

APPENDIX

Table S1: Classification of Myocardial Infarction (MI)**a) 4th Universal Definition of MI: aetiological classification (1)**

Category	Description	MI criteria		Treatment
		Troponin criteria	Supporting Evidence of Myocardial Ischaemia	
Type 1 MI	Spontaneous MI due to primary coronary occlusion from acute plaque rupture, erosion causing coronary artery thrombosis	≥ 20% rise and/or fall of troponin levels with at least one value > 99th percentile of upper reference limit	Clinical symptoms of myocardial ischaemia; new ischaemic ECG changes; imaging evidence of new regional wall motion abnormalities or new loss of viable myocardium or angiographic finding of intra-coronary thrombus	Coronary revascularisation, guideline-directed medical therapy (DAPT +/- heparin, ACEIs, BBs, statins)
Type 2 MI	Secondary to a clinical event that causes myocardial oxygen supply/demand mismatch (e.g. anaemia, hypotension/hypertension, cardiac arrhythmias, sepsis)	≥ 20% rise and/or fall of troponin levels with at least one value > 99th percentile of upper reference limit	Clinical symptoms of myocardial ischaemia; new ischaemic ECG changes; imaging evidence of new regional wall motion abnormalities or new loss of viable myocardium	Treat underlying cause of myocardial ischaemic imbalance, consider judicious use of BBs to reduce myocardial oxygen demand
Type 3 MI	Sudden cardiac death (e.g. cardiac arrest) based on clinical symptomatology, electrocardiographic findings or presence of new coronary artery thrombus identified by angiography or at post-mortem examination, without the availability of cardiac biomarkers	Not applicable	Clinical symptoms of myocardial ischaemia; new ischaemic ECG changes or presence of ventricular fibrillation; post-mortem analyses revealing intra-coronary thrombus	Not applicable
Type 4 MI	4a – MI occurring within 48 hours of PCI	≥ 20% rise in post-procedural troponins to at least 5x upper reference limit	New ischaemic ECG changes; imaging evidence of new regional wall motion abnormalities or new loss of viable myocardium; angiographic finding of post-procedural flow-limiting complication (coronary thromboembolism, dissection)	Same as for type 1 MI
	4b – MI due to coronary in-stent thrombosis (acute → 0 to 24 hours, subacute – >24 hours to 30 days, late – >30 days to 1 year, very late – more than 1 year)	Same as for type 1 MI	Same as for type 1 MI	Same as for type 1 MI
	4c – MI due to coronary in-stent re-stenosis	Same as for type 1 MI	Same as for type 1 MI	Same as for type 1 MI
Type 5 MI	MI after CABG surgery	≥ 20% rise in post-procedural troponins to at least 10x upper reference	New ischaemic ECG changes; imaging evidence of new regional wall motion abnormalities; angiographic finding of either graft vessel or native coronary artery occlusion	Same as for type 1 MI

*ACEIs: ACE inhibitors; BBs: beta blockers; CABG: coronary artery bypass graft; DAPT: dual antiplatelet therapy; MI: myocardial infarction; PCI: percutaneous coronary intervention

b) STEMI vs NSTEMI:

Category	ECG criteria	Extent of Myocardial Ischaemia	Prognosis
STEMI	New onset ST segment elevation in ≥ 2 contiguous leads (with J-point elevation ≥ 2.5 mm in males < 40 years, ≥ 2 mm in males ≥ 40 years and ≥ 1.5 mm in females for leads V2-3, and J-point elevation ≥ 1 mm in all other leads) (1); or Modified Sgarbossa criteria in patients with existing left bundle brunch block (LBBB) (2); or New-onset LBBB in the appropriate clinical context and biochemical profile	Transmural ischaemia	Poorer short-term outcome and higher inpatient mortality risk
NSTEMI	New onset ST segment depression ≥ 0.5 mm in ≥ 2 contiguous leads; and/or New onset T wave inversion > 1 mm in ≥ 2 contiguous leads that have prominent R wave or R/S ratio > 1 (1)	Subendocardial ischaemia	Poorer long-term outcome (possibly contributed by poorer pre-morbid conditions, less likely to be prescribed guideline-directed medical therapy)

*ECG: electrocardiogram; NSTEMI: non-ST segment elevation myocardial infarction; STEMI: ST segment elevation myocardial infarction

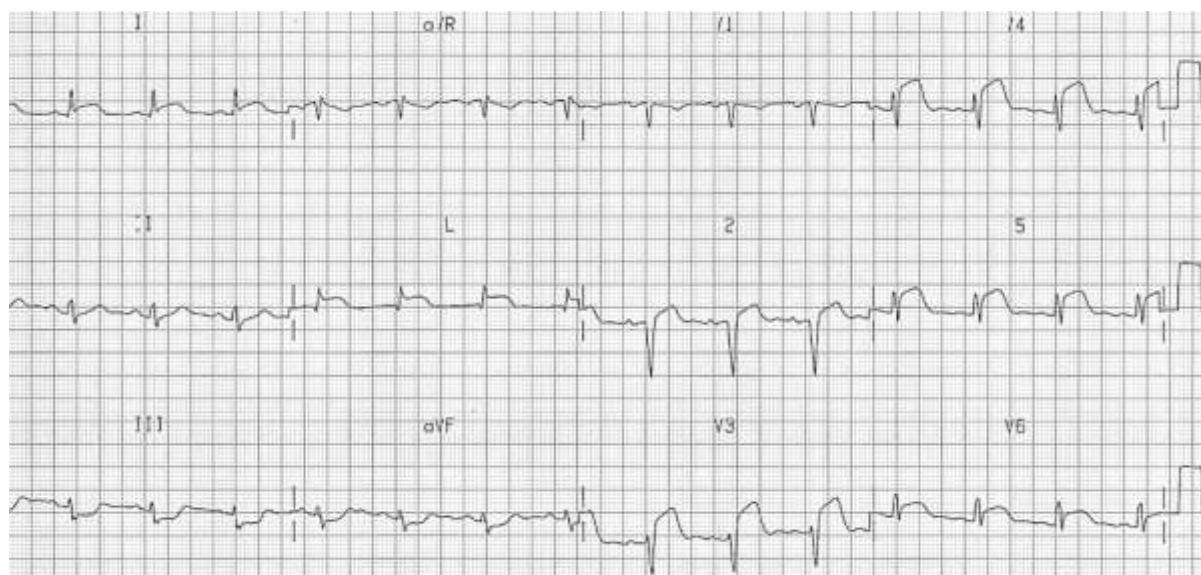
Table S2: Cardiac and Non-Cardiac Causes of Acute Chest Pain

Cardiac	
Pericardium	Acute pericarditis
Myocardium	Acute coronary syndrome (UA, NSTEMI, STEMI) Stable angina/chronic ischemic heart disease Vasospastic (Prinzmetal's) angina Coronary microvascular dysfunction Takotsubo (stress) cardiomyopathy Hypertrophic cardiomyopathy Acute myocarditis
Valvular heart disease	Aortic stenosis Mitral valve prolapse
Non-Cardiac	
Respiratory	Pneumonia Pleuritis Pulmonary embolism Pneumothorax Pulmonary hypertension Asthma/chronic obstructive pulmonary disease (COPD) exacerbation Acute bronchitis Lung cancer Acute chest syndrome (in patients with sickle cell anaemia)
Mediastinum	Aortic dissection/aortic aneurysm Mediastinitis Pneumomediastinum Mediastinal masses
Gastrointestinal	Gastroesophageal reflux disease (GERD)/reflux esophagitis Oesophageal motility disorders Boerhaave's syndrome Peptic ulcer disease Gallstone disease Acute pancreatitis Perforated viscus
Musculoskeletal and Soft Tissue Pathologies	Muscle sprain Rib fractures/trauma Costochondritis (Tietze's syndrome) Cervical/thoracic spine pathologies (referred pain) Herpes zoster (Shingles) Intercostal neuralgia (post-thoracotomy pain syndrome, post-herpetic neuralgia) Xiphoidalgia
Others	Psychogenic (panic attacks, anxiety, somatization disorder)

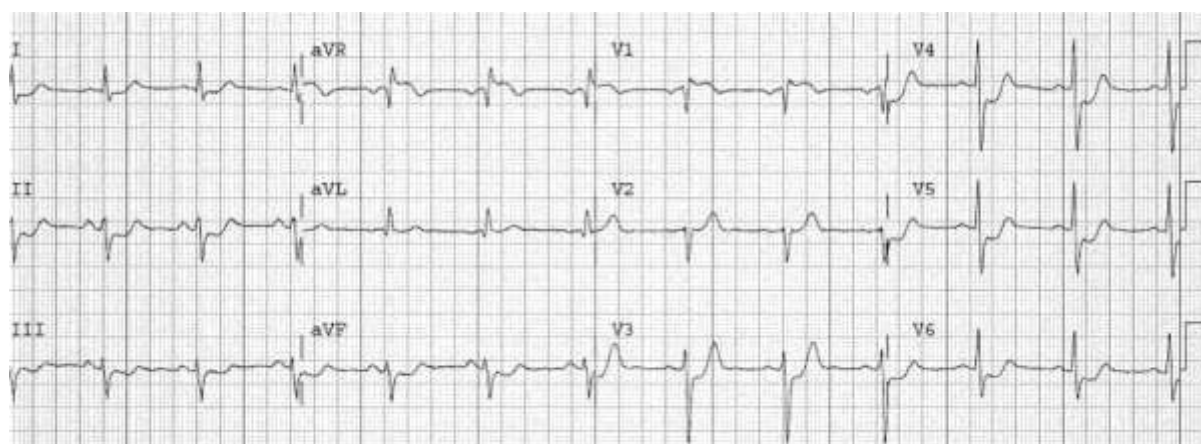
Bold: common causes, highlighted: life-threatening causes

*NSTEMI: non-ST segment elevation myocardial infarction, STEMI – ST segment elevation myocardial infarction, UA – unstable angina

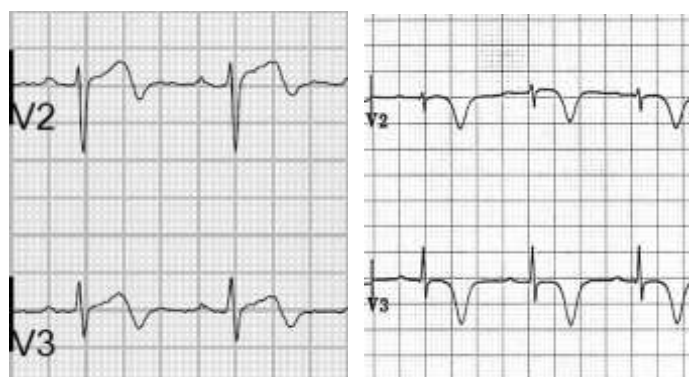
Figure S3: Electrocardiograms of Myocardial Infarction



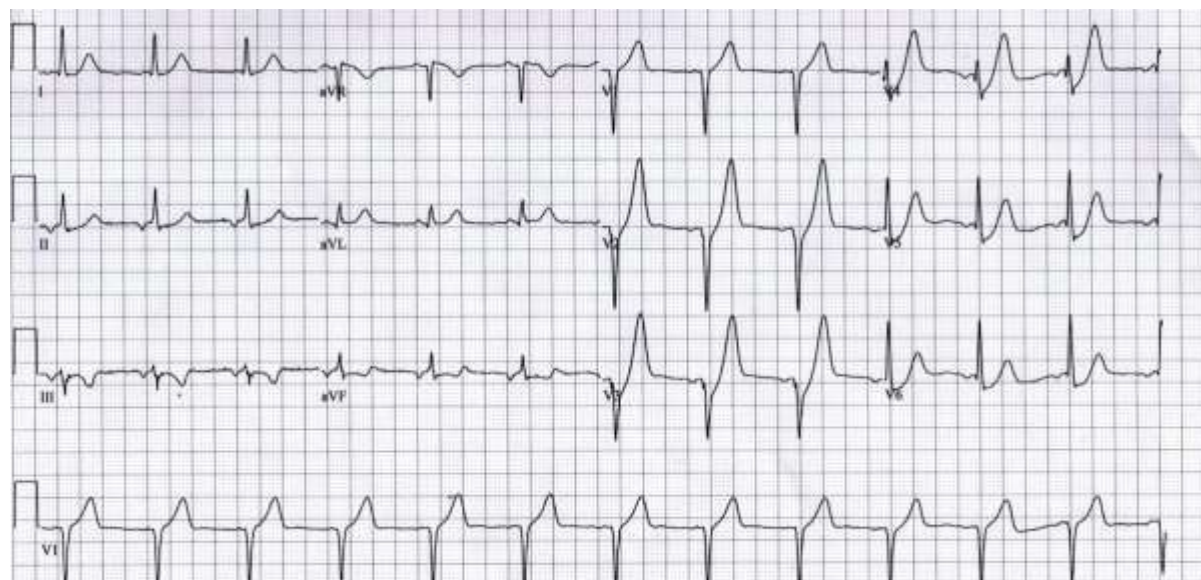
3A) ST segment elevation myocardial infarction (STEMI): this ECG shows ST segment elevation in V2V6, I and aVL, and reciprocal ST depression in III and aVF, suggestive of anterolateral STEMI. Reference: Life in the Fast Lane, 2022 (3).



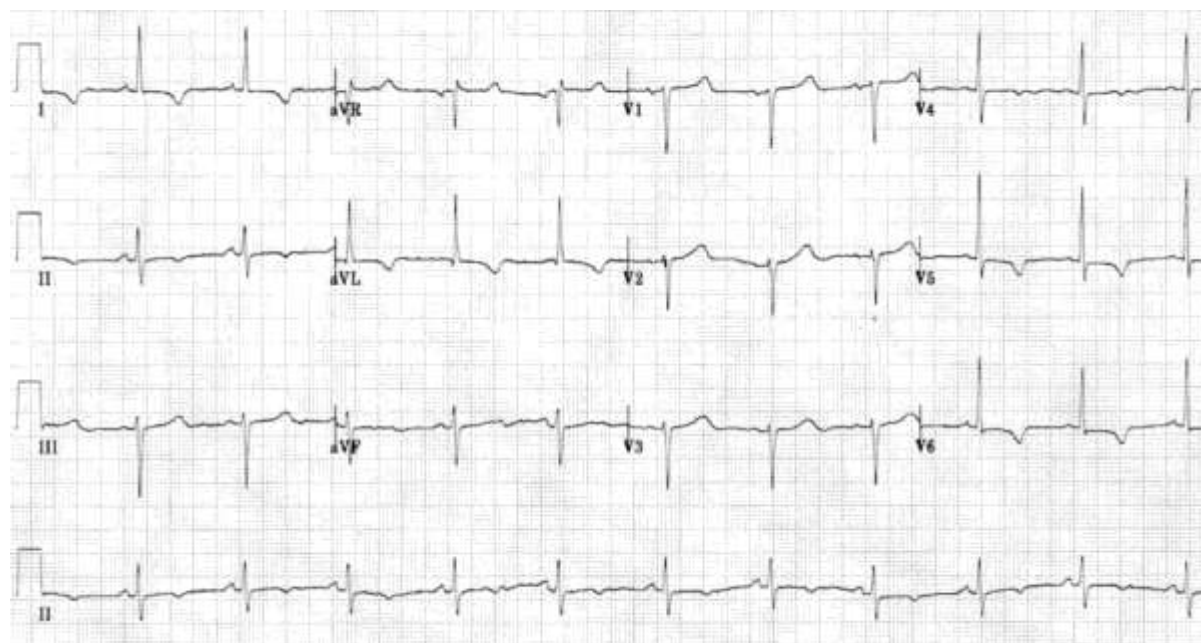
3B) Left main coronary artery (LMCA) occlusion: this ECG shows ST segment elevation in aVR, with global ST segment depressions in multiple leads (V3-V6, I, II, III and aVF). Reference: Life in the Fast Lane, 2021 (4).



3C) Wellen's syndrome – type A (left): biphasic T waves in V2-V3, type B (right): deep T wave inversions in V2-V3. Reference: Life in the Fast Lane, 2021 (5).



3D) de Winter's T waves: this ECG shows upsloping ST segment depression with tall, peaked and symmetrical T waves in precordial leads (V2-6), and ST segment elevation in aVR. Reference: Life in the Fast Lane, 2022 (6).



3E) Non-ST segment elevation myocardial infarction (NSTEMI): this ECG shows T wave inversions predominantly in the lateral leads (I, aVL, V5, V6), which in the appropriate clinical context with hypertroponinaemia showing an acute rise and/or fall in serial troponins, is suggestive of NSTEMI. Reference: Life in the Fast Lane, 2022 (7).

Table S3: Clinical, Biochemical, Electrophysiological and Imaging Features to Differentiate Amongst the Common Causes of Acute Chest Pain

Condition	History-taking	Physical examination	Biochemical tests	ECG	Imaging findings
Stable angina/ chronic IHD	<p><i>Chest pain phenotype:</i> retrosternal/central, crushing/heavy/squeezing pain/discomfort, worse on exertion/emotional stress, better with rest/GTN; may radiate to the neck/jaw/arm</p> <p><i>Associated symptoms:</i> exertional dyspnoea, reduced effort tolerance</p> <p><i>Risk factors:</i> CVRFs (smoking, obesity, DM, HTN, HLD, family history of IHD)</p>	<p><i>Vitals:</i> usually stable</p> <p><i>Stigmata of atherosclerosis or cardiovascular disease or microvascular/macrovascular end-organ damage:</i> Frank's sign (diagonal earlobe crease; associated with atherosclerotic coronary artery disease), Levine's sign (clenched fist over anterior chest; sign of ischaemic chest pain), nicotine stains (teeth/fingers; smoking), obese habitus, xanthelasma/tendon xanthoma (hypercholesterolaemia), insulin injection marks/glucometer needle marks (diabetes mellitus), fundoscopic features of diabetic/hypertensive retinopathy, sallow appearance/presence of dialysis catheters or arteriovenous fistula (end-stage renal disease), "gloves-and-stockings" sensory loss (diabetes mellitus-associated peripheral neuropathy); CABG/GSV harvest scar (IHD), unilateral paraparesis (stroke), poorly felt peripheral pulses/limb pallor/trophic skin changes (smooth, shiny, dry hairless skin)/tissue loss (ulcers, gangrene, amputations) (PAD)</p>	<i>Troponins:</i> not elevated	<i>ECG:</i> no new ischaemic changes	<p><i>CXR:</i> usually normal</p> <p><i>TTE:</i> may show known regional wall motion abnormality, depressed left ventricular/right ventricular systolic function</p>
Unstable angina	<p><i>Chest pain phenotype:</i> crescendo pattern of angina (more severe/intense, longer duration >15 minutes, increased frequency), rest angina/less responsive to GTN; may radiate to the neck/jaw/arm</p>	<p><i>Vitals:</i> usually stable</p> <p><i>Stigmata of atherosclerotic disease or cardiovascular disease or end-organ damage:</i> same as for stable angina/chronic IHD</p>	<i>Troponins:</i> not elevated	<i>ECG:</i> may or may not have acute ischaemic ECG changes (new ST depression or T wave inversions in ≥ 2 contiguous leads)	

	<p><i>Associated symptoms:</i> same as for stable angina/chronic IHD</p> <p><i>Risk factors:</i> same as for stable angina/chronic IHD</p>				
Type 1 NSTEMI	<p><i>Chest pain phenotype:</i> typical anginal chest pain, but may have atypical features (esp. in females, elderly) or even silent; may radiate to the neck/jaw/arm</p>	<p><i>Vitals:</i> may have hemodynamic instability (e.g. hypotension/tachycardia due to cardiogenic shock)</p> <p><i>Complications of myocardial ischaemia:</i> post-MI pericarditis (pericardial friction rub), new-onset pansystolic murmur (e.g. papillary muscle rupture, ventricular septal rupture), acute decompensated heart failure (S3 gallop, bibasal lung crepitations, raised jugular venous pressure, bilateral pedal oedema), cardiac tamponade ("Beck's triad" of muffled heart sounds, elevated jugular venous pressure and hypotension), tachy/bradyarrhythmias</p> <p><i>Stigmata of atherosclerotic disease or cardiovascular disease or end-organ damage:</i> same as for stable angina/chronic IHD</p>	<p><i>Troponins:</i> elevated with $\geq 20\%$ rise and/or fall, with at least one value $> 99^{\text{th}}$ percentile of upper reference limit</p>	<p><i>ECG:</i> may or may not have acute ischaemic ECG changes (new ST segment depression or TW inversions in ≥ 2 contiguous leads)</p> <p><i>ECG:</i> new ST segment elevation in ≥ 2 contiguous leads (significant ST elevation fulfils J-point criteria which differs according to age and gender), new-onset LBBB, fulfilment of Sgarbossa's criteria for existing LBBB</p>	<p><i>CXR:</i> may be normal or show congestive features of pulmonary oedema due to acute heart failure</p> <p><i>TTE:</i> shows new regional wall motion abnormalities</p> <p><i>Diagnostic coronary angiogram:</i> shows significant occlusive coronary artery disease</p>
Type 1 STEMI	<p><i>Associated symptoms:</i> dyspnoea, palpitations, nausea/vomiting, diaphoresis, giddiness/syncope</p> <p><i>Risk factors:</i> CVRFs (same as above)</p>				
T2MI	<p><i>Clinical features:</i> chest pain and associated clinical features inconsistent with T1MI; in clinical context of current condition(s)/medical comorbidities that can lead to myocardial ischaemic imbalance; anaemia, sepsis, AKI, tachyarrhythmias, hypotension</p>			<p><i>ECG:</i> may or may not have acute ischaemic ECG changes (new ST segment depression or T-wave inversions in ≥ 2 contiguous leads, ST elevation may also occur if there is transmural ischaemia from the supply/demand mismatch); may show other pathologies (e.g. tachyarrhythmias like atrial fibrillation)</p>	<p><i>CXR:</i> usually normal or shows other pathologies (e.g. consolidative changes of pneumonia)</p> <p><i>TTE:</i> shows new, regional wall motion abnormalities</p> <p><i>Diagnostic coronary angiogram:</i> may or may not show significant occlusive coronary artery disease (the latter if patient has underlying CAD)</p>
Acute pericarditis	<p><i>Chest pain phenotype:</i> sudden-onset sharp, stabbing, pleuritic chest pain, worse on lying supine, better on sitting up and leaning forward</p> <p><i>Associated symptoms:</i> dyspnoea, low-grade fever</p> <p><i>Risk factors:</i> infections, trauma, autoimmune conditions, malignancy, uraemia, recent MI, cardiothoracic surgery, mediastinal irradiation</p>	<p><i>Vitals:</i> usually stable; if unstable (e.g. hypotension), consider possibility of concomitant pericardial effusion/cardiac tamponade</p> <p><i>Key signs:</i> pericardial friction rub</p>	<p><i>Troponins:</i> usually normal, but may be elevated in myopericarditis</p> <p><i>CRP:</i> may be elevated</p>	<p><i>ECG:</i> global saddle-shaped ST segment elevation and PR segment depression in anterolateral leads (V2-V4, I, aVL, V5-V6) and inferior leads (II, III, aVF), reciprocal ST depression and PR segment elevation in aVR, Spodick's sign (downsloping TP segment)</p>	<p><i>CXR:</i> may be normal or show cardiomegaly</p> <p><i>TTE:</i> may show concomitant pericardial effusion</p>

Acute myocarditis	<p><i>Chest pain phenotype:</i> non-specific chest pain/discomfort, but may be pleuritic (myopericarditis)</p> <p><i>Associated symptoms:</i> dyspnoea, prodromal flu-like symptoms (fever, upper respiratory tract symptoms, myalgia)</p> <p><i>Risk factors:</i> recent infection (esp. viral), autoimmune conditions, allergic/hypersensitivity reactions, toxins</p>	<p><i>Vitals:</i> presence of fever, with tachycardia out of proportion to fever, may be haemodynamically unstable (cardiogenic shock)</p> <p><i>Key signs:</i> may have normal cardiovascular examination (if uncomplicated)</p> <p><i>Complications (acute decompensated heart failure):</i> new S3 gallop, bibasal lung crepitations, raised jugular venous pressure, bilateral pedal oedema</p>	<p><i>Troponins:</i> usually significantly elevated</p> <p><i>CRP:</i> may be elevated</p>	<p><i>ECG:</i> sinus tachycardia, non-specific ST-T changes</p>	<p><i>CXR:</i> may show cardiomegaly or congestive features suggestive of pulmonary oedema in heart failure</p> <p><i>TTE:</i> may show non-coronary regional wall motion abnormalities and depressed left ventricular ejection fraction</p> <p><i>CMR:</i> diagnostic aid based on Lake Louise criteria</p>
Takotsubo cardiomyopathy	<p><i>Chest pain phenotype:</i> anginal type chest pain</p> <p><i>Associated symptoms:</i> dyspnoea, palpitations, giddiness, syncope</p> <p><i>Risk factors:</i> post-menopausal females, significant physical/emotional stressor</p>	<p><i>Vitals:</i> may have hemodynamic instability (e.g. hypotension/tachycardia due to cardiogenic shock)</p> <p><i>Complications (acute heart failure):</i> see above</p> <p><i>Complications (left ventricular outflow tract obstruction):</i> systolic murmur</p>	<p><i>Troponins:</i> may be elevated</p>	<p><i>ECG:</i> may show non-specific ST-T changes</p>	<p><i>TTE:</i> most commonly shows ballooning of left ventricular apical region (i.e. apical akinesis), with compensatory hyperkinesis of basal segments – in the apical form of Takotsubo cardiomyopathy</p> <p><i>Diagnostic coronary angiogram:</i> shows no significant occlusive coronary artery disease</p>
Valvular heart disease (e.g. aortic stenosis)	<p><i>Chest pain phenotype (aortic stenosis):</i> anginal chest pain</p> <p><i>Associated symptoms:</i> dyspnoea, syncope</p> <p><i>Risk factors:</i> advanced age, rheumatic heart disease</p>	<p><i>Vitals:</i> usually stable; if unstable, consider acute decompensated heart failure</p> <p><i>Key signs:</i> presence of cardiac murmurs (e.g. aortic stenosis – ejection systolic murmur loudest at right upper sternal edge that radiates to the carotids)</p> <p><i>Complications (acute heart failure):</i> see above</p>	<p><i>Troponins:</i> usually normal</p>	<p><i>ECG:</i> may show left ventricular hypertrophy (in aortic stenosis), associated atrial fibrillation (in rheumatic heart disease)</p>	<p><i>CXR:</i> may show congestive features suggestive of pulmonary oedema in heart failure</p> <p><i>TTE:</i> shows valvular stenosis/insufficiency</p>
Pneumonia	<p><i>Chest pain phenotype:</i> may be pleuritic</p> <p><i>Associated symptoms:</i> infective/respiratory symptoms such as productive cough, rhinorrhoea, sore throat, fever, chills</p>	<p><i>Vitals:</i> may have fever, tachypnoea, poor oxygen saturation</p> <p><i>Key signs:</i> reduced chest expansion, dullness to percussion, bronchial breath</p>	<p><i>Troponins:</i> usually normal, but may be elevated in infection-precipitated Type 2 MI</p> <p><i>ABG:</i> may</p>	<p><i>ECG:</i> usually normal</p>	<p><i>CXR:</i> may show consolidation, with air bronchograms, loss of Silhouette sign, occasionally with parapneumonic effusion</p> <p><i>POCUS:</i> may show hepatisation of lung</p>

	<p>Risk factors: Elderly, multiple medical comorbidities, immunocompromised, positive contact history, recent viral respiratory tract infection, aspiration risks (dysphagia, impaired consciousness, bedridden)</p>	<p>sounds, crepitations/rhonchi</p>	<p>show type 1 or type 2 respiratory failure</p> <p>CRP, procalcitonin: may be elevated</p>		<p>tissue, air bronchograms, parapneumonic effusion with septations</p>
Pulmonary embolism	<p>Chest pain phenotype: may be pleuritic</p> <p>Associated symptoms: dyspnoea, cough/haemoptysis, giddiness/syncope, calf pain/swelling (suggestive of deep vein thrombosis)</p> <p>Risk factors: long haul flight/prolonged immobility, recent surgery, smoking, pregnancy/post-partum state, use of oral contraceptive pills/hormone replacement therapy/tamoxifen, recent COVID-19 infection, previous venous thromboembolic events, known thrombophilias</p>	<p>Vitals: may have fever, tachycardia, tachypnoea, poor oxygen saturation; if presence of persistent hypotension ≥ 15 minutes or requiring inotropes, this suggests massive PE</p> <p>Key signs: features of right heart failure (e.g. raised jugular venous pressure, pedal oedema), cold/clammy peripheries with weak, thready pulses and increased capillary refill time (obstructive shock)</p>	<p>Troponins: may be elevated (suggestive of right heart strain, with poor prognosis)</p> <p>D-dimer: sensitive, but not specific; consider only in low pre-test probability to rule out pulmonary embolism</p> <p>ABG: may show type 1 respiratory failure</p>	<p>ECG: may show sinus tachycardia, right heart strain patterns (e.g. ST depression, T wave inversion in anteroinferior leads, RBBB, right axis deviation, p pulmonale), S1Q3T3</p>	<p>CXR: usually non-specific changes, rarely may show Fleischner sign, Hampton's hump or Westermark sign</p> <p>POCUS: may show right ventricular dilatation with reduced contractility, poor left ventricular filling, subpleural consolidation, right ventricular thrombus; also may show non-compressible femoral/popliteal vein (concurrent deep vein thrombosis)</p> <p>CTPA: shows filling defect in pulmonary artery, may show right ventricular dilatation</p>
Pneumothorax	<p>Chest pain phenotype: may be pleuritic</p> <p>Associated symptoms: dyspnoea</p> <p>Risk factors: tall/thin young male (primary spontaneous pneumothorax), connective tissue disease, underlying lung disease, trauma, thoracic surgery/procedures</p>	<p>Vitals: if presence of haemodynamic instability (hypotension, tachycardia), suggestive of tension pneumothorax with obstructive shock</p> <p>Key signs: tracheal deviation, elevated jugular venous pressure, cyanosis (tension pneumothorax), reduced chest expansion, hyperresonant percussion note, decreased air entry, subcutaneous emphysema, traumatic chest wounds</p>	<p>Troponins: usually normal</p> <p>ABG: may show type 1 or type 2 respiratory failure</p>	<p>ECG: non-specific</p>	<p>CXR: shows visible visceral pleural edge, devoid of lung markings peripheral to it and a collapsed lung central to it, may demonstrate tracheal deviation/mediastinal shift, subcutaneous emphysema</p> <p>POCUS: absent lung sliding, stratosphere sign</p>
Aortic dissection	<p>Chest pain phenotype: severe, tearing chest pain, sudden and maximal at onset, radiates to the back</p> <p>Associated symptoms: dyspnoea, syncope, neurological</p>	<p>Vitals: likely to show haemodynamic instability, inter-arm systolic blood pressure differentials (> 20 mmHg)</p> <p>Key signs: presence</p>	<p>Troponins: may be elevated (in coronary backflow)</p>	<p>ECG: normal or non-specific ST/T changes</p>	<p>CXR: widened mediastinum (> 8 cm), widened right paratracheal stripe, "calcium sign", tracheal deviation to the right</p> <p>TTE: more</p>

	<p>symptoms</p> <p><i>Risk factors:</i> hypertension, connective tissue disease, trauma</p>	<p>of radial-radial delay, new-onset aortic regurgitation</p> <p><i>Complications (acute heart failure):</i> see above</p>			<p>sensitive/specific for type A dissection than type B dissection</p> <p><i>TEE:</i> preferred in clinically unstable patients</p> <p><i>CT aortogram:</i> preferred in clinically stable patients</p>
Boerhaave's syndrome	<p><i>Chest pain phenotype:</i> sudden onset, severe, subxiphoid chest pain</p> <p><i>Associated symptoms:</i> preceding vomiting/retching, dyspnoea</p> <p><i>Risk factors:</i> binge-eating, alcoholism</p> <p><i>*Mackler's triad:</i> chest pain, preceding vomiting, subcutaneous emphysema</p>	<p><i>Vitals:</i> likely to show haemodynamic instability</p> <p><i>Key signs:</i> reduced breath sounds over lung fields on affected side, subcutaneous emphysema, Hamman's sign (crunching sound over the precordium in sync with heartbeat; suggests pneumomediastinum)</p>	<i>Troponins:</i> usually normal	<i>ECG:</i> usually normal	<p><i>CXR:</i> may show mediastinal widening, pneumomediastinum, subcutaneous emphysema, "V" sign</p> <p><i>CT thorax:</i> shows oesophageal wall thickening, soft tissue gas in thoracic cavity, pneumomediastinum</p>
Gastro-oesophageal reflux disease	<p><i>Chest pain phenotype:</i> burning, retrosternal chest pain, worse after meals/lying flat, better with antacids</p> <p><i>Associated symptoms:</i> sour/bitter taste at back of the throat, nocturnal cough</p> <p><i>Risk factors:</i> smoking, alcohol, obesity, consumption of fatty/spicy food</p>	<p><i>Vitals:</i> stable</p> <p><i>Key signs:</i> usually normal examination</p>	<i>Troponins:</i> normal	<i>ECG:</i> normal	Not required, unless presence of red-flag symptoms
"Acute abdomen" (e.g. PUD, acute cholecystitis/pancreatitis, perforated viscus)	<p><i>Chest pain phenotype:</i> usually substernal, extremely severe; pain radiation to right shoulder (cholecystitis), back (pancreatitis)</p> <p><i>Associated symptoms:</i> nausea/vomiting, bloating/belching</p> <p><i>Risk factors:</i> NSAIDs use (PUD), history of <i>H. pylori</i> colonisation (PUD), known gallstone disease (cholecystitis, pancreatitis), alcoholic binge (pancreatitis)</p>	<p><i>Vitals:</i> may show haemodynamic instability</p> <p><i>Key signs:</i> epigastric/right hypochondrial tenderness, features of peritonism (guarding/rebound tenderness)</p>	<i>Troponins:</i> may be normal or elevated if underlying condition precipitates myocardial injury	<i>ECG:</i> usually normal	<p><i>CXR:</i> may show air under the diaphragm (in perforated viscus)</p> <p><i>US abdomen</i> or <i>CTAP:</i> may aid in diagnosis of intra-abdominal or hepatobiliary pathology</p>
Musculoskeletal/soft tissue pathologies (e.g. rib fractures, muscle sprain, costochondritis, Herpes zoster, intercostal)	<p><i>Chest pain phenotype:</i> usually localised, sharp/stabbing, reproducible with palpation of affected area or on chest wall movements</p>	<p><i>Vitals:</i> usually stable</p> <p><i>Key signs:</i> focal tenderness on palpation of affected region, superficial wounds/bruises, presence of</p>	<i>Troponins:</i> normal	<i>ECG:</i> normal	Not required

neuralgia)	<p><i>Associated symptoms:</i> recent chest wall trauma</p> <p><i>Risk factors:</i> previous VZV infection (for Herpes zoster, post-herpetic intercostal neuralgia)</p>	vesicular rash over anterior chest wall in a dermatomal distribution (Herpes zoster)			
Psychogenic (e.g. somatisation, anxiety, panic attacks)	<p><i>Chest pain phenotype:</i> non-specific</p> <p><i>Associated symptoms:</i> perioral anaesthesia, numbness/tingling sensation, other non-specific symptoms</p> <p><i>Risk factors:</i> history of psychiatric disorders (anxiety, somatisation disorders), recurrent admissions for atypical chest pain</p>	<p><i>Vitals:</i> usually stable</p> <p><i>Key signs:</i> normal examination</p>	<i>Troponins:</i> normal	<i>ECG:</i> normal	Not required

* ABG: arterial blood gas; AKI: acute kidney injury; CABG: coronary artery bypass graft; CAD: coronary artery disease; CKD: chronic kidney disease; CRP: C-reactive protein; CMR: cardiac magnetic resonance imaging; CT: computed tomography; CTPA: computed tomography pulmonary angiogram; CVRFs: cardiovascular risk factors; CXR: chest X-ray; DM: diabetes mellitus; GERD: gastroesophageal reflux disease; GSV: great saphenous vein; GTN: glyceryl trinitrate; HLD: hyperlipidaemia; HTN: hypertension; IHD: ischaemic heart disease; NSTEMI: non-ST segment elevation myocardial infarction; PAD: peripheral arterial disease; POCUS: point-of-care ultrasound; PUD: peptic ulcer disease; STEMI: ST segment elevation myocardial infarction; T1MI: type 1 myocardial infarction; T2MI: type 2 myocardial infarction; TEE: transoesophageal echocardiogram; TTE: transthoracic echocardiogram

Table S4: Causes of Hypertroponinaemia

	Causes
Cardiac (coronary artery)	Myocardial infarction Coronary artery vasospasm Coronary artery dissection Aortic dissection with coronary extension Post-percutaneous coronary intervention
Cardiac (non-coronary artery)	Tachyarrhythmias (e.g. atrial fibrillation, supraventricular tachycardia) Myocarditis/myopericarditis Cardiomyopathies Infective endocarditis Infiltrative cardiac disease (e.g. amyloidosis, sarcoidosis) Heart failure (acute, chronic) Hypertensive crises Chest trauma/cardiac contusions Cardiac procedures (e.g. CPR, defibrillation, transvenous pacing, ablation procedures, cardiothoracic surgery) Cardiotoxins (e.g. anthracyclines, herceptin, recreational drugs such as cocaine)
Non-cardiac	Anaemia Sepsis Critical illness Viral infection (e.g. influenza infection) Pulmonary embolism Pulmonary hypertension Acute respiratory distress syndrome Chronic obstructive pulmonary disease (COPD) exacerbation Renal impairment (acute, chronic) Stroke Subarachnoid haemorrhage Seizures Strenuous exercise Rhabdomyolysis Burns
False positives	Heterophile antibodies Positive rheumatoid factor Elevated alkaline phosphatase Presence of fibrin clots

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