Stevens–Johnson Syndrome and Hypothermia Associated with Anti-tuberculosis Medication in a Patient with Heart Failure

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To the Editor: Although rare, multiple adverse events associated with anti-tuberculosis (TB) medication can occur simultaneously in patients with underlying diseases. Here, we describe a 76-year-old man with several preexisting disorders in whom anti-TB medication caused multiple, simultaneous adverse events, including Stevens–Johnson syndrome (SJS), hypothermia, and lactic acidosis.

He was admitted with a 2-month history of dyspnea and leg edema. He was diagnosed with congestive heart failure with atrial fibrillation and pleural effusion. Despite anticoagulant and diuretic use, pleural effusion remained, and chest computed tomography revealed necrotic mediastinal lymphadenopathy and multiple infiltrative lesions, suggesting pulmonary TB or metastasis [Figure 1a and 1b]. Pleural effusion was transudate and the sputum acid-fast bacillus (AFB) stain and TB polymerase chain reaction assay were negative. The patient was prescribed heart failure medication and discharged.

Five weeks later, sputum AFB culture showed positive result. The patient was diagnosed with pulmonary TB with mediastinal lymphadenopathy and constrictive pericarditis, and anti-TB medications were administered. After 5 weeks of medication, a chest radiograph showed reduced pleural effusion with unchanged cardiomegaly [Figure 1c].

And then 3 weeks later, he was admitted with orthopnea and skin lesions including generalized cutaneous exanthema and erythematous plaques [Figure 1e]. Dermatologist diagnosed the skin lesions as SJS with xerosis cutis. Chest radiography showed increased pleural effusion, and echocardiography revealed markedly reduced ejection fraction (37%), with worsening pericardial constriction [Figure 1d]. Because diuretics were not effective against the pleural effusion, we inserted a chest catheter.

On hospitalization day 6, his body temperature and blood pressure decreased to <35°C and 80/60 mmHg, respectively, and the lactic acidosis worsened (lactate, 6.7 mmol/L). To correct the circulatory deficit, we initiated more aggressive treatment using albumin and inotropes. His hypothermia and severe lactic acidosis continued for 3 days; thereafter, his body temperature began to increase, and the lactic acidosis gradually improved [Figure 1g]. With active topical medication, the skin lesions markedly improved by hospitalization day 15 [Figure 1f]. After overcoming hypothermia and lactic acidosis, his general condition remained stable.

SJS and toxic epidermal necrolysis are life-threatening cutaneous drug reactions in which the epidermis is separated from the dermis. [1,2] There are no universal diagnostic criteria for SJS, and the histologic findings are not diagnostic. The diagnosis of SJS is considered appropriate in patients with the following clinical features: Drug exposure 1–4 weeks prior to symptom onset; acute onset febrile illness and malaise; erythematous macules, targetoid lesions, or diffuse erythema; and necrosis of the epidermis. [1,2] Hypothermia is defined as a core body temperature <35°C and results from prolonged exposure to a cold environment, drug use, underlying pathological conditions such as diabetes or thyroid abnormalities, severe trauma, [1,3] Improving the cardiac function and normalizing the circulatory system are crucial to overcome hypothermia and lactic acidosis in severe heart failure patients. [1,4]

In conclusion, SJS, hypothermia, lactic acidosis, and aggravated constrictive pericarditis are possible adverse events of anti-TB therapy. [1,5] Because treating TB in patients with underlying diseases can cause multiple problems or aggravate underlying diseases, attention is required to promptly identify and treat these adverse events.

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Conflicts of interest

There are no conflicts of interest.

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