Allergy to atropine means a difficulty for future anaesthesia procedures, because of its use in the premedication to avoid vagal reflex during surgery, and to help eliminating the airways secretions. The alternative to atropine would be the glycopyrrolate, not commercialised in some countries; otherwise an alpha-beta blocker could be used, such as Ephedrine, in case of vagal complications during the intervention. The alternative mydriatic drugs to atropine would be the phenylephrine and the adrenaline.

This case highlights once again the reliability of BAT in the diagnosis of drug allergy (4, 5).

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Eosinophilic pneumonia in an asthmatic patient treated with omalizumab therapy: *forme-fruste* of Churg-Strauss syndrome?

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Key words: Churg-Strauss syndrome; omalizumab; severe asthma.

A 25-year-old male with persistent severe asthma since the age of 23 years, who was under regular treatment with fluticasone 250 μg + salmeterol 50 μg every 12 h, montelukast 10 mg/die, and salbutamol on demand, started a treatment with omalizumab 300 mg every 2 weeks. He was sensitized to *Parietaria,* grass and herb pollen with a positive skin test response and total IgE level was 435 IU/ml before starting the treatment. He was also suffering from maxillary sinusitis with polyps. A slight improvement in symptoms was noted at the second omalizumab administration. During the administration of the third omalizumab dose we observed a worsening of clinical conditions. The patient reported that few days before he had an emergency room visit because of worsening of dyspnoea but with a normal chest X-ray. Because of the presence of a non controlled severe asthma, we prescribed oral prednisone (25 mg/day for 5 days, 12.5 mg/day for other 5 days and 6 mg/day for the following 5 days). After the fourth administration of omalizumab (2 months), he was admitted to hospital owing to progressive worsening of dyspnoea and wheezing, cough, haemoptysis and left chest pain. He developed acute respiratory failure (resting PaO_2 of 55 mmHg). Physical examination revealed generalized bronchospasm. An initial chest X-ray showed bilateral parenchymal opacities and laboratory tests revealed a white blood cell count of 16890/mm^3 with 3900/mm^3 (23.1%) eosinophils. C-reactive protein was 23 mg/l, serum total IgE were >1000 IU/ml. A chest CT scan was performed and showed patchy bilateral pulmonary infiltrates and diffuse ground glass opacities (Fig. 1A). As four of the six American College of Rheumatology criteria (asthma, eosinophilia, pulmonary infiltrates and paranasal sinus polyps) were fulfilled (1) but in the absence of evidence of vasculitis and systemic disease, the diagnosis of a *forme-fruste* of Churg-Strauss syndrome (CSS) was formulated. Omalizumab was stopped, and the patient was treated with intravenous methylprednisolone (40 mg/day). After 3 days of treatment, a significant improvement of symptoms was observed, with complete resolution of dyspnoea, and significant decrease in cough severity. A chest CT scan performed after 20 days showed disappearance of pulmonary infiltrates (Fig. 1B). The patient was discharged under therapy with prednisone 5 mg/day, montelukast 10 mg/day, formoterol 12 μg + budesonide 200 μg every 12 h. Our therapeutic plan provided for the discontinuation of treatment with omalizumab.

Omalizumab may facilitate the development of Churg-Strauss syndrome.

This case poses the relevant question to the clinician whether omalizumab may facilitate the development of CSS or not and, if not, whether omalizumab can be considered as a therapy for CSS. Omalizumab is used for the treatment of persistent severe asthma associated with high serum IgE levels (2), but elevated IgE is also a well-known leading laboratory finding in CSS (3). Nonetheless, a link between omalizumab and CSS has been hypothesised, although it is poorly understood. Two mechanisms are possible:

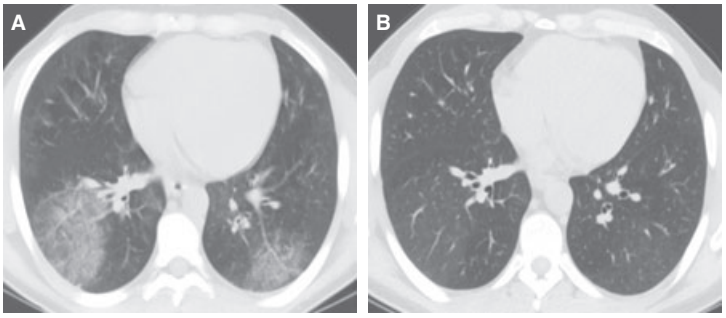

Figure 1. (A) CT scan of the chest, performed when the patient was hospitalized, showing bilateral pulmonary infiltrates in a patient with a *forme-fruste* Churg-Strauss Syndrome. (B) CT scan of the chest, performed 20 days after admission to hospital and a treatment with systemic steroids showing disappearance of pulmonary infiltrates.

(i) omalizumab plays a direct causative role in the development of CSS; (ii) the reduction of corticosteroids, allowed by omalizumab treatment, can unmask CSS, which, beforehand, manifests itself only by asthma, as a *forme-fruste* of CSS (4).

Although the existing evidence seems to indicate that omalizumab can be continued as soon as acute CSS is resolved in order to achieve an optimal control of the asthmatic component (5, 6), we preferred to stop treatment, so as not to expose the patient to a potential risk of further evolution of CSS. In any case, our case suggests to taper corticosteroid with caution and to strictly monitoring patients under omalizumab treatment for signs of CSS, as apparently anti-IgE therapy does not control CSS activity.

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Occupational asthma and rhinitis induced by a cephalosporin intermediate product: description of a case

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Key words: 7-TACA; cephalosporin; occupational asthma; occupational rhinitis; specific inhalation challenge.

7-Amino-3-thiomethyl-3-cephalosporanic acid (7-TACA) is an intermediate product in cephalosporins' synthesis. Few cases of occupational asthma (OA) due to cephalosporins (1) and to their intermediate product 7-ACA (2) have been reported, but no report exists on OA due to 7-TACA nor on occupational rhinitis (OR) induced by cephalosporins or intermediate products.

7-TACA as a new causal agent of occupational asthma and rhinitis.

A 44-year-old man was referred to our department for evaluation of work-related respiratory symptoms. Since July 2007 he had been employed in a pharmaceutical company, assigned to cephalosporins' product line. The product line was enclosed except for loading phases, during which the raw materials and intermediate products (7-TACA) were poured into reactors. Eight months after starting that job, he began to complain of sneezing, and 1 month later of dry cough and shortness of breath. In July 2008 he experienced severe dyspnoea requiring hospitalization after a loading operation. Thereafter he was moved to the corticosteroid product line and became completely asymptomatic.

He came to our observation 4 months after the last exposure to 7-TACA. Personal history of allergy was negative. No similar symptoms were reported by other employees.

The patient underwent the common diagnostic pathway for OA (3) and OR (4). Lung function tests were normal, skin prick tests (SPTs) to common allergens were positive for house dust mites. Serum IgE levels were 98.9 kU/l and peripheral eosinophilia 1.1%.

SPT with 7-TACA was not performed because the compound was not soluble in saline. A Specific Inhalation Challenge (SIC) with occupational method was carried out (5). Before and after SIC nasal secretions (NS) and induced sputum (IS) were collected, methacholine (MCH) challenge test was performed and serum tryptase and eosinophils in peripheral blood were detected.

After a control day in which the patient was exposed to lactose, exposure to 0.5% 7-TACA mixed with 99.5 mg of lactose in a 7.46 m^3 inhalation challenge room (6), elicited dyspnoea after 5 min of exposure and was interrupted. Spirometry showed a 22% FEV_1 fall which decreased up to 32% at 15 min after the end of exposure.