International Journal of Research in Medical Sciences
Parvin S et al. Int J Res Med Sci. 2024 Apr;12(4):1109-1112
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20240833

Original Research Article

Total cholesterol and triglycerides status in autistic spectrum disorder children: a case-control study on Bangladeshi children

Shahana Parvin[1]*, Shorifa Shahzadi[2], Nasir U. Ahmed[3], Mahadi A. Rouf[4], Shahriar Masood[5]

[1]Department of Physiology, Ibrahim Medical College, Dhaka, Bangladesh
[2]Department of Physiology, Bangbandhu Sheikh Mujib Medical University, Dhaka, Bangladesh
[3]Department of Medicine, Armed Forces Medical College, Dhaka, Bangladesh
[4]Department of Physiology, Ad-din Akij Medical College, Khulna, Bangladesh
[5]Department of Physiology, Jahurul Islam Medical College, Dhaka, Bangladesh

Received: 19 February 2024
Revised: 13 March 2024
Accepted: 19 March 2024

***Correspondence:**
Dr. Shahana Parvin,
E-mail: shahanakjsl@gmail.com

ABSTRACT

Background: Autism spectrum disorder (ASD) is a neurodevelopmental condition marked by challenges in social interaction, communication, and repetitive behaviors. The association between lipid profiles, particularly total cholesterol and triglycerides, and ASD in children is a growing focus in pediatric health research. This study aimed to assess the total cholesterol and triglycerides status in autistic spectrum disorder children.
Methods: This cross-sectional study was conducted in the department of physiology, Bangabandhu Sheikh Mujib Medical University, Dhaka from March 2014 to January 2015 with 100 male children, half in a healthy control group (group A) and the other half diagnosed with autism spectrum disorder (group B).
Results: In this study, no significant correlation was found between the groups for age (p=0.94) or BMI (p=0.29). The mean (±SE) serum total cholesterol levels were 146±1.70 mg/dl in group A and 145.00±3.77 mg/dl in group B, showing no significant difference between the two groups (p=0.885). But, the mean (±SE) serum triglyceride levels were 86.14±3.28 mg/dl in group A and 107.74±7.91 mg/dl in group B, with significantly higher levels compared to group A (p<0.01).
Conclusions: Although there is no significant difference in serum total cholesterol levels between healthy children and those with autistic spectrum disorder, there is a significant difference in serum triglyceride levels. Therefore, further studies are needed to provide a clearer understanding of the lipid profile comparison.

Keywords: Total cholesterol, Triglycerides, Autistic spectrum disorder, ASD, Calcium, Magnesium

INTRODUCTION

Autism is a behaviourally defined syndrome characterized by extensive deficits in social interaction, impaired verbal and nonverbal communication, and stereotyped patterns of interests and activities.[1] The American Psychiatric Association associates' autism with behavioural, developmental, neuropathological, and sensory abnormalities.[2] Typically diagnosed between 2 to 10 years of age, with peak prevalence between 5 to 8 years, autism is part of a group of common developmental disorders known as autism spectrum disorders (ASD).[3] The term "spectrum" signifies a wide range of disorders with varying severity across different domains.[2] Autism, within the ASD umbrella, involves deficits in social and behavioural interactions as well as challenges in developing normal interpersonal relationships.[4] Autism, first detailed by Kanner in 1943, is characterized by extreme loneliness and excessive shame from an early age.[5] Core symptomatic domains of autistic disorders include deficits in communication, abnormal social interaction, and restrictive or repetitive interests and

behaviors.[6] Regarded as a metabolic disorder affecting the central nervous system (CNS), autism is associated with various metabolic defects, such as mitochondrial dysfunctions, phenylketonuria, creatine deficiency, and inborn errors of cholesterol biosynthesis.[1,7] Metabolic abnormalities in ASD involve alterations in the tricarboxylic acid (TCA) cycle, energy production, ammonia detoxification, and abnormal cholesterol metabolism.[7] Abnormalities in mitochondrial fatty acid beta-oxidation have been noted in autism.[8] Some studies report that around 19% of individuals with ASD exhibit total cholesterol levels lower than 100 mg/dl, potentially linked to a deficiency of 7-dehydrocholesterol reductase (DHCR7), a crucial enzyme in the final step of cholesterol synthesis. This deficiency is attributed to reduced production rather than excessive breakdown.[8] A recent study by Kim et al compared the complete lipid profiles of children diagnosed with autism to those of healthy, well-matched controls.[9] Autism spectrum disorders involve complex interactions of nutritional and environmental factors.[10] Meguid et al observed significantly lower levels of both forms of vitamin D (25-hydroxy cholecalciferol and 1, 25-dihydroxycholecalciferol) and serum calcium in children with ASD.[11] The active form of vitamin D (1, 25-dihydroxycholecalciferol) facilitates calcium absorption. Deficiency of this vitamin in autism leads to calcium deficiency, contributing to oxidative stress as increased intracellular calcium stimulates mitochondrial oxygen radicals.[12,13] Calcium is crucial for activating essential enzymes in energy-producing pathways, including α-ketoglutarate dehydrogenase, isocitrate dehydrogenase, and pyruvate dehydrogenase.[14] Lozoff et al state that iron deficiency early in life may cause permanent damage to nervous system development and function, leading to impaired cognition, diminished learning capacity, attention deficit, and neuromotor dysfunction.[15] Moreover, besides biochemical factors iron plays a crucial role in the early stages of brain development and function.[16] The objective of this study was to assess the total cholesterol and triglycerides status in autistic spectrum disorder children.

METHODS

This cross-sectional study, conducted at the department of physiology, Bangabandhu Sheikh Mujib Medical University, Dhaka from March 2014 to January 2015, involved 100 randomly selected male children. The participants were divided into two groups: 50 in a healthy control group (group A) and 50 diagnosed with autistic spectrum disorder (group B). The study protocol received approval from the institutional review board at BSMMU, Dhaka, Bangladesh. Autistic children were selected from the parents' forum (DOHS, Mohakhali), while the control group comprised typically developing children from schools. Inclusion criteria ensured a homogeneous group of autistic male children aged 3-8 years with a confirmed diagnosis by a pediatric neurologist. Healthy controls were matched to ASD patients in age, height, weight, BMI, and sex. Rigorous exclusion criteria, including epilepsy, Turner syndrome, Down syndrome, and medication use, were applied to eliminate potential confounding factors, ensuring internal validity and the clarity of study findings. The diagnosis of ASD was made by a pediatric neurologist, ensuring the accuracy of the condition. Control subjects were meticulously chosen to match ASD patients in age, height, weight, BMI, and sex, enhancing the validity of the comparison between the two groups. A thorough physical examination was conducted, capturing anthropometric measurements for height and weight. Furthermore, venous blood samples (5 ml) were collected from the antecubital vein to analyze serum levels of magnesium and calcium, performed at the department of biochemistry at BSMMU. Statistical analysis, employing statistical package for the social sciences (SPSS) for Windows version 16.0, included independent sample 't-tests and 'Z' proportion tests as applicable, with a significance level set at p value <0.05.

RESULTS

In this study, the mean±SE age for group A and group B participants were 6.02±0.21 and 16.90±0.73 years, respectively. No significant correlation was found between the groups for age (p=0.94). Similarly, the mean ±SE BMI for group A and group B participants were 5.93±0.22 and 17.25±0.14 kg/m^2, respectively, with no significant correlation between the groups (p=0.29). In this study, the mean±SE Ca^{++} levels for group A and group B participants were 9.32±0.06 and 8.86±0.05 mg/dl, respectively, showing a significant correlation between the groups (p<0.001). Similarly, the mean±SE Mg^{++} levels for group A and group B participants were 2.13±0.02 and 1.90±0.03 mg/dl, respectively, with a significant correlation between the groups (p<0.001).

The mean (±SE) serum total cholesterol levels were 146±1.70 mg/dl in group A and 145.00±3.77 mg/dl in group B, showing no significant difference between the two groups (p=0.885). However, the mean (±SE) serum triglyceride levels were 86.14±3.28 mg/dl in group A and 107.74±7.91 mg/dl in group B, with significantly higher levels compared to group A (p<0.01).

Table 1: Age and BMI of participants.

Group A (n=50)	Group B (n=50)	P value
Mean±SE age in (year)		
6.02±0.21	16.90±0.73	0.94
Mean±SE age (kg/m^2)		
5.93±0.22	17.25±0.14	0.29

Table 2: S. calcium and magnesium levels.

Group A (n=50)	Group B (n=50)	P value
Mean±SD Ca^{++} (mg/dl)		
9.32±0.06	9.32±0.06	9.32±.06
Mean±SD Mg^{++} (mg/dl)		
2.13±0.02	2.13±0.02	2.13±0.02

Table 3: T. cholesterol triglyceride status.

Group A (n=50)	Group B (n=50)	P value
Mean±SD total cholesterol (mg/dl)		
146.16±1.70	145.00±4.34	0.885
Mean±SD triglyceride (mg/dl)		
86.14±3.28	107.74±7.91	0.007

DISCUSSION

This study aimed to assess the total cholesterol and triglycerides status in autistic spectrum disorder children. In this study, the mean values of all biochemical variables in normal children fell within physiological limits, aligning closely with findings reported by various researchers.[17,18] Both the control and case groups were comparable, with no significant differences in confounding variables such as age, height, weight, and BMI between the two groups. The mean values of magnesium and calcium were below the lower limit of the normal range. In the present study, serum total cholesterol was not significantly higher in the study group compared to the control group. Similar findings were observed in other studies as well.[9,19] However, in some studies, serum total cholesterol was reported to be significantly higher in autistic children, while in another study, it was lower.[17,20,21] Furthermore, the serum total cholesterol level was found to be high in only 6% of autistic children (study group) and 0% in apparently healthy children (control group), which was not statistically significant. There were no similar data available for comparison. In our study, serum triglyceride levels were significantly higher in the study group than in the control group. Similar findings were observed in other studies as well.[9,17] Additionally, serum triglyceride levels were found to be abnormally high in 38% of children in the study group and in 6% of children in the control group, which was statistically significant. Similarly, elevated serum triglyceride levels were also reported by Kim et al in 11% of autistic children.[9] In this present study, serum magnesium was significantly lower in the study group than in the control group. Almost similar findings were observed by Strambi et al.[18] Additionally, the serum magnesium level was found to be abnormally low in 52% of children in the study group and in 4% of children in the control group, which was statistically significant. Similarly, Kozielec and Hermelin observed that 33.6% of autistic children had magnesium deficiency. In this present study, serum calcium was significantly lower in the study group than in the control group. Almost similar findings were observed by Ansary et al and Sun et al.[13,22]

Additionally, the serum calcium level was found to be abnormally low in 74% of children in the study group and in 6% of children in the control group, which was statistically significant. Similarly, Yasuda et al observed that 5.8% of autistic children had calcium deficiency.[23] All the findings of this current study may be helpful in further similar studies.

Limitations

This study was constrained by its single-center design and relatively small sample size. Furthermore, the study duration was short. As a result, the findings may not precisely capture the broader scenario across the entire country.

CONCLUSION

While the research indicates a lack of noteworthy distinctions in serum total cholesterol levels between typically developing children and those diagnosed with autistic spectrum disorder, a notable dissimilarity in serum triglyceride levels has been observed. This underscores the importance of delving deeper into the lipid profiles of individuals with autism, seeking a comprehensive comprehension of the nuanced metabolic aspects associated with the disorder. The identified divergence in triglyceride levels prompts a call for additional investigations, aiming to unravel the intricacies of lipid metabolism in the context of autism. Such endeavors hold the potential to advance our knowledge and contribute to tailored interventions for individuals on the autistic spectrum.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Manzi B, Loizzo AL, Giana G, Curatolo P. Autism and metabolic diseases. J Neurol. 2008;23(3):307-14.
2. American Psychiatric Association 1994. Diagnostic and Statistical Manual of mental disorders, 4th edition. Available at: https://ajp.psychiatry online.org/doi/10.1176/ajp.152.8.1228. Accessed on 02 January 2024.
3. Yeargin-Allsopp M, Rice C, Karapurkar T, Doernberg N, Boyle C, Murphy C. Prevalence of autism in a US metropolitan area. JAMA. 2003;289(1):49-55.
4. Aneja A, Tierney E. Autism: the role of cholesterol in treatment. Int Rev Psychiatry. 2008;20(2):165-7.
5. DiCicco-Bloom E, Lord C, Zwaigenbaum L, Courchesne E, Dager SR, Schmitz C, et al. The developmental neurobiology of autism spectrum disorder. J Neurosci. 2006 Jun 28;26(26):6897-906.
6. Pastural E, Ritchie S, Lu Y, Jin W, Kavianpour A, Khine Su-Myat K, et al. Novel plasma phospholipid biomarkers of autism: mitochondrial dysfunction as a putative causative mechanism. Prostaglandins Leukot Essent Fatty Acids. 2009;81(4):253-64.
7. Clark-Taylor T, Clark-Taylor BE. Is autism a disorder of fatty acid metabolism? Possible dysfunction of mitochondrial beta-oxidation by long chain acyl-CoA dehydrogenase. Med Hypotheses. 2004;62(6):970-5.

8. Tierney E, Bukelis I, Thompson RE, Ahmed K, Aneja A, Kratz L, et al. Abnormalities of cholesterol metabolism in autism spectrum disorders. Am J Med Genet B Neuropsychiatr Genet. 2006;141B(6):666-8.
9. Kim EK, Neggers YH, Shin CS, Kim E, Kim EM. Alterations in lipid profile of autistic boys: a case control study. Nutr Res. 2010;30(4):255-60.
10. Curtis LT, Patel K. Nutritional and environmental approaches to preventing and treating autism and attention deficit hyperactivity disorder (ADHD): a review. J Altern Complement Med. 2008;14(1):79-85.
11. Meguid NA, Hashish AF, Anwar M, Sidhom G. Reduced serum levels of 25-hydroxy and 1,25-dihydroxy vitamin D in Egyptian children with autism. J Altern Complement Med. 2010;16(6):641-5.
12. El-Ansary AK, Ben Bacha AG, Al-Ayadhi LY. Proinflammatory and proapoptotic markers in relation to mono and di-cations in plasma of autistic patients from Saudi Arabia. J Neuroinflammation. 2011;8:142.
13. El-Ansary A, Al-Daihan S, Al-Dbass A, Al-Ayadhi L. Measurement of selected ions related to oxidative stress and energy metabolism in Saudi autistic children. Clin Biochem. 2010;43(1-2):63-70.
14. Castaldo P, Cataldi M, Magi S, Lariccia V, Arcangeli S, Amoroso S. Role of the mitochondrial sodium/calcium exchanger in neuronal physiology and in the pathogenesis of neurological diseases. Prog Neurobiol. 2009;87(1):58-79.
15. Lozoff B, Jimenez E, Wolf AW. Long-term developmental outcome of infants with iron deficiency. N Engl J Med. 1991;325(10):687-94.
16. Oski FA. Iron deficiency in infancy and childhood. N Engl J Med. 1993;329(3):190-3.
17. Dziobek I, Gold SM, Wolf OT, Convit A. Hypercholesterolemia in Asperger syndrome: independence from lifestyle, obsessive-compulsive behavior, and social anxiety. Psychiatry Res. 2007;149(1-3):321-4.
18. Strambi M, Longini M, Hayek J, Berni S, Macucci F, Scalacci E, et al. Magnesium profile in autism. Biol Trace Elem Res. 2006;109(2):97-104.
19. Wiest MM, German JB, Harvey DJ, Watkins SM, Hertz-Picciotto I. Plasma fatty acid profiles in autism: a case-control study. Prostaglandins Leukot Essent Fatty Acids. 2009;80(4):221-7.
20. Kurup RK, Kurup PA. A hypothalamic digoxin-mediated model for autism. Int J Neurosci. 2003;113(11):1537-59.
21. Sikora DM, Pettit-Kekel K, Penfield J, Merkens LS, Steiner RD. The near universal presence of autism spectrum disorders in children with Smith-Lemli-Opitz syndrome. Am J Med Genet A. 2006;140(14):1511-8.
22. Sun C, Xia W, Zhao Y, Li N, Zhao D, Wu L. Nutritional status survey of children with autism and typically developing children aged 4-6 years in Heilongjiang Province, China. J Nutr Sci. 2013;2:e16.
23. Yasuda H, Yasuda Y, Tsutsui T. Estimation of autistic children by metallomics analysis. Sci Rep. 2013;3:1199.

Cite this article as: Parvin S, Shahzadi S, Ahmed NU, Rouf MA, Masood S. Total cholesterol and triglycerides status in autistic spectrum disorder children: a case-control study on Bangladeshi children. Int J Res Med Sci 2024;12:1109-12.