

Title:

DRESS syndrome secondary to carbamazepine

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Dear Editor,

Trigeminal neuralgia is a disabling facial neuropathic pain, such as electric shock. Carbamazepine is a drug of first choice in its treatment, however, it is not free of adverse effects, one of them listed in the data sheet with very rare frequency (<1/10,000) is DRESS¹ syndrome. This consists of a pharmacological reaction with the appearance of skin rash, peripheral eosinophilia and involvement of other systems, including the liver². The latency time from the start of pharmacological treatment and the onset of the clinic varies, between 2 and 8 weeks³. Early detection along with early recognition of the potentially causative drug is important to withdraw it and start treatment.

This is a 62-year-old woman with a history of type 2 DM and trigeminal neuralgia who is treated for 10-day 39°C fever and the appearance of trunk rash in the last 48 hours. He has poor control of neuralgia, which until now he treated with pregabalin, so carbamazepine is started 20 days ago, with a recent dose increase.

In physical examination, a fever of 38.5°C stands out. Normal cardiopulmonary auscultation. Soft, painless abdomen without palpable megalias. Highlights include conjunctival subicteric tint and extensive macular rash on the trunk, face and extremities.

Laboratory tests are performed showing; leukocytes $22500 \times 10^3/\mu\text{l}$ (PMN 38%), eosinophils 8.9%, lymphocytes $10600 \times 10^3/\mu\text{l}$, Hb 13.3 g/dL, platelets $257000 \times 10^3/\mu\text{L}$. Coagulation: TP 50%, INR 1.62. Biochemistry: Na⁺ 131 mEq/L, K⁺ 3.8 mEq/L, urea 26.9 mg/dl, Cr 0.43 mg/dl, filtered >90, AST 236 U/L, ALT 359 U/L, GGT 2267 U/L, BT 1.12 mg/dL, FA 679 U/L, LDH 655 U/L, PCR 76 mg/L.

A study of autoimmunity liver disease (ANA, AMA, ASTHMA, Anti-LKM) and hepatotropic virus serologies (HIV, HBV, HCV, treponema, EBV, CMV, VZV, HSV, Chlamydia, Mycoplasma) that turns out to be normal. Abdominal ultrasound is performed, within normal.

According to the evaluation of the causality of probabilistic CIOMS/RUCAMS for DILI by Carbamazepine, a score of 7 is obtained (probable), so with the data compatible with DRESS syndrome secondary to Carbamazepine with secondary hepatotoxicity, this drug is discontinued and corticosteroid therapy with oral prednisone is initiated. Close monitoring is carried out with control tests, showing at 7 days resolution of rash (Figure 1), disappearance of fever and improvement in liver profile.

We must take into account this multisystem disorder in patients with acute pharmacological hepatitis who present with fever, skin rash and eosinophilia in peripheral blood. The use of corticosteroid therapy has been effective.

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Figure 1. Resolution of rash after 7 days of corticosteroid therapy.



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