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Effects of Ketogenic Therapy on Behavioral and Metabolic Outcomes in a Child with Autism Spectrum Disorder and ADHD Symptoms: A Case Report

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Abstract

Background

Autism spectrum disorder (ASD) frequently co-occurs with attention-deficit/hyperactivity disorder (ADHD). Emerging evidence suggests that metabolic dysregulation may contribute to neurodevelopmental symptomatology, providing rationale for dietary intervention. In this context, the ketogenic diet (KD) has attracted growing interest, yet evidence regarding its effects on co-occurring ADHD symptoms in children with ASD remains limited.

Case presentation

We report a 9-year-old boy with ASD and prominent ADHD symptoms whose parents initiated a professionally supervised KD after limited benefit from prior interventions and adverse pharmacological effects.The diet progressed from low-carbohydrate(≤100 g/day) to full KD (≤20 g/day) over four weeks. At 10-month behavioral follow-up, the Vanderbilt parent rating scale showed reductions in inattentive (9/9 to 3/9), hyperactivity/impulsivity (8/9 to 6/9), and oppositional-defiant symptoms (7/8 to 1/8). Aberrant Behavior Checklist scores improved for Irritability/Agitation/Crying (17 to 8), Lethargy/Social Withdrawal (10 to 4), and Hyperactivity/Noncompliance (34 to 23). Social Communication Questionnaire score remained unchanged (14). Parents reported improvements in daily functioning, most apparent with consistent dietary adherence. Biochemical assessments demonstrated resolution of insulin resistance (HOMA-IR 4.24 to 1.03), normalization of weight-for-age (BMI-SDS +1.77 to −0.18), and improvements in lipid (TG/HDL ratio 0.92 to 0.53; HDL-cholesterol 1.2 to 1.7 mmol/L), inflammatory (CRP 4 to 1 mg/L), and nutritional status markers.

Conclusion

This case demonstrates concurrent behavioral and metabolic changes during KD therapy in a child with ASD and prominent ADHD symptoms. Although causality cannot be established from a single case, these findings support further investigation of ketogenic therapy in pediatric neurodevelopmental disorders.

Introduction

Neurodevelopmental disorders such as autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD) represent a substantial global health burden, together affecting millions of children worldwide (GBD 2019 Diseases and Injuries Collaborators, 2020). ASD is characterised by persistent difficulties in social communication and interaction, together with restricted and repetitive behavior. In contrast, ADHD is characterised by symptoms of inattention, hyperactivity and impulsivity, alongside impairments in executive functioning, emotional regulation and adaptive functioning (Faraone et al., 2024; Hyman et al., 2020). Comorbidity between ASD and ADHD is common, and children with both conditions often present with more severe behavioral, attentional and regulatory difficulties (Narzisi et al., 2026; Rommelse et al., 2010). Both disorders are associated with far-reaching consequences, including reduced quality of life (Van Heijst & Geurts, 2015; Faraone et al., 2024) and

substantial lifetime societal costs (Cakir et al., 2020; Dosie et al., 2012). Despite this impact, current treatment effects remain limited. ASD is typically managed through behavioral and psychopharmacological interventions, whereas ADHD is primarily treated with pharmacological approaches, supplemented by behavioral strategies as part of multimodal care (Aishworiya et al., 2022; Faraone et al., 2024; Hyman et al., 2020). Nonetheless, treatment outcomes are often insufficient, and many interventions fail to address underlying metabolic contributors. This emphasizes the need for additional, alternative therapeutic strategies.

In line with growing interest in alternative therapeutic strategies, dietary approaches have gained attention, with the ketogenic diet emerging as a promising metabolic intervention. This high-fat, moderate-protein, low-carbohydrate regimen induces nutritional ketosis, increasing circulating ketone bodies (Schrickel et al., 2025). These ketone bodies are metabolized within brain mitochondria to support ATP production, providing an alternative cerebral energy substrate for glucose (Hartman et al., 2007). For over a century, ketogenic dietary therapy has been an effective treatment for drug-resistant epilepsy (Martin-McGill et al., 2020), illustrating the relevance of metabolic interventions for brain metabolic function. Building on this, KD has emerged as a potential treatment option for ASD (Schrickel et al., 2025).

Metabolic irregularities reported in ASD include mitochondrial dysfunction, oxidative stress–related disturbances and altered glucose utilization, which have been linked to behavioral and social ASD-related symptoms (Bjørklund et al., 2020; Kumar et al., 2017; Pangrazzi et al., 2020; Rossignol & Frye, 2012). Current hypotheses suggest that KD may modulate these metabolic processes as well as other pathways implicated in ASD, including neurotransmission, mTOR signaling, and epigenetic regulation (Schrickel et al., 2025). The potential therapeutic relevance of KD in ASD is supported by preclinical and clinical observations showing improvements across multiple standardised measures, including the Childhood Autism Rating Scale (CARS and CARS-2), the Autism Treatment Evaluation Checklist (ATEC), and the Autism Diagnostic Observation Schedule (ADOS-2) (Evangeliou et al., 2003; Spilioti et al., 2013; Herbert & Buckley, 2013; El-Rashidy et al., 2017; Lee et al., 2018; Singh et al., 2026; Wang et al., 2026a; Wang et al., 2026b, Żarnowska et al., 2018). Reported benefits include improved cognition, sociability, language and reductions in stereotypies, with some cohort showing improvements in attention, emotional regulation, sensory responses and adaptability (Evangeliou et al., 2003; Herbert & Buckley, 2013; El-Rashidy et al., 2017; Lee et al., 2018; Żarnowska et al., 2018). Other studies similarly describe broad ASD-symptom ameliorations, including reduced anxiety, and improved attention, cognition, communication, social interaction and mood, with fewer adverse effects than conventional psychiatric or antiseizure medications (Coleman et al., 2019; Matthews & Adams, 2023).

Despite these promising preliminary findings, the evidence for efficacy of KD in neurodevelopmental conditions remains limited and has consisted primarily of ASD case studies and small cohorts with substantial variability in dietary protocols and outcome measures, with some studies combining KD with gluten-free and casein-free diets, which makes it difficult to attribute observed effects specifically to KD (Schrickel et al., 2025). Very recently, a randomized controlled trial in 62 young children with ASD found

greater reductions in Autism Behavior Checklist and CARS scores following two months of a modified KD combined with rehabilitation compared with rehabilitation alone (Liu et al., 2026). A second trial in 34 children with ASD reported greater reductions in GARS-3 scores after three months of a KD compared with a balanced diet (Salama et al., 2026).

Emerging evidence indicates that ADHD also involves metabolic dysregulation. Previous research showed mitochondrial dysfunction, increased oxidative stress and altered neuroenergetic metabolism in children with ADHD (Ram et al., 2026; Suwindi et al., 2026). These findings suggest that ADHD, like ASD, may involve neuroenergetic dysregulation, supporting the relevance of metabolic interventions such as KD. However, evidence for the efficiency of KD for ADHD is even more scarce than in ASD. Work consists mainly of indirectly relevant animal models (Liu et al., 2023; Packer et al., 2016), and no controlled human studies have been reported to date. In a recent case report, ADHD remission was described as part of broader psychiatric improvement during ketogenic therapy, although no validated ADHD-specific outcome measure was used (Bellamy & Laurent, 2025). Given the emerging evidence implicating metabolic and neuroenergetic alterations in ADHD, investigation of KD in relation to ADHD symptomatology is therefore warranted.

This case report describes a nine-year-old boy diagnosed with ASD, also presenting prominent ADHD symptoms. Following limited benefit from multiple previous therapeutic interventions and adverse effects associated with pharmacological treatment, professionally supervised KD was initiated at his parents' request. Psychological outcomes were assessed using the NICHQ Vanderbilt ADHD Diagnostic Parent Rating Scale (VADPRS), the Aberrant Behavior Checklist (ABC) and the Social Communication Questionnaire (SCQ), complemented by biological and metabolic measures. Qualitative parental observations were also included. The aim of this case report is to examine concurrent behavioral and metabolic changes during KD therapy in a child with ASD and prominent ADHD symptoms, thereby providing preliminary insights that may inform future research on ketogenic interventions in pediatric neurodevelopmental disorders. To our knowledge, this is the first pediatric case report specifically evaluating prominent ADHD symptoms alongside ASD during KD therapy, using ADHD-specific standardized measures.

Case Presentation

Patient Information and clinical history

The patient is a boy of mixed European and Indonesian ancestry who was nine years old at initiation of the ketogenic diet (KD) therapy. He lives with his parents and his sister, who is 2.5 years older. He was born after a full-term pregnancy marked by high fetal motor activity and significant maternal fatigue. Delivery was complicated by the infant becoming lodged in the birth canal, requiring assisted extraction. He presented with gray skin coloration at birth and required immediate intensive care admission for oxygen support, after which he recovered fully.

Early development was characterized by sleep difficulties, sensory sensitivity, a pronounced need for movement, and difficulties with behavioral regulation. From early childhood, overstimulation was associated with marked motor restlessness, repetitive behaviors, and reduced reciprocal interaction.

At age four, persistent sensory, behavioral, and social difficulties prompted specialized diagnostic assessment, resulting in diagnoses of Autism Spectrum Disorder (ASD) and global developmental delay according to DSM-5 criteria. Cognitive assessment indicated below-average functioning (IQ 83), although the result was considered a snapshot and interpreted with caution given his young age and restless behavior during testing. His difficulties substantially affected educational functioning. After initially starting mainstream primary school at age four, school attendance proved unfeasible, and he moved to a specialized pediatric daycare for nine months. He subsequently returned to mainstream primary school, which he attended part-time, averaging approximately three days per week and frequently required shortened school days due to sensory overload, as a greater school load was not considered feasible.

At age five, the Behavior Rating Inventory of Executive Function (BRIEF-P) indicated impaired inhibition, metacognition, and working memory, especially in group settings, while the Wechsler Preschool and Primary Scale of Intelligence (WPPSI-IV) showed age-appropriate cognitive scores (IQ 102).

From diagnosis onwards, the child received multiple non-pharmacological interventions, including support within a specialized pediatric daycare setting, occupational and sensory integration therapy, behavioral and environmental strategies, pediatric exercise therapy, and speech and language therapy. Speech and language therapy was initiated because of suspected language difficulties, including word-finding difficulties, phonological errors, and difficulties with sentence formulation. Assessment indicated below-average performance in comprehension, production, semantic development, and sentence formulation, and a developmental language disorder or dyslexic difficulties were considered. Limited therapeutic alliance and the high effort required led to discontinuation. These interventions yielded variable benefits; structured routines, visual supports, and sensory regulation strategies were reported to improve selected aspects of attention and behavioral regulation, while substantial difficulties with sensory overstimulation, restlessness, and daily functioning persisted.

School staff and parents reported observed difficulty sustaining attention and remaining seated, pronounced motor restlessness and impulsivity, and marked difficulty waiting his turn, alongside sensory overstimulation. ADHD was clinically suspected based on the persistence and functional impact of these symptoms. Although a formal ADHD diagnosis was not established because further diagnostic assessment was considered too burdensome for the child, these symptoms substantially affected daily functioning at school and prompted several ADHD-targeted medication trials. From age seven onwards, several medications were initiated for ADHD-related symptoms, including modified-release methylphenidate (from 9 June 2023, for 12 days), lisdexamfetamine (from 21 June 2023, for 14 days), clonidine (from 22 August 2023, for 52 days), atomoxetine (from 13 October 2023, for 276 days), and guanfacine (from 10 September 2024, for 149 days). Across these sequential treatments, little to no clinical benefit was observed, while adverse effects included increased restlessness and agitation,

heightened sensory sensitivity, abdominal pain, and headaches, resulting in treatment discontinuation. Aripiprazole (from 7 March 2023, for ~ 330 days), also initiated at age seven to address overstimulation and associated behavioral dysregulation, produced a rapid and noticeable improvement.

Despite increasing educational and behavioral support and effective medication, difficulties with sensory overload, sustained attention, self-regulation, and task-focused behavior persisted throughout standard primary school (from age 4), ultimately resulting in a transfer from mainstream to special education at age nine. The Wechsler Intelligence Scale for Children (WISC-V) administered at age nine showed difficulties with visual-spatial understanding, working memory, and processing speed.

Bupropion was introduced at age nine to improve attention and school functioning. At 150 mg/day, the patient reported some improvement in his ability to focus; a temporary increase to 300 mg/day was poorly tolerated, and the dose was reduced to 150 mg/day and continued during the KD therapy. Although effective, aripiprazole treatment was ultimately discontinued because of reduced satiety and associated weight gain, together with parental concerns regarding potential long-term metabolic effects. The tapering of aripiprazole, which ended with its discontinuation in early 2026 (around M2; exact end date unknown), overlapped with the first months of the KD and is considered a potential confounder in interpreting outcomes.

Given the persistence of ASD-related difficulties and prominent ADHD symptoms despite multiple previous interventions, the parents explored alternative treatment options and decided to pursue a professionally supervised KD. Baseline assessments were obtained before initiation of the dietary intervention in July 2025.

Therapeutic Dietary Intervention

The dietary intervention was initiated on 10 July 2025 with a low-carbohydrate diet (≤ 100 g carbohydrate/day) and gradually transitioned to the full KD (≤ 20 g carbohydrate/day) on 6 August, 2025, under professional supervision by a registered dietitian. Comprehensive baseline biochemical and anthropometric assessments were completed in July 2025 (Metabolic test 1: M1), prior to any dietary changes. At the time of data closure (July 2026), the patient continued the ketogenic diet.

Diet prescription. The intervention followed a carbohydrate-restricted KD with a target of ≤ 20 g carbohydrate/day. No fixed macronutrient ratio was prescribed; fat constituted the predominant energy source and protein was maintained at approximately 1.0–1.5 g/kg body weight/day.

Supplementation. At KD initiation, magnesium (200 mg/day) and omega-3 fatty acids (360 mg EPA + 240 mg DHA) were prescribed. From April 2026, the parents independently added acetyl-L-carnitine (2000 mg/day), vitamin D3 + K2 (1000–2000 IU/day), vitamin C (800 mg/day), ubiquinol/CoQ10 (50 mg/day), R-alpha-lipoic acid (100 mg/day), and N-acetylcysteine (300 mg/day).

Monitoring. Ketosis was monitored daily during the initiation phase through capillary blood beta-hydroxybutyrate (BHB) measurements. Parent-obtained spot measurements were obtained regularly throughout the intervention; the values reported here represent documented patient measurements. Measurements confirmed nutritional ketosis within the first weeks following full KD initiation (BHB 1.6 mmol/L on 17 August 2025 and 0.8 mmol/L on 24 August 2025; Supplementary Table 1); a spot measurement approximately 9 months after full KD initiation yielded 2.9 mmol/L. A planned dietary exception at a school Christmas dinner temporarily interrupted ketosis (BHB 0.03 mmol/L). During a structured monitoring period in July 2026, postprandial BHB was measured daily at approximately 14:00–17:00 h; values ranged from 0.4 to 1.8 mmol/L (Supplementary Table 2), with nutritional ketosis (BHB ≥ 0.5 mmol/L) present on six of eight days. Following a planned dietary deviation, return to ketosis occurred within three days.

Adherence and social adaptation. Parents reported generally good adherence based on regular dietary tracking using a food logging application, though these data were not documented in a traceable way. Strategies to support long-term adherence and social participation included preparing ketogenic meals in advance for attendance at a therapeutic care farm every three weeks and keeping ketogenic snack alternatives at school for social occasions. In addition, structured flexibility was incorporated for selected social occasions, such as the planned dietary exception at the school Christmas dinner. Together, these strategies supported sustained dietary therapy while allowing participation across home, school, care, and social settings.

Adverse effects. No clinically significant adverse effects were observed during the intervention period.

Concurrent medication. Bupropion (150 mg/day) was continued throughout the dietary intervention to support attentional functioning. Aripiprazole, prescribed for overstimulation and behavioral dysregulation, was gradually tapered during the first months of the dietary intervention and discontinued in early 2026 (exact date unknown).

Outcomes

Behavioral and metabolic outcomes were assessed at different time points. Behavioral assessments were conducted at baseline (B1; June 2025) and approximately 10 months after intervention initiation (B2; May 2026). Metabolic assessments were conducted at baseline (M1; July 2025), approximately 7 months (M2; February 2026), and 12 months (M3; July 2026) after intervention initiation. The intervention timeline, assessment time points, and available blood ketone measurements are illustrated in Fig. 1.

Behavioral Assessment

Questionnaires were completed by the parents at two time points: at baseline before initiation of the dietary intervention (B1; June 2025) and at follow-up approximately 10 months after intervention

initiation (B2; May 2026). The Social Communication Questionnaire (SCQ) is a caregiver-screening instrument for symptoms associated with Autism Spectrum Disorder (ASD) (Berument et al. 1999). The SCQ is not a diagnostic tool itself and warrants a follow up ASD psychological assessment. A score above 15 shows a strong indication for the presence of ASD and calls for further ASD diagnostic assessment.

The NICHQ Vanderbilt ADHD Diagnostic Parent Rating Scale (VADPRS) was completed by the parents to characterize ADHD symptomatology (Inattention, Hyperactivity/Impulsivity, Performance) and screen for co-occurring conditions like oppositional Defiant Disorder (ODD), Conduct Disorder (CD) and Anxiety/Depression (NICHQ, 2002; Wolraich et al. 2003). A positive screen for ADHD requires ≥ 6/9 symptoms on the Inattention and Hyperactivity/Impulsivity subscale, combined with at least 1 symptom on the Performance subscale.

The Aberrant Behavior Checklist (ABC) is a caregiver-completed rating scale that measures behavior change in children with intellectual disabilities and ASD (Aman et al. 1985). It is not a diagnostic tool, but rather yields five empirically derived subscale scores - Irritability, Agitation, Crying; Lethargy/Social Withdrawal; Stereotypic Behavior; Hyperactivity/Noncompliance; and Inappropriate Speech - used primarily to quantify treatment-related change in problem-behavior severity over time (Aman et al. 1985; McCracken et al. 2002).

Behavioral Outcomes

Results show that the SCQ total score did not change between baseline and follow-up (total score of 14 at baseline and follow-up). Results of the VADPRS are presented in Table 1. It is apparent that at follow-up, the patient no longer met the clinical threshold for the Inattention subscale for ADHD (≥ 6/9 items ≥ 2 = positive screen), whereas the criteria for Hyperactivity/Impulsivity remained met. The Oppositional-Defiant Disorder subscale likewise showed a great reduction in symptom count (7/8 to 1/8 items scored ≥ 2), thus no longer meeting the positive screen for Oppositional-Defiant Disorder. Moreover, the Performance Subscale also showed a substantial reduction in item scores (from 3/8 to 1/8 items rated ≥ 4). For the Conduct Disorder subscale and the Anxiety/Depression subscale, the symptom count remained below the positive screen and thus showed no meaningful change (0/14 to 0/14 and 2/7 to 1/7, respectively).

The ABC results showed major reductions in the Irritability/Agitation/Crying subscale and the Lethargy/Social Withdrawal subscale (52.9% and 60.0%, respectively; Table 2). At baseline, the Hyperactivity/Noncompliance subscale had the highest score among the five ABC subscales, and it also showed a substantial reduction at follow-up (32.4%).

Table 1

Outcomes of the National Institute for Children's Health Quality (NICHQ) Vanderbilt Attention deficit hyperactivity disorder (ADHD) Diagnostic Parent Rating Scale

Subscale (*)	**Baseline (B1)**	**Follow-up (B2)**
Inattention (*≥ 6/9)*	9	3
Hyperactivity/Impulsivity (*≥ 6/9)*	8	6
Performance (*≥ 1/8)*	3	1
Oppositional-Defiant Disorder (*≥ 4/8)*	7	1
Conduct Disorder (*≥ 3/14)*	0	0
Anxiety/Depression (*≥ 3/7)*	2	1

Note: Baseline assessment was conducted before initiation of the dietary intervention (B1; June 2025); follow-up assessment was conducted approximately 10 months after intervention initiation (B2; May 2026). (*) Positive screen thresholds per subscale (NICHQ, 2002; Wolraich et al. 2003). *A positive screen for ADHD requires ≥ 6/9 symptoms on the Inattention and Hyperactivity/Impulsivity subscale, combined with at least 1 symptom on the Performance subscale (NICHQ, 2002;* Wolraich et al. 2003; Anderson et al. 2022).

Table 2

Outcomes of the Aberrant Behavior Checklist

Subscale (*)	**Baseline (B1)**	**Follow-up (B2)**	**Percentage reduction**
Irritability/Agitation/Crying (45)	17	8	52.9%
Lethargy/Social Withdrawal (48)	10	4	60.0%
Stereotypic Behavior (21)	1	0	100%
Hyperactivity/Noncompliance (48)	34	23	32.4%
Inappropriate Speech (12)	2	2	0.0%

Note: Baseline assessment was conducted before initiation of the dietary intervention (B1; June 2025); follow-up assessment was conducted approximately 10 months after intervention initiation (B2; May 2026). (*) Maximum subscale score.

Biological and Metabolic Outcomes

Biological and metabolic parameters were assessed at baseline (M1; July 2025), prior to dietary intervention initiation, and at approximately 7 months (M2; February 2026) and 12 months (M3; July 2026) after intervention initiation. Laboratory analyses were performed by STAR-SHL Medisch Laboratorium, The Netherlands. All biological and metabolic outcomes are summarized in Table 3.

Anthropometric outcomes

Reductions were observed in weight (46 to 38.1 kg) and BMI-SDS (+ 1.77 to − 0.18) between M1 and M3 At baseline, BMI-SDS was + 1.77 (96th percentile; WHO 2007), representing normalization of weight-for-age status (96th percentile to 43rd percentile; WHO 2007).

Nutritional status

Nutritional markers improved across the KD period. Vitamin D increased from 108 to 125 nmol/L, ferritin from 73 to 126 µg/L, vitamin B12 from 267 to 363 pmol/L, and folate from 12 to 22 nmol/L (all M1 to M3). Homocysteine decreased from 7.6 to 6.2 µmol/L (from M1 to M3, not measured at M2). All values remained within reference ranges throughout.

Thyroid function

Thyroid function remained within reference ranges at all time points. TSH (1.28, 1.83, 1.66 mIU/L) and free T4 (16.7, 17.0, 15.5 pmol/L) levels were stable. Free T3 decreased from 6.31 to 4.83 pmol/L (M1 to M3; not measured at M2), consistent with a recognized physiological adaptation to carbohydrate restriction: reduced insulin levels suppress deiodinase activity, impairing peripheral T4-to-T3 conversion (Vranjić et al., 2025). No clinical features of hypothyroidism were present.

Markers of insulin resistance and glycemic regulation

Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) decreased from 4.24 at M1 to 1.03 at M3, falling below the threshold for insulin resistance (> 2.21); fasting insulin was not measured at M2. Fasting glucose decreased progressively (from 5.1 → 4.9 → 4.5 mmol/L). HbA1c remained stable throughout (31, 29, 30 mmol/mol).

Lipid profile

HDL-cholesterol increased from 1.2 to 1.7 mmol/L (M1 to M3). Triglycerides decreased from 1.1 (M1) to 0.7 mmol/L(M2) and 0.9 mmol/L (M3). The TG/HDL ratio improved from 0.92 (M1) to 0.53 (M3).

Inflammatory and hepatic markers

CRP decreased from 4 mg/L (M1) to 1 mg/L (M2 and M3). Gamma-GT, was mildly elevated at baseline (19 U/L; reference 0–17 U/L), and normalized to 15 U/L at M2 and M3.

Table 3
Biological and metabolic outcomes across three assessment time points.

Parameter	Unit	Reference range	M1 (Jul 2025)	M2 (Feb 2026)	M3 (Jul 2026)
Anthropometric					
Weight	kg	—	46	~ 37 a	38.1
Height	cm	—	—	—	150
BMI	kg/m²	—	—	—	16.9
BMI-SDS (WHO 2007)	SD	—	+ 1.77 (P96)	~+0.61 (P73) a	−0.18 (P43)
Nutritional status					
Ferritin	µg/L	30–400	73	137	126
Vitamin B12	pmol/L	148–584	267	375	363
Folate	nmol/L	5–61	12	19	22
Homocysteine	µmol/L	5.0–15.0	7.6	n.d.	6.2
Vitamin D (25-OH)	nmol/L	50–150	108	97	125
Thyroid function					
TSH	mIU/L	1.12–5.01	1.28	1.83	1.66
Free T3	pmol/L	3.88–8.02	6.31	n.d.	4.83
Free T4	pmol/L	12–22	16.7	17.0	15.5
Glycemic and insulin resistance markers					
HOMA-IR b	—	< 2.21	4.24	n.d.	1.03
Fasting glucose	mmol/L	4.0–6.0	5.1	4.9	4.5
HbA1c	mmol/mol	20–42	31	29	30
Lipid profile					
Triglycerides	mmol/L	0.6–2.2	1.1	0.7	0.9
HDL-cholesterol	mmol/L	0.9–1.7	1.2	1.5	1.7
TG/HDL ratio c	—	< 1.7	0.92	0.47	0.53
Inflammatory and hepatic markers					

Parameter	Unit	Reference range	M1 (Jul 2025)	M2 (Feb 2026)	M3 (Jul 2026)
Anthropometric					
CRP	mg/L	0–10	4	1	1
gamma-GT	U/L	0–17	19	15	15

Note. M1 represents baseline values prior to dietary intervention initiation; M2 and M3 represent approximately 7 and 12 months after intervention initiation, respectively. Reference ranges per STAR-SHL Medisch Laboratorium.

a M2 weight estimated from parental report (~ 37 kg); BMI-SDS calculated indicatively.

b HOMA-IR calculated as [fasting insulin (µIU/mL) × fasting glucose (mmol/L)] / 22.5.

c TG/HDL ratio = triglycerides (mmol/L) / HDL-cholesterol (mmol/L).

Parent- and patient-reported Changes in Daily Functioning

During the KD intervention, the parents reported changes in several aspects of everyday functioning. They observed a marked improvement in his ability to sustain attention; for example, he was able to read independently for approximately 45 minutes, whereas previously he typically stopped after approximately 5 minutes. Changes were also apparent during family meals. While he had previously repeatedly left the table during dinner, he was increasingly able to remain seated throughout the meal, which the parents described as bringing greater calm to family life. The parents further reported fewer incidents of physically aggressive behavior towards other children and less resistance and conflict around bedtime.

The KD was implemented with some intentional flexibility around special occasions. According to the parents, behavioral improvements were most apparent during periods of consistent adherence and diminished when adherence was relaxed. Although based on subjective observation, this recurring pattern suggests a temporal association between dietary adherence and behavioral functioning. The parents further observed that behavioral symptoms consistent with the pre-intervention presentation tended to re-emerge within one to two days following dietary deviation and resolved upon return to ketosis.

The patient himself also reported awareness of this association, describing off-diet experiences as “ants in his head.” He contrasted his mental state on the KD with his off-diet state, characterizing them as a “slow rollercoaster” versus a “fast rollercoaster,” respectively. This patient-reported phenomenology, although anecdotal, was consistent with the parental observations described above.

Discussion

In this case report, implementation of a professionally supervised KD was accompanied by changes across several behavioral and functional domains in a child with ASD and prominent ADHD symptoms. The most pronounced quantitative changes were the observed VADPRS and the ABC scores. At behavioral follow-up (B2) approximately 10 months after dietary intervention initiation, the patient no longer met the positive-screen threshold for the Inattention and Oppositional-Defiant Disorder subscales of the VADPRS, while Hyperactivity/Impulsivity symptoms were reduced but remained above the subscale threshold. On the ABC, reductions were observed in Irritability/Agitation/Crying, Lethargy/Social Withdrawal, and Hyperactivity/Noncompliance. In contrast, the SCQ score remained unchanged, suggesting that the observed changes were more apparent in attentional, regulatory, and behavioral domains than in the social-communication symptoms captured by this measure.

These quantitative findings were accompanied by parent-reported changes in everyday functioning. The parents described improvements in sustained attention, the ability to remain seated during family meals, behavioral regulation, and family functioning, as well as fewer incidents of physical aggression and less conflict around bedtime. These observations broadly corresponded with the domains showing change on the VADPRS and ABC. The parents additionally reported that behavioral improvements were most apparent during periods of consistent dietary adherence and that symptoms tended to re-emerge following dietary deviations and resolve upon return to ketosis. The patient's own descriptions of his mental state on and off the diet were consistent with this reported pattern. These observations are anecdotal and cannot establish a diet-related effect, but provide complementary information on perceived changes in daily functioning alongside the standardized behavioral measures.

The behavioral changes observed in this case are broadly consistent with previous reports of behavioral improvement during ketogenic dietary interventions in children with ASD. Prior studies have reported changes across domains including attention, social functioning, communication, and behavioral regulation (Evangeliou et al., 2003; El-Rashidy et al., 2017; Lee et al., 2018; Żarnowska et al., 2018). More recently, two randomized controlled trials in children with ASD reported greater symptom reductions with a KD than with control conditions (Liu et al., 2026; Salama et al., 2026). With respect to the ADHD-related symptoms central to the present case, Żarnowska et al. (2018) is particularly relevant, as they described improvements in attention and hyperactivity during ketogenic dietary therapy in a child with ASD and co-occurring ADHD. However, ADHD symptoms were not evaluated using an ADHD-specific standardized measure in that case. Evidence specifically addressing ADHD symptoms during ketogenic therapy therefore remains very limited, with no controlled human studies specifically evaluating ADHD outcomes to date. The present case extends these preliminary observations by documenting changes in ADHD-related symptom domains using the VADPRS, while the single-case design and concurrent treatment changes preclude conclusions regarding efficacy or causality.

The metabolic changes observed include Resolution of insulin resistance (HOMA-IR 4.24 to 1.03), normalization of weight-for-age (BMI-SDS + 1.77 to − 0.18), improvement in the TG/HDL ratio (0.92 to 0.53), reduction in CRP (4 to 1 mg/L), and normalization of mildly elevated gamma-GT. These observations are consistent with known effects of sustained carbohydrate restriction (Paskaleva IN et

al., 2025; Paoli A et al., 2023) and with findings reported in other case reports of KD in pediatric neurodevelopmental conditions (Varesio C et al. 2021; Omori NE et al., 2024; Öztürk E et al., 2024). Several mechanisms have been proposed to explain neurobehavioral effects of the KD, including ketone body utilization as an alternative cerebral fuel, modulation of GABAergic tone, reduced neuroinflammation, and mitochondrial effects (Anderson J et al., 2024; Ahire C et al., 2026); which, if any, contributed to the present case cannot be determined. The temporal association between dietary adherence and behavioral functioning lends indirect support to a diet-mediated effect, though pharmacological confounders preclude firm attribution.

Several aspects of this case strengthen its descriptive value, including longitudinal assessment across behavioral, functional, and biological domains, the use of standardized behavioral measures, and professional supervision of the dietary intervention. At the same time, important limitations should be considered. As a single case without a control condition, the observed changes cannot be attributed causally to the KD. Several outcomes relied on parental reporting and may therefore be influenced by expectancy or reporting effects. In addition, concurrent medication changes, particularly the tapering and discontinuation of aripiprazole around the period of dietary intervention initiation, as well as the introduction of several supplements at initiation and later in the intervention period, represent potential confounders.

Taken together, this case should be regarded as hypothesis-generating rather than evidence of treatment efficacy. Future prospective studies should evaluate KD therapy in pediatric neurodevelopmental populations using standardized behavioral outcomes, objective structured measures of dietary adherence and ketosis, and longitudinal metabolic assessments. Combining behavioral and metabolic phenotyping may help determine whether specific symptom profiles or metabolic characteristics are associated with treatment response.

Conclusion

This case describes concurrent behavioral, functional, and metabolic changes during a professionally supervised KD in a child with ASD and prominent ADHD symptoms. Improvements in standardized behavioral measures were accompanied by parent-reported changes in everyday functioning, including sustained attention, behavioral regulation, and family functioning. Although the temporal relationship with KD adherence is noteworthy, the single-case design, concurrent medication and supplementation changes, and reliance on parent-reported outcomes preclude causal inference. This case supports further systematic investigation of KD and other metabolic interventions in children with neurodevelopmental disorders, including the identification of potential metabolic and clinical predictors of treatment response.

Statements and Declarations

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Competing interests

Noor Struik is founder of The Nourishing State, a private dietetic practice that provides ketogenic dietary therapy. The ketogenic dietary therapy described in this case was provided by another dietitian employed by the practice and paid by the patient's parents; Noor Struik did not personally provide the patient's dietary care. The other authors declare no competing interests.

Author contributions

Noor Struik: Data curation, investigation, formal analysis, writing original draft, review and editing.

Kate Dreuning: Data curation, investigation, interviewing, writing original draft.

Jonna Grootveld: Formal analysis, writing original draft.

Eline Dekeyster: Conceptualization, methodology, supervision, project administration, writing original draft, review and editing.

All authors reviewed and approved the final manuscript.

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Data availability

The data presented in this case report contain sensitive clinical information from a minor and are therefore not publicly available.

Ethics and informed consent

Written informed consent for publication of this case report, including the use of clinical information and outcome data, was obtained from the patient's parents.

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Figures

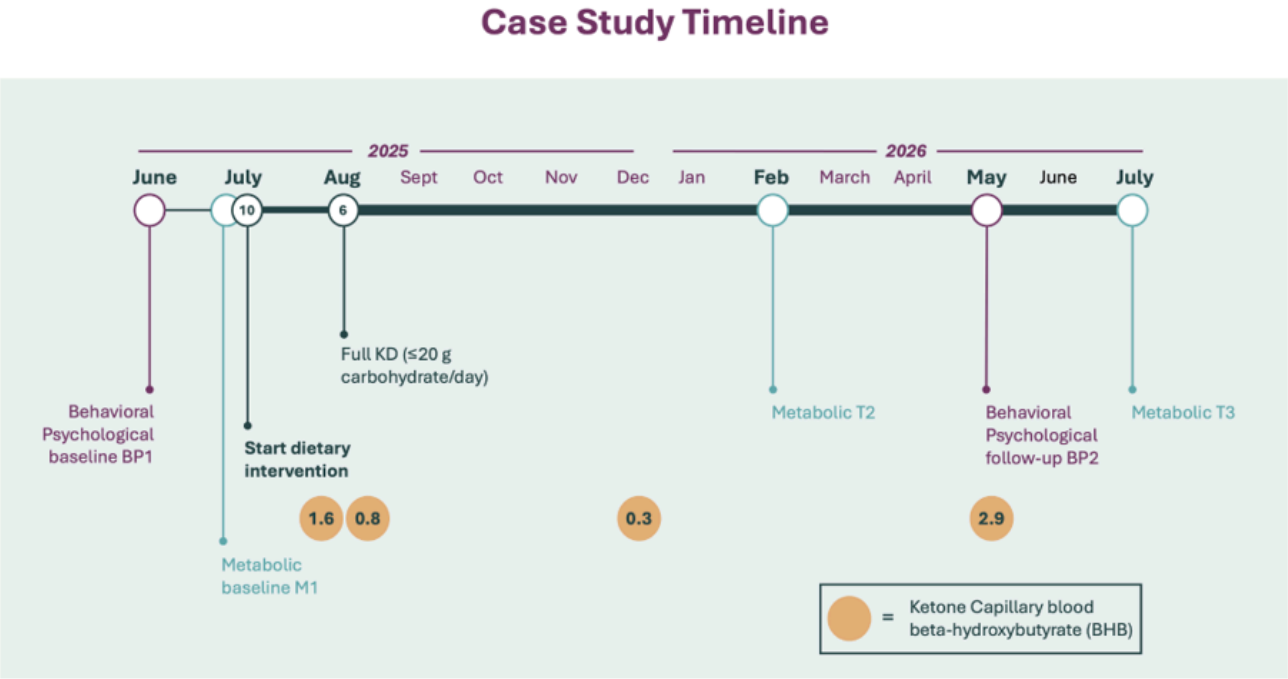


Figure 1

Timeline of the dietary intervention and outcome assessments.

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