**Pseudomonas pneumonia**

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**Disease etiologic agent:**

*Pseudomonas pneumonia* is an infection caused by the microbe known as *Pseudomonas aeruginosa*. It is an opportunistic pathogen because it is capable of attacking its host while its immune system is compromised. It is uncommon for healthy individuals to become infected with this organism and is usually a hospital-acquired bacterium (2).

**Disease Transmission:**

The microbe is found on the skin of humans aka skin flora. The opportunistic characteristics of *Pseudomonas aeruginosa* responds to break down of the skin and enters the body of immunocompromised individuals (2). Catheters inserted into patients, medical equipment, and central lines are common objects contaminated with *P. aeruginosa* and help in passing the bacterium to patients (2).

**Reservoirs:**

Usual reservoirs of this pathogen are water, soil, agricultural plants, animals, and humans (3).

**Special Characteristics/ Taxonomy:**

*P. aeruginosa* is a gram negative rod measuring 0.5 to 0.8 micrometers by 1.5 to 3.0 micrometers. Almost all strains are motile by a single polar flagellum. It is intolerant to a wide variety of physical conditions including temperature. It is resistant to high concentrations of salts and dyes, weak antiseptics, and many commonly used antibiotics (4).

**Specific tests for identification:**

*P. aeruginosa* is obtained using sputum, blood, body fluids, feces, wounds, or lesion cultures. The bacterium produces a pyocyanin (blue-green pigment), fluorescein (yellow-green pigment), and/or a pyorubin (brown-red pigment). In vitro, *P. aeruginosa* typically resembles a pearlescent color and is often foul-smelling. To diagnostically confirm *P aeruginosa* must present with pyocyanin
production of fluorescein production as well as the ability to grow at 42 degrees C. 

_P aeruginosa_ is also identified by its gram negative rod-like appearance, a positive oxidase reaction, and an inability to ferment lactase (4).

**Signs and symptoms:**

Common signs and symptoms of _P. aeruginosa_ include shortness of breath, fever, chills, increases heart rate, decrease appetite, malaise, and systemic inflammatory response. Productive cough, sputum that may have a yellow-green pigment, is thick, and usually foul smelling.

**Pneumonia:** infection in the lungs symptoms include chills, fever, cough that is productive, and difficulty breathing.

**Bacteremia:** bacterial infection of the blood symptoms include fever, chills, fatigue, muscle and joint pains

**Folliculitis:** skin infection, symptoms include itchy rash, bleeding ulcers, and headache.

**Swimmer's ear:** ear canal infection: symptoms include swelling, ear pain, itching, inside the ear, discharge from the ear, and difficulty hearing.

**Eye:** symptoms include inflammation, pus, and pain (5).

**Historical information:**

Gessard first isolated _Pseudomonas aeruginosa_ in 1882 from soldier's wounds. The drainage from their wounds would stain their bandages with a blue-green pigment. In 1889, Charrin and Roger established evidence for acquired immunity in animals inoculated with _P. aeruginosa_ and the mice were able to withhold further infection. During the same year, Bouchard used cell-free filtrates from this organism to help create a vaccine that would stimulate non-specific immunity. Hitschmann and Kreibich first reported in 1897 the skin manifestation leading to septicemia by _P. aeruginosa._

**Virulence Factors:**

_P. aeruginosa_ has multiple virulence factors ranging from cell associated to extra cellular factors that allows it to survive in many conditions as well as cause pathogenesis. Has a single flagellum, also referred to as a biofilm. After colonization and entry into the body, a signaling cascade produces a high amount of
extra cellular virulence factors in the acute phase. The two hemolysis produced by P. aeruginosa act together to break down lipids and lecins along with tissue damage caused by the cytotoxins. The next stage after the acute stage is the chronic phase, however this stage can happen directly after colonization also. The chronic infection has yielding mutants, protects from host immunity, and has a low extracellular production of virulence factors, the tissue damage is mostly a result of the chronic inflammation process.

Control/Treatment:

Current recommended drug therapies for P. aeruginosa include two antipseudomonal drug combinations (1). Patients who are immunocompromised a beta-to prevent Pseudomonas pneumonia lactam antibiotic and aminoglycoside is advised for acute infections (1). The site of infection and how long the infection has been present also depends on the drug therapy used (1). Common antimicrobial medications are ceftazidime, cefepime, meropenem, imipenem, primaxin, zoysn, timentin, gentamicin, streptomycin, sulfate, tetracycline, and acetic acid (1). Control of Pseudomonas pneumonia involves proper technique of hand washing, overall hygiene of patient, isolation precautions and equipment procedures modified.

Prevention/vaccines:

As of date there is no available vaccine to prevent Pseudomonas pneumonia (7). Healthcare providers are advised to consistently follow universal precautions and are monitored to enforce this. Hospital workers should also follow CDC recommendations for inserting catheters. However starting antibiotic therapy prophylactically is not advised due to the increase in multi-drug resistance bacterium (7).

A study recently done on mice consisting of liposomal elements given intraperitoneal or intranasal (8). The goal of the study was to trigger a systemic and airway humoral response to Pseudomonas aeruginosa (8). The study resulted in the vaccines stimulating a production of IgG and/or IgA antibodies against immunogenic peptide from this pathogen. However, this experiment could not be evaluated but may be helpful in other research associate with P. aeruginosa vaccine. However vaccine research is still under development even now at this date. (8).

Global Cases/Outbreaks:
In Warsaw, Poland at a hospital there was 41 *P. aeruginosa* cases with PER-1 extended spectrum beta-lactamase. This clonal complex is normally seen in Turkey but has been identified in the far eastern countries and Europe. In Greece, there was a hospital outbreak of multiple strains of this pathogen, which carried two metallic beta-lactamase gene variants. *Pseudomonas pneumonia* is a common infection found in most hospitals throughout the world.

**Local Cases/Outbreaks:**

In 2006, University hospital located in San Antonio, Texas saw an increase in *P. aeruginosa* infections in its neonatal intensive care unit (NICU), which caused a study to be conducted with data from 2005 to 2007 (9). The NICU generally has a low incidence of this bacterium and has seen fewer cases since revision of control measures (9). The patients with this infection tended to be male and had received mechanical ventilation at one point. During the study, 23 patients were confirmed with *P. aeruginosa*. Only 13% of patients were antibiotic resistant and 30% of the patients died. In US hospitals, there are about 4 per 1000 discharge (0.4%) of *P. aeruginosa* infections. Approximately 10.1% of nosocomial infections result due to this bacterium. Also, this pathogen, 8% of surgical wound infections, and 10% of bacteremia infections (9).

**Works Cited**


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