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LETTER

Can Routine Hematological Markers Improve Obesity Risk Stratification? A Translational Comment on El-Aghbary et al [Letter]

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Dear editor

El-Aghbary et al show that adults with obesity have higher C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) than non-obese controls, reinforcing the notion of low-grade systemic inflammation and drawing attention to readily available tests that could aid clinical screening. The case–control design and emphasis on pragmatic markers are timely. We offer several comments aimed at strengthening the translational value of these observations.[1]

First, although CRP and ESR are sensitive to inflammatory tone, their specificity is limited. Intercurrent infection, autoimmune conditions and commonly used medications may elevate both, complicating interpretation in routine care. Rather than using them in isolation, a more informative strategy is model integration—combining CRP/ESR (and simple ratios such as the neutrophil-to-lymphocyte ratio) with metabolic indices (HOMA-IR, fasting insulin, lipid profile) and measures of central adiposity (waist circumference). Prospective work should test whether such panels improve prediction of incident diabetes, hypertension or cardiovascular disease beyond anthropometrics, using ROC analysis, net reclassification improvement and decision-curve analysis.[2]

Second, the obese cohort was predominantly female, which constrains generalizability. Sex-related differences in fat distribution, adipokine signaling and immune responses can modify biomarker–outcome relationships. Future studies would benefit from prespecified sex-stratified analyses and balanced recruitment across the life course (including adolescents and older adults) to ensure transportability.[3]

Third, the reported associations between low physical activity and constipation with obesity deserve closer attention. These variables point toward a gut–lifestyle–inflammation axis. Stool form and consistency closely track microbiota richness and composition; dietary fiber intake, transit time and microbially derived metabolites can shape systemic inflammation. Incorporating quantitative fiber measures, standardized stool-form indices (eg, Bristol scale) and basic microbiome readouts may reveal modifiable targets whereby lifestyle interventions reduce both adiposity and inflammatory burden.[4]

Finally, for clinical adoption, a pragmatic two-tier approach could be evaluated: (1) in primary care, a minimal panel (CRP, ESR, NLR) plus waist circumference to flag high-risk individuals; (2) in specialty settings, refinement with metabolic markers (HOMA-IR, triglycerides/HDL-C) and testing whether structured exercise and nutrition programs lower hematologic inflammation alongside weight and insulin resistance. Such stepwise evaluation would help define thresholds, use-cases and cost-effectiveness.

In sum, El-Aghbary et al provide a useful foundation. The next step is to determine whether routine hematological markers—embedded within integrated risk models—meaningfully improve prediction and management of obesity-related complications across sexes and care settings.

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