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Coagulation profile in children with idiopathic nephrotic syndrome- A cross-sectional study

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Abstract: ***Background:*** In children, Idiopathic Nephrotic Syndrome (NS) is a very common kidney disease. Massive proteinuria with high urinary protein-creatinine ratio is typical laboratory finding. Apart from it hypoalbuminemia, hypercholesterolemia and hypovolemia contribute to hypercoagulable states. In children and adults, the risk of thromboembolism is significant. ***Objective:*** To determine the coagulation profile among the children with idiopathic nephrotic syndrome. ***Methodology:*** This cross-sectional study was done at the Department of Paediatrics, Dhaka Medical College Hospital, from October 2018 to September 2019 and a total 52 subjects were enrolled. Fifteen cases were initial attack and thirty-seven were relapse cases selected by purposive sampling technique. Prothrombin time (PT), activated partial thromboplastin time (APTT), total platelet count (TPC), serum albumin, serum total cholesterol and protein- creatinine ratio were performed. Data were collected, recorded and analysed by SPSS. ***Result:*** In this study mean age of the children was 5.0±2.44 year. 73% subjects were in 2-6 years age group with male to female ratio being 1.4:1. Increased platelet count (>400000/mm3), prolonged PT (>16 sec) and APTT (>36 sec) were more in relapse cases than in initial attack cases (76% vs. 24%, p<0.001; 3% vs. 0%, p 0.52 and 14% vs. 0%, p 0.13 respectively). The Mean±SD of platelet count was significantly higher in relapse cases than initial attack cases (466081±175989 vs. 352066±103378/mm3, p 0.023). In relapse cases APTT showed significant positive correlation with protein-creatinine ratio (p <0.001) and negatively with s. albumin (p <0.025). ***Conclusion:*** Hypercoagulable state is more prone to develop in relapse cases than in initial attack cases. So, early detection is important to avoid related complications.

Keywords: Coagulation, Nephrotic Syndrome, children, Hypoalbuminemia, Hypercholesterolemia, Glomerular disease.

Original Researcher Article

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Article at a glance:

Study Purpose: *To determine the coagulation profile among the children with idiopathic nephrotic syndrome.*

Key findings: *Increased platelet count (>400000/mm3), prolonged PT (>16 sec) and APTT (>36 sec) were more in relapse cases than in initial attack cases (76% vs. 24%, p<0.001; 3% vs. 0%, p 0.52 and 14% vs. 0%, p 0.13 respectively). The Mean±SD of platelet count was significantly higher in relapse cases than initial attack cases (466081±175989 vs. 352066±103378/mm3, p 0.023). In relapse cases APTT showed significant positive correlation with protein-creatinine ratio (p<0.001) and negatively with s. albumin (p<0.025).*

Newer findings: *Affected male to female ratio is 1.4:1, but previously male to female ratio was 2:1. PT and APTT are mostly normal in this study but in previous studies showed PT were prolonged in initial attack NS and APTT were prolonged in all the relapsed cases.*

Abbreviations: *NS: Nephrotic Syndrome, PT: Prothrombin Time, APTT: Activated Partial Thromboplastin Time, AGN: Acute Glomerulonephritis, FFP: Freshly Frozen Plasma, TPC: Total Platelet Count, SPSS: Statistical Package for Social Science.*

INTRODUCTION

Nephrotic syndrome is a common renal disease among children. It is primarily a disease of children and 15 times more common in children than adults. Massive proteinuria (>1gm/m^2/day), hypoalbuminemia (<2.5gm/dl), generalized edema and hypercholesterolemia (>200mg/dl) are characteristics of nephrotic syndrome in children.[2] Primary or idiopathic nephrotic syndrome is common in 95% of children[2] and 85% of whom is minimal change nephrotic syndrome[3] with good prognosis. Idiopathic nephrotic syndrome is associated with primary glomerular disease without evidence of a specific systemic cause. The prevalence of minimal change nephrotic syndrome is higher in Indian subcontinent.[4] Incidence in Bangladesh is yet unknown.

The incidence of all forms of nephrotic syndrome in children is 2-4 per 100000 population, whereas in the Indian subcontinent it is estimated as 9-10 per 100000 population.[3] In young children, boys are more commonly affected than girls (ratio 3:2) but in teenagers and adults, the sex ratio is approximately equal .[4] Patients with nephrotic syndrome are at a risk for the development of a variety of complications which includes thromboembolism, infections, hypovolemia, shock, dyslipidemia, renal dysfunctions etc. About 5% patients in children with idiopathic nephrotic syndrome are at a risk for developing thromboembolic complications.[5] Thromboembolism in both arterial and venous circulation is significant in children with idiopathic nephrotic syndrome. Deep vein thrombosis, pulmonary embolism and renal vein thrombosis are the most common thromboembolic complications.[6] Thromboembolism is the most significant life threatening complication after infections in nephrotic syndrome.[7]

The pathophysiological mechanisms of thromboembolism in patients with nephrotic syndrome include, alterations in plasma levels of proteins involved in coagulation and fibrinolysis, enhanced platelet aggregation, low plasma albumin, hyper-viscosity and hyperlipidemia as well as treatment with corticosteroids and diuretics.[5] Patient may have a hereditary risk factors such as factor V Leiden mutation that predisposes to clot.[8] There may be intravascular depletion as a result of nephrotic syndrome that may be exacerbated by diuretic use to control edema. When combined with the urinary loss of coagulation cascade regulators (such as antithrombin III) and an increase in hepatic production of procoagulant factors (such as fibrinogen, factor V and factor VIII) that favor thrombus formation. Thrombocytosis and platelet aggregation also occur in nephrotic syndrome and may play a role in Thrombosis.[8, 9]

In addition to urinary loss of hematologic factors there is also loss of immunoglobulin. Beyond the urinary loss of albumin and immunoglobulin, nephrotic syndrome also causes the loss of other important proteins, including vitamin D binding protein and thyroid binding globulin. Thromboembolism in renal disease is divided into the following two groups: Systemic thromboembolism induced by nephrotic syndrome and intragloemrular thrombosis which is frequently observed in acute post streptococcal glomerulonephritis (AGN), diabetic nephropathy and lupus nephritis.[9] The occurrence of venous thrombosis may be influenced by the factors such as an increase in the platelet aggregation and loss of the low molecular weight regulatory proteins in urine and it may be worse by an increase in the viscosity which results from the use of diuretics.[10]

Adults with membranous nephropathy appear to be at the greater risk for developing thrombosis, specially renal vein thrombosis. However, the same is not true for children with membranous nephropathy.[11]There is some evidence that sub-clinical thrombosis may occur quite frequently in children with idiopathic nephrotic syndrome. So, coagulation profile screening before presentation of symptoms is very important to reduce the mortality rate due to this devastating complication.[11]

OBJECTIVE

- To determine the coagulation profile among the children with idiopathic nephrotic syndrome

METHODOLOGY

This cross-sectional study was conducted at the Department of Paediatrics, Dhaka Medical College Hospital, Dhaka for one year from October

2018 to September 2019 which included 52 subjects aged between 2 year to 10 year with idiopathic nephrotic syndrome: initial attack and relapse cases. It included 15 subjects as initial attack and 37 as relapse cases selected by purposive sampling technique. Steroid dependent, steroid resistant, congenital and secondary nephrotic syndromes as well as patient with liver disease on anticoagulant therapy and getting transfusion of albumin, FFP, Vitamin K were excluded. Prothrombin time (PT), activated partial thromboplastin time (APTT), total platelet count (TPC), serum albumin, serum total cholesterol and protein-creatinine ratio were performed for each subject. Data were collected and recorded in a predesigned data collection sheet and analysed by SPSS.

RESULTS

Table 1: Demographic profile of the study subjects (n=52)

Socio-demographic characteristics	Frequency	Percentage (%)
Age		
2-6	38	73
7-10	14	27
Total	52	100
Mean ±SD	5.0±2.44	
Range	2-10 years	
Gender		
Male	30	58
Female	22	42
Total	52	100
Ratio	1.4:1	
Residence		
Rural	33	64
Urban	19	36
Total	52	100
Socioeconomic status	38	73
Poor	12	23
Middle	2	4
Upper	52	100
Total		

Table 1 show Mean±SD of age was 5.0±2.44 with 73% at 2-6 years age group. Male was more (58%), rural subjects was 64%, socioeconomic status of most of the patients guardian was poor (73%). Male to female ratio was 1.4:1.

Table 2: Clinical manifestations of the patients (n=52)

Findings	Frequency	Percentage (%)
Symptoms		
Generalized swelling	52	100
Fever	35	67
Scanty micturition	48	92
Abdominal pain	18	35
Signs		
Generalized edema	52	100
3+ or 4+ proteinuria	52	100
Hypertension	5	10

Table 2 shows generalized swelling, scanty micturition, fever were the commonest presentation, 100%, 92% and 67% respectively of the study subjects. Clinical examination revealed that, edema and 3+ or 4+ proteinuria present in all patients (100%), followed by Hypertension noted in 10% of patients.

Table 3: Laboratory findings of parameters as Mean±SD of study subjects

Laboratory findings	**Initial attack** (n=15) Mean±SD	**Relapse case** (n=37) Mean±SD	**p-value** Unpaired t- test
Serum albumin mg/dl	18.45±7.44	17.10±6.47	0.516
S. total cholesterol mg/dl	281.67±143.29	408.7±125.5	0.003
Urinary spot protein-creatinine ratio	13.49±12.28	14.20±20.35	0.901
Platelet count /cmm	352066±103378	466081±175989	0.023
PT (Sec)	12.29±0.57	12.49±1.04	0.503
APTT (Sec)	31.47±1.18	32.49±4.21	0.363

Table 3 shows that there was no significant difference of s. albumin, urinary spot protein-creatinine ratio, and PT and APTT level between initial attack and relapse case (p >0.05), but there was significant higher serum cholesterol level and platelet count in initial attack and relapse case (p <0.05).

Table 4: Laboratory findings of the study subjects (n=52)

Laboratory parameter	**Initial attack** (n=15)	**Relapse case** (n=37)	**Total** (n=52)	**p-value** Chi-square test
Serum albumin mg/dl				
<25	12 (27%)	32 (58%)	44 (85%)	0.557
25-<50	3(7%)	5(8%)	8(15%)	
S. cholesterol mg/dl				
201-240	11(20%)	2 (6%)	13(26%)	0.001
>240	4 (7%)	35(68%)	39 (75%)	
Urinary spot protein creatinine ratio				
<2	0(0%)	0(0%)	0(0%)	1.000
>2	15(26%)	37(74%)	52 (100%)	
Platelet count / cmm				
150000-400000	11(26%)	9(13%)	20(39%)	0.001
>400000	4(7%)	28(54%)	32(61%)	
PT (Sec.)				
11-16	15(26%)	36(72%)	51 (98%)	0.520
>16	0(0%)	1(2%)	1(2%)	
APTT (Sec.)				
26-36	15(29%)	32(61%)	47(90%)	0.134
>36	0(0.0%)	5(10)	5(10%)	

Table 4 shows that higher platelet count(>400000/cmm), prolong PT and prolong APTT in total, initial attack vs. relapse case were found 61%, 7% vs. 54% (p 0.001); 2%, 0% vs. 2%, (p 0.52) and 10%, 0% vs. 10%, (p 0.134) respectively. In majority of subjects lower serum albumin (85%) and higher S. total cholesterol (75%) was seen and all subjects had protein creatinine ratio >2.

Table 5: Correlation of PT, APTT and platelet count with s. albumin, s. cholesterol, protein creatinine ratio in initial attack and relapse cases (n=52)

	Test variables	Pearson Correlation test			
		Initial attack (n=15)		Relapse case (n=37)	
		r-value	p-value	r-value	p-value
PT	S. Albumin	-.294	0.287	-.099	0.561
	S. cholesterol	+.077	0.784	+.125	0.459
	Protein-creatinine ratio	-.019	0.945	-.035	0.839
APTT	S. Albumin	-.026	0.926	-.368*	0.025
	S. cholesterol	+.052	0.853	+.071	0.678
	Protein-creatinine ratio	+.126	0.655	+.597*	<0.001
Platelet count	S. Albumin	-.438	0.103	+.137	0.417
	S. total cholesterol	+.298	0.280	+.233	0.165
	Protein-creatinine ratio	+.176	0.530	+.143	0.399

*Significant

Table 5: shows that no significant correlation of s. albumin, s. total cholesterol, protein creatinine ratio with PT, and platelet count were found in both initial attack and relapse cases. APTT showed significant positive correlation with protein creatinine ratio (p<0.001) and significant negative correlation with s. albumin in relapse cases (p value 0.025)

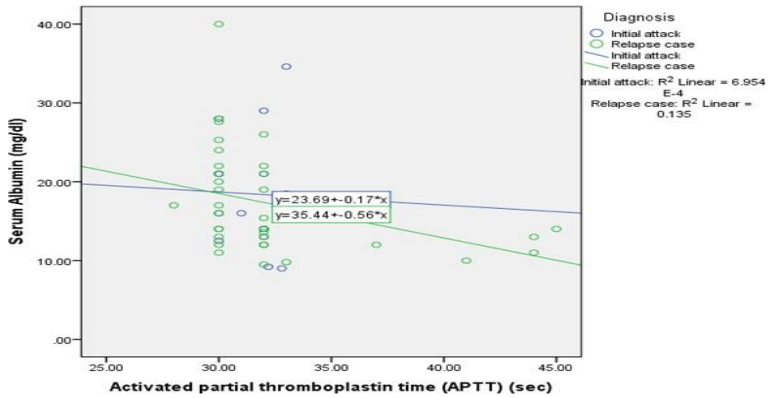

Figure 1: Scatter diagram showing the correlation of activated partial thromboplastin time with serum albumin in initial attack and relapse cases

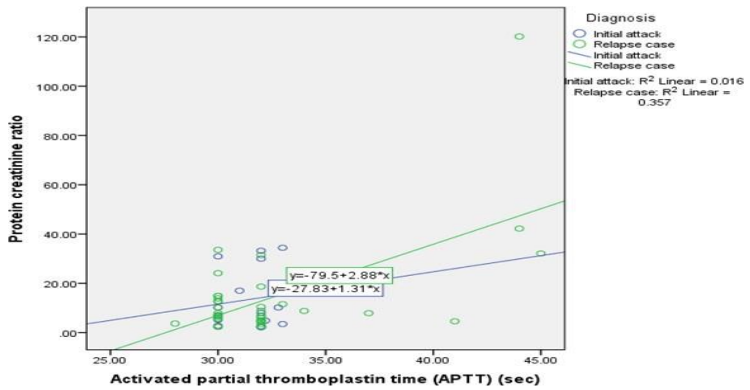

Figure 2: Scatter diagram showing the correlation of activated partial thromboplastin time with PCR in initial attack and relapse cases

DISCUSSION

In children, idiopathic nephrotic syndrome is the most common renal disease.[1,3] It is most commonly seen among school-aged children and adolescents. The prevalence is worldwide approximately 16 cases per 100,000 children with

an incidence of 2 to 7 per 100,000 children. Males appear to be more affected than females at a ratio of 2:1, but this predominance fails to persist in adolescence.[8] In a study in Bangladesh showed, out of 100 patients, majority (67%) were between 2-6 years of age with a mean 5.3±2.1 years and the lowest and highest ages were 2.3 and 10 years respectively. Sixty three of 100 subjects (63%) were male and the rest 37% was female giving a male-female ratio of roughly 2:1.[1]

This study shows (Table 1) that male to female ratio was 1.4:1 with Mean±SD of age was 5.0±2.44 years with 73% subjects were in 2-6 years age group which is consistent with other studies, [12] except this ratio which could be due to awareness of health for female child. Prevalence of idiopathic nephrotic syndrome is more in early age group (2-6 years) then gradually decline. Maximum number of male (60%) and female patients (90%) were affected within 2- 6 years. There is large number of subjects (63%) came from rural area, followed by urban area (37%). Socioeconomic status of the guardian of the participated children shows that 73% cases came from poor socioeconomic background, middle and upper class was 23% and 4% respectively. A study in Bangladesh showed similar findings e.g. 60% of the subjects came from rural, 35% from urban and remaining 5% from slum areas. Socioeconomic condition showed similar results with 59% of the subjects came from poor socioeconomic class followed by 39% from middle class and only 2% from upper class.[1]

Regarding clinical manifestations of idiopathic nephrotic syndrome (Table 2), this study shows generalized swelling and scanty micturition were the commonest presentation, 100%, 92% respectively followed by fever and abdominal pain 67% and 35% in the study subjects. Generalized edema and 3+ or 4+ proteinuria present in all patients (100%), followed by pallor and ascites, 67% and 44% of patients respectively. Both initial attack and relapse cases were more in male than female patients without any significant difference. High blood pressure was found only in 10% of the study subjects and all hypertensive subjects (5 cases) were male and majority (75%) was in relapse cases. A similar finding was also observed in a study of international study of kidney disease in children, 1978.

Present study (Table 3) shows that maximum patients (85%) were hypoalbumenic, where relapse cases and initial attack cases were 62% and 23% respectively. There was no significant difference between the Mean±SD of serum albumin in both initial attack and relapse cases of idiopathic nephrotic syndrome (18.45±7.44 mg/dl vs. 17.10±6.47 mg/dl, p>0.05). Different studies also found lower serum albumin in initial attack.[6,12] Lower serum albumin level in relapse cases of idiopathic nephrotic syndrome also found in a study by Sarker MN et al. (Mean±SD 1.5±0.2gm/dl, p<0.001)[1] and Rani AS *et al.,* (Mean±SD 1.79±0.73 gm/dl, p<0.001).[5] Hypoalbuminemia occur due to renal losses of protein mostly albumin.[5] This study shows that the Mean±SD of prothrombin time (PT) and activated partial thromboplastin time (APTT) in subjects with initial attack vs. relapse case of idiopathic nephrotic syndrome are 12.29±0.57 vs. 12.49±1.04 sec, p 0.503 and 31.47±1.18 vs. 32.49±4.21sec, p 0.363, respectively with no significant difference between two groups. A study conducted in 1996 by Anand NK et al. also found the Mean±SD of PT and APTT in initial attack and relapse cases were within normal limit (12.07±1.27sec vs. 13.23±1.34 sec and 30.58±1.85 sec vs. 35.10±5.63 sec, p >0.05), respectively.[12]

S. cholesterol levels were high among all (100%) study subjects where relapsed cases and initial attack was 75% and 26% respectively in round figures (Table 4). The Mean±SD of total cholesterol was significantly higher in relapse case than in initial case (408.7±125.5 mg/dl vs. 281.67±143.29 mg/dl, p 0.003), respectively. Hypercholesterolemia was also found in other studies (Mean±SD 373.2±137.5) in initial attack and (Mean±SD 482.5±55mg/dl p <0.001) & (Mean±SD 482.5±55mg/dl p <0.001) in relapse case of idiopathic nephrotic syndrome.[1,5,6] Hypoalbuminemia leads to protein synthesis by the liver including lipoprotein which leads to hypercholesterolemia. Higher total platelet count (Table 4) was seen in 61% of the study subjects and more in relapse case (54%) than in initial attack (7%). The Mean±SD of platelet count was significantly higher in relapse case than that in initial attack of idiopathic nephrotic syndrome (466081±175989 vs. 352066±103378/cmm, p 0.023), respectively. Another study also found 58%

patients of idiopathic nephrotic syndrome had higher platelet count both initial attack and relapse case (341300±53820/cm vs. 447600±187460/cmm, p <0.001).[12]

This study shows urinary spot protein creatinine ratio was >2 in all cases (100%) which is consistent with other studies, [5] but no significant difference was found between initial attack and relapse cases of idiopathic nephrotic syndrome (Table 4). Present study shows the level of PT was within normal limit in 98% of the study subjects and only one case out of fifty-two cases, PT was prolonged in relapse case. Similarly normal PT level was also observed as (Mean±SD 12.07±1.27sec, p >0.05)[12] & (Mean±SD 13.31 ± 4.31, p >0.05)[5] but in another study PT level was found significantly lower in initial attack nephrotic syndrome patients (Mean±SD 14.6±8.7, p <0.001).[6] APTT level was found within normal limit in both initial attack and in relapse cases; 100% and 86% respectively. Only 10% patient in relapse cases APTT was prolonged. In a study showed 15% subjects had APTT was prolong in initial attack,[12] but another showed all the relapse cases had prolong APTT level (Mean±SD 49.55 ± 9.23 sec, p <0.001),[5] (Table 4). In Table 5 and Figure 1, 2 Prothrombin time (PT) shows positive correlation with serum cholesterol and negative correlation with both serum albumin and urinary spot protein creatinine ratio in initial attack and in relapse cases of nephrotic syndrome. But correlation with of these laboratory values with PT were not significant (p >0.05).

Activated partial thromboplastin time (APTT) shows negative correlation with serum albumin in initial attack and in relapse cases. Both serum cholesterol and urinary spot protein creatinine ratio shows positive correlation with APTT both in initial and relapses. Negative correlation with serum albumin and positive correlation with urinary spot protein creatinine ratio with APTT in relapse cases of nephrotic syndrome were statistically significant (p <0.05). Platelet count shows positive correlation with both serum cholesterol and urinary spot protein creatinine ratio in initial attack and relapse cases of nephrotic syndrome but showed negative correlation in initial attack and positive correlation in relapse cases of idiopathic nephrotic syndrome. Correlation with of these laboratory values with platelet count were not significant (p >0.05).

No significant correlation of serum albumin, serum total cholesterol, and protein creatinine ratio with PT and platelet count were found in both initial attack and in relapse cases. APTT showed significant positive correlation with protein creatinine ratio (p <0.001) and significant negative correlation with serum albumin in relapse cases (p 0.025) which consistent with a study Rani AS et al. showed that APTT was positively correlated with cholesterol and urinary spot protein creatinine ratio and negatively correlated with serum albumin.[5]

CONCLUSION

Hypercoagulable state is more prone to develop in relapse cases than in initial attack cases. There is significant positive correlation of APTT with protein-creatinine ratio and negative correlation with serum albumin level. So, it should be kept in mind to avoid related complications during managing nephrotic syndrome cases.

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Authors contributions

IH, AKMSA, BT: Concept and design, data acquisition and interpretation, drafting and final approval. BA, RA, TA: Data acquisition, interpretation, drafting, final approval and agree to be accountable for all aspects of the work. SA: Principal guide.

Declarations

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Ethical approval

Ethical approval of the study was obtained from the Ethical Review Committee, BSMMU, Dhaka. The ethical issues were informed and addressed for future development of management to the participants' parents. Verbal consent had been given by the parents of the affected children.

Conflict of interest: There was declared no conflict of interest of the authors.

Consent for publication: Had been taken.

REFERENCES

1. Sarker MN, Islam MMSU, Saad T, Shoma FN, Sharmin LS, Khan H.A, Afrooz F, Fatmi LE, Alam A, Salimullah ASM and Uddin MR. Risk factor for relapse in childhood nephrotic syndrome-a hospital based retrospective study. Faridpur Medical College Journal 2012; 7(1): pp.18-22.
2. Srivastava RN & Bagga A. Nephrotic syndrome. In: Pediatric Nephrology. Jaypee Brothers Medical Publishers, New Delhi, India, 2016; 6th ed: 191.
3. Kayes MI. Recent Update in the Management of Childhood Nephrotic Syndrome. Journal of Bangladesh College of Physicians and Surgeons 2016; 34(1): 26-32.
4. Salcedo JR, Thabet MA, Latta K and Chan JC. Nephrosis in childhood. Nephron 1995; 71(4): 373-385.
5. Rani AS. A Study of PT, APTT, Fibrinogen and urinary protein-creatinin ratio in pediatric patients with nephrotic syndrome. International Journal of Cwontemporary Pediatri 2014; 1(2): pp.89-93.
6. Citak AGOP, Emre S, Sairin A, Bilge I. and Nayır, A. Hemostatic problems and thromboembolic complications in nephrotic children. Pediatric Nephrology 2000; 14(2): 138-142.
7. Kerlin BA, Ayoob R and Smoyer WE. Epidemiology and pathophysiology of nephrotic syndrome–associated thromboembolic disease. Clinical Journal of the American Society of Nephrology 2012; 7(3): 513-520.
8. Andolino TP and Reid-Adam J. Nephrotic syndrome. Pediatrics in review 2015; 36(3): 117-25 (available in: www.pedsinreview.aappublications.org; accessed on August 24, 2019).
9. Kaizu K] and Etoh S. Nephrotic Syndrome and Anticoagulant Therapy. Internal medicine 1998; 37(2): 210-212.
10. Mittal A, Aggarwal KC, Saluja S, Aggarwal A. and Sureka B. Platelet functions and coagulation changes in Indian children with nephrotic syndrome. Journal of clinical and diagnostic research: JCDR 2013; 7(8):1647.
11. Louis CU, Morgenstern BZ and Butani L. Thrombotic complications in childhood-onset idiopathic membranous nephropathy. Pediatric Nephrology 2003; 18(12): pp.1298-1300.
12. Anand NK, Chand G, Talib VH, Chellani H and Pande J. Hemostatic profile in nephrotic syndrome. Indian pediatrics 1996; 33:1005-1012.