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Letters to the Editor

Spontaneous Pneumomediastinum and Pneumopericardium with Respiratory Failure $\stackrel{\scriptscriptstyle \leftrightarrow}{\sim}$

Neumomediastino espontáneo y neumopericardio con insuficiencia respiratoria

Dear Editor:

Spontaneous pneumomediastinum (PM) is an uncommon alteration defined by the presence of free air in the mediastinum that is not preceded by trauma, surgery or other medical interventions.¹ Its presentation is mainly in young men, and chest pain is the most frequent symptom. Other symptoms and signs include dyspnea, cough, neck pain and subcutaneous emphysema.^{1–5} On occasion, chest radiograph is insufficient to detect gas in the mediastinal compartment and a CT scan is necessary.¹ In the absence of an associated subjacent disease, it is considered a process with little clinical impact and its recurrence is very infrequent.^{1–3} Nevertheless, we have treated a patient with spontaneous NM and important gas exchange affectation who presented clinical characteristics that could cause confusion in the differential diagnosis.

Our patient is a 24-year-old male who was admitted to the emergency department due to the sudden appearance of intense precordial pain radiating towards his back, with non-spinning vertigo and breathing difficulties due to the pain. When questioned, he only referred mild watery rhinitis during the previous four days, having received no treatment. He did not smoke, nor did he have any medical history of interest. Physical examination showed no alterations. Blood work revealed leukocytosis of 18.7×10^3 with 16.6×10^3 neutrophils/µl. C-reactive protein was 20.3 mg/l. Blood/arterial gas breathing room air showed pH 7.42, Pa02 54 mmHg, PaCO2 35 mmHg and HCO3 23 mmol/l. Electrocardiogram showed a sinus rhythm with 115 bpm and image of S1Q3. On chest radiograph, we observed a thin pneumopericardium line. In order to rule out pulmonary embolism, thoracic CT angiography was performed, showing no vascular defects and confirming the presence of air in the mediastinal space (Fig. 1) and pneumopericardium. The patient received analgesic treatment and oxygen. Twenty-four hours later, he was asymptomatic, with oxyhemoglobin saturation 98% (Fi02 0.21) and leukocytosis had normalized.

Spontaneous PM is considered a mild process that does not necessitate treatment.¹ However, the case that we have presented showed respiratory failure, leukocytosis and clinical data that required us to rule out other processes. Although the finding of





Fig. 1. Chest CT: presence of air in the mediastinal compartment.

leukocytosis and neutrophilia can be frequent (in the series by Macià et al.,⁴ 42% of the patients with spontaneous PM presented these), we have found no references to the alteration in the gas exchange in similar cases. Exceptionally, PM or tension pneumopericardium can be produced by the persistent entry of air in the mediastinal or pericardial spaces with increased pressure that can affect the venous return and the cardiac function, constituting a medical–surgical emergency¹ that would explain the hypoxemia. In our patient, nevertheless, there were no data for PM or tension pneumopericardium, therefore if they had existed, there would have been transitory alterations.

Our patient had no condition associated with the development of spontaneous PM. This is not uncommon as in more than half of the patients with spontaneous PM there is no identified predisposing condition, such as asthma, interstitial disease or graftversus-host disease.^{1.6} The precipitating factors that are associated with spontaneous PM are those that produce a sudden increase in intrathoracic pressure, mainly vomit, drug inhalation, intense cough and physical exercise. Nevertheless, in between 30 and 50% of patients, no precipitating factor associated with PM can be identified.^{1–5}

We conclude that spontaneous PM, considered a process of limited clinical transcendence, can present with characteristics of greater severity than what are usually reported, although in our case there was a rapid resolution, as is usual in this process.

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Mediastinitis secondary to acute pancreatitis *

Mediastinitis secundaria a una pancreatitis aguda

Dear Editor,

Acute pancreatitis is a serious pathology that can be associated with thoracic complications, fundamentally with pneumonia, pleural effusion and more rarely enzymatic mediastinitis. Of this latter situation, there are few cases reported in the literature. Due to the important morbidity and mortality of mediastinitis and its limited frequency, we present this clinical case.

A 56-year-old male patient, diagnosed with necrotic pancreatitis at his area hospital. After a poor evolution, and with the diagnostic suspicion of mediastinitis on computed tomography (CT), the patient was transferred to our hospital. Initially, as there was no presentation of mediastinal collections, it was decided to initiate conservative treatment with empirical antibiotic therapy. Two days later, the follow-up CT showed an increase in mediastinal fat, mediastinal collections with air content, pericardial and bilateral pleural effusion (Fig. 1). Given the clinical and radiological findings, it was decided to operate. We performed median sternotomy, extirpating the necrotic mediastinal fat, debriding and draining the existing mediastinal collections while draining the bilateral pleural and pericardial effusion. Culture of the drained material was positive for *Candida parapsilosis* and *Pseudomona aeruginosa*.

Postoperative evolution was torpid, with dehiscence and exudate with purulent appearance through the surgical wound, due to which it was decided to operate once again, with drain-lavage of the mediastinum, leaving an open sternotomy for local treatment.

After serial cultures of the surgical wound with no germs isolated and without complications for 2 months, we decided to close the sternotomy using the transposition of the major pectoral muscles. The patient recovered and was discharged 17 days after this last surgery.

Follow-up detected a pancreatic tumor 6 months later. During surgery, multiple hepatic, peritoneal and epiploic metastases were discovered. The patient died months later.

Within the thoracic complications of pancreatitis (15-50% of cases), the most frequent is pneumonia with pleural effusion. What is less common is pancreatic pseudocyst with mediastinal extension, thoracopancreatic fistula and, even more uncommon,

mediastinitis secondary to pancreatitis. Only 4 cases have been published of this latter complication.^{1,2}

The pathogenic mechanism of this type of mediastinitis seems to be the leakage of pancreatic secretions and their ascent through the esophageal or aortic hiatus to the mediastinum.¹ In the case of our patient, necrotic pancreatitis could have caused the erosion of the diaphragmatic parietal peritoneum and allowed the intrathoracic propagation of the pancreatic enzymes.

In a patient with pancreatitis with the appearance of dyspnea, thoracic pain, fever, cyanosis, tachycardia, acute heart failure or a syndrome of the upper vena cava, an intrathoracic complication should be suspected. In our case, the pancreatitis led to respiratory failure, requiring hospitalization in the intensive care unit, and the mediastinal and pleural affectation was observed on CT.

As for the treatment of this type of mediastinitis, all the previously published cases referred the need for surgery using drainage and debridement, as was carried out in the patient that we present. The torpid evolution required open sternotomy to allow for local cleansing and dressing for several weeks until the resolution of the symptoms.

After our experience and the review of the limited bibliography, we would like to conclude by recommending the inclusion of mediastinitis in the differential diagnosis when given suspicion of pancreatitis with the appearance of respiratory symptoms. Due to the high morbidity and mortality of this type of mediastinitis, we recommend aggressive treatment with antibiotic therapy and surgical debridement in addition to exhaustive clinical and



Fig. 1. CT image showing mediastinal widening, collections with air content, pericardial and bilateral pleural effusion and compression atelectasis.

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