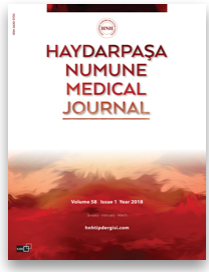
HAYDARPAŞA NUMUNE MEDICAL JOURNAL

DOI: 10.14744/hnhj.2018.82787
Haydarpasa Numune Med J 2019;59(2):186–187



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CASE REPORT

Acute Renal Injury with a Single Oral Dose of Valacyclovir: A Case Report

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Abstract

We report an unusual case of valacylovir-induced nephrotoxicity. The study presents the case of a 38-year-old female patient who developed acute renal injury following treatment with a single oral dose of valacyclovir (2000 mg). Although nephrotoxicity due to rapid intravenous infusion of valacyclovir at high doses is not a rare side effect, oral single doses associated acute renal injury is an uncommon complication. Valacylovir is a commonly used antiviral agent; therefore, physicians should be aware of this rare but potentially serious adverse drug reaction.

Keywords: Acute renal injury; drug-induced nephrotoxicity; valacyclovir.

Drug-induced nephrotoxicity is a common problem in diagnostic and therapeutic medicine. Drugs may cause approximately 20% of community and hospital-acquired episodes of acute kidney injury (AKI) [1].

Valacyclovir hydrochloride is a widely used antiviral agent in the clinical treatment of herpes simplex. After oral administration, it is nearly completely converted to acyclovir. AKI resulting from the accumulation of acyclovir crystals in the kidney has been observed following rapid intravenous infusion of large doses of acyclovir, but it is uncommon and usually reversible. Oral acyclovir therapy is rarely associated with nephrotoxocity [2].

Herein, we have described the case of a 38-year-old female patient who developed acute renal injury following treatment with a single oral dose of valacyclovir (2000 mg).

Case Report

A 38-year-old woman with no significant past medical history was admitted to our emergency department for severe lower back pain and nausea. Her last medical check-up was performed 3 weeks ago, and blood chemistry parameters were within normal ranges. Treatment history disclosed that she had consumed 2 tablets of 1000 mg valacylovir 12 hours before administration for herpes labialis. There was no history of kidney disease, dehydration, and regular medication. She also denied using any complementary alternative medicine.

Physical examination revealed a body temperature of 37.2 °C, pulse of 110 times/min, respiration rate of 18 times/min, and blood pressure of 135/105 mmHg. No abnormality was noted in the heart, lung, or abdomen. Percussion pain was detected in the bilateral kidney. No mass or edema was detected in the bilateral lower extremities.

The patient was hemodynamically stable. Initial work-up was significant for elevated blood urea nitrogen (BUN) of 68 mg/dL and serum creatinine (S-Cr) of 1.65 mg/dL, with electrolytes within normal ranges (normal range for S-Cr

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Submitted Date (Başvuru Tarihi): 29.04.2018 **Accepted Date(Kabul Tarihi):** 19.06.2018

and BUN are 0.6–1.2 mg/dL and 7–18 mg/dL). Complete blood count was in normal limits. Daytime quantity of urine output was normal. Urinalysis revealed 1+ proteinuria. Urine-specific gravity was 1010, +1 of proteinuria with 2 erythrocytes, and 4 leucocytes/mm^3. No glucosuria was detected. Urine was negative for crystals, although polarizing light microscopy was unavailable to identify crystals adequately. Immunological tests were negative (for antinuclear, antineutrophilic cytoplasmic, and antiliver/kidney microsomal). Complement level and serum proteins electrophoresis were normal. Tests for human immunodeficiency and hepatitis B and C viruses were negative. Abdominal and renal ultrasonography revealed no hepatic and kidney abnormalities. Thoracic and abdominal computed tomography was normal.

Her S-Cr level increased gradually, reaching 2.5 mg/dL on day 3. She was diagnosed with acute renal injury and drug-induced nephropathy. The patient refused to undergo renal biopsy and was initiated on aggressive IV fluids. The patient's renal function began to improve toward baseline over the following week.

Discussion

Valacyclovir is a widely used antiviral agent in herpetic infections and is approved as two single-dose therapy (2 g twice daily for 1 day at a 12-hour interval) for the treatment of herpes labialis [3]. After its oral administration, it is nearly completely converted to acyclovir, metabolized by the liver, and excreted in urine (60%–90% as unchanged drug).

Usually, it is a well-tolerated drug and does not appear to cause serious reactions. The risk factors for acyclovir-induced AKI include the use of high-dose bolus injection, decreased blood volume, dehydration, elderly patients, high peak plasma levels of the agent, and pre-existing renal disease [2]. The use of acyclovir can damage the kidney via several mechanisms: dose-dependent cell membrane injury ending in cell necrosis, formed crystals occluding renal tubule, immune factors, and thrombotic microangiopathy [4]. To the best of our knowledge, several studies of valacyclovir–acyclovir-induced renal injury have been reported in the literature [5]; however, no study has reported in term of a single oral dose.

In the present report, the patient was a young female without any history of disease; therefore, this case has more important clinical significance to identify the possible side effects of valacyclovir. Her further evaluation for other causes of AKI was unrevealing, including a negative history for exposure to other nephrotoxic drugs prior to hospitalization. She had a severe lower back pain, and percussion pain was detected in the bilateral kidney. Although AKI is not commonly associated with pain, crystal nephropathy of valacylovir can cause nausea/vomiting and flank or abdominal pain. Moreover, it can be speculated that if the patient was not admitted to the emergency department for lower back pain, she would have not be diagnosed with acute renal failure and would consume the second dose of her treatment.

In summary, we report an unusual case of acute kidney failure caused by valacylovir. Valacylovir is a commonly used antiviral agent; therefore, physicians must be aware of this rare but potentially serious adverse drug reaction.

Informed Consent: Approval was obtained from the patients.
Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

Financial Disclosure: The authors declared that this study received no financial support.

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