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Development and validation of nomograms for survival prediction in Fanconi anemia

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To The Editor:

1. INTRODUCTION

Fanconi anemia (FA) is a hereditary disorder characterized by genomic instability and increased sensitivity to DNA cross-linking agents.[1] FA is typically diagnosed in childhood, with an average age at diagnosis of 7 years.[2] Mutations in any of the 23 genes associated with the FA/BRCA pathway can lead to the onset of FA.[3–5] The clinical manifestations of FA primarily include congenital abnormalities, progressive bone marrow failure, and heightened susceptibility to malignancies.[6–8] Among these manifestations, bone marrow failure is the leading cause of death. Patients with FA have a significantly higher risk for developing acute myeloid leukemia (AML), with an incidence approximately 700 times higher than that in the general population.[9,10] Furthermore, patients with FA encounter a 40-fold increased risk for developing solid tumors. As patients age, squamous cell carcinoma (SCC) in the head, neck, and genital areas becomes the most common solid tumor in patients with FA, with the risk for developing SCC 500 to 1000 times higher than that in age-matched peers.[11,12]

Although hematopoietic stem cell transplantation (HSCT) can improve bone marrow failure and reduce the susceptibility to leukemia,[13,14] its application remains limited by factors such as limited donor availability, graft-versus-host disease (GVHD), and increased tumor risk. More importantly, patients with FA who undergo HSCT remain at risk for intrinsic DNA repair deficiencies in other tissues, which pose lifelong threats for malignancies and other complications.[15] Therefore, the early prediction of progression-free survival (PFS) and overall survival (OS) in patients diagnosed with FA, along with personalized treatment strategies tailored to each patient's specific condition, is crucial.

Nomograms have gained attention due to their high reliability in quantifying disease risk. By integrating key prognostic factors, nomograms convert predictive models into intuitive graphs, providing visual survival rate predictions for clinical application.[16–18] However, no specific nomogram currently exists to predict long-term survival in patients diagnosed with FA. As such, the aim of this study was to develop a nomogram that can estimate the survival probabilities of patients with FA after diagnosis, while offering decision support for subsequent treatment strategies.

2. METHODS

2.1. Study participants

This study included data from patients diagnosed with FA at the Institute of Hematology, Chinese Academy of Medical Sciences (Tianjin, China) between August 2001 and September 2023. The clinical diagnostic criteria for FA are as follows[19]: clinical manifestations including congenital abnormalities (eg, thumb abnormalities and microcephaly), progressive bone marrow failure, hematological malignancies, or solid tumors; a positive chromosomal breakage test; and gene testing revealing mutations in ≥1 of the 23 known pathogenic FA genes.

This study was reviewed and approved by the Ethics Committee of the Institute of Hematology and Hematology Research Institute (IIT2021010-EC-2).

2.2. Data collection

Clinical data were collected from outpatient and inpatient medical records, including general information (place of origin, sex, age at first hematological symptom onset, family history of hematological diseases, and other cancers), clinical manifestations (initial symptoms and physical abnormalities), laboratory investigations at diagnosis (complete blood count, bone marrow examination, chromosomal breakage test, and genetic testing), and treatment history and outcomes (transfusion history, medication use, transplantation status, and follow-up results). Follow-ups were conducted through a review of outpatient and inpatient medical records, analysis of questionnaire data, and telephone interviews. The cutoff date for follow-up was November 12, 2023. For deceased patients, the cutoff date was the date of death; for patients lost to follow-up, the cutoff date was the last follow-up date. The primary endpoint was OS, defined as the time from the initiation of therapy to death caused by any reason, censored, or the last follow-up visit. The secondary endpoint PFS was defined as the time from the initiation

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of therapy to either disease progress or death caused by any reason, censored (patients alive without progression at the last clinic date), or the last follow-up. Variables with missing values exceeding 10% were excluded from the analysis,[20] and patients lacking essential variables were excluded from the analysis.

2.3. Statistical analysis and model construction

This study aimed to develop a nomogram encompassing both OS and PFS to predict patient survival. All statistical analyses were performed using R version 4.3.3 (R Core Team [2024]. R: A Language and Environment for Statistical Computing. R Foundation for Statistical Computing, Vienna, Austria) and RStudio with statistical packages including "rms," "survival," "survival ROC," "dcurves," and "regplot." Categorical variables were compared using the chi-squared test, whereas continuous variables were compared using the Mann–Whitney *U* test.

2.3.1. Univariate and multivariate analysis To identify independent prognostic factors associated with FA survival, univariate analysis was initially performed to select significant variables ($p < 0.05$). Subsequently, multivariate analysis was performed to further confirm independent prognostic factors using stepwise regression, with the Akaike information criterion (AIC) employed to minimize the inclusion of variables. Finally, important variables influencing FA survival were selected, and a predictive model was developed.

2.3.2. Nomogram construction A nomogram was developed to create a predictive model based on the selected independent prognostic factors. The nomogram translates the complex statistical model into an intuitive graph, providing survival rate predictions for patients with FA at various time points (eg, 1, 3, and 5 years). This study adhered to the relevant guidelines outlined in the TRIPOD statement.[21]

2.3.3. Model validation The discriminatory performance of the model was evaluated using the concordance index (*C* index) and the area under the receiver operating characteristic curve (AUC). The *C* index ranges from 0.5 to 1, with 0.5 indicating random predictions and 1 indicating perfect predictions. Generally, a *C* index between 0.50 and 0.70 is considered to indicate low accuracy, between 0.71 and 0.90 indicates moderate accuracy, and >0.90 indicates high accuracy.[22] The AUC is analogous to the *C* index and shares the same value range and interpretation. Calibration curves were used to assess the predictive accuracy of the nomogram, and decision curve analysis (DCA) was used to evaluate the clinical utility of the model. To further validate the robustness of the model, a bootstrap method with 1000 resamples was applied for the internal validation of the training cohort.

3. RESULTS

3.1. Patient demographics

This study included 81 patients (52 male, 29 female) diagnosed with FA, with a median age of 4 years (interquartile range [IQR] 3–7 years). Of these patients, 41 had homozygous *FANC* gene mutations and 40 had compound heterozygous mutations. Additionally, 6 patients had a family history of hematological diseases (aplastic anemia [AA], FA, myelodysplastic syndromes [MDS], and AML), and 7 had a family history of solid tumors (liver cancer, esophageal cancer, rectal cancer, and others). Forty-nine patients presented with varying degrees of physical abnormalities, including head, limb, kidney, gastrointestinal tract, urogenital system, and cardiovascular system. Clinical characteristics of the patients with FA are summarized in Table 1.

Table 1

Demographics and clinic characteristics of the FA patients.

Variables	Total (n = 81)	Survival (n = 47)	Death (n = 34)	Statistic	*p*
Age, y, n (%)				$\chi^2 = 0.01$	0.935
≤5	52 (64.20)	30 (63.83)	22 (64.71)		
>5	29 (35.80)	17 (36.17)	12 (35.29)		
Gender, n (%)				$\chi^2 = 0.15$	0.698
Male	52 (64.20)	31 (65.96)	21 (61.76)		
Female	29 (35.80)	16 (34.04)	13 (38.24)		
WBC, M (Q_1, Q_3)	4.37(3.24, 5.40)	4.50 (3.48, 5.46)	4.22 (2.69, 5.33)	$Z = -0.56$	0.576
HB, mean ± SD	85.42 ± 21.84	90.57 ± 19.22	78.29 ± 23.48	$t = 2.58$	0.012
PLT, M (Q_1, Q_3)	30.00 (23.00, 59.00)	42.00 (26.00, 64.50)	27.00 (19.25, 41.50)	$Z = -2.47$	0.013
Somatic malformations, n (%)				$\chi^2 = 8.77$	0.003
No	32 (39.51)	25 (53.19)	7 (20.59)		
Yes	49 (60.49)	22 (46.81)	27 (79.41)		
Genetic subtyping, n (%)				$\chi^2 = 3.59$	0.058
Homozygous	41 (50.62)	28 (59.57)	13 (38.24)		
Compound heterozygous	40 (49.38)	19 (40.43)	21 (61.76)		
Progression to malignancy, n (%)				$\chi^2 = 7.43$	0.006
No	66 (81.48)	43 (91.49)	23 (67.65)		
Yes	15 (18.52)	4 (8.51)	11 (32.35)		
Family history of blood disorders, n (%)				$\chi^2 = 0.77$	0.381
No	75 (92.59)	42 (89.36)	33 (97.06)		
Yes	6 (7.41)	5 (10.64)	1 (2.94)		
Family history of cancer, n (%)				$\chi^2 = 0.00$	1.000
No	74 (91.36)	43 (91.49)	31 (91.18)		
Yes	7 (8.64)	4 (8.51)	3 (8.82)		
HSCT, n (%)				$\chi^2 = 2.90$	0.089
No	56 (69.14)	29 (61.70)	27 (79.41)		
Yes	25 (30.86)	18 (38.30)	7 (20.59)		

HB = hemoglobin, HSCT = hematopoietic stem cell transplantation, M = median, PLT = platelet, Q_1 = 1st quartile, Q_3 = 3rd quartile; SD = standard deviation, WBC = white blood count.
t: *t* test, *Z*: Mann–Whitney test, χ^2: Chi-square test.

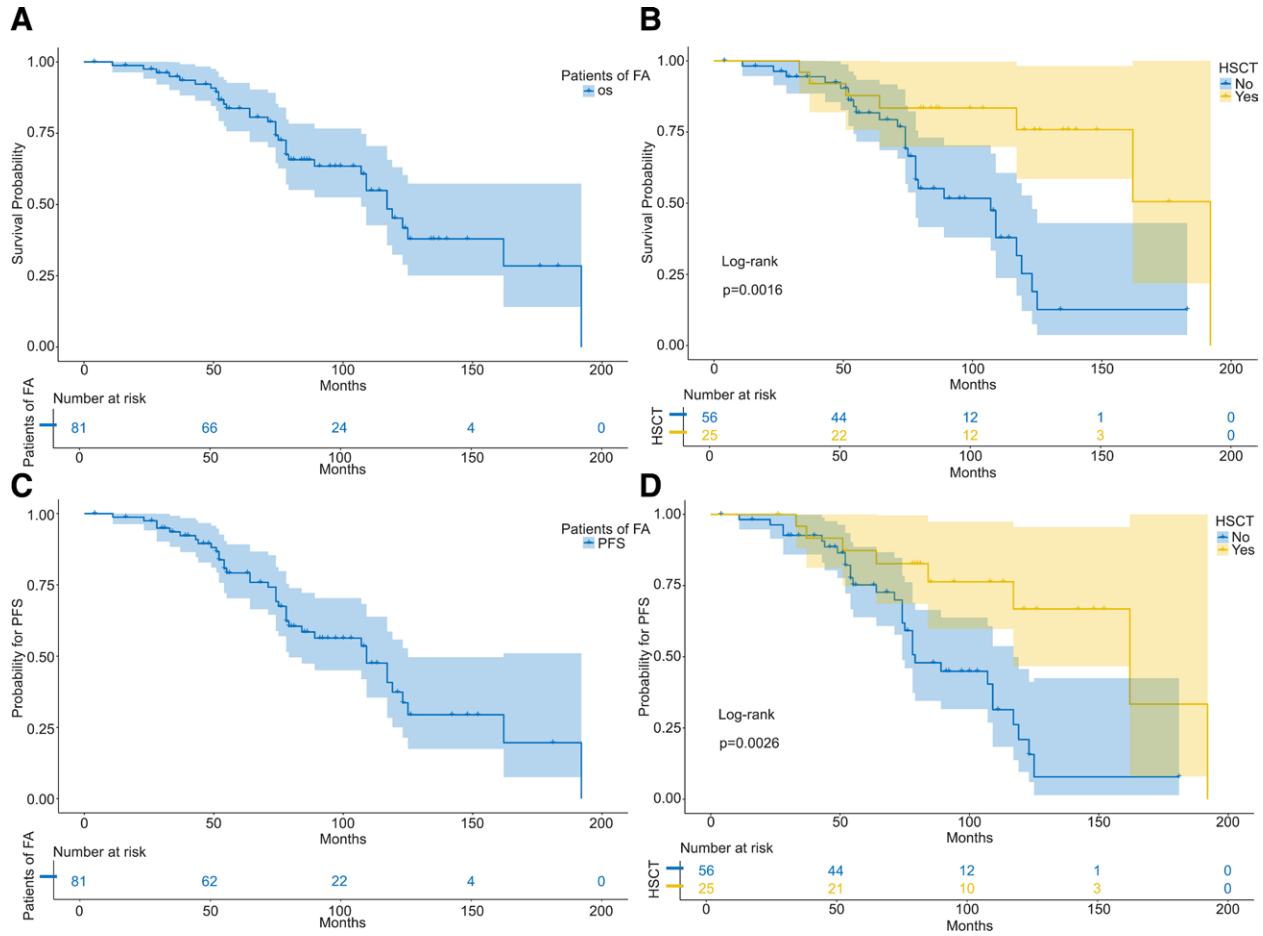

Figure 1. Kaplan–Meier survival curves. (A) Kaplan–Meier curve for OS in patients diagnosed with FA. (B) Kaplan–Meier curve for OS in patients with FA who did and did not undergo HSCT. (C) Kaplan–Meier curve for PFS in patients with FA. (D) Kaplan–Meier curve for PFS in patients with FA who did and did not undergo HSCT. FA = Fanconi anemia, HSCT = hematopoietic stem cell transplantation, OS = overall survival, PFS = progression-free survival.

3.2. Treatment process and outcomes

Of the 81 patients, 25 underwent HSCT, whereas the remaining 56 received drug therapy alone (cyclosporine, stanozolol, and levamisole, among others). The median follow-up was 78 months (range, 4–192 months). The median OS was 117 months (95% confidence interval [CI], 104.5–129.5 months) (**Fig. 1A**) and the median PFS was 109 months (95% CI, 83–134 months) (**Fig. 1C**). During the follow-up period, 15 patients developed malignant tumors, including 7 with MDS and 8 with AML. A total of 34 patients died; the primary causes of death included disease progression, infections, bleeding, and transplant-related complications, including graft failure and GVHD.

3.3. Independent prognostic factors for OS

In the analysis of OS in patients with FA, univariate analysis revealed that hemoglobin (HB) level (p = 0.002) and platelet (PLT) count (p = 0.017) were significant survival-related factors, whereas physical abnormalities (p = 0.042) and malignant tumor progression (p = 0.043) were negatively correlated with prognosis. Notably, patients with FA who underwent HSCT demonstrated better survival outcomes than those who received drug treatment alone (p = 0.003); this difference was also reflected in the Kaplan–Meier survival curves (**Fig. 1B and D**). These factors were included in a multivariate Cox regression analysis, which revealed that HB level, PLT count, malignant tumor progression, and whether the patient underwent HSCT were independent factors influencing OS among patients with FA. Detailed results of the univariate and multivariate analyses are reported in Table 2.

Furthermore, the 25 patients who underwent HSCT based on their initial blood test results were categorized into mild, moderate, and severe groups, and Kaplan–Meier survival analysis was performed. Although differences in survival time were observed among the groups, statistical analysis revealed no significant differences (p = 0.322).

3.4. Construction and evaluation of the OS nomogram

Based on the aforementioned analysis, a survival prediction model for patients with FA—known as a "nomogram"—was constructed. Each variable in the chart was represented by a corresponding line. The specific steps are as follows. First, locate the value corresponding to each variable on the scale, then draw a vertical line from that point to the "score" axis to obtain the corresponding score. Next, sum these scores to derive the total score, which is then positioned on the "total score" axis. Finally, a horizontal line is drawn from the total score axis to the time axis (1, 3, and 5 years) to determine the survival probability at each time point. The application of the nomogram for predicting the survival of patients with FA is illustrated in Figure 2.

The FA nomogram exhibited high accuracy and stability in predicting survival. The C index values in the training and validation sets were 0.732 (95% CI, 0.632–0.838) and 0.729 (95% CI, 0.634–0.830), respectively, indicating a strong discrimination ability and stable clinical predictive performance across different datasets. DCA (**Fig. 3A–C**) revealed that the model curve

Table 2

Univariable and multivariable analysis of OS in FA patients.

	Univariable analysis			Multivariable analysis		
Variables	**Hazzard ratio**	**95% CI**	***p* value**	**Hazzard ratio**	**95% CI**	***p* value**
Age >5	1.058	0.507–2.507	0.881			
Female	1.197	0.589–2.589	0.62			
WBC	1.045	0.983–1.11	0.157			
HB	0.975	0.96–0.991	0.002	0.98	0.964–0.996	0.017
PLT	0.979	0.962–0.962	0.017	0.985	0.968–1.003	0.104
Somatic malformations	2.388	1.034–5.518	0.042			
Progression to malignancy	2.166	1.026–4.573	0.043	1.9	0.884–4.084	0.1
Compound heterozygous	1.297	0.641–2.641	0.469			
Family history of blood disorders	0.33	0.045–2.045	0.276			
Family history of cancer	1.406	0.425–4.425	0.577			
HSCT	0.252	0.101–0.101	0.003	0.242	0.094–0.619	0.003

CI = confidence interval, FA = Fanconi anemia, HB = hemoglobin, HSCT = hematopoietic stem cell transplantation, OS = overall survival, PLT = platelet, WBC = white blood count.

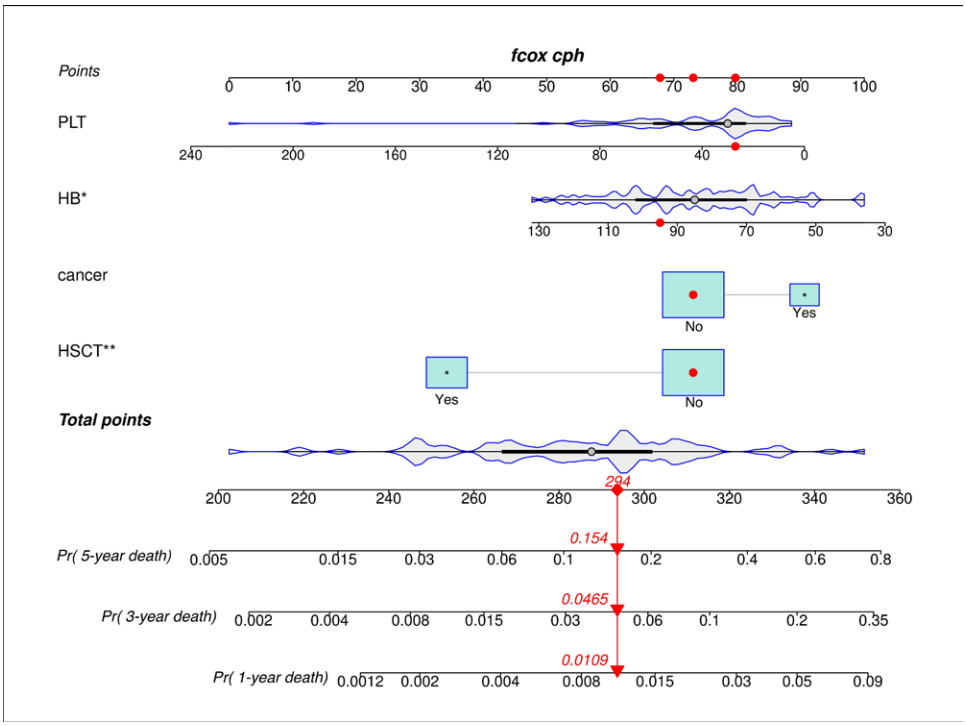

Figure 2. Nomogram for prognostic prediction of OS in patients diagnosed with FA. A nomogram was developed to predict OS of patients with FA. Continuous variables, such as HB and PLT count, are presented as density plots, whereas categorical variables are represented by box-size variations. For a patient with a PLT count of 27 × 10^9/L and an HB level of 95 g/L who did not undergo HSCT or progress to cancer, the total score was 294, corresponding to 1- (98.91%), 3- (95.35%), and 5-y (84.6%) OS probabilities. FA = Fanconi anemia, HB = hemoglobin, HSCT = hematopoietic stem cell transplantation, OS = overall survival, PLT = platelet.

offered a higher net benefit compared to the "treat all" and "treat none" curves, suggesting that the model provided valuable information for clinical decision-making, thereby significantly enhancing treatment outcomes. Furthermore, the 1-, 3-, and 5-year survival calibration plots (**Fig. 3D–F**) demonstrated that the predicted survival probabilities were highly consistent with the actual observations, further validating the high precision and reliability of the nomogram. AUC values for the 1-, 3-, and 5-year survival rates were 0.582, 0.727, and 0.731, respectively (**Fig. 3G**).

The lower AUC at the 1-year survival point was primarily attributed to the majority of patients surviving at the 1-year mark, making it challenging to distinguish between survival and death at this early time point. However, the AUC for the 3- and 5-year survival rates were significantly higher, suggesting that the model performed better at these time points. In summary, the FA nomogram exhibited strong clinical potential for predicting OS, with excellent discrimination and calibration.

3.5. Independent prognostic factors and nomogram for PFS

Results of analysis of PFS in patients with FA closely mirrored those for OS. Univariate analysis revealed that HB level (p = 0.002), PLT count (p = 0.019), whether the patient received HSCT (p = 0.005), and malignant tumor progression (p = 0.0019) significantly influenced PFS. Multivariate Cox regression analysis confirmed that HB level, PLT count, malignant tumor progression, and HSCT were independent prognostic factors for PFS. Detailed results of the univariate and multivariate analyses are reported in Table 3.

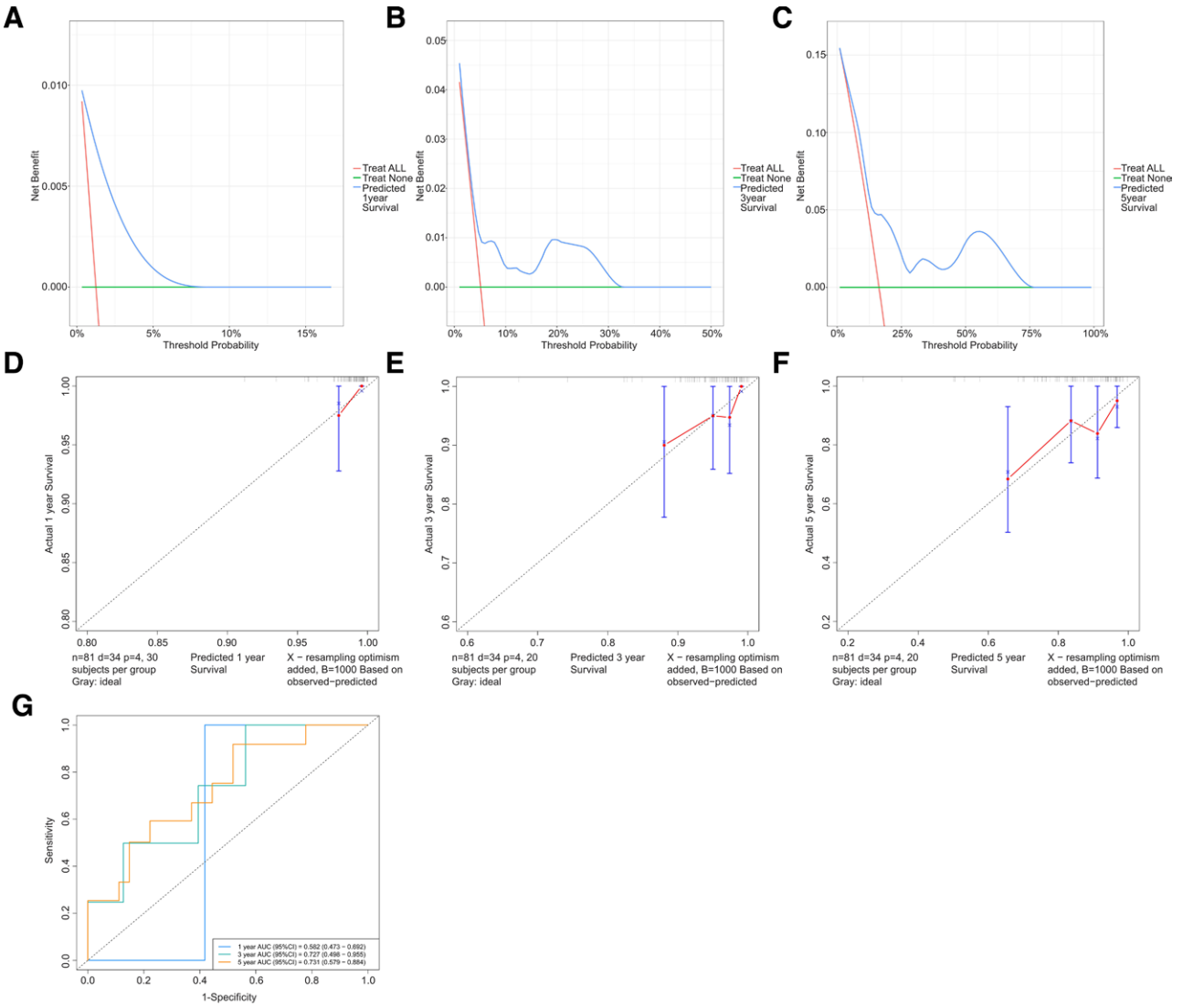

Figure 3. DCA of OS. DCA for 1- (A), 3- (B), and 5-y (C) OS. Calibration plots for 1- (D), 3- (E), and 5-y OS (F). (G) ROC curve for 1-, 3-, and 5-y OS based on the nomogram. AUC = area under the ROC curve, CI = confidence interval, DCA = decision curve analysis, OS = overall survival, ROC = receiver operating characteristic.

Table 3

Univariable and multivariable analysis of PFS in FA patients.

	Univariable analysis			Multivariable analysis		
Variables	**Hazzard ratio**	**95% CI**	***p* value**	**Hazzard ratio**	**95% CI**	***p* value**
Age >5	1.02	0.49–2.13	0.952			
Female	1.28	0.63–2.61	0.492			
WBC	1.04	0.98–1.11	0.197			
HB	0.98	0.96–0.99	0.002	0.982	0.966–0.998	0.024
PLT	0.98	0.96–0.99	0.019	0.987	0.970–1.004	0.139
Somatic malformations	2.09	0.90–4.83	0.085			
Progression to malignancy	2.46	1.16–5.21	0.019	2.010	0.926–4.361	0.077
Compound heterozygous	1.26	0.62–2.54	0.524			
Family history of blood disorders	0.29	0.04–2.11	0.220			
Family history of cancer	1.64	0.49–5.50	0.421			
HSCT	0.27	0.11–0.67	0.005	0.266	0.105–0.672	0.005

CI = confidence interval, FA = Fanconi anemia, HB = hemoglobin, HSCT = hematopoietic stem cell transplantation, PFS = progression-free survival, PLT = platelet, WBC = white blood count.

Based on these factors, a PFS nomogram was developed by incorporating the scores of the independent factors into a clear and intuitive risk prediction tool (**Fig. 4**). In the training set, the C index for the PFS nomogram was 0.741 (95% CI, 0.641–0.841) and, in the internal validation set, was 0.739 (95% CI, 0.638–0.846), indicating superior discrimination ability across

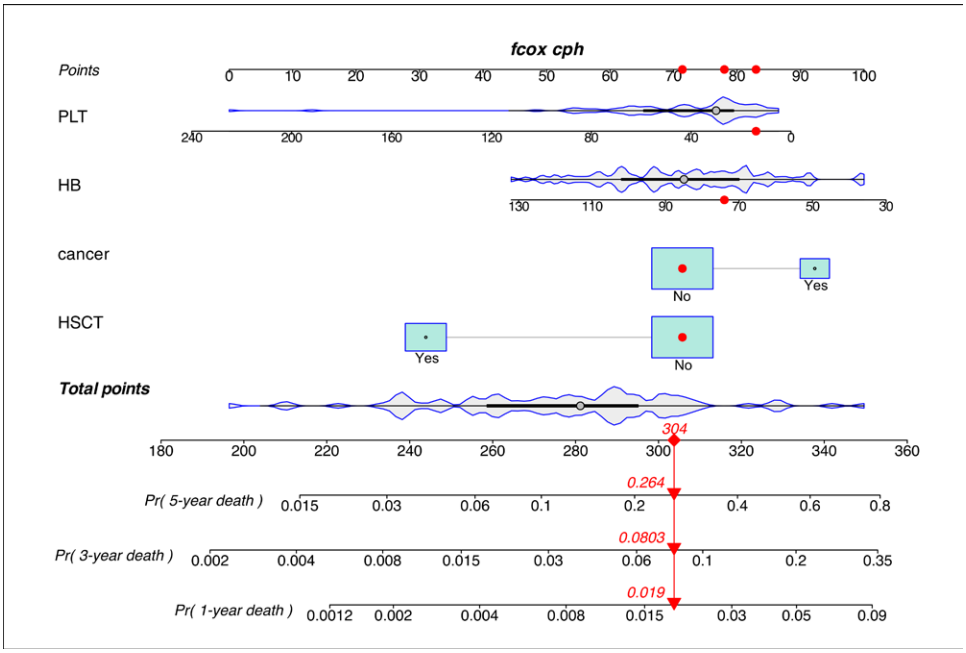

Figure 4. Nomogram for prognostic prediction of PFS in patients diagnosed with FA. Continuous and categorical variables are presented in Figure 2. For a patient with a platelet count of 14 × 10^9/L and a hemoglobin level of 74 g/L, who did not undergo hematopoietic stem cell transplantation or progress to cancer, the total score was 304, corresponding to 1- (98.1%), 3- (91.97%), and 5-y (73.60%) PFS rates. FA = Fanconi anemia, HB = hemoglobin, HSCT = hematopoietic stem cell transplantation, PFS = progression-free survival, PLT = platelet.

different datasets. DCA results (**Fig. 5A–C**) further validated the clinical value of the model. Calibration plots for 1-, 3-, and 5-year PFS (**Fig. 5D–F**) demonstrated that the model's predicted probabilities were highly consistent with the actual observations. AUC analysis revealed that the AUC for 1-, 3-, and 5-year PFS were 0.658, 0.746, and 0.753, respectively (**Fig. 5G**).

Compared with OS prediction, the AUC for PFS was slightly higher at all time points, particularly for 3- and 5-year PFS, suggesting that the model performed well for medium- to long-term PFS prediction. Overall, the PFS nomogram exhibited superior performance in survival prediction compared with the OS nomogram, with higher discrimination and calibration.

4. DISCUSSION

4.1. Main findings

Patients with FA often have a poor prognosis due to progressive bone marrow failure and increased susceptibility to tumors. Consequently, accurate prediction of survival in this patient population is essential for personalized treatment strategies. However, no predictive model has been developed for patients diagnosed with FA. This study aimed to develop a nomogram model that accurately predicted the long-term survival of patients with FA, offers personalized survival estimates, and supports clinical treatment decisions.

Previous studies have demonstrated that patients with FA are predisposed to developing malignant tumors, such as MDS, AML, and SCC.[23,24] In this study, 15 of the 81 patients progressed to malignant tumors, including 7 cases of MDS and 8 cases of AML. Notably, no cases of SCC were observed in this study. Of the 15 patients who developed malignant tumors, 11 died during follow-up. These findings suggest that progression to malignant tumors may be a significant predictor of mortality in patients with FA. Pancytopenia is a hallmark clinical feature of FA[25] and, in patients who progressed to MDS/AML, excessive proliferation of abnormal myeloid cells further disrupts the generation and maturation of white blood cells, red blood cells, and PLT.[26] Thus, we considered initial white blood cell count, HB level, and PLT count as key variables. Patients with FA frequently exhibit congenital malformations, such as VACTERL-H syndrome or PHENOS syndrome[27]; thus, we also considered the presence of physical malformations as a crucial factor influencing survival. FA is a genetic disorder resulting from mutations in 1 of the 23 FANC genes.[28,29] In this study, approximately one-half of the 81 patients had homozygous mutations in *FANC*, whereas the remainder had compound heterozygous mutations. Whether patients with homozygous mutations in *FANC* have better prognosis and longer survival requires further survival analysis. Although the number of non-transplant treatments has increased, HSCT remains the only definitive cure for FA.[30,31] Thus, whether a patient undergoes HSCT remains a critical factor that influences prognosis. Univariate and multivariate analyses of OS and PFS in patients with FA identified the following independent prognostic factors: HB level, PLT count, progression to malignant tumors, and whether the patient received HSCT. The analysis revealed that patients with FA and lower initial PLT and HB levels had poorer prognoses, which is consistent with previous studies, indicating that pancytopenia contributes to worse clinical outcomes. The presence of physical malformations (p = 0.042) was negatively correlated with FA prognosis, suggesting that physical examinations should be prioritized in clinical practice. Patients with FA who underwent HSCT had better prognoses than those who received drug treatment alone (p = 0.003), further underscoring the importance of HSCT in the treatment of FA. Malignant tumor progression increased the risk for death in patients with FA (p = 0.043), suggesting that once patients with FA develop malignant tumors, their condition should be closely monitored, and HSCT or other interventions should be considered to improve survival. The nomogram models for OS and PFS developed using the aforementioned variables demonstrated excellent discrimination and calibration abilities. In clinical practice, the survival probability of patients with FA at 1, 3, and 5 years after diagnosis can be predicted based on initial HB levels, PLT count, presence of physical malformations, progression to malignant tumors, and whether they have undergone HSCT. For patients who have not yet decided whether to undergo HSCT, comparing the prognosis

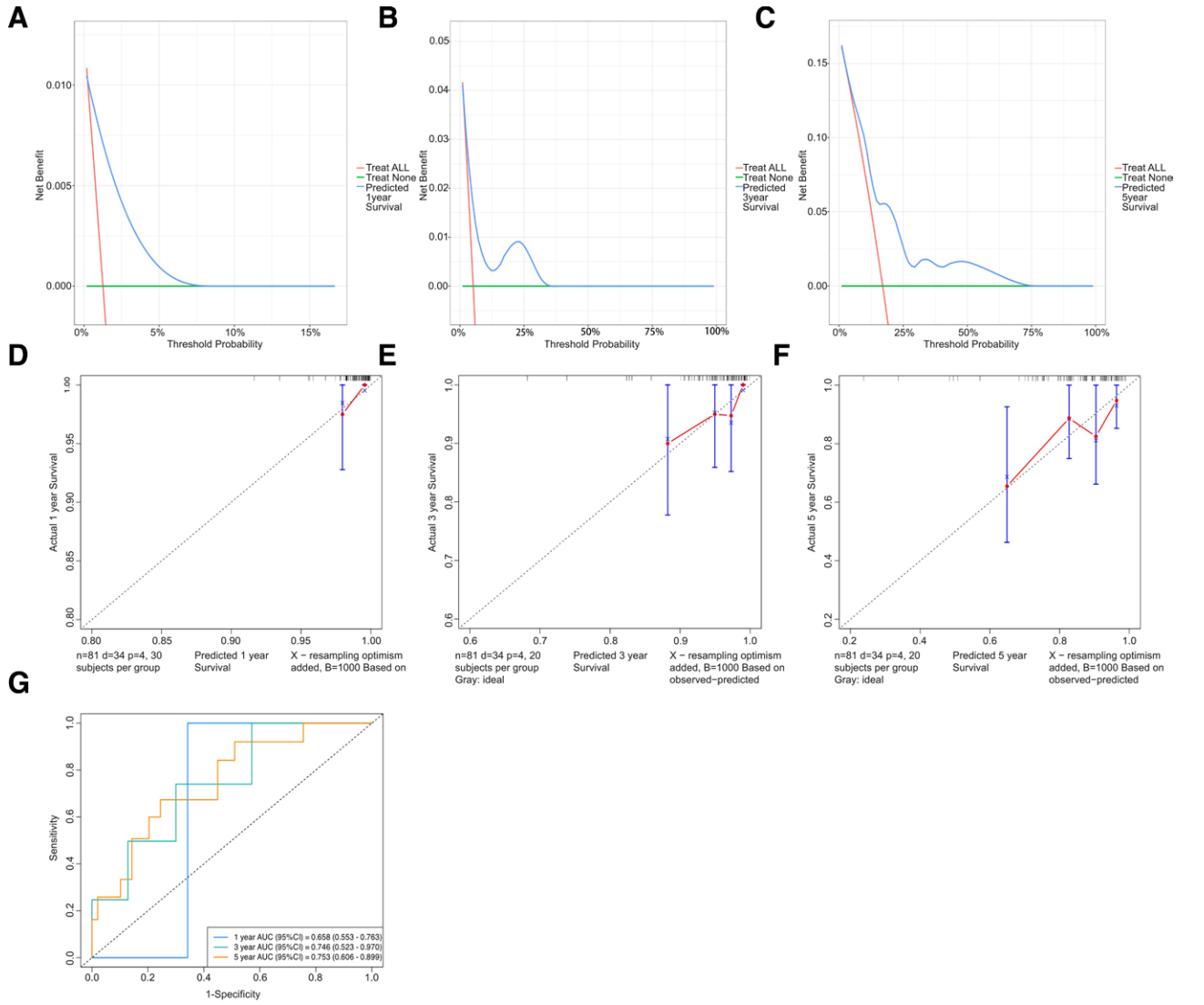

Figure 5. DCA of PFS. DCA for 1- (A), 3- (B), and 5-y (C) PFS. Calibration plots for 1- (D), 3- (E), and 5-y (F) PFS. (G) ROC curve for 1-, 3-, and 5-y PFS based on the nomogram. AUC = area under the ROC curve, CI = confidence interval, DCA = decision curve analysis, PFS = progression-free survival, ROC = receiver operating characteristic.

with and without HSCT can assist clinicians and patients' families in making informed treatment decisions and clarifying the timing and necessity of HSCT. Additionally, when analyzing patients who underwent HSCT separately, we did not observe a statistically significant correlation between initial blood cell counts and survival. This lack of association may be primarily attributable to the overwhelming predominance of patients in the aplastic phase within our FA cohort, which could have substantially influenced the hematopoietic recovery patterns and survival dynamics post-transplantation.

4.2. Limitations of the study

Although the nomogram developed in this study demonstrated strong predictive performance, it had several limitations. First, analysis of family history was limited. Family history may include factors such as consanguinity, congenital malformations, intellectual disabilities, anemia, cancer, and a history of spontaneous abortion. Future studies should collect and analyze these clinical variables. Second, the sex distribution was uneven, which could have influenced the accuracy of the predictive model. Furthermore, because previous studies did not identify other significant variables, this study included only prognostic factors in both univariate and multivariate analyses with a p value <0.05. Moreover, this was a retrospective, single-center study with a relatively small sample size, which limited the generalizability of the nomogram's clinical application to other centers. Additionally, the current OS estimates are constrained by the truncated follow-up duration, evidenced by only 34 mortality events recorded at the data cutoff. To strengthen longitudinal assessment, we will implement extended follow-up period, and subsequent data updates will be disseminated through subsequent publications. In addition, in our preliminary analysis, individual gene mutation subtypes (eg, FANCA, FANCC, FANCG) did not demonstrate statistical significance as independent prognostic variables. Subsequently, we implemented a dichotomized classification system (simple heterozygous mutations vs compound heterozygous mutations), which also failed to reach statistical significance. This finding contrasts with evidence from our prior study[32] demonstrating genotype-prognosis associations in FA, a discrepancy likely attributable to the insufficient sample size. To address this critical limitation, we plan to enlarge the cohort size through extended follow-up monitoring and multicenter collaboration, thereby enabling statistically powered survival analyses. Finally, we found that HB (p = 0.002) and PLT (p = 0.017) levels showed significant negative

linear associations with the risk of adverse prognosis, indicating that lower values were associated with higher risks of poor outcomes. We fully concur that this conclusion poses challenges for direct clinical implementation. This limitation primarily stems from our current insufficient sample size, which precluded stratification according to the FA clinical severity criteria. Upon expanding our sample size in future studies, we plan to conduct survival analyses by classifying both variables as categorical variables based on the FA clinical severity grading criteria.

4.3. Future research directions

The nomogram proposed in this study offers an effective tool for predicting survival in patients with FA and demonstrated significant clinical utility. As the current understanding of FA advances, future research may identify additional key factors influencing FA survival. Furthermore, the FA predictive model requires large-scale multicenter prospective studies to further enhance its accuracy and applicability.

5. CONCLUSION

We developed and validated nomograms for OS and PFS to predict survival outcomes in patients diagnosed with FA. We anticipate that this predictive model will aid clinicians in more accurately assessing the prognosis of this patient population and enhancing their long-term survival.

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