, including six who were diagnosed with

Table 3: Treatment/management of the patients who underwent a high-sensitive cardiac troponin assay and were admitted to the intensive care unit at Sultan Qaboos University Hospital, Muscat, Oman, between January and December 2019 (N = 264)

Treatment/ management	n (%)		*P* value*
	Troponin not raised (n = 136)	Troponin raised (n = 128)	
Dialysis	23 (16.9)	38 (29.7)	0.01
Therapeutic anticoagulation	0	53 (41.4)	<0.001
Prophylactic anticoagulation	86 (63.2)	43 (33.6)	<0.001
Any anticoagulation	86 (63.2)	96 (75.0)	0.51
Antibiotics	99 (72.8)	116 (90.6)	0.01
Inotropes	40 (29.4)	80 (62.5)	<0.001
Dual antiplatelet agents	0 (0.0)	47 (36.7)	<0.001
Single antiplatelet therapy	21 (15.4)	27 (21.1)	0.07
Any antiplatelet therapy	21 (15.4)	74 (57.8)	<0.001
Cardiology consultation	17 (12.5)	110 (85.9)	<0.001

**Using Chi-square test.*

a pulmonary embolus; 13 were designated as 'not for resuscitation' and six had significant bleeding. Patients in whom ACS treatment was initiated did not have significantly different troponin values or ECG changes as compared to those for whom ACS treatment was not initiated.

A coronary angiogram was performed in 30 of the 47 patients who were treated for ACS. The reason for not performing angiogram in the remaining 17 was that seven patients were designated as 'not for resuscitation', six were very unstable and critical and, hence, not fit for invasive procedures and four did not give their consent. There were another two patients for whom ACS treatment was not initiated but an angiogram was performed due to their clinical condition. Of the 32 on whom angiogram was performed, 24 were normal, two had mild disease, three had longstanding chronic total occlusions and only three had a significant disease that required stenting.

Regarding the treatments received by the patients, those with raised troponins were more likely to receive therapeutic anticoagulation and dual antiplatelet therapy. They were also more likely to undergo dialysis and be started on inotropes (62.5% versus 29.4%; *P* <0.001) [Table 3].

Table 4: Final diagnosis of patients who underwent a high-sensitive cardiac troponin assay and were admitted to the intensive care unit at Sultan Qaboos University Hospital, Muscat, Oman, between January and December 2019 (N = 264)

Final diagnosis*	n (%)		*P* value†
	Troponin not raised (n = 136)	Troponin raised (n = 128)	
Sepsis	46 (33.8)	75 (58.6)	<0.001
Heart failure	10 (7.4)	38 (29.7)	<0.001
Pulmonary embolus	0 (0.0)	6 (4.7)	1.000
Central nervous system cause	31 (22.8)	12 (9.4)	<0.001
Respiratory failure	18 (13.2)	2 (1.6)	
Malignancy	22 (16.2)	12 (9.4)	
Trauma	12 (8.8)	2 (1.6)	
OHCA	3 (2.2)	10 (7.8)	
CKD	1 (0.7)	3 (2.3)	
GI cause	14 (10.3)	10 (7.8)	
Others‡	7 (5.1)	2 (1.6)	

OHCA = out of hospital cardiac arrest; CKD = chronic kidney disease; GI = gastrointestinal.

**Values add up to more than the number of patients as some patients had more than one diagnosis.*

†Analysis by Chi-square test.

‡Includes anaphylaxis, mesenteric ischaemia, poisoning and diabetic ketoacidosis.

Patients with raised troponins were more likely to have a diagnosis of sepsis, heart failure or pulmonary embolism, while those with normal troponins received diagnoses that included a central nervous system cause, malignancy, trauma and gastrointestinal bleed [Table 4].

There were 46 patients (age 67.2 ± 14.2 years; n = 28 males) who fulfilled the criteria for a type 2 MI. Although not statistically significant, these patients tended to be older than those who had myocardial injury without infarction; there was also a trend of them having more comorbidities, higher peak creatinine and troponin, higher mortality rates and shorter hospital stays. However, none of these parameters were found to be statistically significant [Table 5].

Patients with raised troponins had a shorter length of hospital stay than those with normal troponins (16 [range: 8–25] versus 19 [range: 13–28] days; *P* = 0.017). Moreover, the in-hospital mortality rate of those with raised troponin was 64.8% compared to 26.5% for those with normal troponin levels. Patients with raised troponins had a poor outcome, with only 45 (35.2%) surviving to discharge compared to 74.3% for those with normal troponin [Table 6].

Table 5: Characteristics of patients with type 2 myocardial infarction versus myocardial injury without infarction (N = 128)

Characteristic	n (%)		*P* value*
	Myocardial injury without infarction (n = 82)	Type 2 MI (n = 46)	
Mean age in years ± SD	62.6 ± 18.1	67.2 ± 14.2	0.14
Male:female ratio	47:35	28:18	0.69
Alive on discharge	33	12	0.12
Median peak troponin in ng/L (IQR)	262 (96–690)	351 (118–899)	0.32
Median peak creatinine in µmol/L (IQR)	118 (72–211)	132 (83–281)	0.43
Haemoglobin in g/L ± SD	10.3 ± 1.7	10.1 ± 2.3	0.91
Hypertensive	51 (62.2)	32 (69.6)	0.41
Diabetic	32 (39.0)	23 (50.0)	0.23
Previous IHD	11 (13.4)	11 (23.9)	0.76
Sepsis	48 (58.5)	27 (58.7)	0.18
Median LOS in days (IQR)	17 (10–27)	12 (5–22)	0.13

MI = myocardial infarction; SD = standard deviation; IQR = interquartile range; IHD = ischaemic heart disease; LOS = length of hospital stay.

**Using Student's t-test, Chi-square test or Mann–Whitney U test, as appropriate.*

Another three patients with raised troponins out of the 45 who were discharged alive died at one month compared to none for the patients with normal troponins. However, there were more hospital re-admissions within one month among the group with normal troponin levels. Underlying malignancy was found in 12 of the 17 patients without raised troponins, and they were readmitted for a worsening condition/sepsis; 10 of these patients died during the second admission. The remaining five patients were admitted for worsening renal functions and required dialysis. All three of the patients with raised troponins who were readmitted had presented with signs of sepsis. All three died during the second admission.

The probability of death for people with a raised troponin level was calculated at 5.32 (95% confidence interval: 3.14–9.03). Based on binary logistic regression, the factors that predicted in-hospital deaths were age (P = 0.01), peak troponin (P = 0.004), lowest haemoglobin (P = 0.01), peak serum creatinine and glomerular filtration rates (P = 0.02) and hypotensive episodes (P <0.001). The other parameters were not predictive.

Table 6: Outcomes at discharge and at one month of patients who underwent a high-sensitive cardiac troponin assay and were admitted to the intensive care unit at Sultan Qaboos University Hospital, Muscat, Oman, between January and December 2019 (N = 264)

	n (%)		*P* value*
	Troponin not raised (n = 136)	Troponin raised (n = 128)	
Median LOS in days (IQR)	19 (13–28)	16 (8–25)	0.017
Alive at discharge	101 (73.5)	45 (35.1)	<0.001
Status at one month for patients discharged alive with follow-up	92 (67.4)	39 (30.5)	
Alive†	75 (81.5)	33 (84.6)	
Dead†	0 (0.0)	3 (7.7)	
Readmitted†	17 (18.5)	3 (7.7)	0.001*
No follow-up†	9	6	0.001†

LOS = length of hospital stay; IQR = interquartile range.

**Using Chi-square test.*

†Percentage calculated out of the patients discharged alive with follow-up.

Discussion

Raised cardiac troponins, in the absence of significant CAD, have been described in many clinical scenarios, including sepsis, renal failure, generalised or localised infections and post-operative states and especially in critically ill patients.[1] Previous studies have stated a median value of 43% (IQR: 21–59%) of ICU patients exhibiting raised troponin levels, with the figure rising to more than 60% for those with sepsis or septic shock.[8,9] The mortality rate of these patients has also shown to be higher than those with normal troponin values.[8] The findings of the current study are largely consistent with the published data and results showed that troponin levels were raised in 48.5% of those who were tested, with 58.6% of these patients having sepsis. The mortality rate of those with raised troponins was five times higher compared to those with normal levels. Patients with raised troponin levels were generally more ill, are more likely to be on inotropes and have multi-organ failure and sepsis, which explains the higher mortality rate.

The reasons for the raised troponins in the absence of CAD range from myocardial inflammation (as seen in sepsis) to myocardial injury due to hypoxia, hypoperfusion and decreased excretion of the molecule,

as seen in the case of renal failure.[2] Many precipitants of myocardial injury in critically ill patients have been described such as tachycardia, anaemia, sepsis and hypotension; indeed, the current study confirms that those with raised troponin had a higher mean heart rate on admission, lower haemoglobin values, lower blood pressure and at least one hypotensive episode.[10] Stress-induced cardiomyopathy is also another entity that can occur in these patients and is not always identified on echocardiography.[11]

Raised troponin levels alone are not diagnostic of an acute MI; patients need to fulfil the criteria, as described earlier. Distinguishing between myocardial injury and either type 1 or type 1 MI is often difficult in the ICU setting.[12–14] Inability to get a proper history of chest pain, lack of 'baseline' ECG and echocardiogram in many cases and lack of specificity of ECG and echocardiogram changes add to this diagnostic difficulty.[15,16] Coronary angiographies are frequently required to differentiate between a type 1 and type 2 MI and further tools such as intravascular ultrasound or optical coherent tomography might be required to assess whether the atheroma is stable or unstable.[5,17]

Physicians tend to err on the side of caution and there is a tendency to over-diagnose MI; indeed, postmortem studies suggest that the rate of type 2 MI is not as high as it is suspected clinically.[16,18] One suggestion to help differentiate myocardial injury from MI is to use different thresholds for the troponin values.[19] This approach, however, is unlikely to be effective, as the amount of troponin leak would depend on multiple factors, such as the underlying health condition of the myocardium (previous MI, cardiomyopathy, etc.) and the presence of non-obstructive atheroma. There are no clear guidelines regarding the management of patients with myocardial injury or type 2 MI, given the paucity of trial data. All the guidelines on ACS management are for type 1 MI patients (either with or without ST-segment elevation) who present with chest pain. The management of patients with type 2 MI is often left to the individual treating clinician. One of the approaches that many physicians take is to treat all patients with raised troponins as ACS, unless there is a clear precipitating cause for non-ischaemic myocardial injury, and perform a coronary angiogram once the patient has been stabilised. However, many constraints often prevent this. Many patients are hypotensive on inotropic support, and as such, anti-ischaemic measures such as beta-blockers or nitrates might not be appropriate. Bleeding issues prevent the initiation of dual antiplatelet therapy.[20] Moreover, many patients with multiple underlying conditions and poor pre-morbid state are labelled 'not for resuscitation', and hence, invasive procedures may be deemed inappropriate in these situations. Indeed, in the study's cohort, only 47 out of the 128 patients were given treatment for ACS management.

As in other studies, the researchers found that a large proportion of the patients having raised troponins were not treated for ACS.[6,21–23] Clear triggering factors for a demand–supply mismatch and alternative diagnosis such as heart failure or pulmonary embolism were the reasons in the current study. Even where ACS was considered, other factors such as altered coagulation profile, bleeding complications and general conditions prevented the initiation of ACS management, thereby reflecting the complex nature of these patients and the practical difficulties encountered in their management in a real-world setting.

Although ACS management might be ineffective in non-coronary-related myocardial injury or type 2 MI, evidence suggests that aspirin, beta-blockers and statin might improve prognosis.[24] It has been postulated that serious or critical illness might trigger a hypercoagulable state and this is countered with the aspirin.[25] Beta-blockers have been suggested to help by reducing the cardiac workload and reducing the heart rate, thereby reducing oxygen demand.[5] However, these data are derived from retrospective studies; prospective studies are required to conclusively show the benefit of this method.

Subjecting these critically ill patients to an invasive coronary angiogram is debatable. Previous studies have shown quite a variable rate of angiography in these patients, with many studies showing a high prevalence of normal coronaries or non-obstructive lesions in patients with sepsis and raised troponins.[21] In the current study, a high proportion of patients treated for ACS underwent a coronary angiogram, with only three patients having significant coronary artery disease (suggesting a type 1 MI) that required stenting.

The management of patients with coincidental coronary atheroma is controversial. It has been suggested that relieving this obstruction may benefit the patient, as in the presence of coronary atheroma, the threshold for the cause of a demand–supply mismatch is lower. Removing this obstruction could theoretically increase the threshold for myocardial injury.[5] However, there are no trials to prove this, and this potential improvement in the flow could be offset by an increased risk of bleeding.[5] The Appropriateness of Coronary Investigation in Myocardial Injury and Type 2 MI trial is an ongoing randomised trial that compares invasive coronary angiography (or coronary computed tomography angiogram) with conservative management on two-year all-cause mortality, which

the researchers hope would provide insights into the management of these patients.[26]

In keeping with other studies, the current study found that patients with raised troponins had a higher mortality rate (five-fold) than those with normal troponin values. These patients had a lower length of stay in the hospital, presumably since many died early during the hospital stay. The reason for this increased mortality could be the severe nature of the underlying illness. In the current study, the majority of the patients with raised troponin had sepsis, and, indeed, many had experienced at least one hypotensive episode and needed inotropes, reflecting the serious nature of their illness.

The current study, being retrospective in nature, is characterised by limitations that are inherent to such types of studies. The quality of the data obtained depends on the quality of the electronic records. In most cases, all of the required information could be extracted. Follow-up information, however, was lacking in a small number of patients. The group considered to be type 2 MI was not analysed separately due to the difficulty in making that diagnosis, which is similar to the approach of other published studies on the topic.[6,7,8,9] It is likely that the hs-cTn assays in this study picked up a higher number of myocardial injury without MI. Indeed, in the cohort, only 48 of the 128 patients with raised troponin fulfilled the criteria for type 2 MI, and it is probable that only a smaller number of these would have had histopathological changes of MI. Despite these limitations, the data are important, as they provide information about myocardial injury in the ICU setting in Oman. In addition, this study is the first of its kind from the region.

Conclusion

A large proportion of critically ill patients admitted to the ICU had evidence of myocardial injury and/or MI. These patients had a worse prognosis and treating them for ACS did not appear to improve their prognosis. Thus, more research is required to fully understand the pathophysiology and management of these patients.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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AUTHORS' CONTRIBUTION

MMS, MAK, WAA, SA, AA, MA and FA collected the data. SKN analysed the data. MA, FA and SKN drafted the manuscript. All authors approved the final version of the manuscript.

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