evidence that accidental ingestion of the sodium nitrite in the salt shaker was the cause of methemoglobinemia in these cases, the interpretation of the events strongly suggests this as the most plausible possibility.

This is a clear example of the potential hazard when laboratory chemicals are used on food, often a consequence of the unsafe practice of eating and drinking in the laboratory. These cases thus emphasize the importance of not only providing and teaching safety procedures but also of strictly enforcing them (1).

References

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Homogyzous alpha Thalassemia 2 Causing a False Negative Solubility Test in Sickle Cell Trait

To the Editor:

We report here a case of the interaction of alpha thalassemia 2 and beta genes and its effect on the solubility test for HbS.

A 10-month-old black girl with a seizure disorder and mild microcytic anemia was recently admitted to the Eugene Talmadge Memorial Hospital. Her hemoglobin concentration was 87 g/L. The MCV, MCH, and MCHC were 67 fl, 22 pg, and 330 g/L, respectively. Her blood smear showed marked microcytosis and targeting. The serum Fe concentration was 1870 µg/L, the transferrin concentration 2350 mg/L. The solubility tests for HbS (“Sickle-Sol”); Dade Division American Hospital Supply Corp., Miami, Florida) was negative (1); however, electrophoresis on cellulose acetate and on citrate agar both showed an A,S pattern. Quantitative HbS by cellulose acetate electrophoresis (2) was 27% (=23 g HbS per liter). HbA2 by column was 3.7%, within the normal range for sickle cell trait. A later solubility test was positive; however, her hemoglobin concentration at that time was 115 g/L, which would correspond to 31 g of HbS per liter.

Normally, there are two pairs of alpha globin genes on each chromosome 16 (alpha/alpha). In blacks the mild alpha thalassemia 2 haplotype is the result of deletion of only one alpha chain gene (-alpha), whereas in orientals the more severe alpha thalassemia 1 haplotype is the result of deletion of both alpha chain genes (-/alpha) (3). The quantity of HbS in sickle cell trait is inversely related to the number of alpha thalassemia 2 genes present. Hence, a trimodal distribution of HbS exists in the black population: with no alpha thalassemia 2 genes, HbS is about 40%, with heterozygosity for alpha thalassemia 2 (-alpha/alpha) HbS approximates 35%, and with homozygosity for alpha thalassemia 2 (-alpha/-alpha) HbS is about 28% (3–5). The above data, especially the degree of microcytosis and the percentage of HbS, are compatible with a homogyzous alpha thalassemia 2, sickle cell trait (alpha/-alpha, beta/beta) genotype for our patient (3). The solubility test can be negative, as it was here in the first instance, when HbS is <26 g/L (6). The positivity of the later solubility test was secondary to greater HbS, which was presumably 31 g/L. Thus, the interaction of alpha thalassemia 2 and beta genes in sickle cell trait can cause a false negative solubility test secondary to the decreased percentage of HbS. To our knowledge, this is the first report of the interaction of alpha thalassemia 2 and beta genes leading to a false-negative solubility test. Sickle cell trait (beta/beta) has an incidence of about 10% in the U.S. (7) and alpha thalassemia “trait” has a frequency of about 6% in American blacks (6), hence the occurrence of this phenomenon may be relatively common. The frequency of the association of alpha thalassemia 2 and beta genes and its effect upon the solubility test for HbS is another reason for the superiority of hemoglobin electrophoresis to solubility testing as the first step in screening for HbS (6).

References


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Falsely High Values for Corticotropin by the CIS-Sorin RIA Method

To the Editor:

We have used the CIS-Sorin RIA method to determine plasma corticotropin (p-ACTH) in 300 patients and in 18 ostensibly healthy subjects. We found the detection limit to be 10 ng/L. The coefficient of variation in duplicate determinations was 17% for values <50 ng/L and 10% for values >50 ng/L. In 19 subjects with normal cortisol response to insulin-induced hypoglycemia (>270 mmol/L), concurrent p-ACTH determinations showed an increase from 26 (SEM 5) ng/L to 163 (SEM 22) ng/L. Thirty-two individuals with a normal urinary 17-hydroxycorticosteroid response (>100% increase) to a standard oral metyrapone test (six 750-mg doses in 24 h) showed an increase in p-ACTH (at 0800 hours) from >90 ng/L before metyrapone to 125–695 ng/L 4 h after the last dose. Our upper normal 0800-hour limit for p-ACTH is 90 ng/L.