Southern Health and Social Care Trust

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# Guidelines

# for the management of

# acute chest pain

# of cardiac origin

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# Table of contents:

## 1. Abbreviations:

| | |
|---|---|
| ACS | Acute Coronary Syndrome |
| AF | Atrial fibrillation |
| BNF | British National Formulary |
| CABG | Coronary artery bypass grafting |
| CTCS | CT calcium score |
| CTCA | CT coronary angiography |
| CXR | Chest X-ray |
| COW | Cardiologist of the Week |
| ECG | Electrocardiogram |
| Hs-TnT | High sensitivity troponin T |
| ICH | Intracranial Haemorrhage |
| LVEF | Left Ventricular Ejection Fraction |
| LBBB | Left Bundle Branch Block |
| MI | Myocardial Infarction |
| MINAP | Myocardial Ischaemia National Audit Project |
| MPI | Myocardial perfusion scan |
| NSTEMI | Non ST elevation Myocardial Infarction |
| PCI | Percutaneous Coronary Intervention |
| PDE5 | Phosphodiesterase type 5 inhibitor |
| PLATO | Study of Platelet Inhibition and Patient Outcomes |
| SAH | Sub arachnoid haemorrhage |
| SHSCT | Southern Health and Social Care Trust |
| STEMI | ST elevation Myocardial Infarction |
| UA | Unstable Angina |

**These guidelines are based predominantly on:**

ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation of the European Society of Cardiology (ESC). European Heart Journal (2020) 00, 1-79.

ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). European Heart Journal (2018) 39, 119–177.

**These guidelines are a summary of the current guidelines and consensus documents of the ESC and are intended for internal use within the SHSCT only.**

**(Amended Sept 2020 due to new ESC guidelines)**

## 2. Introduction

Acute Coronary Syndrome (ACS) describes the combination of signs and symptoms compatible with acute myocardial ischemia including chest pain, chest discomfort / pressure, dizziness, light-headedness, shortness of breath and sweating. The ACS clinical spectrum includes unstable angina (UA), non ST-segment elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction (STEMI).

Disruption of atheromatous plaque is the pathophysiologic basis of ACS. Following plaque rupture and the initiation of thrombotic cascade, myocardial ischaemia and injury sets in and lead to differing clinical forms of ACS. ACS with the presence of myocyte necrosis characterises myocardial infarction. At SHSCHT we currently employ high sensitivity troponin assay to detect myocardial infarction. ACS with no evidence of myocardial injury constitutes the clinical spectrum of UA. Patients with myocardial infarction (MI) are further classified into STEMI and NSTEMI based on the presence or not of persistent ST segment elevation on electrocardiogram (ECG).

The umbrella term “acute coronary syndrome” is useful in that it groups patients with symptoms consistent with acute myocardial ischemia and is the basis for subsequent established diagnostic and treatment decisions

Our aim is to treat all STEMI patients by primary percutaneous coronary intervention (pPCI) transfer to RVH with a door to balloon time of < 90 minutes, as all the evidence points to maximal benefit of pPCI with early revascularisation. We also aim to perform invasive coronary angiography +/- PCI at CAH in all appropriate ACS patients within 72 - 96 hours of admission to hospital in accordance with the national guidelines.

If untreated, the prognosis is poor and mortality high, particularly in people who have had myocardial damage. Appropriate triage, risk assessment and timely use of acute pharmacological or invasive interventions are critical for the prevention of future adverse cardiovascular events (MI, stroke, repeat revascularisation or death).

People who have had an ACS benefit from treatment to reduce the risk of further MI or other manifestations of vascular disease. This is known as secondary prevention.

The following pathway should be implemented for patients with chest pain which is suspected to be due to acute cardiac ischaemia.

## 3. Purpose of this policy

Chest pain is a very common symptom leading to assessment of patients in the emergency department and/or acute medical unit. ACS typically presents with chest pain or discomfort. Assessment of these patients with acute chest pain to identify ACS should include clinical evaluation, 12 lead ECG and serial measurement of markers of myocardial injury (currently by high sensitivity troponin at SHSCT). Prompt pharmacological therapy and coronary intervention is the mainstay of treatment in this group of patients to minimise associated mortality and morbidity. Further long term evidence based drug therapy reduces future cardiovascular morbidity.

This policy aims to assist the attending health care professionals in treating patients with ACS with particular emphasis on immediate pharmacotherapy, risk assessment for urgent coronary angiography, secondary prevention, cardiac rehabilitation and post MI health and lifestyle advice. It is also designed to enable an early `rule out` of an ACS in low risk patients to facilitate early discharge from hospital within four hours of their presentation.

## 4. Scope

This document provides guidance for any professional involved in the clinical management of patients, presenting to either primary or secondary care with chest pain due to suspected or proven ACS. This will include:

- General practitioners
- Specialist nurses
- Junior doctors
- SpRs
- SAS doctors
- Consultants

## 5. Acute coronary syndrome (ACS)

### 5.1. Clinical Classification of MI

After we have documented a significant acute increase in troponin with at least one other criterion for the diagnosis of acute coronary syndrome (ACS) such as symptoms, ECG changes or new regional wall motion abnormality, we still need to determine what type of MI has occurred. Mostly, the question is whether the diagnosis is a type 1 or 2 MI. However, there are also type 3, 4 and 5 MIs**.**

- A type 1 MI is due to a primary coronary problem with plaque rupture, fissuring or dissection causing thrombosis and obstruction to flow resulting in infarction in the territory supplied by the coronary artery i.e. the classical mechanism for a MI.

- Type 2 infarction occurs where there is an imbalance between supply and demand. Common causes include hypotension, arrhythmias and anaemia. Often there are several factors at play.

- Type 3 is sudden death due to MI, type 4 is related to PCI and type 5 is related to CABG.

- Sometimes it can be very difficult, if not impossible to distinguish between a type I or type II MI.

**5.2 History**

Consider the history of the pain, any cardiovascular risk factors, history of ischaemic heart disease and any previous treatment, and previous investigations for chest pain

Symptoms that may indicate ACS include:

- Pain or discomfort in the chest and/or other areas (e.g. the arms, back, neck or jaw) lasting longer than 15 minutes.
- Chest pain with nausea, vomiting, marked sweating and/or breathlessness, or haemodynamic instability.
- New-onset chest pain or abrupt deterioration of stable angina, with recurrent pain occurring frequently with little or no exertion and often lasting longer than 15 minutes.

Chest pain or discomfort is a common presenting complaint in emergency and acute medicine. Although the majority of patients presenting with chest pain or discomfort will not have a serious medical condition, a significant proportion will. Therein lies the dilemma of the acute clinician. There is a desire to avoid inappropriate admission, but anxiety over missing a potential life-threatening diagnosis such as acute MI, pulmonary embolism (PE) or aortic dissection. Thus, the rapid exclusion of a serious medical condition is of paramount importance.

Typically, the pain or discomfort associated with a MI is a retrosternal pressure or ache, but this is not always the case. The discomfort may radiate to the arm(s), back, neck, jaw, teeth or abdomen.

Although patients are commonly asked to rate their pain out of ten, the severity of the pain is not overly helpful in diagnosis. In fact MI can present "silently" with just dyspnoea or autonomic symptoms. This is particularly the case in elderly or diabetic patients. Often a patient with acute MI will manifest autonomic symptoms such as sweating, pallor, nausea and/or vomiting.

The medical history is often helpful in guiding the clinician to the likely cause of chest pain. Clearly, one should have a high index of suspicion in patients with a known history of MI or coronary artery disease (angina, previous coronary artery bypass grafting (CABG), previous PCI etc.) or risk factors for coronary artery disease (age, smoking, diabetes mellitus, hyperlipidaemia, hypertension, family history, recent use of cocaine/similar etc). On the other hand, it may be relatively easy to exclude the diagnosis in patients with few or no risk factors and a likely alternative diagnosis (indigestion, oesophageal spasm, musculoskeletal pain, pericarditis, PE, pneumonia, aortic dissection, psychiatric disorder etc.). Table one highlights different causes of chest pain.

The clinical history, cardiac risk factors and ECG abnormalities can be summarised by the HEART score and Grace score, see appendix 2a / 2b, page 23-24.

**Table 1: Other common and/or important causes of chest pain**

| Possible cause of pain | History | On examination – Are these symptoms present? |
|---|---|---|
| Musculoskeletal Pain | Trauma<br>Over-exertion<br>Arthritis or other musculoskeletal disorder<br>Pleuritic | Tenderness<br>Worse on certain movements |
| PE | Any haemoptysis<br>Any risk for PE in the history – immobility, history of DVT/PE, thrombophilia recent surgery, malignancy etc.<br>Pleuritic (but may be retrosternal dull ache). | Low 02 sats<br>Tachypnoeic<br>Cyanosis |
| Pericarditis | Recent viral infection<br>Recent CABG<br>Previous pericarditis | |
| Chest Infection | Productive (or dry) cough, perhaps with coloured sputum.<br>Fever<br>COPD<br>Pleuritic | Low sats<br>Tachypnoeic<br>Cyanosed |
| Aortic dissection | Hypertensive<br>Tearing pain radiating to neck<br>Undertaking physical effort (e.g. lifting) when pain started<br>Pain on-going | Different BP in arms<br>Asymmetric pulse volumes<br>(e.g. carotids, femorals, radials) |

### 5.3 Electrocardiogram (ECG)

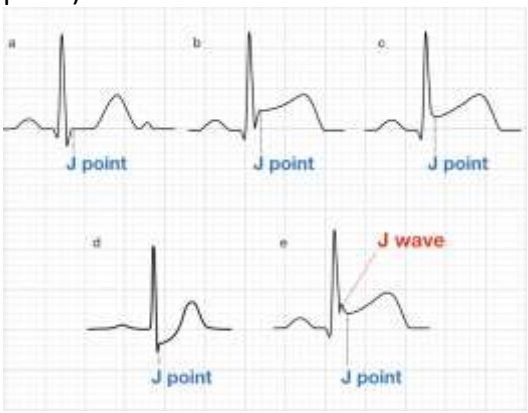
Apart from careful history taking and physical examination, the corner stone of the initial acute assessment of chest pain is the 12 lead ECG. If ST elevation from the isoelectric line to the J point (end of QRS complex) of ≥1mm in two or more contiguous limb leads or ≥2mm in two or more contiguous chest leads (see figure 1) is demonstrated then the pPCI pathway should be activated (see pPCI section).

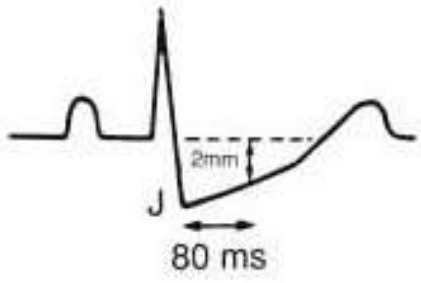
If there is ST depression in leads V1-V3 suggestive of an acute posterior MI then the pPCI pathway should also be activated (see pPCI section).

If there is new LBBB or an unstable NSTEMI then consideration should be given to discussing with the RVH cardiology on-call team.

Otherwise, new ST depression (ideally measured 80ms after the J point, see figure 1) or T wave inversion may be consistent with an acute MI. However, other conditions e.g. PE, dissection, pericarditis, atrial fibrillation (AF), sub-arachnoid haemorrhage (SAH) etc. can also cause acute ST abnormalities on the ECG and indeed the ECG can be normal in NSTEMI. This is where the use of a troponin assay to document acute myocardial damage is useful.

Figure 1: ECG abnormalities showing ST elevation (which should be measured at the J point) and ST depression (which should be measured 80ms [2 small squares] after J point).




**5.4 Highly sensitive troponin T (hsTnT)**

To make the diagnosis of an acute MI a significant alteration (usually an increase) in troponin levels is needed along with at least one of:

- Symptoms consistent with MI (usually chest discomfort due to cardiac ischaemia)
- ECG changes consistent with an acute MI
- New regional wall motion abnormalities consistent with an acute MI on echocardiogram /cardiac MR / myoview / LV ventriculogram

**<u>Six Things to remember about Troponin</u>**

- Do not delay diagnosis of a STEMI until biomarkers are available. The diagnosis of a STEMI is based upon ECG finding and clinical history. This is a medical emergency and requires prompt pPCI at the regional cardiology centre as soon as possible (see guidance on management of STEMI).

- High sensitivity troponin T (hsTnT) should be taken on presentation ($T_0$) and at one hour ($T_1$). A further sample is required at 3 hours ($T_3$) in certain circumstances, see appendix 3, page 25-26.

- The lack of significant change in troponin does not exclude unstable angina.

- There are many other causes of an acutely raised troponin. To make the diagnosis of an acute MI a history of ischaemic chest pain or new ECG abnormalities consistent with MI or new evidence of MI on an imaging test (e.g. echo) must also be present.

- Other causes of an acute troponin elevation include pericarditis, myopericarditis, sepsis, PE, COPD exacerbation, arrhythmia including AF, trauma, cardiac surgery or instrumentation, PCI, etc.

- A chronically elevated hsTnT is common. This is usually secondary to chronic myocardial injury. Causes include renal impairment, heart failure and valvular heart disease.

**6. Initial assessment and treatment of suspected ACS**

- This guideline applies only to patients whose history and clinical examination are suggestive of an ACS as the cause of their chest pain

- Initial assessment should include brief history, physical examination and 12 lead ECG. These are crucial.
- 12 lead ECG – ideally every 15 minutes until pain-free in high risk cases, then preferably at one hour and four hours after pain.
- Use the ECG for initial risk stratification: ST elevation myocardial infarction (*STEMI Immediately proceed to 6.1 and activate the pPCI pathway*).
- Blood pressure should be recorded in both arms.

**6.1. Initial assessment suggests STEMI**

- ST elevation > 1 mm in 2 or more contiguous limb leads or >2 mm in 2 or more chest leads or ST depression of at least 2mm in V1-V3 suggesting acute posterior MI
- **Activate Primary PCI Pathway (see appendix 4, page 27)** for STEMI management without delay.
- Aspirin 300mg

- Ticagrelor 180 mg orally stat unless contraindicated (clopidogrel 600mg if Ticagrelor contraindicated)

  ✓ **Contraindication to Ticagrelor:**

  - Hypersensitivity (e.g. angioedema)
  - History of intracranial haemorrhage (ICH)
  - Active pathologic bleeding (peptic ulcer, ICH)
  - Moderate-Severe hepatic impairment (probable increase in drug exposure)
  - Combination with strong CYP3A4 inhibitors such as Clarithromycin, Ritonavir, Azatanavir, Nefazodone, Ketoconazole

- Left bundle branch block (unless known to have LBBB previously) should be discussed with RVH cardiology on-call team as these don't strictly fall under pPCI remit any longer in Northern Ireland.

**All patients who are declined PPCI must be discussed with on-call cardiologist.**

### 6.2. Initial assessment suggest NSTEMI

- All patients with confirmed NSTEMI should be considered for invasive coronary angiography +/- PCI (percutaneous coronary intervention), where appropriate, taking co-morbidities and patient wishes into consideration.
- Aspirin 300mg
- Ticagrelor 180 mg orally stat unless contraindicated (if contraindicated please load with clopidogrel 600mg or 300mg if elderly / frail – if unsure discuss with a cardiologist).
- Enoxaparin
  - Usual dose 1mg/kg SC BD (max dose 100mg BD) for 3 days then 40mg s/c nocte until discharge.
  - If >75 yrs give 0.75mg/kg SC BD (max dose 75mg BD) for 3 days then 40mg s/c nocte until discharge.
  - If GFR is <30 reduce dosing frequency to OD.

**All unstable NSTEMI / UA patients with ongoing chest pain and / or dynamic ECG changes should be discussed with cardiologist on-call and referred to RVH on-call team for discussion regarding emergency angiography / PCI.**

### 6.3 Initial assessment is unclear if ACS

- **DO NOT load troponin negative patients / 'unstable angina' with DAPT unless agreed with cardiology team**
- Delay loading if diagnosis is uncertain

### 6.4 General management of ACS patients

- Cardiac monitoring
- IV access and blood samples – initial hsTnT, one hour hsTnT and 3 hour hsTnT (if indicated) post maximal chest pain, FBP, U&E, CRP. Lipids, LFT, glucose, HbA1C etc should be sent in all ACS patients during their IP stay to address modifiable risk factors.
- CXR
- Load with Aspirin 300 mg orally (if not already given by ambulance service) and Ticagrelor 180mg unless contraindicated.
- Morphine for pain 2.5-10mg intravenous initially, repeated if necessary after 5 minutes
- Antiemetic should be given with the first dose of Morphine unless already given prior to hospital admission. Metoclopramide 10 mg IV is first line.
- Oxygen should ***not*** be routinely prescribed, but should be initiated if hypoxaemia is evidenced by reduced O2 saturation monitoring or if oxygen saturations cannot be monitored accurately.
- Evaluate hsTnT results (see appendix 3, page 25-26):

  - ✓ Ideally patients will be seen by an ED cardiology chest pain nurse who will arrange onward referral for cardiac investigations directly thus bypassing the need for a RACPC referral.

  - ✓ Otherwise, for patients who present > 3 hours after maximal chest pain, are symptom free with no ECG changes and initial hsTnT ($T_0$) is <5 ng/L with a low risk score then consider discharge home with a referral to the RACPC / cardiology OP clinic using the referral system if their presenting symptoms are felt to be cardiac in origin.

- ✓ This also applies to patients who present > 3 hours after maximal chest pain with an initial hsTnT of <12ng/L and a $T_1$ which does not rise by more than 3ng/L. This constitutes the 'rule out' group.

- ✓ For patients with a high risk history, if the Initial hsTnT is >52ng/L or rises by >5ng/L at $T_1$ then this constitutes the 'rule in' group and should be treated accordingly and referred for admission.

- ✓ If $T_0$ is between 12ng/L and 52ng/L and $T_1$ rises by <5ng/L then this constitutes the 'observation group'. This also encompasses patients with a $T_0$ < 12ng/L with a $T_1$ rising by 3 or 4ng/L.

- ✓ Observation group patients require a $T_3$. If $T_3$ rises by <20% then these patients enter the 'rule out' group. If $T_3$ rises by >20% then these patients enter the 'rule in' group.

Refer to a cardiologist without delay if any of the following apply:

- ST depression of >1mm,
- Initial hsTnT >100ng/L
- Abnormal ECG with dynamic changes
- On-going chest pain / discomfort
- Haemodynamic instability
- Considering the need for intravenous nitrates
- Consideration should also be given for the need of a small molecule GP IIb/IIIa inhibitor **in discussion with a cardiologist.**

***Remember that these are guidelines only and that patients can still have significant coronary artery disease despite negative screening tests. If in doubt, and especially with a good history for ischaemic cardiac symptoms, refer for a specialist opinion.***

### 6.5 Concomitant Pharmacological treatment

- **Aspirin** 75 mg daily
- **Ticagrelor** 90 mg **BD** for 12 months
  (if contraindicated Clopidogrel 75 mg OD)

- **Statins:** See section 6.8 for flowchart

- **ACE inhibitors:** Should be given to all patients post MI unless contraindicated. Renal function must be monitored. Initiate Ramipril 1.25 -2.5 mg OD (or Perindopril 2mg OD) and aim to double after 24-48 hours.

- **Beta blockers:** oral beta blockers should be given to all patients unless there are clear contraindications such as asthma, severe bradycardia, second or third degree AV block or severe heart failure. Initiate Bisoprolol 1.25 -2.5 mg OD and aim to double after 24-48 hours

- **Eplerenone** 25 mg OD should be initiated in any patient with evidence of cardiac failure or LVEF < 40 %

- **Oral anticoagulation:** when used in combination with antiplatelet agents is at the discretion of the interventional cardiology and is an evolving topic.

- **Antianginal medication (see appendix 5, page 28):**

  - **Beta blockers**

  - ✓ Rate limiting action
  - ✓ Contraindicated in severe bradycardia, high degree AV block, sick sinus syndrome and refractory heart failure
  - ✓ Relatively contraindicated in bronchospastic disease. Caution with verapamil and diltiazem

  - **Calcium channel blockers**

  - ✓ Vasodilators (dihydropyridines and phenylalkylamines/ benzothiazepines)
  - ✓ Rate limiting action (phenylalkylamines/ benzothiazepines)
  - ✓ Dihydropyridines contraindicated in cardiogenic shock, significant aortic stenosis
  - ✓ Phenylalkylamines/ benzothiazepines contraindicated in acute porphyria, cardiogenic shock, significantly impaired LV function, HF, AF in WPW, AV and sinoatrial block
  - ✓ Dihydropyridines e.g. amlodipine, nicardipine, nifedipine
  - ✓ Phenylalkylamines e.g. verapamil
  - ✓ Benzothiazepines e.g. diltiazem

- **Nitrates**

✓ Vasodilators
✓ Contraindicated in severe aortic stenosis, relatively contraindicated in HCM (hypertrophic cardiomyopathy)
✓ Should not be used within 24hrs of phosphodiesterase inhibitors due to risk of profound hypotension

- **Ranolazine**

✓ Start with 375mg **BD**
✓ Inhibits the late inward sodium current, indirectly reducing the sodium-dependent calcium current during ischemic conditions and leading to improvement in ventricular diastolic tension and oxygen consumption
✓ Minimal effects on HR and BP
✓ Cautions: Body-weight less than 60 kg; elderly; moderate to severe congestive heart failure; QT interval prolongation

- **Ivabradine**

✓ Start with 5mg **BD**
✓ Selectively inhibits the "funny" channel pacemaker current (If) in the sinoatrial node, contraindicated in acute MI, cardiogenic shock, HR <70, AV block
✓ Do not use in AF

- **Nicorandil**

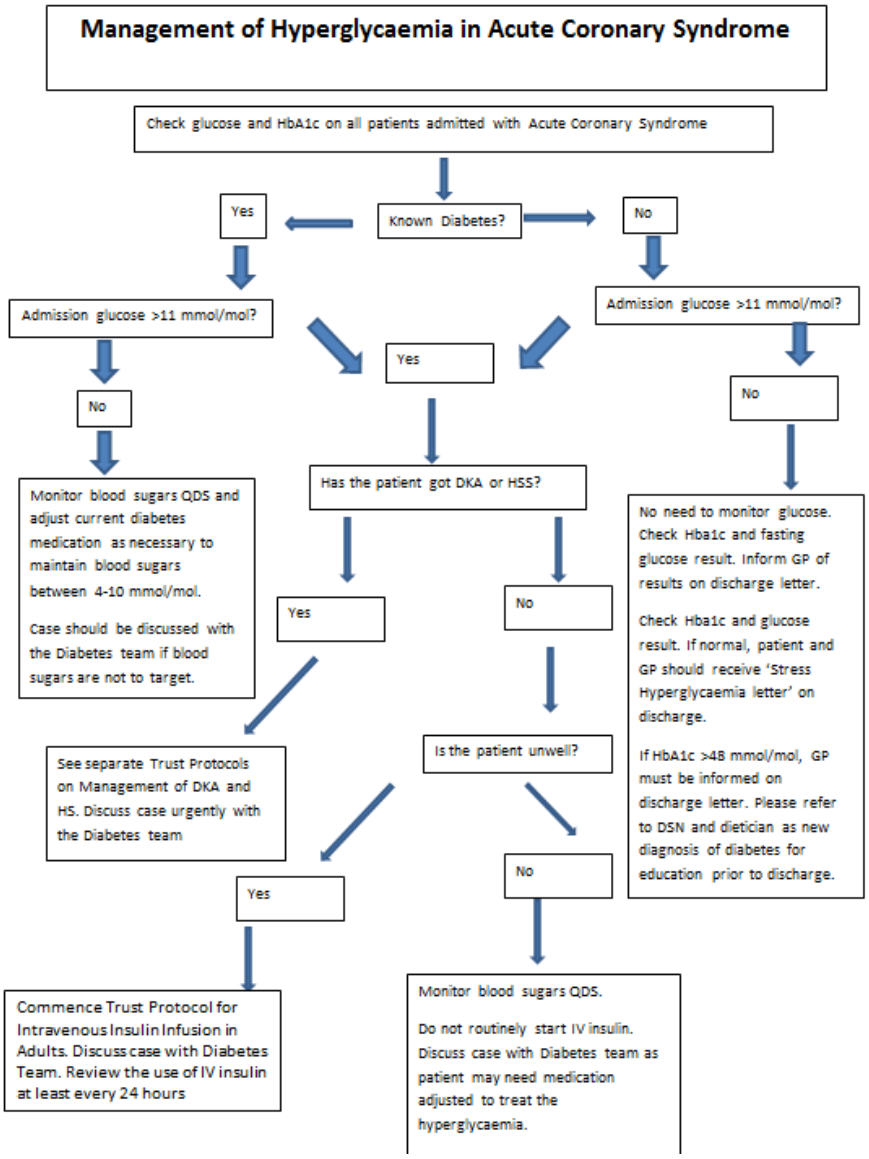
✓ Start with 10mg **BD**
✓ Potassium channel activator causing vasodilation
✓ Contraindicated in cardiogenic shock, hypotension
✓ Can cause significant oral, GI or skin ulceration

### 6.6 Diabetes management in acute myocardial infarction

- All known and newly diagnosed patients with diabetes should have regular glucose monitoring and should be maintained within the strict targets, if needed initiate treatment with intravenous insulin and glucose for at least 24 hours (see CCU protocol).

- Existing oral hypoglycaemic agents should be stopped while intravenous Insulin is being given.
- Patients already on Insulin should be recommenced on their previous regime when stable.
- New diabetics or patients previously on oral hypoglycaemic agents should be referred to a diabetologist for consideration of further management
- Figure 2 summarises the management of hyperglycaemia in ACS

**Figure 2: Management of hyperglycaemia in ACS:**



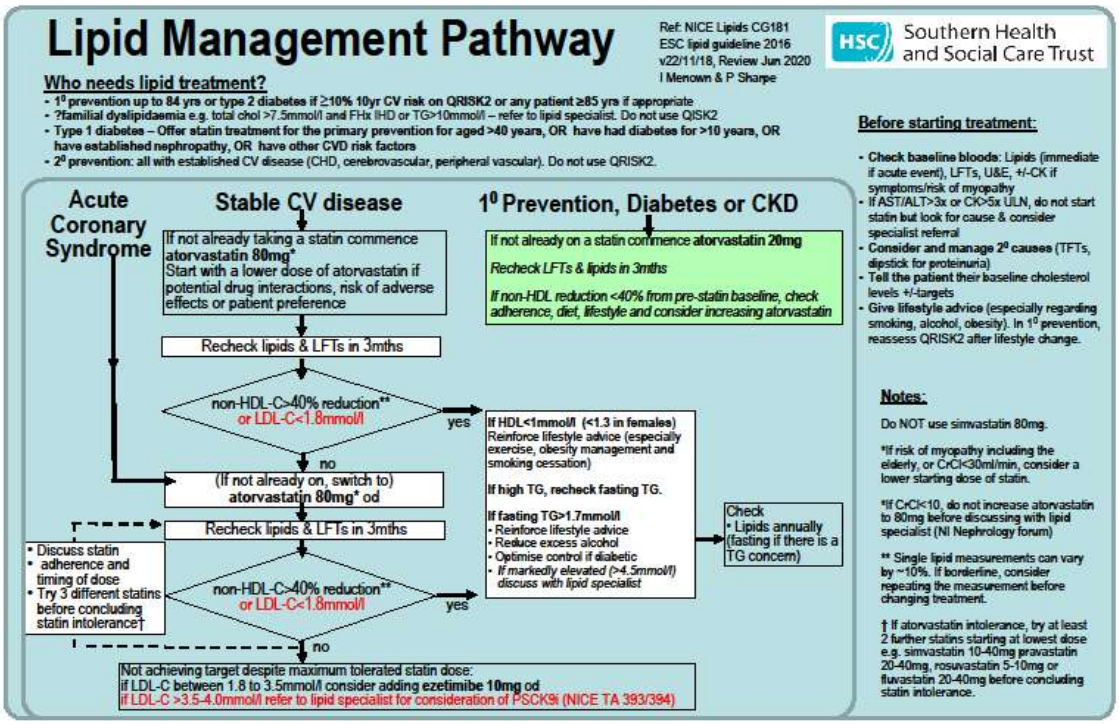
### 6.7 LV assessment in acute myocardial infarction

- All patients should have an echocardiogram pre-discharge to assess LV function not assessed by LV gram during angiogram. If there is evidence of significant LV dysfunction, please request a repeat echocardiogram after 3 months for risk stratification and further cardiology input. If EF<35% at 3 months then consideration should be given to advanced heart failure therapy including Sacubitril / Valsartan (Entresto), device therapy (primary prevention ICD or CRT-D if broad QRS) or transplant workup if necessary.

### 6.8 Lipid management

- Treat all patients post MI regardless of serum cholesterol on admission. First line treatment should be Atorvastatin 80 mg STAT. Please see figure 3 for further lipid guidance and target levels. Lipid guidelines are continuously being reviewed by the Northern Ireland regional cardiovascular formulary group and will be updated in subsequent reviews.

**Figure 3: Lipid management pathway**.



### 6.9 Cardiac rehabilitation

- All patients with STEMI requiring pPCI should be seen by the cardiac rehabilitation team within 48 hours of admission.
- All patients diagnosed with NSTEMI should be seen prior to discharge.
- All patients are referred on to the community rehab team for follow-up post discharge.

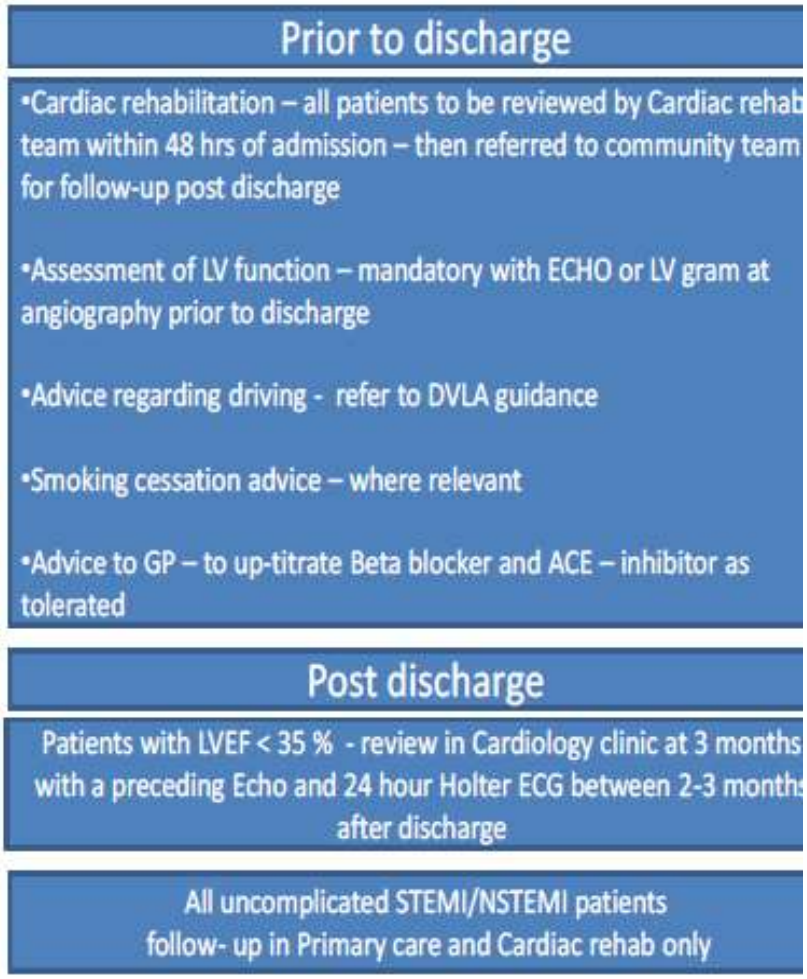
### 6.10 Prevention of contrast – induced acute kidney injury (CI-AKI)

- Contrast induced acute kidney injury (CI-AKI) is currently defined as a delta rise in serum creatinine of ≥26.5 µmol/litre or a relative rise of ≥50% from baseline measured at 48 hours following administration of iodinated contrast.
- Preventative practice in SHSCT includes clear pre- and post-hydration guidance, use of the Mehran score to identify high-risk patients, ensuring nephrotoxic medications are withheld pre and post contrast in appropriate patients, continuous education of staff, use of advice sheets for patients and GPs and a 7 day a week service for renal function sampling 48 hours post contrast (see intranet, CCU and cath lab for further details and protocols).

### 6.11 Follow-up post discharge

- All patients with an LVEF <35% will be reviewed in cardiology outpatient clinic at 3 months with a preceding repeat echo. This will usually be via the heart failure service if suitable.
- ACS patients will be followed up by the discharging consultant or interventional cardiologist who inserted the stent(s) as deemed necessary on a case by case basis.
- Figure 3 is a pre-discharge checklist.

**Figure 3: Pre-discharge checklist**




**6.12 Out-patient investigations**

- Patients may be directly discharged from ED for OP investigations if seen directly by the ED chest pain nurse or in-house cardiology team. This constitutes the 'rule-out' group. OP investigations may include CT calcium scoring (CTCS), CT coronary angiography (CTCA), dobutamine stress echocardiography (DSE), myocardial perfusion imaging (MPI) or diagnostic angiography.

**6.13 Sexual activity**

- Reassure patients that after recovery from an MI, sexual activity presents no greater risk of triggering a subsequent MI than if they had never had an MI. Advise patients who have made an uncomplicated recovery after their MI that they can resume sexual activity when they feel comfortable to do so, usually after about 4 weeks.
- When treating erectile dysfunction, treatment with a PDE5 (phosphodiesterase type 5) inhibitor may be considered in men who have had an MI more than 6 months earlier and who are now stable.
- PDE5 inhibitors must be avoided in patients treated with nitrates or nicorandil because this can lead to dangerously low blood pressure.

### 6.14 Lifestyle changes after an MI

➢ **Changing diet**

- Advise people to eat a Mediterranean-style diet (more bread, fruit, vegetables and fish; less meat; and replace butter and cheese with products based on plant oils).

➢ **Alcohol consumption**

- Advise people who drink alcohol to keep weekly consumption within safe limits (no more than 14 units of alcohol per week for men and women and to avoid binge drinking (more than 3 alcoholic drinks in 1–2 hours).

➢ **Regular physical activity**

- Advise people to undertake regular physical activity sufficient to increase exercise capacity.

- Advise people to be physically active for 20–30 minutes a day to the point of slight breathlessness. Advise people who are not active to this level to increase their activity in a gradual, step-by-step way, aiming to increase their exercise capacity. They should start at a level that is comfortable, and increase the duration and intensity of activity as they gain fitness.

➢ **Smoking cessation**

- Advise all people who smoke to stop and offer assistance from a smoking cessation service. Advise referral to SHSCT smoking cessation nurse.

### 6.15 Advice regarding driving: Please refer to DVLA guidance

https://www.gov.uk/government/publications/assessing-fitness-to-drive-a-guide-for-medical-professionals

- Unfortunately, in view of the frequent changes in DVLA guidance, we cannot add a summary here. The guiding principles include the functional status of the patient, completeness of revascularisation, effect of ACS on the left ventricular function and the type of driving licence. Needless to say, type 2 licence (HGV licence) carries much stricter criteria for regaining the licence. Please see DVLA website.

## 7. Update and review

- This document will be updated every 3 years.

- Revisions will be made ahead of the review date if new, relevant national guidelines are published. Where the revisions are significant and the overall policy is changed, the authors will ensure the revised document is taken through the standard consultation, approval and dissemination processes.

## 8. References

- ESC guidelines for the management of acute coronary syndromes in patients presenting without persistent ST elevation. The Task Force for the management of acute myocardial infarction in patients presenting without ST-segment elevation of the European Society of Cardiology (ESC). European Heart Journal (2020) 00, 1-79.
- ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). European Heart Journal (2018) 39, 119–177.
- Chest pain of recent onset: Assessment and Diagnosis. NICE Guidelines [CG95] Published Date: March 2010
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- Ticagrelor versus Clopidogrel in Patients with Acute Coronary Syndromes N Engl J Med 2009;361:1045-57
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**Appendix 1 – Governance information (amendment agreed on 2nd Sept 2020)**

| | |
|---|---|
| **Document title** | Guidelines for the management of acute chest pain of cardiac origin |
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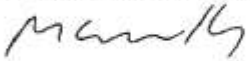
| **Date valid from:** | **5th August 2020** |
| **Date valid to:** | **Initial date: 5th August 2023**<br>**Extended date: 1st Jan 2027 (see below)** |
| **Brief summary of contents** | This document provides guidance for any professional involved in the clinical management of patients presenting to SHSCT with chest pain of cardiac origin. |
| **Policy objectives** | To provide clear speciality agreed guidelines and pathways for the diagnosis and clinical management of patients with chest pain of cardiac origin. |
| **Keywords** | Cardiology<br>Chest pain<br>Acute coronary syndrome<br>Ticagrelor<br>One hour troponin |
| **Authorship** | Dr David Mc Eneaney (cardiology clinical lead, consultant cardiologist)<br>Kay Carroll (cardiology head of service)<br>Dr Mick Connolly (consultant cardiologist)<br>Dr Patricia Campbell (consultant cardiologist)<br>Dr Ian Menown (consultant cardiologist)<br>Dr Janet Toner (associate specialist) |

Addendum Jan 2024:

This policy was reviewed by the consultant cardiologists and discussed at the cardiology governance meeting in Winter 2023. It was sent out for a 6 week consultation period. No amendments were suggested by the wider cardiology team. Following universal unanimous agreement by the consultant body at this governance meeting it has been approved for 3 further years until 1st Jan 2027.

Signed: Dr Mick Connolly Consultant cardiologist



## Appendix 2a: Heart score (www.heartscore.nl)

HEART

### HEART score for chest pain patients

| | | | |
|---|---|---|---|
| History (Anamnesis) | Highly suspicious | 2 | |
| | Moderately suspicious | 1 | |
| | Slightly suspicious | 0 | |
| ECG | Significant ST-deviation | 2 | |
| | Non-specific repolarisation disturbance / LBBB / PM | 1 | |
| | Normal | 0 | |
| Age | ≥ 65 years | 2 | |
| | 45 – 65 years | 1 | |
| | ≤ 45 years | 0 | |
| Risk factors | ≥ 3 risk factors *or* history of atherosclerotic disease | 2 | |
| | 1 or 2 risk factors | 1 | |
| | No risk factors known | 0 | |
| Troponin | ≥ 3x normal limit | 2 | |
| | 1-3x normal limit | 1 | |
| | ≤ normal limit | 0 | |
| | | **Total** | |

**Risk factors for atherosclerotic disease:**

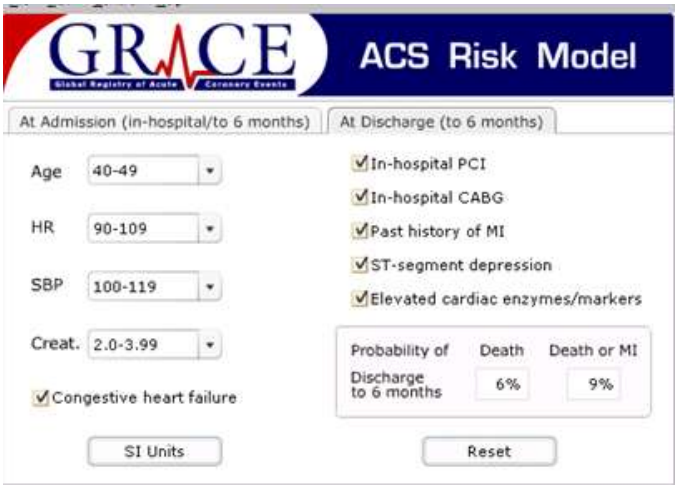
| | |
|---|---|
| Hypercholesterolemia | Cigarette smoking |
| Hypertension | Positive family history |
| Diabetes Mellitus | Obesity (BMI>30) |

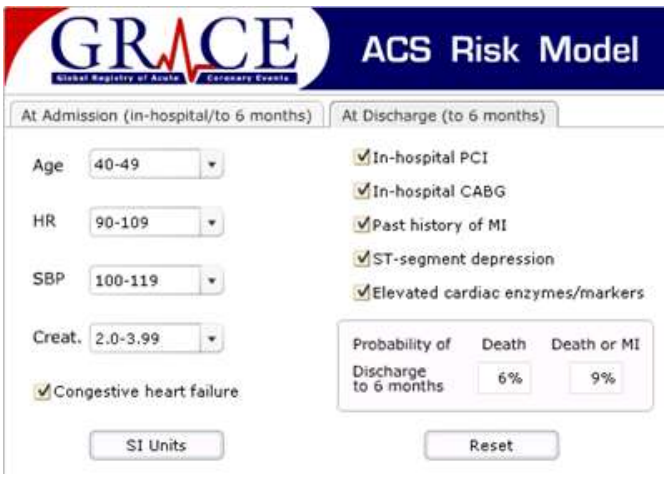
### Risk groups and proposed policy when using the HEART score (pooled results n=6174)

| HEART | ~ % pts | MACE/n | MACE | Death | Proposed Policy |
|---|---|---|---|---|---|
| 0-3 | 32% | 38/1993 | 1.9% | 0.05% | Discharge |
| 4-6 | 51% | 413/3136 | 13% | 1.3% | Observation, risk management |
| 7-10 | 17% | 578/1049 | 55% | 2.8% | Observation, treatment, CAG |

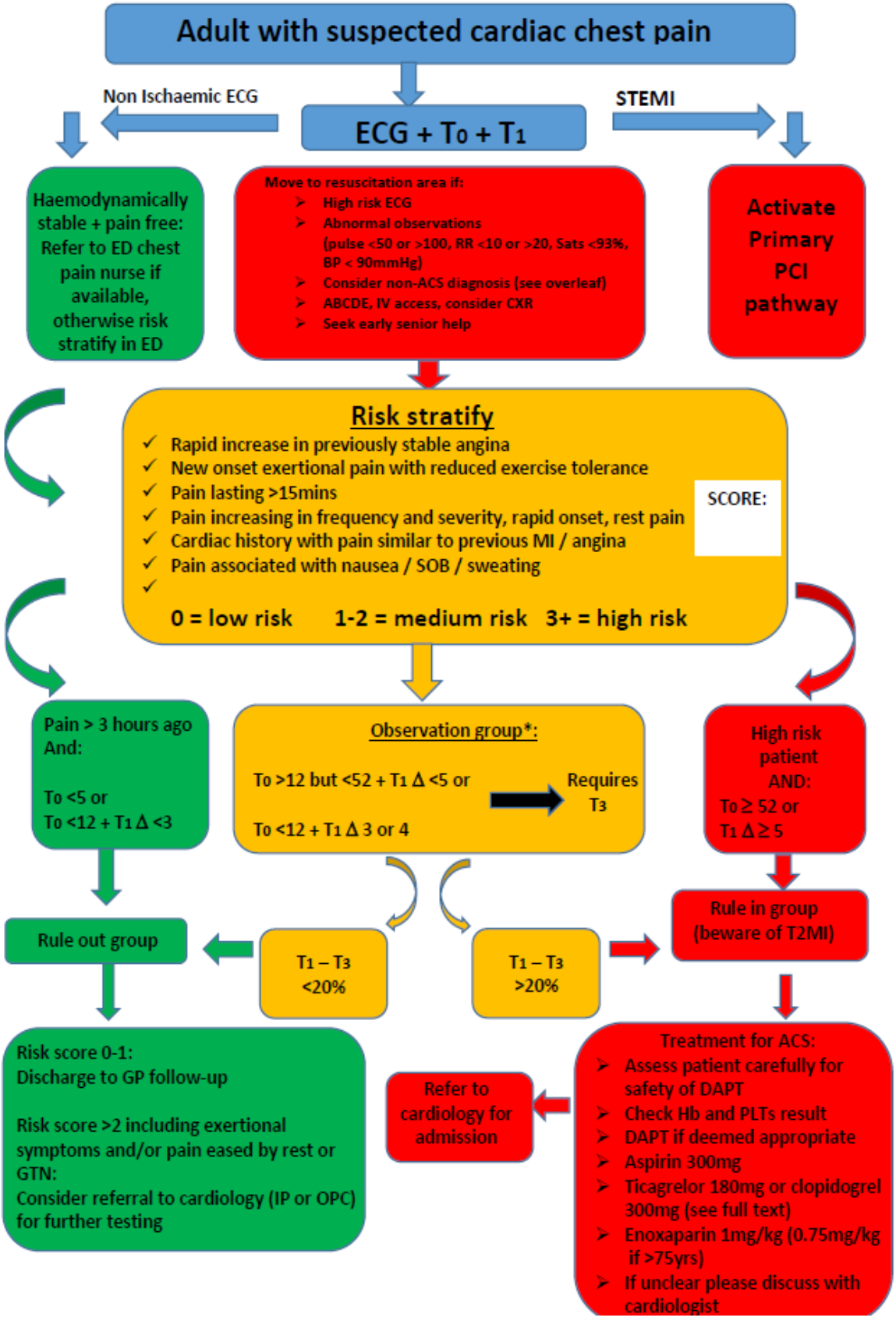
www.heartscore.nl

## Appendix 2b: Grace score (www.outcomes.org/grace)

## Appendix 3: Chest pain pathway



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Patient addressograph

Allergy status:

## Patient times / results

| | Time | hsTnT result |
|---|---|---|
| Pain | | |
| T0 | | |
| T1 | | |
| T3 | | |

## General notes

- Pathways are designed to help, not to override, clinical decision making
- T0 = baseline / arrival highly sensitive troponin
- T1 = 1 hour troponin
- T3 = 3 hour troponin
- Δ = absolute delta change between troponin samples
- If there is a good clinical reason to follow an alternative course of action then it should be done with expert input as necessary
- This pathway is based on European Society of Cardiology guidelines
- This pathway only applies to patients presenting with chest pain
- Be cautious of early presenting patients: If patients present within 3 hours of worst pain then will require T0 + T1 + T3.
- If patient is unsure of onset then perform T0 + T1 as a minimum
- Renal dysfunction: Elevated troponin should not be primarily attributed to impaired creatinine clearance unless GFR<30 and there are no features in the history to suggest an acute cardiac cause
- *The observation group represents those patients requiring further assessment and a 3 hour troponin sample. A change of >20% between T1 and T3 (assuming one value is >14ng/L) is consistent with a diagnosis of ACS.

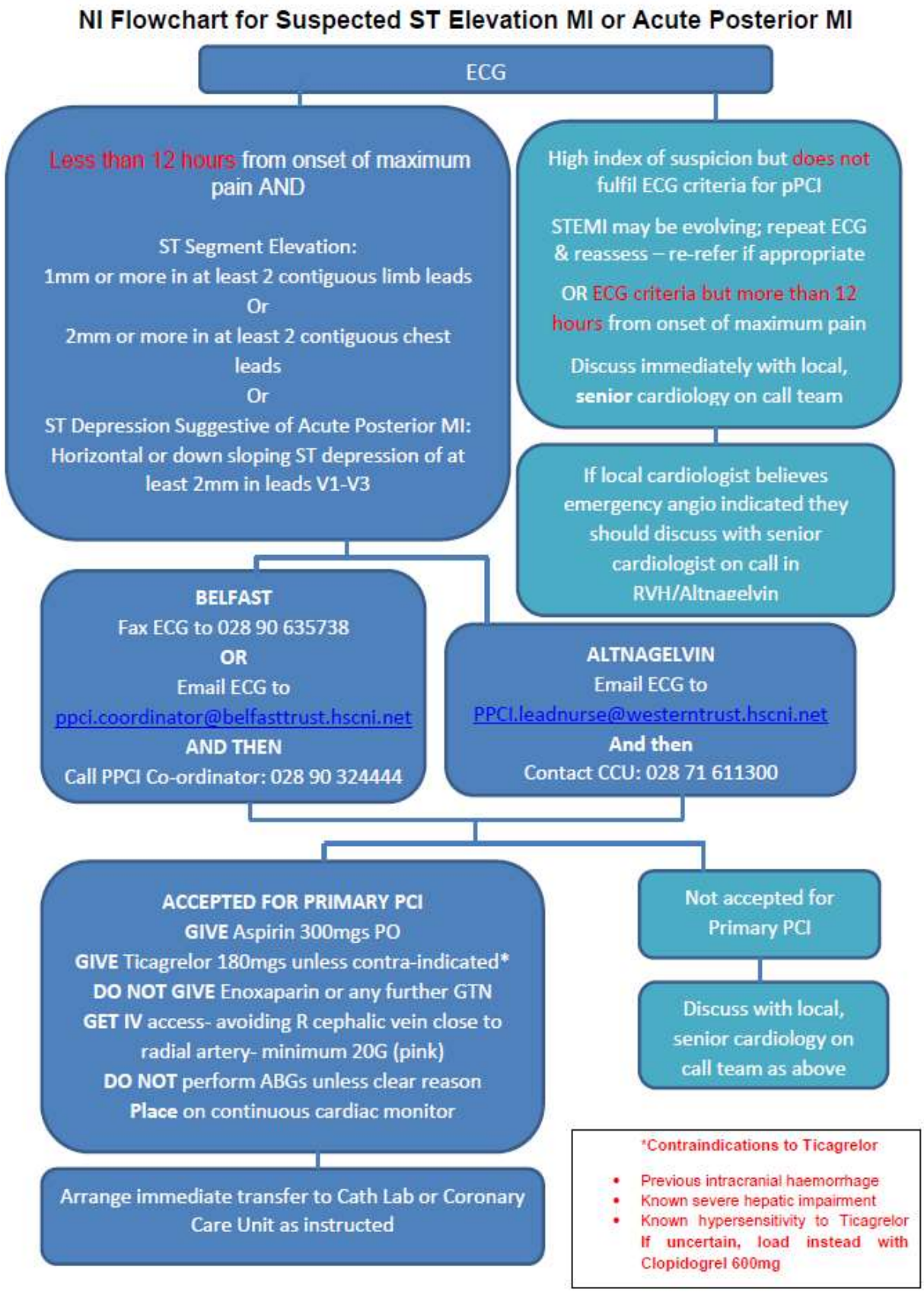
### Cardiac causes of raised troponin

- ✓ Acute coronary syndrome
- ✓ Tachyarrhythmia
- ✓ Cardiac failure
- ✓ Myocarditis
- ✓ Takotsubo cardiomyopathy
- ✓ Aortic dissection
- ✓ Coronary spasm
- ✓ Cardiac contusion
- ✓ Valvular disease e.g. aortic stenosis
- ✓ Hypertensive emergency
- ✓ Post procedure

### Non-cardiac causes of raised troponin

- ✓ Critical illness / sepsis
- ✓ PE / pulmonary hypertension
- ✓ Acute exac COPD
- ✓ Subarachnoid haemorrhage
- ✓ CVA
- ✓ ESRD
- ✓ Seizure
- ✓ Drug toxicity
- ✓ Rhabdomyolysis
- ✓ Strenuous exercise
- ✓ Infiltrative disease

**Appendix 4: PPCI pathway for STEMI or acute posterior MI**



## Appendix 5: Antianginal medication pathway

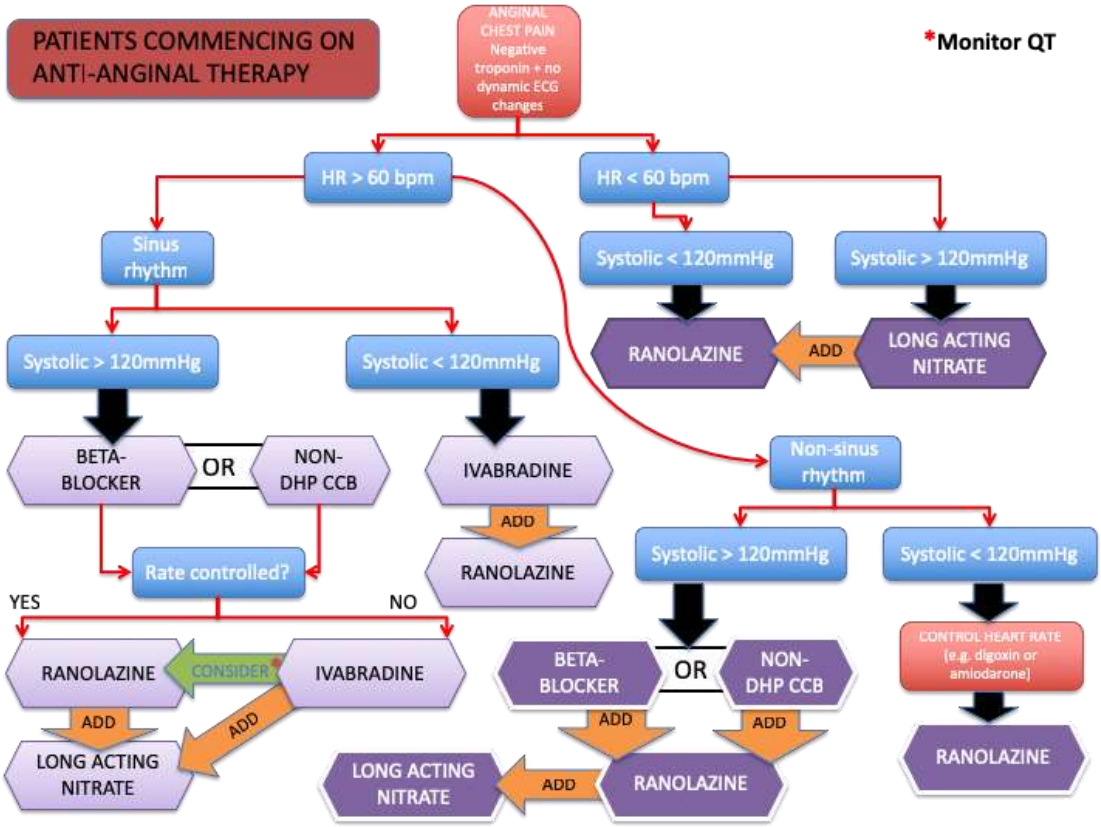



Illustration used with permission from Dr Luke F Smith MBBS M.Sc. (Med Tox) FRCP (Acute) DFMS (many faces of angina meeting, Manchester, 11th October 2019)

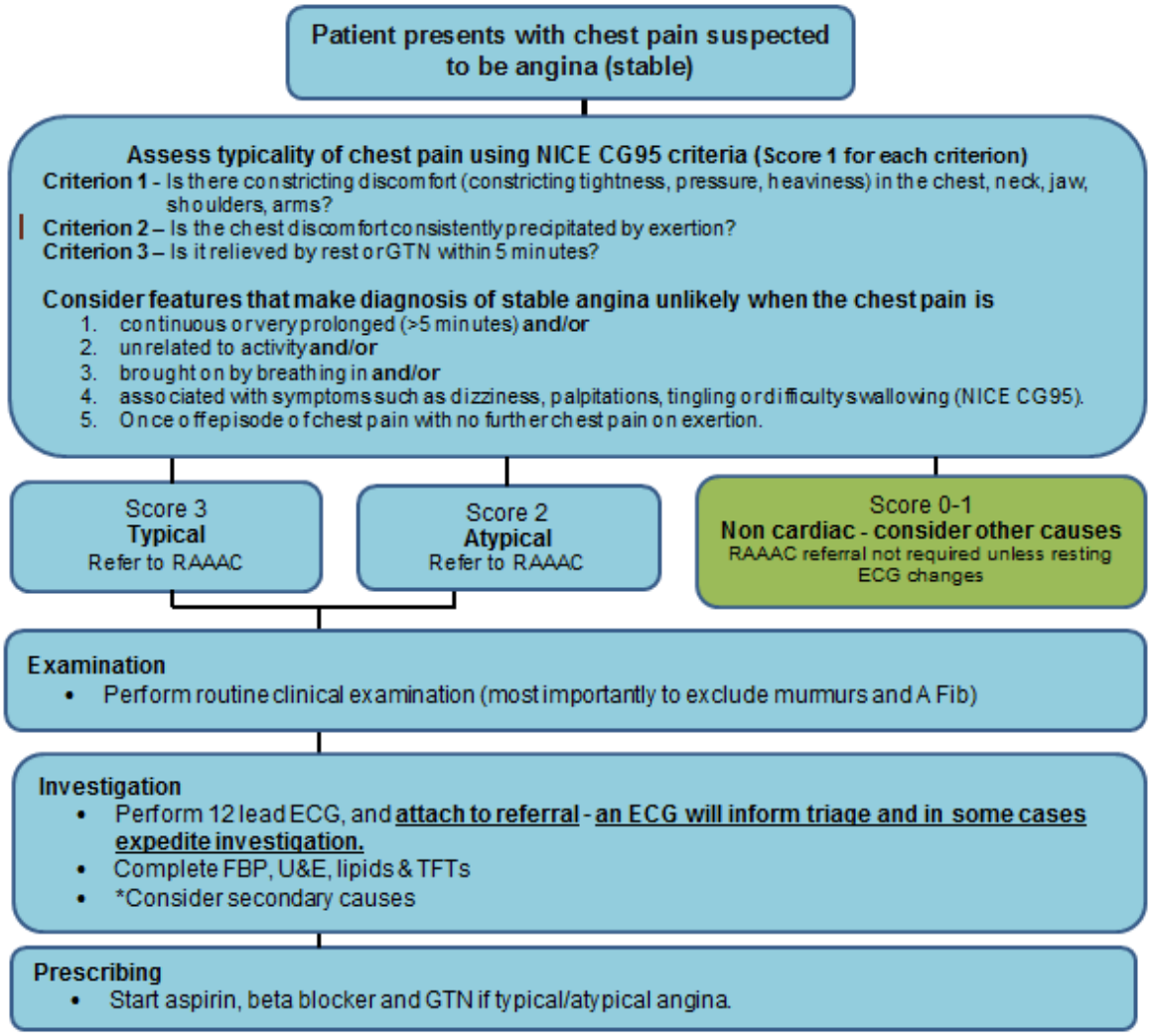
**NB: Avoid non-DHP CCBs in the setting of impaired LVEF or heart failure**

## Appendix 6: Internal referral to RACPC pathway

### NI Protocol for Rapid Access Angina Assessment Clinic (RAAAC) Internal Referral Guidance for Stable Chest Pain

RAAACs are designed for the assessment and diagnosis of new onset chest pain **(stable, non-acute)** suggestive of stable angina and for patients not currently under a cardiologist who have known ischaemic heart disease and recurrent symptoms. It is not appropriate for screening for CHD or definitively diagnosing other causes of chest pain.

Patients who clearly have non-cardiac chest pain are not likely to benefit from attendance and will not be offered an appointment; referrals will be returned if there is insufficient information.




Internal referral information should include:

- Characteristics of presenting chest pain to include NICE CG95 criteria as above
- Patients CV risk factors e.g. smoking, diabetes, hypertension, lipids, and family history (defined as 1st degree relative < 60 years)
- 12 lead ECG and bloods
- Q Risk2 Score if available https://www.qrisk.org/2017/

Advise patient that if pain/discomfort increases in severity or duration to seek urgent medical attention.

NICE CG95 https://pathways.nice.org.uk/pathways/chest-pain#path=view%3A/pathways/chest-pain/assessing-and-diagnosing-suspected-stable-angina.xml&content=view-node%3Anodes-initial-management-and-ecg suggests outruling anaemia, hyperthyroidism etc.

## Appendix 7: GP direct referral to RACPC

### Rapid Access Angina Assessment Clinic (RAAAC) Referral Guidance for Stable Chest Pain

RAAACs are designed for the assessment and diagnosis of new onset chest pain **(stable, non-acute)** suggestive of stable angina and for patients who have known ischaemic heart disease and recurrent symptoms not currently under a cardiologist. It is **not** appropriate for screening for CHD or definitively diagnosing non-cardiac causes of chest pain.

Patients who clearly have non-cardiac chest pain are not likely to benefit from attendance and will not be offered an appointment; referrals will be returned if there is insufficient information.

Patients who are felt to have unstable symptoms i.e. prolonged (>5minutes) episodes of chest pain with a high likelihood of being ischaemic should be referred to ED.

**Patient presents with chest pain suspected to be angina (stable)**

**Assess typicality of chest pain using NICE CG95 criteria (Score 1 for each criterion)**

**Criterion 1** - Is there constricting discomfort (constricting tightness, pressure, heaviness) in the chest, neck, jaw, shoulders, arms?
**Criterion 2** – Is the chest discomfort consistently precipitated by exertion?
**Criterion 3** – Is it relieved by rest or GTN within 5 minutes?

**Consider features that make diagnosis of stable angina unlikely when the chest pain is**

1. continuous or very prolonged (>5 minutes) **and/or**
2. unrelated to activity **and/or**
3. brought on by breathing in **and/or**
4. associated with symptoms such as dizziness, palpitations, tingling or difficulty swallowing (NICE CG95).
5. Once off episode of chest pain with no further chest pain on exertion.

| Score 3 | Score 2 | Score 0-1 |
|---|---|---|
| **Typical** | **Atypical** | **Non cardiac - consider other causes** |
| Refer to RAAAC | Refer to RAAAC | RAAAC referral not required unless resting ECG changes |

**Examination**
- Perform routine clinical examination (most importantly to exclude murmurs and A Fib)

**Investigation**
- Recommended to perform 12 lead ECG where available, and **attach to referral** - **an ECG will inform triage and in some cases expedite investigation.**
- Consider FBP, U&E, lipids & TFTs
- *Consider secondary causes

**Prescribing**
- Start aspirin and GTN if typical/atypical angina and consider starting a beta blocker.

CCG Referral information should include:
- Characteristics of presenting chest pain to include NICE CG95 criteria as above
- Patients CV risk factors e.g. smoking, diabetes, hypertension, lipids, and family history (defined as 1st degree relative < 60 years)
- 12 lead ECG and bloods if available
- Q Risk2 Score if available https://www.qrisk.org/2017/

Advise patient that if pain/discomfort increases in severity or duration to seek urgent medical attention at an ED.

*NICE CG95 https://pathways.nice.org.uk/pathways/chest-pain#path=view%3A/pathways/chest-pain/assessing-and-diagnosing-suspected-stable-angina.xml&content=view-node%3Anodes-initial-management-and-ecg suggests outruling anaemia, hyperthyroidism etc.

## Appendix 8: RACPC assessment pathway

Rapid Access Angina Assessment Clinic (RAAAC) Referral Assessment for Stable Chest Pain

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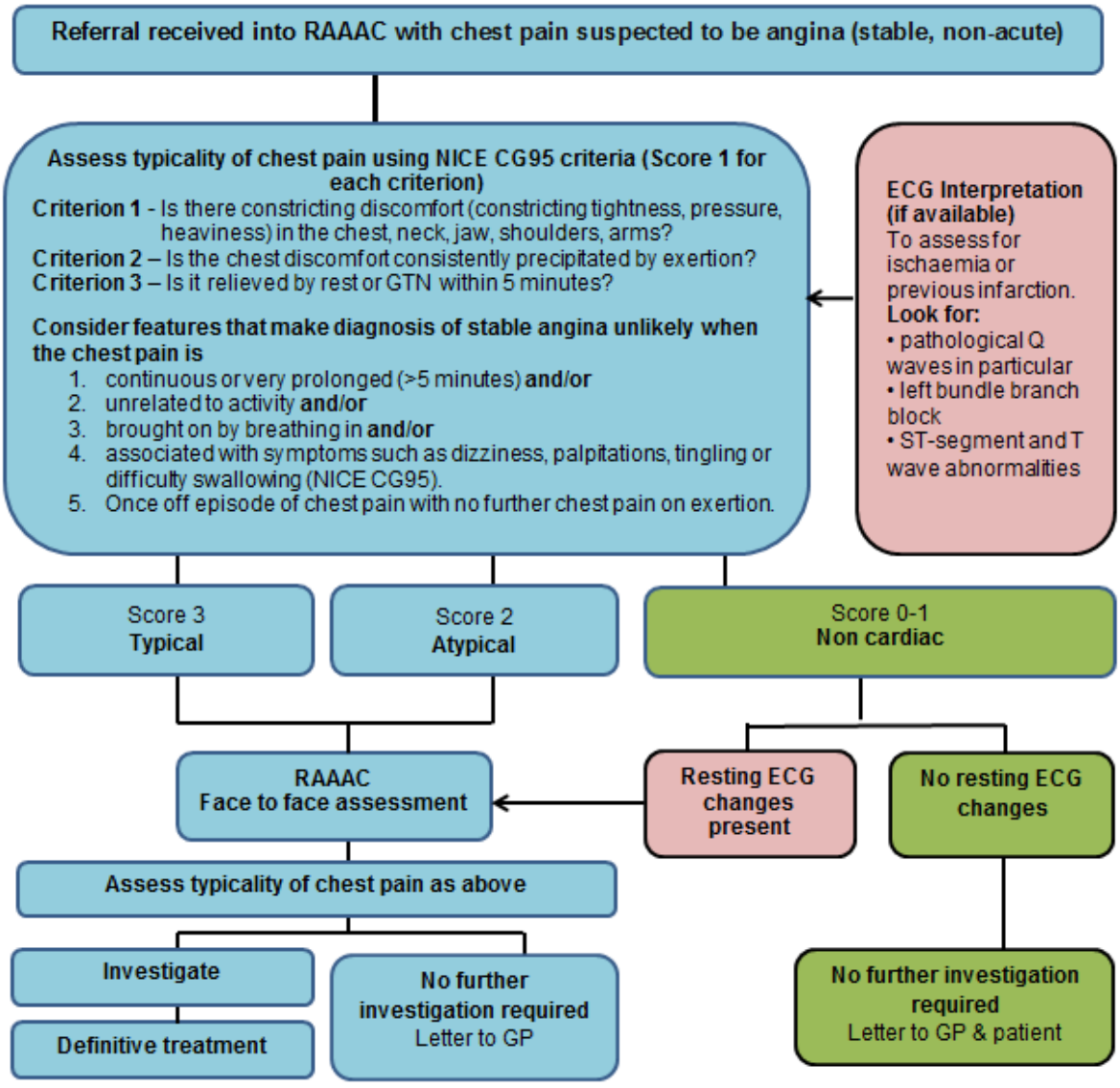


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**Appendix 9: RACPC Trust transfer**

## NI PROTOCOL FOR RAPID ACCESS ANGINA ASSESSMENT CLINIC (RAAAC) PATIENT TRANSFER BETWEEN TRUSTS TO FACILITATE THE MOVEMENT OF PATIENTS TO THOSE UNITS WITH THE SHORTEST WAITING TIMES