**Supplementary Figure Legends**

**Supplementary Figure S1**

**(A) Kaplan–Meier plots of progression-free survival in the overall population and (B) overall survival in the overall population.**

In the overall population, the median PFS duration was 9.6 months (95% CI, 4.2 to not reached), and the median OS duration was 23.2 months (95% CI, 8.4 to not reached), with 23 patients (51.1%) dying.

**Supplementary Figure S2**

**(A) Kaplan–Meier plots of progression-free survival according to *PTEN* mutations**

**(B) Kaplan–Meier plots of overall survival according to *PTEN* mutations**

Patients with mutant PTEN tended to experience shorter PFS and OS durations than those with wild-type PTEN; (median PFS: 4.3 months [95% CI, 1.2 to not reached] vs 15.6 months [95% CI, 4.3 to not reached]; HR: 1.72 [95% CI, 0.76 to 3.92], P = 0.195) and (median OS: 15.2 months [95% CI, 3.8 to not reached] vs 25.7 months [95% CI, 10.3 to not reached]; HR: 1.46 [95% CI, 0.62 to 3.46], P = 0.391).

\*P values were calculated using the Cox proportional hazards model.

**Supplementary Figure S3**

**PTEN-positive tumor and the expression of *PTEN* mRNA according to *PTEN* mutations.**

1. IHC analysis showed that the *PTEN*-positive tumor area was significantly smaller intumors with mutant *PTEN*/mutations in the phosphatase domain than in tumors with wild-type *PTEN* but not in those with mutant *PTEN*/mutations in the *C2* domain.
2. In the transcriptomic analysis, compared with tumors with wild-type *PTEN*, the expression of *PTEN* mRNA was significantly lower in tumors with mutant *PTEN*/mutations in the phosphatase domain but not in those with mutations in the C2 domain.

**Supplementary Figure S4**

**Gene set enrichment analysis of whole transcriptome expression**

**(A) and (B): GSEA enrichment plot for “HALLMARK\_PI3K\_AKT\_MTOR\_SIGNALING” and “HALLMARK\_MTORC1\_SIGNALING” between mutant *PTEN*/mutations in the phosphatase domain and wild-type *PTEN***

**(C) and (D): GSEA enrichment plot for “HALLMARK\_PI3K\_AKT\_MTOR\_SIGNALING” and “HALLMARK\_MTORC1\_SIGNALING” between mutant *PTEN*/mutations in the C2 domain and wild-type *PTEN***

GSEA demonstrated enrichment of signature genes involved in the PI3K-AKT-mTOR and MTORC1 signaling pathways in tumors with mutant *PTEN*/mutations in the phosphatase domain compared to those with wild-type *PTEN*, while the difference was not statistically significant between tumors with mutant *PTEN*/mutations in the C2 domain and those with wild-type *PTEN*.