May 2008

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

Muhammad Faisal Khan
Aga Khan University

Ali Bin Sarwar Zubairi
Aga Khan University, ali.zubairi@aku.edu

Follow this and additional works at: http://ecommons.aku.edu/pakistan_fhs_mc_med_med

Part of the Pulmonology Commons

Recommended Citation
Available at: http://ecommons.aku.edu/pakistan_fhs_mc_med_med/312
population in SHI, which may make this system more complicated rather than helpful.

**Conclusion**

Given present human resource constraints and institutional capacities, operationalizing any government funded SHI scheme on a national level ensuring universal coverage is a huge challenge. Positive vibes are a rising national economy, political will to carry out health sector reforms and the creation of district health system after devolution. The current scenario is ideal to pilot small initiatives, may be at district levels, and then up scale by learning lessons from these pilot projects. Policy makers, health systems specialists and other stakeholders must capitalize on these opportunities and windows to find a way out.

**References**


**Case Report**

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

Muhammad Faisal Khan¹, Ali Bin Sarwar Zubairi²

Department of Anesthesiology¹, Pulmonary and Critical Care Medicine², Aga Khan University Hospital, Karachi.

**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...
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. Tamponade usually takes place due to arrow head endocardial electrode. Fatal myocardial perforation can occur with this electrode and the apex of the right ventricle should be avoided as the site of insertion. 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. 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.

Cardiac tamponade is defined as a haemodynamically significant cardiac compression caused by pericardial fluid. The fluid may be blood, pus, effusion or air. 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, and rarely is the result of blunt trauma. Mortality exceeds 60% if cardiac arrest occurs. 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. Common sites of perforation are the right atrium and right ventricle followed by superior vena cava. 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 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. The definitive treatment of cardiac tamponade is the removal of cardiac diastolic restriction by either pericardiocentesis or thoracotomy. Pericardiocentesis 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.

References

Case Report

De novo primary squamous cell carcinoma of the ovary: A case of a rare malignancy with an aggressive clinical course

Ali Imran Amjad, Inam Pal
Department of Surgery, Aga Khan University Hospital, Karachi, Pakistan.

Abstract
Ovarian squamous cell carcinoma is a rare malignancy and the occurrence is attributable to malignant transformation of an existing ovarian dermoid cyst. The de novo occurrence of squamous cell carcinoma of the ovary, in the absence of an antecedent ovarian dermoid, is extremely rare. The case of a 31 year old Asian woman, evaluated for abdominal distension and discomfort is presented. Abdominal CT was suggestive of a malignant neoplastic process. Laparotomy confirmed a malignant tumour with involvement of the right adnexa and extension into the omentum and bowel. Surgical debulking, hysterectomy, bilateral salpingo-ophorectomy and total omentectomy and bowel resection was performed. Histopathology demonstrated squamous cell carcinoma arising from the right ovary with no co-existing ovarian dermoid. The postoperative period was significant for disease progression despite adjuvant chemotherapy.

Introduction
Squamous cell carcinoma of the ovary is a rare clinical entity, accounting for less than 1% of all malignant tumours of the ovary. Malignant transformation of a pre-existing mature cystic teratoma (dermoid cyst) is appreciated as the underlying pathophysiological mechanism, and this phenomenon is considered rare, as only 1-2% of teratomas demonstrate this change. The de novo development of a primary squamous carcinoma, in an otherwise healthy ovary is an extremely rare occurrence. We present a case of this rare malignancy, not been previously reported in the Southeast Asian population, with a review of literature.

Case Report
A 31 year old, para 2, Asian female (Pakistani heritage) was referred to the general surgery clinic with a one year history of progressive abdominal discomfort and distension. She had a history of weight loss with no change in appetite, bowel habit or menstrual flow. A recent pap smear was negative for dysplasia. On examination, she was anaemic with no jaundice, lymphadenopathy and had normal breast examination. Abdominal examination revealed a firm, mildly tender, mobile mass occupying most of the lower abdomen. There was no clinical evidence of ascites. Pelvic and rectal exams were unremarkable.

Pre-operative evaluation showed anaemia with normal cell counts, and normal kidney and liver functions.