Welcome to the FIT Board Review Corner, prepared by Timothy Chow, MD and Christopher Foster, MD, senior and junior representatives of the College’s Fellows-In-Training (FITs) to the Board of Regents. The FIT Board Review Corner is an opportunity to help hone your Board preparedness.

Review Questions

Allergy and Immunology Review Corner: Middleton’s Allergy Principles and Practice, 8th Edition

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Chapter 80: Hypersensitivity to Aspirin and Other Nonsteroidal Anti-Inflammatory Drugs

Prepared by: Rebecca Koransky, MD

1. Aspirin Exacerbated Respiratory Disease (AERD) describes a clinical syndrome with three features - the “ASA triad.” Which of the following choices below is not included in this triad?
   a. Chronic rhinosinusitis with polyps
   b. Asthma
   c. Increased pulmonary infections
   d. Hypersensitivity reactions to aspirin and other cross-reacting NSAIDs

2. Large amounts of infiltrates of what cell are most often found in the upper and lower airway mucosa of patients with AERD?
   a. Neutrophils
   b. Lymphocytes
   c. Macrophages
   d. Eosinophils

3. Patients with AERD have abnormal arachidonic acid metabolism. Which of the following is seen in patients with AERD?
   a. Decreased production of Prostaglandin E2
   b. Decreased production of leukotrienes
   c. Decreased production of lipoxin A4
   d. Increased production of all anti-inflammatory prostaglandins

4. What allele has been identified as a genetic marker for AERD in Polish and Korean populations?
   a. HLA DQB1*0301
   b. HLA DPB1*0301
   c. HLA DRB1*0301
   d. HLA AB1*0301
5. AERD develops in a distinctive pattern. What is the usual order of symptom development?
   a. Rhinosinusitis with polyps, asthma, aspirin hypersensitivity
   b. Aspirin allergy, rhinosinusitis with polyps, asthma
   c. Asthma, aspirin allergy, rhinosinusitis with polyps
   d. Aspirin allergy, asthma, rhinosinusitis with polyps

6. How do you definitively diagnosis AERD?
   a. Clear history of adverse reaction to aspirin
   b. Improvement of asthma when stopping aspirin
   c. Identifying nasal polyps in at risk patients on exam
   d. Aspirin provocation challenges

7. Aspirin desensitization can be used a treatment option for some patients. Desensitization to which dose of aspirin is recommended to maintain cross-desensitization to any dose of all NSAIDs?
   a. 81mg
   b. 162mg
   c. 325mg
   d. 650mg

8. Which NSAID hypersensitivity likely involves COX-1 inhibition?
   a. Fixed drug eruption
   b. Multiple NSAID induced urticaria
   c. Single drug induced urticaria
   d. Single drug induced anaphylaxis

9. A patient presents with urticaria to multiple NSAIDs. What is the next step in management?
   a. Oral challenge with non-COX-1 inhibitor
   b. Confirmatory oral challenge with COX-1 inhibitor
   c. Desensitization to preferred NSAID
   d. Avoid all NSAIDs

10. A patient presents with angioedema to a single NSAID. What is the next step in management?
    a. Oral challenge test with chemically unrelated NSAID
    b. Confirmatory oral challenge with same NSAID
    c. Oral challenge with non-COX-1 inhibitor
    d. Desensitization
Answers:

1. C. Page 1296. Patients with AERD, or Samter disease, usually present with chronic rhinosinusitis with polyps, moderate to severe asthma, and hypersensitivity reactions to aspirin and other cross-reacting NSAIDs.

2. D. Page 1298, 1301. The pathogenesis of AERD includes development of chronic inflammation of the upper and lower airway mucosa. Abundant amounts of eosinophils are found in mucosa of patients with AERD.


4. B. Page 1299. This allele was identified in studies of a Polish and Korean population. Patients with this allele showed lower FEV1 and high prevalence of rhinosinusitis with nasal polyps.

5. A. Page 1301. Nasal symptoms usually start by middle age and asthma develops a few years later. Aspirin hypersensitivity develops last - usually manifested as bronchospasm, rhinitis, and ocular injection.

6. D. Page 1301. “The diagnosis of AERD can be definitively established only through aspirin-provocation challenges”. Challenges can be oral, inhaled, nasal, or IV. Controlled oral challenge with aspirin is the gold standard.

7. C. Page 1304. The target dose of desensitization depends on the diseases underlying the aspirin desensitization. The target dose for cardiovascular disease prevention is 81mg, the target dose to maintain cross-desensitization to all NSAIDs is 325mg, and the target dose for AERD patients is 650mg twice daily.

8. B. p. 1297, Table 80-1. Cox-1 inhibition is the likely mechanism in urticaria/angioedema induced by multiple NSAIDs. Single drug induced urticaria and anaphylaxis are IgE mediated. A fixed drug eruption is a form of delayed type hypersensitivity.

9. B. p. 1306, Figure 80-3. The first step is a confirmatory oral challenge with a COX-1 inhibitor. If positive, then an oral challenge with a non-COX-1 inhibitor should be done next.

10. A. p. 1306, Figure 80-3. The first step is an oral challenge test with a chemically unrelated NSAID. If positive, patient should be treated as a reactor to multiple NSAIDs.