Case Study 6: Beauty and a (Bacterial) Beast

Jenny had never been happy with her nose, and she had saved for three years for the surgery. She consulted a plastic surgeon, and scheduled surgery for summer vacation. A few days after surgery Jenny was changing the dressings of gauze on her nose when she noticed increased swelling and pain at the incision site. By the next day she had a fever above 100° F. Jenny went back to the surgery clinic, where they took a swab of pus from the infected area for culture and prescribed an antibiotic. The next day the lab called to report that Jenny had a *Staphylococcus aureus* infection. The surgery and the nasal packing with gauze provided both an area for bacterial proliferation and an environment that contained air pockets giving the bacteria necessary amounts of oxygen for growth.

*S. aureus* is a gram positive, nonmotile, catalase positive, coccus found on the outside of the body, especially around the nose. About 30% of the population carries *S. aureus* at any particular time, and about 2/3 of people are at least occasional carriers. The bacteria induce localized inflammation that causes capillary endothelial cell damage and gives the bacteria access to the circulation. *S. aureus* produces an antiphagocytic capsule and surface adhesins. In addition to catalase, it also secretes protease, lipase, and hyaluronidase that destroy tissue, and coagulase that converts fibrinogen to a fibrin clot inside which the bacteria can grow. Many strains of *S. aureus* produce exotoxins and some strains are antibiotic resistant. Fortunately for Jenny, the *S. aureus* infecting her nose did not produce toxic shock syndrome toxin or exfoliative toxin, and her infection responded quickly to antibiotics.

**QUESTIONS:**

1. List as bullets with brief descriptions and in approximate chronological order the steps in the innate response to a bacterial infection. Include both the cells and the molecules that are involved in the response to the bacteria and what each does.

   **Example:**
   
   - Bacteria penetrate the skin and mucous membrane barriers through the surgical incision and enter the deeper layers of tissue.
   - Bacteria begin to replicate at the site of infection.
   - At the site of infection, . . .

2. How do phagocytes eliminate pathogens? What group of pathogens are most easily phagocytosed and why?

3. What is inflammation? What are the four signs/symptoms and what causes them? What is the purpose of inflammation? How are leukocytes (neutrophils and macrophages) signaled where to leave the circulation and enter the infection site?

4. What is complement? In what three ways is complement activation initiated and what are the three functions of complement?
5. Of the virulence factors of *S. aureus* listed below, which promote innate immunity and which protect the bacterium from the immune system? Include in your answer the elements of innate immunity from your list above that are affected by each factor and how. (For example: X stimulates or inhibits phagocytosis, Y activates or blocks a certain activity of complement.)

- Gram positive cell wall: teichoic acid, thick peptidoglycan layer
- Capsule
- Adhesion proteins (adhesins)
- Surface sialic acid
- Catalase (breaks down H\(_2\)O\(_2\) to O\(_2\) + H\(_2\)O)
- Lipase, protease, hyaluronidase
- Coagulase

6. What are acute phase proteins and how do they enhance innate immunity?

7. What is endotoxin shock? How does it differ from inflammation? Was Jenny at risk of endotoxin shock with her Staph infection?

8. Suppose that instead of plastic surgery, Jenny had been bitten by an animal infected with rabies virus. Rabies virus binds to and enters skin cells, where it replicates to produce more virions. Infected cells have rabies virus peptides on their membrane Class I MHC. Compare the innate response to rabies virus at the site of the bite with the innate response to *S. aureus*. What factors would be the same and what would be specific to virus infections?

**Supplementary Materials:** Innate Immunity, Complement, Cytokines, Infectious Disease, Immunity Rules, Antigen

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