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CASE REPORT

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Morphological variability of *Escherichia coli* colonizing human wounds: a case report

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Abstract

Background *Escherichia coli*, known for its adaptability, can cause diverse infections. Morphological variants, such as smooth and mucoid forms, correspond to different infection capabilities and antibiotic resistance profiles. This report presents the isolation of two distinct *E. coli* strains, a smooth strain and a mucoid strain, from a single patient.

Case presentation A 70-year-old woman with a leg wound and lung infection was found to have two *E. coli* strains: a mucoid strain from her wound and sputum and a smooth strain from her rectal swab. Whole-genome sequencing confirmed genetic similarity between the strains with minor SNPs linked to their morphological differences. Both strains were resistant to β-lactam and quinolone antibiotics, complicating treatment. The patient recovered following treatment with Piperacillin/Tazobactam and regular wound care.

Conclusion This case highlights E. coli's phenotypic plasticity within a single host, impacting infection management and antibiotic response. Understanding the genetic basis of such morphological changes could inform more effective treatment strategies.

Keywords *Escherichia coli*, Mucoid morphology, Smooth morphology, Phenotypic plasticity, Whole-genome sequencing, Antibiotic resistance

Background

Escherichia coli is a versatile and ubiquitous bacterium that can colonize both the intestines of healthy individuals and various environmental niches. While most strains are harmless, some variants can cause serious infections, especially in immunocompromised patients. *E. coli* can exhibit diverse colony morphologies, such as smooth, rough, and mucoid forms, depending on environmental and genetic factors. The smooth form is often associated with typical commensal or pathogenic strains, while the mucoid form, characterized by a thick polysaccharide capsule, is linked to increased resistance to phagocytosis and antibiotics, as well as persistence in chronic infections [1]. Similar patterns have been observed in *Klebsiella pneumoniae*, where different morphotypes exhibit distinct adaptations to environmental conditions. The red, dry and rough morphotype is more suited to hospital

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environments due to its increased environmental resistance, while the moist, hypervirulent strains exhibit higher virulence, often causing more severe clinical infections [2]. This pattern has also been observed in urinary tract infections, where mucoid *K. pneumoniae* strains evolved to exhibit enhanced virulence during infection [3].

This study reports the simultaneous isolation of smooth and mucoid *E. coli* from a single patient with bacteremia. While phenotypic variation in *E. coli* is known, the coexistence of these two morphotypes in one host raises concerns for infection management. The mucoid variant is often linked to chronic, difficult-to-treat infections due to its enhanced ability to evade host immune responses [4]. Although instances of *E. coli* infecting the human respiratory tract have been documented [5], there have been no definitive reports of the mucoid phenotype infecting the lower respiratory tract. WGS confirmed that these two morphotypes are genetically homologous, suggesting that *E. coli* can rapidly switch phenotypes within the host environment, potentially contributing to the persistence and recurrence of infections.

The phenotypic plasticity of *E. coli* poses challenges for infection control, especially due to the mucoid variant's resistance to antibiotics and immune defenses. Understanding the genetic mechanisms behind this shift is crucial for improving treatment strategies.

Case presentation

A 70-year-old female presented with chest distress and was diagnosed with valvular heart disease, requiring surgery. A wound on her left lower leg, caused by trauma ten days earlier, had not healed, showing redness, swelling, and purulent discharge. The patient exhibited no systemic symptoms except for fever, and her medical history was unremarkable.

Two distinct *E. coli* strains—smooth and mucoid—were isolated from the patient's wound exudate, indicating an ESBL-producing infection. Concerned about additional infection sites, sputum and rectal swabs were also cultured. The sputum sample yielded the mucoid *E. coli*, while the rectal swab yielded the smooth strain. Drug resistance testing and WGS were conducted on the four isolated *E. coli* strains (two from the wound, one from the sputum, and one from the rectal swab). Virulence assays using *Galleria mellonella* were also performed to assess differences in pathogenicity.

Examination revealed an inflamed, tender wound on the left leg. Laboratory tests showed elevated CRP (36.7 mg/L) and IL-6 (29.1 pg/ml), confirming a skin and soft tissue infection. The patient was treated with intravenous Piperacillin/Tazobactam and daily wound care. CRP levels decreased steadily, and after heart valve surgery, the patient's condition stabilized. One month later, follow-up confirmed complete wound healing.

The genome assemblies for the four isolates (*E. coli* 40143-1, 40143-2, 50009, and XYD-305) have been deposited in GenBank under the accession numbers JBJIAC000000000, JBJIAD000000000, JBJIAE000000000, and JBJIAF000000000, respectively.

Discussion

This case describes two distinct *E. coli* strains, mucoid and smooth, isolated from a single patient. The lung infection was likely caused by the mucoid strain, which is associated with high virulence and resistance to immune defenses [4]. In contrast, the smooth strain was isolated from the patient's rectal swab, suggesting gut colonization without gastrointestinal symptoms such as diarrhea.

Two distinct *E. coli* strains, 40143-1 (mucoid) and 40143-2 (smooth), were isolated from the patient's wound. Strain 40143-1 exhibited a mucoid colony morphology with a raised center, while 40143-2 had a smooth, moist colony (Fig. 1). Virulence testing in the *G. mellonella* model showed that 40143-2 exhibited lower virulence than 40143-1, and both strains had slightly lower virulence levels compared to the CR-HvKP control strain HvKP4 from our previous study [6] (Fig. 2). Both strains exhibited resistance to β-lactam and quinolone antibiotics, posing treatment challenges (Table 1).

The coexistence of two distinct *E. coli* strains raises questions about their potential interaction and infection dynamics. We hypothesize that the mucoid strain from lungs may have migrated to the wound through hematogenous dissemination or direct contamination during medical procedures, while direct contamination during wound care or invasive procedures is also possible. The phenotypic plasticity of *E. coli* is also mentioned in earlier studies, suggesting that selective pressures like antibiotics or immune responses may drive morphological adaptations, enabling colonization across different tissues [7]. Adaptive changes in *E. coli* morphology within the wound may be linked to the patient's prior antibiotic treatment. The role of colonization in wounds not only involves bacterial presence but also exacerbates inflammation and delays healing. Recent studies have shown how commensal bacteria can impair skin barrier repair through prolonged inflammation. In this case, the colonization of smooth and mucoid E. coli strains may have contributed to a pro-inflammatory environment, complicating treatment and recovery [8].

Reports of *E. coli* infections across tissues highlight its phenotypic plasticity and varying virulence. The smooth strain colonizes the gut without causing symptoms, while the mucoid strain, likely responsible for the lung infection, is linked to resistance to phagocytosis and antibiotics, making it a significant hospital risk [9]. Virulence

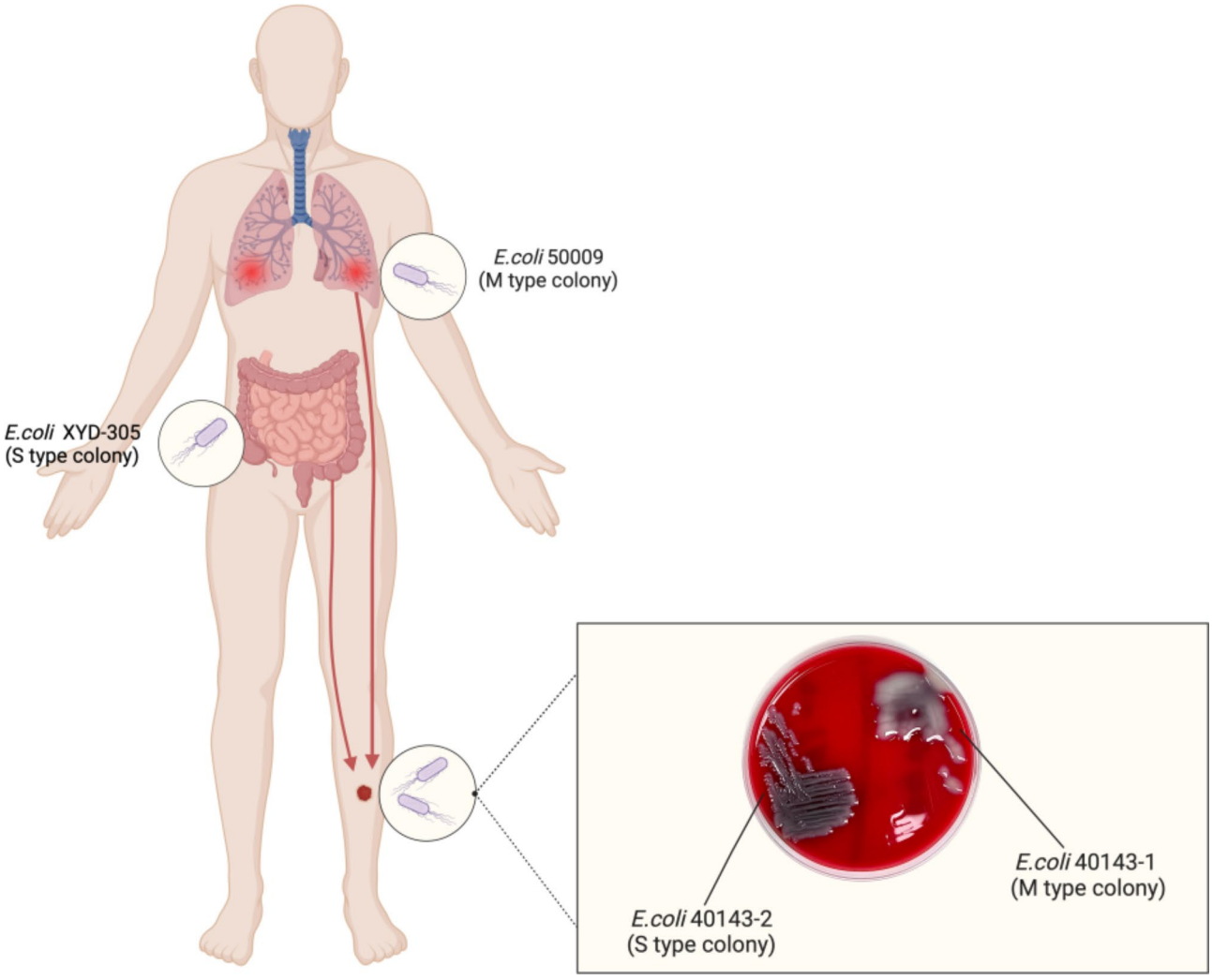

Fig. 1 E. coli translocation process in patients

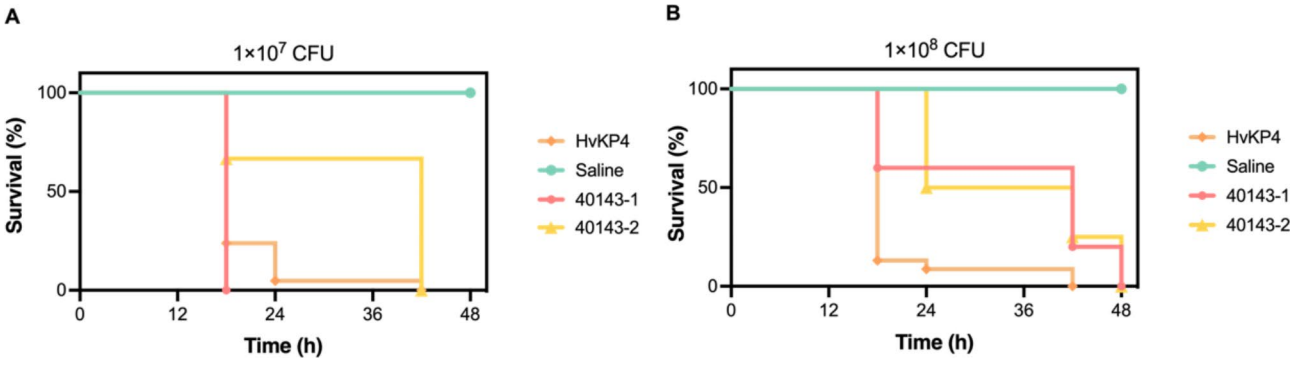

Fig. 2 The virulence evaluation of *E. coli* isolated from patient. (**A**) Survival graph of wax moth larvae that have been challenged by a dose of *E. coli* (10^7 CFU/mL). (**B**) Survival graph of wax moth larvae that have been challenged by a dose of *E. coli* (10^8 CFU/mL). Graphs are representative of 3 independent experiments

Table 1 Antimicrobial susceptibility testing of *E. Coli* isolated from patient (μg/mL)

Strains	XYD305	50,009	40143-2	40143-1
IPM	< 1	< 1	< 1	< 1
MEM	< 1	< 1	< 1	< 1
ETP	< 2	< 2	< 2	< 2
CMZ	< 2	< 2	< 2	< 2
CAZ	64	16	32	16
CTX	> 128	> 128	> 128	> 128
TZP	< 8/4	< 8/4	< 8/4	< 8/4
SCF	16/8	< 8/4	< 8/4	< 8/4
CAV	< 8/4	< 8/4	< 8/4	< 8/4
FEP	> 64	64	> 64	> 64
PB	< 0.5	< 0.5	< 0.5	< 0.5
TGC	< 0.25	< 0.25	< 0.25	< 0.25
CIP	> 32	> 32	> 32	> 32
AK	8	< 4	< 4	< 4
ATM	128	32	128	32

Abbreviations: IPM: Imipenem; MEM: Meropenem; ETP: Ertapenem; CMZ: Cefmetazole; CAZ: Ceftazidime; CTX: Cefotaxime; TZP: Piperacillin/Tazobactam; SCF: Cefoperazone/Sulbactam; CAV: Ceftazidime/Avibactam; FEP: Cefepime; PB: Polymyxin B; TGC: Tigecycline; CIP: Ciprofloxacin; AK: Amikacin; ATM: Aztreonam

testing in *G. mellonella* supports that the mucoid strain may be more pathogenic, as indicated by the patient's lung symptoms.

WGS revealed that smooth-capsulated strains (40143-2 and XYD305) and capsule-adherent strains (40143-1 and 50009) shared identical genes. SNP analysis confirmed they belonged to clonal groups. A four-amino-acid deletion in the *igaA* gene was found in the capsule-adherent strains (40143-1 and 50009), potentially affecting capsular polysaccharide biosynthesis and bacterial virulence and morphology (Fig. 3).

Genetic analysis revealed that, despite their phenotypic differences, the two *E. coli* were genetically homologous, suggesting environmental factors have driven the phenotypic divergence. Studies have demonstrated that *E. coli* can rapidly switch between smooth and mucoid forms in response to immune pressure or antibiotics, with the overproduction of capsular polysaccharides aiding survival in hostile environments [1, 10]. The concurrent isolation of two morphologically distinct strains raises questions about the factors influencing bacterial colony morphology in vivo. Mutations in genes like *bcsA* have been linked to changes in cellulose production and biofilm formation, contributing to the mucoid phenotype and bacterial adaptability [11, 12].

In conclusion, this case highlights the complexity of *E. coli* infections, especially when multiple strains with distinct phenotypes coexist in a single host. Understanding bacterial adaptability is crucial, as it influences infection progression and treatment outcomes. Further research is needed on the genetic and environmental factors driving phenotypic variation and their impact on infection control.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12879-025-10484-7.

Supplementary Material 1

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Not applicable.

Author contributions
Conceptualization: R.Z.; Methodology: X.W.; Data Curation: X.W., H.W; Formal Analysis: J.Z., X.W.; Investigation: R.Z., Y.H.; Writing – Original Draft Preparation: X.W., H.W; Writing – Review & Editing: X.W., H.W; Visualization: J.Z., Y.H.; Supervision: R.Z., Y.W.; Project Administration: Y.N.W., Y. W.; Funding Acquisition: R.Z., Y.H. All authors have read and agreed to the final version of the manuscript.

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Data availability
Sequence data that support the findings of this study have been deposited in the National Center for Biotechnology information (NCBI) with the primary accession code PRJNA1188054.

Declarations

Ethical approval and consent to participate
Ethical permission for this study was agreed by the Ethics Committee of The Second Affiliated Hospital Zhejiang University School of Medicine (2023 – 0733). The studies were conducted in accordance with the local legislation and institutional requirements. The human samples used in this study were acquired from a by- product of routine care. The manuscript presents research on animals that do not require ethical approval for their study.

Consent for publication
Written informed consent was obtained from the individual(s) for the publication of any potentially identifiable images or data included in this article (within Ethical Permission NO. 2023 – 0733).

Competing interests
The authors declare no competing interests.

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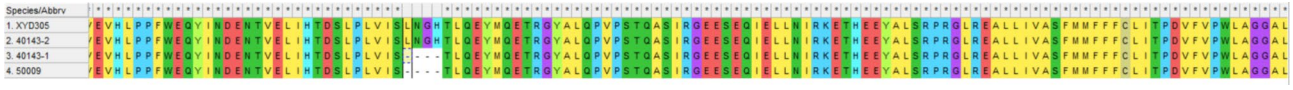

Fig. 3 Alignment of the amino acid sequences

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