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Case Report

Case Report on Cefiderocol-Non-Susceptible *Escherichia Coli* in the Absence of Carbapenemase Genes

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Abstract

Objectives: Cefiderocol is a relatively newer cephalosporin often a last line combat multidrug-resistant Gram-negative bacterium, especially carbapenem-resistant Enterobacterales (CRE). However, resistance has already been observed in several countries despite the recent years of clinical treatment.

Methods: Here we report a fatal case of carbapenem-resistant *Escherichia coli* with cefiderocol non-susceptibility isolated from an intra-abdomen surgical specimen of a renal transplant patient. This isolate was characterized using whole genome sequencing to assess for underlying resistant genes contributing to cefiderocol resistance.

Results: We have identified resistant genes putatively related to cefiderocol non-susceptibility and other antibiotic resistance including carbapenems. In comparison with current literature, several genes may be novel in the context of cefiderocol resistance in a carbapenem-resistant *E.coli*.

Conclusions: Genes related to cefiderocol resistance identified in this isolate include variants of iron-uptake genes (*cirA*, *fepA*, *fepB*, *fepG*, *tonB*), porin uptake genes (*ftsI*, *fecB*), and RNA polymerase gene (*pcnB*).

Keywords: Multi-drug resistant organisms; infectious disease; antibiotic resistance genes; molecular diagnostics.

1. Introduction

Antimicrobial resistance (AMR) is a major global public health threat attributing to 4.95 million deaths in 2019 [1]. The 2024 World Health Organization (WHO) bacterial priority pathogens has urged prioritization of critical group carbapenem-resistant Enterobacterales (CRE) [2]. CRE are a diverse group of pathogens with evolving resistance mechanisms and regional differences in susceptibilities [3].Resistance against the newer beta-lactam beta-lactamase inhibitors (BLBLI) such as ceftazidime-avibactam (CZA), and ceftolozane-tazobactam (CT) have severely limited choices of effective antimicrobials at our disposal [4-7].

Cefiderocol (FDC) holds promise as a last line of defence against extensively drug-resistant (XDR) and pan drug-resistant (PDR) gram negative bacteria. It can overcome most traditional resistance mechanisms (porin mutation, efflux pumps, beta-lactamases) with valuable effectiveness against metallo-beta-lactamases (MBLs) producers, when CZA and CT are ineffective. It is a novel cephalosporin-siderophore conjugate antibiotic with a unique catechol moiety allowing mimicry of naturally occurring siderophores. This allows FDC to 'hijack' TonB-dependent iron transporters (TBDT) and gain access into the periplasmic space in a 'trojan horse' fashion to bind to penicillin binding proteins (PBP) [8-11].

Cefiderocol resistance is uncommon but already reported amongst enterobacterales, and some in *Escherichia coli* too. Current characterization studies show an interplay of multiple mechanisms of resistance that are highly variable and complex. Primarily, FDC resistance relates to mutations affecting the TBDTs (eg, FepA, FecA, CirA, Fiu) and iron transport-related proteins (eg, CirA, FepA, Fiu) [12-15]. Target modifications to PBP-3 (four amino acid YRIN insertion) have been noted in *E.coli* [17]. Other identified targets related to porin expression/function (e.g., OmpC, OmpF) and overexpression of efflux pumps (e.g., MarR) [10-12,16].

The studies so far suggest FDC resistance is not correlated with specific mechanisms but requires combination of different mechanisms comprising predominantly co-expression of various beta-lactamases, mutations affecting siderophore-drug receptors and permeability defects [11]. Certain mechanisms have correlated with higher FDC MICs such as PBP3 mutations and NDM-producers [17-18].

Our current understanding of FDC resistance specifically in *E.coli* is limited. Available reports are mostly NDM producers and from screening specimens [15,20-21]. To our knowledge, our study is the first to characterize a clinical specimen of carbapenemase resistant *E.coli* with no identifiable carbapenemase gene and non-susceptibility to Cefiderocol. This *E.coli* co-incidentally has been identified as *Escherichia coli* ST410, a major dominant global virulent strain [22]. Interestingly, our case demonstrates no prior extensive exposure to beta-lactams or travel history outside of Hong Kong.

This study illustrates a combination of resistant genes contributing to extremely resistant phenotype, providing insights to decipher the complex interactions of these mechanisms in development of cefiderocol resistance.

2. Materials and Methods

2.1 Isolate collction and identification

Collection of isolates and access to clinical and laboratory data on the clinical case was approved by the Joint Chinese University of Hong Kong - New Territories East Cluster Clinical Research Ethics Committee (CREC#2019.013).

Escherichia coli (CUHK_ECOL4484BC_23) was isolated from Columbia blood agar (Thermo Fisher Scientific, USA) in aerobic condition at 37°C prior to antimicrobial susceptibility testing and whole genome sequencing. The isolate was noticed during an active surveillance and whole genome sequencing (WGS) program in our tertiary hospital. Species confirmation was performed using matrix-assisted-laser-desorption ionization time-of-flight mass spectrometry (MALDI-TOF) (Bruker Daltonics, USA).

2.2 Antimicrobial susceptibility testing

Antibiotic susceptibility testing was performed using double disk diffusion test according to CLSI standards with *E.coli* ATCC 25922 serving as control [23]. In vitro synergy between combination of ceftazidime-avibactam (CAZ-AVI) and aztreonam (ATM) was tested with disks containing CAZ-AVI and ATM (Becton Dickinson, USA) placed 15mm apart and incubated overnight at 37°C. Synergism is demonstrated when zones of inhibition around the ATM disks was enhanced towards the CAZ-AVI disc with characteristic 'keyhole' shape.

Synergism was also tested using CLSI broth disk elution method (BDE) [23]. This involves 4 tubes of 5ml Cation-Adjusted Mueller Hinton Broth (CAMHB) (Thermo Scientific, U.S.) Antibiotic disks (Thermo Scientific, U.S.) were placed into the tubes; one with 30μg ATM disk, one with 30/20μg ceftazidime-avibactam (CZA) disk and one with both ATM and CZA disks. The fourth tube had no disks serving as growth control. A 25μL standard inoculum with 0.5 McFarland turbidity standard was added into each of the four tubes, and incubated at 33-35°C in ambient air for 16 to 20 hours. Synergism was demonstrated if there was turbidity in the tubes with individual disks indicating bacterial growth, and no turbidity in the combined disk tube indicating no bacterial growth. The tests were run in parallel with CLSI recommended quality control strains.

2.3 Whole genome sequencing and analysis

Whole genome sequencing was performed as previously described [24] using Promega Wizard Genomic DNA extraction kit (Promega, USA), and the Riptide high throughput library preparation kit (Twist Bioscience, USA) for DNA extraction and library preparation respectively according to manufacturers' protocol. Library DNA was sequenced via Illumina platform Nextseq midoutput 500 with an average of 40x coverage. Short read sequences were assembled *de novo* via Spades (v.3.13.0) after trimming its reads with Trimmomatic (v.0.39). Contigs less than 500 bp were removed and the assembly was annotated with Prokka (v1.14.6). Sequences were matched to NCBI database, VFdB and Plasmidfinder to look for antimicrobial resistance genes (ARGs), virulence factors and plasmids. Plasmid DNA was extracted using Qiagen Plasmid Mini Kit (Qiagen, MD, USA). MinION library preparation kit was used according to manufacturer's protocol using a ligation sequencing kit and native barcoding expansion (Oxford Nanopore Technologies, UK). Library sequencing was performed by running the reaction on the flow cell (Rapid barcoding kit 96) for 24 hours. Long-read sequences were assembled according to manufacturer's instruction. Sequence reads were deposited in

NCBI Bioproject (PRJNA1036298).

3. Case report

This was a case of a middle-aged lady who was a renal transplant recipient with underlying Systemic Lupus Erythematosis on daily prednisolone and cyclosporin A. The last admission two years prior involved isolating extended spectrum beta-lactamase (ESBL) *E.coli* from urine for asymptomatic bacteriuria without any recent antimicrobial use (Figure 1, specimen E0). There was no other record of previous multidrug resistant organisms (MDRO) or carbapenemase-producing enterobacterales (CPE) isolated from screening or clinical specimens. Socially, there was no travel history outside of Hong Kong or admission to other hospitals.

A year later, she was admitted to hospital for Varicella Zoster Encephalitis treated with two weeks of acyclovir. She developed kidney graft failure requiring hemodialysis and admission to intensive care unit (ICU). Ertapenem was given during this time based on ESBL *E.coli* in catheterised urine (CSU) (Figure 1, specimen E1 and E2) Upon clinical improvement, she was able to transition to continuous ambulatory peritoneal dialysis (CAPD). However, feculent material was found in peritoneal dialysis fluid due to small bowel perforation and emergency small bowel resection was promptly performed. Intra-operative peritoneal swab culture yielded heavy growth of multi-drug resistant *E.coli* (Figure 1, specimen E3). This strain of *E.coli* was not susceptible to carbapenems and cefiderocol, but tested susceptible to aminoglycosides; gentamicin and amikacin. The same strain was also isolated later from abdomen surgical wound (Figure 1, specimen E4).

Further susceptibility testing demonstrated strain resistance to CZA and CT, but no synergism was noted between CZA and ATM. The strain was also resistant to fosfomycin, and only susceptible to tigecycline.

By this time antibiotics treatment had already escalated to empirical meropenem and linezolid. This was switched to combination therapy of tigecycline and piperacillin-tazobactam for coverage concomitant isolation of sensitive strain *Pseudomonas aeruginosa* from peritoneal wound swab (Figure 1, specimen E3), and Micafungin later added for coverage of *Candida albicans* isolated from surgical wound swab (Figure 1, specimen E4).

However, there was persistent bile fluid from abdomen drain and emergency laparotomy with double barrel ileostomy was performed just one week after the first operation. Peritoneal wound swab again yielded the same strain of *E.coli* (Figure 1, specimen E5). Additional doses with gentamicin and amikacin were also given in view of suboptimal clinical response. She remained in ICU afterwards with further deterioration and succumbed shortly thereafter. Polymerase chain reaction (PCR) detection for carbapenemase gene (*IMP*, *VIM*, *NDM*, *KPC*, *OXA*) was negative in routine microbiology screening. In addition, further testing at reference laboratory did not detect other carbapenemase genes (*GES*, *IMI*, *SME*, *NmcA*, *GIM*, *SIM*, and *SPM*).

4. Results

4.1 Genome characterization

WGS showed the *E.coli* strain E4 (CUHK_ECOL4484BC_23) belonged to ST410 with five ARGs found (bla_{CMY}, bla_{EC}, $bla_{CTX\text{-}M\text{-}55}$, $bla_{TEM\text{-}1}$, and *fosA*). Three replicon plasmids were found: IncI (with contig length of 40kb), IncF (38kb) and Col (4kb), where IncF carried $bla_{CTX\text{-}M\text{-}55}$, $bla_{TEM\text{-}1}$, and *fosA* genes. The strain was absent of CPE genes, which concurred with PCR results from routine laboratory screening.

Virulence factors related to cefiderocol resistance was looked at with reference to *E.coli* strain K-12 substr MG1655 (Accession No. GCA_000005845.2). We found six amino acid variants across the three genes, *cirA*, *fecB* and *ftsI* (Table 1). I547F was noted in cirA gene, while three mutations (L8V, I57S, and A134T) were found in *fecB* gene. An in-frame insertion (p.333-334insYRIK) and a mutation (A413V) were noted in *ftsI* gene. We then looked at other genes related to iron uptake (*fepA*, *fepC*, *fepG*, *marR* genes) and siderophore- iron uptake (*fepB*, *fecA*, *fhuA*, *exbB*, *exbD*, *tonB*, *envZ*, *baeS/R*, *ompR*, *envZ*, and *aroP* genes) [26] (Supplementary Table 1). Missense variants were found in genes *fecA*, *fepA*, *fepB*, *fepG*, *marR*, *pcnB*, *BaeS*, *BaeR*, and *tonB* where, to the best of our knowledge, most of them were not reported elsewhere.

Table 1. Mutations found in iron transport related genes *cirA, fecB,* and *ftsI* genes.

Gene	Nucleotide pairwise identity	Amino acid variant	Reference
cirA	99%	I547F	[21]
fecB	99.60%	L8V	Not reported[+]
		I57S	[21]
		A134T	Not reported[+]
ftsI	98.37%	p.P333-Y334insYRIK	[21] (ins. YRIN) [15] (both ins.YRIN and YRIK)
		A413V	[15]

[+]Reports of the variant was not found in literature

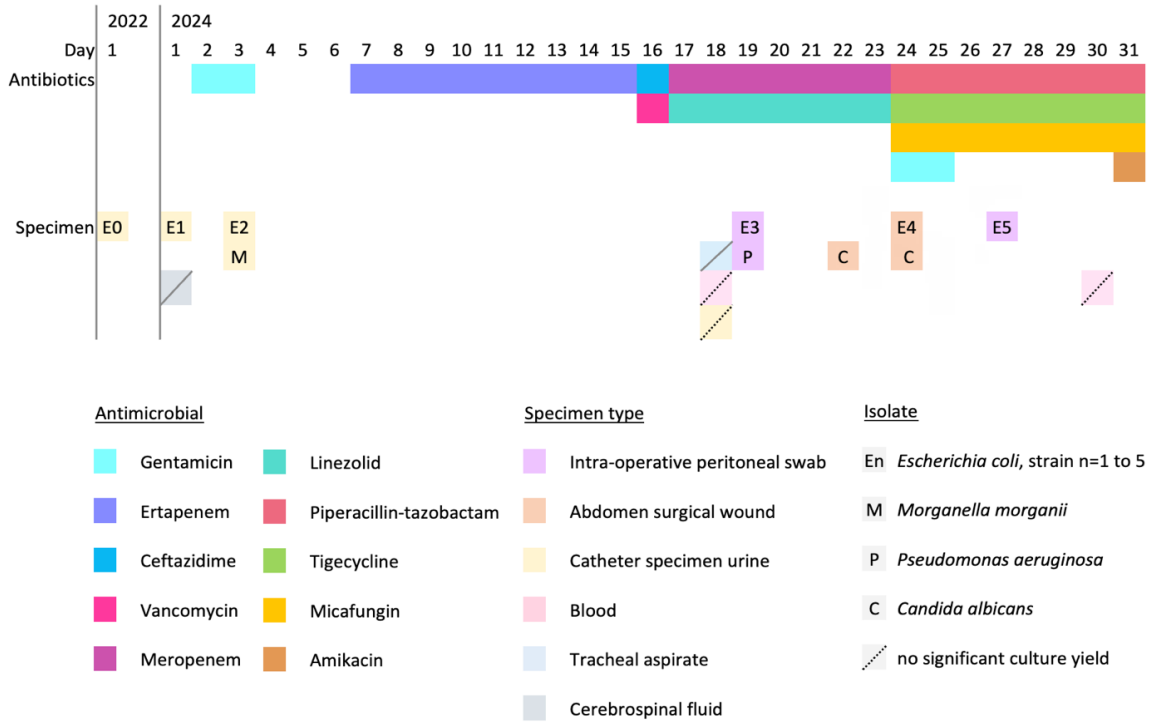

Figure 1: Timeline of clinical events, antimicrobial use and culture isolates. Two admissions were noted for the patient respectively in year 2022 and 2024. Antibiotic treatment during admission is noted in the key legend with colour coding, while the isolation of *E.coli* in various specimens (E0-E5) is indicated with colour coding of specimen type. Isolates of other bacterial species are included.

5. Discussion

5.1 Mutation of genes in Cefiderocol resistance E.coli and ST410

We report a lethal infection caused by *E.coli* ST410 with pan beta-lactam resistance, including FDC resistance and absence of carbapenemase genes despite no prior exposure to FDC.

Analysis of iron transporter genes showed mutation of *cirA* gene with amino acid substitution I547F. Previous studies with FDC non-susceptible isolates had premature stop codons [15,21]. It was also demonstrated that strains of *E.coli* harbouring deletions in both *cirA* and *fiu* result in 16-fold increase in minimal inhibition concentration (MIC) of FDC [12]. This was not seen in our case. We also identified mutations of in several genes relating to iron transport, DNA transcription, two component regulators and TBDT, where most of the variants were not previously reported in literature during our study. Further experiment involving site mutagenesis, gene editing and other functional studies will be necessary to illustrate the importance of these variants in *E.coli* resistance to FDC, but this is beyond the scope of our report.

Baseline non-susceptibility could be mediated by mutations in PBP3 and beta-lactamases blaCMY, blaCTX-M-55, and blaTEM-1. We have identified 4-amino-acid insertion of YRIN at FtsI which encodes for penicillin-binding protein 3 (PBP3 or FTSI) [25]. This was also seen in other studies with FDC resistant *E.coli* [10-11,15,19]. Specifically, we found a A417V substitution in PBP3, which is positioned opposite to PBP3 site hindering substrate binding with the bulkier Valine compared with Alanine [26-28].

The 4-amino-acid insertion of YRIN at FtsI has been shown to significantly reduce susceptibility to cephalosporins [15,26,29]. Reduced susceptibility due to the insertion was further concurred with a functional study where DH5-alpha cell (FDC MIC 0.064ug/ml) carrying the mutant showed increased resistance by four-fold (MIC 0.25 ug/ml), albeit it was overall sensitive [15]. Reports of two FDC susceptible isolates with this insertion sequence indicates that this mutation alone is insufficient to cause resistance [21]. Another insertion of YRIK and TIPY at the region also noted reduced susceptibility to cephalosporins (ceftazidimes, cefepime) and monobactams (aztreonam) [27,28,30].

Cefiderocol resistance was noted in ST167 and ST361 *E.coli* and tend to carry NDM genes [10,11,15,21]. Unlike most other studies, no carbapenemase gene was detected in our *E.coli* ST410 isolate, which raises the question on whether different strains have a propensity for different resistant mechanisms.

ST410 is an emerging multidrug clone with two major sub lineages in Europe and North America circulating since 1980 (B3/H24Rx) and 2003 (B4/H24RxC) respectively [31,32]. It has recently been reported to cause outbreaks in Chinese hospitals between 2017 to 2021, where the hypervirulent strain was lineage B with fimH24 (B5/H24RxC) and showed carbapenem resistance [33]. Our observation of *E.coli* ST410 carrying cefiderocol resistance in the absence of carbapenemase genes renders concern in the emergence of such resistance in the community.

6. Conclusion

In conclusion, cefiderocol resistance in *E.coli* is emerging in the absence of carbapenemase genes, and the resistance mechanism remains elusive. Further functional studies are required to unveil the complex cefiderocol resistance mechanisms, while active surveillance is necessary to monitor the progress of resistance in the community.

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Ethical statement: All patient information is anonymized, and identifiers removed to protect individual's identity and confidentiality.

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Author's contribution: Linda Chan- Clinical isolate, data collection and draft manuscript; Carmen Li - Experiment design and work, data analysis and draft manuscript, Jie Li- genome analysis, Wing Shan Lee- experiment work, Christopher KC Lai - manuscript review, Viola CY Chow- isolate collection and manuscript review, Margaret Ip- project leader and grantee.

Transparency Declaration: None to declare.

Supplementary information

Supplementary Table 1. Mutations in genes related to iron uptake/ siderophore-iron uptake.

Putative resistance mechanism	Gene	Nucleotide pairwise identity	Amino acid variant	Reference
Iron transport	*fecA*	99.8%	N186S	Not reported[+]
			T16A	Not reported[+]
	fepA	97.5%	K69Q	Not reported[+]
			S293A	Not reported[+]
			A312S	Not reported[+]
			I377L	Not reported[+]
			T420A	Not reported[+]
	fepB	97.4%	D312E	Not reported[+]
			M308T	Not reported[+]
	fepG	94.5%	V128I	Not reported[+]
			S121A	Not reported[+]
			L9I	Not reported[+]
DNA-binding transcriptional repressor of multiple antibiotic resistance	*marR*	98.2%	G103S	[19]
			Y137H	[19]
poly(A) polymerase	*pcnB*	99%	V87A	Not reported[+]
			E261D	Not reported[+]
Two component regulator	*baeR*	99.5%	Q184H	Not reported[+]
	baeS	98%	Q376K	Not reported[+]
			G380Q	Not reported[+]
			R382H	Not reported[+]
			D400Q	Not reported[+]
Membrane spanning protein in TonB-ExbB-ExbD transport complex	*tonB*	99.0%	V87A	Not reported[+]

[+]Reports of the variant was not found in literature

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