Pamukkale Medical Journal 2026;19(3):665-666
doi:10.31362/patd.1948929

The prevalence of asthma-COPD overlap (ACO) among COPD patients and the role of prostaglandin D2 (PGD2) in differential diagnosis "about"

KOAH hastaları arasında astım-KOAH overlap (AKO) sıklığı ve ayırıcı tanıda prostaglandin D2 (PGD2)' nin rolü "hakkında"

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Received:11.05.2026 Accepted:07.07.2026

Dear Editor,

We read with interest the study by Aslan and Enli [1] investigating the prevalence of asthma–COPD overlap (ACO) among patients with chronic obstructive pulmonary disease (COPD) and the diagnostic value of prostaglandin D2 (PGD2). While the authors should be commended for addressing an important clinical gray area, we would like to raise several methodological concerns regarding the diagnostic framework and the interpretation of the findings.

Upon reviewing the methodology, ACO was defined solely based on the presence of persistent airflow limitation and bronchodilator reversibility (post-bronchodilator FEV1/FVC <70% and FEV1 increase). However, this approach does not align with the current perspectives of the GINA and GOLD guidelines [2, 3]. Contemporary guidelines have moved away from defining ACO as a distinct disease entity; instead, they emphasize a phenotype-based approach focusing on asthma-related features within COPD and reflecting the heterogeneity between asthma and COPD [2-4]. The lack of a universally accepted definition of ACO and the increasing shift toward phenotype-based frameworks suggest that a reversibility-based classification alone may not adequately capture the clinical complexity of ACO [4].

We believe that this conceptual limitation may have influenced the interpretation of one of the study's key findings regarding PGD2. The authors reported no significant difference in PGD2 levels between ACO and COPD groups (p=0.858), with poor discriminatory performance (AUC≈0.51). Although this finding was interpreted as indicating limited diagnostic value of PGD2, it should be considered that the classification approach itself may have introduced substantial heterogeneity within patient groups. A reversibility-based classification may group patients with different inflammatory profiles under the same category. Moreover, restricting the study population to patients already diagnosed with COPD may have limited the representation of individuals with more prominent asthma features. This may have reduced the ability to detect potential biomarker differences. Therefore, in light of studies supporting the potential diagnostic role of PGD2, the observed negative finding may be more attributable to patient selection and classification limitations rather than the inadequacy of the biomarker itself [5, 6].

In conclusion, although the study by Aslan and Enli [1] provides valuable data, it should be recognized that evaluating a complex clinical spectrum such as ACO using limited diagnostic criteria may influence the interpretation of the findings.

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