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Short reports

Postpneumonectomy interatrial right-to-left shunt

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The mechanism for a right-to-left shunt may often be difficult to determine. Conditions such as pulmonary embolism, interstitial oedema, low cardiac output, and an increase in the closing volume above functional residual capacity may simulate it. The following case report illustrates an unusual mechanism for a right-to-left shunt after right pneumonectomy which was suspected at the bedside from a low blood oxygen content after inhalation of 100% oxygen.

Case report

A 68-year-old white man was admitted to Albert Einstein Medical Center on 14 December 1977, six weeks after a right pneumonectomy for squamous cell carcinoma. At discharge, after pneumonectomy, the patient was hypoxaemic (P_{O_2} 55 mmHg (7·5 kPa)) but otherwise asymptomatic. For several days before admission he suffered dyspnoea at rest, worsened by exertion, without productive cough, fever, or pleuritic pain. He had received six radiotherapy treatments of 200 rads each before admission. His pneumonectomy had been complicated by postoperative hypoxaemia requiring oxygen, and intermittent supraventricular tachycardia. Preoperative spirometry had shown forced vital capacity of 3·14 l (66% of predicted), FEV_1 2·27 l, FEV_1/FVC-73% and $FEF_{25-75\%}$-73% of predicted. His previous medical history included an inferior wall myocardial infarction and asbestos exposure.

Physical examination revealed an acutely ill man in severe respiratory distress. His temperature was 36°C, pulse 88/min, blood pressure 126/70 mmHg, and respirations 40/min. There was cyanosis of the lips, fingers, and toes. Examination of the chest revealed no breath sounds over the right hemithorax; the left hemithorax was resonant to percussion and clear to auscultation. There were no subjective changes in symptoms or signs of respiratory distress with change in posture. Cardiac examination revealed no enlargement, a regular rhythm, and no extra heart sounds or murmurs. The chest radiograph was similar to the post-pneumonectomy film. There was no infiltrate or evidence of heart failure in the left lung. The cardiac outline was not increased. The electrocardiogram showed normal sinus rhythm and diffuse non-specific T wave flattening. Arterial blood gases revealed P_{O_2} 34 mmHg (4·5 kPa) (75·2% saturation), P_{CO_2} 19 mmHg (2·53 kPa), pH 7·55, and bicarbonate 16·2 mmol/l while breathing air.

The initial diagnosis was acute respiratory failure precipitated by pulmonary embolism. The trachea was intubated and ventilation was assisted with a Bennett MA-1 ventilator. The arterial blood gases on 100% oxygen were: P_{O_2} 69 mmHg (9·2 kPa); saturation 94·2%, P_{CO_2} 34 mmHg (4·35 kPa), pH 7·47, and bicarbonate 22·2 mmol/l.

A pulmonary angiogram was performed while the

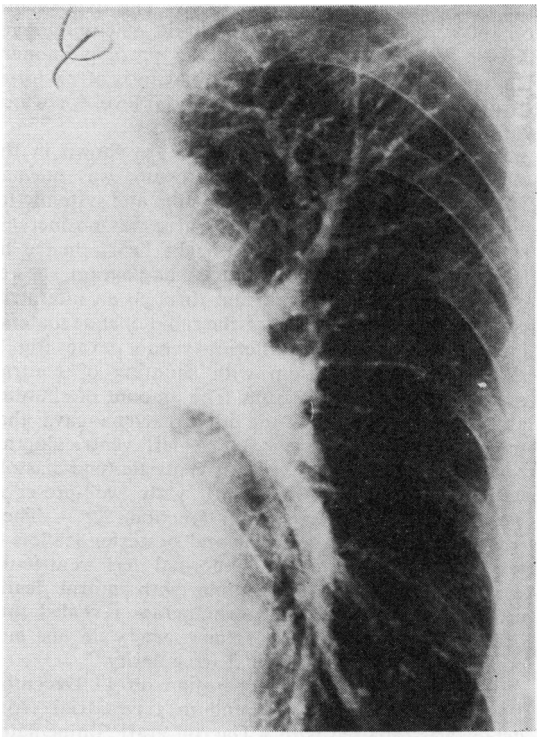

Fig 1 *Normal pulmonary angiogram.*

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Table *Cardiac catheterisation data*

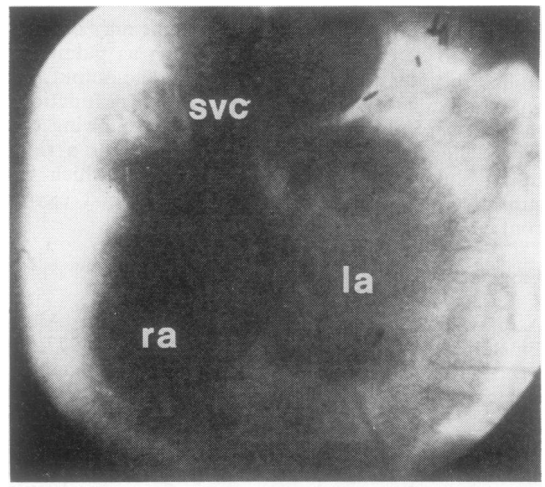
Chamber or blood vessel	*Pressure (mmHg)*	*Mean*
Right atrium	a = 9, v = 1	3
Right ventricle	21/5	
Pulmonary artery	22/9	15
Left ventricle	130/6	
Aorta	130/74	96
Oxygen saturation (%)		
Inferior vena cava		73·4
Superior vena cava		66·3
Right atrium		
High		67·8
Mid		69·3
Low		66·2
Right ventricle		
High		74·5
Low		67·1
Pulmonary artery		
Main		63·5
Left		67·3
Right		70·2
Aorta		86·8
Oxygen consumption		232 l/min
Systemic blood flow		6·22 l/min
Pulmonary blood flow		4·28 l/min
Effective pulm. blood flow		4·28 l/min
Left-to-right shunt		—0—
Right-to-left shunt		1.94 l/min
Systemic: pulmonary blood flow ratio		1·45 : 1
Total pulmonary vascular res.		280 dynes/sec/cm^{-5}
Total peripheral res.		1330 dynes/sec/cm^{-5}

patient was being ventilated artificially (fig 1). The left pulmonary vasculature and pulmonary arterial pressure were within normal limits. The hypoxaemia on 100% oxygen without an apparent new pulmonary disease process on the chest radiograph or pulmonary angiogram led to a presumptive diagnosis of an intracardiac right-to-left shunt at the atrial level for which cardiac catheterisation was performed.

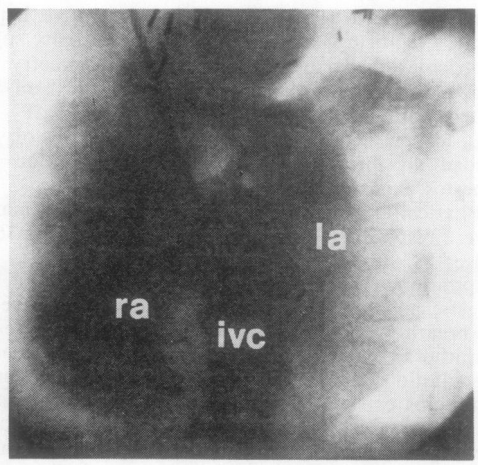
The cardiac catheterisation data are shown in the table. The pulmonary arterial pressure was normal. The calculated shunt was 1·94 l/min, and systemic-to-pulmonary blood flow 1·45 : 1. There was no increase in the oxygen content in the right heart, hence no left-to-right shunt. A right atrial angiogram showed opacification of the left atrium through an interatrial communication. Injection of the radio-opaque material into the superior and inferior venae cavae (fig 2) showed the right atrium with shunting of contrast material into the left atrium. The amount of shunting appeared greater from the inferior vena cava than from the superior vena cava. A left ventriculogram revealed a slightly enlarged left ventricle (end-diastolic volume index of 101 ml/m^2 with well-preserved ventricular function (ejection fraction 82%). There was prolapse of the anterior and posterior leaflets of the mitral valve and an abnormal left ventricular contraction pattern compatible with mitral leaflet prolapse. The coronary cineangiogram revealed total occlusion of the right coronary artery in its mid-portion and proximal luminal irregularity.

The patient underwent operation on 17 December 1977 when a 2 × 2 cm secundum type atrial septal defect was repaired with a Dacron graft. Immediately after closure of the defect, the arterial blood gases on 100% oxygen were: Po_2 238 mmHg (31·7 kPa), saturation 99%, Pco_2 29 mmHg (3·87 kPa), pH 7.43, and bicarbonate -19 mmol/l.

On 40% oxygen the arterial blood gases were: Po_2 97 mmHg (12·9 kPa), Pco_2 39 mmHg (5·2 kPa), pH 7·42, and bicarbonate 19 mmol/l. On the second postoperative day the patient developed a new pulmonary infiltrate and tracheal secretions grew *pseudomonas aeruginosa*. Despite aminoglycoside and carbenicillin therapy, the patient deteriorated and septic shock ensued. The patient died despite efforts to reverse the shock with fluid, antibiotic, and steroid therapy. Necropsy was not performed.



(a)



(b)

Fig 2 *(a) Contrast material is injected into superior vena cava (svc). There is shunting of the radiopaque material into the left atrium (la); ra = right atrium. (b) Contrast material is injected into inferior vena cava (ivc). Left atrium is well opacified.*

Discussion

Post-pneumonectomy dyspnoea caused by a right-to-left interatrial shunt has been described previously in only three patients.[1–3] As in our patient, the symptoms were manifested only after right pneumonectomy. The shunt occurred across either a patent foramen ovale or a secundum atrial septal defect. Patency of the foramen ovale and right-to-left shunt in critically ill patients has been attributed to the pulmonary hypertension associated with pulmonary emboli, pulmonary stenosis, positive pressure ventilation with positive end-expiratory pressure, and severe chronic obstructive pulmonary disease.[3 4]

The present case is of particular interest since there was no pulmonary or right atrial hypertension to account for the right-to-left shunt. In addition, this case differs from those previously described in that our patient developed hypoxaemia which was unaffected by position immediately after operation. From careful review of the angiograms, the most likely mechanism of the shunt seems to be "streaming" of blood flow.

Indeed, as reported by other investigators, a small right-to-left shunt may be present in patients with atrial sepal defect and normal pulmonary artery pressures, the greater proportion of the shunted blood originating from the inferior vena cava.[5] A shunt, whether cardiac or intrapulmonary in origin, may be suspected clinically by a poor response to inhalation of 100% oxygen. Post-pneumonectomy right-to-left interatrial shunting may occur without any increase in right atrial pressure or pulmonary hypertension. Presumably, after pneumonectomy, there is an alteration in the anatomical position of the heart, which allows previously undetected interatrial defects to become clinically significant. In the present case as well as in the three previously reported, the usual electrocardiographic, chest radiographic, and physical findings of atrial septal defect were lacking. The diagnosis was suspected by the lack of response to an inspired oxygen concentration of 100% and the inability to detect a mechanism on the chest radiograph. Confirmation is by right or left cardiac catheterisation and appropriate dye dilution studies. Early surgical correction should be considered as definitive treatment in these cases of atrial septal defect.

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