ent, and the herbal combination appears to slowly improve the FBG concentrations over a period of continuous use. Surprisingly, this effect was rapidly reversed when the treatment ceased, as blood glucose retuned to pretreatment levels within 15–20 days. Those subjects who started to take the herbal remedy again after the withdrawal period showed a more rapid decline in their FBG concentrations than when they first started the treatment. It is unclear from this study whether the FBG would return to euglycemia over a longer period of time, but it appears that the rate of decline slows and may plateau before this was reached.

This study strongly suggests that this combination of traditional Chinese herbs, together with chromium, may be effective in improving glycemic control in people diagnosed with type 2 diabetes. The mechanism of action remains unclear and may be a combination of an increase in insulin responsiveness and glucose uptake. The relative importance of the individual component herbs is also unknown, and we are undertaking further studies to investigate this.

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Severe Injection Site Reaction to Insulin Detemir

Insulin detemir is a new long-acting insulin analog. Though generally well tolerated, injection site reactions have been reported in 2% of insulin detemir users (1). Most commonly, these untoward reactions manifest as mild injection site erythema or discomfort and seldom lead to discontinuation of the product. Herein, I report the case of a patient with a severe local reaction to insulin detemir necessitating its withdrawl.

A 37-year-old Caucasian woman with a 25-year history of type 1 diabetes was switched from NPH (Humulin N; Eli Lilly) to insulin detemir (Novo Nordisk) because of poor glycemic control characterized by undue variability of her blood glucose readings and an elevated HbA_{1c}. She was also being treated with insulin lispro (Eli Lilly), which she remained on. She had no previous history of injection site problems. With both the previous and the new insulin, she maintained the same proper injection technique and injected, as was her custom, into her abdominal wall.

The patient developed injection site problems within hours of her very first injection of insulin detemir with a characteristic and reproducible pattern occurring with all subsequent injections, ultimately necessitating withdrawl of the insulin within a few days of its institution. Within 6 h of an injection of insulin detemir she would develop a slightly raised, indurated, nonerythematous, minimally uncomfortable, nonpruritic, nontender lesion of ~3 cm. Over the subsequent 6 h, a lesion would expand in size reaching a diameter of 5–6 cm and become erythematous (without central sparing), warm, and moderately tender. Over the subsequent 12 h, a lesion would enlarge further, reaching a diameter of 10 cm, and become markedly indurated, hot, and extremely painful. Over the subsequent 12 h, a lesion would gradually and spontaneously resolve. No fever, chills, rigors, or sweats were experienced. Rotating her injection site around various parts of her abdomen was of no benefit, and a trial injection into the thigh resulted in the identical sequence of events. Insulin detemir was discontinued, and the patient reverted back to her former insulin with no further injection site problems.

Recently, a patient was described who was thought to have experienced a type III allergic reaction to insulin detemir (2); however, unlike the current patient, in this previous report, the lesions encountered were said to be "small" and only "slightly painful" as well as being nonerythematous. Whether the patient described in this case report reacted to the insulin detemir per se or one of its excipients is not known.

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A Case of Fulminant Type 1 Diabetes Associated With Painless Thyroiditis

Type 1 diabetes is classified as type 1A and type 1B diabetes, which are considered to be caused by autoimmune and nonautoimmune (idiopathic) mechanisms, respectively (1). Fulminant type 1 diabetes is characterized by a rapid-onset diabetic ketoacidosis within a short period of time, normal to near-normal HbA_{1c} level at onset, and complete β-cell destruction and was originally reported as a subtype of type 1B diabetes (2). Recently, the involvement of viral infections has been suggested to be the triggering mechanism of fulminant type 1 diabetes (3,4). The involvement of T-cell autoimmunity in this disease, however, has also been reported (5–8). Thus, its etiology is still unclear. Here, we report a case of fulminant type 1 diabetes and painless thyroiditis that presented simultaneously.

A 47-year-old woman was admitted to our hospital in a diabetic ketoacidotic coma. She suffered from fatigue and fever 2 days before admission. One day before admission, she visited a clinic and was given drugs for the common cold. On the day of admission, however, she became comatose and was transferred to our emergency room. Her arterial blood pH was 6.9 and bicarbonate was 1.5 mmol/l. She had markedly increased levels of ketone bodies and serum potassium (7.6 mEq/l). Computed tomography of the brain showed no abnormal findings. After admission, she went into cardiac arrest

and was revived by cardiopulmonary resuscitation then treated in the intensive care unit. Her plasma glucose level was 56.3 mmol/l (1,013 mg/dl) and HbA_{1c} was 6.5%. Serum and urinary C-peptide levels were very low (0.2 ng/ml and 3 μg/day, respectively). There was no increase in C-peptide following intravenous administration of 1 mg glucagon. She had no islet-associated autoantibodies (GAD antibody, islet cell antibody, or insulinoma-associated antigen-2 [IA-2] antibody. Her serum amylase was 7,492 IU/l (normal range 30–130). She had elevated lipase and trypsin levels (52 units/l [0–49] and 2,860 ng/ml [100–550], respectively). These findings were consistent with fulminant type 1 diabetes, and she was treated with intensive insulin therapy. The patient had HLA-A24, which is reported to be associated with β-cell destruction (9), and had a homozygous HLA-DR9-DQ3 haplotype, which is strongly associated with autoimmune (type 1A) diabetes (10).

After admission, she had persistent sinus tachycardia. Thirteen days after admission, an echocardiogram revealed paroxysmal atrial fibrillation. At that time, her thyroid hormones were elevated (fT3 10.4 pg/ml, fT4 4.4 ng/dl) and thyroid-stimulating hormone was suppressed (<0.03 μU/ml). Thyroid-stimulating hormone receptor antibody was negative, and a 99m-Tc–labeled thyroid scan revealed a decreased uptake (Tc RI uptake ratio 0.275% [normal range 0.4–3.0]). Thyroid-stimulating hormone measured at the previous clinic 1 day before her admission was within normal limits. Thus, the onset of fulminant type 1 diabetes and painless thyroiditis appeared to be simultaneous.

Cases of fulminant type 1 diabetes with thyroid disease or with thyroid-related antibody were previously reported (11,12), and these cases were suggested to have immunogenetic characteristics. Painless thyroiditis is also generally considered to be an autoimmune disorder (13). This case also suggests participation of autoimmune mechanisms at the onset of fulminant type 1 diabetes. On the other hand, the association of a viral infection cannot be excluded because of preceding symptoms of infection. In a nationwide survey (14), fulminant diabetes comprises ~20% of Japanese type 1 diabetes with ketosis or ketoacidosis at the onset. This new subtype, however, might be a heterogeneous entity. This is the first case of fulminant type 1 diabetes associated with simultaneous painless thyroiditis. It is useful to follow such cases to elucidate fulminant type 1 diabetes etiology, and further study is required to clarify its entity.

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Recurrence of Diabetes After Diarrhea-Associated Hemolytic Uremic Syndrome

We read with interest the article by Suri et al. (1). In this article, we find a systematic review and meta-analysis of articles assessing diabetes during diarrhea-associated hemolytic uremic syndrome (D+HUS). The 21 included studies describe 49 children who developed diabetes during acute D+HUS. Long-term outcome was reported for 44 of 49 children: 13 of 34 survivors were left with persistent diabetes requiring insulin; 11 had persistent diabetes from the outset, while 2 redeveloped diabetes at 3 and 60 months after initial apparent recovery, respectively. The remaining 21 children were reported to have made a complete recovery from diabetes. However, follow-up was <12 months or not reported for these children.