Reduced Exposure to Mercury in Patients Receiving Enteral Nutrition

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The exposure to mercury (Hg) of various groups of people with different dietary backgrounds has been assessed because of its hazardous effects, but little is known about that in patients receiving enteral nutrition. Therefore, we studied the Hg exposure in 25 patients with severe motor disabilities, who received liquid enteral feedings for more than one year, by determining total mercury (T-Hg) in their hair samples with inductively coupled plasma-mass spectrometry. The geometric mean of the T-Hg level in hair from the patients was 88 ng/g hair (± 1 geometric standard deviation [GSD], 34 – 228 ng/g), whereas that for the control group on a normal diet was 1,900 ng/g (± 1 GSD, 1,022 – 3,531 ng/g). The T-Hg levels in the patients’ hair were far lower than those in the controls (p < 0.001). The T-Hg levels in the enteral feedings used were below the detection limit of cold-vapor atomic absorption spectrophotometry (< 10 ng/g). The present study has shown that Hg exposure is low in patients receiving enteral nutrition, indicating that food is a primary source of Hg exposure.

Mercury (Hg) accumulates in the human body mainly as methylmercury (MeHg) via the consumption of fish and sea mammals (Clarkson 2002). The Hg exposure of various groups of people with different dietary backgrounds has been assessed because MeHg is neurotoxic, especially in the developing brain (Nakai and Satoh 2002; Nakai et al. 2004).

The liquid food used for enteral nutrition (EN) is artificial and does not contain sufficient quantities of the components of natural food. In addition, the total amount of liquid nutrient is often reduced to balance the caloric requirements of patients with severe motor disabilities (SMD). These specific nutritional conditions can easily alter micronutrient conditions (Kumode 2003; Munakata et al. 2006). Therefore, it is likely that the exposure to Hg also differs from that of people

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Mercury Exposure in Enteral Nutrition

on a normal diet if the Hg exposure depends primarily on diet. However, the exposure has not been yet examined in patients receiving EN.

The total Hg (T-Hg) concentration in scalp hair is often used as a biomarker for MeHg exposure (Lindberg et al. 2004). Scalp hair is easy to obtain from bedridden, homecare patients receiving EN. In this study, we used inductively coupled plasma-mass spectrometry (ICP-MS) to measure the hair T-Hg as a proxy for the MeHg intake of patients receiving EN in order to assess the exposure to this compound.

SUBJECTS AND METHODS

Table 1 summarizes the details of EN for the patients. Hair samples were obtained from 25 patients with SMD who had received EN for more than 1 year. Their entire daily caloric intake was mainly from liquid enteral feeding via a nasogastric tube or gastrostomy. The total volume of enteral feeding was relatively low because of the low caloric requirement of bed-ridden SMD patients. Considering the volume of sweating, salivation, and urination, water and electrolytes were supplied daily as water, tea, and an oral-rehydration solution containing (in mM): Na$^{+}$ 60, K$^{+}$ 20, Mg$^{2+}$ 1.5, Cl$^{-}$ 50, PO$_4^{3-}$ 5, citrate$^{3-}$ 6.7, and glucose 9 (Solita-T granules no. 2; Shimizu). Fifteen of the 25 patients were given vegetable juice daily; 17 of the 25 patients were treated for epilepsy with antiepileptic drugs. We constructed a control group using hair from age-matched people living in the Tohoku region of Japan. We received informed consent from the parents of the patients and the members of control group for the following examination.

None of the subjects had hair treated by bleaching, dyeing, or permanent waves. Three-centimeter hair specimens, including the roots, were taken from the occipital scalp of the subjects using stainless steel scissors, and 100 mg of each specimen were measured. The hair was washed using a modified method described elsewhere (Puchyr et al. 1998; Yasuda et al. 2005). First, the hair was washed with acetone, then with 0.01% Triton-X 100 (ICN Biomedicals, Costa Mesa, CA, USA), and finally with ultrapure water (> 18 MΩ/cm, Milli-Q water; Millipore, Bedford, MA, USA). Then, 2.5 ml of tetramethyl ammonium hydroxide (Tama Chemicals, Kawasaki) were added to the specimen with 15 μl of an internal standard solution (Yasuda et al. 2005). Finally, ultrapure water was added to give a final volume of 10 ml, and the sample was shaken for 2 hrs at 75°C to digest the hair completely. The samples were cooled to room temperature and topped up to 15.00 g (gravimetric) with ultrapure water. The T-Hg in the samples was analyzed using quadrupole ICP-MS (HP-7500i; Yokogawa, Tokyo) with the internal standard method.

In a different series of experiments, the T-Hg in six kinds of liquid enteral feedings used was below the limit of detection (< 10 ng/g).

RESULTS

Table 1. Details of the enteral nutrition (EN).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
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<tbody>
<tr>
<td>n = 25 (male 13, female 12)</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>10 ± 7 years (range 1-25 years)</td>
</tr>
<tr>
<td>Duration of EN</td>
<td>5.6 ± 3.2 years (range 1-18 years)</td>
</tr>
<tr>
<td>Amount of liquid nutrients</td>
<td>41 ± 23 ml/kg/day (= Kcal/kg/day)</td>
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<tr>
<td>Other liquids: vegetable juice 9 ± 7 ml/kg (n =15) oral-rehydration solution 22 ± 14 ml/kg (n = 9) small quantity of water, tea and soup.</td>
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</table>

The data are given as the mean ± S.D.

Fig. 1 shows the logarithmic plots of the T-Hg levels in the hair of 25 patients with severe motor disabilities receiving EN and of the age-matched control group. The geometric mean T-Hg value was 88 ng/g hair (± 1 GSD range, 34 – 228 ng/g) in the patients and 1,900 ng/g (± 1 GSD range, 1,022 – 3,531 ng/g) in the controls. The T-Hg level was significantly lower in the patient group than in the control group (p < 0.001).

The T-Hg levels in all kinds of liquid enteral feedings used were below the limit of detection (< 10 ng/g).
DISCUSSION

The Hg measured as T-Hg in hair exists mostly as MeHg. T-Hg levels in hair are strongly correlated with MeHg levels in blood, as well as with estimated MeHg intake, but not with inorganic Hg (Lindberg et al. 2004). Here, MeHg exposure is exclusively dietary and originates from contaminated seafood (Clarkson 2002). Therefore, the concentration of T-Hg in hair has been used as a proxy for MeHg exposure and represents the dietary intake of MeHg over a long time frame. In contrast, with a low MeHg exposure, it is postulated that hair T-Hg reflects inorganic Hg exposure, although several lines of evidence exist that hair T-Hg is significantly correlated with MeHg, even with low MeHg exposure (Lindberg et al. 2004).

The hair T-Hg value of our control group was similar to the value reported for a much greater number of people living in Japan (Yasutake et al. 2003). In contrast, the T-Hg levels in patients receiving EN were approximately one-tenth of the control value. The liquid foods used for our patients included both synthetic nutrients and natural terrestrial foodstuffs, such as extracts of soy, corn, and milk (casein). The T-Hg and MeHg levels in terrestrial foods are usually very low (Inskip and Piotrowski 1985; Dabeka et al. 2003). In this study, the T-Hg concentrations in the liquid foods used were below the limit of detection, implying that MeHg in the foods is also extremely low, if any. Therefore, the reduced T-Hg in the patients’ hair is primarily due to reduced Hg intake, thereby confirming the impact of food as a source of exposure to Hg. However, it still remains necessary to determine whether inorganic Hg or MeHg is correlated with hair T-Hg in our patients with an extremely low Hg exposure. In addition, it is still not known how the underlying pathology of the SMD patients and the medications used to treat these patients (anti-epileptic drugs) affect the hair T-Hg level. Further investigation of the mercury kinetics in the pathologic conditions that affect SMD patients is required.

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References


