A case-based update

"Why is my asthma getting worse?"

Karen Binkley, MD, FRCPC

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\mathcal{P}	resentation: History of episodic, severe asthma exacerbations ove
	the past few months; asthma was originally diagnose
	when she was a teenager.
√	Sandra had never been to the emergency department (ED) for he
	asthma until three months ago; since then, she's visited the ED three
	separate times.
	At each ED visit, asthma was rapidly controlled with a short course of
	systemic corticosteroids and additional bronchodilators.
	Asthma is generally under good control with inhaled fluticasone
	125 μg.
	She requires rescue short-acting beta 2 agonist once or twice a month.
	She has no exposure to animals.
	None of her asthma attacks were associated with urticaria, nausea
	vomiting, diarrhea, abdominal cramping or hypotension and the episode
	occurred approximately four weeks apart.
√	The last episode occurred at the onset of Sandra's menstrual cycle. Upon
	questioning, she recalls taking ibuprofen for dysmenorrhea at the onset of
	her menstrual period; Sandra realizes episodic exacerbation
	requiring ED visits all occurred after taking ibuprofen for dysmenorrhea
√	History is otherwise unremarkable.
√	She takes no medications and has no known drug allergies.
V	Review of systems reveals increasing nasal congestion over the past year
	for which she has not sought treatment.
/	Physical exam reveals swollen, pale inferior nasal turbinates. Chest is clear.
V	Rest of exam is unremarkable.

Answer: ASA/NSAID sensitivity

Acetylsalicylic acid/non-steroidal anti-inflammatory drug (ASA/NSAID) sensitivity is the eventual diagnosis, although there appears to be associated asthma and rhinitis. Nasal polyps are not observed on physical exam, but polyps can occur higher in the nasal cavity and, therefore, be obscured by the swollen inferior turbinates.

Sandra is referred to an allergy specialist. Reactions to ASA and NSAIDs are not immunoglobulin (Ig) E-mediated, so skin testing is not appropriate.

Sandra is interested in continuing intermittent use of NSAIDs for dys-

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menorrhea. She therefore elects to undergo testing. Appropriate testing for ASA/NSAID sensitivity involves a graded oral challenge performed in hospital, which is arranged by the allergist. Oral challenge confirms ASA/NSAID sensitivity.

Although desensitization is sometimes possible, Sandra elects not to undertake this, as it would entail using an NSAID on a daily basis, without interruption. Instead, she decides to avoid all ASA and NSAIDs and is given written instructions for a med-

ical identification bracelet to state "allergy to ASA/NSAIDs/ketorolac."

Sandra uses acetaminophen with codeine for her menstrual cramps, but is instructed to keep doses of acetaminophen < 1,000 mg/dose. Higher doses of acetaminophen can trigger reactions in ASA/NSAID-sensitive individuals.

For her perennial rhinitis, allergy skin tests are performed and are negative. This supports the presumptive diagnosis of rhinosinusitis \pm nasal polyposis associated with asthma and ASA/NSAID sensitivity, which is often independent of IgE-mediated mechanisms. An intranasal corticosteroid to reduce

the non-IgE-mediated inflammation is effective in controlling Sandra's symptoms. **D**

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