Figure S1. Knockdown of HHLA2 inhibits TGF-β1-induced EMT of GBC *in vitro*. (A) TGF-β1 (10 ng/ml) increased the expression of HHLA2 in a time-dependent manner, (B) according to the quantification. (C) EMT was inhibited in the TGF-β1-treated GBC cells with HHLA2 expression knocked down compared with that in the TGF + sh-NC group, as shown by Western blot analysis (n=3), (D) which was also quantified. (E) Immunofluorescence staining showed similar results to western blotting. Scale bars, 100 μm. Green, target protein; Blue, DAPI. **P<0.01 and ***P<0.001 (TGF + shHHLA2 group vs. TGF + sh-NC group) HHLA2, human endogenous retrovirus-H long terminal repeat-associating protein 2; EMT, epithelial-mesenchymal transition; Col-I, collagen I; α-SMA, α-smooth muscle actin; shRNA or sh, short hairpin RNA; NC, negative control.

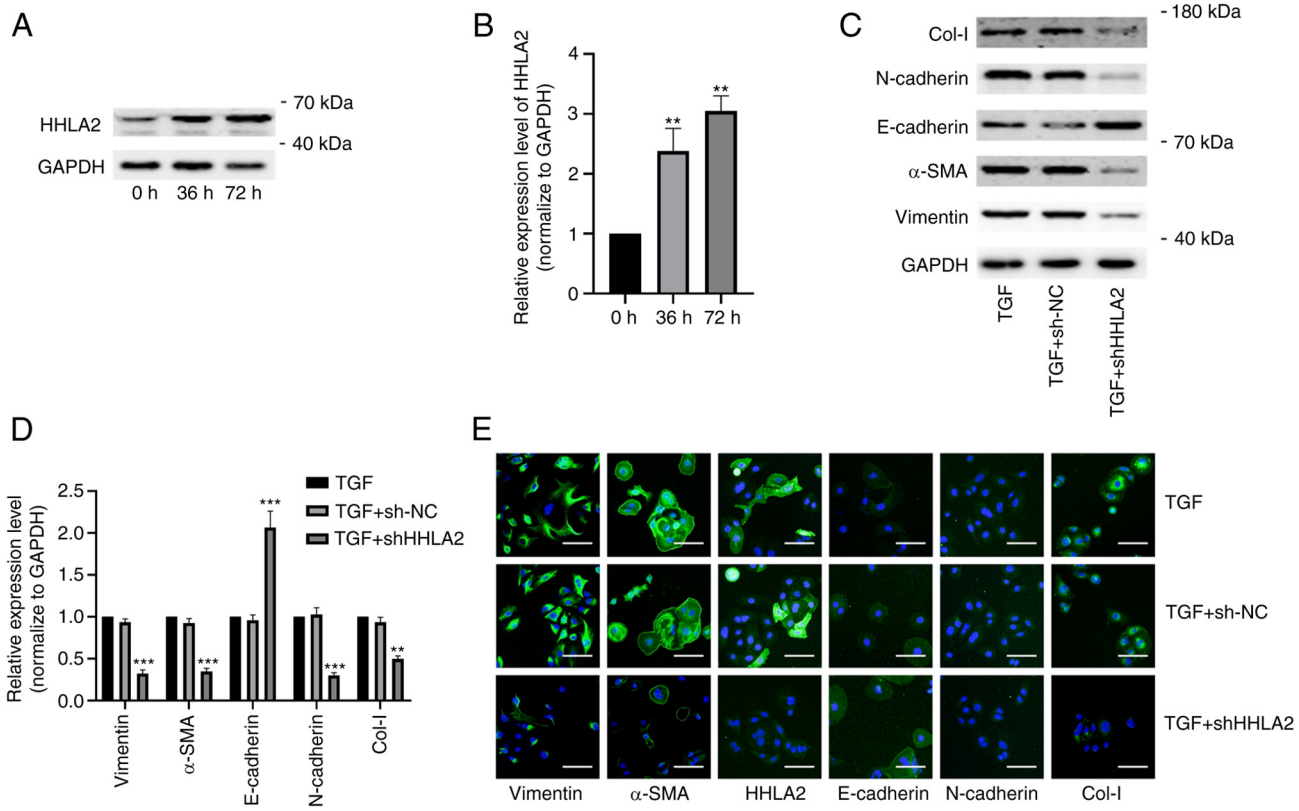

Figure S2. H19 overexpression promotes the EMT process, migration and invasion of GBC cells *in vitro*. (A) Increased EMT was found in the GBC cells overexpressing H19 compared with that in the NC group according to western blot analysis (n=3), (B) which was quantified. (C) Wound healing assay (D) and subsequent quantification showed that H19 overexpression improved cell migration. Scale bars, 100 μm. (E) H19 overexpression was found to increase cell invasion, (F) according to the quantification. Scale bars, 100 μm. **P<0.01 and ***P<0.001 (H19 group vs. H19-NC group). Col-I, collagen I; α-SMA, α-smooth muscle actin; NC, negative control.