

## Letter to the Editor

---

### Organophosphate poisoning in children - atropine, pralidoxime or both?

Dear Sir,

The presentation and management of organophosphate (OP) poisoning is different in young children than in adults.<sup>1</sup> The role of various regimens used in the treatment of OP poisoning like atropine alone or atropine with pralidoxime in addition to supportive measures is debatable.<sup>2</sup> We report the successful recovery of a toddler who presented to us with a history of accidental ingestion of an OP compound and recovered completely on a regimen including atropine, pralidoxime and positive pressure ventilation. A 2 and half-year-old male infant was brought to the Emergency Department in coma with frothing from the mouth, gasping respiration, pinpoint pupils and the characteristic smell of an OP compound. The parents revealed that the child had accidentally ingested an insecticide preparation kept in a "Pepsi" bottle. There was a history of generalized seizure just before arrival at the hospital. The child received intravenous (IV) atropine from the Emergency Department followed by intubation and was transferred to the Pediatric Intensive Care Unit. The infant was ventilated on volume control mode. Chemical analysis of gastric aspirate revealed OP compounds. Other investigations revealed a normal hemogram, urea, electrolytes and liver function tests. X-ray of chest revealed right upper and middle lobe consolidation. Blood culture was sterile. Electrocardiogram was normal. Plasma and red blood cell choline esterase levels were not carried out due to lack of facilities. The child was managed with volume control ventilation, IV atropine at frequent intervals to keep the pupils at normal size and he required 2.5 mg of atropine to achieve this. As the child had symptoms of aggressive atropinization, pralidoxime was also given at a dose of 25 to 50 mg/kg by infusion in 30 minutes, 8 hourly, with a total of 4 doses administered. The dose of atropine was reduced to a minimum. Pneumonia was treated with IV Cloxacillin and Cefotaxime for 7 days with proper chest physiotherapy. With this regimen the child improved and was extubated on the 3rd day and was discharged without any complication on the 10th day after counselling the parents against accidental poisoning.

Organophosphorous compounds act by cholinesterase inhibition. The absence of classic muscarinic side effects does not exclude the possibility of cholinesterase inhibitor agent poisoning in young children with central nervous system (CNS) depression.<sup>2,3</sup> Cholinesterase regenerators are useful

in OP poisoning, especially when the choline esterase levels are less than 25% of normal. Studies have shown that Pralidoxime has direct effect on the CNS in contrast to earlier belief.<sup>4</sup> Chemically, pralidoxime is a 2-formyl-1-methylpyridium chloride oxime (pyridine-2-aldoxime methochloride) and its cholinesterase reactivating ability is due to its 2-formyl-1-methylpyridium ion. The drug is also known as 2-PAM.<sup>4</sup> It is suggested not to withhold Pralidoxime in serious poisoning with a combination of OP compounds and carbamate insecticides or by an unknown cholinergic agent.<sup>5</sup> Addition of pralidoxime has shown to reduce the dose of atropine required to the minimum, thus preventing the side effects of vigorous atropinization. A very high dose of atropine has shown to be associated with ventricular arrhythmia's rarely necessitating pacemaker implantation.<sup>6</sup> In a study conducted at the Medical Toxicology Center, Imam Reza Hospital, Mashhad University of Medical Sciences, Iran has shown that oximes with atropine reduces the fatality rate following OP poisoning. Pralidoxime in high doses (30 mg/kg followed by 8 mg/kg/hr) is free from hepatotoxicity and none of the patients in the group treated with atropine and pralidoxime expired.<sup>1</sup> We observed that the addition of pralidoxime with minimal atropinization was effective and reduced the side effects of atropine in our case. However the role of oximes, pralidoxime in particular, has to be studied on a larger group of patients with OP poisoning to make a definitive conclusion. Accidental poisoning with medications or insecticides is predominantly seen in the age group of 1-4 years of age, especially in boys. Hence parental education and counselling is of paramount importance. Poisonous chemicals should be kept out of sight and of reach of children, preferably locked in cabinets and the used portion should not be kept inadvertently on the table or floor where the ever-exploring toddler can reach. Unnecessary morbidity, hospitalization and even mortality can be prevented to a great extent by proper attention to these minor details. In our particular case, the OP compound was kept in a "Pepsi" bottle which probably stimulated the toddler to drink resulting in dire consequences.

**P. M. C. Nair**  
*Neonatology & Pediatric ICU*  
**Hashim Javad, Zahid A. Al-Mandhiry**  
*Department of Child Health*  
*Sultan Qaboos University Hospital*  
*PO Box 38, Al-Khod 123,*  
*Muscat, Sultanate of Oman*

## References

- Balali-Mood M, Shariat M. Treatment of organophosphate poisoning. Experience of nerve agents and acute pesticide poisoning on the effects of oximes. *J Physiol Paris* 1982; 92: 375-378.
- Lifshitz M, Shakak E, Sofer S. Carbamate and organophosphate poisoning in young children. *Pediatr Emerg Care* 1999; 15: 102-103.
- Sofer S, Tal A, Shahak E. Carbamate and organophosphate poisoning in early childhood. *Pediatr Emer Care* 1989; 5: 222-225.
- Lotti M, Becker CE. Treatment of acute organophosphate poisoning: evidence of a direct effect on central nervous system by 2-PAM (Pyrdine-2-aldoxime methyl chloride). *J Toxicol Clin Toxicol* 1982; 19: 121-127.
- Mortensen ML. Management of acute childhood poisoning caused by selected insecticides and herbicides. *Pediatr Clin North Am* 1986; 33: 421-445.
- Finkelstein Y, Kushmir A, Raikhlin-Eisenkraft B, Taitelman U. Antidotal therapy of severe acute organophosphate poisoning: a multihospital study. *Neurotoxicol Teratol* 1989; 11: 593-596.

## An outbreak of Methicillin Resistant *staphylococcus aureus*

Dear Sir,

The Burns Unit at Hamad Medical Cooperation, Doha, Qatar, (HMC) experienced an outbreak of Methicillin Resistant *Staphylococcus aureus* (MRSA) during May and June 1999. Seven out of 14 patients acquired MRSA one with septicemia. Appreciation of the guidelines for the control of MRSA brought the epidemic to an end. The first index case was admitted to a single room, MRSA was isolated from his burn wound 33 days after his

admission. The average length of patient stay before the acquisition of MRSA was 30 days. Immediately after the first patient was diagnosed, the Unit Head and Head Nurse were alerted and contact isolation was presumed according to the MRSA policy of HMC. The Infection Control Nurse emphasized strict handwashing to all staff. After isolation of 2 more cases, the Infection Control Team started screening the staff and environment. Staff were screened by taking nasal and perineal swabs and finger printing. The patients were screened by taking nasal, perineal and wound swabs. Urine was collected from catheterized patients. Swabs were also taken from the surrounding environment and air was screened by air sampler. Specimens and screening swabs were cultured on mannitol salt agar containing Methicillin 4 mg/l, incubated at 37°C for 48 hours. Yellow colonies were confirmed as *Staphylococcus aureus* (*S.aureus*) by Vitek machine (Biomerieux-France), minimum inhibitory concentration (MIC) of 14 antibiotics were carried out by the same machine.

The results of the screen are shown in Table 1. None of the staff was found to be carriers of MRSA. Seven patients had MRSA isolated from their wounds, 2 from their perineum and one from the nose. The environment of the rooms harboring the MRSA patients was grossly contaminated with MRSA, *Pseudomonas aeruginosa* (*P.aeruginosa*), *Pseudomonas stutzerii*, *Escherichia coli*, *Klebsiella sp.* and *Aspergillus sp.* (combination of skin, fecal and environmental organism). Other environmental swabs were negative for MRSA but *P.aeruginosa* was isolated from the male saline bath and *Aspergillus sp.* from the female saline bath. The MRSA and *P.aeruginosa* isolated from the environment and the patient were identical as indicated by the

**Table 1** - Infection control environmental culture, MRSA and *Pseudomonas* screening.

Samples	Room 3	Room 4	Room 5	Room 6
Airfilter strips	21 colonies	16 colonies	43 colonies	Not done
8 hour settling plates	233 colonies (10 plates)	139 colonies (10 plates)	436 colonies (5 plates)	Not done
24 hour settling plates	235 colonies (10 plates)	>341 colonies (10 plates)	>500 colonies (5 plates)	13 colonies (10 plates) unoccupied
Isolates	MRSA <i>Pseudomonas aeruginosa</i> Coagulase negative spp. <i>Klebsiella spp.</i> <i>Escherichia coli</i> <i>Bacillus spp.</i> <i>Diphtheroids</i> <i>Aspergillus spp.</i> Unidentified fungus	MRSA <i>Pseudomonas stutzerii</i> <i>Pseudomonas aeruginosa</i> <i>Aspergillus spp.</i> Coagulase negative <i>Staphylococcus</i> <i>Bacillus spp.</i> (2 strains) <i>Diphtheroids</i> <i>Klebsiella pneumoniae</i>	MRSA <i>Pseudomonas stutzerii</i> Coagulase negative spp. <i>Bacillus spp.</i> (2 strains) <i>Diphtheroids</i> <i>Alpha Hemolytic Strep</i>	<i>Diphtheroids</i> <i>Bacillus spp.</i> Coagulase Negative spp.
MRSA - Methicillin Resistant <i>Staphylococcus aureus</i> ; Spp. - species.				

## Letter to the Editor

---

susceptibility tests. The index patient was colonized in the groin. This may have been the source of the organism to the unit environment. The gross contamination of the environment and the sharing of nurses may have contributed to the transmission of these organisms.

**Observations during the outbreak.** The Burn Unit is under positive pressure, and 3 filters, which were not routinely checked, filter the air. Space between beds was very small, the common rooms have one washing basin each, which is not elbow or pedal operated. The disinfectants used, savlon and hypochlorite, were not used at the recommended concentration. Nursing staff were hired from other units to help, due to the shortage of nursing staff in Burns Unit.

**Recommendations to contain the outbreak.** Methicillin Resistant *S.aureus* patients should be cohorted in one room and the rooms must be thoroughly cleaned using 0.1% hypochlorite. The Burn Unit should have its own Nursing staff - no hiring from other units. Sinks must be changed to be pedal or elbow operated. Air filters must be checked regularly and documented. Disinfectants must be used at the right concentration. The MRSA policy of corporation must be strictly adhered to.

Methicillin Resistant *S.aureus* is a variant of *S.aureus* that is resistant to all B lactam antibiotics. They may also be resistant to other groups of antibiotics.<sup>1,2</sup> Burns patients colonized by MRSA are at risk of developing bacteremia and wound infection, which can lead to significant morbidity and mortality.<sup>3,4</sup> The rapid spread of MRSA in the outbreak is consistent with cross infection. However strain typing was not carried out, but the similar antibiogram of all isolates suggests identical strains

from the patients and the environment. Environmental contamination with MRSA occurring during outbreaks has previously being reported.<sup>5</sup> Current MRSA guidelines recommend decontamination of the patients environment following discharge from the ward. We endorsed these recommendations and introduced the regular cleaning of the environment with 0.1 hypochlorite. Appropriate use of antibiotics, infection control strategies and educational programs offer additional efforts to control MRSA outbreaks.

**Sittana S. Elshafie**

**Antonio R. Bernardo**

*Department of Laboratory Medicine and Pathology*

*Hamad Medical Corporation*

*PO Box 3050*

*Doha, Qatar*

### References

1. Cookson, BD, Phillips PS. Epidemic methicillin – resistant staphylococcus aureus. *J Antimicrobial Chemother* 1988; 24: 57-65.
2. Hackbarth CJ, Chambers HF. Methicillin - resistant staphylococcus genetics and mechanisms of resistance. *Antimicrobial Agents Chemother* 1989; 33: 991-994.
3. Mulligan ME, Murray-Leisure KA, Ribner BS, Standiford HC, John JF, Korvick JA et al. Methicillin – resistant staphylococcus aureus: a consensus review of the microbial pathogenesis and epidemiology with implications for prevention and management. *Am J Med* 1993; 94: 316-328.
4. Pujol M, Pena C, Pallares R, Ayats J, Ariza J, Gudiol F. Risk factor for nosocomial bacteremia due to methicillin – resistant staphylococcus aureus. *Eur J Clin Microbiol Infection Disease* 1994; 13: 96-102.
5. Rahman M. Epidemic methicillin – resistant staphylococcus aureus (EMRSA): experience from a health district of central England over five years. *Postgrad Med J* 1993; 69: 5126-5129.