Case report

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Ultrasound assessment of cutaneous/subcutaneous dystrophies in insulin-treated patients. A report on two cases.

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Abstract

Insulin treatment can lead to local soft tissue dystrophies. We present the cases of two insulin-treated subjects with two different injecting systems: a basal/bolus and a continuous subcutaneous insulin infusion (insulin pump). The local cutaneous/subcutaneous dystrophies adversely affected their glycaemic values. A conventional and a high resolution ultrasound (20MHz Dermascan) were used to analyze cutaneous and subcutaneous dystrophies. The particularities of these cases were the presence of the cutaneous damages, irrespective of the insulin treatment system or injected layer and the absence of remission, even after 12 months of avoiding the subjected areas.

Keywords: insulin, cutaneous, subcutaneous, dystrophy, ultrasonography

Introduction

Insulin therapy repeatedly performed without good injection practices, can lead to local soft tissue pathology. The most common insulin route is the subcutaneous (SC) one. Under varying conditions, however, other routes can be chosen. The SC damages are lipoatrophic (immune complex-mediated inflammatory lesions) and particularly lipohypertrophic. The known potential dystrophic offenders are: no site rotation, improper needle length and needle re-use (convenience or thrift), number of daily injections, duration of treatment, vicious injecting techniques [1-4]. An erratic insulin absorption, smaller areas for injection/infusion and an unaesthetic appearance are the consequences [5,6]. Two personalized insulin systems are currently in use: insulin pens or syringes for basal/bolus (multiple doses) injections and a continuous subcutaneous insulin infusion (CSII) (insulin pump). The treatment of lipohypertrophies is consistent with the avoidance of dystrophic areas and of the incriminating factors. Lipoatrophies claim to change the insulin type, steroids, or local treatment. Dystrophies are clinically depicted, but rarely US interrogated in practice. A conventional US screening (9MHz linear transducer) and a 20 MHz high resolution Dermascan (Cortex Technology) were used for diagnosis and control in our cases. The depth of tissue and the echogenicity were screened by A mode and B mode.

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Case 1

A 55 year old male, insulin treated for the last 5 years, was clinically examined within a routine presentation. He claimed an unexplained chronic hyperglycaemia within the last 2-3 years despite a flexible basal/bolus insulin therapy. Insulin boluses were repeatedly injected within two outer abdominal wall areas, of roughly 7/7cm each. At physical examination hypertrophic, non inflammatory, unpainful areas with skin tightness were detected. At conventional US examination the affected cutis/SC layers were thickened (maximum 6/20 mm compared to the nearby normal skin- 3.3/14.9 mm), irregular delineated with some emerging vascularized thickened septa crossing the SC.

A 20 MHz high frequency US screening depicted the 3.3 mm normal cutis vs. 5.4 mm thickened, irregular delineated, and inhomogenous pathologic cutis.

This patient used only needles of 5 mm length. We assumed that before the metabolic control declined, the SC insulin absorption was normal. Within time, normal tissues turned into dystrophic ones (cutis/SC layers of 5.4/20mm). From that moment, the injectate exclusively reached the cutis and as a consequence metabolic disturbance appeared. The patient regained near-normal glycemic values after changing injection sites.

Case number 2

A 30 year old woman, insulin treated for the last 15 years was examined during routine follow up. She had been using an insulin pump for 7 years. She had intensively infused the lower outer aspect of the right abdominal wall, changing the device every five days. A few months ago she observed the need for a greater dose of insulin for achieving the normal glycaemic level. A hypertrophic infused area with skin tightness was found. The conventional US depicted more than double nearby normal cutis thickness, irregular delineated, thickened isoechoic connective septa grossly crossing SC layer, with some vascular (C/PW) evidence.

Dermascan confirmed the abnormal cutis thickness between 5.26-6.8 mm and echogenicity, compared to a normal 2.2 mm.

The patient's 9 mm insulin pump cannulae was reaching the SC layer during entirely local evolution from the normal cutis/SC thickness (2.2/10 mm) to a hypertrophic ones (5.2-6.8/16 mm). By changing the insertion site, the patient regained the near-normal metabolic control. We noticed no local and US remission after 12 months.

Discussion

There are mainly two insulin injecting systems: by needles and by insulin pump cannulae both of them being potential local offenders [5,7]. The needle lengths vary from 4-12.7 mm. Despite the manufacturer's recommendation needles are being re-used for 10-30 times, their own damage being more or less visible. The pump users' injecting devices (insertion cannulae of 6 or 9 mm) should be changed every 3rd day, but in practice these were being kept for 4 or 5 days. When injecting, no matter what system is used, the rule is that the rotation of the areas must be within the same anatomic segment, but none of our subjects had respected this. The SC layer must be our patients' target [3,4,8]. An appropriate injection/infusion technique claims to know the cutis/SC thickness. Skin thickness has irrelevant demographical variations but SC tissue – body mass index related varies significantly. Our first patient (an overweight patient), repeatedly injected the same area, with a 5mm needle length. Before metabolic disturbance started, the predictable insulin absorption from a normal abdominal tissue thickness was functioning. Within time, the normal tissue turned into a dystrophic tissue (cutis/SC layers of 5.4/20mm). From that moment, the injectate had mostly reached the cutis, so erratic faster insulin absorption, as mentioned in the literature, appeared [9]. The second patient did not regularly replace the cannulae (9 mm), nor was the insertion rotated according to the rules. The patient was normal-weight. The injectate is always delivered into the SC layer irrespective of its normal cutis/SC thickness (2.2/10mm) or its dystrophic/hypertrophic ones (5.2-6.8/16mm). There is currently no agreement on dystrophies and pump users. The biochemistry of insulin, local and general conditions and disposals are under suspicion [10,11]. The slightly different values of the cutis thickness (of the same subjected area) depicted by conventional and high frequency US, have some explanations: different levels of the soft tissues' diurnal hydration (different days of US examination), nutritional status or distribution of fibrous tissue [12]. We have no cutis biopsies, but, largely connected with a chronic disglycaemia within the clinical context, the data are against scleroderma morphea [13]. The disorganized collagen bundles caused by polyols accumulation and a nonenzimatic glycosilation of collagen are the diabetic's support which probably induces the interstitial fluid and matrix texture dynamic. As a consequence some serial US cutis changes of the above cases could appear. We noticed after 12 months that there was no US remission or changes. To our knowledge there is no US description regarding insulin related dermal abnormalities. These cases have some particularities: 1) The cutis hypertrophic/dystrophic damages appeared irrespective of the injected layer, or injecting/infusion system. 2) The shorter needles (sparing muscularis) are safer but dermal offence can appear – as our first case shows. 3) After 12 months, neither US remission, nor changes were noticed.

Conclusions

From the healthcare professional's perspective cutis/SC thickness have to be assessed when deciding on injection devices and techniques and the validity of the soft tissues particularly in longstanding insulin-treated diabetics. The recovery of the affected tissues must also be US screened by targeting all the subjected soft tissue layers.

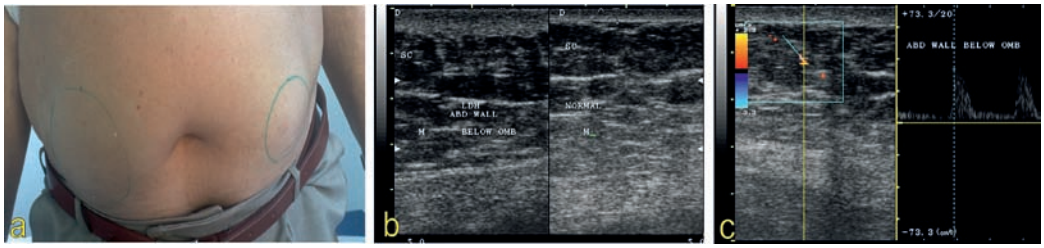


Fig 1. a) The hypertrophic injected area; b) conventional US split screen image. On the left: an inhomogenous hypertrophic/dystrophic cutis/SC of 6/20 mm irregular delineated; thickened septa crossing the SC. On the right: a normal 3.3/14.9mm cutis/SC area. c) The dystrophic SC vascularity.

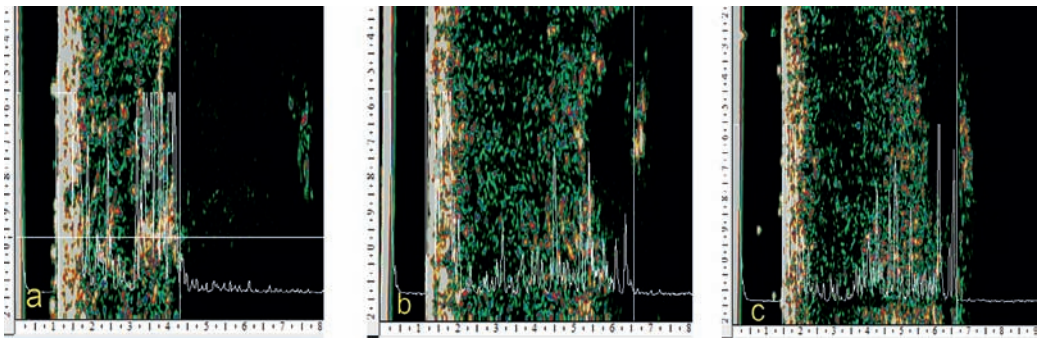


Fig 2. 20 MHz US scanning: a) Normal 3.3 mm cutis b) A 5.4 mm thickened, irregular delineated cutis with hypoechoic scattered areas. c) A 5.2 mm cutis with low echogenic papillary area.

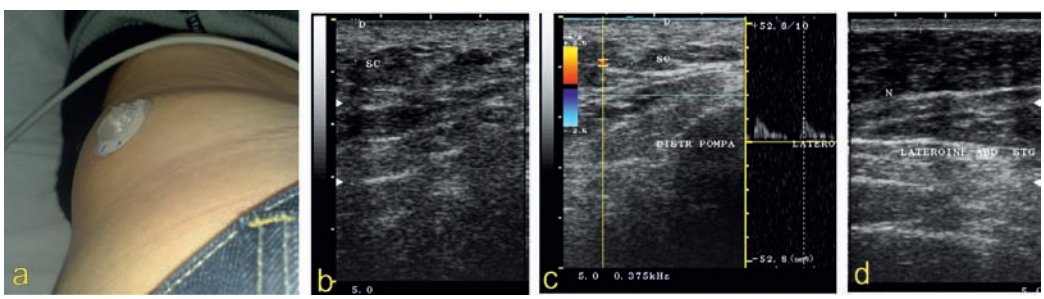


Fig 3. a) Insulin infused hypertrophic area. b) conventional US- inhomogenously thickened cutis/SC of 6.3/16mm irregular delineated; c) emerging septa with arterial signal; d) normal cutis/SC of 2/10 mm.

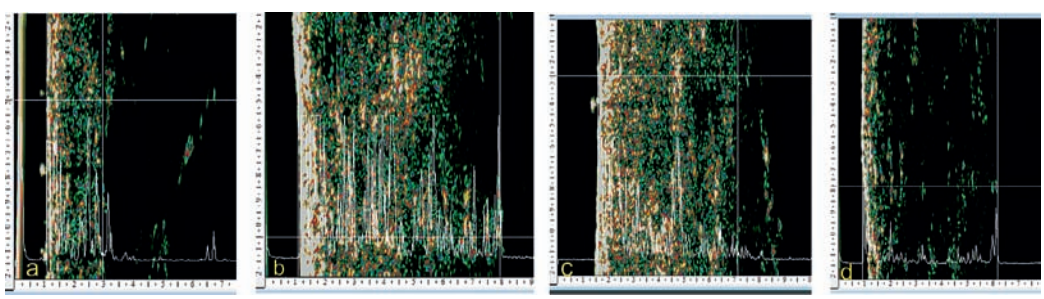


Fig 4. 20 MHz US screening: a) normal cutis of 2,2 mm. b) 6.3 mm thickened, hyperechogenic cutis; apposition of septa gives a thicker appearance of 6.8 mm. c) An inhomogenous echogenicity of 5.7 mm cutis. d) 5.26 mm dystrophic cutis, intensively hypoechoic.

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