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CASE REPORT

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Successful use of intra-aortic counter pulsation therapy for intractable ventricular arrhythmia in patient with severe left ventricular dysfunction and normal coronary arteries

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Abstract

Intra-aortic balloon pumps (IABP) are commonly used in the setting of an acute myocardial infarction that is complicated by cardiogenic shock or mechanical complications such as a ventricular septal defect or papillary muscle rupture. IABP has also been shown to be useful in patients with refractory and hemodynamically unstable ventricular arrhythmias and refractory post-myocardial infarction angina.

We report a case in which IABP was used in a patient with dilated cardiomyopathy and normal coronary arteries, who presented with persistent, recurrent and refractory ventricular tachycardia. His ventricular tachycardia settled immediately with the use of IABP therapy. He subsequently had an implantable defibrillator.

The use of IABP is associated with favorable changes in the left ventricular wall tension and reduction in afterload, which could reduce the excitability of the myocardium, thus making it less prone to arrhythmias. The use of IABP is relatively safe and should be considered in patients with refractory ventricular arrhythmias, even if it is not associated with ischemia. (Cardiol J 2010; 17, 4: 401–403)

Key words: intra-aortic baloon pump, ventricular tachycardia

Introduction

Ventricular arrhythmias either ventricular tachycardia (VT) or ventricular fibrillation (VF) are a common complication seen in patients with impaired ventricular function [1]. VF is associated with hemodynamic compromise and requires prompt electric cardioversion. VT, on the other hand, may be associated with a stable blood pressure. They are often due to re-entry in a region of previous myocardial infarction or ventricular scar. In patients with idiopathic dilated cardiomyopathy, they could result from abnormal circuits and abnormal action potentials due to stretch, damage and fibrosis of the myocardium [2, 3]. If the VT repeatedly recurs and persists for more than half of a 24 hour period, despite repeated attempts to terminate the arrhythmia, it is designated 'incessant'. In these patients, anti-arrhythmic agents can be tried in the first instance. If, however, they fail, urgent electric cardioversion is indicated.

Occasionally, VT fails to respond to repeated electric cardioversion and anti-arrhythmic agents. Ischemia is a common cause of incessant ventricular

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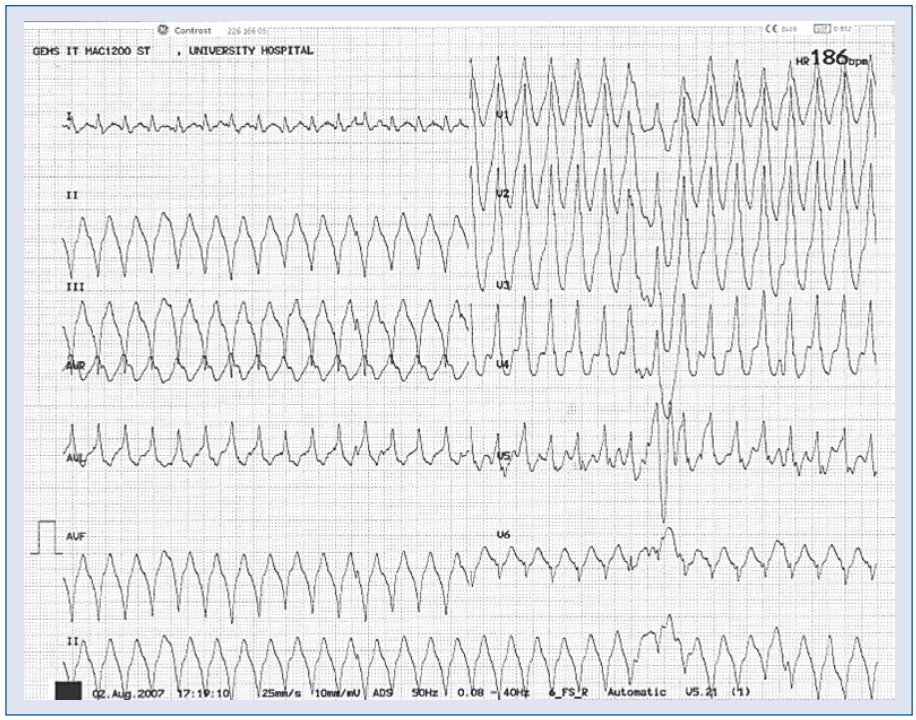

Figure 1. Presenting electrocardiogram showing ventricular tachycardia.

arrhythmias and prompt relief of ischemia by percutaneous coronary intervention helps to relieve the arrhythmia. In conditions where relief of ischemia may not be possible (e.g. where lesions are not amenable to percutaneous coronary intervention), or if there is no ischemia, unconventional methods such as overdrive pacing and the use of an intra-aortic balloon pump may be attempted. Here, we report a case where the VT was not of ischemic origin, and was resistant to various antiarrhythmic agents, responding only after the introduction of an intra-aortic balloon pump (IABP).

Case report

A 52 year-old male with a history of non-insulin dependent diabetes was admitted feeling unwell with increasing shortness of breath. His presenting electrocardiogram showed VT (rate of around 270––300, monomorphic and with an abnormal leftward axis of –75) (Fig. 1) and he was clinically in pulmonary edema which was confirmed radiologically. Arterial blood gases revealed type 2 respiratory failure with acidosis (pH: 7.20, pCO2: 9.45 kPa [normal 4.5––6 kPa]; pO2: 9.06 kPa [normal 10–13 kPa]; HCO3: 16.4 mmol/L [normal 20–24 mmoL/l]).

The VT was initially managed with intravenous lignocaine followed by intravenous amiodarone but these were not successful. In view of the respiratory failure and acidosis, he was ventilated and managed in the intensive therapy unit (ITU). The VT finally responded to electrical cardioversion (150 J of biphasic current) with the restoration of sinus rhythm and the amiodarone infusion was continued. On the day of admission, he had recurrent bouts of VT, each successfully reverting to sinus rhythm with a single shock of 150 J biphasic current.

His bedside echocardiogram revealed a dilated left ventricle (LV) with global hypokinesis and severely impaired LV systolic function. In ITU, he had recurrent episodes of VT (similar morphology to the admission VT) whilst on the amiodarone infusion and despite correction of his electrolytes, requiring repeated electrical cardioversion. He had a total of seven shocks within 12 hours of the first day of admission (two were an hour apart; then he had three within one hour; two hours later he had another two shocks) and a further two shocks on the second day. At first, the VT did not cause hemodynamic compromise (he had maintained a blood pressure of above 120/80 mm Hg at most times) and he never lost his cardiac output or required cardiopulmonary resuscitation. However, the later episodes of VT made him hypotensive and he was commenced on ionotropic support.

He had raised cardiac enzymes (troponin T was elevated at 0.19 μg/L [normal < 0.01 μg/L]). He underwent coronary angiography to rule out underlying myocardial ischemia as the cause of the VT, but it revealed normal coronary arteries with a globally dilated and poorly contracting left ventricle. An IABP was inserted to improve hemodynamics. Immediately prior to the IABP insertion, his blood pressure was 81/54 mm Hg. Once the IABP was inserted, he had no further VT. His blood pressure improved and on 1:1 augmentation he maintained a blood pressure of around 120/70 mm Hg. He was subsequently weaned off the ionotropes as well as the IABP. He subsequently had an implantable cardioverter defibrillator implanted and was discharged home in a couple of days with no further VT.

Discussion

Intra-aortic balloon counter pulsation therapy has been used to treat refractory ventricular arrhythmias in patients with coronary artery disease awaiting revascularisation, and is a class IIa recommended indication for use by the American Heart Association/American College of Cardiology guidelines [4]. However, there are not many reports on the use of this therapy in patients with normal coronary arteries. Fotopoulos et al. [5] used IABP in 21 consecutive patients with refractory ventricular arrhythmias. Eighteen of these patients had significant coronary artery disease. Of the remaining three, two had idiopathic dilated cardiomyopathy and one had amyloidosis. The use of IABP stabilized 18 out of the 21 patients. Complete abolition of arrhythmia was reported in 14 patients, while four other patients had significant reduction in ventricular arrhythmias. Two out of the three patients with normal coronary arteries survived and their arrhythmia responded to treatment with IABP. These two patients had an IABP support period of 3––9 days, while our patient's ventricular arrhythmia was well controlled immediately after IABP insertion, and his IABP was withdrawn within two days of insertion.

In patients with ischemic heart disease, the benefit of IABP in controlling VT could be in improving coronary blood flow and thereby reducing ischemia. However, in patients with normal coronary arteries, the mechanism of action is less clear. It has been suggested that using IABP reduces the adrenergic drive which is arrhythmogenic and thereby reduces the vulnerability of the myocardium to arrhythmias. IABP causes systolic left ventricular offloading and this reduces myocardial wall tension and oxygen demand which could improve oxygenation of the myocardium.

It is possible that the mechanical effect of the IABP has a direct effect in controlling arrhythmias. Animal studies [6] have demonstrated that with progressive increase in the afterload, there is an increase in ventricular ectopy and tachycardia. Similarly, stretching of normal cardiac tissue results in a reduction of action potential duration and an increase in spontaneous depolarization [7]. The use of IABP reduces LV afterload, and thereby reduces myocardial stretching and distension, and could thereby make the myocardium less irritable.

Our patient had refractory VT of a non-ischemic aetiology that responded promptly to IABP use. IABP is a relatively safe procedure [8]. We feel that its use should be considered more frequently in the setting of recurrent and intractable ventricular arrhythmias with or without hemodynamic compromise unrelated to acute ischemic heart disease.

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