Am J Cancer Res 2022;12(9):4428-4438
www.ajcr.us /ISSN:2156-6976/ajcr0146232

Original Article

High expression of SMARCC1 predicts poor prognosis in gastric cancer patients

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Received August 30, 2022; Accepted September 14, 2022; Epub September 15, 2022; Published September 30, 2022

Abstract: The switching/sucrose non-fermenting (SWI/SNF) chromatin remodeling complexes use the energy of ATP hydrolysis to remodel nucleosomes and modulate transcription, which plays an important role in tumors by regulating epigenetics. SWI/SNF Related, Matrix Associated, Actin Dependent Regulator of Chromatin, Subfamily C, Member 1 (SMARCC1) has dual roles in tumors but its role in gastric cancer remains unclear. This study was aimed to find the role of SMARCC1 in gastric cancer. SMARCC1 expression across various tumors from The Cancer Genome Atlas was analyzed using TIMER 2.0 (http://timer.comp-genomics.org/). SMARCC1 mRNA expression profiles in gastric cell lines and gastric tissues were compared with normal tissues and analyzed in the Cancer Cell Line Encyclopedia, Oncomine, and Gene Expression Omnibus databases. SMARCC1 mRNA and protein were then examined in fresh gastric cancer tissues and compared with adjacent normal tissues using quantitative real-time PCR, western blotting, and immunohistochemistry. Associations between SMARCC1 expression and clinicopathological factors, overall survival, and disease-free survival were further evaluated using 130 gastric cancer samples harvested from patients after radical total gastrectomy or subtotal gastrectomy at the Xiangya Hospital of Central South University (Changsha, China). SMARCC1 was frequently upregulated in gastric cancer cells and tissues. SMARCC1 overexpression was significantly associated with tumor size (P=0.002), differentiation (P=0.006), depth of invasion (P=0.001), lymph node involvement (P=0.016), and TNM stage (P=0.007). Furthermore, univariate and multivariate Cox analysis revealed that high SMARCC1 expression, depth invasion, lymph node involvement, and TNM stage were independent risk factors for both overall and disease-free survival in gastric cancer patients (all P<0.05). Kaplan-Meier survival analysis revealed that high SMARCC1 expression predicted poor prognosis in gastric cancer patients (P<0.01). High SMARCC1 expression contributes to poor prognosis in gastric cancer patients. SMARCC1 may be a prognostic biomarker and therapeutic target in gastric cancer.

Keywords: SMARCC1, gastric cancer, prognosis

Introduction

Gastric cancer has the fifth highest incidence rate among all cancers and is the third leading cause of cancer-associated mortality worldwide. It is one of the most lethal malignancies, with over one million people diagnosed every year [1]. Although advances have been made in diagnostic and therapeutic techniques over recent decades, the mortality rate of gastric adenocarcinoma is high and global 5-year survival rates remain unsatisfactory [2], primarily due to metastatic progression. Cancer metastasis is a complex multi-cascade process that unfolds over many biological scales, including molecular signaling networks, protein-protein interactions, metabolism, cell-cell and cell-extracellular matrix interactions, organ-level control, disease manifestations, and epidemics [3].

SMARCC1 functions as a helicase and ATPase to regulate the transcription of certain genes by changing the chromatin structure around these genes, and together with SMARCA4, SMARCA2, and SMARCB1 is a member of the SWI/SNF protein family [4]. Evidence from SWI/SNF-mutant cancers suggests that aberrant activation of gene programs that are involved in cell motility may contribute to invasion and metastasis. SWI/SNF complexes have roles regulating the actin cytoskeleton, and dysregulation of these

pathways has been identified in SNF5-deficient rhabdoid tumors [5]. The mammalian SWI/SNF chromatin remodeling complex has been implicated in a variety of processes including mitosis, DNA replication, DNA damage repair, genomic looping, and gene splicing, in addition to its well-established roles in the transcriptional regulation of genes involved in cellular differentiation, cellular maintenance, and adaptation to stimuli *via* ATP-dependent chromatin remodeling [6]. SMARCC1 is regarded as a tumor suppressor in several cancer types, and it has been reported that SMARCC1 expression is correlated with some human cancers [7, 8]; however, in bladder cancer, colorectal carcinoma, and hepatocellular carcinoma, SMARCC1 was identified to be an oncogene [9-11]. The role of SMARCC1 in gastric cancer remains unclear. Thus, we assessed whether SMARCC1 could be a marker to predict clinical outcomes in gastric cancer.

In this study, we explored SMARCC1 expression in gastric cancer and paired adjacent normal tissues (ANT) as well as correlations between SMARCC1 expression and overall survival (OS) and disease-free survival (DFS) in gastric cancer patients.

Materials and methods

Patients and samples

Between May 2010 and January 2013, 130 gastric cancer samples were harvested from patients after radical total gastrectomy or subtotal gastrectomy at the Xiangya Hospital of Central South University (Changsha, China). All cases were confirmed independently by two pathologists and followed up until March 2018. Furthermore, 30 fresh gastric cancer tissues and matched ANT were collected for quantitative real-time (qRT)-PCR and western blotting. Patients had not received preoperative chemotherapy or radiotherapy prior to diagnosis and surgical treatment.

Bioinformatics analysis of SMARCC1 expression

We used TIMER2.0 (http://timer.comp-genomics.org/) to determine SMARCC1 expression across various tumor types, using data from The Cancer Genome Atlas (TCGA). We used the Cancer Cell Line Encyclopedia (CCLE; https://sites.broadinstitute.org/ccle) to analyze SMARCC1 expression in gastric cancer cell lines. Immunofluorescence analysis of SMARCC1 in U251 cells and representative immunohistochemistry (IHC) images of SMARCC1 were downloaded from the Human Protein Atlas (https://www.proteinatlas.org/). SMARCC1 mRNA expression data from cases in the Wang, Chen gastric cancer database and TCGA were collected from Oncomine (http://www.oncomine.org) and the UCSC Xena browser (http://xena.ucsc.edu/) [12]. SMARCC1 expression profiles in gastric cancer tissues and control samples were obtained from the GSE13911 [13] and GSE27342 [14] datasets from the Gene Expression Omnibus (GEO). We also analyzed SMARCC1 mRNA expression in gastric cancer tissues and control samples using the GSE65801 [15], GSE33429 [16], and GSE-13861 [17] datasets in GEO.

RNA extraction and gene expression analysis by qRT-PCR

Total RNA was extracted from fresh gastric cancer tissues and paired ANT using TRIzol reagent according to the manufacturer's protocol (Thermo Fisher Scientific, Waltham, MA, USA). cDNA was synthesized using a universal cDNA synthesis kit (Toyobo, Tokyo, Japan) and then subjected to qRT-PCR using a SYBR Green PCR Kit (Roche, Basel, Switzerland). SMARCC1 expression was measured using SYBR Green Master Mix (Beyotime, Shanghai, China) on an Applied Biosystems Quantification Studio™ 3 & 5 Real-time PCR system (Thermo Fisher Scientific). The sequences of the primers used were as follows: forward: 5'-TGTTGGAAGTCGTACTCAGGATG-3' and reverse: 5'-TGGATTTCCTGACTGACTGAAGG-3'; All experiments were repeated three times. Data were normalized to β-actin expression and calculated using the $2^{-\Delta\Delta CT}$ method.

Western blot

Total protein was collected using RIPA lysis buffer (CWBIO, Beijing, China). Identical quantities of protein samples were separated by SDS-PAGE and then transferred to polyvinylidene difluoride membranes (Roche). After blocking with 5% skim milk, the membranes were incubated with anti-SMARCC1 (Affinity, Cincinnati, OH, USA; diluted 1:1,000) and anti-β-actin (Affinity; diluted 1:1,000) antibodies overnight at 4°C and then treated with HRP-conjugated secondary antibodies at room temperature for

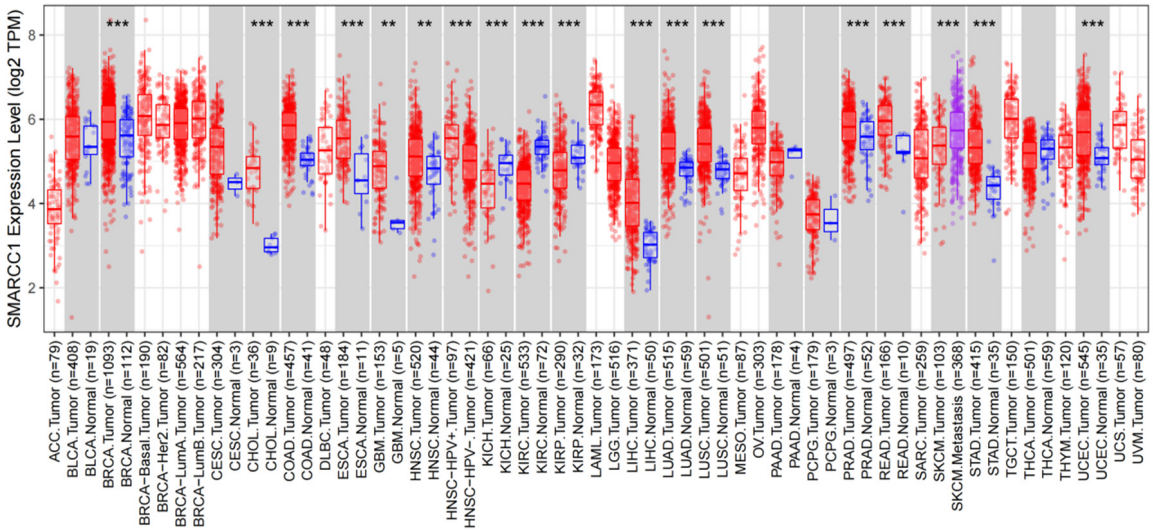

Figure 1. The expression level of the SMARCC1 in different cancer types or specific cancer subtypes was analyzed through TIMER2.0. *P<0.05; **P<0.01; ***P<0.001. Abbreviations: BRCA, breast invasive carcinoma; CHOL, cholangiocarcinoma; COAD, colon adenocarcinoma; ESCA, esophageal carcinoma; HNSC, head and neck squamous cell carcinoma; LIHC, liver hepatocellular carcinoma; LUAD, lung adenocarcinoma; LUSC, lung squamous cellcarcinoma; PRAD, prostatic adenocarcinoma; READ, rectal adenocarcinoma; STAD, stomach adenocarcinoma; SKCM, skin cutaneous melanoma; UCEC, uterine corpus endometrial carcinoma.

30 min. Finally immunoreactive bands were detected with enhanced chemiluminescence reagents (Thermo Fisher Scientific).

IHC

IHC assay were performed using the Universal two-step detection kit (ZSGB-BIO, Beijing, China). After antigen retrieval in the microwave, sections were incubated with anti-SMARCC1 antibody (diluted 1:200) overnight at 4°C. Then the sections were incubated with HRP-conjugated secondary antibody for 30 min and subjected to DAB and hematoxylin treatment. IHC staining of SMARCC1 was scored according to the staining intensity and the percentage of positive cells. We classified staining intensity as: 0 (negative), 1 (weak), 2 (moderate), and 3 (intense); the percentage of positive cells was scored as <5% (0), 5% to 30% (1), 31% to 50% (2), and >50% (3). We defined high SMARCC1 expression as scores ≥4, while low expression was scores <4.

Statistical analysis

We performed all statistical analyses with IBM SPSS Statistics 22 Software (IBM Corporation, Armonk, NY, USA). All quantified data are shown as mean ± standard deviation. Quantitative data were compared between groups using the Student's t-test. Correlations between different SMARCC1 expression levels were analyzed using Pearson's chi-squared test. OS and DFS curves were constructed by the Kaplan-Meier method and analyzed by the log-rank test. Univariate and multivariate analyses were used to verify independent risk factors. P<0.05 was considered statistically significant.

Results

SMARCC1 is highly expressed in gastric cancer according to public datasets

As illustrated in **Figure 1**, SMARCC1 expression was significantly higher in tumor tissues than in NAT in breast invasive carcinoma, cholangiocarcinoma, colon adenocarcinoma, esophageal carcinoma, head and neck squamous cell carcinoma, liver hepatocellular carcinoma, lung adenocarcinoma, lung squamous cell carcinoma, prostate adenocarcinoma, rectum adenocarcinoma, stomach adenocarcinoma, skin cutaneous melanoma, and uterine corpus endometrial carcinoma (P<0.001).

We first analyzed SMARCC1 expression from CCLE data, which showed that SMARCC1 mRNA was upregulated in common gastric cancer cell lines other than BGC823 cells (**Figure 2A**). To determine the localization of SMARCC1,

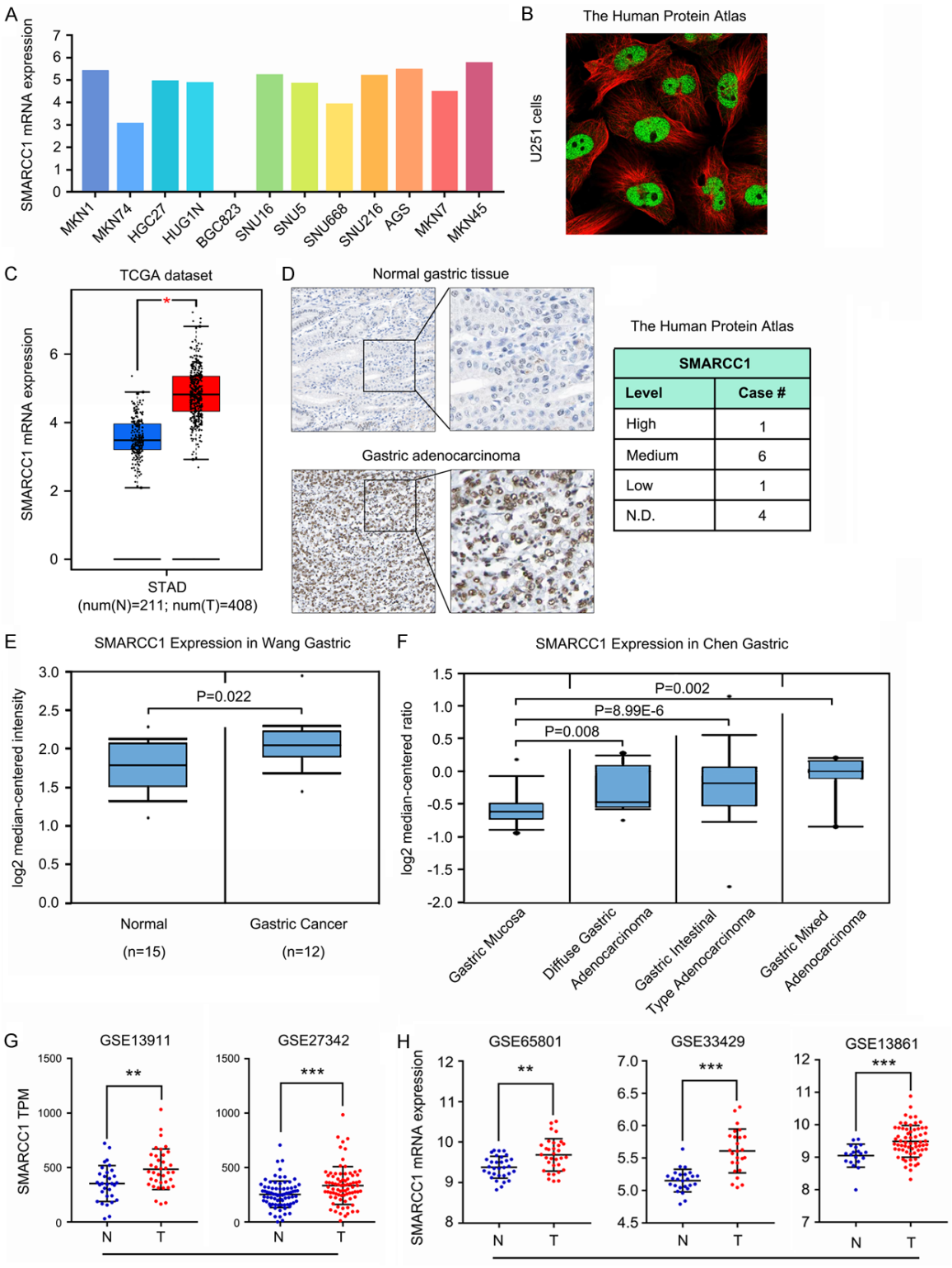

Figure 2. SMARCC1 was upregulated in GC tissues from public datasets. A. SMARCC1 mRNA expression in common GC cell lines from the CCLE. B. The immunofluorescence analysis of SMARCC1 expression in U251 cells is shown. Data were from the Human Protein Atlas. C. The mRNA levels of SMARCC1 in GC samples compared with normal samples from the TCGA dataset are shown. D. Representative images of SMARCC1 IHC results in normal gastric tissue and gastric adenocarcinoma samples are shown. The panel on the right shows the staining results for SMARCC1 in GC tissues. Data were obtained from the Human Protein Atlas. E. SMARCC1 mRNA levels in normal and

GC tissues were analyzed using the Wang gastric microarray data from the Oncomine database. F. SMARCC1 mRNA expression was compared between normal tissue and different GC subtypes using the Chen gastric microarray data from the Oncomine database. G. The SMARCC1 expression profiles were obtained by analysis of normal tissues and GC tissues in the GSE13911 and GSE27342 databases from the GEO. H. The mRNA expression levels of SMARCC1 in normal tissues and GC tissues were analyzed using the GSE65801, GSE33429, and GSE13861 datasets from the GEO. Abbreviations: GC, gastric cancer; N, normal gastric mucosal tissue; T, gastric cancer tissue; ANT, adjacent non-tumor tissue; GEPIA, Gene Expression Profiling Interactive Analysis; CCLE, Cancer Cell Line Encyclopedia; GEO, Gene Expression Omnibus; TCGA, The Cancer Genome Atlas. N.D., not detected. *P<0.05, **P<0.01, ***P<0.001.

we searched the Human Protein Atlas (www.proteinatlas.org) and found SMARCC1 protein mainly localized in the cytoplasm of U251 cells (**Figure 2B**). Furthermore, we analyzed SMARCC1 expression in gastric cancer from TCGA using Gene Expression Profiling Interactive Analysis (GEPIA; http://gepia.cancer-pku.cn). These data showed that SMARCC1 mRNA levels were significantly higher in gastric cancer than in normal gastric tissues (**Figure 2C**). To determine the clinical significance of SMARCC1, we further analyzed the protein expression of SMARCC1 in clinical samples from the Human Protein Atlas. As shown in **Figure 2D**, normal gastric tissues showed weak SMARCC1 expression but gastric cancer tissues presented strong expression. Additionally, SMARCC1 mRNA expression was obtained from the Oncomine dataset (https://www.oncomine.org). In Wang, Chen microarray data for gastric cancer, SMARCC1 mRNA levels were significantly higher than in normal tissues (**Figure 2E**). More importantly, different pathological types of gastric cancer had significantly higher SMARCC1 expression compared with normal gastric mucosa tissues (**Figure 2F**). Consistently, the high SMARCC1 expression in gastric cancer was further confirmed by analyzing mRNA sequencing datasets from GEO (**Figure 2G** and **2H**, GSE13911, GSE27342, GSE65801, GSE33429, and GSE13861, all *P*<0.05). Taken together, bioinformatics analyses indicated that SMARCC1 is highly expressed in gastric cancer cells and gastric cancer tissues, suggesting SMARCC1 may play an important role in gastric cancer development.

SMARCC1 upregulation in clinical gastric cancer samples

Next, we examined SMARCC1 mRNA expression in 30 fresh gastric cancer tissues and paired ANT using qRT-PCR. Our data showed that SMARCC1 mRNA expression was higher in gastric cancer samples than in ANTs (**Figure 3A**). To further detect SMARCC1 protein expression, western blotting was performed in gastric cancer, ANTs, and normal gastric mucosa tissues. Consistent with the mRNA levels, SMARCC1 protein showed higher expression in gastric cancer compared with ANTs and normal gastric mucosa tissues (**Figure 3B**). Furthermore, IHC was performed to detect the protein expression of SMARCC1 in 130 gastric cancer samples. Consistently, SMARCC1 overexpression was confirmed in gastric cancer samples compared with ANTs (**Figure 3C**). SMARCC1 overexpression was frequently observed in gastric cancer (60.8%, 79/130). SMARCC1 showed significantly higher IHC scores in gastric cancer tissues than in ANTs (**Figure 3D**). Collectively, these data suggest that SMARCC1 is upregulated in human gastric cancer samples and may serve a vital role in gastric cancer progression.

SMARCC1 correlates with malignant clinicopathologic features in gastric cancer patients

We conducted Spearmen's correlation analysis to analyze associations between SMARCC1 expression and clinicopathologic characteristics in gastric cancer patients. As shown in **Table 1**, 130 gastric cancer patients were divided into the high (n=79) and low (n=51) SMARCC1 expression groups according to the IHC scores for SMARCC1. High SMARCC1 expression was positively associated with bigger tumor size (P=0.002) and significantly associated with poorer differentiation degree (P=0.006). High SMARCC1 showed a positive association with invasive and metastatic features of gastric cancer, including depth of invasion (P=0.001) and lymph node involvement (P=0.016). However, no significant correlation was detected between SMARCC1 expression and distant metastasis. SMARCC1 overexpression was also related to TNM stage (P=0.007). Finally, SMARCC1 expression was not associated with gender or age in gastric cancer patients.

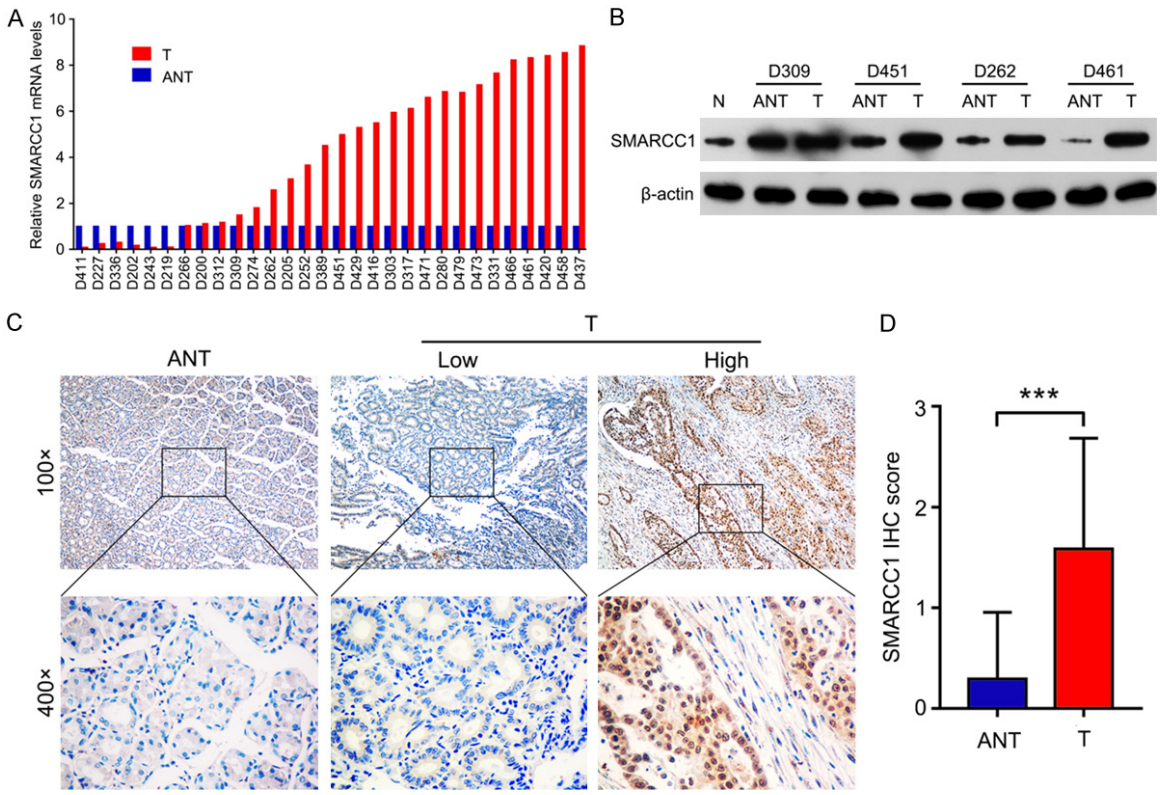

Figure 3. SMARCC1 was overexpressed in human GC tissues. A. The SMARCC1 mRNA level was examined in 30 paired fresh human GC tissues and adjacent non-tumor tissues by qRT-PCR. B. SMARCC1 expression in normal tissue, and four paired GC tissues and matched ANTs was analyzed by western blot. β-actin was used as a control. C. Expression of SMARCC1 in 130 human GC samples and corresponding ANT was detected by IHC. Representative IHC images are shown. Magnification: upper panel, ×100; lower panel, ×400. D. The IHC scores of SMARCC1 expression in GC and ANT are shown. Abbreviations: GC, gastric cancer; qRT-PCR, quantitative real-time polymerase chain reaction; T, gastric cancer tissue; ANT, adjacent non-tumor tissue; N, normal gastric mucosal tissue; IHC, immunohistochemistry. ***P<0.001.

High SMARCC1 expression predicts poor prognosis in gastric cancer patients

To evaluate the ability of SMARCC1 to predict prognosis in gastric cancer patients after resection, we performed Kaplan-Meier analysis to determine associations between SMARCC1 expression and the survival of gastric cancer patients. The results showed that SMARCC1 protein overexpression in gastric cancer was associated with significantly poorer OS (median survival: 60 months vs. 23 months; P<0.001) and DFS (median survival: 38 months vs. 13 months; P=0.013) in gastric cancer patients (**Figure 4A** and **4B**). To further investigate the prognostic potential of SMARCC1 expression, univariate and multivariate analyses were performed to identify prognostic risk factors that impact the survival of gastric cancer patients. Multivariate analysis revealed that high SMARCC1 expression was an independent risk factor for both OS (hazard ratio [HR]=1.796, P=0.017) and DFS (HR=2.601, P=0.016) in gastric cancer patients following gastric resection (**Tables 2** and **3**). Together, these data fully demonstrate that SMARCC1 is closely associated with poor prognosis and could be a novel independent prognostic biomarker for gastric cancer patients after gastric resection.

Discussion

Gastric cancers are lethal malignancies originating in the stomach and are a group of heterogeneous diseases consisting of various phenotypes and genotypes [18, 19]. Despite recent advances in our understanding of the molecular biology and tumor behaviors of gastric cancer, surgical or endoscopic resection remain the most effective treatments [18]. Nevertheless, the prognosis of gastric adenocarcinoma patients who undergo surgical or endoscop-

Table 1. Correlation of SMARCC1 expression with the clinicopathological characteristics of gastric cancer patients

Variables	*n*	SMARCC1 expression		*P*
		High (n=79)	Low (n=51)	
Gender				0.986
Male	84	51	33	
Female	46	28	18	
Age, years				0.273
≤60	43	29	14	
>60	87	50	37	
Tumor size, cm				**0.002**
≤5	60	28	32	
>5	70	51	19	
Differentiation				**0.006**
Well or moderate	57	27	30	
Poor or other	73	52	21	
Depth of invasion				**0.001**
T1+T2	50	21	29	
T3+T4	80	58	22	
Lymph node involvement				**0.016**
Absence	52	25	27	
Presence	78	54	24	
Distant metastasis				0.880
Absence	114	69	45	
Presence	16	10	6	
TNM stage				**0.007**
I-II	55	26	29	
III-IV	75	53	22	

The bold values are statistically significant p<0.05. TNM, tumor-node-metastasis.

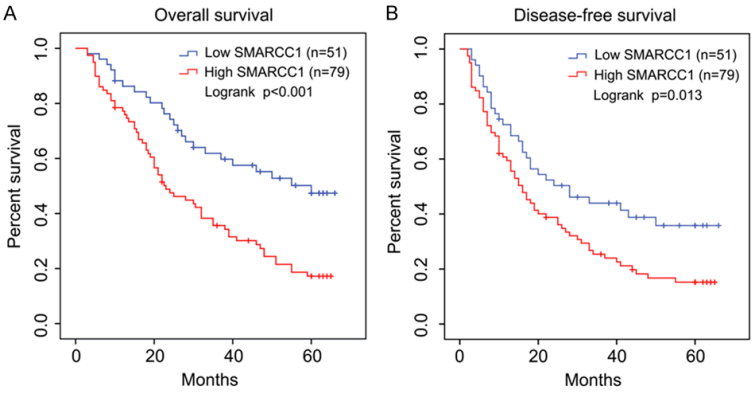

Figure 4. High SMARCC1 expression indicated a poor prognosis in GC patients. A. We divided 130 GC samples into two groups on the basis of the SMARCC1 IHC score. Kaplan-Meier survival analysis and log-rank tests were performed. The OS of GC patients with low or high SMARCC1 expression is shown. B. The DFS of GC patients with low or high SMARCC1 expression is shown. Abbreviations: GC, gastric cancer; IHC, immunohistochemistry; OS, overall survival; DFS, disease-free survival.

ic resection is still unsatisfactory due to high recurrence and metastasis rates [20, 21]. Tumor metastasis is a complex multistep process that involves tumor cell clusters detaching from the primary site, migrating through adjacent tissues, entering and traversing the vasculature, and then growing and proliferating in distant organs [22]. Metastases exhibit remarkable diversity in clinical features depending on cancer type, organ dissemination patterns, and disease course, which reflect different dormant phases [23]. It is critical to identify key predictors of metastasis for individualized treatment and prognostic monitoring in gastric cancer patients.

As a key chromatin remodeling complex, SWI/SNF plays an important role in a variety of cellular biological processes, including transcription and DNA damage repair [24, 25]. The multimeric, combinatorically assembled mSWI/SNF complex can be targeted by multiple chemical approaches to achieve specific functional outcomes [6]. Use of SWI/SNF complex-specific enzyme inhibitors that target the SMARCA2 and/or SMARCA4 ATPase/helicase subunits is associated with multiple human cancers [26-28]. Additionally, mutations in genes encoding subunits of the SWI/SNF (BAF) chromatin remodeling complex are particularly prevalent, occurring in 20% of human cancers [29]. The chromatin remodeler SMARCA1 is frequently methylated in noncancerous tissues of gastric cancer patients and is silenced by methylation in gastric cancer. Furthermore, chromatin remodelers such as ARID1A,

Table 2. Uni- and multivariate analysis of the risk factors affecting overall survival in gastric cancer patients

Variable	Univariate analysis		Multivariate analysis	
	HR (95% CI)	*P*	HR (95% CI)	*P*
Gender				
Female	1			
Male	1.472 (0.855-1.924)	0.375		NA
Age, years				
≤60	1			
>60	1.216 (0.863-2.851)	0.216		NA
Tumor size, cm				
≤5	1		1	
>5	1.439 (1.238-3.289)	**0.021**	0.835 (0.613-1.887)	0.265
Depth of invasion				
T1+T2	1		1	
T3+T4	2.106 (1.365-2.782)	**0.015**	1.431 (1.152-2.658)	**0.034**
Differentiation				
Well or moderate	1			
Poor	1.273 (0.774-2.506)	0.336		NA
Lymph node involvement				
Absence	1		1	
Presence	2.824 (1.803-5.348)	**0.001**	2.208 (1.887-4.225)	**0.004**
TNM stage				
I-II	1		1	
III-IV	3.535 (1.974-6.841)	**<0.001**	2.841 (2.245-4.493)	**0.002**
SMARCC1 expression				
Low	1		1	
High	2.877 (1.932-5.571)	**0.006**	1.796 (1.485-3.966)	**0.017**

Abbreviations: HR, hazard ratio; CI, confidence interval; NA, not applicable; TNM, tumor-node-metastasis. Significant results (*P*<0.05) are shown in bold.

SMARCA1, SMARCA2, and SMARCA4 are frequently mutated in gastric cancer [30].

As a member of SWI/SNF, the role of SMARCC1 in gastric cancer remains poorly understood. A previous study showed that SMARCC1 suppresses prostate cancer cell proliferation and metastasis *via* the PI3K/AKT pathway and is a novel therapeutic [7]. But another study demonstrated that SMARCC1 enters the nucleus *via* KPNA2 to play an oncogenic role in bladder cancer [9]. The tumor-promoting and tumor-suppressive properties of SMARCC1 in different tumors may be due to tumor heterogeneity. Cancer is a dynamic disease. During the course of disease, cancers generally become more heterogeneous [31]. Mutational frequencies of oncogenes and tumour suppressors vary between tumours of different tissues, probably reflecting the importance of distinct signalling pathways within specific tissues or cellular contexts [32]. In various of cancers, the downstream signalling pathways mediated by SMARCC1 are also divergent, so the roles played by SMARCC1 are also different. SWI/SNF complex alterations have also been identified as immunotherapeutic targets and to play important other roles in various cancers [33-35].

In this study, SMARCC1 expression was found to be elevated in gastric cancer at both the mRNA and protein levels by systemic data mining and clinical tissue analysis. Our data also indicated that SMARCC1 expression, invasion depth, lymph node involvement, and TNM stage were associated with poor OS and DFS in gastric cancer. Most importantly, high SMARCC1 expression was related to poor clinical features and decreased OS and DFS in gastric cancer patients. A recent study demonstrated

Table 3. Uni- and multivariate analysis of the risk factors associated with disease-free survival in gastric cancer patients

Variable	Univariate analysis		Multivariate analysis	
	HR (95% CI)	*P*	HR (95% CI)	*P*
Gender				
Female	1			
Male	1.207 (0.816-1.658)	0.306		NA
Age, years				
≤60	1			
>60	1.446 (0.922-1.915)	0.208		NA
Tumor size, cm				
≤5	1		1	
>5	1.622 (1.149-2.292)	**0.022**	1.245 (0.775-1.869)	0.375
Depth of invasion				
T1+T2	1		1	
T3+T4	2.541 (1.272-3.167)	**0.011**	2.094 (1.616-3.391)	**0.025**
Differentiation				
Well or moderate	1		1	
Poor	1.527 (1.389-2.388)	**0.038**	1.326 (0.839-2.208)	0.286
Lymph node involvement				
Absence	1		1	
Presence	2.891 (1.802-4.586)	**0.005**	1.838 (1.266-2.887)	**0.032**
TNM stage				
I-II	1		1	
III-IV	4.357 (2.474-6.405)	**<0.001**	3.696 (2.287-5.935)	**0.003**
SMARCC1 expression				
Low	1		1	
High	1.823 (1.133-3.632)	**0.031**	2.601 (1.545-4.283)	**0.016**

Abbreviations: HR, hazard ratio; CI, confidence interval; NA, not applicable; TNM, tumor-node-metastasis. Significant results (*P*<0.05) are shown in bold.

the differential expression and prognostic value of SMARCC1 in hepatocellular carcinoma, showing that it could serve as a prognostic molecule [11]. Our explorative study demonstrated that elevated SMARCC1 expression is closely associated with poor prognosis in gastric cancer. Consequently, SMARCC1 may be a candidate diagnostic and/or therapeutic target for gastric cancer. Further studies are required to explore the underlying mechanisms and assess the *in vivo* role of SMARCC1.

Conclusion

This study found that SMARCC1 expression is upregulated in gastric cancer tissues compared with paired adjacent normal tissues. High SMARCC1 expression is significantly related to tumor size, differentiation, depth of invasion, lymph node involvement, and TNM stage. Furthermore, high SMARCC1 expression is closely associated with poor prognosis in gastric cancer patients. Thus, SMARCC1 could be an indicator for accurately predicting patient prognosis and/or a therapeutic target.

Acknowledgements

The Project Supported by Natural Science Youth Foundation of Hunan Province (Grant No. 2019JJ50976).

Disclosure of conflict of interest

None.

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