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신이식 후 발생한 혈전성 미세혈관병증 환자에서 Calcineurin inhibitor을 sirolimus로 전환하여 호전된 사례

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Improvement of Post-Renal Transplantation Thrombotic Microangiopathy by Conversion from Calcineurin Inhibitor to Sirolimus

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Introduction: Among patients with end-stage renal disease owing to hemolytic uremic syndrome (HUS), 29.2% later had TMA. However, 0.8% of patients with ESRD owing to other causes had TMA. Post-transplant thrombotic microangiopathy (TMA) is a complication of solid organ transplantation, which remains difficult to treat. In post-transplant TMA immunosuppressants are usually implicated in causing or prolonging the disease. The calcineurin inhibitors (CNI), cyclosporin and tacrolimus are typical causative agents, but sirolimus (SIR) has also been associated with TMA. SIR, a novel macrocyclic lactone that inhibits the mammalian target of rapamycin (mTOR), is used in synergy with CNI as a potent immunosuppressive agent in solid-organ transplantation and in conversion from CNI to less nephrotoxic immunosuppressant. An increase in the occurrence of TMA is reported with the combined use of SIR and CNI in solid-organ transplants. However improvement of post-transplant TMA by convertsion from cancineurin inhibitors to sirolimus is also reported. We report a case of improvement of post-transplant TMA after conversion from tacrolimus to sirolimus.

Case: A 46-year-old woman with unknown cause ESRD underwent living related renal transplantation from her son. Surgery was uneventful, and she had a good urine output immediately after transplantation. Her renal function improved from pre-transplant serum creatinine 9.3 mg/dL to 0.68 mg/dL 1 week after transplantation. Immunosuppressants were cyclosporin A, mycophenolate and prednisolone. She complained of abdominal discomfort, diarrhea and serum level of creatinine increased to 2.99 mg/dL two week after transplantation. Acute humoral rejection was strongly suspected on transplanted kidney biopsy (C4d positive). Plasma exchange, intravenous immunoglobulin injection and rituximab were introduced to overcome acute humoral rejection. And cyclosporine A was replaced with tacrolimus. Serum level of creatinine decreased slowly after those treatments. TMA (many schistocytes on peripheral blood smear, hemoglobin 6.8 g/dL and platelet count 20,000/μL) appeared 1 week after conversion from cyclosporine A to tacrolimus. Plasma exchange was continued and fresh frozen plasma was transfused. And tacrolimus was discontinued and sirolimus was started. Her microangiopatic hemolytic anemia and thrombocytopenia recovered dramatically and serum creatinine level was also normalized two weeks after conversion from tacrolimus to sirolimus.

Key Words: 혈전성 미세혈관병증, Tacrolimus, Sirolimus
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