INTERNAL MEDICINE

□ LETTERS TO THE EDITOR □

Diabetes due to Mitochondrial Adipopathy

Key words: mitochondrial diabetes, adiponectin, insulin resistance

(Intern Med 56: 745, 2017)
(DOI: 10.2169/internalmedicine.56.7950)

The Authors Replies Thank you for your letter regarding our case report about a patient with mitochondrial diabetes who was successfully treated with pioglitazone (1).

We considered that the low plasma adiponectin level (hypoadiponectinemia) and skeletal muscle atrophy would have contributed to the insulin resistance that was observed in the present case. Insulin resistance was also reported to be associated with hypoadiponectinemia in a patient with mitochondrial diabetes in a previous report (2). As you mentioned, insulin resistance is influenced by many other factors. We evaluated the levels of several inflammatory markers and reactive oxygen species, which did not change after pioglitazone treatment, suggesting that these factors had little influence on the patient's hypoadiponectinemia and insulin resistance. Unfortunately, we did not examine the expression levels of insulin receptor mRNA or other factors that would have influenced the patient's insulin sensitivity and we did not perform any immunohistochemical analyses in relation to adipocyte dysfunction.

We considered that her diet would have had little effect on the improvement of her insulin sensitivity. She started a recommended-diet regimen during her third pregnancy when she was diagnosed with diabetes. Indeed, her body weight, her percentage of body fat and her muscle mass did not change over the three-month period.

We assumed that her short stature was derived from mitochondrial disease, because it did not seem to be related to hormonal abnormalities; however, hypopituitarism was not completely ruled out. The patient's serum levels of pituitary hormone, cortisol and thyroid hormone were all within the normal ranges. She had already had three children, which suggested the absence of overt hypogonadism. The serum levels of bone metabolic makers, including PTH, calcium and phosphate, were also within the normal ranges.

Pioglitazone has been effective and safe for at least 2 years in the present case. She did not show any symptoms of heart failure, her blood pressure was within normal limits, her cardiac function, as assessed by echocardiography, did not worsen, and good glycemic control was achieved.

We hypothesize that adipocyte dysfunction, as shown in hypoadiponectinemia, was one of the key pathophysiological aspects of mitochondrial diabetes in the present case. Pioglitazone can be a candidate drug for diabetes control when it is prescribed with careful attention to the cardiac function.

The authors state that they have no Conflict of Interest (COI).

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Received for publication July 5, 2016; Accepted for publication July 11, 2016
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 Journal Website: http://www.naika.or.jp/imonline/index.html