

Fig 1. Coronary angiograms from twin I ( left panel, 1a-c) and twin 2 (Right panel, 2a-c)

We conclude that coronary anatomy is independent of the human genome. Disease lesion sites are at least partly independent of the human genome. In contrast, age at first cardiac event, type of cardiac event and risk factor profile appear to be more closely related to genetic profile. We suggest that when one twin presents with IHD, the second should be subject to increased medical surveillance

The authors have no conflict of interest

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#### DRUGS, ELECTROLYTES AND TAKO-TSUBO CARDIOMYOPATHY: TRIPLE AETIOLOGY OF ACQUIRED LONG QT SYNDROME AND TORSADES DE POINTES.

Editor,

Physical or emotional stress can have unforeseen consequences. We document a 67 year-old female admitted with syncope following emotional stress. She had a history of depression and had been “crying and crying all day”. In addition, she had a history of ileostomy following severe diverticular disease. Her daily medication included ondansetron 4mg b.d. for nausea and fluoxetine 60mg for depression.

On admission, serum magnesium was low at 0.73mmol/l (0.75 – 1.25) and serum potassium was 3.9mmol/l (3.5 – 5.1). Troponin I was mildly elevated at 0.17u/l (0 – 0.04). B-type natriuretic peptide (BNP, Abbott) was grossly elevated at 2569pg/ml (normal < 100). Initial ECG (fig 1) showed new T wave inversion in ECG leads; II, III, aVf and V1 through to V6 with a prolonged corrected QT interval (QTc) of 524ms (upper limit of normal for females = 450ms). An ECG dated June 2007 was normal apart from a QTc of 509ms. She was initially treated as an anterior non-ST segment elevation myocardial infarction. Shortly after admission, she developed polymorphic ventricular tachycardia (torsades de pointes, figure 2). The risk of torsades de pointes increases substantially once QTc is > 500 ms. This was treated with a 200J DC shock, 4mmol of intravenous magnesium with oral beta-blocker, and potassium therapy. Further self-terminating runs of torsades de pointes occurred when her potassium levels dipped below 4mmol/l.

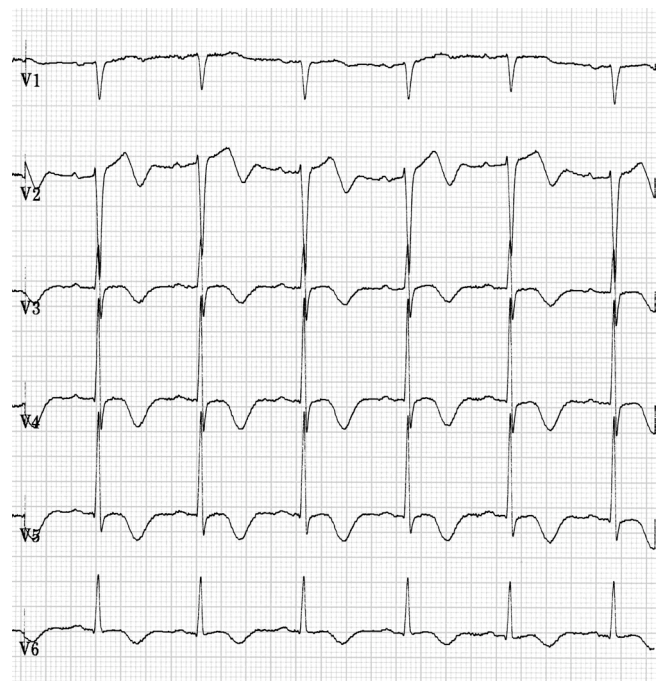


Fig 1. Leads V1 – V6 of admission 12-lead ECG showing T wave inversion resembling non-ST segment elevation MI. QTc is greatly prolonged at 524ms.

On day two, she underwent cardiac catheterisation, which showed normal coronary arteries but marked impairment of systolic function in the apical half of the left ventricle with a characteristic “ballooning” appearance (figure 3). These findings, in association with physical or emotional strain, are diagnostic of tako-tsubo cardiomyopathy. Oral magnesium supplements and bisoprolol 5mg were added in to her medication. Ondansetron and fluoxetine both prolong the QT interval and were stopped. A cardio-defibrillator device was

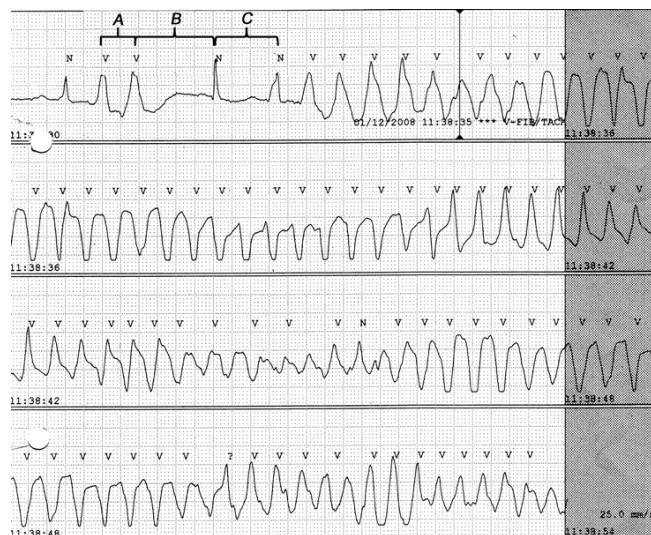


Fig 2. ECG monitor tracing shows a characteristic short (A), long (B), short (C) sequence of ventricular activity initiating polymorphic ventricular tachycardia also known as “torsades de pointes” for its twisting QRS axis about the iso-electric line.

implanted due to continued risk of arrhythmia from electrolyte loss from the ileostomy. The QTc came down to 454ms and BNP fell to 179pg/ml at discharge.

QT prolongation is the surface ECG manifestation of abnormal repolarisation of myocardial cells due to problems with cellular ion channels. The disorder is classified as either congenital or acquired. Acquired QT prolongation may be due to:

1. Electrolyte depletion, particularly potassium or magnesium,
2. Drugs that affect myocardial ion channels
3. A feature of tako-tsubo cardiomyopathy, a catecholamine induced metabolic disorder of myocardial cells caused by physical or emotional stress, especially seen in older females<sup>1,2</sup>.

A reference list of drugs causing QT prolongation is available from the University of Arizona (<http://www.azcert.org>) or the British National Formulary.

Initial presentation and ECGs in tako-tsubo cardiomyopathy are similar to an anterior ST or non-ST segment myocardial infarction but often with QT prolongation. A small troponin rise may be seen but coronary arteries are normal with a characteristic “apical ballooning” or Japanese octopus pot (“tako-tsubo”) pattern seen on ventriculography. Beta-blockade is a key element of treatment. The ventricular changes are mostly reversible if the patient survives the acute phase<sup>3</sup>.

Our patient had all three causes of an acquired QT prolongation - excessive secretion from her ileostomy producing hypomagnesaemia, daily ondansetron and fluoxetine therapy, and acute tako-tsubo cardiomyopathy. We believe the development of tako-tsubo cardiomyopathy exacerbated our patient’s pre-existing QT prolongation to a degree where potentially fatal arrhythmias occurred.

A case of congenital long QT syndrome and tako-tsubo cardiomyopathy with torsades de pointes has been described<sup>4</sup> but MEDLINE and PubMed searching (keywords: long QT

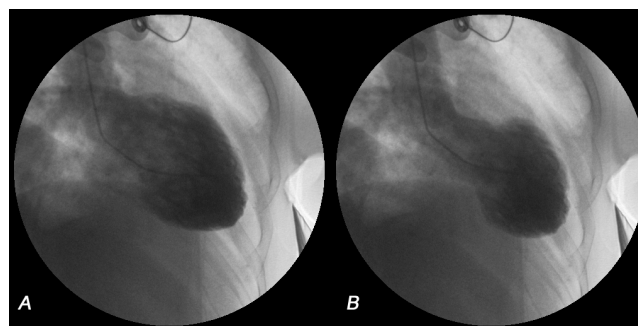


Fig 3. Left ventriculography during diastole (A) and systole (B) demonstrate typical left ventricular apical ballooning and hypercontractile base of tako-tsubo cardiomyopathy.

and cardiomyopathy) revealed no acquired cases. Tako-tsubo cardiomyopathy induced by physical or emotional stress may exacerbate an underlying long QT syndrome with risk of sudden cardiac death.

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#### PSEUDOMYXOMA PERITONEI PRESENTING AS INGUINAL HERNIA.

Editor,

Pseudomyxoma peritonei (PMP) is an uncommon disease with varied presentations. We present two cases presenting at inguinal hernia repair.

**Case 1:** A 41 year-old man presented for right inguinal hernia repair. An encysted swelling was discovered at surgery. Histopathology of the sac showed chronic inflammatory tissue containing lakes of mucin but no neoplastic epithelial cells. Postoperative CT scan showed thickening around the caecum with a fluid collection and abnormality related to the appendix. Colonoscopy and biopsies were normal. The patient was referred to the National Specialist Commissioning Advisory Group Pseudomyxoma Peritonei Centre (Basingstoke) where a laparotomy revealed a