

Should depressive patients undergo a regular diabetes screening test?

Sir,

The article “Association of depression and its relation with complications in newly diagnosed type 2 diabetes” by Bajaj *et al.*,^[1] dealing with the complicated psychological scenario of patients suffering from type 2 diabetes mellitus (T2DM) and its relevance with the systemic complications of the same, is indeed a laudable effort. It has recommended psychological counselling and therapeutic approach to cope with both depression and diabetes to provide better healthcare to the patients. However, the reverse is also true which is greatly overlooked during diagnosis and prevention of new cases of diabetes in the current scenario. This correspondence highlights this little focused arena of endocrinology practice.

Although diabetes and depression apparently appear as two exclusive areas in clinics, there is a common intersecting patient zone. Several studies have shown that diabetes increases the susceptibility of depression. But clinically significant depression, in spite of being a well-identified independent risk factor of developing T2DM,^[2] is not potentially intervened in practice to cut down the occurrence rate of new-onset T2DM because knowledge about the definite mechanism is lacking. Depression can be extrapolated to the development of diabetes by various hypotheses considering the disruption of hypothalamic-pituitary-adrenal (HPA) axis, increased production of interleukin (IL)-6, IL-1, tumor necrosis factor-alpha, C-reactive protein, reduced serum leptin, and increased sympathetic activity in the case of major

depressive disorder. The increased activity of HPA axis in depression leads to elevated serum cortisol level.^[3] Plasma cortisol and free salivary cortisol levels are also typically increased in T2DM.^[3] There is also an inverse correlation between cortisol level and the volume of hippocampus, found to be reduced in T2DM, depicting the cortisol as the missing link.^[3] Pro-inflammatory cytokines linked with T2DM are also important pathophysiological factors of depression and lead to hyperactivity of HPA axis by diminishing the feedback inhibition of corticosteroids.^[4] In this context, the leptin resistance in obese people, the vulnerable group of T2DM, may account for their depressive behavior.^[5] There are also genetic predispositions like tryptophan hydroxylase-2 and P2RX7 polymorphism.^[6] The behavioral outcome of the depression, including improper dietary habit, reduced physical activity, high tobacco use, and low treatment compliance, enhances the risk of developing DM. At the point of treating depression, some anti-depressants, but not all do increase the risk of diabetes.^[7] At this juncture, bromocriptine, a well-known drug for treating depression, is a potential choice to treat diabetes in depressive patients by decreasing serotonergic and sympathetic activity.^[8]

The nation-wide prevalence rate of T2DM among young adults is 9.2%,^[9] whereas lifetime prevalence of depression is about 36%.^[10] So, it is to be noted that depression is more prevalent than diabetes. That is why, it will be a step further to cope up with the “diabetic India” if it could be possible to tweak the risk factor by screening and long-term monitoring of depressive patients for DM and transmogrify the same by some novel therapeutic approach which will wane the migration from depressive population to diabetic population. At this point, a crystal clear molecular understanding of the interwoven patho-mechanisms is highly warranted to reduce the rate of incident DM among the depressive population. We strongly advocate for a study with robust methodology testing the aforesaid hypotheses with an animal model of depression,^[11] which will ultimately open up a new horizon in the healthcare of depression and diabetes, going beyond the multiple management options by creating cutting-edge possibilities of definitive cure.

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