Letters to the Editor

Taxane-Induced Pneumonitis: Our Clinical Experience

Neumonitis por taxanos: nuestra experiencia clínica

Dear Editor,

Paclitaxel is a cytotoxic drug with anti-microtubule activity, widely used in the treatment of different cancers. It has a good safety profile and hematological and systemic toxicity is acceptable. Paclitaxel-associated pneumonitis was first described in 1995 by Goldberg and Vannice,1 and has been attributed to a delayed type IV hypersensitivity reaction. It occurs rarely with an incidence ranging from 1% to 4%, rising when paclitaxel is combined with other cytotoxic drugs or radiation therapy.2

Although it is uncommon, we would like to point out how significant this condition can be, since 4 cases have presented in our hospital in the last 6 months that are clinically compatible with this entity. These were 4 women with breast cancer treated with taxanes. The clinical manifestations began between 7 and 15 days after the last cycle of paclitaxel, characterized by a deterioration in general state of health, dyspnea progressing until it was present even at rest, and cough. In only one case was fever observed, of up to 39°C. Neither the clinical laboratory tests nor the chest X-ray performed on admission showed any significant changes. However, 24 h later, the clinical picture continued to worsen, rapidly deteriorating with corresponding laboratory and radiological tests. In all cases, samples were obtained for microbiology, and all were negative. Wide spectrum antibiotics were initiated with no clear improvement. For this reason, a computed tomography (CT) of the chest was performed, showing diffuse ground-glass pulmonary involvement with thickening of the interlobular septa in the anterior regions of the upper lobes and the middle lobe, all compatible with probable drug-induced pneumonitis. In view of the poor clinical status of the patients, fiberoptic bronchoscopy for obtaining a definitive diagnosis was ruled out and the decision was taken to initiate treatment with corticosteroids, leading rapid clinical improvement of the patients, followed by radiological improvement and resolution of lesions.

In our opinion, these cases serve to illustrate the course of drug-induced pneumonitis, limited by the lack of fiberoptic bronchoscopy which would have allowed samples to be obtained for a definitive diagnosis. However, it should be pointed out that the diagnosis of pneumonitis is made by exclusion. It typically presents with a clinical picture such as those described above, and the diagnostic test of choice is CT, in which bilateral opacities with patchy ground-glass opacities and a reticulonodular pattern, predominantly in the upper fields,3 are observed. If this condition is suspected, infectious causes should be ruled out, in the awareness that neither the clinical picture nor the radiological tests are conclusive. If the patient’s clinical status permits, a fiberoptic bronchoscopy with bronchoalveolar lavage, bronchial aspiration and transbronchial biopsy should be performed, on which mononuclear cell infiltration and granuloma formation characteristic of grade IV hypersensitivity reactions will be seen.4 As for treatment, in some cases, respiratory support measures are required, including orotracheal intubation. When this condition is suspected, empirical administration of corticosteroids should be initiated, although the appropriate dose has not been determined. With our cases, we aim to illustrate this disease that, while uncommon, must be taken into consideration as it can be life-threatening to our patients. This complication becomes especially important in the setting of adjuvant treatment, since this is administered to patients potentially cured of their underlying tumor. Diagnosis and early treatment with corticosteroids are essential to ensure a favorable clinical course.

References


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