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Case Report

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Localized Acanthosis Nigricans Induced by Exogenous Insulin Delivery at Insulin Pump Site in Adolescent with Type 1 Diabetes

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ABSTRACT

Acanthosis nigricans is a hyperkeratotic dermatosis that is commonly associated with severe insulin resistance and rarely in patients with type 1 diabetes mellitus. Here, we report a novel case of an adolescent with type 1 diabetes mellitus who developed localized acanthosis nigricans at his continuous subcutaneous insulin infusion site. This case demonstrates both the unusual cutaneous complication as well as and highlights the importance of pump-site rotation with continuous subcutaneous insulin infusion.

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Introduction

Acanthosis Nigricans (AN) is a hyperkeratotic dermatosis commonly associated with severe insulin resistance, typically in intertriginous areas [1]. AN results from insulin activation of keratinocyte insulin-like growth factor (ILGF) receptors, which can lead to keratinocyte and dermal fibroblast proliferation [2]. AN has been reported in adults with type 2 diabetes mellitus (T2DM) and rarely in adults with type 1 diabetes mellitus (T1DM) at sites of exogenous insulin injection when proper site rotation is not followed [2-4]. Here, we present a novel case of a 14-year-old male with T1DM who developed localized AN at the site of continuous subcutaneous insulin infusion (CSII).

Case Study

A 14-year-old Caucasian male presented to our pediatric endocrinology clinic for a follow-up visit. The patient was initially diagnosed with T1DM at age 11 when he presented with hyperglycemia (HbA1c 10.2%), polyuria, polydipsia, weight loss, and diabetic ketoacidosis. After initial treatment and diagnosis of T1DM, he was managed with subcutaneous basal-bolus insulin for 16 months and then transitioned to CSII therapy. He initially demonstrated a strong preference for placing his CSII on his right abdomen and not following standard pump site rotation [5].

At this follow-up visit, he reported no issues, and his T1DM had been well-controlled in the interval between follow-up visits. His HbA1c was 6.1%, with a total daily insulin dose of 0.8 units/kg/day and a BMI at the 34th percentile. He reported that he had not been rotating his CSII site away from his preferred position on his right anterior abdomen lateral to the umbilicus. On physical exam, he was noted to have a leathery, hyperpigmented patch medial to his CSII (Figure 1) that appeared consistent with AN. No other cutaneous or subcutaneous findings were noted at his CSII site, and no other areas of AN were noted on physical exam other than the area on his abdomen. Multiple providers confirmed the clinical diagnosis of localized AN.

The importance of proper pump site rotation was reiterated and follow up was scheduled for 4 months after the AN was noted [5]. Laboratory workup was obtained at this 4 month follow up visit to again confirm the diagnosis of T1DM as opposed to a state of insulin resistance secondary to a different etiology (Table 1). There was slight improvement at this visit in his AN, and he continued to show resolution in the AN at CSII site with improved pump site rotation at subsequent follow up visits.

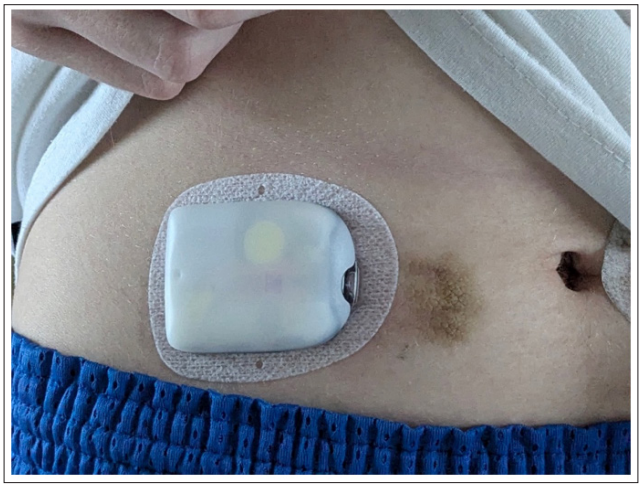


Figure 1: Area of AN at CSII

Table 1

	Result	Reference Range
Insulin	1.2	2.6-24.9 uIU/mL
C-Peptide	0.3	1.1-4.4 ng/mL
Insulin-Like Growth Factor 1	280	123-701 ng/mL
Insulin Autoantibody	53	<5 mU/mL
Glutamic Acid Decarboxylase Antibody	3,354.3	0.0-5.0 U/mL
Zinc Transporter 8 Antibody	234	<15 U/mL
IA-2 Antibody	>120	<7.5 U/mL
Islet Cell Antibody, IgG	positive	negative
Islet Cell Antibody Titer	20	<5 JDF units

Conclusion

This is the first known report of localized AN at the CSII site in an adolescent with type 1 diabetes, and it underscores the importance of appropriate insulin pump site rotation. Labs confirm the diagnosis of type 1 diabetes and not a state of severe insulin resistance [4]. There has been at least one other report of localized AN in an adult with T1DM; however, this patient was on a subcutaneous injection regimen of premix insulin (30% soluble insulin/70% isophane insulin) and was on a consistent dose of 70 units at the time the localized AN was recognized as opposed to a CSII as in our pediatric patient [6-8]. The localized hyperinsulinemia at the site of repeated insulin exposure stimulates IGF-1 receptors on keratinocytes and fibroblasts thus inducing cell overgrowth and darkening causing the changes characteristic of AN. This patient possibly has a genetic difference that results in more sensitive IGF-1 receptors than what are typically seen, and this could explain why localized AN is seen in this patient and not in the many others with T1DM on CSII. Future investigation could include genetic sequencing of the IGF-1 receptor in this patient.

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