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Longitudinal study of bone mass in Swedish children treated with modified ketogenic diet

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Conclusions

- This study demonstrates that MKD is effective for seizure reduction.
- Bone mass remained stable during MKD treatment for 24 months.
- ´MKD could be considered an effective and safe treatment option in childhood and adolescence.

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. Some studies indicate a negative effect on bone mass during KD treatment, probably as a consequence of the chronic acidic environment. Long-term data is missing regarding the effects of MKD on bone mass in children.

This study was designed to prospectively assess the effect on bone mass in children treated with MKD for 24 months.

Patients with intractable epilepsy

Gender	Age at start of MKD	Etiology	Epilepsy syndrome	Physical status	Seizures per month 0/6/12/24 months	DXA and DXL at 0/24 months
M	3.8	Unknown	Lennox Gastaut	A	200/8/8/8	DXA+DXL/ DXA+DXL
F	3.6	Mitochondriopathy	-	NA	200/20/20/10	DXL/DXL
M	2.3	Cortical malformation	Lennox Gastaut	NA	500/300/300/300	DXL/DXL
M	4.0	Genetic epilepsy	Doose	A	200/0/0/0	DXA+DXL/DXA+DXL
M	8.5	Encephalitis	Lennox Gastaut	NA	340/340/340/340	DXA+DXL/DXA+DXL
M	5.7	Unknown	Lennox Gastaut	NA	400/250/250/250	DXA+DXL/DXA+DXL
M	2.3	Genetic epilepsy	West syndrome	NA	500/300/300/300	DXL/DXL
F	3.0	Genetic epilepsy	Doose	A	300/0/0/0	DXL/DXL
M	5.4	Unknown	Lennox Gastaut	NA	1090/280/280/280	DXA+DXL/DXA+DXL
F	10.0	Tuberous Sclerosis	Lennox Gastaut	A	1532/50/50/50	DXA+DXL/DXA+DXL
M	5.5	Tuberous Sclerosis	Lennox Gastaut	A	60/10/10/10	DXL/DXL
F	4.8	Aicardi syndrome	Lennox Gastaut	A	36/22/22/22	DXL/DXL

Patients with GLUT1-DS

Gender	Age at start of MKD	Epilepsy syndrome	Physical status	Seizures per month 0/6/12/24 months	DXA and DXL at 0/24 months
F	17.2	Absence	A	30/0/0/0	DXA/DXA
F	13.2	Absence	A	5/0/0/0	DXA/DXA
F	3.6	Absence	A	40/0/0/0	DXL/DXL
F	1.5	–	A	0/0/0/0	DXL/DXL
M	4.2	Absence	A	30/0/0/0	DXA/DXA
M	16.7	Absence	A	11/0/0/0	DXA/DXA

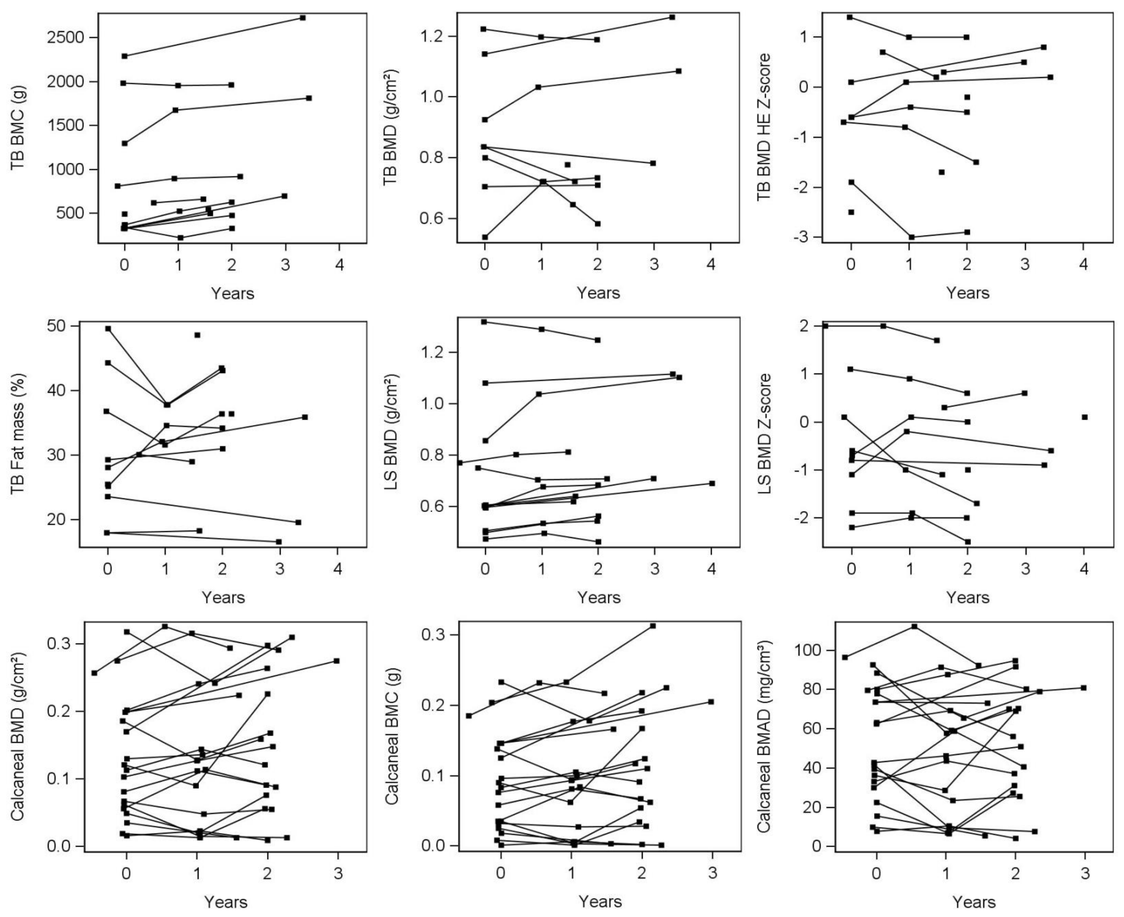
Patients with PDHCD

Gender	Age at start of MKD	Epilepsy syndrome	Physical status	Seizures per month 0/6/12/24 months	DXA and DXL at 0/24 months
F	2.6	–	NA→A	0/0/0/0	DXL/DXL
F	2.0	–	A	0/0/0/0	DXL/DXL
F	9.5	–	A	0/0/0/0	DXL/DXL
M	6.0	–	A	0/0/0/0	DXA+DXL/DXA+DXL
F	6.1	–	A	0/0/0/0	DXA+DXL/DXA+DXL

Methods

The included 23 patients (median age 4.8 years; 12 girls, 11 boys) were evaluated with whole body dual-energy X-ray absorptiometry (DXA) and/or calcaneal DXA and laser (DXL) at baseline and after 12 and 24 months on MKD. Underlying etiologies were genetic epilepsy (n=3), GLUT1-DS (n=6), PDC deficiency (n=5), cortical malformation (n=1), mitochondriopathy (n=1), tuberous sclerosis complex (n=2), encephalitis (n=1), Aicardi syndrome (n=1) and of unknown etiology (n=3). Growth parameters were assessed at baseline, 6, 12 and 24 months. DXA and DXL scans were performed at baseline, 12 and 24 months.

Results



In patients with seizures, 76% responded to the diet with >50% seizure reduction. DXA scans are missing in 11 patients due to low age (<5 years) and movement artefacts. Median (min-max) total body bone mineral density head excluded (TB BMD HE) Z-score was -0.6 (-2.5 to 1.4) at baseline and -0.5 (-2.9 to 1.0) after 24 months, *P*=0.25. Lumbar spine (LS) BMD Z-score was median -0.7 (-2.2 to 2.0) and -1.1 (-2.5 to 0.6), *P*=0.41. TB BMD HE Z-score was <-1 in 2 patients at baseline and <-1 in 3 patients after 24 months. LS BMD Z-score was <-1 in 3 patients at baseline and in 4 patients after 24 months. MKD treatment for 24 months did not have an effect on LS and TB bone mineral content (BMC). No differences in fat mass or lean mass were observed during the study period. Calcaneal BMD and BMC increased slightly during the study period, *P*=0.047 and 0.014, respectively.

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Disclosures of interest: none.

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