Response to Adam Morton's Letter 'Reply to: Timothy A C Snow, Cara A Wasywich and Fiona M Stewart. A case of breathlessness during pregnancy: the difficulty in diagnosing heart failure'

To the Editor

Our thanks to Dr Morton for his thought provoking letter.

He asks whether the clomipramine may have contributed to her dilated cardiomyopathy. Our patient had been on clomipramine for several years prior to her admission. If she had prior left ventricular impairment from clomipramine we would have expected her cardiac function to have decompensated earlier in pregnancy (around 20–28 weeks' gestation, corresponding to the time of maximal rate of increase in cardiac output). She presented at 31 weeks' gestation with at most a two-week history of deteriorating shortness of breath. This patient was re-challenged with clomipramine a year ago due to escalating obsessive compulsive disorder. Over that time we have seen continued improvement of her left ventricular function.

The diagnosis of a peripartum cardiomyopathy was a diagnosis made in the absence of any other identifiable cause despite the relatively early onset of symptoms at about 29 weeks' gestation. The European Society of Cardiology Working Group on Peripartum Cardiomyopathy has extended their definition of peripartum cardiomyopathy earlier than the final four weeks of a pregnancy to encompass the small number of women who present earlier without any other apparent cause for the cardiomyopathy.¹ A cardiac biopsy was performed with no diagnostic histopathology. It was negative for iron and amyloid stains. Coronary angiography was normal. Viral studies were negative although these do not rule out a postviral cardiomyopathy and there was no family history of a dilated cardiomyopathy. It was highly unlikely given the gestation at the onset of symptoms and the

severity of the left ventricular impairment that she had a pre-existing cardiomyopathy. At the time of presentation anti-phospholipid syndrome or phaeochromocytoma were not considered. Neither condition has developed subsequently. Placental abruption is likely to explain the disseminated intravascular coagulopathy and ischaemic complications observed in this case.

Dr Morton asks about the role for aldosterone antagonists for women with dilated cardiomyopathies in pregnancy. Unfortunately in this situation the diagnosis was made at the time of cardiac arrest and fetal death. Spironolactone is a Class C drug in pregnancy mainly because of reports of feminization of male rats but also as a diuretic it will reduce placental perfusion and therefore may reduce fetal growth. There may be a role for the use of this drug in women with refractory heart failure in pregnancy in an attempt to prolong the duration of the pregnancy, to improve fetal outcome. Ideally use of this medication should be carefully monitored and reported to a central registry such as the European Working Group on Peripartum Cardiomyopathy to advance our understanding of the risks and benefits of aldosterone antagonists in pregnancy.

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