LETTERS

Insulin edema in a girl with newly diagnosed diabetes mellitus type I

Dear editor,

Insulin edema is a rare complication of insulin therapy that occurs mostly in patients with diabetes mellitus (DM) type 1[1].

We report a case of a 9.5-year-old girl who presented with swollen feet eight days after her hospitalization for newly diagnosed DM type 1 and ketoacidosis (DKA). She was receiving a subcutaneous basal-bolus insulin regimen with insulin aspart and glargine. On examination, she had visible pitting edema on the shins and back, no signs of cardiac edema, i.e., gallop, normal auscultation of the lungs, non-tender abdomen without organomegaly, and normal peripheral pulses. Her weight had increased by 1.2 kg since admission. Biochemical studies showed serum glucose 140 mg/dl, sodium 141 mEq/l, potassium 4.1 mEq/l, creatinine 0.6 mg/dl, urea 27 mg/dl, albumin 3.9 g/dl, and aldosterone 78 pg/ml (normal 24-86). A urinalysis showed urine pH 6.5, specific gravity 1,010, and 1+glucose. The estimated daily urine protein excretion was 102 mg/day. An electrocardiogram, a chest radiograph, a cardiac echocardiogram andan abdominal Doppler ultrasonogram were normal. She was discharged without specific treatment. One week later,she had a normal physical examination, and no signs of peripheral edema.

Insulin edema is an under-reported complication of intensive insulin therapy. The majority of cases occur within 2 to 4 weeks following the initial presentation with DKA[2]. The most common explanation offered is excessive fluid resuscitation in the setting of DKA. Another explanation is reversal of poor glycemic control with intensive insulin therapy, as poor glycemic control is associated with elevated glucagon, a hormone which hinders the effects of circulating aldosterone. Elevated serum concentrations of antidiuretic hormone (ADH) may also play a role, because the osmotic diuresis associated with DM type I, elevates the serum ADH. Insulin also causes sodium retention, as a result of its direct action on the renal tubule. Finally, the efficacy of ephedrine, a vasoconstrictor in cases of insulin edema indicates that arteriolar vasodilation has a pathogenetic role[3].

Insulin edema is usually a self-limited condition that physicians should be aware of. Its recognition minimizes anxiety and facilitates compliance with life-long insulin therapy.

References

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Conflict of interest

The authors declare no conflict of interest.

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