

Neonatal cholinergic syndrome - organophosphate poisoning or herbal medicine intoxication?

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A single case of neonatal organophosphate-like poisoning is presented, presumed to have been caused by traditional medicine intake. The dangers of traditional medications and naturally occurring anticholinergics are discussed.

Case report

A 10-day-old, previously completely well baby boy experienced sudden onset of feeding difficulties, fever, cough and lethargy. The parents took the baby to the local traditional healer, who treated him with a single dose of oral medication, after which the respiratory symptoms worsened. The baby was then taken to the local clinic, from where he was referred to a tertiary children's hospital with signs of severe respiratory distress.

On admission we noted profound peripheral vasoconstriction, bradycardia (80 beats/min), a decreased level of consciousness and inability to maintain oxygen saturation above 90% despite oxygen therapy. The baby required intubation and was moved to the Neonatal Intensive Care Unit (NICU). On admission to the NICU he was noted to have severe miosis, rhinorrhoea and increased endotracheal secretions. No wheezing was present to indicate bronchospasm. Arterial blood gas analysis revealed severe acidosis with a pH of 6.96 (normal range 7.36 - 7.38) with mixed respiratory (arterial carbon dioxide pressure 8.9 kPa (normal 4.4 - 4.8 kPa), arterial oxygen 5.2 kPa (normal 9.3 - 11.3 kPa))¹ and metabolic components (bicarbonate 13.4 mEq/l (normal 18 - 24 mEq/l), base excess -10). With ventilation the oxygen saturation improved and the pulse normalised, but the level of consciousness remained depressed. Bicarbonate was administered to normalise the metabolic components, but these only resolved after 24 hours. No other abnormalities were noted on clinical examination. A chest radiograph revealed bilateral infiltrates in keeping with a bronchopneumonia.

The baby then developed the following complications:

1. Hypotension requiring inotropic support.
2. Convulsions for which he was given 50 mg/kg phenobarbitone and then diazepam, with final control being achieved with a midazolam infusion.
3. Hyponatraemia requiring correction with hypertonic (5%) saline.

In view of the symptoms the pseudocholinesterase level was measured, and found to be 0.2 kU/l (normal 3 - 9.3 kU/l). An atropine infusion was therefore started, which significantly improved the baby's secretions.

After 3 days of continuous atropine infusion (total dose 3.3 mg, 0.3 mg/kg/d) the increased production of secretions and the miosis normalised. It was then possible to stop the

infusion, and the baby was successfully extubated on day 4 after admission to the NICU.

Bacterial cultures revealed no growth and all biochemical markers normalised. Both a cranial ultrasound scan and echocardiography were reported as normal. Follow-up pseudocholinesterase levels on day 3 after admission had improved to 2.3 kU/l.

Further discussion with the mother indicated that no other medications had been given and that the baby was cared for only by his parents. No known pesticides were used in the home.

The traditional healer was contacted and a sample of the medication that had been administered to the baby was sent to the State Toxicology Laboratory, but the specimen was misplaced during transfer and no analysis could be performed.

The mother was referred to the Social Work Department and no evidence of child abuse was found.

The baby was discharged after 7 days, feeding well, and will be followed up for long-term morbidity.

Discussion

Very few cases of direct neonatal cholinesterase inhibitor poisonings have been reported. Most reported cases have been due to maternal ingestion of insecticides before delivery with subsequent effects on the neonate.^{2,3} Poisonings are difficult to identify in neonates since they require a high index of suspicion, as such incidents are not expected at this stage of life. The symptoms of organophosphate poisoning are well known (miosis, bronchorrhoea, sinus tachycardia, respiratory failure, salivation, depressed level of consciousness, seizures).⁴

A few cases of neonatal poisonings by traditional medicine ('muti') have been reported in South Africa.^{5,6} As far we know, this is the first case of organophosphate-like poisoning by a traditional medicine to be reported.

Most traditional medicines are presumed to be herbal in nature. However, in a study by Stewart *et al.*⁷ it was found that some traditional medications contained various non-herbal substances such as alcohol, lignocaine, carbamazepine and caffeine, as well as the organophosphate pesticides malathion and parathion.



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Naturally occurring anticholinergic compounds are also found as belladonna alkaloids in alkaloid-containing plants, which are often brewed into teas and include hemlock⁶ and the Chinese herb, huperzine.⁸ Solanaceous glycoalkaloids (SGAs) are naturally occurring steroids found in potatoes, tomatoes and eggplants and are able to inhibit acetylcholine (AChE) as well as butyrylcholinesterase (BuChE).⁹ When used in medications, SGAs could cause anticholinergic symptoms.

The clinical results of herbal intoxications include renal and hepatic failure, metabolic derangements, hallucinations and progressive neurodegenerative disease.¹⁰ If traditional medications contain alkaloid substances they may elicit a cholinergic syndrome closely resembling organophosphate poisoning.

The cause of the hyponatraemia in this case could not be defined. Single studies have mentioned hyponatraemia as a metabolic complication of organophosphate poisoning,¹¹ but the mechanism behind this is not clear. Another explanation for the hyponatraemia in our patient may have been sodium loss caused by sulphate, phosphate or magnesium salts contained in the traditional medication. The traditional medicine may also have contained numerous other medications that could have precipitated a syndrome of inappropriate antidiuretic hormone (SIADH).

Conclusion

Although we could not prove the presence of organophosphate in the traditional medication, we have described the symptoms of organophosphate poisoning and showed response to an atropine infusion.

It is not known how traditional healers prepare their medications and what ingredients are used. Medical personnel tend not to ask about the use of traditional medications in young children, especially babies, but given the increasing use

of these medications it would be a wise habit to acquire.

We wish to extend a warning against use of traditional medicine of which the content is not known, to alert clinicians to the possibility of poisoning of neonates, and to stress the importance of a good history.

References

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