Variation in mode of sudden cardiac death in patients with dilated cardiomyopathy

The mode of sudden cardiac death (SCD) in patients with dilated cardiomyopathy (DCM) is often presumed to be secondary to a tachyarrhythmia, although this is not usually witnessed by a reliable observer. We report two cases of SCD in patients with DCM.

Patient 1 was an 82-year-old white female in whom a diagnosis of dilated cardiomyopathy was made in 1992. Echocardiography showed dilated and globally hypokinetic left and right ventricles. She was self-caring and led a relatively active lifestyle. Over the next 2 years she required a number of admissions with congestive cardiac failure but her overall condition remained satisfactory. She was well until admission to her local hospital with diarrhoea. Due to dehydration, her diuretics were discontinued. On transfer she had minimal peripheral oedema, BP 120/60. Resting ECG showed atrial fibrillation, QRS duration 99 ms, QT interval (corrected) 369 ms. A trans-thoracic echocardiogram showed LVEDD 60 cm, LVESD 4-8 cm.

Following reaccumulation of peripheral oedema, her diuretics were recommenced. After experiencing some dizziness, a 24 h tape was requested. Plans were being made for her discharge as she felt considerably better. Whilst wearing a Holter monitor she died in her sleep unexpectedly. The rhythm strips below (Fig. 1,a,b) demonstrate marked ST-segment elevation an hour or so before prolonged bradycardia. The terminal rhythm was asystole. She was asleep when reviewed by the nursing staff and did not awaken.

Patient 2 was a 68-year-old white male who underwent coronary artery bypass grafting in 1986. Cardiac catheterization had shown a dilated poorly contracting ventricle and three-vessel coronary artery disease. He was relatively well until 1993 when he became increasingly short of breath, and developed crescendo angina. Percutaneous transluminal coronary angioplasty and insertion of an intracoronary stent resulted in significant clinical benefit.

In 1994 he was re-admitted with worsening heart failure. Electrocardiogram showed sinus rhythm, PR interval 180 ms, LBBB with QRS duration 188 ms, QT interval (corrected) 631 ms. A transthoracic echocardiogram showed a markedly dilated left ventricle LVEDD 8-0 cm, LVESD 7-0 cm, and prolonged functional mitral regurgitation with reduced filling time.

Due to the ECG and echocardiographic characteristics, a dual chamber pacemaker was implanted. He remained relatively well and was followed up 3 months. He attended for a routine outpatient visit, during which he exercised for almost 6 min of a modified Bruce protocol. He was well when he left the hospital, and had a 24-hour ambulatory recorder attached (as part of a study protocol). Later that evening he collapsed and could not be resuscitated. The rhythm strips relating to this episode are shown below (Fig. 2,a,b).

It is well recognised that patients with DCM are prone to sudden cardiac death[1]. The precise mechanisms underlying this are unclear. Despite this, the classification of mode of death in heart failure trials is often similar. In general, patients are assigned to a cardiovascular or non-cardiovascular cause of death. In CONSENSUS and VHeFT II, patients were categorized as having sudden, unexpected death (<1 h), rapid cardiac death (1–24 h), progressive heart failure, myocardial
infarction, or other[2,3]. In SOLVD, patients were deemed to have sustained: arrhythmia without worsening CCF, arrhythmia with CCF, myocardial infarction or other[4]. In SAVE, although the number of categories of mode of death were similar, all patients in these categories were included under the overall grouping of atherosclerotic heart disease[5].

The two patients described above demonstrate that, in the presence of heart failure that is relatively well controlled, SCD can occur by either bradycardia or tachycardic means. In case 1, there was no history of ischaemic heart disease, but marked ST-segment elevation was noted an hour or so prior to death; the terminal arrhythmia was asystole. In case 2, there was no history of sustained ventricular arrhythmias, and no recent history of worsening angina. However, after an assessment of exercise capacity and clinical condition, at which he had performed relatively very well, he died soon after leaving out-patients from a tachyarrhythmia.

In neither case could the mode of death be predicted. In each case death was sudden, without a deterioration in heart failure, and occurred without reported prior symptoms. These dramatically different ventricular events stress the unreliability of concluding mechanisms of modes of death in heart failure from clinical details in the terminal few hours.

P. KELLY
A. COATS
Royal Brompton Hospital,
London, U.K.

References

Primary chylopericardium due to partial aplasia of the thoracic duct

We report the case of a 29-year-old female patient with massive asymptomatic isolated primary chylopericardium. Chylopericardium is an exceptionally rare entity. Since the first report[6] approximately 44 cases have been reported. The underlying aetiology remains mostly obscure and the disease is referred to as primary chylopericardium. However, in a few cases, lymphangiography has established that abnormalities of the lymphatic system were the cause, as was the case in our patient.

A routine X-ray of the chest showed an enlarged cardiac silhouette suggestive of massive pericardial effusion. Physical examination was completely normal as were standard laboratory tests and the lipid profile. Pericardiocentesis established the diagnosis of chylopericardium: it yielded 950 ml of milky, sterile fluid with a high content of triglycerides (2500 mg . dl ~ ¹). After pericardial aspiration the effusion gradually increased again over the ensuing 7 days. At cardiac catheterization at rest and exercise no signs of constriction or haemodynamic compromise were detected. Lymphangiography showed normal lymphatic vessels up to the level of the 12th thoracic vertebra. Aplasia of the cisterna chyli as well as of the distal part of the thoracic duct were found with collaterals predominantly at the left side. There was evidence of reflux in the left mediastinal and hilar lymphatic channels without direct leakage to the adjacent pericardium (see Fig. 1). Computed tomography of the chest could detect no direct lymphatic channels to the pericardial space. The patient was set on diet containing medium chain triglycerides and stayed well after a follow-up period of 30 months. There were no signs of an increase in pericardial effusion on chest X-ray or echocardiography.