

DOI: 10.21767/2471-8041.100061

SLE with Pulmonary Embolism and Renal Tubule Acidosis

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Rec Date: June 12, 2017, Acc Date: June 20, 2017, Pub Date: June 22, 2017

Citation: Tsai CK, Liao HT, Wu TH, et al. SLE with Pulmonary Embolism and Renal Tubule Acidosis. Med Case Rep 2017, 3:3.

Abstract

We report a middle aged lupus woman with dyspnea, which was caused by renal tubule acidosis, pulmonary embolism, and iron deficiency anemia. The symptoms were eventually completely gone after relief of the individual underlying etiologies one by one.

Keywords: Pulmonary embolism; Renal Tubule acidosis; Systemic lupus erythematosus (SLE)

Case Presentation

Symptomatic renal tubule acidosis ((RTA) in patients with systemic lupus erythematosus (SLE) are common [1]. The clinical presentations of this setting include dyspnea which is body's compensatory mechanism for washing out carbon dioxide to overcome acidosis. Pulmonary embolism (PE) is also infrequently encountered in SLE patients especially when the disease is accompanied by autoantibodies against cardiolipin or involving the coagulation cascade [2,3]. The presentation of PE also includes dyspnea that cannot be easily differentiated from other causes of air hunger. When these two diseases happen together in a same patient with SLE, they pose a big challenge to clinical rheumatologists. We here report a middle aged woman with such an unusual constellation of symptoms who was eventually controlled by short-term low molecular heparin and long-term anti-coagulants as well as replacement of sodium bicarbonate.

A 47-year-old woman, a non-smoker, presented with progressive dyspnea on exertion for two weeks. A diagnosis of SLE has been made 16 years earlier with a setting of symptoms including pleural effusion, joint pain, fever, antinuclear antibodies (ANA) at 1:1280 in a speckled pattern, and anti-dsDNA antibodies. She has been treated with oral prednisolone 15 mg daily for several years.

The dyspnea did not compromise her normal activity except that mild air hunger might develop on prolonged walking. Episodic palpitation was present sometimes. A right lower thoracoscopic segmentectomy has been done to remove harmatomas several years ago. There's a microcytic anemia

resulted largely from menstrual overflow due to adenomyosis. D-dimer level was elevated. A pulmonary function test revealed FEV1 79% with a severe gas exchange reduction. Chest radiograph was unrevealing. However, high resolution computerized tomography (HRCT) showed small nodules and semisolid ground glass opacity in the right lower lobe (RLL) as well as subpleural honey-combing like reticular opacities at the right basal field, compatible with a healing process after surgical procedures.

Discussion

On admission, pale conjunctivae and hyperpnea (25/min) were noted. There were ventricular premature contractions with normal heart ventricles, under treatment with mexiletine/propranolol. Arterial blood gas (ABG) analysis in room air revealed normal oxygenation with a compensated metabolic acidosis (HCO_3 -12 mEq/L, anion gap (AG) 15 with a $\Delta\text{AG}/\Delta\text{HCO}_3 < 1$). Biochemical measurements showed lactate 26 mg/dl, serum creatinine 0.81 mg/dl and urine AG 17.4 with absence of ketone body. With a urine pH of 6.31, she was suspected to have type I RTA. After oral replacement with sodium bicarbonate, the hyperpnea alleviated to some extent but not completely as she still experienced occasionally air hunger on exertion.

Iron replacement was given to overcome iron deficiency anemia (IDA, with $\text{Fe}/\text{TIBC}=15\%$) associated dyspnea. A computed tomographic angiography (CT angiography) showed emboli in the segmental branches of RUL and left lingual as well as bilateral lower lobes. Low molecular weight heparin (LMWH) was given, which soon almost completely relieved the dyspnea. Thus, it was replaced with warfarin later. With subsequent regular supplement of sodium bicarbonate and iron as well as warfarin administration, the dyspnea ameliorated. A latest investigation still failed to show presence of anti-cardiolipin IgM and anti-beta2-glycoprotein I ($\beta2\text{-GPI}$). More recently, because of the menopause, iron replacement was stopped, but regular hydroxychloroquine and low dose oral steroid were still in use.

The presentation of PE includes cough, orthopnea, wheezing, pleuritic pain, calf/thigh pain or swelling, or hemoptysis. There may be hypoxemia, widened alveolar-arterial gradient and respiratory alkalosis. On the other hand, a

severe RTA often results in renal stones, hypokalemia, or failure to thrive. This woman had dyspnea but no other suggesting symptoms for PE. Thanks to CT angiography, it was eventually demonstrated. Unfortunately, a severe metabolic acidosis developed rendering search for etiology of dyspnea more difficult. Type I RTA was finally diagnosed. It is interesting that deep vein thrombosis in the lower limbs or anti-phospholipid syndrome, common precedent diseases for PE, were not present. Because the dyspnea symptoms alleviated to a lesser extent after treatment for PE, dramatically improved after the correction of acidosis, and almost completely alleviated after menopause when the iron loss suddenly stopped, we think that all three pathological factors, PE, RTA and IDA might have similar contribution to her dyspnea.

Conclusion

In conclusion, whatever the causes, the most significant message conveyed by this case is that in taking care of SLE patients with dyspnea, multiple factors which may seem independent, should be taken into consideration to reach a holistic treatment.

Acknowledgements

This is supported by Ministry of Science and Technology (NSC102-2314-B075-067-MY3) and Taipei Veterans General Hospital (V105C-114).

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