Supplementary Figure 1. Distribution of HOTAIRM1 expression level associated with clinical and molecular features. Distribution of HOTAIRM1 expression level in TCGA (A–I) and CGGA (J–O). ns, no significant difference, *p < 0.05, **p < 0.01, ***p < 0.001, ****p < 0.0001, values represented as the mean ± SD, Student’s t-test.
Supplementary Figure 2. Application of HOTAIRM1 in cohorts stratified by clinical and molecular features. (A–D) Prognostic value of HOTAIRM1 for the KPS in glioma or GBM in TCGA. HOTAIRM1 showed predictive value in LGG cohorts stratified by IDH status in TCGA (E, F) and CGGA (N, O) but not in GBM cohorts (G, H, P, Q). High HOTAIRM1 expression indicated a poor prognosis in LGG or GBM with ATRX-wt (J, L, R, U) but not in LGG or GBM with ATRX-mut (K, M, S, V).
Supplementary Figure 3. HOTAIRM1 as a predictor of survival in glioma patients. HOTAIRM1 expression levels predicted one-year, three-year, and five-year survival in TCGA (A–C) and CGGA (D–F).
Supplementary Figure 4. Functional analysis of HOTAIRM1 in glioma. HOTAIRM1 was closely correlated with transcriptome expression profiles in TCGA (A) and CGGA (B). Biologic functions of HOTAIRM1 were confirmed by GSEA in TCGA (C-E) and CGGA (F-N). High-exp, high expression of HOTAIRM1; Low-exp, low expression of HOTAIRM1.
Supplementary Figure 5. HOTAIRM1 promotes cell migration in vitro. (A) Knockdown efficiency of si-HOTAIRM1-1, -2, and -3 in U87 and LN229 cells assessed by qRT-PCR. (B) Wound-healing assays were performed to evaluate the effect of si-HOTAIRM1 on cell migration. *p < 0.05, **p < 0.01, ***p < 0.001, ****p < 0.0001, values represented as the mean ± SD, Student’s t-test.
Supplementary Figure 6. HOTAIRM1 was correlated with EMT. (A) EMT differed between the high-exp and low-exp groups in TCGA and CGGA. (B) GSEA was performed to validate the effect of HOTAIRM1 on EMT in TCGA and CGGA. (C) Photomicrograph of U87 ten days after first transfection (×20 magnification; second transfection was performed five days after first transfection to maintain silence efficiency). EMT, epithelial-mesenchymal transition. High-exp, high expression of HOTAIRM1; Low-exp, low expression of HOTAIRM1.
Supplementary Figure 7. The binding site of hsa-miR-495-3p and hsa-miR-129-5p on hub genes predicted with starBase v3.0 (http://starbase.sysu.edu.cn).
Supplementary Figure 8. Patients with high HOTAIRM1 expression showed a clear immune phenotype. GSEA was performed to confirm the effect of HOTAIRM1 on immune-related gene sets in TCGA (A–E) and CGGA (F–J).
Supplementary Figure 9. HOTAIRM1 was associated with the regulation of inflammatory activities. Effect of HOTAIRM1 on inflammatory activities in TCGA (A) and CGGA (B) was assessed by Pearson correlation analysis.

Supplementary Figure 10. HOTAIRM1 was associated with the regulation of the tumor microenvironment (TME). (A, B) High HOTAIRM1 expression was closely associated with low purity and immune cells including Gamma delta T cells (Tgd), CD8+ T cells, activated dendritic cells (aDC), and Regulatory T cells (Treg).
Supplementary Figure 11. HOTAIRM1 is positively associated with the HOXA gene family. (A, B) The relation between HOTAIRM1 and HOX gene family was investigated by Pearson’s correlation analysis.