Asian Journal of Research and Reports in Endocrinology

Asian Journal of Research and Reports in Endocrinology

2(1): 54-57, 2019; Article no.AJRRE.53944

# A Lipoatrophic Syndrome Acquired in a Type 1 Diabetic Patient

Jemai Chaima<sup>1\*</sup> and Ben Mami Faika<sup>1</sup>

<sup>1</sup>Department of C, National Institute of Nutrition of Tunis, Tunisia.

# Authors' contributions

This work was carried out in collaboration between both authors. Author JC designed the study, wrote the protocol and wrote the first draft of the manuscript. Author BMF managed the literature searches. Both authors read and approved the final manuscript.

#### Article Information

<u>Editor(s):</u> (1) Dr. Arun Kumar Kapoor, Professor, Department of Pharmacology, Rohilkhand Medical College and Hospital, Bareilly, MLN Medical College, Allahabad, India. <u>Reviewers:</u> (1) Umezurike Benedict Chidozie, Nigeria. (2) Gayatri C. Gawade, Delhi Technological University, India. Complete Peer review History: <u>http://www.sdiarticle4.com/review-history/53944</u>

Case Study

Received 15 November 2019 Accepted 19 January 2020 Published 30 January 2020

# ABSTRACT

Aims: Lipoatrophies can have multiple causes. Insulin lipoatrophies are among the rare causes. We report the case of patient with type 1 diabetes, with an acquired lipoatrophic syndrome.
Case Presentation: This was a 19-year-old patient who had been hospitalized in the Diabetology and Nutritional Diseases Department C of the National Nutrition Institute in Tunis for the balancing of diabetes. She has been known to have diabetes for 4 years under Neutral Protamine Hagedorn insulin (NPH insulin) basal bolus complet regimen. She had no other medical history including autoimmune. Her diabetes is poorly monitored, poorly balanced (HbA1C = 9.1%) and uncomplicated. The rotation of the injection sites was well performed by the patient. Her body mass index (BMI) was 19.2 kg/m<sup>2</sup>. The lipoatrophic lesions appeared a year ago and are found at the sites of insulin requirement was 0.4 IU / kg / day. The therapeutic approach was to prohibit injections at the lesions installed, associated with local corticosteroid therapy. Regular monitoring of the progress of these lesions was planned.
Conclusion: This observation suggests that these lipodystophies are most likely of autoimmune

**Conclusion:** This observation suggests that these lipodystophies are most likely of autoimmune origin. They cause diabetes imbalance in addition to their unsightly nature. The treatments currently used have not shown very good effectiveness, especially for extensive lesions already installed.

<sup>\*</sup>Corresponding author: Email: jemaichaima87@yahoo.fr;

Keywords: Diabetes; lipoatrohies; lipodistrophies; insulin.

### **1. INTRODUCTION**

Insulin is a vital therapy in type 1 diabetes. However, it can have multiple side effects, including skin lesions. Lipoatrophies are among the least described skin side effects of insulin [1]. We report the case of a patient with type 1 diabetes who presents lipoatrophic lesions at the sites of insulin injections.

#### 2. CASE PRESENTATION

She was a female aged 19, diabetic type 1 for 4 years, and whose diabetes is poorly monitored. She is treated with NPH insulin basal bolus regimen, with daily basal insulin requirements of 0.4 IU / kg / d. She has no particular pathological history, in particular autoimmune diseases. She was hospitalized for balancing her diabetes. The injection sites have been rotated well. She changed the needles every 2 days and kept the insulin vials at - 25°C.

On examination, she had a body mass index of 19.1 kg/m<sup>2</sup>. She had lipoatrophic lesions at the insulin injection sites. They were spindle shaped, little deep. The largest lesion is located on the right flank. They have appeared insidiously for a year and have been stable for 6 months. No accompanying signs, neither general nor local, were reported by the patient.

In biology, she had an HbA1C of 9.1%, a creatinine clearance, (estimated by the formula CKD-EPI) of 112.56 ml / min. Cholesterol, trglyceridemia, HDL-c and LDL-c were 4.45,

1.36, 0.88 mmol / L and 1.04 g / L respectively. The thyroid checkup and baseline cortisol levels were normal. The serology of celiac disease was negative.

The patient was advised to adhere well to the rotation of the injection sites, and injections at the level of the lesions were proscribed. More frequent change of needles was also recommanded. A local corticotherapy were also prescribed.

#### 3. DISCUSSION

Given the acquired, localized nature of these lipodystrophies at insulin injection sites, we retained the diagnosis of lipoatrophies secondary to the injection of human insulin of autoimmune origin.

Lipodystrophies are a common complication of insulin injections. They are most often hypertrophic (64%), rarely atrophic (4%) [2].

Lipohypertrophies are white, painless, sometimes slightly tender, swelling of the fatty tissue around the sites of subcutaneous insulin injections. Lipoatrophies are characterized by the loss of subcutaneous fat secondary to an immune mediated inflammatory reaction following the injection of insulin into the subcutaneous tissue.

These lesions are secondary to several causes including the traumatic repetition of injections in a site, the absence of rotation and the repeated use of the same needle [3].



Fig. 1. Lipoatrophic lesions diagnosed in the patient

Pathophysiology involves local and general immune responses. The local immune reaction is directed against insulin crystals [4]. The release of cytokines by macrophages, including the tumor necrosing factor (TNF) will contribute to the dedifferentiation of adipocytes [5]. The general immune response is characterized by the presence of circulating anti-insulin antibodies, Ig M deposits and complement deposits [6].

In histology, and at an early stage, there is lymphoid infiltration in the hypodermis, lobar panniculitis, phagocytosis of insulin by histiocytes and active degranulation of eosinophils and mast cells in injection sites [7].

At a more advanced stage, the lobules contain small adipocytes, the capillaries are hyperplasic and there is a degenerative loss of adipose tissue [8].

Lipoatrophic lesions cause glycemic instability in addition to their unsightly nature. The glycemic instability results from the variability of insulin absorption at the injections sites, and the increased risk of insulin injections intramuscularly, because of the scarcity of subcutaneous adipose tissue.

The management of these lesions requires adherence to the rule of rotation of the injection sites, the proscription of insulin injections at the lesions formed, and frequent changing of intralesional needles. Local or iniectable corticosteroid therapy may be prescribed [9]. Sodium chromoglycate may also be prescribed. It stabilizes the mast cell membrane which prevents the release of chemical mediators of inflammation [10]. The use of isulin analogues could be proposed, although lipotrophic lesions with insulin aspart and insulin glargine have been described [11].

# 4. CONCLUSION

Lipoatrphies are a rare complication of injecting human insulin. They cause diabetes imbalance in addition to their unsightly nature. The treatments currently used have not shown very good efficacy, especially for extensive lesions already installed.

# CONSENT

All authors declare that 'written informed consent was obtained from the patient (or other approved

parties) for publication of this case report and accompanying images.

# ETHICAL APPROVAL

As per international standard guideline written ethical approval has been collected and preserved by the author(s).

#### **COMPETING INTERESTS**

Authors have declared that no competing interests exist.

#### REFERENCES

- Jermendy G, Na'das J, Sa' Pi Z. "Lipoblastoma-like" lipoatrophy induced by human insulin: morphological evidence for local dedifferentiation of adipocytes? Diabetologia. 2000;43:955–6.
- Frid A, Hirsch L, Gaspar R, et al. The Third Injection Technique Workshop in Athens (TITAN). Diabetes Metab. 2010;36(Suppl. 2):S19-29.
- Blanco M, Hernández MT, Strauss KW, Amaya M. Prevalence and risk factors of lipohypertrophy in insulin-injecting patients with diabetes. Diabetes Metab. 2013;39: 445-53.
- 4. Atlan-Gepner C, Bongrand P, Farnarier C. Insulin inducedlipoatrophy in type 1 diabetes: a possible tumor necrosisfactora-mediated dedifferentiation of adypocites, DiabetesCare 19. 1996;1283–85.
- Jermendy G, Nadas J, Sa´ Pi Z, Lipoblastoma-likelipoatrophy induced by human insulin: morphologicalevidence for local dedifferentiation of adypocites, Diabetologia 43. 2000;955–56.
- Rademecker RP, Pierard GE, Scheen AJ. Lypodistrophyreactions to insulin, Am. J. Clin. Dermatol. 8/1. 2007;27–28.
- Shimizu I, Furuya K, Osawa H, Fujii Y, Makino H. A case of insulin-induced localized lobular panniculitis with evidence for the phagocytosis of insulin by histiocytes, Endocr. J. 54/3. 2007; 477–80.
- 8. Rademecker RP, Pierard GE, Scheen AJ. Lypodistrophyreactions to insulin, Am. J. Clin. Dermatol. 8/1. 2007;27–28.
- 9. Ramos M. Farias. Human insulin-induced lipoatrophy: asuccessful treatment with glucocorticoid, Diabetes Care 29/4. 2000;926.

Chaima and Faika; AJRRE, 2(1): 54-57, 2019; Article no.AJRRE.53944

- 10. Lopez X, Castells M, Ricker A, et al. Human insulin analog--induced lipoatrophy. Diabetes Care. 2008;31: 442-4.
- 11. Griffin ME, Feder A, Tamborlane WV. Lipoatrophyassociated with lispro in insulin pump therapy, DiabetesCare 24. 2001; 174.

© 2019 Chaima and Faika; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

> Peer-review history: The peer review history for this paper can be accessed here: http://www.sdiarticle4.com/review-history/53944