High Expression of PXN Predicts a Poor Prognosis in Acute myeloid leukemia

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High Expression of *PXN* Predicts a Poor Prognosis in Acute myeloid leukemia

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Abstract

Background: Acute myeloid leukemia (AML) is one of the common malignant diseases of hematopoietic system. Paxillin (*PXN*) is an important part of focal adhesions (FAs), which is related to the poor prognosis of many kinds of malignant tumors. However, no research has focused on the expression of *PXN* in AML. We aimed to investigate the expression of *PXN* in AML and its prognostic significance.

Methods: Using GEPIA and UALCAN database to analyze the expression of *PXN* in AML patients and its prognostic significance. Bone marrow samples of newly diagnosed AML patients were collected to extract RNA, and qRT-PCR was used to detect the expression of *PXN*. The

prognosis was followed up. Chi-square test was used to analyze the relationship between *PXN* expression and clinical laboratory characteristics. Kaplan-Meier analysis was used to draw survival curve, and Cox regression analysis was used to analyze the independent factors affecting the prognosis of patients with AML. The co-expression genes of *PXN* were analyzed by LinkedOmics to explore its biological significance in AML.

Results: Kaplan-Meier analysis showed that the overall survival time of AML patients was related to whether to receive treatment and *PXN* expression ($P < 0.05$). COX regression analysis showed that whether to receive treatment ($HR = 0.227$, $95\%CI = 0.075-0.689$, $P = 0.009$) and high expression of *PXN* ($HR = 4.484$, $95\%CI = 1.449-13.889$, $P = 0.009$) were independent poor prognostic factors in patients with AML.

Conclusion: *PXN* is highly expressed in AML patient, and high *PXN* expression is an indicator of poor prognosis in AML patient.

Keywords: Acute myeloid leukemia, *PXN*, prognosis, Paxillin, overall survival

1. Introduction

Acute myeloid leukemia (acute myeloid leukemia, AML) is a kind of hematopoietic malignant tumor with high heterogeneity, which is characterized by excessive proliferation of bone marrow primordial myeloid cells, accounting for about 60% of adult acute leukemia[1]. Now it is generally believed that the biological characteristics of AML are caused and determined by its own genetic abnormalities[2]. According to the effect of cytogenetic differences on prognosis, AML can be divided into low-risk group, medium-risk group and high-risk group. The five-year overall survival rate (overall survival OS) is 55%, 24% and 5%, respectively[3]. However, due to the limitation of methodological sensitivity, nearly half of AML patients still fail to detect genetic abnormalities using existing cytogenetic detection techniques[4]. Therefore, it is extremely important to look for abnormal molecules related to the prognosis of AML, classify the risk of patients, and then adopt stratified treatment to improve the therapeutic effect.

The paxillin (*PXN*) gene is located on human chromosome 12q24.31 and encodes for a focal adhesion molecule of 68kDa, which is a multidomain adaptor protein connecting extracellular matrices to the cytoskeleton[5, 6]. Paxillin plays an important role in the process of transforming

extracellular signal into intracellular biological response, which is triggered by the binding of integrin and extracellular matrix[7]. *PXN* is generally considered to be related to the movement, migration and invasion of cancer cells, and ultimately related to tumor metastasis. Studies have shown that *PXN* functional mutations are associated with the malignant progression of many tumors, including breast cancer, lung carcinoma[5], prostate cancer[8], melanoma and colorectal cancer[9]. To the best of our knowledge, the function of *PXN* has not been reported in AML. In this study, we focused on the expression and prognostic significance of *PXN* via bioinformatics analysis and detection of expression in clinical samples.

2. Methods

2.1 Data Source

GEPIA (<http://gepia.cancer-pku.cn/>) is a web-based tool based on The Cancer Genome Atlas (TCGA) and Genotype-Tissue Expression (GTEx) project. It provides rapid and customizable functions and key interactive functions, including differential expression analysis, profiling plotting, correlation analysis and patient survival analysis[10]. UALCAN (<http://ualcan.path.uab.edu>) is an easy-to-use interactive portal for in-depth analysis of TCGA gene expression data[11]. In our study, we studied the prognostic value of *PXN* gene in the "Single Gene Analysis" module of GEPIA database. UALCAN was used to verify the prognostic value of *PXN* through the "Survival Analysis" module and the "AML" dataset. Expression data of *PXN* was examined via the "Expression Analysis" module and the "AML" dataset. The P value cutoff was 0.05. The LinkedOmics (<http://www.linkedomics.org/login.php>) is an interactive web-portal including 32 TCGA cancer-associated data[12]. We used Pearson test to analyze the expression of differential genes related to *PXN* in AML, and GESA was used to analyze GO-BP and KEGG pathways. The threshold was based on the following values: FDR of 0.05 and simulations of 500.

2.2 Patients

We collected bone marrow blood from 27 newly diagnosed AML patients in the affiliated Hospital of Southwest Medical University from December 2019 to March 2021. The study was approved by the Clinical trial Ethics Committee of the affiliated Hospital of Southwest Medical University in accordance with Helsinki guidelines for the protection of human subjects. All

participants freely gave written informed consent. The diagnosis of AML was based on the 2008 WHO criteria [13]. The exclusion criteria for selected patients with AML are as follows: (1) relapsed AML, (2) with other malignant tumors, (3) with severe hepatic and renal insufficiency, (4) received hematopoietic stem cell transplantation. All the patients were mainly treated with DA (daunorubicin 45 mg/m²/day for 3 days and cytarabine 100 mg/m²/day for 7 days) and IA regimen (idarubicin 8-10 mg/m²/day for 3 days and cytarabine 150 mg/m²/day for 7 days).

2.3 Quantitative real-time PCR analysis (qRT-PCR)

Fresh bone marrow samples were collected. Total RNA was extracted from fresh bone marrow samples using the TRIzol Reagent (TIANGEN) according to the manufacturers' protocol. RNA was Reverse-transcribed using a Rever Tra Ace qPCR RT Kit (Toyobo). The concentration and quality of RNA samples were examined by Nanodrop (Thermo Fisher). RT-qPCR was conducted using a Light Cycler 96 (Roche) with the following amplification protocol: denaturation at 95°C for 30s, 40 cycles of denaturation at 95°C for 5 s, annealing at 55°C for 10s, and extension at 72°C for 15s. The mRNA expression levels were normalized to GAPDH. The sequences of primers were as follows: *PXN* 5'-GATTCATCCACCAGCAGCCTCAG-3'(forward) and 5'-GCACGGAGAGCCAACACTGTC-3'(reverse); All primers were synthesized by Sangon Biotech (Shanghai, China). Relative messenger RNA (mRNA) levels were measured using the 2^{-Δcycle threshold} (2^{-ΔCT}) method [14].

2.4 Statistical Analysis

SPSS 25.0 software was used for statistical analysis. According to the median level of *PXN* expression, AML patients were divided into high expression group and low expression group. Chi-square test was used to examine the relationship between *PXN* expression and clinicopathological features. The survival curve was drawn by Kaplan-Meier method, and the long-rank test was used in the group comparison. The Cox-regression analysis was used to determine the independent prognostic factors of patients with AML. The hazard ratio (HR) and its corresponding 95% confidence interval (CI) were calculated. The probability value of less than 0.05 indicates statistical significance.

3. Results

3.1 Up-regulation of *PXN* expression in patients with acute myeloid leukemia

We detected the expression of *PXN* gene in bone marrow samples of 27 patients with AML and 10 normal controls. It can be seen from Figure 1A that the expression of *PXN* in AML is higher than that in normal controls ($P < 0.05$), indicating that *PXN* may be a suppressor gene in AML. In addition, we used UALCAN database to study the expression of *PXN* gene in different FAB classification. From Figure 1B, we can see that there are differences in the expression of *PXN* gene among different FAB classifications of leukemia, and the difference is statistically significant. Furthermore, we used GEPIA and UALCAN database to analyze the relationship between *PXN* gene and survival in patients with AML. As shown in Figure 2, the lower expression of *PXN* gene predicts a better prognosis of AML patients.

3.2 The relationship between the expression of *PXN* and clinical features

Bone marrow samples from 27 patients with newly diagnosed AML were collected, and the mRNA level of *PXN* was detected by qRT-PCR method. In order to further evaluate the clinical correlation of *PXN*, 27 patients with AML were divided into two groups according to the median: low *PXN* expression group (n=14) and high *PXN* expression group (n=13), as shown in Figure 3A. Table 1 summarized the relationship between *PXN* expression and clinical features in 27 patients with AML, as shown in Figure 3B-F. There was no significant correlation between *PXN* expression level and age, sex, peripheral blood leukocyte, FAB classification, treatment or not, and the mutation frequency of *CEBPA*, *TET2*, *DNMT3A*, *NPM1* and *IDH1/2*.

3.3 The prognostic value of *PXN* expression in patients with AML

The Kaplan-Meier method was used to evaluate the prognostic factors such as age, gene mutation, whether to receive treatment and *PXN* expression in patients with AML. The prognosis of patients with high *PXN* expression was poorer than that of patients with low *PXN* expression. The Log rank test data is shown in Table 2. Multivariate regression analysis showed that refusal of treatment and high transcript level of *PXN* were independent factors for poor prognosis in patients with AML. The survival time of AML patients with high *PXN* expression was significantly shorter than those with low *PXN* expression, suggesting that the prognosis of AML patients with high *PXN*

transcript level was poor (Table3), and the difference was statistically significant (Figure 4).

3.4 Co-expression Network of *PXN* in Leukemia

We used the LinkedOmics functional module to analyze the co-expressed genes of *PXN* in order to explore its biological significance in AML. The differentially expressed genes related to *PXN* in AML were analyzed by Pearson Test, and the results were shown in Figure 5A. In addition, *PXN* was positively correlated with IL27RA (Pearson correlation= 0.67, P= 1.141E-23), CD52 (Pearson correlation= 0.59, P= 3.044E-17) and GIMAP1 (Pearson correlation= 0.58, P= 1.088E-16), indicating that *PXN* is related to inflammation and immune response (Figure 5B, C).

Through the analysis of Gene Ontology (GO) database, we found that *PXN*-related co-expression genes are mainly involved in the lymphocyte-mediated immune, the positive regulation of defense response and the regulation of intercellular adhesion function (Figure 6A). Besides, through Kyoto Encyclopedia of Genes and Genomes (KEGG) analysis, it was confirmed that *PXN* co-expression genes were enriched in cytokine receptor interaction, T cell receptor signal pathway, Toll-like receptor signal pathway and RAP1 signal pathway (Figure 6B). Therefore, we believe that *PXN* has a wide range of effects on immune response and regulation in AML.

4. Discussion

Acute myeloid leukemia (AML) is a kind of hematopoietic malignant tumor with high heterogeneity, which is mainly characterized by uncontrolled proliferation of immature hematopoietic precursor cells in the bone marrow [1]. Now it is generally believed that the biological characteristics of AML are caused and determined by its own genetic abnormalities [14]. In recent years, great progress has been made in the search for prognostic markers of AML at the genetic level and its application in the re-stratification of AML. The screening for novel molecular markers with gene mutation or abnormal expression will further update people's understanding of the pathogenesis of AML [15].

PXN is located on human chromosome 12q24.31 and encodes a cytoskeleton protein that involves the adhesion of actin membrane to extracellular matrix[15, 16]. The high expression of *PXN* in some tumors is related to tumor stage, poor differentiation, lymphatic metastasis, distant metastasis and tumor lymph node metastasis stage[17, 18]. However, the relationship between *PXN* and AML has not been reported before. Therefore, we collected AML and normal samples to explore

the expression and prognostic value of *PXN* mRNA in AML.

In this study, GEPIA database analysis showed that the level of *PXN* transcription in AML patients was higher than that in normal subjects, and the prognosis of patients with high expression of *PXN* was poor. Subsequently, we collected bone marrow samples from 49 newly diagnosed AML patients for qRT-PCR detection, and reached the same conclusion. Through Kaplan-Meier curve and log rank test, we found that the high expression of *PXN* was significantly correlated with OS in patients with AML, which was consistent with the results of GEPIA and UALCAN database. Therefore, we think that *PXN* can be used as a prognostic marker for AML patients.

In order to further explore the biological significance of *PXN* in AML, we used LinkedOmics functional module to analyze *PXN* co-expression genes. The results show that *PXN* participates in a variety of functional networks related to inflammation and immune response, such as lymphocyte-mediated immunity, positive regulation of defense response, T cell receptor and Toll-like receptor signal pathway.

PXN is an important component of the focal adhesion complex, which is related to the occurrence, development and metastasis of human cancer, and is related to the poor clinical prognosis of tumor patients, which has been reported in many previous studies[19-21]. *PXN* is directly regulated by ETV4, and its overexpression is related to the poor prognosis of human NSCLC[22]. *PXN* promotes tumor progression and metastasis of gastric cancer, breast cancer and colorectal cancer, and its overexpression is associated with poor prognosis[15, 23-25]. CD8⁺T cells play an important role in immune-related tolerance and immunosuppression in tumor microenvironment (TME)[26]. It has been reported that in breast cancer, lung squamous cell carcinoma and cutaneous melanoma, the expression of *PXN* is negatively correlated with the infiltration of CD8⁺ and human epidermal growth factor receptor 2-negative T cells, while the expression of *PXN* in DLBC is positively correlated with CD8⁺T cell infiltration[27]. The binding of *PXN* and CD103 triggers the signal transduction of $\alpha_E\beta_7$ integrin from outside to inside, which promotes the migration behavior and effect function of CD8⁺T cells[28]. Other studies have shown that *PXN* has an important relationship with macrophages and phagocytosis[29]. In macrophages, miR-375 targets *PXN*, directly to enhance the migration and infiltration of macrophages to tumor spheroids and xenografted mice[30]. Taken together, *PXN* has a wide range of effects on immune response, and we speculate that the poor prognosis caused by the high expression of *PXN* in AML

may be related to the immune infiltration and immune escape of tumor cells.

In conclusion, our research shows that the expression of *PXN* in AML patients is higher than that in healthy people, and the AML patients with high expression of *PXN* indicate a poor prognosis. This is the first report of *PXN* in AML. Our group is expanding the sample size, hoping to further study the signal transduction pathway between the high expression of *PXN* and AML.

5. Conclusions

Our research shows that the expression of *PXN* in AML patients is higher than that in healthy people, and the AML patients with high expression of *PXN* indicate a poor prognosis. This is the first report of *PXN* in AML. Our group is expanding the sample size, hoping to further study the signal transduction pathway between the high expression of *PXN* and AML.

Supplementary information

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Authors' contributions

CONCEPTION: Chunlan Huang, Xinwen Zhang, Hao Xiong

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SUPERVISION: Chunlan, Huang

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Ethics approval and consent to participate

Research Involving Human Participants and/or Animals: This study involves blood and bone marrow aspirates from AML patients. Ethical approval was obtained from institutional ethics committee (AF-V4-22.0) to conduct the study.

Informed Consent: Informed consent was obtained from all individual participants included in the study.

Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional committee.

Consent for publication

Not Applicable.

Competing interests

The authors declare that they have no competing interests.

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Figures

Figure 1

The expression levels of PXN based on common data base. A) The expression levels of PXN in AML patients (n=27) and Normal (n=10). B) The expression levels of PXN in AML based on FAB classification (UALCAN).

Figure 2

The effect of PXN expression level on the survival outcomes of patients was analyzed based on datasets. A): The relationship between PXN expression and prognosis in patients with AML based on UALCAN

dataset. B): The relationship between PXN expression and prognosis of patients with AML based on GEPIA dataset.

Figure 3

The relationship between the expression level of PXN and clinicopathological characteristics. A) AML patients were divided into high expression group and low expression group according to the median expression of PXN. B-F) The correlation between PXN expression level and CEBPA, TET2, DNMTA3, NPM1 and IDH1/2 gene mutation.

Figure 4

The Kaplan-Meier survival analysis was used to analyze the prognostic factors of 27 AML patients. A) The relationship between PXN expression and overall survival time in 27 AML patients. B) The relationship between treatment condition and overall survival time in 27 AML patients.

Figure 5

The differentially expressed genes related to PAW in AML (LinkedOmics). (A) Using Pearson test to analyze genes differentially expressed in correlation with PXN in AML.(B) Heat map shows the top 50 genes with significant positive correlation with PXN. (C) Heat map showed that the top 50 genes were significantly negatively correlated with PXN.

Figure 6

GO annotations and KEGG pathways enriched with PXN co-expressed genes in leukemia (LinkedOmics). Using GSEA to analyze the significantly enriched GO-BP and KEGG pathways of PXN co-expression genes in AML. (A) Gene Ontology Biological processes, analyzed by GSEA. (B) KEGG pathway, analyzed by GSEA. The Leading EdgeNum and the false discovery rate (FOR) are shown in blue and orange column, respectively.

Supplementary Files

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