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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии

საქართველოს სამედიცინო სიახლენი

GEORGIAN MEDICAL NEWS

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GMN: Georgian Medical News is peer-reviewed, published monthly journal committed to promoting the science and art of medicine and the betterment of public health, published by the GMN Editorial Board since 1994. GMN carries original scientific articles on medicine, biology and pharmacy, which are of experimental, theoretical and practical character; publishes original research, reviews, commentaries, editorials, essays, medical news, and correspondence in English and Russian.

GMN is indexed in MEDLINE, SCOPUS, PubMed and VINITI Russian Academy of Sciences. The full text content is available through EBSCO databases.

GMN: Медицинские новости Грузии - ежемесячный рецензируемый научный журнал, издаётся Редакционной коллегией с 1994 года на русском и английском языках в целях поддержки медицинской науки и улучшения здравоохранения. В журнале публикуются оригинальные научные статьи в области медицины, биологии и фармации, статьи обзорного характера, научные сообщения, новости медицины и здравоохранения. Журнал индексируется в MEDLINE, отражён в базе данных SCOPUS, PubMed и ВИНИТИ РАН. Полнотекстовые статьи журнала доступны через БД EBSCO.

GMN: Georgian Medical News – საქართველოს სამედიცინო სიახლენი – არის ყოველთვიური სამეცნიერო სამედიცინო რეცენზირებადი ჟურნალი, გამოიცემა 1994 წლიდან, წარმოადგენს სარედაქციო კოლეგიისა და აშშ-ის მეცნიერების, განათლების, ინდუსტრიის, ხელოვნებისა და ბუნებისმეტყველების საერთაშორისო აკადემიის ერთობლივ გამოცემას. GMN-ში რუსულ და ინგლისურ ენებზე ქვეყნდება ექსპერიმენტული, თეორიული და პრაქტიკული ხასიათის ორიგინალური სამეცნიერო სტატიები მედიცინის, ბიოლოგიისა და ფარმაციის სფეროში, მიმოხილვითი ხასიათის სტატიები.

ჟურნალი ინდექსირებულია MEDLINE-ის საერთაშორისო სისტემაში, ასახულია SCOPUS-ის, PubMed-ის და ВИНИТИ РАН-ის მონაცემთა ბაზებში. სტატიების სრული ტექსტი ხელმისაწვდომია EBSCO-ს მონაცემთა ბაზებიდან.

WEBSITE

www.geomednews.com

К СВЕДЕНИЮ АВТОРОВ!

При направлении статьи в редакцию необходимо соблюдать следующие правила:

1. Статья должна быть представлена в двух экземплярах, на русском или английском языках, напечатанная через **полтора интервала на одной стороне стандартного листа с шириной левого поля в три сантиметра**. Используемый компьютерный шрифт для текста на русском и английском языках - **Times New Roman** (**Кириллица**), для текста на грузинском языке следует использовать **AcadNusx.** Размер шрифта - **12**. К рукописи, напечатанной на компьютере, должен быть приложен CD со статьей.

2. Размер статьи должен быть не менее десяти и не более двадцати страниц машинописи, включая указатель литературы и резюме на английском, русском и грузинском языках.

3. В статье должны быть освещены актуальность данного материала, методы и результаты исследования и их обсуждение.

При представлении в печать научных экспериментальных работ авторы должны указывать вид и количество экспериментальных животных, применявшиеся методы обезболивания и усыпления (в ходе острых опытов).

4. К статье должны быть приложены краткое (на полстраницы) резюме на английском, русском и грузинском языках (включающее следующие разделы: цель исследования, материал и методы, результаты и заключение) и список ключевых слов (key words).

5. Таблицы необходимо представлять в печатной форме. Фотокопии не принимаются. **Все цифровые, итоговые и процентные данные в таблицах должны соответствовать таковым в тексте статьи**. Таблицы и графики должны быть озаглавлены.

6. Фотографии должны быть контрастными, фотокопии с рентгенограмм - в позитивном изображении. Рисунки, чертежи и диаграммы следует озаглавить, пронумеровать и вставить в соответствующее место текста **в tiff формате**.

В подписях к микрофотографиям следует указывать степень увеличения через окуляр или объектив и метод окраски или импрегнации срезов.

7. Фамилии отечественных авторов приводятся в оригинальной транскрипции.

8. При оформлении и направлении статей в журнал МНГ просим авторов соблюдать правила, изложенные в «Единых требованиях к рукописям, представляемым в биомедицинские журналы», принятых Международным комитетом редакторов медицинских журналов - http://www.spinesurgery.ru/files/publish.pdf и http://www.nlm.nih.gov/bsd/uniform_requirements.html
В конце каждой оригинальной статьи приводится библиографический список. В список литературы включаются все материалы, на которые имеются ссылки в тексте. Список составляется в алфавитном порядке и нумеруется. Литературный источник приводится на языке оригинала. В списке литературы сначала приводятся работы, написанные знаками грузинского алфавита, затем кириллицей и латиницей. Ссылки на цитируемые работы в тексте статьи даются в квадратных скобках в виде номера, соответствующего номеру данной работы в списке литературы. Большинство цитированных источников должны быть за последние 5-7 лет.

9. Для получения права на публикацию статья должна иметь от руководителя работы или учреждения визу и сопроводительное отношение, написанные или напечатанные на бланке и заверенные подписью и печатью.

10. В конце статьи должны быть подписи всех авторов, полностью приведены их фамилии, имена и отчества, указаны служебный и домашний номера телефонов и адреса или иные координаты. Количество авторов (соавторов) не должно превышать пяти человек.

11. Редакция оставляет за собой право сокращать и исправлять статьи. Корректура авторам не высылается, вся работа и сверка проводится по авторскому оригиналу.

12. Недопустимо направление в редакцию работ, представленных к печати в иных издательствах или опубликованных в других изданиях.

При нарушении указанных правил статьи не рассматриваются.

REQUIREMENTS

Please note, materials submitted to the Editorial Office Staff are supposed to meet the following requirements:

1. Articles must be provided with a double copy, in English or Russian languages and typed or compu-ter-printed on a single side of standard typing paper, with the left margin of **3** centimeters width, and **1.5** spacing between the lines, typeface - **Times New Roman** (**Cyrillic**), print size - **12** (referring to Georgian and Russian materials). With computer-printed texts please enclose a CD carrying the same file titled with Latin symbols.

2. Size of the article, including index and resume in English, Russian and Georgian languages must be at least 10 pages and not exceed the limit of 20 pages of typed or computer-printed text.

3. Submitted material must include a coverage of a topical subject, research methods, results, and review.

Authors of the scientific-research works must indicate the number of experimental biological species drawn in, list the employed methods of anesthetization and soporific means used during acute tests.

4. Articles must have a short (half page) abstract in English, Russian and Georgian (including the following sections: aim of study, material and methods, results and conclusions) and a list of key words.

5. Tables must be presented in an original typed or computer-printed form, instead of a photocopied version. **Numbers, totals, percentile data on the tables must coincide with those in the texts of the articles.** Tables and graphs must be headed.

6. Photographs are required to be contrasted and must be submitted with doubles. Please number each photograph with a pencil on its back, indicate author's name, title of the article (short version), and mark out its top and bottom parts. Drawings must be accurate, drafts and diagrams drawn in Indian ink (or black ink). Photocopies of the X-ray photographs must be presented in a positive image in **tiff format**.

Accurately numbered subtitles for each illustration must be listed on a separate sheet of paper. In the subtitles for the microphotographs please indicate the ocular and objective lens magnification power, method of coloring or impregnation of the microscopic sections (preparations).

7. Please indicate last names, first and middle initials of the native authors, present names and initials of the foreign authors in the transcription of the original language, enclose in parenthesis corresponding number under which the author is listed in the reference materials.

8. Please follow guidance offered to authors by The International Committee of Medical Journal Editors guidance in its Uniform Requirements for Manuscripts Submitted to Biomedical Journals publication available online at: http://www.nlm.nih.gov/bsd/uniform_requirements.html
http://www.icmje.org/urm_full.pdf
In GMN style for each work cited in the text, a bibliographic reference is given, and this is located at the end of the article under the title "References". All references cited in the text must be listed. The list of references should be arranged alphabetically and then numbered. References are numbered in the text [numbers in square brackets] and in the reference list and numbers are repeated throughout the text as needed. The bibliographic description is given in the language of publication (citations in Georgian script are followed by Cyrillic and Latin).

9. To obtain the rights of publication articles must be accompanied by a visa from the project instructor or the establishment, where the work has been performed, and a reference letter, both written or typed on a special signed form, certified by a stamp or a seal.

10. Articles must be signed by all of the authors at the end, and they must be provided with a list of full names, office and home phone numbers and addresses or other non-office locations where the authors could be reached. The number of the authors (co-authors) must not exceed the limit of 5 people.

11. Editorial Staff reserves the rights to cut down in size and correct the articles. Proof-sheets are not sent out to the authors. The entire editorial and collation work is performed according to the author's original text.

12. Sending in the works that have already been assigned to the press by other Editorial Staffs or have been printed by other publishers is not permissible.

Articles that Fail to Meet the Aforementioned Requirements are not Assigned to be Reviewed.

ავტორთა საყურადღებოდ!

რედაქციაში სტატიის წარმოდგენისას საჭიროა დავიცვათ შემდეგი წესები:

1. სტატია უნდა წარმოადგინოთ 2 ცალად, რუსულ ან ინგლისურ ენებზე,დაბეჭდილი სტანდარტული ფურცლის 1 გვერდზე, 3 სმ სიგანის მარცხენა ველისა და სტრიქონებს შორის 1,5 ინტერვალის დაცვით. გამოყენებული კომპიუტერული შრიფტი რუსულ და ინგლისურენოვან ტექსტებში - **Times New Roman (Кириллица)**, ხოლო ქართულენოვან ტექსტში საჭიროა გამოვიყენოთ **AcadNusx.** შრიფტის ზომა – 12. სტატიას თან უნდა ახლდეს CD სტატიით.

2. სტატიის მოცულობა არ უნდა შეადგენდეს 10 გვერდზე ნაკლებს და 20 გვერდზე მეტს ლიტერატურის სიის და რეზიუმეების (ინგლისურ, რუსულ და ქართულ ენებზე) ჩათვლით.

3. სტატიაში საჭიროა გაშუქდეს: საკითხის აქტუალობა; კვლევის მიზანი; საკვლევი მასალა და გამოყენებული მეთოდები; მიღებული შედეგები და მათი განსჯა. ექსპერიმენტული ხასიათის სტატიების წარმოდგენისას ავტორებმა უნდა მიუთითონ საექსპერიმენტო ცხოველების სახეობა და რაოდენობა; გაუტკივარებისა და დაძინების მეთოდები (მწვავე ცდების პირობებში).

4. სტატიას თან უნდა ახლდეს რეზიუმე ინგლისურ, რუსულ და ქართულ ენებზე არანაკლებ ნახევარი გვერდის მოცულობისა (სათაურის, ავტორების, დაწესებულების მითითებით და უნდა შეიცავდეს შემდეგ განყოფილებებს: მიზანი, მასალა და მეთოდები, შედეგები და დასკვნები; ტექსტუალური ნაწილი არ უნდა იყოს 15 სტრიქონზე ნაკლები) და საკვანძო სიტყვების ჩამონათვალი (key words).

5. ცხრილები საჭიროა წარმოადგინოთ ნაბეჭდი სახით. ყველა ციფრული, შემაჯამებელი და პროცენტული მონაცემები უნდა შეესაბამებოდეს ტექსტში მოყვანილს.

6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრამების ფოტოასლები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შეღებვის ან იმპრეგნაციის მეთოდი და აღნიშნოთ სურათის ზედა და ქვედა ნაწილები.

7. სამამულო ავტორების გვარები სტატიაში აღინიშნება ინიციალების თანდართვით, უცხოურისა – უცხოური ტრანსკრიპციით.

8. სტატიას თან უნდა ახლდეს ავტორის მიერ გამოყენებული სამამულო და უცხოური შრომების ბიბლიოგრაფიული სია (ბოლო 5-8 წლის სიღრმით). ანბანური წყობით წარმოდგენილ ბიბლიოგრაფიულ სიაში მიუთითეთ ჯერ სამამულო, შემდეგ უცხოელი ავტორები (გვარი, ინიციალები, სტატიის სათაური, ჟურნალის დასახელება, გამოცემის ადგილი, წელი, ჟურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფჩხილებში უნდა მიუთითოთ ავტორის შესაბამისი N ლიტერატურის სიის მიხედვით. მიზანშეწონილია, რომ ციტირებული წყაროების უმეტესი ნაწილი იყოს 5-6 წლის სიღრმის.

9. სტატიას თან უნდა ახლდეს: ა) დაწესებულების ან სამეცნიერო ხელმძღვანელის წარდგინება, დამოწმებული ხელმოწერითა და ბეჭდით; ბ) დარგის სპეციალისტის დამოწმებული რეცენზია, რომელშიც მითითებული იქნება საკითხის აქტუალობა, მასალის საკმაობა, მეთოდის სანდოობა, შედეგების სამეცნიერო-პრაქტიკული მნიშვნელობა.

10. სტატიის ბოლოს საჭიროა ყველა ავტორის ხელმოწერა, რომელთა რაოდენობა არ უნდა აღემატებოდეს 5-ს.

11. რედაქცია იტოვებს უფლებას შეასწოროს სტატია. ტექსტზე მუშაობა და შეჯერება ხდება საავტორო ორიგინალის მიხედვით.

12. დაუშვებელია რედაქციაში ისეთი სტატიის წარდგენა, რომელიც დასაბეჭდად წარდგენილი იყო სხვა რედაქციაში ან გამოქვეყნებული იყო სხვა გამოცემებში.

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

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ANTIMICROBIAL SUSCEPTIBILITY PATTERNS OF ESCHERICHIA COLI AND ENTEROBACTER SPP. CAUSING COMMUNITY-ACQUIRED INFECTIONS IN MÉRIDA, VENEZUELA

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Abstract.

Enterobacterales are among the leading causes of community-acquired infections and represent an increasing public health concern due to the emergence of antimicrobial resistance. This study aimed to characterize the antimicrobial susceptibility profiles and resistance phenotypes of *Escherichia coli* and *Enterobacter* spp. isolates recovered from clinical specimens processed in a microbiology laboratory in Mérida, Venezuela, between January 2017 and May 2021. A descriptive, retrospective, cross-sectional study was conducted, including 39 clinically relevant isolates corresponding to *E. coli* (n = 17) and *Enterobacter* spp. (n = 22). Bacterial identification was performed using conventional biochemical methods, and antimicrobial susceptibility testing was carried out by disk diffusion according to Clinical and Laboratory Standards Institute (CLSI) guidelines. Phenotypic detection of extended-spectrum β-lactamases (ESBLs) and AmpC β-lactamases was also performed.

Escherichia coli was the predominant pathogen isolated from urine samples (58.8%), whereas *Enterobacter* spp. predominated in skin and wound secretion specimens. Ten antimicrobial susceptibility profiles were identified among *E. coli* isolates and eight among *Enterobacter* spp., revealing substantial phenotypic diversity. *E. coli* isolates exhibited high susceptibility to cefepime, aztreonam, imipenem, and meropenem, but showed high resistance rates to ampicillin, cefuroxime, and trimethoprim-sulfamethoxazole. In addition, susceptibility patterns compatible with ESBL production and multidrug-resistant phenotypes were identified. *Enterobacter* spp. isolates retained susceptibility to third- and fourth-generation cephalosporins, monobactams, and carbapenems, while displaying frequent resistance to β-lactam/β-lactamase inhibitor combinations, a pattern consistent with inducible chromosomal AmpC production. No isolate exhibited a phenotype compatible with carbapenemase production.

These findings demonstrate the circulation of Enterobacterales with diverse resistance profiles in community-acquired infections and highlight the importance of continuous microbiological surveillance to support empirical antimicrobial therapy and prevent the dissemination of emerging resistance mechanisms.

Key words. *Escherichia coli*, *Enterobacter* spp., antimicrobial resistance, extended-spectrum β-lactamases, AmpC β-lactamase, community-acquired infection.

Introduction.

Community-acquired infections represent a major public health concern due to their high prevalence and substantial impact on morbidity and healthcare costs. Among the most important etiological agents are Enterobacterales, a diverse group of Gram-negative bacteria capable of causing urinary tract, gastrointestinal, respiratory, skin, and soft tissue infections in both immunocompetent and immunocompromised individuals [1,2].

Enterobacterales constitute part of the normal human gastrointestinal microbiota; however, certain species possess virulence factors that enable them to colonize extraintestinal sites and cause disease. Within this group, Escherichia coli is the pathogen most frequently associated with community-acquired infections, particularly urinary tract infections and infectious diarrhea. Additionally, *E. coli* may cause severe extraintestinal infections, including bacteremia, hepatobiliary infections, peritonitis, and sepsis, especially when host anatomical barriers are compromised [2,4].

Species belonging to the genus *Enterobacter* have also emerged as clinically significant opportunistic pathogens due to their ability to cause urinary tract infections, respiratory infections, bacteremia, and wound infections. Although traditionally associated with healthcare settings, the isolation of *Enterobacter* spp. from community-acquired infections has increased in recent years, posing an additional challenge for clinical management and epidemiological surveillance [3,5].

The increasing prevalence of antimicrobial resistance among Enterobacterales represents one of the most critical threats to global health. These microorganisms have developed multiple resistance mechanisms, among which the production of extended-spectrum β-lactamases (ESBLs) is particularly concerning. These enzymes hydrolyze penicillins, extended-spectrum cephalosporins, and monobactams, thereby significantly limiting available therapeutic options. Consequently, the treatment of infections caused by these pathogens often requires the use of broad-spectrum or last-resort antimicrobials, further promoting the selection and dissemination of multidrug-resistant strains [1,6].

The spread of antimicrobial-resistant Enterobacterales extends beyond healthcare facilities into the community, where colonized or infected individuals may serve as reservoirs and potential sources of transmission. This phenomenon has highlighted the need for continuous microbiological surveillance programs aimed at monitoring local antimicrobial susceptibility patterns and guiding empirical antimicrobial therapy. Such strategies are essential for optimizing antimicrobial use and mitigating the emergence and dissemination of bacterial resistance [1,7].

In this context, understanding the antimicrobial susceptibility profiles of *E. coli* and *Enterobacter* spp. isolated from

community-acquired infections is crucial for clinical decision-making and the development of effective infection control strategies. Therefore, the present study aimed to characterize the antimicrobial susceptibility profiles and resistance trends of *E. coli* and *Enterobacter* spp. associated with community-acquired infections, isolated at a reference microbiology laboratory in Mérida, Venezuela, between January 2017 and May 2021.

Materials and Methods.

Study design:

A retrospective descriptive observational study was conducted to characterize the antimicrobial susceptibility profiles of *E. coli* and *Enterobacter* spp. isolated from community-acquired infections processed at a reference microbiology laboratory in Mérida, Venezuela, between January 2017 and May 2021.

Study Population and Selection Criteria:

The study population consisted of 39 bacterial isolates identified as *E. coli* and *Enterobacter* spp. recovered from clinical specimens obtained from outpatients with suspected community-acquired infections.

Inclusion Criteria:

Clinical specimens obtained from community patients presenting signs and symptoms suggestive of bacterial infection.

Patients who had not received antimicrobial therapy before specimen collection.

Isolates identified as *E. coli* or *Enterobacter* spp.

Exclusion Criteria:

Specimens collected from patients receiving antimicrobial therapy at the time of sampling.

Isolates with incomplete identification or inconclusive antimicrobial susceptibility results.

Sample Collection and Microbiological Processing:

Bacterial isolates were recovered from urine, pharyngeal swabs, nasal secretions, vaginal secretions, and skin and soft tissue specimens. Samples were transported using Stuart transport medium and processed according to standard microbiological procedures for each specimen type.

Specimens were inoculated onto Blood Agar and MacConkey Agar plates and incubated aerobically at 37 °C for 18–24 h. Following incubation, Gram staining and colony morphology assessment were performed.

Bacterial Identification:

Bacterial identification was carried out using conventional biochemical methods according to the procedures described by Koneman et al. The biochemical tests included oxidase, Kligler Iron Agar (KIA), Lysine Iron Agar (LIA), Motility-Indole-Ornithine (MIO), Simmons citrate, and urease tests.

Isolates were identified as *Escherichia coli* or *Enterobacter* spp. based on their characteristic biochemical profiles and established identification criteria for clinically relevant Enterobacterales.

Antimicrobial Susceptibility Testing:

Antimicrobial susceptibility testing was performed using the Kirby–Bauer disk diffusion method according to the Clinical and Laboratory Standards Institute (CLSI, 2018) [8] guidelines.

Briefly, bacterial suspensions were prepared in sterile 0.85% saline solution and adjusted to a turbidity equivalent to the 0.5 McFarland standard (1.5×10^8 CFU/mL). The inoculum was evenly spread onto Mueller-Hinton agar (HiMedia) plates using sterile cotton swabs.

The following antimicrobial disks (Oxoid®) were tested:

Cefotaxime (CTX, 30 μg)
Ceftazidime (CAZ, 30 μg)
Cefepime (FEP, 30 μg)
Cefoxitin (FOX, 30 μg)
Amoxicillin-clavulanic acid (AMC, 20/10 μg)
Imipenem (IMP, 10 μg)
Meropenem (MER, 10 μg)
Amikacin (AK, 30 μg)
Gentamicin (CN, 10 μg)
Ciprofloxacin (CIP, 5 μg)
Levofloxacin (LEV, 5 μg)
Trimethoprim-sulfamethoxazole (SXT, 1.25/23.75 μg)

Plates were incubated aerobically at 37 °C for 18–24 h. Inhibition zone diameters were measured in millimeters and interpreted as susceptible (S), intermediate (I), or resistant (R) according to CLSI 2018 breakpoints.

Quality Control:

Quality control procedures were performed using *Escherichia coli* ATCC 25922, *Escherichia coli* ATCC 35218, and *Pseudomonas aeruginosa* ATCC 27853 reference strains in accordance with CLSI [8], recommendations. Additionally, a previously characterized metallo-β-lactamase-producing *Pseudomonas aeruginosa* clinical isolate was included as a quality control strain.

Phenotypic detection of extended-spectrum β-lactamases (ESBLs) was performed using the CLSI-recommended combined disk method with cefotaxime (30 μg) and ceftazidime (30 μg), alone and in combination with clavulanic acid. ESBL production was considered positive when an increase of ≥ 5 mm in the inhibition zone diameter was observed in the presence of clavulanate.

Plasmid-mediated AmpC β-lactamase production was detected using the boronic acid disk potentiation method described by Bakthavatchalam et al. [9]. Enterobacteriaceae isolates were tested with cefoxitin (30 μg) alone and cefoxitin (30 μg) supplemented with boronic acid. The disks were placed on Mueller–Hinton agar plates previously inoculated with the bacterial suspension adjusted to a 0.5 McFarland standard and incubated at 35 ± 2°C for 18–24 h. An increase of ≥5 mm in the inhibition zone diameter around the cefoxitin-boronic acid disk compared with the cefoxitin disk alone was interpreted as positive for AmpC β-lactamase production.

Data Processing and Statistical Analysis:

Data were entered into an electronic database and analyzed using descriptive statistics. Absolute and relative frequencies were calculated to describe the distribution of bacterial species and antimicrobial susceptibility profiles.

Results were summarized in tables and figures to illustrate the frequency of bacterial isolates and their susceptibility patterns to the antimicrobial agents tested. Statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS®).

Ethical Considerations.

This study was conducted using microbiological data generated during routine diagnostic procedures at a reference microbiology laboratory in Mérida, Venezuela. All patient-related information was anonymized and handled confidentially to ensure privacy and data protection.

The study was conducted in accordance with the ethical principles for research involving human subjects established in the Declaration of Helsinki and its subsequent amendments. Data were used exclusively for scientific and academic purposes.

Results.

Between January 2017 and May 2021, a total of 66 clinical specimens obtained from patients attending a microbiology laboratory in Mérida, Venezuela, were analyzed. Among these samples, 39 clinically relevant Enterobacterales isolates were recovered, including *E. coli* (n=17; 43.6%) and *Enterobacter* spp. (n=22; 56.4%).

Of the 17 *E. coli* isolates, most were recovered from urine samples (10/17; 58.8%), whereas the remaining isolates originated from wound secretions (7/17; 41.2%) (Table 1).

The distribution of *Enterobacter* species revealed a predominance of isolates recovered from nasal secretions and skin-related specimens. *Enterobacter* spp. was the most frequently identified group (36.36%), being mainly isolated from nasal secretions, whereas *E. cloacae* complex (22.73%) exhibited the broadest distribution across clinical specimens, including respiratory, skin, and vaginal samples. Likewise, *E. vulneris* and *E. agglomerans* were predominantly recovered from upper respiratory tract specimens, suggesting either colonization or involvement in localized infections at these anatomical sites. In contrast, *E. sakazakii* showed the lowest frequency (9.09%) and was detected only in urine and skin samples. These findings highlight the ability of *Enterobacter* species to colonize and infect multiple anatomical sites in community patients.

Ten distinct antimicrobial susceptibility profiles were identified among the 17 *E. coli* isolates recovered from urine and wound secretion samples, revealing substantial phenotypic diversity in resistance patterns. Profiles 1 and 2 were the most prevalent, each accounting for 17.6% (3/17) of the isolates. Profiles 6, 7, and 8 represented 11.8% (2/17) each, whereas the remaining profiles were observed in a single isolate (5.9%) (Table 3).

Profile 1 exhibited broad susceptibility to β-lactam/β-lactamase inhibitor combinations, third- and fourth-generation cephalosporins, carbapenems, monobactams, aminoglycosides, and fluoroquinolones, while remaining resistant only to ampicillin, cefuroxime, and trimethoprim-sulfamethoxazole. This pattern suggests limited antimicrobial selective pressure and the absence of broad-spectrum resistance mechanisms. In contrast, Profile 2 displayed resistance to β-lactamase inhibitor combinations, aminoglycosides, fluoroquinolones, and trimethoprim-sulfamethoxazole, while maintaining susceptibility only to third- and fourth-generation cephalosporins, monobactams, and carbapenems, consistent with a multidrug-resistant (MDR) phenotype.

Profiles 3, 4, and 5 demonstrated resistance to third-generation cephalosporins and aztreonam while retaining susceptibility to carbapenems and cefepime, a pattern consistent with ESBL production. Together, these profiles accounted for 17.7% (3/17) of all isolates, indicating the presence of potential ESBL-producing strains among community-acquired infections.

Profiles 7 and 10 exhibited simultaneous resistance to aminoglycosides, fluoroquinolones, aminopenicillins, and trimethoprim-sulfamethoxazole, representing MDR phenotypes with significant therapeutic implications due to the reduction of available oral treatment options. Likewise, Profile 9 showed resistance restricted to fluoroquinolones while preserving susceptibility to the remaining antimicrobial classes tested.

Eight distinct antimicrobial susceptibility profiles were identified among the 22 Enterobacter spp. isolates, demonstrating substantial phenotypic diversity in resistance patterns. Profile 2 was the most prevalent, accounting for 36.4% (8/22) of the isolates, followed by Profile 7 with 18.2% (4/22), whereas the remaining profiles occurred at frequencies ranging from 4.5% to 13.6% (Table 4).

Profile 2 was characterized by susceptibility to third- and fourth-generation cephalosporins, monobactams, carbapenems, aminoglycosides, and fluoroquinolones, with resistance limited to β-lactam/β-lactamase inhibitor combinations and trimethoprim-sulfamethoxazole. This pattern suggests a relatively low burden of acquired resistance mechanisms and represents the most favorable therapeutic profile observed in the study.

The second most common profile was Profile 7 (18.2%), which exhibited susceptibility to all antimicrobial classes tested except β-lactamase inhibitor combinations. This finding is consistent with the intrinsic or inducible resistance associated with chromosomal AmpC β-lactamase production, a well-recognized characteristic of Enterobacter species. The high prevalence of this profile indicates that resistance to ampicillin/sulbactam and amoxicillin/clavulanic acid is common among the isolates evaluated.

Furthermore, Profiles 1, 6, and 8 exhibited resistance patterns consistent with multidrug-resistant (MDR) phenotypes. Profile 1 demonstrated concurrent resistance to third-generation cephalosporins, β-lactamase inhibitor combinations, and trimethoprim-sulfamethoxazole, while retaining susceptibility to cefepime and carbapenems, a pattern suggestive of AmpC β-lactamase hyperproduction. Profiles 6 and 8 displayed concomitant resistance to multiple antimicrobial classes, including aminoglycosides, fluoroquinolones, β-lactamase inhibitor combinations, and trimethoprim-sulfamethoxazole, thereby meeting the criteria for multidrug resistance.

Table 1. *Distribution of E. coli isolates according to clinical specimen type.*

Clinical specimen type	Number of isolates (n)	Percentage (%)
Urine	10	58.8
Wound secretions	7	41.2
Total	17	100.0

Table 2. *Distribution of Enterobacter species according to clinical specimen type.*

Species	Clinical specimen source	n	%
Enterobacter spp. (unidentified species)†	Nasal secretion (5), Pharyngeal exudate (1), Skin (1), Skin secretion (1)	8	36.36
Enterobacter cloacae complex	Nasal secretion (3), Skin (1), Vaginal secretion (1)	5	22.73
Enterobacter vulneris	Nasal secretion (3), Skin (1)	4	18.18
Enterobacter agglomerans	Nasal secretion (2), Pharyngeal exudate (1)	3	13.64
Enterobacter sakazakii	Urine (1), Skin (1)	2	9.09
Total		22	100.00

† Enterobacter spp. refers to isolates that could not be identified to the species level.

Table 3. *Antimicrobial susceptibility profiles and probable resistance phenotypes of E. coli isolates recovered from urine and wound secretions.*

Profile	n (%)	Antimicrobial susceptibility pattern	Antimicrobial resistance pattern	Resistance phenotype
Profile 1	3 (17.6)	SAM, AMC, CTX, CAZ, CRO, FEP, AZT, IMP, MER, GEN, AK, CIP, LEV	AMP, CXM, SXT	Low-level acquired resistance (Non-MDR)
Profile 2	3 (17.6)	CTX, CAZ, CRO, FEP, AZT, IMP, MER	SAM, AMC, AMP, CXM, GEN, AK, CIP, LEV, SXT	Multidrug-resistant (MDR)
Profile 3	1 (5.9)	FEP, IMP, MER, GEN, AK	SAM, AMC, AMP, CTX, CAZ, CRO, AZT, CIP, LEV, CXM, SXT	ESBL producer + MDR
Profile 4	1 (5.9)	FEP, AZT, IMP, MER, GEN, AK, CIP, LEV	SAM, AMC, AMP, CTX, CAZ, CRO, CXM, SXT	ESBL producer
Profile 5	1 (5.9)	FEP, AZT, IMP, MER, GEN, AK	SAM, AMC, AMP, CTX, CAZ, CRO, CIP, LEV, CXM, SXT	ESBL producer + MDR
Profile 6	2 (11.8)	CTX, CAZ, CRO, FEP, AZT, IMP, MER, GEN, AK, CIP, LEV	SAM, AMC, AMP, CXM, SXT	Resistance associated with β-lactamase inhibitor combinations (Non-MDR)
Profile 7	2 (11.8)	CTX, CAZ, CRO, FEP, AZT, IMP, MER, CIP, LEV	SAM, AMC, AMP, CXM, GEN, AK, SXT	Multidrug-resistant (MDR)
Profile 8	2 (11.8)	CTX, CAZ, CRO, FEP, AZT, IMP, MER, GEN, AK, CIP, LEV, SXT	SAM, AMC, AMP, CXM	Resistance to aminopenicillins and β-lactamase inhibitor combinations (Non-MDR)
Profile 9	1 (5.9)	CTX, CAZ, CRO, FEP, AZT, IMP, MER, GEN, AK, SXT	AMP, CXM, CIP, LEV	Fluoroquinolone-resistant phenotype
Profile 10	1 (5.9)	CTX, CAZ, CRO, FEP, AZT, IMP, MER, SXT	SAM, AMC, AMP, CXM, GEN, AK, CIP, LEV	Multidrug-resistant (MDR)

Abbreviations: *AMP, ampicillin; SAM, ampicillin/sulbactam; AMC, amoxicillin/clavulanic acid; CXM, cefuroxime; CTX, cefotaxime; CAZ, ceftazidime; CRO, ceftriaxone; FEP, cefepime; AZT, aztreonam; IMP, imipenem; MER, meropenem; GEN, gentamicin; AK, amikacin; CIP, ciprofloxacin; LEV, levofloxacin; SXT, trimethoprim-sulfamethoxazole; ESBL, extended-spectrum β-lactamase; MDR, multidrug-resistant.*

Table 4. *Antimicrobial susceptibility profiles and probable resistance phenotypes of Enterobacter spp. isolates recovered from different clinical specimens (n = 22).*

Profile	n (%)	Antimicrobial susceptibility pattern	Antimicrobial resistance pattern	Resistance phenotype
Profile 1	2 (9.1)	FEP, AZT, IMP, MER, GEN, AK, CIP, LEV	AMC, SAM, CTX, CRO, SXT	AmpC hyperproduction + MDR
Profile 2	8 (36.4)	CTX, CRO, FEP, AZT, IMP, MER, GEN, AK, CIP, LEV	AMC, SAM, SXT	Resistance to β-lactamase inhibitor combinations and TMP-SMX (Non-MDR)
Profile 3	3 (13.6)	AMC, SAM, CTX, CRO, FEP, AZT, IMP, MER, GEN, AK, CIP, LEV	SXT	Isolated trimethoprim-sulfamethoxazole resistance
Profile 4	1 (4.5)	AMC, SAM, CTX, CRO, FEP, AZT, IMP, MER, CIP, LEV, SXT	GEN, AK	Aminoglycoside-resistant phenotype
Profile 5	2 (9.1)	CTX, CRO, FEP, AZT, IMP, MER, CIP, LEV, SXT	AMC, SAM, GEN, AK	Resistance to β-lactamase inhibitor combinations and aminoglycosides
Profile 6	1 (4.5)	CTX, CRO, FEP, AZT, IMP, MER	AMC, SAM, GEN, AK, CIP, LEV, SXT	Multidrug-resistant (MDR)
Profile 7	4 (18.2)	CTX, CRO, FEP, AZT, IMP, MER, GEN, AK, CIP, LEV, SXT	AMC, SAM	Resistance to β-lactamase inhibitor combinations (Non-MDR)
Profile 8	1 (4.5)	CTX, CRO, FEP, AZT, IMP, MER, SXT	AMC, SAM, GEN, AK, CIP, LEV	Multidrug-resistant (MDR)

Abbreviations: *AMC, amoxicillin/clavulanic acid; SAM, ampicillin/sulbactam; CTX, cefotaxime; CRO, ceftriaxone; FEP, cefepime; AZT, aztreonam; IMP, imipenem; MER, meropenem; GEN, gentamicin; AK, amikacin; CIP, ciprofloxacin; LEV, levofloxacin; SXT, trimethoprim-sulfamethoxazole; MDR, multidrug-resistant; AmpC, AmpC β-lactamase.*

Profiles 3, 4, and 5 exhibited more limited resistance patterns. Profile 3 showed resistance exclusively to trimethoprim-sulfamethoxazole, whereas Profile 4 was resistant only to aminoglycosides. Profile 5 demonstrated combined resistance to β-lactamase inhibitor combinations and aminoglycosides while maintaining susceptibility to cephalosporins, carbapenems, and fluoroquinolones.

Importantly, all profiles remained susceptible to carbapenems (imipenem and meropenem), indicating the absence of phenotypes compatible with carbapenemase production among the isolates analyzed. Likewise, most isolates retained susceptibility to cefepime, suggesting that this antimicrobial agent continues to be an effective therapeutic option for infections caused by *Enterobacter* spp. in the study population.

Overall, these findings demonstrate a high prevalence of resistance to β-lactamase inhibitor combinations and a low frequency of resistance to carbapenems. However, the identification of isolates exhibiting multidrug-resistant phenotypes highlights the need to strengthen microbiological surveillance and maintain continuous monitoring of antimicrobial susceptibility patterns in community-acquired infections.

Discussion.

The findings of the present study revealed distinct distributions of bacterial genera according to the clinical source of the specimens. Although Escherichia coli accounted for 43.6% of all isolates, it was the predominant pathogen recovered from urinary samples (58.8% of *E. coli* isolates), whereas *Enterobacter* spp. represented the most frequently isolated group from skin infections and wound secretions. These results are consistent with the well-established role of *E. coli* as the leading etiological agent of community-acquired urinary tract infections (UTIs), particularly among sexually active women and older adults, where Enterobacterales remain the most commonly isolated pathogens [10,11].

The antimicrobial susceptibility profiles observed among *E. coli* isolates demonstrated high susceptibility to cefepime, aztreonam, imipenem, and meropenem in both urinary and wound-derived isolates. However, a high frequency of resistance to ampicillin, cefuroxime, and trimethoprim-sulfamethoxazole was detected. These findings are consistent with reports from Spain and several Latin American countries, where a progressive decline in the effectiveness of aminopenicillins and trimethoprim-sulfamethoxazole has been documented for the empirical treatment of community-acquired UTIs caused by *E. coli* [12,13]. In particular, Aguinaga et al. reported elevated resistance rates to amoxicillin-clavulanic acid and trimethoprim-sulfamethoxazole, recommending caution in their empirical use in specific geographical settings [12].

The preservation of susceptibility to carbapenems among all *E. coli* isolates represents a clinically relevant finding. Several studies have documented the increasing emergence of carbapenemase-producing Enterobacterales in Latin America and Europe, particularly among *E. coli* and *Klebsiella pneumoniae*, posing a major public health threat due to the progressive reduction of effective therapeutic options [14,15]. In contrast, none of the susceptibility profiles identified in the present study were compatible with carbapenemase production.

Regarding the genus *Enterobacter*, a higher frequency of isolation was observed in wound and skin specimens, with Enterobacter spp. and *Enterobacter cloacae* complex being the most prevalent species. Although these organisms have traditionally been regarded as opportunistic pathogens associated with healthcare settings, increasing evidence indicates their growing involvement in community-acquired infections, particularly skin and soft tissue infections as well as contaminated wounds [16,17]. This trend may be associated with the remarkable environmental adaptability of these microorganisms and their ability to colonize multiple ecological niches.

AmpC β-lactamases constitute one of the most clinically relevant resistance mechanisms among Enterobacterales, particularly in species such as the *Enterobacter cloacae* complex, *Klebsiella aerogenes*, and *Citrobacter freundii*. These enzymes are capable of hydrolyzing third-generation cephalosporins and β-lactam/β-lactamase inhibitor combinations, when expressed at high levels, thereby compromising their therapeutic efficacy. In organisms harboring inducible chromosomal ampC genes, exposure to β-lactam antibiotics may trigger gene derepression and subsequent AmpC overproduction, leading to the emergence of resistance during antimicrobial therapy. Consequently, cefepime and carbapenems are generally considered more reliable treatment options because of their greater stability against AmpC-mediated hydrolysis [18].

In the present study, the antimicrobial susceptibility profiles of *Enterobacter* spp. isolates demonstrated preserved activity of third- and fourth-generation cephalosporins, monobactams, and carbapenems. In contrast, a consistent resistance pattern was observed against β-lactam/β-lactamase inhibitor combinations, particularly amoxicillin-clavulanic acid and ampicillin-sulbactam. This phenotypic behavior is highly suggestive of inducible chromosomal AmpC β-lactamase production, an intrinsic resistance mechanism widely recognized among *Enterobacter* species. The retention of susceptibility to cefepime and carbapenems in most isolates further supports this hypothesis and indicates that these agents remain effective therapeutic alternatives for infections caused by *Enterobacter* spp. within the studied population .Moreover, these findings emphasize the importance of continuous surveillance of emerging resistance mechanisms and the implementation of antimicrobial stewardship strategies to reduce the selection and dissemination of resistant strains [19].

Additionally, some susceptibility profiles exhibited MDR phenotypes characterized by simultaneous resistance to aminoglycosides, fluoroquinolones, β-lactamase inhibitor combinations, and trimethoprim-sulfamethoxazole. The presence of these MDR phenotypes is clinically relevant because multidrug-resistant Enterobacterales have been associated with treatment failure, prolonged hospitalization, and increased healthcare costs [20]. Although the frequency of MDR isolates was relatively low, these findings underscore the importance of maintaining continuous microbiological surveillance in community settings.

The isolation of a single *Enterobacter* spp. strain from a pharyngeal exudate, which exhibited complete susceptibility to

all β-lactam agents tested and resistance only to trimethoprim-sulfamethoxazole, is consistent with previous reports describing occasional colonization of the upper respiratory tract by Enterobacterales in both ambulatory individuals and healthcare personnel [21]. Nevertheless, the low frequency of respiratory isolation observed in this study suggests a limited role of these microorganisms in community-acquired respiratory infections within the studied population.

Finally, the absence of carbapenem resistance among both *E. coli* and Enterobacter spp. contrasts with reports from Venezuelan hospitals and other Latin American countries, where carbapenemase-producing Enterobacterales, including KPC- and metallo-β-lactamase-producing strains, have been increasingly documented [22]. This discrepancy is likely attributable to the community origin of the isolates analyzed and the lower selective pressure exerted by last-line antimicrobial agents outside hospital environments.

Authorship contribution statement.

M.E.A.: Conceptualization (supporting); Methodology (lead); Formal analysis (equal); Investigation (equal); Writing – original draft (lead); Writing – review and editing (equal); Project administration (lead); Visualization (lead); Supervision (lead).

M.A.A.B.: Investigation (equal); Writing – original draft (supporting); Writing – review and editing (equal); Project administration (supporting); Visualization (supporting).

A.C.G.R.: Conceptualization (lead); Funding acquisition (lead); Investigation (equal); Project administration (supporting).

B.T.G.A.: Investigation (equal).

E.I.Q.R.: Investigation (equal).

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