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Letters to the Editor

Fatal Donor-Acquired Fat Embolism Syndrome Leading to Multiple Organ Failure in a Lung Transplant Recipient

Síndrome mortal de embolia grasa, adquirido a través del donante, que conduce a insuficiencia multiorgánica en un receptor de trasplante de pulmón

To the Editor:

Potential lung donors who have suffered major trauma are at risk for fat embolism syndrome (FES). Donor-acquired FES is manifested in the recipient after organ transplantation and is a potential cause of acute graft failure.[1] Pulmonary emboli diagnosed by lung biopsy after transplantation have been described in 3 case reports.[2-4] We report a case of fatal donor-acquired FES causing primary organ failure after a unilateral lung transplant.

A 41-year-old man with history of silicosis, being treated with oral methylprednisolone and domiciliary oxygen therapy, underwent unilateral lung transplantation. The donor was a 17-year-old man, a smoker, who was brain dead after sustaining a head injury in a traffic accident; he also had a fractured right femur. The chest radiograph following transplantation showed widespread, confluent alveolar markings due to reperfusion edema (ratio of PaO_2 to the fraction of inspired oxygen [FiO_2], 165). The patient was extubated at postoperative day 4, but 12 hours later his condition deteriorated, with progressive hemodynamic instability, respiratory failure requiring mechanical ventilation, a fall in the hematocrit, and renal insufficiency which was treated with dopamine and continuous venovenous hemodiafiltration. Radiographic worsening of the graft (a confluent alveolar pattern) was observed. A transbronchial lung biopsy was performed. Microscopy revealed bone marrow emboli, extensive thrombosis in capillaries and larger-caliber vessels, together with diffuse alveolar damage in exudative-organizing phases. The patient developed acute respiratory distress syndrome and multiorgan failure that led to death 15 days after lung transplantation. Postmortem examination showed that vascular and bronchial sutures had not failed. There was severe diffuse alveolar damage in exudative-organizing phases, and evidence of fat embolism in lung and glomerular capillaries (Figure).

FES usually occurs as a complication of severe trauma. The presence of fat emboli in the pulmonary parenchyma and peripheral circulation does not imply the development of acute FES. FES is manifested by progressive respiratory insufficiency, thrombocytopenia, petechial rash, and impaired level of consciousness.[5] Fat emboli may be detected in more than 90% of patients with long-bone fractures; FES is less common, however, with an incidence between 0.5% and 2.2% in patients with isolated long-bone fractures and between 5% and 10% in patients with multiple long-bone fractures and concomitant pelvic fracture.[6] Recently, Oto et al[1] found that unsuspected pulmonary embolism was relatively common, with an incidence of 38% in a series of 74 lung donors. They also reported that it was significantly related to primary lung graft failure and 1-year survival. However, fat embolism was present in only 9% of the 74 lung donors. In their study, death due to head trauma associated with long bone fracture and a smoking history of more than 20 pack-years were significant donor risk factors for pulmonary embolism.

Both long-bone fracture and a history of smoking were present in the donor in our case. Hypoxia, which occurs in up to 96% of patients with FES,[6] is not present in lung donors, given that a PaO_2/FiO_2 less than 300 is a contraindication for lung transplantation. During early recovery after a lung transplant, recipient hypoxia may be related to reperfusion ischemia, infection, acute cellular rejection, or myocardial dysfunction. All these conditions, as well as suture leak and pulmonary vein thrombosis, were ruled out in our patient by transesophageal echocardiography, biopsy, and microbiology. Ischemic reperfusion injury in the form of diffuse alveolar damage in the exudative-organizing phases was present from the first postoperative days and persisted to the third week; however, at postmortem examination this was not considered to be the cause of graft dysfunction. Sedation and analgesia required for adaptation to mechanical ventilation prevented adequate neurological assessment of our patient. Acute FES was not present before transplantation given that no evidence of fat embolism was found by histopathology of the donor's rejected lung or the recipient's explanted lung.

Treatment of FES includes hemodynamic and respiratory support. Corticosteroids, which the recipient receives from the first postoperative day as part of the immunosuppression regimen, have been used in the treatment of the FES but have not shown a

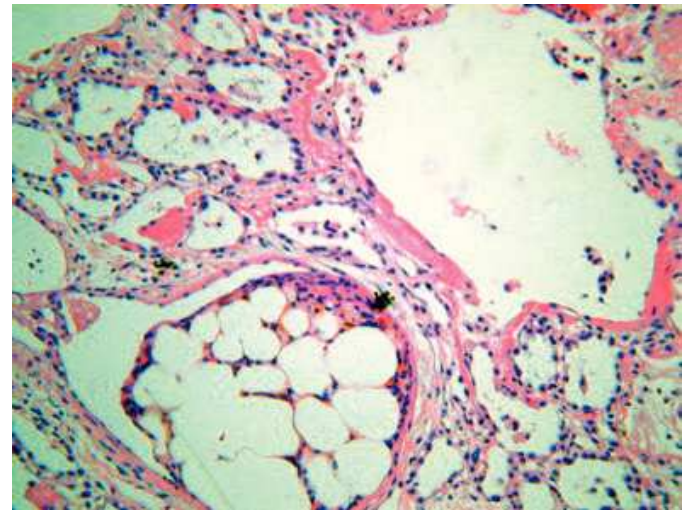

Figure 1. Open lung biopsy showing a pulmonary artery laden with fat and bone marrow cells (hematoxylin & eosin, original magnification × 100).

clear beneficial effect.[5] In fact, corticosteroids seem to have a paradoxical effect, as they have been considered to play a role in the genesis of FES in patients under chronic treatment with these agents.[5]

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Bilateral Pneumothorax after Pleural Drainage

Neumotórax bilateral tras la colocación de un drenaje pleural

To the Editor:

Bilateral primary spontaneous pneumothorax is a rare disease (<1%)[1] that requires urgent diagnosis and treatment. We report a case of presentation that occurred after draining a pneumothorax in the contralateral hemithorax.

A 30-year-old man with a 3-month history of right unilateral pneumothorax presented with dyspnea and pain in the left-hemithorax lasting 3 hours. Physical examination revealed tachypnea, oxygen saturation of 93%, and stable vital signs. A chest x-ray showed a left pneumothorax with contralateral mediastinal shift. During insertion of an 18-F chest tube, the patient complained of moderate pleuritic chest pain; a follow-up chest x-ray was made (Figure) and the right hemithorax was drained with no complications. The new radiologic test showed reexpansion of both lungs. A computed tomography scan (CT) revealed bilateral apical paramediastinal bullae, mainly on the left side. On completion of the preoperative evaluation, bullectomy and mechanical pleurodesis of both lungs were performed in a 2-stage operation. Postoperative recovery was satisfactory and the patient was discharged a week later.

The incidence of bilateral spontaneous pneumothorax is low and is more common in patients with an etiologic trigger (human immunodeficiency virus infection, tuberculosis, sarcoidosis, etc).[1] Although patients with bilateral spontaneous pneumothorax have a lower body mass index than those with unilateral pneumothorax, there are no differences regarding age, sex, or smoking. However, patients with bilateral pneumothorax do present a greater incidence of bullae, which are considered an independent risk factor in the development of bilateral pneumothorax.[1,2]

We emphasize the importance of this case because the contralateral pneumothorax was only detected in the chest x-ray made after the chest tube was inserted; there was no evidence of air accumulation in the first x-ray study. Although the first imaging test revealed no pneumothorax on the contralateral side, the follow-up chest x-ray made after the chest tube was inserted led to its detection. This prompts us to raise the following points:

The contralateral pneumothorax may have been a complication of the reexpansion of the pre-existing pneumothorax. To date, pulmonary edema after abrupt reexpansion is the only known complication of spontaneous pneumothorax. However, if a patient presents with an underlying disease such as bullae, air aspirated through a chest tube from the pleural cavity of one of the hemithoraces would exert sufficient traction on the contralateral pleura and the bullae to trigger a new pneumothorax.

The images might also be due to the effects of pressure; that is, a simultaneous bilateral pneumothorax might have occurred in our patient but gone undetected in the first imaging test because more pressure was exerted on the left side. This would have led to the compression of the right pneumothorax, which would only have become apparent when the former resolved.

Finally, a pleural window may have communicated with both pleural spaces.[3] The window might have been embryonic in origin (due to a defect in the fusion of the pleuropericardial folds in the fifth week of development), or a complication of cardiac and thoracic surgery. In patients with unilateral pneumothorax, this window permits the passage of air from one side to the next, causing bilateral collapse. Our patient had bilateral bullae, however, and pleural windows are typically found in patients with unilateral disease. Although the pleural defects were predominantly left-sided in our

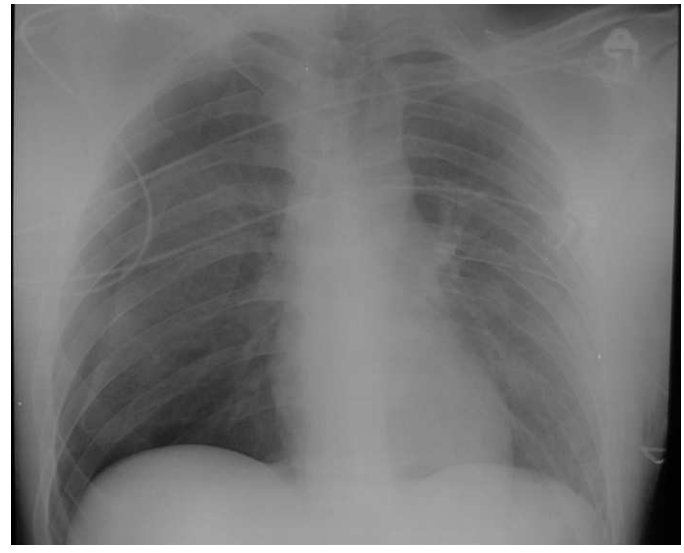

Figure. Posteroanterior chest x-ray: chest tube in the left hemithorax with complete reexpansion of the left lung and appearance of a new right pneumothorax over 3 cm long.