LETTERS IN BIOMATHEMATICS

An International Journal

RESEARCH ARTICLE OPEN ACCESS

Exploring the Role of Toxa and Exos Genes in Pseudomonas Aeruginosa Infection Isolated from Burn

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Abstract

Background: A burn injury sources necrosis of tissue and raw area with serous exudation. The devitalized tissues and moist burn wound is favorable for the colonization and proliferation of microorganisms and following infection, therefore the latent risk of burn wound sepsis and septicemia persists until complete wound healing. The risk of infection is further potentiated due to immune disturbances caused by thermal injury.

Material and methods: 80 specimens have been collected from the burn patients in the period from the initial February to July/2025. This study shown that 50/80(62.5%) specimens Gram negative bacterial culture and 30/80 (37.5 %) from gram-positive bacterial culture.

Result: The identification of bacterial isolates was detected by cultural, biochemical test. The most common bacteria with highest incidence is *Pseudomonas aeruginosa* 27/50 (54%) from other types of gram negative and positive bacteria. The antibacterial resistance rates of the *P. aurogenosa* isolates to Clindamycin amounted 23/27(85.18%) and to Penicillin with 19/27 (70.37%). The moderate resistance rate was observed for Erythromycin 15/27 (59.25%) and the lowest resistance rate was observed for Ciprofloxacin 7/27(25.92%) and Amikacin 4/27(14.81%). Molecular detection for *ToxA and exoS* genes that revealed the presence of key virulence genes. The *ToxA* gene appeared in 20/27(74%) of isolates, indicating genetic diversity among strains, and *exoS* gene were possessed 24(81.48%) of isolates *P. aeruginosa*.

Conclusion: This study that show the patients with burn are at a higher risk of acquiring infections caused by multidrug-resistant Gram-negative bacteria and demonstrated that the lowest resistance rates of the *P. aeruginosa* isolates was observed for Cefuroxime Ciprofloxacin and Amikacin.

ARTICLE HISTORY

Received September 25, 2026
Accepted October 30, 2026

Keywords

P. aeruginosa, Burn, ToxA and exoS gene

Introduction

Burn injuries cause tissue necrosis and create an exposed surface with serous exudate. The necrotic and moist tissue in burn wounds provides a favorable environment for colonization and proliferation of microorganisms, leading to infection. Consequently, the risk of wound sepsis and septicemia persists until the wound has healed. The risk of infection is further increased by the immune system dysfunction resulting from the burn injury [1]. *P. aeruginosa* thrives on the moist surfaces of burn wounds and is highly pathogenic, particularly in immunocompromised burn patients [2]. Patients are often infected through cross-contamination between wounds. Pseudomonas infection is a common complication in burn patients and significantly contributes to increased morbidity and mortality rates [3]. The cell membrane of *P. aeruginosa*, like that of other Gram-negative bacteria, consists of an outer membrane, a peptidoglycan layer, and an inner cytoplasmic membrane [4]. In most

cases, it is composed of phospholipids with randomly distributed protein molecules. These bacteria also produce two chemically distinct types of lipopolysaccharide (LPS): one serotype (B-band) and the other a common antigen (A-band) [5]. Interfering with the biological targets of antibiotics is a common strategy used by bacteria to evade their effects. This is achieved by protecting these targets or altering their sites [6]. Therefore, mutations that modify target sites within *P. aeruginosa* contribute to antibiotic resistance, with quinolone sites being a prime example [7]. *P. aeruginosa* produces several virulence factors that play a significant role in the pathogenesis and toxicity of infections. In some cases, these bacteria develop virulence factors associated with both acute and chronic infections, depending on the strain. Phospholipase C, Exotoxin A, pili, and Exoenzyme S are key factors contributing to acute infections within the body [8].The major aim of the present study is to find whether the exploring the role of exoS and toxA genes in *Pseudomonas aeruginosa* infection isolated from burn.

Material and methods

Specimen collection

Clinical specimens of *P. aeruginosa* were obtained from patients at Al-Sader Teaching Hospital / Burns Center in the period from the initial February to July/2025. Specimens were firstly inoculated onto MacConkey agar and incubated for 24 hours at 37°C. Non-lactose-fermenting and pale colonies were selected on MAC agar. Subsequently, purified colonies were suspended in 0.03% Cetrimide medium and incubated at 37°C for 24 hours. Additional biochemical tests were also performed using single colonies cultured on Nutrient agar to support bacterial identification. All samples were seeded directly onto selective and differential media, including Blood agar Mannitol salt agar, and MAC agar, and then incubated at 37°C for 18–24 hours [3].

Antibiotic susceptibility

The disk diffusion method was used on pure cultures of bacterial isolates according to the guidelines of the Clinical and Laboratory Standards Institute (CLSI) [9]. The isolates were tested using a range of antibiotics to determine their antibiotic susceptibility patterns.

Molecular detection of virulence genes

Genomic DNA from bacterial isolates was prepared using the boiling method to extracted total genomic DNA, as described in [10]. It was concentrated using Nano-drop spectrophotometer at a wavelength of 260 nm. DNA solution properties were obtained with a purity within the range of 1.8 ± 0.2, which is the standard for DNA purity. PCR was performed to detect the presence of ToxA and exoS for the diagnosis of Pseudomonas aeruginosa, according to the manufacturer's protocol and usage software, Promega Biosystems. *ExoS* gene: Forward 5′-GCGGACCTGAATCGCGCTCT-3′, Reverse 5′-CGTACATCCTGTTCCTGGACC -3′ with amplicon size 500 bp [11] and *ToxA* gene: Forward 5′- CGAACTGGACGGTGGAGC-3′, Reverse 5′-CCTGTTCCTTGTCGGGGATG -3′ with amplicon size 535 bp [12].

The PCR reaction mixture was prepared under sterile conditions, and the components were mixed using a refrigerated microcentrifuge at 50 RCF for 5-10 seconds to ensure complete homogeneity. The amplification products were separated and analyzed by electrophoresis on 1.5% agarose gel, pre-stained with ethidium bromide, according to standard procedures [10].

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Result and Discussions

The study results showed that 80 specimens were collected from patients with burn, with 50 isolates (62.5%) being Gram-negative and 30 isolates (37.5%) being Gram-positive. P. aeruginosa that found constituted 27 (54%) isolates from the total Gram-negative bacterial isolates, as shown in Figure (1).

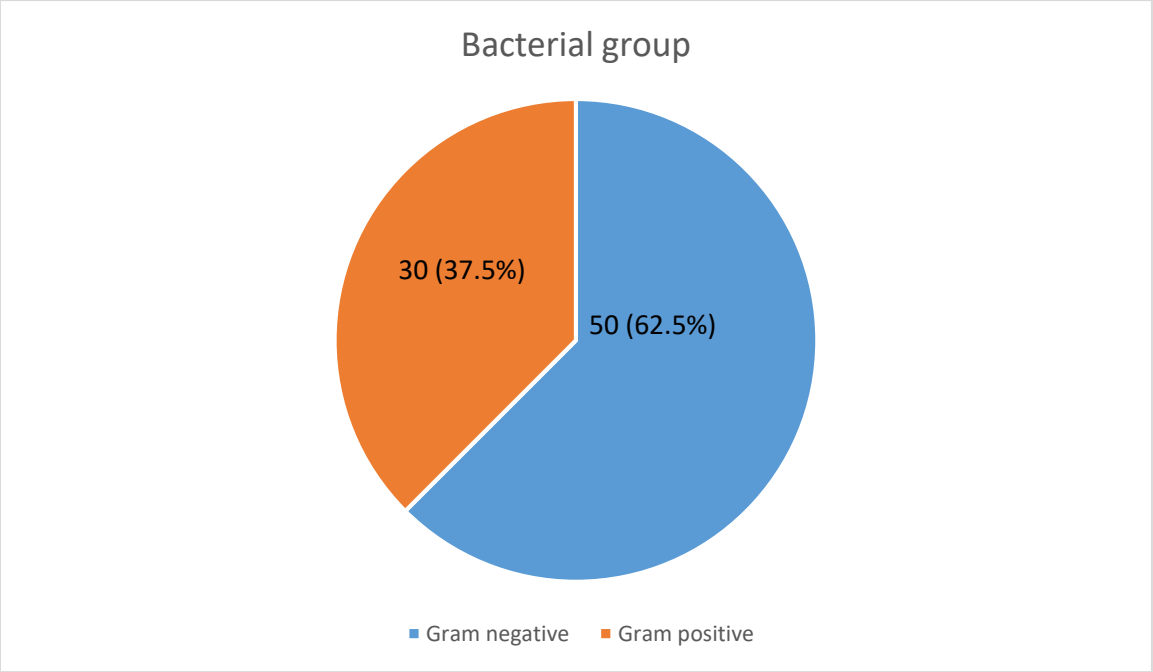


Fig. 1: Distribution of specimens according to group of bacteria

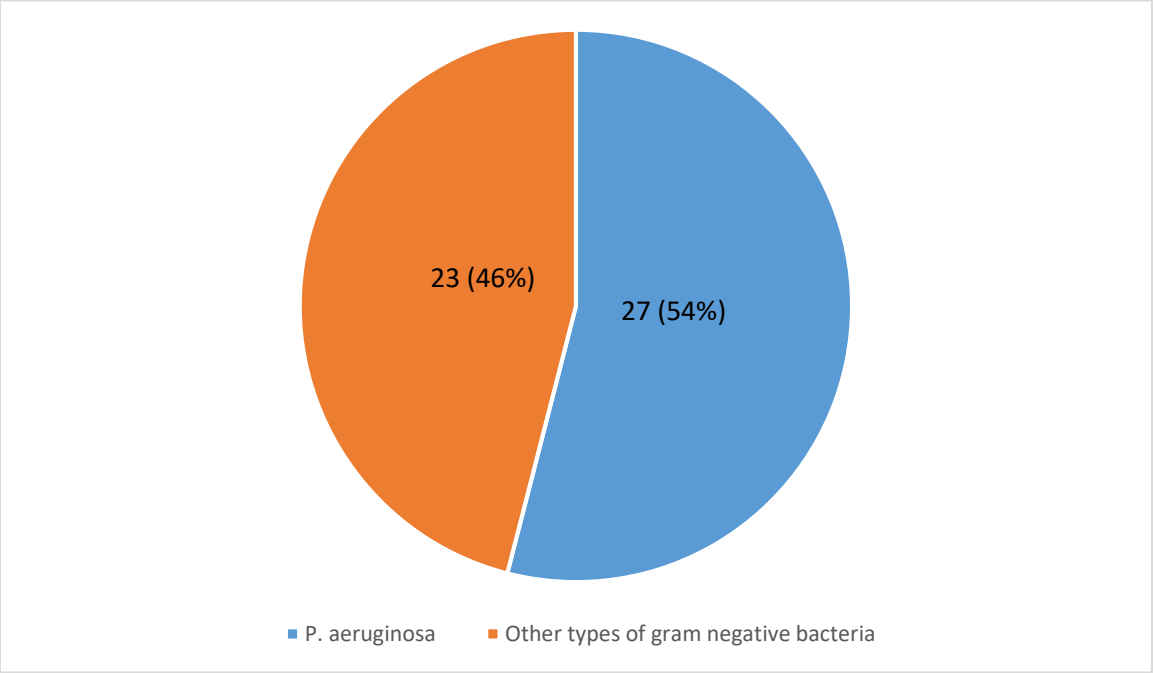


Fig. 2: *P. aeruginosa* distribution from gram-negative bacteria

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Results from this research displayed that Gram-negative bacteria were the greatest public pathogens in diseased burn wounds, manufacture up 62.5 percent of all isolates established. In dissimilarity, Gram-positive bacteria complete up the residual 37.5%. This dispersal is archetypal, as several researches have revealed that Gram-negative bacteria are extra public in burn repair situations due to their adaptableness to humid environments and their propensity toward MDR.

Surrounded by the Gram-negative bacteria, *P. aeruginosa* appeared as the greatest shared pathogen, secretarial for more than half of the Gram-negative isolates, definitely 54 percent. The current research sustenance those of Church et al. [13], who recognized this bacteria as a important risk factor for infections of burn due to its exclusive virulence mechanisms and capability to inhabit wet clinical environments. Gupta et al. [14] maintained the result by recognizing the founding of the presence of MDR and biofilms formation as dangerous elements preserving the pathogens ascendency in settings of burn.

The treatment of acute burn is strictly related with the presence of these bacteria. As well, the biological structures of the burn position stimulate the founding of robust microorganisms like *Pseudomonas aeruginosa*, whose mechanisms of resistance and suppleness environmental make elimination extremely interesting. Gram-positive which represented about 37.5% from all isolates were, which provisions the data providing by Norbury *et al*. on the significance of *S. aureus* in injuries of burn [15]. On the other hand, the quantity of Gram-positive bacteria described in this research was minor than in some previous researches, possible due to variations in the protocols for controlling infection, patterns for antibiotics prescribing, and local variations in the pathogen environment. In this study described the microbiological pattern is found the consistent with international trends, with Gram-negative bacteria mainly *P. aeruginosa* controlling the infection scenery in the units of burn. These showing highlight the continuing need for systematic microbiological investigation and the constant application of evidence grounded prevention of infection approaches in the burn management.

P. aeruginosa Detection

Samples were initially cultured on selective media, including MacConkey agar and blood agar. After incubation for 18–24 hours at 37°C, suspected pure colonies of Gram-negative bacilli exhibiting characteristic features were selected for further analysis.These colonies were re-cultured on blood agar plates and incubated again at 37°C to assess hemolysis patterns . Bacterial identification was based on colony morphology and molecular characteristics, compared to the standard description in Jean F. MacFaddin's Microbiology, as shown in Table 1.

Table (4.1) : *P. aeruginosa* detection by biochemical test

Bacterial species	Indole	Voges-Proskauer	Methyl Red	Catalase	H_2S production	Motility	Citrate	Urease
P. aeruginosa	-	-	-	+	-	+	+	-

The current study identified Pseudomonas aeruginosa isolates based on a combination of culture, morphological, and biochemical characteristics. These isolates exhibited characteristic growth on MacConkey agar as non-lactose fermenting colonies and displayed typical characteristics on Blood agar, including varying patterns of hemolysis. These results are consistent with the known microbiological characteristics of *P. aeruginosa*, which are characterized by its ability to grow in selective media, produce distinctive pigments, and exhibit characteristic colony morphology [16].

Biochemical tests showed that Pseudomonas aeruginosa isolates were negative for indole, phoges-Proscaur (VP), methyl red (MR), hydrogen sulfide (H_2 S) production, and urease, while being positive for catalase, motility, and citrate utilization. This biochemical outline mostly matches standard references, predominantly the MacFaddin Biochemical Identification System, designates these bacteria as oxidase-positive, non-fermenting, motile, capable of citrate utilization without producing indole or urease [9].

Indole, MR, and VP tests gave negative results for *P. aeruginosa* does not shadow butanediol fermentation or mixed acid fermentation pathways. On the contrary, the positive catalase test reproduces the bacteria capability to counteract reactive oxygen, characteristic of a virulence-related that subsidizes to its survival in environments hostile such as host tissues [3]. In addition, the positive motility test is qualified to the presence of tropical flagella, play a central role in the formation of biofilm and colonization, thus attractive the bacteria ability to persuade and persist infection [9].

The citrate positive analyze achieved in this research is constant with earlier reports, representative *P. aeruginosa* aptitude to citrate utilization as sole source of carbon. A number of researches have described comparable biochemical designs [17], who found undistinguishable biochemical reactions in clinical isolates of *P. aeruginosa*. Alternative research [18] established that *P. aeruginosa* isolates reliably show catalase positive, citrate utilization test and positive of motility, whereas negative for urease and indole tests.

However, slight variations in hemolysis patterns observed in the current study may be attributed to strain differences or environmental conditions, as previously suggested by [19]. Such variability is well documented and does not significantly affect the identification of *P. aeruginosa*, which relies more heavily on its biochemical and molecular characteristics.

Antibiotic Susceptibility Testing

The antibiotic resistance pattern of *Pseudomonas aeruginosa* isolates was determined using the Kirby–Bauer disk diffusion method. Results were interpreted based on the diameters of the inhibition zones according to standard guidelines [6]. Clinical isolates exhibited variability in their resistance to the tested antibiotics. All isolates showed highly resistance to Clindamycin amounted 23/27(85.18%) and to Penicillin with 19/27 (70.37%). The moderate resistance rate was observed for Cefepime 15/27 (59.25%) and the lowest resistance rate was observed for Ciprofloxacin 7/27(25.92%) and Amikacin 4/27(14.81%). As shown in figure 3.

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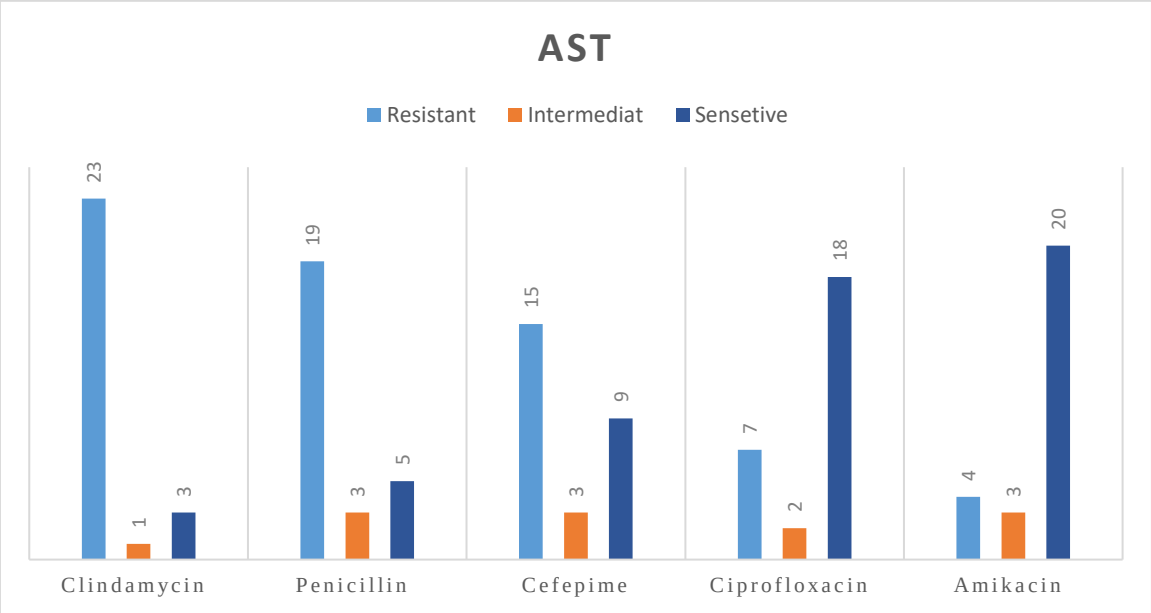


Fig. 3: prevalence of antibiotic resistances in clinical specimens of *P. aeruginosa*

The antibiotic resistance pattern of *Pseudomonas aeruginosa* isolates in the present study revealed considerable variability against the tested antimicrobial agents. The high resistance observed to Clindamycin (85.18%) and Penicillin (70.37%) is expected, as *P. aeruginosa* is intrinsically resistant to many β-lactam antibiotics and lincosamides due to low outer membrane permeability, efflux pump systems, and the production of β-lactamases [20]. Clindamycin, in particular, is generally ineffective against Gram-negative bacteria, including *P. aeruginosa*, which explains the high resistance rate, reported in this study.

The moderate resistance observed for Cefepime (59.25%) may indicate the emergence of extended-spectrum β-lactamases (ESBLs) or AmpC β-lactamase production among the isolates. Cefepime is a fourth-generation cephalosporin often used for treating *P. aeruginosa* infections; however, increasing resistance to this antibiotic has been documented globally due to selective pressure and inappropriate antibiotic use [21]. In contrast, the lowest resistance rates were observed for Amikacin (14.81%) and Ciprofloxacin (25.92%), suggesting that these antibiotics remain relatively effective against *P. aeruginosa* isolates in the current study. Amikacin, an aminoglycoside, is known for its stability against many aminoglycoside-modifying enzymes, which may explain its higher efficacy compared to other antibiotics [22]. Also, Ciprofloxacin, which belongs to the fluoroquinolone group, works by inhibiting the enzymes DNA gyrase and topoisomerase IV, and its relatively low resistance rate indicates limited mutations in the target genes or low expression of efflux pumps in the studied isolates.

The results in this study are consistent with numerous earlier researches, such as [17] who found the isolated of *P. aeruginosa* are highly resistance rates to beta-lactam antibiotics, as well as penicillin's, although the levels of resistance continued inferior to amikacin. Similarly, the study [23] that found the resistance increase to cefepime, qualified to the adaptive resistance mechanisms and beta-lactamase production. A different study [24] that showed the fluoroquinolones and aminoglycosides reserved their effectiveness against some clinical isolates, despite the global increase in MDR strains. The resistances variation detected in this study may be recognized to some factors, such as the hospital environment, local antibiotic use policies, horizontal gene transfer, and

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genetic diversity among the isolates. The comparatively little levels of resistance to both Ciprofloxacin and Amikacin suggest they might be active treatment selections.

Detection of *ToxA and exoS* genes by PCR

1- *ToxA* genes detection

The exotoxin A is a protein whose production controlled by *ToxA gene* responsible, was distinguished by polymerase chain reaction with specific primers, the results of the this study revealed that 20/27 (74%) isolates were positive for *ToxA gene*, as shown in Figure 4.

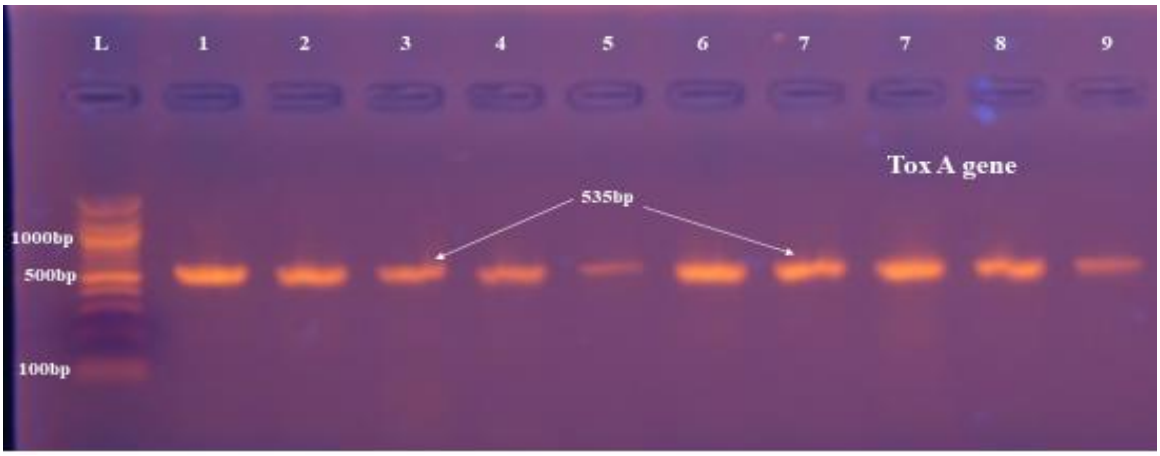


Fig. 4. The amplified *ToxA gene* showed through 1.5% agarose gel with Ethidium bromide-stained. L: DNA marker (100 bp ladder).

Our research showed that the 74% of *P. aeruginosa* isolates gave positive for the *toxA gene*, this gene encodes for exotoxin A. This toxin is vital for development disease by protein synthesis inhibited in host cells through ADP-ribosylation of elongation factor-2, eventually important for death of cell [25].

ToxA gene was found highly detection rate in this study specifies that the mainstream of isolates possess high virulence. The current study is agreement with several preceding studies that reported a high occurrence of this gene between clinical isolates of *P. aeruginosa;* for example, study [26] who found the presence of the *toxA gene* in more than 90% of isolates, representative that it is extremely preserved gene among pathogenic strains. Other local research [27] also shown pervasiveness rates fluctuating from 70% to 85%, which are actual close to the results of the present study. The presence of the gene in 74% of isolates indicates to genetic variation. Some studies have reported inferior frequency rates (60–65%), demonstrating that not all *P. aeruginosa* strains rely on exotoxin A as a main virulence factor, but may use another mechanisms such as the Type III secretion system, phospholipases or elastases [28].

The highly proportion of isolates covering the toxA gene in this research may be related with severity of infection and increased virulence, particularly in patients with immunocompromised. Previous studies have shown a significant association between the presence of virulence genes such as toxA and clinical outcomes, including increased tissue damage and accelerated disease progression [29].

2- Detection of *exoS* genes

The *exoS gene* is responsible for the production of the exoenzyme S enzyme and was detected using polymerase chain reaction (PCR) with specific primers. As shown in Figure 5, the results of the current study showed that 24 isolates (88.88%) of the bacterial isolates were positive for this gene.

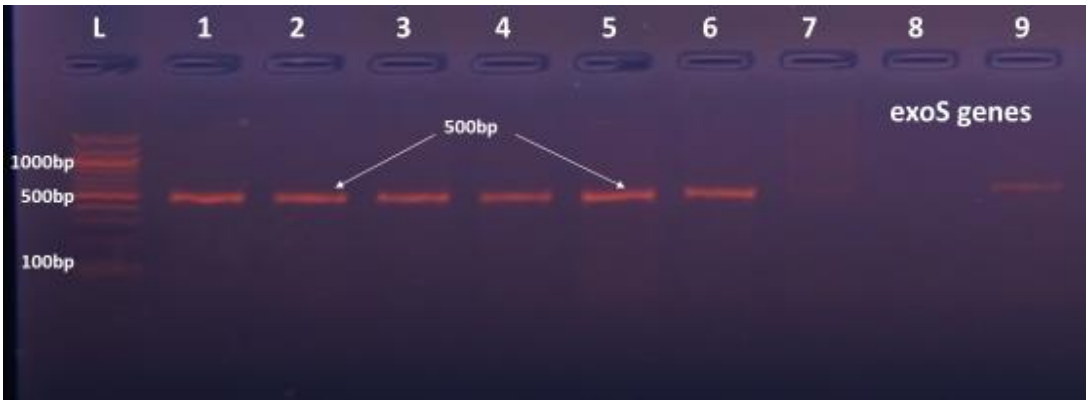


Fig. 5. The amplified *exoS genes* showed through 1.5% agarose gel with Ethidium bromide-stained. L: DNA marker (100 bp ladder).

Our research presented that 24 / 27 (88.88%) isolates of *P. aeruginosa* were gave positive results for the exoS gene, which this gene encodes to exoenzyme S and this play a key virulence factor connected with the type III secretion system (T3SS). This enzyme plays a central role in pathogenesis of bacteria by distracting intracellular signaling pathways passing through ADP-ribosylation activity, principal to dysfunction of cytoskeletal and reduced immune response [30]. The high pervasiveness of *exoS* gene in this research designates that furthermost of the isolates retain a high capability to cause illness, predominantly in relations to invasion of tissue and immune defenses evasion. These results are dependable with numerous earlier researches that showing the prevalent manifestation of this gene among clinical isolates. Such as the study [31] who found the *exoS* gene is usually identified in *P. aeruginosa* invasive strains, with frequency rates more than 80%. Similarly to [32] who found the detection rates reaching from 75% to 90%, outcomes that are mainly dependable with those of the our study. The attendance of the *exoS* gene often related with strains showing an invasive phenotype rather than existence cytotoxic. Conversely, strains comprising the *exoU* gene are naturally more cytotoxic, whereas *exoS* gene positive isolates incline to indorse bacterial spreading within the body and cause long-lasting infections [28]. The importance of difference dishonesties in prominence the latent clinical suggestions of the high occurrence of this gene in the our research, signifying that isolates are subject to causing chronic and invasive infections.

The genetic difference in exoS gene incidence across changed researches can be qualified to numerous factors, including sources of sample, geographical differences and the arrangement of the populations studied. Environmental factors and discerning pressures such as antibiotic use patterns can similarly play an important part in influencing the spreading of virulence genes amongst *P. aeruginosa* isolates [33].

Conclusion

This research established that *P. aeruginosa* isolates are consistent for identification using biochemical, culture, and molecular test. Antibiotic sensitivity designs exposed low sensitive to common antibiotics similar clindamycin and penicillin, whereas higher sensitive was detected to both ciprofloxacin and amikacin. Molecular investigates displayed a high occurrence of genes for virulence, the toxA gene detected in 74% and the exoS gene in 88.88% of the isolates, representative the high pathogenicity of this bacteria.

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