World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-58 May 2026
ISSN: 2749-3644

MORPHOLOGICAL CHANGES IN THE GASTRIC MUCOSA IN CHRONIC KIDNEY DISEASE IN CHILDREN

Turdiqul A. Bobomuratov[1], Abduqakhkhor A. Samadov[2]
Tashkent State Medical University, Tashkent, Uzbekistan
Orcid =0009-0009-8448-4068 E-mail: abdukahhorsamadov@gmail.com

Article history:

Received: 24th March 2026
Accepted: 20th April 2026

Abstract:

This article discusses changes in the mucous membranes of the stomach and duodenum observed by esophagogastroduodenoscopy in children aged 7–17 years with chronic kidney disease that were treated with glucocorticosteroids for six months or longer. As a result of the analysis, chronic gastroduodenitis was detected in 65 (55.1%) cases and gastrobioptates were obtained. Morphological alterations in the cellular composition of the gastric mucosal glands were analyzed in relation to the severity of gastric mucosa inflammation, with morphometric evaluation of the cellular composition conducted to characterize these alterations. Morphometric analysis of the samples taken from 26 healthy children was performed to define reference values to characterize the cellular composition of the gastric mucosal glands as myometria measures.

Keywords: children, chronic kidney disease, glucocorticosteroids, stomach, esophagogastroduodenoscopy, gastroduodenitis, morphometry.

INTRODUCTION

According to the recommendations of the World Health Organization (WHO), the International Pediatric Nephrology Association (IPNA), and the International Study of Kidney Disease in Children (ISKDC), pediatric chronic kidney disease is diagnosed based on the presence of particular clinical characteristics. Those characteristics include proteinuria greater than 1 g/m2/milk, hypoalbuminemia with serum albumin < 25 g/L, dysproteinemia, hypercholesterolemia, and edema ranging from mild peripheral edema to severe anasarca. In severe case, chronic renal failure may also be detected [5,7,8,10,15,17,18,].

In children, chronic kidney disease often has a different presentation than in adults. In some cases, it can develop from an acute episode that deteriorates quickly into chronicity. Taking into account the severity and duration of an acute attack in children, pediatricians recommend immunosuppressors and corticosteroids in addition to the blocking drugs of the renin-angiotensin system, which in turn have a toxic effect on other organs and systems. [1,2,7,17,18,20]

Stages of chronic kidney disease (K/DOQI, 2002)

Stage of CKD	eGFR result (ml/min/1.73 m²)	What it means
Stage 1	**90 or higher**	- Mild kidney damage - Kidneys work as well as normal
Stage 2	**60-89**	- Mild kidney damage - Kidneys still work well
Stage 3a	**45-59**	- Mild to moderate kidney damage - Kidneys don't work as well as they should
Stage 3b	**30-44**	- Moderate to severe damage - Kidneys don't work as well as they should
Stage 4	**15-29**	- Severe kidney damage - Kidneys are close to not working at all
Stage 5	**less than 15**	- Most severe kidney damage - Kidneys are very close to not working or have stopped working (failed)

The biggest reason of chronic kidney disease is nephrotic syndrome.

Classification of nephrotic syndrome: first of all, practical doctors consider it significant to distinguish between congenital, infantile, primary and secondary forms of nephrotic syndrome.[8,14,15,18,23,].

1. Primary neurotic syndrome (PNS).
2. Secondary neurotic syndrome (SNS).

World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-58 May 2026
ISSN: 2749-3644

Depending on the response of the standard course of treatment with prednisolone:
3. Steroid-sensitive neurotic syndrome (SSNS).
low-causal nephrotic syndrome (LCNS).
4. Steroid-dependent nephrotic syndrome (SDNS).

Several authors, including Papayan A.V. and Savenkova N.D. (1997), Nagashima R, Maeda K, Yuda F, et al. (1997), Top C, Vural A, Caglar K, et al. (1998), Zakirov N.Z., Sergeeva T.V., Shavrov A.A. et al. (1997), and Belmer S.V. and Gasilina T.V (1998), have remarked that with long-term treatment of chronic kidney disease (NS) by glucocorticosteroids (GKS) and immunosuppressive drugs (IDs), many children develop diffuse damage of their membranes. This damage results from immunopathological, hemostasiological, and microcirculatory disturbances in the child's body [1,2,5].
In children, a rise in the content of mochevina due to chronic renal failure in deep NS, there changes the background concentration of cytotoxin ammonia in gastric juice to undergone licorice effects of bacterial urease, and the gastric mucosa (pumpkin), and "uremic gastropathy"- denotes inflammation of the mucosal membrane of the GI tract. The inflammatory process causes significant changes in the cellular composition of the glands of the mucous membrane of all parts of the stomach (body, antral, bottom) [2,3,4,6,12,13,16,19,22].

A study of the morphofunctional properties of the digestive and excretory systems shows significant similarities in their histological construction, mechanisms of transport, and engaging regulatory principles of features and functions. The digestive and excretory systems also possess interconnected physiological, microbiological, and immunological shared features, and exhibit functional parallels in the variety of pathological states which may involve both systems [11,14,15,18,21,22].
From the above, it is unconditionally relevant to determine the features of morphological changes in the structure of the gastric mucosal glands cell in nephrotic cidrom in children.

THE PURPOSE OF OUR STUDY

To study the morphological changes in the cellular composition of the glands of the gastric mucosa in nephrotic cidrome in children, their dependence on the level of the inflammatory process.

MATERIALS & METHODOLOGY. From 2024 to 2025, a study was performed at the multidisciplinary clinic of the Tashkent Medical Academy in a sample of 118 children aged 7 to 11 years. The patients were hospitalized and treated in the Department of Nephrology. Specifically, 54 children (45.8%) were evaluated with frequently-relapsing chronic kidney disease (FRNS), 38 children (32.2%) with steroid-dependent chronic kidney disease (SDNS), and the remaining 26 children (22%) did not reveal any issues in the stomach or duodenum and were viewed as clinically well. Through esophagogastroduodenoscopy (EFGDS) and morphometric analysis, mucosal changes in the stomach and duodenum were assessed.

RESULTS

This study**,** EFGDS examination results showed chronic gastritis in 4 cases (7.4%) of FRNS n=54, and chronic gastroduodenitis in 36 cases (66.7%) $p<0.001$. SDNS n = 38 to 3 (7.9%) were diagnosed with chronic gastritis and 29 (76.3%) with chronic gastroduodenitis. of a total of 92 patients, 7 (7.6%) were diagnosed with chronic gastritis and 65 (70.6%) with chronic gastroduodenitis. In 26 practically healthy children, no changes were detected in the mucous membrane of the stomach and 12 fingers of the intestines. The changes detected in the gastric mucosa (esophagus) were due to clinical pictures of chronic kidney disease and were observed mainly in children who took glucocorticosteroids for 6 months or more.
The assessment of the cellular composition of the gastric mucosa salivary glands was described on the basis of a morphological - morphometric examination. To quantify the standard cellular arrangement of glands in the mucous membrane of the body and antral area of the stomach and to evaluate inflammatory infiltration, we estimated key subsets of cells (chief cells, parietal cells, endocrine cells, and accessory cells) in ‰ (per 1,000 glandular epithelial cells) in one microscopic field of view. Inflammatory infiltration was assessed with a visual-analog scale that included evaluation and scoring of both stromal and intraepithelial components, including lymphocytic and granulocytic stromal infiltration. The scoring was based upon the following: 1 = absent or minimal infiltration; 2 = mild infiltration; 3 = moderate infiltration; and 4 = severe infiltration. Lastly, we quantified the number of intraepithelial lymphocytes and neutrophils in ‰ per 1,000 glandular epithelial cells.
To investigate the cellular makeup of the gastric mucosal glands (esophagus) after an EFGDS evaluation, a group of clinically healthy children (n=26) who exhibited no apparent modifications in the gastric mucosa was identified. Gastrobiopsy specimens were collected from these children, and the samples were subjected to morphometric analysis.

World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-58 May 2026
ISSN: 2749-3644

The data showed that 50% of the cellular makeup in the gastric body glands were chief cells, which was approximately equal to one-fifth (194 ± 7‰) of the entire cell population of parietal cells. Endocrine cells represented the least of all cells, making up only 62 ± 3‰ in that cell population. The morphometric data of GGM are given in Tables 1 and 2.

Table 1.Gastric fundal in children normative cell composition of glands (in‰)

Cell name	Number of cells 1000 ‰
Head or main cells	468 ‰
Parietal cells	194 ‰
Endocrinocyte cells	62 ‰
Additional cells	276‰
Total ‰	1000 ‰

Table 2. Normative cell composition of gastric antral part glands in children (in‰)

Cell name	Number of cells 1000 ‰
Additional cells	695 ‰
Endocrinocyte cells	243 ‰
Parietal cells	62 ‰
Total ‰	1000 ‰

More than two-thirds of the cellular type present in the antral region of the esophageal glands was identified as accessory type. The number of endocrinocytes in the esophageal antrum was significantly greater than that of the stomach body, being approximately fourfold larger at a density of 246 throughout the sample area. The prominent role of the antrum is important in relation to digestive physiology. Parietal cells were detected in the pyloric glands of the antrum (61 ± 11‰). (Figure 1).

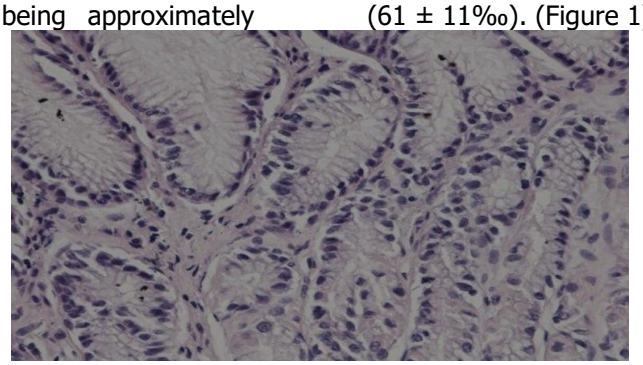


Figure 1. In moderation, the condition of the mucous membrane of the antral part. Appearance of parietal cells in the pyloric glands. Hematoxylin-eosin dyed × 300.

The results of EFGDS testing revealed FRNS n=54 of chronic kidney disease in a sample, 36 (66.7±6.5%, R<0.001) cases and n=38 of SDNS group, 29 (76.3±3.2%) cases in a total of 65 (55.1%) cases, and the cell composition of gastric mucosal glands was assessed morphometrically in relation to the extent of the gastroduodenitis chronic inflammatory process in the gastrobioptats obtained.

With an increase in inflammation in the Fundal glands, a sharp decrease in the number of head cells was observed, which in a clearly expressed process amounted to only 41.1% of the normative indicator (Table 3). In Pprallel, it was noted that the number of parietal cells increases in light, moderate and sharply expressed inflammations by 18.6; 25.1 and 31.7%, respectively. Analog acidification was observed in additional cells. (14.8, 18.9 and 23.5% respectively). Endocrinocyte cells have been found to undergo the most alterations, the amount of which doubles even in mild inflammation (62 to 141‰), while in sharply expressed inflammation it increased by 3.46 times (Figure 2).

3-Table The cellular composition of the glands of the stomach body, depending on the degree of the inflammatory process, ‰

Cells of the gastric	Inflammatory process in the body of the stomach			
	Not available	Lightly expressed	Medial	Sharply expressed

World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-58 May 2026
ISSN: 2749-3644

body glands				
Main	468 ± 11	309±5***	256±8***	188±15***
Parietal	194 ± 10	229±5***	242±7***	256±18***
Additional	276 ± 11	324±8***	338±8***	348±19***
Endocrine	62 ± 3	141±5***	165±7***	211±15***

Note: * differentiation with respect to the non-inflammatory group is reliable (***0,001).

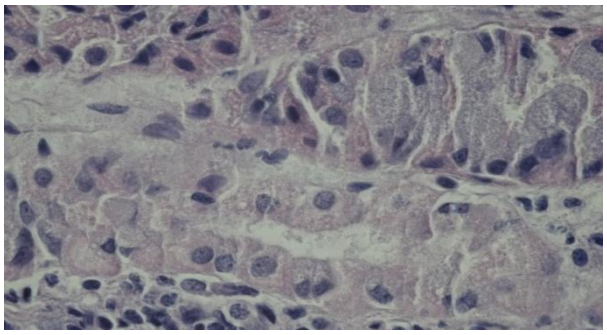


Figure 2. Fundal glands when acute expression of inflammation. Significant increase in the number of Parietal and endocrinocytes. Hematoxylin-eosin dyed × 600

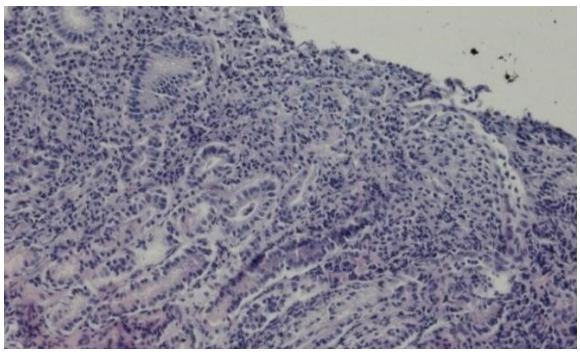
In the morphometric analysis of the cellular composition within the antral glands, endocrinocytic cells were observed to increase substantially during periods of marked inflammation. The number of endocrinocytic cells increased nearly doubled from 246‰ to 462‰ ($r < 0.001$).

4-Table. Cellular composition of the glands of the antral part of the stomach, depending on the degree of the inflammatory process, ‰

Antral part of the gland cells	Inflammatory process in the antral part of the stomach			
	Not available	Lightly expressed	Medial	Sharply expressed
Parietal	61 ± 13	41 ± 6***	30 ± 5*	17 ± 4***
Additional	693 ± 12	617±13***	573±10***	526±12***
Endocrine	246 ± 11	339±13***	396±10***	462±11***

Note: * differentiation with respect to the non-inflammatory group is not available (***0,001).

It was found that the number of parietal cells decreased significantly (61 to 17‰, $p<0.001$). In addition to the increase in the level of inflammation, it was found that the number of additional cells has decreased. (Figure 3).



World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-58 May 2026
ISSN: 2749-3644

Figure 3. Pyloric glands in sharply expressed inflammation. A significant increase in the number of endocrinocytes. Hematoxylin-eosin dyed × 60

DISCUSSIONS: The results demonstrate the implication that the clinical course of chronic kidney disease (i.e., duration, severity, and length of glucocorticosteroid treatment) is an important contributing factor to the presence of gastrointestinal mucosal changes among children diagnosed with chronic kidney disease. The symptoms of gastrodudenitis identified during the EFGDS examination were diagnosed in 100% of cases with a diagnosis of chronic kidney disease, up to 3-5 hospitalizations, taking glucocorticosteroids for 6 months or more, it was observed in children in the FRNS and SDNS groups. Stages of chronic renal failure in children with detected changes, recommendations (K/DOQI, 2002) and Glomerular filtration rate (GFR) Schwartz formula: 2/3 changes in SKF-based analysis (ml/min/1.73m^2) = K×Height(cm)/blood creatinine (mmol/l)K=38 Level III corresponded to moderate-severe 59-30, and level IV-severe 29-15. Over the past five years, a number of domestic and foreign scientists have analyzed the results of endoscopic examination in various somatic, non-communicable chronic diseases, taking into account the negative effects of GCS and other drugs on the condition of the mucous membrane of the gastroduodenal zone. The analysis showed that superficial gastritis and gastroduodenitis often recur (65.1%).[6,7,13,14,15,17,18].

An increase in the overall toxicity of the body as a result of chronic renal failure in children leads to significant changes in the cellular composition of the glands of the gastric mucosa, which leads to uremic gastropathy as an inflammatory effect on the gastric mucosa.[2,3,4,6,12,13,15]. The results of our study also coincided with the data presented in the literature. The results of the study suggest that nephrotic syndromnmng in children needs new scientifically based data with the aim of early diagnosis of the development of changes in the gastric mucosa as a result of prolonged treatment with glucocorticosteroids and the recovery of children.

CONCLUSIONS

In children suffering from chronic kidney disease, clinical esophagogastroduodenoscopy made it possible to assess the severity of inflammation before proceeding with a morphological evaluation of the gastric mucosa.

As a result of the examination, chronic gastritis, (5.9%), chronic gastroduodenitis were found in (55.1%) cases, these changes were found to be associated with impaired maturation in chronic kidney disease and the duration and duration of glucocorticosteroid intake.

In children without any observable morphological alterations in the gastric mucosa, around 50% of the cells within the composition of fundal glands were chief cells. Parietal cells were about the fifth of the composition and accessory cells made up about a quarter. Endocrinocytes were the least abundant at 6.1%.

In children without morphological changes in the gastric mucosa, chief cells comprised 50% of the total cellular composition of the fundal glands. Parietal cells and accessory cells were present in 5:1 and 4:1 ratios, while endocrinocytes were represented the least at 6.1%.

The observation of inflammation in the gastric mucosa causes the mucous membrane glands to significantly change the cell composition. In the fundal glands, as the process progresses, the number of primary cells decreases, the parietal, auxiliary and especially endocrine cells increase.

In the pyloric glands, the set of endocrinocytes increases sharply, against the background of which the number of parietal cells decreases significantly.

REFERENCES

1. Agaronyan A. G., Vashurina T. V., Komarov O. V., Zrobok O. A., Ananyin P. V., Timofeeva A. G., & Tsygin A. N. Immunosuppressive therapy for steroid-dependent chronic kidney disease in children: A literature review (2020). *Nephrology and Dialysis.*4, 474-489.
2. Ankireddypalli S. Peptic ulcer in chronic kidney disease patients due to steroid therapy (2023). *Journal of Pharmacovigilance and Drug Research*.1, 1-4.
3. Barysheva O.Yu., Simanov R.N. The relationship between inflammatory bowel disease and kidney disease*. Bulletin of the North-Western State Medical University named after I.I. Mechnikov.* (2022).1, 31-38.
4. Blanpain, C. Plasticity of epithelial stem cells in tissue regeneration.C. Blanpain, E. *Fuchs Science. (*2014), 344, (6189), P. 1242281.
5. Bobomuratov T. A., Samadov A. A. Changes in the gastrointestinal tract in the treatment of glucocorticosteroids used in children with chronic kidney disease: results of a

World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-58 May 2026
ISSN: 2749-3644

retrospective study.*World Bulletin of Public Health.* (2022). (11), 54-57.
6. Bobomuratov T.A., Rakhmanova L.K., Samadov A.A. Effect of helicobacter pylori in children with chronic kidney disease to the dysfunction of stomach and duodenum*. Acta scientific gastrointestinal disorders*.(2022).5.
7. Bobomuratov T.A., Samadov A.A. Assessment of the functional state of the glandular structure of the gastrointestinal mucosa in children with steroid-dependent chronic kidney disease *.Problems of Biology and Medicine*. (2023). (150), 527-531.
8. Bobomuratov T.A., Samadov A.A. Diagnosis of gastroduodenal diseases in children with chronic kidney disease.*Bulletin of the TMA Special Issue*. (2024).182-186.
9. Bobomuratov T.A., Samadov A.A. Morphological and Functional Features of Gastric and Duodenal Disorders in Children with Chronic Kidney Disease. *New Day in Medicine*.(38/1),383-386.
10. Bobomuratov T.A., Samadov A.A. Chronic kidney disease in children: Etiological and epidemiological aspects, classification, treatment and complications.*Problems of Biology and Medicine.* (2023). (1), 243-249.
11. Bobomuratov T.A., Samadov A.A., Iskanova G.H. Effectiveness of histamine H2-receptor blockers in children with chronic kidney disease receiving glucocorticosteroid therapy*. Journal of Biomedicine and Practice*.(2021).(4),101-106.
12. Bobomuratov T.A., Samadov A.A., Mamadiyorov A.M. Assessment of the Functional State of the Gastric Mucosal Gland Structure in Steroid-Dependent Chronic Kidney Disease in Children. *American Journal of Medicine and Medical Sciences. (*2023). (13), 1087-1090.
13. Hassan F. M., Saleh N. Y., Eldaher M. A., & Abd Elsalam H. B. Prevalence of Helicobacter pylori infection among children with chronic kidney disease. *Menoufia Medical Journal.* (2022). (35), 1288.
14. Ismayilova Z. A., Rajabova N. T. Clinical and laboratory characteristics of the manifestation of chronic kidney disease in children. *Problems of Modern Science and Education*. (2020). (6-2), 68-70.
15. KDIGO Clinical Practice Guideline for Glomerulonephritis*. Kidney International supplements.* (In Russ.) Available at: http://kdigo.org/clinicalpracticeguidelines/pdf /KDIG0%20GN%20Russian%20Full%20Text. pdf. Link active on 02.06.2017.
16. Khamzaev K. A., Sharipov A. M. Clinical and morphological features of the course of steroid-dependent chronic kidney disease in children. *Medical News*. (2020). (6), 75-77.
17. Mashkova A. E. Chronic kidney disease in children .*Proceedings of the Republican Scientific and Practical Conference of Students and Young Scientists dedicated to the 95th anniversary of the birth of Professor Maslakov D.A.*(2022).510-511.
18. Chronic kidney disease in children. (In Russ.) Available at: http://www.pediatr-russia.ru/sites/default/files/file/kr_nefr.pdf. Link active on 02.06.2017.
19. Nikolaevna T. P. The features of the upper gastrointestinal tract lesions in patients with end-stage renal disease. (2021).
20. Samadov A.A. Changes in the Mucous Membrane of the Stomach and Duodenum in Children with Chronic Kidney Disease. *Academic Research in Educational Sciences Scientific Journal.*(2023).(4),126-132.
21. Samadov A.A. Changes of gastric mucous layers in chronic kidney disease in children. *International scientific conference*. (2024).13-14.
22. Samadov A.A., Nurmatova N.F., Habibulloev B.R. Efficacy of histamine h2-receptor blockers in children with chronic kidney disease receiving glucocorticosteroid therapy*. European Journal of Interdisciplinary Research and Development*. (2023). Website: http://ejird.journalspark.org/index.php/ejird
23. Zhdanova O. A., Nastausheva T. L., Batischeva G. A., Savchenko A. P., Stenshinskaya E. V., Zvyagina T. G. Physical development and idiopathic chronic kidney disease in children. *Nephrology*. (2020). 24 (3), 79-89.