Carbamazepine and the lung

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ABSTRACT: A 69 yr old man was admitted with a 10 day history of fever, arthromyalgia, dyspnoea, dry cough and pleuritic pain. Temperature was 38°C; tachypnoea 36 rpm. Extensive crackles were audible over both upper lung fields. Chest X-ray showed bilateral alveolar infiltrates. Forced vital capacity was 49% of predicted, and carbon monoxide transfer coefficient was 32% of predicted value. The patient had been taking carbamazepine for one month because of a trigeminal neuralgia. After withdrawal of the drug he gradually recovered.

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The Carbamazepine pulmonary side-effects are very uncommon. Other effects include hyponatraemia, diffuse hepatic involvement, blood dyscrasia, hypocalcaemia, etc. We report a case of carbamazepine induced pneumonitis.

Patient

A 69 yr old man was admitted into the hospital with a 10 day clinical history of fever, arthromyalgia, dyspnoea, dry cough and pleuritic pain.

One month before admission trigeminal neuralgia was diagnosed and he was initiated on 600 mg·day⁻¹ carbamazepine for two weeks and 400 mg·day⁻¹ thereafter.

On admission the patient was febrile (38°C), tachypnoeic (36 rpm), and extensive crackles were audible on the upper parts of both hemithoraces. The chest X-ray showed bilateral alveolar infiltrates in both upper lobes. The blood analysis gave: erythrocyte sedimentation rate (ESR) 116 mm; Hb 11.3 g·l⁻¹; haematocrit 33%; leucocytes 8.7x10⁹·l⁻¹ with 9 eosinophils; serum Na 129 mEq·l⁻¹; alkaline phosphatase 10.9 µkat·l⁻¹; antistreptolusin (AST) 0.6 µkat·l⁻¹; lactate dehydrogenase (LDH) 4 µkat·l⁻¹; gamma glutamyl-transpeptidase (GT) 5.3 µkat·l⁻¹; serum albumin 23 g·l⁻¹. The blood gas values were: pH 7.48; carbon dioxide tension (Pco₂) 4.4 kPa (33 mmHg); oxygen tension (Po₂) 7.2 kPa (54 mmHg).

Gram stain and culture of the bronchopulmonary secretions and of the sample obtained by transthoracic needle aspiration were negative for conventional bacteria and fungi. The fibroptic bronchoscopy and the bronchoaspirate showed no anatomical and/or cytological evidence of malignancy.

During his hospital stay the patient remained febrile and the pulmonary radiological changes progressed towards both lower lobes, adopting an alveolo-interstitial appearance. The lung function tests indicated a severe restrictive pattern with an important transfer defect: forced vital capacity (FVC) 1.7 l (49% pred) and carbon monoxide transfer coefficient (Kco) 1.28 ml·min⁻¹·torr⁻¹·l⁻¹ (32%).

Ten days after admission the carbamazepine was interrupted due to a suspicion of a drug-induced lung disease. Three days later, the patient became afebrile, the respiratory rate decreased to 20 one week later, and the intensity and extension of the crackles diminished markedly. Twenty days after the withdrawal of the carbamazepine, the lung function values were: FVC 2.3 l (66%) and Kco 2.35 ml·min⁻¹·torr⁻¹·l⁻¹ (60%). The chest X-ray showed a marked decrease in the bilateral pulmonary shadows.

At an out-patient control, one month later, the patient remained afebrile and completely asymptomatic, and the blood biology and chest X-ray were normal.

Discussion

The pulmonary toxicity induced by the carbamazepine has been reported previously [1, 2]. These two patients presented with a clinical picture similar to that of our patient, between 3–6 wks after the introduction of the drug. The most important clinical data were fever, dyspnoea and cough. The chest X-ray and the functional abnormalities are those of a diffuse infiltrative...
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pulmonary process. One case [2] was diagnosed by means of a transbronchial biopsy.

Our patient presented all the clinical, radiological and functional characteristics of a drug-induced pulmonary disease, particularly because these abnormalities reversed after the interruption of the carbamazepine. At the same time in our case, other side-effects of the drug were observed: a diffuse hepatic involvement and hyponatraemia [3].

References

