Cardiac magnetic resonance imaging in suspected blunt cardiac injury: A prospective, pilot, cohort study

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Keywords

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Abstract

Introduction: The aim of this study was to evaluate the incidence and severity of blunt cardiac injury (BCI) as determined by cardiac magnetic resonance imaging (CMR), and to compare this to currently used diagnostic methods in severely injured patients.

Materials and Methods: We conducted a prospective, pilot cohort study of 42 major trauma patients from July 2013 to Jan 2015. The cohort underwent CMR within 7 days, enrolling 21 patients with evidence of chest injury and an elevated Troponin I compared to 21 patients without chest injury who acted as controls. Major adverse cardiac events (MACE) including ventricular arrhythmia, unexplained hypotension requiring inotropes, or a requirement for cardiac surgery were recorded.

Results: 6/21 (28%) patients with chest injuries had abnormal CMR scans, while all 21 control patients had normal scans. CMR abnormalities included myocardial oedema, regional wall motion abnormalities, and myocardial haemorrhage. The left ventricle was the commonest site of injury (5/6), followed by the right ventricle (2/6) and tricuspid valve (1/6). MACE occurred in 5 patients. Sensitivity and specificity values for CMR at predicting MACE were 60%(15-95) & 81%(54-96), which compared favourably with other tests.

Conclusion: In this pilot trial, CMR was found to give detailed anatomic information of myocardial injury in patients with suspected BCI, and may have a role in the diagnosis and management of patients with suspected BCI.

Introduction.

The incidence of blunt cardiac injury (BCI) in chest trauma ranges from 0-50%, and recent estimates suggest over 30 000 cases per year occur in the USA alone. Serious sequelae of BCI include malignant arrhythmia[1], heart failure[2,3] cardiac rupture[4-6], and death[7,8].

Current diagnostic tests in BCI and its complications achieve only moderate sensitivity and specificity[2,8]. These include troponin, creatinine kinase (CK), electrocardiogram (ECG), echocardiography and computerised topography (CT). There is currently no gold standard diagnostic test, making the investigation, diagnosis, risk stratification, and management of these patients challenging[4,5,9,10]. Furthermore, the increasing use of troponin as a screening tool in thoracic trauma, especially in the elderly population who also have risk factors for coronary arterial disease, can lead to patients being over investigated with invasive procedures to rule out acute coronary syndrome (ACS).

Cardiac magnetic resonance (CMR) imaging has been found to be highly effective in the diagnosis of structural heart disease, and is noted for its superior functional and morphological information[11,12], as well as tissue characterisation[10,13,14]. CMR has been demonstrated to effectively diagnose BCI in multiple case reports [6,15-17]. The most recent EAST trauma guidelines[18,19], and others[20,21], have recognised the potential benefits of using CMR, but to date there have been no prospective trials using CMR in BCI.

The aim of this study was to investigate the incidence and severity of BCI as determined by CMR in non intubated, haemodynamically stable patients with

major thoracic trauma, and then to compare this to other currently used diagnostic tests.

Materials and Methods.

Design:

This is a prospective, observational cohort study. The study protocol was approved by the research and ethics committee of the Alfred Hospital, Melbourne, Australia.

Setting and population:

The study was performed at the Alfred Hospital, a Level 1 Adult Major Trauma Centres in Victoria, Australia[22,23] with over 7000 trauma admissions per year. All patients admitted to hospital with major trauma (defined as ISS>12) were screened on weekdays (Monday to Friday) for eligibility by the research team. Patients presenting out of hours, on weekends, or when the CMR scanner was not available were not included. Patients who were haemodynamically unstable, ventilated or who had a contraindication to CMR were excluded from the study. Enrolled patients were divided into two groups. The study group with chest trauma had elevated troponin levels[19,24], and corroborating evidence of thoracic injury, such as fractured ribs, sternum, or significant pulmonary contusions. A second, control group had major traumatic injuries but no evidence of chest trauma as defined by initial trauma CT scan. Demographic data were collected, and Injury Severity Score (ISS), Acute Physiology and Chronic Health Evaluation Score (APACHE) and Trauma Injury Severity Score (TRISS) calculated.

Study protocol:

All patients underwent continuous cardiac monitoring from admission. Serum troponin, CK, as well as ECG were taken upon arrival, and then were repeated daily for three days or until stabilised[25]. ECGs were analyzed and reported by clinicians blinded to the study aims for evidence of acute myocardial injury or conduction defects. A transthoracic echocardiogram (TTE) was performed as soon as was possible (usually <48hours). If the images were suboptimal, then the patient underwent transesophageal echocardiogram (TEE). All echocardiograms were reported by an experienced cardiologist blinded to the study aims.

CMR Protocol:

All patients underwent CMR as per the study protocol within one week of admission (Signa HD 1.5T; GE Healthcare, Waukesha, WI, USA). Patients were offered additional oral analgesia to complete the scan. After initial localizer scans, a contiguous steady state free precession cine stack was acquired to cover both ventricles from the atrioventricular groove to the left ventricular (LV) apex (slice thickness 8mm). To assess myocardial oedema, a T2-weighted short-tau inversion recovery (STIR) sequence was acquired using the same coordinates as the cine stack, and in addition a T2-mapping sequence was acquired in 3 standardised short axis levels (basal, mid and apical). The T2-mapping sequence was a prototype multi-echo double inversion recovery-fast spin echo (MEFSE) technique (Global Applied Science Laboratory, GE Healthcare). In this sequence, the echo train is divided into equal sized segments – one segment per echo time. This information can be used to

generate myocardial T2 weighted SI versus echo time (TE) curves with the myocardial T2 obtained using curve fitting algorithms. Late gadolinium enhancement (LGE) images were acquired 10-15 minutes following the administration of gadolinium– diethylenetriamine pentaacetic acid (DTPA) (0.2 mmol/kg, Magnevist, Bayer Schering Pharma, Leverkusen, Germany) using an inversion recovery gradient echo technique applied to the same coordinates as the cine stack. The total duration of the CMR scan was 60-90 minutes.

CMR Analysis:

The CMR analysis included LV and right ventricular (RV) volume, LV mass and LV systolic global and regional function. All CMR scans were independently interpreted by an experienced cardiologist and radiologist, who were blinded to the the patient group allocation or results of the other investigations. Calculation of LV and RV volume, mass and function was performed using the summation of disc method according to recommended guidelines[2]. The presence of LGE was determined by applying a semi-automated visual threshold. Myocardial oedema was evaluated by calculating the global STIR intensity relative to skeletal muscle, which was averaged over all STIR slices, and in addition regional STIR was calculated for areas with the highest regional intensity. Calculation of myocardial T2 time was performed in the 3 short axis slices by a curve fitting technique using a dedicated research software package (CMR42; Circle Cardiovascular Imaging, Inc., Calgary, Canada). CMR findings of traumatic myocardial injury were defined as any regional wall motion abnormality (RWMA), elevated regional STIR or LGE if also associated with

focal hypokinesis, mild or greater pericardial effusion, intramural haematoma, or new valvular lesion.

Outcomes:

The primary outcome was any evidence of traumatic myocardial injury on CMR. Secondary outcomes included any evidence of major adverse cardiovascular events (MACE), and the ability of the diagnostic tests to predict MACE. In order to reduce potential bias, outcomes were prespecified, and were documented for all patients until discharge, by clinicians unaware of the MRI findings. MACE was defined as malignant arrhythmia, unexplained hypotension requiring inotropes, or injuries requiring cardiac surgery[9]. Patients with an abnormal CMR were then followed up with a repeat CMR at approximately 9 months.

Statistical analysis

Analyses were performed using SPSS (Version 19.0 SPSS Inc, Chicago, IL, USA). All continuous data are presented as mean +/- standard deviation. Continuous data were analysed using Students *t*-test or Mann-Whitney test as appropriate after normality testing. Proportions were analysed with Chi-squared testing, except where value in a cell was less than 5, where the Fisher's Exact test was used. 95% confidence intervals in table 4 were calculated using the exact binomial method. A p-value of <0.05 was considered to be statistically significant.

Results.

Patient enrolment and baseline characteristics

From 1 July 2013 to 31 Jan 2015, 47 patients with major trauma admitted to the Alfred Hospital were screened for eligibility for the study as shown in figure 1. Five patients did not complete the study; three patients refused consent, one patient suffered claustrophobia and was unable to complete the CMR scan, and one patient had a non CMR compatible external fixation device. Thus 42 patients were enrolled in the study until completion, of whom 21 patients had chest trauma, and 21 did not. The scan was tolerated in all patients, but some required addition oral analgesia during breath holds to complete the study. Baseline characteristics are shown in the Table 1. Patients with chest trauma tended to have higher AIS score and TRISS scores, the mechanism of injury was more likely to be motor vehicle accident (MVA), and they were more likely to have been managed in the intensive care unit.

Incidence and distribution of CMR abnormalities

All patients underwent cardiac CMR as per protocol. Of the 42 patients enrolled, 6 (14%) had an abnormal CMR. All abnormal scans were from the chest trauma group 6/21 (28%), while all control patients had normal MRI scans 0/21 (0%) (Table 1). Table 2 shows differences in demographics, initial physiological measures, and MRI findings between chest trauma and control patients. Table 3 shows the distribution of myocardial injuries seen on CMR. RWMAs, with associated focal oedema (with localised elevation of STIR signal) was the commonest abnormality, two patients had reduced right ventricular function and oedema. One patient had evidence of intramural haemorrhage[11], while

another had a rupture of the tricuspid valve with a flail leaflet. The left ventricle was the commonest site of injury, with 5/6 cases demonstrating evidence of LV injury and 2/6 cases demonstrating RV injury (Figure 2).

Echocardiographic findings

Transthoracic echocardiography was performed in 31/42 patients. 3/21 in the chest trauma group had limited views on their echocardiogram but clinicians considered them to be adequate. TTE was abnormal in four chest trauma patients, including at least moderate tricuspid valvular regurgitation, a RWMA, a pericardial effusion, and RV dysfunction. Only one of these patients had MACE. The correlation of TTE with MRI was poor: 3 patients with abnormal MRIs had normal TTEs. TEE was used in one patient to confirm a tricuspid valve lesion which had also been seen on TTE imaging. There were no echocardiographic changes in the control group.

Electrocardiogram findings

Nine of 21 chest trauma patients (43%) had abnormal ECG changes compared to 4/21 (21%) of the controls had an abnormal ECG. The commonest abnormalities were ST changes, T wave inversion, AV block and right bundle branch block.

Elevations in cardiac troponins

Initial troponin was positive in all patients in the chest trauma group (as per protocol), while no patients in the control group had an elevated troponin. Troponin was elevated in all whom subsequent CMR or echocardiography was found to be abnormal, while it was also positive in 9/13 patients with abnormal ECGs.

Prediction of MACE

5/21 (23%) of patients had MACE, all from the chest trauma group, all within the first week of their hospital admission. Two patients had persistently low blood pressure with no other cause found requiring inotropic support, one had evidence of cardiogenic shock requiring adrenalin and recurrent atrial fibrillation, one patient had 2 episodes of ventricular tachycardia, and one patient had a ruptured tricuspid valve with heart failure requiring replacement. Two other adverse events occurred. One patient from the control group had a large MCA stroke and died, and another from BCI group had a clinically non-significant pulmonary embolism and was treated with anticoagulation.

Table 4 shows the performance of the diagnostic tests at predicting MACE during hospital stay, although overall numbers were small and confidence intervals were wide. An abnormal troponin was highly sensitive for predicting MACE, but not specific. ECG was also sensitive but less specific. Echocardiography was the least sensitive but had moderate specificity. CMR

performed moderately well identifying 3/5 or 60% (15-95) of patients with MACE, and performed well at ruling it out, with 13/16 or 81% (54-96) specificity.

9 Month Follow up MRI Scan

Four of the six patients with abnormal CMRs underwent repeat CMR at 9 months (mean 296, SD 20 days) as shown in Table 5. In 3 out of four patients, regional STIR values had fallen while RWMA's persisted, and in 3 out of 4, new regional scar had developed.

Discussion

This is the first study to prospectively analyse CMR in severely injured patients with thoracic trauma. 28% of chest trauma patients had CMR evidence of cardiac injury. We found CMR gives high quality anatomic and pathological information, and within the limits of a pilot study, performed well at predicting adverse outcomes compared to currently used diagnostic tests.

Previous literature has reported a wide range of incidence and outcomes in BCI [10,14]. Some of the challenges have included a lack of uniform diagnostic criteria, the inclusion of heterogeneous populations, and no gold standard diagnostic test.

CMR is a novel diagnostic modality in chest trauma patients, possibly in part due to safety concerns about transporting unstable patients. However very few studies have investigated CMR in this context. For this reason, the population in this study were hemodynamically stable patients who were not intubated, to minimize the transportation of unstable or severely injured patients. MACE occurred in all patients within the first week, such that many patients had their CMR afterwards. Given the superior imaging quality, and potential for improved diagnostic accuracy, it is likely the earlier use of CMR in trauma patients will yield improved diagnostic accuracy, with improved risk stratification and management, which may justify its earlier use in more unstable patients. These advantages however will need to be carefully balanced against any safety concerns, and will be the object of further studies.

The distribution of myocardial injuries on CMR included anterior, septal, and lateral walls of the left ventricle, while the inferior wall was spared (Figure 2). Two patients also had RV injuries, including one with a ruptured tricuspid valve.

Previous autopsy studies have suggested the right heart is more commonly affected, with a rate of injury of 27% to the RV compared with 18% of the LV[6]. In addition to direct trauma, alternative mechanisms such as compression or traction have also been proposed[18]. Huguet proposed RV over-distension causes stress points in the LV at the moderator and RV insertion points[20]. Furthermore, sudden increase in venous return during right ventricular systole when the tricuspid valve is closed can result valvular rupture – a possible mechanism in our patient[22].

The choice of the controls was to address the issue that non chest trauma patients can suffer cardiac related damage, including elevated troponin levels in up to 30% of major trauma[26], and severe TBI patients [27], as well as the potential for right heart damage following abdominal trauma. Thus, as the first prospective CMR study in major trauma, it was not known whether non chest trauma patients would have had abnormal CMR scans. We found however no controls had abnormal CMR scans, an important finding for future studies in this area.

In this study CMR was not inferior and may be superior at predicting adverse events compared to other diagnostic tests although overall numbers were small (table 4). CMR detected 3/5 (60%) of patients who developed MACE, but did not detect two patients with MACE. Possible explanations of this include the delay to perform the CMR until day 7, or the exclusion of more severely injury patients, particularly intubated patients. It may be that earlier CMR, or inclusion of more severely injured patients may increase the rate of detection even further. CMR also performed well at ruling our MACE, with 13/16 (81%) of negative tests being true negatives (although numbers were small). In contrast to other studies,

we found no association with rib fracture or fractured sternum with abnormal CMR or MACE[24]

Although studies have shown troponin to be highly sensitive and specific for other types of myocardial injury[1,28], it is less accurate in chest trauma [3,29,30]. In this study we found it to be highly sensitive (100%) but not specific (21%) at predicting MACE. Increasing the cut off to a level of >1.0 ng/mL had the effect of reducing sensitivity while only marginally increasing specificity. TTE has advantages over CMR in that it is point of care and doesn't require patient transportation. However in this study, we found TTE was not sensitive at predicting MACE. We also found was poor correlation between abnormal TTE and CMR. Previous studies have suggested more routine use of TEE may have improve sensitivity and specificity of echocardiography [4,5,31], however more contemporary studies have found TTE to be similarly effective at detecting cardiac injury[6,7,32] but without the associated morbidity and cost of TEE.

Many patients in our population were elderly, with presentations similar to acute coronary syndrome, including abnormal ECG and elevated troponin levels. In these cases, we found CMR particularly useful in distinguishing between ACS and BCI, as injuries tended to originate from the epicardial border, and cross over coronary territories[8,33]. This circumvented unnecessary interventions such as cardiac catheterization.

There are limited studies investigating the long term effects of BCI. A broad range of effects have been described, from no adverse effects[10,34], persistent structural heart defects[12,35], late arrhythmias[13,36] and the presence of scar on CMR at 6 weeks[15-17,37]. In our study four patients were followed up for 9 months. Three out of the four patients had persistent evidence of scar on CMR,

including in one patient who had intramural haematoma on initial the CMR scan. No patient had MACE or hospitalizations in the 9 months following their initial injury. The clinical significance of scar following BCI is yet to be determined, but it may predict future cardiac complications, such as late ventricular arrhythmias as seen post myocardial infarction[19,38].

Limitations:

In this pilot study, the overall number of BCI patients, as diagnosed by CMR or MACE was low. Although more patients were screened, the exact number and reason for ineligibility was not recorded in many cases. Cardiac computerized topography was not available and therefore a direct comparison of CMR and CT was not performed. The results are limited to haemodynamically stable and non-intubated patients with BCI. Furthermore many Trauma Centres currently do not have access to a CMR service. Further studies will be required to delineate the exact role CMR has in the overall diagnostic algorithm of patients with suspected BCI.

Conclusion

Patients admitted to hospital with major chest injuries may have higher rates of clinically significant BCI than previously recognised. Current EAST guidelines [19,21] suggest echocardiography in patients with suspected BCI, and to consider CMR as an alternative to distinguish BCI from AMI. Whilst the exact role of CMR in BCI remains to be clarified, we have found CMR gives detailed anatomic information of myocardial injury, and may have an important role in the diagnosis and management of patients with suspected BCI.

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Figure 1: Study Flow Diagram

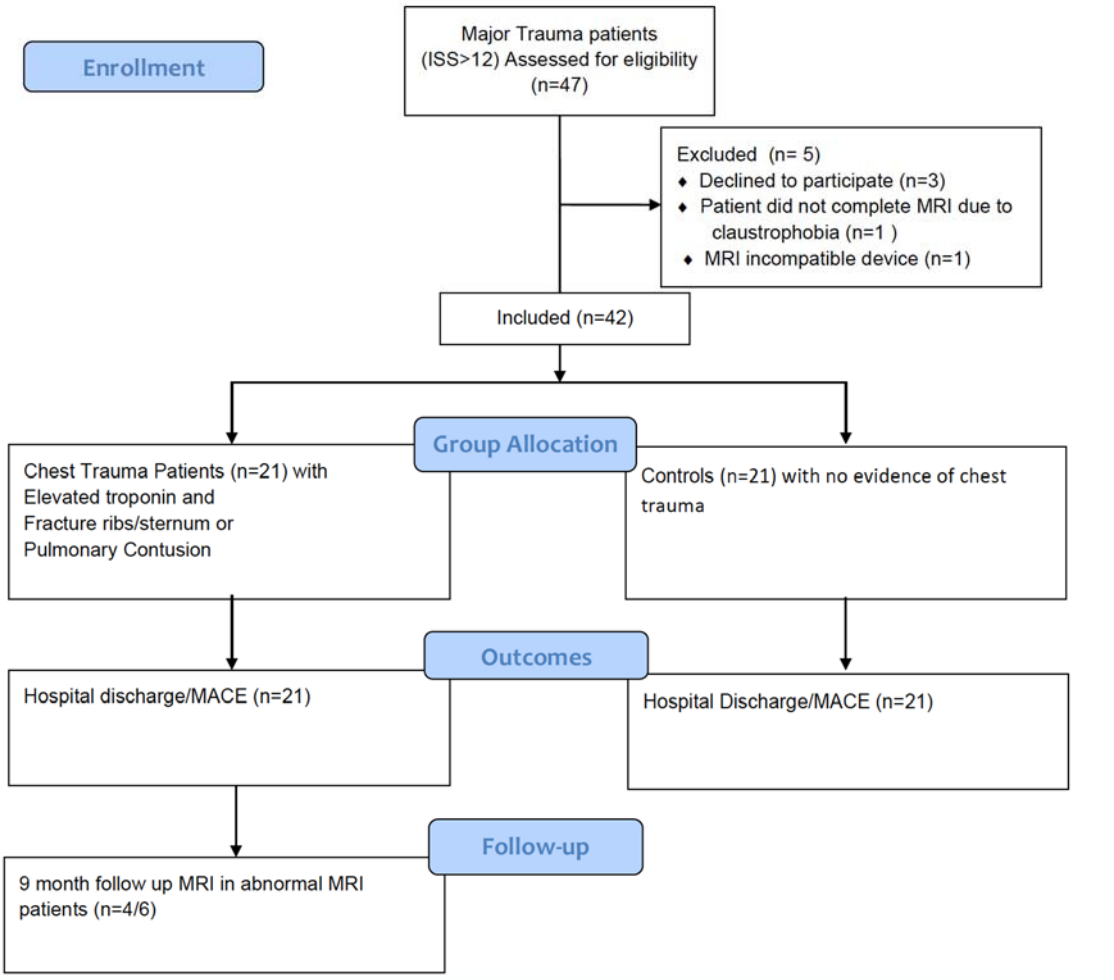

Figure 2: Left and right ventricular distribution of myocardial injuries

Table 1: Baseline Demographics

	Control N=21	Chest trauma N=21
Demographics		
Age, years	42 [15]	40 [22]
Male, no (%)	16/21 (76)	16/21 (76)
Hypertension, no (%)	1/21 (5)	2/21 (10)
Fractured Sternum, no (%)	0 (0)	10/21 (47)
Fractured ribs, no	0 [0]	3.5 [3]
AIS 2008	13 [8]	23 [11]
NISS Score	17 [12]	32 [12]
TRISS Score	0.84 [0.4]	0.90 [0.1]
Mechanism, no (%) -MVA -Cyclist -MBA -Fall -Other	 3 (14) 2 (10) 5 (24) 7 (33) 4 (14)	 12 (57) 2 (10) 3 (14) 2 (10) 2 (10)
Speed at impact, km/hr	69 [35]	66 [27]
Vitals in Emergency Room		
HR, min	82 [14]	106 [26]
SBP, mmHg	125 [58]	140 [32]
GCS	15 [1]	14 [3]
Oxygen Sats (%)	98 [2]	98 [4.0]
Fio2 (%)	23 [10]	51 [28]
Lactate, mmol	1.3 [0.6]	2.3 [1.4]
Investigations		
Troponin initial, ng/mL	0.01 [0.01]	1.61 [2.50]
Troponin peak, ng/mL	0.03 [0.04]	2.80 [5.01]
CK peak, ng/mL	404 [614]	4064 [4420]
Abnormal CMR, no (%)	0/21 (0)	6/21 (29)
ECG changes, n (%)	4/21 (21)	9/21 (43)
Acute Echo change, no (%)	0 (0)	4 (22)
Limited Echo windows, no (%)	0 (0)	3/21 (17)
Echo, days to	7.1 [8.7]	8.8 [5.4]
CMR, days to	3.9 [2.4]	4.6 [2.2]
Outcomes		
Location, no (%) -Ward -Monitored -ICU	 17/21 (77) 0 (0) 1 (5)	 5/21 (23) 2 (10) 14 (67)
ICU duration, hours	41 [21]	114 [57]
Hospital LOS, days	9.1 [10.7]	8.8 [5.4]
Hospital Mortality, %	0/21 (0)	1/21 (5)

Table 2: Patients with abnormal Magnetic Resonance scans.

	Normal CMR N=15	Abnormal CMR N=6
Demographics		
Male	13/15 (87)	3/6 (50)
Speed, km/hr	68 [23]	60 [37]
AIS 2008	25 [11]	22.0 [13]
TRISS	0.91 [0.07]	0.88 [0.21]
In Emergency Room		
HR, min	103 [30]	114 [8]
SBP, mmHg	129 [33]	149 [43]
DBP, mmHg	83 [18]	66 [17]
Lactate, umol/L	2.6 [1.6]	2.2 [1.0]
Troponin initial, ng/mL	1.1 [2.2]	2.8 [2.8]
Troponin Peak, ng/mL	2.1 [5.6]	4.4 [3.4]
ECG Abnormal, (%)	5/15 (33)	4/6 (67)
Echo Abnormal, (%)	1/15 (8)	3/6 (50)
Rib #, %	13/15 (87)	3/6 (50)
Rib # number	4.4 [3.3]	1.2 [1.6]
Bruising, %	7/15 (47)	3/6 (50)
Sternal #, %	9/15 (60)	1/6 (16)
ICU duration, hours	118 [59]	99 [55]
Location Ward Monitored ICU	 5/15 (33) 0/15 (0) 10/15 (67)	 0/6 (0) 2/6 (33) 4/6 (67)
CMR Findings		
LVEDV	167 [44]	147 [48]
LVESV	65 [24]	59 [22]
EF, %	63 [7]	60 [6]
RWMA, %	1/14 (7)	4/6 (67)
RV Dysfunction	0/15 (0)	1/6 (17)
STIR Av	1.7 [0.4]	2.2 [0.6]
STIR regional	1.8 [0.4]	2.5 [0.7]
T2 time, Basal total	44 [10]	43 [5]
T2 time, Mid total	48 [7]	49 [4]
T2 Apical	55 [9]	57 [10]

Table 3. Distribution of myocardial injuries as seen on CMR

Abnormal CMR N=6	Incidence	Comments
RWMA -Ant wall -Anterolateral -Antero septum -Septum	4 1 1 1 1	
RV wall Oedema	1	
Effusion	1	Trivial
STIR global >2.2	4	
Regional >2.2	4	
Valve damage	1	TV rupture
Tissue	1	Intramural haemorrhage

Table 4. Performance of tests at predicting MACE during hospital stay.

Investigation	With MACE N=5 Sensitivity		Without MACE N=16 Specificity	
	Number (%)	95% CI	Number (%)	95% CI
CMR	3/5 (60)	15-95	13/16 (81)	54-96
Echocardiogram	1/4 (25)	1-81	10/13 (77)	46-95
Abnormal Troponin	5/5 (100)	48-100	3/14 (22)	5-51
Troponin Peak>1.0*	2/5 (40)	5-85	8/14 (57)	29-82
EKG	4/5 (80)	28-100	9/16 (69)	30-80

*This level has previously been proposed as a cut off (17)

Table 5. 9 month follow-up scans

	EF (%)		Regional STIR		RWMA		SCAR	
	Initial	9 months	Initial	9 months	Initial	9 months	Initial	9 months
Patient 1	67	-	3.27	3.0	Yes	Yes	No	Yes
Patient 2	65	61	1.97	1.69	Yes	Yes	No	Yes
Patient 3	57	48	1.61	2.3	No	No	No	No
Patient 4	63	69	2.50	1.6	Yes	Yes	No	Yes

Footnote: Patient 5 and 6 did not undergo follow up CMR. Baseline characteristics for patient 5 and 6 includes EF of 57% and 52%, Regional STIR 2.4 and 3.3, RWMA No and Yes, and Scar No and No respectively.