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The reversed halo sign: also think about chronic eosinophilic pneumonia

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TO THE EDITOR:

The reversed halo sign (RHS) is a focal rounded or lobulated area of central ground-glass opacity surrounded by a more or less complete ring of consolidated lung tissue.[1] Although the RHS was initially described in patients with cryptogenic organizing pneumonia, various authors have demonstrated its presence in a wide spectrum of diseases (e.g., tuberculosis, invasive pulmonary aspergillosis, noninvasive fungal infections, *Pneumocystis jirovecii* pneumonia, pulmonary infarction, nonspecific interstitial pneumonia, granulomatosis with polyangiitis, sarcoidosis, lipoid pneumonia, lung adenocarcinoma, metastatic lung disease, and lymphomatoid granulomatosis); therefore, the RHS cannot be considered to be specific for cryptogenic organizing pneumonia.[2-4]

A 28-year-old nonsmoking male undergraduate with a history of allergic asthma—which had been diagnosed on the basis of clinical findings and pulmonary function test results—had been hospitalized 6 months prior because of an acute episode of cough and fever. Chest X-rays showed bilateral infiltrates in the upper and middle lung fields, and a diagnosis of bilateral pneumonia was made. The patient was treated with ceftriaxone and macrolides for 15 days. Laboratory tests revealed high eosinophil levels (13.5%) and ESR (42 mm/h), as well as normal serum IgE levels (53 IU/mL). Autoantibody testing and fecal parasitology were negative. Because of persistent dry cough and asthma, chest HRCT scans were taken 6 months later, revealing patchy bilateral areas of lobulated ground-glass opacity surrounded by crescent-shaped consolidation in the upper lobes and in the apical segment of the right lower lobe (i.e., the RHS; Figures 1A and 1B). On the basis of patient clinical history, blood eosinophilia, and HRCT findings, a presumptive diagnosis of chronic eosinophilic pneumonia (CEP) was made. A fiberoptic bronchoscopy was performed in order to confirm the diagnosis. Analysis of BAL fluid revealed a marked increase in eosinophils (25%), with mixed granulocytic/lymphocytic alveolitis (neutrophils, 10%; lymphocytes, 21%) and a normal CD4/CD8 ratio. The patient was started on corticosteroid therapy (oral

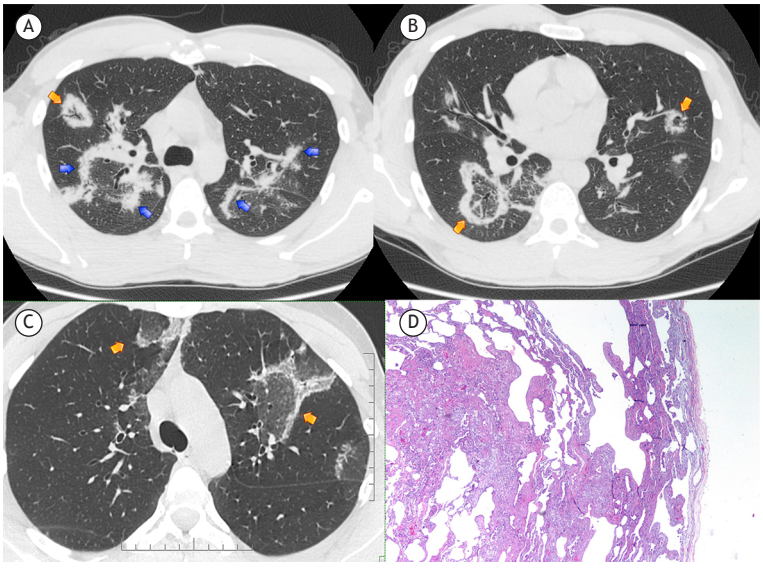

Figure 1. In A (case 1), axial HRCT scan of the chest at the level of the upper lobes showing the reversed halo sign (RHS) in the anterior segment of the right upper lobe (orange arrow), as well as bilateral peripheral and peribronchovascular consolidative opacities (band-like elements; blue arrows). In B (case 1), axial HRCT scan of the chest at the level of the lower lobes showing a well-defined lobulated RHS in the apical segment of the right lower lobe and a small RHS in the posterior segment of the left upper lobe (orange arrows). In C (case 2), axial HRCT scan of the chest at the level of the upper lobes showing the RHS in the anterior segment of the left upper lobe (orange arrow) and a slightly incomplete sign in the anterior segment of the right upper lobe (orange arrow). In D (case 2), note subpleural and septal fibrosis.

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prednisolone at a dose of 12.5 mg/day) after review of the clinical and radiological data. At this writing, he was symptom free, nearly complete resolution of pulmonary infiltrates having been reached. In a second case, a 33-year-old nonsmoking female office worker with a clinical history of surgery for an ovarian cystadenoma was hospitalized because of persistent headache, fever (38.5°C), and dry cough. A chest X-ray showed bilateral pulmonary infiltrates. The patient reported that the symptoms had started after her return from a two-week holiday in Morocco. Laboratory tests showed high eosinophil levels (32.6%), mild anemia (hemoglobin, 9.7 g/dL), and increased ESR (41 mm/h). The patient therefore underwent HRCT, which showed the RHS in the anterior segment of the left upper lobe (Figure 1C) and peripheral RHSs in both lower lobes, with thick, consolidative band-like opacities in the subpleural region on the right. Autoantibody testing, HIV testing, antineutrophil cytoplasmic antibody testing, Mantoux tuberculin skin testing, and microbiological testing were negative. All pulmonary function test results were within normal limits. A fiberoptic bronchoscopy was performed, and the results were negative. An open lung biopsy was performed to assist with diagnosis, showing septal inflammation, septal fibrosis, irregularly distributed lesions, peripheral fibrosis, and eosinophils (Figure 1D). The aforementioned pathological findings were suggestive of advanced healing of CEP. An HRCT scan taken 3 month later showed that the patient had made an almost complete recovery.

Although an uneven distribution of consolidative changes predominantly in the upper lobe has been reported in most cases of CEP, the RHS has only recently been reported in a case of CEP.[5] Given that the RHS is a nonspecific HRCT finding, patient clinical history, laboratory findings, and ancillary radiological findings should also be taken into account in order to narrow the differential diagnosis. In addition, it is useful to determine patient immune status when investigating the RHS, given that delayed diagnosis and treatment can increase mortality. Given that the RHS is a CT finding that is present in multiple diseases, a pathological specimen is needed in order to make an accurate diagnosis (as in our second case); it is equally true, however, that careful clinical and radiological evaluation through a multidisciplinary approach can be diagnostic in the presence of the RHS, without the need for biopsy.

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