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Prognostic significance of excision repair cross complementation group 1 rs2298881 in patients with gastric cancer receiving platinum-based chemotherapy

A PRISMA-compliant meta-analysis

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Abstract

Background: Gastric cancer (GC) is a strong cause of global cancer mortality. Nucleotide excision repair (NER) can modulate platinum-based chemotherapeutic efficacy by removing drug-produced DNA damage. Some studies have found a link between excision repair cross complementation group 1 (ERCC1) rs2298881, one gene in NER pathway, and response to chemotherapy. However, the results have been disputed.

Methods: We conducted a meta-analysis to reevaluate the association between polymorphisms of NER gene (ERCC1 rs2298881) and the clinical outcomes in gastric cancer patients receiving platinum-based chemotherapy. Searching PubMed, Web of Science, EMBASE, Google Scholar, and China National Knowledge Infrastructure, 2 independent searchers found all pertinent literatures up to May 1, 2021. We enrolled studies according to consistent selection criteria, extracted and vitrified data. Crude odds ratios (ORs) and hazard ratios (HRs) with 95% confidence interval (CI) were applied to evaluate the effect of ERCC1 rs2298881 on patients treated by platinum-based chemotherapy.

Results: By the data gathered from 6 independent studies, 1940 cases diagnosed with gastric cancer and treated with chemotherapy were included, containing 1208 Good-Responders and 732 Poor-Responders. With a comprehensive meta-analysis, we found that the patients with ERCC1 rs2298881A allele had a worse response to chemotherapy than those who with rs2298881C allele under allelic model (A vs C), with the pooled OR of 0.780 (95% CI: 0.611–0.996, $P = .046$). And our analysis indicated that AA genotype was associated with unfavorable overall survival (HR = 1.540, 95% CI = 1.106–2.144, $P = .011$) compared with CC genotype.

Conclusions: ERCC1 rs2298881 is suggested as a marker of clinical outcome in gastric cancer patients treated by platinum-based chemotherapy.

Abbreviations: CI = confidence interval, ERCC1 = excision repair cross complementation group 1, GC = gastric cancer, HR = hazard ratio, NER = nucleotide excision repair, OR = odds ratio.

Keywords: chemotherapy, gastric cancer, meta-analysis, single nucleotide polymorphisms

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YL, MX, and YS contributed equally to this work.

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All data generated or analyzed during this study are included in this published article [and its supplementary information files].

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1. Introduction

Gastric cancer (GC) is one of the major causes of death among the common cancers of digestive system.[1] Despite numerous studies to find more ways to treat gastric cancer, it's been the leading cause of cancer death worldwide.[2] Surgical resection is a recommended treatment for most gastric cancer patients diagnosed as early gastric cancer.[3] For those who undergo surgery, adjuvant treatment with chemotherapy (and sometimes radiation) may help them lower the risk of recurrence.[4] And for advanced diseases, chemotherapy (sometimes along with a targeted therapy drug)[5] may lengthen survival in a certain degree. Platinum-based chemotherapy as a first-line treatment regimen for advanced gastric cancer could make a positive effect on the prognosis.[6] Unfortunately, response rates to platinum-based chemotherapy range in a degree of 30% to 50% all the time.[7] Thus, there is an urgent need for potential biomarkers to make predictions about the response to chemotherapy. At the same time, molecular markers are valuable for doctors to make individual treatments for gastric cancer patients.

Nucleotide excision repair (NER), as a versatile DNA repair system, can identify and defend against DNA damage induced by exogenous carcinogen such as platinum.[8] Many studies indicated it has clinical application value in using mRNA or protein levels of NER to predict response to chemotherapy and potentiality in determining resistance simultaneously.[9] Considering the function of NER in response to exogenous toxins, excision repair cross-complementing 1 (ERCC1), one key gene of NER, has been proposed to be correlated with platinum-based therapy efficacy in ovarian cancer, colorectal cancer, and gastric cancer.[10–12] Many investigations have been conducted to access the impact of single nucleotide polymorphisms of ERCC1 on predicting response to chemotherapy and survival in subjects living with gastric cancer.[13–15] Among them, some studies indicated ERCC1 rs2298881 may be useful to predict clinical outcomes of platinum-based chemotherapy in gastric cancer and contribute to the future design of individualized anti-cancer therapies for patients though the results remain controversial.[16,17]

To further understand such correlation, we performed the first meta-analysis, as far as we know, to measure the impact of ERCC1 rs2298881 on clinical outcomes in gastric cancer patients receiving platinum-based chemotherapy with a full use of previously published articles.

2. Materials and methods

Ethical approval statements are not applicable since the present study is a Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA)-compliant meta-analysis and only collates the related literature research.

2.1. Literature and study acquisition

Literature and study acquisition were finished following PRISMA.[18] The following databases were used for potentially related articles that were published before May 1, 2021: PubMed, Google Scholar, EMBASE, Web of Science, and China National Knowledge Infrastructure databases. The search terms included: "platinum or cisplatin or oxaliplatin," "chemotherapy or treatment or therapy or regimen," "gastric cancer or gastric neoplasm or stomach neoplasm," and "excision repair cross-complementation group 1 or ERCC1." Eligibility criteria were as follows: patients were pathologically proven gastric cancer and underwent platinum-based chemotherapy; Cohort study; available data for genotype distribution, objective response rate (ORR) and overall survival (OS). The exclusion criteria were followed: academic conference papers, letters, meta-analysis and systematic reviews; overlapping data; The New Castle-Ottawa Scale (NOS) scores <6.

2.2. Data extraction

Two investigators retrieved the published articles, abstracted the information including publication year, the first author's name, ethnicity, age, sex, TNM stage, tumor histology, sample size, genotypes, clinical outcomes (ORR, OS, and HR with corresponding 95% CIs) and the data of responders and non-responders to chemotherapy and filled out a unified data extraction form. When datasets were not accessible or partial for the requisite data, corresponding authors were connected for more additional information. Gathered information from the included studies was listed in Table 1.

2.3. Quality assessment

The NOS, as an evidence-based tool, was used for each study to appraise the quality.[19] Literature quality scores were obtained from 3 aspects: selection, comparability, and exposure information.

2.4. Choice of genetic model

The rs2298881 polymorphism owns wild-type C allele and variant A allele.[20] We plan to investigate the relationship between rs2298881 polymorphism and platinum-based chemotherapeutic efficacy by utilizing allele model (A vs C), dominant model (AA +

Table 1

Characteristics of the studies included in the meta-analysis for ERCC1 rs2298881 polymorphism at responses of platinum-based chemotherapies in gastric cancer.

				Gender	TNM stage	Tumor histology				Sample size		
First author	Year	Ethnicity	Age	Female/male	I–II/III–IV	Intestinal	Diffuse	Other	Genotypes	Responders	Non-responders	NOS
Chen ZH[26]	2014	Chinese	55.7 ± 9.3	85/170	100/155	142	104	9	AA/AC/CC	156	99	7
Lu ZM[17]	2014	Chinese	55.1 ± 8.4	146/301	42/405	162	172	113	AA/AC/CC	304	143	6
Li J[25]	2014	Chinese	57.1 ± 10.6	103/223	142/184	171	135	20	AA/AC/CC	195	131	6
Zhou J[21]	2015	Chinese	56.2 ± 15.6	143/272	152/263	179	189	47	CC/AC/AA	238	177	8
Yu H[23]	2015	Chinese	55.7 ± 13.8	91/137	81/147	104	88	36	AA/AC/CC	89	139	9
Bai Y[22]	2015	Chinese	64.3 ± 9.1	103/167	155/115	160	110	0	CC/AC/AA	176	93	7

NOS = New Castle-Ottawa Scale.

AC vs CC), recessive model (AA vs AC + CC), heterozygous model (AC vs CC) and homozygous model (AA vs CC).

2.5. Statistical analysis

We computed odds ratios (ORs) with 95% confidence interval (CI) by STATA 11.0 (Stata Corporation, College Station, TX, USA) to assess the absolute impact of ERCC1 rs2298881 on platinum-based chemotherapy under 5 genetic models. For OS, the hazard risks (HRs) and CIs extracted from the raw data of the included articles were calculated to estimate the pooled HRs and 95% CIs in the homozygote genetic model. The heterogeneity among studies was evaluated using the I-squared and the Chi-square test based on Cochran Q statistic. The random effects model was used for meta-analysis when results showed the high between-study heterogeneity ($P < .05$ or $I^2 \geq 50\%$). Otherwise, the fixed effects model was applied. Sensitivity analysis accessed the effect of an individual study on the pooled estimates. Begg test and Egger test suggested statistically significant publication bias ($P < .05$). When necessary, subgroup analyses can be employed.

3. Results

3.1. Results of the literature retrieval and the basic characteristics of the studies

The literature search and selection process are filled in Fig. 1. A total of 683 articles were retrieved. There were 619 records left

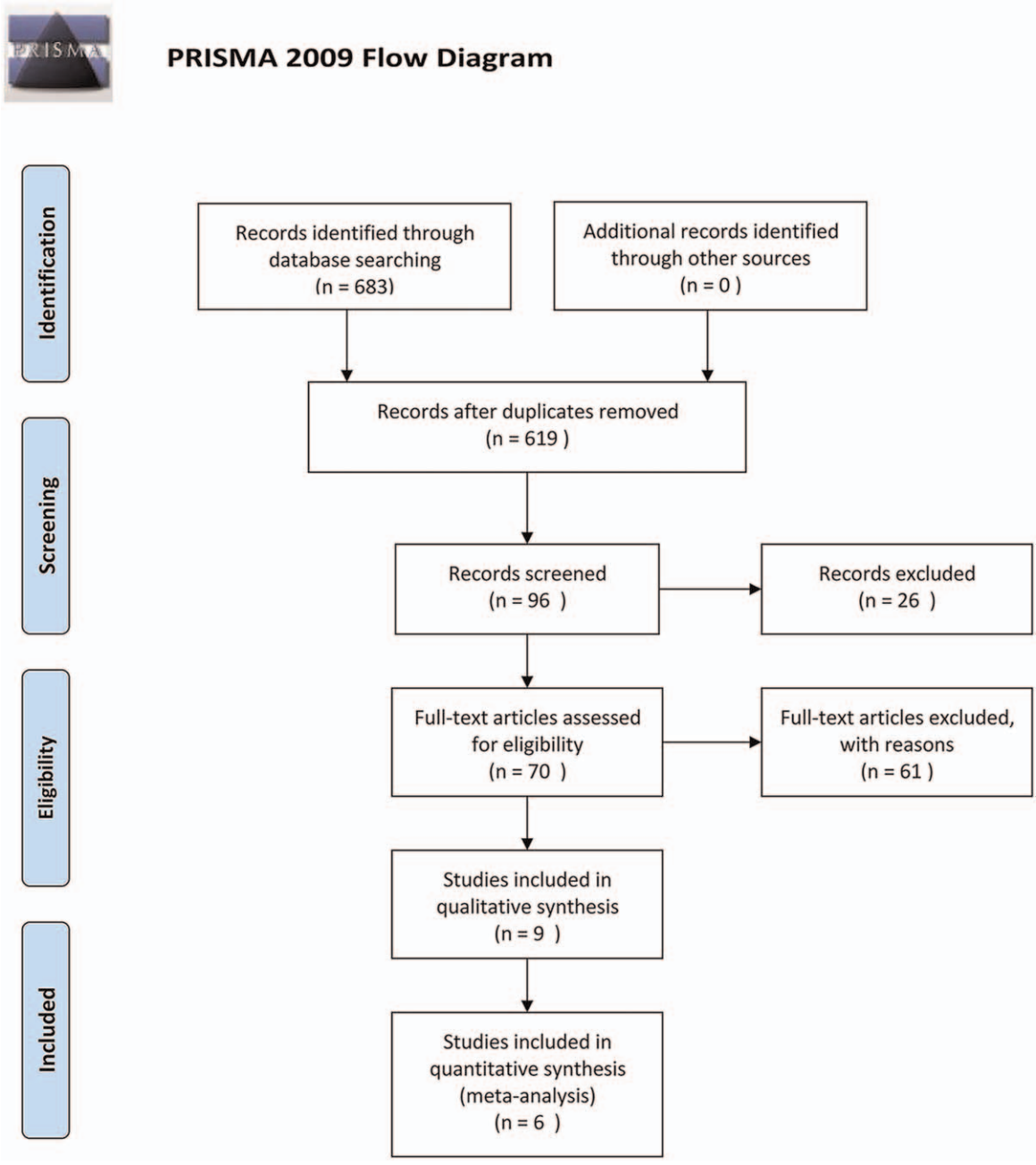

Figure 1. Flow diagram for study selection.

Table 2

Meta-analysis of the association between ERCC1 rs2298881 polymorphism and chemotherapy in tumor response and overall survival for gastric cancer patients.

Genetic comparisons	Test of association		Model	Test of heterogeneity		P_{Egger}	P_{Begg}
	OR/HR (95% CI)	*P*		*P*	I^2 (%)		
Tumor response (OR)							
A *vs.* C	0.780 (0.611, 0.996)	0.046	R	0.028	60.1	0.435	1.000
AC *vs.*CC	0.837 (0.640, 1.096)	0.195	F	0.553	0.0	0.172	0.260
AA *vs.*CC	0.642 (0.474, 0.869)	0.004	F	0.191	32.6	0.555	1.000
AA *vs.* AC+CC	0.764 (0.615, 0.949)	0.015	F	0.223	28.3	0.909	1.000
AA+AC *vs.*CC	0.762 (0.599, 0.970)	0.027	F	0.148	38.7	0.207	0.260
Overall survival (HR)							
AA *vs.*CC	1.540 (1.106,2.144)	0.011	F	0.399	2.7	0.825	1.000

OR = odds ratio, HR = hazard ratio, CI = confidence interval, *vs* = *versus*, F = fixed effect model, R = random effect model.

after removing duplicate records. Twenty six studies were excluded according to title review. And the left 70 studies were for abstract review. We excluded 52 articles because they were reviews or meeting abstracts and 9 articles for no relevance to platinum-based chemotherapy. Amongst the 9 articles left, 3 articles were not available after full-text review. One article could not be found valid data. Two studies were excluded because their sample had been overlapped with other studies. Consequently, 6 articles were enrolled following our inclusion criteria. Table 1 described 6 eligible studies' characteristics including 1940 gastric cancer patients.[21–26] All included studies had high quality (NOS scores > 5) based on the NOS scale and reported ORR, OS, and HR.

As significant heterogeneity was found in the allele model ($P = .028$ and $I^2 = 60.1\%$), we applied the random-effects model to conduct the meta-analysis. And a positive result was found that the response to platinum was related with genetic variation in ERCC1 rs2298881 (OR = 0.780; 95% CI: 0.611–0.996, $P = .046$) (Table 2, Fig. 2). While no significant heterogeneity was observed in other genetic models ($P > .05$ and $I^2 < 50\%$), we chose a fixed-effects model for the statistical analysis. By pooling the data in 6 studies, a significant association was also observed in the AA+AC versus CC (OR = 0.762; 95% CI, 0.599–0.970; $P = .027$), AA versus AC+CC (OR = 0.764; 95% CI, 0.615–0.949; $P = .015$), and AA versus CC (OR = 0.642; 95% CI, 0.474–0.869; $P = .004$) (Table 2, Fig. 3). No significant difference between the different genotypes was found in the AC versus CC (OR = 0.837; 95% CI, 0.640–1.096; $P = .195$) (Table 2, Fig. 3). We tried to find the studies with different ethnicities but we failed. Finally, subgroup analysis could not be conducted on it. For OS, the results of the meta-analysis showed a significant association between the ERCC1 rs2298881 polymorphism and OS in AA versus CC (HR = 1.540; 95% CI, 1.106–2.144; $P = .011$). OS forest plot for AA versus CC is shown in Fig. 4.

3.2. Sensitivity analysis, and publication bias

The stability and reliability of the summary effects were evaluated by sensitivity analysis (Figs. 5–7). The results suggested that the

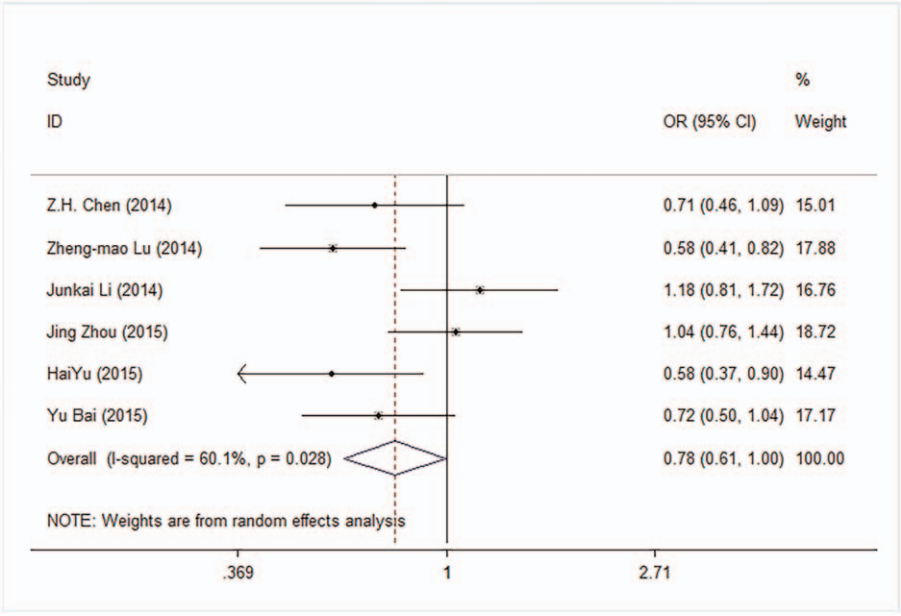

Figure 2. Forest plots for the association between ERCC1 rs2298881 expression and response to platinum-based chemotherapy in patients with gastric cancer including A versus C. ERCC1 = excision repair cross complementation group 1.

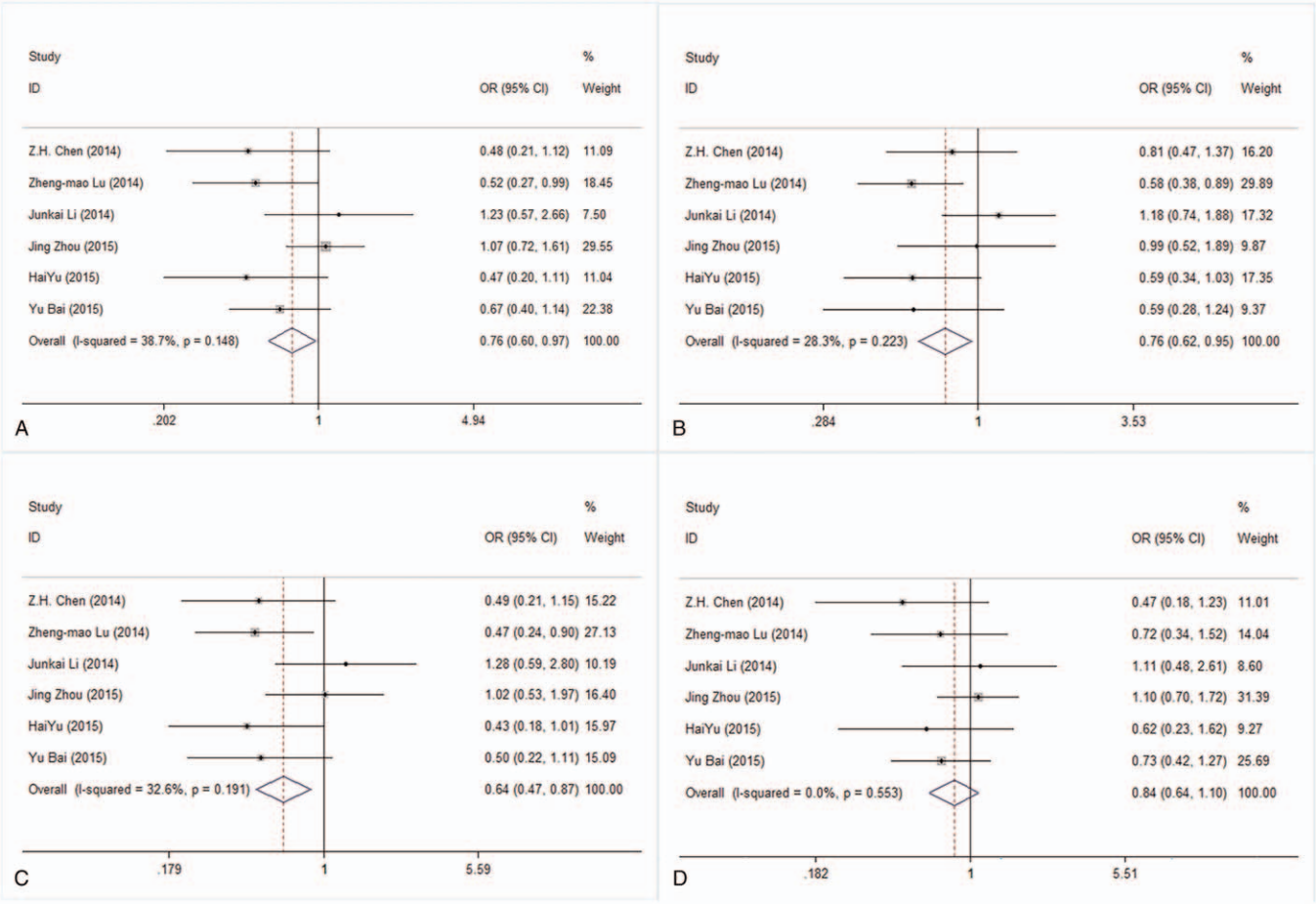

Figure 3. Forest plots for the association between ERCC1 rs2298881 expression and response to platinum-based chemotherapy in patients with gastric cancer including (A) AA+AC versus CC and (B) AA versus AC+CC, (C) AA versus CC, and (D) AC versus CC. ERCC1 = excision repair cross complementation group 1.

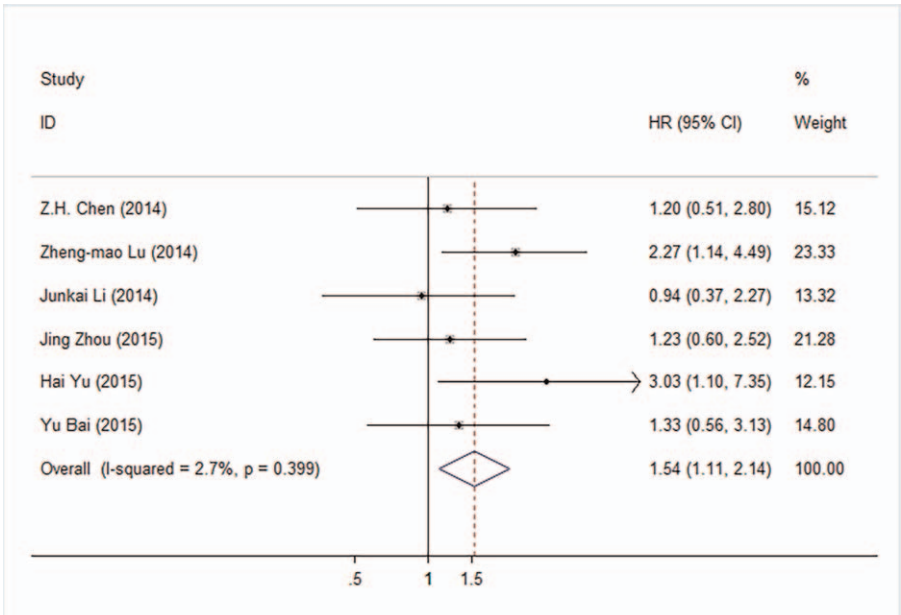

Figure 4. Forest plot of rs2298881 OS (AA vs CC).

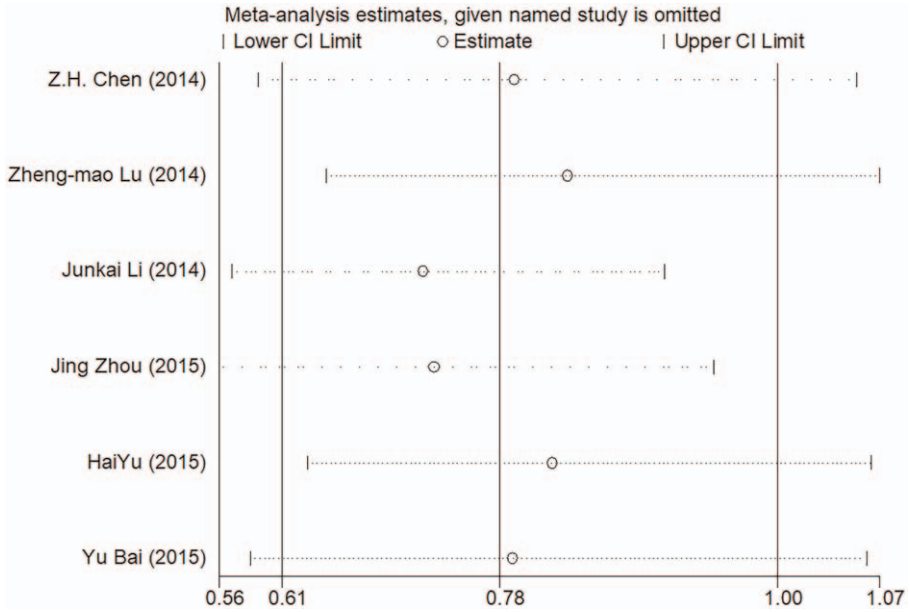

Figure 5. Plots of sensitivity analyses by omitting one study at a time including A versus C.

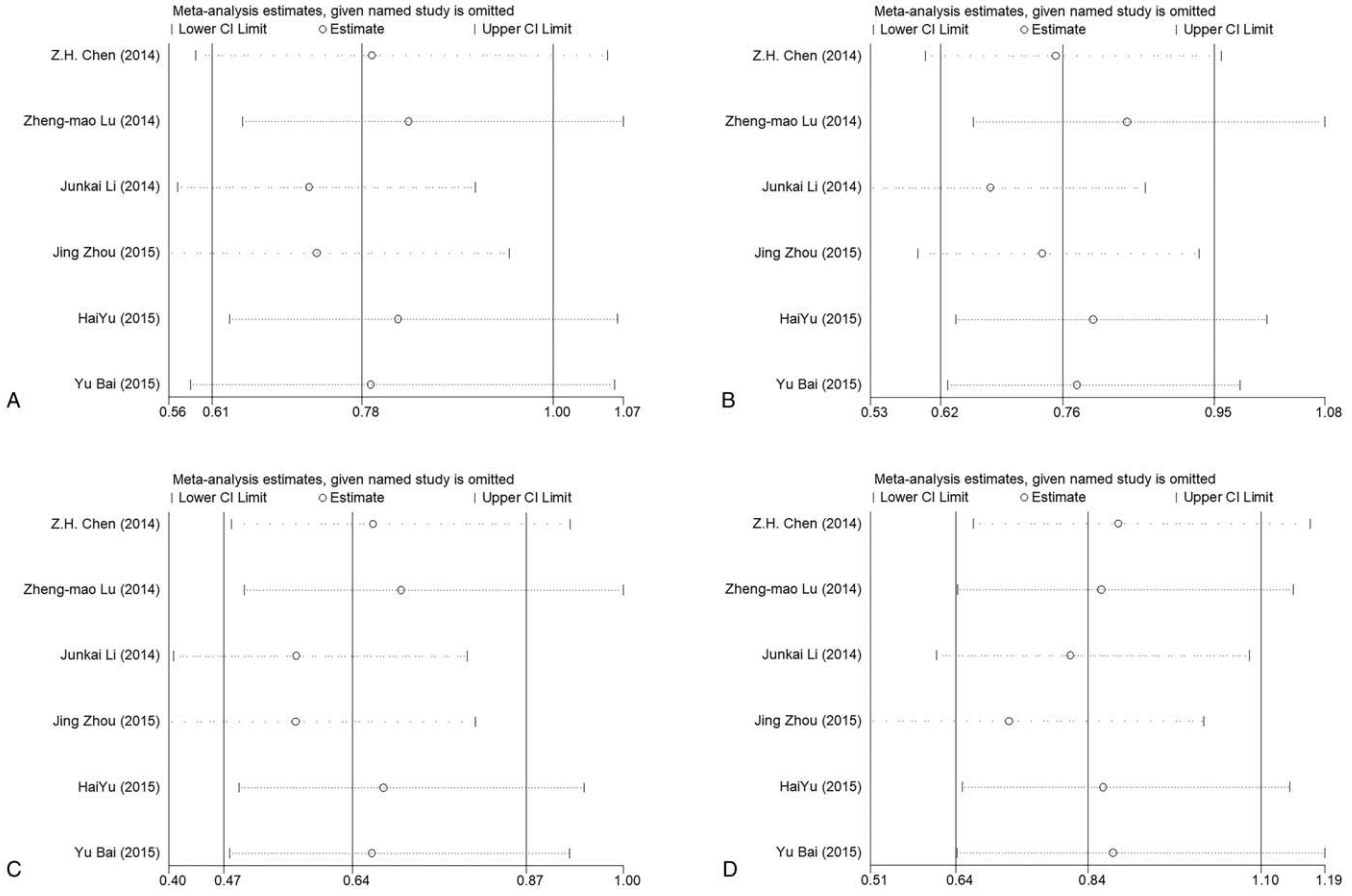

Figure 6. Plots of sensitivity analyses by omitting one study at a time including (A) AA+AC versus CC and (B) AA versus AC+CC, (C) AA versus CC, and (D) AC versus CC.

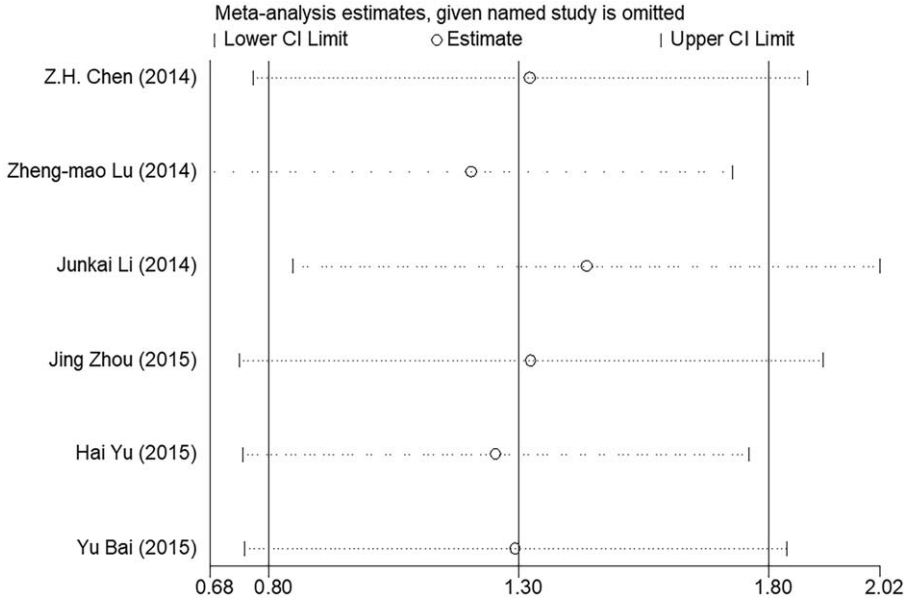

Figure 7. Sensitivity analysis of rs2298881 OS (AA vs CC). OS = overall survival.

conclusions were stable and reliable. And we computed the potential publication bias with Egger test and Begg test. The data showed a satisfactory result to us, there was no obvious publication bias in our study (Table 2).

4. Discussion

Gastric cancer is a common malignant tumor with high mortality. Without specific clinical symptoms in the gastric cancer's early stage, many patients have been with advanced gastric cancer when they are first diagnosed. Chemotherapy can significantly improve patients' quality of life and prolong their survival. It has been found that some DNA damage repair in individual genome sequences can deal with gene mutations and inhibit drug resistance to chemotherapy. NER pathway is the main way to repair DNA damage induced by chemotherapy.[27] As the research moves along, the study of gastric cancer genomics has gradually entered the field of vision. In recent years, more and more important biomarkers related to NER pathway have been found. A recent study revealed that USF1 as a central regulator of NER in response to *Helicobacter pylori* infection play a significant role in gastric carcinogenesis and may be a potential maker of GC susceptibility.[28] Moreover, ERCC1 is a key gene of NER and a study in Croatia declared that positive ERCC1 expression means a worse prognosis for patients with chemotherapy through retrospectively investigating 142 cases of lung adenocarcinoma.[29] It prompts that ERCC1 polymorphism as a genetic factor may play a role in response to chemotherapy though NER pathway. ERCC1 rs2298881 was reported to be correlated with the chemotherapy effects. And it has been proven in several human cancers such as osteosarcoma,[30,31] non-small cell lung cancer,[32–34] and ovarian cancer.[35] Most studies suggested ERCC1 rs2298881 could be used as a valuable biomarker of response to chemotherapy.

However, results remain conflicting in different studies. A single study might be limited by a small sample size. Furthermore, so far there has not been a summative article reporting a sufficiently convincing result to cover all available samples. Covering all previous literatures, we finished the meta-analysis to determine the precise role of ERCC1 rs2298881 in chemotherapy. The studies enrolled 6 studies with 1940 gastric cancer patients. Our meta-analysis results indicated that the patients with rs2298881A allele was an adverse factor while C allele was a protective factor to patients underwent chemotherapy under 4 models except the heterozygous model.

We conducted the meta-analysis comprehensively to combine their inconsistent findings on the same research subject. Despite our efforts in the meta-analysis, this study had a few limitations. Firstly, only 6 articles were involved in the meta-analysis so the comprehensiveness might be impacted. Secondly, this was a meta-analysis utilizing the data from the published studies and individual patient data were not available. Thus, we were unable to assess the effect of the clinical characteristics of the subjects on the outcomes. Thirdly, subgroup analysis can't be performed by ethnicity because all articles were performed in China. Therefore, perspective studies with a larger sample size and more various ethnicities were expected to validate the conclusions.

Consequently, it is fairly important to support more research efforts on basic research and clinical translation about gastric cancer, so as to perform different treatments according to different genotypes and achieving better prognosis and longer survival rates with individualized treatment options. The purpose of this study is exactly to be a supplement when we need the selection or adjustment of chemotherapy for clinical patients. This is the first meta-analysis comprehensively accessing impact of ERCC1 rs2298881 polymorphism on response to chemotherapy in gastric cancer patients. We hope that our findings will help clinicians and patients to select appropriate therapies for gastric cancer.

5. Conclusion

The meta-analysis shows the influence of ERCC1 rs2298881 polymorphism on response to platinum-based chemotherapy in gastric cancer relied on published data. Carriers with ERCC1

rs2298881A allele tend to a poor response to chemotherapy. And ERCC1 rs2298881 could be a predictive biomarker in chemotherapy response for gastric cancer in the future.

Author contributions

Conceptualization: Yidan Sun, Xiaolin Zhang.
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Data curation: Mengyuan Xu, Yidan Sun, Yezhou Liu, Lanfei Bi.
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Methodology: Yalei LV, Mengyuan Xu.
Performed the search: Yidan Sun, Mengyuan Xu, Yezhou Liu, Yalei Lv, Lanfei Bi.
Software: Yidan Sun, Yezhou Liu, Lijuan Zhao, Xuehui Liu, Gaiping Shi, Jinhai Jia.
Writing – original draft: Mengyuan Xu, Yidan Sun.
Writing – review & editing: Mengyuan Xu, Ning Ma, Cheng Qi.

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