Prospective study of growth in Swedish children treated with modified ketogenic diet

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Purpose

Modified ketogenic diet (MKD) is one treatment option for intractable epilepsy and metabolic conditions such as glucose transporter type 1 deficiency syndrome (GLUT1-DS) and pyruvate dehydrogenase complex (PDC) deficiency. MKD is a less restrictive diet than the classical ketogenic diet (KD) and thus more tolerable and, moreover, some studies indicate that prolonged KD treatment can negatively affect linear growth in children. Long-term data is missing regarding the effects of MKD treatment in children. This study was designed to prospectively assess growth in children treated with MKD for 24 months.

Methods

The included patients (n=38; 21 girls, 17 boys) had a mean±SD age of 6.1±4.8 years at MKD initiation. Underlying etiologies were genetic epilepsy (n=6), GLUT1-DS (n=7), PDC deficiency (n=9), cortical malformation (n=3), mitochondrialopathy (n=2), tuberous sclerosis complex (n=2), encephalitis (n=2), stroke (n=2), Alcardi syndrome (n=1) and of unknown etiology (n=4). Thirty patients had seizures prior to MKD. Body weight, height and laboratory tests were assessed at baseline, 6, 12 and 24 months.

Results

After 24 months, 29 patients remained on MKD and 57% responded to the diet with >50% seizure reduction. Weight SDS and height SDS were stable over 24 months (P=0.054 and 0.10 respectively), i.e., weight SDS median (min-max) -0.4 (-2.5 to 3.5) at baseline and 0.2 (-1.8 to 2.1) after 24 months; and corresponding values for height SDS -0.4 (-4.0 to 2.5) to -0.3 (2.9 to 1.4). BMI SDS increased from 0.2 (-3.3 to 4.5) to 0.7 (-0.9 to 2.6) after 24 months, P=0.006. The median plasma 3-hydroxybutyryl acid levels increased from 0.05 mmol/L to 2.35 mmol/L (0.42-5.20 mmol/L) after 6 months, P<0.0001, but remained stable thereafter, i.e., 2.30 mmol/L (0.18-4.90 mmol/L) after 12 months and 2.30 mmol/L (0.16-4.90 mmol/L) at 24 months. Median pH was 7.38 (7.23-7.51) at baseline and 7.39 (7.34-7.45) after 24 months, P=0.45. At baseline, median IGF-I SDS was -0.15, which decreased to -0.85 after 6 months and to -1.00 at 12 months. From 12 to 24 months IGF-I SDS increased to 0.05. Median IGFBP3 SDS at baseline was 1.00 and was stable after 6 months, then decreased at 12 months to 0.10. From 12 to 24 months IGFBP3 SDS increased to 0.60.

Conclusion

This is the first study to assess the long-term effects on growth in children treated with the MKD.

No negative effect was observed on longitudinal growth after MKD treatment for 24 months.

Disclosures of interest: none.