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CASE REPORT

Falsely decreased HbA1c in a type 2 diabetic patient treated with dapsone

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Glycated hemoglobin A1c (HbA1c) is an important indicator of glycemic control. The current recommendation for glycemic control based on HbA1c values has been widely accepted. However, HbA1c values depend on the lifespan of erythrocytes and the assay methods used. Here, we report the case of a patient with type 2 diabetes with unusual falling of HbA1c due to interference from dapsone treatment for leukocytoclastic vasculitis. He was a 52-year-old man, who was diagnosed with type 2 diabetes mellitus 5 years previously and who had been treated in our hospital in the past 3 years. Glycemia was controlled by sulfonylurea and metformin. During the 3-years follow-up period, HbA1c dropped significantly during the addition of dapsone treatment, although plasma glucose levels remained stable. HbA1c levels were raised after discontinuation of dapsone. With rechallenge of dapsone usage, HbA1c decreased again. We conclude that dapsone may be the cause of artificially low HbA1c. Other measurements to monitor glycemic control should be considered when dapsone is used for the treatment of concurrent disorders, such as autoimmune disease and pneumocystis jiroveci pneumonia.

Introduction

Dapsone (4,4'-diaminodiphenylsulfone, a synthetic sulfone) is an old drug used to treat leprosy and malaria. For its anti-inflammatory action, dapsone has become one of the major treatments for autoimmune and allergic disorders.[1–3] In post-transplanted or human immunodeficiency virus-infected patients, dapsone is an alternative drug for prophylaxis of pneumocystis jiroveci pneumonia. In this report, we describe a fall in glycated hemoglobin A1c (HbA1c) readings when a diabetic patient received dapsone treatment. We also discuss the possible mechanisms of artificially low HbA1c when a diabetic patient is co-treated with dapsone.

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Case report

This male patient, born in 1956, was diagnosed with type 2 diabetes mellitus in 2003 and treated with sulfonylurea and metformin from then onwards. He visited our hospital in July 2005 for examination of purpura over the lower limbs. Skin biopsy showed leukocytoclastic vasculitis, for which dapsone 100 mg per day was prescribed from December 2005. He tolerated dapsone well except his hemoglobin levels decreased from 14.3 g/dL to 12.3 g/dL in half a year. Hematologic assessment showed a mean corpuscular volume (MCV) of 106.5 fL; reticulocytes 2.92%; and reticulocyte production index 1.62 in June 2006. For macrocytic anemia and reticulocytosis, the patient received dapsone-induced hemolysis. He was transferred to our diabetic clinic for further diabetic care in June 2006. Examination showed no nephropathy, retinopathy, neuropathy, or abnormal ankle brachial index. The initial fasting plasma glucose was 144 mg/dL, postprandial plasma glucose 141 mg/dL, and HbA1c 4.4% (collected in a fluoride-EDTA-containing tube; assay method used: high-performance liquid chromatography, Primus CLC385, Kansas city, USA, reference range: 3.8—6.0%). The estimated average glucose[4] derived from HbA1c was 79.58 mg/dL (Fig. 1). The patient did not report any hypoglycemic symptoms. He was treated with oral metformin 850 mg and modified-release gliclazide 30 mg per day from July 2006.

In September 2006, cutaneous vasculitis improved and dapsone was discontinued. The HbA1c increased to 6.2% 3-months later without any change of oral antidiabetic agents or lifestyle. He did not complain of any hyperglycemic symptoms, such as polyuria, polydipsia or weight loss. The oral antidiabetic agents were changed to glimepiride 2 mg and metformin 850 mg per day in December 2006. The HbA1c ranged from 5.7 to 7.6%, fasting plasma glucose 112 to 103 mg/dL, and postprandial plasma glucose 109 to 117 mg/dL in the following 6 months.

New purpura lesions recurred and dapsone was then restarted in August 2007. Three months later, the patient's HbA1c level was reported to as low as 4.1% when the fasting plasma glucose was 123 mg/dL and 2-hr postprandial plasma glucose 110 mg/dL. The estimated average glucose derived from HbA1c was 70.97 mg/dL. Antidiabetic treatment was adjusted to only metformin 850 mg per day from then on. Fasting glycemia was elevated to 158 mg/dL and postprandial glycemia to 154 mg/dL; however, we observed that HbA1c remained low at around 4.2% to 5.5%. Hemoglobin electrophoresis was checked and results showed no elevated hemoglobin H, hemoglobin A2 or hemoglobin F. From February 2009, dapsone was discontinued again because of short supply. We found that HbA1c increased to 6.2% 2-months later when fasting glycemia was 132 mg/dL and postprandial glycemia 137 mg/dL. The patient's hemogram results also recovered: hemoglobin increased from 10.3 to 13.3 g/dL, MCV from 105.4 to 96.9 fL, and reticulocyte percentage from 2.55 to 0.74% in 3 months.

Discussion

This patient received dapsone to treat cutaneous condition of leukoclastic vasculitis with a good response but did not achieve complete remission. During a period of over 3 years, we observed an abrupt reduction of HbA1c during two courses of dapsone treatment for recurrent cutaneous lesions. His HbA1c increased to 7.6% after the first course of dapsone treatment. Similar changes in the HbA1c were also observed during the second course of dapsone treatment (Fig. 1). In the literature, low HbA1c induced by dapsone has been reported in one study of NOD mice[5] and six case

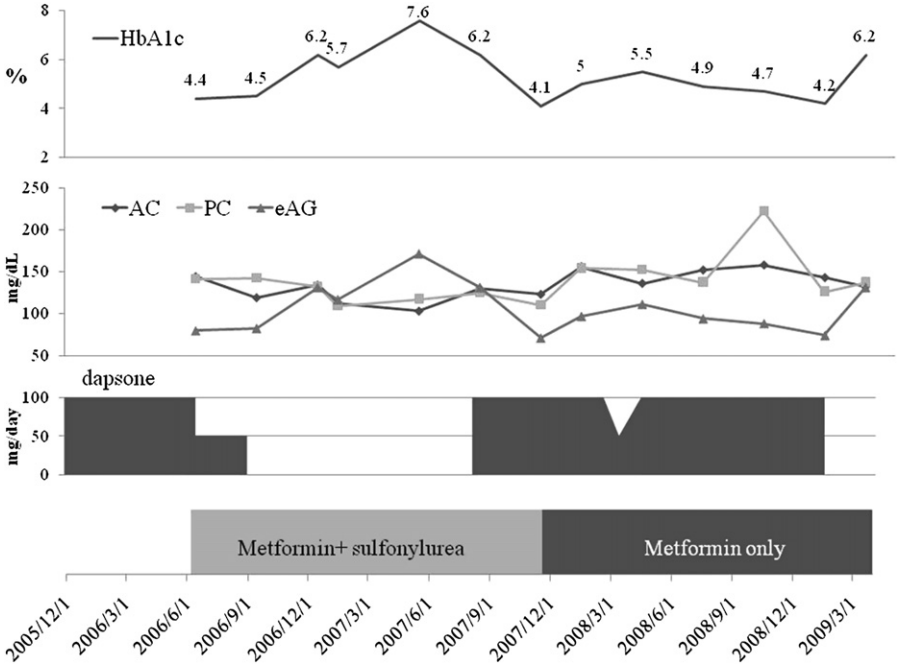

Figure 1 Interference of HbA1c readings in a patient receiving dapsone treatment. AC: fasting plasma glucose; PC: postprandial plasma glucose; eAG: estimated average glucose value = (HbA1c*28.7)-46.7 mg/dL[4].