The North American Children study found a mortality rate of 30%, with 50% of patients experiencing on-going morbidity. It has become clear that some ILD entities are associated with very high mortality, whereas others have a favorable outcome.

References

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Tuberculosis and Microscopic Poliangitis. A Rare Combination

Tuberculosis y poliangitis microscópica. Una asociación muy poco frecuente

To the Editor:

Tuberculosis (TB) is one of the most common causes worldwide of morbidity and mortality due to infection, with a recorded incidence in Spain in 2015 of 21.5 cases/100,000 inhabitants.1

Vasculitis is a general term for a heterogeneous group of diseases characterized by inflammation and destruction of blood vessel walls.2 Most cases are primary, but vasculitis can also be secondary to other diseases, including infections.3 At times the difference between TB and vasculitis can be difficult to determine, because they share similar characteristics, and moreover, both entities can coexist in the same patient.4 The definitions of vasculitis and its different forms are well established.5 We report the case of a patient that presented simultaneous TB and microscopic polyangitis (MPA).

This was a 68-year-old man with a history of TB, who attended the emergency room with a 1-month history of fever, bloody sputum, asthenia, weight loss, and dyspnea. His temperature was 38.4°C, with no other significant findings. Blood tests were normal, and chest X-ray revealed scarring in the upper right lobe and a solid, spiculated parenchymal lesion. Chest computed tomography (CT) showed no changes in the middle and lower fields (Fig. 1A). No upper respiratory tract involvement was observed. Polymerase chain reaction was positive for Mycobacterium tuberculosis in bronchial aspirate and bronchoalveolar lavage. Core needle biopsy of the spiculated lesion revealed necrotizing granulomatous inflammation with multinucleated Langhans giant cells (Fig. 1B), and positive Zielh–Neelsen staining and polymerase chain reaction for M. tuberculosis. The patient developed sudden onset hemoptysis with anaemia (hemoglobin<6.8 g/dL, hematocrit 20.8%), acute renal failure (urea 123 mg/dL, creatinine 8.3 mg/dL), oligoanuria and elevated transaminases (values 5 times the upper limit of normal). Intubation, mechanical ventilation, and hemodialysis were required. In view of the patient’s hepatic and renal insufficiency, antituberculosis treatment began with ethambutol, levofloxacin, and streptomycin. Chest CT showed diffusely increased pulmonary radiodensity, mainly ground glass opacities and areas of consolidation in the peribroncovascular region, with moderate left loculated pleural effusion, with a fissural component, that was interpreted as diffuse alveolar hemorrhage (Fig. 1C). Renal biopsy revealed vasculitis with fibrinoid necrosis of the small arteries associated with focal and segmental necrotizing glomerulonephritis with an absence of immunoglobulin, complement and light chain deposits, suggestive of MPA (Fig. 1D and E). Pleural fluid was a lymphocytic exudate; ADA 45 U/L, with no other significant changes. Anti-neutrophil cytoplasmic antibodies (ANCA) (dilution 1/320; p-ANCA pattern) with anti-myeloperoxidase antibodies>300 U/mL were detected. Anti-glomerular basement membrane antibodies were negative. Treatment was administered with corticosteroids (3 initial boluses of methylprednisolone 500 mg/day, tapered to 15 mg/day of prednisone), plasmapheresis (7 sessions), and rituximab (700 mg/week for 4 weeks). Rifampicin and isoniazid could subsequently be reintroduced. Progress was slow but favorable, with stabilization of respiratory symptoms and radiological improvement (Fig. 1F).

The association between TB and vasculitis has been described, but generally always in association with granulomatosis with polyangiitis.6,7 As far as we know, this is the second case in which TB has been associated with MPA.7 Both diagnoses appear to be confirmed: positive polymerase chain reaction in 2 different samples in the case of TB; and for MPA, granulomatous inflammation with necrosis and multinucleated giant cells in lung tissue

Fig. 1. (A) Chest high-resolution computed tomography (HRCT). No evidence of disease or pleural effusion observed in middle and lower fields. (B) Lung biopsy. Granulomatous inflammation with extensive areas of necrosis and multinucleated Langhans giant cells. (C) Chest HRCT. Diffuse increase of pulmonary radiodensity, mainly ground glass opacities with moderate left loculated pleural effusion, and a fissural component, interpreted as diffuse alveolar hemorrhage. (D) Renal biopsy. Renal glomerulus with area of focal necrosis and karyorrhexis. (E) Renal biopsy with fibrinoid necrosis of the small arteries affecting more than 50% of the circumference, associated with transmural inflammation. (F) Chest HRCT. Reduction of pleural effusion and slight improvement of the ground glass opacities.

with positive Ziehl–Neelsen staining, and confirmed diagnostic criteria with rapidly progressing diffuse alveolar hemorrhage and necrotizing glomerulonephritis.5

The question here is, is this association incidental or does one disease (TB) lead to another (MPA)? The inverse association is possible, and the administration of an immunosuppressive treatment for a diagnosis of vasculitis as a trigger for TB has been documented,8 but this could be ruled out in our patient by the order

of events. Flores-Suárez et al. observed high odds ratios for positive p-ANCA by indirect immunofluorescence in TB patients, compared with asthma patients (odds ratio 2.96 [1.19–7.38]; P<0.027) and healthy subjects (odds ratio 18 [3.88–83.4]; P<0.0001).9 It is difficult to establish an association between TB and MPA, although the coexistence of TB and positive ANCA may be a causative factor. Some drugs, especially isoniazid and rifampicin, can be transformed into active metabolites that develop cytotoxic prod-
Malignant Pleural Mesothelioma: The Last 8 Years of Experience in Our Area

Mesotelioma pleural maligno: experiencia de los últimos 8 años en nuestra área

To the Editor:

Malignant pleural mesothelioma (MPM) is a rare tumor with a poor prognosis. Treatment may include surgical resection, chemotherapy (CT), radiation therapy, or a combination of all 3. We report a descriptive retrospective study that evaluates clinical and pathological features, type of treatment, and survival in patients diagnosed with this disease in our region (Vallés Occidental, Barcelona) between 2008 and 2016.

There were 44 cases of MPM in total. Most patients were men (84%), and 56.8% were 70 years of age or older. Twenty-five patients had epithelioid type disease, 1 biphasic, 2 sarcomatoid, and 16 with no specific histology. Twenty-four (54.6%) were smokers. Nine (20.5%) had a history of asbestos exposure.

Most were in advanced stages at diagnosis, 35 (79.5%) in stages III and IV, and 9 (20.5%) in stages I and II. Twenty-three (52.3%) had performance status (PS) 0–1, and 21 (47.7%) had PS 2–3.

Surgery was indicated in 2 patients (4.5%): in 1 to limit symptoms and for disease diagnosis, and in the other due to very good response to systemic therapy and good PS in a young patient. The remaining patients were treated with talc pleurodesis. Twenty-one patients (47.7%) received first-line CT with cisplatin–pemetrexed, and 4 (9%) showed partial response or stable disease. Ten patients (47.6%) received a second line of treatment after progression, 5 (50%) with vinorelbine and 5 (50%) with gemcitabine.

Mean overall survival (OS), analyzed by the Kaplan–Meier method, was 14 months (95% CI: 11.6–16.4), with no differences between men and women (14 vs 16 months) (P>0.91). There were no significant differences in OS between patients with epithelioid tumors (15 months) and those with other histologies (14 months) (P>0.6). OS in terms of stages I/II/III/IV was 11, 15, 14 and 11 months, respectively (P>0.5). In patients treated with CT, OS was 15 months compared to 11 months in untreated patients (P>0.3).

MPM is a rare tumor that is difficult to diagnose and therapeutic options are limited. In up to 80% of cases, it is associated with occupational exposure to asbestos. 1 Symptoms include the development of pleural effusion, dyspnea or pain. 1 Computed tomography (CT) is the radiological diagnostic procedure of choice; it reveals diffuse pleural thickening and nodular

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