antibiotic policies may be helpful. Implementation of infection control awareness programmes will help to reduce risk factors associated with the emergence of multidrug resistance.

Acknowledgements

Staff of the Bacteriology Department, Institute of Public Health Pakistan, Lahore Pakistan and Microbiology Labs of all four Hospitals in the study are acknowledged for their support.

Conflict of interest statement
Regional differences in MRSA and Increasing drug resistance.

Funding source
No external funding was utilized, Institution resources were used.

References
3. Phoenix PMIC/ID-13, reference 448530, Becton Dickinson and Co. Sparks, MD, USA; Benex Limited, Shannon, Ireland.

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Available online 24 August 2007

Outbreak of multi-resistant Corynebacterium striatum infection in an Italian general intensive care unit

Madam,

Intensive care units (ICUs) are a focus for the emergence and dissemination of multi-resistant bacteria, mainly because the most severely ill patients can be found in the ICU and almost all of these patients will have been exposed to intense antibiotic pressure and exogenous bacterial colonisation.

Corynebacterium spp. are widely disseminated in the environment and constitute part of the normal skin and mucous membrane flora. Although both C. amycolatum and C. jeikeium are currently recognized as important pathogens, the significance and prevalence of C. striatum as a causative agent of disease are not well understood.1

In first months of 2006, 13 strains, which were identified as Kocuria kristinae by an automated system, were isolated from eight patients admitted into ICU. Considering that in our laboratory K. kristinae had never been isolated before and that using standard biochemical analysis misidentification of coagulase-negative staphylococci as Kocuria spp. had been reported, a genotypic assay was performed.2 Surprisingly, all the strains tested were identified as C. striatum.

Our strains were isolated from clinical specimens using routine diagnostic cultures. The identification had been performed and repeated three times using bioMérieux vitek 2 system GP card. The isolates were identified as K. kristinae with a probability of identification of 99.9%. Analysis of the 16S rRNA sequences was performed as described previously by Wauters et al., and showed that all the strains tested were C. striatum.3

All the isolates exhibited the same pattern of antibiotic susceptibility, being resistant to penicillin, amoxicillin, cefalotin, cefoperazone, cefazolin, ceftriaxone, ceftazidime aztreonam, doxycycline,
erythromycin, clindamycin, trimethoprim/sulphamethoxazole, levofloxacin and sensitive to vancomycin, teicoplanin and linezolid.

Overall, C. striatum was isolated from seven bronchial aspirates (five patients), from a central venous catheter tip in one patient and from five blood culture sets in two patients. The demographic and clinical data of the patients are reported in Table I. Patient no. 4 had three positive blood cultures, was clinically septic and died even though he was treated with appropriate antibiotic therapy. In none of the patients, in whom C. striatum was isolated from a bronchial aspirate, was a diagnosis of ventilator-associated pneumonia made.

The temporal distribution of the cases and the fact that all the isolates exhibited the same pattern of antibiotic susceptibility suggest that a single strain selected in the ICU was transferred from one patient to another.

From the analysis of our cases the clinical significance of C. striatum cannot be fully established. In fact, it is hard to assess how many of the symptoms in each patient could be accounted for by infection or by the underlying conditions.

C. striatum may cause endocarditis, pneumonia, empyema, lung abscess, conjunctivitis, endometritis, keratitis, peritonitis, soft tissue infections, meningitis, septic arthritis, osteomyelitis, pancreatic abscess, bacteraemia, and catheter exit site infections especially in patients with underlying diseases. In the literature, four nosocomial outbreaks of C. striatum infection have been reported. Two of these have been in ICU, where person-to-person spread was also documented. Even though a clear pathogenic role was demonstrated only in a few of the above cases, we think that C. striatum cannot be simply dismissed as a contaminant when recovered from clinical specimens and, on the base of our experience, empirical treatment should always include a glycopeptide.

Our outbreak highlights both the growing importance of C. striatum as an emergent multidrug-resistant nosocomial pathogen and the difficulty microbiology laboratories may encounter when trying to identify this species. The possible misidentification of C. striatum as K. kristinae using the bioMérieux Vitek 2 GP card is evidenced. The utilization of a genotypic assay such as 16S rRNA is recommended to confirm species identity for unusual clinical scenarios.

Conflict of interest statement
None declared.

Funding sources
None.

References

Table I  Demographic and clinical data of the patients with Corynebacterium striatum infection

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Hospital stay</th>
<th>Age (years)/sex</th>
<th>Underlying illness</th>
<th>Clinical specimens</th>
<th>Days in ICU</th>
<th>Therapy</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>31 Jan–6 Apr</td>
<td>73/M</td>
<td>Cranial trauma</td>
<td>N=3 bronchial aspirates</td>
<td>7</td>
<td>Teicoplanin</td>
<td>Died</td>
</tr>
<tr>
<td>2</td>
<td>31 Jan–18 Feb</td>
<td>16/M</td>
<td>Multiple trauma</td>
<td>CVC tip</td>
<td>9</td>
<td>Ceftazidime</td>
<td>Recovered</td>
</tr>
<tr>
<td>3</td>
<td>3 Feb–16 Mar</td>
<td>59/M</td>
<td>Stroke</td>
<td>Bronchial aspirate</td>
<td>19</td>
<td>Teicoplanin</td>
<td>Recovered</td>
</tr>
<tr>
<td>4</td>
<td>8 Apr–19 Apr</td>
<td>16/F</td>
<td>Multiple trauma</td>
<td>N=3 sets of blood cultures</td>
<td>5</td>
<td>Teicoplanin</td>
<td>Died</td>
</tr>
<tr>
<td>5</td>
<td>14 Apr–19 Apr</td>
<td>33/M</td>
<td>Multiple trauma</td>
<td>Bronchial aspirate</td>
<td>9</td>
<td>Teicoplanin</td>
<td>Recovered</td>
</tr>
<tr>
<td>6</td>
<td>19 Apr–6 May</td>
<td>80/F</td>
<td>Stroke</td>
<td>N=2 sets of blood cultures</td>
<td>60</td>
<td>Piperacillin</td>
<td>Recovered</td>
</tr>
<tr>
<td>7</td>
<td>5 May–22 May</td>
<td>69/M</td>
<td>Stroke</td>
<td>Bronchial aspirate</td>
<td>5</td>
<td>Teicoplanin</td>
<td>Recovered</td>
</tr>
<tr>
<td>8</td>
<td>10 May–14 May</td>
<td>55/F</td>
<td>Cerebral metastasis</td>
<td>Bronchial aspirate</td>
<td>24</td>
<td>Teicoplanin</td>
<td>Died</td>
</tr>
</tbody>
</table>

ICU, intensive care unit; CVC, central venous catheter.
Pseudo-outbreak of *Aspergillus* keratitis following construction in an ophthalmology ward

Madam,

Outbreaks of fungal keratitis are rare, but have been caused by *Fusarium* species in association with contaminated contact lens cleaning fluid. We report a pseudo-outbreak of *Aspergillus* keratitis in an ophthalmology ward and clinic.

Within our organisation, corneal scrapings are inoculated onto solid media and into broth at the time of specimen collection. Sterile kits containing the required media are prepared by the laboratory and transported to the ophthalmology ward on a weekly basis. During the 6 month period from July to November 2005, *Aspergillus sydowii* was isolated from 40% (23/58) of corneal scrape specimens sent from the ophthalmology ward and outpatient clinic. Prior to July 2005, this species had never been isolated from corneal scrape specimens by our laboratory. Identification was based on the microscopic morphology of the organism. Many members of the *Aspergillus* genus are well known to cause keratitis, and *A. sydowii* has been reported as causing peritonitis in a patient undergoing peritoneal dialysis. However, to our knowledge, *A. sydowii* has not been described as a cause of keratitis in the literature.

On the basis of the low association of this species with disease, and the fact that many colonies were observed outside of the inoculated regions on solid agar, all 23 isolates were thought to be contaminants. Supporting this conclusion, common bacterial or viral pathogens were detected in nine of the 23 cases and only four of the 23 patients were contact lens users.

An initial investigation revealed that all media were sterile and had been refrigerated and sealed until use. In addition, during this time, no specimens other than corneal scrapings were contaminated with *A. sydowii*. On the basis of these observations, it was concluded that contamination was likely to be occurring at the time of specimen collection. Interestingly, it was also noted that the ophthalmology clinic and ward had transferred to a new facility several weeks prior to the first isolation of *A. sydowii*. Major interior construction work within the new ward and adjacent clinic had been completed several weeks prior to the move.

An initial inspection of the collection rooms did not reveal any focus of fungal contamination. More specifically, all furniture, curtains and linen were entirely new with no staining of any surfaces to suggest fungal growth. An air-sampling investigation was then carried out using a hand-held air sampler, the SAS-super90. By this method, 1.5 L of air is taken in over several minutes and particles ≤1 μm in diameter are impacted onto Czapek-dox agar positioned behind a perforated cover plate.

Five clinic rooms were sampled by this method. The rooms were tested in the morning, with all windows closed, and were unoccupied at the time. The mean total colony count in the rooms was

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