Review Article

Helicobacter Pylori Causes Genitourinary Diseases: Review of the Literature

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Abstract

Helicobacter pylori (H. pylori) is among the most prevalent bacterial pathogens globally and is well recognized for its role in gastrointestinal disorders. Emerging evidence indicates that its impact may extend beyond the digestive system, with potential associations involving extra-gastric conditions, including genitourinary (GU) diseases. This review seeks to critically evaluate the existing literature on the relationship between H. pylori infection and various GU disorders, and to explore the biological mechanisms that may explain this association.

Accumulating studies suggest a possible link between H. pylori infection and conditions such as male infertility, prostatitis, benign prostatic hyperplasia, erectile dysfunction, and certain urological cancers. The proposed mechanisms include chronic systemic inflammation, immune system alterations, oxidative stress, endothelial dysfunction, and molecular mimicry, which may collectively contribute to GU pathophysiology. Nevertheless, the current body of evidence remains inconsistent, largely due to methodological variability and limited mechanistic investigations. Robust, well-structured epidemiological and clinical studies are required to confirm causality and to determine the clinical relevance of these potential associations.

Key words: *Helicobacter Pylori, Extragastric Diseases, Genitourinary Diseases.*

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Introduction

As a result of research in various dimensions, new evidence is coming up regarding the pathophysiological processes of different diseases in the human body. So, scientists are eagerly doing investigations to find out the pathological causes and a better understanding of disease processes for ideal treatment and prevention. In this regard, scientists found that if one pathogenic bacterium causes some diseases in one site of the body, it may cause some pathology to other parts of the body, even serious diseases, like cancer. As Helicobacter pylori (H. pylori) mainly causes gastric cancer, which is well known, but researchers found that this notorious bacterium is linked to cause multiple diseases elsewhere in the body, other than the gastrointestinal tract.[1] Interest was growing rapidly to find out the relation of H. pylori with extragastric diseases, and it succeeded.

H. pylori is an atypical gram-negative microaerophilic flagellate, helical bacteria mainly colonize in the gastrointestinal (GI) tract and cause gastritis, gastric ulcer, followed by gastric carcinoma and other gastrointestinal diseases.[2-4]

Doing a study on H pylori, researchers could find and hypothesize that H. pylori bacteria could cause extragastric diseases.[5,6] Among the extragastric diseases, few studies were found that infection by H. pylori is significantly related to genitourinary diseases.[7] Due to pathological effects and association of H. pylori with extragastric diseases, and growing evidence of genitourinary (GU) diseases caused by H. pylori . We conducted the review to explore H. pylori and GU diseases by summarizing epidemiological findings and research evidence, and tried to

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explain the mechanism. Current studies have raised interest in further comprehensive research to examine the association of H. pylori infection with renal stone, cystitis, bladder, and prostate carcinoma. In the future, many urogenital diseases like renal calculi, prostate cancer, bladder cancer, infertility, etc., can be prevented by the eradication of H. pylori may become a reality, like what happened in the treatment of peptic ulcer diseases and gastric cancer. In recent years, it has been suggested that the possible role of H. pylori infection may have several extragastric effects, including neurodegenerative, metabolic, and cardiovascular conditions, as well as hepatobiliary, pancreatic, and colorectal diseases.[8,9]

It has been shown that H. pylori positivity in rheumatic arthritis patients shows more clinical manifestations, such as joint pain, muscle pain etc.[10]

Several studies have shown that H. pylori CagA-positive patients develop more severe rheumatoid arthritis than CagA-negative patients. These imply that H. pylori infection could be one of the pathogeneses of rheumatoid arthritis.[11] The study of H. pylori infection in patients with genitourinary diseases is not as advanced as that of other gastric diseases, but great progress has been made in recent decades.

Materials and Methods

We searched Medline, PubMed, PMC, Google, and Web of Science to gather eligible studies up to 2022. A total of 40 studies were collected, and 37 were finally included in this study. Regarding the relationship between H. pylori infection and genitourinary diseases, various studies-including case-control, case reports, cohort, and epidemiological studies that support a potential link-were searched using combined terms such as: H. pylori with urologic diseases, prostatitis, BPH, PCA, male infertility, sperm disorders, sperm quality, infertility, renal calculus, and bladder carcinoma. A total of 25 relevant articles were reviewed to summarize findings related to H. pylori infection in the genitourinary tract.

Helicobacter Pylori infection influences Reproduction H. pylori Infection could be linked to extragastric diseases, such as those related to human reproduction, concerning the decrease in reproductive potential by strains expressing cytotoxin-associated gene A protein(CagA). This infection is more prevalent in infertility disorders. As the researchers found, anti-H. Helicobacter pylori antibodies in cervical mucus and follicular fluid are more common in infected women, which may decrease sperm motility.[12]

These antibodies react immunologically with sperm, hampering oocyte-sperm fertilization. Not all strains of H. pylori have the same virulence. The CagA gene of H. pylori, which is considered a marker for the presence of Cag in the organisms, induces the production of sperm antibodies detectable by simple serological tests.[13] Researchers in their investigations found that sperm motility, vitality, and the number of normal morphological sperm decrease. The increased cytokines, the inflammatory markers such as TNF-alpha, may damage spermatozoa. This is due to elevated inflammatory cytokine levels in infected individuals. Researchers recommended further studies to explore the possible effect of H. pylori infection on human reproduction.[14]

In a Japanese retrospective study with 204 female patients, it was found that women with idiopathic infertility seropositivity for H. pylori infection were twice as high as in patients with one or more known causes of infertility (38.09% vs 20.2% respectively, OR=2.11). Moreover, infected women had H. pylori antibodies significantly higher in the follicular fluid and cervical mucus.13 High levels of antibodies may contribute to infertility. Studies have shown the role of H. pylori in the development of endocrine pathology, such as polycystic ovary syndrome (PCOS), which may influence reproduction.[15]

In the above study, among 150 infertile women, 58 patients were H. pylori positive and 92 were H. pylori negative. They observed that six out of seven seropositive women for CagA had spontaneous abortion, whereas only one out of 16 seronegative for CagA women had spontaneous abortion ($p=0.0004$, OR=90.01, Fisher Exact test). This difference is highly significant. The difference may be due to H. pylori infection.[15] Levels of Ghrelin and Obestatin in human semen were measured in H. pylori -infected men. Ghrelin semen levels were decreased in H. pylori + men compared with uninfected men ($p<0.05$). Semen obestatin concentration was increased, in a non-significant manner, in H. pylori +/CagA+ men. H. pylori infection may influence the ghrelin seminal concentrations, which may be due to the negative effect of infection on semen quality.[16]

In a study with 109 selected patients, 28 subjects were H. pylori +ve, among them, 12 were CagA seropositive(CagA +ve) and 16 were CagA -ve. Of them, 81 men were H. pylori -ve. Serum TNF-alpha and IL-6 concentrations were increased in H. pylori +ve vs H. pylori -ve groups. In comparison to the H. pylori –ve group CagA+ve group showed reduced sperm motility($p<0.05$), more necrosis($p<0.01$), and increased cytokine levels ($p<0.01$). They concluded that CagA+ve H. pylori infection increases semen levels of inflammatory cytokines and reduces sperm motility, contributing to decreased reproduction in men.[17]

Perez-Perez GI et al. found H. pylori -specific IgG antibodies in H. pylori -infected infertile couples of 314. Anti-sperm antibodies, or ASA, are immune system proteins that destroy sperm. It is found in semen, blood, and vaginal fluid. ASA harms fertility by impacting sperm count, motility, and sperm – egg interaction.[18]

All the results of the studies found that men infected with CagA-positive pylori cause sperm alteration in both men and women, due to an increased amount of proinflammatory cytokines found in semen. We believe that an andrologist should consider routinely testing men and women having idiopathic infertility. In a study sperm quality of men infected with H. pylori strains expressing CagA is reduced. Among the patients they studied, 37(42.5%) were seropositive for CagA. They compared sperm motility and vitality among CagA-positive and CagA-negative groups. Sperm motility (18% versus 32%; $p<0.01$) and sperm vitality (35% versus 48%; $p<0.01$) were significantly reduced in positive cases. They also found

that the percentage of sperm with normal forms (18% versus 22%; p<0.05) was significantly reduced in CagA-positive cases were then in the CagA-negative cases.[18]

In another study, 22 out of 223(9.87%) demonstrated H. pylori IgA antibodies in their seminal plasma. After treatment, mean seminal H. pylori IgA levels demonstrated a significant decrease (1.55+/-0.4 vs 0.52+/-0.26,95% confidence interval 0.83-1.21,p=0.001) in infertile asthenozoospermic men with high seminal H. pylori IgA who were subjected to triple drug treatment.[19]

In conclusion, the evidence gathered until now should be taken into consideration for future research aiming to explore the possible role of H. pylori infection on human reproduction.
Prostatitis, Prostate Enlargement, and Prostate Cancer associated with H. pylori infection

Among the prostate diseases are prostatitis, benign prostatic hyperplasia (BPH), and Prostatic Carcinoma (Pca) is the main and most reviewed association of H. pylori with those diseases. One study was done with 100 patients with prostatic diseases. H. pylori DNA was detected in the prostatic tissue of BPH and Pca. Among the 100 patients, 78% BPH, and 19% had Pca. H. pylori DNA was detected in the prostatic tissue of 5 patients by the PCR procedure. Though BPH and Pca are not significantly associated with H. pylori DNA in prostatic tissue. Interestingly, the H. pylori stool antigen test was positive for those 5 patients. The number of patients was not sufficient. This study will inspire researchers to conduct further multicenter studies to find out the association between H. pylori infection and BPH or Pca.[20]

Another prospective case-control study of both anti- Helicobacter pylori IgG and IgA were found in increased amounts in patients with Chronic prostatitis, and treatment of H. pylori infection was effective in the cure. This finding may support the association with chronic prostatitis.[21]

Abdollah et al. did a prospective case-control study with 126 patients. Forty-two patients had chronic prostatitis. All patients studied had H. pylori +ve. In their study serum level of H. pylori IgG and IgA were 9.36+/- 7.45 u/ml and 6. 25 +/-7.29 u/ml respectively, in the control group. Serum level of anti-H. pylori IgG and IgA were 20.94 +/- 16.98 u/ml and 18.63 +/-15.25 u/ml respectively in the patient group. When comparing these two groups, there was a significant increase in the patient group with chronic prostatitis. (p<0.05).[22]

Researchers found that both anti-H. pylori IgG and IgA are increased in patients with chronic prostatitis, and H. pylori treatment decreased the level of IgG and IgA.[22]

The possible way to enter H. pylori into the prostate to cause prostatitis is by entering directly into the prostate or penetrating the stomach and excreting cytokines and interleukins, which would be circulated in the blood and then into the prostate, causing inflammation or reducing body immunity and causing prostatitis.[23]

A pilot study with patients having chronic prostatitis/chronic pelvic pain syndrome was conducted and compared with prostatitis-free subjects as controls. They found that seropositivity for antibody against H. pylori was significantly higher in the chronic prostatitis/ chronic pelvic pain syndrome than in the control group (p<0.05).[24] So, we can say that, in any way, H. pylori infection has a relation with prostatitis; we recommend further study to find out a more authentic relation.

To find out the association between prostatitis, BPH, and prostate carcinoma, a systematic review and meta-analysis were done. In this meta-analysis, 27 studies were included to describe the association between prostatitis and PCa (OR=1.72, 95% CI=1.44-2.06, I2=90.1%, P<0.001). Among them, 21 studies presented significant evidence about the relation between BPH and PCa (OR=2.16, 95% CI=1.75-2.88, I^2=97.1%, P<0.001.[25]

A case-control study and a cohort study both supported that prostatitis could enhance the risk of BPH. They commented that prostatitis or BPH could lead to the risk of PCa. That`s why people with a history of prostatitis might be more vulnerable to BPH.[26]

A study found that both benign prostatic hyperplasia (BPH) and prostatitis are associated with an increased risk of prostate cancer (PCa), with the combined diagnosis of both conditions showing the highest risk.[26]

Eight studies were retained to evaluate the association between BPH and PCa (OR=3.10, 95% CI=2.87-3.35, I^2=8.4%, P=0.365). As for the relation between prostatitis and BPH, a case-control study and a cohort study both supported that prostatitis could enhance the risk of BPH.26 So, we can say that H. pylori infection is potentially related to prostatitis, BPH, and Pca. If the association of H. pylori infection with BPH and PCa have been confirmed to help with the management of those three diseases by the eradication of H. pylori. In those cases, chronic inflammation stimulates different oncogenic signaling pathways and improves the immune system to promote cancer development.[27]

In a study, H. pylori was experimentally introduced through the urethra into the urinary tract, leading to infection and inflammation in the bladder and renal pelvis. Severe neutrophil infiltration was seen in the mucosal layers of both the bladder and pelvis. Also, H. pylori was detected in the epithelial cells. These observations prompted many researchers to conduct studies to find evidence of H. pylori in BPH and prostate carcinoma.[28]

H. pylori and bladder diseases

A report published that about 87% of the patients with interstitial cystitis (IC), Trigonitis of unknown cause, urethral syndrome without bladder infection, tested for H. pylori infec tion and found positive, whereas only about 62% of those diagnosed with bladder infection.[29, 30]

Research shows that a high H. pylori infection rate is linked to low levels of vitamin B12, folate, C, D, and E in gastric juice

and serum. Low levels of vitamin C and E promote increased nitrosamine formation, which, if excreted through urine, could lead to bladder carcinoma development.[31,32] In a prospective cohort study reported that men with a history of gastric ulcer had a 74% Increase in the risk of bladder cancer compared to those with no history of gastric ulcer. These findings should be interpreted with caution.[33]

H. pylori and renal calculus

H. pylori infection is associated with the development of idiopathic hypercalciuric urinary stones, and this may be attributed to one or more of H. pylori 's virulence factors, such as urease.34 It has been proposed that kidney stone formation is a nanobacteria disease, analogous to Peptic ulcer disease associated with H. pylori infection.[35]

A randomized longitudinal case-control study was carried out, and found that H. pylori infection is related to idiopathic hypercalciuric urolithiasis in children. They investigated 100 cases of urolithiasis patients, and 50 cases were without urolithiasis. They tested H. pylori antigen in the stool and urine of 150 subjects. Of 100 patients, 67% were positive for H. pylori , and in 50, only 18% were positive for H. pylori. Statistical analysis showed a highly significant difference between the two groups ($p<0.0001$). They concluded that H. pylori infection is associated with idiopathic hypercalciuric urinary calculus and H. pylori infection may be an etiological factor.[34]

Our review identified and uncovered the role of H. pylori infection in the pathogenesis of urinary calculus diseases. We postulate that H. pylori infection may cause about 90% of renal calculi either directly by providing calcium and hypercalciuria, or indirectly through the production of the urease enzyme. Urease decomposes urea into carbon dioxide and ammonia by the pylori in the stomach. The ammonia may be absorbed in the blood and ultimately goes to the urinary tract, then induces urolithiasis. Ammonia acts to raise the PH of urine, which makes urine alkaline and promotes the formation of struvite calculus. Struvite is a phosphate mineral and crystallizes for stone formation. This urease-mediated urolithiasis requires further confirmation. Kidney stone formation is a nanobacterial disease, like peptic ulcer disease, which is associated with H. pylori infection. Nanobacteria produce carbonate apatite on their cell walls. Data on Randall's plaques suggest that apatite may initiate kidney stone formation. Randall`s plaque is microscopically a plaque of calcium deposited in the interstitial tissue of the renal papilla. This plaque serves as a nidus for urinary stone formation. Both renal stone formation and peptic ulcer disease are due to bacterial infection, and then endogenous and dietary factors may add to the progression of the diseases.35 H. pylori, which has multisystem effects, extends to malignancies, like hematologic and vascular involvement, through well-defined patho genesis. In a study, researchers claimed that their unique data shows a possible relation between H. pylori and renal stone formation. They also think that the renal stone formation is due to the possible systemic influence, such as the vascular endoluminal effect due to the H. pylori infection.[36]

A randomized double-blind placebo-controlled study demonstrated that a high prevalence of H. pylori infection in patients with primary hyperparathyroidism(PHPT) leading to hypercalcemia. This study showed that H. pylori infection was found frequently (85.7%) in patients with PHPT. In the management of PHPT with or without surgery, patients, especially those with dyspeptic symptoms, should be evaluated for H. pylori infection, which can be effectively eradicated by appropriate therapy.[37]

Discussion

H. pylori penetrates the stomach and releases cytokines and interleukins, which circulate in the bloodstream and eventually reach organs. If we can measure the microorganism's antigen by serological test, it will provide more conclusive results. Based on all the data gathered by researchers, it is clear that H. pylori infection causes extragastric disorders, such as genitourinary issues. Reviewing various studies shows that H. pylori infection can cause infertility in both males and females, either directly or indirectly. In males, it influences conditions like prostatitis, BPH, and Pca, although researchers emphasize that more studies with larger patient groups are needed. Multiple research articles indicate that H. pylori infection is involved in urinary calculus and carcinoma, though further research is recommended. Clarifying these connections could help develop more targeted treatment protocols for GU system diseases. Regarding kidney stone formation, evidence suggests that H. pylori infection causes atherosclerotic changes in the renal papillae, leading to calculus plaque formation and ultimately kidney stones. Similarly, H. pylori infection in the stomach can lead to the release of the carcinogenic compound N-nitrosamine into the urine. We cannot dismiss the possibility that this carcinogen might cause bladder carcinoma. Notably, recent research articles highlight the potential role of H. pylori infection in the pathogenesis of genitourinary diseases. We propose the hypothesis that since H. pylori infection induces inflammatory factors, such as IL-2 and TNF-alpha, these factors may lead to chronic inflammation in the genitourinary system, causing infertility, prostate problems, and bladder tumors. H. pylori antigen may cause other host antigen reactions and ultimately diseases. More research with a large sample size and multicenter studies is recommended.

Possible mechanisms of H. pylori-associated infertility

Helicobacter pylori infection may contribute to infertility through several potential mechanisms. Chronic infection can trigger systemic inflammation, leading to increased levels of pro-inflammatory cytokines that may interfere with reproductive hormone regulation and ovulation.[38,39] The bacterium may also induce autoimmune responses, generating antibodies that cross-react with reproductive tissues, impairing fertility.38,40 Additionally, H. pylori-associated oxidative stress can damage gametes (sperm and oocytes) and the endometrium, reducing implantation success (2,3). Some studies also suggest that H. pylori infection may alter gut microbiota and nutrient absorption, indirectly affecting reproductive health.[38,39]

Possible mechanisms of H. pylori-associated cancers

Chronic infection with Helicobacter pylori is a well-established risk factor for gastric carcinoma and some gastric lymphomas. Several mechanisms have been proposed to explain H. pylori-associated carcinogenesis. Persistent infection induces chronic inflammation of the gastric mucosa, leading to continu

ous production of cytokines, reactive oxygen species (ROS), and reactive nitrogen species that damage DNA and promote genetic instability. Bacterial virulence factors such as CagA and VacA interfere with host cell signaling pathways, disrupt epithelial cell junctions, and activate oncogenic pathways including NF-κB, STAT3, and Wnt/β-catenin, resulting in abnormal cell proliferation and malignant transformation. In addition, H. pylori infection causes oxidative stress, DNA damage, and epigenetic alterations, including abnormal DNA methylation and microRNA dysregulation, which further promote tumor development. The infection also alters apoptosis and immune responses within the gastric microenvironment, facilitating progression from chronic gastritis to atrophic gastritis, intestinal metaplasia, dysplasia, and finally gastric cancer.[41-43]

Conclusion

Based on the above findings in different literature reviews, it can be hypothesized that the genitourinary system may be infected by H. pylori and cause genitourinary diseases. Every study has some limitations, but the different findings will stimulate the collaborative work among researchers and urologists; more exploration will be in future work, which is under investigation now. If it is proven clearly that H. pylori can cause genitourinary diseases, prevention of genitourinary cancer can be done by the eradication of H. pylori infection. Further studies are needed. Eradication of H pylori infection may become a reality, as happened in the treatment of peptic ulcer disease and gastric cancer.

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