COVID-care facility set off apprehension and fear owing to an estranged environment and unfamiliar precautions. We deduced and shifted to the "buddy system"; 1 nephrology fellow and 1 dialysis technician were teamed up to cover COVID-care duties together. On 2 occasions, 1 of the buddies had presyncope and was immediately resituated by the other. This boosted their morale and helped them troubleshoot quickly.

To conclude, we highlight the challenges and their solutions while delivering point-of-care HD amidst the COVID-19 pandemic in a limited-resource setting. Despite limitations, prompt troubleshooting by a motivated team could provide the requisite dialytic facilities. In fact, these measures can be replicated in any far-flung area to provide urgent point-of-care hemodialysis, at times other than the COVID-19 pandemic.

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Letter Regarding "Fibrillary Glomerulonephritis Is Associated With HLA-DR7 and HLA-B35 Antigens"

To the Editor: We read with interest the research letter entitled "Fibrillary Glomerulonephritis Is Associated With HLA-DR7 and HLA-B35 Antigens," by Andeen *et al.*,[1] published on May 20, 2020. The letter reports the association between fibrillary glomerulonephritis (FGN) and specific human leukocyte antigens (HLAs), namely DR7 and B35, in a cohort of 26 patients from 3 institutions. The cases were composed of transplant recipients with FGN, de novo FGN in an allograft, and a donor with FGN.

We have recently reported on the long-term outcomes of our cohort of kidney transplant recipients with FGN[2] ($n = 14$) and have further identified 2 cases of donor-related FGN, totaling 16 cases of FGN with available HLA typing, including DQ data, which is detailed in Table 1. The most common class I and class II antigens in our cohort also included A2 (10/16, 62.5%), DR7 (8/16, 50%), and DQ2 (10/16, 62.5%). In addition, B7 (6/16, 37.5%) was frequently identified. The proportion of B35 was much lower in our cohort, 6.25% (1/16), and DR4 was moderately prevalent at 18.75% (3/16). Compared with the US white population,[3] the proportion of each of the A2 ($P = 0.41$), B7 ($P = 0.32$), DR7 ($P = 0.03$), and DQ2 ($P = 0.08$) alleles was higher in FGN; however, using the χ^2 test, a significant difference was only identified for DR7. In contrast, the proportion of B35 ($P = 0.26$) and DR4 ($P = 0.36$) was lower in FGN, although not statistically significant.

Similar to the previously studied cohort, 87% of subjects in our cohort with the DR7 antigen had DQ2 antigens (7/8) and of the DQ2 subjects, 70% (7/10) had DR7 antigens. Our data provide additional support for the association of DR7 but not B35 with FGN and support the hypothesis for possible underlying genetic cause, which will require further collaborative research studies.

Table 1. HLA typing of kidney transplant recipient with FGN and donor-related FGN

Patient	HLA-A		HLA-B		HLA-DR		HLA-DQ	
1	1	2	51	65	17	11	2	7
2	2	2	56	57	7	15	6	9
3	2	3	35	35	103	7	2	5
4	1	1	8	8	17	17	2	2
5	2	11	7	51	4	11	7	8
6	2	2	57	61	7	8	2	4
7	2	3	51	60	11	13	6	7
8	11	11	18	60	4	7	2	8
9	29	31	27	44	1	7	2	5
10	2	3	7	44	15	15	6	6
11	2	29	8	44	17	7	2	2
12	1	24	7	44	1	7	2	5
13	1	11	8	38	17	13	2	6
14	2	3	7	7	4	15	6	8
15[a]	1	3	7	13	7	15	2	6
16[a]	2	2	7	49	103	13	5	6

FGN, fibrillary glomerulonephritis; HLA, human leukocyte antigen
[a]Donor-related FGN.

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The Authors Reply: We reported a significant positive association of each of human leukocyte antigen (HLA)-DR7 and HLA-B35 with fibrillary glomerulonephritis (FGN) in a cohort of 26 patients with FGN.[1] We are pleased that El Ters *et al*.[2] have observed a similar association between HLA-DR7 and FGN in their cohort of 16 patients with native kidney failure due to FGN or donor-derived FGN. Together, these findings support a genetic component to this rare glomerulonephritis.

Unlike our study, HLA-B35 was not significantly associated with FGN in the Mayo Clinic cohort,[2] raising uncertainty about the significance of HLA-B35 association with FGN. HLA antigens have associations with race and infectious and autoimmune diseases, that may confound analyses of small cohorts. HLA-B35 is one of the largest allelic Class I molecules.[3] HLA-B35 appears to increase susceptibility to chronic hepatitis C virus infection in particular populations,[4] and hepatitis C infection is significantly associated with Black patients in the setting of FGN.[S1,S2] In small cohorts, differences in ethnicity and concurrent diseases may affect the ability to confirm genetic associations.

Larger, ethnicity-matched cohorts are needed to confirm the associations that have been described between HLAs and FGN. Importantly, future investigations may benefit from molecular typing for both HLA Class I and Class I, and from applying genome-wide association studies to more specifically decipher genetic susceptibility loci in patients with FGN beyond serologic typing of HLA antigens.

SUPPLEMENTARY MATERIAL

Supplementary File (PDF)

Supplementary References.

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