

SEVERE HYPEREMESIS GRAVIDARUM AND INTRA-UTERINE FETAL DEMISE: IS GESTATIONAL MALNUTRITION A FACTOR?

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One in three cases of severe hyperemesis gravidarum (HG) results in spontaneous abortion (1) (2), however the etiologies of these intra-uterine fetal demises (IUFD) has not been fully examined. Our hypothesis is severe caloric and nutrient deficiencies may contribute.

The Recommended Dietary Allowances of the Institute of Medicine endorses increase in all nutritive substrates ---3 caloric sources (carbohydrate, protein and fat), 13 vitamins and 10 minerals in pregnancy. Most – but not all- increase approximately 18% in the second trimester and 23% in the third trimester of gestation. There has never been a recommendation for a hypo-caloric state in pregnancy, however women with HG frequently fails to achieve nutritional adequacy. (3) As a consequence, the developing fetus is chronically deprived of vital nutrients for growth and cell differentiation. Based on known consequences of malnutrition in the adult with some nutrient reserve, the fetus likely suffers nutrient deprivation more acutely in all developing organ systems.

The nutritional composition of the human brain is unknown, however extrapolation from nutrient analysis of animal brain for food, provides a basis for consideration. (4) The estimated basal metabolic rate of the adult brain, comprising 3% of body weight, is 28% of daily requirements. (5) It is highly probable the fetal head/brain, comprising a greater percent of its body habitus, could be more energy demanding. Brain tissue is highly dependent upon an adequate supply of nutrients, including phosphate, thiamine and glucose and other vitamers. Phosphorous is critical for ATP production and required by all major vital organs including brain, heart, kidneys, lung and liver for energy metabolism. Failure to maintain an adequate phosphate level can be lethal. (6) The brain is an avid and obligate glucose consumer as are red blood cells however thiamine and magnesium required co-factors in these energy transactions.

Choissi (2) reviewed fetal loss in 49 women suffering HG also diagnosed with Wernicke's encephalopathy, a consequence of thiamine deficiency. Half of the women suffered weight loss of 11.77 kg with duration of illness of 7.7 +/- 2.8 weeks. It is highly plausible these fetuses also suffered thiamine deficiency concurrently, as well as severe caloric deprivation. Thiamine deficiency manifests as wet and dry beriberi: the former affecting cardiac muscle and the latter cognitive/cerebral functioning. The rate of IUFD was 37%.

Selenium deficiency (Keshan disease) has been identified in childhood cardiomyopathies. (7) In March 2017 we documented a selenium value of 65 ng/mL (normal range: 70-150 ng/mL) in a HG woman with 30 lb weight loss who had a growth restricted fetus at 5% at 28 weeks with poor fetal testing. She was also deficient in vitamin A, 25-OH vitamin D 25 and zinc.

Low potassium and magnesium also contribute to cardiac irregularities, which are lethal if not corrected. (8) Fetal bradycardia (decreased cardiac rhythm) has been observed in a pregnant woman with hypokalemia. (9)

To answer the question, can some cases of hyperemesis-related fetal losses be attributed gestational malnutrition? The evidence says YES!

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