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Introduction

Hepatocellular Carcinoma (HCC) is one of the most common and aggressive malignant tumors in the world, and it is also the third most common cause of death in the world [1,2]. e disease causes nearly 600,000 deaths worldwide every year, and its morbidity and mortality have been increasing in recent years. Although surgical treatment and medical management strategies for HCC have progressed, the overall prognosis of HCC patients is still not satisfactory because liver cancer patients undergoing treatment o en su er from terminal-stage cancer. e 5-year survival rate of HCC is approximately 17% [3,4]. Although the *Alpha-Fetoprotein*(*AFP*) tumor marker is routinely used in early screening for HCC, which has reduced the mortality of liver cancer, its sensitivity and speci city are still limited [5,6]. erefore, discovering a valuable cellular biomarker speci c for HCC and exploring new diagnosis and treatment strategies are crucial.

e alteration of DNA methylation is a key epigenetic event in cancer. Such alteration has been con rmed to play a vital role in cancer and other human diseases [7]. DNA methylation o en occurs in Tumor Suppressor Genes (TSGs), and Hypermethylation can cause inappropriate transc2 63(u)12(p)19(t)-5(io)1e mao(a)-4.-8(enc-6(in)8[8,9], l-8(e)-d12(in)8(g6)-8(o t)-5.93v)7.9(n)]TJ8387 Tw 0 -1.278 T

addition, the methylation levels of *TBX15* in all 61 tumor tissues were higher than those in the paired adjacent paracancerous tissues. e di erence was statistically signi cant (P<0.001, (Figure 1).

Relationships between the *TBX15* methylation level and clinicopathological characteristics of patients with HCC

is study explored the association between the *TBX15* methylation level and clinicopathological characteristics of patients with HCC. Several factors including age, sex, *AFP* level, HBV status, TNM stage and tumor size, presence of thrombi and/or vascular invasion and envelope integrity were included in this study. As indicated in (Table 3), male patient *TBX15* methylation levels were signi cantly higher than those of female patients (P<0.05). *TBX15* methylation levels were increased in tumor tissues with vascular invasion (P:(c)6(h)3(a)9(83:(c)6(h)3(a)9(83:(c)6(h)3(a22.9 -1(l)3a)3(sio)12(n (P:(c)6(h)3(a)9(83:(c)6
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potential of *TBX15* methylation. When the threshold was 38% for the methylation level, the Youden index was the largest, with a sensitivity of 96%, speci city and AUC of 0.98 (CI=0.952-0.998, P<0.01), (Figure 2). e diagnostic concordance rate was 90%.

Association between *TBX15* methylation and the prognosis of HCC patients

Kaplan-Meier survival analysis according to the relapse-free survival rate was performed to evaluate the prognostic potential of *TBX15* methylation. Based on the methylation level threshold obtained by the ROC curve analysis, HCC patients were divided into a Hypermethylation group (n=49) and a hypomethylation group (n=12). During the follow-up, 21 people experienced relapse in the Hypermethylation group, with two dying from cancer. Four people had a relapse in the hypomethylation group, with two dying from cancer. During the follow-up period, 3 patients in the Hypermethylation group lost contact with the research team. e relapse-free survival time of the Hypermethylation group was signi cantly lower than that of the hypomethylation group (P<0.01), (Figure 3). Cox proportional hazards regression model analysis was used to further study the prognostic value of *TBX15* methylation in HCC patients. e results showed that the risk of relapse in the hypomethylation group was 5.6% lower than that in the Hypermethylation group, (P<0.01) (Figure 4 and Table 4).

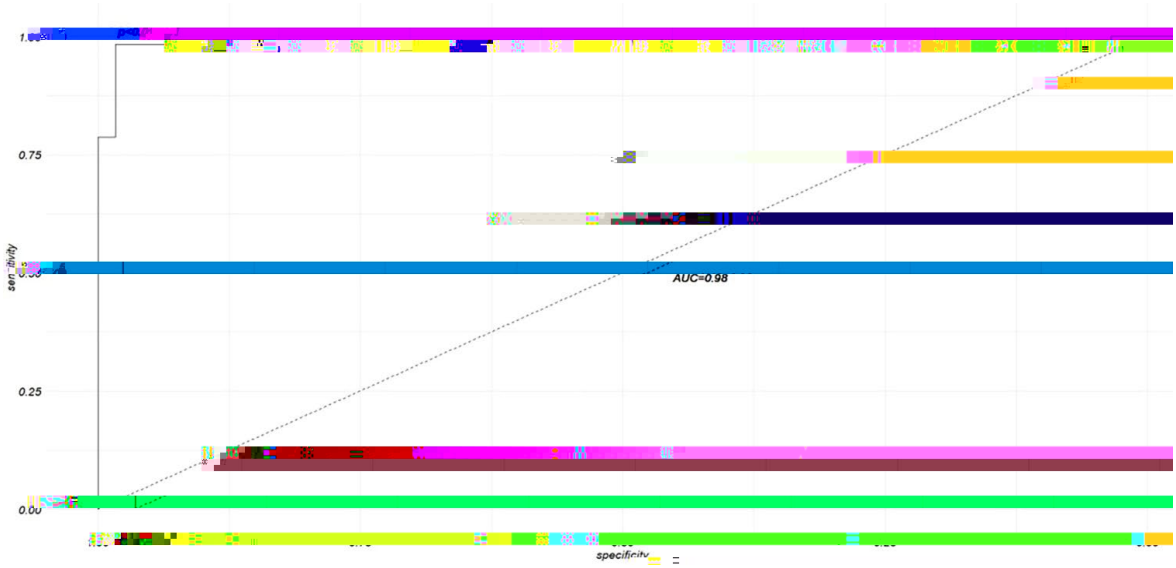

Figure 2: Receiver Operating Characteristic (ROC) curve analysis.

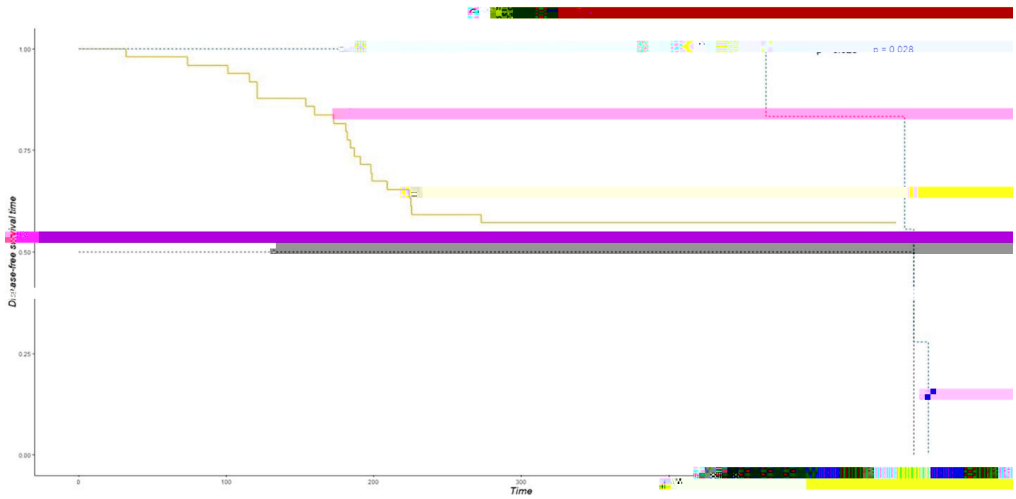

Figure 3: Disease-free survival analysis.

free survival time than those with hypermethylated *TBX15*. erefore, patients with hypermethylated *TBX15* can receive earlier intervention to achieve better curative e ects. Due to the limited follow-up time, the relationship between *TBX15* methylation and overall survival was not evaluated. In the future, more patients who complete follow-up can be studied for a longer duration than was used in this study to obtain more meaningful results regarding the correlation between *TBX15* and the overall survival rate.

Conclusion

e results of research on DNA methylation have been applied clinically. Some reports have shown that gene methylation can be detected in body uids such as urine, plasma, and serum and tissues. In addition, gene silencing caused by TSG methylation is reversible. Demethylating reagents such as 5-azacytidine can re-express previously silenced genes in cancer cells. Clinically, Demethylating reagents such