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The Effectiveness of Low-Dose Colchicine in the Treatment of Steroid- Resistant Primary Focal Segmental Glomerulosclerosis

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Abstract:

Background: Focal Segmental Glomerulosclerosis (FSGS) is a rare condition that is detected in only 9% of all kidney biopsies. with consistent rates in recent years. This study aimed to investigate the effect of Low-Dose Colchicine (LoDoCo) (0.5mg once daily) as Add-On therapy on the response and progression of steroid resistant primary FSGS in patients receiving standard care. **Methods:** The study was carried on 40 patients with primary FSGS resistant to corticosteroids admitted to Nephrology Department at Benha University Hospitals. **Results:** The eGFR was significantly increased during follow-up (P<0.001). The eGFR was significantly higher at 1, 2 and 3 months compared to baseline (P<0.001, <0.001, <0.001), was significantly at 2 and 3 months compared to 1 month (P <0.001, <0.001) and was significantly higher at 3 months compared to 2 months (P <0.001). The response to LODOCO after 3 months, 22 (55%) patients showed complete remission whereas 18 (45%) patients showed partial remission. **Conclusion:** LoDoCo (0.5 mg once daily) is promising therapy for steroid resistant FSGS with 55% with complete remission whereas 45% with partial remission. A three-month course of low-dose colchicine (0.5 mg once daily) may enhance albuminuria and estimated glomerular filtration rate when administered alongside standard treatment for focal segmental glomerulosclerosis that has become resistant to corticosteroids. Specifically, 55% of patients experienced complete remission, while 45% experienced partial remission. Achieving remission of proteinuria is crucial for slowing disease progression.

Keywords: Low-Dose Colchicine; Primary Focal Segmental Glomerulosclerosis; Steroid-Sensitive, Estimated Glomerular Filtration.

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Introduction

Proteinuria, hypoalbuminemia, and edema are the hallmarks of idiopathic nephrotic syndrome (INS), a primary renal disorder. In INS, there are numerous histopathological subtypes; however, the three most prevalent are diffuse mesangial proliferation, minimal change disease, and focal segmental glomerulosclerosis (FSGS). Two forms of INS are distinguished by the response to steroid treatment: While proteinuria resolves in steroid-sensitive nephrotic syndrome (SSNS), it persists in steroid-resistant nephrotic syndrome (SRNS) [(1)]. At present, renal biopsy is not being performed on patients with SSNS, and the preponderance of patients with minimal change disease are generally effective with steroid treatment. This has resulted in the association of the term "minimal change disease" with SSNS. The identification of causative mutations in cases of infantile and juvenile SRNS and familial nephrotic syndrome has highlighted the significance of genetic manifestations of nephrotic syndrome. Histologic evidence indicates the existence of FSGS in the kidneys of most patients with SRNS, whether it is syndromic or nonsyndromic. Consequently, to save money on treatments that don't work, we need to work toward excluding genetic forms of FSGS. [(2)].

An anti-inflammatory and anti-fibrotic drug, colchicine is a lipophilic alkaloid. Inhibiting neutrophil adhesion and recruitment and disrupting the microtubule system are the main mechanisms in which colchicine exerts its effects. It is recommended that colchicine be used to prevent and treat familial Mediterranean fever and gouty arthritis [(3)]**.** An anti-inflammatory response is induced by colchicine, which disrupts neutrophil function by inhibiting microtubule polymerizations. Furthermore, colchicine exhibits anti-fibrotic characteristics via a wide variety of unique pathways. The beneficial effects of colchicine in preventing fibrosis and postponing the progression of renal disease have been consistently shown in experimental studies. This is not unexpected, as fibrosis is a terminal pathway that is prevalent in a wide range of renal diseases. The use of colchicine in renal disease is the subject of a scarcity of clinical data, despite the existence of encouraging experimental evidence [(4)].

Primary FSGS management remains a challenge due to the frequent need for additional immunosuppressive therapies and the limited therapeutic options available as a result of steroid resistance [(5)]. The 2021 KDIGO Clinical Practice Guideline on Glomerular Diseases states that corticosteroids are the initial treatment for primary FSGS. For a minimum of six months, calcinurin inhibitors should be administered in instances where steroids are ineffective. However, steroid-resistant disease is not an uncommon occurrence, and alternative agents have been suggested, including rituximab, mycophenolic acid, and cyclophosphamide; These agents have the potential to elevate the probability of infection, malignancy, and other complications. The therapeutic alternative for primary FSGS is not yet definitively established [(6)].

Colchicine is traditionally the first-line treatment for acute gout, as it has the capacity to reduce inflammation and irritation. Colchicine appears to inhibit a variety of pro-inflammatory mechanisms in addition to increasing the concentration of anti-inflammatory molecules, which provides a clinical advantage in gout patients. Typically, colchicine is employed to alleviate acute gout; however, it has recently been identified as possessing anti-fibrotic properties in a broad spectrum of nephropathies. Colchicine's protracted use is associated with nephrotoxicity, which can lead to renal function impairment, contingent upon the dosage. The risk of colchicine therapy is increased in patients with chronic kidney disease (CKD);

consequently, a dose reduction is required in accordance with the stage of CKD [7].

Colchicine significantly enhanced renal function by approximately 25% in a rabbit model of anti-glomerular basement membrane antibody-induced renal glomerulosclerosis, according to a study. Weight was not influenced by colchicine; however, anti-guinea pig immunoglobulin levels were , fibro cellular crescent formation, tissue hydroxyproline, or urine protein: creatinine ratio, and its exact mechanism of action couldn't be pinpointed, these findings suggest that colchicine, at doses relevant for human use, is beneficial in mitigating kidney damage in severe crescentic nephritis [8].

Patients with type 2 diabetes and microalbuminuria showed a consistent and effective decrease in NRCI when given low doses of colchicine. Still, it failed to show that these patients were significantly less likely to experience overt nephropathy when given low doses of colchicine. Similarly, no significant improvement in eGFR, UAE, or UACR could be found when low-dose colchicine was used. In order to prove that anti-NRCI does not slow the early stage progression of DKD, researchers performed a study [9].

With its new FDA approval for secondary prevention in coronary artery disease, low-dose colchicine (0.5 mg daily) is a well-tolerated and safe medication that can be used continuously, even by patients taking statins. A comprehensive review of current data reveals that common side effects are limited to mild, transient diarrhea upon initiation, which typically resolves within a week. It is crucial to note that renal, liver, and cognitive function are not adversely affected by continuous low-dose colchicine. Additionally, the risks of bleeding, wound healing complications, fertility issues, pregnancy complications, cancer, severe infections, or cause-specific mortality are not increased thereby [10].

The aim of this study was to investigate the effect of low dose colchicine (LODOCO) (0.5mg once daily) as Add-On therapy on the response and progression of steroid resistant primary FSGS in patients receiving standard care.

Patients and methods

This was a **prospective interventional single-arm clinical study** conducted on 40 patients with primary FSGS resistant to corticosteroids admitted to the Nephrology Department at Benha University Hospitals. An informed written consent was obtained from the patients. The purpose of the study was explained to each patient, and they were assigned a secret code number. The research was conducted with the approval of the Research Ethics Committee at the Faculty of Medicine at Benha University from November 2023 to November 2024.

Inclusion criteria biopsy-proven FSGS, and nephrotic syndrome that were resistant to corticosteroids were present in patients who were 18 years of age or older. Persistent or increasing proteinuria levels (>3.0 g) despite tolerated angiotensin-converting enzyme inhibitor/angiotensin-receptor blocker treatment and a minimum of 8 weeks of prednisone therapy were defined as treatment resistance or intolerance. The term "steroid resistance" was developed to denote the persistence of nephrotic syndrome in the presence of prednisone therapy (1 mg/kg per day) for a period more than four months.

Exclusion criteria included serum creatinine >1.4 mg/dL an estimated glomerular filtration (eGFR) <60 ml/min per 1.73 m^2, hepatic dysfunction, liver diseases, malignancy and secondary causes of FSGS (Diabetes mellitus, hypertension, obesity, hepatic viral infections (hepatitis C virus (HCV) and hepatitis B virus (HBV) and drugs).

All studied cases were subjected to the following: Full history taking included patient identification including (age, gender, occupation, and demographic details), social history (smoke or consume alcohol), past medical history (exclusion of secondary causes of FSGS) in primary FSGS, there is usually no history of

secondary factors such as human immunodeficiency virus (HIV) infection, nephrotoxic drugs or systemic diseases, (steroid use) : patients often have a history of steroid therapy for nephrotic syndrome but fail to achieve remission or see a significant reduction in proteinuria, indicating steroid resistance, family history(kidney disease or other autoimmune disorders) and response to corticosteroids and standard care treatment (control of hypertension , lowering of lipid levels , reduction in daily salt ,use of diuretics, ACEI and ARBs and immunosuppressive drugs) .**Examination included general examination** as measurement of height, weight, body mass index , vital signs as (pulse, temperature , blood pressure, respiratory rate,), general appearance, cardiovascular system, respiratory system, abdomen, skin and neurological examination. **Laboratory investigations including** (complete blood picture, fasting blood glucose (mg/dl), and glycated hemoglobin, serum creatinine (mg/dl), eGFR (ml/min/1.73m^2) by the chronic kidney disease epidemiology collaboration (CKD-EPI), blood urea (mg/dl), serum calcium (mg/dl), serum phosphorus (mg/dl), high-sensitivity C-reactive protein (mg/dl), S. albumin(g/dl), S. uric acid (mg/dl), lipid profile, SGOT (IU/L), SGPT (IU/L) and alkaline phosphatase (IU/L)., HCV and HBV viral markers, urine analysis, urinary 24 Protein (g/24hr), urinary albumin / creatinine ratio (mg/g), abdominal ultrasonography and renal biopsy.

We collected blood using three tubes: one with 4.5 mL of citrate, one with 10 mL of serum, and one with 10 mL of ethylene diamine tetraacetic acid (EDTA). The samples underwent centrifugation at room temperature for 10 minutes at 2000 times gravity, all within 4 hours of collection. The collected serum or plasma was chilled to -80°C for later use. The samples were kept for an average of 2.5 years before analysis. Renal function biomarkers included creatinine and blood urea nitrogen (BUN). In order to determine the eGFR, the (CKD-EPI) coefficient was utilized. A normal eGFR was defined as more than 90 mL/min/1.73 m2. The categories that renal function was placed into were mild decline (60-89 mL/min/1.73 m2), mild to moderate decline (45-59 mL/min/1.73 m2), moderate to severe decline (30-44 mL/min/1.73 m2), severe decline (15-29 mL/min/1.73 m2), or kidney failure (< 15 mL/min/1.73 m2).

Therapeutical trial design: pre- add-on vs post-Add-On low dose colchicine (LODOCO) (0.5 mg once daily) effects and post- add-on phase: Patients continue their standard therapy and LODOCO 0.5 mg once daily for 3 months and follow up the outcome monthly.

Outcomes

Before and during the first, second, and third months following the administration of low-dose colchicine, serum creatinine, eGFR, S. albumin, urinary 24 protein, urinary albumin/creatinine ratio, and high-sensitivity C-reactive protein (hsCRP) were monitored. Proteinuria levels below 0.3 g/d is considered complete remission, while levels between 0.3 and 3.5 g/d indicate partial remission or a 50% reduction in proteinuria. Patients were further divided into these two groups.

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Statistical analysis

The Armonk, New York, USA-based IBM SPSS v26 was important to our study. Using histograms and the Shapiro-Wilks test, we assessed if the data was normally distributed. Displayed quantitatively, parametric data was the mean plus standard deviation (SD). The non-parametric quantitative data was shown using the median and interquartile range. The frequency and percentage (%) of qualitative characteristics were displayed. To compare the same subjects across different settings or times, researchers employed repeated measures ANOVA tests. Statistical significance was

determined by a two-tailed P value less than 0.05.

Results

Table 1 shows the baseline characteristics, comorbidities and vital signs and of the studied patients.

Table 2 shows the laboratory investigation and lipid profile of the studied patients. The viral markers, there were no patient had HCV and HBV.

The pathological findings of the renal biopsy of the glomeruli showed that the % segmental sclerosis ranged from 17 to 59 with a mean of 42.33± 11 and the % global sclerosis ranged from 5 to 30 with a mean of 18± 7.48. The % interstitial disease was ≤1+ in 27 (67.5%) patients and was ≥2+ in 13 (32.5%) patients. The % vascular disease was ≤1+ in 33 (82.5%) patients and was ≥2+ in 7 (17.5%) patients. **Table 3**

The hs - CRP level was significantly decreased during follow-up (P<0.001). The hs - CRP was significantly lower at 1, 2 and 3 months compared to baseline (P<0.001, <0.001, <0.001), to 1 month was significantly higher compared to 2 and 3 months (P <0.001, <0.001) and was significantly higher at 2 months compared to 3 months (P <0.001). **Table 4**

Table 1: Baseline characteristics, comorbidities and vital signs (pre-Add-On LODOCO).

			Total (n= 40)
Baseline characteristics	**Age (years)**	**Mean± SD**	61.55± 7.34
		Range	45-75
	Sex	**Male**	31 (77.5%)
		Female	9 (22.5%)
	Weight (kg)	**Mean± SD**	72.58± 8.18
		Range	60-85
	Height (m)	**Mean± SD**	1.67± 0.04
		Range	1.59-1.73
	BMI (Kg/m^2)	**Mean± SD**	25.28± 3.2
		Range	20.1-30
Comorbidities	**Smoking**		9 (22.5%)
	Hyperlipidaemia		17 (42.5%)
	History of cardiovascular diseases		4 (10%)
	Previous history of operation		3 (7.5%)
Vital signs	**HR (beats/min)**	**Mean± SD**	80.95± 7.49
		Range	70-95
	SBP (mmHg)	**Mean± SD**	114.79± 4.03
		Range	110-120
	DBP (mmHg)	**Mean± SD**	74.25± 4.01
		Range	70-80
	RR (breaths/min)	**Mean± SD**	16.4± 1.1
		Range	15-18
	Temp (°C)	**Mean± SD**	36.92± 0.12
		Range	36.7-37.1

Data was presented as mean± SD or range or frequency (%).BMI: Body mass index. HR: Heart rate, SBP: systolic blood pressure, DBP: diastolic blood pressure, RR: respiratory rate, Temp: temperature.

Table 2: Laboratory investigation, lipid profile and viral markers (pre-Add-On LODOCO) .

		Total(n=40)
Laboratory investigation		
Hb (g/dl)	**Mean± SD**	11.53± 1.04
	Range	9.5-13.7
WBCs (*10^9/L)	**Mean± SD**	8.01± 2.05
	Range	4.9-11.5
Platelets (*10^9/L)	**Mean± SD**	216.6± 57.51
	Range	121-302
Fasting blood glucose (mg/dl)	**Mean± SD**	89.85± 6.16
	Range	80-100
HbA1c (%)	**Mean± SD**	4.49± 0.33
	Range	4-5.5
Blood urea (mg/dl)	**Mean± SD**	65.43± 21.09
	Range	31-100
Serum calcium (mg/dl)	**Mean± SD**	9.39± 0.59
	Range	8.5-10.2
Phosphorus (mg/dl)	**Mean± SD**	3.88± 0.32
	Range	3.4-4.5
Creatinine (mg/dL)	**Mean± SD**	1.19± 0.09
	Range	1.1-1.4
eGFR (mL/min/1.73 m2)	**Mean± SD**	77.1±7.1
	Range	63-88
Serum uric acid level (mg /dl)	**Mean± SD**	5.95± 1.06
	Range	3.8-7.3
SGOT (IU/L)	**Mean± SD**	27.23± 5.09
	Range	20-35
SGPT (IU/L)	**Mean± SD**	22.2± 4.7
	Range	15-30
ALP (IU/L)	**Mean± SD**	65.05± 8.79
	Range	50-80
Lipid profile		
Cholesterol (mg/dL)	**Mean± SD**	190.15± 24.29
	Range	137-230.2
Triglyceride (mg/dL)	**Mean± SD**	182.48± 58.65
	Range	91-275
HDL (mg/dL)	**Mean± SD**	51.23± 6.12
	Range	41-60
LDL (mg/dL)	**Mean± SD**	102.43± 20.93
	Range	71-139
Viral markers	**HCV**	0 (0%)
	HBV	0 (0%)

Data was presented as mean± SD or range. Hb: hemoglobin, PLT: platelet count, WBCs: white blood cells, HDL: high-density lipoproteins, LDL: low-density lipoprotein. HbA1c: glycated hemoglobin. SGOT: Serum Glutamic-Oxaloacetic Transaminase, SGPT: Serum Glutamic Pyruvic Transaminase, ALP: Alkaline Phosphatase.

Table 3: Pathological findings of the renal biopsy (pre-Add-On LODOCO) .

			Total (n= 40)
Glomeruli	**% Segmental sclerosis**	**Mean± SD**	42.33± 11
		Range	17-59
	% Global sclerosis	**Mean± SD**	18± 7.48
		Range	5-30
Interstitial disease		**%≤1+**	27 (67.5%)
		%≥2+	13 (32.5%)
Vascular disease		**%≤1+**	33 (82.5%)
		%≥2+	7 (17.5%)

Data was presented as numbers.

Table 4: High sensitivity CRP (pre-Add-On VS Post-Add-On LODOCO)

		Pre-Add-On LODOCO	***Post-Add-On LODOCO***			
		Baseline	*1 month*	*2 months*	*3 months*	*P value*
hs - CRP (mg/dL)	*Mean±SD*	52.57± 22.39	48.65± 22.22	37.81± 18.51	27.34± 15.29	<0.001*
	Range	12.3-86.2	10.1-83.5	7.7-77.5	6-70.5	
		P1	< 0.001*	< 0.001*	< 0.001*	
			P2	< 0.001*	< 0.001*	
				P3	< 0.001*	

hs - CRP: High sensitivity c - reactive protein, *: significant difference<0.05, P1: P value compared to baseline, P2: P value compared to 1 month, P3: P value compared to 2 months,

Serum creatinine was significantly lower during follow-up (P<0.001), whereas urinary albumin/creatinine was significantly decreased during follow-up (P<0.001). Serum albumin was insignificantly different along the different measurement during the follow-up. Serum creatinine and urinary albumin/ creatinine were significantly lower at 1, 2 and 3 months compared to baseline (P<0.05), was significantly lower at 2 and 3 months compared to 1 month (P <0.05) and was significantly lower at 3 months compared to 2 months (P <0.05). During the follow-up, the proteinuria was significantly reduced (P<0.001). The proteinuria was significantly lower at 1, 2, and 3 months compared to baseline (P<0.001, <0.001, <0.001), at 2 and 3 months compared to 1 month (P <0.001, <0.001), and at 3 months compared to 2 months (P <0.001). During the follow-up period, the eGFR significantly increased (P0.001). At baseline, the eGFR was significantly lower than at 1, 2, and 3 months (P<0.001, <0.001, <0.001). It was significantly higher at 2 and 3 months compared to 1 month (P <0.001, <0.001) and at 3 months compared to 2 months (P <0.001). **Table 5** The response to Add-On low dose colchicine (LODOCO) after 3 months, 22 (55%) patients showed complete remission whereas 18 (45%) patients showed partial remission. **Table 6**

Table 5: Creatinine, albumin and urinary albumin/ creatinine ratio, proteinuria and estimated glomerular filtration (eGFR) (pre-Add-On VS Post-Add-On LODOCO)

		pre-Add-On LODOCO	***Post-Add-On LODOCO***			
		Baseline	*1 month*	*2 months*	*3 months*	*P value*
Creatinine (mg/dL)	*Mean± SD*	1.19± 0.09	1.11± 0.11	1.03± 0.16	0.95± 0.23	*<0.001**
	Range	1.1-1.4	0.9-1.4	0.7-1.3	0.6-1.3	
	P1		< 0.001*	< 0.001*	< 0.001*	--
	P2			< 0.001*	< 0.001*	
	P3				< 0.001*	
Albumin (g/dL)	*Mean± SD*	4.29± 0.41	4.09± 0.42	4.08± 0.44	4.12± 0.44	*0.088*
	Range	3.4-4.9	3.4-4.9	3.4-4.9	3.4-4.9	
Urinary albumin / creatinine (mg/g)	*Mean± SD*	3453.78± 129.08	2594.03± 119.32	1527.68± 87.82	1108.93± 48.93	*<0.001**
	Range	3226-3698	2411-2790	1406-1699	1011-1192	
	P1		< 0.001*	< 0.001*	< 0.001*	--
	P2			0.002*	< 0.001*	
	P3				0.001*	
Proteinuria (g/24hrs.)	*Mean± SD*	3.98± 0.25	2.72± 0.47	1.68± 0.89	1.03± 0.92	*<0.001**
	Range	3.6-4.4	1.7-3.7	0.1-3.2	0.096-3.2	
	Median (IQR)	3.9 (3.8 - 4.2)	2.8 (2.5 - 3.0)	1.6 (0.9 - 2.5)	0.8 (0.2 - 1.8)	
	P1		< 0.001*	< 0.001*	< 0.001*	--
	P2			0.002*	< 0.001*	--
	P3				0.001*	--
eGFR (mL/min/1.73 m2)	*Mean± SD*	77.1±7.1	79± 6.94	80.13± 7.1	81.8± 6.78	*<0.001**
	Range	63-88	66-90	68-92	70-95	
	P1		< 0.001*	< 0.001*	< 0.001*	--
	P2			< 0.001*	< 0.001*	
	P3				< 0.001*	

Data was presented as mean± SD or range *: statistically significant as P value <0.05. eGFR: estimated glomerular filtration.

Table 6: Response to Add-On low dose colchicine (LODOCO) after 3 months

		Total (n= 40)
Response	**Complete remission**	22 (55%)
	Partial remission	18 (45%)

Data was presented as frequency (%).

Discussion

The CRP level decreased significantly (P<0.001) during the follow-up period. At every time point, the CRP was significantly lower compared to the previous one or two months, three months to two months (P<0.001), and one and two months to baseline (P<0.001, ~0.001, ~0.001).

In accordance with our study, Sun et al. [11] stated that low dose colchicine for FSGS associated with decreasing inflammatory markers as CRP.

As well, Fiolet et al., [12] was told that after being exposed to low-dose colchicine for one month, two inflammatory markers, hs-CRP and IL-6, showed a decrease.

The NLRP3 inflammasome, microtubule-based inflammatory cell chemotaxis, phagocytosis, and the production of leukotrienes and cytokines are among the numerous pathways that colchicine inhibits to be associated with inflammation. The rationale for these findings is as follows. The accumulation

of colchicine in white blood cells leads to a variety of effects, including the reduction of motility, the loosening of adhesion and chemotaxis, the inhibition of the formation of superoxide anions, and the disruption of mast cell degranulation. As a result, Colchicine inhibited the synthesis of IL-1γ/IL-18/IL-6, increased the production of pro-inflammatory cytokines, and generated neutrophil extracellular traps, all of which served to prevent endothelial injury. Endothelial cells and white blood cells interact much less as a consequence [13].

Serum creatinine decreased considerably during follow-up (P<0.001), while urinary albumin/creatinine decreased significantly during follow-up (P<0.001). When comparing serum albumin levels measured at various points in the follow-up period, no statistically significant differences were found. At 1, 2, and 3 months, urinary albumin/creatinine levels and serum creatinine levels were found to be significantly lower than the baseline levels (P<0.05). At 2 and 3 months, they were significantly lower (P<0.05) when compared to 1 and 2 months, respectively. Proteinuria also decreased significantly (P<0.001) during the duration of the follow-up. At 1, 2, and 3 months, proteinuria was significantly lower than at the beginning (P<0.001, <0.001, <0.001), at 2 and 3 months, it was lower than at 1 month (P<0.001, <0.001), and at 3 months, it was lower than at 2 months (P<0.001). The eGFR also increased significantly (P<0.001) throughout the follow-up period. Compared to baseline, 1 month, and 3 months, the eGFR was significantly lower at 2 months (P<0.001, <0.001, <0.001), but it was significantly higher than 2 months and 1 month, respectively, when compared to a month later.

These findings are in accordance with Barut et al. [14] who reported that colchicine is an interesting compound with potential benefits for FSGS, particularly in preventing inflammation and fibrosis and decreasing serum creatinine and A/C ratio.

Additionally, Solak et al. [15] Colchicine is an alkaloid derived from plants that has been shown to accumulate in neutrophils, interfere with the microtubule network in cells, and halt the recruitment and adhesion of neutrophils. In addition, colchicine blocks the entry of other inflammatory and fibrotic mediators into cells. The most common and last stage of chronic renal disease is fibrosis of the kidneys.

On explanation of the results, low dose LODOCO (0.5 mg once daily) as Add-On therapy might be beneficial in FSGS as anti-inflammatory effects that FSGS often involves inflammation in the kidneys, The anti-inflammatory and anti-fibrotic effects of colchicine have the potential to alleviate this inflammation and slow the development of FSGS-defining fibrosis (scarring) in the kidneys, and anti-proliferative effects as colchicine's ability to inhibit cell growth may help prevent the proliferation of cells in the kidneys that contribute to FSGS [7, 16].

The response to Add-On low dose LODOCO after 3 months, 22 (55%) patients showed complete remission whereas 18 (45%) patients showed partial remission.

Conclusion

Low dose LODOCO (0.5 mg once daily) is a prospective therapy for steroid-resistant FSGS, with 55% of patients experiencing complete remission and 45% experiencing partial remission. Patients with focal segmental glomerulosclerosis that has not responded to corticosteroids may see an improvement in albuminuria and eGFR after three months of using low-dose colchicine (0.5 mg once daily) as an add-on medication. Specifically, 55% of patients experience complete remission, while 45% experience partial remission. Achieving proteinuria remission is essential for decelerating the progression of the disease.

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Conflicts of interest

No conflicts of interest

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