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A CASE OF CARBAMAZEPINE INDUCED SYSTEMIC LUPUS ERYTHEMATOUS (SLE)

Shahin Asgari[1], Hamid Rouhi Boroujeni*[2], Mahmoud Mirhoseini[3], Hojjat Rouhi Boroujeni[4], Minasadat Khoddami[5]

[1]*Chief Resident of Internal Medicine, Shahrekord University of Medical Sciences, Shahrekord, IRAN*
[2*]*Pulmonologist and Associated Professor of Biochemical Research Center, Shahrekord University of Medical sciences, Shahrekord, IRAN*
[3]*Endocrinologist and Assistant Professor of Internal Medicine, Shahrekord University of Medical Sciences, Shahrekord, IRAN*
[4]*Student Research Committee, Medical Plants Research Center, Shahrekord University of Medical Sciences, Shahrekord, IRAN*
[5] *Shahrekord University of Medical sciences, Shahrekord, IRAN*

ABSTRACT

Carbamazepine is an anticonvulsant drug that used to treat seizures and nerve pain and such as most drugs have side effect. Systemic lupus erythematous is one of connective tissue disorders that often caused by an auto immune mechanism of unknown, although some drugs cause SLE. This report aimed to demonstrate the carbamazepine induced SLE after its oral consumption for Convulsion. This is the report carbamazepine-induced systemic lupus in a 16- year-old patient who had been treated with carbamazepine for 2 years because of seizures. . After cessation of consumption and treatment, symptoms improved and antinuclear antibodies disappeared and indicated the patient's definite recovery. So far, few cases have been reported of carbamazepine-induced lupus after years the start of treatment. This is report about carbamazepine-induced systemic lupus erythematosus with serological confirmation after 2 years of treatment. Systemic lupus erythematosis is a syndrome of positive ANA associated with symptoms such as fever, malaise, arthritis or intense arthralgias/myalgias, serositis, and/or rash. The syndrome appears during therapy with certain medications and biologic agents, occurs predominantly in whites, has less female predilection than SLE, rarely involves kidneys or brain. Many drugs like carbamazepine. Hydralazine and phenytoin, cause SLE, but carbamazepine is a rare cause in literature. But according to physical exam and laboratory data this case occurred.

KEY WORDS
Carbamazepine, systemic lupus, erythematous (SLE), Hydralazine

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***Corresponding Author**
Email:
hammfer@yahoo.com

INTRODUCTION

Carbamazepine is an anticonvulsant that used to treat seizures and nerve pain. Although carbamazepine is usually well tolerated by most people, the potential side effects of therapy can vary from mild symptoms to severe, which may cause side effect such as slurred speech, vomiting, severe weakness, heart disease, dizziness, liver or kidney disease, glaucoma, drowsines, or dry mouth, swollen tongue, jaundice, headache, high blood pressure, high cholesterol or triglycerides, a thyroid disorder; lupus; porphyria [1].

Drug-Induced Lupus is a syndrome of positive ANA associated with symptoms such as fever, malaise, arthritis or intense arthralgias/myalgias, serositis, and/or rash. The syndrome appears during therapy with certain medications and biologic agents, occurs predominantly in whites, has less female predilection than SLE, and rarely involves kidneys or brain [2]. The list of substances that can induce lupus-like disease is long such as the anticonvulsants carbamazepine and phenytoin. ANA usually appears before symptoms [3].

MATERIALS AND METHODS

A 16-year-old man with a medical history of seizure disorder whose use anticonvulsant carbamazepine from 24 months prior to this admission, presented with the chief complaint of edema and dyspnea. His symptoms begun two weeks ago. His vital signs were BP of 100/60 mm Hg; heart rate, 114 beats/min; respiratory rate, 30 to breaths/min; and temperature, 38C. The patient had not a pulsus paradoxus. On physical examination, the patient's chest was decreased auscultation bilaterally, and cardiac auscultation heart sounds, with regular S1, S2.

laboratory data included a BUN, 11 mg/dL; creatinine, 0.7 mg/dL; total bilirubin, 3.7 mg/dL; aspartate amino transaminase (serum glutamic oxalacetic transaminase), 431 IU/L; alanine aminotransaminase (serum glutamic pyruvate transaminase), 183 IU/L; albumin, 3.7 g/dL; creatinine phosphokinase, 143 IU/L; WBC count, 10. × 103/μL with a normal differential; hemoglobin 13.2 g/dL; and hematocrit, 40.5%. The ECG demonstrated sinus tachycardia and low voltages without electrical alternant.

RESULTS

A chest radiograph revealed a "water bottle" cardiac silhouette [Fig. 1], bilateral pleural effusion without evidence of pulmonary infiltrate.

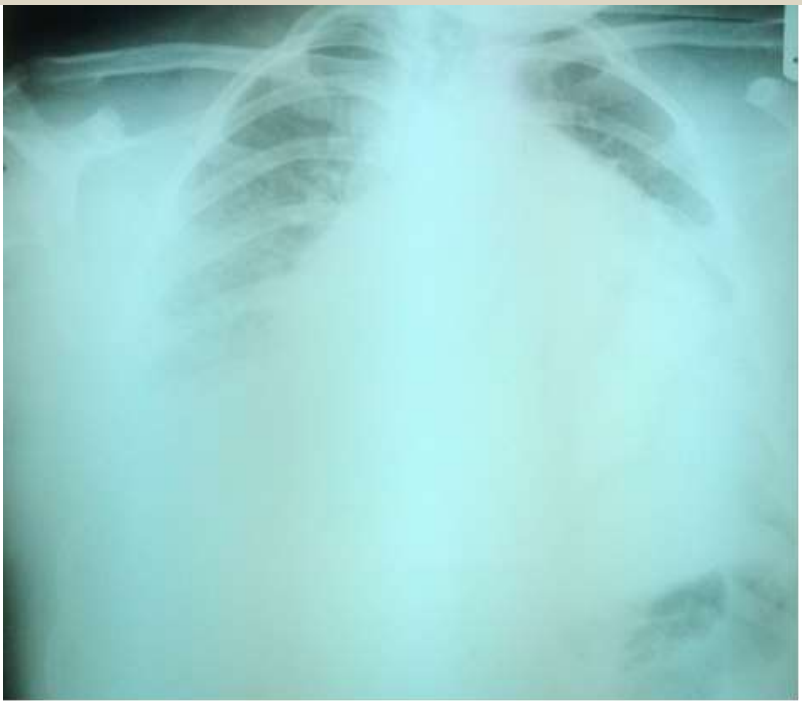

Fig. 1: "water bottle" cardiac silhouette, bilateral pleural effusion without evidence of pulmonary infiltrate.

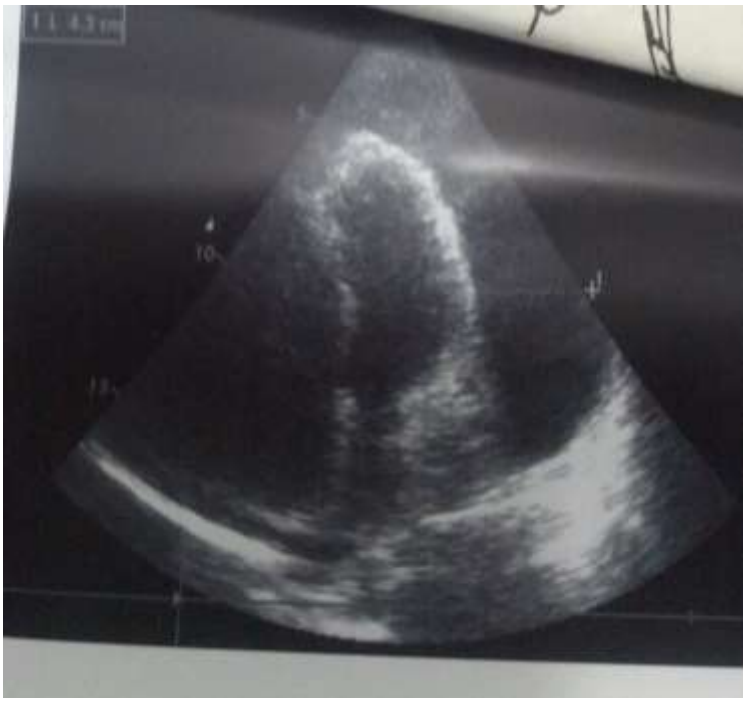

Fig. 2: A massive pericardial effusion in carbamazepine induced SLE.

An emergent echocardiogram showed a massive pericardial effusion, no tamponade, no RA and RV collapse.[Fig. 2]

Pericardiocentesis was performed with approximately 1000 mL of fluid removed. The patient began to improve breathing. Echocardiogram repeated and showed reduction in the pericardial effusion. pericardial fluid studies showed a; RBC count, 8500 cells/μL; WBC count, 235 cells/μL (neutrophils 65%, lymphocytes 35%); protein, 6 mg/dL; glucose, 20 mg/dL; and lactate dehydrogenase, 14 IU/L. Blood serologic studies showed antinuclear antibodies positive (1:320), anti-double-stranded-DNA (dsDNA) negative, antihistone antibodies positive. Serum studies for ADA and HIV were negative. Complement levels of C3, C4, and CH50 were normal limits. According neurology consult begun for antiseizure therapy and was discharged.

DISCUSSION AND CONCLUSION

Although some drugs cause pleural effusion and pericardial effusion and even ascites like concato disease without collagen vascular disorder, that reported by Hamid Rouhi and et al [4], but some drugs can cause systemic disease, that serositis is one of the presented symptoms.

Drug –induced SLE is generally equally common in males and females, and more common in older people and white populations. The risk for developing drug-induced lupus varies substantially between different medications, ranging from 15 to 20 percent of those taking procainamide, and 7 to 13 percent of those taking hydralazine, to as low as 2 per 1000 for those taking anti-tumor necrosis factor (TNF) agent, and 5 per 10,000 of those taking minocycline The diagnosis of SLE is based on characteristic clinical features and autoantibodies [5-6].

In many patients, criteria accrue over time. Antinuclear antibodies (ANA) are positive in >98% of patients during the course of disease. High-titer IgG antibodies to double-stranded DNA and antibodies to the Sm antigen are both specific for SLE and, therefore, favor the diagnosis in the presence of compatible clinical manifestations , but drug –induced SLE is rarely associated with anti-dsDNA, is commonly associated with antibodies to histones [2], and usually resolves over several weeks after discontinuation of the offending medication. In this case ANA and Anti histone anti body is positive and Anti ds DNA is negative, according manifestations and serologic studies drug-induced SLE considered for patient.

CONFLICT OF INTEREST
There is no conflict of interest.

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FINANCIAL DISCLOSURE
None

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