



Figure S1. Kaplan-Meier curves for PFS; (a) (b) with *BRAF*^{V600E} mutation or wild type and negative or positive PD-L1 expression and (c) (d) with negative or positive CD8 + expression and negative or positive PD-L1 expression. P values were calculated by the log- rank test. *BRAF* wild type and PD-L1 positive group (n=1) was excluded from analysis due to small sample. *BRAF*^{V600E}, PD-L1 negative vs. PD-L1 positive; $\chi^2=0.673$, $p=0.412$. PD-L1 negative, *BRAF*^{V600E} mutation vs. *BRAF* wild type; $\chi^2=5.615$, $p=0.018$ (a). CD8 + negative/ PD-L1 positive group (n=2) was excluded from analysis. CD8+ positive, PD-L1 negative vs. PD-L1 positive; $\chi^2=0.317$, $p=0.573$. PD-L1 negative, CD8+ positive vs. CD8+ negative; $\chi^2=1.140$, $p=0.286$ (c).

Table S1. Results summary

Results	Data details
Table 1. Correlations between PD-L1 expression and clinicopathological factors in thyroid cancer.	
1. The present findings indicate the rate of PD-L1 positive expression and clinicopathologic factors which were associated with PD-L1 expression.	With a PD-L1 staining cut-off value of 1%, 13 (39.4%) of 33 patients were classified as positive. PD-L1 positive was positively associated with low stimulation Index (SI) levels ($p = 0.046$).
2. <i>BRAF</i> ^{V600E} mutation was significantly associated with increasing expression of PD-L1.	12 (50%) of 24 patients with <i>BRAF</i> ^{V600E} mutation were PD-L1 positive, compared to one (11.1%) of nine patients with <i>BRAF</i> wild type ($p = 0.047$).
3. CD8+ expression was significantly associated with increased PD-L1 expression.	Of the 17 patients with CD8+ positive, 11 (64.7%) were PD-L1 positive, compared to six (35.3%) CD8+ negative patients ($p = 0.003$).

Table 2. Univariate analysis of clinicopathological factors associated with PFS.

The results of the univariate analysis revealed that 9 variables which were considered significantly associated with poor PFS. PD-L1 expression was not associated with PFS.	9 variables which were considered significantly associated with poor PFS were, <ul style="list-style-type: none"> • <i>BRAF</i> wild type (HR = 0.27 [CI 0.10–0.76], $p = 0.013$), • high CRP (HR = 5.08 [CI 1.26–64.76], $p = 0.002$), • high WBC (HR = 30.93 [CI 2.81–340.82], $p = 0.005$), • high VEGF (HR = 9.02 [CI 1.559–6.383], $p = 0.029$), • primary tumor size ≥ 20mm (HR = 7.79 [CI 1.74–34.78], $p = 0.007$), • pT4 (HR = 8.12 [CI 2.07–31.86], $p=0.003$), • extrathyroidal extension (Ex) 2 (HR = 9.3 [CI 2.28–37.9], $p = 0.002$),
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	<ul style="list-style-type: none"> • metastasis (M)1 (HR = 5.73 [CI 1.98–16.58], p = 0.001), and • anaplastic thyroid cancer (HR = 4.97 [CI 1.53–16.18], p = 0.008).
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Table3. Multivariate analysis of clinicopathological factors associated with PFS.

In the multivariate analysis, patients with the <i>BRAF</i> wild type tended to have poor prognosis than with <i>BRAF</i> ^{V600E} mutation.	<i>BRAF</i> wild type (p = 0.022) and high CRP (p = 0.039) were independent and significant predictive factors for poor PFS.
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Figure 3 and Supplementary Figure S1. Kaplan-Meier estimation of PFS with negative or positive PD-L1 expression, with *BRAF*^{V600E} mutation or wild type, and with negative or positive CD8+ expression.

Patients with <i>BRAF</i> ^{V600E} mutation had a significantly longer survival than with <i>BRAF</i> wild type. Moreover, CD8+ negative patients might tend to have a poor prognosis.	<i>BRAF</i> wild type patients were significantly associated with poor PFS (p = 0.007).
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*SI, Stimulation Index is one of the markers of inflammation; VEGF, vascular endothelial growth factor; BRAF, v-raf murine sarcoma viral oncogene homolog B1; PFS, progression-free survival.