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Mercury toxicity following merthiolate ear irrigations

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AQUEOUS MERTHIOLATE, an aqueous solution containing 0.1% thimerosal and 0.14% sodium borate, has been recommended as a treatment of purulent otitis media with spontaneous perforations, after insertion of tympanostomy tubes, for external otitis, and as an irrigant during mastoid surgery.^{1,2} Although aqueous merthiolate has been used for years as a topical antiseptic, a recent review of its use by the Food and Drug Administration resulted in its classification as "less than effective."³ Furthermore, two of the ingredients (thimerosal and borate) in merthiolate are toxic if absorbed or injected.

We describe mercury toxicity in a child after ear irrigations with aqueous merthiolate for 1 month.

CASE REPORT

An 18-month-old white infant girl was admitted with a diagnosis of chronic otitis media, ataxia, and irritability. One year previously, bilateral tympanostomy tubes had been inserted. Six weeks prior to admission, she developed purulent otitis media refractory to antibiotic therapy (amoxicillin, trimethoprim/sulfamethoxazole, then erythromycin/sulfisoxazole). One month prior to admission, daily ear irrigations with 1 oz aqueous merthiolate were prescribed. The frequency of irrigations was increased to twice daily 3 weeks prior to admission. A total of 1.2 L merthiolate

was used over the 4 weeks.

Admission findings included a history of staring spells, ataxia, unprovoked screaming episodes associated with opisthotonic posturing, hand tremors, inability to feed herself, and vomiting. Her past medical history, including exposure to other toxins, was unremarkable. Growth and development had been appropriate for age. Initial physical examination showed only a small, irritable child who was difficult to console, with a left-sided draining otitis media and marked ataxia. Rectal temperature was 38.8° C, pulse 164, respirations 30, BP 108/70. Height was 76 cm (5% for age), and weight 9.42 kg (20%).

Blood and CSF cultures, viral serologic findings (including Epstein-Barr virus and hepatitis), CT scan, and mastoid films were all normal. The first EEG showed generalized slowing. By day 4 of admission the child was lethargic, and on day 5 she developed metabolic acidosis, dehydration, and hyperglycemia. Stage II coma and rotary nystagmus were evident by day 7, and on day 10 she required tracheal intubation and mechanical ventilation. The EEG on day 7 was even slower and more abnormal, with increased delta activity, and by day 10 showed burst suppression. The SGOT activity at that time was 1789 U/L (normal 8 to U/L), SGPT 1807 U/L (nl 5 to 35 U/L), prothrombin time 20.9 sec (n 10.4 to 12.5 sec), and activated PTT 190.3 sec (nl 26.4 to 40.0 sec). Serum lactate concentration was 24.7 mg/dl (nl 5 to 20 mg/dl), and pyruvate 0.6 mg/dl (nl 0.3 to 0.9 mg/dl). The urine showed a generalized aminoaciduria.

The patient developed persistent metabolic acidosis, with a large anion gap, hyperkalemia, renal and hepatic failure, hypertension, and congestive heart failure. She later developed a scaled skin picture, and culture proved staphylococcal sepsis. A toxicology consultation was obtained, and the history of previous exposure

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