Screening haematology patients for carbapenem-resistant *Klebsiella pneumoniae*

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Abstract

Following a cluster of haematology patients with carbapenem-resistant *Klebsiella pneumoniae* (CRKP) septicaemia, we initiated screening for rectal carriage of CRKP and multidrug-resistant *K. pneumoniae* (MDRKP) in this patient group. Haematology inpatients submit a rectal swab once weekly. When plated onto chromogenic Brilliance™ UTI Agar (Oxoid), and incubated overnight with a 10 µg ertapenem disc (Oxoid), *K. pneumoniae* is identified and semi-automated antibiotic susceptibility testing is performed using the Vitek 2 analyser (Biomérieux). When no zone of inhibition occurs, immediate intervention through patient isolation and enhanced environmental cleaning can be instigated to control further spread while empirical antibiotic prescribing is adapted to take account of identified resistances. Over 2 years, six patients with CRKP and 20 patients with MDRKP were identified. These isolates were resistant to first-line empirical treatment choices for neutropenic sepsis and presented a clinical risk of treatment failure for sepsis post cytotoxic chemotherapy. We describe how this rectal screening methodology was developed and how the results influenced appropriate antibiotic prescribing, patient placement in single rooms and the cleaning of the ward environment to prevent person-to-person transmission of MDRKP and CRKP.

Background

Multidrug-resistant (MDR) *Enterobacteriaceae* (Magiorakos et al, 2012) have been increasingly recognised in Scotland (HPS, 2010) and carbapenem resistance has been observed locally (Findlay et al, 2011). This presents problems in accurate microbiological detection, identification and susceptibility testing, but also limits the treatment options

available to manage sepsis resulting from these organisms. There are public health concerns regarding accurate surveillance for such bacteria and a need for adequate containment of them to prevent health-care associated outbreaks (CDC, 2009; HPA, 2011).

Patients with fever $\geq 38.0^{\circ}\text{C}$ and neutrophils $< 0.5 \times 10^9/\text{L}$ should receive effective antibiotics within 1 hour of presentation (NHS Scotland, 2011; National Institute for Health and Clinical Excellence (NICE), 2012). In particular, Gram negative bacteraemia in neutropenic patients has been associated with a mortality of between 2 and 21% (NICE, 2012). Recognised empirical antibiotic regimens for Gram negative bacteraemia in neutropenic sepsis are an antipseudomonal β lactam agent, a carbapenem or piperacillin-tazobactam (Freifeld et al, 2011). Locally, when screening was introduced, first-line antibiotics for neutropenic sepsis were piperacillin-tazobactam with gentamicin, and second line was meropenem.

In October 2010 we detected a cluster of patients presenting to the clinical haematology unit with neutropenic Gram negative sepsis due to *Klebsiella pneumoniae* subspecies *pneumoniae* which was resistant to all antipseudomonal β lactam agents including piperacillin-tazobactam and was not fully susceptible to carbapenems except imipenem.

We recognised that by detecting carriage of this organism in the intestinal flora of patients prior to the development of neutropenic sepsis, we could adapt our neutropenic sepsis policy so that the first-line antibiotic choice for such patients was imipenem. The rationale for this was that it would avoid treatment delays, through receiving ineffective initial antibiotics, potentially reduce the risk of death from untreated sepsis and reduce selection pressures promoting growth of the resistant bacteria, all of which were considered to be unacceptable clinical risks. In addition, screening and identification of carriers helps to target infection control interventions that may prevent onward spread.

Figure 1. Blue colonies of a carbapenem-resistant *K. pneumoniae* grown on Brilliance™ UTI Agar demonstrating no zone of inhibition at all to ertapenem

This observational study describes the laboratory methodology used for screening this patient group for multidrug-resistant *K. pneumoniae* (MDRKP) and carbapenem-resistant *K. pneumoniae* (CRKP) and reports the prevalence of these organisms during the first 24 months of the screening programme.

The haematology ward is a purpose-built 20-bedded clinical haematology ward of which only 16 beds are currently utilised. The ward configuration is of three shared rooms of four beds each (one room of which is not currently used) and eight single rooms. The entire ward is under positive air pressure and the air is HEPA-filtered in all areas. All rooms have their own toilet and shower. The ward has its own regular cleaning staff.

Surveillance for gastrointestinal colonisation with CRKP is recognised as an important component of infection control protocols where infections due to such organisms have occurred (CDC, 2009). An existing method is cumbersome, labour intensive, takes a minimum of 48 hours to process and assumes that the *K. pneumoniae* will be resistant to imipenem (Landman et al, 2005). As such it would not detect the organism causing infections locally. In the absence of national guidance regarding how to screen for CRKP we designed our own method. Prior to October 2010 no screening of this patient group for CRKP was performed. The only bacteriological screening was for methicillin-resistant *Staphylococcus aureus* (MRSA) in accordance with the national MRSA screening programme that was implemented in Scotland in January 2010 (HPS, 2009).

Methods

The benefits of a screening programme were discussed initially at problem assessment group meetings, held to investigate the cluster of CRKP, by clinical haematology unit staff, infection control and microbiology staff and Health Protection Scotland. It was decided that taking rectal swabs would be easier than collecting stool samples for culture from patients and so, once per week, every inpatient in the clinical haematology unit has a rectal swab performed after it is explained that the test is to assess whether they have carriage of antibiotic-resistant bacteria which might influence the choice of antibiotics required should they become septic. Swabs are either self taken or taken by nursing staff. Swabbing occurs on a Tuesday to allow the laboratory to generate a result before the end of the working week. All patients are eligible for swabbing. No-one has refused to have it performed.

The implementation of the screening involved co-ordination between the clinical haematology unit, the infection control team and

the microbiology laboratory. As screening was launched, nursing staff were reminded at weekly Haematology Unit Multidisciplinary Team Meetings of the need to screen by the consultant microbiologist and opportunistically on the ward by the infection control nurses until this became standard practice. All consultant haematologists working on the ward and the senior nursing staff were aware of the need for antibiotic prescribing for neutropenic sepsis to be adapted if the patient was identified as a carrier of MDRKP. A confidential database of patients who were carriers was kept, and their electronic record in the hospital's patient tracking software was adapted with an alert status so that their carrier state would be alerted to medical and nursing staff at presentation when re-admitted. Once identified, carriers of MDRKP were accommodated in single rooms whether or not they were neutropenic. The cleaning frequency for these rooms was increased to twice daily rather than once daily as directed for a BI High Risk Inpatient Area in the National Cleaning Specification (HFS, 2009).

Rectal swabs were delivered to the microbiology laboratory and on the same day plated onto chromogenic agar (Brilliance™ UTI Agar, Oxoid). Chromogenic agar has been described as a means of effectively and rapidly selecting out Extended Spectrum β -Lactamase (ESBL)-producing bacteria from other bowel organisms growing from clinical samples (Reglier-Poupet et al, 2008). By growing the *K. pneumoniae* on chromogenic agar in the presence of a 10 μ g ertapenem antibiotic disc we are easily able to screen patients within 24 hours for *K. pneumoniae* which is resistant (i.e. no zone of inhibition) to ertapenem (Figure 1). If no zone of inhibition is seen this is immediately communicated to the consultant microbiologist to inform ward staff. If a zone of inhibition is seen, this cannot be interpreted as to whether there is carbapenem resistance or not, as illustrated in Figure 2.

Further antimicrobial susceptibility testing of *K. pneumoniae* isolates, regardless of any zone of inhibition, is performed using a Vitek 2 analyser (Biomérieux) which determines antimicrobial minimum inhibitory concentrations (MICs) using a semi-automated method. Final results are made available electronically to clinical staff via the laboratory results browser, accessible from all ward computers. If an MDRKP is identified this is communicated to the ward medical staff and senior charge nurse and the infection control team by telephone by the senior laboratory biomedical scientists or the consultant microbiologist. Antimicrobial resistance data is also sent electronically to Health Protection Scotland as part of mandatory national surveillance of antibiotic resistance (SAPG, 2010). When CRKP isolates were identified, the health protection team in the local Department of Public Health were also notified.

Figure 2. Disc diffusion antibiotic susceptibility testing using 10 µg meropenem disc (top) and 10 µg imipenem disc (bottom) using Clinical and Laboratory Standards Institute criteria. On the left, a pre-treatment meropenem-susceptible *K. pneumoniae* isolate (MIC for meropenem <0.25 mg/L) is shown, while on the right the *K. pneumoniae* has an MIC for meropenem of 8 mg/L, which arose in the same neutropenic patient being treated for *K. pneumoniae* septicaemia. The isolate on the right no longer is fully susceptible to meropenem despite having a visible zone of inhibition and also features in Figure 1 demonstrating resistance to ertapenem

Results

We have been performing screening for 2 years, during which 392 patients were screened. Twenty carriers of MDRKP (resistant to piperacillin-tazobactam, gentamicin and ciprofloxacin but carbapenem susceptible) were identified. Three (50%) carriers of CRKP were detected prior to the development of sepsis, and three (50%) carriers of CRKP were identified during episodes of neutropenic sepsis during which all three grew it from their blood cultures and rectal swabs concurrently, and CRKP was attributed to being the cause of their neutropenic sepsis. In one patient, although CRKP was detected in the bowel it was not detected from an invasive site and neutropenic sepsis was attributed to proven *C. difficile* infection. One carrier of CRKP, although experiencing periods of neutropenia, had no episodes of sepsis concurrent with rectal carriage of the CRKP. One patient grew CRKP from sputum secondary to bronchiectasis concurrent with rectal carriage of CRKP having developed carbapenem resistance after being exposed to meropenem to treat an exacerbation of bronchiectasis. Of the 26 affected patients only nine were still alive 2 years into the screening programme. The majority of patients died due to non-infective reasons resulting from their underlying malignancy, although two patients died with sepsis and were receiving appropriate antimicrobial therapy at the time of death.

The CRKP and MDRKP in our cohort have been identified as a CTX-M ESBL-producing organism by the Antimicrobial Resistance and Healthcare Associated Infections (AMRHA) Reference Unit at Public Health England. Variable resistance to colistin, temocillin, tigecycline and fosfomycin is illustrated in Table 1.

In two patients being treated with meropenem, through screening we observed progression to carbapenem resistance. In one such patient, molecular typing (using variable number tandem repeat (VNTR) analysis) was performed at the AMRHA Reference Unit on both the carbapenem fully susceptible and carbapenem-resistant phenotypes of MDRKP, and both isolates were identical. In fact, all the tested CRKP isolates have the same VNTR profile (3,3,3,0,1,1,4,1,1) and the same banding pattern on pulsed field gel electrophoresis, indicating that the CRKP isolates are representative of a single strain, which has been termed MONK04KL-2. It is thought, through investigations performed at the AMRHA Reference Unit, that resistance has arisen due to changes in outer membrane porins, as the organism does not carry any identifiable carbapenemase enzyme, having been tested negative by polymerase chain reaction for Ambler class A (KPC, IMI, NMC and SME), Ambler class B (IMP, VIM, AIM, GIM, KHM,

SIM and SPM) and Ambler class D (OXA-23-like, OXA-24-like, OXA-48-like, OXA-51-like and OXA-58-like) carbapenemases as well as being negative for the *bla*KPC like non-metallo-carbapenemase gene and the *bla*OXA-48 like non-metallo-carbapenemase gene (Cohen Stuart et al, 2010). This mechanism may explain the susceptibility to imipenem despite the organism not being fully susceptible to meropenem or ertapenem. There is greater heterogeneity among the MDRKP isolates which were not CRKP. Sporadic VNTR analysis of these isolates shows at least two different profiles which, although resistant to cephalosporins, aminoglycosides and fluoroquinolones, have differences in resistances to other antibiotics tested (Table 1).

Figure 3 illustrates the incidence per month of carriers of both CRKP and MDRKP, while the prevalence of rectal carriage of MDRKP per month of inpatients being managed by the haematology unit can be seen in Figure 4. Of the nine surviving patients seven have not been re-swabbed since discharge and their carriage state was unknown in November 2012. Patient 2 in Figure 4, who initially was infected with CRKP, remained a carrier of MDRKP for the entire 24 months of screening, although the carbapenem-resistant phenotype was only observed during the first 2 months. Of the carriers who have died, seven were considered still to be carriers when they died (based on having growth of MDRKP from a rectal swab within 4 weeks of death). In four patients more than three consecutive negative swabs indicating no detection of MDRKP were observed over several months prior to death. For the other patients who have died, their carriage status at death was unknown.

Discussion

During the period of surveillance described there was no standard method regarding how to screen for carbapenem resistance among *Enterobacteriaceae* in the United Kingdom. After an initial cluster of four patients with CRKP who shared a room and toilet we devised a screening method which proved to provisionally identify ertapenem-resistant *K. pneumoniae* after one overnight culture on chromogenic agar, but through utilising a Vitek 2 analyser also reproducibly identifies an extensive antibiogram. This can determine whether colistin, temocillin, tigecycline, amikacin and fosfomycin may be used as adjuncts to imipenem (which has become our first-line antibiotic treatment for CRKP and MDRKP-affected patients), as well as monitor whether the organism develops resistance to such agents while on treatment. This is useful as tigecycline and colistin have been proposed as potential treatments for CRKP (Freifeld et al, 2011), but Table 1 illustrates that we have

Table 1. Documentation of the highest Minimum Inhibitory Concentration (MIC) for Tazocin (Piperacillin-tazobactam), amikacin, gentamicin, ciprofloxacin, colistin, meropenem, ertapenem, imipenem, tigecycline and fosfomycin identified from isolates grown from each of the 26 patients who were either carriers or infected with CRKP or MDRKP during the 24 months of surveillance. MICs were determined in mg/L by Vitek 2 (Biomérieux). Interpretation of the MIC as sensitive (S), intermediate (I) or resistant (R) is according to Clinical and Laboratory Standards Institute breakpoints from 2011. NT= Not Tested

	Patient 1		Patient 2		Patient 3		Patient 4		Patient 5		Patient 6		Patient 7		Patient 8		Patient 9		Patient 10	
	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R
Tazocin	≥128	R	≥128	R	≥128	R	>16	R	>64	R	>64	R	>64	R	>64	R	>64	R	>64	R
Amikacin	≥32	R	8	S	16	R	≥64	R	≥32	R	16	R	≤2	S	16	R	16	R	16	R
Gentamicin	≥16	R	≥16	R	≥16	R	≥16	R	≥16	R	≥16	R	≤1	S	≥16	R	≥16	R	≥16	R
Ciprofloxacin	≥4	R	≥4	R	≥4	R	≥4	R	≥4	R	≥4	R	≥4	R	≥4	R	≥4	R	≥4	R
Colistin	≥16	R	2	S	8	R	≤0.5	S	2	S	1	S	2	S	2	S	2	S	2	S
Meropenem	4	I	2	S	4	I	>16	R	2	S	≤0.25	S	≤0.25	S	≤0.25	S	≤0.25	S	≤0.25	S
Ertapenem	4	R	4	R	4	R	>16	R	4	R	≤0.5	S	≤0.5	S	≤0.5	S	≤0.5	S	≤0.5	S
Imipenem	≤1	S	<1	S	<1	S	2	S	≤1	S	≤1	S	<2	S	<2	S	2	S	2	S
Tigecycline	8	R	>8	R	≤0.5	S	2	R	2	R	≥8	R	≤0.5	S	4	R	>2	R	4	R
Fosfomycin	≥256	R	<16	S	<16	S	128	R	≥256	R	32	R	<16	S	<16	S	≥256	R	≥256	R
	Patient 11		Patient 12		Patient 13		Patient 14		Patient 15		Patient 16		Patient 17		Patient 18		Patient 19		Patient 20	
	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R
Tazocin	>64	R	>16	R	>16	R	>16	R	≥128	R	16	R	16	R	>16	R	>16	R	>16	R
Amikacin	16	R	16	R	≤2	S	16	R	16	R	32	R	≤2	S	≤2	S	≤2	S	>64	R
Gentamicin	≥16	R	≥16	R	≤1	S	≥16	R	≥16	R	≥16	R	≥16	R	≥16	R	≤1	S	≥16	R
Ciprofloxacin	≥4	R	≥4	R	≥4	R	≥4	R	≥4	R	≥4	R	≤0.25	S	≤0.25	S	≤2	S	≥4	R
Colistin	2	S	≤0.5	S	<2	S	≤0.5	S	1	S	1	S	<0.5	S	≤0.5	S	NT	NT	≤0.5	S
Meropenem	≤0.25	S	≤0.25	S	≤0.25	S	≤0.25	S	≤0.25	S	≤0.25	S	≤0.25	S	≤0.25	S	1	S	≤0.25	S
Ertapenem	≤0.5	S	≤0.5	S	≤0.5	S	≤0.5	S	≤0.5	S	≤0.5	S	≤0.5	S	≤0.5	S	≤0.5	S	≤0.5	S
Imipenem	2	S	≤1	S	<1	S	≤0.5	S	≤2	S	≤1	S	NT	NT	<1	S	NT	NT	≤1	S
Tigecycline	>8	R	≥8	R	2	R	≤0.5	S	≥8	R	1	S	≤0.5	S	≤0.5	S	NT	NT	≤0.5	S
Fosfomycin	<16	S	32	R	<16	S	<16	S	32	R	32	R	32	R	NT	NT	NT	NT	16	I
	Patient 21		Patient 22		Patient 23		Patient 24		Patient 25		Patient 26									
	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R	MIC	S/I/R								
Tazocin	>16	R	>16	R	>16	R	>16	R	>64	R	32	R								
Amikacin	16	R	≤2	S	8	I	NT	NT	4	S	≤2	S								
Gentamicin	≥16	R	≥16	R	≥16	R	≥16	R	≥16	R	≥16	R								
Ciprofloxacin	≥4	R	≤0.25	S	≥4	R	≥4	R	≤0.25	S	≥4	R								
Colistin	2	S	<2	S	<2	S	2	S	<0.5	S	NT	NT								
Meropenem	≤0.25	S	≤0.25	S	≤0.25	S	≤0.25	S	8	I	<0.25	S								
Ertapenem	≤0.5	S	≤0.5	S	≤0.5	S	≤0.5	S	>16	R	<0.5	S								
Imipenem	2	S	2	S	2	S	<1	S	2	S	<1	S								
Tigecycline	≤0.5	S	≤0.5	S	1	S	NT	NT	2	S	NT	NT								
Fosfomycin	<16	S	32	R	<16	S	32	R	≥256	R	<16	S

Figure 3. Incidence of CRKP and MDRKP among haematology patients. Although 4/6 with the CRKP phenotype were part of a cluster, 2/6 CRKP cases occurred de novo during treatment with meropenem. The carriers of MDRKP *K. pneumoniae* appear to be sporadic cases

Figure 4. Timeline illustrating the duration of screening and duration of rectal carriage of CRKP and MDRKP in the 26 affected haematology inpatients. From this the number of inpatients per month with CRKP or MDRKP carriage requiring single room isolation can also be seen. Dates of death are added to illustrate that for some patients screening stopped in the terminal phase of their haematological illness when death was felt to be imminent, while for others screening stopped because no further inpatient treatment was necessary. Consequently the longitudinal carriage data arising from rectal screening for some patients equates to less than a month, whereas for some others screening has been performed regularly for several months

identified resistance to these antibiotics too. By checking the full antibiotic susceptibility profile of the organism on the Vitek 2 analyser, our method is semi-automated with less hands-on processing (2–3 days from receipt of swab to complete result) than previously described methods, which can take up to 4 days to perform (CDC, 2008).

By improving time to effective antibiotic treatment for CRKP carriers with neutropenic sepsis, Healthcare Quality Strategy for Scotland Quality Ambitions are met by facilitating appropriate treatment in a timely manner, while reducing time to generate a result and potential variation in the antibiotic susceptibility result which can occur using a fully manual method. Figure 2 illustrates this, as a slightly reduced zone of inhibition to meropenem is present with the potential that such a zone could be mis-identified as sensitive using disc diffusion methodology. We suggest that the semi-automated method using Vitek 2, which determines MICs, is more accurate for the detection of carbapenem non-susceptibility than disc diffusion methodology albeit with its own limitations (Woodford et al, 2010; Hunt and Gibb, 2012).

Earlier identification of carriers of MDRKP also reduces antibiotic treatment costs, as an empirical treatment regimen using imipenem 1 g twice daily was found to be cheaper (£25 per day) than either a piperacillin-tazobactam 4.5 g four times daily with gentamicin 240 mg once daily (£61 per day) or meropenem 1 g three times daily-based regimen (£44). It is unfortunate that imipenem is more associated with seizures (Leo and Ballow, 1991) than other carbapenems, but it is possible that it may generate less selection pressure on the *K. pneumoniae* to develop carbapenem resistance via a porin-related mechanism than does meropenem or ertapenem. Twice we have had to discontinue empirical treatment with imipenem, because of a rash in one patient and seizures (identified later to be secondary to underlying intracranial pathology) in a second patient. The patient whose rash was attributed to imipenem (patient 9) has subsequently required treatment with a colistin and fosfomycin-based neutropenic sepsis regimen, as the isolate from this patient was also resistant to tigecycline. Resistance to fosfomycin also developed after minimal exposure to this antibiotic.

Duration of carriage is difficult to assess, as once patients no longer require inpatient treatment they are no longer swabbed. Neither can we be certain that carriage is genuinely lost when repeated swabs are negative, as rectal swabbing may be too insensitive a method to fully exclude continuing carriage elsewhere in the gastrointestinal tract (or lung, as in the patient with lung carriage due to bronchiectasis). In some cases continued rectal screening was felt to be inappropriate, when patient management became palliative with the expectation that death was imminent, and was discontinued.

Staff screening has never been performed as the sporadic nature of newly identified MDRKP carriers and the slightly different antibiograms of isolates have never implicated a point source such as a staff member. It has also been observed through audits of practice that compliance with hand hygiene and standard infection control precautions in the unit was good for all staff groups, making transmission from staff less likely. Environmental screening was undertaken once to investigate possible person-to-person transmission of MDRKP within a four-bedded room, but swabbing of the room and toilet did not yield any growth of MDRKP.

Screening on admission for CRKP has also enabled us to conclude that acquisition of the MDRKP appears to be occurring in the community rather than from person-to-person transmission in hospital. This is because there are several different antibiograms seen among the MDRKP strains, and sporadic VNTR typing demonstrates more than one profile. Patients who acquired MDRKP, having been known not to be carriers through initial screens, did so after periods in the community “on pass” or were identified as carriers on their first screen after readmission for a further cycle of chemotherapy having been in the community. We have also observed MDRKP in urine isolates of primary care patients and patients from other hospital specialties who have no connection with the haematology unit. Poultry have been implicated as a means by which

ESBL-producing *Enterobacteriaceae* enter the food chain to colonise humans (Leverstein-van Hall et al, 2011) which might explain suspected community acquisition. We did not identify any significant epidemiological links between cases to suggest community transmission and as such this was not further investigated, but we did see evidence that suggested *de novo* changes in the resistance patterns of *K. pneumoniae* that was already identified as part of the intestinal flora of such patients. The observation of transformation into a carbapenem-resistant phenotype from a carbapenem-susceptible phenotype in carriers of MDRKP after exposure to meropenem on two occasions (patients 5 and 25) is of interest, particularly since both phenotypes have the same genotype. This is compatible with the suspected resistance mechanism which causes this change in phenotype being a change in the complement of outer membrane porins. We have also observed this change in phenotype to occur in a non-haematology patient whose only antibiotic exposure was to nitrofurantoin in the community and never a carbapenem.

We have also observed a dramatic fall in the incidence of both the carbapenem-resistant and carbapenem-susceptible MDRKP, as illustrated in Figure 3. After July 2011, identification of new carriers of MDRKP has been more infrequent. The reasons for this may be multifactorial, including increased awareness within the haematology unit staff to avoid unnecessary antibiotic use and the need for appropriate patient placement and room cleaning to minimise risk of person-to-person transmission. However, since acquisition of this organism may be in the local community, changes in community prescribing may be more influential. Interestingly, during the period 2010–2011 there was a marked reduction in prescribing of quinolones and cephalosporins in primary care in our health board in response to efforts to reduce overall antibiotic prescribing. Reduction in the use of quinolones has been linked to reduced resistance to quinolones, piperacillin-tazobactam and meropenem in Gram negative bacteria (Troughton et al, 2011), and this reduction in quinolone prescribing may be reducing the incidence of MDRKP seen in the haematology unit.

Conclusions

Microbiological pathogens adapt to overcome the strategies used to treat and eradicate them. Consequently, infection specialists need to be aware of changing and increasing resistance to antimicrobial drugs and have strategies to detect and treat such emerging pathogens. We have shown that a simple method can be rapid, accurate and effective in aiding the clinical management and infection control management of MDRKP. We have also observed that traditional methods of identifying antibiotic resistance using antibiotic discs alone are not accurate enough to identify CRKP.

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The author(s) declare that there is no conflict of interest.

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