

Prognostic Nomogram on Clinicopathologic Features and Serum Uric Acid for Precancerous Lesions and Gastric Cancer

Shanshan Tang

Department of Endocrinology and Metabolism, Shanghai General Hospital, Shanghai Jiao Tong University School of Medicine, 100 Haining Road, Shanghai, 200080, China

Ying Chen

Department of Endocrinology and Metabolism, Shanghai General Hospital, Shanghai Jiao Tong University School of Medicine, 100 Haining Road, Shanghai, 200080, China

Chenhong Fu

Department of Gastroenterology, Shanghai General Hospital, Shanghai Jiao Tong University School of Medicine, 100 Haining Road, Shanghai, 200080, China

Xin Xie

Department of Endocrinology and Metabolism, Shanghai Traditional Chinese and Medicine integrated Hospital, 18 Baoding Road, Hongkou District, Shanghai, 200080, China

Ziyu Song

Department of Chinese Medicine, Shanghai Hongkou District Guangzhong Road Community Health Service Center, No.80 Guangling 1st Road, Hongkou District, Shanghai, 200083, China

Jianzhong Xu

Department of Chinese Medicine, Shanghai Hongkou District Guangzhong Road Community Health Service Center, No.80 Guangling 1st Road, Hongkou District, Shanghai, 200083, China

Yihan Zhang

Department of Endocrinology and Metabolism, Shanghai General Hospital, Shanghai Jiao Tong University School of Medicine, 100 Haining Road, Shanghai, 200080, China

Nengguang Fan

Department of Endocrinology and Metabolism, Shanghai General Hospital, Shanghai Jiao Tong University School of Medicine, 100 Haining Road, Shanghai, 200080, China

Yongde Peng

Department of Endocrinology and Metabolism, Shanghai General Hospital, Shanghai Jiao Tong University School of Medicine, 100 Haining Road, Shanghai, 200080, China

Qifa Zhang

Department of Urology, Shanghai Jiangqiao Hospital (Shanghai General Hospital Jiading Branch), Shanghai, 201803, China

Huanbai Xu (✉ huanbaixu@126.com)

Department of Endocrinology and Metabolism, Shanghai General Hospital, Shanghai Jiao Tong University School of Medicine, 100 Haining Road, Shanghai, 200080, China

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TITLE PAGE

RESEARCH ARTICLE

Nomogram for predicting the risk of GC and GPL based on SUA

Prognostic Nomogram on Clinicopathologic Features and Serum Uric Acid for Precancerous Lesions and Gastric Cancer

Shanshan Tang¹, Ying Chen¹, Chenhong Fu², Xin Xie³, Ziyu Song⁴, Jian Zhong Xu⁴, Yihan Zhang¹, Nengguang Fan¹, Yongde Peng¹, Qifa Zhang^{5*}, Huanbai Xu^{1*}

¹Department of Endocrinology and Metabolism, Shanghai General Hospital, Shanghai Jiao Tong University School of Medicine, 100 Haining Road, Shanghai, 200080, China

²Department of Gastroenterology, Shanghai General Hospital, Shanghai Jiao Tong University School of Medicine, 100 Haining Road, Shanghai, 200080, China

³Department of Endocrinology and Metabolism, Shanghai Traditional Chinese and Medicine integrated Hospital, 18 Baoding Road, Hongkou District, Shanghai, 200080, China

⁴Department of Chinese Medicine, Shanghai Hongkou District Guangzhong Road Community Health Service Center, No.80 Guangling 1st Road, Hongkou District, Shanghai, 200083, China

⁵Department of Urology, Shanghai Jiangqiao Hospital (Shanghai General Hospital Jiading Branch), Shanghai, 201803, China

***Corresponding author:** Huanbai Xu and Qifa Zhang. Huanbai Xu, MD, PhD. Department of Endocrinology and Metabolism, Shanghai General Hospital, Shanghai Jiao Tong University School of Medicine, 100 Haining Road, Shanghai, 200080, China. Qifa Zhang, MD, PhD. Department of Urology, Shanghai Jiangqiao Hospital (Shanghai General Hospital Jiading Branch), Shanghai, 201803, China

Email: huanbaixu@126.com (HX) and tss8235078@163.com (ST)

Telephone number:15800622510(HX) and 15800853279(ST)

Fax number:021- 63240090

Co-first author: Shanshan Tang and Ying Chen contributed equally to this work.

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Abstract

The relationship between Uric acid (UA) and malignant tumor are still confusing. Gastric cancer(GC) is recognized to be closely related to *Helicobacter pylori* (*H. pylori*) infection, early diagnosis rate is very low. In this study, we aimed to investigate the relationship between *H. pylori* and hyperuricemia (HUA), and evaluate the predictive value of serum uric acid (SUA) in gastric precancerous lesion (GPL) and gastric cancer (GC). This retrospective study included 486 patients who underwent gastroscopy (155 controls, 272 GPL, 59 GC patients). The risk factors for GPL and GC were identified by multiple logistic regression analysis and nomogram was constructed to evaluate the ability of SUA to predict the risk of these diseases based on SUA score. We found that in healthy controls, HUA is positively correlated with *H. Pylori* (+). SUA was an independent risk factor for GPL and GC. Verification shows that the nomogram was better fitted for GC than for GPL. In conclusion, our study established nomogram based on SUA to predict the risk of GPL and GC, suggested that the incidence of GPL and GC is higher in *H. pylori* (+) HUA patients, so early intervention and vigilance should be raised.

Keywords:

Uric acid; Helicobacter pylori; Gastric precancerous lesion; Gastric cancer; Predictive value

INTRODUCTION

Gastric cancer (GC) is one of the most common malignant tumors in China, ranking the third in the incidence of malignant tumors, the second in the mortality rate, and second only to lung cancer [1]. Although the incidence of GC is on the decline in most countries around the world, more than half of the new GC cases in the world each year still come from east Asia, with China, Japan and South Korea especially serious. And due to its low early diagnosis rate, about 70% of patients diagnosed with GC can no longer be treated by surgery[2]. Therefore, early screening and diagnosis and intervention of GC should be strengthened.

Uric acid (UA) is a terminal metabolite of human purine compounds. Previous studies have shown that UA is both a inflammatory medium and an antioxidant. An higher UA level has often been observed with gout, non-alcoholic fatty liver disease, metabolic syndrome, cardiovascular disease [3]. Although the antioxidant properties of serum uric acid (SUA) are believed to prevent the occurrence of malignant tumors, recent epidemiological studies have shown that hyperuricemia (HUA), a component of the metabolic syndrome (MS), increases the risk of colorectal, breast, prostate and other cancers [4,5]. The relationship between UA and malignant tumor remains to be further studied.

In recent years, *Helicobacter pylori* (*H. pylori*) infection has attracted increasing attention due to changes in diet and environment, as well as the popularity of testing and the improvement of people's sense of self-care. Gastric carcinogenesis is recognized to be a multistep and multifactorial process, *H. pylori* infection is also recognized to play an important role in this process [6,7]. The final outcome of HUA and *H. pylori* infection was low grade inflammation. Whether SUA level and *H. pylori* infection are involved in and influence each other in the progression of GC has

not been conclusively concluded. Therefore, we attempted to study not only the relationship between *H. pylori* and SUA, but also the relationship between SUA level and gastric precancerous lesion (GPL) patients as well as GC patients. Meanwhile, We also establish a personalized visual model based on the research results.

MATERIALS AND METHODS

Study Population

We conducted a retrospective cohort analysis of adults aged 18 years or older who underwent routine health screening at Shanghai General Hospital from February 2017 to December 2019. All participants underwent esophagogastroduodenoscopy (EGD) to detect gastrointestinal lesions. Urease detection for *Helicobacter pylori* was performed during EGD. Basic blood test data were also obtained on the same day as EGD. Those who had at least two screening tests were considered for analysis. EGD diagnosed as normal or chronic superficial gastritis from subjects participating in health screening at the same time were normal controls.

Inclusion and exclusion criteria

Inclusion criteria: (1) Subjects were a minimum of 18 years of age; (2) Complete clinical and laboratory data.

Exclusion criteria:(1) Acute complications, diabetes, serious cardiovascular and cerebrovascular diseases, severe liver and kidney dysfunction, malignant tumors, leukemia; (2) Recently used uric-acid-lowering drugs or other drugs that affect the production or excretion of UA, drugs for the treatment of *H. pylori* (diuretics, lipid-regulating drugs, aspirin, angiotensin-converting enzyme inhibitors, angiotensin receptor antagonists); (3) SUA level is less than 3mg/dL (180 μ mol/L); (4) Lack of *H. pylori* urease test results for gastric biopsy specimens or basic blood test data.

Based on these criteria, the final study population consisted of 486 subjects (239 men and 247 women). At the same time, 486 patients were divided into healthy control group, GPL group and GC group according to EGD pathological diagnosis.

All the pathological diagnoses were made following the updated Sydney Gastritis Classification and World Health Organization Classification of Tumors of the Digestive System [8,9].

Study definitions

Hyperuricemia: fasting serum uric acid ≥ 7 mg/dL (adult, male and female) can be diagnosed [10]. The following variables are defined and grouped in this study[11]: Body mass index (BMI) was calculated as weight in kilograms divided by height in squared. According to the WHO guide-lines for the Asia-Pacific population [12], normal weight was defined as $18.5 \leq \text{BMI} < 24.0\text{kg/m}^2$, overweight was defined as $24.0 \leq \text{BMI} < 28.0\text{kg/m}^2$, obesity was defined as $\text{BMI} \geq 28.0\text{kg/m}^2$, and underweight was defined as $\text{BMI} < 18.5\text{kg/m}^2$. According to the Chinese adult dyslipidemia prevention guide (2016 edition), Total cholesterol (TC) ≥ 6.2 mmol/L (240 mg/dL) were considered to be hypercholesterolemia, Triglycerides (TG) ≥ 2.3 mmol/L (200 mg/dL) were considered to be hypertriglyceridemia, Low-density lipoprotein-cholesterol (LDL-C) ≥ 4.1 mmol/L (160 mg/dL) were considered to be LDL-hypercholesterolemia, and high-density lipoprotein-cholesterol (HDL-C) ≤ 1.0 mmol/L (40 mg/dL) were considered to be HDL-hypocholesterolemia [13].

Laboratory Assays

Peripheral blood samples were taken at the first visit. At the same time, blood samples were taken before patients were pretreated. After fasting for 8 hours, subjects

underwent standard venipuncture in the anterior cubital fossa vein (anterior cubital vein) in the morning, and blood samples were taken for biochemical testing. SUA was quantitatively determined using a commercially available kit (Roche Diagnostics, Mannheim, Germany) enzymatic colorimetry.

Ethical Statement

This study have been performed in accordance with the Declaration of Helsinki and approved by the Ethics Committee of Shanghai General Hospital of Jiaotong University in Shanghai, China. All experiments were performed in accordance with relevant guidelines and regulations. All subjects provided written informed consent to use any clinical data for the study.

Statistical Analysis

Variations recorded for each subject were age, sex, BMI, HbA1c, lipid levels, smoking status, family history of GC, and H. pylori status. In addition, SUA levels were recorded. Continuous variable data are expressed as mean standard deviations, while classified data are expressed as numbers (percentages). We divided the subjects into quartiles, according to SUA level (Q1: < 5 mg/dL($Q1 < 300\mu\text{mol/L}$), Q2: $5 - 6$ mg/dL($300 \leq Q2 < 360\mu\text{mol/L}$), Q3: $6 - 7$ mg/dL($360 \leq Q2 < 420\mu\text{mol/L}$), Q4: ≥ 7 mg/dL($Q4 \geq 420\mu\text{mol/L}$)). The Chi-square test or Fisher's exact test was used to compare categorical variables. All P values were two-sided and $P < 0.05$ was considered to indicate statistical significance. SPSS software (Version 24) was used for statistical analysis. Variables with statistically significant differences in univariate analysis were included in multiple logistic regression analysis to determine the independent risk factors for GPL or GC. According to the results, R software version

3.6.1 was used to visualize the forest plot. Besides, Chi-square test and Spearman correlation analysis were used to study the correlation between *H. pylori* (+) and HUA, as well as the occurrence of GPL or GC in *H. pylori* (+) patients with HUA. Receiver operating characteristics (ROC) curve was used to evaluate the diagnostic value of SUA in predicting precancerous gastric lesions and gastric cancer, and to determine the optimal cut-off concentrations of SUA respectively. According to the results of multiple logistic regression analysis, R software was used to construct a nomogram model to evaluate the ability of SUA predicting the risk of GPL or GC based on SUA score. The discrimination of the nomogram was measured by Harrell's concordance index (C-index). Through Bootstrap sampling method to calculate C-index. The value of the C-index ranged from 0.5 to 1.0, with 0.5 indicating random chance, and 1.0 indicating the perfect ability to correctly predict the outcome with the model [14]. On this basis, the calibration curve of nomogram is established to analyze and evaluate the predictive performance of the model. The calibration curve is the comparison of the actual risk and the predicted risk. The higher the coincidence rate of the curve, the better the prediction effect. Decision-curve analysis can be used to incorporate the clinical consequences of a decision into evaluations of diagnostic test results or prediction models [15], which represents a potential net benefit of each decision strategy at each threshold probability.

RESULTS

Baseline Characteristics among Healthy, Gastric Precancerous Lesion and Gastric Cancer

According to gastroscopy and pathological results, 486 participants were divided into GPL group (272 case, 56.3%), GC group (59 cases, 11.8%), and 155 healthy (31.9%)

subjects which were selected as the control group are shown in Table 1. Among them, *H. pylori* infection was negative in 214 patients with GPL (78.7%), positive in 58 patients (21.3%). Of the 59 patients with GC, 17 patients (28.8%) were positive for *H. pylori* and 42 patients (71.2%) were negative. Besides, we found that compared with the control group, the incidence of HUA (Q4) in both the GPL group and the GC group was significantly increased (38.6% vs. 11.0%, $P < 0.001$), (40.7% vs. 11.0%, $P < 0.001$). According to Chi-square test, there were significant difference in gender, age, BMI, LDL-C, *H. pylori* infection and SUA between the control group and the GPL group as well as the GC group are shown in Table 1. Therefore, gender, age, BMI, LDL-C, *H. pylori* infection were considered risk factors for GPL and GC and were used as confounding factors in the following statistical analysis to correct the relationship between SUA and GPL and the relationship between SUA and GC, respectively.

Moreover, in order to exclude the effect of secondary hyperuricemia, we only studied the relationship between *H. pylori* and HUA in healthy controls. Our study revealed a positive correlation between *H. pylori* (+) and HUA ($r = 0.207$, $P < 0.05$) in healthy controls (Table 2). The incidence of GPL or GC in patients with HUA after *H. pylori* infection was significantly higher than those patients without HUA (33.3% vs. 46.6%; $P = 0.021$), (33.3% vs. 47.1%; $P = 0.043$) (Table 3). Therefore, when patients with *H. pylori* (+) developed HUA ($\geq 7\text{mg/dL}$), they were more prone to gastrointestinal pathological changes, and the degree of precancerous lesions was more serious.

SUA is An Independent Prognostic Factors for Gastric Precancerous Lesion and Gastric Cancer

Multivariate logistic regression analysis (Table 4) showed that gender (OR = 2.054),

BMI (OR = 1.269), *H. pylori* infection (OR = 2.502) and SUA (OR = 2.275) were significantly associated with GPL in patients; and age (OR = 1.826), *H. pylori* infection (OR = 3.434) and SUA (OR = 2.493) were significantly associated with GC in patients. According to the results of multivariate logistic regression analysis (Table 4), the results are shown in the forest plots (Fig.1A, B), respectively. The results have shown that HUA is an independent risk factor for the occurrence and development of GPL and GC, and the risk of GPL and GC increases with the increase of SUA level. With SUA level as the test variable, ROC curve analysis showed whether GPL or GC occurred or not as the outcome variable. The AUROC of SUA was calculated to assess the diagnostic accuracy for prediction of GPL and GC (Fig.2). The AUROC of the prediction of SUA on GPL was 0.801(95%CI: 0.759 ~ 0.843, $P < 0.001$), the sensitivity was 77.9%, the specificity was 72.9%, and the optimal diagnostic value was 5.24mg/dL (341.5 μ mol/L) (Fig.2A). The AUROC of the prediction of SUA on GC was 0.854 (95%CI: 0.796 ~ 0.913, $P < 0.001$), the results were statistically significant. The sensitivity was 74.6%, the specificity was 85.8%, and the optimal diagnostic value was 6.26mg/dL (375.5 μ mol/L) (Fig.2B). These results suggested that SUA could be used to predict the risk of GPL and GC.

Nomogram was Established to Evaluate the Ability of SUA to Predict The Risk of Gastric Precancerous Lesion and Gastric Cancer

We constructed a new predictive risk model (Fig.3A, B) named nomogram for GPL and GC, respectively. The model consisted of gender, BMI, *H. pylori* infection and SUA, which were significantly correlated with GPL ($p < 0.05$) (Fig.3A) The model consisted of age, *H. pylori* infection and SUA, which were significantly correlated with GC ($p < 0.05$) (Fig.3B).

Validation of the Nomogram Model for Predicting Gastric Precancerous Lesion and Gastric Cancer

The calibration of the nomogram was evaluated using a calibration curve (Fig.4A, B), accompanied with the Hosmer Lemeshow test. The clinical value of the nomogram and SUA was analyzed by the decision curve and showed in (Fig.4C, D). The calibration curve (Fig.4A) and Hosmer–Lemeshow test statistic ($P < 0.001$) performed poor calibration, which shows the prediction model of GPL was not well fitted. But the C-index of the nomogram about GPL was 0.781, as shown in Table 5. The calibration curve (Fig.4B) and Hosmer–Lemeshow test statistic ($P = 0.058 > 0.05$) performed favorable calibration, which shows the prediction model of GC was well fitted. The C-index of the nomogram about GC was 0.835, as shown in Table 5. The results show that the GC model has good prediction effect. When the threshold of high risk is 0.08 ~ 0.60, the net benefit rate is > 0 , which has clinical significance, suggesting that SUA has important clinical value in the prediction of GPL in Fig.4C. When the threshold of high risk is 0.40 ~ 0.88, the net benefit rate is > 0 , which has clinical significance, suggesting that SUA has important clinical value in the prediction of GC in Fig.4D.

DISCUSSION

The prevalence of HUA in China has been increasing over the past decades [16]. The prevalence of HUA in Shanghai in 2015 was 17.2% [11]. Currently, there are many reports on the epidemiological evidence of elevated SUA levels and cancer incidence, but the conclusions are inconsistent. Studies have suggested that the risk of cancer is significantly increased with elevated baseline SUA levels [17,18]. Previous study

suggested that increasing SUA was associated with poorer outcomes of renal cell carcinoma (RCC) [19]. It has also been reported that HUA is not a risk factor for cancer, but a protective factor. This result suggested that high preoperative SUA levels were identified as an independent prognostic factor associated with improved clinical outcomes among laryngeal squamous cell cancer (LSCC) patients [20]. A retrospective study from the UK also showed a negative correlation between SUA levels and lung cancer risk, although this association was limited to smokers [21]. Considering the inconsistency of the above conclusions and the fact that few people pay attention to the risk factors of precancerous gastric lesions, we studied the relationship between SUA and GPL and the relationship between SUA and GC, hoping to provide more data support for the relationship between SUA and malignant tumors.

The progression from normal to GPL to GC is a multistep and multifactorial dynamic process. *H. pylori* infection is also recognized to play an important role in this process. Previous studies have proved that *H. pylori* is related to chronic metabolic diseases, including type 2 diabetes, hyperlipidemia and MS [22-24] . However, few people have paid attention to the relationship between *H. pylori* and HUA. Besides, whether both of them are involved in and influence each other in the progression of GC has not been conclusively concluded. In order to exclude the influence of secondary HUA during tumor development, that is, a large amount of cell destruction accelerates nucleic acid metabolism, the relationship between *H. pylori* and HUA in this study was only conducted in healthy controls. In our study, we revealed a positive correlation between *H. pylori* (+) and HUA in the healthy control group. This result supports that the occurrence of HUA reported in Africa is significantly correlated with *H. Pylori* infection ($P < 0.01$) [24]. Both HUA and *H. pylori* are chronic systemic

inflammatory diseases. The mechanism by which *H. pylori* affects HUA may be that oxidative stress reaction and autoimmune reaction jointly lead to insulin resistance (IR), while IR and HUA promote each other and thus eventually lead to the occurrence of HUA[25-27].The disorder of glucose and lipid metabolism caused by HUA can also promote the occurrence of *H. pylori* infection[27]. Therefore, *H. pylori* infection and HUA may have some common pathogenesis and interact with each other.

Besides, we found that compared with the control group, the incidence of HUA ($\geq 7\text{mg/dL}$) in both the GPL group and the GC group was significantly increased. After adjustment for several prognostic variables such as age, gender and BMI, LDL-C, *H. pylori* in our study, the results showed that high SUA levels were significantly and independently associated with a higher risk of GC. The AUROC of the prediction of SUA on GPL was 0.801, the sensitivity was 77.9%, the specificity was 72.9%, the optimal diagnostic value was 5.24mg/dL (341.5 $\mu\text{mol/L}$), the AUROC of SUA on GC was 0.854, the sensitivity was 74.6%, the specificity was 85.8%, and the optimal diagnostic value was 6.26mg/dL (375.5 $\mu\text{mol/L}$), which supported the good diagnostic efficacy of SUA. Moreover, we found the incidence of GPL and GC in patients with HUA after *H. pylori* infection was significantly higher than that in patients without HUA respectively. Taken together, our study demonstrated that SUA is a risk factor for GPL and GC.

UA acts as a systemic antioxidant, its pro-inflammatory effect can mediate the mutation process of tumor cells and participate in the development of malignant tumors. In addition, obesity, type 2 diabetes and MS are also associated with cancer related inflammation (CRI) and HUA, suggesting that UA may play an important role in these diseases and the development of malignant tumors. The specific mechanism

by which SUA is involved in the occurrence and development of gastric cancer is still unclear. It may be due to the presence of UA in the body in the form of sodium urate crystals or soluble factors, both of which promote inflammation. Inflammatory mediators are an important part of tumor local environment, and inflammatory response can promote tumor proliferation and survival. Soluble UA can enter human cells to activate mitogen-activated protein kinase(MAPK), further activate the nuclear factor kB (NFkB) inflammatory pathway, and induce the expression of inflammatory mediators such as monocyte chemoattractant protein-1 (MCP-1) and c-reactive protein(CRP) [28]. MSU(monosodium urate) also can be recognized by toll-like receptor 4 and promote the production of various inflammatory cytokines by leukocytes. And CRP, adiponectin and leptin, together with UA, constitute a chronic inflammatory environment, and long-term chronic inflammatory response may promote tumor progression[29]. In addition, when UA enters cancer cells, it inhibits intracellular Xanthine oxidoreductase (XOR) expression. Low level of XOR stimulates the expression of differentiation protein inhibitors to increase the aggressiveness of cancer cells by regulating the secretion of cyclooxygenase-2 (COX-2) and matrix metalloproteinase-1 (MMP-1)[30]. The pathophysiological process of normal - GPL - GC involves many factors, SUA may affect the occurrence and development of GC in a certain way, forming an internal environment that promotes the mutation and proliferation of tumor cells. The mechanism of this effect needs further study and analysis.

In our study, nomograms were constructed to predict the risk of GPL and GC based on SUA score and the diagnostic ability of SUA respectively. The accuracy and discriminability were determined by the C- index and verified by the calibration curve and the decision curve, confirming that the nomogram of gastric cancer in this study

had good consistency and differentiation. But the prediction model of precancerous gastric lesions based on SUA score is not well fit. The difference between these two results may be due to the fact that the whole pathological process from normal mucosa to multifocal atrophy and intestinal metaplasia to finally to dysplasia or even cancer is a multistep and multifactorial process. The mechanism is too complex, SUA is not the only thing that can explain the difference.

Our study has some limitations. First of all, this study is a retrospective analysis of a single base and a single center. Secondly, due to the limitation of time and tools, there is no long-term follow-up in this physical examination population, and there is a lack of data affecting patient outcome or survival.

CONCLUSIONS

Our study is the first to study the relationship between HUA and *H. pylori*, as well as the relationship between SUA and GPL and the relationship between SUA level and GC. SUA is a valuable factor for predicting risk of GPL and GC. Since the incidence of GPL and GC in *H. Pylori* positive patients with HUA was significantly higher than that in non-HUA patients, some attention should be paid to the treatment and management of *H. Pylori* (+) HUA patients. In addition, the predictive model of GC constructed in this study showed good diagnostic value and predictive ability, while the GPL model was poor fitted. The difference between the two models may be due to the many factors involved in the pathophysiological process from GPL to GC, and SUA may affect its occurrence and development in a certain way, the mechanism of which remains to be further studied and analyzed.

Data availability

The datasets generated during and/or analysed during the current study are available

from the corresponding author on reasonable request.

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Author Contributions

Study concept, design and supervision: **HB Xu** And **QF Zhang**. Acquisition, analysis, or interpretation of data and drafting of the article: **SS Tang** And **Y Chen**. Collect data: **CH Fu**, **X Xie**, **ZY Song**, **JZ Xu**, **YH Zhang** And **NG Fan**. Critical revision of the article for important intellectual content: **HB Xu** And **QF Zhang** . Obtaining funding: **SSTang**, **HB Xu** And **QF Zhang** . Administrative, technical, or material support: **YD Peng**.

Competing interests

The authors have not declared any conflicts of interest.

Figure legend

Figure1. Forest Plot for Outcome of Logistic Regression Analysis

In the plane cartesian coordinate system, with an invalid vertical line (horizontal coordinate scale of 1 or 0) as the center, multiple line segments parallel to the horizontal axis are used to describe each included variable and confidence interval (CI), and a prism is used to describe multiple variables and confidence interval

combined in the study. It simply and intuitively describes the statistical results of multiple logistics regression analysis.

(A) Forest Plot for Gastric Precancerous Lesion

(B) Forest Plot for Gastric Cancer

Figure2. The Predictive Value of Uric Acid in Gastric Precancerous Lesion and Gastric Cancer

The y-axis indicates the true positive rate of the risk prediction. The x-axis indicates the false-positive rate of the risk prediction. The blue line represents the performance of the nomogram.

(A) The Predictive Value of Uric Acid in Gastric Precancerous Lesion

(B) The Predictive Value of Uric Acid in Gastric Cancer

Figure3. Nomogram Model to Predict the Risk of Gastric Precancerous Lesion and Gastric Cancer

The scores corresponding to different values of each factor and risk of each factor are shown in this figure. The total scores of each factor are calculated. The total scores range from 0 - 200, 0 - 220, respectively. And the corresponding risk rates range from 0.2 - 0.9, 0.1 - 0.9, respectively. The higher the total score, the greater the risk of GPL and GC.

(A) Nomogram Model to Predict the Risk of Gastric Precancerous Lesion

(B) Nomogram Model to Predict the Risk of Gastric Cancer

Figure4.Validation of the Nomogram Model about Gastric Precancerous Lesion and Gastric Cancer

The x-axis represents the predicted incidence risk. The y-axis represents the actual diagnosed GPL or GC. The diagonal dotted line represents a perfect prediction by an ideal model. The solid line represents the performance of the nomogram; a closer fit to the diagonal dotted line represents a better prediction; a calibration plot compares the model's predicted probabilities and observed proportions. The diagonal line reflects the ideal situation (predicted probability = observed proportion) (Fig4A, B). The y-axis indicates the net benefit. The dotted line represents the incidence risk nomogram of GPL or GC. The thin solid line represents the assumption that all patients are diagnosed as GPL or GC. Thin thick solid line represents the assumption that no patients are diagnosed as GPL or GC (Fig4C, D).

(A)The Calibration Curve of Gastric Precancerous Lesion

(B)The Calibration Curve of Gastric Cancer

(C)The Decision Curve Analysis for Gastric Precancerous Lesion

(D)The Decision Curve Analysis for Gastric Cancer

Figures

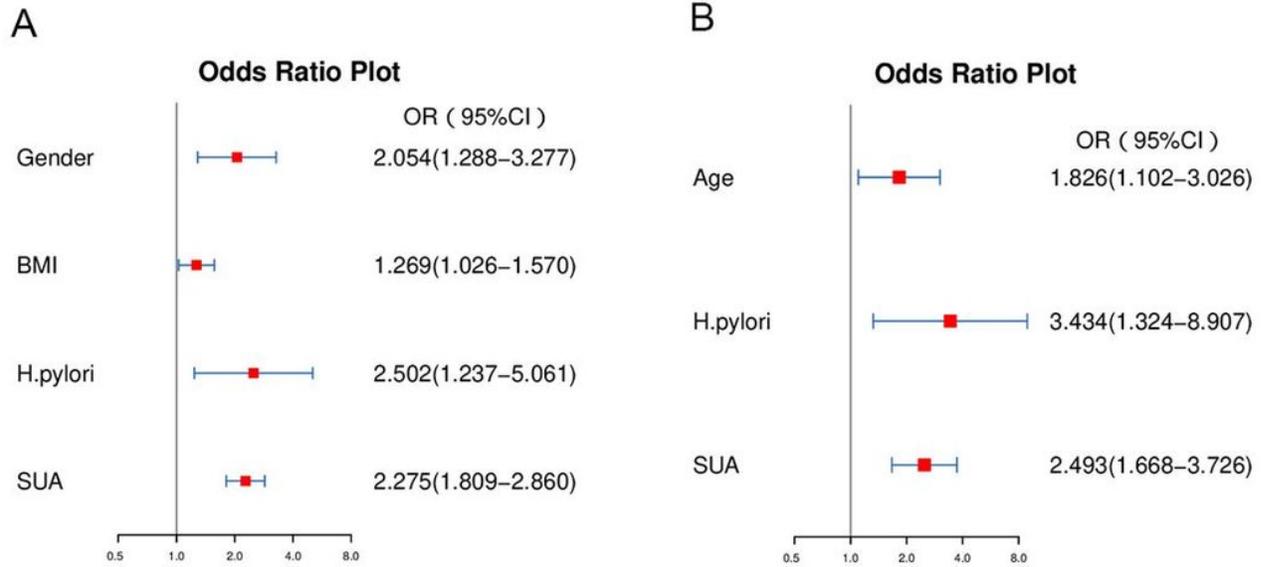


Figure 1

Forest Plot for Outcome of Logistic Regression Analysis In the plane cartesian coordinate system, with an invalid vertical line (horizontal coordinate scale of 1 or 0) as the center, multiple line segments parallel to the horizontal axis are used to describe each included variable and confidence interval (CI), and a prism is used to describe multiple variables and confidence interval combined in the study. It simply and intuitively describes the statistical results of multiple logistics regression analysis. (A) Forest Plot for Gastric Precancerous Lesion (B) Forest Plot for Gastric Cancer

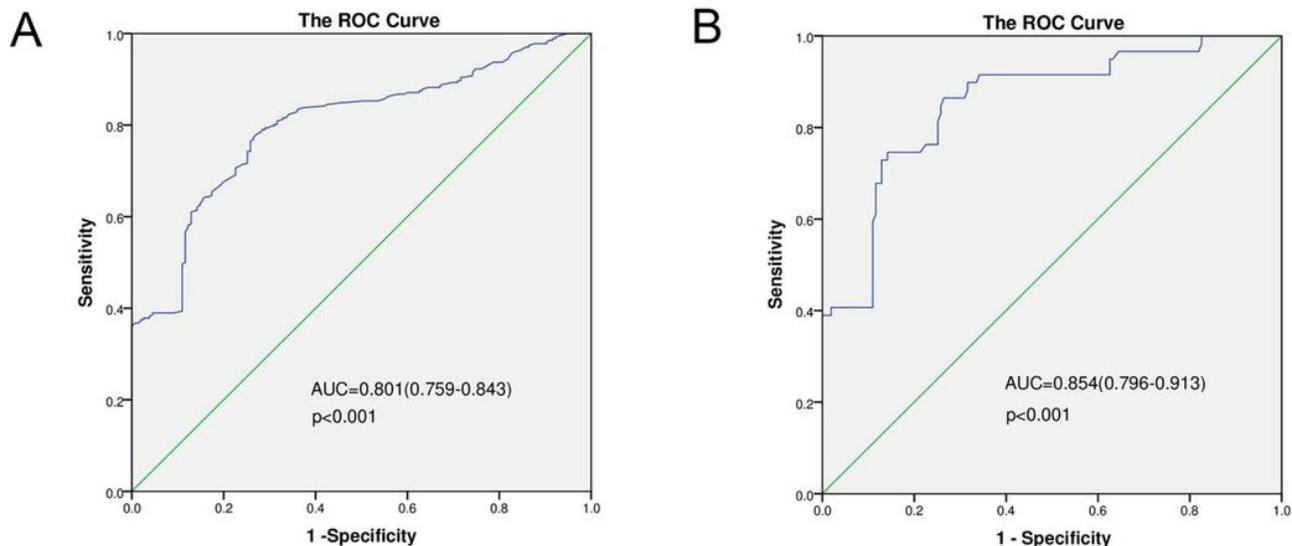
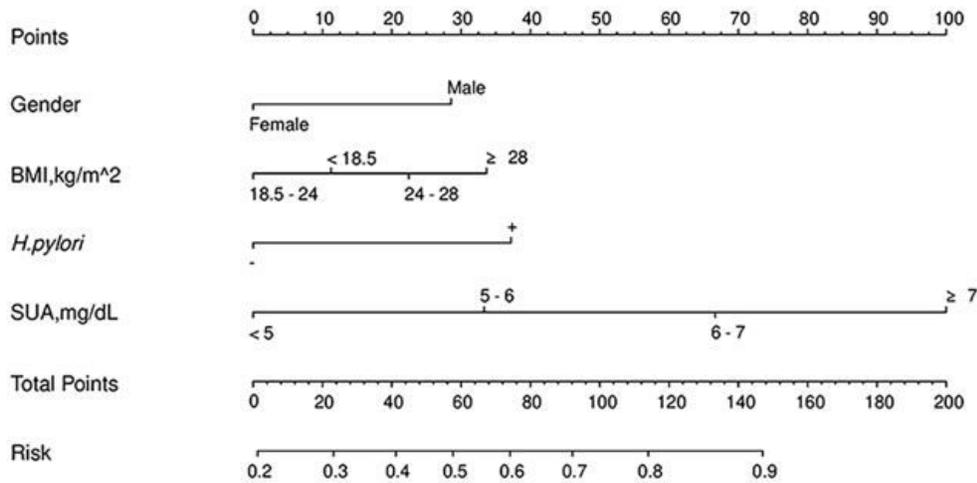


Figure 2

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A



B

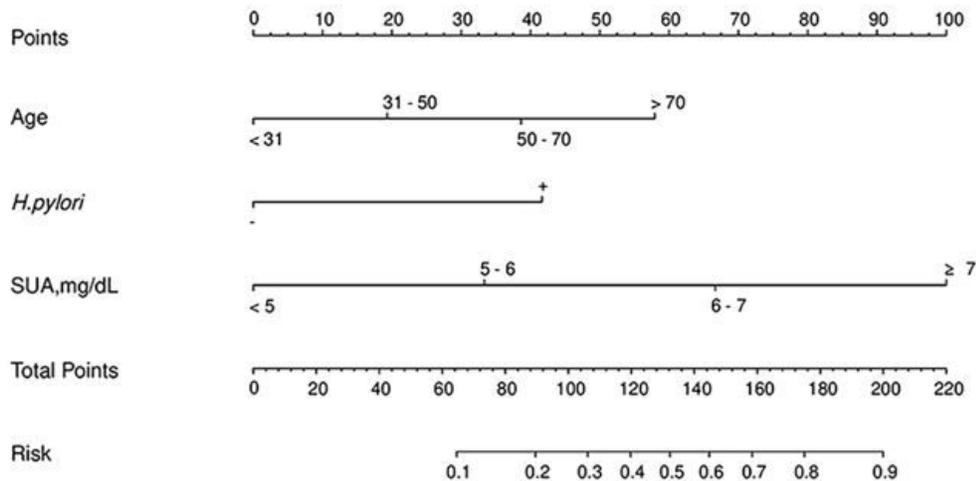


Figure 3

Nomogram Model to Predict the Risk of Gastric Precancerous Lesion and Gastric Cancer The scores corresponding to different values of each factor and risk of each factor are shown in this figure. The total scores of each factor are calculated. The total scores range from 0 - 200, 0 - 220, respectively. And the corresponding risk rates range from 0.2 - 0.9, 0.1 - 0.9, respectively. The higher the total score, the greater

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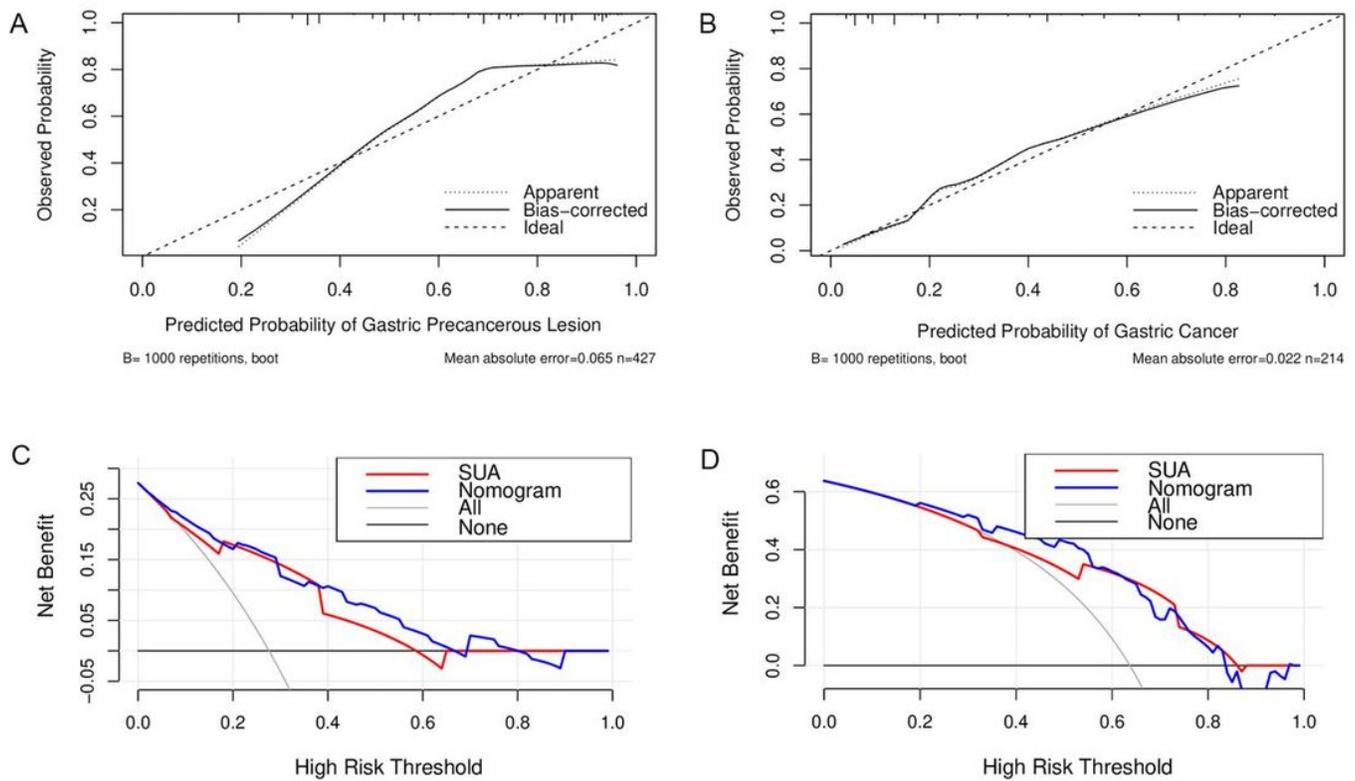


Figure 4

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