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CASE STUDY

Does My Patient Have Asthma?

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Introduction

Physicians all know what asthma is. Southern California Kaiser Permanente (KP) physicians treated 211,350 patients during 458,868 encounters for asthma (International Classification of Diseases code = 493.xx) in 2009 (data from internal, password-protected KP research database). We do a great job of providing at least one inhaled steroid canister annually for every patient who meets the current Healthcare Effectiveness Data and Information Set (HEDIS) criteria for persistent asthma. We give them to virtually 100% of these patients. So why might this be a problem? Why ask: "Does my patient have asthma?"

Imagine if one-third of the individuals you treated for type 2 diabetes really had some other problem and did not really need or benefit from the medications that you gave them over the long term. This is essentially what often happens with asthma management. It is important to remember that asthma is a syndrome and that the diagnosis is often made clinically, based on wheezing and shortness of breath, but must be confirmed to justify long-term treatment with inhaled steroids with or without long-acting bronchodilators. Self-reported asthma symptoms and even physician-diagnosed "asthma" are more common with obesity, but reversible airflow obstruction is not.

Case Report

Now for the case:

Ms X, age 57 years, transferred her medical care to KP in late December 2009. She was initially seen in primary care on January 6, 2010, with a diagnosis of steroid-dependent "asthma," along with obesity, depression, reflux, sleep apnea, pollen allergy, hypertension, hyperlipidemia, and prediabetes. She was quickly referred to the Allergy Department where she was initially seen on January 11, 2010. She had been taking oral steroids daily since 2003, averaging about 20 mg of prednisone per day. She had episodically taken as much as 60 mg/d. Tapering had been tried in the past but was always stopped secondary to myalgias, shortness of breath, and depression. These symptoms would worsen markedly when dosage reached 10 mg/d. She was a 45-pack-year smoker who quit in 1998. Her shortness of breath did not start until 2002. She underwent environmental skin testing in 2002 and was noted to be allergic to pollens only. She had gained >27 kg in the decade before symptom onset. Her body mass index was 39.6. She underwent sinus surgery in 2003. She had no childhood history of asthma, and she had not undergone lung function tests to document reversible airflow obstruction before being seen at KP. She had not undergone a methacholine challenge. She had been getting poor-quality sleep for years. Sleep apnea was initially diagnosed in 2005, and she had been using continuous positive airway pressure (CPAP) when initially seen, but she did so irregularly because it did not seem to help.

When initially seen in the Allergy Department, she had normal spirometry results (forced vital capacity, 91%; forced expiratory volume in the first second of expiration, 96%; ratio of forced vital capacity to forced expiratory volume in the first second of expiration, 84%), without obstruction or restriction. She had a low normal fraction of exhaled nitric oxide (16 parts per billion). She had normal findings on sinus radiographs, with no air fluid levels. Steroids were initially tapered by 10 mg every other week. The combination steroid and long-acting bronchodilator she had been using was stopped. The leukotriene inhibitor she had been given was stopped. The angiotensin-converting enzyme (ACE) inhibitor that she had been taking was stopped, and she was given an angiotensin-receptor blocker instead. Her CPAP machine was retitrated, her anti-reflux therapy was reinforced, and she began an exercise and weight-loss program. She lost >18 kg by November 2010. When prednisone dosage was down to 10 mg/d, the taper was slowed to 1 mg every other week. When she caught viral infections, the steroid taper was slowed. She was no

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longer taking oral prednisone by early November 2010. Because the patient received help in controlling her weight, sleep apnea, iatrogenic cough, and reflux laryngitis, her "asthma" symptoms disappeared. She still coughs when she has viral infections, but with her assistance and understanding, her health care team is resisting future long-term treatment with oral steroids.

Discussion

Physicians learn in medical school that asthma is a chronic inflammatory lung disease. It is clinically characterized by shortness of breath and wheezing and physiologically verified by documenting reversible airflow obstruction or bronchial hyperreactivity. We know that there are many other conditions that will cause asthma-like symptoms, including obesity, heart failure, smoking, reflux laryngitis, viral infection, sinusitis, laryngeal dysfunction, use of ACE inhibitors, and aspiration pneumonia, but we still tend to rely on the clinical symptoms of coughing, wheezing, and shortness of breath to diagnose asthma. Asthma treatments are extremely effective in individuals with reversible airflow obstruction caused by small-airway inflammation. Overuse of bronchodilators can contribute to worsening cough, laryngitis, and reflux. Use of high-dose inhaled steroids increases the risk of diabetes.[1] Asthma treatments can seem to provide transient symptomatic relief of shortness of breath in individuals without asthma, and asthma medications are heavily advertised directly to consumers. We know there has been an epidemic of "asthma" diagnosed since the 1980s, but we still don't want to miss the diagnosis or fail to provide symptomatic therapy to our patients.

When my colleagues and I studied individuals in 2005 who were continuous long-term KP members and who met the HEDIS criteria for persistent asthma in any year between 1999 and 2002, a surprisingly high 48% of them met the criteria in only one of the four years. We found that in that four-year period, only 19% of them met the criteria every year.[2] Partly because of that study, the national HEDIS definition of persistent asthma was changed. Now we look at individuals who meet the criteria for at least two years in a row and see whether they are getting any preventive medication.

When we randomly sampled individuals in the KP Asthma Case Identification Database with a clinical diagnosis of asthma in San Diego in 2001, we found that more than one third of them had no evidence of reversible airflow obstruction or bronchial hyperreactivity when tested in 2003.[3] These patients' had carried a physician diagnosis of asthma for a mean of >22 years, yet before the study evaluation, only 13% of them had ever had undergone measurement of their forced expiratory volume in the first second of expiration pre- and postbronchodilator. This was surprising because 40% of them had been seen at least once by Allergy or Pulmonary Departments during their care at KP and almost 80% had at least one spirometry in their medical records. KP physicians are not the only clinicians who tend to over diagnose asthma. Similar findings have been published for large trials in Ontario, Canada.[4]

Most patients who currently have a diagnosis of asthma in their KP records have still never undergone pulmonary function testing to document reversible airflow obstruction. Among patients whose pulmonary function has been tested, many still have a diagnosis of asthma in their medical records and receive chronic asthma medications, even when the test findings were normal or negative. Methacholine challenges are done rarely to confirm bronchial hyperreactivity in individuals with clinical asthma symptoms yet repetitively normal findings on spirometry. Methacholine challenges are available in all KP medical centers, either in the Allergy Department or in the Pulmonary Laboratory, depending on the site. KP HealthConnect now can use flow sheets to track spirometry results obtained in Allergy Departments. Results for complete lung function tests done in the KP pulmonary laboratories are still generally available only as scanned reports or text.

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Patients "with asthma" who require chronic oral steroids for "control" frequently have one or more other chronic conditions that are their primary problems. They typically also have iatrogenic comorbidities. When these patients are assessed in-depth and their comorbidities are addressed, they rarely require daily oral steroids for symptom management. Some patients with true asthma will require recurrent oral steroids, but this is a rare exception, and these patients should all be treated in the Allergy or Pulmonary Department. Aspirin-exacerbated respiratory disease is often the cause of the need for oral steroids; it can be treated with aspirin desensitization.[5] Exhaled nitric oxide is a marker for eosinophilic inflammation, and a low fraction of exhaled nitric oxide argues against active asthma as the cause of shortness of breath in a person with normal spirometry findings.[6] Oral

steroids can help calm laryngeal irritation mimicking asthma, but at the price of greater obesity and severe somatic symptoms when withdrawn.

For all of the patients whose condition has been diagnosed as "asthma" and who take chronic inhaled steroids and long-acting bronchodilators or leukotriene inhibitors, the diagnosis should be confirmed by pulmonary function tests before and after bronchodilator use to document reversible airflow obstruction or should undergo a methacholine or mannitol challenge. For patients found to have normal lung function and no bronchial hyperreactivity, the other causes of their "asthma" symptoms should be addressed and treated. ❖

Disclosure Statement

The author(s) have no conflicts of interest to disclose.

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Wheeze

All that wheezes is not asthma.

— Chevalier Jackson, 1865-1958, a laryngologist and pioneer of the modern science of endoscopy