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Assessment of Serum Vitamin D3 and Lipid Profile Level in Children with Autism Spectrum Disorder in Sana'a City, Yemen

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Abstract

Background: Autism spectrum disorder (ASD) is a complex neurodevelopmental condition characterized by persistent deficits in social communication, restricted patterns of behavior, and a wide range of cognitive and sensory processing differences. The global prevalence of ASD has increased significantly over the past two decades, raising scientific interest in understanding its multifactorial etiology. The pathogenesis of autism spectrum disorder (ASD) remains unclear and is considered highly complex. However, current evidence suggests that ASD arises from a multifactorial interplay between genetic predisposition and environmental factors that alter brain development and neural function. This study aimed to assess the levels of serum vitamin D3 and lipid profiles in children with autism spectrum disorder in Sana'a city, Yemen.
Methods: This case-control study was conducted from January to December 2025. The sample size will be 120, divided into 60 cases and 60 controls each. A venous blood sample (approximately 5 mL) was collected from each participant in the morning after an overnight fast (8-10 hours) into labelled plain test tubes under aseptic conditions. Demographic and clinical information, including age, sex, weight, height, body mass index (BMI), type of birth, and mothers' and fathers' ages at childbirth, were collected. Data were entered and analyzed using the Statistical Package for the Social Sciences (SPSS) version 25.
Result: This study population included children with autism spectrum disorder (ASD) who were attending specialized autism centers in Sana'a city, Yemen, to assess the levels of serum Vit D3 and lipid profiles in children with autism spectrum disorder. We found a statistical significance of VitD3 and HDL among cases and controls, with a median (IQR) of VitD3 (18.5 (10.2)) compared to controls (23.5 (11)) with P = 0.01. The median (IQR) of HDL of case 40 (14) compared to control 44 (13), with P = 0.01. Non-statistically significant findings of cholesterol, triglyceride, and LDL with mean ± SD and median (IQR) of the control 159±27.7, 115.5(26), and 87(23), respectively, compared to the mean ± SD and median (IQR) of the case 159±20, 116(50), and 88.5(36), respectively. Although there was a non-significant difference between the case and control groups in terms of BMI, type of birth, and father and mother age at the baby's birth.
Conclusion: This study highlights whether there are differences in vitamin D3 and lipid profile levels between children and typically developing children in Sanaa City, Yemen.

Article Info

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1. INTRODUCTION

Autism spectrum disorder (ASD) is a multifaceted neurodevelopmental condition marked by ongoing challenges in social communication, limited behavioral patterns, and a broad array of differences in cognitive and sensory processing [1]. The global prevalence of autism spectrum disorder (ASD) has markedly increased over the past two decades, prompting heightened scientific

interest in elucidating its multifactorial etiology [2].The male-to-female prevalence ratio of ASD is estimated to be approximately 4.5:1[3]. According to the 2021 Global Burden of Disease study, the global prevalence of autism spectrum disorder (ASD) is estimated to be approximately 788.3 per 100,000 people, or roughly 0.79%, indicating a widespread neurodevelopmental condition[4]. Although genetic predisposition plays a central role, accumulating evidence suggests that environmental, metabolic, immunological, and hormonal factors contribute substantially to the pathophysiology of ASD. Among these biological determinants, vitamin D status and lipid profile have emerged as critical areas of investigation because of their essential roles in neurodevelopment, brain maturation, and immune modulation[5]. Vitamin D is increasingly recognized as a neuroactive steroid rather than as a simple micronutrient. It regulates neuronal differentiation, synaptic plasticity, oxidative stress pathways, and immune responses in the developing brain[1]. Evidence indicates a significant deficiency in Vitamin D levels among children diagnosed with ASD compared to their neurotypical peers[5]. The proposed mechanisms linking vitamin D deficiency to ASD include impaired neurotrophic signalling, increased neuroinflammation, oxidative stress, and altered serotonin synthesis, each of which may contribute to characteristic ASD symptoms[6]. Moreover, prenatal and early life vitamin D insufficiency has been associated with greater ASD risk, suggesting that vitamin D may influence the neurodevelopmental trajectory long before clinical symptoms become apparent[2]. Recent evidence has highlighted the potential role of metabolic markers, particularly lipid profile abnormalities, in the pathophysiology of ASD. Several studies have reported altered levels of triglycerides, total cholesterol, LDL, and HDL in children with ASD compared to typically developing controls[7, 8]. Dyslipidemia may contribute to neurodevelopmental disturbances through multiple mechanisms, including increased oxidative stress, impaired neuronal membrane fluidity, disruption of synaptic signalling, and heightened inflammatory responses[8]. Increased triglyceride levels have been identified as a potential indicator of greater symptom severity in children diagnosed with ASD, suggesting that lipid imbalance may not only reflect metabolic dysfunction but could also influence behavioral and cognitive outcomes [9]. Additionally, abnormalities in HDL levels, given its antioxidant and anti-inflammatory properties, may play a role in modulating neuroinflammation, which is frequently reported in ASD[7]. These findings indicate that lipid metabolism is an important biomarker domain that may interact with hormonal and nutritional factors, such as vitamin D and thyroid hormones, further highlighting the multifactorial nature of ASD[9]. Despite the growing interest in these biological factors, data remain limited in many regions, particularly in developing countries, where vitamin D deficiency is prevalent in children with ASD, and screening is not consistently performed. Therefore, this study aimed to assess vitamin D levels and lipid profiles in children diagnosed with autism spectrum disorder and compare these parameters with those of age-matched typically developing children. Ultimately, understanding these relationships may support early intervention efforts and offer opportunities for improved patient care in populations with limited data. Therefore, this study aimed to assess the levels of serum Vit D and lipid profiles in children with autism spectrum disorder.

2. MATERIALS AND METHODS

2.1. Subjects and Data Collection

A case-control study will be conducted from January to April 2025. The study population included 120 children aged 3-15 years: 60 children with autism spectrum disorder (ASD) and 60 healthy children in Sanaa city. Children with autism spectrum disorder (ASD) who attended specialized autism centers in Sana'a city (Al-Tahadi, Khatwah, and Ebni centers, Humanitarian Cooperation Foundation for the Care of People with Special Needs, and CARS center) had their diagnoses of ASD confirmed by the Childhood Autism Rating Scale (CARS-2) in this center. The study protocol was approved by the Institutional Review Board (IRB) of the Faculty of Medicine and Health Sciences, Sana'a University, Sana' a, Yemen. Informed consent was obtained from all individuals after explaining the purpose and nature of the study. Multiple clinical assessments and biochemical tests were performed for each participant. These included weight and height assessments that were used to calculate the body mass index (BMI), mother and father age at the baby's birth, type of birth, and age. The exclusion criteria were children with autism who had received vitamin D or omega-3 supplementation within the last 6 and 2 months, respectively, before joining the study.

2.2. Sample collection

A venous blood sample (approximately 5 mL) was collected from each participant in the morning after an overnight fast (8-10 hours) into labelled plain test tubes under aseptic conditions. All tubes were protected from sunlight by placing them in a sample icebox for biological samples. The samples were then left to clot at room temperature, then transported to the laboratory within 2 hours, and sera were separated by centrifugation at 3000 rounds per minute for 10 minutes, then transferred and separated into 2 labelled Eppendorf tubes. One of the covered Eppendorf tubes was used to estimate the level of 25(OH)D, and the other covered Eppendorf tube was used for the lipid profile (total cholesterol, triglycerides, HDL-c, and LDL-c). The samples were then analyzed immediately.

2.3. LABORATORY TEST

Samples will be measuring Vitamin D by using a fully automated hormone machine (Cobas e411, Germany) and a semi-automated machine (Evolution 3000 semi-auto biochemistry analyzer, Italy) to measure total cholesterol, HDL-c, LDL-c, and triglyceride levels.

2.4. STATISTICAL ANALYSES

Data were entered and analyzed using the Statistical Package for the Social Sciences (SPSS) version 25. Descriptive statistics, including the mean ± standard deviation (SD) for continuous variables and frequencies with percentages for categorical variables, were used to summarize the data. The Shapiro-Wilk test was applied to assess the normality of the data distribution. Comparisons between the case and control groups were performed using the independent samples t-test for normally distributed variables, the Mann-Whitney U test for non-normally distributed data, and the one-way ANOVA test. The chi-square test (χ^2) was used to compare categorical variables between groups. The risk and its significance will be calculated using the odds ratio test with a 95

3. RESULTS

Table 1 shows the general demographic data of children with ASD and the controls. Age, sex, BMI, type of birth, age of mother at birth, and age of father at birth. We found no significant differences between the terms. Among the 120 participants, 90 (75%) were male and 30 (25 %) were female. The male-to-female prevalence ratio of ASD is estimated to be approximately 4.5:1[3]. There was no statistically significant difference between the two groups in terms of type of birth, father, or mother's age at birth.

Table 2 presents a comparison of biochemical parameters (serum vitamin D, total cholesterol, triglycerides, LDL-c, and HDL-c mean and mean rank between study groups. There was a significant difference in vitamin D3 and HDL levels between the case and control groups. The median (IQR) of the Vit. D_3 is 18.5 (10.2), compared to the control of 23.5 (11) with P = 0.01. The median (IQR) of HDL of case 40 (14) compared to the control 44 (13) with P=0.01. There was also no statistical significance in total cholesterol, triglycerides, and LDL between the case and control groups. The mean ± SD of total cholesterol of the case group (159.7±20.6) compared to the control (159.2±27.7) with p = 0.9. The median (IQR) of triglycerides in the case group was 116(60) compared to the control group (115.5(26)) with a P=0.9. The median (IQR) of LDL in case 88.5 (36). Compared to the control 87(23) with p=0.4.

Table 3 presents a comparison of biochemical parameters based on ASD severity to show the significance of the severity of ASD on triglyceride levels (P=0.01). non-significance of severity of ASD, 25(OH)D, cholesterol, LDL, and HDL (P=*0.6*, P=0.29, P=0.9, and P=0.3, respectively).

4. DISCUSSION

The present study evaluated serum vitamin D3 and lipid profiles (TC, TG, HDL-c, and LDL-c) in children diagnosed with autism spectrum disorder (ASD) compared to age- and sex-matched healthy controls in Yemen. The case-control study comprised 120 participants, divided equally into the ASD and control groups. A male predominance was observed among participants, aligning with established epidemiological data indicating that the prevalence of ASD is approximately four times higher in males than in females[10].This study showed a significant reduction in serum vitamin D3 levels in the ASD group. This result corroborates several previous studies; for instance, Singh et al. (2022) reported lower vitamin D3 concentrations in the ASD group[11]. Similarly, Mansour et al. (2024) found that 63.8% of children with ASD exhibited vitamin D insufficiency, while 28.8% suffered from deficiency[10]. Further support is provided by Desoky et al. (2017) and Savas et al. (2024), who identified statistically significant reductions in vitamin D3 levels in patients with ASD[12, 13]. Conversely, some studies, such as that by Hashemzadeh et al. (2015), found no significant association between vitamin D3 and ASD, potentially due to limited sample sizes[14]. Mechanistically, vitamin D acts as a hormone that regulates approximately 900 genes involved in brain development[15, 16]. It activates the transcription of tryptophan hydroxylase 2 (TPH2), the rate-limiting enzyme for serotonin synthesis in the brain, while repressing TPH1 in peripheral tissues[16–18]. Consequently, vitamin D deficiency during critical developmental windows may impair neuronal growth, synaptic plasticity, and neuroprotection, all of which are central to the pathophysiology of ASD[16]. This study also identified a significant reduction in high-density lipoprotein (HDL) levels in the ASD group. This is consistent with the findings of Kim et al. (2010), who observed lower mean HDL levels in autistic subjects than in controls[19]. Tierney et al. (2021) further noted that one-fifth of ASD participants exhibited hypolipidemia and deficiencies in apolipoprotein A1 (ApoA1), the primary protein component of HDL[20]. Meta-analyses by Dhanasekara et al. (2023) and Al-Beltagi et al. (2024) have also confirmed dyslipidaemias as a common metabolic feature in ASD.ASD[21, 22]. HDL plays a vital role in reverse cholesterol transport and exerts antioxidant and neuroprotective effects on the brain. Given that cholesterol is essential for myelin integrity and synapse formation, disturbances in HDL-mediated transport may contribute to the altered neuronal connectivity and behavioral deficits characteristic of ASD[23]. Furthermore, decreased activity of the

Table 1. The demographic data of ASD children and controls(n=120)

Characteristic		Control (n=60)	ASD(n=60)	*P value*
Age (years)	**1-6**	17(14.2%)	17(14.2%)	0.88*
	7-11	33(27.5%)	31(25.8%)	
	12-20	10(8.3%)	12(10%)	
BMI (Kg\m^2)		15.3(2.5)	15.4(3.3)	*0.*57**
gender	**Male**	45(37.5%)	45(37.5%)	1.0*
	Female	15(12.5%)	15(12.5%)	
Type of birth	**Natural**	46(38.3%)	42(35%)	0.40*
	Caesarean	14(11.7%)	18(15%)	
Mother's age at baby's birth(years)	**17-26 yrs**	26(21.9 %)	28(23.5%)	0.90*
	27-36 yrs	30(25.2%)	30(25.2%)	
	>36yrs	3(2.5%)	2(1.7%)	
Father's age at baby's birth(years)	**17-26 yrs**	13(10.8%)	11(9.2%)	0.65*
	27-36 yrs	26(21.7%)	31(25.8%)	
	>36yrs	21(17.5%)	18(15%)	

Data were presented as percentages for all variables except BMI, which was expressed as median (interquartile range). Significant differences between the two groups were illustrated as $p<0.05$. The chi-square test and the Mann-Whitney U test were used to check if there were differences between the two groups.

HDL-associated enzyme paraoxonase-1 (PON1) may exacerbate oxidative stress [24]. These lipid abnormalities may be driven by genetic mutations in the APOA1 gene, chronic low-grade inflammation, or selective eating behaviors common in children with ASD.[20, 21]. Additionally, in our study, there was no significant difference in total cholesterol, triglyceride, and LDL-C levels between the ASD and control groups. This finding is consistent with Kim et al. (2010), who found no significant difference in cholesterol and LDL between children with ASD groups and control groups [19], and Parvin et al. (2024), who found no significant difference in cholesterol between the ASD and control groups[25]. However, some studies have reported findings that contradict the results of the present study, such as Alyaseen et al. (2024), where low-significant cholesterol levels were found in ASD; this difference from my study may be due to the samples being taken from non-fasting children. Regarding triglycerides, most studies contradict my study, like Parvin et al. (2024) and Kim et al. (2010), where they found a highly significant triglyceride level in ASD compared to the control group. The reason for the difference between my study and that of Parvin et al. may be that they selected a younger age group under 10 years. The difference between our study and that of Kim et al. may be due to the variation in the BMI of children with ASD compared to healthy children, which affected triglyceride levels. In contrast, the BMIs in our study were similar. In addition, no study has found results that match ours, possibly because the environment in Yemen differs from that in other countries, as well as poor nutrition. However, our study showed significantly higher triglyceride levels in patients with severe ASD. These findings suggest that abnormalities in lipid metabolism are linked to the pathophysiological mechanisms underlying symptom severity. Similar results were reported in a Brazilian study, where more intense ASD symptoms correlated with higher TG values[26], and a Korean study that identified elevated TG as a significant marker in boys with ASD [19]. Elevated triglyceride levels may reflect broader disturbances in lipid transport and cellular membrane composition, influencing neurodevelopmental trajectories [27, 28]. The relationship between vitamin D and HDL cholesterol is multifaceted. Vitamin D influences lipid homeostasis by modulating hepatic apoprotein synthesis and the hypothalamic-pituitary-thyroid (HPT) axis[22]. Conversely, because vitamin D is fat-soluble, it requires lipid transporters; thus, reduced HDL levels may impair the bioavailability and transport of vitamin D, leading to lower serum concentrations[29].Furthermore, my study investigated several risk factors, such as the type of birth and the father's and mother's age at the baby's birth;

Table 2. comparison of biochemical parameters levels in study group(n=120):

Parameter	Control(n=60)	Case(n=60)	*P* value
Vitamin D3(25-OH) ng/ml	23.5(11)	18.5(10.2)	0.01*
Total cholesterol mg/dl	159.2±27.7	159.7±20.6	0.9**
Triglycerides mg/dl	115.5(26)	116(50)	0.99*
HDL mg/dl	44(13)	40(14)	0.010*
LDL mg/dl	87(23)	88.5(36)	0.485*

Data were presented as median (interquartile range) for all variables except total cholesterol, which was expressed as mean ± SD. A P-value $\leq$ 0.05 was considered significant. **An independent t-test was conducted to determine if there is a difference in total cholesterol between cases and controls. *A Mann-Whitney U test was conducted to determine if there is a difference in vitamin D3, triglycerides, LDL, and HDL among cases and controls.

Table 3. Biochemical parameters of severity of ASD according to CARS-2 (n=60).

parameter	Mild ASD (n=20)	Moderate ASD(n=20)	Sever ASD(n=20)	*P value*
Vitamin D3(25-OH) ng/ml	18.1(14.3)	20(7.9)	17.6(12.1)	*0.6***
Total cholesterol mg/dl	167±28	158±29	153±26	*0.29**
Triglycerides mg/dl	114(60)	102(28)	141(56)	*0.01***
HDL mg/dl	41(13)	41(15)	38 (17)	*0.9***
LDL mg/dl	95(23)	89(40)	81(42)	*0.3***

Data were presented as median (interquartile range) for all variables except total cholesterol, which was expressed as mean ± SD. P-values $\leq$ 0.05 were considered significant. We conducted a one-way ANOVA test to compare cholesterol mean with severity of ASD. *Kruskal-Wallis H was performed on median VitD3, triglyceride, HDL, and LDL for the case group with severity of ASD. The statically significant difference across ASD severity groups of triglycerides (P=0.01) with higher median values was observed in the severe ASD group compared to the mild and moderate groups.

no significant difference was found in these risk factors between the ASD and control groups.

5. CONCLUSION

- The primary aim of this study was to determine whether measurable differences exist between children with ASD and healthy children in terms of vitamin D and lipid profiles.
- The results successfully achieved this objective, confirming the presence of these differences in the data. These outcomes underscore the importance of early screening and monitoring of metabolic and nutritional parameters in children.
- Early detection may support timely intervention and potentially improve clinical outcomes.

6. RECOMMENDATION

For future research. It is recommended that investigators conduct more in-depth studies to explore whether gastrointestinal dysfunction, which is potentially common among children with ASD, may impair nutrient absorption and contribute to metabolic irregularities. Understanding these underlying mechanisms could provide important insights into the biological pathways linking nutritional status, appearance, and ASD severity.

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