Evaluation of vildagliptin and fixed dose combination of vildagliptin and metformin on glycemic control and insulin dose over 3 months in patients with type 2 diabetes mellitus

Sir,

We thank Mundra for a detailed analysis of our paper, and for his attempt to improve the impact of our findings.[1] In our study, 152 subjects were on plain Vildagliptin while 148 were on a fixed dose combination of vildagliptin and metformin. The uptitration of the dose was based on the glycemic control achieved. There was no protocol followed for the uptitration of the dosages

A significant drop was reported in both the groups (on vildagliptin as well as vildagliptin and metformin combination). The baseline fasting plasma glucose in plain and combination groups was 195.61 ± 57.91 and 194.26 ± 54.56 mg% respectively, which came down to 121.84 ± 27.52 and 128.11 ± 32.33 mg% respectively. Also, the baseline postprandial glucose in plain and combination of 287.43 ± 85.62 and 287.77 ± 69.94 mg% came down to 165.60 ± 34.00 and 178.67 ± 39.65 mg% respectively. The HbA1c changes were significant: Values of 9.04 ± 1.45 and 8.99 ± 1.12% came down to levels of 7.61 ± 1.04 and 7.69 ± 0.99% respectively.

Dipeptidyl peptidase (DPP)-4 inhibitors are generally weight-neutral, although modest weight loss has been observed with the DPP-4 inhibitor, vildagliptin, in patients with relatively low baseline glycemia. The weight neutrality of vildagliptin likely results in part from its intrinsically low risk for hypoglycemia. Recent studies point to additional potential mechanisms. One study found that drug-naïve patients randomized to vildagliptin exhibited significantly lower chylomicron lipid and apolipoprotein levels than placebo patients, suggesting that vildagliptin may inhibit intestinal fat extraction. Another trial found that patients randomized to vildagliptin versus placebo experienced paradoxical postprandial increases in markers of fatty acid mobilization and oxidation, in conjunction with increased sympathetic stimulation. Elaboration of these and other pathways could further clarify the origins of the favorable weight profile of vildagriptin.[2]

Adverse event reporting was only kept for hypoglycaemia, which was not observed during the trial period in any of the group.

We agree with Dr. Mundra that we definitely need more randomized trials to further elucidate the role of DPP-IV inhibitors and their benefit in terms of cardiac, lipids and insulin sensitivity.[1]

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Quick Response Code:	Website: www.ijem.in
	DOI: 10.4103/2230-8210.107877