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Pseudomonas aeruginosa as a pathogenic Organism

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Abstract

Pseudomonas aeruginosa, is a member of the Pseudomonadaceae family. *Pseudomonas aeruginosa* is an opportunistic pathogen commonly found in the environment mainly in soil and water, but is also regularly found on plants and sometimes on animals, including humans. *Pseudomonas aeruginosa* is an important pathogen in healthcare-associated infections. *Pseudomonas aeruginosa* cause a variety of infections such as chronic CF lung infection, acute septicemia, wound infection, urinary tract infection, corneal ulceration, endocarditis, and pneumonia. *P. aeruginosa* also has a large number of virulence factors such as adhesions exotoxin A, exoenzyme S, nan 1 and Las genes, Mucoid exopolysaccharide, Protease enzymes, Lipopolysaccharide, Pigments eg. pyocyanin, 1-hydroxyphenazine, pyoverdin, Phospholipase C, Rhamnolipid, Pili, Lipase, Histamine, and Leukocidin. During pathogenesis *P. aeruginosa* quorum sensing plays a critical role for survival and colonization by coordinating phenotypic alterations at early stages of infection. *Pseudomonas* skin and soft tissue infections can be destructive and can cause massive necrosis and gangrene. *Pseudomonas aeruginosa* is resistant to quinolones, tetracycline, chloramphenicol while It is sensitive to gentamicin. This information about *Pseudomonas aeruginosa* will help to a better understanding of infectious processes and will allow to develop new effective procedure for prevention and clinical treatment.

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Introduction

Pseudomonas aeruginosa, is a proteobacteria and member of the Pseudomonadaceae family. Discovered in 1882 by the French bacteriologist and chemist Carle Gessard, this gram-negative bacteria structure includes a 5-8µm by 1.5-3µm rod with single polar flagellum. *Pseudomonas aeruginosa* is an opportunistic pathogen commonly found in the environment mainly in soil and water, but is also regularly found on plants and sometimes on animals, including humans. *Pseudomonas aeruginosa* is an important pathogen frequently implicated in healthcare-associated infections (HAIs), particularly in critically ill or immunocompromised patients (Elizabeth and Vincent, 2010). The bacteria often produce the blue-green pigment pyocyanin, a redox-active phenazine, which is known to kill mammalian and bacterial cells through the generation of reactive oxygen intermediates (Pollack, 2000). *Pseudomonas* infections often have a characteristic sweet odor and have become a substantial cause of infection in patients with immune deficiencies (Govan and Deretic, 1996).

P. aeruginosa is nosocomial pathogen and Its infections is common in hospitalized patients, particularly those who are debilitated or immunocompromised such as in intensive care units, HIV-infected patients, particularly those in advanced stages are at risk groups. (Lowbury *et al*., 1970). The genus Pseudomonas consists of different species such as*P. aeruginosa* , *P. paucimobilis, P. putida, P. fluorescens*, or *P. acidovorans* (Euzeby, 1997). This study is conducted to identified the virulance factors involve in pathogenesis of *Pseudomonas aeruginosa*.

Pathogenesis of Pseudomonas aeruginosa

The pathogenicity of *P. aeroginosa* depends on its ability to produce different proteases and toxins and on its ability to resist phagocytosis (Khan *et al*., 2002). Pathogenesis of *Pseudomonas* is multifactorial and complex because *Pseudomonas* species are both invasive and toxigenic. There are 3 stages are bacterial attachment and colonization, local infection, and bloodstream dissemination and systemic disease . The importance of colonization and adherence is most evident when studied in the context of respiratory tract infection in patients that need complicate mechanical ventilation, such as those with cystic fibrosis (Pollack, 2000). *Pseudomonas aeruginosa* has been found to cause a variety of infections in clinical practice besides chronic CF lung infection, including common acute septicemia from burn or surgical wound infection, urinary tract infection, corneal ulceration (from wearing contact lenses), endocarditis (caused by intravenous drug use, etc.), and pneumonia (from use of ventilator and endotracheal tube) (Bodey *et al*., 1983).

Virulence factors

Pseudomonas aeruginosa possesses a variety of virulence factors that may contribute to its pathogenicity. *P. aeruginosa* also has a large number of virulence factors such as adhesions exotoxin A, exoenzyme S, nan 1 and Las genes (Van and Iglewski, 1998). The outer membrane proteins of *P. aeruginosa* OprI and OprL play important roles in the interaction of the bacterium with the environment as well as the inherent resistance of *P. aeruginosa* to antibiotics where the consequence of the presence of these specific outer membrane proteins that have been implicated in efflux transport systems that affect cell permeability (Nikaido, 1994).

As these proteins are found only in this organism, they could be a reliable factor for rapid identification of *P. aeruginosa* in clinical samples (Vos *et al*., 1997). Other Virulence factors which are responsible for its pathogenicity of this bacterium are Mucoid exopolysaccharide (alginate), Protease enzymes, Exotoxin A, Lipopolysaccharide, Pigments eg. pyocyanin, 1-hydroxyphenazine, pyoverdin, Phospholipase C, Rhamnolipid, Pili, Lipase, Histamine, Exoenzyme S and Leukocidin. (Ras GJ *et al*., 1990).

Extracellular toxins

P. aeruginosa virulence factors include a variety of extracellular toxins that could cause extensive damage to host tissues through their enzymatic activities (Bitter,2003). These factors play important roles in the acute infections.

Flagellum, pilus and alginate

There are various virulence factors of *Pseudomonas aeruginosa* which help in pathogenesis such as extra cellular toxins, exotoxins, adherence .Another group of virulence factors are the attachment and motility organelles, including flagella (swimming), pili (twitching), and extropolysaccharide (alginate) (Alex and Simon,1994).

Quorum sensing

During pathogenesis *P. aeruginosa* QS plays a critical role for survival and colonization by coordinating phenotypic alterations at early stages of infection (González and Keshavan, 2006). The progress of acute to chronic infection is critically influenced by QS-dependent gene expression. More than 10% of *P. aeruginosa* genes are regulated by QS. These genes are mainly involved in virulence factor production, motility, motility-sessility switch and biofilm development, antibiotic resistance mechanisms and the adjustment of metabolic pathways for stress responses (Barr *et al.*, 2015).

Pseudomonas aeruginosa in Burn wound infections

Systemic manifestations of burn wound sepsis may include fever or hypothermia, dis orientation, hypotension, oliguria, ileus and leukopenia. The organism also flourishes on moist skin. *Pseudomonas* is a common cause of hot tub or swimming pool folliculitis. Patients present with pruritic follicular, maculopapular, vesicular, or pustular lesions on any part of the body that was immersed in water (Armour *et al.*, 2007).

Addtionally, green nail syndromeis a paronychia infection that can develop inindividuals whose hands are frequently submerged in water. Secondary wound infections occur in patients with decubiti, eczema, and tinea pedals. These infections may have a characteristic blue-green exudate with a fruity odor (Ratjen *et al.*, 2010). *Pseudomonas* skin and soft tissue infections can be destructive and can cause massive necrosis and gangrene (Heal *et al.,* 2009).

Pseudomonas aeruginosa in Skeletal infection

The most common sites of involvement are the vertebral column, the pelvis, and the sternoclavicular joint. These sites are usually infected due to secondary seeding (eg. Bacteremia or UTI) (Osama *et al.*, 2017).

Pseudomonas aeruginosainRespiratory tract infections

P. aeruginosa is well known for its ability to establish permanent residency in the airways of cystic fibrosis (CF) patients, resulting in the recurrence of chronic lung infections, progressive decline in lung function and increased morbidity and mortality rates (Nixon *et al.*, 2001; Smith *et al.,* 1996).

Pseudomonas aeruginosa in urinary tract infections

Urinary tract infections caused by *P. aeruginosa* usually occur secondary to catheterization, instrumentation or surgery. Catheterization of the urinary tract is the major cause of nosocomial acquired-UTI by *P. aeruginosa* (Mittal *et al.*, 2009).

Pseudomonas aeruginosain Keratitis

P. aeruginosa is the leading cause of bacterial keratitis (Nagachandrikaa *et al.*, 2011), and occurs in patients with pre-existing ocular disease, in post-ocular surgery patients and in individuals who use contact lens. *P. aeruginosa* has been shown to adhere to the disrupted corneal epithelial cells, and internalize rapidly (Ramphal *et al.*, 1981). Contact-lens associated keratitis is mediated by the extended use of contact lens that has been shown to disrupt the epithelial surface of the cornea, causing cornea abrasions (Roberston *et al.*, 2007).

Pseudomonas aeruginosain Swimmers Ear infections

Otitis externa, commonly known as 'swimmers ear' and an inflammation or infection of the external auditory canal, due to prolonged exposure to moisture and or the insertion of foreign objects (e.g. cotton tips) (Wang *et al.,* 2005). It is well known that *P. aeruginosa* is the most common pathogen of otitis externa, strongly associated with swimming in contaminated recreational pools (Ninkovic *et al.*, 2008).

Antibiotic resistance to Pseudomonas aeruginosa

P. aeruginosa is intrinsically resistant to various classes of antibiotics like some beta-lactam antibiotics due to a chromosomally encoded Amp C

cephalosporinase and tetracyclines, chloramphenicol, macrolides, trimethoprim and sulfonamide due toless permeability and eflux pumps. Resistance to other antibiotics like carbapenems, aminoglycosides and fluoroquinolons can be acquired. These resistance mechanisms include mutations that influence expression of eflux pumps, mutations in target sites, membrane modification and expression of enzymes that inhibit or modify antibiotics. while it is sensitive to gentamicin (Jombo *et al.*, 2008).

Efflux pump mediated resistance

Another resistance attribute to the mechanisms of *P. aeruginosa* are its efflux pumps, proteins found in nearly all bacteria that function to deport antimicrobials. Efflux pumps can be categorized into five families. Major facilitator (MF) multidrug and toxic efflux (MATE) resistance nodulation-division (RND) small multidrug resistance (SMR) and ATP binding cassette (ABC). All these families have been studied and shown to be found within *P. aeruginosa*. However, of these pumps, RND is the most common in *P. aeruginosa* (Stover *et al*., 2000).

Laboratory Diagnosis of Pseudomonas aeruginosa

The media used for *Pseudomonas aeruginosa* are Cetrimide Agar, Mac Conkey Agar,blood agar (Akoglu and Gokce, 2012; Douraghi *et al*., 2014). *Pseudomonas aeruginosa* is biochemically identify by catalase test, oxidase test, citrate utilization test, indole test, triple sugar iron test, urease test, methyl red test, voges proskauer test and gel liquification (Prabhat *et al*., 2010).

Conclusion

*Pseudomonas aeruginosa*is a pathogenic organism it caused number of infection in human. Their pathogenicity is promoted by number of virulence factors such as adhesions, exotoxin A, exoenzyme S, nan 1 and Las genes, Mucoid exopolysaccharide, Protease enzymes, Lipopolysaccharide, Pigments eg. pyocyanin, 1-hydroxyphenazine, pyoverdin, Phospholipase C, Rhamnolipid, Pili, Lipase, Histamine, and Leukocidin.This information about *Pseudomonas aeruginosa* will help to a better understanding of infectious processes and will allow to develop new effective procedure for prevention and clinical treatment.

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