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Letter to the Editor

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Letter to the Editor

Dear Sir,

The illustration on the cover of the November–December 2005 issue of *Ultrastructural Pathology* of thin basement membrane disease prompted the following recollections.

Before my retirement, the years ago, I had collected several cases of benign hematuria caused by thin basement membrane disease. Light microscopy showed normal glomeruli and immunofluorescence was negative. In addition to the thin glomerular basement membranes, I also observed the following changes.

The contour of the lamina rara interna was often undulating crating spaces (lacunae) bordered by the lamina densa and the endothelial cytoplasm, which sometimes formed protrusions (channels) into the vascular lumen. The lacunae contained flocculent material of nearly the same electron density as the lamina densa, which segmentally had a frayed appearance. Portions of red blood cells were occasionally noticed in the channels and lacunae and in the urinary space. Segmental swelling and obliteration of the foot processes of the epithelial cells were noted.

None of the ultrastructural changes described above are specific for thin basement membrane disease and can be found in other diseases with glomerular hematuria, such as IgA nehropathy, acute glomerulonephritis, and Alport syndrome.

Since glomerular hematuria is only caused by complete discontinuities of the glomerual basement membrane in crescentic clomerulonephritis, maybe the hematura in the other forms of glomerulonephropathy is caused by red blood cells entering through the endothelial channels, passing through the lacuna, squeezing through the lamina densa and out in between the foot processes of the epithelial cells without causing major damage to these structures.

Yours sincerely,

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