A
bacavir is a potent inhibitor of human immunodeficiency virus type 1 reverse transcriptase [1]. It has a significant penetration of the blood-brain barrier, synergy with other antiretroviral agents, good oral bioavailability and a metabolic pathway that is not dependent on cytochrome P450 liver enzymes [2]. Recently it has been demonstrated that in antiretroviral-naive HIV-infected adults the triple nucleoside regimen of abacavir, lamivudine and zidovudine is equivalent to a standard protease inhibitor regimen in achieving a virologic suppression (HIV RNA concentration of 400 copies/ml at 48 weeks) [3].

Like other antiretroviral agents, abacavir can induce various side-effects. The most common side-effects, seen during abacavir therapy, are gastrointestinal and neurologic. In the CNAA2001 trial the most commonly reported adverse events were nausea, headache, asthenia, diarrhea, insomnia, dizziness, abdominal pain, fever and vomiting [4].

An important clinical side-effect related to the use of abacavir is hypersensitivity reaction which can be fatal [5]. The median incidence of this syndrome is low at 3% (range 2-5%). The hypersensitivity events begin at a mean of 11 days after the initiation of abacavir (usually within the first 6 weeks of treatment), although this reaction may occur at any time during therapy [5].

Although presentation of this idiosyncratic reaction varies markedly among HIV-infected patients the frequently occurring symptoms are fever, skin rash, gastrointestinal symptoms (nausea, vomiting and diarrhea) and fatigue or malaise [5]. Recently, respiratory symptoms have been recognized as part of the hypersensitivity reaction, occurring in approximately 20% of patients having this reaction (dyspnea, cough or pharyngitis) [6]. If an induced hypersensitivity reaction is suspected, abacavir should be stopped and not be restarted.

We report a case of an HIV-infected patient who showed enanthema and fever after a week of treatment with an abacavir-containing regimen.

A 43-year-old man, HIV-positive since 1986 (CDC B3) and with chronic hepatitis C, was admitted to our unit because of a *Clostridium difficile* related diarrhea. He had never had an AIDS defining illness and had always refused antiretroviral therapy, taking only cotrimoxazole three times a week. Before discharge, on the basis of his viral load (*HIV RNA > 500000 copies/ml*) and severe immunosuppression (T CD4 lymphocytes 103/mm³, 10%) we convinced the patient to start an antiretroviral therapy, taking only cotrimoxazole three times a week. Before discharge, on the basis of his viral load (*HIV RNA > 500000 copies/ml*) and severe immunosuppression (T CD4 lymphocytes 103/mm³, 10%) we convinced the patient to start an antiretroviral therapy. Considering his poor predisposition to taking the medicine we proposed an antiretroviral therapy comprising combivir (zidovudine + lamivudine) and ziagen (abacavir). He started therapy on 13th March 2001. After a week the patient returned to our unit with a slight increase in temperature (37.8 °C) and appearance of enanthema in the soft palate. The patient did not refer any
other symptom. We invited him to continue his therapy. After three days he returned with fever (38.3 °C) and a worsening enanthema characterized by an extension of lesions to the hard palate and resembling petechiae tending to confluence. We stopped the abacavir treatment, continuing the zidovudine/lamivudine combination and cotrimoxazole. Laboratory evaluation disclosed the following: WBC count 3500/mm$^3$; AST 49 IU/L (n.v. 5-45 IU/L); ALT 83 U/L (n.v. 5-50 IU/L) and GGT 70 IU/L (n.v. 5-60 IU/L). Coagulation tests (prothrombin time, partial thromboplastin time and fibrinogen) were normal like platelet count (154000/mm$^3$; n.v. 150000-400000).

Suspecting an abacavir-induced hypersensitivity reaction we stopped abacavir. After its discontinuation symptoms improved rapidly and resolved in five days. We performed no biopsy of the lesions or microbiological examinations for the rapid resolution of the clinical picture. At the beginning of April we added sustiva (efavirenz) to his antiretroviral therapy but the patient stopped antiretroviral therapy after five days. Our experience confirms the importance of a careful physical examination of HIV-patients taking abacavir during the first month of therapy (hypersensitivity syndrome occurs usually in this period). If hypersensitivity reaction is suspected abacavir should be discontinued and not be restarted because of the risk of more severe hypersensitivity reactions, including life-threatening hypotension and death.

Key words: abacavir, ipersensibilità, enantema

**REFERENCES**


