

sitivity has not been shown.³ We describe a patient who was successfully desensitized to aspirin despite a history of CIU.

The patient is a 49-year-old man with a history of diabetes mellitus, childhood asthma, hypertension, gastroesophageal reflux disease, myocardial infarction, and intermittent hives. He first noticed the hives when he was 16 years old. He developed generalized pruritic lesions that lasted 24 hours and spontaneously disappeared. At the age of 33 years, he was prescribed an adult dose of a nonsteroidal anti-inflammatory drug (NSAID) for a leg injury. A few days later, he noticed hives that began on his legs and spread upward 10 to 15 minutes after taking the NSAID. He took diphenhydramine, and the hives resolved. A few months later, the patient was having mild arthritic pain and took an adult dose of aspirin. Ten minutes later he began to develop hives. The hives again were relieved by oral diphenhydramine treatment. Since that time, despite avoiding NSAIDs and aspirin, the patient reports having outbreaks of hives 5 to 6 times per year with no apparent triggers. He had no respiratory complaints, swelling of eyes or lips, or gastrointestinal problems associated with the hives.

The patient was referred to us for aspirin desensitization because of his history of myocardial infarction. Physical examination findings were normal. There were no hives or evidence of dermatographism. Spirometry showed normal lung function, and autologous serum skin test results were negative. The patient was diagnosed as having CIU and aspirin sensitivity. He was advised of the risks of desensitization and agreed to the procedure.

Aspirin desensitization treatment began with 40 mg given at 10 AM. The patient had some mild itching of his ears, but this spontaneously abated. At 11:30 AM, he was given aspirin, 81 mg. He was observed for 2 hours and had no reaction. Later that afternoon, 3.5 hours after the aspirin administration, the patient had a severe flare-up of generalized urticaria. He took diphenhydramine, 25 mg, and the hives subsided. The next day, he was given aspirin, 81 mg, at 8:30 AM. He had no reaction after 2 hours and was given aspirin, 162 mg, at 10:30 AM. At 12:30 PM, the patient began to have a mild flare of hives, with a few lesions seen on his neck. He was given cetirizine, 10 mg, and the hives resolved in 30 minutes. The next day he was given aspirin, 162 mg, at 8:30 AM. At 11:15 AM he was given aspirin, 325 mg, and remained asymptomatic. The patient was advised to continue with the aspirin, 325 mg/d, and cetirizine, 10 mg nightly. Since the patient's desensitization, his dose of aspirin was reduced from 325 to 81 mg/d after 2 months by his cardiologist, and he is taking cetirizine only as needed. In the 5 months after his desensitization, he had 2 mild urticarial outbreaks that readily resolved with cetirizine treatment.

Aspirin desensitization has been proved to be a safe and effective office procedure,⁴ but its use in patients with aspirin-induced urticaria remains controversial. Grzelewska-Rzymowska et al⁵ and Asad et al⁶ demonstrated that patients with urticaria or angioedema reactions solely related to aspirin could be desensitized in a few days. Wong et al² studied 11 patients, most of whom had acute coronary syndrome and a history of aspirin-induced urticaria and angioedema. They underwent a rapid desensitization protocol, which was successful in 9 patients. This illustrates the possibility that desensitization is possible in single drug-induced urticaria/angioedema.

Simon³ studied aspirin desensitization in patients with a history of chronic urticaria. He desensitized 25 patients to aspirin, 650 mg. However, within 1 day of desensitization, the hives flared. This outbreak was severe, and prednisone was administered to adequately control it. Further aspirin desensitization attempts after the initial

trial only triggered more exacerbations of the hives. Our patient experienced acute urticaria with desensitization but was controlled with oral antihistamines.

The present case demonstrates that aspirin desensitization may be possible in patients with a history of aspirin sensitivity and CIU. We should emphasize that this patient may represent the milder end of the spectrum in that he was responsive to a histamine₁ antihistamine and the autologous serum skin test result was negative. Further studies with more patients are needed to better characterize the possibility of aspirin desensitization in this difficult-to-treat population.

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EMPOWER YOUR PATIENTS: SAVE LIVES

Epinephrine is the initial drug of choice in anaphylactic reactions. Some patients, such as those allergic to foods, latex, or Hymenoptera venom, are at such a high risk for systemic reactions (SRs) that they should always carry an epinephrine autoinjector. Proper treatment depends on the availability of epinephrine, knowledge of indications, and accurate use of the device when needed.¹ Several studies^{2–4} have shown that many patients do not always carry their epinephrine device, are deficient in their knowledge of indications for use (84% of parents), and cannot use the device correctly (63%). One survey¹ of patients' parents showed that training, previous use of epinephrine, and empowerment improved comfort with parental administration of epinephrine autoinjectors. In the severe anaphylactic reactions that have occurred, a delay in administration, inaccurate use, and lack of availability of epinephrine have been cited as contributing to the severity of the reaction.⁵ Physicians are already encouraged in practice parameters to educate patients on the importance of always having an epinephrine autoinjector available.^{1,5} Education by the physician or team is typically achieved using a training device,^{1,3,6,7} but patients may still fear the needle and medication from the autoinjector itself. A survey by Kim et al¹ showed that parental comfort was not affected by their child's previous

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anaphylactic reaction but was correlated with previous epinephrine administration.

One brief correspondence in the *Annals*⁸ described one physician's experience with demonstrating the use of a live epinephrine autoinjector on an asymptomatic, nonreacting patient. Although we believe that the use of medication, especially epinephrine, when not clinically indicated is completely inappropriate and unsafe, we recognize from previous literature that epinephrine autoinjector trainer education alone often falls short of addressing patients' fears of live epinephrine use.

In the SR emergency cart at the Department of Allergy and Immunology, Wilford Hall Medical Center, we now stock epinephrine autoinjectors (EpiPen and EpiPen Jr) in addition to predrawn syringes of epinephrine. The physician of the day (POD) who responds to an SR now has the discretionary option of using the EpiPen. In addition, if the patient is identified to be at high risk for future reactions (food, latex, or venom), hemodynamically stable, and willing, the POD may elect to guide the patient through self-administration under direct physician supervision. We briefly describe a case highlighting the importance and impact of this intervention.

In response to an SR alarm, the POD went promptly to the clinic treatment area, where a mother was found consoling her 2-year-old daughter with a history of peanut allergy. The child had completed peanut skin testing and within 5 minutes had a positive wheal and flare, with generalized urticaria and facial swelling. A focused history described a child with peanut ingestion who developed diffuse urticaria on 3 separate occasions since the age of 11 months. The mother had never administered EpiPen, but the child had received it during a previous emergency department visit. Previously, her primary allergist had demonstrated use of the EpiPen with the trainer at their initial appointment. In less than 60 seconds, the POD elected to use the EpiPen Jr option and asked the mother if she was willing to administer the EpiPen Jr to the child herself with supervision and assistance.

The mother was nervous but willing. We helped position the child on the mother's lap. The POD quickly reviewed the correct use of the EpiPen Jr and then handed the mother the device. Under the POD's supervision, the mother used the autoinjector correctly and was praised for a perfect injection. Within 5 minutes, the child's urticaria began to resolve, her eye swelling resolved, and her affect improved dramatically.

Shortly afterward, we again reviewed use of the EpiPen Jr with the mother. She admitted to having felt intimidated by the device. She related that while she had been instructed by her primary allergist on the indications for use of EpiPen Jr and had been allowed to use the trainer, she was still uncertain whether she could use the device if the time came. After this day's experience, she said she felt very comfortable with its use and extremely confident that if her child unintentionally ingested peanuts in the future she would be ready to administer the EpiPen Jr.

A MEDLINE literature review finds no previous study of in-clinic administration of epinephrine autoinjectors on patients, parental or provider perceptions of comfort, and likelihood of future administration. What better opportunity for training high-risk patients for future SRs is there than the SR that occurs in your office? This case exemplifies the importance of empowering our patients through real-time physician education.

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SUCCESSFUL ADMINISTRATION OF CYTARABINE IN A 16-MONTH-OLD GIRL WITH ACUTE MYELOGENOUS LEUKEMIA AND CYTARABINE SYNDROME

We describe a 16-month-old girl with acute myelogenous leukemia who developed cytarabine syndrome during intravenous cytarabine treatment. She subsequently successfully completed a 5-day course of high-dose cytarabine after 2 days of premedication with methylprednisolone, diphenhydramine, and ranitidine.

Cytarabine syndrome was initially described by Castleberry et al¹ in 1981 as observed in 6 children undergoing treatment with cytarabine alone. The symptoms included fever, malaise, joint or bone pain, maculopapular rash, conjunctivitis, and chest pain. Treatment with corticosteroids 1 day before administration of cytarabine prevented the recurrence of symptoms. Ek et al² reported that pretreatment with corticosteroids decreased the incidence of fever in pediatric patients receiving high-dose cytarabine. The package insert for injectable cytarabine states, "Corticosteroids have been shown to be beneficial in treating or preventing [cytarabine] syndrome. If the symptoms of the syndrome are deemed treatable, corticosteroids should be contemplated as well as continuation of therapy with cytarabine."³ However, in these 3 references and in a review of the English-language literature, specific dosing and timing of the premedication protocol are not given. We report a successful protocol of premedication to prevent the occurrence of cytarabine syndrome.

A 16-month-old, white girl diagnosed as having acute myelogenous leukemia received the first induction of high-dose intrathecal and intravenous cytarabine, etoposide, and daunorubicin without incident. One month later, after the second dose of the second induction with intrathecal and intravenous cytarabine, she developed intermittent fever. Vancomycin and meropenem were prescribed empirically for suspected infection. A day later, the fever persisted and she developed hypotension. Gentamicin was added. She developed a raised erythematous rash during the gentamicin infusion,

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