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Methylprednisolone-Responsive Leptospiral Acute Pulmonary Syndrome

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To the Editor,

The article by Johan-Arief et al highlights the role of early methylprednisolone therapy to reduce morbidity and mortality of the acute pulmonary syndrome in leptospirosis,¹ and the authors emphasize the ideal administration of this drug within 12 hours after diagnosis. They reported a 26-year-old man with fever of one-week duration, who on the third day had presented with cough and breathlessness, and episodes of hemoptysis occurred two days later.¹ His professional work involved frequent close contact with soil, and he was swimming in a waterfall a week before the onset of the high-grade fever associated with chills, rigors and body aches.¹ Physical signs were jaundice, subconjunctival suffusion, and bilateral pulmonary crepitations. Blood tests showed neutrophilic leucocytosis, low platelets, disordered coagulation, liver and renal functions; and the chest x-ray revealed scattered alveolar infiltrates. The Weil's disease was diagnosed by positive IgM ELISA, and the specific microscopic agglutination test (1:400).¹ Intravenously, he underwent tranexamic acid (500 mg 3 times daily), plus benzyl penicillin (1200 mg every 6 hours) during a week, and methylprednisolone (15 mg/kg daily) for 3 days.¹ His clinical response to treatment was good and he was soon discharged home asymptomatic. Environmental factors and typical features of severe leptospirosis raised the diagnostic suspicion; as the early diagnosis is the cornerstone of prompt adequate treatment of severe leptospirosis, one must consider some main concerns that constitute frequent challenges in clinical practice.^{2,3} During the first week of acute leptospirosis, the blood borne bacteria almost invariably do not evoke an immune response enough to yield antibody levels and false negative tests can occur;^{1,3} so, the goal of starting adequate therapy within the first seven days of disease may be hindered.³ Pulmonary involvement with hemoptysis has been more often reported in severe Weil's disease, and the coexistence of accentuated thrombocytopenia may play a role on the poorest prognoses. Significant pulmonary changes as well as low platelet counts can pose other concerns about diagnostic pitfalls with Hantavirus and dengue

infections, especially in developing countries.²⁻⁵ Since the first reports in North American Indians, the possibility of mistakes involving the acute pulmonary syndrome caused by leptospirosis and HPS was commented in Brazilian Journals.⁴ Brazilian authors described a 19-year-old man with the diagnosis of anicteric leptospirosis, who had a productive cough without hemoptysis, reduced lung sounds on the right lower third of the thorax, and normal kidney function. Images of computed tomography confirmed the acute inflammatory process in the right lung, and he underwent a successful course of intravenous ceftriaxone.³ Worthy of note, the patient had clinical and electrocardiographic findings of pericarditis; moreover, the count of bands was high (up to 1020) and the platelet count was low ($82 \times 10^9/L$). Although not confirmed, these clinical and complementary findings were suggestive of HPS; a condition that has been described in the Brazilian Central Plateau either isolated or as a coinfection.^{3,5} On a practical standpoint, corticosteroids have been useful to control both the syndromes.^{1,5}

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