Received: 19 July 2023 | Revised: 3 October 2023 | Accepted: 19 October 2023

DOI: 10.1002/cam4.6669

BRIEF COMMUNICATION

Cancer Medicine Open Access WILEY

The clinical features and prognostic implications of *PTPN11* mutation in adult patients with acute myeloid leukemia in China

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Funding information
1·3·5 project for disciplines of excellence-Clinical Research Incubation Project, West China Hospital, Sichuan University, Grant/Award Number: 2022HXFH040

Abstract

Background: The clinical significance of protein tyrosine phosphatase non-receptor type 11 mutation (*PTPN11*^{mut}) in acute myeloid leukemia (AML) is underestimated.

Methods: We collected the data of AML patients with mutated *PTPN11* and wild-type *PTPN11* (*PTPN11*^{wt}) treated at our hospital and analyzed their clinical characteristics and prognosis.

Results: Fifty-nine *PTPN11*^{mut} and 124 *PTPN11*^{wt} AML patients were included. *PTPN11*^{mut} was more common in myelomonocytic and monocytic leukemia, and was more likely to co-mutate with *KRAS*, *KMT2C*, *NRAS*, *U2AF1*, *NOTCH1*, *IKZF1*, and *USH2A* mutations than *PTPN11*^{wt}. The overall survival for AML patients with *PTPN11*^{mut} was significantly shorter than that for those with *PTPN11*^{wt} ($p = 0.03$). The negative impact of *PTPN11*^{mut} on overall survival was pronounced in the "favorable" and "intermediate" groups of ELN2017 risk stratification, as well as in the wild-type *NPM1* group ($p = 0.01$, $p = 0.01$, and $p = 0.04$).

Conclusion: *PTPN11*^{mut} is associated with distinct clinical and molecular characteristics, and adverse prognosis in AML patients.

KEYWORDS

acute myeloid leukemia, event-free survival, overall survival, *PTPN11*

1 | INTRODUCTION

Acute myeloid leukemia (AML) is a hematologic malignancy caused by clonal proliferation and differentiation arrest of myeloid precursors.[1] Somatic mutations, such as *NPM1*, *FLT3-ITD*, and *TP53*, enable hematopoietic stem and progenitor cells to acquire the ability of self-renewal, which is crucial for the pathogenesis and prognosis of AML.[2]

Mutation in tyrosine-protein phosphatase nonreceptor type 11 (*PTPN11*^{mut}) can be found in 1.5%–12% of adult AML patients.[3] The *PTPN11* gene, located on chromosome 12q24, encodes a protein composed of a N-terminal Src homology 2 (N-SH2), a protein tyrosine phosphatase (PTP)

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catalytic domain and a C-terminal SH2.[4] N-SH2 can prevent the PTP domain from being overactivated.[5] In leukemogenesis, *PTPN11*^{mut} blocks self-inhibition of SH2 catalytic activity, which induces increased sensitivity of hematopoietic stem and progenitor cells to granulocyte-macrophage colony-stimulating factor and RAS signaling pathway hyperactivation, leading to leukemic transformation.[6–9]

Currently, the prognosis of *PTPN11*^{mut} in AML is controversial. Some studies showed that *PTPN11*^{mut} was associated with an adverse prognosis,[10–13] while another report uncovered that AML patients with *PTPN11*^{mut} presented an improved prognosis.[14] In addition, other studies did not observe a significant prognostic effect of *PTPN11*^{mut} in AML.[15,16] Hence, the prognostic value of *PTPN11*^{mut} in AML needs further exploration. Herein, we report a single-center data from China to compare the clinical and molecular features, and outcomes between adult AML patients with mutated *PTPN11* and wild-type *PTPN11* (*PTPN11*^{wt}).

2 | METHODS

We retrospectively collected the data of adult AML patients with *PTPN11*^{mut} (≥18 years of age) from West China Hospital between May 2015 and October 2022. Of 1531 adult AML patients, *PTPN11* mutations were detected in 59 patients (3.9%). Among these 59 *PTPN11*^{mut} patients, 41 patients received chemotherapy. Then, patients with *PTPN11*^{wt} were randomly included in a 3:1 ratio to patients with *PTPN11*^{mut} who received chemotherapy based on diagnosis year, age, sex, and ELN2017 risk stratification. Detailed information about treatment, outcome indicators, detection methods for cytogenetics and molecular biology, and statistical analysis are presented in the Appendix S1 (Figure S4).

3 | RESULTS

3.1 | Baseline characteristics

The baseline characteristics of the *PTPN11*^{mut} and *PTPN11*^{wt} groups were similar and are shown in Table 1. Patients with *PTPN11*^{mut} were significantly more common in acute myelomonocytic and monocytic leukemia (AMML/AMOL) than those with *PTPN11*^{wt} (53.7% vs. 35.5%, $p=0.04$).

3.2 | Mutation landscape

Seventy-four *PTPN11* mutations were identified in 59 patients. Forty-nine (83.1%) patients harbored a single mutation, and 10 (16.9%) carried more than one different *PTPN11* variant (double mutated, $n=7$; triple mutated, $n=1$, quadruple mutated, $n=2$). The multiple mutations among 6 patients were on the same alleles. *PTPN11* mutations were exclusively missense single nucleotide variants. Most mutations (50/74; 67.6%) were localized in the N-SH2 domain, whereas a minority of mutations (21/74; 28.4%) were localized in the PTP domain. These mutations were localized in exons 3, 8, 12, and 13. A72T, the most common amino acid substitution, was found in eight patients (11.1%) (Figure S1). The median VAF of *PTPN11*^{mut} patients was 8.6%, ranging from 1.0% to 57.9%.

In addition, patients with *PTPN11*^{mut} were more likely to co-mutate with *KRAS* (22.0% vs. 4.8%, $p<0.01$), *KMT2C* (10.2% vs. 0.8%, $p<0.01$), *NRAS* (20.3% vs. 8.9%, $p=0.03$), *U2AF1* (11.9% vs. 4.0%, $p=0.045$), *NOTCH1* (5.1% vs. 0%, $p=0.03$), *IKZF1* (5.1% vs. 0%, $p=0.03$), and *USH2A* (5.1% vs. 0%, $p=0.03$) mutations than those with *PTPN11*^{wt} (Figure S2 and Table S1).

3.3 | Response and survival

Although no significant differences were found in complete remission (61.0% vs. 67.7%, $p=0.43$) or event-free survival (EFS) (8.4 vs. 10.2 months, $p=0.26$, Figure 1A) between the *PTPN11*^{mut} and *PTPN11*^{wt} groups, patients with *PTPN11*^{mut} showed shorter overall survival (OS) than patients with *PTPN11*^{wt} (16.2 vs. 34.8 months, $p=0.03$, Figure 1B). In addition, the adverse prognosis was pronounced in the "favorable" and "intermediate" groups of ELN2017 (20.5 months vs. undefined, $p=0.01$; 14.1 vs. 34.8 months, $p=0.01$; Figure 1C). However, in the "adverse" group, the median OS between patients with *PTPN11*^{mut} and *PTPN11*^{wt} was similar (16.2 vs. 13.5 months, $p=0.87$, Figure 1C).

The impact of the coexistence of *NPM1* mutation (*NPM1*^{mut}) and *PTPN11*^{mut} on the prognosis of AML patients was also investigated. In the *NPM1*^{mut} group, there was no difference in OS between patients with *PTPN11*^{mut} and *PTPN11*^{wt} (20.5 months vs. undefined, $p=0.25$, Figure 1D). However, in the *NPM1* wild type (*NPM1*^{wt}) group, patients harboring *PTPN11*^{mut} had a shorter OS than patients with *PTPN11*^{wt} (16.2 vs. 34.8 months, $p=0.04$, Figure 1E).

Moreover, we further studied the prognosis of AMML/AMOL and non-AMML/AMOL patients with *PTPN11*^{mut} and *PTPN11*^{wt}. There were no significant differences between patients with *PTPN11*^{mut} and *PTPN11*^{wt} in either the AMML/AMOL or non-AMML/AMOL groups (16.2 vs. 17.4 months, $p=0.36$; 20.5 vs. 47.1 months, $p=0.07$) (Figure S3).

TABLE 1 The clinical and molecular characteristics of AML patients with *PTPN11*^{mut} and *PTPN11*^{wt}.

Characteristic	*PTPN11*^{mut} ($n=41$)	*PTPN11*^{wt} ($n=124$)	*p* value
Age (years)	54.0 (19.0–75.0)	50.0 (16.0–76.0)	0.52
Sex			
Female	25.0 (61.0)	83.0 (66.9)	0.49
Male	16.0 (39.0)	41.0 (33.1)	
Disease status			
De novo AML	35.0 (85.4)	116.0 (93.5)	0.19
sAML/t-AML	6.0 (14.6)	8.0 (6.5)	
Laboratory			
WBC ($\times10^9$/L)	15.4 (0.6–182.4)	17.7 (0.3–286.8)	0.91
ANC ($\times10^9$/L)	1.0 (0–42.3)	0.9 (0–24.6)	0.58
Platelet ($\times10^9$/L)	63.0 (8.0–634.0)	48.5 (2.0–429.0)	0.16
Hemoglobin (g/dL)	7.4 (3.9–12.3)	7.8 (3.1–13.0)	0.65
LDH (IU/L)	342.5 (136.0–3466.0)	395.0 (118.0–4410.0)	0.83
PB blasts (%)	25.0 (0–99.0)	29.0 (0–99.0)	0.84
BM blasts (%)	51.5 (7.0–96.0)	57.0 (7.0–94.0)	0.47
AMML/AMOL	22.0 (53.7)	44.0 (35.5)	0.04
Extramedullary involvement	4.0 (9.8)	7.0 (5.6)	0.58
Cytogenetics			
Normal	19.0 (46.3)	62.0 (50.0)	0.69
Complex	7.0 (17.1)	11.0 (8.9)	0.24
t(8;21)(q22;q22)	0 (0)	11.0 (8.9)	0.11
inv(16)(p13q22)/t(16;16)(p13;q22)	3.0 (7.3)	8.0 (6.5)	1.00
inv(3)(q21q26)/t(3;3)(q21;q26)	2.0 (4.9)	0 (0)	0.06
−7	1.0 (2.4)	0 (0)	0.25
+8	0 (0)	5.0 (4.0)	0.44
Others	4.0 (9.8)	19.0 (15.3)	0.37
Insufficient	5.0 (12.2)	8.0 (6.5)	0.40
ELN-2017 Risk			
Favorable	18.0 (43.9)	47.0 (37.9)	0.71
Intermediate	8.0 (19.5)	31.0 (25.0)	
Adverse	15.0 (36.6)	46.0 (37.1)	
Treatment			
High intensity	29 (70.7)	102 (82.3)	0.11
Low intensity	12 (29.3)	22 (17.7)	
BM transplant			
allo-HSCT	6.0 (14.6)	22.0 (17.7)	0.65
auto-HSCT	0 (0)	1.0 (0.8)	1.00
CR	25 (61.0)	84 (67.7)	0.43

Note: Values are *n* (%) or median (range).

Abbreviations: allo-HSCT, allogeneic hematopoietic stem cell transplantation; AML, acute myeloid leukemia; AMML/AMOL, acute myelomonocytic and monocytic leukemia; ANC, absolute neutrophil count; auto-HSCT, autologous hematopoietic stem cell transplantation; BM, bone marrow; CR, complete remission; ELN, European LeukemiaNet; LDH, lactate dehydrogenase; PB, peripheral blood; *PTPN11*^{mut}, *PTPN11* mutation; *PTPN11*^{wt}, *PTPN11* wild type; sAML, secondary AML; t-AML, therapy-related AML; WBC, white blood cell.

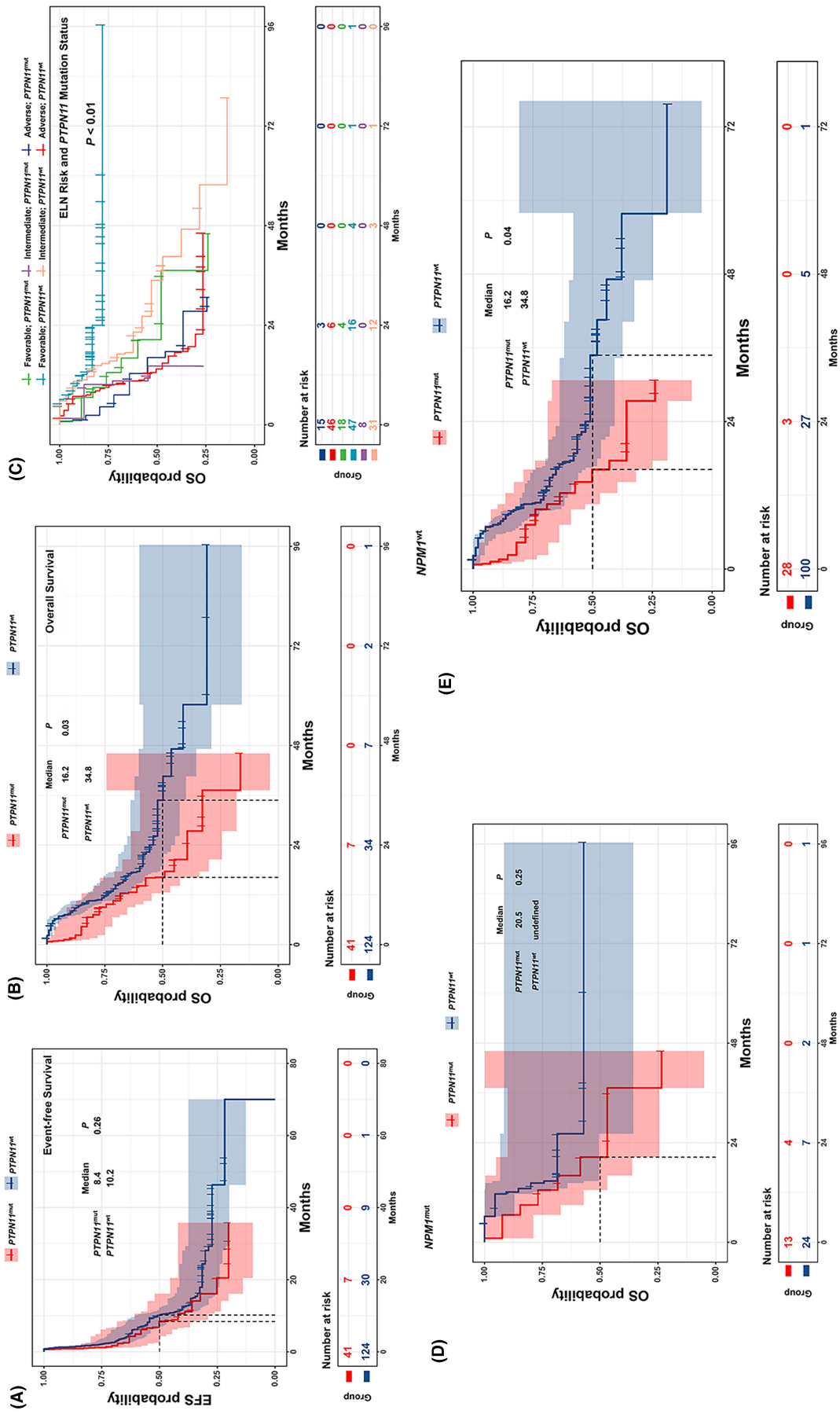

FIGURE 1 (A) EFS of AML patients with *PTPN11*^{mut} and *PTPN11*^{wt}. (B) OS of AML patients with *PTPN11*^{mut} and *PTPN11*^{wt}. (C) OS of AML patients with *PTPN11*^{mut} and *PTPN11*^{wt} based on ELN-2017 risk stratification. (D) OS of *NPM1*^{mut} AML patients with *PTPN11*^{mut} and *PTPN11*^{wt}. (E) OS of *NPM1*^{wt} AML patients with *PTPN11*^{mut} and *PTPN11*^{wt}.

4 | DISCUSSION

Herein, we report the clinical characteristics, gene mutations, and prognosis of adult AML patients with *PTPN11*^{mut} and *PTPN11*^{wt} in a large cohort. We identified that *PTPN11*^{mut} was independently associated with distinct clinical and molecular features and adverse outcomes in AML patients.

Alfayez et al. reported that *PTPN11*^{mut} was more commonly associated with the AMML/AMOL subtype.[11] However, the impacts of *PTPN11*^{mut} on prognosis in AMML/AMOL and non-AMML/AMOL patients have not been reported. In our study, patients with *PTPN11*^{mut} seemed to have a poorer prognosis than patients with *PTPN11*^{wt} in the non-AMML/AMOL group ($p=0.07$). However, in the AMML/AMOL group, both the *PTPN11*^{mut} and *PTPN11*^{wt} groups showed similar OS.

Gene mutations are critical for the prognosis of AML patients. Previous studies have shown differences in gene mutation profiles among patients with *PTPN11*^{mut}. In our study, there were seven patients (12%) with *FLT3-ITD* mutations. Similarly, some studies revealed that the incidence rates of *FLT3-ITD* in *PTPN11*^{mut} AML patients were 16%–27%.[11,16,17] However, Hou et al. identified that *PTPN11*^{mut} and *FLT3-ITD* mutations were mutually exclusive. This difference might be related to the smaller sample size of Hou et al.[15] Moreover, the incidence rates of *NPM1* and *DNMT3A* in AML patients with *PTPN11*^{mut} are controversial. Some researchers reported high incidence rates of *NPM1* and *DNMT3A* with 60.5%–63% and 37%–56.1% in AML patients with *PTPN11*^{mut}, respectively.[14,16,17] However, others uncovered that the incidence rates of *NPM1* and *DNMT3A* in AML patients with *PTPN11*^{mut} were 22%–29% and 24%–27%, respectively.[11,12] Our study supported the results of the latter. Although the most common mutations coexisting with *PTPN11*^{mut} in our study were *DNMT3A* and *NPM1*, the incidence rates of these two mutations were only 25% and 25%, respectively. In addition, our study found that patients with *PTPN11*^{mut} were more likely to harbor *KRAS*, *KMT2C, NRAS*, *U2AF1*, *NOTCH1*, *IKZF1*, and *USH2A* mutations than those with *PTPN11*^{wt}. Given that *PTPN11* can modulate the RAS/MAPK signaling axis,[18] further in vitro and clinical studies are needed to verify whether *PTPN11*^{mut} combined with these mutations has a synergistic effect on the occurrence and development of AML.

A few published studies have focused on the relationships between *PTPN11*^{mut} and the clinical outcomes of AML patients. The impacts of *PTPN11*^{mut} on OS in AML patients are controversial. Some studies showed that *PTPN11*^{mut} were associated with shorter OS.[10,11,17,19] However, others reported that the median OS was similar in both the *PTPN11*^{mut} and *PTPN11*^{wt} groups.[15,20] Metzeler et al.[14] found that patients with *PTPN11*^{mut} had relatively favorable survival outcomes. Our study also showed shorter OS in patients with *PTPN11*^{mut} than in those with *PTPN11*^{wt}. In addition, Alfayez et al. reported that *PTPN11*^{mut} negatively impacted OS across all ELN risk categories.[11] However, Stasik et al. reported that the negative impact of *PTPN11*^{mut} was mainly limited to the ELN favorable risk group.[17] Our study showed that *PTPN11*^{mut} was poor prognostic factor for the "favorable" and "intermediate" groups of ELN2017, which can facilitate further stratification of the two groups. Moreover, our subgroup analysis supported the idea that *PTPN11*^{mut} suggested significantly short OS in AML patients with *NPM1*^{wt}, instead of in patients with *NPM1*^{mut}.[12,15,16,21] Furthermore, our study showed that no significant difference between *PTPN11*^{mut} and *PTPN11*^{wt} patients was observed for EFS, which was similar to the report of Stasik et al.[17] Prospective trials with larger sample sizes and longer follow-up periods are warranted to explore the impacts of *PTPN11*^{mut} on OS and EFS in AML patients.

Our study had some limitations. First, this was a retrospective study, and AML patients with *PTPN11*^{mut} and *PTPN11*^{wt} at a 1:3 ratio were included, which may cause selection bias. To reduce the risk of bias, patients were randomly included based on the year of diagnosis, age, sex, and ELN2017 risk stratification in this study. Moreover, our study did not explore the detailed mechanism of *PTPN11*^{mut} in poor prognosis of AML. Previous studies indicated that the poor prognosis of *PTPN11*^{mut} may be related to its increased resistance to venetoclax and azacitidine.[22,23] More prospective clinical and basic studies are required to elucidate the impact of *PTPN11*^{mut} in AML patients and possible molecular mechanisms in AML progression.

5 | CONCLUSIONS

This study demonstrates that AML patients with *PTPN11*^{mut} are associated with distinct clinical characteristics and poor prognosis.

AUTHOR CONTRIBUTIONS

Jinjun Yang: Conceptualization (equal); data curation (equal); formal analysis (equal); investigation (equal); methodology (equal); software (equal); visualization (equal); writing – original draft (equal). **Lei Zhao:** Conceptualization (equal); data curation (equal); investigation (equal); software (equal); visualization (equal). **Yu Wu:** Resources (equal). **Ting Niu:** Resources (equal). **Yuping Gong:** Resources (equal). **Xinchuan Chen:** Resources (equal). **Xiaoou Huang:** Resources (equal). **Jiazhuo Liu:** Resources (equal). **Yang Dai:** Resources (equal). **Hongbing Ma:** Conceptualization (equal);

resources (equal); supervision (equal); writing – review and editing (equal).

ACKNOWLEDGMENTS

We thank the guidance and assistance of all colleagues from the Department of Hematology and Institute of Hematology of West China Hospital.

FUNDING INFORMATION

The study was funded by 1·3·5 project for disciplines of excellence–Clinical Research Incubation Project, West China Hospital, Sichuan University (No. 2022HXFH040) to Hongbing Ma.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

All the data of our study can be obtained from the corresponding author.

ETHICS STATEMENT

The study was approved by the West China Hospital Institutional Review Board and in accordance with the Helsinki declaration.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Yang J, Zhao L, Wu Y, et al. The clinical features and prognostic implications of *PTPN11* mutation in adult patients with acute myeloid leukemia in China. *Cancer Med*. 2023;12:21111-21117. doi:10.1002/cam4.6669