The 50 cases were retrieved from the Surgical Pathology database of Thomas Jefferson University Hospital. All biopsies were obtained for cause and examined by both light and electron microscopy (Figure 1). C4d staining was by immunofluorescence microscopy until 12/2008 and thereafter by immunohistochemistry. DSAs were detected by Luminex Lab Screen.

The mean age of the 50 patients was 50.5 years (Table 1). Eleven patients were retransplants. The mean donor age was 39.3 years (31 deceased and 17 living). DSAs were absent in 24 of the 50 patients and detected in 26. 23 patients were completely negative and 1 patient had a weak antibody titer of 1600 MFI. The mean time to biopsy in those with DSAs was 62 months and 74 months in those without. The mean creatinine (mg/dL) at biopsy in the DSA group was 3.0 ± 1.2 (SD) and 2.8 ± 1.4 in the negative group. The mean proteinuria at biopsy in the same groups was 1.7 g ± 1.6 and 3.6 ± 3.9, respectively. None of the DSA-negative patients experienced acute CMR, whereas 10 patients with DSAs did. Two of the 26 DSA-positive patients had acute AMR, whereas none of the DSA-negative patients did. None of the DSA-negative patients had C4d staining of PTC, whereas 7 of the DSA-positive patients did. Fifteen of the DSA-negative and 21 of the positive patients showed PTC basement membrane multilayering (Figure 2).

**CONCLUSIONS**

Half of our 50 patients with TG documented by EM had no DSAs or positive C4d staining in PTCs. Almost 70% of the patients evidenced PTC basement membrane multilayering. These patients were all diagnosed with chronic active CMR independently of the presence of TG. Cellular rejection mechanisms are likely the cause of the TG in this group. Patients with TG and DSAs are at greater risk for episodes of acute AMR and CMR. C4d staining of PTCs was evident in less than 40% and in the glomeruli in less than half. Interestingly, 25 of the 26 DSA+ patients were independently diagnosed with chronic active CMR.