

Serum creatinine, urea and dietary protein intake in Bangladeshi children with autism spectrum disorder

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Abstract

Background and objective: Autism spectrum disorder (ASD) frequently co-occurs with mitochondrial dysfunction and with restrictive, selective eating. Both may raise serum creatinine. We assessed serum creatinine and urea in Bangladeshi children with ASD. Dietary protein intake was examined as a possible driver of any difference.

Materials and methods: This cross-sectional study ran from July 2019 to June 2020 at Dhaka Medical College. We enrolled 30 ASD children (Group A) and 30 matched healthy controls (Group B), aged 5–18 years. Serum creatinine, urea and blood glucose were measured on a Dimension EXL-200 analyzer. Dietary protein intake was estimated from parent-interview food frequency data using the Food Composition Table for Bangladesh. SPSS (Statistical Package for the Social Sciences) version 20.0 was used for analysis; $p < 0.05$ was considered statistically significant.

Results: Mean serum creatinine was significantly higher in Group A (0.74 ± 0.22 vs. 0.51 ± 0.13 mg/dl; $p < 0.001$). This exceeded the pediatric reference range. Serum urea ($p = 0.180$) and blood glucose ($p = 0.657$) did not differ. ASD children consumed fish and eggs more frequently ($p = 0.032$ and $p = 0.041$). Mean protein intake was 58.4 ± 12.3 g/day. This exceeded the recommended safe allowance per kilogram. Dietary protein correlated strongly with serum creatinine in Group A ($r = 0.70$; $p < 0.001$; $R^2 = 0.49$). Socioeconomic profiles were comparable ($p = 0.287$).

Conclusion: Serum creatinine was significantly higher in Bangladeshi ASD children than in matched controls and exceeded the pediatric reference range, whereas serum urea and blood glucose were unchanged. The creatinine elevation was strongly correlated with a higher dietary protein intake from preferred fish and egg consumption. Routine serum creatinine monitoring and dietary counseling should be integrated into the standard clinical management of children with ASD.

Introduction

Autism spectrum disorder (ASD) is a neurodevelopmental condition defined by a

distinct mix of features. These include impaired social communication, repetitive behaviors, highly restricted interests and unusual sensory responses,

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all beginning early in life. The clinical presentation is markedly heterogeneous across the spectrum. Its global prevalence is close to 1%. Rates are generally higher in high-income countries [1]. Up to 40 percent of affected individuals carry identifiable genetic or metabolic disturbances [2].

Metabolic comorbidities like disruptions in cellular energy production, gastrointestinal physiology and micronutrient homeostasis have been documented in children with ASD [3]. Of these biological findings, mitochondrial dysfunction is the best supported and most often replicated [4,5]. In one case-control study, immune cells from ASD children showed much lower mitochondrial complex I activity. The activity was about two-thirds below that of typically developing children [6].

Cellular energy production normally follows a sequential chain. Glucose is first broken down to pyruvate through glycolysis in the cytosol. Pyruvate is then converted to acetyl-CoA and enters the tricarboxylic acid (TCA) cycle in the mitochondrial matrix. The TCA cycle produces the reduced electron carriers NADH and FADH₂. These donate electrons to the electron transport chain (ETC) in the inner mitochondrial membrane. This process drives oxidative phosphorylation and produces ATP [5,7]. In ASD, impairment of ETC complexes I, III and IV reduces the efficiency of this primary ATP-generating pathway [5,6]. To cope with this energy shortfall, cells lean on the creatine kinase/phosphocreatine (CK/PCr) shuttle as a buffer [8]. In this shuttle, creatine kinase in the mitochondrial intermembrane space uses ATP to convert creatine to phosphocreatine. Cytosolic creatine kinase then regenerates ATP at sites of energy consumption [8,9]. Increased reliance on this shuttle accelerates the non-enzymatic, irreversible dehydration of creatine and phosphocreatine to creatinine [10,11]. Creatinine is the final end product of this pathway. It cannot be reconverted and is excreted by the kidneys [11]. Sustained activation of this pathway therefore increases creatinine production and can raise the serum creatinine concentration [10,11].

Children with ASD are more than five times as likely to have significant feeding problems as typically developing children [12]. This restrictive pattern often narrows food choices to a small group of preferred items [13,14]. Fish and eggs

are typically among the most preferred foods, while most vegetables are avoided [14]. In Bangladesh, almost the whole population eats fish. It provides much of the daily animal protein [15]. This selective preference is therefore likely to raise habitual animal-protein and creatine intake. Both fish and eggs are concentrated dietary sources of creatine [16].

Amino acids from digested protein, particularly arginine and glycine, serve as precursors for endogenous creatine biosynthesis via the L-arginine:glycine amidinotransferase (AGAT) and Guanidinoacetate N-methyltransferase (GAMT) enzymatic pathway in the liver and kidney [9]. Creatine-rich foods also directly expand the intracellular creatine pool in muscle, further increasing creatinine output [16]. When dietary intake of animal protein is high, the muscle creatine pool expands and creatinine efflux rises independently of any change in glomerular filtration [16,17]. But urea comes mainly from the urea cycle in the liver. This pathway converts ammonia from amino acid catabolism to urea through enzymatic steps located partly in the mitochondrial matrix and partly in the cytosol [7,11]. Serum urea, being a product of hepatic nitrogen metabolism, is therefore a far less specific reflection of muscle creatine turnover than creatinine [11]. Systemic glucose homeostasis, when intact, helps exclude diabetic nephropathy as a confounding factor in evaluating creatinine changes.

Few studies have examined serum creatinine, urea and glucose levels in children with ASD. Earlier case-control studies found no notable change in serum creatinine or urea in this group [18,19]. Findings on blood glucose have been inconsistent, with some reports suggesting lower fasting levels in ASD children compared with controls. None of these studies, however, assessed diet at the same time. They also did not evaluate populations with high habitual fish and animal protein consumption. The mechanism by which dietary protein selectivity and mitochondrial dysfunction together affect renal metabolic markers in ASD is therefore unclear.

We measured serum creatinine, serum urea and random blood glucose in Bangladeshi children with ASD and matched healthy controls. Dietary protein

intake and socioeconomic characteristics were also quantified. This study asked whether ASD is linked to altered renal metabolic markers. It also examined how far dietary protein selectivity explains any such change.

Materials and methods

This study followed a cross-sectional analytical design. Data were collected over one year, from July 2019 to June 2020. It was carried out at the Department of Biochemistry, Dhaka Medical College, Dhaka, Bangladesh.

Sixty children between 5 and 18 years of age were recruited using a purposive sampling technique. Group A comprised 30 children with a confirmed diagnosis of ASD. All fulfilled DSM-5 criteria (Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition) [20]. They were recruited from the outpatient department of NIMH, Dhaka. Group B consisted of 30 apparently healthy children. They were matched for age and body mass index (BMI), with a comparable gender distribution. They were recruited from residential localities in Dhaka city. Children with epilepsy, obsessive-compulsive disorder, attention deficit hyperactivity disorder (ADHD) or any other current psychiatric diagnosis were excluded from both groups. The parent or legal guardian of each child provided written informed consent before any study procedure was undertaken.

Five milliliters of venous blood were obtained under aseptic conditions by a trained phlebotomist. The sample was transferred into a dry red-capped tube. It was allowed to clot at ambient temperature for 20 minutes. Serum was obtained after centrifuging the clotted sample at 3,000 rpm over a 10-minute period. Blood glucose at the time of sampling was quantified by the enzymatic hexokinase assay. Kinetic Jaffe method was applied in measurement of serum creatinine. Urea concentration in serum was determined using the enzymatic urease assay. Biochemical analyses were performed on a Dimension EXL-200 automated analyzer housed in the Clinical Pathology department at Dhaka Medical College and Hospital.

Supplementary information was obtained by interviewing the parent or guardian of each

participant. The interview used a pre-designed structured format. Questions addressed weekly food consumption patterns, estimated daily protein intake, physical activity habits and household economic status. Daily protein intake was calculated using the Bangladesh Food Composition Table [21]. Protein content per 100 g of each food item was multiplied by the portion size reported for that item. This figure was adjusted for how often the food was eaten each week. Contributions from all food items were then added together. The final intake was expressed per kilogram of body weight and compared against the safe protein intake level (0.84–1.14 g/kg/day for children aged 5–18 years) [22]. Household income was categorized using the Bangladesh Bureau of Statistics HIES 2016 classification [23].

For normally distributed continuous variables, results are presented as mean $\pm$ SD. Between-group differences were evaluated with the unpaired Student's t-test. For categorical data, the Chi-square test was applied; Fisher's exact test was substituted when any expected cell count fell below five. Within Group A, Pearson's product-moment correlation coefficient (r) was calculated to evaluate the linear association between estimated daily protein intake and serum creatinine. The coefficient of determination (R^2) was derived to quantify the proportion of creatinine variance attributable to protein intake. All analyses were performed using SPSS version 20.0. Statistical significance was defined as a two-tailed p-value of less than 0.05, corresponding to a 95% confidence level.

Results

Sixty children were enrolled. Group A had 30 ASD children. Group B had 30 healthy matched controls. Table-1 presents the baseline demographic and socioeconomic characteristics of the study subjects. The two groups were well matched for age, BMI and gender distribution. Household income and maternal education level were also comparable between the groups ($p=0.287$ and $p=0.812$ respectively), confirming that any observed dietary differences were not attributable to socioeconomic disparities.

Table-1: Baseline demographic and socioeconomic characteristics of the study subjects (N=60)

Variable	Group A – ASD (n=30)	Group B – Controls (n=30)	p-value
Age (years), Mean $\pm$ SD	7.53 $\pm$ 3.01	7.50 $\pm$ 2.94	0.966
BMI (kg/m ²), Mean $\pm$ SD	16.63 $\pm$ 2.11	16.43 $\pm$ 1.98	0.712
Male, n (%)	25 (83.3)	20 (66.7)	0.243
Female, n (%)	5 (16.7)	10 (33.3)	
Household income, n (%)			0.287
Low (<15,000 BDT)	4 (13.3)	8 (26.7)	
Middle (15,000–50,000 BDT)	18 (60.0)	17 (56.7)	
High (>50,000 BDT)	8 (26.7)	5 (16.7)	
Maternal education, n (%)			0.812
Primary or below	6 (20.0)	5 (16.7)	
Secondary to higher secondary	16 (53.3)	17 (56.7)	
Graduate and above	8 (26.7)	8 (26.7)	

Unpaired t-test for continuous variables; Chi-square test for categorical variables. Income classification based on Bangladesh Bureau of Statistics HIES 2016 categories [23]. $p < 0.05$ = statistically significant.

Table-2 shows the serum biochemical parameters in both groups. Mean serum creatinine was significantly elevated in Group A (0.74 ± 0.22 vs. 0.51 ± 0.13 mg/dl; $p < 0.001$). Values in Group A exceeded the pediatric reference upper limit of

0.70 mg/dl. Group B values fell entirely within the normal range. Neither serum urea nor random blood glucose differed significantly between the groups.

Table-2: Serum biochemical parameters in Group A (ASD) and Group B (Controls) (N=60)

Parameter	Group A (ASD) Mean $\pm$ SD (Range)	Group B (Control) Mean $\pm$ SD (Range)	p-value
Random blood glucose (mmol/L)	5.71 $\pm$ 0.66 (4.00–6.70)	5.61 $\pm$ 0.98 (3.50–7.10)	0.657
Serum creatinine (mg/dl)	0.74 $\pm$ 0.22 (0.47–1.34)	0.51 $\pm$ 0.13 (0.20–0.70)	<0.001*
Serum urea (mg/dl)	16.77 $\pm$ 5.35 (10.00–27.00)	18.67 $\pm$ 5.48 (12.00–33.00)	0.180

Unpaired t-test. $p < 0.05$ = statistically significant. Reference ranges: random glucose 3.3–7.8 mmol/L; creatinine (child) 0.20–0.70 mg/dl; urea 10–35 mg/dl [24].

Table-3 shows the frequency of dietary protein-rich food consumption. ASD children consumed fish (73.3% vs. 46.7%; $p = 0.032$) and eggs (66.7% vs. 40.0%; $p = 0.041$) more frequently than controls.

Vegetable consumption was markedly lower in Group A (33.3% vs. 80.0%; $p = 0.001$). No significant differences were found for lentils, meat or dairy products.

Table-3: Frequency of dietary protein-rich food consumption in Group A (ASD) and Group B (Controls) (N=60)

Food item (frequency)	Group A (ASD) n (%)	Group B (Control) n (%)	p-value
Fish (≥5 days/week)	22 (73.3)	14 (46.7)	0.032*
Egg (≥5 days/week)	20 (66.7)	12 (40.0)	0.041*
Lentil/Dal (≥5 days/week)	24 (80.0)	22 (73.3)	0.548
Meat/Poultry (≥3 days/week)	18 (60.0)	16 (53.3)	0.606
Dairy/Milk (≥5 days/week)	16 (53.3)	18 (60.0)	0.606
Vegetables (≥5 days/week)	10 (33.3)	24 (80.0)	0.001*

Chi-square test or Fisher's exact test. $p < 0.05$ = statistically significant. Frequency data collected by structured parent interview using weekly food-frequency recall.

Table-4 shows the estimated daily protein intake in the two groups. ASD children had significantly higher estimated daily protein intake than controls (58.4 ± 12.3 vs. 44.7 ± 9.8 g/day; $p < 0.001$). Per-kilogram intake was 2.8 ± 0.6 g/kg/day, approximately double the upper limit of the recommended safe range.

Table-4: Estimated daily protein intake in Group A (ASD) and Group B (Controls) (N=60)

Parameter	Group A (ASD) Mean ± SD	Group B (Control) Mean ± SD	p-value
Estimated daily protein intake (g/day)	58.4 ± 12.3	44.7 ± 9.8	<0.001*
Protein intake per kg body weight (g/kg/day)	2.8 ± 0.6	2.1 ± 0.5	0.002*

Unpaired t-test. $p < 0.05$ = statistically significant. Recommended safe protein intake for children aged 5–18 years: 0.84–1.14 g/kg/day [22].

Table-5 presents the Pearson correlation analysis performed within the ASD group (Group A) between estimated daily protein intake and serum creatinine. A strong, statistically significant positive linear correlation was observed ($r = 0.70$; $p < 0.001$), and the coefficient of determination ($R^2 = 0.49$) indicated that dietary protein intake alone accounted for approximately 49% of the variance in serum creatinine among these children.

Table-5: Correlation between estimated daily protein intake and serum creatinine within Group A (ASD) (n=30)

Correlated variables	Pearson r	R ²	p-value
Daily protein intake vs. serum creatinine	0.70	0.49	<0.001*

Pearson's product-moment correlation within Group A. $p < 0.05$ = statistically significant. $R^2 = 0.49$ indicates that dietary protein intake accounts for 49% of the variance in serum creatinine among ASD children.

Discussion

In this cohort, ASD children showed clearly raised serum creatinine compared with matched healthy

controls. The levels exceeded the pediatric upper reference limit of 0.70 mg/dl. Serum urea and random blood glucose did not differ between the

two groups. This selective rise in creatinine is attributable mainly to dietary protein selectivity.

Mitochondrial dysfunction has been proposed in the literature as a contributor to altered creatine metabolism in ASD. A systematic review and meta-analysis reported biomarkers of mitochondrial dysfunction, including elevated creatine kinase, in 30 to 50% of children with ASD [5,10]. However, mitochondrial function was not assessed in the present study. This pathway is therefore mentioned only as a hypothetical mechanism and cannot be inferred from the present data.

Dietary protein selectivity is a contributing mechanism supported by the present data. ASD children in this study consumed fish and eggs significantly more often than controls [13,14]. This dietary pathway is supported by the strong positive correlation between daily protein intake and serum creatinine observed in Group A, consistent with experimental evidence that a single standardized protein-rich meal measurably raises serum creatinine in humans within hours [17].

These findings differ from two earlier studies in Egyptian children with ASD, which reported no significant change in serum creatinine or urea [18,19]. Lower habitual fish consumption in those populations may explain this discrepancy. These studies also did not include a concurrent dietary assessment, which further limits direct comparison.

Clothier and Absoud (2021) separately highlighted the overlap between ASD and renal pathology in children and called for closer clinical attention to kidney function in this population [25]. The present findings show that serum creatinine elevation can occur in ASD children without overt renal disease. Dietary protein selectivity alone accounted for close to half of the variance in serum creatinine, making it a modifiable target for structured dietary counseling.

Random blood glucose did not differ significantly between Group A and Group B, and mean values in both groups fell within the normal reference range. This contrasts with Al-Bazzaz et al. (2020), who reported significantly lower fasting blood glucose in autistic patients compared with controls [26]. In that study, the low fasting glucose was attributed mainly to poor intestinal glucose uptake. The authors linked this to the gut microbial changes

and feeding difficulties commonly seen in ASD. The present study measured random rather than fasting blood glucose, and the habitually carbohydrate-rich Bangladeshi diet is likely to keep blood glucose within the normal range. Because hyperglycemia was absent in this cohort, diabetic nephropathy can be excluded as an explanation for the elevated creatinine seen in Group A.

Household socioeconomic status was comparable between the two groups, indicating that the higher fish and egg intake among ASD children was not driven by household income or purchasing power. Zimmer et al. (2012) showed that food variety in ASD is shaped by the child's sensory and behavioral characteristics rather than family resources [27]. The food choices observed in this study are consistent with sensory sensitivity and behavioral rigidity rather than socioeconomic factors.

Serum urea did not differ significantly between the groups, consistent with Fahmy (2016) and ElBaz et al. (2014) [18,19]. Urea, by contrast, responds only weakly and non-specifically to muscle creatine breakdown [11], which likely explains why it did not track the same dietary influence in this cohort.

The small single-center sample is a key limitation. Reliance on parent-reported dietary recall is also a limitation. A larger multi-center study with objective dietary and physical activity measurement is warranted.

Conclusion

In summary, serum creatinine was significantly elevated in Bangladeshi ASD children, while serum urea and blood glucose remained within normal limits. High dietary protein intake from preferred fish and egg consumption was the main contributing factor identified in this study. Routine serum creatinine monitoring and dietary counseling should be incorporated into standard clinical management of children with ASD.

Ethical statement

Ethical clearance was obtained from the Ethical Review Committee, Dhaka Medical College [Memo No. ERC-DMC/ECC/2020/84]. Permission for blood sample collection from NIMH was granted by the

Director-cum-Professor, NIMH [Memo No. NIMH/2020/342]. Informed written consent for participation and publication of anonymized data was obtained from the parent or legal guardian of each participant.

Conflict of interest

The authors declare no competing financial or non-financial interests in relation to this work.

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References

1. Lord C, Brugha TS, Charman T, Cusack J, Dumas G, Frazier T, et al. Autism spectrum disorder. *Nat Rev Dis Primers*. 2020; **6**(1): 5. doi: 10.1038/s41572-026-00706-w.
2. Genovese A, Butler MG. Clinical assessment, genetics, and treatment approaches in autism spectrum disorder (ASD). *Int J Mol Sci*. 2020; **21**(13): 4726. doi: 10.3390/ijms21134726.
3. Kohane IS, McMurphy A, Weber G, MacFadden D, Rappaport L, Kunkel L, et al. The comorbidity burden of children and young adults with autism spectrum disorders. *PLoS One*. 2012; **7**(4): e33224. doi: 10.1371/journal.pone.0033224.
4. Cheng N, Rho J, Masino S. Metabolic dysfunction underlying autism spectrum disorder and potential treatment approaches. *Front Mol Neurosci*. 2017; **10**: 34. doi: 10.3389/fnmol.2017.00034.
5. Rossignol DA, Frye RE. Mitochondrial dysfunction in autism spectrum disorders: a systematic review and meta-analysis. *Mol Psychiatry*. 2012; **17**(3): 290-314. doi: 10.1038/mp.2010.136.
6. Giulivi C, Zhang YF, Omanska-Klusek A, Ross-Inta C, Wong S, Hertz-Picciotto I, et al. Mitochondrial dysfunction in autism. *JAMA*. 2010; **304**(21): 2389-2396. doi: 10.1001/jama.2010.1706.
7. Frye RE, Rossignol DA. Mitochondrial dysfunction can connect the diverse medical symptoms associated with autism spectrum disorders. *Pediatr Res*. 2011; **69**(5 Pt 2): 41R-47R. doi: 10.1203/PDR.0b013e318212f16b.
8. Wallimann T, Tokarska-Schlattner M, Schlattner U. The creatine kinase system and pleiotropic effects of creatine. *Amino Acids*. 2011; **40**(5): 1271-1296. doi: 10.1007/s00726-011-0877-3.
9. Wyss M, Kaddurah-Daouk R. Creatine and creatinine metabolism. *Physiol Rev*. 2000; **80**(3): 1107-1213. doi: 10.1152/physrev.2000.80.3.1107.
10. Frye RE. Biomarkers of abnormal energy metabolism in children with autism spectrum disorder. *N Am J Med Sci*. 2012; **5**(3): 141-147.
11. Patel SS, Molnar MZ, Tayek JA, Ix JH, Noori N, Benner D, et al. Serum creatinine as a marker of muscle mass in chronic kidney disease: results of a cross-sectional study and review of literature. *J Cachexia Sarcopenia Muscle*. 2013; **4**(1): 19-29. doi: 10.1007/s13539-012-0079-1.
12. Sharp WG, Berry RC, McCracken C, Nuhu NN, Marvel E, Saulnier CA, et al. Feeding problems and nutrient intake in children with autism spectrum disorders: a meta-analysis and comprehensive review of the literature. *J Autism Dev Disord*. 2013; **43**(9): 2159-2173. doi: 10.1007/s10803-013-1771-5.
13. Bandini LG, Anderson SE, Curtin C, Cermak S, Evans EW, Scampini R, et al. Food selectivity in children with autism spectrum disorders and typically developing children. *J Pediatr*. 2010; **157**(2): 259-264. doi: 10.1016/j.jpeds.2010.02.013.
14. Cermak SA, Curtin C, Bandini LG. Food selectivity and sensory sensitivity in children with autism spectrum disorders. *J Am Diet*

- Assoc. 2010; **110**(2): 238-246.
doi: 10.1016/j.jada.2009.10.032.
15. Bogard JR, Farook S, Marks GC, Waid J, Belton B, Ali M, et al. Higher fish but lower micronutrient intakes: temporal changes in fish consumption from capture fisheries and aquaculture in Bangladesh. *PLoS One*. 2017; **12**(4): e0175098.
doi: 10.1371/journal.pone.0175098.
 16. Brosnan ME, Brosnan JT. The role of dietary creatine. *Amino Acids*. 2016; **48**(8): 1785-1791.
doi: 10.1007/s00726-016-2188-1.
 17. Nair S, O'Brien SV, Hayden K, Pandya B, Lisboa PJ, Hardy KJ, et al. Effect of a cooked meat meal on serum creatinine and estimated glomerular filtration rate in diabetes-related kidney disease. *Diabetes Care*. 2014; **37**(2): 483-487. doi: 10.2337/dc13-1770.
 18. Fahmy SF, Sabri NA, El Hamamsy MH, El Sawi M, Zaki OK. Vitamin D intake and sun exposure in autistic children. *Int J Pharm Sci Res*. 2016; **7**(3): 1043-1049.
doi: 10.13040/IJPSR.0975-8232.7(3).1043-49.
 19. ElBaz F, Zaki M, Youssef A, ElDorri G, Elalfy D. Study of plasma amino acid levels in children with autism: an Egyptian sample. *Egypt J Med Hum Genet*. 2014; **15**(2): 181-186.
doi:10.1016/j.ejmhg.2014.02.002.
 20. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders (DSM-5). 5th ed. Arlington, VA: American Psychiatric Publishing; 2013.
doi: 10.1176/appi.books.9780890425596.
 21. Shaheen N, Rahim ATMA, Mohiduzzaman M, Banu CP, Bari ML, Tukun AB, et al. Food Composition Table for Bangladesh. Dhaka: Institute of Nutrition and Food Science, Centre for Advanced Research in Sciences, University of Dhaka; 2013.
 22. Joint WHO/FAO/UNU Expert Consultation. Protein and amino acid requirements in human nutrition. *World Health Organ Tech Rep Ser*. 2007; (935): 1-265.
 23. Bangladesh Bureau of Statistics. Household Income and Expenditure Survey 2016. Dhaka: Statistics and Informatics Division, Ministry of Planning, Government of Bangladesh. 2017; p. 204.
 24. Burtis CA, Ashwood ER, Bruns DE. Textbook of Clinical Chemistry and Molecular Diagnostics. 4th ed. New York: Elsevier Saunders; 2006. p. 604-607.
 25. Clothier J, Absoud M. Autism spectrum disorder and kidney disease. *Pediatr Nephrol*. 2021; **36**(10): 2987-2995.
doi: 10.1007/s00467-020-04875-y.
 26. Al-Bazzaz A, A-Dahir K, Almashhadani A, Al-Ani I. Estimation of fasting serum levels of glucose, zinc, copper, zinc/copper ratio and their relation to the measured lipid profile in autistic patients and non-autistic controls in Jordan. *Biomed Pharmacol J*. 2020; **13**(1): 481-488.
doi: 10.13005/bpj/1909.
 27. Zimmer MH, Hart LC, Manning-Courtney P, Murray DS, Bing NM, Summer S. Food variety as a predictor of nutritional status among children with autism. *J Autism Dev Disord*. 2012; **42**(4): 549-556.
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