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Treatment of Severe Subcutaneous Emphysema by Microdrainage. A Case Report☆

Tratamiento de enfisema subcutáneo severo por microdrenaje. A propósito de un caso

To the Editor:

Subcutaneous emphysema (SE) is defined as tumefaction produced by the presence of air or gas in the skin.[1] It is a primarily aesthetic problem which does not usually cause complications, unless it is very extensive. We present a case of severe SE due to iatrogenic pneumothorax treated with a fenestrated angiocatheter, according to the method described by Beck,[2] used for the first time in our centre.

A 77-year-old male, with a history of GOLD stage III COPD, was admitted for percutaneous pulmonary biopsy for the study of a lung nodule suggestive of bronchogenic carcinoma. After the procedure, pneumothorax was detected, requiring the placement of a drainage tube which was only maintained for 24 h before it was accidentally removed. The patient subsequently developed extensive SE (face, neck, upper and lower limbs) and reappearance of the pneumothorax. A new drainage tube with aspiration was placed, resolving the pneumothorax, but not the SE. The decision was taken to withdraw the chest drainage and continue with conservative treatment. However, the SE progressed, causing dyspnoea and difficulty seeing and swallowing, so treatment was initiated with a fenestrated angiocatheter, according to the technique described by Beck,[2] consisting of several fenestrations made in a spiral pattern along a 14G-calibre angiocatheter. Two fenestrated catheters were inserted in the subcutaneous space, 2 cm lateral the midclavicular line at the level of the third rib (Fig. 1), achieving immediate improvement after placement. It was connected to continuous suction and the nursing staff and family members were instructed in performing massage manoeuvres from the distal to the medial regions to facilitate aspiration. In less than 48 h, the patient could open his eyes and his dyspnoea had resolved. He was discharged after resolution of symptoms.

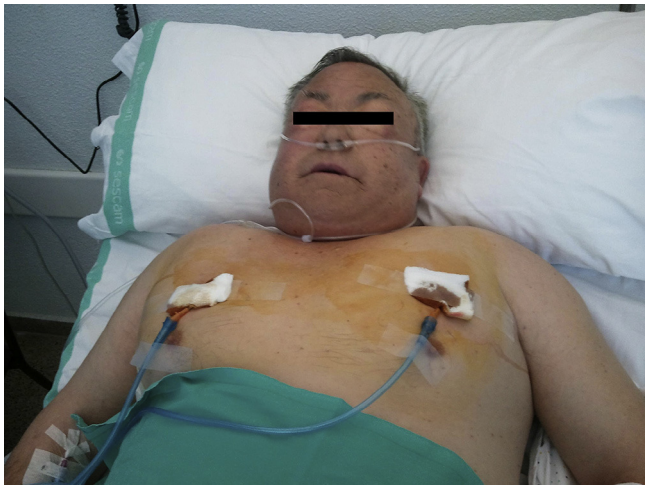

Fig. 1. Severe subcutaneous emphysema. Fenestrated catheters placed according to the technique described by Beck, connected to continuous aspiration.

Studies on the mechanism of pulmonary interstitial emphysema, pneumomediastinum and SE have shown that air begins to migrate from a rupture in the alveoli to the pulmonary interstitial tissue and then on to the perivascular space until it reaches the mediastinum.[2,3] When the passage of air is greater than the pleural resorption, SE occurs. This may also develop in the case of SE due to iatrogenic pneumothorax. Serious complications have been described, such as pacemaker malfunction, compromised airway, intracranial hypertension or respiratory failure, but these are uncommon. Conservative management is generally sufficient for the resolution of SE,[1] although several therapies have been proposed, such as making holes in the skin, placement of chest tubes, pig-tail drainage or trocar drainage, all of which have limited use and are associated with risks of infection, scarring and patient discomfort.[1,2] Fenestrated catheter placement is easy, fast and simple and is minimally invasive, since the risks of infection and skin scarring are reduced, although its use for periods longer than 72 h is not recommended.[4] This technique for the management of SE must be used on an individualised basis, depending on the clinical context and severity of the symptoms, although its ease of use, high effectiveness, low cost, minimal invasiveness and low risk of complications make it a good option in selected cases.

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A Case of Interstitial Lung Disease With Apical Pleural Thickening: Idiopathic Pleuroparenchymal Fibroelastosis☆,☆☆

Un caso de neumopatía intersticial con engrosamiento pleural apical: fibroelastosis pleuroparequimatosa idiopática

To the Editor,

Idiopathic pleuroparenchymal fibroelastosis (IPPFE) is a very rare, recently described condition, characterised by fibrotic thickening of the pleural and subpleural parenchyma, predominantly in the upper lobes.[1] Clinical manifestations and lung function tests are similar to those observed in restrictive interstitial pneumonias, and in some of the cases described, there was a history of recurrent infections, such as allergic bronchopulmonary aspergillosis or cystic fibrosis.[2,3] Radiological findings include intense pleural thickening associated with signs of fibrosis, particularly in the upper lobes, with loss of volume and structural distortion, as observed in the case presented here.

This is an 82-year-old female patient with suspected usual interstitial pneumonia on CT imaging, who consulted her pulmonologist due to worsening of dyspnoea on effort and non-productive cough. The patient stated that she was a non-smoker and had not had any previous exposure to environmental allergens, radiotherapy, drugs or history of contact with asbestos. On the physical examination, the patient had a healthy appearance, with normal vital signs and 95% PaO_2 (with room air). Chest examination showed reduced breath sounds and bilateral rales in the lower lobes. Heart examination showed regular frequency and rhythm, and the extremities were normal without finger clubbing. Lung function tests showed moderate restrictive ventilatory defects (FVC: 57%, FEV1: 72%). The 6-min walking test gave a distance walked of 314 m (74%), no desaturation and 3–3 on the Borg scale.

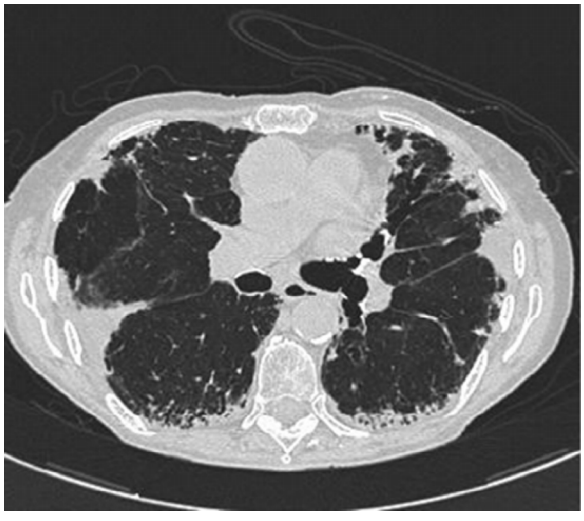

Figure 1. Chest CT scan revealed bilateral irregular pleuroparenchymal thickening, principally in the upper and middle areas, associated with fibrotic signs.

Chest X-rays showed intense apical pleural thickening and upper hilar retraction. The chest CT revealed bilateral irregular pleuroparenchymal thickening, principally in the upper and middle areas, associated with fibrotic signs (Fig. 1). Serology tests were negative for anti-scl-70, anti-Jo-1 and anti-DNA antinuclear antibodies, antineutrophil cytoplasmic antibodies, rapid plasma reagin and rheumatoid factor. A videobronchoscopy with bronchoalveolar lavage was carried out and transbronchial biopsies were obtained, giving negative cytology and microbiology results. Histopathology examination of the lung biopsy showed intraalveolar fibrosis without granulomas. The complementary imaging studies and examinations were compatible with IPPFE and the patient was initially treated with low-dose oral azathioprine and corticosteroids. After 24 h, the patient showed disease progression with no response to treatment, requiring home oxygen therapy. A recent chest CT showed loss of volume and progression of pleural thickening, along with signs of fibrosis in the lower lobes. Follow-up lung function tests showed FVC 58% and FEV1 67%.

IPPFE is a very rare disease entity. Diagnosis is based on clinical, radiological and histopathological examinations. Histopathological findings include marked thickening of the visceral pleura and prominent subpleural fibrosis, with elastosis of alveolar walls. Reddy et al. describe the “definite” characteristic of IPPFE as pleural fibrosis in the upper lobes, associated with intraalveolar fibrosis accompanied by alveolar elastosis. They considered the presence of intraalveolar fibrosis as “consistent” with IPPFE but when (a) not associated with pleural fibrosis; (b) not located predominantly below the pleura; or (c) not located in the upper lobes.[4,5] The treatment of IPPFE has not been determined. Kusagaya et al. described 5 cases of Japanese patients who did not receive any type of treatment with a mean follow-up of 45.2 months (7–83 months), all of who remained alive, but with clinical and functional deficit.[3] In another series of 12 European patients, 9 were treated with low-dose corticosteroids, immunosuppressive drugs or N-acetylcysteine. Five (5) of these patients died within a period of 4–24 months after diagnosis.[4]

In conclusion, IPPFE is a very rare entity and our case is the first description of the disease in Latin America. Identification of this disorder is very important for defining its prognosis and promoting the development of alternative treatments.

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