

7 separate pages. In addition, the report includes flawed instructions for using a peak flow meter and for determining the patient's personal best peak expiratory flow rate (PEFR). Providers who use these directions to instruct their patients may provide less than optimal care.

A comprehensive asthma diary is a powerful tool to help patients manage their asthma at home.² Patients can use a diary to identify triggers, track their medicines, determine when to start and stop taking medicines, and assess whether they are getting better or worse. A PEFR-based diary can be used to monitor patients 5 years and older.^{3,4} A signs-based diary⁵ can be used for young children. Because the report neither displays nor explains how to use a diary, providers cannot appreciate its value in daily management.

Experts agree that PEFR is an objective and more reliable^{6,7} measure of the patient's status than are the symptoms of asthma. The PEFR is a particularly useful way for patients to monitor their status at home. However, many physicians do not use the PEFR to guide treatment because their patients have blown inconsistent scores. This usually occurs when patients block airflow in their mouth, causing pressure to build up before release. Unfortunately, the instructions in the EPR-3 do not fully prevent this problem. The EPR-3 advises, "Place the mouthpiece in your mouth and close your lips around it. Do not put your tongue inside the hole."^{1(p122)} But the report neglects to mention that spitting⁸ or coughing during the maneuver might also block airflow. The PEFR is reliable and consistent if patients use the proper technique.

The concept of a personal best is the same for PEFR as it is in sports. It is the patient's best score ever (when using proper technique). The EPR-3 states, "Your personal best peak flow number is the highest peak flow number that you can achieve over a 2-week period when your asthma is under good control. Good control is when you feel good and do not have any asthma symptoms."^{1(p122)} The instructions in the EPR-3 are based on the inaccurate assumption that a patient's airflow is normal when he or she feels good. In fact, many asthmatic patients feel good even when their lung function is reduced by as much as 50%.^{9,10} For this reason, physicians who follow the instructions in the EPR-3 may establish a "personal best" that is falsely low. The patient's personal best PEFR serves as the basis for developing an asthma action plan that will guide home treatment. A patient who relies on an action plan based on a falsely low personal best score may delay timely treatment.

Because the PEFR increases steadily with treatment, the personal best is determined after it has reached its highest point and is then held steady for at least a week. Measurements should be made in the afternoon, when peak flow levels are highest. It may require several weeks of optimal treatment before the PEFR plateaus. Until that time, physicians can use the predicted PEFR (mean PEFR for height, age, and sex) to guide treatment.

The EPR-3 includes many useful recommendations. However, physicians will need to supplement those recommendations with a fuller understanding of proper PEFR technique and the directions for calculating the personal best PEFR.

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ACUTE GENERALIZED EXANTHEMATOUS PUSTULOSIS CAUSED BY RADIOCONTRAST MEDIA

Acute generalized exanthematous pustulosis (AGEP) is a rare pustular skin eruption usually triggered by drugs. AGEP is characterized by nonfollicular sterile pustules on erythematous skin and a rapid clinical course, with favorable prognosis in most patients.¹ Its pathogenesis remains uncertain, but drug-specific positive patch test results and in vitro lymphocyte proliferative reactions in patients with AGEP strongly suggest a T-cell-mediated process.^{2,3} We describe a patient who developed AGEP after intravenous injection of radiocontrast media.

A 27-year-old man who had been diagnosed as having chronic renal failure due to IgA nephropathy in August 1995 and who had undergone 2 kidney transplant operations in 1997 and 2002 because of short-term rejection after his first kidney transplant developed chronic allograft nephropathy in 2005 and was given hemodialysis 3 times a week through a left arteriovenous fistula. A few days before his hospital visit, he had experienced increased venous return pressure and decreased flow during hemodialysis but without reduction of thrill or bruit. Angiographic evaluation showed segmental stenosis at the anastomosis of the polytetrafluoroethylene prosthesis and the axillary vein. To improve the patency of the vascular access, balloon percutaneous transluminal angioplasty was scheduled 5 days later.

On that evening, approximately 6 hours after the angiography using radiocontrast dye (ioversol, 10 mL), the patient noticed a rash involving the posterior aspect of his neck and both axillary areas. The primary lesions were multiple 1-mm red papules accompanied by pruritus. The next day, he developed fever and chill, with widespread skin eruption over the entire body, progressing to pustules. He has neither experienced this type of rash previously nor been diagnosed as having psoriasis. Initial physical examination revealed a temperature of 38.9°C but no inflammatory signs, such as redness, swelling, or tenderness overlying the graft. The rash was composed of fine papules and pustules on erythematous skin involving the entire body (Figure 1). Laboratory examination revealed an elevated white blood cell count of 20,400/ μ L with an increased neutrophil count (19,726/ μ L) and

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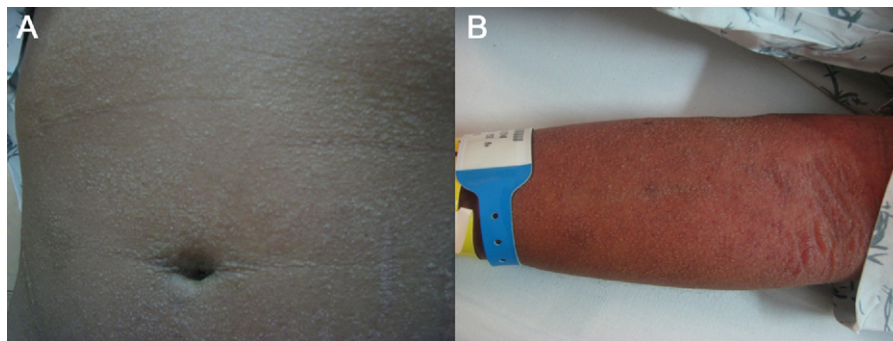


Figure 1. Multiple erythematous pustular eruptions involving the trunk (A) and upper extremities (B).

an elevated C-reactive protein concentration (11.23 mg/dL). All other laboratory values were unremarkable. A biopsy specimen was not taken. The patient was treated with 0.05% topical desonide lotion for the rash, the antihistamines hydroxyzine and levocetirizine for the pruritus, and empirical antibiotics for possible systemic infection. On day 4, his fever had subsided completely and all blood culture results were negative. On day 6, generalized cutaneous desquamation occurred with marked resolution of his erythema and pustules.

AGEP is characterized by a sudden onset of fever and widespread numerous nonfollicular sterile pustules and has a benign, self-limiting course.¹ Although most cases of AGEP (>90%) have been associated with drugs, in particular antibiotics, its origin is variable, and it may be triggered by viral infection, hypersensitivity to mercury, or spider bites. Several case reports have described patients with AGEP induced by contrast media, including 2 patients with delayed, generalized, and protracted cutaneous reactions compatible with AGEP after contrast medium administration,⁴ 1 patient with contrast-induced AGEP,⁵ and 1 patient who developed a pruritic pustular eruption on 2 separate occasions after administration of a nonionic contrast medium.⁶

The main histopathologic findings in most patients with AGEP include spongiform superficial pustules, papillary edema, polymorphonuclear cells with some perivascular eosinophils, and leukocytoclastic vasculitis with fibrinoid deposits. Our patient had the characteristic morphologic features and course of AGEP, except that histologic features could not be identified because no biopsy specimen was obtained.

To our knowledge, this is the first case of AGEP induced by contrast media ever reported in Korea. Physicians should bear in mind that AGEP can be induced by radiocontrast media.

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THE SAFETY AND INTERPRETABILITY OF SKIN TESTS WITH OMALIZUMAB

Omalizumab is an anti-IgE antibody used to reduce exacerbations in patients with moderate to severe allergic asthma. There have been reports of systemic reactions after injection with omalizumab consistent with anaphylactic reactions; however, it remains unclear whether these reactions are caused by IgE sensitization to omalizumab or its excipients vs an alternative non-IgE-mediated mechanism.¹ The product, omalizumab (Xolair; Genentech/Novartis, San Francisco, California) includes the omalizumab antibody and the following excipients: sucrose, L-histidine, L-histidine hydrochloride monohydrate, and polysorbate 20.

To advance our understanding of the potential role of IgE antibodies to omalizumab (or one of its excipients) relative to these reactions, an interpretable result on a skin test to omalizumab and its vehicle is needed. A study to examine whether omalizumab skin testing is safe and to establish an appropriate nonirritating concentration for prick and intradermal testing was conducted. This protocol, the informed consent form, and relevant supporting information were submitted to the Mid* Land Institutional Review Board (Overland, Kansas) by the principal investigator for review and approval before the study was initiated.

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