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Case Series

Helicobacter pylori and chronic kidney disease: controversy and possibility

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ABSTRACT

Chronic kidney disease (CKD) is rapidly increasing worldwide, constituting a major public health burden that will stress healthcare systems in all countries, with end-stage renal disease (ESRD) emerging as a growing challenge, particularly in developing nations. The increasing burden of CKD, coupled with limited financial and epidemiological resources in less developed countries, places a severe strain on existing health policies. Hypertensive illness and diabetes mellitus (DM) are significant predisposing risk factors for the development and progression of chronic kidney disease. However, the literature lacks sufficient knowledge about the influence of *Helicobacter pylori* on chronic kidney disease, and while there is controversy surrounding the association of *H. pylori* with DM and hypertension, current knowledge does not support a direct role of *H. pylori* in causing CKD. This prospective case series, conducted in Medina, Saudi Arabia, from October 2023 to October 2024, involved nine patients with varying grades of renal dysfunction associated with the presence of *H. pylori*, which was confirmed through specific tests; colon clearing was performed on all patients. The results indicated a marked improvement in kidney function, highlighted by decreased blood urea and serum creatinine levels. Consequently, colonic *H. pylori* strains could be a hidden contributor to CKD through toxic or immune mechanisms, suggesting that careful attention to colonic health may be crucial for individuals with impaired kidney function associated with *H. pylori* existence.

Keywords: Chronic kidney disease, Colon clear, *Helicobacter pylori*, Haemodialysis

INTRODUCTION

Chronic kidney disease (CKD) is rapidly increasing worldwide constituting major public health burden that will stress the healthcare system in all countries. End-stage renal disease (ESRD) is also a growing challenge particularly in developing countries. In the light of the increasing burden of CKD worldwide, the limited financial and the lack of epidemiological resources in developing and less developed countries would put rather severe strain on the existing health policies.[1-3]

Hypertensive illness and diabetes mellitus (DM) are major predisposing risk factors that can lead to the development and progress of chronic disease of the kidneys. The prevalence of the metabolic syndrome is increasing worldwide in both developing and developed countries. It has been documented to increase the risk of cardiovascular disease and CKD; however, few studies are available in

developing countries. Obesity, high triglycerides, high blood pressure and impaired fasting glucose were significantly associated with an increased prevalence of CKD. As much as experimental and clinical studies have revealed that metabolic syndrome plays an important role in the development of CKD, emerging evidences also suggest that CKD may actually cause metabolic syndrome since the kidney is an important organ of glucose and lipid homeostasis.[4,5]

CKD is a common and costly health problem in Saudi Arabia and in the Middle East. The incidence of CKD is still unknown, and the incidence of the end-stage renal disease (ESRD) is estimated at 100-140 incident cases per million populations in the Middle East countries. Published population-based studies for the incidence and prevalence of CKD and ESRD are still lacking in most of the Middle East countries; this would negatively affect the health planning together with effectiveness of preventive and therapeutic measures in these regions.[6]

Acute kidney injury or insult (AKI) is considered to be a potential reason for developing CKD particularly in less developed or poor countries where the origin of AKI in those disadvantaged populations could be also associated with endemic infections, obstetric problems, poisons, toxins and natural disasters.[7] Acute injury of the kidneys is not necessary to be always traumatic; it could be just totally a toxic or biological insult to the kidneys.

The worldwide prevalence of *Helicobacter pylori* and the flare up of a lot of *H. pylori*-related medical challenges through immune or different unknown reasons have been reported in literature; DM and hypertension (HTN) which are known major predisposing risk factors for CKD lie among these challenges.[4,8] The literature lacks sufficient knowledge about the influence of *H. pylori* in causing chronic disease of the kidney. A definite knowledge about the role of *H. pylori* in leading to CKD is not available in literature; most of the available knowledge mainly entails the risk related to the association of *H. pylori* with chronic renal failure in dialysis patients.[9,10] The aim of this study is the demonstration of a possible influence of *H. pylori* in leading to chronic disease of the kidney.

CASE SERIES

A prospective case series study done in Medina, Saudi Arabia between October 2023 and October 2024. The scientific interest of this study included 9 patients with renal dysfunction (multiple-case study); 4 patients with early kidney dysfunction, 4 patients on border of undergoing dialysis while one patient was early on dialysis. Their age ranged between 31-44 years, and they were investigated for existence of *H. pylori* by specific tests; urea breath test and *H. pylori* fecal antigen.[8] Their blood urea level ranged between 24.6 to 71.8 mg/dl (reference range 5 to 20 mg/dl) while creatinine level was between 4.5-5.9 mg/dl (Reference rage 0.6-1.2 mg/dl) (Table 1). Colon clear for natural eradications of *H. pylori* was employed for them employing the potent natural senna leaves extract purge.[11] The study employed descriptive statistics via SPSS to summarize the standard deviation, and the mean for blood urea nitrogen (BUN) and serum creatinine levels before and after the intervention. Paired t-test was conducted to compare the pre- and post-intervention levels of BUN and serum creatinine within the same patients.

H. pylori fecal antigen test was proved positive in all patients while urea breath test was positive in one patient; the one on dialysis. The four patients with early kidney dysfunction recovered normal kidney function, their blood urea levels were rendered between 9.9 and 16.8 mg/dl while serum creatinine reached between 0.8 and 1.01 mg/dl. The four patients on border of dialysis improved dramatically and were saved from dialysis, their blood urea levels became ranging between 28.1 and 32.6 mg/dl while serum creatinine between 2.2 and 3.8 mg/dl (Table 1). The patient on dialysis also improved markedly with lowering of blood urea and serum creatinine levels, his dialysis sessions were reduced to once weekly instead of three sessions, but he did not complete the study (Figures 1 and 2).

BUN levels

The bar plot shows the mean BUN levels before and after the intervention, with error bars representing the standard deviation. The significant decrease post-intervention is evident (Figure 1).

Creatinine levels

The bar plot displays the mean creatinine levels before and after the intervention, with error bars. A substantial reduction is observed, indicating improved kidney function (Figure 1).

In Figure 2, each line represents an individual patient's values before and after colon clearance.

BUN levels

The lines show a consistent decrease across all patients.

Creatinine levels

Most lines also trend downward, reflecting significant improvements in kidney function.

Table 1: Summary of findings (before and after colon clearance).

S. no.	Age	BUN before (mg/dl)	Creatinine before (mg/dl)	On dialysis	Urea breath test	Fecal antigen	BUN after (mg/dl)	Creatinine after (mg/dl)	BUN % change	Creatinine % change
1	31	26.4	1.5	No	N	P	11.5	0.8	-56.44	-46.67
2	35	33.9	1.9	No	N	P	16.2	0.9	-52.21	-52.63
3	36	60.5	5.1	No	N	P	28.1	2.9	-53.55	-43.14
4	37	24.6	1.4	No	N	P	9.9	1.01	-59.76	-27.86
5	39	62.9	4.9	No	N	P	30.3	2.2	-51.83	-55.10
6	40	31.9	1.7	No	N	P	16.8	0.9	-47.34	-47.06
7	41	71.8	5.9	Yes	P	P	60.3	4.9	-16.00	-16.95
8	41	63.2	5.0	No	N	P	28.4	3.8	-55.06	-24.00
9	44	64.2	4.8	No	N	P	32.6	3.1	-49.22	-35.42

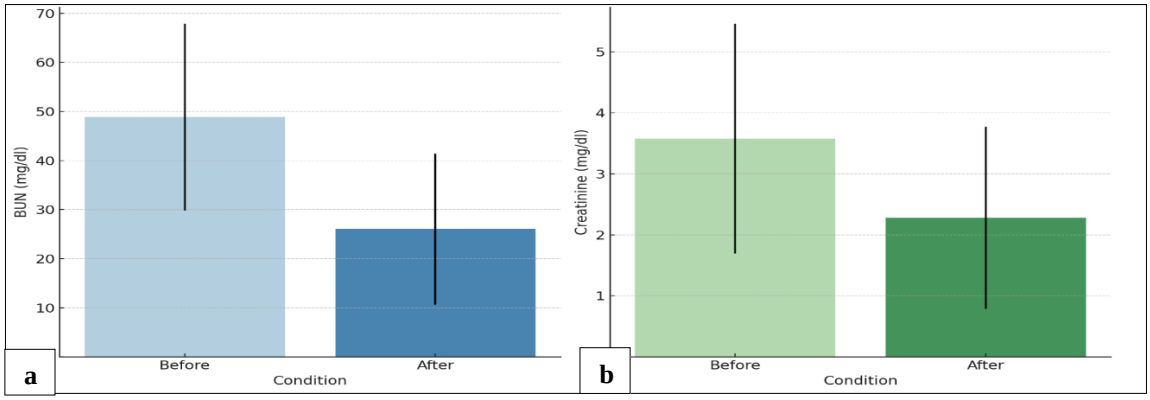

Figure 1: Mean of (a) BUN and (b) creatinine levels before and after intervention.

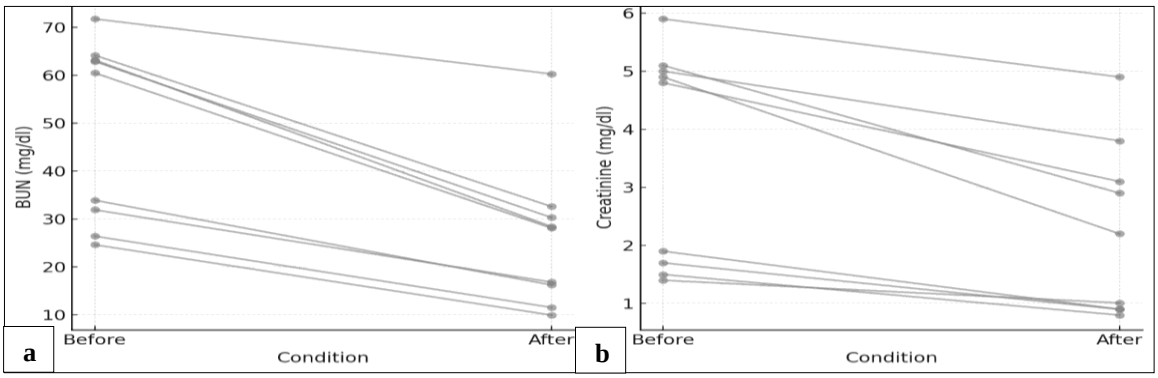

Figure 2: Paired t-test charts for (a) BUN and (b) creatinine levels.

DISCUSSION

H. pylori existence is common among diabetic patients; an illness which is famous to lead to CKD. It is important to elicit in *H. pylori*-associated medical problems which one of these associated conditions is leading to the other or the matter is just a coincidence. Published evidences have been showing that the prevalence of *H. pylori* is higher in patients with type II DM as compared to normal population and it has been illustrated also that *H. pylori* itself is behind the development of DM in many patients.[10,11]

The pathologic toxic reasons which lead to the development of *H. pylori*-associated DM could be the same reasons leading to CKD.

It seems controversial that the association of *H. pylori* with DM, HTN and CKD is common with a possible role of *H. pylori* behind the spread of DM and HTN while the available current knowledge is devoid of any influence of *H. pylori* in causing CKD.[4,5,11] It is unclear whether *H. pylori* is directly associated with progression of renal dysfunction and prognosis of chronic renal failure patients or not. It was reported that the high serum magnesium level in diabetic patients with CKD as well as its higher

concentration in the gastric mucosa might facilitate the colonization of *H. pylori* in the stomach of patients on hemodialysis (HD) but not in patients with various stages of renal failure who are not on HD.[10] On the contrary, some reports showed that the prevalence of *H. pylori* in chronic renal failure patients is significantly lower than in subjects with normal renal function. In particular, the prevalence of *H. pylori* existence decreases in HD patients as dialysis periods progresses.[9] Moreover, it was reported that chronic kidney failure was suggested to have a protective effect against *H. pylori* infection in adults, which does not seem scientifically sound as deterioration of a critical illness might cause progression of an associated chronic illness but not protecting from it.[12] The clue which could have been overlooked here is the mass migration of *H. pylori* to the colon giving an impression of its diminution or absence from the stomach while its massive existence in the colon could constitute a potential source of toxins that could influence toxic nephritis or constitutes a reason of autoimmunity leading to progression of the CKD into parenchymal damage.

H. pylori could travel to the colon possibly because of the stream of food or bowel movement. The bacterium would continue producing ammonia in the colon for a reason or no reason leading to accumulation of profuse toxic amounts of colonic ammonia, un-opposed or buffered by any acidity that could lead to multiple colonic spasms.[8,11] A colonic re-absorptive error could establish with excess retention of fluids, salts and toxins inside the body from the colon with rise of blood urea and serum creatinine giving in this situation a false impression that the kidney function is impaired.[11,14]

H. pylori colonization, once existed in the colon, will remain life-long unless eradicated; accumulation of ammonia in high concentrations is toxic to the body which can lead to toxic nephritis in predisposed disadvantaged population.[8,13]

The key or idea of the study could be emphasized as a toxic insult of the kidneys in the form of toxic nephritis in susceptible patients due to accumulation of ammonia in high toxic levels in the colon together with the toxic effect of raised blood urea due to its undue colonic re-absorption. This acute insult of both kidneys could be further worsened with the development of diabetes and HTN as complications of the existent colonic *H. pylori* strains.[11,14]

In case *H. pylori* has colonized the stomach as its natural habitat, this could signify that the bacterium would be considered a foreign structure to other tissues in the body like the colon constituting a reason for auto-immunity that could lead to a sort of immune nephropathy among susceptible individuals; the matter could end into an established chronic failure of the kidneys or an ESRD.[8]

CKD is a major health burden and the lack of knowledge about a possible influence of *H. pylori* in leading to CKD is per se a motive to illustrate this concept. The worldwide prevalence of *H. pylori* particularly in developing countries and the evolution of a lot of *H. pylori*-related chronic degenerative conditions as DM and HTN is coinciding with the flare up of CKD during the latest decades; this should attract the attention towards a role of *H. pylori* in leading also to CKD. In addition, it has been referred in literature that there is an irrelevant relation between *H. pylori* and metabolic syndrome while it has been confirmed that metabolic syndrome can predispose to CKD; this should motivate the interest to empathize the role of *H. pylori* in leading to CKD either directly or indirectly.[4,5,11,14]

The observation that some patients who were seeking medical assessment for their kidney function, they were advised to start dialysis but when they returned for re-assessment, they were informed that the need for dialysis is not existing anymore. This might indicate an underlying pathologic error possibly in the colon which sometimes improves, and other times goes bad.

A further striking motive for this study is a dramatic improvement which has been confronted in three patients under HD after employing a natural purgative to clear the colon; they needed less frequent sessions of dialysis.

Limitations

The study has several limitations, including a small sample size of only 9 patients, which restricts the statistical power and generalizability of the findings to larger populations. Additionally, the absence of a control group complicates the ability to dismiss other potential factors, such as spontaneous recovery or dietary changes, that may have contributed to the observed improvements. Some patients had short follow-up periods, and notably, the patient on dialysis did not complete the study, hindering the evaluation of long-term outcomes for this subgroup. Furthermore, uncontrolled variables like diet, hydration, and concurrent medications that could affect kidney function were neither specified nor managed during the research.

CONCLUSION

In spite of all controversial reasons, the abnormal-existence colonic *H. pylori* strains could constitute a possible hidden reason behind CKD via toxic or immune influence, therefore; carefulness about the colon together with colon clear whenever necessary could be integral for those susceptible individuals in particular. Colon care together with colon clear could constitute a wonderful dramatic measure that could protect the kidney function and save from dialysis in those patients with impaired kidney function associated with *H. pylori* existence.

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