of SIADH by lopinavir was probably responsible for the severe hyponatraemia in our patient.

Conflict of interest: No conflict of interest to declare.

References


M.T.M. Roberts*
S.H. Aliyu
Department of Infectious Diseases, Box 25, Addenbrooke’s Hospital, Cambridge, CB2 2QQ, UK

*Corresponding author. Tel.: +44 1223 245151; fax: +44 1223 586874
E-mail address: mtmr1@cam.ac.uk (M.T.M. Roberts)  

Corresponding Editor: Salim S. Abdool Karim, Durban, South Africa
26 September 2005


Antimicrobial resistance of Pseudomonas aeruginosa in pediatric infections

Problems encountered in the management of pediatric infections include treatment failure, which often occurs as a result of antimicrobial-resistant bacterial strains; especially among Pseudomonas aeruginosa.1, 2 For this reason antimicrobial drug activity surveillance is necessary, especially for opportunistic and nosocomial P. aeruginosa.2 In Latin America, resistant bacteria are emerging as a real threat in the community as well as in hospital-acquired infections, including pediatric infections.4 We reviewed susceptibility data of isolates cultured from hospitalized pediatric patients with suspected community-acquired infections in the West General Hospital (WGH) Caracas, Venezuela between 1997 and 2003. The WGH is a 300-bed general community hospital serving people from west Caracas. Samples were taken before antimicrobial drug therapy was commenced. Samples were processed and organisms identified by traditional methods. In vitro antimicrobial susceptibility of the isolates was assessed by an agar disk diffusion method using Mueller—Hinton agar as recommended by the Clinical and Laboratory Standards Institute (formerly NCCLS). Antipseudomonal third generation cephalosporins and carbapenems are freely prescribed in hospitalized patients but previous antimicrobial drug exposure in this patient series was not measured; there were no chronic conditions reported.

During this seven-year period, P. aeruginosa accounted for 137 (4%) of 3425 bacterial isolates from children: 49% from otorhinolaryngological (ORL) infections, 18% urinary tract infections, 7% skin, and 7% gastrointestinal tract, among others. Overall susceptibility rates are shown in Figure 1. Better antimicrobial activity was observed with ciprofloxacin, meropenem, and imipenem (<5% resistant) than for gentamicin, piperacillin and piperacillin/tazobactam (>10% resistant). For urinary isolates, we found strains resistant to norfloxacin (13%) and gentamicin (8%), but only intermediate resistance to aztreonam, ceftazidime, and ciprofloxacin (8% for each). Susceptibility to imipenem, piperacillin/tazobactam, and tobramycin was 100%. In ORL infections, we found significant resistance to carbenicillin (18%) and some resistance to meropenem (5%) and imipenem (3%) but 100% susceptibility to ciprofloxacin, ofloxacin, and piperacillin/tazobactam.

**Figure 1** Overall antimicrobial drug susceptibility (%) of Pseudomonas aeruginosa isolated from pediatric infections against tested antibiotics (WGH, Venezuela, 1997—2003).
Although multidrug resistance has commonly been reported in nosocomial *P. aeruginosa*, community-acquired data are less frequently reported. For this reason, epidemiological studies on the prevalence and antimicrobial susceptibility patterns of resistant isolates in different geographical settings, would provide useful information for both empirical treatment of suspected infections and better management of patients.

Our results show that in our setting imipenem is still very active against strains of *P. aeruginosa* in pediatric infections, although other studies have reported higher resistance rates.3,4 Recent studies have reduced safety concerns over the use of quinolones in pediatric patients. Combined with the good antimicrobial activity shown in this and other studies,5 this makes them a good empirical choice for community-acquired infections. Local surveillance of antimicrobial activity should be done periodically to guide antimicrobial therapy, especially as carbapenem-resistant *P. aeruginosa* has been reported as an emerging problem in Latin America.6

**Acknowledgments**

This work was presented in part at the 24th International Congress of Chemotherapy, Manila, Philippines, June 4–6, 2005. Abstract No. PP1-035. A.J. Rodriguez-Morales was the recipient of an Inter-American Society for Chemotherapy Travel Award (IASC) to attend this meeting.

**Conflict of interest:** No conflicting interest declared.

**References**


Alfonso J. Rodríguez-Morales

**Instituto Experimental José Witremundo Torrealba,**

**Universidad de Los Andes, Trujillo,**

**Venezuela**

Cruz N. Rodríguez*

Ada García

Bileida Pastran

Ivette Jiménez

Pilar Meijomil

Laboratory of Microbiology,

José Gregorio Hernandez West General Hospital (WGH),

Caracas, DC, Venezuela

*Corresponding author. Tel.: +58 212 963 4053
doi:10.1016/j.ijid.2005.10.010

Unrecognized near-fatal hyperlactatemia in an HIV-infected infant exposed to nucleoside reverse transcriptase inhibitors

Nucleoside analogue reverse transcriptase inhibitors (NRTIs) inhibit mitochondrial DNA (mtDNA) polymerase gamma thereby interfering with mtDNA replication.1–3 The relative potency of the NRTIs in causing mitochondrial dysfunction is highest for zalcitabine, followed by didanosine, stavudine, and zidovudine. Lamivudine, abacavir, and tenofovir have lower affinity for mtDNA polymerase.4 In utero and perinatal exposure to NRTIs are known to cause hyperlactatemia (HLA) from mitochondrial toxicity.5,6

An HIV-infected mother detected at 30 weeks of pregnancy was treated with lamivudine, stavudine, and nevirapine. A live male baby weighing 2135 g was delivered by elective cesarean section at 38 weeks of gestation with intrapartum intravenous zidovudine cover as per PACTG 076 protocol.7 He was discharged well with oral zidovudine on the third day.

At three weeks old he developed fever, abdominal distension, diarrhea, and vomiting, which required treatment with intravenous fluids and antibiotics, and was discharged well after 12 days. At five weeks old he returned with diarrhea, poor feeding, dehydration, and acidotic breathing. Arterial blood pH was 7.13, pO2 128 mmHg, pCO2 18.7 mmHg, base excess 21.9 mmol/L, and anion gap 24 mmol/L. Serum lactate and ammonia levels were 4.07 mmol/L, and anion gap 24 mmol/L, respectively. Serum albumin was 28 g/L with prolonged activated partial thromboplastin time (APTT) and increased prothrombin time (PT) ratio. Repeated hemoculture and stool cultures did not yield any pathogen. His urine was screened for inborn errors of metabolism but proved normal. On the second hospital day he developed jerking movements...