The risks of delay in diagnosis of breathlessness in pregnancy

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Be aware of cardiac dyspnoea in pregnant women when they are breathless

Heart disease is an important cause of dyspnoea in pregnancy. The diagnosis can easily be missed by the unwary unless it is specifically looked for. Owing to the physiological change and hyperdynamic circulation of pregnancy, patients often deteriorate suddenly and unexpectedly, catching both them and their doctors by surprise. If the diagnosis is not made early this may prove fatal. We describe five cases (over two years) of cardiac disease presenting for the first time during pregnancy.

Case reports

CASE 1

A 33 year old Asian (Indian subcontinent) woman presented in the 16th week of pregnancy (para 3+0). Since her arrival from Pakistan five years previously she had been complaining of shortness of breath with poor exercise tolerance, and had a copious productive cough with clear sputum. She was treated by her general practitioner with antihistamines for "allergy." She was admitted acutely to the obstetrics unit as she was thought to be draining liquor. The medical team was then asked to see her in view of dyspnoea at rest. A few days before her acute admission she had also been complaining of a worsening cough with greenish sputum.

On examination she was dyspnoeic at rest, feverish, in sinus tachycardia, and had widespread rhonchi and right basal crepitations. No heart murmurs were heard. A chest x ray was not requested until the next day as it was thought that this was a case of infective exacerbations of previously undiagnosed asthma. There was also some reluctance to perform a chest x ray as she was pregnant. She improved initially with oxygen, nebulisers, and antibiotics, but several hours later she suddenly became very dyspnoeic and started coughing copious amounts of sputum.

She suffered respiratory arrest, was intubated successfully, resuscitated, and transferred to the intensive care unit. The chest x ray film showed a "white out" of both lung fields with normal heart size but some left atrial dilatation. Swan-Ganz catheterisation showed a pulmonary capillary wedge of 25 mm Hg. A cardiological opinion confirmed severe mitral stenosis, and urgent echocardiography showed severe mitral commissural fusion with a Doppler estimated mitral valve area of 0.7 cm. She was transferred for urgent open valvotomy but died in refractory pulmonary oedema within 24 hours of operation.

CASE 2

A 24 year old white woman, a known asthmatic and 24 weeks pregnant (para 1+1), presented with a one day history of increasing shortness of breath. She was treated as having an acute asthmatic attack and responded initially to treatment but suddenly deteriorated after 2 days and required ventilation. A chest x ray film showed severe pulmonary oedema. This was thought to be due to consolidation or aspiration with pulmonary oedema. Shortly afterwards her right pupil became fixed and dilated and urgent computed tomography of the head confirmed a large infarct.

Postmortem examination showed vegetations on the mitral valve but there was no sign of any previous rheumatic damage. There was similar vegetative material on the brain. The diagnosis was subacute bacterial endocarditis on a prolapsing mitral valve, complicated by cerebral embolus.

CASE 3

A 21 year old Asian woman presented in the 20th week of pregnancy (para 1+0). One week before her acute admission she had been complaining of dyspnoea. Her general practitioner treated her with antibiotics for a presumed chest infection. On the day of admission she suddenly became worse and on arrival in hospital had a respiratory arrest, for which she was successfully ventilated and transferred to the intensive care unit. On examination she had bilateral crepitations, but there was no heart murmur. Echocardiography showed she was in sinus rhythm. A chest x ray film showed bilateral shadowing of the lung fields and an enlarged left atrium. The pulmonary wedge pressure was 25 mm Hg and pulmonary pressure was 44 mm Hg. An urgent cardiological opinion was sought, and echocardiography showed an enlarged left atrium at 4.5 cm, and severe rheumatic mitral stenosis with mitral valve gradient estimated at 20 mm Hg. She was resuscitated with diuretics and cautious use of cardiac inotropes. She was then transferred for cardiac surgery, and mitral valvotomy was performed successfully. Unfortunately intrauterine death ensued and the fetus had to be aborted.

CASE 4

A 21 year old Asian woman, 33 weeks pregnant, presented with a three month history of shortness of breath and dry cough, not previously investigated. She had cough, dyspnoea, haemoptysis, and pleuritic chest pain. On admission she was in sinus rhythm, and had a loud first heart sound and mid-diastolic murmur. Echocardiography showed P mitrale and a chest x ray film showed evidence of pulmonary oedema. An urgent echocardiogram showed severe mitral stenosis with valve area and gradient estimated at 0.6 cm and 19 mm Hg respectively. Left atrial size was 4.7 cm. The posterior mitral leaflet was calcified and mobility of the anterior leaflet was reduced. Left ventricular function was good. A ventilation-perfusion scan showed evidence of a ventilation and perfusion mismatch. She was therefore anticoagulated with heparin for pulmonary embolus and her pulmonary oedema was treated with diuretics. A cardiological opinion was then sought while she was in the intensive care unit. A healthy baby boy was delivered by caesarean section. Two months later she had her mitral valve replaced with a Bjork-Shiley metal prosthesis.

CASE 5

A 20 year old Asian woman, 16 weeks pregnant (para 1+0) and previously healthy, presented with a four day history of increasing dyspnoea. On examination she was in fast atrial fibrillation, had a loud pansystolic murmur radiating to the axilla, and had bilateral crepitations. A chest x ray film showed cardiomegaly, left atrial enlargement, and pulmonary oedema.

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Cardiological opinion was then sought. Urgent echocardiography showed severe mitral regurgitation with an enlarged left atrium at 7 cm and left ventricular volume overload. She was treated with diuretics and given digoxin. Her pregnancy was terminated and she later had a successful valve replacement.

Discussion

These five cases illustrate the importance of excluding cardiac dyspnoea as a cause of dyspnoea in pregnancy. In each case the correct diagnosis was not made until the patient had become very unwell. In cases 1, 3, 4, and 5 cardiological opinion was sought only when the patients were in intensive care—in other words, after they had suddenly deteriorated. They were previously seen either by their general practitioner or by hospital doctors.

Only in two out of the five cases were heart murmurs detected. Perhaps that, and the reluctance to perform chest x ray examinations during pregnancy, the lack of suspicion of heart disease, and the failure to recognise left atrial enlargement on x ray film, explain why the diagnoses were unnecessarily delayed.

Doctors worry about irradiating pregnant women because of the risk of fetal damage. This risk is probably limited to the first trimester, however, and none of the five patients was seen in the first trimester. Therefore, this reservation should be overruled, particularly in the second and third trimesters of pregnancy.

All five cases occurred over two years, and all four cases of mitral valve disease were in Asian women. The relative rarity of mitral stenosis in young British women may have led to a deterioration in its management during pregnancy in the United Kingdom, although it is being seen increasingly in young Third World immigrants. This presumably is due to an increased prevalence of rheumatic heart disease in Asian women.

Heart disease remains the most important cause of non-obstetric death in pregnancy.¹ The main hazards are pulmonary oedema when it occurs suddenly in mitral stenosis, infective endocarditis, pulmonary

hypertension, and fulminating peripartum cardiomyopathy. Pregnancy and the peripartum period are associated with substantial circulatory changes. In the woman with heart disease these changes can lead to rapid clinical deterioration, resulting in greater morbidity and possibly death.

Mitral stenosis is the commonest and most commonly fatal of all cardiac conditions during pregnancy.² It is essential that it is diagnosed and carefully assessed in pregnancy. Symptoms may have been mild or even absent, and the patient and doctor may have been unaware of the heart disease. These patients can develop pulmonary oedema rapidly as tachycardias during pregnancy reduce the diastolic time for the left atrium to empty. This reduces stroke volume, and in order to maintain cardiac output heart rate further increases. This then leads to further reduction in left atrial emptying and eventually left atrial pressure rises, resulting in pulmonary oedema.

Valve surgery is no more risky to a pregnant woman than to a non-pregnant woman, but there is a risk to the fetus from the cardiopulmonary bypass and general anaesthetic, as illustrated in case 3. Open and closed valvotomy, balloon valvuloplasty, and mitral valve replacement have been performed in severely symptomatic patients during pregnancy, all with good results.^{3,4} Only one maternal death was reported in 68 cases in one series, together with a fetal survival rate of more than 80%.⁴ Open procedures carry a higher risk of fetal loss than closed procedures. Balloon valvuloplasty is therefore likely to be the treatment of choice if the mitral leaflets are mobile and not calcified. β blockers may be used to reduce tachycardia provided the maternal haemodynamic state is stable.

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2 McFaul PB, Dornam JC, Lamki H, Boyle D. Pregnancy complicated by maternal heart disease: a review of 519 women. *Br J Obstet Gynaecol* 1988;95:861.

3 Bernal JM, Mirallees PJ. Cardiopulmonary bypass during pregnancy. *Obstet Gynecol Surv* 1986;41:1.

4 Safian RD, Berman AD, Sacho B, Diver DJ, Come PC, Bairn DS, et al. Percutaneous balloon mitral valvuloplasty in a pregnant woman with mitral stenosis. *Cathet Cardiovasc Diag* 1988;15:103-8.

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WAYS WITH WORDS

Changing names

I was sitting on a tribunal at what used to be the Countess of Chester Hospital, waiting for an appellant who was late. He arrived eventually with profuse apologies and explained that he was a stranger to the area and had driven past the "Countess of Chester Health Park," not realising that it was a hospital.

The new NHS trusts seem to be in the business of throwing out well established dirty words, like "hospital," presumably in favour of something more user, consumer, client—or should it be punter (ugly word)?—friendly; never, of course, the politically incorrect "patient." Some of the new titles are pretentious or dull. In the city where I used to practise East Birmingham Hospital is now Heartlands and Dudley Road Hospital has become the City Hospital Trust—as though there were not any other city hospitals in that large metropolis.

How many of the hundreds of hospitals that have now become trusts have changed names, have had to design new logos, print runs of stationery for every department, and make changes to signposts and notice boards? Not to mention the implications for items like medical and telephone directories.

Does it matter that well known (and in a few cases well

loved) names should be relegated to history? Ironically a book called *Hospital Names* has just been published, perhaps in anticipation of their imminent demise. Those of us with a touch of paranoia will see this as the latest move to "disappear" consultants. It began in one of the many previous reorganisations when hospitals were renamed district health authorities. Consultants now worked for an authority and it was difficult to identify their hospital. With new titles which presumably do not have to include hospital trusts will no longer need to employ medical consultants, only consultants who have to sort out the overdraft (overspend). My advice is that hospital doctors should quickly rename themselves vice presidents (for corporate affairs?).

Perhaps some good will result. Reading about the newish specialty of adolescent medicine, I see that there are now adolescent physicians and psychiatrists. I suppose that they are more grown up than baby consultants—recently appointed? Or do they look after infants but are not yet wise enough to be elderly or geriatric physicians? Diabetic physicians will no doubt resist abolition, but I shall not miss being called an alcoholic physician.—ALEX PATON is a retired physician in Oxfordshire