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Myocardial infarction in intensive care units: A systematic review of diagnosis and treatment

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Abstract

Introduction: Patients in the intensive care unit are vulnerable to myocardial injury from a variety of causes, both ischaemic and non-ischaemic. It is challenging for ICU clinicians to apply the conventional guidance concerning diagnosis and treatment. We conducted this review to examine the evidence concerning diagnosis and treatment of myocardial infarction in the ICU.

Methods: A systematic review was performed to identify relevant studies.

Results: 19 studies concerning use of ECG, cardiac enzymes, echocardiography and angiography were identified. 4 studies considered treatment of myocardial infarction.

Conclusions: Regular 12 lead ECG or 12 lead ECG monitoring is more sensitive than 2 lead monitoring, regular measurement of cardiac enzymes is more sensitive than when provoked by symptoms. Coronary angiography rarely identifies treatable lesions, without regional wall motion abnormality on echocardiography. Evidence relating to treatment was limited. A potential strategy to diagnose myocardial infarctions in the ICU is proposed.

Keywords

Myocardial infarcation, myocardial ischemia, critical care, troponin, electrocardiography

Introduction

Myocardial infarction (MI) is defined as myocardial cell death due to prolonged ischaemia.[1] This may be identified under situations outlined in Box 1.

MI is classified into five types: Type 1 – MI with intraluminal thrombus; Type 2 – where a condition other than coronary artery disease (such as arrhythmia or anaemia) contributes to an imbalance between myocardial oxygen supply and demand; Type 3 where MI results in death before biomarker values are available. Type 4 and 5 relate to PCI and CABG.

Cardiac troponins (cTn) are regulatory proteins of the cardiac contractile mechanism, with subtypes cTnT and cTnI.[2] Creatine kinase isoenzymes are phosphate carriers in myocytes with the –MB isoenzyme being most abundant in the heart.[3] However, as noted in the 3rd Universal definition, owing to low specificity of CK isoenzymes, cTn is the preferred biomarker.[1] A cTn increase greater than the 99th percentile of the upper limit of normal is defined as positive and the value will depend on the assay used.[1]

Non-ischaemic conditions may also cause a cTn rise and should be classed more broadly as myocardial injury. Examples include: purely non-ischaemic conditions such as cardiac contusions, rhabdomyolysis, myocarditis or cardiotoxic agents and pre-dominantly non-ischaemic conditions such as heart failure, renal failure, arrhythmia and pulmonary embolism.[1] Therefore any cTn increase should be interpreted considering the pre-test probability of myocardial ischaemia.

Myocardial injury is a common event within an intensive care unit (ICU).[4,5] In systematic review of 20 studies of patients who had a cTn measured during their ICU stay, a median frequency of cTn elevation of 43% was reported, which was associated with increased risk of death.[6] A review from 2012 summarised evidence showing that an elevation in cTn

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Box 1. Third universal definition of myocardial infarction.[1]

Detection of a rise and/or fall of cardiac biomarkers (preferably cardiac troponin – cTn) with at least one value above the 99th percentile upper reference limit and with at least one of the following:
- Symptoms of ischaemia.
- New or presumed new ST segment – T wave changes, or new left bundle branch block.
- Development of pathological Q waves on the ECG.
- Imaging evidence of new loss of viable myocardium or new RWMA.
- Identification of an intra-coronary thrombus by angiography or autopsy.

Box 2. ESC guidelines for treatment of NSTEMI.

Pharmacological treatment of ischaemia
- Oxygen where SpO2 <90%.
- Sublingual or IV nitrates to relieve angina.
- Early beta blockade.
- Aspirin.
- A $P2Y_{12}$ inhibitor (e.g. ticagrelor).
- Parenteral anti-coagulation (e.g. fondaparinux) at the time of diagnosis.

Requirement for angiography in discussion with a cardiologist with timing according to:
- Haemodynamic stability.
- Ongoing angina.
- Arrythmia or cardiac arrest.
- Acute heart failure.
- ECG and cTn changes.
- Comorbidities.

in ICU patients is associated with an increase in mortality.[7]

The distinction between myocardial injury and MI presents a challenge in ICU and the universal definition authors acknowledge that the development of ischaemia may not be apparent in critically ill patients.[1] Patients in ICU may not be able to complain of ischaemic symptoms; electrocardiogram (ECG) changes may not be observed; the rise in biomarkers may be due to non-ischaemic causes; a regional wall motion abnormality (RWMA) on echocardiography or angiography changes will only be identified in the case of either routine surveillance, or the investigation being prompted by clinical suspicion. In the absence of chest pain, factors such as hypotension, arrhythmias or evidence of heart failure may be the prompts to search for evidence of ischaemia.[8,9]

The authors of the universal definition note that the relationship between underlying coronary artery disease and MI is less secure in the ICU setting than a general population, and that careful clinical judgment is required to evaluate the coronary circulation.[10] For example, in one study, two intensivists were retrospectively asked to determine the most likely cause of cTn increase in 49 ICU patients. MI was suggested in 26; type 1 MI in 10, and type 2 MI in 16. The remaining 23 were judged to have had other non-ischaemic causes of cTn elevation.[11]

The significance of MI within ICU was demonstrated by a review of post-mortem examinations performed on ICU patients which showed the most frequently missed diagnosis likely to impact outcome was MI.[12]

Furthermore, the authors of the universal definition note that it 'may be a challenge for the clinician, caring for a critically ill patient . . . to decide on a plan of action when the patient has elevated cTn values'.[1] European Society of Cardiology (ESC) consensus management guidelines for STEMI and NSTEMI (see Box 2 for NSTEMI guidance) are written in the context of patients presenting to hospital with MI.[13,14]

The NICE guidance on diagnosis of acute coronary syndromes[15] is also based on the universal definition of MI. Equally, the NICE management guidance on NSTEMI[16] and STEMI[17] relates to those presenting with MI. The precise role of scoring systems such as Global Registry of Acute Coronary Events (GRACE) remains unclear in the ITU setting, as it was validated in patients presenting to hospital with acute coronary events.[18]

In addition, decisions regarding treatment in critically ill patients are complicated by conditions which increase the potential of iatrogenic harm from treatment: recent surgery and the presence of coagulopathy and thrombocytopenia may limit use of antiplatelets (both pre and post coronary intervention); cardiovascular instability may preclude the use of nitrates and beta blockade.

The difficulty in making treatment decisions in this patient group is further demonstrated by the variety of intensivists practice when managing patients with elevated cTn, but without symptoms of MI or ECG changes.[19] To our knowledge, there are no guidelines regarding the management of MI in ICU.

In view of the above uncertainties, we performed a systematic review of the available literature considering two questions:

When the clinician suspects an ICU patient is undergoing an MI, what diagnostic tests are most helpful?
When an ICU patient is diagnosed with MI, what treatment is most appropriate?

Methods

We searched EMBASE, PUBMED and Cochrane databases using the MeSH terms in Box 3 from title, abstract or full text.

Box 3. Search terms used.

Pathological terms
- Myocardial infarct.
- Myocardial ischaemia.
- Heart muscle ischaemia.
- Heart infarction.

Location terms
- Critical illness.
- Intensive care units.
- Critical care.
- ICU.

Diagnosis or treatment terms
- Myocardial infarction/diagnosis.
- Myocardial ischaemia/diagnosis.
- Myocardial infarction/therapy.
- Myocardial ischaemia/therapy.
- Emergency treatment.
- Medication therapy/management.

The search was limited to those studies in English and concerning patients over 16 years old. This identified 2811 potential papers (Figure 1). The titles were reviewed by one author (IC) according to the following criteria:

Was the study population relevant to our question?
Did it concern adult patients?
Did the study concern MI acquired within an ICU?
Did the study consider the diagnosis or treatment of MI?

After elimination of duplicates, a total of 96 papers were identified. We reviewed references and contacted prominent authors regarding recently published articles or those pending publication, identifying a further 17 abstracts.

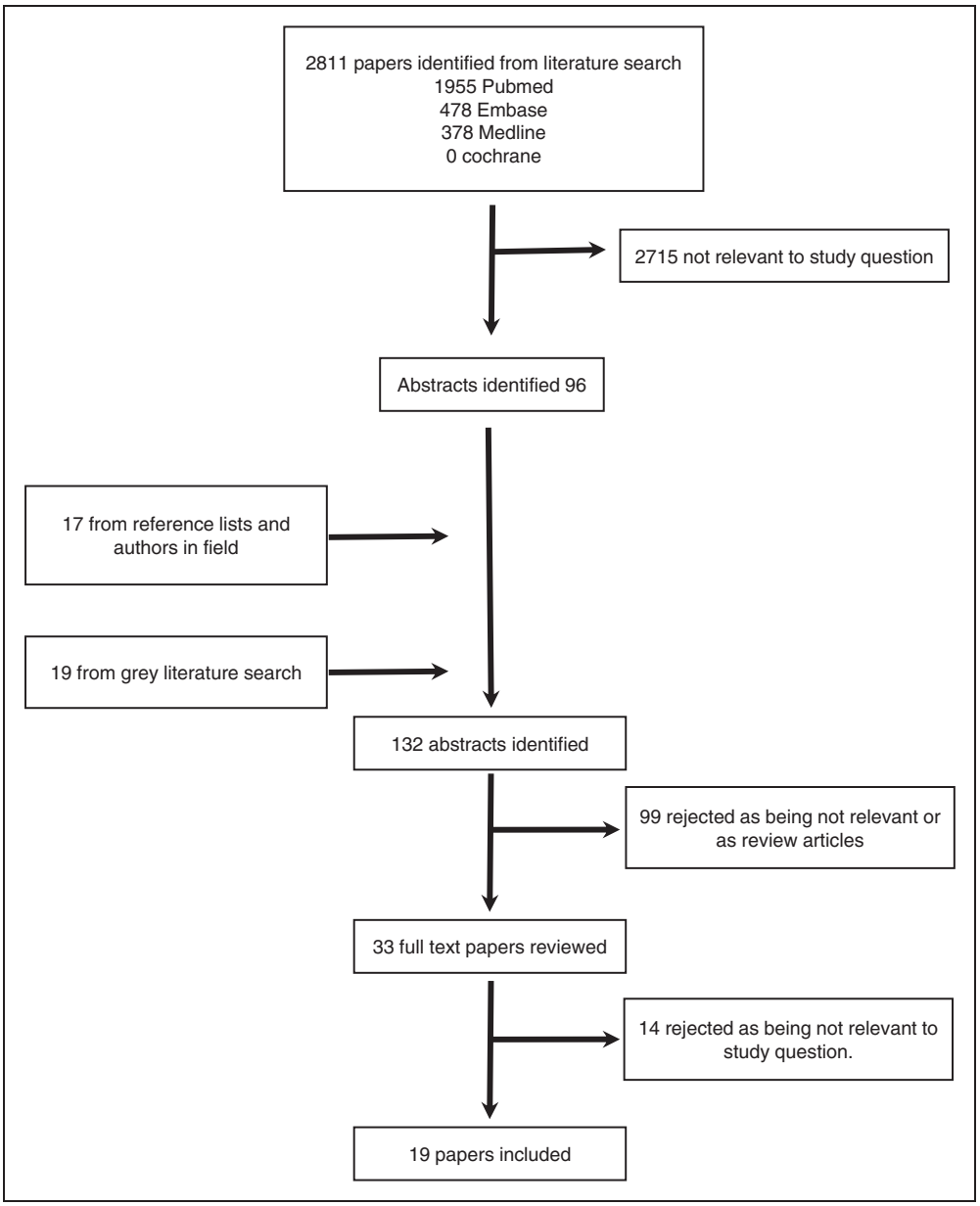

Figure 1. Flow chart of papers reviewed.

Box 4. Categories of papers chosen.

(a) Three papers considered the ability of ECG monitoring to diagnose MI.
(b) Eight papers compared the relationship of cardiac enzymes to clinical (symptom and ECG) findings.
(c) Three papers considered the use of echocardiography in diagnosis.
(d) Three considered the role of angiography in diagnosis.
(e) Two considered autopsy findings.
(f) Four considered treatment modalities available.

A grey literature search for unpublished abstracts and conference proceedings using an internet search engine identified 19 abstracts.

These abstracts were reviewed by IC and TM. The criteria above were reapplied, and review articles, letters and comments excluded. In cases of disagreement, the full text paper was included; 33 papers were identified.

On consideration of full texts, eight reviews and four non-relevant papers were rejected; 19 papers were therefore considered fully. They are categorised in Box 4, with some featuring in more than one category.

Discussion

Nineteen papers were included, which presented highly variable evidence. They are outlined in Table 1. Some studies included an unselected ICU population, while others concerned patients with only one pathology.

The diagnostic studies considered presented evidence for a range of different tests; ECG monitoring with a variety of lead combinations, interval 12-lead ECGs; echocardiography; functional cardiac studies and cardiac enzyme measurements.

In the summary below, the pertinent conclusions are highlighted.

Evidence concerning the use of ECG

12-lead ECGs have been shown to have several limitations.

There was limited agreement on ECG findings on separate readings by the same doctor and between experienced doctors interpreting ECGs, but agreement increased when further clinical information, such as results of cTn assays were presented.[20] This suggests that the role of single ECG in diagnosis is undermined by the difficulty in agreement between clinicians. Automated ECG interpretation software of 12-lead ECGs showed a significant false positive rate.[21]

Two-lead ECG monitoring with daily 12-lead ECGs was shown to have a low sensitivity of 12%, but a specificity of 98% for detecting the first episode of ischaemia when compared to 2 minutely 12-lead ECG in post-operative vascular patients.[22]

Evidence concerning the use of cardiac enzymes

Studies in this group can be divided into two categories:

1. Those that compared incidence of cTn rise with incidence of ECG abnormality and clinical findings.[23–25] In these studies, patients were identified who had a cTn rise which was not detected in routine care and who lacked other features of myocardial ischaemia.

If an enzyme rise is documented, without corroboration from other factors, then there are two diagnostic possibilities:

   a. The situation is a non-ischaemic myocardial injury.
   b. There has been a MI (Type 1 or 2), but either the tests lacked sensitivity, or were not performed at an appropriate time.

2. Those that compared standard ICU surveillance with a form of 'enhanced surveillance'. Consequently, it was possible to reach additional diagnoses of MI. This was achieved by the following methods:

Continuous 12-lead ECG surveillance. This also showed the rarity of clinical symptoms as only 2 out of 37 episodes were associated with clinical symptoms.[8]
Continuous 12-lead ECG surveillance and daily cTn measurement.[26]
Serial repetition of 12-lead ECG and cTn.[27]
Focused clinical assessment and daily cTn measurement.[9]

The results of these studies are shown in more detail in Table 1 and may reflect the lack of sensitivity of routine surveillance, or that the elevation of cTn reflected non-ischaemic myocardial injury. In the context of the first group of studies, it is likely that a significant proportion of the patients had 'missed' myocardial ischaemia, where the ECG was not recorded at an appropriate time.

In a separate style of study, Lim et al.[28] noted how the agreement between four experienced intensivists in reaching a diagnosis of MI when presented with ECG and cTn details varied extensively from slight to substantial, according to the which two of the doctors were compared.

Papers considering the use of echocardiography or angiography in diagnosis

Three of the papers in this category[29–31] identified that the greater the increase in the serum cTn, the more likely there were to be echocardiographic abnormalities detected.

Table 1. Summary of included studies

Question addressed	Paper	Key question addressed	Style of design	Number of patients	Type of unit	Exclusions	Key outcomes
Use of ECG in diagnosis of myocardial infarction in ICU	Lim et al.[20]	Reliability of ECG interpretation. Study investigated comparisons between the same doctors on two occasions, and agreement between two doctors.	Prospective cohort	114	Medical/surgical		Intraobserver reliability noted as fair to moderate Interobserver reliability slight when blinded to cTn values, moderate when aware of cTn. The greater the cTn rise, the greater the likelihood of the ECGs being interpreted as ischaemic
	Rennyson et al.[21]	Ability of automated ECG interpretation software to diagnose STEMI	Retrospective cohort	46	Medical/surgical	Patients admitted with ACS. Patients who did not have two cTn assays measured	An expert only agreed with a positive diagnosis of STEMI in 18/46 cases, of these only 6 had a significant cTn rise. An expert disagreed with the interpretation in 28 cases, of which only 1 had a significant cTn rise.
	Martinez et al.[22]	Comparison of routine ICU monitoring (clinical and 2-lead ECG monitoring with ST segment alarm) in detecting myocardial ischaemia when compared with gold standard of 2 min 12-lead ECG	Prospective cohort	149	Post infra-inguinal or aortic vascular surgery	Pre-op LBBB, PPM, contraindication to B blockade or EF <20%. Age less than 40.	Routine surveillance had 12% sensitivity and 98% specificity of detecting initial ischaemia when compared to the gold standard
Use of cardiac enzymes in diagnosis of myocardial infarction in the intensive care unit.	Andrews et al.[23]	Comparison of frequency of MI diagnosis made by CK, CKMB and ECG changes to cTnI elevation.	Prospective observational	99	Elective/emergency vascular surgical cases	One non-operative case	Twelve patients had an MI as identified by classical criteria. Another five had a cTn rise and retroscpectively identified as having features suggestive of MI. Considering cTn to be the gold standard for diagnosis of MI, the sensitivity of CKMB was 55%, specificity 92.7%, sensitivity of ECG

(continued)

Table 1. Continued

Question addressed	Paper	Key question addressed	Style of design	Number of patients	Type of unit	Exclusions	Key outcomes
							changes was 61% with specificity 76.8%
	Arlati et al.[24]	Did patients admitted to ICU with sepsis, septic shock or hypovolaemic shock have undiagnosed episodes of myocardial infarction	Prospective observational	31	Mixed medical/surgical	Those admitted with features of cardiac illness	ECG abnormalities were noted in five patients, but rise in CKMB in 21, CK in 23, and cTnI in 23 patients. Median level of the cardiac markers increased with duration hypotension.
	Klein Gunnewiek and van de Leur[25]	Comparison of frequency of cTn elevation with ECG findings	Prospective observational	34	Thoracic/vascular	None	Eleven patients had a cTn rise. Four of these had ECG changes diagnostic of MI; three non-specific ECG changes and three normal ECGs
	Booker et al.[8]	Comparison between ischaemia on continous 12-lead ECG monitoring and cTnI elevation	Prospective observational	76	Mixed medical/surgical	Non-English speakers, greater than 24 h admission, paced rhythm	8 of 76 patients had ischaemia detected on ECG screening, with a total of 37 'ischaemic episodes'. Of these 35 episodes were symptomless; 6 of the eight patients had a rise in cTnI. A further six patients had a rise in cTnI without ECG ischaemia being noted.
	Landesberg et al.[26]	Relationship between myocardial ischaemia as detected by routine clinical care and enhanced care with continuous 12-lead ECG monitoring, and daily cTn measurement	Prospective observational	101	Mixed	Patients without known CAD or risk factors.	Myocardial infarction was clinically suspected in only four patients; 21 patients had ECG evidence of ischaemic episodes, of whom 14 had cTn elevation. Greater than 60 min ischaemia had a sensitivity of 31.6% and specificity of 95.2% for detecting a cTn increase.
	Lim et al.[27]	Comparison of frequency of MI diagnosis made by standard care and	Prospective observational	103	Mixed medical/surgical	None	Standard care identified 18 patients as having had an MI, of which 4 were judged false

(continued)

Table 1. Continued

Question addressed	Paper	Key question addressed	Style of design	Number of patients	Type of unit	Exclusions	Key outcomes
		standard care plus serial cTn and ECG measurement.					positives. A further 23 diagnoses were made with the enhanced care. MI associated with increased mortality and increased use of anti-platelet, coagulation modifiers, β blockers and diuretics. Making a diagnosis had no impact on mortality.
	Guest et al.[9]	Comparison of frequency MI diagnosis made by standard care (CKMB and ECG as required by clinical team) and standard care plus daily cTnI measurement	Prospective observational	209	Medical/respiratory ICU	Admissions due to lack of other beds. Inability to measure daily blood samples	In standard care, 16 diagnoses of MI were made. Four of these were judged false positives by the lack of cTnI elevation. A further 20 cases were identified in the enhanced care group. Combined ECG and CKMB sensitivity was quoted at 37.5%, specificity 97.7%. Patients with clinically undiagnosed injury tended to be younger and have less significant rises in cTnI. Those with cTnI increase had greater mortality
	Lim et al.[28]	Agreement between clinicians when interpreting ECG and cTn results without clinical information	Prospective observational	45	Medical/surgical	Those who did not consent	Variable agreement between assessors, ranked from slight to substantial
Use of echocardiography in diagnosis	Ver Elst et al.[29]	To compare cTnI concentrations with TEE findings.	Prospective observational	46	Early sepsis in a general ICU	Recent cardiothoracic event, immunosuppression, irreversible medical conditions	Raised cTnT, cTnI and CK-MB in 50%, 36% and 41% of patients, respectively, all of which were strongly associated with each other. Despite no patients having evidence of ECG changes, a highly significant association was found between cTnI and LV

(continued)

Table 1. Continued

Question addressed	Paper	Key question addressed	Style of design	Number of patients	Type of unit	Exclusions	Key outcomes
							dysfunction. In the 21 non-survivors, 12 autopsies were performed. One patient had evidence of LV rupture, and one of acute myocardial infarction. In 10 other patients, no evidence of myocardial infarction was seen.
	Relos et al.[30]	Association between cTn increase, ECG or echocardiography abnormality with diagnosis of MI and mortality	Retrospective observational	869	Post-surgical	Those who did not have a cTn measured	Incidence of ECG abnormality, echocardiography abnormality, and mortality increased with magnitude of cTnI elevation. The use of B blockers and aspirin was associated with improved survival, in patients where cTnI was most significantly elevated.
	Ko et al.[31]	Contribution of coronary arterial disease to ischaemic events within ICU	Retrospective observational	56	Medical	Those with a background of coronary arterial disease.	Fifty-six ICU patients had had an angiogram due to suspicion of MI while ICU inpatients. Of these only 18 had significant coronary arterial disease and 9 required treatment to a culprit lesion.
	Ruiz Bailen et al.[32]	To investigate echocardiographic abnormalities in critical care patients and relationship to angiography findings.	Prospective observational	574	Medical/surgical	Those with background of cardiovascular disease or associations	Patients underwent serial echocardiography due to a variety of clinical indications; 33 had evidence of myocardial dysfunction, all of which normalised over the first month. None had angina. Coronary angiography was performed in seven patients, all of which were normal.
	Ammann et al.[33]	Relationship between elevated cardiac cTn results in ICU and angiographic findings.	Prospective observational	58	Medical	ACS, MI, recent cardiac surgery, admission	Thirty-two patients had an elevated cTn; 23 of these were shown to have no associated coronary artery disease (by

(continued)

Table 1. Continued

Question addressed	Paper	Key question addressed	Style of design	Number of patients	Type of unit	Exclusions	Key outcomes
						expected to be less than 24 h	either DSE, angiography or autopsy); 2 had autopsy evidence of CAD and 7 were not investigated. LVEF was inversely associated with cTn elevation.
	Ko et al.[31]	See above					
Use of autopsy findings	Berlot et al.[34]	Comparison of clinical and autopsy diagnosis of myocardial infarction in intensive care	Retrospective observational	600	All non-trauma/ paediatric/ obstetric	None	Seventy-five patients had autopsy evidence of recent acute myocardial infarction, of whom 25 had evidence of CAD and 50 did not. A clinical diagnosis had only be made in 11 cases. A further eight patients were clinically suspected of MI, but had no post-mortem evidence.
	Ver Elst et al.[29]	See above					
Treatment modalities available	Hébert et al.[35]	Effect of liberal vs restrictive transfusion strategy in critical care patients.	Non-blinded randomised trial	357 patients	General ICU	Initial Hb <9.0 g/dl, age <16, refusal of transfusion.	No difference, in mortality between restrictive and liberal groups, when either those with any cardiovascular disease or the subset with ischaemic heart disease alone was considered.
	Lim et al.[36]	Cohort study investigating incidence of new MI, or isolated cTnI rise during ICU admission	Prospective observational	198	Medical/surgical	Trauma, neuro, orthopaedic, cardiac surgery, pregnancy, palliative care.	Seven patients had a new MI during the admission, a further two had cTnI rise without ECG changes. The use of anti-ischaemic or thrombotic treatment did not impact mortality.
	Relos et al.[30]	See above					
	Lim et al.[26]	See above					

In the ver Elst study of patients with septic shock, an association was shown between the magnitude of elevation of cTn elevation and evidence of left ventricular (LV) dysfunction on trans-oesophageal echocardiography. Additionally, cTnI was found to be more sensitive than cTnT; seven subjects who were cTnI positive remained cTnT negative.[29]

Relos showed an increased incidence of ECG change, echocardiographic abnormality, and mortality with greater cTnI increase in 869 post-surgical patients.[30]

Ko et al.[31] identified 56 patients who had had an angiogram performed for investigation of coronary artery disease while on ICU; 18 out of 56 patients were identified as having coronary artery disease, of whom 9 required interventional treatment. Angiographic abnormalities requiring treatment were observed in only 9 out of 56 studies performed and were more common if patients had RWMA on transthoracic echocardiography.

In the Ruiz Bailen study, 574 patients who had an echocardiogram performed in ICU were identified; 33 had myocardial dysfunction, primarily in the basal and apical segments. These were not associated with other evidence of ischaemia and the changes all normalized during follow-up interval echocardiography. Seven of the patients proceeded to angiography, which was normal in each case.[32]

Ammann et al.[33] studied 58 patients admitted to a medical ICU with sepsis, SIRS, or other severe disease (mean Simplified Acute Physiology Score II of 42); 32 had raised cTn (I or T isoform) but only two had evidence of coronary arterial disease; 23 were shown to have no coronary arterial disease (by either dobutamine stress echocardiography, coronary angiography or, in six cases by autopsy), seven were untested, and two had evidence of coronary artery disease on autopsy. Ethical considerations had not permitted the investigators to submit every patient to coronary angiography.

It may be concluded that the utility of angiography for patients who have had a diagnosis of MI while on ICU is questionable, with a relatively limited probability of identifying any lesions which are remedial to treatment. This should be considered in the context of the practical difficulties in arranging such tests for ICU inpatients, as well the possibility of the abnormalities which prompted angiography normalising over time.

Autopsy findings

These studies demonstrate the discrepancy between clinical suspicion of MI and pathological diagnoses.

Berlot et al.[34] investigated the 600 autopsies performed on ICU patients in a single unit over a 10-year period. Evidence of MI was found in 75 cases; 25 patients had evidence of coronary artery disease and 50 did not and may therefore be concluded to represent type 2 MI. Of the 75 cases, 55 had been investigated clinically, but a diagnosis of MI was clinically confirmed in only 11. Additionally, during the time period, eight patients were clinically suspected of having suffered an MI, but no post-mortem evidence was identified.

In the ver Elst study of 46 patients with septic shock, autopsies were performed on 12 patients, seven of whom were cTn positive. One cTn positive patient had evidence of MI, while a cTn negative patient had an LV free wall rupture. In the 10 other post-mortems performed, there was no evidence of MI or inflammatory infiltrate.[29]

Treatment modalities available

A subgroup analysis of the TRICC study showed no difference in mortality of 357 patients with cardiovascular disease, or the subset of 257 patients with acute or chronic coronary arterial disease, when randomised to either a restrictive Hb (7–9 g/dl) or liberal (10–12 g/dl) transfusion strategy. This was the only randomised study included.[35]

There was no other convincing evidence identified regarding treatments for patients having suffered MI on ICU.

The Lim 2006 cohort identified seven patients who had an MI during the ICU stay. Patients were managed according to the judgement of the treating physician. No difference in mortality was noted according to whether the patient had received anti-platelet, anticoagulant treatment, β blockade or nitrates. However, the use of vasopressors and inotropes was an independent predictor of ICU mortality.[36]

A separate cohort study by the same author identified that patients identified as having an MI were more likely to receive conventional treatment for MI. However, there was no difference in mortality between those who were identified with ischaemic disease and those who were not.[27]

Conversely, Relos's retrospective cohort of 869 patients considered the impact of the treatment used in patients suspected of having had an MI. They found that where cTn rise was greatest, the use of B blockers and aspirin were associated with decreased hospital mortality.[30]

Conclusion

In reviewing the evidence regarding diagnosis of MI in intensive care, it is clear that a different strategy is required than in patients presenting with chest pain. Patients rarely present with symptoms of ischaemia[8,9,31] and when tests only are used in response to symptoms, signs or clinical suspicion, diagnosis of MI is frequently missed.[8,9,26,27]

Single 12-lead ECGs were shown to have a limited role owing to lack of sensitivity and lack of agreement both between different interpreters, and the same interpreter on different occasions. Regular 12-lead ECGs have improved sensitivity to 2-lead monitoring.

Box 5. Suggested strategy for monitoring and diagnosis of MI.

Monitoring on leads V2, V3 or V4
Daily 12-lead ECG or 12-lead monitoring.
Avoidance of using automated ECG interpretation.
Daily troponin measurement in all patients.
Evaluation of ECG by two clinicians in conjunction with enzyme results.
Use of echocardiography to identify RWMA, especially where the troponin increase is of greater magnitude.
If there are no echocardiographic abnormalities, angiography is unlikely to reveal a treatable lesion.

It is possible to increase the sensitivity of regular monitoring for detecting MI used in many ICUs (e.g. clinical signs, 2-lead ECG monitoring, 12-lead ECG on demand and cardiac enzymes as prompted by suspicion of ischaemia) by several methods: continuous 12-lead ECG surveillance; regular 12-lead ECG and regular cTn measurement unprompted by clinical suspicion.

Echocardiographic abnormalities were more common the greater the increase in cTn, and in one study were associated with increased likelihood of angiographic abnormality. However, lesions requiring interventional treatment remained unusual.

Therefore, a potential strategy to diagnose MI in the ICU setting is outlined in Box 5.

Regarding treatment, when a diagnosis of MI is made in an ICU patient, it remains unclear whether the conventional anti-platelet, anti-anginal and interventional treatments (see Box 2) are appropriate or beneficial. In cohort studies, there has been no evidence of reduced mortality in patients who were treated with conventional anti-ischaemic regimes compared to those who were not. However, we were not able to identify any randomised trials conducted in this area, and it remains an area for future research opportunities. No study in intensive care has considered the relative benefit of treatment in type 1 or 2 MI. Early liaison with cardiologists and admitting teams with careful consideration of likelihood of test results representing myocardial ischaemia and risks and benefits of treatment to the individual patient will remain critical in this area.

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