Table 1. *Spa* Types

***Spa*-type:**
# of isolates with valid value: 129
# of different values: 51

Item	# of Isolates	% of isolates with valid values
t002	16	12.4
t008	42	32.6
t012	1	0.8
t024	4	3.1
t062	1	0.8
t064	1	0.8
t067	1	0.8
t084	1	0.8
t088	4	3.1
t091	1	0.8
t105	1	0.8
t1107	1	0.8
t1154	1	0.8
t121	1	0.8
t1259	1	0.8
t1544	1	0.8
t1577	1	0.8
t160	1	0.8
t179	1	0.8
t18738	1	0.8
t18739	1	0.8
t18740	2	1.6
t18741	1	0.8
t18742	1	0.8
t197	3	2.3
t209	1	0.8
t211	2	1.6
t216	5	3.9
t233	1	0.8
t242	2	1.6
t267	1	0.8
t2724	1	0.8
t2743	1	0.8
t305	1	0.8
t3136	1	0.8
t3240	1	0.8
t334	6	4.7
t338	1	0.8
t355	1	0.8
t359	1	0.8
t363	1	0.8
t3732	1	0.8
t4277	1	0.8
t4454	1	0.8
t450	1	0.8
t4727	1	0.8
t539	1	0.8
t723	2	1.6
t779	1	0.8
t786	1	0.8
t9821	2	1.6

Typability

Item	# of Isolates used	# of Isolates rejected	# of Isolates not typable	Typability
Spa-type	129	0	0	100.0%

Discriminatory index

Item	# of Isolates	# of Isolates rejected	# of Different types	Discriminatory index	Confidence intervall (95% CI)
Spa-type	129	0	51	0.876	[0.827 - 0.925]

Disclosures. **All authors:** No reported disclosures.

234. Reversal of Carbapenem and Amikacin Susceptibilities in Isogenic Klebsiella pneumoniae From a Patient with Persistent Bacteriuria

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Background. Genomic tools permit a detailed analysis of antibiotic resistance determinants in bacteria, or resistome. Here we discuss variations in antibiotic resistance in *K. pneumoniae (Kp)* not explained by changes in the resistome

Methods. We compared *Kp* strains with divergent carbapenem and aminoglycoside susceptibilities. After identification of bacteria, antibiotic susceptibility testing was performed according to CLSI guidelines. Draft genome sequences were generated using Illumina MiSeq (Nextera paired-end library) and assembled using CLC Genomics Workbench (CLC bio, Cambridge, MA). Resistome, plasmid types and MLST were investigated using the CGE platform (http://cge.cbs.dtu.dk), while capsular type and virulence genes were investigated using the Pasteur BIGsDB database (https://bigsdb.pasteur.fr).

Results. While receiving amoxicillin-clavulanate, a 44-year old man with diabetes mellitus and paraplegia with neurogenic bladder grew*Kp* resistant to carbapenems and amikacin from urine. He was treated with fosfomycin and amikacin, followed by imipenem and plazomicin, prior to lithotripsy. Three months later, while off antibiotics, urine cultures grew *Kp* susceptible to carbapenems and amikacin (figure). Genetic comparison between resistant (November 20, 2018) and susceptible (January 30, 2019) strains revealed they were isogenic, only differing by 559 SNPs (table). Both were ST14, presented capsular type 16, and shared cehalosporinase (bla_{SHV-28}, $bla_{CTX-M-15}$, bla_{TEM-1B}, bla_{OXA-1}) and aminoglycoside modifying enzyme (AME) (*aph(3")-Ib, aph(6)-Id, aac(6')-Ib-cr*) genes. Although both had mutations in the outer membrane porin OmpK36, these differed (stop AA125 and frameshift AA183, respectively)

Conclusion. Carbapenem resistance in the initial *Kp* is likely explained by overexpression of cephalosporinases in combination with changes in membrane permeability, while amikacin resistance is likely due to AMEs. Since no significant gene variation was observed in the susceptible *Kp*, reversal of resistance was likely due to decreased expression of cephalosporinases and AMEs after antibiotics were stopped. Incorporation of antibiotic history and host factors can explain clinically important changes in antibiotic resistance

Table 1. Summary of genomic analysis of isogenic isolates

Date of isolation	11/20/18	1/30/19
ST	14	14
Virulence genes	kfuA 3, kfuB 3, kfuC 1 mrkA 3, mrkB 3, mrkC 3, mrkC 302, mrkD 1, mrkI 4, mrkJ 3	
Capsular type	K16	K16
Resistance genes	bla_{SHV-28}, $bla_{CTX-M-15}$, bla_{TEM-1B}, bla_{OXA-1}, fosA, sul2, tet(A), qnrB1, dfrA14, aph(3")-Ib, aph(6)-Id, aac(6')-Ib-cr	bla_{SHV-28}, $bla_{CTX-M-15}$, bla_{TEM-1B}, bla_{OXA-1}, fosA, sul2, tet(A), qnrB1, dfrA14, aph(3")-Ib, aph(6)-Id, aac(6')-Ib-cr, catB3, aac(3)-Iia
Plasmids	IncFIB(K), IncFII(K), IncL/M	IncFIB(K), IncFII(K), IncL/M
OmpK 35	WT	WT
OmpK36	Stop AA125	Del AA183 (frameshift)

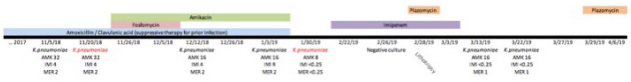

Figure 1. Timeline of antibiotic exposure and collected isolates. Isolates in red were selected for WGS. AMK: amikacin, IMI: imipenem, MER: meropenem.

Disclosures. **All authors:** No reported disclosures.

235. Next-Generation Sequencing for Investigation of Hospital Outbreak of Carbapenem--Resistant Klebsiella pneumoniae

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Background. Carbapenem--resistant Enterobacteriaceae constitute an urgent public health problem worldwide. In 2018, carbapenem--resistant *Klebsiella pneumoniae* (CR-KP) caused outbreaks of infection in 4 intensive-care units (ICUs)in a tertiary-care hospital in Egypt. We aimed to identify the clonal relatedness of isolates by whole genome (WGS).

Methods. Identification and antibiotic susceptibility testing was done by VITEK-2. Eleven isolates showed identical resistance pattern (resistant to Amikacin, gentamicin, Imipenem, meropenem, levofloxacin, and Piperacillin/Tazobactam) and were susceptible only to colistin. Caba-NP test was positive for carbapenemase production. The 11 isolates were studied by WGS by Illumina Miseq in a reference lab in Cairo University Hospital.

Results. In only one ICU, WGS identified 4 outbreak isolates of CR-KP that group together as a tight clonal cluster, suggestive of intra-ward transmission event. The outbreak isolates belonged to MLST 147. All isolates carried $bla_{CTXM-15}$, $bla_{oxa-48,}$ and bla_{NDM1} encoding ESBL and carbapenemase activity. Other identified resistance genes were Str, AadA, MsrE, Tet, and DfrA, encoding resistance to aminoglycosides, macrolide–lincosamide–streptogramin, tetracycline and trimethoprim/sulphonamides. Virulence genes included Yersiniabactin, aerobactin, rmpA, rmpA2 and wzi64, which has been associated with pathogenicity and hypervirulent *K. pneumoniae* lineages. No clonal relationships were identified between the isolates from other ICUs.

Conclusion. WGS is a powerful tool that goes beyond high-resolution tracking of transmission events into identifying the genetic basis of drug-resistance and virulence.

Disclosures. **All authors:** No reported disclosures.

236. The Comparative Utility Of Metagenomic Next-Generation Sequencing and Universal PCR for Pathogen Detection on Cerebrospinal Fluid: A Retrospective Analysis From a Tertiary Care Center

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