A Distribution of endocrine therapy partners

Endocrine therapy	Palbociclib-based setting N (%)	Abemaciclib / tucidinostat-setting N (%)
FUL	76 (51.0)	49 (32.9)
AI	67 (45.0)	69 (46.3)
TAM/TOR	6 (4.0)	13 (8.7)
Progesterone	0	18 (12.1)

B Transition of endocrine therapy partners

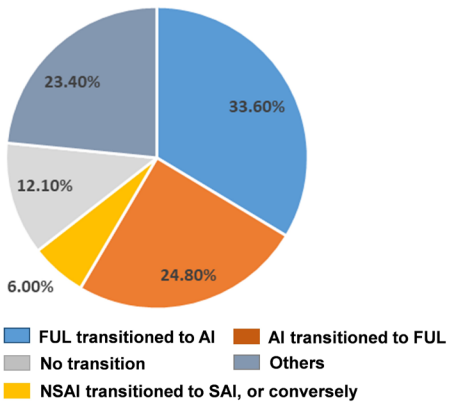

Figure S1 Endocrine partners. FUL, fulvestrant; AI, aromatase inhibitor; TAM, tamoxifen; TOR, toremifene; SAI, steroidal aromatase inhibitor; NSAI, non-steroidal aromatase inhibitor.

https://dx.doi.org/10.21037/tbcr-23-9

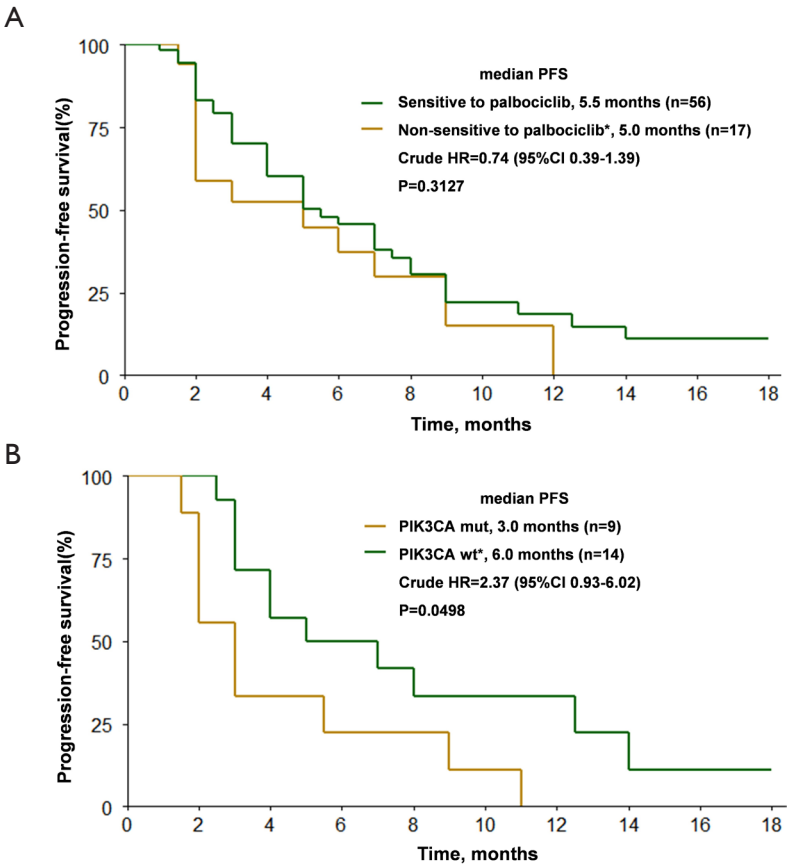

Figure S2 Progression-free survival of abemaciclib-based therapy by sensitivity to palbociclib and *PIK3CA* gene type. (A) Progression-free survival of abemaciclib-based therapy in patients sensitive and non-sensitive to palbociclib. (B) Progression-free survival of abemaciclib-based therapy in patients with wild type and mutant type *PIK3CA* gene. *, the reference group.

Table S1 Dose and reduction, discontinuation information

	ET + abemaciclib (n=73)		ET + tucidinostat (n=76)	
Initial dose[†]	150 mg	32 (43.8%)	30 mg	57 (75.0%)
	100 mg	12 (16.4%)	25 mg	3 (3.9%)
	50 mg	1 (1.4%)	20 mg	14 (18.4%)
Reduced dosage due to adverse reactions	6 (8.2%)		8 (10.5%)	
Discontinuation of targeted drugs due to adverse reactions	0 (0%)		1 (1.3%)	

[†], 28 cases in abemaciclib group lost information, 2 cases in tucidinostat group lost information.

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Table S2 Multigene sequencing results and methods

No. of patients	Genomic alterations	Method	Platform
1	None	NGS	High-throughput sequencing platform (HiSeq)
2	BRCA2	NGS	HiSeq
3	AKT1 p.E17K; HER2 p.R896G; FSL	NGS	HiSeq
4	ESR1	NGS	HiSeq
5	PTEN	NGS	HiSeq
6	None	NGS	HiSeq
7	AKT1	NGS	HiSeq
8	CDH1; IGF1R; PIK3CA (p.E5769Q,p.E726k); TP53 (c.67311_687delTTATCTCCT)	NGS	HiSeq
9	FGFR1 all exon; NF1 p.S1329* EX30; PIK3CA p.N345K EX5; TP53 p.T253Nfs*11	NGS	HiSeq
10	None	NGS	HiSeq
11	None	NGS	HiSeq
12	PALB2 p.R131*fs*1; NF1 (p.E602*, p.E1667*); PIK3CA (p.E545K, p.E726K); TP53 p.E285K; APC p.D1186Y	NGS	HiSeq
13	PIK3CA (p.E542K); PTEN	NGS	HiSeq
14	PIK3CA (p.E545K); TP53 (c.T712C; p.C238R)	NGS	HiSeq
15	CCND1; ESR1 D538G	NGS	HiSeq
16	AKT1, c.49G>A (p.E17K); ESR1, c.1610A>C (p.Y537S); MAP2K4, (p.K198_V206delinsL*)	NGS	HiSeq
17	ESR1, c.1607T>C (p.L536P); FGFR1; RB1, c.1498+2T>C	NGS	HiSeq
18	CCND1; FGFR1; MYC; TP53, p.R158Afs*12	NGS	HiSeq
19	AKT1, p.E17K; HER2, p.G776delinsV C; ESR1, (p.D538G; p.L536H); PIK3CA, (p.N1044K; p.E453Q; p.N345K)	NGS	HiSeq
20	ARID1A, c.4385_4401del, p.D1462Afs*23; c.6251T>G, p.V2084G; NTRK1	NGS	HiSeq
21	ATR; PIK3CA, H1047R; TP53, Y163C	NGS	HiSeq

Table S2 (*continued*)

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Table S2 (*continued*)

No. of patients	Genomic alterations	Method	Platform
22	CCND1; FGFR1; PIK3CA, p.H 1047R	NGS	HiSeq
23	BRCA1; BRCA2, p.R18Lfs*12; HER2; MAP2K4, p.P232L; NTRK1; NTRK2; NTRK3; PIK3CA	NGS	HiSeq
24	CDH1, p.S851*, exon16; PIK3CA, p.H1047R, exon21; TP53, p.L257Q, exon7	NGS	HiSeq
25	None	NGS	HiSeq
26	PIK3CA E545K; APC; TP53 splice site 782+1G>C	NGS	HiSeq
27	ARID1A, D1850Gfs*4; ARID2 R1769*; GATA3, P409Afs*99; MDM2	NGS	HiSeq
28	BRCA2; ESR1; PIK3CA; TP53	NGS	HiSeq
29	None	NGS	HiSeq
30	PIK3CA	NGS	HiSeq
31	PIK3CA	NGS	HiSeq
32	ESR1, p.D538G; PIK3CA, p.H1047L	NGS	HiSeq
33	ESR1; FGFR1; PTEN	NGS	HiSeq
34	PIK3CA p.N345K; APC p.N859S	NGS	HiSeq
35	ESR1; PIK3CA p.E545K	NGS	HiSeq
36	ESR1, p.D538G; PIK3CA, p.E545K	NGS	HiSeq
37	None	NGS	HiSeq
38	None	NGS	HiSeq
39	None	NGS	HiSeq
40	CCND1; CDKN2A; ESR1 (p.D538G, p.V187A)	NGS	HiSeq
41	AKT1; CDKN2B; FGFR1; TP53	NGS	HiSeq
42	BRCA1; PIK3CA	NGS	HiSeq
43	BRCA1; PTEN	NGS	HiSeq

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