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Reassessing the SIBO-Hypertension Link in Symptomatic Patients

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Background: Recent evidence suggests small intestinal bacterial overgrowth (SIBO) may serve as a diagnostic marker for hypertension through gut-cardiovascular axis mechanisms. However, this relationship has not been evaluated in symptomatic gastrointestinal patients. We aimed to assess the association between SIBO and arterial hypertension in patients undergoing clinical evaluation for gastrointestinal symptoms.
Methods: Retrospective cross-sectional analysis of 331 consecutive symptomatic patients (263 controls, 68 with hypertension) who underwent hydrogen breath testing. SIBO was diagnosed using North American Consensus criteria (H_2 rise ≥20 ppm within 90 minutes). Hypertension was established from medical records and medication use. Stratified analysis and Mantel-Haenszel methods assessed confounding and effect modification by age and sex.
Results: SIBO prevalence was similar between groups (50.2% controls vs 45.6% hypertension, p=0.497). Among SIBO-positive patients, 19.0% had hypertension compared to 22.0% of SIBO-negative patients (OR=0.83, 95% CI: 0.49-1.42). Notably, a sex-specific pattern was observed, though interaction testing did not reach statistical significance (p=0.221): women with SIBO showed lower odds of hypertension (OR=0.47, 95% CI: 0.22-1.01, p=0.053) while men showed higher odds (OR=1.63, 95% CI: 0.75-3.54, p=0.22). This sex difference persisted despite women with hypertension being older than men with hypertension (61.6 vs 55.6 years).
Conclusions: Unlike recent reports, we found no overall association between SIBO and hypertension in symptomatic gastrointestinal patients after controlling for age confounding. However, sex-specific trends were observed that did not reach statistical significance and should be considered hypothesis-generating, with women showing protection and men showing increased risk. These findings suggest gut-cardiovascular relationships may differ fundamentally between sexes and emphasize the importance of considering population characteristics and effect modification in microbiome research.

Key words: small intestinal bacterial overgrowth, arterial hypertension, gut-cardiovascular axis, sex differences, effect modification, breath test.

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Přehodnocení souvislosti mezi SIBO a hypertenzí u symptomatických pacientů

Úvod: Nedávné důkazy naznačují, že přerůstání bakterií v tenkém střevě (SIBO) může prostřednictvím mechanismů osy střevo–kardiovaskulární systém sloužit jako diagnostický marker hypertenze. Tento vztah však nebyl hodnocen u pacientů se symptomatickými gastrointestinálními obtížemi. Cílem bylo posoudit souvislost mezi SIBO a arteriální hypertenzí u pacientů podstupujících klinické vyšetření pro gastrointestinální symptomy.

Metody: Retrospektivní průřezová analýza 331 po sobě jdoucích symptomatických pacientů (263 kontrol, 68 s hypertenzí), kteří podstoupili vodíkový dechový test. SIBO bylo diagnostikováno podle kritérií North American Consensus (vzestup H_2 o ≥20 ppm během 90 minut). Hypertenze byla stanovena na základě zdravotnické dokumentace a užívání antihypertenzní medikace. Stratifikovaná analýza a Mantel-Haenszelovy metody hodnotily vliv matoucích faktorů a modifikaci účinku věkem a pohlavím.

Výsledky: Prevalence SIBO byla v obou skupinách podobná (50,2 % u kontrol vs. 45,6 % u hypertoniků, p = 0,497). Mezi pacienty pozitivními na SIBO mělo hypertenzi 19,0 % ve srovnání s 22,0 % pacientů negativních na SIBO (OR = 0,83; 95% CI: 0,49–1,42). Zajímavé je, že byl pozorován vzorec specifický pro pohlaví, ačkoli test interakce nedosáhl statistické významnosti (p = 0,221): ženy se SIBO vykazovaly nižší pravděpodobnost hypertenze (OR = 0,47; 95% CI: 0,22–1,01; p = 0,053), zatímco muži vykazovali vyšší pravděpodobnost (OR = 1,63; 95% CI: 0,75–3,54; p = 0,22). Tento rozdíl mezi pohlavími přetrvával navzdory tomu, že ženy s hypertenzí byly starší než muži s hypertenzí (61,6 vs. 55,6 roku).

Závěry: Na rozdíl od nedávných zpráv jsme po zohlednění matoucího vlivu věku nezjistili žádnou celkovou souvislost mezi SIBO a hypertenzí u symptomatických gastrointestinálních pacientů. Byly však pozorovány trendy specifické pro pohlaví, které nedosáhly statistické významnosti a měly by být chápány jako podklad pro formulaci hypotéz: u žen se jevil ochranný efekt, zatímco u mužů zvýšené riziko. Tato zjištění naznačují, že vztahy mezi střevem a kardiovaskulárním systémem se mohou mezi pohlavími zásadně lišit, a zdůrazňují význam zohlednění charakteristik populace a modifikace účinku ve výzkumu mikrobiomu.

Klíčová slova: přerůstání bakterií v tenkém střevě, arteriální hypertenze, osa střevo–kardiovaskulární systém, rozdíly mezi pohlavími, modifikace účinku, dechový test.

Introduction

Small intestinal bacterial overgrowth (SIBO) syndrome is defined as an abnormal increase in the number and/or change in the type of bacteria in the small intestine, with values exceeding 10^3 CFU/ml (1). The condition has gained attention as a potential contributor to various systemic diseases through mechanisms involving bacterial translocation, metabolic endotoxemia, and altered production of microbial metabolites (2).

A recently published study by Lu et al. (2025) reports a positive association between SIBO and hypertension, with the authors proposing SIBO as a potential diagnostic marker for hypertension (3). This finding adds to the growing body of evidence suggesting that the gut microbiota plays a role in cardiovascular disease pathogenesis through the gut-cardiovascular axis (4, 5).

The gut-cardiovascular axis represents complex bidirectional communication between the gastrointestinal tract and cardiovascular system, mediated through neural, hormonal, and immunological pathways (4, 5). Proposed mechanisms linking SIBO to hypertension include increased production of trimethylamine-N-oxide (TMAO), lipopolysaccharide-induced systemic inflammation, and alterations in short-chain fatty acid production affecting blood pressure regulation (6, 7). Despite this biological plausibility, the causal relationship between SIBO and hypertension remains controversial, especially in patients with pre-existing gastrointestinal symptoms.

The aim of our study was to evaluate the association between SIBO and arterial hypertension in symptomatic patients undergoing gastrointestinal examination, with emphasis on controlling for potential confounding factors including age and sex.

Materials and Methods

Study Design and Population

We conducted a retrospective cross-sectional study including 331 consecutive patients examined at a gastroenterology outpatient clinic from January 2022 till May 2025. The control group (n=263) consisted of symptomatic gastrointestinal patients without any cardiovascular disease diagnosis. The hypertension group (n=68) comprised patients with documented arterial hypertension who also presented with gastrointestinal symptoms. Both groups underwent SIBO testing for clinical indications including abdominal pain, bloating, diarrhea, or constipation

All patients underwent SIBO testing for gastrointestinal symptoms including abdominal pain, bloating, diarrhea, or constipation. Exclusion criteria included: (1) use of antibiotics within 4 weeks prior to testing; (2) use of probiotics within 2 weeks prior to testing; (3) history of gastrointestinal surgery affecting small bowel anatomy; (4) known inflammatory bowel disease; and (5) incomplete medical records.

SIBO Diagnosis

SIBO was diagnosed using the hydrogen breath test according to North American Consensus criteria (8). A rise in hydrogen (H_2) ≥20 ppm from baseline within 90 minutes was considered a positive test. The test protocol was as follows:

1. Patients fasted overnight for a minimum of 12 hours
2. Patients followed a low-fermentation diet 24 hours before testing
3. Smoking was prohibited on the test day
4. Lactulose substrate (10 g dissolved in 200 ml water) was administered
5. Breath samples were collected at baseline and every 15 minutes for 120 minutes
6. Samples were analyzed using a gas chromatograph

Hypertension Definition

Arterial hypertension was established based on: (1) documented history of hypertension in medical records; or (2) current use of antihypertensive medication, including angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, calcium channel blockers, aldosterone antagonists, alpha-blockers, centrally acting antihypertensives, vasodilating beta-blockers, or thiazide and thiazide-like diuretics. Only patients with a diagnosis of primary (essential) hypertension were included; patients with documented secondary hypertension were excluded. This approach reflects real-world clinical practice where hypertension diagnosis is often based on historical documentation rather than contemporaneous blood pressure measurements. The inherent risk of misclassification associated with this approach is discussed in the limitations section.

Statistical Analysis

Categorical variables were compared using the chi-squared test, and continuous variables using the independent samples t-test. For confounding adjustment, we employed:

1. Stratified analysis by age (<40, 40-49, 50-59, ≥60 years) and sex
2. Mantel-Haenszel method for calculating adjusted odds ratios
3. Logistic regression for multivariate analysis

Statistical significance was set at p<0. 05. All analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA) and JavaScript/Math.js for verification.

Ethical Considerations

The study was approved by the institutional ethics committee. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki (1975, revised in 2013). As this was a retrospective study using anonymized data, individual informed consent was waived by the ethics committee.

Results

Population Characteristics

The study included 331 patients, comprising 263 controls (without cardiovascular comorbidites) and 68 patients with hypertension. Baseline characteristics are presented in Table 1. The hypertension group was significantly older than the control group (58.6±13.3 vs 44.4±13.9 years, p<0.001). Women comprised 63.5% of the control group versus 50% of the hypertension group (p=0.043).

SIBO as a Predictor of Hypertension

Among patients with SIBO, 31/163 (19.0%) had hypertension compared to 37/168 (22.0%) among those without SIBO (Table 2). The odds ratio for hypertension given SIBO status was 0.83 (95% CI: 0.49-1.42, p=0.497), indicating no significant association. This finding contrasts with Lu et al., who reported OR=1.478 (95% CI: 1.039-2.102, p=0.03) (3).

Age-Stratified Analysis

Stratification by age revealed substantial variation in the SIBO-hypertension association across age groups (Table 3). The Mantel-Haenszel age-adjusted odds ratio was 1.10 (95% CI: 0.60–2.01), representing a 32.5% change from the crude OR and indicating meaningful age confounding, although the age-adjusted estimate did not reach statistical significance.

Sex-Stratified Analysis

Women with SIBO had 53% lower odds of hypertension (OR=0.47, CI 0.22–1.01, p=0.053), while men with SIBO showed 63% higher odds (OR=1.63, CI 0.75–3.54, p=0.22).

Tab. 1. *Baseline characteristics of study groups*

Parameter	Control (n=263)	Hypertension (n=68)	p-value
Age (years), mean±SD	44.4±13.9	58.6±13.3	<0.001
Women, n (%)	167 (63.5)	34 (50.0)	0.043
Men, n (%)	96 (36.5)	34 (50.0)	0.043
SIBO positive, n (%)	132 (50.2)	31 (45.6)	0.497

Tab. 2. *Association between SIBO and hypertension prevalence*

SIBO Status	Hypertension n (%)	No Hypertension n (%)	Total
SIBO Positive	31 (19.0)	132 (81.0)	163
SIBO Negative	37 (22.0)	131 (78.0)	168
Total	68 (20.5)	263 (79.5)	331
OR (95% CI)	0.83 (0.49-1.42)		p=0.497

Tab. 3. *Age-stratified analysis of SIBO predicting hypertension*

Age Group	n	HTN in SIBO+	HTN in SIBO-	OR	95% CI
<40 years	110	5/66 (7.6%)	4/44 (9.1%)	0.82	0.31-2.18
40-49 years	76	4/38 (10.5%)	3/38 (7.9%)	1.37	0.42-4.47
50-59 years	65	5/27 (18.5%)	9/38 (23.7%)	0.73	0.23-2.31
≥60 years	80	17/32 (53.1%)	21/48 (43.8%)	1.46	0.64-3.33
Mantel-Haenszel adjusted OR				1.10	0.60–2.01

Tab. 4. *Sex-stratified analysis of SIBO predicting hypertension*

Sex	HTN in SIBO+	HTN in SIBO-	OR	95% CI	p-value
Women (n=201)	12/102 (11.8%)	22/99 (22.2%)	0.47	0.22-1.01	0.053
Men (n=130)	19/61 (31.1%)	15/69 (21.7%)	1.63	0.75-3.54	0.22
Overall	31/163 (19.0%)	37/168 (22.0%)	0.83	0.49-1.42	0.497

Tab. 5. *Age distribution by sex and disease status*

Group	n	Mean Age (years)	SD
Overall			
All Women	201	47.7	16.1
All Men	130	46.8	12.9
By Hypertension Status			
Control Women	167	44.8	15.2
Control Men	96	43.6	11.1
Hypertension Women	34	61.6	12.8
Hypertension Men	34	55.6	13.7
SIBO+ Patients			
SIBO+ Women with HTN	12	60.2	14.1
SIBO+ Women without HTN	90	42.9	14.8
SIBO+ Men with HTN	19	54.7	12.9
SIBO+ Men without HTN	42	41.1	11.3

Age Distribution by Sex

Analysis of age distribution revealed that the sex-specific effects were not confounded by age differences (Table 5). Women and men had similar overall mean ages, but critically, women with hypertension were older than men with hypertension, yet still showed the protective association.

Multivariate Analysis

After adjustment for age and sex using logistic regression, the odds ratio was 1.09 (95% CI: 0.63-1.88, p=0.77), confirming no significant association between SIBO and hypertension.

Effect Modification Analysis

Formal testing for effect modification by sex revealed a trend toward heterogeneity (likelihood ratio test: $\chi^2 = 1.50$, $p = 0.221$). The interaction coefficient was $\beta = 0.75$, indicating that the SIBO-hypertension association differs between men and women. Women with SIBO showed lower odds of hypertension (OR = 0.77), while men with SIBO showed higher odds (OR = 1.62), representing a 2.1-fold difference in effect magnitude between sexes.

Age–SIBO interaction was not significant (likelihood ratio test: $\chi^2 = 1.09$, $p = 0.296$), with OR = 0.78 in patients aged <60 years and OR = 1.46 in patients aged ≥60 years (ratio 1.87), supporting age as a confounder rather than an effect modifier. However, stratified analysis revealed substantial heterogeneity across age groups, with odds ratios ranging from 0.78 in middle-aged patients to 1.46 in the oldest age group (Table 3). This pattern suggests that age acts primarily as a confounder rather than as an effect modifier, with both SIBO and hypertension increasing with age but not necessarily interacting.

Power analysis

Post-hoc power analysis revealed that with 68 hypertension patients, our study had 80% power ($\alpha = 0.05$) to detect an odds ratio of 2.20 or greater; for the observed OR = 0.83, actual power was only 10.4%. In sex-stratified subanalyses, power was further reduced: 80% power was achievable only for OR ≥ 3.18 in women and OR ≥ 3.16 in men, while observed effects of OR = 0.47 (power 50%) and OR = 1.63 (power 23%) remained below these thresholds. These results confirm that all analyses were substantially underpowered and that non-significant findings do not exclude clinically meaningful effects.

Discussion

Our study found no association between SIBO and arterial hypertension in symptomatic gastrointestinal patients, contrasting with recent findings by Lu et al. (3). However, we identified sex-specific effects that cannot be explained by age confounding. This complex pattern highlights the importance of considering effect modification in microbiome-disease association studies.

Population Differences and Their Implications

The fundamental difference between our study and that of Lu et al. lies in the patient population (3). Our cohort consisted exclusively of symptomatic patients with gastrointestinal complaints undergoing SIBO testing for clinical indications. This represents a pathophysiologically distinct group where the relationship between SIBO and systemic diseases may be altered by underlying gastrointestinal dysfunction (9).

The high SIBO prevalence in both our groups (50.2% in controls, 45.6% in hypertension) compared to Lu et al. (37.5% in controls, 49% in hypertension) supports this interpretation. Symptomatic patients often have impaired gastrointestinal motility, altered dietary patterns, and medication exposures affecting both the gut microbiome and cardiovascular risk factors (9). This high baseline prevalence may create a ceiling effect that obscures associations detectable in populations with lower SIBO rates.

Furthermore, the presence of symptoms may indicate chronic adaptation to SIBO. The hypothesis that SIBO contributes to hypertension through increased production of metabolites such as trimethylami-

Tab. 6. *Formal testing for effect modification by sex and age*

Interaction	LR χ^2	p-value	OR Women	OR Men	OR Ratio
Sex × SIBO	1.50	0.221	0.77	1.62	2.11
Age × SIBO	1.09	0.296	0.78[a]	1.46[a]	1.87

[a]Age < 60 vs ≥ 60 years. LR – likelihood ratio; OR – odds ratio from stratified analyses

ne-N-oxide (TMAO) and lipopolysaccharide assumes uniform operation across populations (20, 21). However, symptomatic patients may have developed compensatory mechanisms or immunological tolerance through repeated exposure (22, 23).

Age Confounding: A Critical Factor

Our analysis revealed substantial age confounding, with the OR changing from 0.83 (crude) to 1.10 (age-adjusted)—a 32.5% change. The 14-year age difference between groups was clinically meaningful, as hypertension prevalence increases from 7-10% in individuals under 40 years to over 70% in those over 65 years (12, 16). Similarly, SIBO prevalence increases with age due to decreased gastric acid production, slowed transit, and immunosenescence (11, 17).

The age-stratified analysis revealed non-uniform effects across age groups, suggesting age acts not only as a confounder but also as an effect modifier. The protective association in younger groups (<40 years: OR=0.82) contrasted with increased risk in older groups (≥60 years: OR=1.46). This heterogeneity indicates that any SIBO-hypertension relationship may be age-dependent.

While Lu et al. also reported age differences, they used logistic regression adjustment rather than stratified analysis (3). Our Mantel-Haenszel approach, which does not assume linearity or constant effects across strata, may provide more robust adjustment for complex confounding patterns.

Sex as an Effect Modifier: A Real Phenomenon

Our identification of sex-specific effects represents a finding that cannot be explained by age confounding. Critical analysis revealed that women and men had nearly identical mean ages overall (47.7 vs 46.8 years), definitively ruling out age as an explanation for the sex difference.

More strikingly, women in the hypertension group were older than men with hypertension (61.6 vs 55.6 years), yet still showed a protective trend between SIBO and hypertension. Among SIBO-positive patients, women had markedly lower hypertension prevalence (11.8%) compared to men (31.1%), despite hypertensive women being 5.5 years older. The age gap between those with and without hypertension was larger in women (17.3 years) than in men (13.6 years) among SIBO-positive patients. Despite this greater age disadvantage, SIBO-positive women still had lower hypertension rates, suggesting the protective trend works against the age gradient.

Possible mechanisms for this sex difference include estrogen's effects on gut barrier function and inflammation (18), sex-specific immune responses, and different SIBO phenotypes between sexes. The higher prevalence of functional gastrointestinal disorders in women may mean that symptomatic women represent a different phenotype with different SIBO-systemic disease relationships (19).

However, these observations must be interpreted with considerable caution. The formal likelihood ratio test for sex–SIBO interaction did not reach statistical significance ($\chi^2 = 1.50$, $p = 0.221$), meaning that the difference between female and male estimates is statistically compatible with chance. Combined with the low observed power in sex-stratified subanalyses (50% for women, 23% for men), the apparent sex-specific pattern cannot be distinguished from a type I error. We therefore caution against over-interpreting these findings: they represent an exploratory observation that justifies hypothesis formulation and pre-specified testing in future adequately powered studies, but do not constitute evidence of a confirmed sex-specific SIBO–hypertension relationship.

Mechanistic Considerations

The lack of association in our symptomatic population challenges proposed mechanisms linking SIBO to hypertension through the gut-cardiovascular axis (4, 5). The specific bacterial species involved in SIBO may differ between symptomatic and asymptomatic individuals, with different metabolic consequences (24, 25). Symptomatic patients may have chronic low-grade inflammation that paradoxically blunts systemic responses to SIBO (26).

A further unresolved issue is the absence of blood pressure values for both groups. Without these data, we cannot determine whether hypertensive patients were adequately controlled at the time of SIBO testing, nor can we assess blood pressure levels in controls—some of whom may have had undiagnosed hypertension. This limits our ability to characterize the true relationship between blood pressure status and SIBO.

Without metagenomic data, we cannot determine whether bacterial composition differs between our population and that studied by Lu et al. However, the sex-specific patterns suggest that any mechanistic pathways linking SIBO to hypertension operate differently in men versus women, possibly through interactions with sex hormones or sex-specific microbiome profiles.

Clinical Implications

Our findings indicate that SIBO testing should not be used as a cardiovascular risk marker in symptomatic gastrointestinal patients. The absence of overall association suggests treating SIBO in this population would not be expected to reduce hypertension risk. However, the sex-specific findings, if confirmed, might support targeted approaches in specific subgroups.

The observed effect modification by sex suggests personalized approaches to both SIBO and hypertension management may be necessary. Future clinical trials should stratify by sex and consider symptom status when assessing cardiovascular outcomes of SIBO treatment.

Strengths and Limitations

Strengths include rigorous statistical methodology with stratified analysis and formal interaction testing, and the use of standardized diagnostic criteria allowing direct comparison with other studies.

Several limitations merit consideration. First, the retrospective single-center design precludes causal inference and limits generalizability. Second, the hypertension group was small (n=68), resulting in low statistical power—particularly in sex-stratified subanalyses—and precluding detection of small-to-moderate effects. Third, hypertension classification based on medical records and medication use carries a substantial risk of misclassification: some antihypertensive drug classes are also prescribed for non-hypertensive indications such as arrhythmias or heart failure; office measurements may reflect white coat effect;

and the complete absence of blood pressure values precludes any assessment of hypertension severity, compensation status, or whether controls had undiagnosed hypertension—all of which may have biased group allocation in unknown directions. Fourth, we lack data on several confounders that are independently and strongly associated with both SIBO and hypertension: BMI and obesity, metabolic syndrome, diabetes mellitus, smoking, alcohol consumption, dietary patterns, physical activity, and medications known to alter gut microbiota composition such as proton pump inhibitors and metformin. These unmeasured confounders may substantially bias our results and the conclusions of this study must therefore be interpreted with considerable caution.

Conclusions

Our study found no overall association between SIBO and arterial hypertension in symptomatic gastrointestinal patients after appropriate confounding adjustment. However, we observed sex-specific trends that should be considered strictly hypothesis-generating: women showed a trend toward lower odds of hypertension (OR=0.47) while men showed a trend toward higher odds (OR=1.63), though neither reached statistical significance, the sex–SIBO interaction was not significant (p=0.221), and all subgroup analyses were substantially underpowered. These observations provide a rationale for prospective studies with pre-specified sex-stratified analyses and adequate sample sizes.

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