A Bizarre Complication of Levothyroxine Therapy

Sir,

We describe the case of a 49-year-old gentleman who presented with a generalized urticarial rash with wheals, itching, and angioedema 6 weeks after the initiation of levothyroxine (Brand-1) tablets for hypothyroidism. He was diagnosed with hypothyroidism while being evaluated for carpal tunnel syndrome. His thyroid-stimulating hormone (TSH) level before initiation of therapy was 170.9 mIU/L. There was no history of allergy to drugs, foods, or any other medications before the development of the present allergic reaction. There was no history to suggest chronic urticaria. He discontinued the medication by himself following which his rash subsided with complete resolution of urticarial lesions in 3 weeks. Clinical examination at the time of presentation was unremarkable. On follow-up after stopping the medication, his TSH was 28.3 mIU/L, T4 was 4.4 mcg/dL, and free T4 was 1.1 ng/dL. The titers of antithyroglobulin and antithyroid peroxidase were 1226 IU/mL and 705 IU/mL, respectively**.** Immunoglobulin E (IgE) was 102.2 U/mL (N: 0–378). Besides IgE, antinuclear antibody (ANA), C3, and C4 were done, these were normal. Anti-C1q antibody testing was also negative. He was started on levothyroxine (Brand-2) 12.5 mcg and on the same day developed urticarial rash [Figure 1] that subsided completely after stopping the medication. Subsequently, he was initiated on 12.5 mcg of levothyroxine (Brand-3), which he tolerated well, without the development of allergic reactions. It was decided to gradually increase the dosage of levothyroxine over the next 4 weeks.

The annual incidence of hypothyroidism is 1 in 10,000 in males and 1–2/1000 in females.[1] Levothyroxine supplementation is the routinely accepted treatment for hypothyroidism worldwide.[2] There are previous cases of thyroxin allergy that have been reported.[3,4] Allergic reactions to thyroxin may be due to its active ingredient or other chemical excipients. The active ingredient in thyroxin tablets is levothyroxine sodium. In addition to this, there are several inactive ingredients that vary from brand to brand. A few of the common inactive ingredients are microcrystalline cellulose, maize starch, purified talc, colloidal anhydrous silica, magnesium stearate, lactose, and acacia.[5] Our patient did not develop an allergy to Brand-3 of levothyroxine and hence the probable causative agent could be one of the inactive excipients. Drug desensitization under supervision with gradual uptitration of the dose may be done as a treatment option in patients who develop recurrent allergic reactions to different brands of thyroxine.[6]

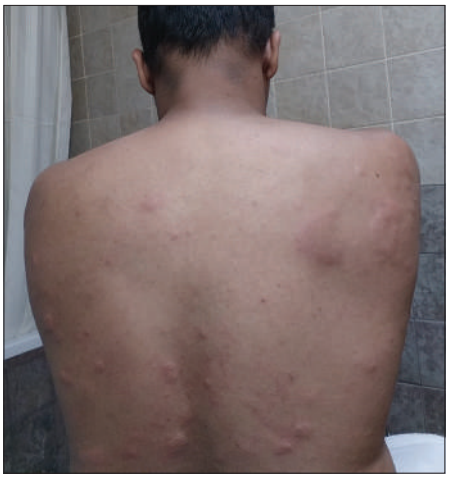

Figure 1: Urticarial rashes over the trunk following levothyroxine treatment

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Conflicts of interest

There are no conflicts of interest.

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Pneumomediastinum Complicating Diabetic Ketoacidosis in Type 1 Diabetes Patient with COV-19 Reinfection: A Case Report

Sir,

Spontaneous pneumomediastinum is a rare complication, characterized by the presence of air in the mediastinum that is not related to trauma, surgery, or other medical procedures.[1] It is thought to be caused by an alveolar rupture secondary to factors leading to a sudden increase in intrathoracic pressure, such as coughing, sneezing, and vomiting. These factors are often present during DKA but also during SARS-COV 19 infection.[2]

We describe a case of a pneumomediastinum in a patient with severe DKA and SARS-COV19 infection.

Case Presentation

An 18-year-old woman was diagnosed with type-1 diabetes by two elevated fasting blood glucose (220 and 190 mg/dl) and HbA1c at 7.5%, established by positive anti GAD65 and anti IA2 antibodies in October 2020 following a first confirmed SARS-CoV 2. This was a severe acute respiratory infection with SpO2 of <94% in ambient air and lung infiltration of >50% on the computed tomography (CT) scan of the chest; CORAD score of chest CT was 6. The patient completed a course of cefotaxime, ciprofloxacin, dexamethasone, and enoxaparin. In January 2021, the patient presented to the emergency department with diabetic ketoacidosis secondary to covid-19 reinfection (confirmed by PCR test). The clinical examination showed a lethargic patient with generalized weakness, confusion, incoercible vomiting, and signs of dehydration. She was hemodynamically stable with a blood pressure of 100/60 mm Hg and a heart rate of 90 b/min. She was tachypneic with a respiratory rate of 41/minute and her oxygen saturation was 99% in ambient air. There was no cough or fever noted.

Laboratory tests revealed metabolic acidosis with venous blood pH at 7.10, bicarbonate of 4.7 mmol/l, hyperglycemia of 380 mg/dl, hypokalemia of 3 meq/l. Urinalysis was positive for ketones (3+). Her leukocyte count was 7.75 × 10^9/l with neutrophils at 3.26 × 10^9/l, and a lymphocytes count of 3.57 × 10^9/l, an elevated C-reactive protein at 4.4 mg/dl, and normal liver and kidney functions [Table 1].

Table 1: Results of the biological exams

Variables	Value	Range
Hematological exam		
Leukocyte (10^9/l)	7.55	4-10
Lymphocyte (10^9)	3.57	1.5-4
Hemoglobin (g/l)	91	120-150
Platelets (10^9/l)	250	150-400
Coagulation tests		
APTT (seconds)	20	25-35
PT (%)	100	70-100
D-dimer (µg/ml)	6.46	<0.5
Fibrinogen (mg/dl)	327	150-350
Biochemical exams		
GPT (u/l)	70	10-35
GOT (u/l)	66	10-35
Urea (mg/dl)	15	10-50
Serum creatinine (mg/dl)	0.6	0.6-1.2
Fasting blood sugar (mg/dl)	380	70-100
ALP (U/l)	43	<125
LDH (U/l)	313	240-480
CRP (mg/dl)	4.4	<0.6
pH	7.10	7.38-7.42
PCO2 (mm Hg)	15	35-45
HCO3 (mmol/l)	4.7	22-26
Lactate level (mmol/l)	0.62	0.5-1
NA+ (meq/l)	138	135-145
K+ (meq/l)	3	3.5-5
CL (meq/l)	102	98-105