Severe Asymptomatic Essential Fatty Acid Deficiency (EFAD) in a Patient Enrolled in a Quality Improvement Project for HPN Patients (QIP-PN)

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Objectives: Introduction. A 97-year-old, 47 kg female with a history of dysphasia, and failed enteral feeding was on HPN for 35 months. PN provided 150 grams dextrose, 75 grams amino acids, electrolytes, MVI and trace elements. Intravenous lipid emulsion (ILE) was held because liver dysfunction and line infections.

Methods: The patient enrolled in Amerita QIP-PN program. Essential fatty acid and trace element levels were obtained.

Results: Severe deficiencies of linoleic (c18:2w6; 524.3; (nl) 2653.4–6130.3 umol/L) and ȧ-linolenic acids (c18:3w3; 4.12; nl 26.1–150.1) were detected. Eicosatrienoic (Mead) acid (c20:3w9) was markedly elevated at 135.33 (nl 10.3–41.3). The eicosatrienoic to arachidonic acid (triene to tetraene) ratio was markedly elevated (0.27; nl 0.02–0.05). Stearic (267.2; nl 590.2 – 1377.2) and arachidic (8.86; nl 16.8 – 38.5) acids were low. Total polyunsaturated (1.6; nl 3.57 – 8.11) and total n-6 fatty acids (1.3; nl 3.3 – 7.1) were low. Seven fatty acid levels were elevated, apparently from de novo lipogenesis in the presence of insulin: myristic = 299.15 (nl 39.4 – 258.2), hexadecanoic = 300.41(nl 19.82 – 59.93), palmetoleic = 1197.60 (nl 68.5 – 570.2), vaccenic = 458.11 (nl 84.82 – 260.8), docosapentanoic = 32.8 (nl 9.2–32.1), docosenoic = 20.88 (nl 5.73 – 11.92). Interventions. Four-oil ILE was added as 20 grams once weekly, providing ∼4 grams of linoleic and ∼0.5 grams of ȧ-linolenic acids. Follow up free fatty acids showed normalization of ȧ-linolenic (27.07) and improvement in linoleic (889.74) and mead (60.34) acids. The triene to tetraene ratio improved to 0.17.

Conclusions: An elevated Mead acid level and elevated triene to tetraene ratio confirmed severe EFAD. Mead acid is produced from the elongation of oleic acid (n-9). The n-3, n-6, and n-9 fatty acids all compete for the same desaturases. But the desaturase enzymes have a preference for the fatty acids based on their n-terminal structure. The sequence is n-3 > n-6 > n-9. Therefore, increased Mead acid (20:3n-9) synthesis occurs only with very low availability of n-3 and n-6 fatty acids. In severe EFAD, Mead acid serves as a precursor to specific prostaglandins and leukotrienes via the cycloxygenase and lipoxygenase pathways. This case highlights the value of the QIP-PN in identifying EFAD in long-term HPN patients.

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