Stevens-Johnson syndrome induced by allopurinol in patients with chronic kidney disease

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Abstract

Allopurinol is a xanthine oxidase inhibitor that prevents the production of uric acid to reduce plasma uric acid levels to a normal range. It is the most frequently used anti-hyperuricemic agent in the world due to its long-term pharmacological effect. However, allopurinol is also one of the most frequent causes of Severe Cutaneous Adverse Drug Reactions (SCAR) including Stevens–Johnson Syndrome (SJS), therefore, SJS is a serious problem in allopurinol therapy, in spite of the ideal anti-hyperuricemia effect of allopurinol. We report two cases report of Stevens-Johnson syndrome induced by allopurinol in patients with chronic kidney disease (CKD). Case Report-1: N.E 70 years old, followed for CKD stage-IV for three months put on allopurinol 300 mg/day for one month for asymptomatic hyperuricemia, admitted for Stevens Johnson syndrome, he was conscious, feverish at 38.5 °C, hemodynamically stable, having a prominent bullous detachment on the back, anuric for 24 hours. He had kidney failure at 76 mg/l of serum creatinine, urea at 3.34 g/l, a hyper eosinophilia at 720 e/mm3. Allopurinol was stopped; he was put on antihistamines, corticosteroids and hyperhydration. He received a total of three hemodialysis sessions. The evolution was marked by the aggravation of the dermatological lesions with improvement of the renal function. The evolution was marked by the aggravation of skin lesions with improved renal function, but he died seven days after admission in an array of multiple organ failure. Case Report-2: S.O 74 years old, followed for CKD stage-IV for three years, turning on allopurinol 300 mg/ day for five weeks for asymptomatic hyperurcemia, admitted to Stevens Johnson syndrome. She was conscious, feverish at 38 °C, hemodynamically stable, she had bilateral conjunctivitis, cheilitis, and macular lesions with early skin peeling and preserved diuresis. She had kidney failure at 66 mg/L of serum creatinine, urea 2.21 g/l. Allopurinol was arrested and she was put on antihistamines and corticosteroids and hyperhydaration. The evolution was marked by the return of the glomerular filtration rate to baseline value after 15 days. Discussion: Allopurinol is the first xanthine oxidase inhibitor to be marketed more than 40 years ago. It constitutes the reference treatment of symptomatic hyperuricemia. Although it is generally well tolerated, the severe toxidermias associated with it constitute a major risk when it is used. Thus, allopurinol is currently the leading cause of severe bullous toxidermia including Lyell and Stevens-Johnson syndromes in Europe and one of the first providers of Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) in the world. The action is due to a hypersensitivity reaction type-III probably triggered by the Oxipurinol, main metabolite of allopurinol. The clearance of Oxipurinol depends mainly on renal function, which explains the high frequency in patients with CKD. Conclusion: These cases illustrate the significant morbidity associated with the irrational use of allopurinol in patients with chronic kidney disease.

Biography:

Sanae Ezzaki has completed her Graduation from the University of Medicine and Pharmacy of Rabat, Morocco. She is currently Resident in Nephrology Hemodialysis at the Ibn Rochd University and Hospital Center in Casablanca, Morocco.