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RESEARCH

# Elevated STMN1 Expression Correlates with Poor Prognosis in Patients with Pancreatic Ductal Adenocarcinoma

**Jian Li · Guohuang Hu · Fujiao Kong · Kemin Wu · Kun Song · Jianfeng He · Weijia Sun**

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**Abstract** STMN1 is a cytosolic phosphoprotein that not only participates in cell division, but also plays an important role in other microtubule-dependent processes, such as cell motility. Furthermore, STMN1 acts as a "relay protein" in several intracellular signaling pathways that influence cell growth and differentiation. Thus, STMN1 is likely to support cellular processes essential for tumor progression: survival and migration. Indeed, elevated STMN1 expression has been reported in various types of human malignancies and is correlated with poor prognosis in these human malignancies. However, the clinical and prognostic significance of STMN1 in pancreatic ductal adenocarcinoma (PDAC) remains unknown. Thus, we assessed STMN1 in PDAC in this retrospective study. We first examined STMN1 expression in PDAC tissues from 27 cases and matched adjacent non-cancerous tissues by quantitative polymerase chain reaction (PCR) and western blot analyses. Next, immunohistochemistry was used to evaluate STMN1 expression in 87 archived paraffin-embedded PDAC specimens. STMN1 mRNA and protein expression levels were to a large extent up-regulated in PDAC tissue compared with their adjacent non-cancerous tissues. Moreover, STMN1 expression was closely correlated with histological differentiation, lymphatic metastasis, and TNM stage ($P$= 0.023, 0.047, and 0.014, respectively). In addition, PDAC patients with higher STMN1 expression died sooner than those with lower STMN1 expression ($P$<0.01). Multivariate analysis demonstrated that STMN1 expression was an independent prognostic factor for PDAC patients ($P$<0.01). Herein, we provide the first evidence that up-regulated STMN1 may contribute to tumor progression and poor prognosis in PDAC patients and may serve as a novel prognostic marker.

**Keywords** Pancreatic ductal adenocarcinoma (PDAC) · STMN1 · Prognosis

## Introduction

Pancreatic cancer is one of the most lethal solid malignancies and the seventh most frequent cause of cancer death worldwide, causing approximately 265, 000 deaths, of which were 280, 000 new cases in 2008 [1]. Despite recent advances in surgical and medical therapy, the mortality rate from this disease remains generally unchanged [2–5]. This poor outcome is mainly attributed to its aggressive growth, difficulty of early diagnosis, rapid metastatic dissemination, and lack of effective therapies. This situation signifies an urgent need for the exploration of diagnostic molecular factors to improve the survival rates for this disease, particularly in the case of pancreatic ductal adenocarcinoma (PDAC), which accounts for the majority (>90 %) of pancreatic malignancies.

STMN1, also known as stathmin, metablastin, phosphoprotein 19, LAP18 and Op 18, is the prototype member of a phosphoprotein family that shares a so-called stathmin-like domain that contains up to four phosphorylation sites (stathmin: Ser16, Ser25, Ser38, and Ser63). The

J. Li
Department of PET center, Xiangya Hospital, Central South University, Changsha, China

G. Hu
Department of General Surgery, The fourth Hospital of Changsha, Changsha, China

F. Kong
Department of Anesthesiology, Xiangya Hospital, Central South University, Changsha, China

K. Wu · K. Song · J. He · W. Sun (✉)
Department of General Surgery, Xiangya Hospital, Central South University, Changsha, China
e-mail: sunweijia2013@126.com

dephosphorylation of STMN1 by phosphatases promotes microtubule catastrophe by facilitating depolymerization and sequestration of soluble tubulin heterodimers [6, 7]. A tightly regulated sequenced pattern of STMN1 phosphorylation and dephosphorylation is essential for entry into prophase and, terminally, into cytokinesis [8].

Apart from its role in cell division, STMN1 not only participates in other microtubule-dependent processes, such as cell motility [9–12], but also acts as a "relay protein" in several intracellular signaling pathways that influence cell growth and differentiation [9–12]. Thus, STMN1 is likely to support cellular processes that are vital for tumor progression: survival and migration. Indeed, overexpression of STMN1 has been reported in various types of human malignancies, such as breast cancer [13], hepatocellular carcinoma [14], sarcoma [15–17], lung adenocarcinomas [18], gastric cancer [17], oral squamous-cell carcinoma [15], and prostate cancer [19]. Furthermore, STMN1 is known to be involved in the oncogenesis of a wide variety of human cancers [11, 17, 20]. All of these findings indicate that STMN1 plays an important role in tumorigenesis. However, the clinicopathological significance and prognostic role of STMN1 in PDAC remains largely unknown. Thus, in this retrospective study, we use real-time polymerase chain reaction (PCR), western blot, and immunohistochemistry to evaluate STMN1 expression and its correlation with survival in PDAC patients.

## Materials and Methods

### Tissue Specimen Collection

This retrospective study was approved by the Ethics Committee of Central South University, and informed consent was obtained from each patient. Twenty seven primary PDAC specimens and matched adjacent non-tumor tissues (taken >2 cm from the tumor margin) were collected from patients at the Department of General Surgery, Xiangya Hospital, Central South University, People's Republic of China in 2011 (Table 1), and used for quantitative reverse-transcription (qRT)-PCR and western blott tests. Patients were histopathologically diagnosed as suffering from primary PDAC, which was newly diagnosed and untreated, and had no history of any other tumors. All samples were obtained immediately following surgical removal, snap-frozen in liquid nitrogen, and stored at −80 °C until further processing.

For immunohistochemical assay, a total of 87 paraffin-embedded PDAC samples were obtained from patients who underwent radical or palliative surgery between June 2004 and June 2007 at the Department of General Surgery at Xiangya Hospital of Central South University, People's Republic of China. Next, 40 normal pancreatic tissue samples were collected at surgery from patients suffering from acute pancreatic injury or benign pancreatic diseases, such as pseudocyst, cystadenoma, or microcystic adenoma. The criteria for study enrollment were: histopathological diagnosis of primary PDAC, and no received chemotherapy or radiotherapy before surgery, or postoperative adjuvant therapy, and no history of previous tumors. The patients included 54 males and 33 females, aged from 35 to 79 years (mean age, 60.7 years). As shown in Table 2, we recorded clinicopathologic variables, such as gender, age, tumor site, tumor size, differentiation, TNM stage, and lymphatic metastasis. The follow-up period was defined as the interval between the date of the operation and the date of either patient death or the final follow-up. Deaths from non-PDAC related causes were treated as censored cases. The follow-up time was 5 years for 69 patients who had undergone radical surgery, over a period ranging from 7 to 65 months. Follow-ups were performed over the telephone.

### Quantitative Real-Time RT-PCR for STMN1

Total RNA was extracted using Trizol reagent (Invitrogen, Carlsbad, USA) according to the manufacturer's instructions. RNA was reverse transcripted into cDNAs using RevertAid H Minus First Strand cDNA Synthesis Kit (Fermentas, CA). Briefly, 300 ng of each RNA sample was mixed with reaction mixture consisting of 4 μl 5× reaction buffer, 1 μl Oligo(dT) 18 primer, 2 μl dNTP mix, 0.5 μl Ribonuclease Inhibitor, 1 μl RevertAidTM M-MulV Reverse Transcriptase, and 12 μl DEPC-treated water. They were incubated at 42 °C for 60 min and then 70 °C for 10 min. Quantitative PCR was carried out using Applied Biosystems 7900HT Fast Real-Time PCR System (Applied Biosystems, CA USA) and SYBR Green PCR Master Mix (Applied Biosystems). The sample mixture of a total 10 μl reaction volume contained complementary DNA, 2× SYBR Green PCR Master mix, primers and nuclease-free water. The reaction conditions were 95 °C for 5 min, then 35 cycles at 94 °C for 30 s, 60 °C for 30 s, and followed by 72 °C for 30 s. The primer sequence was designed using Prime 3.0 (ProMab, Changsha, People's Republic of China). The primers for STMN1 were (forward) 5′- TCAGCCCTCGGTCAAAAGAAT-3′, and (reverse) 5′-TTCTCGTGCTCTCGTTTCTCA-3′. The housekeeping gene glyceraldehyde-3-phosphate dehydrogenase (GAPDH; ProMab) was used as an internal control, with the primer sequences (forward) 5′- ACAACTTTGGTATCGTGGAAGG-3′, and (reverse) 5′- GCCATCACGCCACAGTTTC-3′. Each sample was analyzed in triplicate, and the Ct value for each sample was calculated using the ΔΔCt method and the results are expressed as $2^{-\Delta\Delta Ct}$ [21].

**Table 1** Patient clinical features and STMN1 expression profile

| Case | Age (years) | Gender | Tumor size (cm) | TNM stage | Normalised STMN1 amount in tumor tissue relative to adjacent nomoral tissue $2^{-\triangle\triangle Ct}$ |
|---|---|---|---|---|---|
| 1 | 60 | F | 3.6×3.9×4.5 | II | 16.6410 |
| 2 | 38 | M | 2.8×3.6×4.4 | IV | 15.9631 |
| 3 | 63 | M | 2.7×2.8×2.6 | I | 2.4453 |
| 4 | 58 | M | 1.9×1.8×2.3 | I | 7.5162 |
| 5 | 75 | F | 2.2×2.8×3.6 | III | 22.3675 |
| 6 | 66 | M | 2.5×3.5×4.4 | II | 1.8661 |
| 7 | 63 | M | 3.0×3.0×3.1 | II | 10.7034 |
| 8 | 73 | F | 2.5×3.2×3.3 | I | 6.1475 |
| 9 | 61 | M | 4.0×5.0×3.5 | II | 5.1575 |
| 10 | 70 | F | 2.0×3.0×2.2 | I | 1.3915 |
| 11 | 62 | M | 2.1×2.6×3.1 | I | 1.3044 |
| 12 | 48 | M | 3.4×3.8×4.5 | III | 10.6787 |
| 13 | 57 | F | 3.8.×4.0×3.6 | III | 6.6192 |
| 14 | 73 | M | 2.5×2.9×3.6 | II | 9.7360 |
| 15 | 71 | M | 1.7×2.4×2.6 | I | 2.4680 |
| 16 | 56 | F | 2.3×3.0×2.2 | II | 4.0935 |
| 17 | 77 | M | 3.2×4.2×4.1 | II | 3.0738 |
| 18 | 59 | F | 4.0×4.2×4.6 | III | 6.2477 |
| 19 | 79 | M | 6.0×5.0×4.2 | IV | 24.1397 |
| 20 | 77 | M | 3.0×4.0×4.0 | III | 28.3118 |
| 21 | 69 | F | 3.1×4.3×3.7 | III | 9.8037 |
| 22 | 65 | F | 2.7×3.5×3.0 | III | 4.8906 |
| 23 | 56 | M | 3.8×4.5×3.3 | II | 5.3765 |
| 24 | 61 | M | 3.3×2.9×2.7 | II | 10.2437 |
| 25 | 64 | F | 4.2×4.5×4.4 | III | 19.6529 |
| 26 | 39 | M | 2.6×1.9×3.0 | II | 3.1383 |
| 27 | 57 | F | 2.5×3.8×3.6 | I | 16.2234 |

*M* male, *F* female. Relative quantification was performed by the $2^{-\triangle\triangle Ct}$ method with adjacent normal pancreatic tissue sample as a calibrator. Data show the means from three independent analyses. Every independent analysis was carried out after the RNA extraction step. Total RNAwas extracted, reverse transcripted, and then real-time PCR tested. $\triangle$Ct obtained from real-time PCR was subject to Wilcoxon signed rank test ($\triangle$Ct=Ct STMN1-Ct GAPDH). The expression levels of STMN1 in tumor tissues were significantly higher than in adjacent normal tissues ($p<0.01$, $Z=5.004$)

## Western Blot

STMN1 protein was extracted using a Total Protein Extraction Kit (ProMab). Briefly, all proteins were resolved on 10 % sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) in running buffer (Bio-Rad, USA), and transferred to nitrocellulose membranes (Pierce, USA). After blocking with 5 % nonfat milk for 2 h, the membranes were incubated with primary antibodies against STMN1 (1:1500; ABCCam, USA), and GAPDH (1:15000; Santa Cruz, USA) at room temperature for 1 h, and then the membranes were washed with phosphate-buffered saline-Tween20 (PBST) and incubated with secondary antibody goat anti-rabbit IgG/hourseradish peroxidase (HRP; 1:5000; ProMab) for 1 h at room temperature. Enhanced chemiluminescence detection of the target protein was performed using an ECL chemiluminescence system (Pierce).

## Immunohistochemistry

All specimens were fixed in 4 % formaldehyde, dewaxed, embedded, and cut into 3 μm serial sections. The sections were then incubated overnight at 4 °C with an anti-STMN1 antibody (Abcam). After washing with PBS, sections were incubated with secondary antibodies for 30 min at 37 °C. Then, the sections were washed three times with phosphate buffered (PBS) and treated with DAB for approximately 5 min. Finally, sections were counterstained with hematoxylin, dehydrated, mounted, and examined via light microscopy. Sections incubated with PBS instead of the primary antibody

**Table 2** Correlation between STMN1 expression and clinicopathologic features of PDAC patients

| Parameters | Case | STMN1 expression (*n*) | | $\chi^2$ | *p* |
|---|---|---|---|---|---|
| | | Positive | Negative | | |
| Gender | | | | | |
| Male | 54 | 38 | 16 | 1.421 | 0.175 |
| Female | 33 | 27 | 6 | | |
| Age (years) | | | | | |
| ≤60 | 38 | 30 | 8 | 0.640 | 0.292 |
| <60 | 49 | 35 | 14 | | |
| Tumor size (diameter) | | | | | |
| ≤5 cm | 53 | 41 | 12 | 0.503 | 0.322 |
| <5 cm | 34 | 24 | 10 | | |
| Tumor site | | | | | |
| Head-Neck | 54 | 41 | 13 | 0.111 | 0.465 |
| Body-Tail | 33 | 24 | 9 | | |
| Histological differentiation | | | | | |
| High | 54 | 36 | 18 | 4.878 | **0.023** |
| Middle-Low | 33 | 29 | 4 | | |
| TNM stage | | | | | |
| I+II | 57 | 38 | 19 | 5.664 | **0.014** |
| III+IV | 30 | 27 | 3 | | |
| Lymphatic metastasis | | | | | |
| Yes | 47 | 39 | 8 | 3.697 | **0.047** |
| No | 40 | 26 | 14 | | |

Bold values represent *p* values which are considered to be statistically significant at <0.05

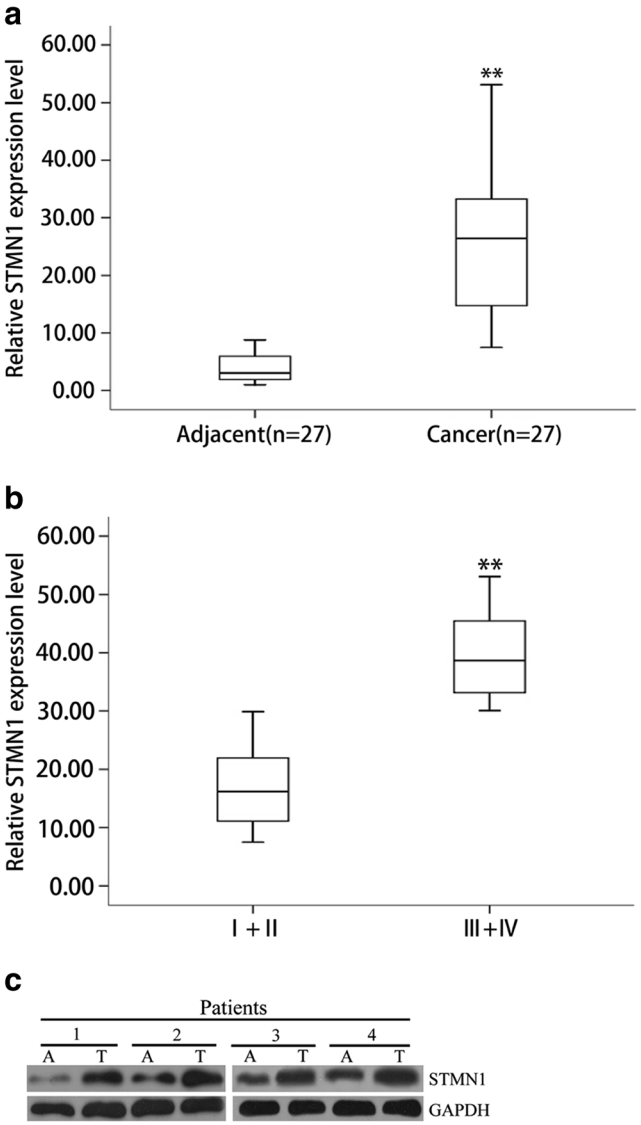


**Fig. 1** Comparison of STMN1 levels in PDAC, adjacent non-tumor tissue. **a** and **b** STMN1 expression levels were obtained by real-time-PCR. Wilcoxon signed rank test was used to determine statistical significance. **a** Relative STMN1 expression levels in different tissue: cancer, PDAC; adjacent, adjacent non-tumor tissue samples. **b** Relative STMN1 expression levels in PDAC tissue at I+II and III+IVstages. **c** It is a representative Western blot analysis of STMN1 expression in all specimens

were used as negative controls. All sections were examined and scored independently by two experienced pathologists who had no knowledge of clinical or pathological information regarding the samples. STMN1 expression was evaluated according to the ratio of positive cells per specimen and the staining intensity. The ratio of positive cells per specimen was evaluated quantitatively and scored as follows: 0, staining of ≤1 %; 1, staining of 2–25 %; 2, staining of 26–50 %; 3, staining of 51–75 %; and 4, staining of >75 % of the examined cells. Staining intensity was divided into four groups: 0, no signal; 1, weak staining; 2, moderate staining; and 3, strong staining. A total score of 0–12 was determined as follows: total score = ratio of positively staining cells (score) × intensity of immunoreactivity (score). Scores were graded as negative (−; score: 0–1), weak (+; score: 2–4), moderate (++; score: 5–8) or strong (+++; score: 9–12).

## Statistical Analysis

All results are expressed as means±standard deviation (SD) based on data form at least three separate experiments. Possible differences between groups were analyzed using the Mann–Whitney *U*-test. The association between STMN1 and clinicopathological features was analyzed using the chi-square test. Survival curves were obtained using Kaplan–Meier curves and log-rank tests. Multivariate prognostic factors were examined using Cox's proportional hazards model and a receiver operating curve (ROC) curve was drawn. SPSS 13.0 (SPSS Inc., Chicago, IL, USA) software was used for statistical analysis. A *P*-value <0.05 was considered statistically significant.

## Results

### Upregulation of STMN1 in PDAC

To explore the roles of STMN1 in PDAC, 27 pairs of PDAC tissue and corresponding adjacent pancreatic tissues were collected, and the expression levels of STMN1 were measured by quantitative qRT-PCR and western blot. As shown in Fig. 1a, STMN1 mRNA levels in PDAC and adjacent tissue were 25.6352±13.0395 and 3.9170±2.2652 ($P<0.001$), respectively. Furthermore, STMN1 mRNA expression in advanced stage PDACs was significantly higher than early stage PDACs (Fig. 1b; $P<0.001$). Western blot analysis showed that the STMN1 protein expression in PDAC tissues was significantly higher than in adjacent pancreatic tissues (Fig. 1c; $P<0.05$). In addition, 87 PDAC tissue samples and 40 normal pancreatic tissue samples were analyzed via immunohistochemistry. According to immunohistochemical staining results, high STMN1 expression was detected in 65 of 87 (74.7 %) PDAC cases, and was mainly located in the cytoplasm of cancer cells (Fig. 2). By contrast, only 11 of 40 (27.5 %) cases were positive for STMN1 in normal pancreatic tissue (Table 3). Taken together, the results suggest that STMN1 is involved in PDAC initiation and aggressive progression.

### Correlation Between STMN1 Protein Expression and PDAC Clinicopathological Features

On the basis of immunohistochemical results, we further investigated the relationship between STMN1 protein expression and the clinicopathological characteristics of PDAC. As shown in Table 3, STMN1 protein expression correlated significantly with histological differentiation, lymphatic metastasis, and TNM stage ($P<0.05$). However, STMN1 protein expression was not associated with other clinicopathological features, such as age, sex, tumor size, or tumor site.

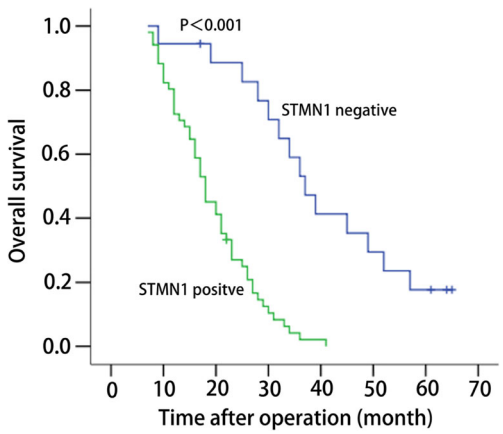


**Fig. 2** Immunohistochemical staining of STMN1 in normal pancreatic and pancreatic ductal adenocarcinoma tissue. Tissue sections were stained with antibodies against STMN1 and counterstained with hematoxylin. **a** STMN1 negative staining (−) was shown in normal pancreatic tissues. **b**–**d** STMN1 immunoreactivity was detected in pancreatic ductal adenocarcinoma tissue with staining (+, ++ and +++) predominant in the cytoplasm (×400)

**Table 3** expression of STMN1 in pancreatic tissue samples

| Group | *n* | STMN1 expression | | *p* |
|---|---|---|---|---|
| | | Positive expression | % | |
| PDAC | 87 | 65 | 74.7 | **<0.01** |
| NP | 40 | 11 | 27.5 | |

*NP* normal pancreatic tissue. Bold values represent *p* values which are considered to be statistically significant at 0.05

### Prognostic Significance of STMN1 Expression in PDAC

Given the close association between STMN1 and several malignant parameters in PDAC, we further investigated whether STMN1 could be used as a prognostic predictor for PDAC patients who underwent radical surgery. Kaplan–Meier survival analysis indicated that PDAC patients in the positive STMN1 expression group had poorer overall survival rates compared with patients in the negative STMN1 expression group (median survival, 19.10 vs 38.94 months, respectively; $P<0.001$; Fig. 3). The results from univariate and multivariate analyses of overall PDAC patient survival are shown in Table 4. Univariate analysis showed that the following factors were significantly related to postoperative survival: histological differentiation, TNM stage grading, lymphatic metastasis, and STMN1 expression. Multivariate analysis, performed using the Cox proportional hazards model, indicated that histological differentiation, STMN1 expression and TNM stage were all significantly independent prognostic factors of patients who underwent radical surgery for PDAC. Furthermore, the ROC curve showed that the ROC area of STMN1 expression in patients (0.791) was higher than for histological differentiation (0.648) and TNM stage (0.689; Fig. 4).

## Discussion

In this retrospective study, we found that the expression of STMN1 was upregulated in most primary PDAC tissues compared with their matched adjacent non-tumor pancreatic tissues at both mRNA and protein levels. Furthermore, STMN1 protein level was significantly correlated with PDAC histological differentiation, lymphatic metastasis, and TNM stage. Consistently, PDAC patients with high STMN1 expression had shorter survival times compared with those with low STMN1 expression. In addition, our results revealed that tumor size was not a significant prognostic factor of PDAC,

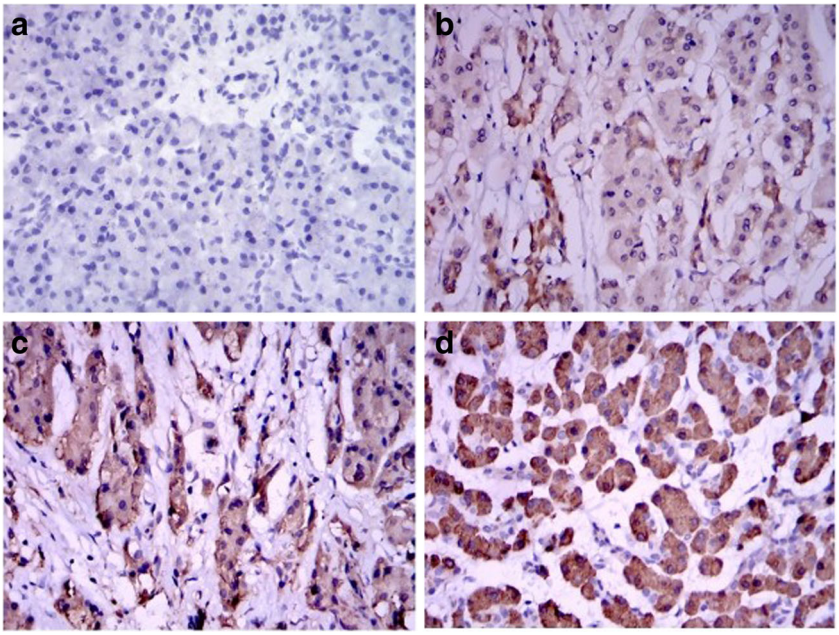


**Fig. 3** Over survival in 69 PDAC pantients who underwent radical surgery based on the expression of STMN1. The graph summarizes Kaplan-meier survival analysis for patients with positive or negative STMN1 expression ($p<0.001$)

which is consistent with some previous results [22, 23]. However, this result contradicts other previous studies [24–26]. The reasons underlying these controversial observations and conclusions may be the different tumor

**Table 4** Univariate and multivariate analysis of overall survival in 69 PDAC pantients who underwent radical surgery

| Parameters | Univariate analysis | | Multivariate analysis | |
|---|---|---|---|---|
| | RR (95 % CI) | *p* | RR (95 % CI) | *p* |
| Gender | | | | |
| Male<br>Female | 0.971 (0.590–1.599) | 0.909 | | |
| Age | | | | |
| ≤60<br><60 | 1.475 (0.894–2.433) | 0.128 | | |
| Tumour size | | | | |
| ≤5 cm<br><5 cm | 1.103 (0.652–1.867) | 0.714 | | |
| Tumour site | | | | |
| Head-Neck<br>Body-Tail | 0.703 (0.410–1.204) | 0.199 | | |
| Histopathologic differentiation | | | | |
| High<br>Middle-Low | 2.825 (1.566–5.098) | **0.001** | 2.127 (1.161–3.897) | **0.015** |
| TNM stage | | | | |
| I<br>II+III | 12.677 (5.877–27.344) | **<0.001** | 7.956 (3.029–20.898) | **<0.001** |
| Lymphatic metastasis | | | | |
| Yes<br>No | 0.196 (0.108–0.354) | **<0.001** | 0.836 (0.395–1.772) | 0.641 |
| STMN1 expression | | | | |
| Negative<br>Positive | 5.797 (2.786–12.060) | **<0.001** | 3.892 (1.773–8.543) | **0.001** |

Bold values represent *p* values are considered to be statistically significant at <0.05

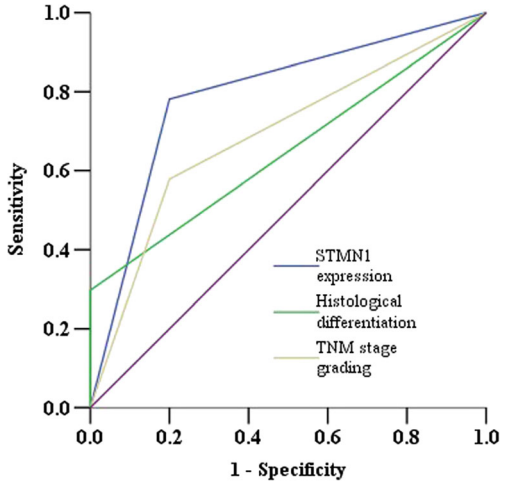


**Fig. 4** Receiver operating characteristic curves of histological differentiation, STMN1 expression and TNM stage for predicting the prognosis of PDAC. The areas under the curve for histological differentiation, STMN1 expression and TNM stage were 0.648, 0.791 and 0.689

size cut-off points and the small number of cases analyzed.

STMN1 is a cytosolic phosphoprotein that was first identified in neuroendocrine cells [27]. As a cytosolic phosphoprotein, STMN1 has always been considered as microtubule destabilizer, which plays important roles in the construction and function of the mitotic spindle [28, 29]. However, in recent years, ample evidence has indicated that STMN1 overexpression is a consistent feature of malignant human neoplasia. For example, STMN1 is abnormally overexpressed in various types of malignant tumors including breast cancer [13], hepatocellular carcinoma [14], sarcoma [15–17], lung adenocarcinomas [18], gastric cancer [17], oral squamous cell carcinoma [15], and prostate cancer [19]. Moreover, recent studies have shown that STMN1 overexpression correlates significantly with a number of parameters known to be associated with poor prognosis. For instance, Curmi et al. [30] reported that increased STMN1 mRNA expression was significantly associated with histological grade III disease, and the loss of estrogen receptors or progesterone receptors in breast cancer. Similarly, Zheng et al. [31] also reported that STMN1 overexpression was positively related with depth of tumor invasion, lymph node status, Dukes classification, and TNM staging system of colorectal cancer (CRC) patients. In addition, several other studies reported that STMN1 was highly expressed in more advanced stages of the disease [15–17]. Taken together, these findings lead us to the conclusion that STMN1 participates in the development and progression of human carcinomas. In this study, we observed that the expression levels of STMN1 mRNA and protein in PDAC tissues were significantly higher than those in adjacent tissues ($P<0.01$). Moreover, STMN1 protein expression correlated significantly with histological differentiation, lymphatic metastasis, and TNM stage grading, with a particular emphasis on the TNM stage grading ($P=0.014$; Table 2). Therefore, STMN1 overexpression is related to tumor development and progression in PDAC, suggesting that STMN1 is a potential novel therapeutic pharmacological target.

Apart from investigating of STMN1 expression and its relationship with clinicopathologic features, we also analyzed the prognostic significance of STMN1 expression in PDAC patients who had undergone radical surgery. In this study, we found that increased STMN1 protein expression in PDAC was significantly associated with poor prognosis and low overall survival. Furthermore, univariate and multivariate analyses showed that STMN1 protein expression was an independent prognostic factor associated with overall survival rate ($P<0.01$; Table 4). A previous study of oral squamous cell carcinoma also found that STMN1 overexpression is closely related to disease-free survival [15]. In gastric cancer, the association of STMN1 expression with recurrence-free survival rate was more significant in diffuse type of gastric cancer [17]. In addition, overexpression of STMN1 protein correlated with poor outcomes of CRC patients [31, 32]. Taken together, these observations suggest that STMN1 overexpression is a potential independent poor prognostic factor of patients with malignant tumors, including PDAC.

In summary, for the first time, we investigated the relationship between STMN1 expression and clinicopathological and prognostic value in PDAC. Our data indicate that STMN1 expression is significantly higher in PDAC tissues compared with adjacent normal pancreatic tissues, and increased expression is positively associated with histological differentiation, lymphatic metastasis, and TNM stage grading. Furthermore, with respect to PDAC patients who underwent radical surgery, patients with higher STMN1 expression have poorer postoperative prognosis. Our study suggests that STMN1 might serve as a prognostic marker and potential molecular target for tumor therapy for PDAC patients with high STMN1 expression.

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