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Original Article

Unveiling the Barcodes of Biofilm Formation Genes (*algD*, *pslA*, *pslB*, and *pelA*) and Antibiotic Resistance Genes (*bla-OXA* and *blaCTX-M*) of *Pseudomonas aeruginosa* Strains Isolated from Wound Burn Infections in Iraq

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Abstract

Pseudomonas aeruginosa is a common opportunistic pathogen found in burn wound infections. Multi-drug resistance, coupled with its ability to form biofilms, makes treatment outcomes even more difficult to achieve. The molecular characterization of the biofilm genes of virulence and the determinants of extended-spectrum β-lactamase (ESBL) is vital concerning the epidemiology and clinical behavior of the circulating strains. The aim of this study was to create molecular barcodes for the P. aeruginosa isolates from burn wound infections in Iraq, focusing on the biofilm formation genes (algD, pslA, pslB, and pelA) and antibiotic resistance genes (blaOXA and blaCTX-M). Clinical samples were obtained from 70 patients with burn wounds. Selective media, the VITEK 2 system, and 16S rRNA gene sequencing were used to identify 20 isolates of P. aeruginosa. ESBL-encoding genes and biofilm-related genes were detected using conventional PCR. Ten isolates were selected to represent the set, and their partial 16S rRNA sequences were analyzed for phylogenetic relationships and submitted to GenBank. The findings showed that 100% of isolates had the biofilm-forming genes algD, pslA, pslB, and pelA. The blaOXA gene was present in all the isolates examined, and blaCTX-M was present in 19 out of 20 isolates (95%). Successful amplification of gene-specific fragments showed the widespread co-occurrence of biofilm and resistance determinants. The phylogenetic analysis showed the presence of close genetic relatedness of the isolates and P. aeruginosa reference strains. The prevalence of biofilm-producing P. aeruginosa and ESBL genes in reported cases from Iraqi healthcare facilities indicates the need for focused molecular-based/incision antimicrobial management.

Keywords: burn units; clinical microbiology; genetic surveillance; selective culture media; virulence profiling

Introduction

In clinical practice, complications arising from burn wound infections can result in extended occurrences of hospital stays, unanticipated delays in the healing process, and increased risk of fatalities. Among the many types of bacteria, *Pseudomonas aeruginosa* is frequently found in hospital burn wards and plays a significant role in contributing to the high rates of negative clinical outcomes, especially those that involve the development of infections that are systemically spread or that result in the development of sepsis [1]. The organism is recurrently isolated from burn units and has been consistently found to correlate with poor clinical outcomes. [2] Among the numerous types of bacteria, *Pseudomonas aeruginosa* is adaptive and survives in hostile settings, including low-nutrient environments, oxidative stress, and the presence of antimicrobial agents [1].

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Infection from burn injuries caused by P. aeruginosa in burn wounds has a unique characteristic that is the ability to create biofilms. The development of biofilms generates communities of microorganisms that are encased in gelatinous matrix materials, and this allows individual bacterial cells to be protected from the various immune responses and the therapeutic devices that are available to the host [3]. There are numerous genes that are related to the synthesis of sugar polymers, such as pslA, pelA, and related regulatory factors. These genes are essential to the biofilm development, as well as to the overall process of maturation and long-lasting persistence on the surfaces of the damaged skin [4]. Clinical studies have shown that isolates that are able to produce biofilms are much harder to eradicate, and this characteristic is found consistently in the case of chronic or recurrent infections of the wounds [5]. Antimicrobial resistance has simultaneously emerged with biofilm formation as a critical concern in P. aeruginosa infections related to burns. The detection of more than one type of extended-spectrum ß-lactamase (ESBL) genes, including blaOXA and blaCTX-M, has been associated with the diminished effectiveness of the first-line β-lactam antibiotics [6]. Genome studies showed the integrative presence of both virulence factors and resistance genes in the same strain, thus elevating the possibility of survival and spread in the hospital settings [7]. The presence of multidrug (MDR) and extensively drug-resistant (XDR) strains now poses a significant risk to the infection control measures in burn units [8].

The recent works have pointed out that biofilm formation and antibiotic resistance are not isolated phenomena, but rather a synergistic relationship that strengthens one another. Cells that are within biofilms exhibit unique gene expression that lends itself to the development of resistance, and resistant strains exhibit the ability to reinforce biofilms under antimicrobial stress [9]. This relationship calls for studies at the molecular level that tackle, at the same time, the virulence and resistance profiles in the clinical isolates.

Regardless of the regional evidence available, much of the data concerning the molecular features of P. aeruginosa associated with burn wounds in numerous low-and middle-income nations remains scant. Thorough molecular barcoding of the circulating strains is critical in determining local epidemiology and informing targeted treatment approaches and antimicrobial stewardship efforts [10]. Therefore, studying the dual presence of biofilm-related genes and resistance determinants offers valuable perspectives on the clinical and epidemiological relevance of P. aeruginosa in burn wound infection.

The aim of this study was to create molecular barcodes for the P. aeruginosa isolates from burn wound infections in Iraq, focusing on the biofilm formation genes (algD, pslA, pslB, and pelA) and antibiotic resistance genes (blaOXA and blaCTX-M).

Materials and methods

Patients (Cohort Study)

In this cohort study, we included a total of seventy patients who suffered from burn wound infections and consulted a dermatologist at the Dermatology Clinic, Marjan Teaching Hospital, Iraq, from January 2023 to May 2023. Our study population consisted of both male and female patients, and their ages ranged from 1 to 60 years. For each study participant, we completed a structured questionnaire that included the patient's name, age, gender, and clinical symptoms related to the case.

Molecular Detection

PCR, agarose, and a 100 base pair DNA ladder were acquired from the Promega Company (USA). For this study, all primers were ordered and synthesized from Macrogen Ltd. (South Korea). The bacterial genomic DNA isolation kit was from Promega Company (USA). The GeneJET PCR purification kit

was obtained from Thermo Fisher Scientific Company (USA).

Samples Collection

Using a sterile cotton swab, we transported soft cotton swab samples from the surface of the burn wound skin lesions of the participants. Without any delay, we transported all clinical specimens, which were contained in Amies transport medium and sealed cotton swabs, to the laboratory.

Microbiology Culture Media

Microbiology Media samples stored in Amies medium were enriched in sheep blood agar. From there, Pseudomonas selective agar (CFC) was applied to isolate clinical strains of Pseudomonas spp. from skin lesions of burn wounds. During the activation process, nutrient broth and nutrient agar were used for the overnight cultivation of the isolated bacteria to make bacterial pellets for genomic DNA extraction. The clinical strains were all stored at -80°C after the addition of glycerol at a final 50% (v/v) concentration. The agar media for this study were also made using Pseudomonas CHROMagar™ (Paris, France) and supplied from Oxoid–Thermo Fisher Scientific Company (USA).

VITEK 2 System and Clinical Strains Phenotypic Characterization

Cotton swabs with samples suggestive of *Pseudomonas aeruginosa* were enriched in sheep blood agar and incubated for 24 hours at 37 °C. The resulting colonies were used to inoculate Pseudomonas selective agar (CFC) and Pseudomonas CHROMagar™, and these were incubated for 24 hours at 37 °C. The colonies formed after this incubation were subjected to phenotypic characterization using the VITEK® 2 system.

Isolation of Genomic DNA

The genomic DNA from each bacterial isolate was acquired with a genomic DNA purification kit (Thermo Fisher Scientific, USA) based on the guidelines provided by the manufacturer. The extracted genomic DNA was tested for quality by performing agarose gel (1%) electrophoresis for a time period of 10 minutes, followed by visualization on a U.V. transilluminator (Labcompare Co., USA). The extracted genomic DNA was quantified by the use of a NanoDrop™ 2000/2000c (Thermo Fisher Scientific, USA).

Sequencing 16S rDNA Genes and Creation of a Phylogenetic Tree

For the analysis of 16S rDNA genes of the 10 clinical isolates, and to validate the identity of *10Pseudomonas aeruginosa* strains, a 16S rRNA gene fragment (668 bp) from each isolate was amplified by using the primer pair specific to *Pseudomonas aeruginosa*: F16S-P. aeruginosa (5′-TCAACCTGGGAACTGCATCC-3′) and R16S-P. aeruginosa (5′-CAGACTGCGATCCGGACTAC-3′).

Reaction mixtures for the PCR were made in a total of 25 µL containing 50 ng of genomic DNA, 12.5 µL of 2× PCR master mix (Promega, USA), and 0.2 µM of the forward and reverse primers. Using the Biometra thermal cycler (Germany), the PCR amplification was done under the following conditions: initial denaturation for 5 minutes at 95 °C, then 30 cycles of 1 minute of denaturation at 94 °C, 30 seconds of annealing at 55 °C, and 30 seconds of extension at 72 °C, followed by a final extension for 5 minutes at 72 °C.

After the completion of the polymerase chain reaction, the products were examined after the electrophoresis process and the addition of a 100 bp ladder, which happened on a 1.5% agarose gel. The PCR products were subjected to sequencing after purification with the GeneJET PCR purification kit (Thermo Fisher Scientific, USA). The same primer sets were used for the sequencing process, which was carried out by Macrogen Company (South Korea). Using BioEdit version 7.0, the sequences were edited and assembled. The sequences were edited, and the BLASTn tool, which is found inside the National Center for Biotechnology Information (NCBI) database, was used to analyze the sequences

for genetic similarity to the database. With MEGA software, the author performed a multiple sequence alignment and built a phylogenetic tree for the purpose of analyzing the evolutionary relationships between the isolates. The sequences for the 16S rRNA genes from this study were assigned accession numbers and deposited into the GenBank database.

Detection of Extended-Spectrum β-Lactamase (ESBL) Genes via PCR

Based on twenty P. aeruginosa clinical isolates, the genes blaOXA and blaCTX-M and the distribution of antibiotic-resistant genes were analyzed using conventional PCR with specified primers. The primer sets Fbla-OXA (5′-ATATCTCACTGTTGCATCTCC-3′) / Rbla-OXA (5′-AAACCCTTCAAACCATCC-3′) and Fbla-CTX-M (5′-CGCTTTGCGATGTGCAG-3′) / Rbla-CTX-M (5′-ACCGCGATATCGTTGGT-3′) were used to amplify the target gene fragments of 618 bp for blaOXA and 550 bp for blaCTX-M.

For each clinical isolate, two discrete PCR reactions were conducted. Each PCR mixture was prepared with 40 ng of genomic DNA, 0.3 µM of both primers, 12.5 µL of 2× PCR master mix (Taq, Promega, USA), and was brought to 25 µL with nuclease-free water. Amplification of PCR products was executed with a Biometra thermal cycler (Germany) under the following parameters: initial denaturation at 96 degrees Celsius for 3 minutes; denaturation at 94 degrees Celsius for 30 seconds for 30 cycles, followed by annealing at 57 degrees for blaOXA and 58 degrees for blaCTX-M for 45 seconds, and extension at 72 degrees for 30 seconds; and a final extension of 72 degrees Celsius for 3 minutes. 1.5% agarose gels were used to resolve the PCR products, which were compared against a 100bp DNA ladder to verify the sizes of the expected amplicons.

Detection of Biofilm Formation Genes through PCR (algD, pslA, pslB, and pelA)

Using conventional PCR and gene-specific primers, four biofilm-forming genes (algD, pslA, pslB, and pelA) were confirmed. Amplification occurred for partial fragments measuring 219 bp (algD), 230 bp (pslA), 220 bp (pslB), and 214 bp (pelA). For PCR reactions, 40 ng of genomic DNA, 0.3 µM of both forward and reverse primers, and 12.5 µL of 2× PCR master mix (Taq, Promega, USA) were combined, and a final volume of 25 µL was reached with nuclease-free water.

The PCR cycling conditions included an initial denaturation set to 96 °C for 3 minutes, followed by 30 cycles of denaturation set to 94 °C for 30 seconds, annealing at 57 °C for 45 seconds for algD and at 58 °C for pslA, pslB, and pelA, and an extension at 72 °C for 30 seconds. A final extension at 72 °C was set for 3 minutes. PCR products were examined on 1.5% agarose gel electrophoresis with a 100 bp DNA ladder to verify bands for the expected fragment sizes.

Results and discussion

Patient Distribution and Sample Collection

The current study, which took place from January 2023 to May 2023, included seventy patients with burn wound infections. Clinical specimens from all the affiliated patients were collected employing the use of sterile cotton swabs. All samples were promptly transported in an Amies transport medium, which was used to process samples upon arriving to the microbiology laboratory. No samples were discarded due to collection errors, transport failures, or contamination.

Isolation and Phenotypic Identification of *Pseudomonas aeruginosa*

Bacterial growth was noted in the majority of collected specimens after the first cultivation period in the sheep blood agar. Further selective cultures, with both Pseudomonas selective agar (CFC) and Pseudomonas CHROMagar™, established characteristic colonies of Pseudomonas. Presumptive Pseudomonas isolates were selected for additional identification based upon several distinct characteristics, which included colony morphology, pigment, and growth (Figure 1).

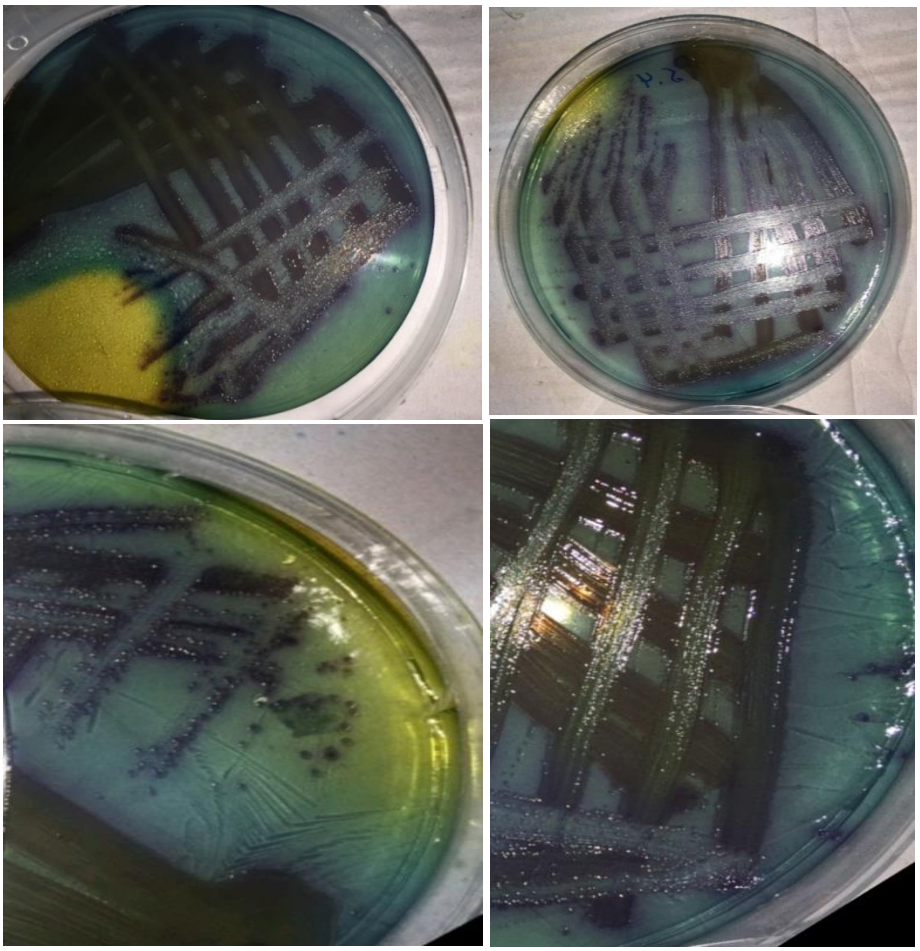

Figure 1: *Pseudomonas aeroginosa* on *Pseudomonas* CHROMagar

Of the seventy clinical samples, twenty isolates were, after phenotypic evaluation, identified as *Pseudomonas aeruginosa*. The VITEK® 2 automated identification system, which is used for species-level identification, confirmed this identification.

This study presents a thorough molecular analysis of *Pseudomonas aeruginosa* isolates from burn wound infections, focusing on the coexistence of the biofilm-associated genes and extended-spectrum β-lactamase determinants. Evidence shows burn environments as selective niches for highly adaptive P. aeruginosa strains capable of colonization and antimicrobial avoidance. Studies show burn wound isolates as having a higher burden of both virulence and resistance genes than isolates from other clinical sources, which shows the selective pressure of the tissue damage and immune suppression with concomitant antimicrobial treatment [11,12].

Extraction of Genomic DNA and Evaluation of Extract Quality

Genomic DNA was extracted from all twenty P. aeruginosa isolates. All samples were checked for DNA integrity and degradation through agarose gel electrophoresis. The extracted DNA showed no evidence of degradation and appeared as an intact high-molecular-weight DNA. A NanoDrop™ was used to analyze DNA concentration and purity ratios. The ratios were acceptable for subsequent applications to molecular biology.

Molecular Confirmation Using 16S rRNA Gene Amplification and Sequencing

Molecular confirmation was performed on 10 clinical P. aeruginosa isolates by 16S rRNA gene amplification. The LAS was used to visualize gel electrophoresis results. PCR amplification was positive for all 10, as evidenced by a fragment of 668 bp (Figure 2).

The isolates were confirmed as *Pseudomonas aeruginosa* by sequencing of PCR products. A NCBI BLASTn search was done to confirm the location of the P. aeruginosa strains. The phylogenetic analysis showed that the sequenced isolates all grouped within the P. aeruginosa sub-tree with respect to other described strains. This indicates all sequenced strains were closely related to each other. The phylogenetic relationships of the sequenced strains and the described reference strains are shown in Figure 3.

Identification of Genes for Extended-Spectrum β-Lactamases

Among the twenty P. aeruginosa isolates, the ESBL-encoding genes, blaOXA and blaCTX-M, were confirmed using conventional PCR. All isolates (100%) showed amplification for the blaOXA gene, which produced the expected 618 bp PCR product (Figure 4).

Gene amplification for blaCTX-M was observed in 19 of the 20 isolates (95%), for which a PCR product of 550 bp was generated. One isolate failed to amplify the blaCTX-M gene (Figure 5).

The extensive prevalence of ESBL genes confirmed through this study supports the already-established alarming potential resisting capability in burn wound-associated P. aeruginosa. Identifying all isolates with blaOXA and most with blaCTX-M corresponds with recent studies in burn units from varying regions of the world, reinforcing the embedding of β-lactamase genes in the commensal P. aeruginosa population. These resistance factors continuously progress the already alarming multidrug and pan-drug resistant specimens found within burn units, while also furthering the ineffectiveness of β-lactam class antibiotics [13, 15, 16].

Barcode Identification of Genes Responsible for Biofilm Formation

In all isolates examined, the genes algD, pslA, pslB, and pelA, which are associated with biofilm formation, were confirmed. Biofilm formation was evidenced by the amplification of the expected product sizes of 219 bp (algD), 230 bp (pslA), 220 bp (pslB), and 214 bp (pelA) in all isolates (Figure 6).

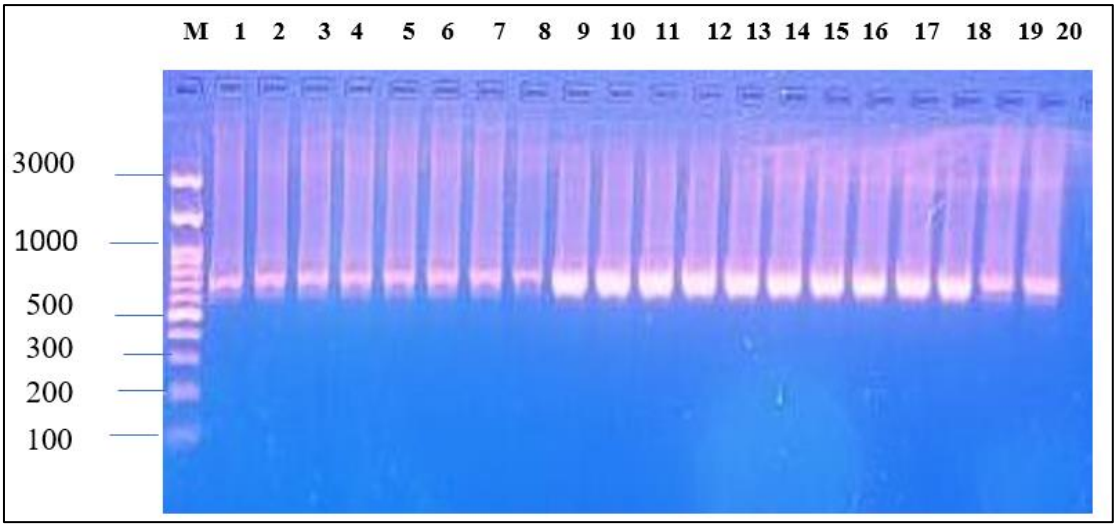

Figure 2 Agarose gel electrophoresis (1%) indicating partial amplification in16S r RNA gene specific for P. aeruginosa by PCR from 10 clinical strains of P. aeruginosa isolated from burn wound infection samples enrolled in this study. M: 100 bp DNA ladder. Lanes 1-19: PCR product (665 bp) of 16S rRNA gene partially amplified in 10 clinical strains of P. aeruginosa.

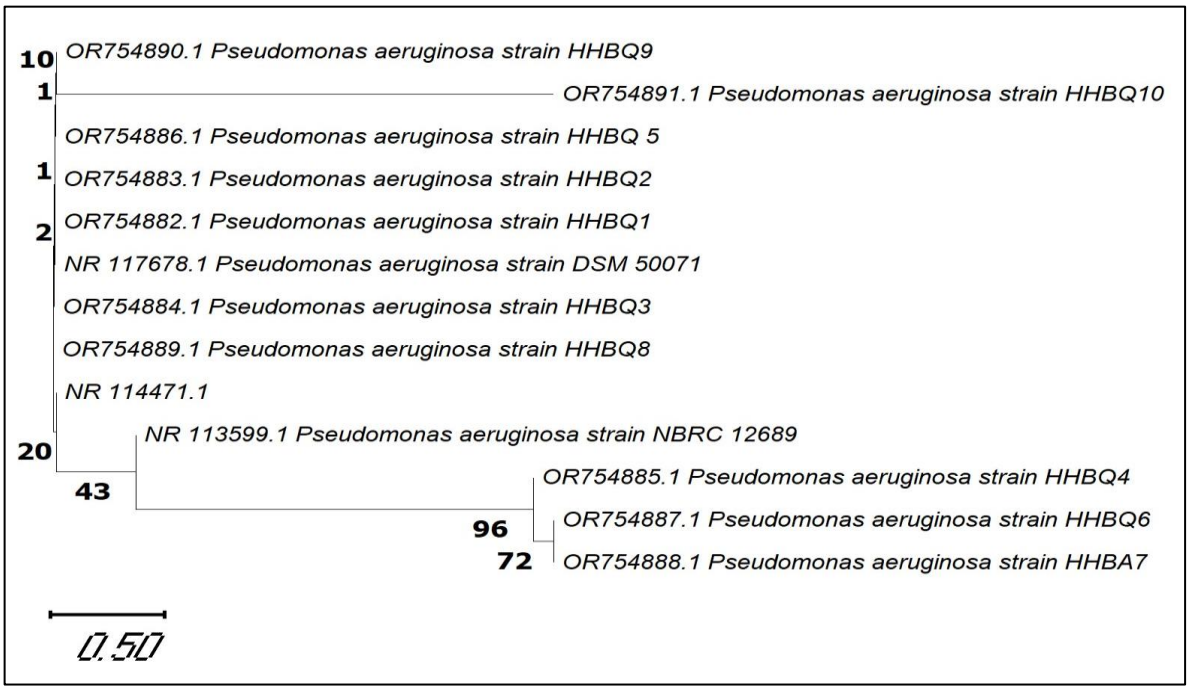

Figure 3: A phylogenetic tree constructed by MEGA 11.0 showing the evolutionary relationship of ten clinical strains of *P. aeruginosa* isolated from burn infections, samples enrolled, in relation to other closely related strains with the accession numbers: OR754882.1, OR754883.1, OR754884.1, OR754885.1, OR754886.1, OR754887.1, OR754888.1, OR754889.1, OR754890.1, and OR754891.1. The sequences with the accession numbers NR_113599.1, NR_114471.1, and NR_114471.1 are reference sequences retrieved from GenBank. Values displayed on arms represent bootstrapping values of 1000 re-samplings. The length of the arm is 0.05.

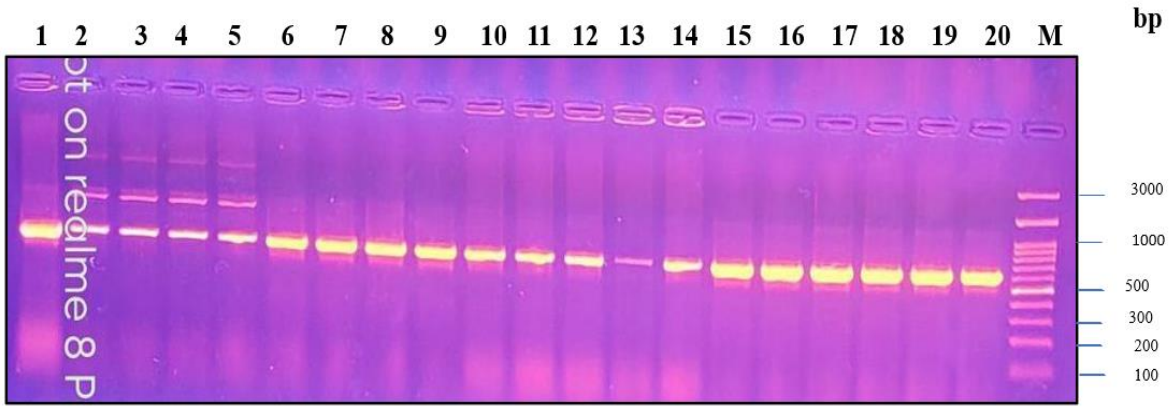

Figure 4 Agarose gel electrophoresis (1%) indicating partial amplification in the bla-OXA gene by PCR of 10 clinical strains of P. aeruginosa isolated from burn wound infection samples enrolled in this study. M: 100 bp DNA ladder. Lanes 1-20: PCR product (618 bp) of bla-OXA gene partially amplified from 10 clinical strains of P. aeruginosa.

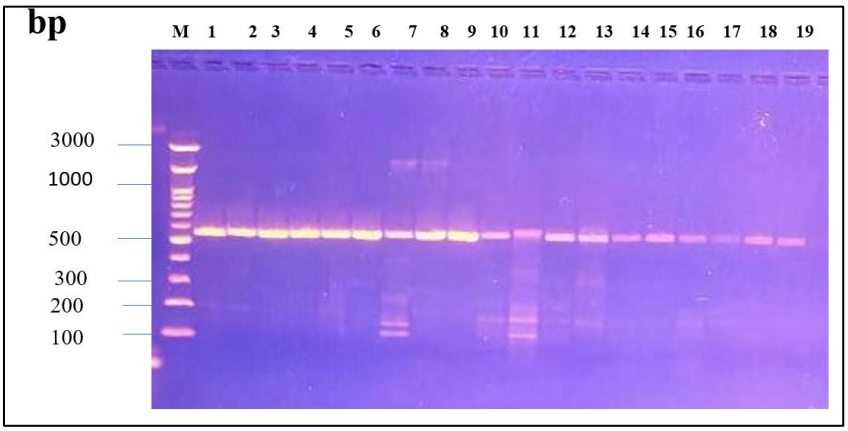

Figure 5 Agarose gel electrophoresis (1%) showing partial amplification in the bla CTX-M gene by PCR of 10 clinical strains of P. aeruginosa isolated from burn wound infection samples enrolled in this study. M: 100 bp DNA ladder. Lanes 1-19: PCR product (550 bp) of bla CTX-M gene partially amplified from 10 clinical strains of P. aeruginosa.

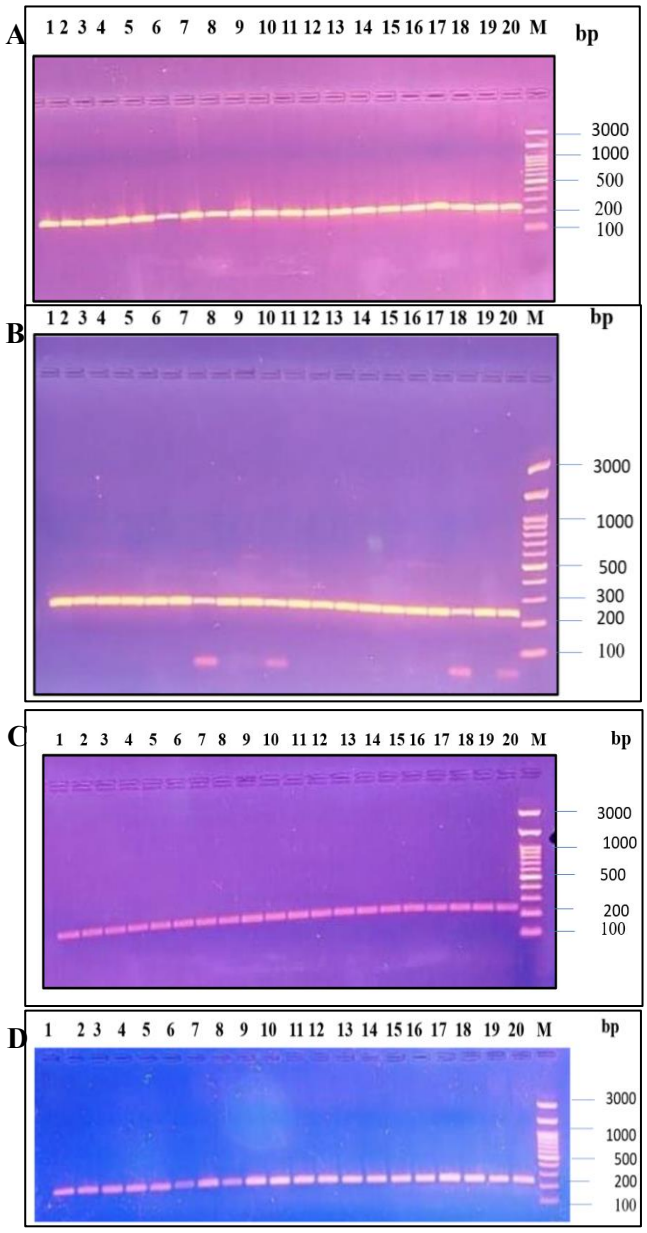

Figure 6. Agarose gel electrophoresis showing PCR amplification of biofilm-associated genes in clinical Pseudomonas aeruginosa isolates from burn wound infections. (A) Partial amplification of the algD gene (219 bp) on 1% agarose gel. (B) Amplification of the pslA gene (230 bp) on 2% agarose gel. (C) Partial amplification of the pslB gene (220 bp) on 2% agarose gel. (D) Partial amplification of the pelA gene (214 bp) on 1% agarose gel. M: 100 bp DNA ladder; lanes 1–20 represent PCR products from 10 clinical P. aeruginosa strains.

Recent studies in biofilm and molecular genetics show a relationship between biofilm and antibiotic resistance. P. aeruginosa cells within biofilm have unique metabolic and genetic expression profiles that result in the retention of resistance genes and the active transfer of resistance genes [14, 18]. This may clarify the observation in this study that isolates with strong biofilm genotypes also have multiple resistance genes. Salem et al. [17] demonstrated that antibiofilm strategies could re-establish antibiotic sensitivity in resistant P. aeruginosa isolates. This research also supports the observation made by Salem et al. that biofilm and resistance are reinforcing processes.

The majority of burn wound infections involve highly conserved lineages; the phylogenetic confirmation of the isolates as P. aeruginosa suggests that such lineages are dominating the infection. As Pseudomonas lineages that are pathogenic and prevalent in hospitals have been shown to possess similar core virulence structures, the reports also provide evidence of the presence of some form of resistance through mobile genetic elements [18,19]. Such elements increase both the capability to persist in the environment and the potential to spread in settings where burn care is practiced.

The need to combine control of infection and responsive approaches is also advocated in this setting. The reports confirming the presence of pan-drug-resistant P. aeruginosa in burn and even pediatric patients illustrate how the effectiveness of antimicrobial therapies is diminishing [15]. There is hope that some of the newer strategies, such as biofilm disruption, the use of nanoparticles to deliver drugs, and even the use of adjunctive antibiofilm therapies, will be effective as alternatives to the use of antibiotics [17,20].

These findings are in alignment with studies done globally at the level of regions, indicating that P. aeruginosa infection of burn wounds is converging with aspects of virulence, biofilm formation, and antimicrobial resistance. Antibiofilm-oriented treatment strategies, along with the ongoing molecular surveillance burnout, are necessary to reduce the additional morbidity and mortality risks high risk pathogens are adding to burn care.

Conclusions

The current research identified *Pseudomonas aeruginosa* isolates obtained from infection of burn wounds showing a remarkable and consistent high carriage of biofilm-associated genes (algD, pslA, pslB, and pelA). Adding to this finding, there was also a worrying high incidence of extended spectrum beta-lactamase (ESBL) genes, especially blaOXA and blaCTX-M. The biofilm and ESBL positive molecular traits reflect an 'environmentally persistent' pathogen population to the burn wound infection. The biofilm plus antimicrobial resistance "survival mechanisms" are adaptive to the wound infection. Confirmatory molecular and subsequent phylogenetic evaluations supported the conclusion of a clinical isolate being conserved dominant P. aeruginosa lineage. Overall, this research presented P. aeruginosa from burns as a coping research problem, and the requirement for sustained molecular monitoring, rigid infection control, and biofilm and antimicrobial resistance pairing in therapeutic approaches is to improve clinical outcomes in the field of burns.

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Declaration of Generative AI and AI-Assisted Technologies in Scientific Writing

The author declares that no generative artificial intelligence tools or AI-assisted technologies were used in this study

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