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**References**

- Rosenmann E, Eliakim M. Nephrotic syndrome associated with amyloid-like glomerular deposits. *Nephron* 1977;18:301-8.
- Alpers CE, Rennke HG, Hopper Jr J, Biava CG. Fibrillary glomerulonephritis: An entity with unusual immunofluorescence features. *Kidney Int* 1987;31:781-9.
- Rosenstock JL, Markowitz GS, Valeri AM, Sacchi G, Appel GB, D'Agati VD. Fibrillary and immunotactoid glomerulonephritis: Distinct entities with different clinical and pathologic features. *Kidney Int* 2003;63:1450-61.
- Dasari S, Alexander MP, Vrana JA, Theis JD, Mills JR, Negron V, et al. DnaJ heat shock protein family B member 9 is a novel biomarker for fibrillary GN. *J Am Soc Nephrol* 2018;29:51-6.
- Nasr SH, Valeri AM, Cornell LD, Fidler ME, Sethi S, Leung N, et al. Fibrillary glomerulonephritis: A report of 66 cases from a single institution. *Clin J Am Soc Nephrol* 2011;6:775-84.
- Van Ginneken EE, Assmann KJ, Koolen MI, Jansen JL, Wetzels JF. Fibrillary-immunotactoid glomerulopathy with renal deposits of IgA lambda: A rare cause of glomerulonephritis. *Clin Nephrol* 1999;52:383-9.
- Kawashima M, Horita S, Nakayama H, Honda K, Uchida K, Nitta K, Yumura W, Nihei H. Immunoelectron microscopic analysis of intra-glomerular deposits in IgA-dominant immunotactoid glomerulopathy. *The Japanese journal of Clinical Pathology* 2002;50:1085-9.
- Moriyama T, Honda K, Tsukada M, Koike M, Itoh K, Nitta K, et al. A case of immunotactoid glomerulopathy with IgA2, kappa deposition ameliorated by steroid therapy. *Nihon Jinzo Gakkai Shi* 2003;45:449-56.
- Lodhi MU, Saleem TS, Usman MS, Chowdhury W, Kuzel AR, Iqbal HI, et al. Fibrillary glomerulonephritis in a patient with Sjogren's syndrome. *Cureus* 2018;10:e2483. doi: 10.7759/cureus. 2483.
- Yun YS, Song HC, Lee K, Choi EJ, Kim YS, Min JK, et al. Fibrillary glomerulonephritis in rheumatoid arthritis. *Nephrology* 2010; 15:266-7.
- Kim HJ, Kang SW, Park SJ, Kim TH, Kang MS, Kim YH. Fibrillary glomerulonephritis associated with Behçet's syndrome. *Ren Fail* 2012;34:637-9.
- Pronovost PH, Brady HR, Gunning ME, Espinoza O, Rennke HG. Clinical features, predictors of disease progression and results of renal transplantation in fibrillary/immunotactoid glomerulopathy. *Nephrol Dial Transplant* 1996;11:837-42.
- Collins M, Navaneethan SD, Chung M, Sloand J, Goldman B, Appel G, et al. Rituximab treatment of fibrillary glomerulonephritis. *Am J Kidney Dis* 2008;52:1158-62.
- Hogan J, Restivo M, Canetta PA, Herlitz LC, Radhakrishnan J, Appel GB, et al. Rituximab treatment for fibrillary glomerulonephritis. *Nephrol Dial Transplant* 2014;29:1925-31.
- Czarnecki PG, Lager DJ, Leung N, Dispenzieri A, Cosio FG, Fervenza FC. Long-term outcome of kidney transplantation in patients with fibrillary glomerulonephritis or monoclonal gammopathy with fibrillary deposits. *Kidney Int* 2009;75:420-7.
- El Ters M, Bobart SA, Cornell LD, Leung N, Bentall A, Sethi S, et al. Recurrence of DNAJB9-positive fibrillary glomerulonephritis after kidney transplantation: A case series. *Am J Kidney Dis* 2020;76:500-10.

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## Sapovirus-Associated Diarrhea in Renal Transplant Patient Treated Without Altering Immunosuppression

**Abstract**

Solid organ transplant (SOT) recipients are at increased risk of infective diarrheas. In such patients, diarrhea can be complicated by dehydration, leading to acute kidney injury or vascular thrombosis. Viral diarrhea in SOT is reported to be commonly due to cytomegalovirus and norovirus. As sapovirus is not routinely included in diagnostic evaluations, its epidemiology and natural history is not well documented. Anecdotal cases of sapovirus-associated diarrhea in renal transplant recipients have been treated with oral nitazoxanide, often with simultaneous reduction in immunosuppressants. We report sapovirus-associated diarrhea in a renal transplant recipient which responded well within two days to oral nitazoxanide. This, possibly, is the first such report from the Indian subcontinent of adequate control of sapovirus-associated diarrhea without any alteration in the immunosuppressant medications.

**Keywords:** Sapovirus, Diarrhea, Transplant recipients, Nitazoxanide, Immunosuppressant

**Introduction**

Chronic immunosuppression increases the risks of infectious diarrhea among solid organ transplant (SOT) recipients. Common viral etiologies include cytomegalovirus and norovirus. As more sensitive diagnostic tools become available, emerging causes of viral enteritis are being identified. We present a renal transplant recipient with sapovirus-associated diarrhea treated successfully using nitazoxanide.

**Case Report**

In September 2014, a 17-year-old girl with history of chronic kidney disease stage 5 secondary to biopsy-proven chronic tubulo-interstitial nephritis had received a live related renal transplant. She was on triple-drug immunosuppression and maintained stable serum creatinine levels at 1.2 mg/dL. In April 2023, at 26, she presented to our outpatient department with two weeks

of diarrhea. She was passing 6–8 stools daily and said it had started after consumption of around 100 mL of palm wine called 'kallu' or 'toddy' locally. Stools were watery, whitish to yellowish, and of medium volume. There was occasional abdominal cramping but no fever, nausea or anorexia. She was managed with loperamide and ricedotril without much relief. Probiotics were avoided to prevent superadded infections by *Lactobacilli*, *Bifidobacterium*, *Peptostreptococci*, etc.

In May 2023, she was admitted for closer monitoring and intravenous fluid management. At admission, her vitals were normal with no pallor, icterus, cyanosis, lymphadenopathy, or edema. She was clinically volume depleted. The serum creatinine was 1.9 mg/dL. Stool routine microscopy showed no parasitic ova or cysts and stool culture showed no pathogenic bacterial growth. The BioFire FilmArray™ gastrointestinal panel of polymerase chain reaction (PCR) tests was positive for sapovirus and Shiga toxin producing *E. coli* (STEC)/*E. coli* O157. Supportive care with intravenous fluids was continued and tablet nitazoxanide 500 mg twice daily was started. Within two days, the stool frequency reduced and serum creatinine level returned to 1.2 mg/dL. Nitazoxanide was continued for a total of seven days. Immunosuppression therapy was not altered.

## Discussion

Sapovirus, like norovirus, is a small, single-stranded RNA virus of the Caliciviridae family. While norovirus accounts for 90% of gastroenteritis outbreaks among adults worldwide, neither of the viruses is very well-studied in the immunocompromised population.<sup>1</sup>

We found only one multiplex PCR comparison of the infective etiology of diarrhea among renal transplant patients and non-transplant patients from the Indian subcontinent.<sup>2</sup> The commonest cause was bacterial infection. Individually, among transplant patients, norovirus was the most common organism isolated. In contrast, sapovirus was detected during only one of the

304 diarrhea-related hospitalizations (in both transplant and non-transplant groups) analyzed.

Though the presentation is very similar to norovirus infection, there are scanty reports of sapovirus-associated diarrhea in renal transplant.<sup>1–5</sup> One treated case of norovirus-associated diarrhea has also been reported where a superimposed sapovirus infection developed.<sup>6</sup>

Compared to bacterial and parasitic infections, norovirus/sapovirus infections are associated with greater weight loss, longer duration of symptoms, and more frequent need to reduce the dose of immunosuppressants.<sup>3</sup> This can potentially cause graft dysfunction and rejection. A large proportion of patients experience acute kidney injury. Some cases of biopsy-proven active graft rejection and oxalate nephropathy have also been reported. Viral shedding in stool, in some cases, lasted up to 581 days.<sup>3</sup>

Due to lack of specific therapies for STEC and viral diarrhea, management focuses on hydration and antimotility agents. In SOT patients, dose of immunosuppressants is often reduced on diagnosis of norovirus or sapovirus, in order to help control the infection and prevent superadded infections. A fine balance is needed between managing the infection and preventing graft dysfunction. Some researchers suggest “an alternative management plan with use of oral nitazoxanide”.<sup>1</sup>

Nitazoxanide, a thiazolide, has antibacterial, antiviral, and antiparasitic properties. Given orally, it inhibits viral replication in the intestinal mucosa probably by targeting cellular pathways in the synthesis of viral proteins.<sup>1</sup>

We had used the same dose as Ghussan and Vasquez<sup>1</sup> who demonstrated a negative sapovirus PCR in a repeat stool infectious panel one month after nitazoxanide therapy [Table 1]. In our patient, the diarrhea improved within two days—a day earlier than their patient. They had gradually tapered and discontinued the immunosuppressant mycophenolate mofetil and replaced it with azathioprine. We were able to safely and successfully control sapovirus-associated diarrhea with oral

**Table 1: Sapovirus diarrhea cases successfully treated with oral nitazoxanide**

Study	Age (y) & sex	Time since transplant	Signs, symptoms, & lab findings at admission	Treatment	Outcomes	Remarks
Ghussan & Vasquez <sup>1</sup>	30 F	10 y	4–5 stools/d; ↑ creatinine; 15 lb unintentional weight loss; ↓ Hb	IV fluids; antidiarrheals; NTZ 500 mg orally twice daily for 7d	Diarrhea improved within 3d of NTZ; Sapovirus -ve PCR in 1 mo after therapy	MMF dose ↓ & stopped - replaced with AZT;
Myat and May <sup>5</sup>	32 F	11 mo	Frequency of stool NR; ↑ creatinine	NTZ for 3d (dose NR); Supportive care NR	Resolution of diarrhea; Return to baseline creatinine; Rapid resolution of Sapovirus PCR (time to PCR negativity NR)	No reduction of immunosuppression; timeline of resolution of diarrhea NR; Time of repeat PCR is NR.
Present Case	26 F	9.5 y	6–8 stools/d; ↑ creatinine; No pallor	IV fluids; antidiarrheals; NTZ 500 mg orally twice daily for 7d	Diarrhea improved within 2d of NTZ	No reduction of immunosuppression

NTZ: Nitazoxanide; PCR: Polymerase chain reaction; MMF: Mycophenolate mofetil; AZT: Azathioprine; NR: Not reported; Hb: Hemoglobin; IV: Intravenous.

nitazoxanide without reducing the immunosuppression. There possibly is only one earlier report of sapovirus diarrhea successfully treated with nitazoxanide without reducing immunosuppression.<sup>5</sup> The chronic norovirus and subsequent sapovirus diarrhea reported by Wright *et al.*<sup>6</sup> required prolonged treatment with nitazoxanide and reduction of immunosuppression leading to graft rejection. That patient as well as the one managed by Ghusson and Vasquez,<sup>1,6</sup> had substantial unplanned weight loss before the PCR-based diagnosis.

Stool PCR, if available, should be done for all SOT patients presenting with diarrhea. Though it is more expensive than conventional tests and cannot differentiate active and asymptomatic carriers, in transplant patients where risks are higher, delays in diagnosis should be avoided. We also suggest adding nitazoxanide to the treatment regimen without reducing immunosuppression.

#### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

#### Conflicts of interest

There are no conflicts of interest.

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## References

1. Ghusson N, Vasquez G. Successfully treated norovirus- and sapovirus-associated diarrhea in three renal transplant patients. *Case Reports in Infectious Diseases* 2018;2018:e6846873.
2. Tiwari V, Anand Y, Gupta A, Divyaveer S, Bhargava V, Malik M, *et al.* Etiological spectrum of infective diarrhea in renal transplant patient by stool PCR: An Indian perspective. *Indian J Nephrol* 2021;31:245–53.
3. Roos-Weil D, Ambert-Balay K, Lanternier F, Mamzer-Bruneel MF, Nochy D, Pothier P, *et al.* Impact of norovirus/sapovirus-related diarrhea in renal transplant recipients hospitalized for diarrhea. *Transplantation* 2011;92:61–9.
4. Dargan A, Tofani C, Dhanekula RK, Quirk D. Chronic diarrhea secondary to human sapovirus in a renal transplant recipient. *Annals of Clinical and Laboratory Research* 4:1–2.
5. Myat L, May S. Treatment of sapovirus infection in a renal transplant patient with nitazoxanide. *Nephrology* 2020;25:40–82.
6. Wright S, Kleven D, Kapoor R, Kavuri S, Gani I. Recurring norovirus & sapovirus infection in a renal transplant patient. *IDCases* 2020;20:e00776.

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## NELL-1 as a Target Antigen in Asbestosis Associated Membranous Nephropathy — A Case Report

### Abstract

An 80-year-old male with a history of prolonged asbestos exposure presented with 24-hour urine protein of 8 gm, and serum albumin of 1.7 gm/dl. Renal biopsy disclosed features of membranous nephropathy. Immunohistochemistry showed positivity for neural epidermal-like growth factor-like 1 (NELL1) (2+/3+). Further assessment uncovered an incidental finding of asbestos-related pleural plaques and left hemithorax volume loss on computed tomography (CT) chest, leading to a diagnosis of asbestosis. This case highlights the rare association between asbestosis and NELL-1 positive membranous nephropathy.

**Keywords:** Membranous nephropathy, Asbestosis, NELL-1, Secondary membranous

### Introduction

NELL-1 positive membranous nephropathy has been seen various causes like malignancy, infections like hepatitis B, autoimmune disorders, indigenous medicines (containing mercury).<sup>1,2</sup> Exposure to various toxic environmental substances like asbestos, lead, mercury, have been linked with membranous nephropathy.<sup>3</sup> Here we describe NELL-1 positive membranous nephropathy in an individual with asbestosis.

### Case Report

An 80-year-old male presented with lower limb swelling and periorbital edema for 10 days. He worked as military personnel for 35–40 years; and he stayed in a house with asbestos roofing for over 30 years. On evaluation, he was found to have urine albumin 4+ without any active urinary sediments, a 24-hr urine protein 8 gm, serum albumin 1.7 gm/dl, total cholesterol 340 mg/dl, serum LDL 263 mg/dl, triglycerides 166 mg/dl, and serum creatinine 0.9 mg/dl. A