contained medium sized malignant cells with vesicular pleomorphic nuclei and brisk mitotic figures arranged in sheets, cords, and small glands. The tumour extended into half of the muscle layer with lymphatic permeation. There was no evidence of further infiltration to the parametrium. The tumour was classified as stage Ib grade III. The patient made an uneventful recovery and showed no evidence of recurrence at follow up.

One year later she complained of ankle swelling, abdominal distension, and decreased exercise tolerance and on 21 October was readmitted with severe right sided heart failure. Two dimensional echocardiography (fig) showed a large dumb-bell shaped tumour occupying the whole right ventricular cavity.

Apical four chamber view of heart showing the tumour (T) occupying whole right ventricular cavity.

As no difficulty had been encountered in pacing the ventricle in 1983 and an atrial myxoma was unlikely to grow to this size within a year cardiac metastasis was suspected. At operation on 29 October the tumour was found to be arising from the lower half of the anterior right ventricular wall and protruding into the atrium. It appeared to have an endothelial lining with subendothelial haemorrhage. Two of the chordae of the tricuspid valve had ruptured but the valve was normal. The tumour was removed piecemeal and as completely as possible. Histologically irregular masses of malignant tissue were scattered in a background of organised thrombus and fibrosis. The tumour consisted of highly pleomorphic anaplastic cells with numerous mitotic figures arranged in glandular pattern. The picture was consistent with metastasis from the endometrial carcinoma. Postoperative recovery was uneventful, and as there was no evidence of metastasis elsewhere she was given progestogen treatment.

Comment

Cardiac metastasis occurs in 1.5-18.3% of patients who die of malignant disease. The most frequent sites of origin include lung, breast, lymphoma, leukaemia, and melanoma.¹ Cardiac metastasis associated with gynaecological malignancies is rare. Of 1100 cases of gynaecological cancer, only six patients with terminal squamous carcinoma of cervix or vulva had cardiac metastasis.² In tumour metastasis to the heart the pericardium, myocardium, or endocardium may be affected singly or in combination.³ Endocardial implantation is the least common and usually occurs on the valves. Owing to the relative avascularity of the endocardium and rapidity of blood flow through the chambers, a degree of valvular damage may be necessary for implantation.⁴

Our case, which illustrates the value of two dimensional echocardiography in the prompt diagnosis of cardiac metastasis,⁵ showed several unusual features. Finding an isolated intracavitary metastasis complicating a stage I tumour is atypical in endometrial carcinoma. Furthermore, implantation occurred at an uncommon site corresponding to the endocardial pacing site. Thus we postulate that the temporary pacing wire being left in situ for one week may have traumatised the right ventricular endocardium, the tissue reaction and fibrin deposition providing a nidus for implantation by the tumour cells, which might have been released into the circulation around the time of total hysterectomy.

In view of this case we suggest that caution should be exercised when considering temporary pacing in patients with malignant disorders.

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Acute renal failure presenting in pregnancy secondary to idiopathic hydronephrosis

Acute renal failure secondary to idiopathic hydronephrosis has not been documented in pregnancy. We report a case of anuria occurring in pregnancy in a patient with previously undiagnosed obstruction at the ureteropelvic junction.

Case report

A 25 year old primigravid patient was booked for confinement at a gestational age of 13 weeks. There was no relevant history. She was normotensive and urine analysis showed nothing abnormal. Pregnancy progressed uneventfully until she was admitted as an emergency at 27 weeks because of vomiting, anorexia, left sided back pain, and a small antepartum haemorrhage. She was not feverish and blood pressure was 150/90 mm Hg. Tenderness was elicited in the left loin. Uterine size was appropriate for gestational age and the fetal heart was audible. The urine contained blood and protein.

Haemoglobin concentration was 9.5 g/dl, white cell count $13.7 \times 10^9/l$, blood urea concentration 31.3 mmol/l (189 mg/100 ml), and potassium concentration 5.8 mmol(mEq)/l. An ultrasound scan showed bilateral hydronephrosis. Only 25 ml urine was passed during the first 24 hours after admission, and the blood urea value rose to 41.0 mmol/l (247 mg/100 ml) and potassium to 7.8 mmol/l. Haemodialysis was therefore carried out on two occasions within 72 hours of admission. In view of the bilateral hydronephrosis an attempt was made to pass retrograde ureteric catheters, but the stenosis could not be negotiated on either side. Consequently, bilateral percutaneous nephrostomies were performed under ultrasound control and the nephrostomy catheters left in situ. After this procedure large amounts of urine drained from the catheters, and blood urea and potassium concentrations returned to normal within 48 hours. The urine remained sterile.

On the sixth day of admission (gestational age 28 weeks) the amniotic membranes ruptured spontaneously, followed within 24 hours by uterine contractions. Labour progressed rapidly and she had a vaginal delivery of a live boy weighing 1400 g. The progress of the baby was essentially satisfactory apart from rather persistent apnoeic attacks during the neonatal period.

On the 10th postpartum day antegrade pyelography was performed using each nephrostomy tube and bilateral obstruction of the ureteropelvic junctions confirmed. On the 12th day a bilateral Anderson-Hynes operation was carried out. The postoperative course was uneventful and the patient was discharged from hospital seven days later. At follow up she remained well and free from loin pain, and renal function values continued to be within normal limits.

Comment

Idiopathic hydronephrosis due to obstruction at the ureteropelvic junction is usually partial and presents with loin pain. The obstruction becomes complete only if complicated by infection or stone. Neither was present in this case. We recognise that the previously accepted concept that progesterone is responsible for the ureteric dilatation in pregnancy^{1,2} has been strongly challenged in favour of an obstructive cause.³ Nevertheless, we believe that the increased circulating hormones in pregnancy must have had a part to play in the complete obstruction at the level of the ureteropelvic junction in this case. Surgical correction of the bilateral obstructions was carried out on the 12th postpartum day to prevent future deterioration in renal function, and by this time the thrombogenic effects of pregnancy are reversed.

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Captopril and domiciliary oxygen in chronic airflow obstruction

Inhibition of angiotensin converting enzyme has been reported to reduce pulmonary arterial pressure in primary pulmonary hypertension¹ and to reduce total pulmonary resistance and increase cardiac output in some patients with pulmonary hypertension secondary to collagen vascular disease.² We therefore investigated the effects of captopril (3-mercapto-2-methylpropionyl-proline), a potent and relatively specific angiotensin converting enzyme inhibitor, in five patients with pulmonary hypertension secondary to hypoxic chronic airflow obstruction.

Patients, methods, and results

The study group comprised five patients with chronic airflow obstruction (mean forced expiratory volume in one second (FEV₁) 0.71 l; mean forced vital capacity (FVC) 1.36 l; mean FEV₁/FVC 52%) and resting arterial hypoxaemia (mean arterial oxygen pressure (PaO₂) 6.8 kPa (51 mm Hg) breathing room air) who took domiciliary oxygen with proved good compliance for at least four months before the study (mean daily oxygen use 19.4 hours, assessed by a concealed meter attached to the oxygen concentrator machines).

Baseline measurements (see table) were made, and after an initial test dose of 12.5 mg, which was well tolerated, captopril was given in a dose of 25 mg thrice daily for three days. All measurements were repeated frequently during this period. Statistical analysis of the results at baseline and after three days of captopril was carried out by paired Student's *t* test.

Cardiac output was measured in triplicate using the thermodilution method (Edwards 9520 A cardiac output computer) and mean values recorded. Haemodynamic measurements were calculated by computer (Cardiac Recorders Ltd, Memory Monitor 3220) from pressure transducers (Bell and Howell type 4-327-1). All measurements, including blood gas analysis, were made after the patients had breathed room air for at least 30 minutes.

Baseline results (table) showed increased pulmonary vascular resistance and pulmonary arterial pressure, appreciable arterial hypoxaemia and mild hypercapnia, and a greatly increased alveolar-arterial oxygen gradient (A-aDo₂).

After three days of captopril both pulmonary arterial pressure and pulmon-

ary vascular resistance were significantly reduced ($p < 0.05$) and cardiac output significantly increased ($p < 0.01$). Although systemic blood pressure and systemic vascular resistance were reduced ($p < 0.01$), no patient developed symptoms related to inadequate perfusion. Both oxygen delivery to the tissues and mixed venous Po₂ (PvO₂) were significantly increased ($p < 0.01$), reflecting the increased cardiac output, although the rise in A-aDo₂ at the end of the study ($p < 0.05$) suggested that hypoxic pulmonary vasoconstriction was impaired by the drug.

Comment

Although the presence of pulmonary hypertension is associated with a poor prognosis in chronic airflow obstruction,³ a reduction in pulmonary arterial pressure alone may not be therapeutic unless tissue oxygenation is also improved.^{4,5} In our patients the reduction in pulmonary arterial pressure achieved with captopril was accompanied by significantly increased PvO₂ values and it therefore seems reasonable to suggest that in addition to its haemodynamic effects, tissue oxygenation was also increased by the drug.

Since three days' treatment with captopril had a beneficial effect on factors known to be associated with a poor prognosis in chronic airflow obstruction, and similar results have been reported with other vasodilators, further studies seem indicated in the context of a controlled clinical trial (with and without domiciliary oxygen) to assess the long term risks and benefits of vasodilator drugs in chronic airflow obstruction.

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Summary of results at baseline and after three days of captopril

Measurement	Baseline		Captopril		p Value
	Mean	SD	Mean	SD	
Pulmonary vascular resistance (PVR) (dyn-s-cm ⁻⁵)*	470.4	216.0	274.8	129.4	< 0.05
Cardiac output (CO) (l/min)	4.62	1.05	5.66	1.01	< 0.01
Mean pulmonary arterial pressure (PAP) (mm Hg)	33.8	7.3	26.0	8.1	< 0.05
Pulmonary artery capillary wedge pressure (PACWP) (mm Hg)	8.8	1.3	7.6	2.1	NS
Heart rate (beats/min)	93.2	19.3	101.6	24.0	NS
Systemic vascular resistance (SVR) (dyn-s-cm ⁻⁵)†	1552.0	316.4	1075.8	211.9	< 0.01
Mean systemic blood pressure (MBP) (mm Hg)	94.0	11.3	78.0	9.2	< 0.001
Right atrial pressure (RAP) (mm Hg)	6.8	4.1	3.6	2.6	< 0.05
PVR SVR	0.30	0.13	0.26	0.14	NS
Pressure of arterial oxygen (PaO ₂) (kPa)	6.8	0.9	6.6	0.9	NS
Pressure of arterial carbon dioxide (PaCO ₂) (kPa)	5.8	1.1	5.6	1.1	< 0.05
Mixed venous oxygen tension (PvO ₂) (kPa)	3.8	0.7	4.1	0.6	< 0.01
Alveolar-arterial oxygen gradient (A-aDo ₂) (kPa)‡	5.6	1.4	6.4	1.2	< 0.05
Arterial O ₂ content (CaO ₂) (ml %)	17.0	1.9	16.8	1.7	NS
Oxygen delivery (ml/min)*	793.4	235.8	962.0	269.9	< 0.01

NS = Not significant.

*[(PAP-PACWP)/CO] × 80.

†[(MBP-RAP)/CO] × 80.

‡Conversion: SI to traditional units—PaO₂, PaCO₂, PvO₂, A-aDo₂: 1 kPa ≈ 7.5 mm Hg.

‡[150-(PaCO₂/0.8)]-PaO₂.

§Haemoglobin (g/dl) × 1.34 × % saturation + 0.003 × PaO₂.

¶CaO₂ × 10 × CO.