Commentary

In this issue, Rout *et al*. in their article "Anthropometric parameters as indicators of metabolic derangements in schizophrenia patients stabilized on olanzapine in an Indian rural population" report the results of their hospital-based case-control study assessing whether body mass index (BMI) or waist hip ratio (WHR) can

better predict dyslipidemia and insulin resistance in rural Indian patients with schizophrenia taking olanzapine as antipsychotic treatment.[1] This study is motivated by the now widely recognized clinical observations of increased risk of metabolic derangement for this study's target population, based on their underlying illness, medication exposure, and, for this group, their cultural makeup.

Patients with chronic mental illness, such as psychotic illnesses like schizophrenia, have been shown to face a significantly shortened life span and greater burden of medical illnesses, such as cardiovascular illness; this is thought to be related to a variety of patient, provider, and system-level factors that have been identified in several countries.[2] For this vulnerable population, antipsychotic medication is typically part of a long-term treatment plan prescribed with the good intentions of diminishing patient suffering and improving quality of life, with the unintended but predictable consequences of harmful and often cumulative side effects. Second generation antipsychotics (SGAs) as a class have increased risk of metabolic syndrome – including central obesity, hypertension, dyslipidemia, and insulin resistance – which bears clinical importance given contributions to increased risk of cardiovascular morbidity and mortality.[3] Increased risk of metabolic syndrome with SGAs has highlighted the need for a careful assessment of medication choice; olanzapine, the atypical antipsychotic studied here, has been studied with rates of metabolic syndrome of 40% at 3 years in some studies, likely reflective of medication effects in combination with inadequate screening and treatment for at-risk patients.[4] Given the finding then that Asian Indians have been found to have more total and abdominal body fat – important markers of dyslipidemia and insulin resistance - it becomes important to understand what can be done to better predict who is already at increased risk of diabetes and cardiovascular disease.[5]

The clinical issue of how to best screen patients for metabolic derangements has been addressed by several guidelines, which have unfortunately continued to fall short of full implementation by clinicians for a variety of reasons.[6] McDonell *et al*. recently studied barriers to screening for metabolic syndrome by healthcare provider survey, finding the most frequently ranked barriers were (lack of) patient resources, inadequate mental health training in primary care, and fragmented medical and mental health systems, highlighting again a combination of patient, provider, and system-level factors.[7] A simply administered, easily communicated screening measurement, such as BMI or WHR, would potentially be a useful first step in addressing screening barriers and could serve as a necessary, and potentially sufficient, measurement to assess for metabolic derangement. While this question has been asked in other populations, it has not been addressed in rural India.

This study uses a methodology that makes use of the well-validated homeostasis model assessment of insulin resistance (HOMA-IR) as both, a measure of insulin resistance and a proxy to metabolic syndrome to answer this exact question: Whether BMI or WHR could sufficiently predict dyslipidemia and insulin resistance in their sample population. HOMA-IR scores can be calculated with only a single measurement of insulin and glucose, minimizing patient study dropout and need for multiple study points. The study makes use of both male and female outpatients stabilized on at least 6 months of olanzapine, which would potentially blind results to expected gender differences, though it simplifies findings. The study excludes those with type 2 diabetes already, family history of diabetes in first degree relatives, substance use disordered patients, and non-olanzapine antipsychotics; while these exclusions limit the generalizability of the findings, this does allow for fewer confounding factors in the sample population. The results showed mean fasting serum insulin and insulin resistance indicator HOMA in the case group ($P < 0.001$) without a change in fasting blood glucose (FBG), validating that the case population as a whole was showing signs of increased insulin resistance prior to onset of diabetes or even a recognizable significantly different FBG. The significantly higher case group serum triglycerides ($P < 0.001$) and lower HDL ($P = 0.016$) are consistent with evidence of an expected increased rate of metabolic syndrome, further validating the study findings. The primary study result that HOMA-IR was dependent on WHR, but not BMI, lends support toward a recommendation of prioritizing WHR over BMI in this population.

While it was known that both total body obesity and central obesity can increase from antipsychotic therapy and contribute to cardiovascular disease, this study takes a step toward understanding the relative importance of WHR compared to BMI in patients with schizophrenia taking olanzapine in India. It will remain to be seen whether this finding will hold true for other populations and other antipsychotics.

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