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Genomic and immune features of anti-PD-1 response in diverse solid tumours

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Immunotherapy using checkpoint inhibitors has demonstrated clinical efficacy for cancers ranging from melanoma to non-small-cell lung cancer and other types. Despite some success, many patients do not respond to the therapy, and a subset of patients develops "hyper-progressive" disease. Biomarkers that predict response or toxicity to checkpoint inhibitors will help to select patients who are most likely to benefit from these novel immune agents. Clinical-grade next generation sequencing is becoming more prevalent, providing information about genomic alternations, which could serve as potential new biomarkers to immunotherapy agents.

In this retrospective study, we included 43 patients treated in a community cancer center who had diverse solid organ malignancies, completed sequencing of tumour DNA before the initiation of immunotherapy, and received at least one cycle of a PD-1 or PD-L1 inhibitor. Half of the patients had breast and gynecologic cancers, which have been less investigated for checkpoint blockade.

We found that response to checkpoint inhibitors was associated with i) fewer previous treatment lines, ii) longer duration of immunotherapy, iii) higher frequencies of peripheral blood monocytes and lymphocytes after treatment, and iv) higher tumour mutational burden (TMB). In addition, base substitutions and indels in PRKDC and LRP1B and amplification of BCL6 occurred more frequently in responders. PRKDC and LRP1B mutations showed significant association with higher levels of TMB, which was confirmed by large-scale cancer genomics data.

Results from this study may be used to inform patients who will have a better response to PD-1/PD-L1-based immunotherapy, possibly indicating a first-line therapy in their treatment.

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