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Cardiac Emergencies in Intensive Care Unit

An Essay

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Cardiac Emergencies in ICU

Cardiovascular emergencies are life-threatening disorders that must be recognized immediately to avoid delay in treatment and to minimize morbidity and mortality. Patients may present with severe hypertension, chest pain, dysrhythmia, or cardiopulmonary arrest. One and half million Americans will have a heart attack every year. Two out of three will survive, but that number could be much higher ***(Berg, 2004).***

Consider this: Of the 500,000 deaths that will occur, 300,000 will be within the first hour of the onset of symptoms. Most of these people will die before they reach a hospital. Almost three-quarters of these sudden deaths occur at home during a heart attack, the heart is deprived of oxygen, but it may continue to function ***(Berg, 2004).***

Cardiopulmonary resuscitation is a combination of breathing for the victim-to supply oxygen-and compressing the chest wall, which squeezes the heart and pushes blood out to vital organs. In this way, oxygen is delivered to the brain and to the arteries of the heart itself. Unless breathing and circulation are established within four to six minutes of a cardiac arrest, irreversible brain damage occurs, and eventually the individual dies ***(Berg, 2004).***

Aim of the Work

The aim of this work is to spotlight on cardiac emergencies in intensive care unit patients and how to diagnose and manage.

Cardiac Tamponade (Pericardial Effusion)

Cardiac tamponade is a life-threatening condition that can be palliated or cured, depending on its cause and on the timeliness of treatment. Cardiac tamponade occurs when fluid in the pericardial space reaches a pressure exceeding central venous pressure ***(Schiavone, 2013)***.

Cardiac tamponade is defined as an accumulation of fluid in the pericardial sac, creating an increased pressure within the pericardial space that impairs the ability of the heart to fill and to pump. As the pump function of the heart becomes impaired then there is a fall in cardiac output and systemic perfusion leading to life-threatening organ dysfunction ***(Rafique et al., 2011)***.

Importantly the presence of abnormally large volumes of fluid within the pericardial sac does not always necessitate the development of tamponade. This is because slowly accumulating fluid allows physiological compensatory mechanisms to limit the rise in pericardial pressure and therefore avoid cardiac pump failure ***(Imazio et al., 2010)***.

Consequently a pericardial effusion should be distinguished from tamponade as an *anatomical* diagnosis, describing the presence of abnormal fluid within the pericardial sac with no hemodynamic compromise. In contrast cardiac tamponade is a *physiological* diagnosis in which the pericardial effusion must be accompanied by evidence of obstructive shock ***(Imazio et al., 2010)***.

Epidemiology:

The incidence of cardiac tamponade is 2 cases per 10,000 population in the United States. Approximately 2% of penetrating injuries are reported to result in cardiac tamponade ***(Imazio and Hoit, 2013)***.

Pathophysiology of cardiac tamponade:

1. Physiology of the pericardial sac:

The pericardial sac consists of 2 layers of tough, fibrous tissues surrounding and protecting the heart. An inner visceral layer is separated from the parietal pericardium by a small amount of lubricating pericardial fluid. Usually a volume of around 15-50mls of pericardial fluid is present ***(Huang et al., 2011)***.

2. Pressure-volume relationship:

Fluid within the pericardial space exhibits a pressure-volume relationship. When plotted the pressure-volume relationship exhibits 2 distinct phases, as illustrated in (Fig. 1-1) ***(Halpern et al., 2012)***.

If fluid accumulates gradually then during the first phase an increase in volume of the pericardial fluid causes only a small increase in pericardial pressure. During the second phase the reserve volume, below which pressure changes are minimised by the compliance of the pericardial membranes, is exceeded and pericardial pressure begins to rise more rapidly ***(Halpern et al., 2012)***.

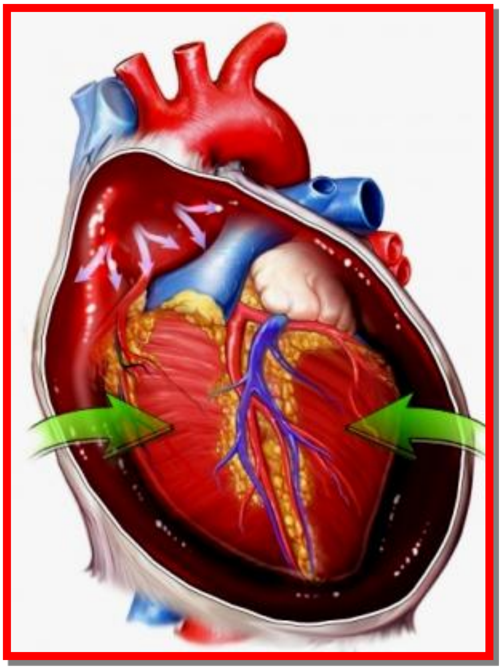

Fig.1-1: Cardiac effusion ***(Halpern et al., 2012)***.

In addition a number of factors influence the rate of pressure increase within the pericardial space. Most important is the duration of time over which fluid accumulates. In the acute situation as little as 150ml of fluid may result in cardiac tamponade. In marked contrast the pericardial membranes can stretch significantly if the build up of fluid is sufficiently chronic, with the pericardial sac volume reaching 2000ml in extreme cases ***(Hayashi et al., 2012)***.

Etiology:

A trauma patient first encountered in the emergency department may have an underlying disease, but the focus is squarely on the effects of trauma or violent injury. In a patient with multiple trauma, hypotension and tachycardia that do not respond to intravenous volume replacement when there is an obvious rise in central venous pressure should be clues to cardiac tamponade. If the patient has recently undergone a cardiac procedure, knowing about the procedure narrows the differential diagnosis when hypotension, tachycardia, and jugular venous distention develop ***(Schiavone, 2013)***.

For all patients, malignant diseases are the most common cause of pericardial tamponade. Tamponade can occur as a result of any type of pericarditis ***(Figueras et al., 2010)***.

Likely to progress to cardiac tamponade
- Neoplastic diseases
- Infections (i.e. viral: EBV(Epstein barr virus), CMV(cytomegalo virus) enteroviruses, HIV(human immune deficiency virus), bacterial, especially tuberculosis)
- Iatrogenic hemopericardium
- Post-traumatic pericardial effusion
- Post-cardiotomy syndrome
- Hemopericardium in aortic dissection and rupture of the heart after acute myocardial infarction
- Renal failure

Rarely progressing to cardiac tamponade
- Systemic autoimmune disease
- Autoreactive pericardial effusions
- Hypo- or hyperthyreosis
- Early and late pericarditis (Dressler's syndrome) in acute myocardial infarction
- Any other aetiology of pericardial disease (i.e. cholesterol pericarditis, chylopericardium)

Never progressing to cardiac tamponade
- Pericardial transudates caused by heart failure or pulmonary hypertension
- Pericardial transudates in the last trimester of normal pregnancy

Precipitating factors
- Drugs
  - Antihypertensive medications
  - Anticoagulants, thrombolytics, etc.
- Injury
  - Complex PCI (percutaneous coronary intervention)
  - Pacemaker implantation
  - Endomyocardial biopsy
  - Recent cardiac surgery
  - Indwelling instrumentation
  - Blunt chest trauma
- Septicaemia
- Dehydration, diuretics (reduced circulating volume)

Table 1-1: Causes of pericardial disease and precipitating factors causing cardiac tamponade ***(Imazio and Adler, 2013)*.**

Infective Endocarditis

Infective endocarditis has an estimated annual incidence of 3-9 cases per 100,000 persons in industrialized countries. The highest rates are observed among patients with prosthetic valves, intracardiac devices, unrepaired cyanotic congenital heart diseases, or a history of infective endocarditis ***(Hoen and Duval, 2013)***.

Other risk factors include chronic rheumatic heart, age related degenerative valvular lesions, hemodialysis, and coexisting conditions such as diabetes, HIV infection, and intravenous drug use ***(Federspiel et al., 2012)***.

The clustering of several of these predisposing factors with age probably explains the increased incidence of infective endocarditis among persons 65 years of age or older ***(Suty et al., 2012)***.

Microbiology:

Streptococci and staphylococci account for 80% of cases of infective endocarditis, with proportions varying according to valve (native or prosthetic), source of infection, patient age, and coexisting conditions ***(Duval et al., 2012)***.

Cases of infective endocarditis in which a blood culture is negative (10% of cases) may reflect one of two situations:

infective endocarditis in patients exposed to antibiotic agents before the diagnosis of infective endocarditis or infective endocarditis caused by fastidious microorganisms ***(Sy and Kritharidis, 2010)***.

In the latter case, serologic testing, valve or blood polymerase-chain-reaction (PCR) assay, and highly specialized microbiologic techniques lead to the identification of the pathogen in 60% of cases with the most frequent microorganisms being bartonella species, brucella species, *Coxiellaburnetii* (the agent causing Q fever), haemophilus species, Actinobacillus and Eikenellacorrodens ***(Fournier et al., 2010)***.

Pathogenesis:

Normal valvular endothelium is naturally resistant to colonization by bacteria. In the conventional model of native-valve infectious endocarditis, infection results from the colonization of damaged valvular endothelium by circulating bacteria with specific adherence properties ***(Hoen and Duval, 2013)***.

Endothelial damage may result from so-called jet lesions due to turbulent blood flow or may be provoked by electrodes or catheters or by repeated intravenous injections of solid particles in intravenous-drug users ***(Correa et al., 2010)***.

Chronic inflammation, as in chronic rheumatic heart disease and degenerative valvular lesions, may also promote infective endocarditis. However, the conventional model may not accurately explain the pathogenesis of infective endocarditis due to intracellular microorganisms, such as *C. burnetii*, or bartonella species, in which the exposure and immune response of the host may play a prominent role ***(Hoen and Duval, 2013)***.

Cardiac Arrest

History:

History and associated symptoms depend on the underlying etiology of sudden cardiac death (SCD). For example, SCD in an elderly patient with significant coronary artery disease may be associated with preceding chest pain due to a myocardial infarction, while SCD in a young patient may be associated with history of prior syncopal episodes and/or a family history of syncope and SCD and due to inherited arrhythmia syndromes ***(Berdowski et al., 2011)***.

Risk factors that relate to coronary artery disease and subsequent myocardial infarction and ischemic cardiomyopathy also are important and include a family history of premature coronary artery disease, smoking, dyslipidemia, hypertension, diabetes, obesity, and a sedentary lifestyle ***(Holzer, 2010)***.

Specific considerations include the following table, ***(Holzer, 2010)***:

Coronary artery disease

- Previous cardiac arrest
- Syncope
- Prior myocardial infarction, especially within 6 months
- Ejection fraction less than 30-35%
- History of frequent ventricular ectopy (more than 10 premature ventricular contractions (PVCs) per h or nonsustained ventricular tachycardia (VT)

Dilated cardiomyopathy

- Previous cardiac arrest
- Syncope
- Ejection fraction less than 30-35%
- Use of inotropic medications

Hypertrophic cardiomyopathy

- Previous cardiac arrest
- Syncope
- Family history of SCD
- Symptoms of heart failure
- Drop in SBP or ventricular ectopy upon stress testing
- Palpitations
- Most are asymptomatic

Valvular disease

- Valve replacement within 6 months
- Syncope
- History of frequent ventricular ectopy
- Symptoms associated with severe uncorrected aortic stenosis or mitral stenosis

Long QT syndrome

- Family history of long QT and SCD
- Medications that prolong the QT interval
- Bilateral deafness

Wolff-Parkinson-White (WPW) syndrome (with atrial fibrillation or atrial flutter with extremely rapid ventricular rates)

With extremely rapid conduction over an accessory pathway, degeneration to VF can occur.

Other

- Brugada syndrome, arrhythmogenic right ventricular (RV) cardiomyopathy/dysplasia, and others

Table 1-2: Risk factors for developing cardiac arrest ***(Holzer, 2010)***.

Physical examination:

The physical examination may reveal evidence of underlying myocardial disease or may be entirely normal, depending on the underlying cause. The cardiac arrest score, developed by **Thompson and McCullough**, can be used for patients with witnessed out-of-hospital cardiac arrest and is defined by the following criteria ***(Merchant and Amoundas, 2012)***.

Clinical characteristic points:

Emergency department (ED) systolic blood pressure (SBP) greater than 90 mm Hg = 1 point

ED SBP less than 90 mm Hg = 0 points

Time to return of spontaneous circulation (ROSC) less than 25 minutes = 1 point

Time to ROSC more than 25 minutes = 0 points

Neurologically responsive = 1 point

Comatose = 0 point

Maximum score = 3 points

Patients with a score of 3 points can be expected to have an 89% chance of neurologic recovery and an 82% chance of survival to discharge ***(Fishrman et al., 2010)***.

Causes:

① Ischemic heart disease:

Cardiac arrest due to ventricular arrhythmias may be due to post-(MI) myocardial infarction remodeling of the heart with scar formation and interstitial fibrosis or to acute MI/ischemia ***(Mallinson, 2010)***.

In postmortem studies of people who have died from SCD, extensive atherosclerosis is a common pathologic finding. In survivors of cardiac arrest, coronary heart disease with vessels showing greater than 75% stenosis is observed in 40-86% of patients, depending on the age and sex of the population studied ***(Studnek et al., 2010)***.

Reperfusion of ischemic myocardium with thrombolysis or direct percutaneous coronary intervention can induce transient electrical instability by several different mechanisms ***(Ong et al., 2011)***.

Coronary artery spasm is a condition that exposes the myocardium to both ischemia and reperfusion insults ***(Xiao et al., 2013)***.

Non-atherosclerotic abnormalities in coronary arteries, including congenital lesions, coronary artery embolism, coronary arteritis, and mechanical abnormalities of the coronary artery,