

Antimicrobial resistance of Shiga toxin-producing *Escherichia coli* O157 isolates from Northern Palestine

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Abstract - The present study was initiated to assess and evaluate antimicrobial resistance in one hundred eighteen isolates of Shiga toxin-producing *Escherichia coli* STEC O157 obtained from Northern Palestine. Eighty-three percent of isolates were resistant to ampicillin, followed by 59% to norfloxacin, 58% to tetracycline, 55% to gentamicin, 50% to chloramphenicol, 48% to amikacin, 40% to co-trimoxazole, and 25% to ceftazidime. Multidrug resistance was seen in more than two-third of the isolates (68%), and there was no common resistance pattern among the isolates. Our findings suggest that use of antimicrobials, including tetracycline derivatives, sulfa drugs, aminoglycosides, and penicillins, has selected for multiple antimicrobial resistance phenotypes in STEC O157.

Keywords: STEC O157, drug resistance

The incidence of antimicrobial resistance is increasingly reported at alarming rates in recent years so that it is considered today a major public health problem in both developed and developing countries throughout the world.^{1,2} Recently, multidrug-resistant STEC O157 isolates have been documented and considerably increased all over the world.³⁻⁷ Antibiotics such as tetracyclines, sulfa drugs, cephalosporins, and penicillins in therapy are a major factor in the emergence and dissemination of antimicrobial resistant STEC O157.^{7,8,9} The present study aimed to obtain a snapshot of STEC O157 resistance in northern Palestine, a part of the world not previously surveyed for this type of resistance.

Materials and Methods

A total of 118 STEC O157 isolates collected during an outbreak of diarrhoea between February and June 1999 in the north of Palestine were used in this study. All the isolates carried the gene for Shiga toxin type 1 (*stx1*) and 112 (90.3%) carried *stx2*. The intimin encoding gene locus *eae* was detected in 16 (12.9%) and the enterohaemolysin encoding gene, *hlyA*, in 18 (14.5%). ERIC-PCR analysis of DNA from 80 isolates revealed three different clonal populations.¹⁰ The susceptibility of isolates to antimicrobials was determined by disk diffusion¹¹ in accordance with National Committee for Clinical Laboratory Standards.¹² The following antibiotics were used: ampicillin (Amp; 10 µg), tetracycline (Tet; 30 µg), co-trimoxazole (Sxt; 25), chloramphenicol (Chl; 30 µg), amikacin (Amk; 30), gentamicin

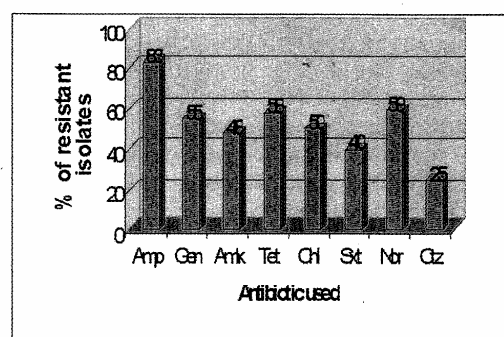


Fig.: Antibiotic resistance patterns of Shiga Toxin-Producing *Escherichia coli* O157 isolates. Abbreviations: Amp, ampicillin; Tet, tetracycline; Sxt, Co-trimoxazole; Chl, chloramphenicol; Amk, amikacin; Gen, gentamicin; Nor, norfloxacin; Cz, ceftazidime.

(Gen; 10 µg), norfloxacin (Nor; 10), and ceftazidime (Ctz; 10)

Results

Eighty-three percent of isolates were resistant to ampicillin, followed by 59% to norfloxacin, 58% to tetracycline, 55% to gentamicin, 50% to chloramphenicol, 48% to amikacin, 40% to co-trimoxazole, and 25% to ceftazidime (Fig.). Multidrug resistance was seen in more than two-third of the isolates (68%), and there was no common resistance pattern among the isolates.

Discussion

Of the 118 STEC O157 isolates characterized in this study, multiple antimicrobial resistance was seen in

more than two-thirds of the isolates (68%), a finding mirrored elsewhere.^{7,9} The isolates examined in this study came from clinical cases and therefore may have been exposed to elevated concentrations of antimicrobials as a result of treatment efforts. Nonetheless, our findings suggest that use of antimicrobials, including tetracycline derivatives, sulfa drugs, aminoglycosides, and penicillins has selected for multiple antimicrobial resistance phenotypes in STEC O157. On the other hand, this phenomenon may be due to the frequent contact with livestock that are commonly given antimicrobial growth promoters, the reservoir of STEC.^{13,14,15}

Multiple antimicrobial resistance in STEC O157 may partly result from the spread of genetic elements including plasmids, transposons, and integrons⁷ that may confer resistance to numerous antimicrobials. The recovery of three different ERIC-PCR patterns among 80 isolates and the lack of correlation between antimicrobial resistance patterns and molecular types of the isolates suggest convergent acquisition of resistance determinants by genetically unrelated STEC O157 strains rather than epidemic spread of resistant isolates in community. Because the emergence and dissemination of antimicrobial resistance in STEC O157 may complicate future therapeutic options that are being developed for treatment of haemolytic-uraemic syndrome (HUS) and thrombotic thrombocytopenic purpura, continued surveillance of emerging antimicrobial resistance, therefore, should be taken into account in planning therapy for STEC infections.

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