ACUTE CORONARY SYNDROME
ANAESTHESIA TUTORIAL OF THE WEEK 210

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QUESTIONS

Before continuing, try to answer the following questions. The answers can be found at the end of the article, together with an explanation.

1. Which of the following statements is correct?
    a. Acute coronary syndrome patients include those with unstable angina and all types of myocardial infarction.
    b. Acute coronary syndrome patients include those with stable angina.
    c. Myocardial infarction causes serum cardiac enzymes to be raised.
    d. It is possible to have a myocardial infarction without having ECG ST segment changes.

2. What signs and symptoms might indicate a diagnosis of myocardial infarction?

3. Which of these systems have been used to predict cardiac risk?
    a. Lees cardiac risk index
    b. Detsky cardiac risk index
    c. Eagle cardiac risk index
    d. Goldman cardiac risk index
    e. ACC AHA guidelines
    f. All of the above

4. How would you manage a patient you suspect is having an intraoperative myocardial infarction?

DEFINITIONS

- Acute coronary syndrome (ACS): unstable angina or myocardial infarction with or without ST segment elevation on ECG.
- Unstable angina: myocardial ischaemia of increasing duration or severity in the absence of cardiac myocyte necrosis.
- ST segment elevation myocardial infarction (STEMI): myocardial necrosis (evidenced by raised cardiac enzymes on blood test) with ST segment elevation on ECG.
- Non-ST segment elevation myocardial infarction (NSTEMI): myocardial necrosis (evidenced by raised cardiac enzymes on blood test) with no ST segment elevation on ECG.

CLASSIFICATION IS IMPORTANT

Distinction between angina, unstable angina, NSTEMI and STEMI is clinically relevant for several reasons. The prognosis is progressively worse for each clinical group. The STEMI group have the poorest prognosis, with one third of this group dead within 24 hours of onset of ischaemia.

Treatment is different depending on the clinical classification. Patients with STEMI are treated with urgent attempt at reperfusion. Percutaneous coronary intervention is the preferred treatment if available. ACS patients not having a STEMI are generally not rushed to urgent reperfusion procedures. They are often stratified into low and high risk groups. Medical management is optimized and some are considered for reperfusion treatment.

RISK FACTORS

The two most significant risk factors for developing coronary vessel atherosclerosis are increasing age and male gender. Other risk factors include cigarette smoking, hypertension, obesity and sedentary lifestyle, high cholesterol, diabetes mellitus, family history of premature coronary artery disease.

PATHOPHYSIOLOGY

Atherosclerosis has multiple pathological processes. Coronary arteries become stenosed over time with lipid rich atheroma deposited in the sub-endothelial layer. An initial tissue injury and accumulation of macrophages at the area leads to plaque development. Eventually the plaque includes a macrophage rich lipid core covered by a protein fibrin mesh cap. Plaque deposits tend to accumulate around bifurcations of arteries, and cause symptoms once the lumen diameter is reduced by over fifty percent.

Blood flow is compromised when the lumen is blocked by a combination of vessel plaque, thrombus and inflammatory mediators. Thrombus formation can occur when plaques rupture or ulcerate causing exposure of tissue factor and initiation of clotting at the site. Glycoprotein IIb/IIIa receptors on platelets are activated causing growth and stabilisation of the thrombus. Chemical mediators include collagen, adrenaline, adenosine, and serotonin. Thromboxane A2 is a potent vasoconstrictor and its release further compromises blood flow.

Inflammation is increasingly becoming recognised as a major factor. Inflammatory cells are present in plaques and are important in the cascade of events leading to plaque rupture. Inflammatory markers such as C-reactive protein and fibrinogen are increased in people at high risk of ischaemic heart disease.

Plaques that rupture and lead to acute coronary occlusion (i.e. STEMI) are rarely large enough to occlude the vessel by plaque alone. Flow restrictive plaques that cause angina are less likely to rupture. Abrupt decrease in luminal blood flow causes STEMI. Occasionally STEMI can occur from acute spasm of the coronary artery or coronary artery embolus.

DIAGNOSIS

A clinical history of worsening angina or new chest pain is common. Pain may also be reported in the neck, jaw, arms and back. Symptoms may include: sweating, feeling generally unwell, fatigue, dyspnoea and nausea. A typical description of ‘crushing’ central chest pain is not always given, indeed as many as half of all myocardial infarctions are clinically silent. A ‘silent’ myocardial infarction is much more likely in patients with diabetes mellitus.

Diagnosis of acute myocardial infarction requires at least two of the following;

1. chest pain
2. electrocardiograph (ECG) changes consistent with myocardial infarction.
3. increase and decrease of serum cardiac enzymes.

Every patient with suspicious symptoms or clinical history should receive timely assessment, ECG and serum cardiac enzyme evaluation. These tests can confirm STEMI or NSTEMI, but if negative do not necessarily exclude a diagnosis of ACS. Further assessment by a cardiologist and exercise stress testing, pharmacological stress testing, or coronary angiography may be required.

Diagnosis of ACS in an unconscious patient under anaesthesia can be more challenging. Here the anaesthetist is guided by clinical history, relevant risk factors, physical examination, and intraoperative monitoring (including ECG and haemodynamics). Trends in filling pressures and cardiac output can be useful if a pulmonary artery catheter is present. Transoesophageal echocardiography (TOE) is very sensitive for detecting acute segmental wall motion abnormalities and can be detected before ECG changes occur. TOE is a very useful intraoperative procedure in the patient at high risk of intraoperative ACS.

Table 1: Diagnosis of myocardial infarction

SIGNS	SYMPTOMS	BEDSIDE INVESTIGATIONS	LABORATORY AND IMAGING STUDIES
Anxiety Pallor Diaphoresis Sinus tachycardia Hypotension Pulmonary oedema Tachypnoea Cardiac murmur	Chest Pain Jaw, neck, arm pain Generally unwell Fatigue Dyspnoea Nausea Fear 'impending doom'	ECG Hypertension or hypotension Tachycardia Hypoxia (if pulmonary oedema from right heart failure)	Troponin CKMB Echocardiography Radionucleotide imaging (is of limited use in acute MI)

TREATMENT

General principles

It is critical to urgently assess the patient and commence treatment. The general principles are to improve myocardial oxygen supply and minimise oxygen demand. Oxygen supply can be increased by adequate ventilation, adequate oxygenation, low normal heart rate and normal haemoglobin. Oxygen demand can be decreased by maintaining low normal heart rate, maintaining normal blood pressure, avoiding shivering, avoiding acidosis and treating sepsis. Good initial and subsequent management can prevent angina from progression to MI, and prevent MI from progression to massive MI.

Initial management

Timely patient assessment is imperative. This includes administration of supplemental oxygen, assessment of haemodynamic stability and obtaining a twelve lead ECG. Pain relief with morphine and GTN are important to decrease preload and catecholamine release and hence minimise myocardial work. Aspirin is administered to minimise further clot development. Patients who are intolerant of aspirin can be given an ADP receptor antagonist such as clopidogrel.

Subsequent management

The focus of subsequent treatment is to re establish coronary perfusion as soon as possible. The patient must be carefully monitored with continuous ECG looking for development of dysrhythmias. There are medical, surgical and cardiac catheter interventional treatments available.

Direct coronary angioplasty

This should ideally be performed within 90 minutes of arrival at hospital and within 12 hours of symptom onset. This is the treatment of choice in patients where thrombolytic agents are contraindicated (such as recent surgery), or if severe cardiac or respiratory failure are present. Five percent of patients with acute MI having immediate angioplasty will require conversion to a surgical treatment due to unsuitable anatomy or failed angioplasty. Gylcoprotein IIb/IIIa inhibitors are commonly used during emergency PCI procedures to enhance retrograde coronary flow and decrease the need for subsequent revascularisation procedures.

Coronary artery bypass graft

CABG is performed to restore perfusion in an occluded coronary artery. The increased time to achieving reperfusion for CABG over thrombolytic or PCI treatment is clinically relevant in acute MI. Emergency CABG is used for patients who have coronary anatomy unsuitable for PCI, patients with

failed attempt at PCI, and those with infarction related ventricular septal defect or mitral regurgitation. Mortality after CABG is significant in the first 3 to 7 days after acute MI.

Reperfusion therapy

Options for thrombolytic therapy include streptokinase, tissue plasminogen activator, reteplase and tenecteplase. Thrombolytic therapy should be initiated within 60 minutes of arrival at hospital. The aim of this treatment is to restore coronary antegrade ciriculation. Delay in initiating therapy produces less effective clot resolution. Thrombolytic therapy can cause significant harm due to haemorrhage. Intracranial haemorrhage is more common in patients over 75 years of age and gastrointestinal haemorrhage is not uncommon. Thrombolytic therapy is not recommended in patients with unstable angina or NSTEMI, and can not be used in the perioperative period.

Adjunctive medical therapy

Intravenous heparin is commonly used for the first 48 hours after MI to decrease thrombus generation. This can be unfractionated heparin, low molecular weight heparin, or direct thrombin inhibitors. Low molecular weight heparin has the advantages of a more predictable pharmacological profile, long plasma half-life and convenient subcutaneous dosing.

Use of betablockers is helpful in long term management of ACS, however its use in the actue MI setting is controversial. Early administration can decrease infarct size by decreasing heart rate, contractility and blood pressure. Other evidence suggests that use of IV beta blockers in acute MI can worsen acute cardiogenic shock.

Statin therapy has been shown to be useful outside of the acute setting by preventing major coronary events, and decreasing risk of death and major coronary events after ACS is diagnosed.

ACE inhibitors or angiotension II receptor antagonists are given to patients with large anterior wall MI, clinical evidence of left ventricular failure, an ejection fraction less than 40% or diabetes.

PREOPERATIVE ASSESSMENT OF PATIENTS WITH ISCHAEMIC HEART DISEASE

Various models of cardiac risk assessment have been described over the years. The Goldman Cardiac Risk index (1977) was a popular system for many years, and Lee's modified Cardiac Risk Index (1999) is based on the Goldman system. Other models included the Detsky (1986) and Eagle (1989) Cardiac Risk Indices.

Lees Cardiac Risk Index gives one point each for various risk factors (high risk surgery, history of ischaemic heart disease, history of congestive cardiac failure, history of cerebrovascular disease, preoperative insulin, preoperative creatinine > 177 mmol/L).

The ACC/AHA guidelines for preoperative assessment of patients with heart disease (2002/2006/2007/2009) describe classification of patients into a) surgical risk factors (high, intermediate, low) and b) patient risk factors (active cardiac conditions, clinical risk factors, minor predictors). The perioperative risk and planned pre operative intervention are determined by these classifications. For more details of the ACC AHA guidelines and how they effect preoperative planning, see the weblinks at the end of this article.

Table 2a: ACC AHA Recommendations for preoperative risk assessment for cardiac patients (2007): Surgical Factors.

HIGH RISK >5% risk death/non fatal MI	INTERMEDIATE RISK 1-5% risk death/non fatal MI	LOW RISK <1% risk death/non fatal MI
1. major emergency surgery 2. aortic or other major vascular surgery 3. peripheral vascular surgery 4. surgery with large fluid shifts/blood loss	1. intraperitoneal and intrathoracic surgery 2. carotid surgery 3. head and neck surgery 4. prostate surgery 5. orthopaedic surgery 6. endovascular AAA	1. endoscopy 2. superficial surgery 3. breast surgery 4. cataract surgery 5. ambulatory surgery

Table 2b: ACC AHA Recommendations for preoperative risk assessment for cardiac patients (2007): Patient Factors.

ACTIVE CARDIAC CONDITIONS	CLINICAL RISK FACTORS	MINOR PREDICITORS
1. unstable coronary syndromes (MI within 1 month, Canadian class III or IV angina). 2. decompensated heart failure 3. significant arrhythmia 4. severe valvular disease	1. ischaemic heart disease 2. compensated or prior congestive heart failure 3. stroke 4. diabetes 5. renal impairment (creatinine >175 mmol/L)	1. age over 70 years 2. abnormal ECG 3. rhythm other than sinus 4. uncontrolled HTN

PERIOPERATIVE ACUTE CORONARY SYNDROME

Perioperative MI occurs with suprising frequency. For all surgical patients over 40 years of age there is a 2.5% risk of perioperative MI, which increases up to 25% risk in patients with pre-existing ACS. Surgery places a patient at increased risk of ACS events for many reasons. Possible harmful effects of anaesthesia and surgery include: hypoxia, hypotension, hypertension, blood loss causing anaemia, arrhythmogenic electrolyte disturbances, inflammation, blood sugar instability in diabetics, acute stress response and avoidance of anticoagulation.

PERIOPERATIVE ACUTE CORONARY SYNDROME RISK REDUCTION

Risk reduction for perioperative MI can be guided by the ACC AHA surgical and patient risk factors for that patient. Those with active cardiac conditions, unstable lesions, or critical lesions of the left main coronary artery should have surgery delayed, cardiologist review, active problem stabilized, optimisation of medical management and may require revascularisation procedures. If a patient has had a coronary stent placed recently, surgery must be delayed until a minimum course of antiplatelet treatment is completed (1 month for bare metal stents, and 1 year for drug eluting stents).

For patients with no active cardiac conditions and stable ischaemic heart disease, invasive cardiac investigation and treatment is not generally recommended unless it would be recommended in the routine treatment of the patients ischaemic heart disease regardless of the proposed surgery. There is evidence that preoperative revascularisation for patients with stable ischaemic heart disease is not beneficial. This is in part due to the fact that patients with stable coronary disease are much more likely to suffer perioperative MI due to acute plaque rupture without critical stenosis preoperatively. For these patients prevention should focus on stabilization of plaques and minimizing the surgical stress response.

There is evidence for perioperative cardiac risk reduction through use of: statins, aspirin, ADP receptor antagonists (such as clopidogrel), and normothermia. There is some evidence for risk reduction with insulin therapy and beta blockers, although the appropriate dose and duration is still being determined. Volatile anaesthetic agents are known to be useful for ischaemia preconditioning and may have a role in risk reduction, although this has not yet been well demonstrated.

INTRAOPERATIVE ACUTE CORONARY SYNDROME MANAGEMENT

It is important to be alert for the evolution of intraoperative MI. Continuous monitoring of heart rate, blood pressure and ECG trends are imperative. The anaesthetist must be vigilant for warning signs. If

introperative MI is suspected, this emergency situation should be discussed between the surgical and anaesthetic team. Concluding the surgery should be considered and senior staff members must be involved. If STEMI is present, cardiologist involvement is required urgently as revascularistaion may be the treatment of choice.

The main anaesthetic aims to manage intraoperative MI include: oxygenation, maintain optimal haemodynamics, minimise cardiac work, treat arrhythmias, consider use of aspirin and heparin, consider use of gylceryl trinitrate, and to consider use of intraoperative TOE (where available). It is important to monitor for cardiogenic shock (look for elevated filling pressures, low cardiac output, and signs of tissue hypoperfusion). If cardiogenic shock does not respond to pharmacologic support, an intra aortic balloon pump may be required (if there is not significant aortic valve incompetence).

The priorities are to detect intraoperative MI early, give effective management and transfer the patient urgently to further cardiac care.

POSTOPERATIVE ACUTE CORONARY SYNDROME MANAGEMENT

Appropriate follow up will depend on the clinical situation. For minor ECG changes intraoperatively with stable haemodynamics, the minimum post operative care would include 12 lead ECG in recovery and monitoring for changes with serum cardiac enzymes. If there is a diagnosis of STEMI, NSTEMI or unstable angina, cardiology referral must be made. Suitable post operative ward locations might include cardiology wards with continuous telemetry, high dependency or intensive care.

SUMMARY

- Patients with ACS have a high perioperative risk for MI
- The anaesthetist must carefully assess and optimise ACS patients prior to surgery
- Intraoperative MI requires prompt management to prevent further myocardial damage or patient death

ANSWERS TO QUESTIONS

1a: True
1b. False, patients must progress to unstable angina before they meet the criteria for diagnosis of ACS.
1c. True, MI typically causes raised serum troponin and CKMB. There may also be chest pain and ECG changes.
1d. True, an MI without ST changes on ECG is classified as a non ST segment elevation MI (NSTEMI).

2. See table One.

3. f is the correct option. All these systems have been used as predictors of cardiac risk. The ACC AHA guidelines are very commonly used in many countries currently.

4. Intraoperative MI is an emergency. Discuss with the surgeon and consider completing surgery quickly. Control haemodynamics, minimize cardiac work, treat arrhythmias, consider drug treatment (aspirin, heparin, GTN), consider further investigation such as trans oesophageal echocardiography, and consider if urgent revascularisation is indicated.

WEBLINKS

European heart journal guidelines for pre operative cardiac risk assessment: http://www.escardio.org/guidelines-surveys/esc-guidelines/GuidelinesDocuments/guidelines-perioperative-cardiac-care-FT.pdf

ACC AHA 2009 updates on guidelines for managing STEMI, and PCI: http://circ.ahajournals.org/cgi/reprint/CIRCULATIONAHA.109.192663

ACC AHA 2009 update of guidelines for assessment of cardiac patients preoperatively: http://circ.ahajournals.org/cgi/reprint/CIRCULATIONAHA.109.192690

ACC AHA 2007 guidelines for assessment of cardiac patients preoperatively: http://www.asefiles.org/2007GuidelinesonPerioperativeCardiovascular.pdf

More on ACC AHA guidelines for cardiac patient preoperatively: http://www.medigraphic.com/pdfs/rma/cma-2010/cmas101bj.pdf

REFERENCES and FURTHER READING

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