An Irish outbreak of New Delhi metallo-β-lactamase (NDM)-1 carbapenemase-producing Enterobacteriaceae: increasing but unrecognised prevalence.

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Running title: New Delhi Metallo-β-lactamase (NDM)-1 outbreak in Ireland
**Structured Summary**

**Background**
Carbapenemase-producing Enterobacteriaceae (CPE) can cause healthcare–associated infections with high mortality rates. New Delhi metallo-beta-lactamase-1 (NDM-1) is amongst the most recently discovered carbapenemases.

**Aim**
To report the first outbreak of NDM-1 CPE in Ireland, including microbiological and epidemiological characteristics, and assessing the impact of infection prevention and control measures.

**Methods**
Retrospective microbiological and epidemiological review. Cases were defined as patients with a CPE positive culture. Contacts were designated as roommates or ward mates.

**Findings**
This outbreak involved ten patients, with a median age of 71 years (range 45-90 years), located in three separate but affiliated healthcare facilities. One patient was infected (the index case); the nine others were colonised. Nine NDM-1-producing *Klebsiella pneumoniae*, a NDM-1-producing *Escherichia coli* and a *K. pneumoniae* carbapenemase (KPC)-producing *Enterobacter cloacae* were detected between week 24 2014 and week 37 2014. Pulsed field gel electrophoresis demonstrated similarity. NDM-1 positive isolates were meropenem resistant with MICs ranging from 12 to 32 µg/ml. All were tigecycline susceptible (MICs ≤1 µg/ml). One isolate was colistin resistant (MIC 4.0 µg/ml; mcr-1 gene not detected). In 2015, four further NDM-1 isolates were detected.

**Conclusions**
The successful management of this outbreak was achieved via the prompt implementation of enhanced infection prevention and control practices to prevent transmission. These patients did not have a history of travel outside of Ireland, but a number had frequent hospitalisations in Ireland, raising concerns regarding the possibility of increasing but unrecognised prevalence of NDM-1 and potential decline in value of travel history a marker of colonisation risk.

**Keywords**

**Introduction**

Enterobacteriaceae are Gram-negative colonisers of the human gut. Carbapenemase-producing Enterobacteriaceae (CPE) are resistant to most classes of antimicrobials. New Delhi metallo-beta-lactamase-1 (NDM-1) is amongst the most recently discovered carbapenemase enzymes. The responsible $\textit{bla}_{\text{NDM-1}}$ gene is thought to have originated in the environment from plant pathogens and is plasmid-borne. NDM-1 confers broad spectrum beta-lactam resistance mediated by hydrolysis of all $\beta$-lactam antimicrobials, with the exception of monobactams, such as aztreonam. Many NDM-1-producing bacteria remain susceptible only to colistin, fosfomycin and tigecycline. Since first reported as implicated in human disease, NDM-1-producing bacteria have been recovered from numerous infection sites including device-associated infections, intra-abdominal, urinary tract, bloodstream and surgical wounds. Publications have described most variants of the enzyme as having originated in Asia.

Acquisition of NDM-1-producers has been reported as associated with travel to known reservoirs areas, notably the Indian subcontinent (Pakistan, India, Sri Lanka) and the Balkan countries, where prevalence of community carriage is estimated to be 5-15%. Global dissemination is facilitated by intercontinental travel, including healthcare tourism, and migration. International spread has been rapid. The NDM isolates identified in Ireland prior to this outbreak were isolated or paired cases from a number of hospitals countrywide and generally with an identifiable link with travel. Dissemination of the $\textit{bla}_{\text{NDM-1}}$ gene, like other similar resistance mediators, is facilitated by inadequate infection prevention and control practice in healthcare settings, uncontrolled or poorly controlled antimicrobial use, inadequate practices related to food preparation, water treatment and general sanitation.
The largest reported NDM outbreak to date in a non-endemic country was reported from Poland in 2015, where 374 cases of infection or colonisation, with a variety of NDM-producing Enterobactericeae, were identified from 40 hospitals over a two-year period. In this report, we describe what we believe to be the first outbreak of NDM-1-producing Enterobacteriaceae in Ireland, which occurred in 2014.
Methods

Setting

The Department of Clinical Microbiology at University Hospital Limerick (UHL) provides a centralised microbiology service for six acute hospital sites (800 beds; population circa 380,000 people). As an aid to contextualising this outbreak, it is notable that 48 *K. pneumoniae* carbapenemase (KPC) and one imipenem-hydrolyzing beta-lactamase (IMI)-producing isolates were detected at UHL between February 2009 and May 2015, as previously published.²¹

Study definitions

Cases were defined as patients with a NDM-1 positive culture from any site during their hospitalisation. Contacts were designated as roommates or ward mates.

Microbiological and molecular detection of NDM-1

Since 2011, CPE surveillance at UHL had been performed on stool samples or rectal swabs using KPC-producer selective chromogenic agar (CHROMagar™ KPC, Paris, France). MALDI-TOF MS (Bruker Diagnostics) identification was performed on all colonies, as previously described.²² Antimicrobial susceptibility testing was performed using broth microdilution (ARIS Sensititre® system-Thermo Fisher Scientific Inc, Massachusetts, USA). Elevated carbapenem minimum inhibitory concentrations (MICs) for meropenem and ertapenem were confirmed by E-test (AB Biodisk, Solna, Sweden) following the European Committee on Antimicrobial Susceptibility Testing (EUCAST) guidelines; ertapenem resistance MIC >1 g/l, meropenem resistance MIC >8 g/l. Isolates with elevated carbapenem MICs were further evaluated using the modified Hodge test (MHT). Commercially available
diagnostic kits (Rosco Diagnostica A/S, Taastrup, Denmark) consisting of meropenem discs supplemented with β-lactamase inhibitors: meropenem + dipicolinic acid; meropenem + boronic acid; and meropenem + cloxacillin were used to phenotypically distinguish CPE isolates. Isolates were referred to the National Carbapenemase-Producing Enterobacteriaceae (CPE) Reference Laboratory Service (CPERLS) at University Hospital Galway, Ireland for CPE confirmation by molecular methods. Genetic relationship of NDM-1 isolates was determined by pulsed field gel electrophoresis (PFGE).

*Details of NDM-1 positive patients*

A retrospective chart review assessing clinical and epidemiologic characteristics was completed for all patients involved including: dates of admission, transfers, and hospital discharges; locations within the hospital; procedures and operative notes; use of invasive devices; biochemical and haematological blood test results; antimicrobials received and documentation of a travel history.

*Infection control interventions*

The isolation of a NDM-1-producing *K. pneumoniae* triggered initiation of the hospital’s outbreak management protocol. Rectal swabs or stool samples were obtained from all contacts of the index case. Information leaflets were distributed to all patients and family as appropriate. The Public Health England CPE toolkit was implemented during the outbreak. All infected or colonised patients were barrier nursed utilising long-sleeved disposable gowns and gloves, and single rooms were used when available. Chlorhexidine gluconate wash-cloths were employed for bathing of patients. Dedicated equipment was prioritised for NDM-positive inpatients, both infected and colonised, but was not available for all NDM-contacts. A semi-automated electronic data surveillance system, ICNet™ (Baxter), was used to collate
the records of the outbreak meetings and patients involved. All patients identified as CPE positive or as CPE contact were flagged on the ICNet™ system and their medical charts were assigned a CPE alert sticker, placed on the front cover.

During the thirteen weeks over which this outbreak occurred, there were an additional 13 KPCs and one oxacillin-hydrolyzing carbapenamase (OXA-48) identified. This was the first OXA isolated at UHL. At a practical level, staff were familiar with the term ‘KPC’, but the introduction of the term ‘NDM’ and ‘OXA’ created confusion and the concept of three different types of CPE circulating simultaneously generated alarm amongst clinical staff. Members of the infection prevention and control team provided additional education sessions at ward level to nursing staff and healthcare assistants. New CPE posters were designed explaining the different CPE types in simple and clear language, and these were placed in the doctors’ residence (communal living space) and on all wards. Anecdotal feedback received regarding the posters was positive. An electronic link to the location of the CPE guideline on the hospital intranet was disseminated on a memo to all staff.

Hand hygiene audits were performed with greater frequency in affected areas, which involved twice weekly observational audits at ward level. Enhanced cleaning, twice daily, of all implicated clinical areas and patient equipment was instigated in parallel with increased auditing of cleaning practice. The index cases’ room in the ICU and all ward areas, where positive NDM-1 patients had been admitted, underwent routine cleaning followed by hydrogen peroxide vapour decontamination post-discharge. A deep clean of the emergency department (ED) including the waiting room and resuscitation areas was performed as seven patients involved in the outbreak had been admitted via the ED. High-touch surfaces such as door handles, bedside lockers and chairs and bed rails were emphasised by the hospital
hygiene nurse manager for cleaning to reduce cross-transmission. An ultraviolet torch was used to assess the quality of cleaning performed and face-to-face feedback regarding cleaning deficits was provided to cleaning operatives. Environmental sampling was not performed during this outbreak.

A further initiative was introduced involving, on a daily basis, a joint pharmacist/clinical microbiologist handover of all in-house carbapenem prescriptions, and subsequent discussion by the either the microbiology consultant or registrar with clinical teams regarding alternative agents where appropriate.
Results

Index Case

The index case was a community-dwelling Irish female. In the summer of 2014, she was admitted with sepsis. Blood and peritoneal fluid cultures confirmed *Escherichia coli*. Initial admission was to a six-bed bay in a general medical ward preceding transfer the following day to a single room in the intensive care unit (ICU). Admission screens confirmed prior colonisation with meticillin-resistant *Staphylococcus aureus* (MRSA) and vancomycin-resistant *Enterococcus faecium* (VRE). A rectal screen at that time did not detect CPE. Empiric therapy utilised intravenous (IV) ceftazidime 2g every 8 hours and gentamicin. As she did not respond to initial therapy, treatment was changed to meropenem 1g IV every 8 hours, metronidazole 500mg IV TDS and vancomycin 1.5g IV every 12 hours but was de-escalated to meropenem monotherapy.

Seven days post admission, a rectal CPE screen (routine for ICU patients) detected a CTX-M extended spectrum beta-lactamase (ESBL)-producing *K. pneumoniae* and NDM-1-producing *K. pneumoniae*. A skin rash developed and diagnostic biopsies were performed on the left forearm. An abscess developed at the skin biopsy site and a CTX-M ESBL-producing *K. pneumoniae* and NDM-1-producing *K. pneumoniae* were isolated from the abscess discharge. IV tigecycline 50mg every 12 hours was initiated and the site was debrided surgically. The patient died 2 months following admission secondary to refractory soft tissue sepsis.
Microbiological

Prior to this outbreak, a case of NDM-producing Enterobacteriaceae had not been identified in our laboratory. None of the patients in this outbreak were known to have prior colonisation with extended-spectrum beta-lactamase (ESBL) producers based on previous screening. Three patients had been screened for CPE before this outbreak but CPE was not detected on those occasions.

In summary, nine NDM-1-producing *K. pneumoniae*, a NDM-1-producing *Escherichia coli* and a KPC-producing *Enterobacter cloacae* were detected. Of note, the NDM-1-producing *Escherichia coli* and a KPC-producing *Enterobacter cloacae* were identified in one patient. The isolates were detected in samples from UHL and two affiliated regional hospitals, located 10km and 40km away, respectively.

Clinical specimens that were positive for NDM-1-producing isolates included mid-stream urine samples (n=2), rectal swabs (n=8) and skin biopsy samples (n=3) from the index case. NDM-1-producing isolates were meropenem-resistant with MICs ranging from 12 to 32 µg/ml. All isolates were tigecycline susceptible (MICs ≤1 µg/ml). One isolate was colistin-resistant (MIC 4.0 µg/ml; *mcr-1* gene negative). PFGE demonstrated that the isolates were closely related (Figure 1). Multilocus sequence typing (MLST) was not performed.

The Outbreak: Patient demographics & epidemiological factors

The patient demographics are summarised in Table 1. All were in full-time residence in Ireland. The screening policy in operation at the time of this outbreak, and currently, is that all patients are screened on admission if: admitted to the ICU or high dependency unit (HDU) at UHL; transferred from another hospital in Ireland; have had an acute admission in the past
12 months to any hospital within our hospital group (except for paediatric, maternity or orthopaedic); or if hospitalised abroad. Haemodialysis patients are screened every three months. Patients in ICU and HDU are screened weekly until discharge. During an outbreak, screening of all patients with epidemiological and environmental links to CPE positive patients, via rectal swab or stool specimen, is performed weekly. CPE screening is performed for a period of four weeks until no new cases of CPE colonisation or infection are detected.

In June 2014, two contacts of the index case prior to her ICU admission were identified (Patients B, D). Both had been on the medical ward with the index case during her 24-hour admission prior to transfer to ICU. Patient B was screened, identified as NDM-1-producer positive and was isolated. Patient D was discharged prior to CPE screening. She re-presented for admission in July 2014 and was re-admitted to a 6-bedded area on the same medical ward as she had been admitted to previously in June 2014. She had a CPE screen performed and was confirmed as NDM-1-producer positive and she was isolated immediately. Patients E, F and H, all were ward contacts of the index case, were NDM-1-producer positive. Patients G and I were identified from routine admission rectal CPE screens performed during the outbreak period. Both had admissions to UHL in the prior 12 months; neither had contact with the index case.

Two additional NDM-1-producer positive patients were identified during the outbreak period but their CPE positivity was not explained by any apparent epidemiologic link with other outbreak related cases. These were not contacts of the index case and had no contact with UHL during the defined outbreak period, including the outpatients and the emergency departments. Patient C was identified as CPE positive from a screening rectal swab that was performed at a regional hospital 10km from UHL. This CPE screen was performed as per
screening protocol because the patient had been admitted to UHL in the prior 12 months. His last admission to UHL was almost five months before the outbreak was declared. Patient J, identified as being CPE positive following a MSU sample sent for routine culture and sensitivity as the patient had symptoms of cystitis, was located in a regional hospital 40km away from UHL. Notably, this latter isolate was determined by PFGE (Figure 1) to be the isolate most distantly related to the other outbreak isolates.

Given the identification of NDM-1 at three different associated hospitals, a decision was made to perform contact tracing and screening of all contacts at each site. As a result of that exercise, during the outbreak, 2204 CPE screens, including contact tracing and routine CPE screening, were processed in our laboratory, which in addition to detecting the NDM-1 isolates, also identified 13 new KPCs and one OXA-48.

Carbapenem consumption

Only the index case had been prescribed meropenem during the current admission. This was prior to the isolation of a NDM-positive culture and, in total, the patient was administered five days of meropenem.

NDM-1 isolates identified in 2015

In week 31 2015, a NDM-1 producing K. pneumoniae was identified in an MSU sample from an 81-year-old female residing in a private LTCF. This patient was a contact of the index case during the outbreak but CPE was not detected at the time. PFGE demonstrated similarity to the 2014 isolates. In week 32 2015, a NDM-1-producing K. pneumoniae was identified from a rectal swab of a 71-year old public LTCF patient who was known to be previously colonised with a KPC-producing Citrobacter freundii. This isolate did not demonstrate
similarity to previous isolates. In week 42 2015, a NDM-1-producing *K. pneumoniae* was isolated by rectal swab from an admission CPE screen of a patient repatriated from Bosnia. She had not been admitted to UHL previously and had never had any specimens processed in the UHL microbiology laboratory. This isolate did not demonstrate PFGE similarity. In week 48 2015, a NDM-1-producing *E. coli* was detected in a 60-year patient who had been recently hospitalised in India. Again this isolate did not demonstrate similarity.
Discussion

The source of the index case patient’s NDM-1-producer acquisition remains uncertain although acquisition during the hospital admission of June 2014 is considered likely. The index case had been on haemodialysis for 16 years and switched to peritoneal dialysis in 2013 (five months prior to NDM-1 detection). In our care, all haemodialysis patients undergo surveillance CPE screening every three months, but the same screening is not conducted for peritoneal dialysis patients. The index case patient was screened for rectal CPE in December 2013, at which time CPE was not detected. Following her transition to peritoneal dialysis, no further CPE screening was performed prior to her transfer to ICU on this final admission to UHL, at which time CPE was likewise undetected. She had never worked in a healthcare setting nor lived with any healthcare workers. She had no known travel to NDM-1 endemic areas.

In this outbreak, international travel was not a recognised factor, implying that there may be a hospital and/or community burden of blaNDM-1 than had not been previously appreciated. Struelens et al. 25 reviewed 77 NDM-1 producing Enterobacteriaceae reported from 13 European countries from 2008 to 2010. Among 55 of the cases with recorded travel history, 31 had involved travel to, or admission to a hospital in, India or Pakistan, and five patients had been hospitalised in the Balkan region. Possible nosocomial acquisition accounted for 13 of 77 cases (17%). In contrast, our outbreak more closely resembled the outbreak reported by Borgia et al. that occurred in Brampton, Ontario, Canada where five patients were identified as carrying NDM-1–producing K. pneumoniae; all of them epidemiologically linked with each other, but none with a relevant travel history 26.
NDM-1-producing *K. pneumoniae* and NDM-1-producing *E. coli* were identified from one hospital (UHL) and NDM-1 producing *K. pneumoniae* found in the two other affiliated hospitals. The dominant species in our NDM-1 outbreak was *K. pneumoniae* (Figure 1). A successfully controlled outbreak in Mexico City reported the isolation of NDM-1 producing *E. coli* and NDM-1 producing *E. cloacae* from the same patient in addition to three NDM-1 producing *K. pneumoniae* isolates derived from three other epidemiologically-related patients. In that outbreak, one plasmid (*IncFII*) was borne by all of the isolates. In a large outbreak reported from South Africa (as in our case, also from three acute hospitals), which persisted for 16 weeks in 2012, *K. pneumoniae* was also the dominant species and accounted for 28/38 isolates (74%) with *E. cloacae* accounting for the 5/38 (13%).

**Learning from the outbreak**

In 2013, Lin et al. reported substantial community reservoirs of CPE in the United States. Currently, there are no data available in Ireland regarding national prevalence of CPE in long-term care facilities (LTCF) as a national point prevalence study of LTCF relating to multidrug-resistant organisms has never been performed. However, three of the ten patients in this outbreak were permanent residents of three separate LTCF (two public, one private) and one other patient was a permanent resident in a residential care facility for adults with learning disabilities (Table 1). Such a study is needed, justified by our data and the fact that, between 2009 and 2015, 140 CPE isolates were identified from clinical specimens of which 12 CPE isolates originated in local public (n=10 isolates) and two private LTCF (n=2 isolates).

Influenced by this outbreak, our antimicrobial stewardship has been modified. Overall hospital antibiotic consumption rate in defined daily doses (DDD) per 100 bed days used (BDU) demonstrates a reduction in carbapenem consumption. Between 2014 and the end of
2015, carbapenem consumption decreased by 21% (2014: 4.43 DDD/100BDU, 2015: 3.49 DDD/100BDU). This compares very favourably with a 4% increase from 2013 (4.24 DDD/100BDU) to 2014, 25% increase from 2012 (3.39 DDD/100BDU) to 2013, a 36% increase from 2011 (2.50 DDD/100BDU) to 2012.\(^{30}\)
Conclusions

This outbreak and our other sporadic isolates indicate the changing epidemiology of NDM-1 CPE. In Ireland, as elsewhere (e.g. Canada), absence of a history of travel to a known endemic area is of decreasing value in differentiating between those at risk of and those not at risk of colonisation or infection with NDM-1 producers. As the successful management of this outbreak demonstrates, prompt infection prevention and control practices are essential to prevent transmission. No staff or environmental screening was performed but extensive resources directed towards education, hand hygiene compliance, environmental disinfection, cleaning standards and reducing carbapenem consumption were successful in controlling rapid in-hospital transmission of NDM-1 producers. The subsequent detection of additional cases, in particular the related isolate from a nursing home resident in week 31 2015, demonstrates the difficulty of definitely eradicating these organisms once established in the “revolving door” systems of nursing homes and hospitals.
Table 1: Clinical characteristics of patients involved in the outbreak

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age</th>
<th>F/M</th>
<th>Place of residence</th>
<th>Date admitted</th>
<th>Admitting diagnosis</th>
<th>Treated with meropenem during admission</th>
<th>Specimen</th>
<th>Organism(s) isolated</th>
<th>Carbapenemase enzyme(s) detected</th>
<th>Date of culture</th>
<th>Infected/Colonised</th>
<th>Outcome</th>
<th>Previous admission to hospital in prior 12 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>44</td>
<td>F</td>
<td>Community</td>
<td>Week 24 (2014)</td>
<td>Peritonitis</td>
<td>Yes</td>
<td>Rectal swab</td>
<td>Klebsiella pneumonia</td>
<td>NDM-1</td>
<td>Week 25 (2014)</td>
<td>Infected</td>
<td>Died</td>
<td>Yes</td>
</tr>
<tr>
<td>B</td>
<td>78</td>
<td>F</td>
<td>Community</td>
<td>Week 24 (2014)</td>
<td>Streptococcus faecalis bacteremia</td>
<td>No</td>
<td>Mid-stream urine</td>
<td>Klebsiella pneumonia</td>
<td>NDM-1</td>
<td>Week 25 (2014)</td>
<td>Colonised</td>
<td>Discharged to a LTCF*</td>
<td>Yes</td>
</tr>
<tr>
<td>C</td>
<td>61</td>
<td>M</td>
<td>Community</td>
<td>Week 25 (2014)</td>
<td>Urinary retention</td>
<td>No</td>
<td>Rectal swab</td>
<td>Klebsiella pneumonia</td>
<td>NDM-1</td>
<td>Week 25 (2014)</td>
<td>Colonised</td>
<td>Discharged to the community</td>
<td>Yes</td>
</tr>
<tr>
<td>D</td>
<td>65</td>
<td>F</td>
<td>Residential care facility</td>
<td>Week 30 (2014)</td>
<td>Urinary infection</td>
<td>No</td>
<td>Rectal swab</td>
<td>Klebsiella pneumonia</td>
<td>NDM-1</td>
<td>Week 32 (2014)</td>
<td>Colonised</td>
<td>Discharged to residential care</td>
<td>Yes</td>
</tr>
<tr>
<td>E</td>
<td>81</td>
<td>F</td>
<td>Public LTCF</td>
<td>Week 30 (2014)</td>
<td>Respiratory infection</td>
<td>No</td>
<td>Rectal swab</td>
<td>Klebsiella pneumonia</td>
<td>NDM-1</td>
<td>Week 32 (2014)</td>
<td>Colonised</td>
<td>Discharged to a LTCF</td>
<td>Yes</td>
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<tr>
<td>F</td>
<td>71</td>
<td>F</td>
<td>Community</td>
<td>Week 30 (2014)</td>
<td>Collapse</td>
<td>No</td>
<td>Rectal swab</td>
<td>Klebsiella pneumonia</td>
<td>NDM-1</td>
<td>Week 32 (2014)</td>
<td>Colonised</td>
<td>Discharged to a LTCF</td>
<td>Yes</td>
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<tr>
<td>G</td>
<td>89</td>
<td>F</td>
<td>Private LTCF</td>
<td>Week 32 (2014)</td>
<td>Congestive cardiac failure</td>
<td>No</td>
<td>Rectal swab</td>
<td>E. coli, Enterobacter cloacae</td>
<td>NDM-1, KPC</td>
<td>Week 33 (2014)</td>
<td>Colonised</td>
<td>Discharged to a LTCF</td>
<td>Yes</td>
</tr>
<tr>
<td>H</td>
<td>90</td>
<td>F</td>
<td>Community</td>
<td>Week 30 (2014)</td>
<td>Respiratory infection</td>
<td>No</td>
<td>Rectal swab</td>
<td>Klebsiella pneumonia</td>
<td>NDM-1</td>
<td>Week 33 (2014)</td>
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<td>Died</td>
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<tr>
<td>I</td>
<td>75</td>
<td>M</td>
<td>Public LTCF</td>
<td>Week 35 (2014)</td>
<td>Respiratory infection</td>
<td>No</td>
<td>Rectal swab</td>
<td>Klebsiella pneumonia</td>
<td>NDM-1</td>
<td>Week 35 (2014)</td>
<td>Colonised</td>
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</tr>
<tr>
<td>J</td>
<td>53</td>
<td>F</td>
<td>Community</td>
<td>Week 37 (2014)</td>
<td>Skin and soft tissue infection</td>
<td>No</td>
<td>Mid-stream urine</td>
<td>Klebsiella pneumonia</td>
<td>NDM-1</td>
<td>Week 37 (2014)</td>
<td>Colonised</td>
<td>Discharged to the community</td>
<td>No</td>
</tr>
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</table>

*LTCF: long term care facility
Figures

Figure 1: Pulsed field gel electrophoresis of 12 *K. pneumoniae* isolates. ME 140282 is the index case.

Figure 2: Pulsed field gel electrophoresis of the two NDM-1 *E. coli* isolates.
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Transparency declaration
None to declare.

Author contribution
All authors made a substantial contribution to the acquisition and interpretation of data. CD performed final drafting of the manuscript.

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