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Prognostic significance of miR-378 in cancers: a meta-analysis

Lei Shi[1†], Jie Wei[2†], Yankun Shen[3†], Xiaoqiang Zhang[1], Lixin Li[1] and Ying Deng[1*]

Abstract

Background The prognostic significance of miR-378 in cancers remains controversial. We carried out the meta-analysis to clarify the issue.

Methods Related researches were obtained from PubMed, Web of Science and EMBASE. The search was conducted up until 10 September 2023. Hazard ratios (HRs) or odds ratios (ORs) with 95% confidence intervals (CIs) were calculated to access the correlation between miR-378 and survival outcomes and clinicopathological features.

Results 16 articles were included in the meta-analysis. The pooled analysis showed that low miR-378 expression predicted poor overall survival (OS) (HR: 1.73, 95% CI: 1.09–2.74). No significant difference was discovered between low miR-378 expression and disease-free survival/ recurrence-free survival (DFS/RFS) (HR: 1.43; 95% CI: 0.79–2.59). Subgroup analysis revealed that the low miR-378 expression had better predictive value for CRC (HR: 2.97, 95% CI: 1.27–6.96), GC (HR: 2.12, 95% CI: 1.23–3.67) and glioma (HR: 3.87, 95% CI: 1.52–9.81). In addition, low miR-378 expression had positive correlation with lymph node metastasis (yes vs. no)(OR: 2.42, 95% CI: 1.55–2.87) and distant metastasis (yes vs. no) (OR: 1.26, 95% CI: 1.04–2.35).

Conclusion MiR-378 could be an efficient prognostic biomarker in cancers.

Keywords miR-378, Prognosis, Cancer, Meta-analysis

Introduction

Cancer has become the leading cause of death in many regions [1]. In 2020, the quantity of cancer cases attained 19.3 million, nearly 10 million people died of cancer worldwide [2]. The cancer burden would continue to increase in the next 20 years [3].

MicroRNAs (miRNAs) are short RNA molecules with a size of 19–25 nucleotides that adjust post-transcriptional gene expression by integrating the 3' untranslated region (UTR) of mRNA [4]. An independent miRNA can affect hundreds of RNAs and the expression of genes [5]. Abnormal expression of miRNAs can interfere with the expression of oncogene or tumor suppressor target genes. Many miRNAs are down- or up-regulated in human cancers and play a role in promoting or inhibiting tumors [6]. MiR-378 is one of the most valuable miRNAs.

MiR-378 has situated in the intron of its gene peroxisome proliferator-activation receptor γ [7]. MiR-378 as a hinder in the mitogen-activation protein kinase pathway, affects extracellular signal-adjusted kinase genes, and participates in cell proliferation, differentiation,

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transcriptional regulation and development [8]. A study illustrated that.

miR-378 can target and inhibit the anti-proliferative TOB2 to achieve the oncogenic effect [9]. It was portrayed that the aberrant level of miR-378 was associated with the survival outcomes of many cancers [10–25]. However, their results were inconsistent. In this study, we carried out a meta-analysis to comprehensively explore the prognostic value of miR-378 in cancers.

Methods

Literature search

Three authors(Lei Shi, Xiaoqiang Zhang and Yankun Shen) conducted a comprehensive search of the PubMed, Embase, and Web of Science databases to identify articles investigating the association between miR-378 and prognosis of cancer patients.The search was conducted up until 10 September 2023. The searching strategy applied the following clauses: 'miR-378' or 'microRNA-378' or 'miRNA-378' and'cancer' or 'carcinoma' or 'tumor' or 'tumour' or 'neoplasm' or 'malignancy' and 'prognosis' or 'survival' or 'prognostic' or 'outcome. There were no language restrictions. The references for the selected studies were carefully examined for possible studies.This analysis followed the PRISMA guidelines(Supplementary material).

Inclusion and exclusion criteria

Articles that met the following criteria were included: (1) explored the correlation between miR-378 expression and prognosis of cancer patients. (2) provided adequate data to calculate hazard ratios (HRs) or odds ratios (ORs) with 95% confidence intervals (CIs). Studies that met the following standard were eliminated: (1) reviews, conference abstracts, animal investigations and deficient data. (2) duplicate publications and data from public databases.

Data extraction and quality assessment

Two independent researchers collected all the data. The following data was collected: author name, year of publication, country, study design, tumor type, sample size, detected sample, detected method, analysis type, survival analysis and source of HR. For studies that did not directly provide survival data, we extracted it from survival curves [26]. Newcastle-Ottawa Quality Assessment Scale (NOS) was utilized to evaluate the quality of each article [27].

Statistical analysis

All data analysis was performed by STATA 12.0 software (STATA Corporation, College Station, TX, USA). The heterogeneity was assessed by the I^2 statistics. $I^2 < 50$ was considered as slight heterogeneity, and the fixed effect model was adopted. Otherwise, the random-effects model was used. The stability of the meta-analysis was tested by sensitivity analysis. Begg's test, Egger's test and trim-and-fill method were applied to evaluate publication bias [28]. $P < 0.05$ indicated statistically significant.

Results

Literature selection

Articles about the prognostic value of miR-378 in cancers were collected from the databases. A total of 420 articles were initially identified. After expurgating duplicated articles and articles that did not meet the inclusion criteria, 16 articles were included in the study [10–25]. The flow chart was displayed in Fig. 1.

Study characteristics

All incorporated studies were published between 2013 and 2021. They were from China, Germany and Greece, respectively. 13 different tumors were presented, including prostate cancer (PC), breast cancer (BC), glioblastoma (GBM), esophageal cancer (EC), hepatocellular carcinoma (HCC), renal cell carcinoma (RCC), glioma (GM), gastric adenocarcinoma (GC), non-small cell lung cancer (NSCLC), cholangiocarcinoma (CCA), oral squamous cell carcinoma (OSCC), acute myeloid leukemia (AML) and colorectal cancer (CRC). Survival outcomes such as overall survival (OS) disease-free survival (DFS), recurrence-free survival (RFS) were reported.The NOS scores of the studies ranged from 5 to 8 (mean: 6.18). Detailed information was shown in Table 1.

Relationship between low miR-378expression and OS

Due to significant heterogeneity ($I^2 = 87.2\%$), we utilized a random effects model to compute the pooled HRs. The results revealed the low miR-378 was significantly related to the poor OS (HR: 1.73, 95% CI: 1.09–2.74)(Fig. 2).

Subgroup analysis and meta-regression

To further examine the predictive significance of miR-378, we conducted subgroup analysis and meta-regression based on cancer type, country, analysis type and sample size (Table 2). We discovered that low miR-378 expression was significantly related to unfavorable OS in the subgroups, such as CRC, GC, Glioma, China, univariate analysis and sample size<100. No significant difference was discovered in other subgroups.Meta-regression showed that cancer type may contribute to heterogeneity($P = 0.043$).

Relationship between low miR-378expression and DFS/RFS

5 studies reported the relationship between low miR-378expression and DFS/RFS. Owing to the significant heterogeneity ($I^2 = 75.6\%$), the random effect model was used. No significant difference was discovered between

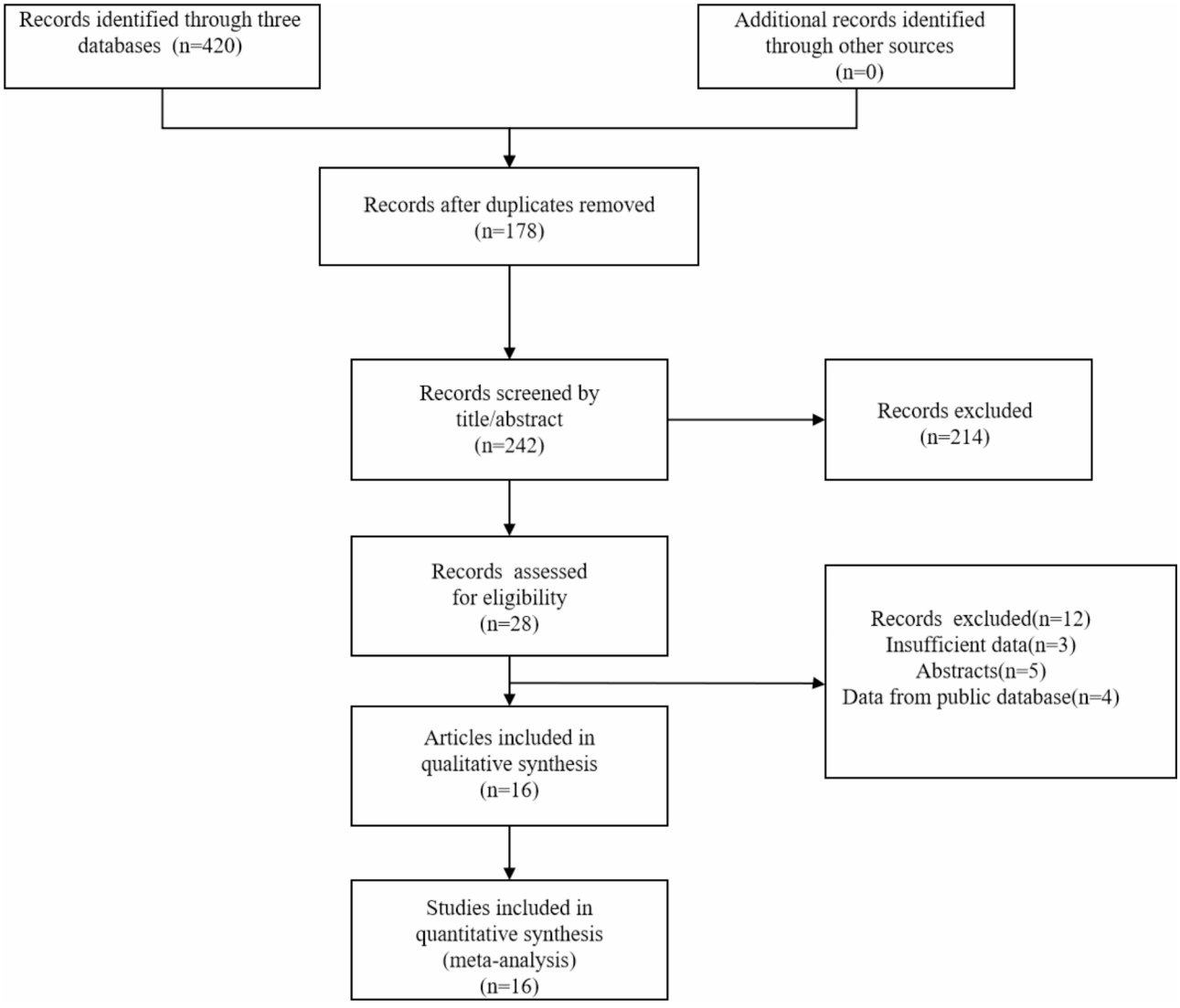

Fig. 1 Flow diagram of the literature search

low miR-378 expression and DFS/RFS (HR: 1.43; 95% CI: 0.79–2.59) (Fig. 3).

Relationship between low miR-378 expression and clinicopathological features

We gathered clinical data, including gender, age, tumor diameter, tumor stage and lymph node status to explore the correlation between the low miR-378 expression and clinicpathological characteristics (Table 3). The results showed that low miR-378 expression had positive correlation with lymph node metastasis (yes vs. no) (OR: 2.42, 95% CI: 1.55–2.87) and distant metastasis (yes vs. no) (OR: 1.26, 95% CI: 1.04–2.35).

Sensitivity analysis

Sensitivity analysis was performed by excluding individual study. The results were consistent with the comprehensive analysis, confirming that the outcomes of the OS and DFS/PFS were stable (Fig. 4A, B).

Publication bias

In order toassess publication bias, Begg's and Egger's tests were applied. The p-values of Begg's and Egger's tests for OS(Fig. 5A) were 0.029 and 0.001, indicating there existed publication bias. Therefore, the trim-and-fill method was applied. It was found that the publication bias did not influence the pooled analysis(HR:1.367, 95% CI: 1.01–2.23)(Fig. 5B). The p-values of Begg's and Egger's tests for DFS/RFS (Fig. 5C) were 0.462 and 0.638, suggesting that there was no publication bias.

Table 1 Basic information of all included articles

Study	Year	Country	Study type	Tumor type	Sample size	Detected sample	Detected method	Analysis type	Survival analysis	Source of HR	NOS score
Avgeris	2014	Greece	R	PC	73	tissues	PCR	Multivariate	DFS	Reported	6
Ding	2019	China	R	OSCC	96	tissues	PCR	Univariate	OS	SC	6
Zheng	2016	China	R	GC	87	tissues	PCR	Univariate	OS	Reported	5
Gong	2021	Germany	R	BC	103	tissues	PCR	Univariate	OS, DFS	Reported	7
Guo	2019	China	R	Glioma	53	tissues	PCR	Univariate	OS	SC	5
Jin	2021	China	R	EC	135	tissues	PCR	Multivariate	OS	Reported	6
Li	2015	China	R	Glioma	100	tissues	PCR	Multivariate	OS	Reported	6
Lin	2020	China	R	HCC	100	tissues	PCR	Univariate	OS, DFS	Reported	8
Pan	2019	China	R	RCC	45	tissues	PCR	Multivariate	OS	Reported	6
Qian	2013	China	R	AML	84	Serum	PCR	Univariate	RFS	SC	6
Shi	2018	China	R	Glioma	52	tissues	PCR	Univariate	OS	SC	5
Wang	2014	China	R	CRC	34	tissues	PCR	Univariate	OS	SC	6
Yang	2019	China	R	GC	50	tissues	PCR	Univariate	OS, DFS	SC	6
Zhang	2014	China	R	CRC	84	tissues	PCR	Multivariate	OS	Reported	7
Zhang	2020	China	R	NSCLC	103	Serum	PCR	Multivariate	OS	Reported	7
Zhou	2018	China	R	CCA	120	tissues	PCR	Multivariate	OS	Reported	7

Abbreviations: R, Retrospective; OS, overall survival; DFS, disease-free survival; RFS, relapse-free survival; SC, survival curve; PC, prostate cancer; BC, breast cancer; EC, esophageal cancer; HCC, hepatocellular carcinoma; RCC, renal cell carcinoma; GC, gastric adenocarcinoma; NSCLC, non-small cell lung cancer; CCA, cholangiocarcinoma; OSCC, oral squamous cell carcinoma; AML, acute myeloid leukemia; CRC, colorectal cancer

Discussion

Cancer morbidity and mortality were increasing at express speed. There were 2.7 million new cancers cases and 1.3 million death cases in 2020 [29]. Growing cancer burden reflected changes in the economy, society and lifestyle related to globalization and socio-economic development [30]. High miR-378 expression can promote tumor cell proliferation, angiogenesis and metastasis [31]. A study displayed that miR-378 promoted cancer growth and cell self-renewal [32]. In contrast, many studies found that low level of miR-378 could induce cancer cell proliferation, cell migration and invasion [33–35]. These contradictory consequences suggested that miR-378 acted as a duple character in cancers.

Our study was the first meta-analysis to explore the prognostic significance of miR-378 in cancers. The results discovered that low miR-378 expression was significantly related to the poor OS. Subgroup analysis revealed that the low miR-378 expression had better predictive value for CRC (HR: 2.97, 95% CI: 1.27–6.96), GC (HR: 2.12, 95% CI: 1.23–3.67) and glioma (HR: 3.87, 95% CI: 1.52–9.81). We also found that low miR-378 expression was related to poor prognosis in subgroup of China (HR: 1.92, 95% CI: 1.18–3.11). In addition, low miR-378 expression was significantly correlated with lymph node metastasis (yes vs. no) and distant metastasis (yes vs. no).

MiR-378 regulated tumor development in a variety of ways. MiR-378 can impede the invasion of colon cancer cells and the development of colon cancer cells by hindering the Wnt/β-catenin pathway [33]. MiR-378 can also play an anti-prostate cancer role by affecting multiple target genes [34]. In vitro, experiments suggested that high miR-378 expression can hinder the invasion of prostate cancer cells by mitogen-activated protein kinase 1 [35]. In HCC, miR-378 may block HCC development by regulating insulin-like growth factor 1 receptor 3'-UTR [36]. Furthermore, another study also found that the up-regulation of miR-378 can inhibit the proliferation of hepatocellular carcinoma cells and cancer growth through G2/M arrest [37]. Moreover, exogenous miR-378 can down-regulate the level of vascular endothelial growth factor and act a tumor suppressor character in GC by inhibiting cyclin-dependent kinases 6 and vascular endothelial growth factor signaling pathways [38]. The results indicated that miR-378 played different important roles in different tumors.

The study had some limitations. Firstly, data collected from the survival curves would not precisely represent the authentic standard. Secondly, the sample of the included studies was small. Thirdly, the most of the studies were from China, which may affect the generalization of the meta-analysis. Fourth, all included articles were retrospective studies.

The study also had merits. Firstly, it was the first meta-analysis to explore the relationship between miR-378 expression and prognosis of cancer patients. Secondly, the results of the meta-analysis were steady and credible. Thirdly, the trim-and-fill method revealed that the results for OS were unaffected by the publication bias.

In conclusions, our results indicated that low miR-378 expression predicted adverse survival outcomes in cancer patients. MiR-378 could be a promising prognostic

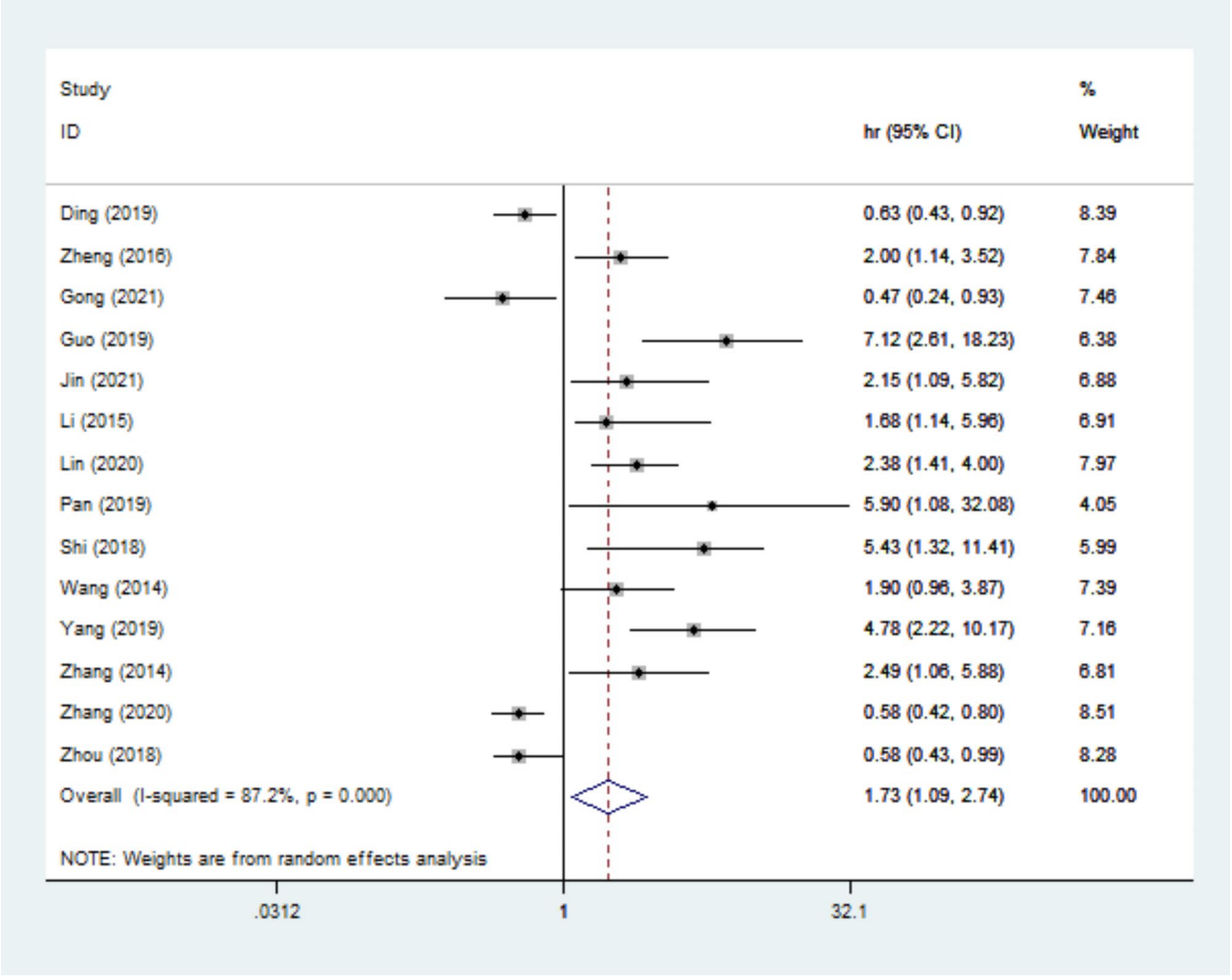

Fig. 2 Forest plot of the relationship between low miR-378 expression and OS

Table 2 Subgroup analysis and meta-regression for OS

Variables	No. of studies	Pooled HR (95%CI)	*P*-value	Heterogeneity			Meta-regression		
				I^2 (%)	P	Model	Tau2	Adj R2 (%)	P
Cancer type							0.448	34.81	0.043
CRC	2	2.97(1.27–6.96)	0.012	69.2	0.071	Fixed			
GC	2	2.12(1.23–3.63)	0.007	0	0.631	Random			
Glioma	3	3.87(1.52–9.81)	0.004	65	0.058				
Others	7	0.96(0.58–1.59)	0.889	83.9	<0.01	Fixed			
Country							0.62	10.06	0.142
China	13	1.92(1.18–3.11)	0.008	87.4	<0.01	Random			
Germany	1	0.47(0.24–0.93)							
Analysis type							0.7	-2.12	0.46
Multivariate	6	1.31(0.71–2.42)	0.388	81.2	<0.01	Random			
Univariate	8	2.05(1.06–3.98)	0.034	88.5	<0.01	Random			
Sample size							0.55	23.43	0.050
>100	6	1.01(0.57–1.79)	0.974	85.2	<0.01	Fixed			
<100	8	2.75(1.36–5.54)	0.005	86	<0.01	Random			

Abbreviations: GC, gastric adenocarcinoma; CRC, colorectal cancer

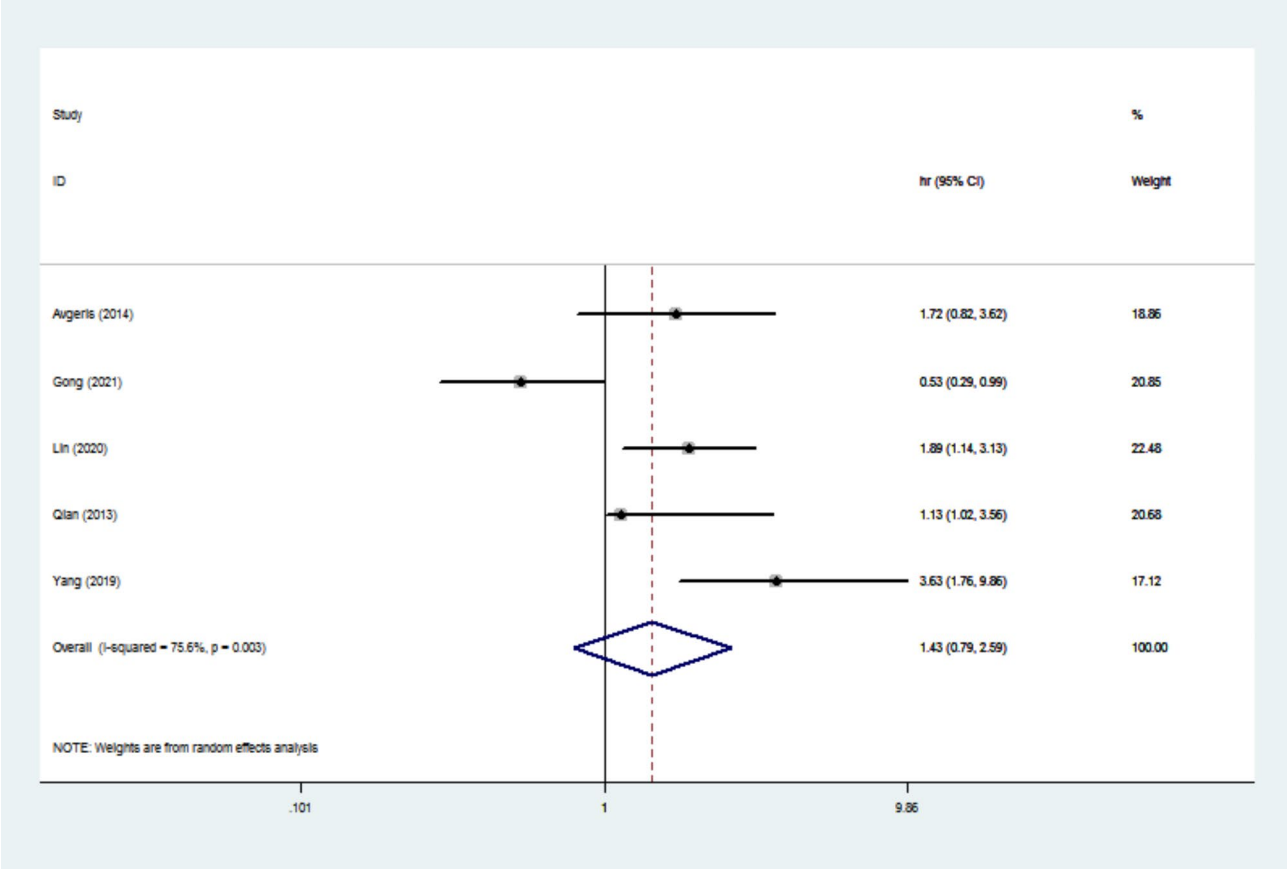

Fig. 3 Forest plot of the relationship between low miR-378 expression and DFS/RFS

Table 3 Relationship between low miR-378 expression and clinicopathological features

Clinicopathologic features	No. of studies	Estimate OR (95%CI)	*p*-value	Heterogeneity		
				I^2(%)	*p*-value	Model
Gender (Male VS. Female)	9	0.92(0.65–1.45)	0.75	0	0.843	Fixed
Tumor differentiation (Poor VS Moderate/Well)	5	1.68(0.64–4.53)	0.275	76.9	0.04	Random
Lymph node metastasis (Yes VS No)	6	2.42(1.55–2.87)	0.003	28.6	0.204	Fixed
Distant metastasis (Yes VS No)	4	1.26(1.04–2.35)	0.35	33.2	0.76	Random

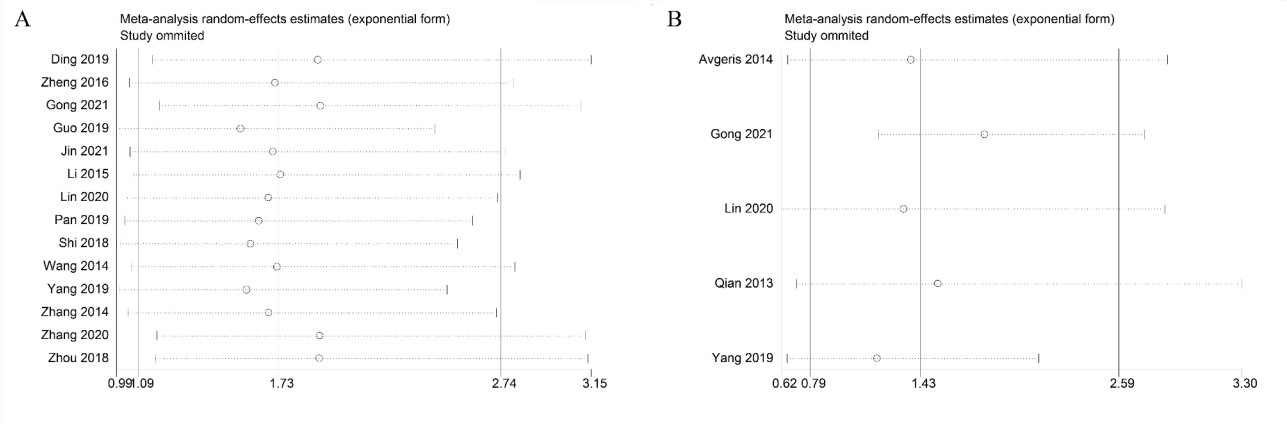

Fig. 4 Sensitivity analysis. (**A**) Sensitivity analysis for OS. (**B**) Sensitivity analysis for DFS/RFS

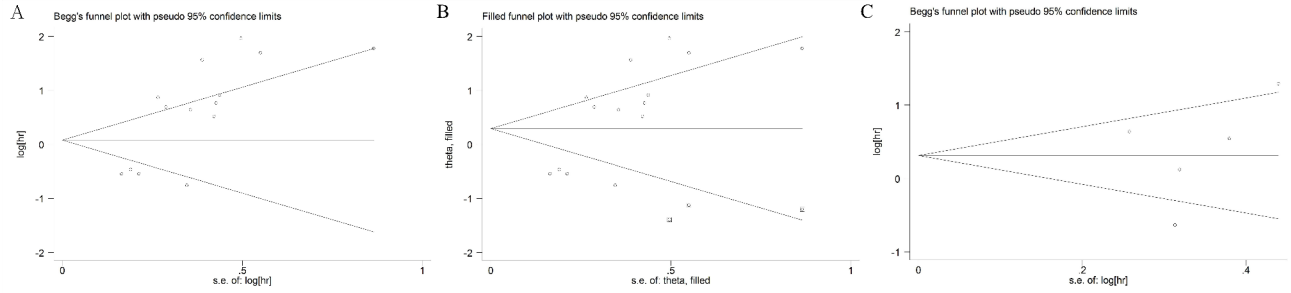

Fig. 5 Publication bias. (**A**) Publication bias for OS. (**B**) Trim and fill method to evaluate OS data. (C)Publication bias for DFS/RFS

marker in cancers, especially for CRC, GC and Glioma. Due to the limitations, future prospective studies were necessary to verify our findings and further assess the relationship between miR-378 and cancers.

Abbreviations

R	Retrospective
OS	overall survival
DFS	disease-free survival
RFS	relapse-free survival
SC	survival curve
PC	prostate cancer
BC	breast cancer
EC	esophageal cancer
HCC	hepatocellular carcinoma
RCC	renal cell carcinoma
GC	gastric adenocarcinoma
NSCLC	non-small cell lung cancer
CCA	cholangiocarcinoma
OSCC	oral squamous cell carcinoma
AML	acute myeloid leukemia
CRC	colorectal cancer

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12885-025-13619-w.

Supplementary Material 1

Author contributions

Ying Deng contributed to the study inception and design. Lei Shi, Jie Wei and Yankun Shen equally contributed to the literature search, analysis and writing of the manuscript. Xiaoqiang Zhang and Lixin Li contributed to the study design and study supervision. All authors approved the final version of the manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Not applicable.

Competing interests

The authors declare no competing interests.

Patient consent for publication

Not applicable.

Abbreviations

GC, gastric adenocarcinoma; CRC, colorectal cancer.

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