Thiamine deficiency in ill children

Dear Sir:

Thiamine deficiency is increasingly recognized in varied parts of the world. In many areas of rural Southeast Asia, thiamine deficiency (seemingly related to rice preparation practices) is the accepted cause of wet beriberi, which presents with respiratory distress and heart failure and rapidly improves after thiamine administration (1). Lima et al (2) have provided important data showing that low thiamine concentrations were common in critically ill children at their center in Brazil. These data add a compelling global dimension to pediatric thiamine deficiency and prompt further questions.

First, is thiamine deficiency a cause, an effect, or an incidental finding unrelated to the illness that prompted admission to an intensive care unit? Thiamine deficiency might have been the major cause of illness in some of Lima’s patients, but only about a third were <1 y of age, the age at which wet beriberi is most commonly seen in Asian children. We recently studied Cambodian infants with a clinical diagnosis of wet beriberi and found that thiamine concentrations were low not only in clinical cases but also in most seemingly healthy matched control infants from the same district (K Shelton-Dodge, personal communication, 2010). Similarly, a recent report from Laos suggests that sick children without clinical signs of beriberi often have biochemical evidence of thiamine deficiency (4). Hence, low thiamine concentrations do not in themselves prove that an acute illness is beriberi. In some sick children with low thiamine blood concentrations, thiamine deficiency may be unrelated or may be an important cofactor that, together with another factor (eg, intercurrent infection), precipitates or worsens clinical illness. It is currently unclear how beriberi is best distinguished from thiamine deficiency accompanying another acute cardiorespiratory illness in infants. Criteria that might be useful include the presence of dysphonia (a distinctive and perhaps fairly specific sign of infantile beriberi), absence of findings of infection, and rapid resolution of symptoms after thiamine administration. Lima et al did not report these clinical features in their patients.

Second, how frequently is thiamine deficiency implicated in severe childhood disease and death? Lima et al (2) observed thiamine deficiency in 28% of their severely sick children but did not show a statistically significant association between thiamine concentrations and mortality. In Laos, however, thiamine deficiency conferred a 9-fold risk of illness-related mortality in sick children (4). In Mesang District, Prey Veng province of Cambodia, where clinical beriberi is frequently diagnosed, we recently conducted a verbal autopsy survey and identified 51 deaths during the first year of life (48 of the 51 during the first 6 mo of life) among 910 live births from January 2005 through 15 April 2008. Thirty-seven (73%) of the children who died were reported to have had tachypnea or dyspnea during the illness leading to death, and 25 (49%) of the children who died had dysphonia [21 of 33 (64%) who died after 7 d of age had dysphonia]. Heart failure with tachypnea is typical of but not specific for beriberi. Using a conservative case definition of beriberi as dysphonia with ≥2 of 4 other typical findings (respiratory distress, irritability, vomiting, wheezing), 23 of the 51 (45%) dying babies had beriberi. Of course, some of these children could have died of pneumonia (with a hoarse voice due to irritability and crying) unrelated to beriberi or thiamine deficiency. But, because not all patients with beriberi have dysphonia, it is also possible that more than half of infant deaths were due to beriberi; thiamine deficiency could be a major contributor to childhood mortality in Cambodia where 6% of children die before their fifth birthday (5).

We are grateful to Lima et al (2) for providing new data about the high frequency of thiamine deficiency in sick Brazilian children. Their data raise important questions about the role of thiamine in acute illnesses other than wet beriberi (isolated thiamine deficiency). Although thiamine deficiency appears to be a major cause of infant mortality in Southeast Asian populations, it remains unclear whether it is a significant contributor to mortality in sick children from other parts of the world.

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in critically ill children during their hospitalization. We seek to identify the potential factors contributing to this deficiency. Low blood concentrations during the ICU stay. Future studies should investigate the clinical significance of thiamine deficiency in critically ill children does not necessarily reflect the prevalence of thiamine deficiency in the overall pediatric population. In critically ill patients, factors such as increased consumption due to increased metabolism, impaired intestinal nutrient absorption, loss of water-soluble vitamins, and subsequently, the phase of anabolism, which higher values of C-reactive protein were shown to be associated with low thiamine blood concentrations on admission, suggesting that the magnitude of the inflammatory response is a risk factor for thiamine deficiency in critically ill children.

We cannot affirm that thiamine deficiency was a major cause of disease in our patients. Although one patient had a confirmed diagnosis of Wernicke-Korsakoff encephalopathy, it would be difficult to identify specific signs of thiamine deficiency in our patients, because clinical signs lack sensitivity and specificity for diagnosing deficits in critically ill children. In our study, low blood concentrations of thiamine on admission were not associated with overall mortality of patients admitted to the ICU. However, a trend of association was particularly with intakes of marine omega-3 fatty acids from a serving of fish per day. Therefore, the association was particularly with intakes of marine omega-3 fatty acids. A similar association was observed with fish intake, but additional adjustment for docosahexaenoic acid led to the elimination of the association. Thus, the association was particularly with intakes of marine omega-3 fatty acids from >0.20 g omega-3/d or >2 servings of fish/d.

In a recent article on dietary omega-3 fatty acids and fish consumption in relation to the risk of type 2 diabetes (T2D) from the Women’s Health Study, Djoussé et al (1) reported epidemiologic data suggesting an increased risk of T2D with intake of marine omega-3 fatty acids. A similar association was observed with fish intake, but additional adjustment for docosahexaenoic acid led to the elimination of the association. Thus, the association was particularly with intakes of marine omega-3 fatty acids from >0.20 g omega-3/d or >2 servings of fish/d.

T2D is strongly associated with proinflammatory products in obese tissue, and it has been established that insulin resistance results from inflammation of the adipose tissue in which cytokines such as tumor necrosis factor-α (TNF-α), interleukin (IL)-1β, and IL-6 as well as products such as PAI-1 (plasminogen activator inhibitor-1) are part of the development and progression of T2D (2). In this regard, omega-3 fatty acids should be beneficial and prevent T2D because they have the potential of suppressing the production of tumor necrosis factor-α (TNF-α), interleukin (IL)-1β, and IL-6 as well as products such as PAI-1 (plasminogen activator inhibitor-1) are part of the development and progression of T2D (2). In this regard, omega-3 fatty acids should be beneficial and prevent T2D because they have the potential of suppressing the production of.

None of the authors had a conflict of interest to declare.

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