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Efficacy of hemodialysis and plasma exchange for ceftriaxone-induced encephalopathy in an elderly patient with acute kidney injury due to rapidly progressive glomerulonephritis: A case report

Shohei Nagayama[1], Yuki Kawai[1], Ayana Kunii[1], Kenji Baba[1], Shiro Komiya[1], Kazuaki Matsumoto[2], Tamio Iwamoto[1], Kouichi Tamura[3]
[1]Department of Division of Nephrology and Hypertension, Saiseikai Yokohamashi Nanbu Hospital, Japan
[2]Department of Division of Pharmacodynamics, Keio University Faculty of Pharmacy, Japan
[3]Department of Department of Medical Science and Cardiorenal Medicine, Yokohama City University Graduate School of Medicine, Japan

Case Study : An 88-year-old woman was admitted to our hospital with a diagnosis of acute kidney injury (AKI, stage 3) due to rapidly progressive glomerulonephritis (RPGN) by anti-glomerular basement membrane (anti-GBM) disease. She was treated with emergent hemodialysis (HD), corticosteroids, plasma exchange (PE) and antibiotic therapy (CTRX 2g, every 24 hours). However, she showed a decreased level of consciousness and myoclonus on the fifth day of hospitalization. We suspected ceftriaxone-induced encephalopathy because there were no specific findings on head CT/MRI or blood tests, and electroencephalogram showed high-amplitude slow waves. We discontinued CTRX, and at a later date, the plasma CTRX concentration was found to be as high as 198.7 μg/mL. Although we anticipated PE to enhance CTRX removal due to its low volume of distribution and high protein-binding affinity rate, the plasma CTRX concentration didn't decrease after PE. In addition, while a temporary decrease in plasma CTRX concentration was observed after HD, effective drug elimination didn't occur on the next day due to redistribution from the tissue to the blood (rebound phenomenon). Hypoalbuminemia and kidney dysfunction due to RPGN in this patient might lead to a decrease in the protein-binding affinity of CTRX, resulting in unchanged plasma CTRX concentration after HD and PE. After discontinuation of CTRX, the patient's level of consciousness improved to the same level as on admission on the 13th day of hospitalization. As elderly patients with kidney dysfunction often have multiple risks for antibiotic-associated encephalopathy, ceftriaxone-induced encephalopathy is more likely to develop. In acute inflammatory diseases such as RPGN, drug removal may not be achieved as effectively as it should be. It is important that apheresis therapy take into account the background and pathology of each individual patient.

Figure1 Clinical course.png

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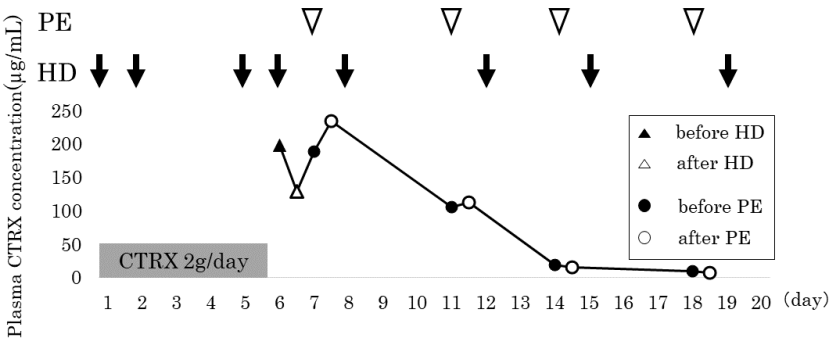

Figure1 Clinical course.png

Figure2 Electroencephalogram