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A review of the Royal Perth Hospital Bali experience: an infection control perspective

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Abstract
Thirty five patients were transferred to Royal Perth Hospital (RPH) after the Bali bombings. The patients had severe burn injuries and were considered to be at high-risk of both the carriage and acquisition of multi-resistant organisms (MROs). Whilst seeking to protect the Bali patients with a comprehensive infection control response, we also sought to protect other high-risk patients from nosocomial acquisition of MROs. MROs were detected from 25 (82%) of the 29 Bali patients admitted to RPH. Bali patients were colonised, or infected, with one or more of the following MROs: multi-resistant *Acinetobacter baumannii* (MRAB) (19 patients), extended-spectrum β-lactamase (ESBL) producing Gram-negative bacteria (15 patients), vancomycin-resistant enterococci (VRE) (nine patients), multi-resistant *Pseudomonas aeruginosa* (MRPA) (six patients), multi-resistant *Chryseobacterium* sp. (four patients), and methicillin-resistant *Staphylococcus aureus* (MRSA) (three patients). Five Bali patients developed a total of eight bacteraemic episodes, with MRPA sepsis contributing to death in two patients. Since the Bali bombings horizontal transmission of Bali MROs has occurred in 41 non-Bali patients in RPH. MRPA has had the greatest clinical impact. Eight non-Bali patients developed a total of 11 bacteraemic episodes, with MRPA sepsis contributing to death in four patients. However, apart from MRPA, we have now controlled transmission of the other MROs in RPH.

The emergency response to the Bali disaster required strong leadership, good communication and multi-disciplinary teamwork. The infection control strategy contributed to good outcomes for most Bali bombing patients. However, many patients within the Bali cohort were heavily colonised with MROs, and some developed invasive infection. Subsequent nosocomial transmission of these MROs to non-Bali patients has been a legacy of the Bali tragedy.

Introduction
Since the September 11, 2001 attacks in the United States, the anthrax and white powder scares in many countries and with the Bali bombings, there has been increasing awareness of terrorist activity. These events, together with the recent war in Iraq and the current severe acute respiratory syndrome (SARS) epidemic, have highlighted the need at the national, state and individual hospital level for training and preparedness for emergency responses. Major incident plans must include contingencies to manage situations where caseload exceeds available resources, and require strong leadership, good communication and multi-disciplinary teamwork to provide efficient and effective patient care.

The Bali bombings caused much carnage in Kuta, which resulted in many casualty admissions for a number of Australian hospitals. The infection management service (IMS) at Royal Perth Hospital (RPH) played an important role in the coordinated emergency response, including a comprehensive infection control management response to the casualties.
At preliminary briefings, several key questions were critical to the development of our strategic plan. What multi-resistant organisms (MROs) might these patients be carrying? How could we protect these patients from acquiring nosocomial infection? How might we prevent horizontal transmission of MROs in units where these patients were accommodated and elsewhere in the hospital? Finally, was bioterrorism a possibility? We were also cognisant of the necessity to consider carefully the on-going infection control needs of the rest of the hospital.

We believe it is timely to report our experiences and the lessons learnt from the RPH response to the bombings. Therefore, we report how the IMS collaborated with the RPH Bali Disaster Management Executive and implemented a comprehensive infection control response. Secondly, we report the subsequent nosocomial transmission of Bali MROs within RPH.

Background
On Saturday 12 October 2002 at 2300hrs, two bombs exploded within minutes of one another in the popular night spots of the Sari Club and Paddy’s Bar in the Kuta district of Bali. Most casualties of the bombing were initially treated at Sanglah Hospital but, with more than 200 dead and 400 injured, the medical facilities and resources of the Sanglah Hospital were pushed well beyond available resources – and not just for the living. The limited mortuary facility at Sanglah Hospital, plus difficulties in the identification of badly charred and decomposing human remains, added to the enormity of the tragedy. Bali has limited medical resources, and significant political, cultural, religious and demographic differences from Australia.

Several Australian doctors were directly involved in management of patients at Sanglah Hospital. The ‘Australia Ward’ at Sanglah Hospital was a large open ward with no air-conditioning, but crowded with the general public and the media. Almost all patients had severe full-thickness burns (ranging from 25-85%). Many had shrapnel wounds or intra-abdominal injuries, and some had blast-related limb-threatening vascular or crush injuries, and severe head or orthopaedic injuries.

Problems encountered after the bombings included the language barrier, lack of adequate experienced medical staff, challenging triage decisions and insufficient resources e.g. shortage of beds, oxygen, intravenous fluids, anaesthetic and analgesic medication, blood, sterile gloves, scalpel blade handles and gowns. For example, gloves had to be re-used after rudimentary cleaning and disinfection. Nevertheless, on the first day at Sanglah Hospital, many, if not all, patients were prescribed empirically one or more doses of intravenous cephalothin [Southwick Cl, Plastic Surgeon, Melbourne Institute of Plastic Surgery, Melbourne. Personal e-mail communication January 27 2003].

The Australian Defence Force (ADF) coordinated the evacuation of most patients, expatriating 66 critically ill patients from Bali to Royal Darwin Hospital (RDH) within 30 hours of the bombings. Patients were stabilised, then under the direction of Emergency Management Australia, were transferred to various centres in Australia including RPH.

Major incident planning
The Western Australian (WA) State Emergency Management Committee met early on 13 October and a major incident plan was activated and coordinated from a command post in RPH. The command post facilitated communication between a Perth airport medical team, other Perth metropolitan hospitals and RPH. Regular metropolitan and internal RPH Bali Disaster Management Executive meetings were held to communicate developments and to share information.

The RPH burns unit (BU) has seven single bed rooms and one two bedded room with positive pressure ventilation. The RPH medical intensive care unit (ICU) has four single bed negative pressure isolation rooms and an open ward area with eight beds. Thus patients admitted to ICU could not be cared for in positive pressure protective isolation rooms. The plastic surgery ward (PSW) had only four bedded bays and no single rooms.

RPH has the only adult BU for WA, and it was full prior to the bombings. Over 13-14 October the RPH Bali Disaster Management Executive moved all patients from the medical ICU and PSW to accommodate burnt patients from Bali. An extended BU area was developed, including the BU and ICU areas, with the PSW becoming the designated overflow burns ward for Bali patients.
The initial management of the Bali bombing patients at RPH

In total, 35 of the Bali bombing burn victims were referred to RPH. Thus RPH treated the largest number of patients of any single Australian referral burn service. Twenty nine of these patients required hospitalisation; six patients were managed as outpatients.

The first 11 seriously injured Bali burn patients were referred to RPH early on 14 October, expatriated directly from Bali by various means including private Learjet. Early on 15 October a further 13 seriously injured patients evacuated by the ADF (via RDH) came to RPH. Additional patients arrived over the next 4 days including two Balinese burn patients. The final two WA patients arrived from an interstate BU on 14 November. During the first week, nine of the sickest patients required ICU care. ICU patients were then transferred to the BU or adjacent PSW as their condition permitted.

Initial infection control strategies

The infection control strategies were twofold. Firstly, to provide an environment to prevent infection in this highly susceptible group, and secondly to prevent the transmission of MROs to other patients. The IMS focused on the implementation of a multi-faceted infection control response.

Regular RPH Bali Disaster Management Executive meetings were held at 1700hrs to discuss issues ranging from surgical planning, staffing, supplies, infection control, customer and counselling services to media management. In addition to routine IMS meetings, ad hoc IMS meetings were held to deal with unexpected problems as they arose. Two members of the IMS attended all meetings of the RPH Bali Disaster Management Executive. Recommendations were taken to the RPH Bali Disaster Management Executive meeting for consideration and resolution of competing interests. Once the RPH Bali Disaster Management Executive endorsed agreed infection control strategies, then all parties actively implemented these infection control plans. These plans were regularly reviewed and modified as new laboratory data became available.

The IMS recommended cohorting of all Bali patients, where possible. In addition, ward access was further restricted, posters outlining required infection control practices were used and increased environmental hygiene, dedicated individual patient equipment, emphasis on cleaning and disinfection of patient care equipment, increased staff education and feedback, and monitoring of healthcare workers’ (HCWs) adherence with infection control protocols were undertaken.

IMS initiatives included creation of a ‘new front door’ to the PSW, complete with keypad lock and intercom, to control PSW traffic. A four bedded bay was commandeered in the PSW to provide a comfortable waiting room – including television, coffee making facilities, vending machines and soft furnishings – for visitors and the media. Likewise, with cancellation of elective surgery, the day surgery ward adjacent to ICU was annexed to provide additional waiting room space. This helped reduce relatives’ frustration and also limited ICU traffic.

Secondly, alcohol hand hygiene was introduced beyond pre-existing areas of use (ICU and the BU) to include all areas with Bali patients.

Thirdly, the IMS maintained a high profile on all involved wards, with each area visited by a member of the IMS at least daily (often two to three times per day), to provide both up-to-the-minute information regarding laboratory results and to advise on patient care. Non-RPH volunteer HCWs and agency nurses were specifically targeted for additional support, education and feedback regarding infection control issues. Regularly up-dated written management plans were also provided as an adjunct to verbal infection control instructions.

Finally, an outpatient dressing and physiotherapy clinic was specifically established for Bali patients to separate those outpatients who were colonised (or potentially colonised) with MROs from other burn patients.

The diagnostic microbiology laboratory response

There were significant laboratory resource implications, with a 20% increase in the number of wound swabs processed in October-November 2002. The greatest laboratory challenge was the rapid detection and susceptibility testing of MROs from diagnostic samples and screening swabs. In October-November the number of isolates requiring susceptibility testing doubled, and was more complex because of the wide range of MROs detected.

Burn wound surveillance swabs were collected regularly and the results used to guide empiric antimicrobial therapy when clinically indicated. MROs were isolated from clinical specimens, especially burn wounds. Most patients had many diagnostic samples as part of their routine management because of the severity of their injuries.

An endotracheal aspirate from one patient collected on 14 October yielded a Bacillus sp., and because of the patient’s clinical condition and the possibility of bioterrorism, urgent exclusion of Bacillus anthracis (i.e. inhalational anthrax) was required, but the isolate was identified as Bacillus cereus.

On 18 October the Communicable Diseases Network of Australia (CDNA) raised concerns about the possibility of unscreened Indonesian blood being given to expatriated Bali patients. Prompt serological screening for blood-borne viruses (BBVs) was performed, albeit with significant human and laboratory resource implications. Based on individual risk assessment and pre-test counselling, none of our patients were considered to require post-exposure prophylaxis for HIV. Prior to discharge, patients were given post-test counselling. Repeat serology for BBVs at 6 weeks and then 3 months after the incident was arranged at discharge.
Fresh (unrefrigerated) screened blood was used for all major burn surgery procedures at RPH. Aliquots of all warm ultra-fresh blood were screened for bacterial contamination; this required significant microbiology laboratory resources. No samples yielded bacteria.

Screening procedures
Bacteria were identified and susceptibility testing was performed by standard microbiological techniques. Admission screening of all patients for Gram-positive MROs - methicillin-resistant *Staphylococcus aureus* (MRSA) and vancomycin-resistant Enterococci (VRE) - was implemented as per hospital policy. Screening of all patients for Gram-negative MROs, particularly multi-resistant *Acinetobacter baumannii* (MRAB), extended-spectrum β-lactamase (ESBL) producing bacteria and multi-resistant *Pseudomonas aeruginosa* (MRPA) were recommended on 16 October and introduced on 18 October.

MRPA was defined as resistance to three or more of the following antimicrobial classes: β-lactam/β-lactamase inhibitor combinations (ticarcillin-clavulanic acid and/or piperacillin-tazobactam), aminoglycosides (gentamicin and/or tobramycin and/or amikacin), celtazidine, fourth-generation cephalosporins (cefepine and/or cefpirome), quinolones (ciprofloxacin) and/or carbapenens (meropenem and/or imipenem).

Screening results
MROs colonised 24 (82%) of 29 of the Bali bombing victims admitted to RPH. MROs identified from admission screening swabs included VRE, MRAB and ESBL-producing Gram-negative bacilli. Conversely, MROs such as MRPA, MRSA, *Stenotrophomonas maltophilia* and *Chryseobacterium sp.* were detected from Bali patients a week or more after admission to RPH. Details of MROs grown from the Bali patients are shown in Table 1.

The IMS response to Bali patients with MROs
The numerous MROs identified required a broad and reactive infection control plan. Management of patients carrying MROs varied depending on patient location and availability of single rooms. ICU patients carrying VRE on admission screening were accommodated in single rooms from admission. However, some ICU patients carrying MRAB isolates could not initially be isolated because of a shortage of single rooms.

After identification of MRAB and VRE carriers in ICU, environmental surveillance swabbing was instigated. No environmental VRE contamination was found. However, MRAB was isolated in the vicinity of six MRAB-positive ICU patients. Once the open area in ICU was cleared of patients, thorough environmental cleaning and disinfection were performed. Environmental cleaning and disinfection of the ICU were continued until two sets of environmental swabs were negative.

Patients carrying MROs were isolated with additional precautions from admission in the BU. MRAB environmental contamination was identified in the BU from surfaces in the female staff change room and general office areas. Environmental cleaning and disinfection of the BU was continued until two sets of environmental swabs were all negative.

Separation of Bali patients with MROs in the PSW was not possible, as there were no single rooms. However PSW Bali patients with MROs were cohorted with additional precautions. Three carriers of MROs were moved to an adjacent medical ward under additional precautions.

Finally, concerns regarding adequate environmental cleaning, disinfection and hand hygiene were sporadically identified in the ICU. These problems were improved with the appointment of an infection control liaison nurse (see below), and a dedicated cleaning team. Clutter was also problematic, particularly in the ICU.

Clinical outcomes of Bali patients
The severely injured Bali burn patients were heavily colonised with multiple types of MROs. Colonisation with MROs resulted in eight episodes of bacteraemic sepsis in Bali patients (Table 1).

Two Bali patients died within the first week and another death occurred approximately 2 months after admission. Eight episodes of bacteraemia - caused by 12 organisms (including ten different MROs, one non-VRE *E. faecium* and one non-MRPA) - occurred in five Bali patients (Table 1).

Using the AICA definition, the EWMA control chart detected a significant rise in the hospital wide healthcare related bloodstream infection rates associated with the Bali patients (i.e. during October-November 2002 - data not shown). Bacteraemia were putatively associated with burn wound infections in most cases, as burn wound tissue biopsies and/or swabs yielded the same organism concurrently in all cases. However, some patients also had urine and/or respiratory tract specimens yielding the same organism concomitantly. No bacteraemia was line-related.

The most significant organisms associated with bacteraemia were *Pseudomonas* species. Five episodes of *Pseudomonas* bacteraemia - caused by MRPA (three episodes), multi-resistant *Pseudomonas putida* (one episode) and *P. aeruginosa* (one episode) - occurred in three Bali patients (Table 1). In two severely burned patients *Pseudomonas* bacteraemia (all with polymicrobial bacteraemia) contributed to their sepsis related deaths.

Empiric antimicrobial therapies for patients presumed to be infected with multiple MROs were difficult as few therapeutic options were available. Therefore, broad-spectrum antibiotics (e.g. meropenem) were widely used empirically in the ICU. The resultant evolution of carbapenem resistant Gram-negative bacilli required empiric
colistin therapy (despite significant renal impairment) in two Bali patients on four occasions.

Four patients’ burn wounds were colonised and/or infected with B. cereus. Four patients were treated for fungal burn wound infection. Fungal pathogens grown from burn wound biopsies and/or swabs included Candida sp. (four patients; Candida albicans two patients, Candida parapsilosis one patient, Candida tropicalis one patient), Fusarium sp. (three patients; Fusarium sp. two patients, Fusarium solani one patient) and Aspergillus flavus (one patient). One patient developed a varicella-zoster superinfection of a burn wound.

**Horizontal transmission of MROs to non-Bali patients**

Forty one non-Bali patients have had presumed horizontal acquisition of a Bali originated MRO. Transmission of MROs included sporadic cases and three clusters. Details of MROs associated with horizontal transmission to non-Bali patients are shown in Table 2.

The first cluster was MRAB spread to three non-Bali patients which occurred in the PSW. Transient staff hand carriage was the presumed cause of nosocomial dissemination.

The second cluster occurred after the transfer of two known MRSA-positive Bali patients from an interstate hospital. This MRSA spread to a BU patient from Bali, and then, after the patient was transferred into a four bedded bay, to three other non-Bali room contacts in the PSW. Pulsed-field gel electrophoresis (PFGE) showed that all these isolates had identical clamped homogeneous electric field (CHEF) patterns (data not shown), supporting single-strain horizontal transmission.

The third and most significant cluster was MRPA transmission. A total of 29 non-Bali patients were colonised and/or infected with MRPA. The last of the Bali patients was discharged from ICU on 9 December. Despite this, we continue to experience an ongoing problem of MRPA, with the ICU the recognised focal point. Sixteen ICU patients have been colonised and/or infected with MRPA; seven were local WA burns patients who were treated in the ICU.

Horizontal transmission of MRPA extended beyond the ICU and included the BU and the neurosurgical ward. Six BU patients who were never admitted to ICU have become colonised and/or infected with MRPA. An ICU patient with MRPA on ICU discharge screening swabs was transferred to the neurosurgical ward, and a further four additional cases of MRPA carriage and/or infection are also putatively linked. The index patient on the neurosurgical ward later died and MRPA ventriculitis and meningitis (associated with an external ventricular drain) contributed to his death.

In total, of the 41 non-Bali patients who became colonised with MROs, eight patients developed bacteraemia, seven patients with MRPA and one with an ESBL-producer, causing a total of 11 episodes of bacteraemia. In four non-Bali patients’ MRPA, sepsicaemia contributed to their deaths.

PFGE typing of some MRPA isolates revealed three genotypically closely related clusters when using the Tenover criteria. All three clusters had Bali burn patients as presumed index cases, strongly suggesting horizontal transmission.

**Additional infection control strategies following horizontal transmission of MROs to non-Bali patients**

Additional infection control interventions were implemented in response to further horizontal transmission of MROs to non-Bali patients. A barrier previously used to separate the ICU isolation rooms from the rest of the ward was re-erected. A poster on the barrier alerted visitors and staff of the need to adhere to additional precautions.

An ICU infection control liaison nurse, with past IMS experience, was seconded to educate staff and enforce additional precautions in the isolation rooms and hand hygiene after every patient contact. The role included observation and auditing of environmental cleaning and disinfection to ensure adequate hygiene. Regular updates were provided to all staff to increase their awareness of the ongoing problem.

An outbreak investigation and review of practices associated with the use of water sources was undertaken. The draining of dialysate drainage bags into a sluice was considered sub-optimal – thus reuse of these bags was ceased. Extensive environmental sampling of known sources of hospital acquired P. aeruginosa sepsis found one MRPA positive sink drain in the ICU clean stock room. Dedicated cleaning teams were employed to thoroughly clean the ICU weekly to reduce environmental contamination.

**Discussion**

Burn patients are among the patients at highest risk for hospital-acquired infections. The burn wound includes necrotic eschar combined with serum proteins which provides a rich culture medium for microorganisms. Burns patients have defects in local and systemic immunity, and are at high risk of burn wound infection, bacteraemia and nosocomial pneumonia. Microorganisms rapidly colonise the burn wound, initially with Gram-positive bacteria, but between 3 and 21 days the wound becomes colonised with Gram-negative bacilli from the patient’s own gastrointestinal tract or from other patients in the burn care facility. Principal causes of death in burns patients includes sepsis, usually from burn wounds or the lungs.

A prominent feature of the Bali patients was the extent and diversity of multi-drug resistant Gram-negative bacilli. Most patients were colonised on initial (or early) screening and/or diagnostic swabs and/or biopsies. Although very predictable, why was this so prominent? Common risk factors for MROs, such as length of hospital stay (the strongest predictor), advanced age, gastrointestinal surgery, transplantation, prolonged exposure to invasive devices and antimicrobials...
did not initially apply to the Bali patients. Potential explanations include the importance of pathogens such as MRAB in community and hospital acquired infections in the tropics\textsuperscript{10-13}; immersion of some Bali patients in hotel pool water after injury; sub-optimal facilities for burns patients at Bali hospitals; administration of empiric cefalo-thrun; and possibly the delay in performing early cleansing and debridement of heavily soiled burn wounds at RPH.

\textit{A. baumannii} are ubiquitous organisms found in water and soil that readily acquire resistance to many antimicrobials. Environmental levels are increased during the summer months, possibly due to biofilm bloom in tapwater. Skin and mucous membrane carriage is higher in the tropics than in temperate climates. Community acquired \textit{A. baumannii} pneumonia is well described in Darwin and in other tropical/sub-tropical climates\textsuperscript{14}.

\textit{A. baumannii} are also emerging nosocomial opportunists among debilitated patients, particularly in ICUs. Most hospital (and many community) isolates are multi-resistant. MRAB are recognised pathogens associated with trauma related wound infections (e.g. following missile injuries during the Iran-Iraq war\textsuperscript{15}), and has been reported from infected wounds from the recent conflict in Iraq\textsuperscript{16}. Outbreaks of MRAB have been described in BUs\textsuperscript{17,18}. We had recurrent outbreaks of MRAB in RPH over the 5 year period 1992-1997\textsuperscript{19}, but never involving the BU. We have seen only one sporadic case of MRAB bacteraemia in the RPH BU over the last 5 years.

In Bali patients, MRAB caused bacteraemia in three patients, and colonised and/or infected the burn wounds of 19 Bali patients; it also caused cross-transmission to eight non-Bali patients. Many isolates were initially resistant to all antimicrobials apart from carabapenems and colistin, but pan-resistant strains emerged (data not shown).

ESBL-producing Gram-negative bacteria are significant nosocomial pathogens\textsuperscript{20} which are usually spread within hospitals by HCW hands. Klebsiellae in particular survive long periods on environmental surfaces\textsuperscript{21}. Outbreaks of ESBL-producers have been described in BUs\textsuperscript{22}, and we reported a small cluster of ESBL-producing \textit{Klebsiella pneumoniae} related sepsis in the RPH BU in 1997\textsuperscript{2}. However, we had not seen ESBL-producers cause sepsis in the RPH BU from then until the Bali patients were admitted.

ESBL-producers caused bacteraemia in two Bali patients, and colonisation and/or infection of the wounds in 14 other Bali patients. An ESBL-producing \textit{Enterobacter cloacae} caused bacteraemia in one non-Bali BU patient. Six other non-Bali patients had ESBL-producers detected from screening swabs. Most isolates were initially susceptible to quinolones and carbapenems, but quinolone resistant strains emerged (data not shown).

Pseudomonads are ubiquitous organisms found in soil and potable water and other water sources. Human disease

| Table 1. Summary of the MROs grown from Bali burns patients. |
| Organism | No. patients | No. isolates | Bacteraemias: No. patients | No. episodes* |
| --- | --- | --- | --- | --- |
| MRAB | 19 | 19 | 3 | 3 |
| ESBL Gram-negative bacteria\textsuperscript{1} | 15 | 15 | 12 | 2 |
| VRE\textsuperscript{1} | 9 | 11 | 0 | 0 |
| \textit{P. aeruginosa} | 6 | 10 | 3 | 5 |
| \textit{Chryseobacterium} sp.\textsuperscript{2} | 4 | 4 | 0 | 0 |
| MRSA\textsuperscript{2} | 3 | 3 | 0 | 0 |
| \textit{S. maltophilia} | 3 | 3 | 1 | 1 |

* Summary of MRO bacteraemias in Bali patients: one patient had three episodes of bacteraemia with four MROs grown; one patient had two episodes of bacteraemia with three MROs grown; one patient had one episode of bacteraemia with three pathogens grown; and two patients each had one episode of bacteraemia and one MRO grown. Thus, five Bali patients had eight bacteraemic episodes, caused by 12 MROs.

\textsuperscript{1} Included K. pneumoniae (12 patients), \textit{E. cloacae} (8 patients), \textit{E. coli} (4 patients) and \textit{P. stuartii} (1 patient).

\textsuperscript{2} ESBL-producing \textit{E. cloacae} caused bacteraemia in two patients.

\textsuperscript{3} Included \textit{E. faecalis} (6 patients), \textit{E. gallinarum} (3 patients) and \textit{E. faecium} (2 patients). All VRE isolates were phenotypically and genotypically vanA isolates. Genetic analysis with PFGE analysis of CHEF patterns and plasmid typing showed no evidence of horizontal transmission.

\textsuperscript{4} Non-VRE \textit{E. faecium} bacteraemia in one Bali patient (not included in Table 2).

\textsuperscript{5} \textit{P. aeruginosa} bacteraemias due to MRPA (2 patients – 3 episodes), non-multi-resistant \textit{P. aeruginosa} (1 patient – 1 episode) and \textit{P. putida} (1 patient – 1 episode).

\textsuperscript{6} Includes \textit{C. indologenes} (3 patients) and \textit{C. meningosepticum} (1 patient).

\textsuperscript{7} All MRSA isolates were multi-resistant strains that were identical by both phage typing and PFGE with analysis of CHEF patterns.

outside hospitals is associated with water reservoirs, including swimming pools, and whirlpools\textsuperscript{11-12}. Thus the history of exposure to swimming pool water in Bali may be significant. Risk factors for \textit{P. aeruginosa} infection are multifactorial, but infection is mostly frequently seen in the hospital setting, and burn patients are at very high risk of Pseudomonas sepsis\textsuperscript{10-13}. In hospitals, respiratory equipment, clean solutions, medicines, disinfectants, sinks, mops and even food are important sources\textsuperscript{11-13}. In spite of strict infection control measures, epidemiological investigation and PFGE
Table 2. Summary of MROs grown from non-Bali patients, with presumed horizontal transmission from Bali burns patients.

| Organism                          | No. patients with positive screening samples | No. patients with positive clinical specimens | No. bacteraemic patients (episodes)* |
|-----------------------------------|---------------------------------------------|---------------------------------------------|-------------------------------------|
| Multi-resistant *P. aeruginosa*    | 16                                          | 25                                          | 7 (10)                              |
| Gram-negative bacilli with ESBL-production  | 6                                           | 2                                           | 1 (1)                               |
| MRAB                              | 8                                           | 4                                           | 0                                   |
| MRSA – multi-resistant            | 3                                           | 0                                           | 0                                   |
| S. maltophilia                    | 3                                           | 0                                           | 0                                   |
| C. indologenes                    | 3                                           | 0                                           | 0                                   |

* Summary of MRO bacteraemias in non-Bali patients: one patient had 3 episodes of bacteraemia, one patient had 2 episodes of bacteraemia; and six patients each had 1 episode of bacteraemia. Thus, eight non-Bali patients had 11 bacteraemic episodes.
† P. aeruginosa bacteraemias due to MRPA in seven patients (9 episodes). One non-Bali burns patient in ICU has also had a further episode of bacteraemia due to non-multi-resistant isolate of P. aeruginosa (data not shown in Table 3).
‡ E. cloacae ESBL-producers detected in two patients (one patient also had 1 episode of bacteraemia) and K. pneumoniae ESBL-producers detected in four patients.
Ω All MRSA isolates were multi-resistant strains that were identical by both phage typing and PFGE with analysis of CHEF patterns.

typing of strains showed that nosocomial transmission occurred.

Most isolates of *Pseudomonas* sp. in Bali patients were initially relatively resistant to antimicrobials, and progressively became more resistant. Frequent use of empiric meropenem undoubtedly contributed to evolution of carbapenem resistant strains (usually colistin susceptible, but pan-resistant in some cases) (data not shown) – an emerging global resistance concern.

Lessons learnt

What lessons did the IMS learn from the RPH response to the Bali bombings and its aftermath, and what would we do differently with a large number of severely burnt patients from another disaster? What are our plans for the future?

Firstly, good leadership after a major incident is crucial. A key to good administration was the development of the RPH Bali Disaster Management Executive early to coordinate activities and provide information to all relevant departments. All departments acknowledged the benefit of this group and the leadership that emanated from it. The result was broad cooperation throughout the hospital, thereby providing efficient and effective patient care in what was a well-coordinated hospital response to an extraordinary event.

Secondly, good communication after a major incident is crucial. This was achieved with the RPH Bali Disaster Management Executive. Communication between the ICU and our department has been further enhanced. The ICU have seconded a clinical nurse to act as an infection control liaison nurse to continue to facilitate the implementation and monitoring of infection control practices. Consultants from both the ICU and our department have been nominated to undertake clinical liaison responsibilities, thus ensuring rapid communication of any infection control concerns or issues as they arise.

Thirdly, additional facilities to care for burn patients (and other patients requiring protective isolation) in RPH would have been helpful. Access to more burns rooms and the ability to maintain the Bali cohort as an isolated group may have prevented some subsequent transmission of MROs. Prior to the Bali bombings, plans were being drafted to convert four beds in the open ICU area into four single bed rooms – all with positive pressure ventilation and HEPA filtered air and each with an anteroom. Air will be exhausted to make the anteroom air pressure negative with respect to both the isolation room and the ICU. Clutter in the ICU was another problem. Storage space for equipment necessary for large numbers of burn patients was inadequate. Renovations of the ICU in the latter half of 2003 will provide the four single rooms and more storage space.

Fourthly, environmental cleaning is crucial for the control of MROs. In future incidents, cleaning teams will be assembled forthwith to ensure adequate cleaning is performed and monitored. In April 2003 single stream cleaning teams were introduced at RPH which should improve the overall standard of environmental cleaning in the wards.
Fifthly, for a trial period, we have implemented routine screening of patients for multi-resistant Gram-negative bacilli when they are admitted from the tropics, especially south-east Asia.

Finally, improved antimicrobial stewardship may have helped limit the development and spread of MROs. Daily ward rounds of areas with Bali patients by a consultant from our department may have limited antimicrobial utilisation and, in some cases, provided more narrow-spectrum therapies. The appointment of an infectious diseases pharmacist, together with electronic antimicrobial approval and software support systems, was planned at RPH before the Bali disaster. A multi-disciplinary antimicrobial stewardship programme is currently being formed to establish this initiative. Surveillance of antimicrobial utilisation with control charts is also planned.

In summary, the overall response from RPH staff to the Bali tragedy was exceptional. We implemented an infection control strategy that contributed to good outcomes for most Bali bombing patients. However, most Bali patients were colonised, and some became infected with one or more MROs. Finally, nosocomial transmission of MROs at RPH has been a legacy of the Bali tragedy.

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