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A Few Words about Myocardial Infarction

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Abstract

Myocardial infarction means damage and extinction of heart muscle cells that occurs when there is a reduction or complete cessation of blood flow to the heart muscle. The main cause of myocardial infarction is atherosclerosis of the arteries that supply the heart muscle with oxygen and nutrients. Risk factors that contribute to cardiovascular disease are age and gender, genetic factors (60% higher risk in individuals with one parent having cardiovascular disease), elevated cholesterol and triglyceride levels, smoking, exposure to chronic stress, poor eating habits, hypertension, diabetes, obesity and some chronic diseases.

Keywords: Heart; ECG; Heart Attack; Thrombosis; Health

Abbreviations: ECG: Electrocardiogram; MI: Myocardial Infarction; RBC: Red Blood Cells; HF: Heart Failure; UA: Unstable Angina; TIMI: Thrombolysis In Myocardial Infarction; PCI: Percutaneous Coronary Intervention.

Introduction

The typical presentation of an electrocardiogram (ECG) for an acute MI (myocardial infarction) include ST segment elevation in two “consecutive” or “contiguous” leads, which represents the same coronary artery territory, and not how they appear in sequence on the ECG. ST elevation should be ≥ 1 mm or 0.1 mV [1]. Other ECG findings include inversion of T waves and finally the development of a Q wave. The area that may be elusive on an ECG includes the inferior lateral wall. The presentation here would include increased voltage over the R waves, peaked T waves, and ST depression on leads V1 –V2. Also, the development of a new left bundle branch block should be approached as an acute MI and managed as such until biomarker data exclude the likelihood of an acute event.

The biomarkers follow a pattern of progression that often makes it challenging to assess for reinfarction. At the onset of a MI, creatine phosphokinase and creatine kinase-MB serum levels begin to rise at 4-8 hours, peak at approximately 18 hours, and return to baseline after 2-4 days. Troponin levels are more specific and are the better biomarkers to follow. Troponin serum levels rise at six hours, and may remain elevated for a few days, particularly if the patient has coexisting renal insufficiency. Specific levels of biomarkers should be referenced with the specific institutional standards, as they may vary.

Discussion

Dysrhythmias

Cardiac dysrhythmias affect up to 29% of patients having non-cardiac surgery and are a significant cause of morbidity and mortality in the perioperative period [2]. Although dysrhythmia may be the manifestation of an underlying cardiac disorder, they are an independent risk factor for future cardiac events. The incidence of dysrhythmias is greater following cardiac surgery. Postoperative atrial fibrillation (AF) is associated with a 2.3-fold increase in the risk of stroke, as well as an increased incidence of ventricular dysrhythmias, myocardial infarction, congestive cardiac failure and renal failure.

The majority of cardiac dysrhythmias occur in patients with pre-existing heart disease who have sustained an additional insult in the perioperative period. Cardiac, thoracic and laparoscopic surgeries are the commonest settings for perioperative dysrhythmias, with more than 15% of thoracic patients having an arrhythmia.

Long QT syndrome, whether congenital or acquired, is significant as it predisposes to the development of polymorphic ventricular tachycardia torsade de pointes, which can degenerate to ventricular fibrillation and sudden death. Patients with cardiomyopathy are at a significantly increased risk of PMI (perioperative myocardial ischaemia) or sudden death during non-cardiac surgery.

ECG (electrocardiography) monitoring is standard during anaesthesia but a 12-lead ECG may be required postoperatively to confirm the diagnosis. Evaluation of the EC regularity, the presence of p-waves and the configuration of the QRS complex. It is also important to note the existence of myocardial ischaemia, which may be a cause or consequence of the dysrhythmia. Assessment of the degree of cardiovascular compromise caused by the dysrhythmia is also crucial.

Sinus arrhythmias (tachycardia and bradycardia) are the most frequently encountered intra-operative dysrhythmias. Bradycardia may follow activation of vagal reflexes such as the oculocardiac reflex during ophthalmic surgery, or the Brewer–Luckhardt reflex during anal or cervical stretching. Hypoxia, high-sympathetic block, acute myocardial infarction or drugs may all lead to bradycardia. Sinus tachycardia may be due to pain, light anaesthesia, sepsis, hypoxia, hypovolaemia, hypercapnia or drugs.

Conduction abnormalities may cause complete heart block, which may require temporary or permanent pacing. However, it is rare for patients with intraventricular conduction delays even in the presence of left- or right-bundle branch block to progress to complete heart block intra-operatively.

Anemia

Anemia is associated with adverse outcomes and remains the most common indication for RBC (red blood cells) transfusion in patients admitted to the ICU, even when adherence to a conservative transfusion threshold is high [3]. Preventing the onset and progression of anemia requires a multifaceted approach adapted to the specific clinical context. Major surgery requiring elective ICU admission represents a large patient cohort where a pre-emptive approach is preferable. Approximately one in three patients scheduled to undergo major surgery is anemic, a potentially modifiable risk for perioperative adverse events, including myocardial infarction, stroke and mortality. A recent international consensus statement recommends routine screening of all patients undergoing surgery with an expected blood loss >500 mL and consideration of intravenous iron for patients with anemia and evidence of iron deficiency when oral iron is inefficacious, not tolerated or surgery is planned to occur in less than 6 weeks. In contrast, a Cochrane review on the use of preoperative iron therapy to correct anemia identified only three small RCTs (randomized controlled trial) and found no significant reduction in allogeneic RBC transfusion requirements. The safety and efficacy of preoperative intravenous iron therapy is now the focus of several ongoing large scale RCTs in cardiac, abdominal and orthopedic surgery.

Heart Attack

Diagnosing a heart attack is often difficult [4]. A heart attack (acute myocardial infarction) occurs when an artery that supplies blood to the heart muscle is blocked. Initially, the area of heart muscle supplied by the artery will become injured (myocardial ischemia). If this continues, the affected area of heart muscle will die (myocardial infarction). Because of this, it is important for emergency personnel to recognize heart attacks, now referred to as acute coronary syndrome, so that treatment can be provided and blood flow restored before a significant mass of the heart muscle is permanently damaged.

Several diagnostic tools are routinely used to determine whether a heart attack has occurred. These include the electrocardiogram (ECG), X-rays, laboratory tests, and others. Following injury to the heart muscle, its chemical components are released into the circulation. Laboratory assays of these chemicals can aid in the diagnosis of heart attack.

Thrombosis

The localized formation of a blood clot (thrombosis) is a normal part of the body's repair and healing response [4]. A physiological thrombus serves to limit hemorrhage that results from microscopic or macroscopic vascular injury. Physiological thrombus is counterbalanced by physiological anticoagulation and physiological fibrinolysis. In the normal setting, a physiological thrombus is confined to the immediate area of injury and does not obstruct blood flow to critical areas. Certain pathological conditions can lead to thrombus formation. In addition, under pathological conditions, a thrombus can expand into otherwise normal blood vessels, and obstruct blood flow to critical tissues. An abnormal thrombus can occur anywhere in the body but is particularly problematic when it causes acute coronary

syndrome, deep vein thrombosis, pulmonary embolism, acute nonhemorrhagic stroke, or blockage of peripheral arteries.

A thrombus in a coronary artery interrupts blood supply to a portion of the myocardium, which results in acute coronary syndrome. Likewise, the formation of a thrombus within the vessels of the brain can cause a stroke. The time from blockage of the vessel until irreversible tissue injury occurs is short. In the setting of acute myocardial infarction, treatment must be provided in six hours or less in order to be most effective. When fibrinolytics are administered to treat nonhemorrhagic strokes, they generally must be administered within three hours.

Patients

HF (heart failure) syndrome continues to progress relentlessly with decompensation of failure state and is a leading reason for hospital admission of elderly patients [5]. Readmission rates for acute decompensated heart disease remains the highest among all medical admissions to the hospital. Acute decompensation is often due to progression of underlying cardiomyopathic pathology with overwhelmed compensatory processes or can be de nova from acute myocardial injury (endocarditis, myocarditis, myocardial infarction). After treating precipitating cause, treatment is directed towards improving cardiac performance by altering loading conditions and enhancing contractility or mechanical support, if necessary. These patients can be classified into different clinical profiles depending on the presenting signs and symptoms. This profiling is derived from the initial Forrester Classification of HF in patients presenting with acute myocardial infarction, and has shown to have prognostic implications with cold and wet patients having a 6-month mortality of up to 40 %.

These patients with significant perioperative implications are treated in monitored settings, quite often in intensive care units (ICU) for early detection of clinical deterioration and to titrate the treatment options to clinical effect. Supplemental oxygen is administered to maintain adequate peripheral oxygen saturation and, if necessary, noninvasive ventilation is used to decrease work of breathing. Caution needs to be exercised when altering the intrathoracic pressure in patients with right HF. Positive pressure ventilation and tracheal intubation is necessary if the patient is in respiratory distress or unable to protect the airway secondary to neurological deterioration.

It may be possible to predict where there is a risk of deterioration [6]. This can occur when patients in hospital are relatively stable, but have the potential to develop critical illness, such as while waiting to undergo major surgery, or following surgery that carries a high risk of complications. The patient can have a period of physiological deterioration before they become critically ill, or the patient may suddenly become critically unwell (as in a severe myocardial infarction).

Recognizing and judging the degree of patient deterioration is complicated by the fact that it is a process that can result in complete recovery or death, with or without clinical intervention. In addition, the patient's past medical history and current diagnosis can affect the context within which physiological deterioration is viewed. In some patients it is an inevitable precursor to death following a life-limiting illness, while in other patients it is an undesirable complication that needs prompt and effective management to prevent further deterioration.

The pathophysiological course of deterioration is common to most patients regardless of their specific underlying clinical condition, and the signs and symptoms can be observed as the body responds to poor organ perfusion. These signs and symptoms arise through the activation of the autonomic nervous system that is a physiological response to hypoxia, hypotension and stress. This 'flight-orfight' response is the body's attempt to maintain adequate circulation to oxygenate vital organs and results in increased heart rate and respiratory rate, dilation of airways, dilation of the blood vessels supplying vital organs such as the heart, and constriction of blood vessels to non-essential organs. Where this physiological compensation is failing, the common signs may be confusion due to decreased oxygen to the brain, a decrease in blood pressure and/or poor urine output due to the body's attempt to preserve a circulating volume of fluid. The deteriorating patient can therefore be identified by changes in their vital signs that can include temperature, heart rate, respiration rate, blood pressure and oxygen saturation. Other symptoms, such as new confusion or reduced conscious level, changes in skin colour (mottled, grey or blue) and skin temperature (very hot, cool or cold) can give additional clues to the severity of the patient's deteriorating physical condition.

ECG

The cornerstone of diagnosis of acute coronary syndromes is the ECG [7]. Because findings on the initial ECG form a critical branch point in therapy, patients presenting to the emergency department with chest pain suggestive of ACS (acute coronary syndrome) should have an ECG within 10 minutes of arrival. Identifying STEMI (ST-elevation myocardial infarction) by ECG as soon as possible is the first step toward rapidly establishing reperfusion and reducing mortality. In contrast to STEMI, ECG findings may be subtle or absent in NSTEMI (non–ST-elevation myocardial infarction)

and UA (unstable angina), and are not required for diagnosis and initiation of therapy. However, certain findings, such as ST-segment depression or deep T-wave inversions, particularly those that change in accord with symptoms, can rapidly establish the diagnosis of UA and NSTEMI.

Unfortunately, the ECG is frequently nondiagnostic in ACS. Even in patients eventually diagnosed with MI, the initial ECG is nondiagnostic in about 50% and completely normal in up to 8%. Comparing the current ECG to old tracings is crucial, because subtle changes may be seen. Serial ECGs performed at 15- to 30-minute intervals, or continuous ST-segment monitoring, may reveal the subtle dynamic changes of UA, or those of an evolving MI.

In the face of a normal or nondiagnostic ECG, the decision whether to further evaluate for ACS depends on the likelihood that the pain is actually of cardiac origin and on the patient's overall risk profile. Inquiring about traditional risk factors for CHD remains a standard component of the chest pain evaluation. High risk is easily established if there is a prior history of definite CHD such as prior MI or abnormal coronary angiogram. Patients who are young, without a family history of premature CHD, and with an atypical history and a normal or nondiagnostic ECG can usually be safely discharged without further evaluation for ACS. Shortterm prognosis in those with known or suspected UA or NSTEMI can be calculated with the thrombolytics in myocardial infarction (TIMI) risk score.

With respect to ACS, certain risk factors have been associated with the probability of developing cardiac disease over a lifetime but have been shown to be of very little utility in the acute setting [8]. Age, sex, family history, hypertension, diabetes mellitus, elevated cholesterol, obesity, smoking, and physical inactivity, while important, are not predictive of acute disease. One of the more common tools for risk stratifying patients with suspected ACS is the Thrombolysis In Myocardial Infarction (TIMI) score. Associated with each score is a specific risk of poor outcome defined as death, myocardial infarction, or need for acute percutaneous coronary intervention (PCI).

Physical examination for patients with ACS is most useful for evaluating other potential etiologies of the patient's complaint, although the exam has utility in predicting patients who may be at risk for a poorer outcome or who have developed a complication related to their myocardial infarction. For example, patients with unstable vital signs, jugular venous distention, pulmonary edema, and/or an S3 gallop are indicative of heart failure. A new murmur is suggestive of papillary muscle rupture. Hemiparesis can indicate aortic dissection. Each of these is associated with worse prognosis than those without these complications. Caution must be exhibited, though, when it comes to physical examination and the possibility of ascribing a patient's symptoms to a process more benign than ACS. For example, a significant proportion of ACS patients may in fact have pleuritic, positional, or reproducible chest pain on examination. 11 Simply stated, there is no single feature of physical examination that safely rules out ACS.

Treatment

One of the first steps in managing acute MI is to identify which chamber is being affected so that one can implement the appropriate strategy for achieving target hemodynamic goals [1]. The primary method of improving delivery of oxygen to the myocardium is by improving blood flow, because the myocardium is already maximally extraction oxygen at a ratio of approximately 75%. The higher the coronary perfusion pressure, the better the blood flow. At this time we should define the determining variables for coronary perfusion pressure (CPP). For the left ventricle, the CPP is diastolic blood pressure (DBP) minus LV end diastolic pressure (LVEDP) (CPP = DBP – LVEDP). This is particularly unique to the left ventricle, because it primarily perfuses during diastole. Coronary arteries supplying the LV travel epicardially and then into the myocardial wall, where the vessels are sounded by the myocardium. If the systolic blood pressure (SBP) in the aortic root is 120 mmHg, then the pressure generated within the LV must be greater than 120 mmHg for forward flow to exist. Thus, the pressure surrounding the LV coronary arteries will be greater than the pressure within them, and therefore will not have flow during systole. During diastole, the pressure in the aortic root is 80 mmHg. Because the aortic valve closes, the pressure in the LV a healthy patient would be approximately 10 mmHg. As a result, the LV coronary branches only have 10 mmHg of resistance. Therefore, the LV CPP would be 80 mmHg (DBP) – 10 (LVEDP) for an LV CPP of 70 mmHg. Therefore, to improve blood flow to the LV, one must either increase the DBP or decrease the LVEDP.

Thrombolytics prevent recurrent thrombus formation and rapidly restore hemodynamic stability in selected patients with MI [9]. These agents can dissolve pathologic intraluminal thrombus or embolus not yet dissolved by the endogenous fibrinolytic system. When given within 12 hours of symptom onset, they restore patency of occluded arteries, salvage myocardium, and reduce morbidity and mortality of AMI. Thrombolytic agents studied for MI include SK and alteplase (recombinant tPA). Alteplase is more expensive but has shown a greater benefit in the GUSTO-1 (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) trial with a 15% relative risk

reduction (1% absolute risk reduction) compared to SK. Newer agents like tenecteplase and reteplase are as effective as alteplase. Benefit from thrombolytic therapy is largely correlated with time to therapy. Early therapy has the greatest impact on infarct size and left ventricular ejection fraction. Thrombolytic treatment should be started within 30 minutes of arrival (door-drug time). Maximum benefit occurs when administered within 1–3 hours of symptom onset. American College of Cardiology/American Heart Association guidelines recommend thrombolytic therapy for patients without contraindications within 12 hours of symptom onset. It is reasonable to give thrombolytic therapy within 12–24 hours after symptom onset if persistent ST elevations or continuing symptoms are present. Achievement of TIMI-3 (Thrombosis in Myocardial Infarction Trial 3) flow (normal flow which fills distal coronary bed completely) after thrombolytic therapy occurs in only 50–60% of patients-much less than that achieved with percutaneous coronary intervention (PCI) which is almost 90%. TIMI-3 flow postintervention is highly correlated with long-term survival. Risks of thrombolytic therapy for MI include bleeding-with the most worrisome complication being hemorrhagic stroke.

Research

The biomedical focus on the three narratives has led to anomalies and substantial controversy over guidelines and policies [10]. It has also narrowed the research horizon, slowing the development of innovative treatment alternatives that are based on new and better understanding of the pathophysiology.

The intuitive concept in acute and critical care of a need to be “fast” and “early” following the motto “time is tissue” has improved outcome in many areas of medicine, such as myocardial infarction or stroke, in which timing of an intervention, typically re-establishing oxygen availability, is of the essence. This concept has been rolled out to other areas in critical care, most notably sepsis. The evidence base, however, to justify this generalization is shaky. Extrapolation of the data from other forms of shock, e.g., hypovolemic shock, to septic shock is not as solid as it may seem at first glance. While this approach will improve oxygen delivery when an inappropriately low preload contributes to anaerobic metabolism, the pathogenesis of hyperlactatemia in sepsis is multifactorial and includes several causes unrelated to an impaired preload, such as mitochondrial uncoupling and Warburg metabolism. Moreover, mandatory fulfillment of a predefined volume load even in those patients that are not volume responsive is wrong. As such, the lack of documented benefit of the volume challenge in healthcare systems in highincome countries does not come as a surprise and is reflected in data that a positive fluid balance is associated with worsening outcome.

Conclusion

Myocardial infarction or heart attack is the result of a complete cessation of blood flow to the coronary artery. It usually occurs suddenly, due to thrombosis that clogs the lumen of the coronary artery or its branches, and leads to a heart attack. It varies depending on the location and size of the infarction and other factors. It usually begins suddenly, with strong, penetrating pain, which does not subside either at rest or not in response to nitroglycerin. If help is not provided in time, cardiogenic shock develops: the patient turns pale, cold sweat pours over him, the pressure drops, and the pulse becomes weak, rapid and irregular.

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