correction of the contribution of these two excellent scientists, *Silk Route disease* is correctly called *Adamantiaides-Behçet’s disease.*

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**REFERENCES**


**More About Churg-Strauss Syndrome and Montelukast Treatment**

**To the Editor:**

It has been suggested that the reduction or withdrawal of systemic or inhaled corticosteroid therapy because of the clinical improvement of asthma after leukotriene-modifying drug treatments may play a decisive role in the appearance of a fruste form of Churg-Strauss syndrome (CSS) that previously existed.1 We present the case of a 49-year-old woman with a 15-year history of allergic rhinitis and mild-to-moderate asthma, for which she received only the inhaled β2-agonist salmeterol twice daily, but with good control of her disease. She had never received inhaled or systemic corticosteroids. Five months after her physician changed her regimen from salmeterol to montelukast, she presented with arthralgias in her hands and feet, vomiting, severe abdominal pain, anemia, leukocytosis with hyper eosinophilia (40%), and an accelerated erythrocyte sedimentation rate. The results of tests for antineutrophil cytoplasmatic antibodies (MPO and PR3) and antinuclear antibodies were normal or negative. Esophagogastroduodenoscopy, ultrasonography, and CT scanning of the abdomen showed no abnormalities. So, a laparotomy was performed, and inflammation in the first portion of the duodenum was observed. Histologic examination revealed a necrotizing vasculitis with extravascular granulomas, which is compatible with CSS. Montelukast therapy was discontinued, and high doses of corticosteroids and an IV bolus of cyclophosphamide were prescribed. However, 6 months later, the patient experienced a new episode of acute abdominal pain, vomiting, and anemia, and a new surgical procedure was required. A pyloric stenosis due to a granuloma that was 5 × 10 cm in diameter was found, and an antrectomy was performed. The histologic study showed the same findings of necrotizing vasculitis.

Thus, we report an unusual and severe case of CSS with GI involvement that developed 5 months after montelukast therapy was started in a patient who had never used systemic or inhaled corticosteroids for asthma treatment. We do not know whether the appearance of CSS after montelukast use in our patient could be due to a casual association, but we cannot rule out a possible pathogenic link between leukotriene-modifier drug use and CSS development.

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**REFERENCE**


**Perimenstrual Asthma Exacerbations and Positioning of Leukotriene-Modifying Agents in Asthma Management Guidelines**

**To the Editor:**

The excellent review in CHEST (May 2001) by Salvi et al.1 discussing the combined anti-inflammatory and bronchodilating effects of leukotriene-modifying agents and their positioning in asthma management guidelines, is both timely and needed in updating guidelines for asthma therapy. Perimenstrual exacerbations of persistent asthma, in addition to exercise- and aspirin-induced asthma, is an example of a subset of asthma better controlled by the addition of a leukotriene-modifying agent to a regimen containing inhaled steroids. Forty percent of asthmatic female patients have perimenstrual exacerbations of asthma uncontrolled by steroid therapy.2 Female asthmatics have an increase in bronchial reactivity to adenosine monophosphate and dysregulation of β2-receptors concomitant with fluxes of estrogen and progesterone during the transition from the luteal to the follicular phase of the menstrual cycle.3 Studies4–5 have noted the inability of therapeutic steroids to inhibit clinical perimenstrual exacerbations of persistent asthma. The efficacy of leukotriene-modifying agents in preventing perimenstrual exacerbations and concomitant increases in leukotrienes have been cited in two reports.4,5 Additional studies examining the role of leukotriene-modifying agents in perimenstrual asthma exacerbations and on the neuroendocrine immune system are needed.

The combined anti-inflammatory and bronchodilating properties of leukotriene modifiers described by Salvi and colleagues1 highlight both the early (step 2) role for these controller agents as well as a role in all stages of severity of persistent asthma. Leukotriene modifiers, particularly in perimenstrual-, exercise-, and aspirin-triggered asthma and asthma with comorbid rhinitis, merit consideration as an addition to inhaled steroid therapy as a controller targeting inflammation not adequately controlled by steroids.

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