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Neurotoxicity Induced by Acyclovir in a Hemodialysis Patient with Herpes Zoster: A Case Letter

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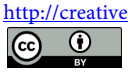

Abstract

Letter to Editor.

Subject Areas

Neurology, Nephrology

Keywords

Letter to Editor

To the Editor,

We report a case of a chronic hemodialysis patient with Herpes zoster (HZ) who had developed acyclovir neurotoxicity.

A 67-year-old woman with end-stage renal disease secondary to vascular nephropathy, who had been on hemodialysis for 10 years, presented to the hospital with a 1-day history of confusion. Her medical history included hypertension, controlled by renin-amgiotensin system inhibitor.

Three days prior, the patient had developed an erosive erythematous plaque surmounted by confluent bullous vesicles in the left eye. A diagnosis of ophthalmic herpes zoster was made by the ophthalmologist, who prescribed oral acyclovir 1600 mg daily; three days later, she suddenly complained of visual hallucinations and confusion. On physical examination, the patient's blood pressure was 140/70 mmHg with a heart rate of 85 beats per minute and oxygen saturation of 93% on room air.

On neurological examination, she was confused and disoriented. We noted no nuchal rigidity and no focal neurological deficits. The patient had an erythematous

erosive plaque surmounted by confluent vesicular bubbles giving way to yellowish and hemorrhagic crusts affecting the ophthalmic branch of the trigeminal nerve (V) of the left eye (Figure 1). Brain computed tomography revealed no specific findings except for left periorbital inflammatory thickening. Laboratory examination revealed normal blood sugar levels.

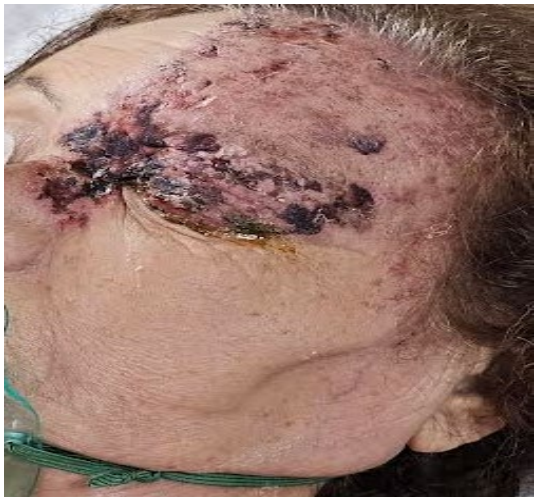

Figure 1. Image showing ophthalmic herpes zoster lesion.

Our primary consideration was viral encephalitis. However, the absence of fever, meningeal signs (nuchal rigidity), or symptoms (headache), despite the presence of confusion, made it less likely. A lumbar puncture was performed, and cerebrospinal fluid (CSF) analysis showed normal results. Microbiological studies for bacterial and viral encephalitis were negative, and polymerase chain reaction (PCR) testing of the patient's CSF returned negative for herpes simplex virus and varicella-zoster virus.

Our second consideration was acyclovir neurotoxicity. To address this, the acyclovir dose was adjusted, and the patient underwent daily dialysis. Despite the dose adaptation of acyclovir, the patient's neurological symptoms improved minimally. However, clinical symptoms completely resolved three days after intensifying the hemodialysis sessions. This, combined with the absence of fever and negative microbiological tests, strongly suggested a diagnosis of acyclovir neurotoxicity.

Acyclovir is highly effective and widely used in treating herpes zoster and herpes simplex. However, it is crucial for physicians to be aware of appropriate dosing to prevent serious adverse effects [1] [2]. Although acyclovir has a wide therapeutic index and is generally well-tolerated, adverse effects are not uncommon. Nephrotoxicity and neurotoxicity are the two most significant adverse effects of concern [3].

Neurotoxicity induced by acyclovir is more common in patients with kidney failure. Since acyclovir is primarily eliminated by the kidneys, its clearance rate can be significantly prolonged in renal failure. Therefore, dose adjustment should

be considered in hemodialysis patients [4].

For oral acyclovir, the manufacturer's technical sheet recommends a dose of 400 mg/day for dialysis patients. Hemodialysis can also serve as a diagnostic tool for acyclovir toxicity, as there will be no clinical response in cases of viral encephalitis [5].

In our case, the high doses of acyclovir administered caused neurotoxicity, with the simultaneous emergence of neurological symptoms. This highlights the importance of careful clinical monitoring in renal failure patients with HZ who are receiving acyclovir.

Conflicts of Interest

The authors declare no conflicts of interest.

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