

Helicobacter pylori and hypertension-a cross-sectional study based on a healthy population

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Abstract

Objective: In this study, we aimed to elucidate the potential association between *H. pylori* infection and the prevalence of hypertension in the Inner Mongolia population, while also exploring the impact of sex and age on the relationship between helicobacter pylori and hypertension.

Method: We collected physical examination data from a cohort of 10,517 patients at the Second Affiliated Hospital of Baotou Medical College between January and December 2020. The patients were categorized into three groups based on their blood pressure and *H. pylori* infection status. Our findings revealed a significant correlation between *H. pylori* infection and blood pressure levels in the patients.

Result: The infection rate of *H. pylori* was higher in patients with hypertension compared to those without hypertension. Additionally, sex and age exerted an influence on the association between helicobacter pylori and hypertension. Among the hypertensive patients, males displayed a greater likelihood of *H. pylori* infection and more severe infections than females. Furthermore, within the

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hypertensive patients infected with *H. pylori*, the degree of infection was lower in young adults compared to middle-aged adults and the elderly.

Conclusion: Hypertension is associated with *Helicobacter pylori* infection.

Keywords

Hypertension, *H. pylori*, sex, age, across-sectional

Introduction

Hypertension, a prevalent cardiovascular disease with a high fatality rate, still lacks a detailed understanding of its pathophysiological mechanism. In recent years, the influence of inflammation on hypertension has gained significant attention.¹ Inflammatory responses not only contribute to the development of hypertension but also increase the risk of other cardiovascular diseases, such as stroke and heart failure.¹ Currently, the prevalence of hypertension in China has reached 23%.² Therefore, it is necessary to study the detailed mechanisms underlying hypertension. The prevalence of *H. pylori* in the Chinese population was higher than that in the Asian population (55.8% vs. 44.7%) probably due to the fecal-oral route of transmission and the common dietary pattern in China³⁻⁶. Recent studies have shown that inflammation is associated with hypertension, which is reminiscent of whether hypertension is associated with *H. pylori* infection, and whether there are differences in the prevalence of *H. pylori* infection in different populations. Numerous researchers have discovered a close association between inflammatory responses caused by intestinal dysbiosis and *H. pylori* infection and hypertension. Dysbiosis of intestinal flora leads to the production of short-chain fatty acids and corticosterone, which promote the development of hypertension.⁷⁻⁹ This avenue may provide insights into the mechanisms of hypertension. *H. pylori* may be associated with sodium retention, vascular endothelial damage, insulin resistance, disorders of lipid metabolism, and altered intestinal flora, all of which potentially contribute to elevated blood pressure and the severity of hypertension.¹⁰⁻¹⁴ A cross-sectional study by Wang et al. found a positive correlation between the prevalence of *H. pylori* infection and hypertension.¹⁵ Xiong et al. further revealed that *H. pylori* infection not only affects the incidence of hypertension but also leads to a higher infection rate in hypertensive individuals compared to those with normal blood pressure.¹⁶ Consequently, we investigated the association between hypertension and *H. pylori* by collecting various indicators from a physically examined population in Baotou City, Inner Mongolia, China. Through this study, the relationship between hypertension and *H. pylori* infection rate was verified, and the differences in different sex and age groups were analyzed, to provide a basis for further research on the correlation and influencing mechanism between hypertension and *H. pylori*. Future studies can refer to this study to exclude relevant influencing factors.

Subjects and methods

Our study was retrospective and in accordance with the 1975 Declaration of Helsinki (revised in 2013). Its reporting conforms to STROBE guidelines.¹⁷

Study design and grouping criteria

We collected data from 10,517 patients who underwent physical examinations at the Second Affiliated Hospital of Baotou Medical College from January to December 2020. All participants were informed of the study's purpose and provided informed consent.¹⁶ (In terms of sample size, we did not calculate the sample size by formula, but all the people who met the sample during this period were included in the study). Inclusion criteria: ¹⁴C breath test, blood pressure measurement, age, and past medical history were recorded. People who took antibiotics and proton pump inhibitors in the past two weeks, and those with serious liver and kidney diseases, serious blood diseases, malignant tumors, and autoimmune diseases were excluded. All patients underwent ¹⁴C breath tests and blood pressure measurements. Based on their blood pressure status, participants were divided into hypertensive and normotensive groups. Within the hypertensive group, individuals were further categorized into those with and without *H. pylori* infection. Sex-based divisions were made for both hypertensive and normotensive populations, and age-based divisions included young, middle-aged, and elderly groups. (We defined the age group between 20 and 40 years as young, 41–65 years as middle-aged, and over 66 years as elderly).

Evaluation of hypertension

Blood pressure measurements were conducted using a reliable electronic sphygmomanometer (automatic sphygmomanometer HPP-9020). Prior to measurement, participants were required to refrain from smoking, drinking tea or coffee, empty their bladder for at least 30 min, and then sit in a quiet environment for at least 5 min to ensure complete relaxation. The average value of three measurements was recorded as the test result.¹⁸ Hypertension was defined as systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg.

Assessment of Helicobacter pylori infection

The presence of *H. pylori* infection was determined by a ¹⁴C breath test in the examined population. Participants were tested on an empty stomach or two hours after eating, and they orally consumed one urea [¹⁴C] capsule. After 15 min, the *H. pylori* tester HUBT-20A was used to measure the breath test results, expressed as 20-min DPM (disintegrations per minute). Results equal to or greater than 100 were considered positive for *H. pylori* infection.

Covariate measurement

During blood pressure measurements, the patient's age, sex, and past medical history were recorded. Designated professional physicians recorded and uploaded the data, simultaneously making corresponding diagnoses. The laboratory department of the hospital employed an automatic blood cell analyzer BC-6800PLUS to measure blood routine examination parameters (white blood cells, red blood cells, neutrophils,

etc.). The Sysmex UF-50 urine sediment analyzer was used to detect urea, uric acid (UA), and creatinine (CREA). Automatic biochemical immunoassay CoBAS 8000 was employed to measure fasting plasma glucose (FPG), triglyceride (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), total bilirubin (TBil), direct bilirubin (DBil), alanine aminotransferase (ALT), aspartate aminotransferase (AST), γ -glutamyltransferase (GGT), globulin (GLb), and albumin/globulin ratio (A/G).

Informed consent was obtained from all participants, who signed the relevant informed consent form. Ethical approval was granted for the study (approved by the Ethics Committee of the Second Affiliated Hospital of Baotou Medical College, LW-014).

Data analysis

All data were analyzed using SPSS version 26.0. Measurement data with a normal distribution were expressed as means $\pm$ standard deviation ($\bar{x} \pm s$), and a t-test was employed for between-group comparisons. For example, urea, creatinine, and other indicators with specific numerical expression. Numerical data were presented as percentages such as sex, urinary protein, *Helicobacter pylori* infection status, and hypertension grade. The chi-square test was used for these indicators. For multi-group comparison, *P* values were derived from one-way ANOVA (continuous variables) or Chi-square test (categorical variables). All tests were two-sided, and *p* values < 0.05 . Graphs were prepared in Graphpad Prism (version 7).

Results

Comparison of various clinical indicators between hypertensive and non-hypertensive populations in Inner Mongolia

A total of 10,517 physical examination subjects were included in this study, with 3335 diagnosed with hypertension, accounting for 33.3% of the examined population (Figure 1). The number of patients infected with *H. pylori* was 2,815, representing 28.1% of the total population. After excluding patients who did not complete all examinations, the remaining sample consisted of 9363 cases. Among them, 3015 had hypertension and 2599 were infected with *H. pylori*. The screened population was divided into hypertensive and normotensive groups, with 2044 patients in the hypertensive group after excluding those with white coat hypertension. Using SPSS, we randomly selected 2044 normotensive patients (without any other diseases) as the control group. The differences in clinical indices between the two groups were analyzed using SPSS. The population was divided into hypertensive and normotensive groups, and the differences in examined population indicators were analyzed. Significant differences were observed between the hypertensive and normotensive populations in terms of age, sex, urea, uric acid (UA), creatinine (CREA), urine protein, fasting blood glucose (FPG), triglycerides (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), total bilirubin (TBil), direct bilirubin (DBil), indirect bilirubin (IBil), alanine aminotransferase (ALT), γ -glutamyltransferase (GGT), globulin (GLB),

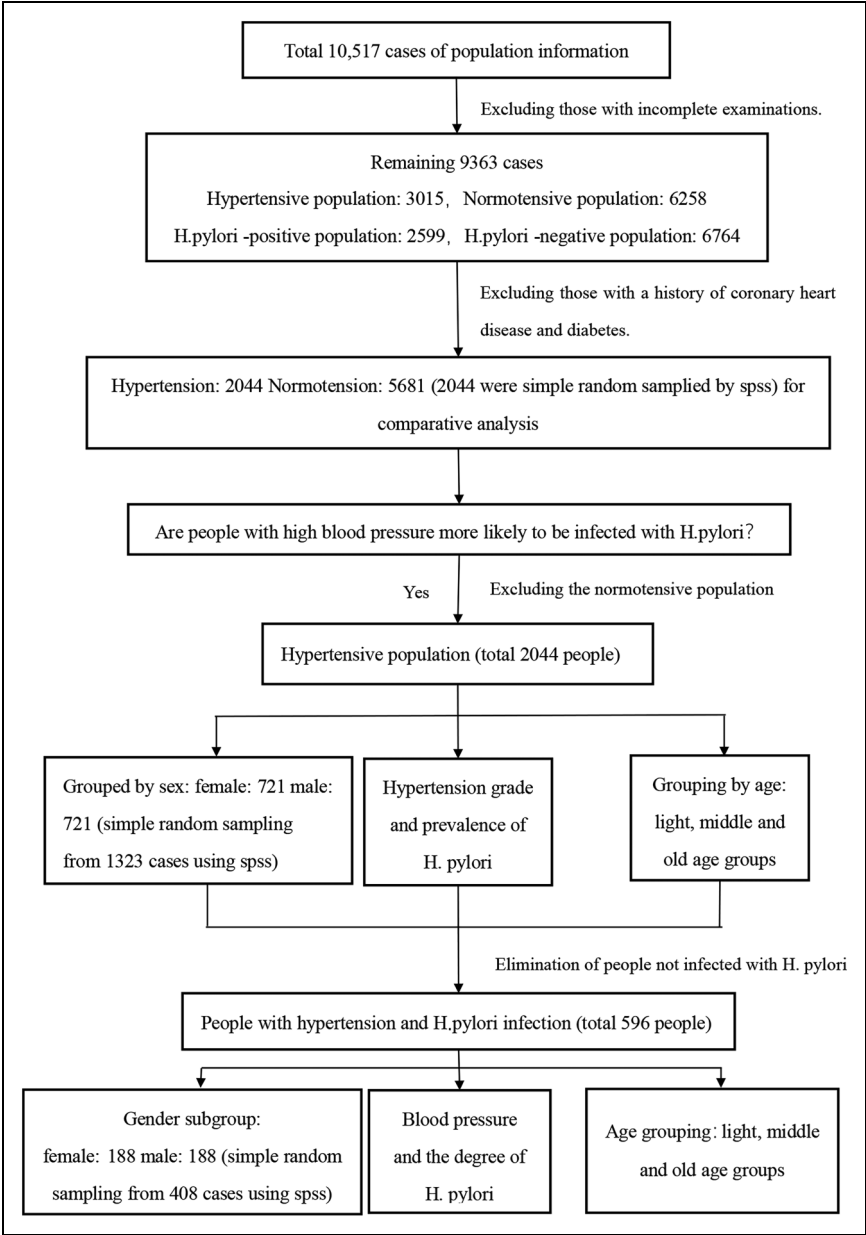


Figure 1. The detailed protocol of category volunteers.

Legend: After the statist analysis of *H. pylori* infection rate and other clinical indicators in the whole population, the relationship between age and sex and *H. pylori* infection rate was analyzed by separating the hypertensive population according to blood pressure. In the hypertensive population, the *H. pylori* co-infection population was sorted out according to the status of *H. pylori* infection, and the effect of age and gender on other clinical indicators was analyzed.

white blood cells (WBC), red blood cells (RBC), hemoglobin (Hb), platelets (PLT), lymphocytes, neutrophils, monocytes, and the infection rate of *H. pylori* (see Table 1). The infection rate of *H. pylori* in the hypertensive group was higher than that in the normal blood pressure group (29.16% vs. 25.44%, $p < 0.05$).

Table 1. Characteristics between the hypertensive group and non-hypertensive group.

Variable	no Hypertension <i>n</i> = 2044	Hypertension <i>n</i> = 2044	t/χ^2	<i>p</i> -value
Age (y)	44.93 ± 12.71	52.52 ± 13.27	-18.675	<0.001
Sex (female)	928(45.40%)	721(35.27%)	43.553	<0.001
Renal function test				
Urea (mmol/L)	4.66 ± 1.20	4.80 ± 1.20	-3.633	<0.001
Crea (μmol/L)	73.05 ± 13.93	74.75 ± 14.65	-3.807	<0.001
Uric acid (μmol/L)	335.56 ± 92.47	360.50 ± 92.75	-8.612	<0.001
Urine protein	86(4.21%)	203(9.93%)	50.970	<0.001
FPG (mmol/L)	5.22 ± 1.08	5.64 ± 1.36	-10.897	<0.001
Blood lipid test				
TG (mmol/L)	1.56 ± 1.01	1.99 ± 1.26	-11.789	<0.001
TC (mmol/L)	4.70 ± 0.91	4.95 ± 0.94	-8.668	<0.001
HDL-C (mmol/L)	1.26 ± 0.28	1.23 ± 0.28	3.537	<0.001
LDL-C (mmol/L)	2.92 ± 0.79	3.08 ± 0.78	-6.821	<0.001
TBil (μmol/L)	14.18 ± 6.07	14.69 ± 5.98	-2.744	0.006
Dbil (μmol/L)	2.02 ± 1.25	2.11 ± 1.16	-2.466	0.014
IBil (μmol/L)	12.15 ± 5.35	12.58 ± 5.30	-2.579	0.010
Liver function test				
ALT (U/L)	22.56 ± 16.81	26.71 ± 21.77	-6.831	<0.001
GGT (U/L)	28.71 ± 30.17	39.77 ± 39.51	-10.062	<0.001
TP(g/L)	75.90 ± 22.19	76.37 ± 21.86	-0.679	0.497
ALB(g/L)	47.33 ± 13.63	46.81 ± 13.23	1.683	0.092
GLB(g/L)	28.27 ± 3.42	28.84 ± 3.67	-5.111	<0.001
A/G	1.74 ± 1.38	1.73 ± 1.76	0.117	0.907
Blood routine examination				
WBC (×10 ⁹ /L)	5.95 ± 1.51	6.30 ± 1.59	-7.129	<0.001
RBC (×10 ⁹ /L)	4.96 ± 0.44	5.06 ± 0.47	-6.524	<0.001
Hb(g/L)	155.33 ± 17.31	160.42 ± 18.86	-8.999	<0.001
PLT (×10 ⁹ /L)	245.01 ± 56.27	240.35 ± 59.27	2.578	0.010
lymphocyte (×10 ⁹ /L)	2.00 ± 0.55	2.09 ± 0.60	-4.855	<0.001
neutrophil (×10 ⁹ /L)	3.46 ± 1.15	3.71 ± 1.26	-6.521	<0.001
monocyte (×10 ⁹ /L)	0.33 ± 1.10	0.35 ± 0.13	-5.309	<0.001
EOS (×10 ⁹ /L)	0.14 ± 0.11	0.14 ± 0.12	-1.093	0.275
B (×10 ⁹ /L)	0.03 ± 0.02	0.03 ± 0.02	-1.923	0.055
hp	520(25.44%)	596(29.16%)	7.119	0.008

Note: Crea: Creatinine; FPG: fasting blood-glucose; TG: triglyceride; TC: total cholesterol; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; TBil: total bilirubin; Dbil: direct bilirubin; IBil: indirect bilirubin; GGT: γ-glutamyltransferase; ALT: alanine aminotransferase; TP: total protein; ALB: albumin; GLB: globulin; A/G: ratio of albumin to globulin; WBC: white blood cell; RBC: red blood cell; Hb: hemoglobin; PLT: platelet; EOS: eosinophilic granulocyte; B: basophilic granulocyte; there is helico differencesori Legethereise some difference bedifferencesrtensive people and the normotensive people in the prevmeane of *H. pylori* infection, m, mean age, sex composition, n and other clinical indicators.

Differences in the prevalence of H. pylori infection between men and women in a hypertensive population. The hypertension group was divided by sex, including 721 females and 1323 males. We selected 721 females as one group and randomly selected 721 males from the 1323 male population. After analysis with SPSS, no statistical difference was found between the two groups, except for hypertension level and A/G. Among the female hypertensive patients, 188 were infected with *H. pylori*, while among the male hypertensive patients, 224 were infected. The prevalence of *H. pylori* infection was higher in male hypertensive patients than in female hypertensive patients (31.07% vs. 26.07%, $p=0.036$) (Figure 2).

Differences in clinical indicators between men and women in a population with hypertension co-infected with H. pylori. Among them, 596 patients had hypertension and *H. pylori* infection. They were divided into groups based on sex, with 188 females and 188 males (randomly selected from a total of 408 cases using SPSS). Statistical analysis showed significant differences in diastolic blood pressure (DBP), urea, creatinine (Crea), uric acid, urine protein, fasting blood glucose (FPG), total cholesterol (TC), HDL-C, total bilirubin (TBil), direct bilirubin (DBil), indirect bilirubin (IBil), γ -glutamyltransferase (GGT), albumin (ALB), globulin (GLB), A/G ratio, white blood cells (WBC), red blood cells (RBC), hemoglobin (Hb), platelets (PLT), neutrophils, monocytes, eosinophils, and the infection degree of *H. pylori*(The degree of *H. pylori* infection was divided into according to the ^{14}C breath test. 1:0-60 2:61-120 3: > 120) between the two groups (Table 2, Figure 3).

Effect of age on the relationship between H. pylori and hypertension

Variation in H. pylori infection rates among young, middle-aged, and elderly patients with hypertension. The hypertensive group was divided into three age groups: young,

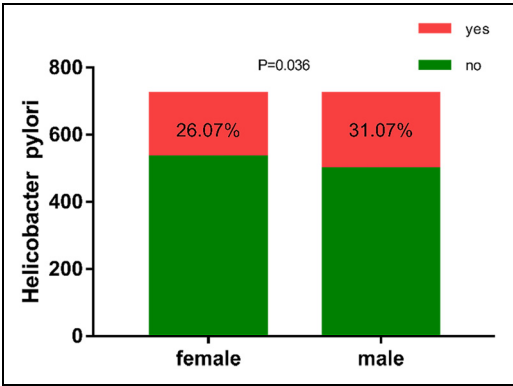


Figure 2. Differences in the prevalence of *Helicobacter pylori* infection in male versus female hypertensive patients.
Legend: In the hypertensive population, the prevalence of *H. pylori* infection was 26.07% in females and 31.07% in males, and the prevalence of *H. pylori* infection in the male population was higher than that in females.

Table 2. Characteristics between males and females in hypertensive combined with *H. pylori* populations.

Variable	Female <i>n</i> = 188	Male <i>n</i> = 188	t/χ^2	<i>p</i> -value
Age(y)	54.39 ± 11.62	52.99 ± 14.21	1.041	0.298
Blood pressure				
Level of hypertension	127(67.55%)	122(64.89%)	3.924	0.141
SBP (mmHg)	151.80 ± 12.85	152.74 ± 13.95	-0.677	0.499
DBP (mmHg)	88.14 ± 11.21	92.78 ± 10.60	-4.128	<0.001
Renal function test				
Urea (mmol/L)	4.58 ± 1.14	5.09 ± 1.24	-4.161	<0.001
Crea (μmol/L)	64.27 ± 10.72	81.47 ± 12.76	-14.150	<0.001
Uric acid(μmol/L)	316.31 ± 87.48	380.95 ± 88.65	-7.115	<0.001
urine protein	9(4.79%)	22(11.70%)	5.941	0.023
FPG (mmol/L)	5.53 ± 1.10	5.87 ± 1.69	-2.307	0.022
Blood lipid test				
TG (mmol/L)	1.81 ± 1.07	2.00 ± 1.32	-1.514	0.131
TC (mmol/L)	5.15 ± 0.92	4.91 ± 0.91	2.527	0.012
HDL-C (mmol/L)	1.33 ± 0.28	1.19 ± 0.25	5.040	<0.001
LDL-C (mmol/L)	3.18 ± 0.78	3.04 ± 0.74	1.830	0.068
TBil (μmol/L)	12.56 ± 4.54	15.20 ± 6.08	-4.788	<0.001
Dbil (μmol/L)	1.61 ± 0.68	2.37 ± 0.98	-8.778	<0.001
IBil (μmol/L)	10.92 ± 4.19	12.83 ± 5.42	-3.810	<0.001
Liver function test				
ALT (U/L)	25.13 ± 23.53	27.94 ± 22.06	-1.192	<0.234
GGT (U/L)	30.74 ± 37.38	46.73 ± 41.85	-3.907	<0.001
TP(g/L)	80.41 ± 51.37	75.15 ± 4.09	1.402	0.163
ALB(g/L)	46.12 ± 3.87	47.19 ± 2.85	-3.043	0.003
GLB(g/L)	30.58 ± 3.73	27.95 ± 3.46	7.069	<0.001
A/G	1.53 ± 0.21	1.80 ± 1.21	-3.037	0.003
Blood routine examination				
WBC (×10 ⁹ /L)	6.22 ± 1.68	6.66 ± 1.57	-2.569	0.011
RBC (×10 ⁹ /L)	4.76 ± 0.33	5.17 ± 0.43	-10.596	<0.001
Hb (g/L)	146.58 ± 13.36	167.19 ± 11.33	-16.134	<0.001
PLT (×10 ⁹ /L)	259.55 ± 60.03	224.03 ± 59.68	5.753	<0.001
lymphocyte (×10 ⁹ /L)	2.11 ± 0.61	2.14 ± 0.62	-0.535	0.593
neutrophil (×10 ⁹ /L)	3.65 ± 1.30	3.95 ± 1.18	-2.319	0.021
monocyte (×10 ⁹ /L)	0.32 ± 0.15	0.37 ± 0.11	-3.675	<0.001
EOS (×10 ⁹ /L)	0.12 ± 0.14	0.15 ± 0.12	-2.409	0.016
B (×10 ⁹ /L)	0.03 ± 0.01	0.03 ± 0.02	-0.948	0.344
hp	1(31.38%)	1(48.40%)	16.660	<0.001

Note: Crea: Creatinine; FPG: fasting blood-glucose; TG: triglyceride; TC: total cholesterol; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; TBil: total bilirubin; Dbil: direct bilirubin; IBil: indirect bilirubin; GGT: γ -glutamyltransferase; ALT: alanine aminotransferase; TP: total protein; ALB: albumin; GLB: globulin; A/G: ratio of albumin to globulin; WBC: white blood cell; RBC: red blood cell; Hb: hemoglobin; PLT: platelet; EOS: eosinophilic granulocyte; B: basophilic granulocyte Hp: helicobacter pylori.

middle-aged, and elderly. The young group consisted of 372 patients, the middle-aged group had 1330 patients, and the elderly group included 342 patients. We randomly selected 342 patients from each group as the final study subjects. Statistical analysis

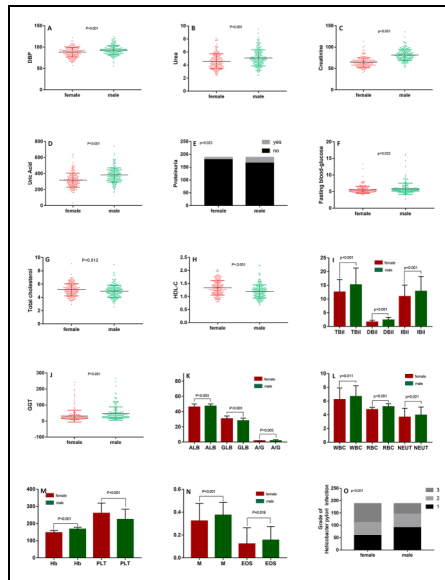


Figure 3. Characteristics between men and women in hypertensive combined with *H. pylori* populations.

Legend: Differences between males and females with co-infected *H. pylori* hypertension in terms of ABCDEFGHIJKLMNOP. [A]: DBP (diastolic blood pressure), for female, the mean $\pm$ SD (standard deviation) is 88.14 ± 11.21 mmHg, and that of male was 92.78 ± 10.60 mmHg, $p < 0.001$. [B]: Urea, The mean $\pm$ SD value of female was 4.58 ± 1.14 mmol/L and that of male was 5.09 ± 1.24 mmol/L, $p < 0.001$. [C]: Crea (creatinine), the mean $\pm$ SD value of female was 64.27 ± 10.72 μ mol/L and that of male was 81.47 ± 12.76 μ mol/L, $p < 0.001$. [D]: Uric acid, the mean $\pm$ SD of female was 316.31 ± 87.48 μ mol/L and that of male was 380.95 ± 88.65 μ mol/L, $p < 0.001$. [E]: urine protein, the positive rate was 4.79% in female and 11.70% in male, $p = 0.023$. [F]: FPG (fasting blood glucose), The mean $\pm$ SD of female was 5.53 ± 1.10 mmol/L and that of male was 5.87 ± 1.69 mmol/L, $p = 0.022$. [G]: TC (total cholesterol), the mean $\pm$ SD of female was 5.15 ± 0.92 mmol/L and that of male was 4.91 ± 0.91 mmol/L, $p = 0.012$. [H]: HDL-C (high-density lipoprotein cholesterol), the mean $\pm$ SD of female was 1.33 ± 0.28 mmol/L and that of male was 1.19 ± 0.25 mmol/L, $p < 0.001$. [I]: TBil (total bilirubin), Dbil (direct bilirubin), IBil (indirect bilirubin), for female, the mean $\pm$ SD were 12.56 ± 4.54 , 1.61 ± 0.68 , 10.92 ± 4.19 , and for male were 15.20 ± 6.08 , 2.37 ± 0.98 , 12.83 ± 5.42 , (μ mol/L) $p < 0.001$, $p < 0.001$, $p < 0.001$. [J]: GGT (γ -glutamyltransferase), the mean $\pm$ SD of female was 30.74 ± 37.38 U/L and that of male was 46.73 ± 41.85 U/L, $p < 0.001$, [K]: ALB (albumin), GLB (globulin), A/G (ratio of albumin to globulin), For female, the mean $\pm$ SD were 46.12 ± 3.87 g/L, 30.58 ± 3.73 g/L, 1.53 ± 0.21 , and for male were 47.19 ± 2.85 g/L, 27.95 ± 3.46 g/L, 1.80 ± 1.21 , $p = 0.003$, $p < 0.001$, $p = 0.003$. [L]: WBC (white blood cell), RBC (red blood cell), The mean $\pm$ SD value of female were $6.22 \pm 1.68 \times 10^9$ /L, $4.76 \pm 0.33 \times 10^9$ /L, and that of male were $6.66 \pm 1.57 \times 10^9$ /L, $5.17 \pm 0.43 \times 10^9$ /L, $p = 0.011$, $p < 0.001$. [M]: Hb (hemoglobin), PLT (platelet), For female, the mean $\pm$ SD were 146.58 ± 13.36 g/L, $259.55 \pm 60.03 \times 10^9$ /L, and for male were 167.19 ± 11.33 g/L, $224.03 \pm 59.68 \times 10^9$ /L, $p < 0.001$, $p < 0.001$. [N]: neutrophil, monocyte, EOS (eosinophilic granulocyte), The mean $\pm$ SD of female were $3.65 \pm 1.30 \times 10^9$ /L, $0.32 \pm 0.15 \times 10^9$ /L, $0.12 \pm 0.14 \times 10^9$ /L, and that of male were $3.95 \pm 1.18 \times 10^9$ /L, $0.37 \pm 0.11 \times 10^9$ /L, $0.15 \pm 0.12 \times 10^9$ /L, $p = 0.021$, $p < 0.001$, $p = 0.016$. [O]: infection degree of *H. pylori* in males and females with co-infected *H. pylori* hypertension. 31.38% of female patients were grade I, and 48.40% of male patients were grade I, $p < 0.001$.

using SPSS showed that among these study subjects, 100 young patients, 98 middle-aged patients, and 109 elderly patients were infected with *H. pylori*, accounting for 29.24%, 28.65%, and 31.87% of the population in each group, respectively. However, there was no statistical difference in the *H. pylori* infection rate (Figure 4).

Clinical indicators in young, middle-aged, and elderly individuals with co-infection of *H. pylori* in the hypertensive population. According to the aforementioned age grouping method, the patients were divided into three categories: young, middle-aged, and old. Among them, there were 110 young patients, 377 middle-aged patients, and 109 elderly patients. After excluding other diseases, we randomly selected 109 patients from each age group the young group, middle-aged group, and elderly group. After statistical analysis, significant differences were observed among the three groups in terms of systolic blood pressure (SBP), DBP, urea, uric acid, triglycerides (TG), alanine aminotransferase (ALT), γ -glutamyltransferase (GGT), albumin (ALB), globulin (GLB), A/G ratio, WBC, RBC, Hb, PLT, lymphocytes, neutrophils, monocytes, basophilic granulocytes, and the degree of *H. pylori* infection (Table 3). The expression levels of urea, ALT, ALB, RBC, Hb, and PLT differed significantly between the young and middle-aged groups, middle-aged and elderly groups, and young and middle-aged groups ($p < 0.05$). Differences in SBP, DBP, GGT, GLB, and A/G ratio were mainly observed between the young and elderly groups and between the middle-aged and elderly groups ($p < 0.05$). Uric acid, TG, WBC, lymphocytes, neutrophils, basophilic granulocytes, and monocytes showed significant differences between the young and middle-aged groups and between the young and elderly groups ($p < 0.05$). The difference in monocyte levels was only observed between the young and middle-aged groups ($p = 0.009$). Regarding *H. pylori* test results, the infection level was lower in the young population compared to the middle-aged population (196.39 ± 91.34 vs. 239.49 ± 125.16 , $p <$

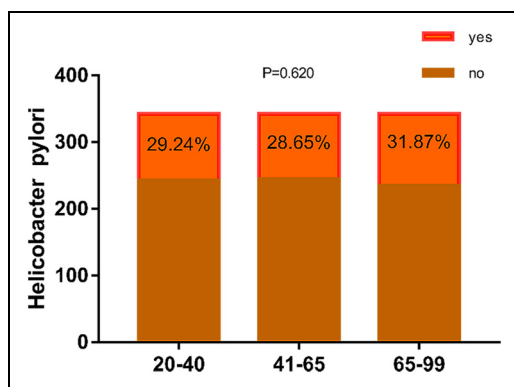


Figure 4. Prevalence of *H. pylori* infection in young, middle-aged, and elderly people. Legend: In the hypertensive population, the highest rate of *H. pylori* infection was 37.87% in those aged 65–99 years, followed by 29.24% in those aged 20–40 years and the lowest rate of 28.65% in those aged 41–65 years, but this difference was not statistically significant.

Table 3. Description of the characteristics of people of different ages with hypertension combined with *H. pylori*.

Variable	20–40 n = 109	41–65 n = 109	66–99 n = 109	F/ χ^2	p-value
Sex (female)	21(19.27%)	36(35.78%)	33(30.28%)	5.795	0.055
Blood pressure					
Level of hypertension	75(68.81%)	75(68.81%)	63(57.80%)	4.282	0.369
SBP (mmHg)	147.82 ± 11.22	149.12 ± 11.90	157.80 ± 13.43	21.499	<0.001
DBP (mmHg)	94.21 ± 9.83	92.49 ± 10.10	85.69 ± 11.26	20.410	<0.001
Renal function test					
Urea (mmol/L)	4.55 ± 1.12	4.87 ± 1.21	5.22 ± 1.25	8.775	<0.001
Crea (μmol/L)	76.95 ± 12.76	75.28 ± 13.38	78.03 ± 16.37	1.024	0.360
Uric acid (μmol/L)	412.20 ± 99.87	353.16 ± 91.46	333.51 ± 72.51	23.245	<0.001
Urine protein	14(12.84%)	15(13.76%)	17(15.60%)	0.354	0.838
FPG (mmol/L)	5.53 ± 1.37	5.66 ± 1.22	5.91 ± 1.48	2.160	0.117
Blood lipid test					
TG (mmol/L)	2.44 ± 1.78	1.83 ± 1.23	1.65 ± 0.97	9.805	<0.001
TC (mmol/L)	4.94 ± 0.90	4.90 ± 0.84	4.91 ± 0.92	0.055	0.946
HDL-C (mmol/L)	1.17 ± 0.29	1.24 ± 0.23	1.25 ± 0.25	2.558	0.079
LDL-C (mmol/L)	3.06 ± 0.75	3.02 ± 0.70	3.18 ± 0.76	1.528	0.218
TBil (μmol/L)	13.99 ± 5.29	14.60 ± 5.69	14.59 ± 6.14	0.406	0.666
Dbil (μmol/L)	2.14 ± 0.77	2.13 ± 0.90	2.25 ± 1.12	0.533	0.588
IBil (μmol/L)	11.85 ± 4.79	12.47 ± 5.01	12.33 ± 5.27	0.455	0.635
Liver function test					
ALT (U/L)	44.19 ± 35.80	28.18 ± 21.74	18.38 ± 9.31	30.160	<0.001
GGT (U/L)	58.20 ± 51.18	45.77 ± 46.82	28.48 ± 20.01	13.982	<0.001
TP(g/L)	76.90 ± 4.18	75.63 ± 4.35	75.89 ± 3.82	2.900	0.056
ALB(g/L)	48.06 ± 2.55	46.61 ± 3.40	45.60 ± 2.87	18.972	<0.001
GLB(g/L)	28.84 ± 3.81	28.81 ± 3.84	30.26 ± 3.68	5.245	0.006
A/G	1.82 ± 1.30	1.65 ± 0.25	1.53 ± 0.24	3.832	0.023
Blood routine examination					
WBC (×10 ⁹ /L)	7.28 ± 1.98	6.36 ± 1.55	6.32 ± 1.46	11.303	<0.001
RBC (×10 ⁹ /L)	5.25 ± 0.43	5.05 ± 0.47	4.89 ± 0.44	17.558	<0.001
Hb (g/L)	165.91 ± 11.25	161.40 ± 14.80	156.90 ± 14.34	12.038	<0.001
PLT (×10 ⁹ /L)	257.45 ± 58.71	236.15 ± 55.81	213.64 ± 61.48	15.173	<0.001
lymphocyte (×10 ⁹ /L)	2.43 ± 0.65	2.16 ± 0.62	2.04 ± 0.60	11.369	<0.001
neutrophil (×10 ⁹ /L)	4.26 ± 1.47	3.69 ± 1.24	3.75 ± 1.14	6.418	0.002
monocyte (×10 ⁹ /L)	0.39 ± 0.12	0.35 ± 0.12	0.36 ± 0.11	3.729	0.025
EOS (×10 ⁹ /L)	0.16 ± 0.11	0.14 ± 0.11	0.13 ± 0.11	1.933	0.146
B(×10 ⁹ /L)	0.04 ± 0.02	0.03 ± 0.02	0.03 ± 0.02	3.122	0.045
hp	196.39 ± 91.34	239.49 ± 125.16	273.84 ± 157.93	10.062	<0.001

Note: Crea: Creatinine; FPG: fasting blood-glucose; TG: triglyceride; TC: total cholesterol; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; TBil: total bilirubin; Dbil: direct bilirubin; IBil: indirect bilirubin; GGT: γ -glutamyltransferase; ALT: alanine aminotransferase; TP: total protein; ALB: albumin; GLB: globulin; A/G: ratio of albumin to globulin; WBC: white blood cell; RBC: red blood cell; Hb: hemoglobin; PLT: platelet; EOS: eosinophilic granulocyte; B: basophilic granulocyte; Hp: helicobacter pylori.

0.01), and also lower in the young group compared to the elderly group (196.39 ± 91.34 vs. 273.84 ± 157.93 , $p < 0.001$).

Discussion

Through this cross-sectional study, we have discovered a significant association between *H. pylori* infection and hypertension. Patients with *H. pylori* infection exhibited higher systolic blood pressure (SBP), diastolic blood pressure (DBP), and a higher prevalence of hypertension compared to those without *H. pylori* infection. Furthermore, we observed differences in the prevalence and severity of *H. pylori* infection between males and females, with males showing higher rates and more severe infections. Age also played a role, with higher *H. pylori* infection rates in the elderly and middle-aged patients compared to young patients.

The overall incidence of *H. pylori* infection in our study was 28.1%, which is lower than in other studies. For instance, a cross-sectional study in Wuhan, China, reported an *H. pylori* infection rate of 32.6% among healthy people.¹⁴ The prevalence of *H. pylori* infection is influenced by regional factors and is strongly associated with family size, education level, lifestyle habits, and income level.¹⁹ Our analysis revealed a higher prevalence of *H. pylori* infection in the hypertensive population compared to the normotensive population (29.16% vs. 25.44%). This finding aligns with the results of a previous study.²⁰ However, early studies on *H. pylori* and metabolic syndrome did not find a significant association with blood pressure.²¹ It is worth noting that these studies used serological indicators to determine *H. pylori* infection, which may have lower sensitivity and specificity compared to the urea breath test (UBT), currently recognized as the gold standard for *H. pylori* detection.²² Significant differences were observed in age and sex between the hypertensive and normal blood pressure groups. Data from the National Health and Nutrition Examination Survey also indicate sex and age differences in hypertension control.²³

We also identified differences in the prevalence of *H. pylori* infection between males and females. The infection rate and severity of *H. pylori* were higher in males than in females. This finding is consistent with the studies conducted by Sun and Chen, which also reported a sex-related association with *H. pylori* infection.^{24,25} Males and females exhibit variations in intragastric acidity and plasma gastrin concentration profiles, which may influence *H. pylori* infection.²⁶ Xiong and other researchers found a correlation between *H. pylori* infection and elevated blood pressure.¹⁶ Upon *H. pylori* infection, the body produces proteins involved in cytotoxic immune responses, leading to the generation of *H. pylori* antibodies. These antibodies cross-react with antigens on blood vessel walls, triggering the release of inflammatory factors that stimulate vascular walls. This immune response affects microvascular systolic and diastolic functions, with the release of IL-8 activating norepinephrine. Disturbed immune responses to *H. pylori* infection result in vasoconstriction and endothelial dysfunction, impacting blood pressure regulation.^{27,28} The expression of IL-8 varies between sex, often exhibiting a protective effect in women but a risk factor in men.²⁹ This may explain the higher *H. pylori* infection rate and severity observed in males. Further analysis of clinical indicators in hypertensive patients with *H. pylori* infection revealed that females had higher levels of high-density

lipoprotein cholesterol (HDL-C) compared to males. This may be attributed to the influence of *H. pylori* on lipid profiles, which may vary by sex.³⁰

Among hypertensive patients with *H. pylori* infection, age played a significant role in the severity of infection. The degree of *H. pylori* infection was lowest in young individuals (<40 years old) compared to middle-aged and elderly individuals. Wenjun and other scholars found that people over 40 years of age in China are more likely to develop homocysteinemia,³¹ a condition in which *H. pylori* infection may play a role. *H. pylori* infection may decrease the absorption of folic acid and vitamin B12, leading to increased levels of homocysteine (Hcy). Elevated Hcy levels damage the vascular endothelium and stimulate collagen fiber synthesis, smooth muscle cell proliferation, and elastic fiber degradation in the vascular wall, resulting in vascular remodeling. Additionally, Hcy inhibits nitric oxide secretion by endothelial cells, leading to platelet aggregation and vasoconstriction. Endothelial damage also increases the levels of cyclooxygenase-1 (COX-1) and cyclooxygenase-2 (COX-2), promoting prostaglandin production, platelet aggregation, and the development of ischemic heart disease.^{16,28,32} However, the rate of *H. pylori* infection in hypertensive individuals did not show a significant association with age. Some studies have reported that *H. pylori* infection rates increase with age up to a certain point (around 36 years old), after which the prevalence does not vary significantly with age.¹⁹

While our study had a large sample size and comprehensive statistical analysis, establishing a clear causal relationship between *H. pylori* and hypertension requires further experiments. Our study did not include the height, weight, and lifestyle habits of the study population, which have been shown in previous studies to be associated with *H. pylori* infection. For instance, studies have found that individuals with *H. pylori* infection tend to have higher body mass index (BMI) compared to those without *H. pylori* infection.^{33–35} In this study, we found that there were significant differences in age, sex, Renal function test, FPG, Blood lipid test, Liver function test, and Blood routine examination between hypertensive and non-hypertensive people. We did not examine the possible reasons for these differences or whether these factors also affect the rate of *H. pylori* infection, which is a limitation of our study. In addition, our study had limitations in the diagnostic criteria for hypertension and *H. pylori* infection. Diagnostic criteria for hypertension are recommended that 2–3 visits be made to confirm hypertension in patients. Since this study was a retrospective study and the study population was a physical examination population, the study subjects could not be required to visit the doctor 2–3 times. Although ¹⁴C breath tests are currently used as the clinical standard for *Helicobacter pylori* infection, according to the studies of other scholars, compared with the gold standard examinations such as histology, ¹⁴C breath tests have a certain degree of false negative, and some patients may be infected with *Helicobacter pylori* but this test is negative.^{36,37} These errors will inevitably affect our study, which is the weakness of this study. The correlation between *H. pylori* infection and alcohol consumption and smoking in daily life remains debated among scholars. Some studies suggest that moderate alcohol consumption may reduce *H. pylori* infection,^{38,39} while others conclude that alcohol can damage the gastric mucosa or enhance *H. pylori* adherence, promoting infection. The relationship between alcohol consumption, smoking, and *H. pylori* infection may also vary by sex, with both alcohol consumption and smoking being risk factors

for males but not females.^{40,41} Further research and analysis are needed to clarify the correlation between *H. pylori* infection and alcohol consumption and smoking, which were not addressed in our study. In conclusion, this study provides a foundation for conducting further research on the association and mechanism of influence between hypertension and *H. pylori*. The current study may be used as a reference for future studies to exclude relevant influencing factors.

Conclusion

This study confirms the association between *H. pylori* infection and hypertension. Among individuals with established hypertension, males are more likely to be infected with *H. pylori* and exhibit more severe infections. Additionally, *H. pylori* infection is less prevalent in younger individuals. Therefore, male middle-aged and elderly patients with hypertension and *H. pylori* infection should receive increased attention and timely treatment.

Summary: This study adds to the existing knowledge by establishing a significant association between *H. pylori* infection and hypertension. It highlights the sex and age differences in *H. pylori* infection rates and severity among hypertensive individuals. The study also emphasizes the need for further research to explore the causal relationship between *H. pylori* and hypertension, as well as the impact of lifestyle factors on *H. pylori* infection.

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Author contribution

This study was proposed by Guanhua Huang who was responsible for the overall coordination, with Ping Yang in charge of paper writing, We Shi in charge of statistical guidance, Chi Wang in charge of research scheme design, Hai Liu in charge of data collation, Xianbin Wang and Zengshuai Wang in charge of data collection, and Guanrong Huang in charge of results analysis.

Consent

We hereby declare by signing below that this manuscript is original, has not been published before, and is not currently under consideration for publication elsewhere. We confirm that the manuscript has been read and approved by all named authors, and that there are no others who meet the criteria for authorship but are not listed. We further confirm that the order of authorship as listed in the manuscript has been approved by all of us.

Data availability statement

Data is available on request from the authors.

Declaration of conflicting interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical considerations and consent

This study was approved by the Ethics Committee of the Second Affiliated Hospital of Baotou Medical College, No. LW-014, on January 4, 2020.

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Patient consent

Consent for this study has been obtained from all participants. Our study subjects are adults who take part in the health examination, and all of them have signed the relevant informed consent form.

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Supplemental material

Supplemental material for this article is available online.

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