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Detection of Oxa10, 23 and 48 Genes in ESBL Gram Negative Bacteria Isolated from Hospitalized Patients at Red Sea Teaching Hospitals

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Abstract: ***The presence of ESBLs in many Gram-negative strains are of serious concern, since these organisms are the most common cause of different human infections. ESBL positive phenotypically were tested for the presence of ESBL encoding genes using PCR with specific primers for the detection of OXA Genes then the amplicons were sequenced to characterized gene content. The presence of OXA genes was confirmed in 17/ 163 of the isolates. The ESBL genes were detected in Escherichia coli, Klebsiellapneumoniae, Pseudomonas aeruginosa and Proteus mirabilis, the nucleotide sequences were subjected to BLAST for sequences similarity and homology.***

Keywords: E. coli, K. Pneumoniae, ESBLs *Pseudomonas aeruginosa, Proteus mirabilis,* OXA Genes, Sudan

1. Introduction

Antimicrobial resistance (AMR) represents one of the major global health threats of the 21st century, particularly in low- and middle-income countries, where surveillance, infection control and access to effective antibiotics are limited. Among Gram-negative pathogens, those producing extended-spectrum β-lactamases (ESBLs) which have been designated as (TEM-1, TEM-2, and SHV1). Although most of the ESBLs are mutants of the TEM and the SHV enzymes, the CTX-M type beta lactamases have become more important. and carbapenemases (notably KPC, VIM, and various OXA types) are of particular concern, because they render many β-lactam antibiotics ineffective. ESBL-producing bacteria compromise third-generation cephalosporins and related drugs; carbapenemase producers can inactivate even carbapenems, often the last resort in serious infections.

In Africa, the burden of ESBLs and carbapenemase genes is growing. Studies have shown increasing prevalence of genes such as bla_CTX-M, bla_SHV, bla_TEM for ESBLs, and emerging presence of carbapenemase genes including bla_OXA-48, bla_NDM, bla_KPC, bla_VIM. Surveillance is patchy, and countries with fewer resources often lack detailed molecular data to guide prevention and treatment.

In Sudan, several studies in recent years have begun to characterize the prevalence of ESBL and carbapenemase genes among clinical and environmental isolates. However, detailed data on co-existence of specific genes (KPC, VIM, OXA) in ESBL-producing, Gram-negative isolates from hospitals remain limited, which hampers infection control and antibiotic policy. Despite the growing body of literature on antimicrobial resistance in Sudan, most studies remain geographically restricted to Khartoum and Gezira states, with limited or no data from the Red Sea region. Furthermore, many investigations have focused primarily on ESBL genes (bla_CTX-M, bla_SHV, bla_TEM), while overlooking clinically significant carbapenemase genes such as bla_KPC, bla_VIM, and bla_OXA-48. This represents a critical gap, as the co-occurrence of ESBL and carbapenemase determinants severely limits therapeutic options and contributes to higher morbidity and mortality rates. Given the Red Sea Teaching Hospitals serve as referral centers for a wide catchment area, systematic screening of ESBL and carbapenemase genes in Gram-negative bacilli from hospitalized patients is urgently needed. The resulting molecular data will inform local antimicrobial stewardship policies, guide empiric therapy decisions, and strengthen infection prevention and control measures in this under-studied region.

ESBLs (commonly encoded by genes such as bla_CTX-M, bla_SHV, bla_TEM) compromise the efficacy of third-generation cephalosporins, while carbapenemases (including KPC, VIM, OXA-48-like NDM, IMP) can render carbapenems ineffective, removing a key last-line option for severe infections. The co-occurrence of ESBL and carbapenemase genes on mobile genetic elements accelerates dissemination between species and across healthcare and community settings

Globally, ESBL-producing Gram-negative bacilli have been widely reported in both hospital and community settings. The most frequent ESBL genes include bla_CTX-M, bla_SHV, and bla_TEM. In many countries, carbapenemases such as bla_NDM, bla_OXA-48, bla_KPC, bla_VIM are increasingly documented, posing a threat to the efficacy of last-resort antibiotics. A systematic review and meta-analysis (2023) estimated the prevalence of ESBL-producing K. pneumoniae in sub-Saharan Africa at 8.6% across human, animal, and environmental samples, with bla_CTX-M-15 being the dominant genotype [1].

A study of Shigella species in Africa (2024) found a pooled ESBL prevalence of 41.2%, though carbapenemase genes were less common [2]. In Alexandria (2024), bla_NDM was present in 92.5% of carbapenem-resistant K. pneumoniae isolates, bla_OXA-48 in 51.1%, while bla_KPC was detected

rarely (1%) [3]. Other studies confirm high frequency of ESBL genes (bla_SHV, bla_CTX-M) and carbapenemase genes (bla_NDM, bla_OXA-48) [4].

Studies from Sudan paint a concerning picture. Environmental surveillance in Khartoum detected carbapenemase genes in E. coli from drinking water; OXA-48 was the most commonly identified carbapenemase, with smaller numbers positive for KPC, and no VIM or IMP detected (5). Clinical investigations in Gezira documented high frequencies of ESBL genes of bla_CTX-M (85%), bla_SHV (70%), bla_TEM (33.3%), with carbapenemase genes bla_OXA-23 and bla_OXA-51 (~10%) [6]. Soba and Khartoum Hospitals non-glucose fermenting Gram-negative bacilli carried multiple carbapenemases (bla_NDM-1, bla_VIM, bla_KPC, bla_OXA-48-like, bla_OXA-23, bla_OXA-24, bla_OXA-51, bla_OXA-58). bla_OXA-51 was most common frequent [7].

2. Materials & Methods

2.1 Bacterial Isolates

Bacterial isolates were obtained from various clinical specimens including Urine, Wound swabs, and Sputum were collected from infected patients at Port Sudan Teaching hospital. The microbiology laboratory proceeds the specimens for the isolation and identification of significant bacterial pathogens following standard conventional procedures [8].

Specimens of urine were collected from the patients into sterile plastic containers and were transported to the microbiology laboratory and they were processed immediately for detection of pathogenic Grame negative bacteria. However, the Sputum samples were collected under aseptic condition. Specimens from wounds were taken by swabs, then placed on transport media and were analyzed as soon as possible.

2.2 Isolation and Identification of Gram-negative Bacilli

Isolation and identification of gram-negative bacilliwere carried out in a systemic way according to standard microbiological methods [9, 10]. A general procedure for isolated bacteria included isolation, identification, antimicrobial susceptibility testing and screening to presence of nosocomial isolates expressing an extended-spectrum beta-lactamaseas (ESBLs) by detection of reduced zone of inhibition around the third generation cephalosporins disc as recommended by the Clinical and Laboratory Standards Institute (CLSI). These isolates were confirmed for phenotypic ESBL production by the double disc synergy test (DDST) and the confirmatory double disc diffusion test (DDDT) and then were further confirmed by genotypic method (PCR) [11].

2.3 Cultivation of Specimens

Isolation of Gram-negative bacteria from specimens of urine was done by culturing directly onto CLED, MacConkey and Blood agar plates (Oxoid, Basingstoke England), using sterile nichrome wire calibrated loop. The isolation of Gram-negative bacteria from clinical specimens of wound swabs was done by inoculating directly onto MacConkey agar plates by streaking the swabs onto a small area of the plate. Then the sterile loop was used for cross-streaking to spread the inoculum over the surface of the plate to obtain single colonies. Specimen of sputum was received in the microbiology laboratory incubated aerobically overnight at a temperature of 37°C. After overnight incubation. The plates were then incubated overnight under aerobic conditions. On the second day, the first subcultures were observed for growth, and any growth identified. The samples that did not record any growth were re-incubated for another 24 hours under the same conditions. Up to three subcultures were performed similar to the procedure mentioned above if there was no growth from previous subcultures [10].

All cultured plates were incubated aerobically for 24 hours at 37°C and were examined for countable colonies. Each single significant growth of Gram-negative bacteria isolates were identified on the basis of cultural characteristics, gram stains, oxidase test and conventional biochemical tests, then confirmed by API 20E identification system (biomerieux Marcy-l'Etoile, France). Culture plates which yielded more than two organisms per specimen were excluded from the study.

2.4 Antimicrobial Susceptibility Test

Antimicrobial susceptibility testing of Gram-negative bacteria isolates was performed on Mueller-Hinton agar plate (Oxoid, Basingstoke England) by the Kirby-Bauer disk diffusion method following the CLSI recommendations. All isolates were tested for their susceptibility against 14 antimicrobial agents including; amikacin (30 µg), amoxicillin (10 µg), amoxicillin-clavulanic acid (30 µg), ceftazidime (30 µg), ceftriaxone (30 µg), cefuroxime (30 µg), chloramphenicol (30 µg), ciprofloxacin (5 µg), gentamicin (10 µg), nalidixic acid (30 µg), nitrofurantoin (50 µg), tetracycline (30 µg), tobramycin (10 µg) and trimethoprim sulfamethoxazole (25 µg), (Liofilchem Co. Italy). Standardized inoculum conforming to 0.5 McFarland standard turbidity of each isolate was inoculated on two Mueller-Hinton agar plates using a sterile cotton swab by streaking the swab over the entire sterile agar surface 3 times. Then onto each plate, 8 to 9 antimicrobial disks were placed at the recommended distance from each other. All plates were aerobically incubated at 37°C for 18 hours before the zone sizes were recorded. *E. coli* ATCC 25922, which were obtained from the American Type Culture Collection was used as control strains and tested each time when susceptibility testing was performed. Test results were only validated in the cases where inhibition zone diameters of the control strains were within performance range in accordance to CLSI guidelines [11].

2.5 Phenotypic Detection of ESBLs

2.5.1. ESBL Screening Test

2.5.1. Double Disc Synergy Test (DDST)

The double disc synergy test was carried out on Muller Hinton agar plate seeded by bacterial suspension. A disc containing the amoxiclav (amoxicillin 20 µg plus clavulanic

acid 10 µg) was placed on the center of Muller-Hinton agar, four discs of the following cephalosporins; cefepime 30µg, ceftazidime 30µg, cefotaxime 30 µg, and aztreonam 30 µg were placed around amoxiclave (Augmentin 20/10µg) at distance 25mm center to center. After overnight incubation, if there is an extension of the zone towards the disc containing amoxicillin-clavulanic acid it indicated that the strain possesses an ESBL [11].

2.5.3 Double Disk Diffusion Test (DDDT)

According to NCCLS, and CLSI-ESBL, phenotypic confirmatory test with Ceftazidime 30 µg, cefotaxime 30 µg, and cefepime 30 µg were performed for all the isolates by disk diffusion test (DDDT). Each disk was placed on Muller Hinton agar plates with and without 10µg of clavulanic acid. A difference of ≥5mm between the zone diameters of either of the cephalosporin disks and their respective cephalosporin/ clavulanate disk was considered to be phenotypic confirmation of ESBL production [11]. *E. coli* strain ATCC 25922 was used as a negative control and *Klebsiellapneumoniae* ATCC 700603 was used as a positive control.

2.6. Genotypic Detection of ESBLs

2.6.1. DNA Extraction

The genomic bacterial DNA was extracted using QIAamp DNA Kit (Qiagen, Germany). As per the manufacturer's instructions.

The concentration of the extracted DNA was measured using a Nanodrop 2000 (Thermo Scientific).

All isolates were screened for the resistance genes bla (OXA-10-23-48-) by polymerase chain reaction (PCR), using their specific primers.

Target Genes were amplified using COSMO PCR RED Master Mix (Willow Fort) and analyzed on a Biosystem 2720 thermal cycler. Prepare a PCR mixture reaction of 25 µL volume containing 12.5 µL master mix, 1 µL forward primer, 1 µL reverse primer, 2 µL DNA, and 8.5 µL deionized sterile water. This mixture was performed under the following thermocycler conditions: an initial polymerase activation step at 95 C° for 2 min, followed by 35 cycles of 95 C° for 15 sec, annealing temperature according to the gene C° for 40 sec, 72 C° for 40 sec, and final extension at 72 C° for 2 min [12].

2.6.2 DNA Sequencing for Characterization of KPC, VIM and OXA Genes

Sequencing is a method for determining the nucleotide sequence of a DNA molecule, thus it is a very precise method. DNA purification and standard sequencing was performed for both strands of OXA Genes Selected positive sample were sealed in sterile Eppendorf tubes and sent to the DNA sequencing by CEB Company (dokki, Egypt).

2.6.3 Bioinformatics Analysis

The nucleotide sequences of the OXA-10, OXA-23, OXA-48, genes were analyzed and modified using BioEdit software. They were then aligned and checked for sequence similarity using Nucleotide BLASTN, which is included in the BLAST program (Basic Local Alignment Search Tool) (http: //blast.ncbi.nlm.nih.gov/Blast.cgi). [13].

3. Results

3.1 Antibiotic Susceptibility Pattern of Gram-Negative Bacilli

Among the ESBL-producing Gram-negative bacilli, high resistance rates were observed for ceftriaxone (100.0%), tetracycline (100.0%), ciprofloxacin (100.0%), amoxyclav (98.9%), cefuroxime (98.9%), nalidixic acid (98.9%) and amoxicillin (95.5%). The highest antimicrobial activities of ESBL-producing organisms were observed with amikacin (96.6%), followed by Chloramphenicol (63.6%), Tobramycin (53.4%) and nitrofurantoin (50.0%). ESBL-producing Gram negative bacilli isolates were significantly more resistant to trimethoprim-sulfamethoxazole, tetracycline, nalidixic acid, cefuroxime, ceftazidime, ceftriaxone, ciprofloxacin, gentamicin, nitrofurantoin, amoxiclav, tobramycin and chloramphenicol compared to non-ESBL producing isolates.

3.2 Phenotypic Detection of ESBLs

A total of 163 Gram-negative bacilli isolates were tested for ESBL production. Of the 163 isolates, 17 (10.4%) were found to be ESBL-producers by double-disk synergy test (DDST)

3.3 Distribution of ESBL and Carbapenemase Genes among Bacterial Isolates

A total of **35 resistance genes** were identified among the 17 bacterial isolates, representing a gene-to-isolate ratio of approximately 2.05 genes per isolate. This indicates that most isolates harbored more than one gene. *Escherichia coli* accounted for the highest proportion, carrying 13/35 genes (37.1%). *Klebsiella pneumoniae* isolates contributed 9/35 genes (25.7%), while *Pseudomonas aeruginosa* carried 9/35 genes (25.7%). *Proteus mirabilis* had the lowest contribution with 4/35 genes (11.4%). These findings demonstrate although all species carried ESBL and carbapenemase genes, *E. coli* and *P. aeruginosa* showed the greatest genetic burden.

3.4 Frequencies of Genes among the 17 isolates:

Analysis of 35 resistance genes among the 17 isolates revealed distinct distribution patterns across bacterial species. The most frequent genes were OXA-48 and OXA-10, detected in 9/17(52.9%) and 7/17 isolates (41.2%), respectively. OXA-58 was found in 5/17 isolates (29.4%), followed by OXA-23 5/17 (29.4%), KPC 4/17 isolates (23.5%), VIM 4/17 (23.5%), whereas OXA-51 was the least common, detected in only 1/17 isolate (5.9 %).

3.5 Multiple Sequence Alignment

The multiple sequence alignment of the mutant isolate with similar nucleotide sequences that obtained from BLASTn was carried out to find the homology and evolutionary relation between these sequences. As shown by Bio Edit software there is an inserted and deleted of amino acid at very conserved region, see Figure: (1, 2).

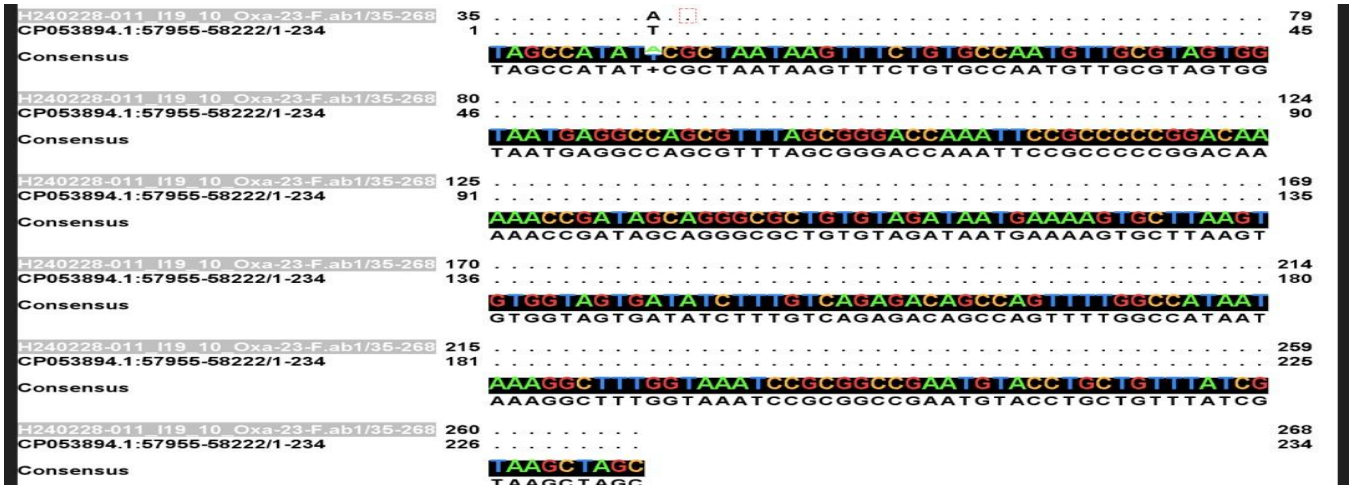

Figure 1: Sequencing alignment for the Mutant OXA-23 gene sequence chromatogram. showed deletion of Thymine at position 44 compared to the reference sequence.

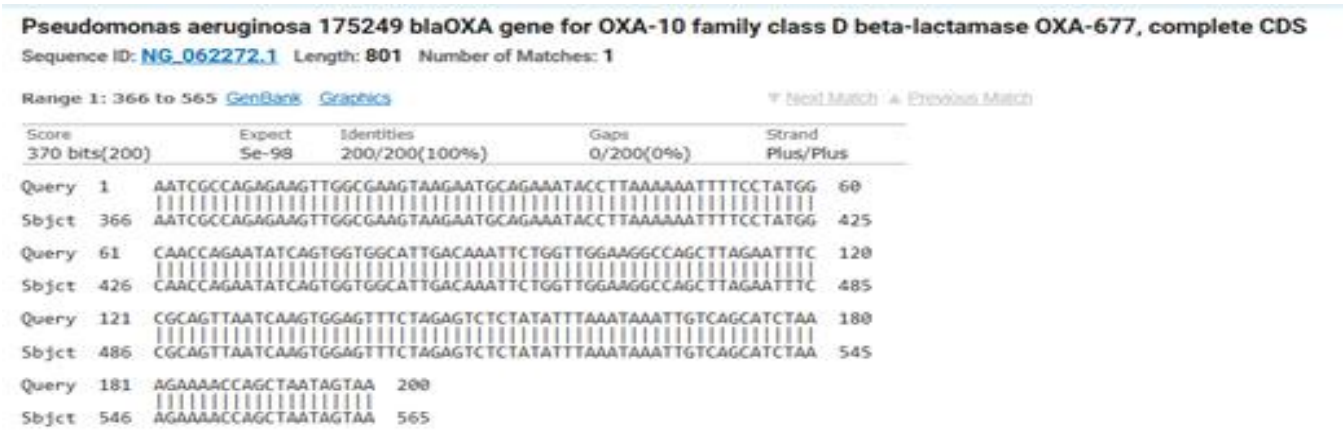

Figure 2: Sequencing alignment for the OXA-10 gene sequence chromatogram from another sample showed a complete match.

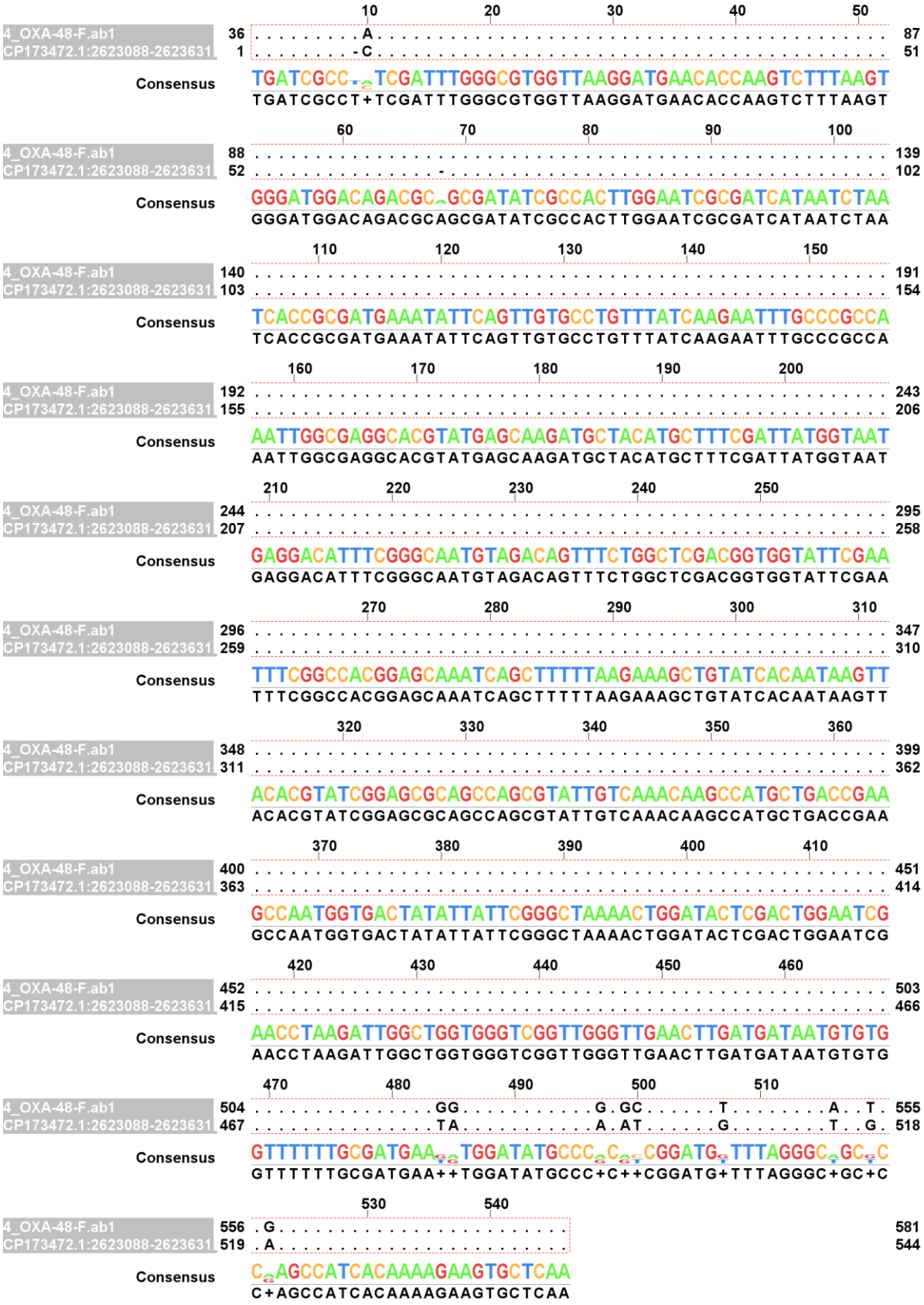

Figure 3: Sequencing alignment for the Mutant OXA-48 gene sequence chromatogram.

According to the pairwise alignment of the *bla*OXA-48 gene from Isolate 4 against the reference sequence from *Escherichia coli* strain 98224 (NCBI Accession: CP173472.1), a total of twelve nucleotide variations were detected. These include **two Insertions**, located at **positions 44 and 103**, relative to (OXA-48-F.ab1).

In addition to the insertion, **ten single-nucleotide substitutions** were identified throughout the gene sequence. These substitutions include the following mutations: C→A at position 45, T→ G at position 482, A→ G at position 483, A→ G at position 495, A→ G at position 497, T→C at

position 498, G→T at position 505, T→A at position 514, G→T at position 517, A→G at position 520 (positions relative to (OXA-48-F.ab1).

Collectively, these variations indicate that Isolate 4 carries a **distinct variant of the *bla*OXA-48 gene**, characterized by both early-frame deletions and dispersed point mutations. Such modifications may influence the structural configuration or enzymatic function of the resulting OXA-48 β-lactamase, potentially altering its antimicrobial resistance profile.

Insertions (2 Total)
The two insertions occur consecutively, resulting in a loss of two base pairs in the query sequence relative to the reference. This event is located near the end of the aligned region, preceding a highly divergent section.

Table 1: Frequencies of Genes among the isolates

GENES	E. Coli	Klebsiella pneumoniae	Proteus mirabilis	Pseudomonas aeruginosa
OXa-48	4	3	1	1
OXa-10	3	2	1	1
OXa-23	2	2	0	1

From these the existence of (OXa10-23-48), genes were detected among 17 (73.9%) ESBL producers. The most frequent gene detected was OXa-48 (9/17) followed by OXa10 gene (7/17), OXa23 gene (5/7). This difference in the frequency of ESBL genes was found significant (p = 0.00).

4. Discussions

ESBL detection is not routinely carried out in many microbiology laboratories of hospitals in developing countries, as well as in Sudan. The emergence of ESBL producing strains creates a need for laboratory testing methods for detection of these enzymes among bacterial pathogens. In this study, Escherichia coli was the predominant ESBL producer (41.2%), followed by Klebsiella pneumoniae and Pseudomonas aeruginosa. This pattern is consistent with recent global reports showing E. coli as the leading pathogen among multidrug-resistant (MDR) isolates, particularly from urinary sources. The high prevalence of OXA-48 (52.9%) among our isolates is of particular concern, as this gene has emerged as the most widespread carbapenemase in Africa and the Middle East, and is often associated with silent dissemination due to low-level resistance expression (14,15).

The detection of OXA-10 (41.2%) and OXA-23 (29.4%) further highlights the genetic diversity of β-lactamase determinants in our setting. OXA-23, traditionally associated with Acinetobacter, has been increasingly reported in Enterobacterales, indicating horizontal gene transfer and expansion beyond its classical hosts (16). Similarly, the presence of OXA-48 gene mirrors recent findings from Sub-Saharan Africa where metallo-β-lactamases (MBLs) are emerging at worrying rates (17).

Sanger sequencing confirmed multiple substitutions and deletions in OXA genes, which may enhance hydrolytic activity and compromise inhibitor binding. Comparable structural mutations in OXA-48 variants were recently linked to increased carbapenem resistance and reduced susceptibility to novel β-lactam/β-lactamase inhibitor combinations (18,19). These observations underscore the evolutionary adaptation of resistance genes under antibiotic pressure, which may limit therapeutic options. Our findings emphasize the urgent need for continuous molecular surveillance and antibiotic stewardship programs in Sudan. The predominance of OXA-48, coupled with the emergence of diverse carbapenemase mutations, indicates a high potential for treatment failures and outbreaks. Integration of molecular diagnostics into routine clinical practice is essential for early detection and containment. In our study, bioinformatics analysis of the sequenced **OXA** genes using **BLAS Tn** revealed several nucleotide and amino acid variations, including **insertions, deletions, and substitutions** when compared with reference β-lactamase sequences from NCBI. These mutations were located in highly **conserved regions** of the genes, particularly within the **β-lactamase/transpeptidase-like domain**, which plays a critical role in antibiotic hydrolysis and resistance mechanisms. (20). Such amino acid alterations may affect the configuration of the **active site serine residue** or alter the **hydrophobic pocket** that interacts with β-lactam antibiotics, consequently enhancing catalytic efficiency and broadening substrate specificity. This structural modification could explain the extended-spectrum activity observed among the isolates, as similar findings were reported by several studies that demonstrated that point mutations within conserved motifs (such as *S70, K73, S130,* and *E166*) significantly increase β-lactamase activity and confer resistance to newer β-lactam compounds. (21).

Overall, the bioinformatics results support the hypothesis that **specific mutations within conserved catalytic domains** are responsible for the enhanced enzymatic activity of ESBLs and their ability to inactivate a broad range of β-lactam antibiotics. Continuous genomic monitoring and comparative sequence analysis are therefore essential for understanding the molecular evolution of resistance genes and for guiding the development of new therapeutic inhibitors.

5. Conclusion

ESBL types of OXA genes are predominant among Gram-negative bacilli isolates and ESBL producer strains carried one or more than OXA genes. ESBLs genes are rapidly evolved among pathogenic bacteria, thus study like this to detect a new antibiotic-resistant gene variant could guide the choice of optimal antibiotic therapy for successful treatment, thus improving the outcomes for patients with severe bacterial infections.

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