AN UNUSUAL CASE OF ACQUIRED PARTIAL LIPODYSTROPHY PRESENTING WITH ACANTHOSIS NIGRICANS

N. Ozdemir Kutbay^{1,*}, B. Sarer Yurekli², Z. Yasar³, B. Akinci⁴

¹Celal Bayar University, Faculty of Medicine, Division of Endocrinology and Metabolism, Manisa,

²Ege University - Endocrinology, Izmir, ³Selahaddin Eyyubi State Hospital, Diyarbakir, ⁴Dokuz Eylul University - Division of Endocrinology, Izmir, Turkey

Abstract

About 250 patients with acquired partial lipodystrophy (Barraquer-Simons) syndrome have been reported so far. It is characterized by the loss of adipose tissue from the face and upper extremities, and accumulated fat in the rest of the body. The disease usually starts in females during childhood or adolescence, and usually after a febrile illness. Fat loss often comes into view in months or years. We present a 23-year-old female patient with acquired partial lipodystrophy, which is rarely seen.

Key words: Acquired partial lipodystrophy, insulin resistance, diabetes.

Dear editor,

Acquired partial lipodystrophy (APL) is a rare disease characterized by the loss of adipose tissue from the face, upper extremities and upper trunk. Adipose tissue loss usually starts during childhood or early adulthood. The disease is more common in females (1). In contrast to other types of lipodystrophy, clinically significant metabolic abnormalities associated with insulin resistance develop in a small proportion of patients (2).

Here we report a young female with APL who applied to our clinic because of *Acanthosis nigricans* that became obvious six years after she started losing adipose tissue. This 23-year-old-female first noticed fat loss on her face that started after an inflammatory disease when she was 12 years old. Progressive loss of subcutaneous adipose tissue appeared sequentially and spread from the face to the neck, upper limbs and upper trunk in a caudocephalic manner. She underwent autologous fat tissue transfer for esthetic reconstruction several times. She first noticed brown, poorly defined hyperpigmentation of the skin in her

body folds such as the lateral and posterior folds of the neck and the armpits when she was 18. She developed hypertriglyceridemia and diabetes at age 21. She was on metformin 2 grams a day at her admission. On physical examination; her height was 151 cm, weight was 52.2 kg, and BMI was calculated as 22.9 kg/m². Although she recently had subcutaneous adipose tissue harvested from the buttocks and transferred to her face, adipose tissue loss was still remarkable on her face, arms and upper trunk. Skin changes of *Acanthosis nigricans* were present in her armpits and around her neck (Fig. 1). Her laboratory results were as follows: fasting glucose: 111 mg/dL, HbA1c: 6.1%, fasting insulin: 34.3 uIU/mL, HOMA-IR score: 9.4, AST: 40 U/L, ALT: 65 U/L, triglyceride: 393 mg/dL, HDL cholesterol: 32 mg/dL, LDL cholesterol: 75 mg/dL, TSH: 1.97 uIU/mL, C3: 1.02 g/L (0.9-1.8), C4:0.29 g/L (0.1-0.4). Abdominal ultrasonography showed hepatic steatosis.

Although the etiology of APL still remains unknown, reports indicate that APL is associated

Figure 1. *Acanthosis nigricans* was noticed on her armpits and around her neck.

*Correspondence to: Nilufer Ozdemir Kutbay MD, Celal Bayar University, Faculty of Medicine, Division of Endocrinology and Metabolism, Manisa, 45125, Turkey, E-mail: nozdemirkutbay@hotmail.com

with abnormalities of complement pathway and other autoimmune diseases. Previous studies reported that metabolic abnormalities were rarely observed in APL (2). However, a recent study from our group showed that metabolic abnormalities associated with insulin resistance might develop in patients with APL during long term follow-up (3). Even so, severe metabolic disturbances are rarely observed in patients with APL. The presence of *Acanthosis nigricans* is an indicator of insulin resistance in our patient that can be further supported by increased HOMA-IR score, the presence of diabetes and lipid abnormalities that all developed at a relatively young age. Current knowledge fails to explain why some patients with APL develop metabolic abnormalities associated with insulin resistance; however, our case is an illustrative example of the causal relationship between APL and insulin resistance.

Conflict of interest

The authors declare that they have no conflict of interest.

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