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A case of Sirolimus-induced De Novo Focal Segmental Glomerulosclerosis in Kidney Transplant Recipient

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INTRODUCTION: Sirolimus has been used as an alternative immunosuppressant for calcineurin inhibitors or antimetabolites in kidney transplant recipients (KTRs). However, sirolimus has been reported to cause several complications such as proteinuria, delayed wound healing, and hyperlipidemia. In previous study, we have reported a case of sirolimus-induced focal segmental glomerulonephritis (FSGS) in KTRs. Herein, we report the clinical course of sirolimus-induced FSGS after discontinuation of sirolimus.
PRESENTAION OF CASE: A 58-year-old male patient who underwent deceased donor kidney transplantation (KT) six years ago was admitted to investigate the cause of proteinuria and allograft dysfunction at 4 years after KT. The cause of end-stage renal disease was diabetes mellitus. He was diagnosed with laryngeal cancer at the first year after KT and mycophenolate mofetil (MMF) was converted to sirolimus. He had been cured after radiation therapy. At 3 years after the cure of cancer, proteinuria was detected and allograft function was deteriorated. He was diagnosed with FSGS by allograft kidney biopsy, which was determined by the impact of sirolimus. After confirming the cure of laryngeal cancer, sirolimus was converted to MMF. Although sirolimus was discontinued, the amount of proteinuria was increased and allograft function was deteriorated. He underwent allograft kidney biopsy, and the result showed the progression of previous lesions without rejection or other specific findings. Finally, he is on hemodialysis due to the progression of end-stage renal disease.

CONCLUSION: If proteinuria or tissue change such as FSGS by sirolimus occurs, irreversible deterioration will occur despite discontinuation of sirolimus. Therefore, we should pay attention to the use of sirolimus in KTRs as shown in this case.