rhabdomyosarcoma of the bladder could be causing the obstruction.

Ifosfamide is an ozazophosphorine derivative of cyclophosphamide. This chemotherapeutic agent is an attractive alternative for the treatment of cyclophosphamide-resistant tumors. It also has lower bone marrow toxicity. Specific damage to the proximal tubule results in a Fanconi-type renal tubular syndrome. This consists of diminished phosphate resorption from the proximal tubules and a hypophosphatemic state (*9*). Normal plasma calcium is maintained. Excess phosphate in the urine can lead to a radiographic pattern of rickets (*10*). In this report, the damage to the kidney, as shown by the decreased DMSA uptake, occurred before any radiographic evidence of rickets at the growth plate. Other renal tubular resorptive mechanisms are also damaged, resulting in glycosuria, renal tubular acidosis and abnormal vitamin D metabolism. This complication is more common in smaller children in the first decade of life and is described as producing rickets more commonly in children with a single kidney, such as after a nephrectomy for Wilms tumor (*11*). This complication is presumed to be dose-related, but there is a single case report of a child who developed Fanconi syndrome after the first dose of ifosfamide (*12*), which suggests that the nephrotoxicity of ifosfamide to the proximal renal tubules may not be completely dose-related. Earlier treatment with nephrotoxic drugs such as cisplatin or carboplatin exaggerates the damage from ifosfamide.

DMSA has been shown to be exquisitely sensitive in detection of renal tubular damage from ifosfamide. A quantitative serial study in children receiving ifosfamide documented a cumulative pattern of injury (*13*). This pattern showed decreased renal and background activity with increased bladder activity. None of the children showed activity in a dilated renal pelvis and ureters as our patient. The sensitivity to renal tubular damage was superior to laboratory measurements, including β_2 microglobulin values in the urine and renal tubular resorption of phosphate. DMSA was recommended as a suitable method to access ifosfamide-induced renal tubular damage. This case report also documents the superiority of the DMSA scan in detecting renal tubular damage over radiographic changes of rickets at the growth plate. This patient's radiographs of the wrists and knees were normal at the time of the DMSA scan.

In the present report, damage to the renal tubular cells from ifosfamide also interrupted the transport mechanism for binding DMSA. Without this binding to the renal tubular cells, DMSA was excreted by glomerular filtration, thereby accounting for the unusual pattern seen in Figures 1 and 2.

CONCLUSION

Ifosfamide may produce damage to the renal tubular transport mechanism, which is responsible for the binding of DMSA. We have described an altered uptake pattern on the DMSA scan that more closely resembles that of a glomerular filtration agent as seen, for example, with DTPA. This unusual uptake pattern can be recognized as a side effect of chemotherapy from ifosfamide.

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Lung Scintigraphy in Postpneumonectomy Dyspnea Due to a Right-to-Left Shunt

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We report the case of a 50-yr-old man who experienced exertional dyspnea 5 mo after a left pneumonectomy for carcinoma. As the clinical features pointed toward a pulmonary embolism, we performed a ventilation plus perfusion radionuclide lung scan. It showed no evidence of pulmonary embolism, but it did show a systemic uptake of the isotope, suggesting a right-to-left shunt that was confirmed by contrast echocardiography, which revealed an atrial septal defect. Right-to-left shunts after pneumonectomy have already been reported and can be diagnosed by lung scintigraphy. Usually, a patent foramen ovale is encountered, but the underlying physiopathology remains under discussion. Clinically, right-to-left shunts are often related to platypnea-orthodeoxia.

Key Words: ventilation; perfusion lung scan; pneumonectomy; dyspnea; platypnea-orthodeoxia

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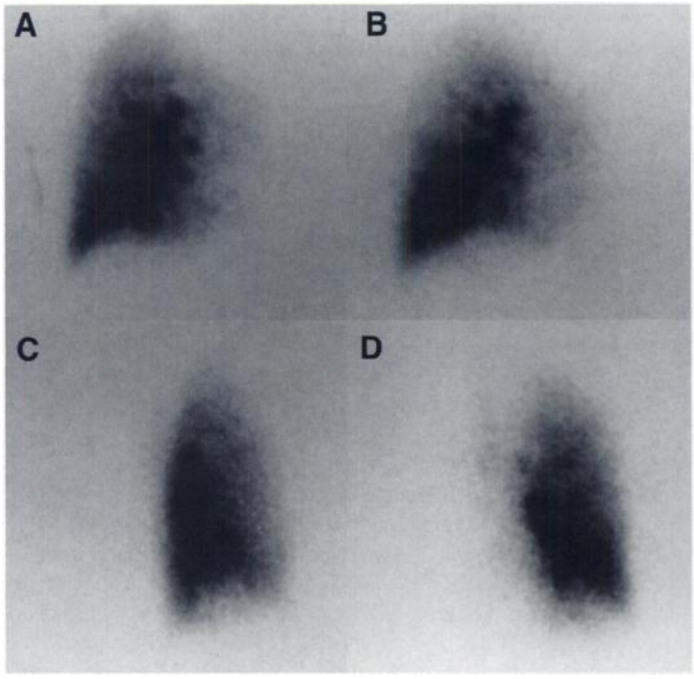

FIGURE 1. Ventilation lung scan (250,000 counts): (A) anterior; (B) right anterior oblique; (C) right posterior oblique; (D) posterior. The remaining right lung appears homogeneous.

Lung scintigraphy is usually performed during the exploration of a dyspnea to diagnose pulmonary embolism, but it has proven useful in the detection of right-to-left shunts (RLSs) in several diseases (*1–9*). Here, we present the case of a RLS occurring after a left pneumonectomy.

CASE REPORT

We report the case of a 50-yr-old man, an ex-smoker, who was in good health until an hemoptysis led to the discovery of a mixed bronchial carcinoma (adenocarcinoma and small cell carcinoma) at the origin of the left lower bronchus, with mediastinal adenopathy. A left pneumonectomy was performed, and three cycles of chemotherapy were begun, but they were interrupted because of anemia and renal failure.

Five months after the pneumonectomy, he started to experience dyspnea, first at exercise and then at rest. The clinical and radiological findings of the right lung were normal. The total vital capacity was 44% of normal capacity, and the first second forced expiration capacity was 56% of the total vital capacity. The patient's arterial blood showed a partial pressure of oxygen (PaO_2) of 6.5 kPa (49 mmHg) and a $PaCO_2$ of 4.4 kPa (33 mmHg) while he was breathing room air.

As these features suggested a pulmonary embolism, a ventilation and perfusion lung scintigraphy was performed.

The patient breathed an aerosol (ultravent design) of ^{99m}Tc-labeled phytates (1.1 GBq) to get four 250,000-count ventilation frames, and then we injected 0.2 mg (185 MBq) of ^{99m}Tc-labeled albumin macroaggregates (MAAs), i.e., at least 120,000 particles in an antecubital vein, while the patient was supine and made 500,000-count perfusion frames.

The ventilation was homogeneous (Fig. 1), and there was no evidence of perfusion defect in the remaining right lung (Fig. 2). However, after the MAA injection, an important activity was seen in the spleen, the myocardium and the kidneys. We made another view of the skull (Fig. 3); the thyroid, the encephalon, the salivary glands and the upper airway were clearly seen. The brain-to-lung uptake ratio was high (2.5%). The labeling yield of the injected MAAs was controlled by Tech-Kit (under 0.2% of free technetium).

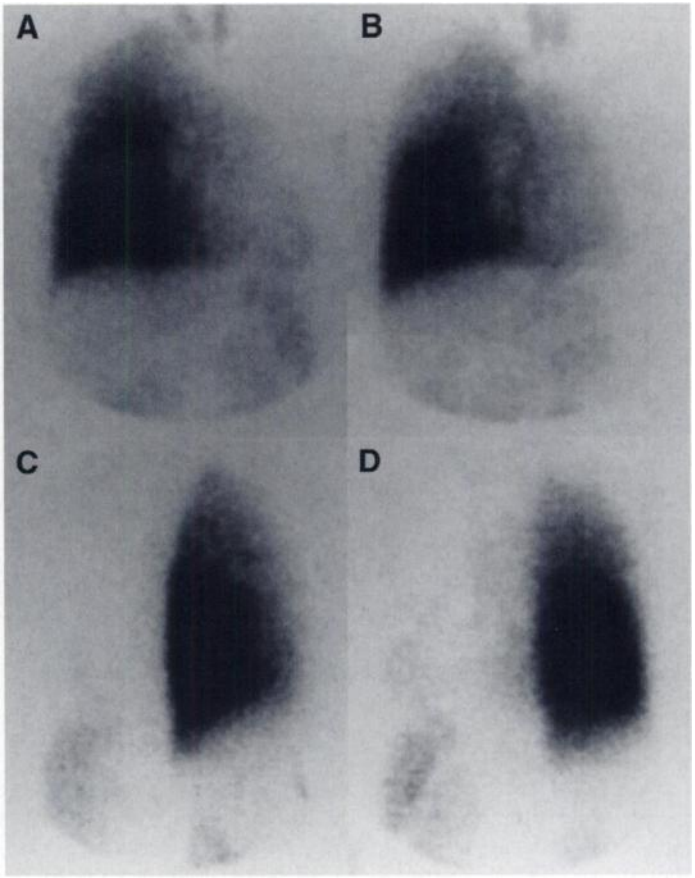

FIGURE 2. MAA perfusion lung scan (500,000 counts): (A) anterior; (B) right anterior oblique; (C) right posterior oblique; (D) posterior. The remaining right lung appears homogeneous. MAA is visible in spleen, myocardium and kidneys.

We concluded that there was no recent pulmonary embolism but that there was a right-to-left shunting because of the presence of the MAAs in the systemic circulation.

The transthoracic cardiac ultrasonographic examination only showed a right ventricle hypertrophy, whereas the transesophageal ultrasonography demonstrated a large atrial septal defect (ASD) with a bidirectional shunt (mostly RLS). The microbubbles injected intravenously were immediately seen in the left heart chambers. The Doppler ultrasonography failed to measure the intracardiac pressures by the tricuspid regurgitation method. Because of the poor prognosis of this patient, there were no other investigations, such as catheterization, nor was there treatment for this ASD.

We kept the assumption of an ancient ASD with a nonsymp-

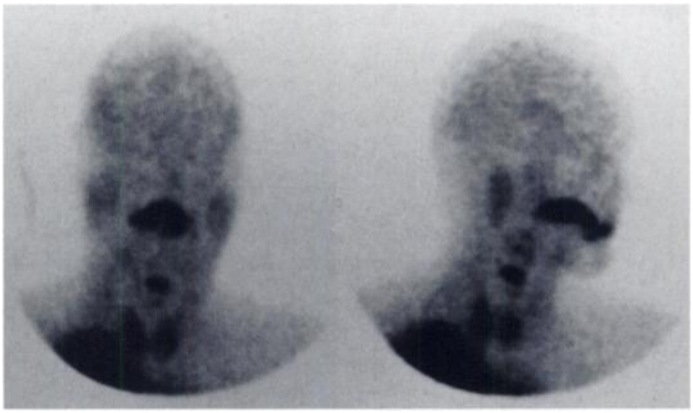

FIGURE 3. Image of the skull after MAA injection (300 sec): (left) anterior; (right) right. MAA is visible in brain, thyroid and salivary glands. The aerosol is seen in the airway.

TABLE 1
RLS Etiology

Pulmonary shunts	Cardiac shunts
Congenital: Pulmonary arteriovenous fistulae (*5,10–14*) (may be part of a Weber–Rendu–Osler syndrome)	Patent foramen ovale (*2,7,16–18*)
	True ASD (*19,20*)
	Ostium secundum (the most frequent)
Secondary to: (*6,8–11,13,15*)	Ostium primum
Pulmonary hypertension	Sinus venosus
Iterative pulmonary embolisms	Single atrium
Chronic obstructive pulmonary disease (COPD)	Abnormal venous drainage into the right atrium (*8*)
Cavopulmonary shunts	
Pulmonary metastasis	
Actinomycosis	
Schistosomiasis	
Sarcoidosis	
Liver cirrhosis	
Primary biliary cirrhosis	
Kimura's syndrome (angiolymphoid hyperplasia)	

tomatic left-to-right shunt. The pneumonectomy caused the inversion of the shunt, thus leading to a respiratory decompensation.

DISCUSSION

Right-to-left shunting may occur in either the lungs or the heart. The different causes are listed in Table 1 (*5–20*). A specific scintigraphic feature of the pulmonary shunt is an early uptake, quickly followed by a local defect with washout in the surrounding lung field (*5,12*). The main cause of cardiac shunting is patent foramen ovale, which is common (incidence of 10%–35%) (*2,7,16–18*). A foramen ovale may be opened by the postpneumonectomy intrathoracic distortion (*2,16*). However, because the pressure is higher in the left chambers, the shunt is usually left-to-right. Such a shunt is nonsymptomatic so long as it is moderate, but it may be reversed by pulmonary hypertension, right atrium hypertension (*17,21*) (a pneumonectomy may decrease right atrium compliance and cause high pressure in the right atrium) (*7*) or transient respiratory right atrial hypertension (*18*). Nonetheless, there are RLSs with no interatrial pressure gradient, mostly after a right (*2,7,22–25*) or left (*2,26,27*) pneumonectomy or lobectomy (*2,28*) or after a thoracotomy undergone for coronary surgery (*29*) or a thorax trauma (*30*) or even without overt pulmonary disease (*17,18,22*). Possible explanations are:

1. Despite a mean pressure lower in the right atrium, the instantaneous pressure may be transiently higher than in the left atrium during brief intervals of the cardiac cycle, leading to bidirectional shunt and hypoxemia (*18,31*).
2. The mediastinal distortion after a pneumonectomy may redirect the blood flow from the inferior vena cava toward an ASD or patent foramen ovale (*2,17,18,32*). Indeed, some RLSs are better demonstrated by contrast echocardiography (or scintigraphy) when the injection is performed into femoral vein rather than cephalic vein (*4,7,18*). An enlarged eustachian valve may induce this streaming (*17,18,22*).
3. Increased right ventricular compliance (*17,18,32*).

Clinically, such RLSs, either cardiac or pulmonary, may be associated with platypnea-orthodeoxia; this syndrome, quoted by Seward et al. (*22*), was first described in 1949 by Burchell. Platypnea is an upright dyspnea that is relieved in a recumbent position. Orthodeoxia is the same phenomenon for arterial hypoxemia, i.e., a fall in the arterial oxygen pressure when the patient stands up (*13,22*). Conversely, chronic obstructive pulmonary disease gives a dyspnea that is impaired in recumbent position (*13*). This syndrome is often undetected because clinical examination and arterial sampling are usually performed on a lying patient and thus are mildly disturbed, whereas the patient might feel marked symptoms (*13,22,28*). Platypnea-orthodeoxia may be caused by chronic obstructive pulmonary disease, severe pulmonary embolism, pulmonary arteriovenous fistulae, thoracotomy and intracardiac RLSs, usually after pneumonectomy (*16,21,22,24*) and as a late consequence of it (*25*). Contrast echocardiography has proven that the postpneumonectomy RLS can be induced or increased by erect position (*14,22*). The right heart pressures measured by catheterization are usually normal (*22*); the reasons for this surprising fact have already been mentioned. Three phenomena may explain why erect position enhances the reversed shunt: the decreasing venous return, worsened by fluid depletion (*2,22,24*); a mediastinal postpneumonectomy distortion, which may open a foramen ovale or redirect the streaming of blood from inferior vena cava to an ASD; and an increasing flow through a basal arteriovenous fistula due to gravity (*13,29*).

Usually, ASD is well tolerated. When the defect remains open, 14% of patients develop pulmonary hypertension with secondary reversal of the shunt (*19*) and may develop transcardiac embolism (*17,22*). Nonetheless, surgical closure is recommended because patients with unrepaired ASD are generally thought to have a shorter life expectancy (*20*). Conversely, the prognosis of postpneumonectomy RLS is spontaneously poor (*21*), so in this situation, closure is needed, either surgically or percutaneously. It provides good results, with fair improvement of PaO_2 and symptomatic relief (*7,16–19,21–23,27,28*).

In an onset of dyspnea occurring after a pneumonectomy, a physical and radiological examination should first check for a parenchymal or pleural disease of the remaining lung (*16,21*). Pulmonary function tests and bronchofibroscopy may show a late respiratory failure (either restrictive or obstructive). The arterial blood gas may show hypoxemia and hypocapnia, due to either a ventilation/perfusion mismatch (such a hypoxemia is improved by 100% oxygen breathing) or a true shunt (*2,10,21*). Perfusion lung scintigraphy is commonly performed to rule out a pulmonary embolism, but it is also quite efficient in the diagnosis of RLS. Cardiac shunts can also be assessed by left and right heart catheterization with pressure measurement (*6,16*) and angiogram. Contrast ultrasonography with intravenous injection of microbubbles (agitated saline) is a sensitive test for RLS; the appearance of the contrast into the left chambers is evidence for intracardiac shunt, if early, and extracardiac shunt, if delayed (*1,10,12,14,22*). Moreover, this technique can image intrathoracic malformations (*14*). Better results are obtained when using transesophageal echography rather than the classical transthoracic echography, all the more because intrathoracic anatomy has been modified by pneumonectomy (*14–16,25*). Magnetic resonance imaging was recently suggested for assessing intracardiac shunts (*25*), and it can also image intrathoracic malformations.

A good technique is required for lung scintigraphy, especially when looking for a shunt; the size of MAA particles should range from 10 μm to 50 μm (some authors advise checking under a microscope) (*1,3,4*). Their total weight should not exceed 0.2 mg, to avoid significant systemic infarction. This amount has been proven safe (*3,4*). The recommended injected activity is 185 MBq for adults (*5*). The labeling yield should be checked: free pertechnetate should not be excessive (under 2%) (*4,6*) lest it could be mistaken with MAA systemic uptake.

Besides usual planar images of the lungs, the quantification of a RLS requires the scanning of the kidneys and brain too (5-min acquisition static frames) or, better, a whole-body scan. All the frames should be acquired shortly after injection to avoid the releasing of fragmented MAAs from the pulmonary capillaries network (*1*), which may otherwise cause an overestimation of the shunt.

The tracer's uptake into kidneys, spleen, thyroid and brain is consistent with RLS (*1,2,4–9*), whereas gastric, bladder or salivary uptake can be the result of a poor labeling (*1,4,6*). To our knowledge, myocardial uptake has not been described previously, probably because RLS usually occurs after right pneumonectomy. A typical RLS feature, extrapulmonary mottled uptake (not found in our case), has been described as providing better sensitivity and specificity than mere quantification (*1*). As about 100,000 particles are injected, there should be about 10,000 scattered extrapulmonary particles for a 10% RLS; the random distribution into spots would be compatible with this mottled uptake.

Three formulas may quantify the RLS.

Using a whole-body scan (*1,3,4*):

$$\%\ \text{Shunt} = 100 \times \frac{\text{Whole-body uptake} - \text{Pulmonary uptake}}{\text{Whole-body uptake}}.$$

Using a kidney frame and assuming the renal blood flow is about 25% of the cardiac blood flow (*6*):

$$\%\ \text{Shunt} = 100 \times \frac{4 \times \text{Renal uptake}}{4 \times \text{Renal uptake} + \text{Pulmonary uptake}}.$$

Using the normal brain-to-lung activity ratio, the normal value of which is 0.43% ± 0.30% (s.d.) in children (*15*):

$$\text{Shunt index} = 100 \times \frac{\text{Cerebral uptake}}{\text{Pulmonary uptake}}.$$

If a ventilation scan is performed before the perfusion scan, this might affect the results of the quantification. Hence, if the main point is a shunt assessment, only a perfusion scan should be done.

CONCLUSION

Because the general incidence of patent foramen ovale is about 20%, right-to-left shunting is probably underestimated and should be checked for after a pneumonectomy, especially when the symptoms felt by the patient are not explained by physical examination and arterial blood gas. When this syndrome is suspected, the examination and arterial blood sampling should be repeated while the patient is upright or at least seated because the shunt is frequently increased in this position. Lung scan may yield useful information, provided a good technique is used. Probably the injection of MAA in the upright position would increase the sensitivity of the test in this indication, as for echography, but this has yet to be to proven.

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