

Although rare, the neoplastic transformation of LP has been reported.¹⁵ In clinical trials of omalizumab, malignant neoplasms (mostly solid tumors) were observed in 20 of 4,127 omalizumab-treated patients (0.5%) compared with 5 of 2,236 control patients (0.2%). The onset of most tumors preceded treatment with omalizumab, and 60% of tumors presented within less than 6 months of treatment.^{14,16} Omalizumab therapy was discontinued in this patient to prevent further recurrences of DiLP and a theoretical risk for LP-associated malignant transformation. Our observation suggests that omalizumab should be considered as a cause of DiLP.

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MONTELUKAST-ASSOCIATED CHURG-STRAUSS VASCULITIS: ANOTHER ASSOCIATED REPORT

A 37-year-old woman presented to her allergist's office after 6 months of dry cough. Before her appointment, she was treated sporadically with inhaled corticosteroids, albuterol, and antibiotics but showed no improvement. On evaluation, aeroallergen skin prick test results were negative; a complete blood cell count was normal, with 4% eosinophils; and her chest radiograph showed only peribronchial thickening. She was empirically administered montelukast for possible cough variant asthma.

Within 1 month, her cough began to worsen, and she developed dyspnea that required 2 pulse corticosteroid treatments for relief. Inhaled salmeterol and budesonide were added. Chest computed tomography revealed a left-sided suprahilar opacity. The patient was referred to a pulmonologist for bronchoscopy. At that visit, a repeated chest radiograph showed infiltrates in the left upper lobe and lingula, and lavage fluid from the bronchoscopy showed predominant eosinophilia. Shortly thereafter, the patient developed a vesicular rash on her head and arms, fever, myalgias, hemoptysis, pleuritic chest pain, and worsening dyspnea. On examination, she had distended neck veins with bilateral infiltrates and cardiomegaly on her chest radiograph.

On admission to the hospital, her echocardiogram showed a depressed left ventricular ejection fraction of 42% with a pericardial effusion. Pericardiocentesis yielded fluid with a differential count significant for eosinophilia of 42%. She also had peripheral eosinophilia of 24%. A biopsy specimen of the hand revealed necrobiotic collagen with granulomas and an eosinophilic infiltrate (Fig 1). The patient improved with administration of intravenous inotropes, diuretics, and corticosteroids. Montelukast use was discontinued. During the next year, she was weaned off oral corticosteroids, and she now has intermittent asthma that is controlled by daily inhaled mometasone therapy. She also developed nasal polyps that were treated successfully with surgery.

Churg-Strauss vasculitis (CSV) is a small- to medium-sized vessel vasculitis associated with asthma and peripheral eosinophilia. This patient had 4 of the 6 American College of Rheumatology criteria for a CSV diagnosis (asthma, peripheral eosinophilia, pulmonary infiltrates, and extravascular eosinophils),

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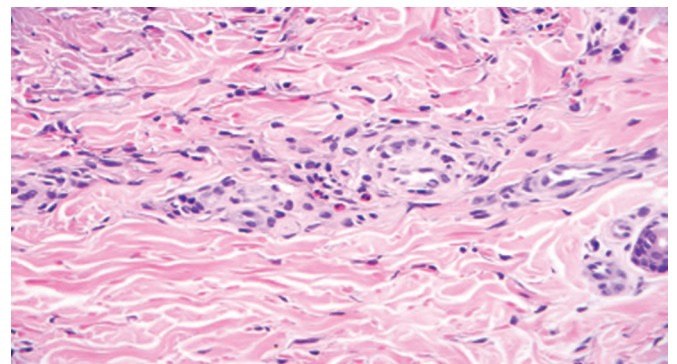


Figure 1. Perivascular eosinophils and granuloma formation in a skin biopsy sample, findings consistent with Churg-Strauss vasculitis.

and she later developed a fifth (nasal polyps). In patients with CSV, indicators of poor prognosis include cardiac and gastrointestinal involvement.¹ In a study by Guillevin et al,¹ 96 patients with CSV were followed up to evaluate outcomes. This study showed 39% mortality with myocardial disease, and most of these patients died during the acute phase of their illness. Corticosteroids are the first-line treatment for CSV, and remission rates are 80% to 90% with this therapy.

In the past decade, there seems to be an increase in the diagnosis of CSV with the introduction of leukotriene modifiers, such as montelukast. Wechsler et al² published a 6-patient case series in 2000 that concluded that the relationship is coincidental. The authors postulated that the vasculitic component of CSV is suppressed by corticosteroids and that the addition of montelukast allows for the withdrawal of corticosteroids, thus allowing the vasculitis to be "unmasked." Lofdahl et al³ showed in a double-blind, placebo-controlled trial that montelukast can allow for tapering of inhaled corticosteroids, but this may not be the case in all patients.

Other case reports have shown a diagnosis of CSV in association with montelukast while inhaled corticosteroids were not being weaned. Sabio et al⁴ described a 49-year-old patient who developed rash and duodenal necrotizing vasculitis 5 months after switching from salmeterol to montelukast. Villena et al⁵ described a patient who developed rash, eosinophilia, and bilateral pulmonary infiltrates 4 months after beginning montelukast treatment while taking inhaled corticosteroids and bronchodilators only. Solans et al⁶ described an asthmatic patient who had never received oral corticosteroids who developed CSV 4 months after initiating montelukast treatment.

In summary, this patient developed corticosteroid-dependent asthma and biopsy-proven CSV with a poor prognostic indicator of cardiac involvement after starting montelukast treatment. She survived with cessation of montelukast treatment, and currently her asthma is controlled with mometasone only. Although there is strong circumstantial evidence of a relationship in this patient, Weller et al⁷ showed in an extensive summary that no evidence exists to support the causation of CSV by leukotriene receptor antagonists. As cases continue to be reported, there should be further investigation to determine whether a true relationship exists.

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β -AGONIST USE AS AN INDICATOR OF CHANGE IN ASTHMA CONTROL DURING PREGNANCY

Asthma is a common chronic disease that affects care during pregnancy in approximately 8% of pregnant women.¹ Asthma during pregnancy has adverse effects on both mothers and infants, including increased risk for preeclampsia, pregnancy-induced hypertension, preterm delivery, and birth weight of less than 2,500 g.^{2,3} A commonly quoted generalization based on data from several small and select study populations is that the course of asthma worsens in one-third of pregnant women, improves in one-third, and remains unchanged in one-third.⁴

In a large population-based cohort, we studied 8,149 pregnant women with asthma from a larger group of 112,171 pregnant white and black women aged 15 to 44 years who were enrolled in TennCare, Tennessee's Medicaid managed care program from 1995 to 2001 and who had at least 180 days of continuous enrollment before their last menstrual period. The 112,171 eligible women represented 48% of all deliveries to women enrolled in Tennessee Medicaid and 21% of all live births in Tennessee during the study period. Data were obtained from linked Tennessee Medicaid database and vital records files, providing information on maternal demographics, *International Classification of Diseases, Ninth Revision (ICD-9)* diagnoses, and pharmacy claims. The protocol was approved by the institutional review boards of Vanderbilt University and the Tennessee Department of Health. A more complete discussion of these methods is contained in the article by Enriquez et al.⁵ Asthma control was defined by use of short-acting β -agonists. Pharmacy claims containing short-acting β -agonist dose and days of supply were available for all study participants from 25 weeks before last menstrual period until the end of pregnancy. These data were used to determine short-acting β -agonist use for each week from 20 weeks before last menstrual period through the last full week of pregnancy or 40 weeks. Women were classified as current users for each week that was within 30 days of their last dispensed prescription; otherwise, they were classified as nonusers for that week. In Figure 1 (bottom), 95% confidence bands were estimated by bootstrap simulations.⁶

Among the 8,149 pregnant asthmatic women, 77% used short-acting β -agonists during follow-up. Overall, pregnant women had an initial decrease in β -agonist use in the first trimester before experiencing peak use in the third trimester, with 31.8% experiencing worsening asthma control, 31.4% experiencing unchanged asthma control (defined as <5% variation from baseline), and 36.8% experiencing improved asthma control during pregnancy. Changes in use for black and white women are given in Figure 1 (top). Figure 1 (center) shows that asthma control improves during the first half of pregnancy and then worsens. Among pregnant white women control improves during the first half of pregnancy and then reverts to control similar to baseline (Figure 1). However, black women show

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