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A General Overview on Pseudomonas aeruginosa bacteria and its pathogenicity

Ahmed Sabah Al-Jasimee, Abbas Mayar Hezam*, Wurood Jasim Mohammed, Mohammed M Alkhuzaie, Zinah Abdulkadhim Oudah

College of Science, University of Al-Qadisiyah, Diwaniyah, Iraq

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Abstract

The first case of bacterial infection was recorded in 1862, while it was first isolated in 1882 from the scientist Gessard, who was called Bacillus pyocyaneus. The most common infections caused by bacteria are the first bacteremia in patients with serious burns, chronic lung injuries in patients with cystic fibrosis, and acute ulcerative keratitis in people who use contact lenses. The gastrointestinal tract is an important gateway for entry into the blood infection caused by bacteria, and the bacteria cause endocarditis, where the bacteria infects the heart valves from the direct invasion of the bloodstream, as it causes meningitis and brain abscesses, and it can invade the central organ The inner and nasal sinuses can also be accessed from a site far from the injury, such as the urinary tract. Other pathogenic infections caused by bacteria are pulmonary injuries, as bacteria are the most common disease associated with lung injuries. They are caused by bacteria Hospitalized lung with a mortality rate greater than 70%. Bacteria are a common cause and acquired by hospitals for urinary tract infections due to their ability to adhere to urinary epithelial cells in the bladder, as they cause cystitis and urinary tract infections. The percentage of deaths caused by bacteria can reach 50% due to many factors, including weak body defenses and bacteria resistance to anti-life as well as the production of bacteria, enzymes and external toxins.

*Corresponding Author

Name: Abbas Mayar Hezam
Phone:
Email: abbas.hezam@qu.edu.iq

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INTRODUCTION

Studies conducted in the United States of America through data collected from the Centers for Disease Control for the period 1990-1996 (1996) indicate that this bacterium is the second common cause of pneumonia acquired from hospitals (Nosocomial Pneumonia) 17%, and the third common cause of systemic infections Urinary system 11%, the fourth common cause of injuries to medical surgical sites (Surgical wound site) 8%, the fifth common cause of various clinical injuries 9%, and the seventh common cause of bacteremia 3% (Delden and Iglewski, 1998; Ehrlich, 2003). Human nurses, where research indicates that in most diagnostic laboratories, your PC has been isolated. RIA repeat loud and promised one of the most important three clinical isolates with both Staphylococcus aureus and Escherichia coli (Cowell, 2003; Abed and Salwan, 2017; Abed and Salim, 2019b).

The ferocity factors for Pseudomonas aeruginosa

Bacteria are multifactorial, including secretory and cross-linked, with the cell (Prithiviraj *et al.*, 2005).

The bacteria have the ability to produce a number of Proteases enzymes such as basal protease, Elastase, and Staphylolytic Protease (LasA), and protease IV in addition to producing the blood-sensitive and heat-stable enzyme (Heat-labile and heat-stable Hemolysin), and Phospholipase C, and many types of toxins, including exogenous enzymes T, S, U).

(Exoenzymes and exotoxin A (Nagano *et al.*, 2001; Pillar and Hobden, 2002) are all important virulence factors in keratitis, as many of these factors show tissue damage or damage The cornea when it is injected into the eye alone or through the bacteria that produce it (Zhu *et al.*, 2002; Mariencheck *et al.*, 2003).

Many other virulence factors of this bacterium contribute to corneal injury, including Glycocalyx, LipoPolySaccharide (LPS), exogenous toxins, proteases, whips and fringes, where the role of Glycocalyx is important in the attachment of bacteria to host cells and has a role in resisting the phagocytosis process, and also has a role In stopping the interaction of host immune cell receptors with antibodies linked to bacterial surface antigens (Lyczak *et al.*, 2000). As well as the bacteria have defensive antioxidant enzyme systems, among them are Superoxide Dismutase, Peroxidase, Catalase (Kim, 2003).

Firm factors associated with the cell

LPS contributes to bacterial pathogenesis, as its presence as a bacterial product increases the inflammatory response of the host in the cornea (Khatri *et al.*, 2002). It was found that the susceptibility of bacteria to in vitro infection in the eyes of animals does not stimulate when bacterial isolation has a deficiency in the production of complete LPS (Preston *et al.*, 1995).

Alginate is another conjugating agent, which is an external polysaccharide (a polymer consisting of Mannuronic and Glucuronic Acid), and it is in the form of a mucous membrane (Biofilm) in which bacterial cells are anchored, and thus it protects bacteria from the body's defenses such as cells From the ciliary movement of the respiratory canal and from the antibody and complement. The biofilm is a developing layer of the microscopic organism on the smooth surfaces and adheres to it by secreting polysaccharides and glycoproteins (Head and Yu, 2004).

Among the other factors associated with flagella is that these bacteria have three main types of flagella: (A1, A2, B), and it was found that the difference in the chemical composition of these types does not affect the virulence of bacteria. Studies indicate that flagella are among the bacteria that aid in adhesion, as they are able to bind to glycosphingolipids (GSL) receptors in a similar way to cilia (Hahn, 1997; Lyczak *et al.*, 2000).

The bacteria also have the ability to produce other adhesion factors associated with the surface that helps in the adhesion of bacteria to the epithelial cells and then contribute to the virulence of bacteria. Among these factors is the fourth type of cilia (Pili), which is responsible for 90% of the adhesion to bacteria. It was found that the tremor movement of bacteria by cilia has a role in the infection of the cornea (Zolfaghar *et al.*, 2003). Whereas, bacterial isolates (which do not contain cilia) have less harm in infection events in laboratory animals (Comolli *et al.*, 1999).

External virulence factors are cellular

Bacteria produce cytotoxin, which is a perforating protein, also called leukocidin, because of its effect on neutrophil cells, as it is toxic to eukaryotic cells and has a role in the sweep process (Geiser *et al.*, 2001).

Hemolysin (Hemolysin) is one of the important factors for the virulence of this bacterium, and it consists of several types: Rhamnolipid, Phospholipase and Lecithinase, which are observed to work synergistically to break down fats and Lecithin. Bacteria produce the enzyme Phospholipase C, which most studies indicate is an unimportant virulence factor in causing eye damage. This is due to the ability of mutant bacterial isolates (not producing this enzyme) to cause keratolytic infection.

The production of Siderophores (iron-bound compounds) is one of the mechanisms of bacteria to obtain iron. These compounds are produced by P. aeruginosa as Pyoverdine or Pyocheline. Because of their chemical composition, Pyoverdine is the main type, while Pyocheline is the least prevalent in the environment (Meyer *et al.*, 1996; Takase *et al.*, 2000).

Another external virulence factor of P. aeruginosa is exotoxin A. Its toxic effect on corneal cells was investigated when it was added externally to the eye, and it was found to be an important virulence factor in eye diseases.

Toxin (Exotoxin S) is one of the exogenous toxins of P. aeruginosa, which has the ADP-ribosylating activity of various eukaryotic proteins (Geiser *et al.*, 2001; Ewaid and Abed, 2017). It was found that 38% of the isolates of these bacteria isolated from ulcerative keratitis are productive of this toxin (Winstanley, 2005; Ewaid *et al.*, 2019a,b).

Another type of exogenous toxin is Exotoxin T, which is more virulent in keratitis and its devel-

opment because it has the effectiveness of Anti-internalization from which the bacteria produced for it when present in the cornea can resist the phagocytic action by immune cells (Geiser *et al.*, 2001). One study indicated that bacterial isolates isolated from keratitis had a 49% T toxin effectiveness. This toxin has a key role in the development of keratitis (Krall *et al.*, 2000; Al-Zaidy *et al.*, 2019).

There are other types of exogenous toxins: ExotoxinU and ExotoxinY, which are also cytotoxic (Geiser *et al.*, 2001). These toxins are an important pathogen in bacterial infections.

One study indicates that the binding of P. aeruginosa in the cornea is stimulated by the proteases enzymes that can digest corneal proteins (collagens and protocols), resulting in severe corneal damage.

Bacterial isolates with high production of Proteases are able to analyze mucin, while non-producing of these enzymes cannot analyze mucus, which is the primary barrier of the cornea and protects the epithelial layers below it from bacterial attack, where each of the two enzymes has the Elastase IV Protease has greater susceptibility to mucus analysis than basal protease (Marquart *et al.*, 2005). Several studies also indicate that protease enzymes such as Elastase and basal protease have the ability to inhibit the functions of many cells that have a role in the immune response such as T-cells, Natural Killer (NK) PMN, (Twining *et al.*, 1993).

Keratitis caused by P. aeruginosa

Bacterial keratitis is one of the most important serious lesions in pathology due to the recurrence of its occurrence and its complications, and one study indicates that there is

One eye loses daily in the world due to the use of contact lenses, and P.aeruginosa is one of the most important factors that cause microbial keratitis, which leads to corneal ulceration, which if not treated can lead to a loss.

Al-Basr (Matsumoto, 2004), Incidence of microbial keratitis in the United States of America is estimated at 25,000 to 30,000 cases annually, and the cost of treatment is estimated from $ 15 million to $ 30 million, and studies indicate that P. aeruginosa is the most common cause of exacerbation of keratitis (Khatri *et al.*, 2002).

Staphylococcus epidermidis is among the natural fluoride isolated from the eyes of healthy people, while S. aureus, P. aeruginosa, and Streptococcus pneumonia are nurses who have virulence factors and cause serious eye infections (Aristoteli and Willcox, 2003).

A corneal puncture can occur in less than 24 hours during its infection with P. aeruginosa. Another complication is corneal opacities that lead to visual impairment. These complications are eliminated only in the case of a new patch of the cornea.

The inflammation of the cornea caused by P.aeruginosa is characterized by the infiltration of the inflammatory cells with rapid tissue damage and that this injury can lead to corneal perforation, intraocular injuries, iris damage, lens opacity, and finally it causes endophthalmitis (Twining *et al.*, 1993). The infection of the cornea with bacteria is rapid and often results in vision damage due to the scar that occurs in the cornea, and is usually characterized by liquid necrosis (Liquefative Necrosis) associated with severe ulceration and perforation of the cornea.

Both bacteria and host factors free from infiltration of inflammatory cells contribute to the multiplication and speed of progress of necrosis of the stroma as fluidized agents of stroma during injury (Thakur *et al.*, 2001). The bacteria produce various external virulence factors.

Capability and damage to eye tissue Pillar & (Cheng *et al.*, 1996; Hobden, 2002). It was found that bacterial isolates isolated from keratitis are highly effective for Elastase enzyme, basal protease, and ExoenzymeU which is cytotoxic. Many sources indicate that all isolated bacterial isolates from keratitis are all productive of Proteases, which play an important role in pathology. There is a study that indicates that bacterial isolation of virulence of the cornea can produce at least three different Proteases enzymes outside the living body so that these enzymes have the ability to cause rapid and comprehensive damage to the cornea of the rabbit.

Corneal infection of bacteria can be divided into three stages: adhesion and colonization of bacteria, followed by localized invasion, and finally, the spread of systemic disease (Fleiszig *et al.*, 1997). Because the affinity for bacteria to adhere to the epithelial cells of the healthy cornea is small, but the ability to adhere to it may be increased by exposure to hidden receptors, or when localized defenses are exposed as a result of tissue damage. Among the important factors that help bacteria adhere to are: cilia, Alginate, and Exoenzyme S. The latter plays an important role in invading the epithelial cells of the cornea outside the living body (Fleiszig *et al.*, 1997). Protease enzymes contribute to the adhesion process by analyzing Fibronectin, which helps to detect numerous receptors present below it on the surfaces of epithelial cells (Cowell, 2003). It has been found that there are special molecules found

in the cornea that increase the pathology of bacteria. Among these is the Intercellular adhesion molecule1 (ICM-1), where studies indicate that the presence of this molecule contributes to the severity of the response to the disease.

The severity of pathogenic bacteria, as it was found that mice that do not possess these molecules are less likely to be infected with the disease compared to mice that own these molecules.

The lacrimal membrane is the first barrier between the external environment and the epithelial cells of the cornea under the membrane, and therefore the breakdown of mucin (Mucin), which is the main component of the lacrimal membrane by bacteria, is one of the ferocity factors that the isolates that cause injury to the eye should possess, as it is the result of removing mucus From the cornea is the increased adhesion of bacteria to the surface of the cornea, as the isolates of bacteria that do not produce Proteases are not able to analyze mucus, that is, the ability of bacteria to consume mucus is associated with the ability of bacterial isolates to produce Proteases.

Immune changes associated with corneal infection in P.aeruginosa

Eye diseases of the bacterium (keratitis) include the following steps: first, bacterial settlement of the cornea, followed by stimulation of a number of cytokines such as TNF.

Interleukin-1 (IL-1) and finally the migration of Polymorphonuclear leukocytes cells (PMNs) to the cornea to get rid of the pathogen, the flow or entry of PMNs cells due to the induction of cytokines that attract these cells, that is, the damage resulting from keratitis by bacteria caused by the factors associated With bacterial settlement, this also results from the response of PMNs cells (Cheng *et al.*, 1996). As a result of the host's inflammatory response to bacterial corneal infection, the response is at the beginning of the entry of PMNs cells that cause corneal tissue breakdown, although it mainly analyzes the injury, as PMNs migrate from the lacrimal membrane and from the iris and peripheral vessels to the areas of the vascular cornea (Hobden *et al.*, 1999; Thakur *et al.*, 2004). The infiltration of PMNs cells is a central feature of bacterial pathology of the eye, and although these cells are necessary to get rid of living bacteria and result in the final recovery of the cornea, the survival of these cells in the cornea increases the severity of the stroma of the cornea (Thakur *et al.*, 2001).

During the bacterial infection of the cornea, complicated immune reactions occur, including inflammation, cellular and humoral immune response, and degeneration of stroma proteins. During the inflammatory process, leukocyte cells adhere to the endothelium and this adhesion is stimulated by cytokines such as TNF, IL-1. The inflammation caused by eye infection with the bacterium P.aeruginosa begins with a series of localized host reactions such as edema, infiltration of white blood cells, and angiogenesis. As a result of stromal dissolution, corneal ulceration and perforation occur, and this inflammatory response results as a result of the products of the bacteria secreted and paired with the cell. It is noteworthy that primitive cellular infiltration during infection consists initially of PMNs cells and then followed by macrophages (Dong *et al.*, 2000). One of the studies indicates that on the sixth day of the injury, a thickening of the cornea is observed due to edema and infiltration of inflammatory cells, where PMNs cells are spread throughout the cornea and accompanied by the formation of capillaries in the cornea, while on the ninth day of the injury, PMNs cells decrease and the phagocytic cells increase (Dong *et al.*, 2000; Abed and Salim, 2019a).

There are several studies conducted to compare the production of (IL-1) Interleukine-1 after bacterial infection of the cornea in mice sensitive to infection (which led to perforation of the cornea (and other mice resistant to infection). The cornea was cured), where high IL-1 was observed in both groups. IL-1 production peak one day after the injury, it was noted that IL-1 production remained high in the susceptible group while its productivity decreased in the infection-resistant group, and to study the importance of high IL-1 in mice sensitive to infection, IL-antibodies were used 1, which is used to treat infected mice, where antibodies are associated with IL-1, resulting in reduced injury The cornea, as reducing the severity of injury in the group of sensitive mice is associated with reducing the number of PMNs for the cornea, and studies also indicate that IL-2 has an important contribution to the infection of the cornea with bacteria, where there is a specific mechanism that leads to high expression of IL-2, which contributes to Invertible corneal tissue breakdown, as a result of which increased the entry of PMNs into the cornea, while less IL-2 eliminates bacterial infection with minimal damage to the cornea (Szliter *et al.*, 2006; Salim and Abed, 2017).

It was also found that IL-8 has a role in the development of injury, as it stimulates the attraction of PMNs cells, and attraction occurs after (10-8) hours after corneal damage, and corneal infiltration appears by inflammatory cells, where corneal opacities appear that are surrounding the affected tissues, and immunity begins to act with a specificity.

Dark edges that are surrounded by a number of lymphocytes that are part of the mucous membrane-associated with lymphoid tissue, as well as that the antibodies secreted to stimulate the phagocytic process of the bacteria (Schaefer, 2001; Abed *et al.*, 2019).

CONCLUSION

Bacteria are a common cause and acquired by hospitals for urinary tract infections due to their ability to adhere to urinary epithelial cells in the bladder, as they cause cystitis and urinary tract infections. The percentage of deaths caused by bacteria can reach 50% due to many factors, including weak body defenses and bacteria resistance to antilife as well as the production of bacteria, enzymes and external toxins.

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