RESEARCH ARTICLE

pISSN: 0126-074X | eISSN: 2338-6223
https://doi.org/10.15395/mkb.v57.4406
Majalah Kedokteran Bandung. 2025;57(4):328–335

Majalah Kedokteran Bandung (MKB)

Received: May 16, 2025
Accepted: November 18, 2025
Available online: December 31, 2025

Left Ventricular Function in Steroid-Resistant Nephrotic Syndrome at Dr. Hasan Sadikin Hospital

Aghnia Ghassani Kusumaputri,[1] Sri Endah Rahayuningsih,[2] Rini Rossanti,[2] Putria Rayani Apandi,[2] Ahmedz Widiasta[2]

[1]Faculty of Medicine Universitas Padjadjaran, Indonesia
[2]Department of Child Health Faculty of Medicine Universitas Padjadjaran
Dr. Hasan Sadikin General Hospital Bandung, Indonesia

Abstract

Steroid-resistant nephrotic syndrome (SRNS) is a nephrotic syndrome (NS) which does not respond to high-dose corticosteroid therapy after 4 to 8 weeks. SRNS occurs in approximately 10–20% of childhood idiopathic nephrotic syndrome. SRNS patients are at risk of developing cardiovascular complications due to the long duration of the disease. This retrospective descriptive study investigated left ventricular function in pediatric patients with steroid-resistant nephrotic syndrome (SRNS) at Dr. Hasan Sadikin General Hospital, Bandung, Indonesia, from 2018 to 2022. A total of 42 patients aged 1 month to 18 years who underwent transthoracic echocardiography (TTE) were included. Left ventricular function was assessed using ejection fraction (EF) and fractional shortening (FS). Most patients (61.9%) had normal EF and FS values, while 38.1% showed decreased systolic function. The mean EF and FS were 55.8% and 30.2%, respectively, with median values of 59.3% and 31.2%. An important finding in this study is that some children with SRNS exhibited decreased EF and FS values despite having no other identifiable risk factors for ventricular dysfunction, such as congenital heart disease (CHD), rheumatic heart disease (RHD), chronic kidney disease (CKD), or hypertension. This finding sugest that ventricular dysfunction may still occur independently in some cases of SRNS.

Keywords: Childhood idiopathic nephrotic syndrome, echocardiography, left ventricular function, steroid-resistant nephrotic syndrome

Introduction

Nephrotic syndrome is characterized by massive proteinuria, which leads to hypoalbuminemia, edema, and hyperlipidemia. Nephrotic syndrome that does not respond after four to eight weeks of high-dose corticosteroid therapy is classified as steroid-resistant nephrotic syndrome (SRNS).[1] SRNS occurs in approximately 10–20% of children with idiopathic nephrotic syndrome.[2] SRNS are relatively vulnerable to complication, due to prolonged hyperlipidemia, persistent proteinuria, and hypoalbuminemia that fail to achieve remission. These conditions may lead to to cardiovascular complication. Major cardiovascular complications such as left ventricular dysfunction and remodeling may develop. Through the SMAD–TGF-β signaling pathway, SRNS may contribute to progressive stiffening of the blood vessels and myocardium, with these alterations typically developing gradually over a span of two to five years. [3,4]

Focal segmental glomerulosclerosis (FSGS) is the most common histological finding in SRNS, with various associated genetic mutations. These mutations include alterations in genes encoding nephrin, podocin, and CD2-associated protein (CD2AP), which are essential components of the podocyte slit diaphragm, as well as ACTN4, a key structural protein of the podocyte cytoskeleton.[5,6] Mutations in these genes can damage the filtration membrane, resulting in persistent proteinuria. Such damage, along with ongoing proteinuria, contributes to complications including dyslipidemia, thrombosis, hypertension, and cardiovascular disease.[4,5]

Thrombocytosis and hypertension are additional complications observed in children with SRNS. Thrombosis, in particular, has been linked to an increased risk of cardiovascular events.[1,7] Hypertension is a condition that may

Corresponding Author:
Sri Endah Rahayuningsih.
Department of Child Health Faculty of Medicine Universitas Padjadjaran/Dr. Hasan Sadikin General Hospital Bandung, Indonesia
Email: sri.endah@unpad.ac.id

occur in children with nephrotic syndrome, including children with SRNS. Both renal and extrarenal factors contribute to hypertension in nephrotic syndrome. Renal factors include albuminuria, sodium retention, activation of the RAAS (renin-angiotensin-aldosterone system), and fibrosis or decreased glomerular filtration rate (GFR). Extrarenal factors include the effects of medications, comorbid conditions, and genetic predisposition factors.[8] Hypertension is a risk factor for ventricular dysfunction. In children with hypertension, diastolic dysfunction of the left ventricle is observed owing to increased afterload, myocardial fibrosis, and inflammation.[9,10]

Another possible complication is endothelial dysfunction, which may be induced by excessive albuminuria, oxidative stress, or prolonged steroid therapy. Several markers indicate endothelial dysfunction, such as tissue plasminogen activator (t-PA), plasminogen activator inhibitor-1 (PAI-1), and von Willebrand factor (vWF). These markers reflect endothelial activation and damage, and are linked to atherosclerotic and thrombotic diseases. In children with SRNS, endothelial dysfunction tends to be more severe than in children with frequently relapsing or steroid-sensitive nephrotic syndrome, thereby significantly increasing the risk of atherosclerosis.[11]

Previous studies have shown that children with NS, especially those with SRNS, are at an increased risk for developing ventricular dysfunction. A deeper understanding of these risks in pediatric NS and SRNS patients is essential. However, there remains a scarcity of studies examining ventricular dysfunction in children with nephrotic syndrome, particularly those with SRNS, and this is especially true in the Indonesian context. This study represents the first Indonesian investigation assessing left ventricular function in pediatric SRNS using both ejection fraction (EF) and fractional shortening (FS), which are standard echocardiographic parameters for evaluating systolic function.[12] This study aims to identify potential risk factors that may serve as predictors of left ventricular function decline in this patient population.

Methods

This study was conducted using a retrospective descriptive research design. The sample comprised medical records of children aged over 1 month and under 18 years diagnosed with steroid-resistant nephrotic syndrome (SRNS) and treated at Dr. Hasan Sadikin General Hospital Bandung, between 2018 and 2022. The primary objective was to evaluate left ventricular function through transthoracic echocardiography. All echocardiographic examination were performed at Dr. Hasan Sadikin General Hospital Bandung by pedriatic cardiologist.

A total sampling method was applied, in which all patients who met the eligibility criteria during the study period were included.. The inclusion criteria were children with a confirmed diagnosis of SRNS, characterized by massive proteinuria, edema, and hypoalbuminemia,[1] who had undergone transthoracic echocardiography. Patient with incomplete records were excluded. Ethical approval and permission to access medical records were granted by the Health Ethics Research Committee Universitas Padjadjaran and Dr. Hasan Sadikin General Hospital (Approval No.197/UN6.KEP/EC/2023 and No.DP.04.03/X.2.2.1/5947/2023, respectively).

Data extracted from the medical records included sex, age, presence of congenital heart disease (CHD), rheumatic heart disease (RHD), other congenital disorders, chronic kidney disease (CKD) status, SRNS etiology, nutritional status, blood pressure (BP), heart rate (HR), laboratory results including platelet count, history of hematuria, and echocardiographic findings. Echocardiographic evaluation of left ventricular function was based on measurements of ejection fraction (EF) and fractional shortening (FS). Normal EF values were defined as 56–78%, and normal FS values as 28–45%.[12–14]

CKD stages were classified according to the KDIGO guidelines into non-CKD, CKD stage I, stage II, stage III, stage IV, and stage V.[1] Blood pressure categoriesnormal, prehypertension, stage I hypertension, and stage II hypertension were determined using the 2017 American Academy of Pediatrics (AAP) guidelines. Heart rate was categorized as bradycardia, normal, or tachycardia based on age-adjusted norms in *Park's Pediatric Cardiology for Practitioners*.[15] Platelet counts were classified as low, normal, or high using reference values from the *Nelson Textbook of Pediatrics*.[16] Hematuria was classified as either absent or present, also following criteria in the *Nelson Textbook of Pediatrics*.[16]

Results

This study included 42 out of 59 children with

Table 1 Distribution of Characteristics in Children with SRNS

Variables	Children with SRNS	
	n=42	%
Age (years)		
0 – 1	1	2.4
2 – 3	0	0
4 – 6	0	0
7 – 12	16	38.1
13 – 18	25	59.5
Gender		
Male	18	42.9
Female	24	57.1
Nutritional Status		
Severely wasted	6	14.3
Wasted	3	7.1
Normal	32	76.2
Overweight	1	2.4
SRNS Etiology		
Primary	31	73.8
Secondary	11	26.2
History of CHD/RHD/Other Congenital Diseases		
Yes	8	19.0
No	34	81.0
CKD History		
No CKD	17	40.5
Stage I	2	4.8
Stage II	1	2.4
Stage III	1	2.4
Stage IV	2	4.8
Stage V	19	45.2
Blood Pressure		
Normal	15	35.7
Pre-hypertension	4	9.5
Stage 1 hypertension	5	11.9
Stage 2 hypertension	18	42.9
Heart Rate		
Bradycardia	0	0
Normal	39	92.9
Tachycardia	3	7.1
Platelet Count		
Low	8	19.0

Table 1 Continued

Variables	Children with SRNS	
	n=42	%
Normal	29	69.0
High	5	11.9
Hematuria History		
Negative	9	21.4
Positive	33	78.6

steroid-resistant nephrotic syndrome (SRNS) who underwent echocardiographic examination and met the inclusion criteria. Of these, 31 children were diagnosed with primary SRNS, while 11 had secondary SRNS. Eight children had SRNS accompanied by congenital heart disease (CHD), rheumatic heart disease (RHD), or other congenital disorders. Two children had SRNS with chronic kidney disease (CKD) stage I, and 23 children had CKD stage I. The characteristics of the study sample are presented in Table 1.

Table 2 displays the distribution of echocardiographic findings in children with SRNS. The majority of children (61.9%) had normal ejection fraction (EF) and fractional shortening (FS) values, while 38.1% had decreased EF and FS. The mean EF and FS values were 55.8% and 30.2%, respectively, with median values of 59.3% and 31.2%.

The distribution of echocardiographic findings in children with steroid-resistant nephrotic syndrome (SRNS), according to various patient characteristics, is presented in Table 3. Among children with SRNS and poor nutritional status, the majority showed decreased ejection fraction (EF) and fractional shortening (FS) values (4 samples; 66.7%). Conversely, most children with primary SRNS had normal EF and FS values (18 samples; 58.1%), although a substantial proportion (13 children; 41.9%) exhibited decreased EF and FS values. Similarly, most children with secondary SRNS showed normal EF and FS values (8 samples; 72.7%), while 3 children (27.3%) had decreased values.

Most children with SRNS and a history of chronic kidney disease (CKD) demonstrated normal EF and FS values. However, among those at CKD stage V, a notable number exhibited decreased EF and FS values (8 samples; 42.1%). In contrast, among children with SRNS but no history of CKD, the proportions were nearly balanced between those with decreased (8 samples; 47.1%) and those with normal EF and FS values (9 samples; 52.9%).

Children with SRNS and a history of congenital heart disease (CHD), rheumatic heart disease (RHD), or other congenital conditions predominantly showed normal EF and FS values (6 samples; 75%), with the remainder (2 samples; 25%) demonstrating decreased values. All children with SRNS and a history of prehypertension, stage I hypertension, or stage II hypertension exhibited normal EF and FS values (19 samples). In contrast, among children with no history of hypertension, the proportion of those with decreased EF and FS values (8

Table 2 Echocardiographic Findings in Children with Steroid-Resistant Nephrotic Syndrome

Variables	Children with SRNS		Mean ± SD	Median
	n	%		
EF[1]				
Decreased	16	38.1	55.8±17.1	59.3%
Normal	26	61.9		
FS[2]				
Decreased	16	38.1	30.2±13.6	31.2%
Normal	26	61.9		

[1]EF, ejection fraction; [2]FS, fraction shortening

Table 3 Distribution of Echocardiographic Findings According to Clinical Characteristics in Children With Steroid-Resistant Nephrotic Syndrome

Characteristic	Category	EF Decreased n (%)	EF Normal n (%)	FS Decreased n (%)	FS Normal n (%)
Age (years)	0–1	0 (0.0)	1 (100.0)	0 (0.0)	1 (100.0)
	2–3	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
	4–6	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
	7–12	7 (43.8)	9 (56.3)	7 (43.8)	9 (56.3)
	13–18	9 (36.0)	16 (64.0)	9 (36.0)	16 (64.0)
Sex	Male	7 (38.9)	11 (61.1)	7 (38.9)	11 (61.1)
	Female	9 (37.5)	15 (62.5)	9 (37.5)	15 (62.5)
Nutritional status	Severely wasted	4 (66.7)	2 (33.3)	4 (66.7)	2 (33.3)
	Wasted	1 (33.3)	2 (66.7)	1 (33.3)	2 (66.7)
	Normal	11 (34.4)	21 (65.6)	11 (34.4)	21 (65.6)
	Overweight	0 (0.0)	1 (100.0)	0 (0.0)	1 (100.0)
Etiology	Primary	13 (41.9)	18 (58.1)	13 (41.9)	18 (58.1)
	Secondary	3 (27.3)	8 (72.7)	3 (27.3)	8 (72.7)
CKD stage	No CKD	8 (47.1)	9 (52.9)	8 (47.1)	9 (52.9)
	CKD I	0 (0.0)	2 (100.0)	0 (0.0)	2 (100.0)
	CKD II	0 (0.0)	1 (100.0)	0 (0.0)	1 (100.0)
	CKD III	0 (0.0)	1 (100.0)	0 (0.0)	1 (100.0)
	CKD IV	0 (0.0)	2 (100.0)	0 (0.0)	2 (100.0)
	CKD V	8 (42.1)	11 (57.9)	8 (42.1)	11 (57.9)
CHD/RHD/other congenital disease	Yes	2 (25.0)	6 (75.0)	2 (25.0)	6 (75.0)
	No	14 (41.2)	20 (58.8)	14 (41.2)	20 (58.8)
Blood pressure	Normal	8 (53.3)	7 (46.7)	8 (53.3)	7 (46.7)
	Pre-hypertension	1 (25.0)	3 (75.0)	1 (25.0)	3 (75.0)
	Hypertension I	2 (40.0)	3 (60.0)	2 (40.0)	3 (60.0)
	Hypertension II	5 (27.8)	13 (72.2)	5 (27.8)	13 (72.2)
Heart rate	Bradycardia	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
	Normal	14 (35.9)	25 (64.1)	14 (35.9)	25 (64.1)
	Tachycardia	2 (66.7)	1 (33.3)	2 (66.7)	1 (33.3)
Platelet count	Low	4 (50.0)	4 (50.0)	4 (50.0)	4 (50.0)
	Normal	11 (37.9)	18 (62.1)	11 (37.9)	18 (62.1)
	High	1 (20.0)	4 (80.0)	1 (20.0)	4 (80.0)
Hematuria history	Negative	3 (33.3)	6 (66.7)	3 (33.3)	6 (66.7)
	Positive	13 (39.4)	20 (60.6)	13 (39.4)	20 (60.6)

EF=ejection fraction; FS=fractional shortening;;CKD=chronic kidney disease; CHD=congenital heart disease; RHD=rheumatic heart disease.

samples; 53.3%) slightly exceeded those with normal values (7 samples; 46.7%).

In children with SRNS and a normal heart rate, most had normal EF and FS values (25 samples; 64.1%). However, children with tachycardia predominantly showed decreased EF and FS values (2 samples; 66.7%).

Among children with SRNS who had normal platelet counts or a history of thrombocytosis, most exhibited normal EF and FS values (22 samples). However, among those with thrombocytopenia, EF and FS values were evenly distributed between decreased and normal (4 samples each; 50.0%). Similarly, children with SRNS and a history of hematuria mostly demonstrated normal EF and FS values (20 samples; 60.6%).

Discussion

This is the first study in Indonesia to evaluate left ventricular function in children with steroid-resistant nephrotic syndrome (SRNS) using ejection fraction (EF) and fractional shortening (FS). The majority of patients demonstrated values within the normal range for both EF and FS, although a notable proportion (38.1%) showed reduced systolic function. The average EF and FS were 55.8% and 30.2%, respectively, with normal reference ranges of 56–78% for EF and 28–45% for FS.

This aligns with findings from Kamel et al., who reported that 20% of children with nephrotic syndrome (NS), including those with SRNS, exhibited decreased left ventricular function. The difference in prevalence between the two studies may be attributed to differences in the study populations. Kamel et al. primarily included children with steroid-sensitive nephrotic syndrome (SSNS), whereas the present study focused specifically on children with SRNS. Furthermore, changes in ventricular function are influenced by several factors, including the duration of the disease, treatment regimens, relapse frequency, and response to steroid and immunosuppressive therapies. It has also been reported that children with SRNS are more likely to experience impaired ventricular function than those with SSNS. Based on these findings, it can be concluded that while children with SRNS are at risk for reduced left ventricular function, this does not necessarily progress to overt dysfunction.[17]

Hypercoagulability, resulting from endothelial dysfunction, proteinuria, and dyslipidemia, is a significant risk factor for cardiovascular complications in children with NS, including SRNS.[5] Endothelial dysfunction can contribute to thrombus formation and is itself a risk factor for cardiovascular complications.[1118] In this study, most children with SRNS had normal platelet levels and did not exhibit signs of hypercoagulability. This may partially explain why the majority of these children demonstrated normal left ventricular function. Supporting this interpretation, most children with normal platelet counts also had normal EF and FS values.

Hypertension is another recognized risk factor for ventricular dysfunction in children with NS. It can lead to diastolic dysfunction of the left ventricle through mechanisms such as increased afterload, myocardial fibrosis, and inflammation.[9,10] Hypertension may also eventually cause systolic dysfunction; however, during the early stages, systolic function often remains preserved, and EF values may stay within the normal range despite underlying myocardial deformation.[19] In this study, most children with SRNS and a history of hypertension had EF and FS values within normal limits, likely due to effective management of their blood pressure. Similarly, previous studies have observed that systolic function, as indicated by EF, is generally preserved in the early stages of hypertension.[19]

Among children with poor nutritional status, a greater proportion showed reduced EF and FS values. Malnutrition, particularly in severely wasted children, is a known risk factor for decreased ventricular function. Previous studies have shown that malnourished children often exhibit diminished cardiac contractility, especially involving systolic function and the cardiac conduction system.[20] Additional research has reported that children with protein-energy malnutrition (PEM) may develop myocardial hypertrophy along with both systolic and diastolic dysfunction of the left ventricle.[21] Significant reductions in EF have also been documented in this population.[22]

Importantly, some children in this study exhibited reduced EF and FS values despite the absence of identifiable risk factors such as congenital heart disease, rheumatic heart disease, chronic kidney disease, or hypertensio. This finding highlights the inherent cardiovascular risk associated with SRNS itself. Cardiovascular complications in SRNS can stem from endothelial dysfunction caused by proteinuria and dyslipidemia, thromboembolic events due to hypercoagulability and thrombocytosis, and hypertension.[5,23] Reduced ventricular function

has previously been reported in children with idiopathic NS,[24] with an even higher risk observed in those with SRNS.[25] Notably, children with SRNS have been found to exhibit thickening of the tunica intima and media of the carotid arteries, which is a marker of increased cardiovascular risk.[23]

Transforming growth factor-beta (TGF-β) may also contribute to cardiovascular risk in SRNS. TGF-β levels are elevated in SRNS and are associated with vascular and cardiac remodeling, playing a key role in disease progression toward CKD or end-stage kidney disease (ESKD).[2]

This study has several limitations. The data were collected at a single time point, which limits the ability to assess changes in cardiac function over time. A prospective cohort study is needed to evaluate the long-term impact of SRNS on left ventricular function. Despite these limitations, the findings suggest that some children with SRNS experience left ventricular systolic dysfunction, even in the absence of known risk factors.

In conclusion, most of children with SRNS in the present study (61.9%) exhibited normal EF and FS values. However, children with SRNS remain at risk for left ventricular systolic dysfunction. This concern is underscored by the finding that 38.1% of participants demonstrated reduced EF and FS values. Furthermore, a decrease in EF and FS was observed in a subset of children despite the absence of identifiable risk factors or comorbidities, indicating that ventricular dysfunction may still occur independently in some cases of SRNS.

References

1. Kher KK, Greenbaum LA, Schnaper HW. Clinical pediatric nephrology. 3rd ed. United States: CRC Press; 2016. doi:10.1201/9781315382319.
2. Widiasta A, Wahyudi K, Sribudiani Y, Rachmadi D. The level of transforming growth factor-β as a possible predictor of cyclophosphamide response in children with steroid-resistant nephrotic syndrome. Biomedicine (Taipei) 2021;11:68–75. doi:10.37796/2211-8039.1205.
3. Widiasta A, Sribudiani Y, Nugrahapraja H, Rachmadi D. miRNAs involved in the TGFB signaling as possible markers of steroid-resistant nephrotic syndrome in children. Gene Rep 2025;39:102173. doi:10.1016/j.genrep.2025.102173.
4. Patnaik SK, Kumar P, Bamal M, Patel S, Yadav MP, Kumar V, et al. Cardiovascular outcomes of Nephrotic syndrome in childhood (CVONS) study: A protocol for prospective cohort study. BMC Nephrol 2018;19. doi:10.1186/s12882-018-0878-5.
5. Nourbakhsh N, Mak RH. Steroid-resistant nephrotic syndrome: past and current perspectives. Pediatric Health Med Ther 2017;8:29–37. doi:10.2147/PHMT.S100803.
6. Moore KL, Dalley AF, Agur AMR. Moore Clinically Oriented Anatomy. 8th ed. Netherlands: WoltersKluwer; 2018.
7. Politano SA, Colbert GB, Hamiduzzaman N. Nephrotic Syndrome. Prim Care 2020;47:597–613. doi:10.1016/J.POP.2020.08.002.
8. Shatat IF, Becton LJ, Woroniecki RP. Hypertension in childhood nephrotic syndrome. Front Pediatr 2019;7:287. doi:10.3389/FPED.2019.00287.
9. Zhou D, Yan M, Cheng Q, Feng X, Tang S, Feng Y. Prevalence and prognosis of left ventricular diastolic dysfunction in community hypertension patients. BMC Cardiovasc Disord 2022;22(1):265. doi:10.1186/S12872-022-02709-3.
10. Nadruz W, Shah AM, Solomon SD. Diastolic dysfunction and hypertension. Med Clin North Am 2017;101:7–17. doi:10.1016/J.MCNA.2016.08.013.
11. Sharma B, Saha A, Dubey NK, Kapoor K, Anubhuti, Batra VV, et al. Endothelial dysfunction in children with idiopathic nephrotic syndrome. Atherosclerosis 2014;233:704–6. doi:10.1016/J.ATHEROSCLEROSIS.2014.01.055.
12. Park MK. Special tools in evaluation of cardiac patients. park's pediatric cardiology for practitioners. 6th ed. Philadelphia: Elsevier; 202. p. 77–95.
13. Forshaw N, Broadhead M, Fenton M. How to interpret a paediatric echocardiography report. BJA Educ 2020;20(8):278–86. doi:10.1016/j.bjae.2020.03.010.
14. Tissot C, Singh Y, Sekarski N. Echocardiographic evaluation of ventricular function-for the neonatologist and pediatric intensivist. Front Pediatr. 2018;6:79. doi: 10.3389/fped.2018.00079.
15. Park MK. Arrhythmias and atrioventricular conduction disturbances. park's pediatric cardiology for practitioners. 6th ed. Philadelphia: Elsevier; 2021. p. 407–8.
16. Kliegman RM, St. Geme JW, Blum NJ, Shah SS, Tasker RC, Wilson KM. Nelson textbook of

paediatrics. 21st ed. Philadelphia: Elsevier; 2020.
17. Kamel AS, Abo Elnour SI, Ragaey Mahmoud MM, Sayed Kamel A. Cardiac performance evaluation in children with nephrotic syndrome. Fayoum University Medical Journal. 2020;6:18–27.
18. Chen X, Geng X, Jin S, Xu J, Guo M, Shen D, et al. The association of syndecan-1, hypercoagulable state and thrombosis and in patients with nephrotic syndrome. Clinical and Applied Thrombosis/Hemostasis 2021;27:1–6. doi: 10.1177/10760296211010256.
19. Sibel CE. The Role of echocardiography in hypertension. Clinical Experimental Cardiol. 2016;7:1–5. doi:10.4172/2155-9880.1000486.
20. Akdeniz O, Yılmaz E, Çelik M, Özgün N. Cardiac evaluation in children with malnutrition. Turk Pediatri Ars 2019;54:157–65. doi:10.14744/TURKPEDIATRIARS.2019.43815.
21. Dimiati H, Wahab AS, Juffrie M, Julia M, Gani BA. Study of NT-proBNP and Hs-Troponin I biomarkers for early detection of children's heart function of proteinenergy malnutrition. Pediatr Rep. 2019;11:25–30. doi:10.4081/PR.2019.7997.
22. Arshad MS, Khan S, Ali I, Arshad R, Abbas A, Irshad S. Echocardiographic based cardiac evaluation in children with severe acute malnutrition. J Pak Med Assoc. 2022;72:2391–4. doi:10.47391/JPMA.3094.
23. Kari JA, Quinlan C, Deanfield J, Shroff R, Tullus K. Endothelial Dysfunction in Children with Steroid-Resistant Nephrotic Syndrome. Inn J Pediatr. 2017;27(4):e8026. doi:10.5812/IJP.8026.
24. Ahmed HM, Ameen EED, Awad MS, Botrous OE. Assessment of carotid intima media thickness and left ventricular mass index in children with idiopathic nephrotic syndrome. Vasc Health Risk Manag. 2021;17:349–56. doi:10.2147/VHRM.S295868.
25. Candan C, Canpolat N, Gökalp S, Yildiz N, Turhan P, Taşdemir M, et al. Subclinical cardiovascular disease and its association with risk factors in children with steroid-resistant nephrotic syndrome. Pediatr Nephrol 2014;29:95–102. doi:10.1007/S00467-013-2608-3.