pISSN 1738-6586 / eISSN 2005-5013 / J Clin Neurol 2021;17(1):140-141 / https://doi.org/10.3988/jcn.2021.17.1.140

Posterior Reversible Encephalopathy Syndrome in a Case of Vancomycin-Induced Acute Kidney Injury

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Dear Editor,

Posterior reversible encephalopathy syndrome (PRES) is a neurological disorder that typically presents with seizures, headache, visual disturbances, and altered mental status. PRES most often occurs in the setting of a hypertensive crisis or with cytotoxic immunosuppressive therapy. Impaired renal function has been observed in about half of PRES cases, but it is not known whether the impaired renal function itself or the accompanying arterial hypertension is the primary triggering factor for this condition.[1] Here we present a patient who developed PRES 12 days after experiencing acute kidney injury (AKI) secondary to vancomycin use.

A 32-year-old female with no previous history of hypertension or renal disease was initially admitted to another hospital. She had developed AKI with a serum creatinine level of 8.5 mg/dL after being treated with vancomycin for cellulitis of the right hip. Elevated blood pressure was not documented throughout this admission. She was discharged on day 9, at which time her creatinine level was 4 mg/dL. Three days following her discharge, the patient was admitted to our facility due to bilateral vision loss, altered mental status, and seizures. On admission, her creatinine level was 3.22 mg/dL and her blood pressure were in the ranges of 107−156/68−108 mm Hg. Brain magnetic resonance imaging (MRI) showed bilateral occipital and posterior parietal T2-weighted/FLAIR subcortical white-matter hyperintensities suggestive of PRES. She was treated with the antiepileptics (levetiracetam and fosphenytoin), and her blood pressure was controlled with a goal of less than 140/90 mm Hg. Her creatinine level continued to improve, reaching 2.49 mg/dL on day 6 of admission. Repeat MRI on day 7 of admission showed resolution of the T2-weighted/FLAIR hyperintensities in the previously involved areas. The patient's neurological symptoms resolved, and she was discharged after 13 days of hospitalization.

The pathophysiological mechanism of PRES is not yet fully understood. The most commonly accepted theories include a loss of autoregulation in the cerebral blood flow secondary to uncontrolled hypertension, and endothelial dysfunction due to chemotherapy or immunosuppressive medications. Impaired renal function is observed in about half of PRES cases, and arterial hypertension is the primary triggering factor for PRES in most of these cases.[1,2] Normal or slightly elevated blood pressure not exceeding the normal upper autoregulation limit has been observed in about 30% of PRES cases. Only three cases of AKI-related PRES without the degree of documented severe hypertension typically seen in PRES were found in a review of the literature (Table 1). The first case was a 17-year-old male who presented with typical clinical and radiological findings of PRES after developing exercise-induced AKI.[3] The second case was a 25-year-old male who reportedly experienced symptoms and typical MRI findings of PRES about 32 days after developing AKI in the setting of acute hepatitis A.[4] The third case was a 6-year-old female who experienced symptoms of PRES after developing hypovolemic AKI following a traumatic amputation.[5] These previous findings

Received July 21, 2020
Revised October 5, 2020
Accepted October 6, 2020

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Table 1. Comparison of cases of AKI-related PRES

Case	Age (years)/ sex	Features of AKI			Clinical and radiological presentations of PRES						
		Cause of AKI	Cr at time of AKI (mg/dL)	Time from AKI to PRES (days)	Seizures	HA	Visual disturbance	AMS	Radiological findings	BP range (mm Hg)	Time to resolution of PRES (clinical and/or radiological)
Present	33/female	Vancomycin	8.50	12	Yes	Yes	Bilateral vision loss	Yes	Bilateral occipital and posterior parietal T2-weighted/ FLAIR subcortical white-matter hyperintensities	107–156/ 68–108	6 days
Kimura et al.[3]	17/male	Strenuous exercise	9.00	5	Yes	Yes	Visual agnosia	Not mentioned	Increased signal intensity in the subcortical white matter predominantly in posterior and parietal lobes of cerebrum and in cerebellum	158–168/ 88–108	25 days
Kim et al.[4]	25/male	Acute hepatitis A	5.19	32	Yes	Yes	Yes	Yes	Hyperintense signal alterations in the bilateral subcortical regions of temporoparietal and occipital lobes	120–180/ 50–90	14 days
Aggarwal et al.[5]	6/female	Hypovolemia after trauma	1.70*	1	Yes	Not mentioned	Not mentioned	Yes	Noncontrast head CTrevealed bilateral multiple hypodense foci in white matter predominately in parieto-occipital lobes	150–160/ 90–100†	4 days

*Normal Cr range for a 6-year-old: 0.3–0.7 mg/dL, †Normal BP range for a 6-year-old: 97–115/57–76 mm Hg.
AKI: acute kidney injury, AMS: altered mental status, BP: blood pressure, Cr: creatinine, CT: computed tomography, HA: headache, PRES: posterior reversible encephalopathy syndrome.

combined with our case suggest that the occurrence of PRES in the setting of AKI without hypertension favors endothelial dysfunction as a cause of PRES in such conditions.

Several medications have been reported as predisposing factors for the development of PRES, most of which are immunosuppressive agents such as methotrexate and tacrolimus.[2] To our knowledge, the present patient is the first reported case of vancomycin-related PRES. Another intriguing aspect of our patient is that she had a delayed onset of PRES and developed symptoms 12 days after the initial onset of her AKI despite improvement in her renal function. A prompt diagnosis can result in a benign clinical course of PRES, and complete recovery of both symptoms and radiological findings can be expected, as seen in our case.

Author Contributions

Conceptualization: Laura J Wu. Data curation: Lily Kim, Leah Kolar. Formal analysis: Laura J Wu. Supervision: Laura J Wu. Writing—original draft: Lily Kim. Writing—review & editing: all authors.

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Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

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