Letters

LOBAR PNEUMONIA TREATED BY MUSGRAVE PARK PHYSICIANS.

Editor,

In the excellent historical article by John Hedley-Whyte, I saw the photograph of Sir Alexander Fleming with Professor William Thomson on the doorsteps of Number 25 University Square and I remembered that in my grandfathers visitors book there were the signatures of Sir Alexander and Lady Fleming not only at number 12 University square (figure 1), but also at Greenlawn in Donaghadee (figure 2). The exact date is not clear but I suspect about April 1942.

Professor CG Lowry (known as CG) and Professor Thomson (known as WWD) were close friends, colleagues and neighbours both in University Square, CG at number 12, and WWD at Number 25, and also next door at Donaghadee, and hence this accounts for the above records of those events.

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CORONARY ARTERY DISEASE: ANATOMY AND PRESENTATION IN IDENTICAL TWINS

Editor,

A 47 year old man (twin 1) was admitted electively for coronary angiography following an acute myocardial infarction (MI) one month previously. His risk factor profile included smoking, a positive family history, hypertension and hypercholesterolaemia. On the day of admission, it was discovered that his identical twin brother (twin 2) was an elective inpatient for coronary angiography. His history included acute MI aged 42 years, with subsequent percutaneous coronary intervention to the circumflex. His risk factor profile included previous MI, a positive family history, hypertension and hypercholesterolaemia.

Coronary angiograms were performed on consecutive days. Coronary arterial anatomy was discordant between the twins. Angiographic images from twin 1 are shown in figure 1 (panels 1a-1c), beside matched images from twin 2 (panels 2a-2c). In twin 1 the left main stem bifurcates into left anterior descending (LAD) and circumflex (CX) branches (panel 1a), while in twin 2 it trifurcates into an LAD, CX and ramus intermedius branch (panel 2a). The first obtuse marginal branch (OM1) arises and bifurcates proximally in twin 1 (panels 1a and 1b) but arises and bifurcates more distally in twin 2 (panels 2a and 2b). The right coronary artery supplies a prominent sinus node branch in twin 1 (SA node, panel 1c) which is not apparent in twin 2 (panel 2c).

Coronary artery disease distribution was also discordant between the twins. Twin 1 was found to have a normal left main stem, with a long area of moderate to severe disease in the mid part of the LAD. A large diagonal branch had a 90% ostial lesion. There was a 50% lesion in the main CX and a 90% lesion in its first marginal branch. The right coronary artery was diffusely diseased. Twin 2 had a normal left main stem, with an angiographically near-normal LAD. The CX was diffusely diseased. The right coronary artery was diffusely diseased but with no significant stenosis.

Our observation of discordant coronary artery distribution and coronary atherosclerosis in identical twins supports the findings of previous observational studies. Furthermore age at first cardiac event, type of cardiac event and risk factor profile show concordance in this pair of identical twins, also consistent with previous observations.
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 POINTEES.

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.

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 added.

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

**Fig 2.** Leads V1 – V6 of admission 12-lead ECG showing T wave inversion resembling non-ST segment elevation MI. QTC is greatly prolonged at 524ms.

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