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## LETTER

## Neonatal cholinergic syndrome - organophosphate poisoning or herbal medicine intoxication?

**To the Editor:** May I commend Van Wyk and Els on their successful management of a 10-day-old baby with organophosphate poisoning?<sup>1</sup> A low plasma pseudocholinesterase (butyrylcholinesterase) level as reported in the patient is a confirmation of organophosphate poisoning irrespective of the source of the poison or the mode of exposure. Perhaps 'Organophosphate poisoning in a newborn' would have been a more appropriate title. I would like to raise a few points relating to the potential source of poisoning and management of the patient.

A history of organophosphate exposure may not be evident in neonates presenting with cholinergic symptoms;<sup>2,3</sup> the effects depend on the type of chemical, its concentration and length of exposure. Exposure can occur in various ways, such as inhalation or absorption through intact skin from contact with contaminated work clothes of an adult, which can explain cases where no pesticides were used in the home. Clothes may remain contaminated even after being laundered,<sup>4</sup> at which stage the dose may not be toxic to adults but enough to cause symptoms in babies. Sudden onset of feeding difficulties, fever, cough, respiratory distress and lethargy, as initially presented by Van Wyk and Els's patient, are similar to symptoms reported for early severe organophosphate poisoning,<sup>3</sup> suggesting that exposure might have occurred before the baby was taken to the traditional healer.

A possible differential diagnosis not considered in this patient was cyanide poisoning. Herbal products can contain high levels of heavy metals and cyanide.<sup>5</sup> It might have been useful to analyse heavy metals and cyanide, in addition to organophosphate, in the samples sent for toxicological studies.

The authors did not report the baby's blood glucose level, and yet 5% dextrose saline was infused. Moderate to severe hyperglycaemia has been reported in patients with organophosphate poisoning,<sup>6</sup> which is believed to be due to secondary release of catecholamines from the adrenal medulla<sup>7</sup> and may require insulin. The relative risk of dying from hyperglycaemia has been reported to be increased when the maximum glucose level is over 150 mg/dl on admission.<sup>8</sup>

Atropine has been successfully used in the treatment of children, but is not without adverse effects.<sup>9</sup> Combination with an oximine may reduce atropine toxicity. Oximine is more useful early in the presentation (36 - 48 hours),<sup>10</sup> and is recommended when the plasma pseudocholinesterase level is below 25% of normal and the cholinergic poison is unknown,<sup>11</sup> it might have been worth a trial in this patient.

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