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Adiponectin levels in nonalcoholic fatty liver disease

To the Editor:

We read with great interest the recent review produced by Sofer et al [1] on the effect of metformin treatment (at a dose of 850 to 1700 mg/d) on arterial properties, metabolic parameters, and liver function in patients with nonalcoholic fatty liver disease (NAFLD). Adiponectin is exclusively secreted by adipocytes and is considered as an anti-inflammatory adipokine. It reduces body fat; improves hepatic and peripheral insulin sensitivity; and is inversely associated with body mass index, insulin resistance, and hepatic fat. Low serum adiponectin level predisposes to the development of NAFLD and insulin resistance. On the contrary, a high level of this adipokine suggests lack or low grade of liver steatosis [2]. In the "Results" paragraph, the authors reported that serum adiponectin level tended to increase during 4 months of the treatment with metformin. However, this increase did not reach statistical significance ($P = .171$). Recently, we have reported the efficacy of a treatment with low-dose metformin compared with dietary measures alone in obese nondiabetic patients with NAFLD in a 6-month prospective randomized study [3]. Fifty patients were enrolled and randomized into 2 groups: the first group (n = 25) was given metformin (1 g/d) plus dietary treatment, and the second group (n = 25) was given dietary treatment alone. At the end of the study, plasma adiponectin measurement, available in 12 patients of metformin group and in 19 patients of diet group, increased in both groups. This increase reached statistical significance only in the metformin group (from 5.8 ± 2.7 to 7.0 ± 3.3 μg/mL, $P = .005$, in the metformin group and from 7.9 ± 4.4 to 8.5 ± 4.6 μg/mL, $P = .17$, in the diet group). This result is subsequent to the improvement of insulin sensitivity observed in the metformin group as compared with the diet group and suggests a possible role of this cytokine as a marker of efficacy treatment in NAFLD patients. However, in accordance with the authors, it seems reasonable to suppose that the increase in adiponectin levels in metformin-treated patients with NAFLD is not rapid and requires long-term therapy to improve glucose tolerance and proinflammatory state.

Conflict of Interest

The author has declared that there is no conflict of interest.

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