EDITORIAL CORRESPONDENCE

Editorial correspondence is subject to critical review and to current editorial policy in respect to publication in part or in full. Preference is given to letters related to articles published in THE JOURNAL, but letters on topics of current interest may be accepted if space is available. Letters are restricted to 300 words or less, and 3 supporting references.

Disseminated coccidioidomycosis in children

To the Editor:

Drs. Kafka and Catanzaro presented a fine review of the various clinical presentations of disseminated coccidioidomycosis (cocci) in children.[1] At the United Stated Public Health Service Phoenix Indian Medical Center (Phoenix, Ariz.) I have seen two cases of cocci meningitis occurring one and ten years, respectively, after amphotericin B treatment for disseminated cocci.

The first child was a 2-year-old Papago Indian girl who presented with malnutrition, fever, and a chest radiograph showing a markedly widened mediastinum and bilateral perihilar infiltrates. Her coccidioidin skin test was negative and her cocci serum complement fixation (CF) titer rose to 1:520. The initial lumbar puncture was negative for any evidence of cocci. After four months of treatment with amphotericin B iv, there was a progressive drop in the CF titer and she was discharged. Unfortunately, she was lost to follow-up. One year later she returned with fever, lethargy, and nuchal rigidity. The cerebrospinal fluid had a positive cocci CF titer and culture. After two months of intracisternal amphotericin B therapy via reservoir, she died.

The second child was a 4-year-old Apache Indian boy who presented with fever, cough, and supraclavicular lymphadenopathy. His chest radiograph showed bilateral hilar adenopathy and a widened superior mediastinum. A supraclavicular lymph node biopsy revealed cocci spherules and a positive cocci culture. His cocci CF titer rose to 1:256. He was treated for approximately six weeks with amphotericin B iv, with subsequent decline in his cocci CF titer. Six months after treatment was discontinued he developed a subcutaneous abscess on the left forearm, which subsequently was drained; examination and culture demonstrated spherules and a positive cocci culture. He was again treated with amphotericin B for one month. From age 6 no follow-up was done until he returned at age 14 years with progressive headaches, lethargy, personality change, and weakness. A lumbar puncture at that time revealed cloudy cerebrospinal fluid which subsequently had a CF titer of 1:512. He presently is being treated with intracisternal amphotericin B installation, resulting in a slow, progressively improved clinical response.

These two cases illustrate the importance of continued long-term follow-up in all children with disseminated coccidioidomycosis.

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REFERENCE

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Risk of hypernatremia with oral rehydration

To the Editor:

The article by Cleary et al[1] entitled "The relationship of oral rehydration solution to hypernatremia in infantile diarrhea" emphasizes the importance of health education as the major component of any community-based oral rehydration program. They also observed that diarrhea patients with hypernatremia were successfully treated with oral rehydration solution (ORS) which is consistent with our experience.

We are concerned, however, that their article implies that ORS given in the home commonly leads to hypernatremia. This has certainly not been our experience, nor do the methods used in their study allow this conclusion. (1) The children admitted to the hospital who had received ORS at home are a biased sample of all children receiving ORS. Most children with diarrhea do not require hospitalization and the authors give no data concerning this larger population of children in the community who receive ORS. (2) The authors give no data regarding other risk factors for hypernatremia.[2] (3) The Table shows certain internal inconsistencies. The group of patients with hypernatremia completely corrected their hypernatremia within six hours. This seems unlikely since it implies that the average patient would have absorbed 100 ml/kg of plain water, yet the patients were receiving ORS

containing 90 mEq sodium and gained only 7% body weight. Also, the reported change in sodium occurred without a comparable change in chloride. Assuming that the patients were somewhat acidotic on admission, the serum anion gap is unusually high (> 35 mEq/L) with no apparent explanation for this wide gap. These inconsistencies make interpretation of their data very difficult and one would certainly not advise correcting significant hypernatremia so rapidly.[3]

We are reassured of the safety of ORS in ambulatory patients by our experience at the U.S. Public Health Service Hospital on the Fort Apache Reservation at Whiteriver, Ariz., where over 1,500 outpatients with diarrhea were treated. Since there have been no cases of ORS-related hypernatremia admitted to the PHS Hospital (the only hospital on the reservation) during this time,[4] it is unlikely that ORS commonly leads to hypernatremia.

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2. Finberg L: Hypernatremic (hypertonic) dehydration in infants, N Engl J Med **289**:196, 1973.
3. Haddow JE, and Cohen DL: Understanding and managing hypernatremic dehydration, Pediatr Clin North Am **21**:435, 1974.
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Reply

To the Editor:

Several points raised by Dr. Sack et al require comment. The major difference between their experience and the one we described is that in their studies oral rehydration solution (ORS) is given *after instruction* of the mothers. In our setting mothers had obtained ORS packets from local pharmacies and used them with no instruction or supervision. As a result their use of ORS was *incorrect* in *all* cases. Documented errors included mixing three times too much ORS with the required volume of water, mixing ORS with enough water to make a slurry, and giving ORS sprinkled over cereal. The predicted Na+ concentrations of the fluid as used in this unsupervised fashion ranged from 150 mEq/L to greater than 900 mEq/L. Furthermore, the mothers typically believed that withholding water would help stop the diarrhea. In this setting that ORS might predispose to hypernatremia is hardly a surprise. Admittedly our sample is biased, but it demonstrates that unsupervised use of ORS can be expected to be complicated on occasion by significant hypernatremia. Indeed Nalin et al[1] have demonstrated that use of ORS containing 90 mEq/L *without* extra free water is associated with a 16% incidence of hypernatremia by six hours of therapy. Our findings and his underscore the need for ORS use to be supervised to assure that adequate free water is given. Simply passing out packets to vast populations without adequate instruction and supervision can be expected to result in some disasters; with fairly minimal instruction mothers can learn to use this therapy correctly.

Serum sodium concentrations were determined on an Instrument Laboratories flame photometer which had been serviced by the manufacturer immediately prior to use in this project. Standards were determined immediately before and at the completion of each run. Determinations were made in duplicate by a board-certified pathologist. These results agree quite well with other data recently published. Pizarro et al[2] documented similar drops in serum Na+ (156 to 146 mEq/L) during the early rehydration phase of ORS treatment of hypernatremic infants. The estimate of Sack et al that 100 ml/kg of water would be required to lower Na+ from 157 to 142 mEq/L is mathematically correct if the patients had been anephric. These infants took approximately 120 ml/kg of ORS in the first six hours plus extra water in a 2:1 ratio and had good urine output during rehydration. Presumably they lowered their serum sodiums at least in part by renal mechanisms. The change in serum chloride that they mention is less easily explained. Serum chlorides were determined in a routine clinical chemistry laboratory not under our direct supervision and may have been affected by whatever error occurs in routine chemistry laboratories.

The final point they make is that rapid correction of hypernatremia is generally not advised. Our purpose in giving fluid in this way was to simulate what is being done in an unregulated fashion during routine use and measure changes in sodium related to it. Since during home use the mother is unaware of whether the infant is hypernatremic or not, she does not change her method of delivery based on this consideration. Prior to our study there were published data on only eight hypernatremic infants treated with ORS.[3] It now appears that ORS can in fact be used for much more rapid correction of mild to moderate hypernatremia than has been traditionally recommended. The approximately 6% seizure rate with no permanent residua compares favorably to results with any previously described regimen. Further study, particularly of very hypernatremia infants, should clarify the role of ORS in this setting.

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