3-1-2012

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Recommended Citation
Yee J. The Renal Amalgam: Where Renal Pathology Fits. Advances in Chronic Kidney Disease 2012; 19(2):51-52.

ACKD
Advances in Chronic Kidney Disease

National Kidney Foundation

Vol 19, No 2, March 2012

EDITORIAL

The Renal Amalgam: Where Renal Pathology Fits

An amalgam is composed of metallic alloys that somehow by virtue of their individual properties fit together and become more than the sum of its constituent parts: this is the essence of nephrology. Recalling that the kidney performs a myriad of functions, it is only apropos that the discipline has required a coalition of several constituencies: electrolyticians, endocrinologists, dialyticians, hypertensionologists, and renal pathologists. This coalition, somewhat amorphous at first, congealed to become what we now acknowledge as the discipline of nephrology.

Renal pathologists themselves only coalesced as a club in November 1977, during an American Society of Nephrology meeting at the Washington Hilton hotel.[1] However, they had been around for years and may be traced back to at least 1914, the year of Theodor Volhard's seminal publication on glomerulonephritis, nephrosis, and hypertensive nephrosclerosis.[2] Subsequently, the club met at the annual meetings of the American Society of Nephrology, National Kidney Foundation, and the United States and Canadian Academy of Pathology under the leadership of Presidents Conrad Pirani and Robert Heptinstall. Club status was shed in March 1993 with rebirth as the Renal Pathology Society (RPS), and 2 of its more than 400 members—from more than 30 countries—serve as coast-to-coast Guest Editors of this issue of *Advances in Chronic Kidney Disease*: Cynthia Nast from the University of California, Los Angeles (RPS Past President, 2003) and Laura Barisoni from New York University. Long overdue and 16 RPS presidents later, this is the first time that this journal has been stewarded by direct purveyors of kidney pathology.

Renal pathologists have contributed mightily to this understanding and at all levels—clinically and pathophysiologically. The importance of an understanding of the fundamentals of kidney pathology to our discipline is underscored as an intrinsic requirement of any nephrology training program. Lamentably, it is still never enough. However, a solution has been provided, and mini renal pathology courses are provided at many of the top nephrology society meetings. They are wildly popular and perennially oversubscribed. There is no "standing room only."

In this issue, "The Role of Glomerular Disease in Progressive Renal Disease," the Guest Editors have compiled 8 articles that inform the reader on what is most poorly understood in nephrology, namely, glomerular disorders. Before 1950, our appreciation of glomerulopathies lay primarily within the advanced disease of autopsied specimens. Until today's routine technique of periodic acid-Schiff staining appeared, histological examination had been severely hampered.[3,4] Now, we can more fully correlate the differential contributions of the cells that are contained in the glomeruli to renal pathology. Phenotypical alterations correspond with biomolecular changes and are manifest as hematuria and proteinuria (see ACKD Volume 18, No. 4) and may be characterized, in part, by the urinary proteome (see ACKD Volume 17, No. 6).

Drs. Bomback and Markowitz initiate this series with a discussion of renal biopsy in elderly patients. The kidney biopsy, now routine, was once fraught with danger until proven safe and effective by Iversen and Brun, in their milestone publication of the aspiration technique during intravenous pyelography.[5] Notably, success was present in 42 of the original 66 patients. Now, much more safely done, renal biopsy should not be eschewed based on age alone.

When examining the causes of ESRD worldwide, it is frighteningly apparent that type 2 diabetes mellitus and hypertension do not always constitute approximately 70% of the causes of ESRD, as they do in the United States. Infection-related glomerular disorders play a distressingly prominent role in other parts of the world,

1548-5595/$36.00
doi:10.1053/j.ackd.2012.02.016

where diabetes is less of a problem, and Dr. Nast will expound on the topic of postinfectious glomerulonephritis.

The term "glomerulosclerosis" is often used by clinical nephrologists as a diagnosis rather than a morphological description. The reasons and correction of this error will be discussed by Dr. Barisoni, as she provides mechanistic insights into the role of the podocyte with regard to repair and progression of this descriptive lesion that often portends progression of CKD.

Drs. Vernon and Cook provide a salient discussion of complement. Activation of this primordial defense system is described as a host mediator of tubulointerstitial compartmental damage. Given that the tubulointerstitium is engaged in crosstalk with its upstream glomerular counterpart, complement-mediated inflammation here is a harbinger of progression. Vernon and Cook's discussion is complemented by the review of Dr. Satirapoj, which focuses on mechanisms through which glomerular damage initiates and perpetuates tubulointerstitial damage, rather than the converse situation, whereby an inflamed tubulointerstitium injures glomeruli.[6] Likely, both are true with conflation of injury, as the inverse and contrapositive circumstances clearly are observed microscopically.

The final 3 disease-specific articles review disorders for which there is a large amount of emerging knowledge. Dr. Cohen (RPS Past President, 1997) reviews the collagenofibrotic glomerulopathies as proteinuric disorders and their characterization by immunostaining of type III collagen and ultrastructural appearance. Drs. Haas and Reich cogently discuss the requirement for means other than clinical parameters to independently predict prognosis in IgA nephropathy. Here, renal biopsy may more greatly inform the clinician, and the utility of kidney biopsy scoring systems in IgA nephropathy is critically reviewed. Finally, exciting observations regarding the pathogenesis of membranous nephropathy and the prospect of a nonproteinuric method to monitor this in situ immune complex disorder are provided by Drs. Segal and Choi.

Clearly, nephrology represents the amalgam of multiple groups of differing expertise, all focused on the function and diseases of the kidney. Each constituent of the amalgam holds it together, including those properties of renal pathologists, who provide a light where clinicians cannot see.

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Editor

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