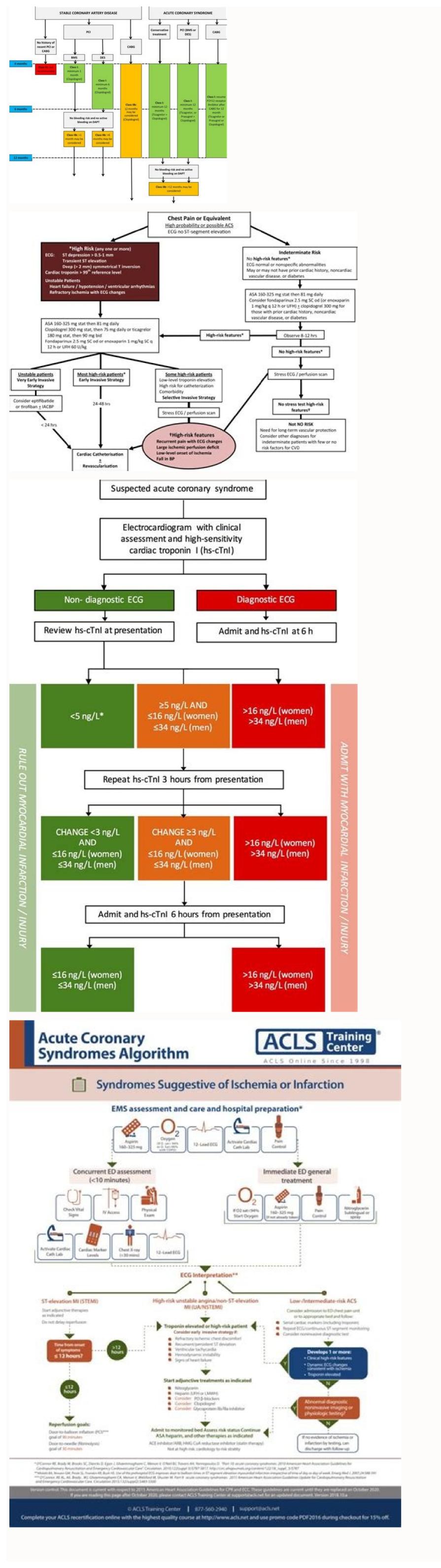
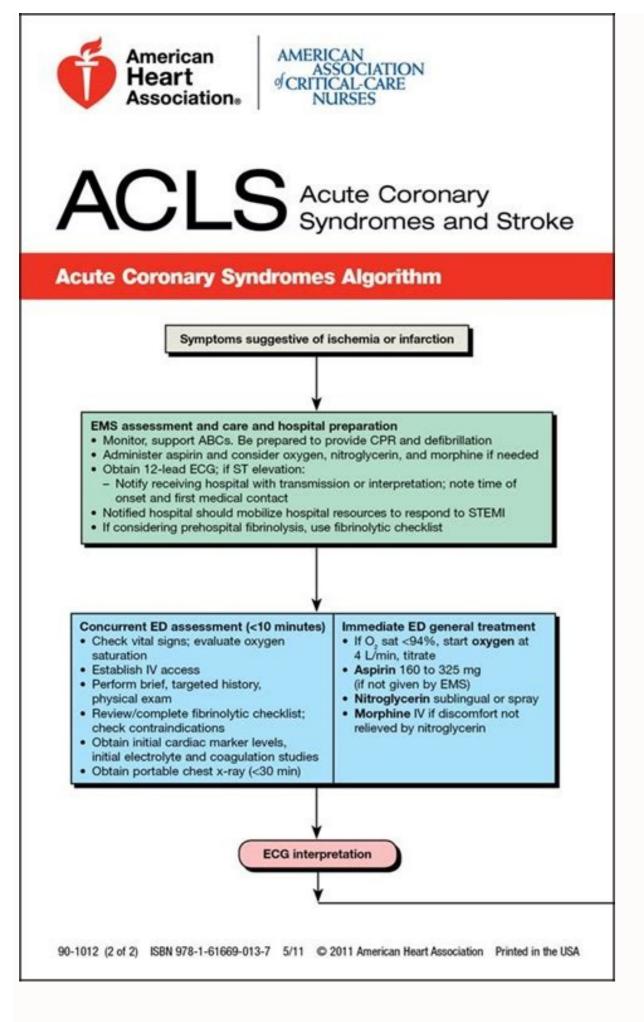
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Chest pain is a common presentation, and the diagnoses are variety of diagnoses and can be subcategorised into: You might also be interested in our medical flashcard collection which contains over 2000 flashcards that cover key medical topics. Aetiology Two of the three diagnoses that exist under the umbrella of ACS are types of myocardial infarction (STEMI and NSTEMI). Definition of MI is as follows... Detection of a rise and/or fall in cardiac biomarker values (preferably troponin) with at least one value above the 99th percentile (upper reference limit) with at least one of the following: Symptoms of ischaemia (the patient's history and clinical presentation of the tests you are about to perform) New or presumed new significant ST segment or T wave changes or new left bundle branch block (LBBB) Pathological Q wave changes on the ECG Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality Identification of an intracoronary thrombus by angiography A 'new left bundle branch block' does not mean 'a left bundle branch block that has just been found because the patient has never had an ECG before'. Nor does it mean 'a left bundle branch block that is present now but was not present on an ECG in 2006'. A new LBBB due to ischaemia is the result of an occluded proximal left anterior descending (LAD) or left main stem artery. A large amount of myocardium and conductive tissue needs to be affected to cause this ECG appearance and these patients are usually acutely unwell. Development of Q waves on the ECG The Q wave reflects septal depolarisation. If the ventricular wall is dead, a 'window' is created that allows the septal depolarisation to show up on the surface ECG. Imaging evidence of new regional wall abnormality The sequence of changes and signs of ischaemic cascade) on various tests is shown in Figure 1. Ischaemic cascade flowchart Troponin A troponin level is only a number. It is only relevant when applied in the context of other clinical findings. There are many causes of a raised troponin, including: Myocardial infarction Tachy/bradyarrhythmias Aortic dissection Severe anaemia Coronary spasm Heart failure Stroke Subarachnoid haemorrhage Classification of myocardial infarctions Type 1: spontaneous myocardial infarction This is your 'typical' myocardial infarction. Atherosclerosis originates from damage to the endothelium and a build-up of cholesterol. The inflammatory reaction to atherosclerosis by macrophages causes a plaque of foam cells, lipids, cellular debris and (eventually) calcium to build up with a fibrous cap on top. When this cap cracks, the exposed debris triggers thrombus formation in the confines of the affected coronary artery (which are only typically between 2 and 5mm in diameter), causing partial or total occlusion of the artery. Type 2: myocardial infarction is a common event in hospitals where patients with stable coronary artery disease +/- previous coronary intervention (PCI or CABG) are unwell and put additional stress on their heart that would not normally be present. If there is a sufficient imbalance between the supply of blood (e.g. sepsis/hypovolaemic shock, tachyarrhythmia) then the myocardium can become ischaemic without a plaque rupture event. Type 3 MI: post-mortem A type 3 myocardial infarction is diagnosed post-mortem. Type 4a MI: percutaneous coronary intervention (i.e. caused by an angioplasty procedure blocking a side branch or damaging the main coronary artery causing ischaemia) Type 4b MI: stent thrombosis This is related to stent thrombosis or NSTEMI if complete sudden thrombosis or NSTEMI if gradual re-stenosis over time). Type 5 MI: bypass Type 5 myocardial infarction is related to a bypass graft (CABG) operation. Clinical features The clinical features of ACS are similar, regardless of the underlying diagnosis (e.g. NSTEMI, STEMI or unstable angina). Typical clinical features of ACS are similar, regardless of the underlying diagnosis (e.g. NSTEMI, STEMI or unstable angina). with nausea and vomiting, marked sweating and breathlessness chest pain associated with haemodynamic instability new-onset chest pain occurring frequently and with little or no exertion, and with episodes often lasting longer than 15 minutes. For more information, see the Geeky Medics guide to a chest pain history. Management When ACS is suspected, initial management should be commenced as soon as possible: Resting 12-lead ECG Morphine Oxygen: only if low SpO2 Nitrates (e.g. glyceryl trinitrate) Aspirin (300 mg) and a second rapid-acting antiplatelet such as Ticagrelor or Prasugrel For more information, see the Geeky Medics guide to the emergency management of ACS. ST-elevation ACS ST-elevation and persistent (>20 minutes) ST-segment elevation. ST elevation is a sign of complete occlusion of an epicardial coronary artery by thrombus causing immediate myocardial death relating to the territory affected: Anterior: V1-V4 Inferior: V1-V4 Inferior: II, III, aVF High lateral: I, aVL Low lateral: V5, V6 Posterior: a dominant R wave in V1-3 with ST depression in V1 the artery. Management of STEMI3 The current gold standard treatment option is primary percutaneous intervention (PCI) to allow the vessel to be opened as quickly as possible (as 'time is muscle'). Studies have shown that it is beneficial to delay treatment for up to 90 minutes to allow transfer to a primary PCI centre rather than resorting to thrombolysis. There is a benefit associated with performing primary PCI up to 12 hours from the occluded artery as the damage is already done. 4 The recommended treatment used to be thrombolysis to try and break down the clot in the artery. However, this has significant bleeding risks attached to it and often failed to open the artery up requiring the patient to undergo 'rescue angioplasty' with higher bleeding risks associated due to the pre-medication. Non-ST-elevation myocardial infarction (NSTEMI) Non-ST elevation myocardial infarction (NSTEMI) are often seen as a more 'routine' heart attack. However, they have worse long-term outcomes than STEMIs (although a lower risk of death in the short term). They tend to be associated with partial coronary occlusion. Diagnosis of an NSTEMI involves a combination of, clinical assessment, serial troponin measurement and ECG analysis (see definition of MI earlier in the article). For more information, see the Geeky Medics guide to NSTEMIs. Management of NSTEMIs is confirmed (with serial troponin measurement): Beta blockade (or alternative rate-limiting agent if contraindicated - aim HR of 50-60 bpm) ACE inhibitor (unless contraindicated - aim for a systolic blood pressure of 120 mmHg or less) Atorvastatin 80mg OD Once someone is diagnosed with a type I NSTEMI and commenced on appropriate medical therapy, there are various risk scores that can be calculated to assess the value of invasive angiography. NICE recommends the use of the GRACE score which is used to predict in-hospital and post-discharge to 6-month mortality. Other factors that should be considered before proceeding to angiography include: Renal function (there is a risk of contrast nephropathy and this can leave predialysis patients permanently dependent on dialysis) Bleeding risk (as above) Can the patient lie flat? Although angiography can be performed whilst patients with severe heart failure are sat upright, it is not fun for the operator, cath lab team or the patient. In a non-urgent setting, angiography should be delayed until the patient is able to tolerate lying flat. Other significant co-morbidities: patients with poor quality of life and/or short life expectancy (age/dementia/malignancy) may not gain any prognostic benefit from the procedure and should be managed medically. Offer conservative management without early coronary angiography (with follow-on PCI if indicated) to patients initially assessed to be at low risk of adverse cardiovascular events (predicted 6-month mortality 3.0% or less) if ischaemia is subsequently experienced or is demonstrated by ischaemia testing. Offer coronary angiography (with follow-on PCI if indicated) within 96 hours of first admission to hospital to patients who have an intermediate or higher risk of adverse cardiovascular events (predicted 6-month mortality above 3.0%) if they have no contraindications to angiography (such as active bleeding or comorbidity). Perform angiography as soon as possible for patients who are clinically unstable or at high ischaemic risk. Unstable angina Stable (or 'exertional') angina is defined as: Typical cardiac pain Brought on by exertion and relieved by rest Lasting less than 20 minutes Unstable angina is an acute coronary syndrome that is defined by the absence of biochemical evidence of myocardial damage. It is characterised by specific clinical findings of: 6 prolonged (>20 minutes) angina at rest new onset of severe angina angina that is increasing in frequency, longer in duration, or lower in threshold angina that occurs after a recent episode of myocardial infarction Diagnosis of unstable angina is based on clinical assessment. Troponin measurements will be normal as there has not been any ischaemic damage (yet) and the ECG may be normal for similar reasons. Management of unstable angina Management of unstable angina is the same as for NSTEMI discussed above. Editor Dr Chris Jefferies References Third Universal Definition of Myocardial Infarction); NICE Quality Standard, Sept 2014. Available from: [LINK] NICE Guideline. Myocardial infarction with ST-segment elevation: acute management. Clinical guideline [CG167]. Published date: 10 July 2013. Available from: [LINK] Yousef ZR, Redwood SR, Bucknall CA, Sulke AN, Marber MS. Late intervention after anterior myocardial infarction: effects on left ventricular size, function, quality of life, and exercise tolerance: results of the Open Artery Trial (TOAT Study). J Am Coll Cardiol 2002;40:869-876 NICE Guideline. Unstable angina and NSTEMI; NICE Clinical Guideline (March 2010 - last updated November 2013). Available from: [LINK] Roffi M, Patrono C, Collet JP, et al. 2015 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). Eur Heart J. 2016 Jan 14;37(3):267-315.

Aug 19, 2020 · ESC Clinical Practice Guidelines aim to present all the relevant evidence to help physicians weigh the benefits and risks of a particular diagnostic or therapeutic procedure on Acute Coronary Syndromes (ACS) in patients presenting without persistent ST-segment elevation. They should be essential in everyday clinical decision making. Drug therapy and choice of revascularization depend on the type of acute coronary syndrome as well as ... intensive glucose control is no longer recommended; guidelines call for an insulin-based ... representatives of the European Association ... May 18, 2021 · Acute coronary syndrome refers to many conditions that cause sudden, low blood flow to the heart. Know the symptoms, causes and ... Aug 29, 2019 · Guidelines summarize and evaluate available evidence with the aim of assisting health professionals in proposing the best management strategies for an individual patient with a given condition Guidelines and their recommendations should facilitate decision making of health professionals in their daily practice. However, the final decisions concerning an ... Dec 14, 2020 · The term 'acute coronary syndrome' (ACS) covers a range of disorders, including a heart attack (myocardial infarction) and unstable angina, that are caused by the same underlying problem. ... 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management ... May 18, 2021 · Acute coronary syndrome usually results from the buildup of fatty deposits (plaques) in and on the walls of coronary arteries, the blood vessels delivering oxygen and nutrients to heart muscles. When a plaque deposit ruptures or splits, a blood clot forms. This clot blocks the flow of blood to heart muscles. When a plaque deposit ruptures or splits, a blood clot forms. This clot blocks the flow of blood to heart muscles. When a plaque deposit ruptures or splits, a blood clot forms. This clot blocks the flow of blood to heart muscles. When a plaque deposit ruptures or splits, a blood clot forms. This clot blocks the flow of blood to heart muscles. Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation (NIV) is widely used in the acute care setting for acute respiratory failure (ARF) across a variety of aetiologies. This document provides European Respiratory Society/American Thoracic Society recommendations for the clinical application of NIV based on the most current literature. The guideline committee was composed of clinicians, ...

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