# KSN 2016 Abstract Submission

*Acute Kidney Injury*

KSN2016ABS-1482

**Acute Kidney Injury after Intravenous methylprednisolone pulse therapy in idiopathic nephrotic syndrome in children**

Mi Young Kim* [1], Eujin Park[1], Yo Han Ahn[1], Hee Gyung Kang[1, 2], Il Soo Ha[1], Hae Il Cheong[1, 2]

[1]Pediatrics, Seoul National University Children's Hospital, [2]Research Coordination Center for Rare Diseases, Seoul National University Hospital, Seoul, Korea, Republic Of

**Background:** For steroid-resistant nephrotic syndrome, intravenous methylprednisolone pulse therapy is considered as a second line treatment. While this high-dose steroid therapy often effective, bradycardia, hypertension, or fluid retention may occur as infusion reaction. Rarely, transient deterioration of renal function has been reported to accompany methylprednisolone pulse therapy.

**Methods:** Here we report two pediatric cases of acute kidney injury (AKI) coinciding intravenous methylprednisolone therapy.

**Results:** Case 1) A 6 year-old boy with idiopathic nephrotic syndrome was transferred to our center due to relapse while on initial treatment of full dose oral steroid therapy for his first episode of the disease. Initially his proteinuria had disappeared after one week treatment of oral steroid treatment, however the proteinuira relapsed after 2 days. He did not response to subsequent oral steroid therapy anymore, so was treated by intravenous methylprednisolone therapy (30mg/kg per day, 3 consecutive days) at our center. His estimated GFR was declined from 195.22 to 28.93 without any other causes of renal dysfunction. Kidney biopsy revealed pathology of minimal change disease. After cessation of steroid pulse therapy, his renal function recovered gradually. However his proteinuria persisted, and now he is treated with calcineurin inhibitor Tacrolimus and Rituximab.

Case 2) A 14 year-old girl with minimal change nephrotic syndrome was transferred to our center due to subsequence steroid non-responsiveness. Before transfer, she had cerebral venous thrombosis complicating her nephrotic syndrome and had been treated with intravenous methylprednisolone therapy of 3 doses, every other day. On transfer her estimated GFR was deteriorated to 25.83 from about 100 at her first hospital; she had diarrhea for a few day with Norovirus infection. Kidney biopsy showed acute tubular injury. Her renal function recovered in three months, along with disappearance of proteinuria.

**Conclusion:** AKI is a known complication of nephrotic syndrome, considered to occur due to intravascular volume depletion along with severe edema (pre-renal), renal interstitial edema (renal), or other nephrotoxic agents. While diuretics or antibiotics are often cause AKI in nephrotic patients, above-reported cases indicate that methylprednisolone pulse therapy may also be associated with AKI. The pathogenesis of methylprednisolone pulse therapy associated AKI is yet to be elaborated; hemodynamic effect or nephrotoxicity

of this therapy should be considered. While methylprednisolone pulse therapy is often used as rescue therapy for nephrotic syndrome, monitoring of renal function is necessary.

**Keywords**: Acute kidney injury, methylprednisolone, nephrotic syndrome