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Cardiac tamponade after removal of temporary pace maker in multidiscipinary intensive care unit.

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Case Report

Cardiac Tamponade after removal of temporary pace maker in multidiscipinary Intensive Care Unit

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Abstract

Cardiac tamponade is a medical and surgical emergency, which needs early recognition and treatment. Myocardial perforation leading to cardiac tamponade is a rare complication after pace maker insertion. We are reporting a case of cardiac tamponade after removal of temporary pace maker in a multidisciplinary intensive care unit.

Introduction

Cardiac tamponade is a medical emergency which is characterized by the accumulation of fluid in the pericardial space, resulting in reduced ventricular filling and subsequent haemodynamic compromise. Myocardial perforation leading to cardiac tamponade is a rare complication after pace maker insertion. This condition requires urgent recognition since the prompt drainage of the pericardial fluid may be lifesaving. We present a case report of myocardial perforation complicated by cardiac tamponade after removal of a pacemaker which was successfully managed surgically.

Case Report

A 60 year-old female with hypertension, diabetes mellitus and ischaemic heart disease with mild to moderate systolic dysfunction, was admitted through emergency room with cardiogenic shock due to Non-ST elevated MI (Troponin I >3), hyponatraemia (Na = 123 Meq/L) and severe metabolic acidosis. She was intubated in emergency room due to respiratory distress. Post intubation, she went into cardiac arrest. Temporary pacemaker was inserted in the emergency room (Figure) and she was transferred to intensive care unit (ICU). She was successfully extubated on day 4. Patient regained her own heart rhythm 24 hours after insertion of pacemaker as shock and metabolic acidosis improved. Within one hour of removal of pacemaker she complained of dizziness and difficulty in breathing. Blood pressure was 60/40 mmHg and heart rate

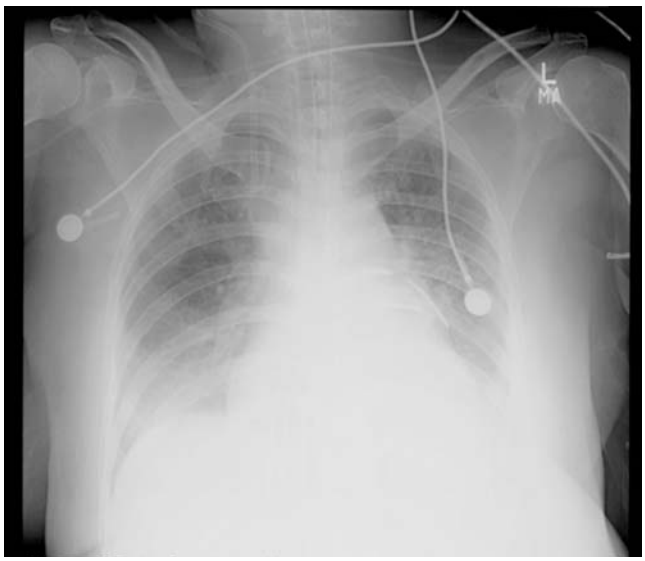

Figure 1. Portable Chest X-Ray showing ET tube, right IJ swan sheath and temporary pacemaker in appropriate position. Lung fields show mild bilateral pleural effusions, silhouetting of left hemi diaphragm and haze over both lower lung zones consistent with pulmonary edema.

90/min. Pulse was weak. EKG showed low voltage waves with ST depression in V3 to V5. Urgent echocardiogram showed large pericardial effusion with tamponade. She was resuscitated with IV fluids and immediate pericardiocentesis was performed and 400 ml of clotted blood was removed. Pericardial window was made. Patient became haemodynamically stable and was extubated on the next day and shifted to special care unit. She was discharged home seven days after the event.

Discussion

Myocardial perforation is a rare complication following pacemaker implantation that may cause cardiac tamponade. If it does occur, it is usually at the time of lead insertion.[1] Tamponade usually takes place due to arrow head endocardial electrode.[2] Fatal myocardial perforation can occur with this electrode and the apex of the right ventricle should be avoided as the site of insertion3. This condition requires urgent recognition since the prompt drainage of the pericardial fluid may be lifesaving.

The pericardium consists of a thin serous membrane covering the epicardial surface (visceral pericardium) and a serous membrane-lined fibrous sac (parietal pericardium), which has limited elastic properties. The pericardial space separates the two layers and contains approximately 25-35 ml of serous fluid.[1] An acute accumulation of pericardial fluid of greater than 100 ml will produce haemodynamic effects of tamponade whereas a chronic pericardial collection of fluid up to 2000 ml may occur without imposing any effect upon cardiac output.[2]

Cardiac tamponade is defined as a haemodynamically significant cardiac compression caused by pericardial fluid.[3] The fluid may be blood, pus, effusion or air.[4] Pericardial tamponade may arise from multiple traumatic and non traumatic etiologies, resulting in unrecognized rapid deterioration and often death. Traumatic pericardial tamponade occurs in only 2% of all penetrating chest injuries[5], and rarely is the result of blunt trauma. Mortality exceeds 60% if cardiac arrest occurs.[6] Non traumatic causes of pericardial tamponade include haemopericardium due to anticoagulant therapy or as a rare complication of acute myocardial infarction. In addition, various non- traumatic causes such as infection, drugs, neoplasm, uraemia, myxoedema, collagen vascular disease, and hypersensitivity states may produce large effusion with tamponade. Pericardial tamponade has resulted from cardiac catheterization, central venous catheterization, pericardiocentesis, intracardiac injection, cardiac surgery, and sternal bone biopsy and transvenous pacemaker insertion.[7] Common sites of perforation are the right atrium and right ventricle followed by superior vena cava.[8] Perforation has also been reported in the left atrium (patent foramen ovale) and the left pericardiophrenic vein. Endocardial injury is thought to be caused by either movement of the catheter tip, by movements of cardiac chambers and lower superior vena cava (cardiac cycle) or by direct trauma. Injury causes thrombus formation and eventually adherence of the wire to the endocardium. Erosion occurs which may lead to perforation.

Early recognition and treatment of cardiac tamponade is essential to prevent fatal outcome. Symptoms and signs are usually sudden and include nausea, fatigue, light-headedness, dyspnoea, retrosternal chest pain, cyanosis, venous engorgement, pulsus paradoxus and confusion. The most common findings noted by Nasim and colleagues[9] from case reports were hypotension (88%), raised central venous pressure (70%) and a disturbance in cardiac rhythm (67%) mainly tachycardia. However in 29% of these cases death occurred suddenly after 'vague premonitory signs'. Diagnosis of cardiac tamponade is difficult in sedated, ventilated and post-operative patients.

EKG and chest radiograph findings may not always assist the diagnosis. EKG findings such as low voltage QRS complex or electrical alternans may not always be present. Chest radiograph may not show abnormalities until considerable fluid has accumulated in the pericardial sac. Transthoracic or transesophageal echocardiography is diagnostic. These techniques are unfortunately not always available and delaying treatment to obtain these investigations may be fatal.

The patient is initially resuscitated with intravenous fluids to promote maximum filling of the heart. In general, inotropic agents that increase the stroke volume and support

systemic resistance are used, although some recommend isoprenaline, as it reduces the cardiac size and diminishes the effective degree of tamponade while increasing cardiac output.[2] The definitive treatment of cardiac tamponade is the removal of cardiac diastolic restriction by either pericardiocentesis or thoracotomy. Pericardiocnetesis is usually performed for urgent management of an acute tamponade (the acute removal of as little as 50 ml of fluid is often sufficient to correct the hypotension). A thoracotomy is often required when a tamponade exists following coronary artery bypass grafting, penetrating or closed cardiac trauma and aortic dissection. It is also indicated when pericardiocentesis has failed to relieve the tamponade.

Conclusion

Cardiac tamponade is a life-threatening emergency. Immediate recognition and treatment are imperative if a disastrous outcome is to be prevented.

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