

# *Helicobacter Pylori* Infection and Gastroduodenal Lesions in Patients with Chronic Kidney Disease: A Comparative Study

Winnie T. Bekolo Nga<sup>1,2\*</sup>, Servais A. F. Eloumou Bagnaka<sup>1,3</sup>, Nancy Halle-Ekane<sup>1,4</sup>, Antonin Ndjitoyap Ndam<sup>5</sup>, Guy R. Senga Ndjapa<sup>6</sup>, Hermine Fouda<sup>5</sup>, Lionel P. J. Elimby Ngande<sup>5</sup>, Agnès Malongue<sup>1</sup>, Dominique Noah Noah<sup>5</sup>, Mathurin Kowo<sup>5</sup>, Firmin Ankouane Andoulo<sup>2</sup>, Henry N. Luma<sup>1,5</sup>, Marie P. Halle-Ekane<sup>1,2</sup>

<sup>1</sup>Internal Medicine Department, Douala General Hospital, Douala, Cameroon

<sup>2</sup>Faculty of Medicine and Pharmaceutical Sciences, University of Douala, Douala, Cameroon

<sup>3</sup>Internal Medicine Department, Gyneco-Pediatric and Obstetric Hospital of Douala, Douala, Cameroon

<sup>4</sup>Faculty of Health Sciences, University of Buea, Buea, Cameroon

<sup>5</sup>Faculty of Medicine and Biomedical Sciences, University of Yaoundé I, Yaounde, Cameroon

<sup>6</sup>Faculty of Medicine, University of Dschang, Dschang, Cameroon

Email: \*winbek@yahoo.fr

**How to cite this paper:** Nga, W.T.B., Bagnaka, S.A.F.E., Halle-Ekane, N., Ndam, A.N., Ndjapa, G.R.S., Fouda, H., Ngande, L.P.J.E., Malongue, A., Noah, D.N., Kowo, M., Andoulo, F.A., Luma, H.N. and Halle-Ekane, M.P. (2023) *Helicobacter Pylori* Infection and Gastroduodenal Lesions in Patients with Chronic Kidney Disease: A Comparative Study. *Open Journal of Gastroenterology*, 13, 49-60.

<https://doi.org/10.4236/ojgas.2023.131006>

**Received:** December 10, 2022

**Accepted:** January 16, 2023

**Published:** January 19, 2023

Copyright © 2023 by author(s) and Scientific Research Publishing Inc.

This work is licensed under the Creative Commons Attribution International License (CC BY 4.0).

<http://creativecommons.org/licenses/by/4.0/>



Open Access

## Abstract

**Introduction:** Gastroduodenal lesions are common in chronic kidney disease (CKD). They are linked to various factors including *Helicobacter pylori* infection (*H. pylori*). Few data are available in Africa on *H. pylori* infection and chronic kidney disease. The aim of this study was to assess the impact of *H. pylori* infection and to describe the gastroduodenal lesions found in patients with chronic kidney disease. **Patients and Methods:** A cross-sectional study was conducted, February 1<sup>st</sup> to May 31<sup>st</sup>, 2021, at the Douala General Hospital in Cameroon. We included patients with CKD classified as stages 3 to 5 according to KDIGO classification, on hemodialysis or not, who agreed to participate in the study. They were matched with a “control” population including patients with normal renal function according to sex and age (ratio 1:2). Patients on antibiotics and/or proton pump inhibitors were excluded. We collected data from CKD patients and from medical records for non-CKD group. Each patient underwent an upper digestive endoscopy and identification of *H. pylori* using a urease rapid test. Logistic regression was used to identify independent associations for a significance level set at  $p < 0.05$ . **Results:** We included 99 patients including 33 with CKD and 66 control patients. Among patients with CKD, the predominance was male ( $n = 18/33$  or 54.5%). The mean age was  $51.2 \pm 12.8$  years. Arterial hypertension was the first etiology of CKD ( $n = 13$  or 39.4%). The prevalence of *H. pylori* in pa-

tients with CKD was 63.6% versus 37.9% in control patients (p-value = 0.015). The main endoscopic lesions were erosive gastropathy (n = 14 or 42.4%) and erythematous gastropathy (n = 7 or 21.2%). Patients with CKD were 5 times more likely to have *H. pylori* infection (OR = 5.69; CI 95% 0.14 - 0.82; p = 0.017). Factors associated with *H. pylori* infection were chronic kidney disease (aOR = 1.02; CI 95% 0.14 - 0.82; p = 0.017) and hemodialysis (aOR = 10; CI 95% 1.08 - 91.9; p = 0.042). **Conclusion:** The prevalence of *H. pylori* infection is higher in patients with CKD. Endoscopic lesions are inflammatory. Factors associated with *H. pylori* infection are chronic kidney disease and hemodialysis.

## Keywords

Chronic Kidney Disease, *H. Pylori*, Gastroduodenal Lesions, Comparative Study, Cameroon

---

## 1. Introduction

*Helicobacter pylori* is a spiral-shaped, gram-negative flagellated bacterium that usually resides in the gastric mucosa [1]. It affects approximately 50% of the world's population, even 80% in lower and middle outcome countries [1] [2]. The prevalence according to a study conducted by Ankouane *et al.* in Yaoundé (Cameroon) is up to 72.5% in hospital [3]. The main route of transmission is person-to-person transmission and often occurs in the first 5 years of life [4]. The risk factors for infection with *H. pylori* are low socioeconomic level, promiscuity, family history of *H. pylori* infection or gastritis, alcohol consumption, smoking [5]. The diagnosis of *H. pylori* is made by noninvasive methods (the rapid urease test, the breath test, serology, stool antigen test) or invasive methods biopsy-based tests (culture and histology) [6] [7]. The gold standard is histology, but current methods have been developed, using high-definition endoscopy [7].

It has close associations with gastroduodenal disorders [8] [9]. Recent studies suggest some extra gastroduodenal disorders like chronic kidney may be related to *H. pylori* [10] [11] [12] [13]. Although evidence demonstrating an association between *H. pylori* infection and renal disease remain unknown. About 25% - 75% of chronic kidney disease (CKD) patients suffer from multiple gastrointestinal lesions and their complications (gastric erosions, peptic ulcer disease, angiodysplasia, and gastrointestinal bleeding) [14] [15] [16]. *H. pylori* plays a major role in the genesis of many gastrointestinal conditions both in individuals with normal renal function and CKD. It has been hypothesized that the uremic state of CKD patients increase the risk of *H. pylori* infection [17] [18]. This stems from the notion that *H. pylori* possess several pathogenetic determinants, including urease activity, converting urea to ammonia, providing protection against low pH [18]. Thus, the gastric mucosa of uremic patients could be prone

*H. pylori* mucosal colonization, leading to mucosal damage and gastrointestinal lesions [19]. However, this was contradicted by some studies that reported no association between the uremic state and *H. pylori* colonization [19] [20].

The prevalence of *H. pylori* infection in CKD patients ranges from 20% to 64% [21] [22] [23] [24] [25]. However, the association of *H. pylori* and CKD patients compared to individuals with normal renal function (NRF) remains controversial. In Africa, very few data are available on the association between these two frequent conditions. We aimed to study the prevalence and the determinants of *H. pylori* infection and gastroduodenal lesions in CKD patients.

## 2. Patients and Methods

We conducted a cross-sectional study at the Douala General Hospital over 5 months from January to May 2021. We included patients over the age of 18, with CKD between stage 3 and 5 according to KDIGO classification [26], and who had freely consented to participate in the study. We excluded patients recently on proton pump inhibitors two weeks or less prior to the interview, on nonsteroidal anti-inflammatory drugs, histamine 2 receptor inhibitors, antibiotics (Amoxicillin, Clarithromycin, Metronidazole, Quinolones, Tetracycline) within one last month. A group of comparison was selected with normal renal function (NRF). Two NRF age and sex corresponding patients were included for one CKD patient.

CKD was defined as documented chronic kidney failure by a nephrologist for more than 3 months with  $\text{GFR} \leq 60 \text{ ml/min}$  [26]. The stage defined according KDIGO guidelines [26]. NRF was considered as having a normal serum creatinine level and no history of kidney disease or no risk factors of CKD for patients without serum creatinine [26]. The patients were contacted through the nephrology department of the Douala general hospital. The sample size calculation was calculated using the formula for comparing two proportions [27]:

$$n = \frac{p_1(1-p_1) + p_2(1-p_2)}{p_1 - p_2} \times c_{p,\text{power}}$$

where  $n$  is sample size;  $p_1$  and  $p_2$  are expected sample proportions of the two groups and  $c_{p,\text{power}}$  the critical value, for a confidence interval of 95% the critical value is 1.96.

Nardone *et al.* reported the proportion of CKD patients with *H. pylori* infection as 74% ( $p_1$ ) and in patients with NRF as 36% ( $p_2$ ). Applying the above formula, the 23 participants per group, and to cover non-consenting patients, we added 20% to the minimum sample size. Therefore, the minimum sample size for our study was 28 participants per group.

After signing the informed consent form, each patient was interviewed by the principal investigator and the data collected was noted on an anonymous and individual data collection sheet. We collected data on socio-demographic characteristics (age and sex), past history (high blood pressure, diabetes, HIV status, viral hepatitis), lifestyle (notion of alcohol consumption, and/or smoking), CKD

(date of diagnosis, etiology, onset of dialysis, number of weekly dialysis sessions). Then, an appointment was scheduled for the upper GIT endoscopy. Upper GIT endoscopy was performed by a single endoscopist using a FUJINON® EPX-2200 endoscopy column. This endoscopist was a senior endoscopist. During the endoscopy examination, five biopsies are taken: two in the antrum, two in fundus and one in corner of lesser curvature. These biopsies were used to check for *H. pylori*. The biopsies were placed in a urease rapid test kit (Helicobacter UT® Plus). The result on the urease rapid test kit is given within 30 minutes to 2 hours according to the instructions available. The lesions found and the results of the urease rapid test kit were recorded in the data collection sheet for each patient. We classified endoscopic lesions according to location and the type. In the stomach, lesions were gastritis (erythematous, erosive, pseudo-nodular) and ulcer. The lesions of esophagus were esophagitis. Ulcer and bulbitis were the lesion described in duodenum.

Data were analyzed using SPSS version 28 software. Results were expressed as mean  $\pm$  SD and percentages. Comparison between groups was made using chi-square test for categorical variables and Student T-test for continuous variables. Logistic regression was performed to determine factors independently associated to *H. pylori* infection in CKD patient (95% Confidence Interval). Results were considered statistically significant for  $p$ -value  $< 0.05$ .

#### **Ethical considerations**

Ethical approval was granted by the institutional review board (IRB) of the Faculty of Health Sciences (FHS), University of Buea n°2021/1275-02/UB/SG/IRB/FHS. Administrative authorization was obtained from the Administration of the Douala General Hospital n°002AR/MINSANTE/HGD/M/01/21.

**Respect for autonomy:** Participants were given consent forms to sign after understanding the details (nature, risks, benefits) of the study. The participants were free to withdraw from the study at any time they pleased.

**Confidentiality:** Participants were given codes instead of using their names for identification. The consent forms containing names were kept separate from the data collection forms. These forms were kept secure.

**Beneficence:** Participants benefited from a gastroenterology consultation and knowing their *H. pylori* and gastroduodenal disease status for free. They were also counselled and given prescriptions if needed. The information obtained at the end of the study will improve scientific knowledge, which could help their management.

**Non-maleficence:** There were no risks involved in this study. All efforts were made to maximize the safety of participants.

**Justice:** Participants were treated equally and fairly.

### **3. Results**

We included 33 CKD and 66 NRF patients in this study. 54.4% were males (54.4%) with a M/F ratio of 1.2. The mean age was  $51.1 \pm 12.8$  years ranging from 26 - 77 years. Hypertension (32.3%) and diabetes (7.1%) were the main comorbidities. 75.8% of CKD patients were at stage 5, and 66.7% were on dialy-

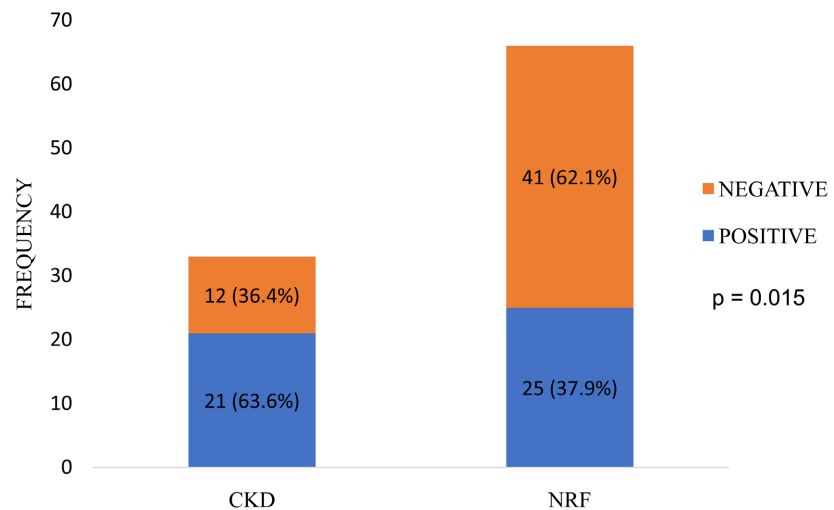
sis. The median duration of hemodialysis was 5 years (IQ 1.5 - 6.5). The main etiology of CKD was hypertension (**Table 1**). In both groups, we had a common history of chronic epigastric pain, which was more present in CKD patients, 96.9% vs 43.9% (p-value < 0.001). Epigastric pain, pyrosis, and eructation were the commonest presenting symptoms in both groups (**Table 2**). The CKD patients significantly presented anorexia and asthenia. The prevalence of *H. pylori* infection among CKD patients was 63.6% and 37.9% in patients with NRF (p-value = 0.015) (**Figure 1**).

Gastroduodenal lesions were present in 87.9% (95% CI: 69.9% - 98.2%) of CKD patients vs 72.7% (95% CI: 61.74% - 83.6%) in patients with NRF (p = 0.73). The most common endoscopic finding in patients with *H. pylori* infection was erythematous gastropathy (**Table 3**). The difference between CKD patients

**Table 1.** General characteristics of study population.

	All patients (n = 99) N (%)	CKD patients (n = 33) N (%)	NRF patients (n = 66) N (%)
<b>Mean Age (years)</b>		51.2 ± 12.8	51 ± 12.8
<b>Sex</b>			
Males		18 (54.5)	36 (54.5)
Females		15 (45.5)	30 (45.5)
<b>Comorbidities</b>			
Hypertension	32 (32.3)	26 (78.8)	6 (9.1)
Diabetes	7 (7.1)	6 (18.2)	1 (1.5)
HIV	7 (7.1)	4 (12.1)	3 (4.5)
<b>Etiologies of CKD</b>			
Hypertension		13 (39.4)	
Diabetes		4 (12.1)	
Chronic glomerunephritis		4 (12.1)	
Others <sup>(1)</sup>		7 (21.2)	
Unknow		5 (15.2)	
<b>Stages of CKD</b>			
3		2 (6.1)	
4		6 (18.2)	
5 non dialyzed		3 (9.1)	
5 dialyzed		22 (66.7)	
<b>Duration of dialysis (years)</b>			
Median (IQR)		5 (1.5 - 6.5)	

(1): mixed nephropathy, HIV, Polycystic kidney disease, chronic interstitial nephritis.



**Figure 1.** Prevalence of *H. pylori* infection.

**Table 2.** Clinical Presentation of study population.

Clinical characteristics	CKD (N = 33) N (%)	NRF (N = 66) N (%)	Total (N = 99) N (%)	p-Value
Chronic epigastric pain	32 (96.9)	29 (43.9)	61 (61.6)	<0.001
Nausea	13 (39.4)	30 (45.5)	43 (43.4)	0.56
Pyrosis	23 (69.7)	51 (80.3)	74 (74.7)	0.23
Bloating	10 (30.3)	28 (42.4)	38 (38.3)	0.24
Regurgitation	5 (15.2)	22 (33.3)	27 (27.2)	0.05
Eructation	20 (60.6)	52 (78.8)	72 (72.7)	0.05
Anorexia	16 (48.5)	4 (6.1)	20 (20.2)	<0.001
Asthenia	13 (39.4)	2 (3.0)	15 (15.2)	<0.001
Epigastric tenderness	17 (51.5)	30 (45.5)	47 (47.5)	0.67

**Table 3.** Endoscopic findings.

	CKD (n = 33) N (%)	NRF (n = 66) N (%)	Total (n = 99)	p-value
<b>Normal exam</b>	4 (12.1)	18 (27.3)	22 (22.2)	0.064
<b>Esophagitis</b>	3 (9.1)	0 (0.0)	3 (3.0)	0.051
<b>Erythematous gastritis</b>	14 (42.4)	13 (19.7)	27 (27.3)	0.314
<b>Erosive gastritis</b>	7 (21.2)	1 (1.5)	8 (8.1)	<b>0.009</b>
<b>Gastric ulcer</b>	2 (6.1)	2 (3.0)	4 (4.0)	0.855
<b>Pseudonodular gastritis</b>	1 (3.0)	3 (4.5)	4 (4.0)	0.385
<b>Erythematous bulbitis</b>	3 (9.1)	4 (6.1)	7 (7.1)	0.872
<b>Bulbar ulcer</b>	1 (3.0)	3 (4.5)	4 (4.0)	0.385
<b>Hiatal hernia</b>	4 (12.1)	0 (0.0)	4 (4.0)	<b>0.022</b>
<b>Cardiac incontinence</b>	3 (9.1)	2 (3.0)	5 (5.1)	0.495

and NRF patients was significant with erosive gastritis ( $p = 0.009$ ) and hiatal hernia ( $p = 0.002$ ) (**Table 3**). CKD (aOR = 1.02; CI 95% 0.14 - 0.82;  $p = 0.017$ ) and dialysis (aOR = 10; CI 95% 1.08 - 91.9;  $p = 0.042$ ) were independently associated to *H. pylori* infection (**Table 4**). We had no association between sex, age, endoscopic lesion, duration of dialysis and *H. pylori* infection in CKD patients.

#### 4. Discussion

Upper gastrointestinal disorders are common among CKD patients. Although their etiologies are multifactorial, *H. pylori* infection is a known cause. In addition, there is a discrepancy in the frequency of *H. pylori* infection in CKD patients compared to NRF patients. In this study, we evaluated the prevalence of *H. pylori* infection and gastroduodenal lesions by upper GI endoscopy and a concomitant rapid urease test. Although our sample size was larger than calculated, many patients could not be included in the study because they were treated with proton pump inhibitors in an almost systematic manner; this is a bias in estimating the prevalence of *H. pylori* infection. In addition, the lack of follow-up does not allow us to assess the long-term impact of chronic kidney disease on *H. pylori* infection and the evolution of endoscopic lesions.

The prevalence of *H. pylori* infection was significantly higher in CKD patients than in patients with NRF (63.6% vs 37.9%). Similar findings are reported by Khedmat *et al.* and Nardone *et al.* [28] [29]. We nevertheless found contradictory results in certain studies [14] [15] [16] [20] [21]. The variation in these results may be related to different diagnostic methods, but also to the type of patients chosen. The exact relationship between *H. pylori* infection and CKD is still to be determined. We found no association between age or sex and *H. pylori* infection in CKD, according to some studies [30]. The CKD and dialysis were significantly associated with this infection. Asl *et al.* found high prevalence of *H. pylori* infection in hemodialyzed patient without a significant difference [25]. Low gastric motility, low chloride level, elevated uremia and immunosuppression in CKD patients could be synergistic risk factors for gastric colonization with *H. pylori*. Concerning dialysis, many study suggest that a long time in dialysis can have a protective effect to *H. pylori* infection [31] [32] [33] [34]. Sugimoto *et al.*

**Table 4.** Factors associated to *H. pylori* infection in CKD patients.

Variables	Odd Ratio	Min CI at 95%	Max CI at 95%	p-Value
Dialysis	10.00	1.08	91.98	<b>0.042</b>
Chronic kidney disease	1.02	0.02	0.64	<b>0.017</b>
Duration of dialysis $\leq 4$ years	1.525	0.058	1.907	0.217
Presence of endoscopic lesions	1.90	0.232	15.582	0.550
Age $\leq 55$ years	0.667	0.152	2.926	0.591
Sex male	0.79	0.000	16.163	0.350



reported that being on dialysis, and specifically for less than four years is associated with *H. pylori* infection [23]. Rasmi *et al.* conducted a study with IgG serologic markers and reported that *H. pylori* infection significantly increased with the duration of dialysis [32]. We found no association between *H. pylori* infection and duration of dialysis.

Previous studies have reported multiple upper GI endoscopic lesions in CKD patients which are present in 25% - 75% of these patients [14] [15] [16]. We observed a high prevalence of gastroduodenal lesions in CKD patients is like those found in several studies [27]-[32]. It is slightly higher than that found by Cisse *et al.* in Senegal or Serme *et al.* in Burkina Faso which had respective prevalence's of 76% and 72.9% [35] [36].

The gastroduodenal lesions were frequent in CKD patient more than NRF like Nardone *et al.* studies [28]. In a study on asymptomatic patients from both groups, gastroduodenal lesions were more frequent patients with NRF compared to CKD patients (95% vs 81.5% respectively) [29]. The pathogenesis of upper gastrointestinal mucosal lesions in CKD patients remains undefined, and it is uncertain whether these patients are more prone to have these lesions compared to the normal population. However, there are hypothesis on the role of uremia. Hyper uremia increases hydrogen ions back-diffusion across mucosa. In addition, CKD is associated with a lower clearance of gastrin. Hence, both factors subsequently cause acid hypersecretion which may lead to mucosal injury. Moreover, gastrin decreases pyloric sphincter tone, predisposing to biliary reflux, thus worsening the mucosal injury [37] [38] [39]. In this study we found no significant association between CKD and the type of gastroduodenal lesions. Erythematous gastritis was the commonest gastroduodenal lesion observed in *H. pylori*-infected patients from both groups. This is like the literature [28] [29] [30]. Karari *et al.* demonstrated that duodenal ulcers and duodenal bulb deformities were more frequent in *H. pylori*-infected CKD patients than those with NRF [24]. This is not consistent with our findings. Erosive gastritis and hiatal hernias were more common in the *H. pylori*-infected CKD group. We found no significant association between *H. pylori* infection and the existence of gastroduodenal lesions.

## 5. Conclusion

The prevalence of *H. pylori* infection in CKD patients is higher compared to those with NRF. There was no significant difference in the frequency of gastroduodenal lesions between both groups. Being CKD patients on dialysis contributed to the occurrence of this infection in CKD patients. We need more studies to evaluate the long-term impact of CKD and duration of dialysis on *H. pylori* infection and gastroduodenal lesions.

## Author's Contributions

Writing: Bekolo Nga Winnie, Halle-Ekane Nancy.



Data collection: Bekolo Nga Winnie, Halle-Ekane Nancy.

Corrections: Bekolo Nga Winnie, Eloumou Baganaka Servais, Ndjitoyap Ndam Antonin, Sengha Njapa Guy Roger, Fouda Hermine, Malongue Agnès, Kowo Mathurin, Noah Noah Dominique, Ankouone Andoulo Firmin, Luma Namme Henry, Halle-Ekane Marie Patrice.

## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

## References

- [1] Hooi, J.K.Y., *et al.* (2017) Global Prevalence of *Helicobacter pylori* Infection: Systematic Review and Meta-Analysis. *Gastroenterology*, **153**, 420-429. <https://doi.org/10.1053/j.gastro.2017.04.022>
- [2] Smith, S., Fowora, M. and Pellicano, R. (2019) Infections with *Helicobacter pylori* and Challenges Encountered in Africa. *World Journal of Gastroenterology*, **25**, 3183-3195. <https://doi.org/10.3748/wjg.v25.i25.3183>
- [3] Andoulo, F.A., Noah, D.N., *et al.* (2013) Epidémiologie de l'infection à *Helicobacter pylori* à Yaoundé: De la particularité à l'énigme Africaine. *Pan African Medical Journal*, **16**, Article 115. <https://doi.org/10.11604/pamj.2013.16.115.3007>
- [4] Kayali, S., Manfredi, M., Gaiani, F., Bianchi, L., Bizzarri, B., Leandro, G., Di Mario, F. and De' Angelis, G.L. (2018) *Helicobacter pylori*, Transmission Routes and Recurrence of Infection: State of the Art. *Acta Biomedica*, **89**, 72-76.
- [5] Mabeku, L.B.K., Ngamga, M.L.N. and Leundji, H. (2018) Potential Risk Factors and Prevalence of *Helicobacter pylori* Infection among Adult Patients with Dyspepsia Symptoms in Cameroon. *BMC Infectious Diseases*, **18**, Article No. 278. <https://doi.org/10.1186/s12879-018-3146-1>
- [6] Abadi, A.T.B. (2018) Diagnosis of *Helicobacter pylori* Using Invasive and Noninvasive Approaches. *Journal of Pathogens*, **2018**, Article ID: 9064952. <https://doi.org/10.1155/2018/9064952>
- [7] Dore, M.P. and Pes, G.M. (2021) What Is New in *Helicobacter pylori* Diagnosis. An Overview. *Journal of Clinical Medicine*, **10**, Article 2091. <https://doi.org/10.3390/jcm10102091>
- [8] Marshall, B.J. and Warren, J.R. (1984) Unidentified Curved Bacilli in the Stomach of Patients with Gastritis and Peptic Ulceration. *The Lancet*, **1**, 1311-1315. [https://doi.org/10.1016/S0140-6736\(84\)91816-6](https://doi.org/10.1016/S0140-6736(84)91816-6)
- [9] WHO (2002) IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. *Lyon: International Agency for Research on Cancer*, **82**, Article 601.
- [10] De Korwin, J.-D., Ianiro, G., Gibiino, G. and Gasbarrini, A. (2017) *Helicobacter pylori* Infection and Extragastric Diseases in 2017. *Helicobacter*, **22**, e12411. <https://doi.org/10.1111/hel.12411>
- [11] Lin, S.-Y., Lin, C.-L., Liu, J.-H., Yang, Y.-F., Huang, C.-C. and Kao, C.-H. (2015) Association between *Helicobacter pylori* Infection and the Subsequent Risk of End-Stage Renal Disease: A Nationwide Population-Based Cohort Study. *International Journal of Clinical Practice*, **69**, 604-610. <https://doi.org/10.1111/ijcp.12602>
- [12] Aydogan, T., Ulas, T., Selcoki, Y., Alkan, R., Yilmaz, O.C., Yalcin, K.S., *et al.* (2012)

- Effects of *Helicobacter pylori* Eradication on Proteinuria: A Prospective Study. *Wiener Klinische Wochenschrift*, **124**, 241-244. <https://doi.org/10.1007/s00508-012-0150-0>
- [13] Liu, X.-Z., Zhang, Y.-M., Jia, N.-Y. and Zhang, H. (2020) *Helicobacter pylori* Infection Is Associated with Elevated Galactose-Deficient IgA1 in IgA Nephropathy. *Renal Failure*, **42**, 539-546. <https://doi.org/10.1080/0886022X.2020.1772295>
- [14] Thomas, R., Panackal, C., John, M., Joshi, H., Mathai, S., Kattickaran, J., *et al.* (2013) Gastrointestinal Complications in Patients with Chronic Kidney Disease—A 5-Year Retrospective Study from a Tertiary Referral Center. *Renal Failure*, **35**, 49-55. <https://doi.org/10.3109/0886022X.2012.731998>
- [15] Sotoudehmanesh, R., Asgari, A.A., Ansari, R. and Nouraie, M. (2003) Endoscopic Findings in End-Stage Renal Disease. *Endoscopy*, **35**, 502-505.
- [16] Niknam, R., Barfei, M. and Mahmoudi, L. (2019) *Helicobacter pylori*, Endoscopic, and Histologic Features among Kidney Transplant Candidates in Southern Iran. *Infection and Drug Resistance*, **12**, 3687-3693. <https://doi.org/10.2147/IDR.S228026>
- [17] Wee, A., Kang, J.Y., Ho, M.S., Choong, H.L., Wu, A.Y. and Sutherland, I.H. (1990) Gastroduodenal Mucosa in Uraemia: Endoscopic and Histological Correlation and Prevalence of *Helicobacter*-Like Organisms. *Gut*, **31**, 1093-1096. <https://doi.org/10.1136/gut.31.10.1093>
- [18] Kao, C.-H., Hsu, Y.-H. and Wang, S.-J. (1995) Delayed Gastric Emptying and *Helicobacter pylori* Infection in Patients with Chronic Renal Failure. *European Journal of Nuclear Medicine*, **22**, 1282-1285. <https://doi.org/10.1007/BF00801614>
- [19] Neithercut, W.D., Rowe, P.A., El Nujumi, A.M., Dahill, S. and McColl, K.E. (1993) Effect of *Helicobacter pylori* Infection on Intragastric Urea and Ammonium Concentrations in Patients with Chronic Renal Failure. *Journal of Clinical Pathology*, **46**, 544-547. <https://doi.org/10.1136/jcp.46.6.544>
- [20] Blusiewicz, K., Rydzewska, G. and Rydzewski, A. (2005) Gastric Juice Ammonia and Urea Concentrations and Their Relation to Gastric Mucosa Injury in Patients Maintained on Chronic Hemodialysis. *Roczniki Akademii Medycznej w Białymstoku*, **50**, 188-192.
- [21] Shin, S.P., Bang, C.S., Lee, J.J. and Baik, G.H. (2019) *Helicobacter pylori* Infection in Patients with Chronic Kidney Disease: A Systematic Review and Meta-Analysis. *Gut Liver*, **13**, 628-641. <https://doi.org/10.5009/gnl18517>
- [22] Chang, S.-S. and Hu, H.-Y. (2015) Association between Early *Helicobacter pylori* Eradication and a Lower Risk of Recurrent Complicated Peptic Ulcers in End-Stage Renal Disease Patients. *Medicine (Baltimore)*, **94**, e370. <https://doi.org/10.1097/MD.0000000000000370>
- [23] Sugimoto, M., Sakai, K., Kita, M., Imanishi, J. and Yamaoka, Y. (2009) Prevalence of *Helicobacter pylori* Infection in Long-Term Hemodialysis Patients. *Kidney International*, **75**, 96-103. <https://doi.org/10.1038/ki.2008.508>
- [24] Karari, E.M., Lule, G.N., McLigeyo, S.O. and Amayo, E.O. (2000) Endoscopic Findings and the Prevalence of *Helicobacter pylori* in Chronic Renal Failure Patients with Dyspepsia. *East African Medical Journal*, **77**, 406-409.
- [25] Asl, M.K.H. and Nasri, H. (2009) Prevalence of *Helicobacter pylori* Infection in Maintenance Hemodialysis Patients with Non-ulcer Dyspepsia. *Saudi Journal of Kidney Diseases and Transplantation*, **20**, 223-226.
- [26] Levin, A. and Stevens, P.E. (2014) Summary of KDIGO 2012 CKD Guideline: Behind the Scenes, Need for Guidance, and a Framework for Moving Forward. *Kidney*

- International*, **85**, 49-61. <https://doi.org/10.1038/ki.2013.444>
- [27] Whitley, E. and Ball, J. (2002) Statistics Review 4: Sample Size Calculations. *Critical Care*, **6**, 335-341. <https://doi.org/10.1186/cc1521>
  - [28] Nardone, G., Rocco, A., Fiorillo, M., Del Pezzo, M., Autiero, G., Cuomo, R., *et al.* (2005) Gastroduodenal Lesions and *Helicobacter pylori* Infection in Dyspeptic Patients with and without Chronic Renal Failure. *Helicobacter*, **10**, 53-58. <https://doi.org/10.1111/j.1523-5378.2005.00291.x>
  - [29] Khedmat, H., Ahmadzad-Asl, M., Amini, M., Lessan-Pezeshki, M., Einollahi, B., Pourfarziani, V., *et al.* (2007) Gastro-Duodenal Lesions and *Helicobacter pylori* Infection in Uremic Patients and Renal Transplant Recipients. *Transplantation Proceedings*, **39**, 1003-1007. <https://doi.org/10.1016/j.transproceed.2007.03.034>
  - [30] Abdulrahman, I.S., Al-Mueilo, S.H., Ismail, M.H., Yasawy, M.I., Al-Qahtani, F.N. and Al-Qorain, A.A. (2004) Does *Helicobacter pylori* Infection in Chronic Renal Failure Increase the Risk of Gastroduodenal Lesions? A Prospective Study. *Saudi Journal of Gastroenterology*, **10**, 78-85.
  - [31] Mortazavi, F. and Rafeey, M. (2008) Endoscopic Findings and *Helicobacter pylori* in Children on Long-Term Hemodialysis. *Pakistan Journal of Biological Sciences*, **11**, 1840-1843. <https://doi.org/10.3923/pjbs.2008.1840.1843>
  - [32] Rasmi, Y., Farshid, S. and Makhdomi, K. (2012) Effect of Duration on Hemodialysis on Prevalence of *Helicobacter pylori* Infection. *Saudi Journal of Kidney Diseases and Transplantation*, **23**, Article 489.
  - [33] Chang, S.-S. and Hu, H.-Y. (2014) Lower *Helicobacter pylori* Infection Rate in Chronic Kidney Disease and End-Stage Renal Disease Patients with Peptic Ulcer Disease. *Journal of the Chinese Medical Association*, **77**, 354-359. <https://doi.org/10.1016/j.jcma.2014.04.004>
  - [34] Al-Mueilo, S.H. (2004) Gastroduodenal Lesions and *Helicobacter pylori* Infection in Hemodialysis Patients. *Saudi Medical Journal*, **25**, 1010-1014.
  - [35] Cisse, M.M., Fary, K.E.H., Daouda, D., Mahamat, A.G., Nzambaza, J.D.D., *et al.* (2015) Upper Digestive Endoscopic Lesions in Chronic Kidney Disease (CKD): Experience of a Senegalese Center; About 50 Cases. *Journal of Nephrology and Therapeutics*, **5**, Article 202.
  - [36] Serme, A.K., Lengani, A., Ilboudo, P.D., Sawadogo, N. and Sombie, R. (2003) Les lésions endoscopiques digestives hautes dans l'insuffisance rénale chronique sévère en Afrique noire. *Medecine d'Afrique noire*, **50**, 31-36.
  - [37] Tytgat, G.N. (1994) *Helicobacter pylori*: Recent Developments. *Journal of Gastroenterology*, **29**, 30-33. <https://doi.org/10.3109/00365529409091408>
  - [38] Ryabov, S.I., Ryss, E.S., Prochukhanov, R.A., *et al.* (1980) Present-Day Concepts on Gastric Pathology in Patients with Chronic Renal Failure. *International Urology and Nephrology*, **12**, 189-197. <https://doi.org/10.1007/BF02217135>
  - [39] Tani, N., Harasawa, S., Suzuki, S., *et al.* (1980) Lesions of the Upper Gastrointestinal Tract in Patients with Chronic Renal Failure. *Gastroenterologia Japonica*, **15**, 480-484. <https://doi.org/10.1007/BF02773912>

## **Abbreviations**

CKD: Chronic Kidney Disease;

NRF: Normal Renal Function;

PUD: Peptic Ulcer Disease.