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Case Series
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Type 2 Diabetes Mellitus Associated with Behçet's Disease: A Case Series

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Abstract

Behçet's disease (BD) is a systemic vasculitis widely prevalent in countries along the ancient Silk Road and around the Mediterranean. Endocrine complications are uncommon in this vasculitis. Non-corticosteroid-induced diabetes mellitus remains exceptional in BD. We report three cases of BD associated with diabetes mellitus type 2 in three Tunisian patients. They are two women and a man aged 42, 37, and 48 years old respectively with mean body mass index at 22.33 kg/m². Diabetes occurred at 8, 10, and 17 years respectively from the initial diagnosis of BD without signs of insulin resistance and without any prior use of systemic corticosteroids.

Keywords: Behçet's Disease; Diabetes Mellitus; Systemic Vasculitis; Diabetes

Introduction

Behçet's disease (BD) is a systemic vasculitis of still unknown etiology affecting both arteries and veins of all calibers [1]. It is characterized by the classic triad: recurrent oral aphthosis, genital ulcerations, and ocular involvement primarily in the form of anterior uveitis with hypopyon [2], but which can cause varied and sometimes severe systemic manifestations: cardiovascular, neurological, pleuropulmonary, digestive, renal, and others [3-9]. Endocrine complications remain exceptional in this vasculitis, and hypophysis, adrenal glands, and thyroid appear to be the most affected gland in BD [10, 11]. The association of BD with diabetes mellitus is not typical except in corticosteroid-induced forms [10, 11].

We report three cases of BD associated with diabetes mellitus type 2 in Tunisian patients, without signs of insulin resistance and without any prior use of systemic corticosteroids.

Cases Reports

Case 1

A 42-year-old Tunisian woman, followed for BD for 8 years with mucocutaneous involvement (recurrent oral aphthosis, vaginal ulcerations, necrotizing pseudofolliculitis, and a positive Pathergy test), and positive human leukocyte antigen (HLA) B51, presented with polyuria and polydipsia. She had no systemic manifestations of the disease and was well-treated with colchicine (1 mg/day) and salicylate (100 mg/day). Laboratory tests confirmed the diagnosis of diabetes mellitus type 2: fasting blood glucose at 7.90 mmol/L, postprandial blood glucose at 15 mmol/L, and glycosylated hemoglobin (HbA1c) at 7%. Anti-glutamic acid decarboxylase 65 antibodies (anti-GAD 65) were negative. She was treated with Metformin (2g/day) and Vildagliptin (100mg/day) with normalization of glycemic parameters.

Case 2

A 37-year-old Tunisian woman followed for BD for 17 years with simple mucocutaneous and articular involvement (oral aphthae, genital ulcerations, necrotizing pseudofolliculitis, seronegative polyarthritis, and erythema nodosum), and positive HLA-B51. She was treated long-term with colchicine (1 mg/day) and aspirin (100 mg/day), and short-term with nonsteroidal anti-inflammatory drugs (NSAIDs) for the articular involvement and erythema nodosum. Investigations for recurrent urinary tract infections led to a diagnosis of type 2 diabetes mellitus: Fasting blood glucose of 10 mmol/L and HbA1c of 8%. Anti-GAD65 autoantibodies were negative. She was treated with combined dual therapy (Metformin-Vildagliptin 850/50: 2 tablets/day) bringing her glycemic parameters to the target.

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Case 3

A 48-year-old Tunisian man, followed for BD for 10 years, presented with simple mucocutaneous involvement (oral aphthae, scrotal ulcerations, necrotizing pseudofolliculitis, and positive Pathergy test), and positive HLA-B51. He was well-stabilized on colchicine (1 mg/day) and aspirin (100 mg/day). Routine monitoring showed a fasting blood glucose level of 7.8 mmol/L. A follow-up examination confirmed the diagnosis of diabetes mellitus with a postprandial blood glucose level of 14 mmol/L, and an HbA1c of 7%. Anti-GAD65 antibodies were negative. He was treated with metformin (2 g/day), resulting in normalization of his blood glucose levels. The clinical course was marked by a severe flare-up of the disease with bilateral anterior uveitis requiring systemic corticosteroid therapy and a temporary switch to insulin analogs to control the diabetes.

None of our patients had received corticosteroids prior to their diabetes diagnosis. The workup for micro- and macro-vascular degenerative complications of diabetes was negative in all three patients, and their respective body mass indices (BMI) were 22,25, and 20 kg/m^2.

Discussion

We report three cases of type 2 diabetes mellitus occurring in patients followed for BD at respective intervals of 8, 10, and 17 years, in the absence of any prior systemic corticosteroid therapy or signs of insulin resistance (mean BMI at 22.33 kg/m^2). These findings suggest a non-random association between these two conditions.

The association between BD and diabetes mellitus is rare [12-14]. Indeed, in the recent Saudi series by Alharthy F et al, of 91 patients with BD, the association with diabetes mellitus was noted in only 6 patients (6.6%) [12].

Similarly, the meta-analysis by Chen T et al, found a pooled prevalence of diabetes mellitus in BD of 7.45% [14]. This prevalence is statistically significantly higher than that of the healthy control population (7.00%), making the association between BD and diabetes mellitus significant, with an odds ratio of 1.23 [14].

This association seems to be favored by peripheral insulin resistance, which is significantly increased in BD [13-15]. The increased peripheral insulin resistance in BD could be a consequence of the chronic inflammation and endothelial dysfunction that characterize this disease [16]. This insulin resistance occurs independently of obesity in patients with BD [17] thus reinforcing the direct incrimination of BD in its genesis.

Less commonly, diabetes mellitus could result from direct and specific pancreatic involvement in BD: destruction of pancreatic islets by the chronic inflammatory process or by severe pancreatic vasculitis. Indeed, a few cases of chronic pancreatitis and pancreatic vasculitis have been reported in the context of BD [18, 19].

Furthermore, a few anecdotal cases of type 1 diabetes mellitus associated with BD have also been reported [20], suggesting a common autoinflammatory mechanism [21].

Conclusion

Non-corticosteroid-induced diabetes mellitus remains exceptional in BD. Its presence is often synonymous of metabolic syndrome and peripheral insulin resistance resulting from disease activity (inflammation and endothelial dysfunction), and more rarely of acute or chronic pancreatic involvement specific to the disease. Therefore, it is essential to screen all patients followed for BD for diabetes, especially before prescribing any systemic corticosteroid therapy, which risks decompensating the diabetes.

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