Systemic Toxocariasis Presenting With Eosinophilic Ascites and Pericardial Effusion

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Visceral larva migrans (VLM) syndrome is a clinical manifestation of systemic organ involvement by Toxocara species. It is often asymptomatic and self-limiting while some present various manifestations. Here we report a case of a 48-year-old male patient admitted for abdominal distension and chest discomfort. Initial blood tests showed hemoglobin of 13.1 g/dL, hematocrit 38.7%, 14,290 leukocytes with 55.3% eosinophils, 155,000/mm³ platelets, mildly increased AST 67 IU/mL and ALT 216 IU/mL, serum total protein 5.9 g/dL, albumin 3.6 g/dL, and proBNP 1,674.2 pg/mL. The anti-nuclear antibody (ANA), anti-mitochondrial antibody (AMA), anti-smooth muscle antibody were all negative. Abdominal CT (Fig. A) and ultrasonography (Fig. B) revealed moderate amount of ascites and both pleural effusion. Echocardiography showed scanty pericardial effusion with preserved ejection function (Fig. C). The ascitic fluid was clear and straw-colored, which cell count showed 4,500 cells/mm³ with 90% eosinophils, indicating eosinophilic ascites. We considered eosinophilic gastroenteritis and parasitic infestation for differential diagnosis. Total IgE was 189.9 IU/mL and serology results were positive for toxocariasis Ab IgG. The diagnosis of active systemic toxocariasis was made and albendazole 800 mg was initiated. 2 months later, follow-up CT revealed no apparent ascites, pleural effusion and pericardial effusion (Fig. D). We report this case as systemic involvement of toxocariasis may delay the diagnosis as serologic testing is not routinely performed at clinical settings.

Telbivudine-induced myopathy in a patient with chronic hepatitis B

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Telbivudine (L-deoxythymidine) is well tolerated for long-term use in treatment of chronic hepatitis B (CHB). But there have been reports on several side effects. Here we report a case of telbivudine-induced myopathy in patients with CHB. A 38-year old man who received telbivudine monotherapy for CHB presented with general weakness and loss of muscular mass and strength over the past 6 months. He was diagnosed with CHB in his high school years. Since his liver function tests showed abnormal result on his regular check-up, he started taking 600 mg of telbivudine once daily in January, 2012. The serum HBV-DNA level decreased from 9,158,793 IU/mL to less than 20 IU/mL and the serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels were also normalized. However, he began to complain of fatigue and progressive weakness of extremities in July, 2014. Laboratory analyses showed AST of 54 IU/L, ALT of 83 IU/L, total bilirubin of 1.05 mg/dL. His serum creatine kinase (CK) level was elevated up to 291 IU/L. Electromyography (EMG) was performed in the right upper and lower extremity muscles. Increased polyphasic motor unit action potential with slightly early to early recruitment patterns were noted in the right and lower extremity muscles. The electrophysiologic findings are suspicious of myopathy with dominant involvement of proximal muscles. A muscle biopsy on biceps was performed. The pathologic confirmation of the atrophy of muscle fibers with a few T lymphocytes infiltration was reported. The patient was diagnosed with telbivudine-induced myopathy. He stopped taking telbivudine and changed antiviral agent to 300 mg of tenofovir once daily. After a week after withdrawal of telbivudine, the serum CK levels decreased to 142 U/L and his clinical symptoms were improved. Even though there have been few cases of telbivudine-induced myopathy in Korea, when a patient administered antiviral agents for CHB presents with general weakness or changes in muscle strength, physicians should consider the possibility of drug-induced myopathy. And this reversible adverse event can be detected with careful examinations, inspections and monitoring of serum CK level.