■ S-517 ■

Successful management of cytomegalovirus-induced lower GI bleeding in renal transplant recipients

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CMV induced lower gastrointestinal (GI) bleeding in renal recipients is a frequently observed complication and its incidence is estimated as 0.9%. This opportunistic infection is clinically important in points that it comprised almost half of lower GI bleeding, and it showed high mortality rate up to 70%. Here, we report our experience of Cytomegalovirus-induced massive lower GI bleeding in renal transplant recipients. We retrospectively analyzed the medical records of 66 CMV-complicated patients who had undergone kidney transplantation at the Kangnam St.Mary's Hospital between January 1991 and April 2004. Of these, 3 patients who were diagnosed CMV induced lower GI bleeding cases were included. The diagnosis of CMV colitis was made by the following: i) clinical evidence of GI disease or bleeding and ii) documentation of CMV inclusion bodies or CMV DNA by in situ hybridization in a biopsy specimen of the lesion. Out of 66 patients who were diagnosed to have CMV infection, 3 patients (4.5%) were diagnosed to have CMV colitis. Chief complaints of these patients at the time of diagnosis were massive lower GI bleeding one month after transplantation. To control massive lower GI bleeding, all patients should be had transfusion more than 32 units on an average. Our cases showed positive signal for CMV using in situ hybridization but failed to demonstrate CMV viremia using CMV Ag assay. Our cases show successful management of local CMV infection with early withdrawal of immunosuppressants and ganciclovir treatment, dramatically. We can conclude that early colonoscopy is needed for transplant recipients with GI bleeding associated with anemia to confirm exact tissue diagnosis. But CMV viremia detected by CMV antigen assay is not effective in detecting local activation of CMV.

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A case of acute tubular necrosis presenting as sudden visual loss

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A 45-year-old alcoholic male presented with sudden visual loss at our hospital. He had been taking medications including NSAIDs (mefenamic acid) for the treatment of a common cold. His visual acuity determined by an ophthalmological examination was at the level of hand motion detection. The patient was oliguric, and his laboratory tests revealed an extremely high serum level of blood urea nitrogen and creatinine along with significantly increased anion gap metabolic acidosis. An ultrasound scan revealed moderately echogenic but normal-sized kidneys. Magnetic resonance imaging (MRI) revealed a vasogenic edema in the parietal-occipital regions of the brain. After two rounds of dialysis, his vision improved, and completely recovered after 10 days. A follow up brain MRI also revealed improvement. A kidney biopsy indicated severe acute tubular necrosis (ATN). Ten weeks after his hospital admission, his renal function was normal. Here, we report a patient with severe ATN that presented as sudden vision loss due to uremic posterior leukoencephalopathy syndrome (PLS).