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CASE REPORT

Pulmonary fat embolism presenting as chronic respiratory failure

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Pulmonary fat embolism presenting as chronic respiratory failure. D. Galdermans, D. Coolen, I. Neetens, J. Bultinck, G. Parizel.
ABSTRACT: A patient is presented with progressive respiratory failure, caused by pulmonary fat embolism as proved by an open lung biopsy. Four and a half yrs earlier, she underwent a right hip operation. We surmise that a loosening hip prosthesis caused the marrow embolisation. *Eur Respir J., 1989, 2, 185–187.*

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Case Report

A 76 yr old white woman is presented with a five month history of gradual onset of dry cough and dyspnoea. Four and a half yrs earlier, she underwent a right hip replacement surgery for severe coxarthrosis.

Previous history included stripping of the saphenous veins and a cholecystectomy. Twelve yrs ago, a pancytopenia of unknown cause was found and treated with stanozolol (Strombaject®) and mesterolon (Pro-viron®) with a good result until now. The patient was a non-smoker.

There was no history of fever, angina pectoris, palpitations, haemoptysis or ankle oedema. The patient denied earlier episodes of recurrent bronchitis and there was no significant history of occupational exposure. On admission, the patient complained of severe dyspnoea at rest. Physical examination revealed her to be a cyanotic, elderly woman in severe respiratory distress. Wheezes and dry crackles in early inspiration were present bilaterally. A chest X-ray showed an interstitial pattern in the lower lobes. The dyspnoea and cough had not improved following treatment, started in another hospital, for left ventricular failure with furosemide, captopril and digoxin.

In addition, echocardiography and a radionuclide ventriculogram showed a normal left ventricular function. The sedimentation rate was 47 mm. Cytology and culture of sputum and bronchoscopy with transbronchial biopsy were not conclusive. Pulmonary function tests showed a severe restrictive pattern and hypoxaemia at rest, arterial oxygen tension (Pao_2) - 45 mmHg or 6 kPa (table 1).

The patient was treated with methylprednisolone (32 mg per day) and oxygen. Since only minor improvement occurred after several weeks of treatment, an open-chest lung biopsy of the left lower lobe was performed. The colour of the lung was normal, but it showed a solid shape.

Pathological findings

After fixation in Bouin's fluid (750 ml picric acid saturated aqueous solution, 250 ml 37–40% formaldehyde, 50 ml glacial acetic acid), routine paraffin sections were made and stained with haematoxylin, eosin and safrane.

In multiple pulmonary artery branches, bone marrow emboli were found (fig. 1). The emboli did not consistently fill the lumen of the artery. They consisted of fat cells and scattered precursors of the red and white cell lines, plasmocytes, polymorphonuclear leucocytes and a few megakaryocytes. The bronchoalveolar supporting stroma was oedematous and showed many, thin-walled, dilated vessels of various diameters which are considered as arteriovenous shunts. There was no infarction of surrounding lung tissue. Focally there was slight, non-specific, interstitial, chronic inflammation and intra-alveolar and intra-lobular emphysema. Some alveoli showed desquamation of alveolar cells.

We tried to find a possible cause for these pulmonary fat emboli. A lung perfusion ^{99m}Tc macroaggregated albumin (MAA)-ventilation (^{81m}Kr) scintigraphy showed a normal ventilation distribution but perfusion defects in the apical segments of the left and right lower lobes. Multiple pathological "hot spots" were found on the bone ^{99m}Tc maximum permissible dose (MPD) scintigraphy. A bone marrow biopsy of two "hot spots" was normal and the X-rays revealed only severe arthrosis. The X-ray

Table 1. – Pulmonary function tests

Parameter	Units	Normal Value	Initial Value	After 1* months treatment	After 2* months treatment
Pao_2	mmHg	80–100	45	49	79
	kPa	10.7–13.3	6	6.5	10.5
$Paco_2$	mmHg	40	37	34	35
	kPa	5.3	4.9	4.5	4.6
Sao_2	%	90–100	82	85	96
TLC	*l*	5	2.7	2.9	2.8
RV	*l*	2	1.2	1.4	0.9
VC	*l*	2.8	1.6	1.5	1.9
FEV_1	*l*	2.1	–	1.1	1.5
DLCO	$ml \cdot min^{-1} \cdot mmHg^{-1}$	16.5	–	4.8	8.2

*: treatment (oxygen and bed rest); Sao_2: arterial oxygen saturation; TLC: total lung capacity; RV: residual volume; VC: vital capacity; FEV_1: forced expiratory volume in one second; DLCO: diffusing capacity of the lung for carbon monoxide.

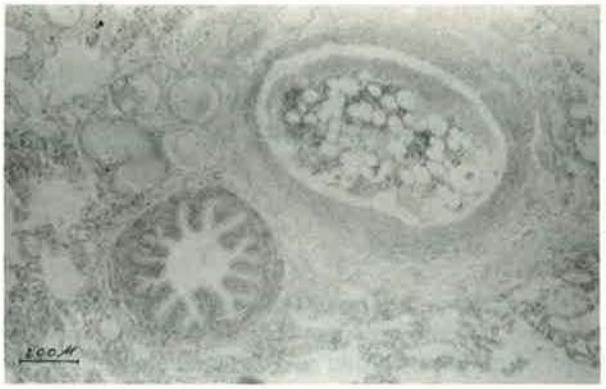

Fig. 1. – Specimen from open lung biopsy.

of the right hip showed a loosening Moore prothesis, surrounded by severe osteoporosis.

Following hospital treatment for three months with rest, oxygen and corticosteroids, the pulmonary condition improved. The arterial oxygen tension became normal at rest but there was still dyspnoea on exertion. The pulmonary function pattern remained restrictive with a diffusion capacity of 50% of the predicted value (single-breath method table 1).

Discussion

Classically, the pulmonary fat embolism syndrome is a symptom complex of acute respiratory failure occurring 12–30 h after severe long-bone fractures [1]. The signs are hypoxaemia, anaemia, thrombocytopenia, lipuria, tachypnoea, petechiae on the upper thorax and arms, fever and usually altered consciousness [2, 3]. It is thought to be caused by deposition of embolic fat within the pulmonary capillaries resulting in capillary leakage. The source appears to be bone marrow fat.

Recently, this syndrome was also described after bone surgery [4, 5], bone marrow transplantation, severe skeletal metastasis with hypercalcaemia and bone necrosis after decompression sickness. Deposition of lipid globules can also be found after hysterography, after treatment of premature babies with intravenous intralipid [6], and after trauma of a fatty liver. Spontaneous resorption of the aggregates of fat usually occurs after several weeks. Oxygen therapy is usually sufficient, and the role of corticosteroids remains controversial.

Our patient developed a progressive respiratory failure over several months, with a chest X-ray suggestive of interstitial lung disease. An open-lung biopsy revealed the pulmonary fat embolism (fig. 1).

The lung perfusion-ventilation scintigraphy confirms the perfusion defects in the lower lobes and this also explains the restrictive lung pattern. A right-to-left intrapulmonary shunt, secondary to the arterial pulmonary fat embolism, probably explains the dilated vessels and oedematous, supporting stroma. On the other hand, we would expect a more extensive pulmonary response with granulomatous reaction and fibrosis, after the chronic release of these emboli.

Fat emboli have already been described after surgery of the long-bone or insertion of hip prostheses, in association with polymethyl methacrylate bone cement [5]. We surmise that during exercise, modifications of the pressure forces of the right hip prosthesis on the surrounding osteoporotic bone cause marrow embolisation. This could also explain the favourable effect of rest on respiratory function.

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Observation clinique. Embolie graisseuse pulmonaire se présentant comme une insuffisance respiratoire chronique. D. Galdermans, D. Coolen, I. Neetens, J. Bultinck, G. Parizel.
RÉSUMÉ: Observation d'un cas de décompensation respiratoire progressive due à une embolie graisseuse pulmonaire prouvée par une biopsie pulmonaire à ciel ouvert. La patiente avait subi une intervention sur la hanche droite quatre ans et demi plus tôt. Nous supposons que cette prothèse de hanche mal assujettie fut la cause de l'embolisation de moelle.
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