BACKGROUND

• Breast cancer is a heterogeneous disease (6) with PI3K- and AKT-dependent signaling being key drivers of breast cancer (7–10).

• Understanding the deregulation of PI3K signaling has led to further development of a PI3K inhibitor class for breast cancer (11).

• BBG-295 and ALP-302 are two PI3K inhibitor drugs that specifically target the PI3K δ and γ isoforms.

• The recent availability of PI3K inhibitor therapy may improve clinical outcomes in patients with PIK3CA-mutant and PIK3CA-WT breast cancer.

METHODS

Study Design

• Two ongoing open-label clinical trials investigating BBG-295 and ALP-302 in patients with either triple-negative or hormone-receptor-positive breast cancer (NCT03054276, NCT03778937).

• Consenting patients with breast cancer who were determined to be candidates for a PI3K inhibitor in a real-world setting were included in the present analysis.

• The study design and treatment selection for patients with breast cancer was determined between the treating physicians and patients.

• All patients were analyzed on an intent-to-treat basis.

• Patients included in the analysis were determined by the treating physicians.

• Overall survival was calculated from the date of the first BBG-295/ALP-302 treatment dose.

• Data collected from patients treated with BBG-295/ALP-302 were compared with clinical and nonclinical data collected from PI3K inhibitor naïve patients with breast cancer treated with standard of care therapies in ABC (Figure 1).

RESULTS

• In this real-world study, patients treated with PI3K inhibitors or standard care therapies were similar among patients with and without PI3KCA mutation, and patient clinical characteristics corresponded across treatment groups.

• Mutations in PIK3CA were analyzed in 35% of patients with breast cancer.

• The median age at ABC diagnosis was 59.5 years (interquartile range: 1.0–8.0) for patients with PIK3CA-mutant ABC, the median OS from ABC diagnosis was 12.7% (median: 12.4 months, 95% CI: 10.4–15.1 months) for patients with PIK3CA-mutant ABC, and the median OS from ABC diagnosis was 7.1 months (95% CI: 6.1–10.1 months) for patients with PIK3CA-wild-type ABC.

• Clinical data on PIK3CA-mutant and PIK3CA-wild-type ABC are shown in Figure 4.

CONCLUSIONS

• This analysis of PIK3CA-mutant and PIK3CA-WT breast cancer demonstrates that patients treated with BBG-295 and ALP-302 were similar among patients with and without PI3KCA mutation, and patient clinical characteristics corresponded across treatment groups.

• In this real-world study, patients treated with PI3K inhibitors or standard care therapies were similar among patients with and without PI3KCA mutation, and patient clinical characteristics corresponded across treatment groups.

• Clinical data on PIK3CA-mutant and PIK3CA-wild-type ABC are shown in Figure 4.

LIMITATIONS

• Limited data availability

• This analysis is a retrospective analysis of patients treated with PI3K inhibitors in a single cancer center, and thus may not be generalizable to other patients with ABC.

• This analysis was performed with a PI3K inhibitor used as the standard of care therapy.

• This analysis was conducted in the context of a larger clinical trial

REFERENCES

