Elmer Press

Editorial

J Clin Med Res. 2016;8(8):567-568

White Coat Hyperglycemia: The Forgotten Syndrome

Jamila Abdedaim Benmoussa[a, b], Matthew Clarke[a], Dennis Bloomfield[a]

There are many examples of medical forgetfulness. In the early 1900s, heart attack was called "coronary thrombosis", but this pathological process was forgotten in the excitement of cholesterol in all its variations and implications. It was not until the concept of thrombolysis became practical that treatment was refocused on the true pathology.

White coat hyperglycemia has suffered the same neglect but ignorance of its prevalence, its possible connection to white coat hypertension and its therapeutic relevance now require consideration.

White coat hyperglycemia was first described in the early 1990s, defined as a condition in which elevated blood glucose levels are found in a clinical setting (clinician's office or laboratory) while normal glucose levels are recorded in a self-measured unchallenging home environment [1, 2]. It has not been mentioned in the literature since 1994. Its prevalence is unknown, and its occurrence is presumably forgotten.

On the other hand, white coat hypertension is commonly recognized and considered to be present in between 25% and 40% of the population [3]. With the increased use of ambulatory blood pressure monitoring (ABPM), white coat hypertension has gained an accepted and important place in hypertension research, knowledge and literature. It has been defined as elevated (systolic) blood pressure when measured in a doctor's office or in a hospital setting while being measured at a significantly lower level when recorded by ABPM or by the patient in the unchallenging environment of the home.

We have been following a healthy middle-aged male who not only has white coat hyperglycemia with hemoglobin A_1C (HbA1c) correlating with the home-measured glucose levels, but white coat hypertension as well. The patient is a 67-year-old Caucasian retired teacher with no family or personal history diabetes mellitus. He is a thin active person who exercises daily. He described himself as a nervous individual but this is not at a level to impede his work, personal relationships or social interactions. On routine laboratory testing, he was found to have morning fasting blood glucoses above 126 mg/dL with HbA1c of only 5.3%. After instruction of the patient on proper technique of taking and processing blood samples, the home morning fasting fingersticks blood glucose levels ranged from 86 to 100 mg/dL. Laboratory blood glucose levels during the same period ranged from 127 to 143 mg/dL. For more than 10 years, the patient had been followed with a diagnosis of white coat hypertension, consistently recording office blood pressures of 150 - 190/90 mm Hg and home and ambulatory blood pressure readings of 110 -130/80 mm Hg. Physical examination was normal except for the blood pressure and there was no ventricular hypertrophy, retinopathy, glycosuria or albuminuria. HbA1c measurements in the following consecutive years were 5.5% and 5.3%. There have never been symptoms of hyperglycemia or end organ damage.

There were no confounding co-morbidities such as chronic anemia, major blood loss, hemolysis, uremia or liver failure that would render the HbA1c value falsely low or explain the discordance between the home and laboratory values.

The patient's ability to perform and correctly record results of fingerstick blood glucose and blood pressure measurements was assessed at multiple office visits.

The original study done to identify patients with high discrepancy between fasting blood glucose in clinical settings compared with self-reported values in patient with diabetes mellitus, found white coat hyperglycemia in about 50% the patients, with errors in technique accounting for the rest of the discrepancies [1].

As with blood pressure, the usual stresses of daily life, worry about being in a medical setting and acute illness may elevate the blood glucose [4, 5]. In a study to identify an association between blood glucose concentration, acute stress, and blood variables, healthy cats were exposed to extreme stresses. Blood glucose, lactate, insulin, glucagon, cortisol, epinephrine and norepinephrine concentrations were measured. There was a strong correlation between the mean glucose concentrations and mean lactate and mean norepinephrine concentrations. Gluconeogenesis, stimulated by lactate release, is the likely mechanism for white coat hyperglycemia while norepinephrine increase stimulates the white coat hypertensive episode. The same study showed rapid increase in glucose, lactate and norepinephrine concentrations within 5 min of acute stress and a slow resolution within 90 min mimicking the white coat episode response [6].

HbA1c and fructosamine, despite the fact that they are obtained in the laboratory setting, do not reflect the transient, episodic elevations of the white coat event [4, 5]. Capillary serum fructosamine level is an alternative marker that can be used to monitor blood glucose level in the clinic setting [7].

This patient, with the ability to register normal fingerstick

Manuscript accepted for publication May 30, 2016

[a]Department of Internal of Medicine, Richmond University Medical Center, Staten Island, NY 10310, USA
[b]Corresponding Author: Jamila Benmoussa, Department of Internal of Medicine, Richmond University Medical Center, 355 Bard Avenue, Staten Island, NY 10310, USA. Email: Jabenmoussa@rumcsi.org

doi: http://dx.doi.org/10.14740/jocmr2626e

blood glucose levels in multiple occasions in the unchallenging home environment that correlate with HbA1c, while losing this control in the formal biological laboratory setting, is an example of the long forgotten syndrome of white coat hyperglycemia.

The combination of high fasting blood glucose obtained in a laboratory setting and established white coat hypertension should be an alerting pattern for the clinically significant diagnosis of white coat hyperglycemia. The dual white coat effect may be much more prevalent than presently recognized.

Conflicts of Interest

None.

References

1. Campbell LV, Ashwell SM, Borkman M, Chisholm DJ. White coat hyperglycaemia: disparity between diabetes clinic and home blood glucose concentrations. BMJ. 1992;305(6863):1194-1196.
2. Lardinois CK. 'White coat' hyperglycemia. Arch Fam Med. 1994;3(5):461-464.
3. Sipahioglu NT, Sipahioglu F. Closer look at white-coat hypertension. World J Methodol. 2014;4(3):144-150.
4. Bonora E, Tuomilehto J. The pros and cons of diagnosing diabetes with A1C. Diabetes Care. 2011;34(Suppl 2):S184-190.
5. Saudek CD, Herman WH, Sacks DB, Bergenstal RM, Edelman D, Davidson MB. A new look at screening and diagnosing diabetes mellitus. J Clin Endocrinol Metab. 2008;93(7):2447-2453.
6. Rand JS, Kinnaird E, Baglioni A, Blackshaw J, Priest J. Acute stress hyperglycemia in cats is associated with struggling and increased concentrations of lactate and norepinephrine. J Vet Intern Med. 2002;16(2):123-132.
7. Sullivan C, Chambers T, Goldie D, Gillett M, Woods A. White coat hyperglycaemia. BMJ. 1993;306(6871):208.