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Casein Kinase 1 Alpha 1 Is Over-expressed in Pancreatic Adenocarcinoma Tissues and Correlates With Shorter Patient Survival

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Abstract

Background/Aim: A quarter of a century has passed since the start of the 21st century, and cancer, once considered an incurable disease, has become manageable thanks to the development of various treatments. However, pancreatic adenocarcinoma (PAAD) remains one of the deadliest cancers in the world, with over 95% of patients dying within five years. This is because the anatomical location of the pancreas makes it very difficult to detect with imaging, and symptoms often do not appear until the cancer invades the nerve plexus in its terminal stages, meaning that by the time it is diagnosed, it is often too late. Therefore, the development of prognostic markers is an urgent task. Casein kinase 1 alpha 1 (CSNK1A1) is a serine/threonine protein kinase deeply involved in Wnt signaling and the tumor suppressor mechanisms of p53. While the association between increased or decreased CSNK1A1 expression and prognosis has been reported in many types of cancer tissue, its association in PAAD is not yet fully understood. This study investigated the potential of CSNK1A1 as a prognostic marker for PAAD.

Materials and Methods: We used Gene Expression Profiling Interactive Analysis (GEPIA) and the University of Alabama Birmingham Cancer Data Analysis Portal (UALCAN) bioinformatics platforms to analyze *CSNK1A1* mRNA expression, protein levels, and survival rates of patients with PAAD obtained from The Cancer Genome Atlas (TCGA) database.

Results: CSNK1A1 mRNA and protein levels were significantly higher in PAAD tissue compared to normal pancreatic tissue, and this increase was associated with a poor prognosis in patients with PAAD.

continued

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Conclusion: In PAAD tissue, increased expression of *CSNK1A1* mRNA and protein was observed compared to normal pancreatic tissue, and this increased expression correlated with poor patient prognosis. Therefore, CSNK1A1 is considered a promising prognostic biomarker in PAAD.

Keywords: CSNK1A1, pancreatic adenocarcinoma, Kaplan–Meier survival plot, prognosis.

Introduction

Pancreatic adenocarcinoma (PAAD) is one of the cancers with the poorest prognosis. According to "Cancer Statistics 2025" the 5-year relative survival rate for PAAD in Japan is 8.5% (1). PAAD is often diagnosed at an advanced stage, with most cases detected at stage IV, and it is extremely rare for it to be detected at an early stage where it can be resected. The poor prognosis is due to the anatomical difficulty of early detection, the tendency of cancer cells to disseminate within the abdominal cavity, and the lack of effective treatment other than surgical resection. While the use of gemcitabine and S-1 has improved survival rates compared to the past, the prognosis is still not satisfactory. A useful biomarker for early detection screening has yet to be established. The carbohydrate antigen 19-9 (CA19-9) is the most commonly used biomarker, but its low sensitivity and specificity make it unreliable for early detection. Based on these findings, it is considered important to select target molecules and prognostic markers that are strongly associated with poor prognosis and that can be targeted by molecular targeted therapeutic drugs in patients with PAAD. Currently, active research is underway to identify factors that determine the malignant potential of PAAD.

In studies aiming to identify molecules closely associated with prognosis in patients with PAAD, large-scale molecular expression data from human PAAD tissues must be correlated with clinical outcomes. However, the number of PAAD cases available at our institution alone is too limited to identify meaningful prognostic molecules.

The Cancer Genome Atlas (TCGA) is a molecular analysis database established in 2006 as a joint project between the National Cancer Institute and the National Human Genome Research Institute. It contains data on over 20,000 primary cancers and their corresponding normal tumor samples from 33 cancer types (2). This database is available to the public for use in cancer diagnosis, treatment, and other research. To investigate the relationship between gene expression levels and prognosis in various cancers using the TCGA database, a platform suitable for research purposes is required. The University of Alabama at Birmingham Cancer Data Analysis Portal (UALCAN) is an easy-to-use, interactive web portal for performing in-depth analysis of TCGA gene expression data, and is free for anyone to use (3). Gene Expression Profiling Interactive Analysis (GEPIA) is another free platform for analyzing the TCGA database. GEPIA offers key interactive and customizable features, including differential expression analysis, profiling plots, correlation analysis, patient survival analysis, similar gene detection, and dimension reduction analysis (4). Previously, using the UALCAN and GEPIA platforms, we identified several molecules from the TCGA database that may be significantly associated with the prognosis of patients with pancreatic cancer, adrenocortical carcinoma, uveal melanoma, and renal cell carcinoma (5-13). In this study, we analyzed the TCGA database of patients with pancreatic cancer and identified gene that was more strongly expressed in pancreatic cancer tissue compared with normal pancreatic tissue and associated with poor prognosis using UALCAN and GEPIA.

Casein kinase 1 alpha 1 (CSNK1A1) is a Ser/Thr protein kinase belonging to the casein kinase I subfamily. Casein kinases are known to be involved in various signal transduction pathways and have also been reported to be involved in various diseases such as inflammation, cancer,

and neurological disorders (14). CSNK1A1 can phosphorylate multiple proteins and is involved in Wnt signaling. CSNK1A1 phosphorylates β-catenin, inducing its degradation and inhibiting downstream Wnt signaling (15). CSNK1A1 has also been implicated in regulating DNA damage response pathways, suggesting a potential role in promoting genomic instability (16, 17). Changes in *CSNK1A1* mRNA expression in tumor cells and tissues, and the association between expression levels and prognosis, have been reported in various tumor types, and Schittek *et al.* reported that *CSNK1A1* mRNA expression was increased in brain tumor tissues, prostate cancer tissues, lymphoma cells, and leukemia cells, whereas *CSNK1A1* mRNA expression was decreased in bladder cancer, lung cancer, and melanoma tissues (18). Sinnberg *et al*. have confirmed that in the case of melanoma, *CSNK1A1* mRNA and protein expression decreases as the tumor progresses (19). Richter *et al*. reported that *CSNK1A1* is over-expressed in colorectal cancer tumor tissues compared to normal tissues, and that over-expression of *CSNK1A1* in tumor tissues correlates with a poor prognosis in patients with colorectal cancer (20). Liu *et al.* reported that suppressing *CSNK1A1* expression or inhibiting it with CSNK1A1 inhibitors effectively suppressed the proliferation of glioblastoma multiforme cells, while over-expression of *CSNK1A1* promoted cell proliferation and colony formation (21). Järås *et al*. showed that CSNK1A1 is essential for the survival of acute myeloid leukemia cells, and that inhibition of CSNK1A1 selectively eliminates leukemia cells (22).

Based on these findings, CSNK1A1 mRNA expression appears to be elevated in some cancer tissues but decreased in others. Therefore, in this study, we used the TCGA database to determine whether CSNK1A1 mRNA expression is elevated in pancreatic cancer and to evaluate the relationship between CSNK1A1 expression levels and survival in patients with pancreatic cancer.

Materials and Methods

Evaluation of CSNK1A1 expression in cancer tissues from patients with pancreatic adenocarcinoma. The gene name "*CSNK1A1*" is registered in the TCGA database. The expression levels of *CSNK1A1* mRNA in tumor tissues from patients with PAAD were investigated using the GEPIA platform (4). mRNA expression data were normalized, and expression values are presented in transcript per million (TPM).

Analysis for protein expression of CSNK1A1 in pancreatic adenocarcinoma tissues. The UALCAN platform was used to investigate protein levels of CSK1A1 in PAAD tissues (23). From the Clinical Proteomic Tumor Analysis Consortium (CPTAC) PAAD dataset, protein levels of CSK1A1 in cancer tissues of patients with PAAD were investigated using UALCAN platform.

Survival analysis according to CSNK1A1 mRNA expression levels in pancreatic adenocarcinoma tissues. Survival analysis was performed using the UALCAN platform to investigate the effect of *CSNK1A1* expression level on the survival of patients with PAAD. The gene name "*CSNK1A1*" was entered into the TCGA database to generate Kaplan-Meier curves for patients with PAAD. Furthermore, the effect of *CSNK1A1* mRNA expression level on overall survival and disease-free survival in patients with PAAD was investigated using the bioinformatics platform GEPIA.

Statistical analysis. Both UALCAN and GEPIA perform differential analysis automatically within their respective programs. In UALCAN, mRNA levels are calculated using Student's *t*-tests, with disease status (tumor or normal) as the variable for calculating differential expression, and survival curves are calculated using Log-rank tests. In GEPIA, mRNA levels are calculated using unpaired two-tailed Student's *t*-tests, and survival curves are calculated using Log-rank tests. A *p*-value less than 0.05 was considered statistically significant.

Results

CSNK1A1 mRNA expression was up-regulated in pancreatic adenocarcinoma tissues. To investigate whether *CSNK1A1*

mRNA expression in PAAD tissues is increased compared with normal pancreatic tissues, we analyzed the TCGA dataset using the GEPIA platform. Figure 1 shows the expression levels of *CSNK1A1* mRNA in PAAD tissues and normal pancreatic tissues. *CSNK1A1* mRNA levels were significantly elevated in PAAD tissues compared with normal pancreatic tissues (p<0.05).

The protein expression levels of CSNK1A1 were increased in pancreatic adenocarcinoma tissues. We analyzed the CPTAC dataset using the UALCAN platform to assess whether the protein expression level of CSNK1A1 is increased in PAAD tissues. The results showed that the protein levels of CSNK1A1 were increased in PAAD tissues (n=137) compared with normal tissues (n=74) (p<0.01) (Figure 2).

Increased CSNK1A1 mRNA expression levels are associated with shorter survival in patients with pancreatic adenocarcinoma. Using the UALCAN platform, Kaplan-Meier survival curves were generated for patients with PAAD tissue exhibiting high (n=45) and low/moderate (n=132) CSNK1A1 expression levels. The results showed that elevated *CSNK1A1* mRNA expression levels correlated with shorter patient survival (p=0.0028) (Figure 3). Furthermore, GEPIA analysis showed no significant difference in overall survival between patients with low *CSNK1A1* expression and those with high expression, but patients with high expression had a shorter overall survival period (p=0.055) (Figure 4A). However, the use of GEPIA patients with high *CSNK1A1* expression had a significantly shorter disease-free survival period (p=0.038) (Figure 4B).

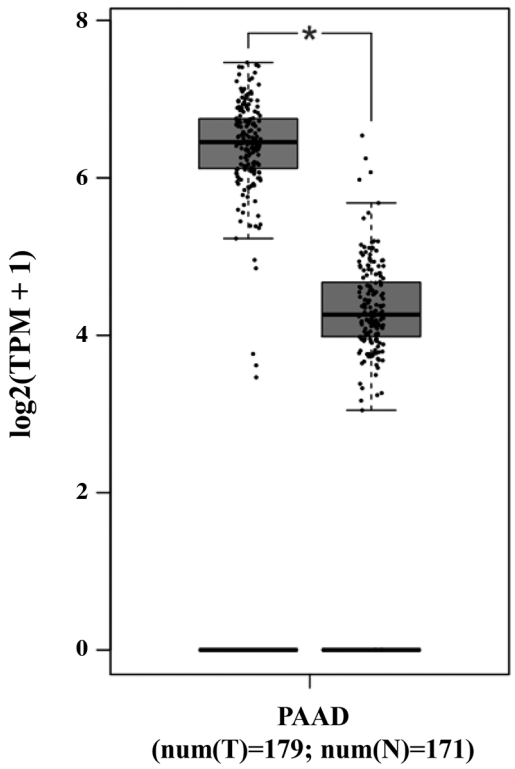

Figure 1. *Casein kinase 1 alpha 1 (CSNK1A1) mRNA expression analysis of pancreatic adenocarcinoma (PAAD) tissues from The Cancer Genome Atlas (TCGA) database. Box plots were downloaded from the Gene Expression Profiling Interactive Analysis (GEPIA) platform based on the PAAD dataset from TCGA. CSNK1A1 mRNA expression in PAAD tissues was analyzed. The left box represents CSNK1A1 mRNA expression in PAAD tissues (n=179), whereas the right box represents CSNK1A1 mRNA expression in normal pancreatic tissues (n=171). p<0.05 was regarded as statistically significant.*

Discussion

In the present study, *CSNK1A1* mRNA expression and Kaplan-Meier survival in patients with PAAD were analyzed using the TCGA database. The results showed that *CSNK1A1* mRNA and protein expression levels were significantly increased in PAAD tissues compared with normal pancreatic tissues. Furthermore, increased *CSNK1A1* mRNA expression correlated with shorter overall survival and disease-free survival in patients with PAAD.

CSNK1A1 is a serine/threonine protein kinase capable of phosphorylating multiple proteins and is involved in many signaling pathways, including Wnt signaling, and circadian rhythm regulation. Increased or decreased expression of CSNK1A1 has been reported in many types of cancer tissues. The increased mRNA expression has been reported in brain tumor tissues, prostate cancer tissues, colorectal cancer tissues, lung adenocarcinoma tissues, lung squamous cell carcinoma tissues, lymphoma cells, and leukemia cells, while decreased expression has

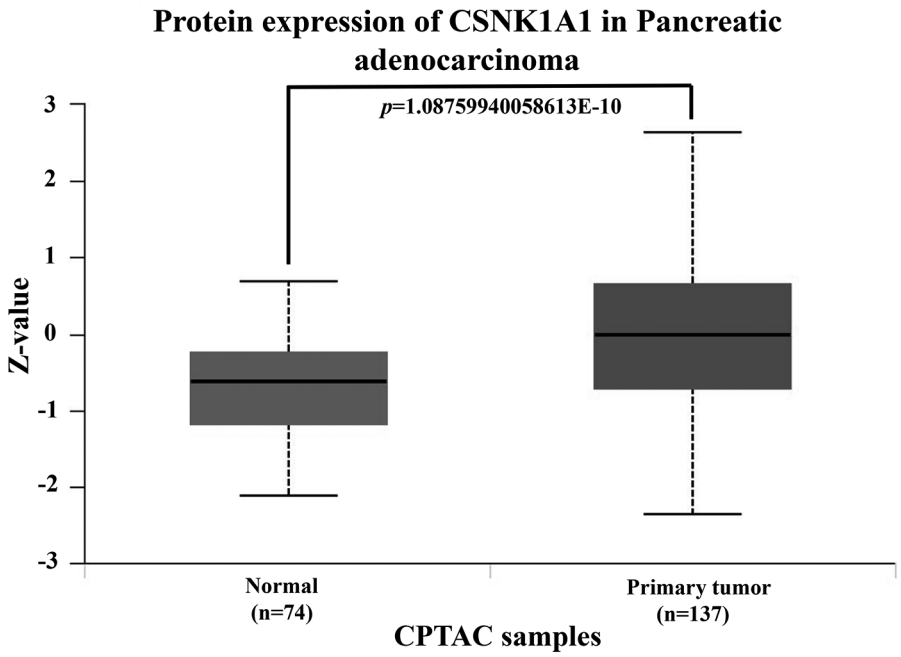

Figure 2. *Casein kinase 1 alpha 1 (CSNK1A1) protein expression analysis for pancreatic adenocarcinoma (PAAD) tissues from the Clinical Proteomic Tumor Analysis Consortium (CPTAC) database. The boxplots were downloaded from the University of Alabama at Birmingham Cancer Data Analysis Portal (UALCAN) based on Clinical Proteomic Tumor Analysis Consortium (CPTAC) PAAD dataset. The left box represents CSNK1A1 protein expression in normal pancreatic tissues (n=74), whereas the right box represents CSNK1A1 protein expression in pancreatic adenocarcinoma tissues (n=137) (p<0.001). p<0.05 was regarded as statistically significant.*

been reported in bladder cancer tissues, small cell lung cancer tissues, and melanoma tissues, indicating that the increase or decrease in expression depends on the type of tumor cell (18-20, 24, 25).

Furthermore, the relationship between the degree of expression of this gene and prognosis depends on the tumor. One type of cancer in which high expression of *CSNK1A1* negatively impacts prognosis is colorectal cancer. *CSNK1A1* is over-expressed in colorectal cancer tissues, and over-expression of *CSNK1A1* is correlated with poor prognosis in colorectal cancer patients (20). In glioblastoma cells, suppression or inhibition of *CSNK1A1* expression effectively suppressed glioblastoma cell proliferation, while over-expression of *CSNK1A1* promoted cell proliferation and colony formation (21). Furthermore, since inhibition of CSNK1A1 was shown to selectively eliminate leukemia cells, it became clear that CSNK1A1 is essential for the survival of acute myeloid leukemia cells (22). Conversely, in the case of melanoma, suppression of *CSNK1A1* in melanoma cells induces the switching of β-catenin signaling and promotes metastasis (19). Based on the reports mentioned above, it appears that CSNK1A1 expression depends on the type of tumor and that this also affects patient prognosis differently.

How does CSNK1A1 worsen prognosis of patients with cancer? As mentioned above, CSNK1A1 phosphorylates a specific site of β-catenin, thereby promoting subsequent phosphorylation by GSK3β and ubiquitination. As a result, it maintains low β-catenin levels when Wnt signaling is off, suppressing transcriptional activation. In other words, it acts as a negative regulator of Wnt signaling (26). This appears to contradict our *in silico* analysis results, which indicated that high expression of *CSNK1A1* negatively impacts the prognosis of patients with pancreatic cancer. In some cancers, such as melanoma, patients with high *CSNK1A1* expression may have a better prognosis due to this mechanism (19).

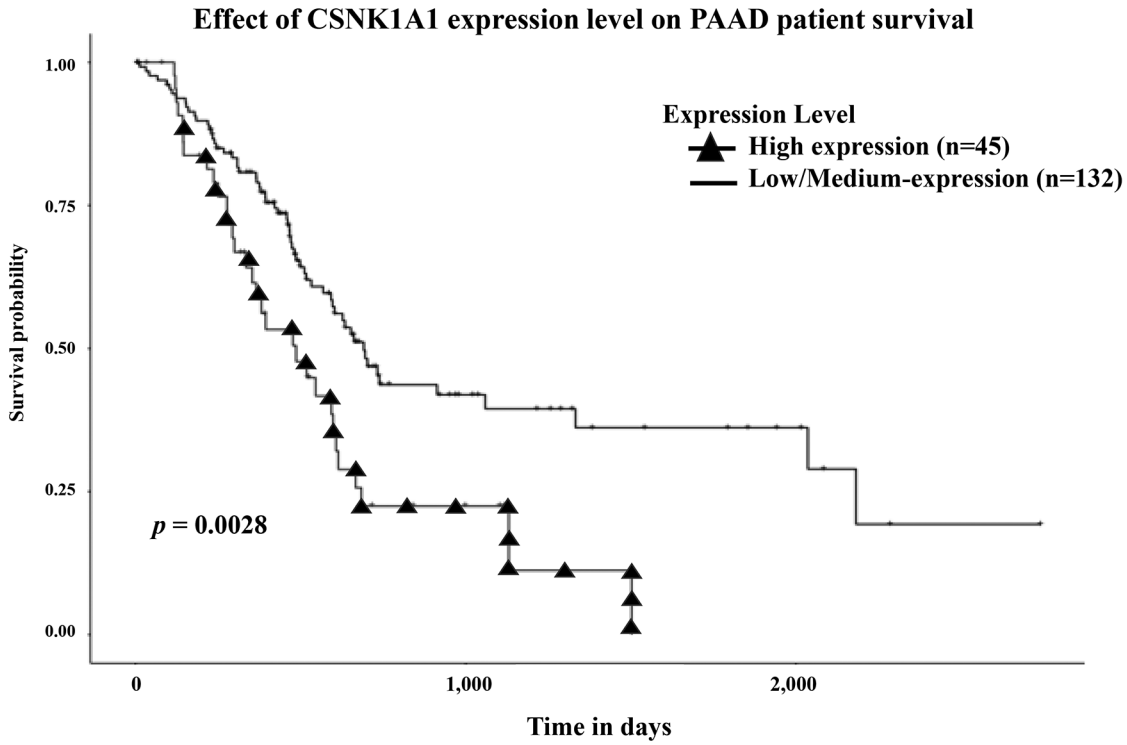

Figure 3. *Kaplan-Meier survival curves of pancreatic adenocarcinoma patients with high and low/medium levels of casein kinase 1 alpha 1 (CSNK1A1). The overall survival rate of patients with pancreatic adenocarcinoma (PAAD) was analyzed based on CSNK1A1 expression levels using the University of Alabama at Birmingham Cancer Data Analysis Portal (UALCAN) platform. Survival curves of patients with PAAD were compared between patients with high CSNK1A1 expression levels (▲, n=45) and patients with low/intermediate CSNK1A1 expression levels (n=132) (p=0.0028). p<0.05 was considered statistically significant.*

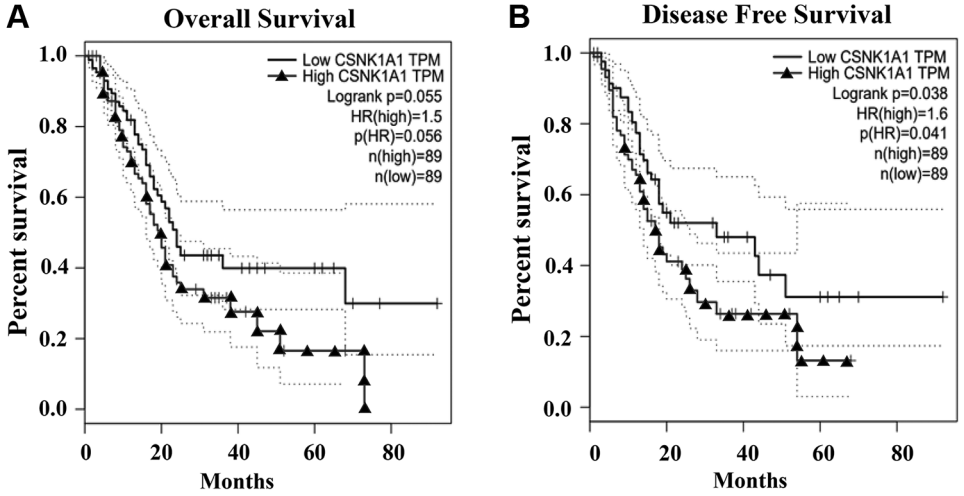

Figure 4. *Kaplan-Meier survival curves of overall survival and disease-free survival in patients with pancreatic adenocarcinoma (PAAD) with high and low casein kinase 1 alpha 1 (CSNK1A1) expression levels. (A) Overall survival analysis and (B) disease-free survival analysis of PAAD patients were performed using the Gene Expression Profiling Interactive Analysis (GEPIA) platform based on CSNK1A1 mRNA expression levels. Overall survival and disease-free survival curves for PAAD patients were compared between the high CSNK1A1 expression group (▲, n=89) and the low CSNK1A1 expression group (n=89). p<0.05 was considered statistically significant.*

However, CSNK1A1 may also influence cancer cell progression through a different mechanism. CSNK1A1 phosphorylates MDMX and promotes the binding of MDMX to p53. As a result, the DNA-binding activity of p53 is suppressed, and the transcription of p53 target genes such as *p21* is reduced. Therefore, apoptosis in cancer cells is suppressed, and cancer progresses. However, homozygous deficiency of *CSNK1A1* in mouse colonic epithelium has been reported to induce β-catenin accumulation and potent activation of p53, thereby inducing senescence and suppressing tumorigenesis. In other words, *CSNK1A1* deficiency itself enhances p53-dependent tumor suppression at least in colorectal cancer (27).

Although few studies have examined the relationship between CSNK1A1 and pancreatic cancer, Venkat *et al*. reported that knockdown or pharmacological inhibition of CSNK1A1 reduces the proliferation and clonal capacity of PAAD cells (28).

Our *in silico* analysis results clearly show that CSNK1A1 worsens the prognosis of patients with pancreatic cancer. Therefore, future research will aim to clarify how CSNK1A1 is involved in the progression of malignancy within pancreatic cancer cells.

Conflicts of Interest

The Authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Authors' Contributions

All Authors contributed to the conception and design of the study. Data collection and analysis were performed by Shin-nosuke Yamashita, Yoshiatsu Tanaka, Shajedul Islam and Yasuhiro Kuramitsu. Shin-nosuke Yamashita and Yoshiatsu Tanaka wrote the first draft of the manuscript, Takao Kitagawa and Yasuhiro Kuramitsu commented on an earlier version of the manuscript. All Authors read and approved the final manuscript.

Artificial Intelligence (AI) Disclosure

No artificial intelligence (AI) tools, including large language models or machine learning software, were used in the preparation, analysis, or presentation of this manuscript.

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