SZAKORVOSI TOVÁBBKÉPZÉS

CLINICAL NEPHROLOGY

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IRODALOM: 2005. JANUÁR 1. – JÚNIUS 30.

GYŐR, 2005. JÚLIUS 31.

C O N T E N T S

Part One

SECTIONS

TITLE OF PUBLICATIONS – AUTHORS – PUBLICATIONS

Part One

I. EPIDEMIOLOGY

1. Chronic kidney disease: the global challenge

A Meguid El Nahas, Aminu K Bello

Lancet 2005; 365: 331-40.

2. Epidemiology and risk factors for chronic kidney disease

William M McClellan

Med Clin N Am 89 (2005) 419-445.

3. Glomerulonephritis

S J Chadban, R C Atkins

Lancet 2005; 365: 1797-806.

4. Increasing incidence of focal segmental glomerulosclerosis and examination of demografic patterns

Dragovic D, Rosenstock J L, Wahl S J, Panagopoulos G, DeVita M V, Michelis M F

Clin Nephrol 2005 63 (1): 1-7.

5. Role of urinary screening programmes in children in the prevention of chronic kidney disease

Yap H K, Quek C M, Shen Q, Joshi V, Chia K S

Ann Acad Med Singapore 2005 34 (1): 3-7.

6. The investigation of hematuria

Kinkaid-Smith P, Fairley K

Semin Nephrol 2005 25 (3): 127-35.

7. Hematuria in adolescents

Gordon C, Stapleton F B

Adolesc Med Clin 2005 16 (1): 229-39.

8. Proteinuria screening for children

Murakami M, Hayakawa M, Yanagihara T, Hukunaga Y

Kidney Int Suppl 2005 (94): S23-7.

9. Renal manifestations of sexually transmitted disease: sexually transmitted diseases and the kidney

Abitbol C L, Friedman L B, Zilleruelo G

Adolesc Med Clin 2005 16 (1): 45-65.

10. Hypocomplementemia and membranoproliferative glomerulonephritis in children

Itaka K, Nakamura S, Moriya S, Koshino H, Iwanami N, Sakai T, Motoyama O

Clin Exp Nephrol 2005 9 (1): 31-3.

11. Prevention of the progression of chronic kidney disease: practice in China

Wang H, Zhang L, Lv J

Kidney Int Suppl 2005 (94): S63-7.

12. Lupus nephritis in children in Malaysia

Khoo J J, Pee S, Tevarajah B, Yap Y C, Chin C K

J Pediatr Child Health 2005 41 (1-2): 31-5.

13. Cardiovascular risk factors in hemodialysis patients: results from baseline data of kaleidoscopic approaches to patients with end-stage renal disease study

Ohsawa M, Kato K, Itai K, Onoda T, Konda R, Fujioka T, Nakamura M, Okayama A

J Epidemiol 2005 15 (3): 96-105.

14. Five-year follow-up of patients with epidemic glomerulonephritis due to Streptococcus zooepidemicus

Sesso R, Pinto S W

Nephrol Dial Transplant 2005 May 26 [Epub ahead of print]

15. HCV associated glomerulopathy in Egyptian patients: clinicopathological analysis

Sabry A, E-Agroudy A, Sheashaa H, El-Husseini A, Mohamed Taha N, Elbaz M, Sobh M

Virology 2005 30; 334 (1): 10-6.

II. ETIOLOGY

1. Existence and significance of hepatitis B virus DNA in kidneys of IgA nephropathy

Wang N S, Wu Z L, Zhang Y E, Liao L T

World J Gastroenterol 2005 7; (5): 712-6.

2. HIV-associated immune complex glomerulonephritis with „lupus-like" features: a clinicopathologic study of 14 cases

Haas M, Kaul S, Eustace J A

Kidney Int 2005 67(4): 1381-90.

3. Glomerulonephritis in a patient with chronic active Epstein-Barr virus infection

Kano K, Yamada Y, Sato Y, Arisaka O, Ono Y, Ueda Y

Pediatr Nephrol 2005 20 (1): 89-92.

4. A case of necrotizing glomerulonephritis presenting with nephrotic syndrome associated with pulmonary cryptococcosis

Nakayama M, Hori K, Ishida I, Masutani K, Katafuchi R

Clin Exp Nephrol 2005 (1): 74-8.

5. A case of renal sarcoidosis with complement activation via the lectin pathway

Hagivara S, Ohi H, Eishi Y, Kodama F, Tashiro K, Makita Y, Suzuki Y, Maeda K, Fukui M, Horikoshi S, Tomino Y

Am J Kidney Dis 2005 45 (3): 580-7.

6. Henoch-Schönlein purpura associated with esophagus carcinoma and adenocarcinoma of the lung

Weiler-Bisig D, Ettlin G, Brink T, Arnold W, Glatz-Krieger K, Fischer A

Clin Nephrol 2005 63 (4): 302-304.

7. Light chain muscle deposition caused rhabdomyolysis and acute renal failure in patients with multiple myeloma

Farah R, Farah R, Kolin M, Cohen H, Kristal B

Clin Nephrol 2005 63(1): 50-53.

8. Psoriatic nephropathy – does an entity exist?

Singh N P, Prakash A, Kubba S, Ganguli A, Singh A K, Sikdar S, Agarwal S K, Dinda A K, Grover C

Ren Fail 2005 27 (1): 123-7.

9. Neutrophilic dermatosis associated with propylthiouracil-induced p-ANCA (p-antineutrophil cytoplasmic antibodies)

Boulenger-Vazel A, Kupfer-Bessaquet I, Gouedard C, Leberre R, Leroy J P, Sonnet E, Desvignes O, Misery L, Sassolas B

Ann Dermatol Venereol 2005 132 (1): 27-31.

10. Epitope analysis of myeloperoxidase-specific antineutrophil cytoplasmic autoantibodies (MPO-ANCA) in childhood onset Graves' disease treated with propylthiouracil

Fujieda M, Suzuki K, Sato H, Hattori M, Wada N, Tsuchiya M, Okamoto N, Murata T, Matsudaira M, Shimizu M, Ohta K, Naruse K, Sugihara S, Wakiguchi H

Clin Nephrol 2005 63 (6): 437-45.

11. Necrotizimg glomerulonephritis and pulmonary hemorrhage associated with carbimazole therapy

Calanas-Continente A, Espinosa M, Manzano-Garcia G, Santamaria R, Lopet-Rubio F, Aljama P

Thyroid 2005 15 (3): 286-8.

12. IgA nephropathy in a young man with primary hyperparathyroidism

Jochum E, Brandenburg V M, Brodersen H P, Janssen U

Clin Nephrol 2005 63 (1): 46-9.

13. Acute renal failure due to mesangial proliferative glomerulonephritis in a pregnant woman with primary Sjogren's syndrome

Adam F U, Torun D, Bolat F, Zumrutdal A, Sezer S, Ozdemir F N

Clin Rheumatol 2005 May 26; [Epub ahead of print]

14. Membranous nephropathy associated with familial chronic ulcerative colitis in a 12-year-old girl

Ridder R M, Kreth H W, Grone H J, Gordjani N

Pediatr Nephrol 2005 Jun 22; [Epub ahead of print]

15. Renal involvement in patients with polymyositis and dermatomyositis

Yen T H, Lai P C, Chen C C, Hsueh S, Huang J Y

Int J Clin Pract 2005 59 (2): 188-93.

16. Nephrotic syndrome after stem cell transplantation

Stevenson W S, Nankivell B J, Hertzberg M S

Clin Transplant 2005 19 (1): 141-4.

17. Development of glomerulonephritis during anti-TNF-{alpha} therapy for rheumatoid arthritis

Stokes M B, Foster K, Markowitz G S, Ebrahimi F, Hiness W, Kaufman D, Moore B, Wolde D, D'Agati V D

Nephrol Dial Transplant 2005 20 (7): 1400-6.

18. Case of inflammatory vasculopathy and encephalopathy caused by treatment with tacrolimus

Ringelstein A, Bongs K, Sorge-Hadicke B, Berlit P

Nervenarzt 2005 76 (4): 475-8.

19. Cyclooxygenase-2 inhibitor-associated minimal-change disease

Almanson M, Kovithavongs T, Quarni M U

Clin Nephrol 2005 63 (5): 381-384.

20. COX-2 inhibitor induced anuric renal failure in a previously healthy young woman

Mühlfeld A S, Floege J

Clin Nephrol 2005 63 (3): 221-224.

21. COX-2 inhibitors and acute interstitial nephritis: case report and review of the literature

Esteve J-B, Launay-Vacher V, Brocheriou I, Grimaldi A, Izzedine H

Clin Nephrol 2005 63 (5): 385-389

22. Acute renal failure

Lameire N, Van Biesen W, Vanholder R

Lancet 2005 365: 417-30.

III. PATHOGENESIS

1. Glomerulonephritis

Chadban S J, Atkins R C

Lancet 2005 365: 1797-806.

2. Genetics of common progressive renal disease

Kai Ming Chow, Teresa Yuk Hwa Wong, Philip Kam-Tao Li

Kidney Int Suppl 2005 94: S41-5.

3. Complement and glomerulonephritis: new insight

Daniel Turnberg, H. Terence Cook

Curr Opin Nephrol Hypertens 2005 17: 223-228.

4. Ageing as a determinant of renal and vascular disease: role of endothelial factors

Matthias Barton

Nephrol Dial Transplant 2005 20: 485-490.

5. Apolipoprotein E and progression of chronic kidney disease

Charles C. Hsu, W.H. Linda Kao, Josef Coresh, James S. Pankow, Jane Marsh-Manzi, Eric Boerwinkle, Molly S. Bray

JAMA 2005 293 (23): 2892-2899.

6. The link between circulating markers of endothelial function and proteinuria in patients with primary glomerulonephritis

B. Mackinnon, C.J. Deighan, J. Norrie, J.M. Boulton-Jones, N. Sattar, J G Fox

Clin Nephrol 2005 63 (3): 173-180.

7. Regression of glomerulosclerosis with high-dose angiotensin inhibition is linked to decreased plasminogen activator inhibitor-1

Li-Jun Ma, Shinya Nakamura, Jean Claude Aldigier, Michele Rossini, Haichun Yang, Xiubin Liang, Ikuko Nakamura, Carmelita Marcantoni, Agnes B Fogo

J Am Soc Nephrol 2005 16: 966-976.

8. Expression and activation of STAT3 in chronic proliferative immune complex glomerulonephritis and the effect of fosinopril

Wuxing Zhang, Xiangmei Chen, Souzhu Shi, Ribao Wei, Jianzhong Wang, Nobuaki Yamanaka, Quan Hong

Nephrol Dial Transplant 2005 20: 892-901.

9. Apoptosis and proliferation in childhood acute proliferative glomerulonephritis

Ozaltin F, Besbas N, Bakkaloglu A, Gucer S, Topaloglu R, Ozen S, Kale G, Caglar M

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10. Glomerular plasmin-like activity in relation to nephritis-associated plasmin receptor in acute poststreptococcal glomerulonephritis

Oda T, Yamakami K, Omasu F, Suzuki S, Miura S, Sugisaki T, Yoshizawa N

J Am Soc Nephrol 2005 16 (1): 247-54.

11. Fibrillary and immunotactoid glomerulonephritis: report of a case and review of the literature

Vera Mendez F J, Molina Nunez M, Hernandez Garcia M A, Garcia Solano J

An Med Interna 2005 22 (1): 35-8.

12. HCV associated glomerulopathy in Egyptian patients: clinicopathological analysis

Sabry A, E-Agroudy A, Sheashas H, El-Husseini A, Mohamed Taha N, Elbaz M, Sobh M

Virology 2005 334 (1): 10-6.

13. Vascular endothelial growth factor (VEGF-C1)-dependent inflammatory response of podocytes in nephrotic syndrome glomerulopathies in children: an immunohistochemical approach.

Ostalska-Nowicka D, Zachwieja J, Nowicki M, Kaczmarek E, Siwinska A, Witt M

Histopathology 2005 46 (2): 176-83.

14. Expression of Fas, Bcl-2 and p53 molecules in glomerulonephritis and their correlation with clinical and laboratory findings

Uguz A, Gonlusen G, Ergin M, Tuncer I

Nephrology (Carlton) 2005 10 (3): 311-6.

15. Immunohistochemical detection of immunoglobulins and complements in formaldehyde-fixed and paraffin-embended renal biopsy tissue: an adjunct for diagnosis of glomerulonephritis

Chowdhury A R, Ehara T, Higuchi M, Hora K, Shigematsu H

Nephrology (Carlton) 2005 10 (3): 298-304.

16. Hepatitis C virus RNA and core protein in kidney glomerular and tubular structures isolated with laser capture microdissection

Sansonno D, Lauletta G, Montrone M, Grandaliano G, Schena F P, Dammacco F

Clin Exp Immunol 2005 140(3): 498-506.

17. Relative interstitial volume is correlated with renal function even in non-representative biopsy

Okon K, Szumera A, Kuziewski M, Smolenski O

Pol J Pathol 2005 56 (1): 9-13.

18. Study of the biopsied nephrotic syndrome for 20 years in the Cadiz Bay Area: histological correspondence, renal prognosis and clinical prognostic factors

Quiros P L, Ceballos M, Remon C, Lozano A, del Castillo R, Aznar E, Perez-Ruilopez M A, Rivero M, E

Nefrologia 2005 25 (2): 147-54.

19. Immunotactoid glomerulopathy with microtubular deposits, with reference to the characteristics of Japanese cases

Fukuda M, Morozumi K, Oikawa T, Motokawa M, Usami T, Yoshida A, Kimura G

Clin Nephrol 2005 63 (5): 368-74.

20. Renal effects of non-steroidal anti-inflammatory drugs and selective cyclooxygenase-2 inhibitors

H. F. Cheng, R. C. Harris

Current Pharmaceutical Design 2005 11 000-000

21. Thrombotic thrombocytopenic purpura associated with rapidly progressive lupus nephritis: report of two cases

S. Kapoulas, S. Liakos, G. Karkavelas, M. Ageloudi, D. Grekas

Clin Nephrol 2005 63 (4): 297-301.

22. Association between polymorphisms of the renin-angiotensin system and more severe histological forms of lupus nephritis

S.R.S. Sprovieri, Y.A. Sens, D. Martini Filho

Clin Nephrol 2005 64 (1): 20-27.

23. Inhibition of protein kinase CK2 prevents the progression of glomerulonephritis

Yamada M, Katsuma S, Adachi T, Hirasawa A, Shiojima S, Kadowaki T, Okuno Y, Koshimizu T A, Fujii S, Sekiya T, Miyamoto Y, Tamura M, Yumura W, Nihel H, Kobayashi M, Tsujimoto G

Proc Natl Acad Sci USA 2005 102 (21): 7736-41.

24. Lupus erythematosus proliferative glomerulonephritis in fetus

Daikha-Dahmane F, Bault J P, Molina-Gomes D, Hillion D, Ville Y

Lupus 2005 14 (4): 326-7.

25. HIV-associated immune complex glomerulonephritis with „lupus-like" features: a clinicopathological study 14 cases

Haas M, Kaul S, Eustace J A

Kidney Int 2005 67 (4): 1381-90.

26. Apoptosis and proliferating cell nuclear antigen in lupus nephritis (class IV) and membranoproliferative glomerulonephritis

Kirim S, Tamer T, Saime P, Gonlusen G

Ren Fail 2005 27 (1): 107-13.

27. Quantitative morphometry of lupus nephritis: the significance of collagen, tubular space, and inflammatory infiltrate

Hunter M G, Hurwitz S, Bellamy C O, Duffied J S

Kidney Int 2005 67 (1): 94-102.

28. Glomerular podocytopathy in patients with systemic lupus erythematosus

Kraft S W, Schwartz M M, Korbet S M, Lewis E J

J Am Soc Nephrol 2005 16 (1): 175-9.

29. Kidney disease associated with primary antiphospholipid syndrome: clinical signs and histopathological features in an experience of five cases

Saracino A, Ramunni A, Pannarale G. Coratelli P

Clin Nephrol 2005 63 (6): 471-476.

30. Histopathological atlas of renal disesases: ANCA-associated vasculitis (First part)

Ferrario F, Rastaldi M P

J Nephrol 2005 18 (2): 113-6.

31. Antineutrophil cytoplasmic antibodies (ANCA) in Chinese patients with anti-GBM crescentic glomerulonephritis

Yang G, Tang Z, Chen Y, Zeng C, Chen H, Liu Z, Li L

Clin Nephrol 2005 63 (6): 423-428.

32. Pathogenesis of Wegener's granulomatosis: concepts

Sarraf P, Sneller M C

Expert Rev Mol Med 2005 7 (8): 1-19.

33. Complex genetics of Wegener granulomatosis

Jagiello P, Gross W L, Epplen J T

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34. Manifestations mimicking relapsing polychondritis in a patient with microscopic polyangiitis

Ogawa H, Nishi E, Amano K, Takeuchi T

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35. Activation of the lectin complement pathway in Henoch-Schönlein purpura nephritis

Hisano S, Matsushita M, Fujita T, Iwasaki H

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36. A case of renal sarcoidosis with complement activation via the lectin pathway

Hagiwara S, Ohi H, Eishi Y, Kodama F, Tashiro K, Makita Y, Suzuki Y, Maeda K, Fukui M, Horikoshi S, Tomino Y

Am J Kidney Dis 2005 45 (3): 580-7.

37. Recurrent Goodpasture's disease secondary to a monoclonal IgA1-kappa antibody autoreactive with the alpha1/alpha2 chains of type IV collagen

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Am J Kidney Dis 2005 45 (2): 397-406.

38. Detection of mutations in the COL4A5 gene by analyzing cDNA of skin fibroblasts.

Wang F, Wang Y, Ding J, Yang j

Kidney Int 2005 67 (4): 1268-74.

39. Thin basement membrane nephropathy in pregnancy

Packham D

Semin Nephrol 2005 25 (3): 180-3.

40. Thin basement membrane nephropathy associated with other glomerular diseases

Norby S M, Cosio F G
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41. The risk of thin basement membrane nephropathy

Tonna S, Wang Y Y, MacGregor D, Sinclair R, Martinello P, Power D, Savige J

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42. Histopathological atlas of renal disease: minimal change disease and focal glomerulosclerosis

Ferrario F, Rastaldi M P

J Nephrol 2005 18 (1): 1-.

43. Glomerulonephritis in a patient with chronic active Epstein-Barr virus infection

Kano K, Yamada Y, Sato Y, Arisaka O, Ono Y, Ueda Y

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44. Elevated macrophage migration inhibitory factor (MIF) leveles in the urine of patients glomerular sclerosis

Matsumoto K, Abe M, Satomura A, Fujita T

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46. Case of inflammatory vasculopathy and encephalopathy caused by treatment with tacrolimus

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47. Characteristics and risk factors of intrarenal arterial leions in patients with IgA nephropathy

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J Lab Clin Med 2005 145 (5): 233-8.

50. Association of C-509T and T869C polymorphisms of transforming growth factor-beta gene with susceptibility to and progression of IgA nephropathy

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51. Urinary glycosaminoglycan composition in chronic glomerulonephritis

De Muro P, Faedda R, Satta A,

J Nephrol 2005 18: 154-160.

52. Tissue specific expressin of renin-angiotensin system components in IgA nephropathy

Miyake-Ogawa C, Miyazaki M, Abe K, Harada T, Ozono Y, Sakai H, Koji T

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53. Inhibition of protein kinase CK2 prevents the progression of glomerulonephritis

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Proc Natl Acad Sci USA 2005 102: 7736-41.

54. Role of coagulation factor Xa and protease-activated receptor 2 in human mesangial cell proliferation

Tanaka M, Arai H, Liu N, Nogaki F, Nomura K, Kasuno K, Olda E, Kita T, Ono T

Kidney Int 2005 67 (6): 2123-33.

55. The predictive value of peritubular capillaries C3d deposition in IgA glomerulonephritis

Gerghiceanu M, Penescu M, Mandache E

J Cell Mol Med 2005 9 (1): 143-52.

56. Genetics of common progressive renal disease

Chow K, M, Wong T Y, Li p K

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57. Mechanisms of tubulointerstitial injury in IgA nephropathy

Lai K N, Chan L Y, Leung J C

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58. Characteristics and risk factors of intrarenal arterial lesions in patients with IgA nephropathy

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Nephrol Dial Transplant 2005 20(4): 719-27.

59. Role of macromolecular IgA in IgA nephropathy

van der Boog P J, van Kooten C, de Fijter J W, Daha M R

Kidney Int 2005 67 (3): 813-21.

60. Activation of tubular epithelial cells by mesangial-derived TNF alpha: glomerular communication in IgA nephropathy

Chan L Y, Leung J C, Tsang A W, Tang S C, Lai K N

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63. Association of a single-nucleotide polymorphism in the immunoglobulin mu-binding protein 2 gene with immunoglobuln A nephropathy

Ohtsubo S, Iida A, Nitta K, Tanaka T, Yamada R, Ohnishi Y, Maeda S, Tsunoda T, Takei T, Obara W, Akiyama F, Ito K, Honda K, Uchida K, Tsuchiya K, Yumura W, Ujite T, Nagane Y, Miyano S, Y

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J Zhejiang Univ Sci B 2005 6 (1): 61-8.

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66. Urinary glycosaminoglycan composition in chronic glomerulonephritis

De Muro P, Faedda R, Satta A, Finetti D, Masala A, Cigni A, Sanna G M, Cherchi G M

J Nephrol 2005 18: 154-160.

67. Hypocomplementemia and membranoproliferative glomerulonephritis in children

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68. Myeloid related protein 8 expression on macrophages is a useful prognostic marker for renal dysfunction in children with MPGN type I

Kawasaki Y, Hosoya M, Takahashi A, Isome M, Tanji M, Suzuki H

Am J Kidney Dis 2005 45 (3): 510-8.

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Schena F P, Gesualdo L,

J Am Soc Nephrol 2005 16: S30-33.

70. Genetics of common progressive renal disease

Kai Ming Chow, Teresa Yuk Hwa Wong, Philip Kam-Tao Li

Kidney Int 2005 67 S94: S41-45.

71. Adiponectin level is reduced and inversely correlated with the degree of proteinuria in type 2 diabetic patients

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Clin Nephrol 2005 64 (1): 12-19.

72. Apolipoprotein E and progression of chronic kidney disease

Hsu C C, Kao W H L, Coresh J, Pankow J S, Marsh-Manzi J, Boerwinkle E, Bray M S

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73. Lower serum magnesium levels are associated with more rapid decline of renal function in patients with diabetes mellitus type 2

Pham P-C T, Pham P-M T, Pham P-A T, Pham S V, Pham H V, Miller J M, Yanagawa N, Pham P T

Clin Nephrol 2005 63 (6): 429-436.

74. Acute renal failure

Lameire N, Van Biesen W, Vanholder R

Lancet 2005 365: 417-30.

75. Acute renal failure in chronic kidney disease – clinical and pathological analysis of 104 cases

Zhang L, Wang M, Wang H

Clin Nephrol 63 (5): 346-350.

IV. CLINICAL PRESENTATION

1. Glomerulonephritis

Lau K K, Wyatt R J

Adolesc Med Clin 2005 16 (1): 67-85.

2. The investigation of hematuria

Kincaid-Smith P, Fairley K

Semin Nephrol 2005 25 (3): 127-35.

3. The clinical significance of asymptomatic gross and microscopic hematuria in children

Bergstein J, Leiser J, Andreoli S

Arch Pediatr Adolesc Med 2005 159 (4): 353-5.

4. Microscopic hematuria in pregnancy: relevance to pregnancy outcome

Brown M A, Holt J L, mangos G J, Murray N, Curtis J, Homer C

Am J Kidney Dis 2005 45 (4): 667-73.

5. Biochemical risk markers: a novel area for better prediction of renal risk?

Stuveling E M, Stephan J, Bakker L, Hillege H L, de Jong P E, Gans R O B, de Zeeuw D

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6. Assessment of renal function in recently admitted critically ill patients with normal serum creatinine

Hoste E A J, Damen J, Vanholder R C, Lameure N H, Delanghe J R,, Van den Hauwe K, Colardyn F A

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7. Study of the biopsied nephrotic syndrome for 20 years in the Cadiz Bay Area: histological correspondence, renal prognosis and clinical prognostic factors

Quiros P L, Ceballos M, Remon C, Lozano A, del Castillo R, Aznar E, Perez-Ruilopez M A, Rivero M, Fernandez Ruiz E

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Abitbol C L, Friedman L B, Zilleruelo G.

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clinicopathologic study of 14 cases

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Kano K, Yamada Y, Sato Y, Arisaka O, Ono Y, Ueda Y

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V. TREATMENT

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VI. TRANSPLANTATION

1. Are autoimmune diseases or glomerulonephritis affecting the development of panel-reactive antibodies in candidates for renal transplantation?

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C O N T E N T S

Part Two

SECTIONS

I. EPIDEMIOLOGY [EP]

II. ETIOLOGY [ET]

III. PATHOGENESIS [PG]

IV. CLINICAL PRESENTATION [CP]

V. TREATMENT [TR]

VI. TRANSPLANTATION [TP]

TITLE OF PUBLICATIONS – AUTHORS – SUMMARY OF PUBLICATIONS

Part Two

I. EPIDEMIOLOGY

1. Chronic kidney disease: the global challenge

El Nahas AM, Belko AK.

Lancet 2005; 365: 331-40.

The worldwide rise in the number of patients with chronic kidney disease (CKD) and consequent end-stage renal failure necessitiating renal replacement therapy is threatening to reach epidemic proportions over the next decade, and only a small number of countries have robust economies able to meet the challenges posed. A change in global approach to CKD from treatment of end-stage renal disease (ESRD) to much more agressive primary and secondary prevention is therefore imperative. In this seminar, we examine the epidemiology of CKD worldwide, with emphasis on early detection and prevention, and the feasibility of methods for detection and primary prevention of CKD. We also review the risk factors and markers of progressive CKD. We explore current understanding of the mechanisms underlying renal scarring leading to ESRD to inform on current and future interventions as well as evidence relating to interventions to slow the progression of CKD. Finally, we make strategic recommendations based on future research to stem the worldwide growth of CKD. Consideration is given to health economics. A global and concerted approach to CKD must be adopted in both more and less developed countries to avoid a major catastrophe.

2. Epidemiology and risk factors for chronic kidney disease

Wlliam M, McClellan

Med Clin N Am 2005; 89: 419-445.

Summary. Kidney disease is highly prevalent in the United States population and groups at high risk for increased prevalence of chronic kidney disease (CKD) include individuals with family history of end-stage renal disease (ESRD), diabetes, hypertension, and cardiovascular disease. Despite the increased risk of ESRD observed for blacks compared with whites, racial disparities in the prevalence of kidney disease have not been consistently demonstrated in the United States population. Although the reasons for this discrepancy in risk of ESRD and CKD have not been estabilished, clinicians should be aware that more rapid progression of CKD among blacks is a possible explanation for this observation and that closer monitoring and intensive care of risk factors associated with progressive renal injury is warranted for blacks with CKD and in other high-

risk groups. Therapeutic interventions that delay or prevent progressive kidney disease are well estabilished and incorporated into widely disseminated clinical practice guidelines. These interventions include agressive blood pressure control with agents that block the renin-angiotensin system, reduction of dietary protein to recommended levels for the American diet, weight loss, smoking cessation, and control of hyperlipidemia. These interventions also reduce the risk of cardiovascular disease and should be regarded as essential components of care of CKD. Achieving high levels of medically appropriate care of CKD patients and reduction in risk of progressin to ESRD may be delayed by barriers created by individual and regional poverty.

3. Glomerulonephritis

Chadban SJ, Atkins RC.

Lancet 2005; 365:1997-806.

Abstract. The term glomerulonephritis encompasses a range of immune-mediated disorders that cause inflammation within the glomerulus and other copartments of the kidney. Studies with animal models have shown the crucial interaction between bone-marrow-derived inflammatory cells and cells intrinsic to the kidney that is both fundamental and unique to the pathogenesis of glomerulonephritis, The mechanisms of interaction between these cells and the mediators of their coordinated response to inflammation are being elucidated. Despite these pathophysiological advances, treatment for glomerulonephritis remain non-specific, hazardous, and only partly successful. Glomerulonephritis therefore remains a common cause of end-stage kidney failure worldwide. Molecule-specific approaches offer hope for more effective and safer treatments in the future.

4. Increasing incidence of focal segmental glomerulosclerosis and an examination of demographic patterns

Dragovic D, Rosenstock JL, Wahl SJ, et al.

Clin Nephrol 2005; 63 (1): 1-7.

Abstract. Background: Idiopathic focal segmental glomerulosclerosis (FSGS) is one of the leading cuses of the nephrotic syndrome in adults and an important cause of end-stage renal disease. Its incidence has dramatically increased in the last two decades and it is especially prevalent among black patients. The trend of FSGS incidence has not been reported beyond 1997. Methods: We retrospectively reviewed all renal biopsies performed at our institution between 1986 and 2002 and identified patients with diagnoses consistent with primary glomerulopathy (PG), which included: minimal-change disease (MCD), idiopathic focal segmental glomerulosclerosis (FSGS), membranous nephropathy (MGN), IgA nephropathy (IgA), membranoproliferative glomerulonephritis (MPGN) and mesangioproliferative glomerulonephritis. Patients with possible secondary causes for their renal disease were excluded. Clincal data at the time of biopsy and

follow-up data were collected and analyzed. Results: During the period from January 1986 – December 2002, 299 renal biopsies were performed and 132 patients were diagnosed with PG. FSGS was the most common form of PG representing 37,8% of all PG followed by IgA 27,3%, MGN 16,6% and MCD 9,1%. Among FSGS patients 59% were females, 64% had nephrotic range proteinuria and 54% had the nephrotic syndrome. Mean serum creatinine was 2,0+/- 0,2 mg/dl and mean protein excretion was 6,1 +/- 1,0 g/day. The incidence of FSGS increased from 19,3% (1986-1991) and 16,6% (1992-1997) to 58,5% in the period from 1998-2002. The increase occured among black and Hispanic patients (33,3 – 79,2%) as well as white patients (12,5 – 5,15%). Black and Hispanic patients with PG presented for renal biopsy at a significantly younger age than white patients (p = 0,003), with mean age 37,5 +/- 2,0 vs 50,3 +/- 1,8 years. White FSGS patients were significantly older than white non-FSGS patients (mean age 56,4+/- 3,2 years vs. 48,0 +/- 2,0 years, p=0,03). Black and Hispanic FSGS patients were also older when compared to their non-FSGS counterparts (mean age 40,6 +/- 2,8 vs. 32,1 +/- 2,0 years, p = 0,04). When patients were stratified by age (< 45 years and ≥45 years), FSGS was the most common diagnosis in both groups among black and Hispanic patients (55,1% and 88,8%) but only among older white patients (36,2%). Conclusions: The incidence of FSGS as a proportion of PG in our population has increased markedly in the most recent time time period analyzed (1998 – 2002). The increase has occured among both white and black and Hispanic patients. We also found that FSGS was most prevalent in patients ≥45 years.

5. Role of urinary screening programmes in children in the prevention of chronic kidney disease

Yap HK, Quek CM, Shen Q, et al.

Ann Acad Med Singapore 2005; 34 (1): 3-7.

Abstract. Introduction: This article reviews published literature on the usefulness of population-based urinary screening in the Asian pediatric population. Methods: Articles were found in the Medline database using the key words „pediatrics", „urine screening", „hematuria" and „population". The Asian countries which had carried out population-based urinary screening of the pediatric population included Taiwan, Japan and Korea. One study on urinary screening in a select population in Malaysia. Preliminary results of the urinary screening of school children in a Singapore are presented and compared with the results found in the above-mentioned countries. Results: Overall, the proportion of children found to have urinary abnormalities ranged from less than 0,1% of the population screened to almost 50% of a select cohort referred from the srceening programmes for the evaluation of urinary abnormalities. In the pilot Singapore school screening programme, the prevalence of clinically significant proteinuria was 1,2 per 1000 children screened. Multivariate analysis showed that low body weight was associated with a 1,8-fold greater risk for proteinuria. The major cause of haematuria and proteinuria in those studies where renal biopsies were performed was glomerulonephritis. The Taiwanese experience also showed a reduction in the incidence of end-stage renal failure diagnosed in children after the onset of urine screening. Conclusion: These studies showed that urinary screening programmes in school children allow the early detection of disease. The cost-benefit ratio for specific populations should be determined before the implementation of such programmes.

6. The investigation of hematuria

Kincaid-Smith P, Fairley K.

Semin Nephrol 2005; 25 (3): 127-35.

Persistent microscopic hematuria is present in about 6% of the population, but probably only a small minority have hematuria that those not originate from the glomerulus. Careful analysis of phase-contrast urine microscopy by a skilled observer is critically important in the investigation of hematuria. In glomerular disease, urine microscopy often is second only to renal biopsy examination in helping make a diagnosis. Glomerular and nonglomerular hematuria are distinguished easily on phase-contrast urine microscopy or by an automated peripheral blood cell counter. However, urine microscopy provides additional information about casts and other features that may enable such disparate diagnosis as Fabry's disease, sickle cell disease, and cystine calculi to be made. Macroscopic nonglomerular hematuria id of particular significant because it is much more likely than microscopic hematuria to be associated with malignancy. Macroscopic hematuria originated from the glomerulus indicates the presence crescentic disease, which requires urgent assessment by renal biopsy examination. We advocate a renal biopsy examination in any individual with a persisting urinary eryrthrocyte count greater than 100,000/mL. Thirty percent of patients with isolated microscopic hematuria have mesangial immunoglobulin A glomerulonephritis (IgAN) shown on biopsy examination and 20% to 40% of these patients will progressed to renal failure without treatment.

7. Hematuria in adolescents

Gordon C, Stapleton FB.

Adolesc Med Clin 2005; 16 (1): 229-39.

Hematuria is not a rare finding during adolscence. The high prevalence of microscopic hematuria is not surprising when one consider the vast number of ways in which RBC can end up in the urine. The adolescent presenting with gross hematuria, proteinuria, or microscopic hematuria in combination with other symptoms of genitourinary disease is more likely to require a therapeutic intervention than is the individual found incidentally to have microscopic hematuria. Screening for hematuria is not supperted by current evidence. When it is discovered as the reasults of a screening examination, persistent microscopic hematuria in an otherwise asymptomatic individual may not require further investigation; however, the renal ultrasound examination has little risk and is helpful in diagnosing many of the conditions amenable to intervention. Serum studies offer little useful information in the evaluation of microscopic hematuria. Addressing isolated hematuria in a systemic, evidence-based fashion can help avoid untoward patients and parental worry and excessive heath care costs, without missing treatable or progressive disease entities.

8. Proteinuria screening for children

Murakami M, Hayakawa M, Yanagihara T, et al.

Kidney Int Suppl 2005; 94: S23-7.

Background: In Japan, urine screening are performed annualy at school for proteinuria and hematuria, but the effectiveness of this practice has not been clarified. Methods: urine screening at school was performed, and we investigated the prevalence of urine abnormalities and incidence and the causes of their diseases. Therefore, we studied effectiveness of the school-screening program. Results: The prevalence of urinary abnormalities was 0,52% amomg elementary school children and 0,75% among junior high school children. The school-screening program is effective in early detection of glomerulonephritis , so the number of new end-stage renal disease (ESRD) patients starting treatment has been changing. Discussion: The school-screening program is effective for early detection of glomerulonephritis. In case of generations who underwent the school-screening program, the age that one develops ESRD has been rising year by year, and the number of new ESRD patients starting treatment before 20 years old is lower in Japan than in America. Conclusion: The school-screening program in Japan represents a highly effective mass screening technique.

9. Renal manifestation of sexually transmitted disease: sexually transmitted disease and the kidney

Abitbol CL, Friedman LB, Zilleruelo G.

Adolesc Med Clin 2005; 16 (1): 45-65.

The adolescent population is particularly vulnerable to sexually transmitted diseases (STDs). Those that cause significant kidney disease are viral origin. The primary VVD are HIV-1, HBV, and HCV. Screening of high-risk populations should inclide quantitation of proteinuria, including total protein and microalbumin, to asses severity of renal damage and potential for progression. Renal biopsy is indicated for diagnosis and for planning important treatment interventions if there is significant proteinuria or decreased renal function. Causes of acute renal failure are frequently reversible and should be treated aggressively. These include HUS, vaso-motor or ischemic acute tubular necrosis, and drug toxicities. The spectrum of chronic kidney disease associated with VVD is broad and may include systemic manifestations of vasculitis. HIV-associated nephropathy is the prototype, with the most prevalent lesion remaining focal segmental glomerulosclerosis (FSGS). Progression occurs in up to 15% of the patients, who are overhelmingly of African lineage. Significant advances in management include ongoing development of HAART, angiotensin antagonists to control proteinuria, and novel immune-modulating drugs such as MMF, CsA, and rituximab. Dialysis therapies have offered improved survival, especially in pediatric patients. Moreover, transplantation is no longer considered experimental and should be offered to select patients.

10. Hypocomplementemia and membranoproliferative glomerulonephritis in children

Itaka K, Nakamura S, Moriya S, et al.

Clin Exp Nephrol 2005, 9 (1): 31-3.

Background: The incidence of hypocomplementemia detected inthe school urinary screening program in Kanagawa Prefecture, Japan, and number of new patients with membranoproliferative glomerulonephritis (MPGN) diagnosed in our institution were decreasing during the period between 1974 and 1997. Follow-up of this study was performed during the period between 1998 and 2003. Methods: A total of 1,230,398 urine specimens in elementary and junior high school were examined between 1980and 2003. serum C3 was measured in children with abnormal urinary findings. Fifty-nine children were diagnosed as having idiopathic MPGN in our hospital between 1974 and 2003. Results: Serum C3 was measured in 1546 school children with abnormal urinary findings, and 34 had hypocomplementemia between 1980 and 2003. Among 264 children in whom C3 was measured between 1998 and 2003, only 4 had mild hypocomplementemia of 83-86 mg/dl (normal, 87-157 mg/dl). Between 1974 and 2003, 59 children (8,8%) were diagnosed having idiopathic MPGN in our hospital, whereas only 2 were diagnosed during the period between 1998 and 2003. Conclusion: The incidence of hypocomplementemia detected in school urinary screening and new cases of MPGN continue to decrese in our experience.

11. Prevention of the progression of chronic kidney disease: practice in China

Wang H, Zhang L.

Kidney Int Suppl 2005; 94: S63-7.

With the epidemic rise of end-stage renal disease (ESRD) in many countries od the world, there is an urgent need to develop and implement strategies aiming at preventing the development and progression of chronic kidney disease (CKD), and the sutuation is the same in China. Glomerulonephritis is still the most common cause of ESRD in China; however, epidemiologic studies have revealed that the prevalence of diabetes and hypertension, which both are major causes of ESRD in many developed countries, are increasing dramatically. Additional studies about the prevalence of albuminuria in diabetes mellitus (DM) patients , and the prevalence of kidney lesion in certain high-risk population (e.g., hypertension and atherosclerosis) are undergoing. According to a questionnaire survey and some reports, education program for Chinese nephrologists and practioners should to be strengthened.

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12. Lupus nephritis in children in Malaysia

Khoo JJ, Pee S, Thevarajah B, et al.

J Pediatr Child Health 2005; 41 (1-2): 31-5.

Objectives:To determine the pattern of renal histolgy, clinical outcome of children with lupus nephritis and to identify any associated risk factors predicting renal failure in these children. Methods: Retrospectively, 27 children under 16 years of age with lupus nephritis who had renal biopsies done at Sultanah Aminah Hospital Johor, Malaysia from 1994 to 2002 were studied. The renal histology was graded according to WHO classification system (1982). The medical records, laboratory data and the clinical outcome of the patients were studied. Results: There were 24 cases of WHO Class IV, two cases of WHO Class II and a case of WHO Class V. Twenty children were in the good renal outcome group while six children progressed into the poor renal outcome group and required renal replacement. One child was lost to follow-up. All six children in the poor renal outcome group had WHO Class IV histology. The 5-year patient and renal survival rates were 84% and 75%, respectively. Age, sex, activity and chronicity indices in the renal histology, anaemia, elevated serum creatinine, depressed levels of C3 and C4, heavy proteinuria or presence of urinary active sediments were not associated with progression to renal failure. Conclusion: Presently, children with lupus nephritis appeared to have better patient and renal survival rates. Assessment of renal histology in these children was important for diagnosis, treatment and probably prognosis. In this study, there was a 25% incidence of loss of renal function over 5 years in children with WHO Class IV renal histology.

13. Cardiovascular risk factors in hemodialysis patients: results from baseline data of kaleidoscopic approaches to patients with end-stage renal disease study.

Ohsawa M, Kato K, Itai K, et al.

J Epidemiol 2005; 15 (3): 96-105.

Background: The prevalence of cardiovascular risk factors and the prevalence of comorbidities in adult hemodialysis patients in Japan are not fully understood. Methods: In „Kaleidoscopic Approaches to Patients with End-Stage Renal Disease Study" (the KAREN Study, 2003), trained research stuff examined 1,214 adult patients in northern areas of Iwate Prefecture. Cardiovascular risk factors and the prevalence of comorbidities in hemodialysis patients were compared with those in the general population using direct age-adjusment methodology and standardized morbidity ratios (SMRs). Results: In hemodialysis patients, common causes of end-stage renal disease were chronic glomerulonephritis (29,8%), diabetic nephropathy (24,5%), and other diseases. Prevalence and SMR of myocardial infarction were 5% and 9,6 respectively, and those of stroke were 13% and 5,7. The prevalence of hypertension and diabetes mellitus were 87% and 29%, respectively. Mean systolic blood pressure and mean diastolic blood pressure were 155 mmhg and 85 mmHg, respectively. Mean levels of total serum cholesterol, high-density lioprotein cholesterol, and albumin in patients with end-stage renal disease were lower than those of the general population (160,6 vs. 203,3 mg/dL, 48,5 vs. 59,7 mg/dL, and 3,7 vs. 4,4 g/dl, respectively). Mean levels of C-reactive protein were higher than those of the general population (3,80 vs. 1,16 mg/L).

Conclusion: Hemodialysis patients have a high prevalence of cardiovascular risk factors and comorbidities. Levels of nutrition-related markers were lower, and C-reactive protein levels were higher, in hemodialysis patients than in the general population.

14. Five-year follow-up of patients with epidemic glomerulonephritis due to Streptococcus zooepidemicus

Sesso R, Pinto SW.

Background: In 1998 there was a large oubreak of acute glomerulonephritis in Nova Serrana, Brazil, caused by group C Streptococcus zooepidemicus. This study describes the follow-up of these patients, after a mean time of 5,4 years of the acute episode. Methods: Of 135 cases idintified in 1998, 56 were re-examined in a prospective study and had measurements of blood pressure, creatinine clearence (estimated by the Cockcroft and Gault formula), microalbuminuria (radioimmunoassay), urine sediment analysis and a protein dipstick test. Results: Of the original group of 135 subjects, 3 died in the acute phase and 5 (3,78%) required chronic dialysis. Of the 56 cases re-evaluated, 54 (96%) were adults (mean +/- SD age, 43/17 years) and 36 (64%) females. At the follow-up examination, we found arterial hypertension in 30% (n = 17/56) of the subjects, reduced creatinine clearence (<80 m/min) in 49% (n=26/53) and increased microalbuminuria (> 20 microg/min) in 22% (n=11/51). Compared to the evaluation carried out 3 years before, the number of cases with creatinine clearence lower than 80 ml/min increased from 20to 26 (of 56 cases). Increased microalbuminuria and/or reduced creatinine clearence were detected in 57% (n=32/56) of the subjects. Patients with reduced creatinine clearence were older than those without reduced renal function (54+/-12 years, p<0,001). Conclusions: After a mean time of 5,4 years, a relatively high proportion of patients with epidemic poststreptococcal glomerulonephritis due to S. zooepidemicus present hypertension, reduced renal function and increased microalbuminuria.

15. HCV associated glomerulopathy in Egyptian patients: clinicopathological analysis

Asbry A, E-Agroudy A, Sheashaa H, et al.

Virology 2005; 334 (1): 10-16.

Background: Hepatitis C virus (HCV) infection in Egypt has reached an epidemic proportion and is associated with many extra hepatic manifestations. Glomerulonephritis (GN) is one of the most consequences of HCV infection oten resulting in end stage renal disease in some cases. Detection of viral genome or particles within the kidney biopsies from HCV-infected patients has proven to be difficult. Histological characterization of renal lesions still represents a major challenge. The aim of our work was to describe the histological pattern of HCV-associated nephropathy. Methods: Fifty patiens – out of 233 – presented to Mansoura Urology and Nephrology Clinic with manifestations of glomerular disease were screened for HCV antibodies by a 3rd generation ELISA test. Those tested positive for HCV antibodies were confirmed by PCR for HCV-RNA and subjected to more detailed clinical, biochemical and histological study. Kidney biopsies and in appropriate cases liver biopsies were examined by LM and electron microscopy (EM). Results: Histological study of renal biopsies revealed membranoproliferative [MPGN] type I to be the most common lesion encountered (54%), folloved by focal segmental glomerulosclerosis [FSGS] (28%), mesangioproliferative GN (18%), membranous nephropathy [MN] (4%) in that order. EM examinations of renal biopsies were successful in identifying HCV like particles in frozen renal tissue. Conclusion: HCV-associated

glomerulopathy is a distinct category of glomerulonephritis. Results of LM showed some peculiar features. In addition, we were successful in location and detection of HCV particles in renal tissues by EM.

II. ETIOLOGY

1. Existence and significance of hepatitis B virus DNA in kidneys of IgA nephropathy

Wang NS, Wu Zl, Zhang YE, et al.

World J Gastroenterol 2005; 11 (5): 712-6.

Aim: To investigate the existence and significance of hepatitis B virus (HBV) DNA in the pathogenesis of IgA nephropathy (IgAN). Methods: Fifty cases of IgAN with HBV antigenemia and/or hepetitis B virus antigens (HBAg, or HbsAg, HbcAg) detected by immunohistochemically in renal tissue were enrolled in our study. The distribution and localization of HBV DNA were observed using in situ hybridization. Southern blot analysis was performed to reveal the state of renal HBV DNA. Results: Among the 5 patients with IgAN, Hbs antigenemia was detected in 17 patients (34%), HBAg in renal tissue was detected in 48 patients (96%), the positive rate of HBAg, HbsAg, and HbcAg was 82% (41/50), 58% (29/50), and 42% (21/50) in glomeruli, respectively; and was 94% (47/50), 85% (28/50) and 78% (39/50) in tubular epithelia, respectively. Positive HBV DNA was detected in 72% (36/50) and 82% (41/50) cases in tubular epithelia and glomeruli respectively by in situ hybridization, and the positive signals were localized in the nuclei of tubular epithelial cells and glomerular mesangial cells as well as infiltrated interstitial lymphocytes. Moreover, 68% (34/50) cases proved to HBV DNA positive by Southern blot analysis, and all the integrated form. Conclusion: HBV infection might play an important role in occurence and progress of IgAN. In addition to humoral immune damages mediated by HBAg-HBAb immune complex, renal tissue of some IgAN are directly infected with HBV and express HBAg in situ, and the cellular mechanism mediated by HBV originating from renal cells in situ may also be involved in the pathogenesis of IgAN.

2. HIV-associated immune complex glomerulonephritis with „lupus-like" features: a clinicopathologic study of 14 cases

Haas M, Kaul S, Eustace JA.

Kidney Int 2005; 67 (4): 1381-90.

Background: While the most common glomerular lesion associated with human immunodeficiency virus (HIV) infection is collapsing focal segmental glomerulosclerosis (FSGS) [HIV-associated-nephropathy (HIVAN)], immune complex-mediated forms of glomerulonephritis have been increasingly reported. One form of glomerulonephritis that has been destricted in the HIV-infected population is immune complex glomerulonephritis with „lupus-like" features, characterized by histologic, immunohistologic, and ultrastructural features resembling lupus nephritis, but occuring in patients without evidence of systemic lupus erythematosus (SLE). Data regarding clinical outcomes in patients with this form of glomerulonephritis are very limited. Methods: We reviewed patholgy reports for all nativ renal biopsy specimens from HIV-positive patients processed at our center from January 1999 through December 2003. Of 77 total specimens, 14 met the following criteria for lupus-like glomerulonephritis: (1) immunofluorescence microscopy showed granular glomerular staining for IgA, IgM, C3 and C1q, with >or-1+(O to 4+ scale) staining for C1q; and (2) the patient's serum was negative for antinuclear antibodies (ANA), or weakly positive (titer < or = 1:80) for ANA and negative for antidouble-stranded DNA. Results: Clinically, ten of the 14 patients with lupus-like glomerulonephritis presented with nephrotic syndrome, all had microscopic hematuria, and nine had serum creatinine >3,0 mg/dL. All but one were African American. Histologically, seven biopsies diffuse proliferative glomerulonephritis, six focal proliferative glomerulonephritis, and one membranous nephropathy. All but two biopsies showed moderate or severe chronic change, and three showed concurrent HIVAN. Ten of the 14 patients developed end-stage renal disease (ESRD) within 1 year of the biopsy. Nine of these ten patients presented with proteinuria >5,0g/24 hours and nephrotic syndrome, while three of four patients who did not develop ESRD had proteinuria < or = 3,0 g/24 hours. Conclusions: Lupus-like glomerulonephritis, defined by immunohistologic features and absence or serologic evidence of SLE, is not an uncommon form of glomerular disease in HIV-infected patients undergoing a renal biopsy. Renal outcomes in these patients were poor, although this may be due largely to most patients presenting with advanced disease.

3. Glomerulonephritis in a patients with chronic active Epstein-Barr virus infection

Kano K, Yamada Y, Sato Y, et al.

Pediatr Nephrol 2005; 20 (1): 89-92.

Renal involvement is rare in chronic active Epstein-Barr virus (EBV) infection. We report an 11-year-old girl who had focal mesangial proliferative glomerulonephritis with cellular crescents and renal tubular atrophy with foam cells in the lumen at the time of the first admission. However, the patient was not diagnosed with chronic active EBV infection until the third admission, 18 months later, because she did not exhibit typical clinical manifestations of infectoius mononucleosis, i.e., fever, lymphadenopathy, hepatomegaly, or increased atypical lymphoctes. We performed in situ hybridization of EBV in renal biopsy and renal autopsy tissue and found genome-positive cells in the enlarged vascular areas surrounding the renal tubules in both specimens. The relationship between mesangial proliferative glomerulonephritis with crescents and chronic active EBV

infection id unknown.

4. A case of necrotizing glomerulonephritis presenting with nephrotic syndrome associated with pulmonary cryptococcosis

Nakayama M, Hori K, Ishida I, et al.

Clin Exp Nephrol 2005; 9 (1): 74-8.

We describe a 68-year-old man with necrotizing glomerulonephritis who presented with nephrotic syndrome accompanied by pulmonary cryptococcosis. He developed rheumatoid arthritis in July 1999 and was treated with low-dose prednisolone. He was admitted to our hospital on November 22 following the appearance of bilateral leg edema in October 2000. Laboratory tests at presentation revealed nephrotic syndrome with renal impairment. Renal biopsy specimens revealed necrotizing glomerulonephritis with crescent, but immunofluorescence study showed lack of staining for immunoglobulins or complement components. Chest X-ray and CT showed abnormal shadows in the right upper lung field, and Cryptococcus neoformans was isolated in a transbronchial lung biopsy. After the diagnosis of pulmonary cryptococcosis was made, the patient was treated with 200 mg/day fluconazole. The pulmonary abnormal shadows immediately improved and urinary protein excretion dramatically decreased. A second renal biopsy, performed about 2 months after the first biopsy, showed disappearance of crescent. Electron microscopic examination of the second renal biopsy showed partial effacement of foot processes without electron-dense deposits. Our findings suggest that necrotizing glomerulonephritis with nephrotic syndrome in this patient represented pauci-immune T-cell-mediated injury related to pulmonary cryptococcosis.

5. A case of renal sarcoidosis with complement activation via the lectin pathway

Hagiwara S, Ohi H, Eishi Y, et al.

Am J Kidney Dis 2005; 45 (3): 580-587.

A 57-year-old woman with pulmonary sarcoidosis was admitted to the hospital because of an elevation of serum creatinine and blood urea nitrogen. On admission, the laboratory data suggested interstitial nephritis without proteinuria and hematuria, whereas a renal biopsy showed granulomatous interstitial nephritis and mild mesangial proliferative glomerulonephritis. Immunoglobulin and C1q deposits were negative, but mannose-binding lectin, C3, C4d, and C5b-9 deposits were marked in the glomerular mesangial areas. The lectin pathway of complement activation may have contributed to the development of glomerular injury in this patient. DNA of *Propionibacterium acnes,* which is now strongly suspected as the pathogen of sarcoidosis, was detected in the patient's glomerular mesangial cells; tubular epithelial cells, which were involved in granulomatous inflammation; and mononuclear cells in epithelioid granulomas by in situ hybridization. These findings may be add new insights to the pathogenesis of renal sarcoidosis, including its relation to infection, because mannose-binding lectin plays a crucial role in the host

defense against various pathogens. From this case of renal sarcoidosis, it is hypothesized that *P acnes* may be involved in pathogenesis of granulomatous interstitial nephritis and that it plays a role in glomerular complement activation via the lectin pathway.

6. Henoch-Schönlein purpura associated with esophagus carcinoma and adenocarcionoma of the lung

Weller-Bisig D, Ettlin G, Brink T, et al.

Clin Nephrol 2005; 63 (4): 302-304.

Abstract. Henoch-Schönlein purpura (HSP) is known exist in association with a variety of malignant diseases including squamous and small cell lung cancer and hematological malignancies. We report first cases of HSP associated with carcinoma of the esophagus and adenocarcinoma of the lung, respectively. We compare the main features of our patients with 23 previously published cases. We recommend that patients with HSP, especially men over 40 years of age, should undergo screening for occult neoplasia.

7. Light chain muscle deposition caused rhabdomyolysis and acute renal failure in patients with multiple myeloma

Farah R, Farah R, Kolin M, et al.

Clin Nephrol 2005; 63 (1): 50-53.

Abstract Case report of a 70-year-old woman with the diagnosis of multiple myeloma and acute renal failure due to rhabdomyolysis (RDM) that was caused from the deposition of k light chain in muscle fibers. In addition, the deposition was found in the liver.

8. Psoriatic nephropathy – does an entity exist?

Singh NP, Prakash A, Kubba S, et al.

Ren Fail 2005; 27 (1): 123-7.

Psoriasis is an immune-mediated chronic inflammatory disorder of the skin. Association with kidney disease has been debated for a long time. Secondary renal amyloidosis in psoriatic arthropathy and drug-induced renal lesions secondary to methotrexate or cyclosporine are accepted

accompaniments of psoriasis. IgA nephropathy is also known to occur in psoriatics. We report three interesting cases of renal involvement in long-standing estabilished psoriasis on topical therapy alone. The patients presented with hypertension, significant proteinuria, hypoalbuminemia, and dyslipidemia. Kidney biopsies revealed „mesangioproliferative glomerulonephritis with IgA nephropathy", „focal proliferative glomerulonephritis", „and membranous glomerulopathy" . The former two had marked active urinary sediment. Patients improved on prednisolone and angiotensin-converting enzyme inhibitors. Contrary to the belief that renal involvement in psoriasis is coincidental, we propose that kidney disease may be a common accompaniment of psoriasis, which may be labeled as „psoriatic nephropathy" or „psoriatic kidney disease". The exact mechanism of this entity is yet to be elucidated.

9. Neutrophilic dermatosis associated with propylthiouracil-induced p-ANCA (p-antineutrophil cytoplasmic antibodies)

Boulenger-Vazel A, Kupfer-Bessaquet I, Gouedard C, et al.

Ann Dermatol Venereol 2005; 132 (1): 27-31.

Introduction: We report on a patient who progressively developed polymorphic expression of neutrophilic dermatosis (Sneddon-Wilkinson subcorneal pustulosis and pyoderma gangrenosum) associated with p-antineutrophil cytoplasmic antibodies (p-ANCA), while receiving propylthiouracil for hyperthyroidism. To our knowledge, such associations have never been published so far. Case-report: A 40 year-old woman was treated with propylthiouracil for Graves' disease. After 16 months of therapy, she noted flares of pustular lesions surrounded with erythematous halo mainly localized on the trunk. The lesions became chronic, and were not improved by potent topical corticosteroids. When first seen in our department in February 2003, the eruption was typical of Sneddon-Wilkinson subcorneal pustulosis. This diagnosis was confirmed by the histological examination of a skin biopsy of pustule. One month later, she developed an inflammatory progressively ulcerative lesion on the right ankle, typical of pyoderma gangrenosum. The diagnosis was confirmed by the histological examination of a skin biopsy take on the evolving border of the lesion and showed polynuclear neutrophilc infiltration without vasculitis. Direct immunofluorescence was negative. The presence of serum anti.myeloperoxidase p-ANCA was known for this patients since Octóber 22. No IgA monoclonal gammopathy was revealed on extensive biological check-up. Systemic oral corticosteroid therapy)1mg/kg/day) dramatically improved skin lesions with complete healing within 8 weeks. Discussion: Propylthiouracil is well known to induce the occurence of ANCA in patients treated for Graves' disease. The mechanisms involved are badly recognized so far. Cutaneous vasculitis, glomerulonephritis, and polycondritis may be clinically associated with those antibodies. Rare observations of neutrophilic dermatosis, mostly Sweet's syndrome, have been described in patients with propylthiouracil-induced ANCA. One case-report described a 44 year-old woman who developed pyoderma gangrenosum associated with propylthiouracil-induced p-ANCA. These manifestation usually appear within 2 years , as our patient. The data in the literature, allows us to report the polymorphic expressions of neutrophilic dermatosis in this patient with p-ANCA which could be related to propylthiouracil. Such association of Sneddon-Wilkinson subcorneal pustulosis and pyoderma gangrenosum with p-ANCA has never been described in this endocrinologic context so far. Furthermore we propose that neutrophilic dermatosis should be inscribed in the list of side effects induced by propylthiouracil therapy.

10. Epitope analysis of myeloperoxidase-specific antineutrophil cytoplasmic autoantibodies (MPO-ANCA) in childhood onset Graves' disease

Fujieda M, Suzuki K, Sato H, et al.

Clin Nephrol 2005, 63 (6): 437-45.

Aim: This study aimed to elucidate the relationship between epitope profiles and clinical manifestations of patients with myeloperoxidase antineutrophil cytoplasmic autoantibodies (MPO-ANCA) positive childhood onset Graves's disease treated with propylthiouracil (PTU). Methods: Sixteen patients were studied. The patients were grouped into ten without clinical vasculitis and nephritis (non-vasculitis group) and six with biopsy.proven pauci-immune necrotizing crescentic glomerulonephrits (vasculitis group). Epitope analysis was performed on serum samples by an enzym-liked immunosorbent assay (ELISA) using a panel of recombinant deletion mutants of MPO. Results: The high frequency sites were region upstream of Met341 (Ha region) near the N-terminus of the heavy chain, and regions downstream of Gly598 (Hf and Hg regions) near the C-terminus. Most patients in the non-vasculitis group had polyclonal MPO-ANCA recognizing both above linear sites and other epitope sites of the haevy chain of MPO. Only one of ten patients in the non-vasculitis group, and six patients in the vasculitis group, two had nephritis, like rapidly progressive glomerulonephritis and one had alveolar hemorrhage. Conclusion: These findings suggest that most with childhood onset Graves' disease treated with PTU who manifest no vasculitis have polyclonal MPO-ANCA recognizing both the linear and other epitope sites of the heavy chain of MPO. However, some patients who have develop nephritis have MPO-ANCA recognizing only the linear sites of the heavy chain of MPO. This clonality of MPO-ANCA may be a risk factor that induces clinical vasculitis and nephritis in patients treated with PTU. Therefore, patients exposed to PTU should be monitored for MPO-ANCA level and epitopes.

11. Necrotizing glomerulonephritis and pulmonary hemorrhage associated with carbimazole therapy

Calanas-Continente A, Espinosa M, Manzano-Garcia G, et al.

Thyroid 2005; 15 (3): 286-8.

Methimazole, carbimazole, and propylthiouracil (PTU) are the maintays of antithyroid drug therapy. Adverse effects of these drugs have been documented in less than 15% of patients undergoing treatment for hyperthyroidism. Common problems include fever, skin rash, urticaria, arthralgias, and arthritis. Vasculitis associated with antineutrophil anticytoplasmic antibodies (ANCA) has been reported on several occasion following treatment with PTU. However, vasculitis rarely appears to

be associated with carbimazole. We report the clinical hystory of a women with necrotizing glomerulonephritis and pulmonary hemorrhage associated with carbimazole therapy.

12. IgA nephropathy in a young man with primary hyperparathyroidism

Jochum E, Brandenburg VM, Brodersen HP, et al.

Clin Nephrol 2005; 63 (1): 46-9.

We report the first documented case of IgA nephropathy occuring after treatment of primary hyperparathyroidism. A 29-year-old man with history of kidney stones and primary hyperparathyroidism underwent kidney biopsy for persistent proteinuria and microhematuria 18 months after resection of an ectopic parathyroid adenoma with subsequent normalization of serum calcium and parathyroid hormon levels. On ultrasound, renal intraparenchymal calcifications were noted. Renal biopsy revealed IgA nephropathy in addition to tubulointerstitial microcalcifications. The development of IgA nephropathy may have been influenced by hyperparathyroidism and/or its treatment. The case highlights the role of renal biopsy in patients with a history of kidney stones and abnormal urinary findings.

13. Acute renal failure due to mesangial proliferative glomerulonephritis in a pregnant woman with primary Sjogren's syndrome

Adam FU, Torun D, Bolat F, et al.

Clin Rheumatol 2005 May 26 [Epub ahead of print]

The most common form of renal involvement in Sjogren's syndrome (SS) is tubulointerstitial nephritis. Renal dysfunction is usually mild and subclinical. Glomerulonephritis (GMN) is rare in patients with SS. We report a 28-year-old multigravida patient with primary Sjögren's syndrome (pSS) and associated manifestations, whor presented with acute renal failure inthe 20th week of her fifth pregnancy. The complaints and clinical findings, positive Schirmer's test, findings of dry eye on ophtalmologic examination, and the salivary glans biopsy were compatible with SS. The patients exhibited no other clinical and laboratory findings indicative of other collagenous disease and/or rheumatoid arthritis. She refused renal biopsy, hesitating for fear fetal loss; thus based on the vasculitis, prednisolone, plasmapheresis, and one dose of cyclophosphamide were administered during the pregnancy. Hemodialysis five times weekly was performed. At the 28th week of gestation, she underwent a cesarean section due to early rupture of membranes and fetal distress. A healthy male boy was delivered. The renal biopsy performed 2 weeks after labor revealed mesangial proliferative glomerulonephritis. After the fourth cyclophosphamide treatment, her urinary output increased and she was discharged from hemodialysis program. She remains in follow-up at our outpatient clinic free of hemodialysis for 4 months. This is the first report of mesangial prolferative GMN requring dialysis in a pregnant pSS patients that has featured good maternal and fetal outcomes.

14. Membranous nephropathy associated with familial chronic ulcerative colitis in a 12-year-old girl

Ridder RM, Kreth HW, Kiss E, et al.

Pediatr Nephrol 2005 Jun 22 [Epub ahead of print]

Glomerulonephritis is a rare complication in patients with inflammatory bowel disease. We report a case of membranous nephropathy (MN) in a 12,6-year-old girl with chronic ulcerative colitis. The girl was referred to the hospital with bloody diarrhea and arthralgia. Routine urinalysis showed 1 g/m(2) protein excretion in 24h serum ANCA titers were positive. The diagnoses were confirmed by colonoscopy and kidney biopsy. The patient's mother had also suffered from ulcerative colitis in adolescence. Proteinuria normalized under treatment with prednisone (60 mg/m(2)/day) and azathioprine, which was initiated to treat the colitis. Chronic ulcerative colitis can be associated with glomerulonephritis.

15. Renal involvement in patients with polimyositis and dermatomyositis

Yen TH, Lai PC, Chen CC, et al.

Int J Clin Pract 2005; 59 (2): 188-93.

Renal invovement in patients with polymyositis (PM)/dermatomyositis (DM) is previously thought to be uncommon, but two main types of renal lesion have been described. First, acute tubular necrosis with renal failure related to myoglobulinemia and myoglobulinuria is a well-recognised feature of acute rhabdomyolysis. Second, chronic glomerulonephritis has been infrequently reported in a small group of patients with PM/DM. This study aims at investigating the incidence, severity and prognosis of renal disease in PM/DM patients, admitted to a single centre in a 10-year interval. The hospital records of 65 Taiwanese patients with PM/DM, examined between 1992 and 2002, were studied retrospectively. Of the 65 patients, 14 were found to have suffered varying degree of renal involvement, and the incidence rate was 21,5%. All the 14 patients had varying degree of haematuria and proteinuria. Acute tubular necrosis with renal failure developed in four patients with PM and in five patients with DM. Renal biopsy in two DM patients with overt proteinuria revealed IgA nephropathy in one and membranous nephropathy in the other. We, therefore, concluded that renal involvement in PM/DM patients is not as uncommon as previusly thought.

16. Nephrotic syndrome after stem cell transplantation

Stewenson WS, Nankivell BJ, Hertzberg MS.

Clin Transplant 2005; 19: 141-144.

Abstract: Nephrotic syndrome occurs rarely after bone marrow transplantation. We describe three patients with myeloid malignancy who developed nephrotic syndrome from 5, 22 and 25 months after allogenic stem cell transplantation (SCT) confirmed by electron microscopy as membranous glomerulonephritis in two and minimal change glomerulonephritis in one. Proteinuria was initially severe in all and clinically distinct from prior graft-vs.-host disease in two patients. While all responded initially to prednisolone and cyclosporine therapy, two recipients with hihgh-risk leukemia developed late solid organ and bone marrow relapse of their disease, which ultimately proved fatal. The third patient remains alive and disease-free with minimal proteinuria off immunosuppressive therapy. Hence, the onset of *de novo* high-grade proteinuria after allogenic SCT should prompt renal histological confirmation, and a trial of immunosuppressive therapy after other causes of nephritic syndrome have been excluded.

17. Development of glomerulonephritis during anti-TNF-(alpha) therapy for rheumatoid arthritis

Stokes MB, Foster K, Markowitz GS, et al.

Nephrol Dial Transplant 2005; 20 (7): 1400-6.

Background: Treatment of rheumatoid arthritis with anti-tumor necrosis factor alpha (TNFalpha) agents may lead to autoantibody formation and flares of vasculitis, but renal complication are rare. Methods: We report the clinical and pathologic findings in five patients with longstanding rheumatoid arthritis (duration of rheumatoid arthritis, 10-30 years; mean, 23 years) who developed new onset of glomerular disease after commencing therapy with anti-TNFalpha agents (duration therapy, 3-30 months; median, 6 months). Results: At presentation, three patients were receiving etanercept, one adalimumab and one infliximab. Two subjects presented with acute renal insufficiency, hematuria, nephrotic-range proteinuria, positive lupus serologia, and hypocomplementemia, and renal biopsies showed proliferative lupus nephritis. Two individuals presented with new onset renal insufficiency, hematuria and proteinuria, and renal biopsies showed pauci-immune necrotizing and crescentic glomerulonephritis. One of these subjects, who had antimyeloperoxidase autoantibodies, also developed pulmonary vasculitis. The fifth patient presented with nephrotic syndrome and renal biopsy findings of membranous glomerulonephritis, associated with immune complex renal vasculitis. A pathogenic role for anti-TNFalpha therapy is suggested by the close temporal relationship with development of glomerular disease, and by the the improvement in clinical and laboratory abnormalities after drug wihdrawal and initiation of immunosuppressive therapy in most cases. Conclusion: Rheumatoid arthritis patients receiving anti-TNFalpha agents may develop glomerulonephritis via the induction of rheumatoid arthritis-related nephropathy or de novo autoimmune disorders.

18. Case of inflammatory vasculopathy and encephalopathy caused by treatment with tacrolimus

Ringelstein A, Bongs K, Sorge-Hadicke B, et al.

Nervenarzt 2005; 76 (4): 475-8.

The case of inflammatory vasculopathy and encephalopathy caused by treatment with tacrolimus is reported. This 49-year-old woman developed progressive gait ataxia and right-sided hemiparesis after 7 years of tacrolimus therapy for focal sclerosing glomerulonephritis. MRI presented multifocal cerebral lesions with contrast enhancement. Oligoclonal banding was positive. When treatment with tacrolimus was stopped, the clinical symptoms resolved completely and the MRI findings improved with corticoid monotherapy.

19. Cyclooxigenase-2 inhibitor-associated minimal-change disease

Almanson M, Kovithavongs T, Qarni MU.

Clin Nephrol 2005; 63 (5): 381-384.

Abstract. Selective cyclooxigenase-2 (COX-2) inhibitors are relatively newer antiinflammatory drugs that produce comparable antiinflammatory and analgesic effects to the nonselective nonsteroidal antiinflammatory drugs (NSAIDs); but with fewer symptomatic gastric and duodenal ulcers. Limited data are available concerning the toxicity associated with COX-2 inhibitors outside the gastrointestinal tract. The NSAIDs have been known for their nephrotoxic potentials including minimal-change disease (MCD) with interstitial nephritis. Although the recent data suggest that COX-2 inhibitors may have the same adverse renal effect as NSAIDs, there is only one case report describing minimal change disease and acute interstitial nephrits (AIN) associated with a COX-2 inhibitor, celecoxib. We are reporting a case of MCD and acute tubular necrosis (ATN) but without interstitial nephritis in a patient treated with celecoxib. Although the proteinuria in our patient resolved completely after discontinuation of celecoxib, the renal function did not. We suggest that heightened suspicion of this side effect of COX-2 inhibitors should be maintained in all patients taking this class of drugs who present with nephrotic syndrome.

20. COX-2 inhibitor induced renal failure in a previously healthy young woman

Mühlfeld AS, Floege J.

Clin Nephrol 2005; 63 (3): 221-224.

Abstract. Side effects of nonsteroidal anti-inflammatory drugs (NSAIDs) most commonly affect the gastrontestinal tract and the kidney. The recent release of selective cyclooxygenase-2 (COX-2) inhibitors has been associated with a decrease in adverse gastrointestinal effects. However, the

nephrotoxic potential of these drugs still remains controversial. Here, we eight days of anuria following the administartion of valdecoxib, a newly released selective cyclooxygenase-2 inhibitor, during episode of acute febrile pyelonephritis. We suggest that selective COX-2 inhibitors should not be used in patients with volume contraction and underlying renal disease.

21. COX-2 inhibitors and acute interstitial nephritis: case report and review of the literature

Esteve JB, Launay-Vacher V, Brocheriou I, et al.

Clin Nephrol 2005; 63 (5): 385-389.

Abstract. We report a case of biposy-proven acute interstitial nephrits (AIN) in a 50-year-old diabetic women, who had been treated with celecoxib for 4 weeks before presentation. She presented with clinical findings of renal proximal tubulopathy, aseptic leukocyturia and acute renal failure. Kidney biopsy specimen showed AIN with intense tubuli and eosinophilic infiltrate in the interstitium. She recovered normal reanl function two weeks after cessation of celecoxib and use of a corticosteroid. A review of the literature yielded eight cases of COX-2 inhibitor-associated AIN with a biopsy-proven diagnosis. Among the reported cases, AIN was diagnosed after an average of 8,3 months of therapy (SD 12 months, range 3 days – 3 years) with 25 mg rofecoxib or 200 mg celecoxib daily. Common symptoms included asthenia, anorexia, nause and womiting. The classic triad of fever, rash and eosinophilia. Renal failure was common at the time of diagnosis. Mean serum creatinine levels were 0,86±0,11 mg/dl, 5,66±3,50 mg/dl and 1,15±0,4 before treatment, at time of diagnosis and 1-2 months after COX-2 inhibitor withdrawal, respectively. Three patients required emergency hemodialysis. After cessation of CO-2 inhibitor treatment, patients recovered completely with a normalized serum creatinine level after one to two months. Manegement consisted of withdrawal of the COX-2 inhibitor drug and in four patients, corticosteroid therapy was well-tolerated and may have been beneficial.

22. Acute renal failure

Lameire N, Van Biesen W, Vanholder R.

Lancet 2005; 365: 417-30.

This seminar covers the most recent information on definition, epidemiology, and clinical causes of acute renal failure. The mechanisms of acute prerenal failure and the potential interference by commonly used drugs of autoregulation of renal blood flow are discussed. We summarise some basic and recent insights into the haemodynamic and cellular pathophysiology mechanisms, mainly of postischaemic acute renal failure. Recent findings on the repair mechanisms of renal injury and the potential future therapeutic possibilitie are discussed. We provide some differential diagnostic approaches for patients with acute renal failure and summarise preventiom of the disorder and management of critically ill patients by dialysis and by other means. Finally, some information on the influence of gene polymophisms on the prognosis of acute renal failure is given.

III. PATHOGENESIS

1. Glomerulonephritis

Chadban SJ, Atkins RC.

Lancet 2005; 365: 1797-806.

The term glomerulonephritis encompassess a range of immune-mediated disorders that cause inflammation within the glomerulus and other copartments of the kidney. Studies with animal models have

shown the crucial interaction between bone-marrow-derived inflammatory cells and cells intrinsic to the kidney that is both fundamental and unique to the pathogenesis of glomerulonephritis. The mechanisms of interaction between thes cells and the mediators of their coordinated response to

inflammation are being elucidated. Despite these pathophysiological advances, treatments for glomerulonephritis remain non-specific, hazardous, and only partly successful. Glomerulonephritis therefore remains a common cause of en-stage kidney failure worldwide. Molecule-specific approaches offer hope for more effective and safer treatments in the future.

2. Genetics of common progressive renal disease

Chow KM, Wong TY, Li Pk.

Kideny Int Suppl 2005; 94: S41-5.

Familial aggregation of common chronic kidney diseases provides a unique opportunity to investigate the susceptibility genetic and environmental factors. In the past decade, a wealth of new data has become available concerning the genetic susceptibiliy leading to numerous nephropathies. Knowledge of the genetic components allows better understanding of initiation and progression of these chronic kidney diseases. In addition, one can envision that identification of genetically susceptible individuals might lead to erlier diagnosis and potential reversal of the current epidemic of end-stage renal disease. The goal of the current discussion is to review various issues pertaining to the role of genetic factors in common chronic kidney diseases as exemplified by two leading causes of end-stage renal diseases woldwide, nephropathy of type 2 diabetes and IgA nephropathy. The genetic and environmental interplay leading to the nephropathies is highlighted.

3. Complement and glomerulonephritis: new insights

Turnberg D, Cook HT.

Curr Opin Nephrol Hypertens 2005; 14: 223-228.

Purpose of review: The last few yers have seen a huge increase in our understanding of the role of the complement system its regulation in glomerular disease. Our aim is to summarize the most important advances in this field. Recent findings: The role of complement in systemic lupus erythematosus continues to be elucidated. Classical pathway components protect from the development of autoimmunity, at least in part through their role in the clearence of apoptotic cells in contrast, the alternative pathways plays a direct role in exacerbating glomerular injury. Ant-C1q antibodies are related to activity in lupus nephritis and recent studies have shown that they are directly pathogenic in animal models. Proteinuria, whatever the cause, may lead to tubulointerstitial injury and complement activation adds to this process. In particular, deposition of terminal components of complement in the tubular lumen contributes to interstitial myofibroblast activation. There is increasing evidence for the role of complement regulatory proteins in glomerular injury. In particular, abnormalities of factor H or of CD46 may predispose to atypical haemolytic uraemic syndrome. The control proteins also protect against injury in immune complex glomerulonephritis. Summary: Advances in our understanding of the role of complement in glomerular injury point to the likely therapeutic benefits of targeting the complement system. Many new drugs are becoming

available. Careful dissection of the pro- and antiinflammatory effects of complement system which the experimental models allow will assist in designing directed therapy that will avoid the detrimental effects of nonspecific systemic complement inhibition.

4. Ageing as a determinant of renal and vascular disease: role of endothelial factors

Barton M.

Nephrol Dial Transplant 2005; 20: 485-490.

In developed countries, ageing is the most important risk factor for age and death after age 28. Age also determines the onset and development of the most prominent vascular and renal disease, atherosclerosis and glomerulosclerosis. Increased vascular and renal oxidative stress, and, as a consequence, abnormal activity of endothelium derived molecules, such as nitric oxide (NO), angiotensin II and endothelin, are now recognized as important mechanisms controlling these disease process. In this article, I will discuss current evidence for the involvement of endothelial factors in the genesis of vascular dysfunction and cardiorenal disease seen with ageing and present therapeutic approaches to actively interfere with these disease process.

5. Apolipoprotein E and progression of chronic kidney disease

Hsu CC, Kao WHL, Coresh J, et al.

JAMA 2005; 293 (23): 2892-2899.

Context: Apolipoprotein E (APOE) genetic variation has been implcated in diabetic nephropathy with the epsilon(E) allele increasing and E4 allele decreasing risk. APOE allelic associations with chronic kidney disease beyond diabetic nephropathy are unknown, with no studies reported in high-risk African populations. Objective: To quantify the risk of chronic kidney disease progression associated with APOE in a population-based study including white, African American, diabetic, and nondiabetic individuals. Design, Setting,and Participants: Prospective follow-up (through January 1,2003) of Atherosclerosis Risk in Communities (ARIC) study participants, including 3859 African American and 10 661 white adults aged 45 to 64 years without severe renal dysfunction at baseline in 1987-1989, sampled from 4 US communities. Main Outcome Measures: Incident chronic kidney disease progression, defined as hospitalization or death with kidney disease or increase in serum creatinine level of 0,4 mg/dL (35 umol/L) or more above baseline, examined by APOE genotypes and alleles. Results: During media follow-up of 14 years, chronic kidney disease progression developed in 1060 individuals (incidence per 1000 person-years; 5,5 overall; 8,8 in African Americans 4,4 in whites). Adjusting for major chronic kidney disease risk factors, E2 moderately increased and E4 decreased risk of disease progression (likelihood ratio test, P=0,3). Further adjusment for low- and high-density lipoprotein cholesterol and triglycerides did not attenuate relative risks (RRs) (E2: 1,08 [95% CI, 0,93-1,25] and E4: 0,85 [95% CI, 0,75-0,95] compared with E3; likelihood ratio test, P=0,008). E2 was associated with a decline in renal function (RR, 1,25

[95% CI, 1,02-1,53]), though not with events, such as hospitalization or end-stage renal disease. Risk was similar stratified by race, sex, diabetes, and hypertension (all P values for interaction >0,05). Excess risk of chronic kidney disease in African Americans was not explained by APOE alleles. Coclusion: APOE variation predicts chronic kidney disease progression , indpendent of diabetes, race, lipid, and nonlipid risk factors. Our study suggest that nonlipid-mediated pathways, such as cellular mechanisms of kidney disease remodeling, may be involved in the association of APOE alleles and progression of chronic kidney disease.

6. The link between circulating markers of endothelial function and proteinuria in patients with primary glomerulonephritis

Mackinon B, Deighan CJ, Norrie J, et al.

Clin Nephrol 2005; 63 (3): 173-180.

Abstrat. Introduction: It is well-estabilished that there is an increase in the incidence of cardiovascular mortality in patients with proteinuric renal disease. The magnitude of the increase in risk is unlikely to be explained by traditional risk factors for cardiovascular disease alone. Proteinuria itself may cinstitute an additonal risk factor, and proteinuric patients are known to have a degree of endothelial dysfunction. The nature of this relationship between proteinuria and endothelial function is the subject of intense investigation. Aim: The aim of this study was to axamine the relationship between proteinuria and endothelial dysfunction, as reflected by serum von Willebrand factor (vWF), and the soluble cell adhesion molecules VCAM and ICAM, in patients with primary glomerulonephritis (GN). A secondary aim was to discern whether any relationship could be explained by renal function, lipid profile, inflammation or blood pressure. Methods: A cross-sectional study was undertaken in consecutive patients attending a general nephrology clinic with biopsy-proven primary GN. Patients with end-stage renal disease (ESDR), those on immunosuppressive drugs, or with intercurrent infective illness were excluded. Blood pressure and body mass index were recorded. Routine lab assays were undertaken for serum creatinine, lipid profile, and 24-hour urinary protein (UProt). Additional serum samples were stored at –80 C for subsequent measurement of vWF, VCAM, ICAM and sensitive C reactiv protein (sCRP). Results: Data were collected from 129 (86 Male) patients. Mean (standard dviation) estimated creatinine clearence was 64 (32 ml/min, and median (interquartile range) 24-hour proteinuria was 1,1 (0,22-2,9)g. Mean vWF was 173 (68) IU/dl, median VCAM, ICAM and sCRP were 594 (410-708)ng/ml, 235(208-286)ng/ml, and 2,33 (0,83-5,68) mg/l, respectively. There was a significant positive correlation between vWF and UProt (Spearman rank correlation, r- 0,41, p<0,001). When split into tertiles, accordong to UProt(0—500mg, 500-2000mg, and >2,000mg), there was a significant, stepwise increase in mean vWF (p<0,001), log VCAM (p<0,001), and log ICAM (p=0,002)). On multivariate analysis with vWF as the continous dependent variable, UProt, age, total cholesterol and sCRP were only significant independent correlates (model-adjusted R^2=33%). Conclusion: In patients with primary GN, there is a significant association between endothelial activation as reflected by vWF, VCAM or ICAM and increasing proteinuria. Elevations in vWF, as well as being related to classical risk factors, are associated with increases in total proteinuria and low-grade inflammation. Thus, future prospective studies should examine the extent to which vWF and other circulating markers of endothelial activation predict coronary heart disease risk in patients with proteinuric renal disease.

7. Regression of glomerulosclerosis with high-dose angiotensin inhibition is linked to decreased plasminogen activator inhibitor-1

Ma LJ, Nakamura S, Aldigier JC, et al.

J Am Soc Nephrol 2005; 16: 966-976.

The potential and possibel mechanisms for regression of existing glomerulosclerosis by angiotensin II type 1receptor antagonist (AT1RA) and/or angiotensin I converting enzyme inhibitor (ACEI) were investigated. Adult male Sprague Dawley rats underwnt 5/6 nephrectomy (Nx). Glomerulosclerosis was assessed by renal biopsy 8 wk later, and rats were divided into goups with equal biopsy sclerosis and treated for next 4 wk untily were killed at 12 wk as follows: Control with no further treatment (CONT), high-dose AT1RA, high-dose ACEI, and varying AT1RA+ACEI combinations. Hypertension and proteinuria induced by 5/6 Nx were significantly decreased by all treatments, except high-dose ACEI, which showed persistent proteinuria. High-dose AT1RA and ACEI markedly decreased progression of sclerosis, with –2,3% average decrease in sclerosis from biopsy to autopsy in AT1RA *versus* 194% increase in CONT (P<0,0001). Glomerulosclerosis regressed, with loss severe lesions at the time when the rats were killed than at biopsy in 62% of AT1RA-treated and 57% of ACEI-treated rats. In contrast, only 17 to 33% of rats in combination groups had regression. Alternatively, thes data might be viewed as reflecting halting of progression, as some goups had higher BP and proteinuria. However, this potential confounding effect does not negate the effects to achive regression of sclerosis in these rats. Regression was not explained by changes in mRNA of TGF-ß1 and matrix metalloproteinase-2 and –9 but linked to decreased tissue inhibitor of metalloproteinase-1 and plasminogen activator inhibitor-1. It is concluded that angiotensin inhibition mediates regression in part by effects on matrix modulation.

8. Expression and activation of STAT3 in chronic proliferative immune complex glomerulonephritis and the effect of fosinopril

Zhang W, Chen X, Shi S, et al.

Nephrol Dial Transplant 2005; 20: 892-901.

Background: Signal transducers and activators of transcription (STATs) are cytoplasmic proteins that are activated in response to stimulation from varous cytokines. Among these, STAT3 is an importan member that has been implicated in the inflammatory proliferation of cells. We hypothesized that STAT3 may be activated in kidneys of rats having modified chronic immune complex glomerulonephritis, and that angiotensin-converting enzyme (ACE) inhibition with fosinopril may prevent the activation of STAT3 and subsequent upregulation of tissue inhibitor of metalloproteinase-1 (TIMP-1), which are effects of fosinopril on nephritis. Methods: Fifty-one Wistar rats were randomly divided into three groups that included a control group, a model group and a fosinopril group. Bovine serum albumin (BSA) nephritis was induced by subcutaneous immunization and daily intraperitonel (i.p.) administration of BSA. To accentuate the nephritis, we performed uni-nephrectomy and gave 100ug of lipopolysaccharide (LPS) as an i.p. injection.

Macrophage infiltration (ED-1) was assessed with immunohistochemistry. The expression and activation of STAT3 and the expression of TIMP-1, one of the STAT3 downstrem genes, were observed in renal tissues of rats by means of immunohistochemistry, electrophoretic mobility shift assay (EMSA), western blot and northen blot. The relationship between STAT3 phosphorilation, 24h urinary protein excretion were also analysed. Results: Northern blot showed that the mRNA expression of both STAT3 and TIMP-1 was significantly increased in kidneys from the model group, but significantly decreased inthe fosinopril group (P<0,05). Western blot analysis revealed similar increases in the expression of STAT3. phopho-STAT3)p-STAT3) and TIMP-1 in the model group. Analysis of immunohistochemistry showed that STAT3 and p-STAT3 were expressed in very few cells of normal rats, that expression was strong in model rats and that this increased expressin was attenued in the fosinopril group (P<0,05). The expression of p-STAT3 in glomeruli was positively correlated with 24h proteinuria as well as with glomerular TIMP-1 expression. Double staining showed that some ED-1 positive cells also contained p-STAT3-positive staining. Conclusions: The present study showed that STAT3 is expressed and activated in kidneys of rats with modified immune complex glomerulonephritis. These rats also had increased ED-1-positive cells, with some cell showing simultaneous expression of p-STAT3 and ED-1, which may contribute to glomerular inflammatory proliferation and extracellular matrix accumulation. Finally, fosinopril downregulated STAT3 activation and ED-1 influx, which are effects that may attenuate renal damagae in this model.

9. Apoptosis and proliferation in chilhood acute proliferative glomerulonephritis

Ozaltin F, Besbas N, Bakkaloglu A, et al.

Pediatr Nephrol 2005 Jun 18 [Epub ahead of print]

Acute proliferative glomerulonephritis is characterized by glomerular hypercellularity that can be caused by many different etiologies and pathogenetic mechanisms. A balance between cell birth by mitosis and cell death by apoptosis is crucial. In this study, apoptosis and regenerative activity (Ki 67/apoptosis index) were investigates in acute proliferative glomerulonephritis. Thirty-five children with biopsy-proven acute proliferative glomerulonephritis and five controls with MCD were studied retrospectively. According to the clinical outcome, patients were divided into 2 groups: group 1 (n=21) were patients with normal renal functions at follow-up; group 2 (n=8) were patients with end-stage renal failure or those who died. Immunohistochemical staining of proliferating cells (Ki67) was done. In situ end labeling of DNA was used to evaluate apoptosis .Glomerular cell apoptosis was 45% in the patients with acute proliferative glomerulonephrits and 3% in controls (p<0,001). Apoptotic cells were identified in the tubulointerstitial compartment with higher and heavier immunostaining in patients in patients than controls (p=0,001). Tubular proliferative index (=tubular proliferatin/tubular apoptosis ratio) was significantyl higher in group 1 patients than group 2 patients (2,0+/-2% versus 0,32+/-0,6%). Tubulointerstitial regenerative ratio (=tubular proliferation/interstitial proliferation ratio) was significantly higher in controls than in patients (3,4+/-1,9 versus 1,52+/-0,8, p= 0,01). In addition , it was significantly increased in group 1 patients when compared with those group 2 patients (1,89+/-0,8 versus 0,73+/-0,2 p=0,001). Since 17 patients presented with postinfectious proliferative glomerulonephritis, which is known to exhibit better course, we also evaluated those parameters in patients with postinfectious proliferative glomerulonephritis separately. We found statisticaly significant differences only in the tubulointerstitial regenerative ratio, which was higher in postinfectious cases when compared with those in other cases [1,60 interquartile rang (IQR) 1,54 versus 1,22 IQR 1,26; respectively,

p=0,003]. In conclusion, tubular proliferative index and tubulointerstitial regenerative ratio might be useful parameters for predicting final functional outcome in acute proliferative glomerulonephrits. Further studies, however, are still needed to clarify the importance of these histoptahological parameters.

10. Glomerular plasmin-like activity in relation to nephritis-associated plasmin receptor in acute poststreptococcus glomerulonephritis

Oda T, Yamakami K, Omasu F, et al.

J Am Soc Nephrol 2005; 16(1): 247-54.

A nephritogenic antigen for acute poststreptococcal glomerulonephritis (APSGN) was isolated recently from group A streptococcus and termed nephritis-assocated plasmin receptor (NAP1r). In vitro experimental data indicate that the pathogenic role of NAP1r occurs through its ability to bind to plasmin and maintain its proteolytic activity. However, the mechanism whereby this antigen induces glomerular damage in vivo has not been fully elicidated. Renal biopsy tissues from 17 patients with APSGN, 8 patients with rapidly progressive glomerulonephritis, and 10 normal kidney were analyzed in this study. Plasmin-like activity was assessed on cryostat sections by in situ zymography with a plasmin-sensitive synthetic substrate. Serial section were simultaneously assessed for NAP1r deposition by immunoflurescence staining. Glomerular plasmin-like activity was absent or weak in normal and in patients with rapidly progressive glomerulonephritis, although tubulointerstitial activity was occasionally detected. Prominent plasmin-like activity was found in patients who had APSGN and in whom glomerular NAPlr was positive, whereas it was absent or weak in patients who had APSGN and in whom glomerular NAP1r was negative. The distribution of glomerular plasmin-like activity was identical to that of NAP1r deposition but was generally different from that of fibrin(ogen) deposition as assessed by double staining. The activity was abolished by the addition of aprotonin to the reaction mixture but was not altered by the addition of a matrix metalloproteinase inhibitor, a cysteine protease inhibitor, or inhibitors of plasminogen activators. Thus, upregulated glomerular plasmin-like activity in relation to NAP1r deposition in APSGN was identified. This result supports the nephritogenic chararter of NAP1r and offers insight into the mechanism whereby this antigen induces nephritis.

11. Fibrillary and immunotactoid glomerulonephritis: report of a case and review of the literature

Mendez FJ, Nunez MM, Garcia MA, et al.

An Med Interna 2005; 22 (1): 35-8.

We describe the case of a 67-year-old female with nephrotic syndrome and rapidly progressive renal failure. The nephropathy was characterized by deposits of randomly oriented fibrills with a diameter of about 18-20 nm on electron microscopy. Immunofluorescence microscopy was performed and there was no staining for immunoglobulins and complement. We diagnosed atypical fibrillary

glomerulopathy with absence of immune deposuts. The patient developed end-stage renal failure rapidly. We rivew in the literature new clinical and pathogenetic features related to fibrillary and immunotactoid glomerulopathy.

12. HCV associated glomerulopathy in Egyptian patients: clinicopathological analyisis

Sabry A, El-Agroudy A, Sheashas H, et al.

Virology 2005; 334 (1): 10-6.

Summary: See Part 2/EP/15.

13. Vascular endothelial growth factor (VEGF-C1)-dependent inflammatory response of podocytes in nephrotic syndrome glomerulopathies in children: an immunohistochemical approach

Ostalska-Nowicka D, Zachwieja J, Nowicki M, et al.

Histopathology 2005; 46(2): 176-83.

Aims: To analyse expression and distribution of VEGF-C1, podocalyxin and synaptopodin within renal tissue in nephrotic syndrome glomerulopathies in children. Methods and Results: Renal biopsies performed at the time and in the manner recommended by the World Health Organization. The study group consisted of submicroscopic glomerulonephritis (n=10), diffuse mesangial proliferation (n=14) and focal segmental glomerulosclerosis (n=5). The control tissue consisted of macroscopically normal appearing cortex taken from kidneys resected for localized neoplasms (n=3). Material for immunohistochemistry was fixed in Bouin's solution and embedded in paraffin. Indirect immunohistochemistry using monoclonal anti-human antibodies directed against VEGF-C1, podcalyxin and synaptopodin was employed. The distribution of markers was quantified by computerized image analyisi. In non-sclerosed glomeruli (within podocyte cytoplasm), VEGF-C1 was more expressed in podocytes of all groups (<0,0002), while the distribution of synaptopodin was less expressed in all groups (p<0,0002). There was no statistical difference between all groups in the expression of podocalyxin. Conclusion: The increased permeability of the filtration barrier in steroid-resitant glomerulopathies may be a consequence of subcellular changes in podocytes resulting from decreased expression of synaptopodin. Moreover, impaired permeability of endothelium could be secondary to increased expression of podocyte-derived VEGF-C1.

14. Expression of Fas, Bc1-2 and p53 molecules in glomerulonephritis and their correlations with clinical and laboratory findings

Uguz A, Gonlusen G, Ergin M, et al.

Nephrology (Carlton) 2005; 10 (3): 311-6.

Summary: Background and Aim: Apoptosis plays a crucial role in glomerulonephritis (GN) as a regulatory mechanism and is controlled by various molecules including Fas antigen, Bcl-2 and p53 oncoproteins. The aim of the present study is to evaluate the correlation between the expression of these molecules and clinical and laboratory data in different types of GN. Results: The expression of Fas antigen, Bcl-2 and p53 protein in five normal human kidney specimens and 55 tissues from patients with several types of GN were examined by immunohistochemistry and correlated with clincal and laboratory findings. The number of Fas-positive intraglomerular cells was significantly increased in proliferative GN when compared with non-proliferative cases. Numbers of Bcl-2- and p53-positive cells in proliferative GN were not different from the non-proliferative cases and there was no correlation between the changes is Fas, Bcl-2 and p53 themselves. Significant correlation of expression of these molecules with clinical and laboratory findings was not found, except between p53 and blood urea nitrogen levels. Conclusion: Apoptosis is a complex molecular process and the results of the present study should be supported with other methods to understand wheter apoptosis contributes to progression or resolution of GN. Increased glomerular expression of Fas, Bcl-2 and p53 molecules in all types of GN might contribute new therapeutic approaches by modulating the expression of these molecules.

15. Immunohistochemical detection of immunoglobulins and complements in formaldehyde-fixed and paraffin-embedded renal biopsy tissues: an adjunct for diagnosis of glomerulonephritis

Chowdhury AR, Ehara T, Higuchi M, et al.

Nephrology (Carlton) 2005; 10 (3): 298-304.

Summary: Background: The present study was undertaken to demonstrate the deposition of immunoglobulins or complements in formaldehyde-fixed and paraffin-embedded renal biopsy tissues throght the unmasking of antigens with microwave treatment plus protease digestion or trypsin digestion. Methods: Biposy samples from patients with IgA nephritis (n=7), lupus nephritis (7), membranous nephropathy (7), and mesangiocapillary glomerulonephritis (3) were used. Antigen unmasking was performed with (i) microwave treatment plus protease digestion for 10, 30 or 60 min, or (ii) digestion with 0,25% trypsin for 60 or 120 min. Results: Microwave treatment plus protease digestion for 30 or 60min and trypsin digestion for 120min provided good results for the unmasking of immnunoglobulins in glomeruli with structural preservation. The IgA deposits in IgA nephritis and IgG in lupus nephritis and membranous nephropathy were revealed in more than 80% of cases by both pretreatments. Microwave treatment plus protease digestion for 30min revealed the deposition of C3 in all cases of mesangiocapillary glomerulonephritis and lupus nephritis and was superior to trypsin digestion. Characteristic patterns of C3 deposition were observed for these forms of glomerulonephritis, although C3 deposition in membranous nephropathy were detected only 50% of cases. It was not possible to unmask all of the antigens in the glomeruli, especially those with weak immunoflurescence. Conclusion: Microwave treatment plus protease digestion is effective for the unmasking of antigens in paraffin sections and as useful for the diagnosis of immune-mediated glomerulonephritis as trypsin digestion.

16. Hepatitis C virus RNA and core protein in kidney glomerular and tubular structures isolated with laser capture microdissection

Sansonno D, Lauletta G, Montrone M, et al.

Clin Exp Immunol 2005; 140(3): 498-506.

The role of hepatitis C virus (HCV) in the production of renal injury has been extensively investigated, though with conflicting results. Laser capture microdissection (LCM) was performed to isolate and collect glomeruli and tubules from 20 consecutive chronically HCV-infected patients, namely 6 with membranoproliferative glomerulonephritis, 4 with membranous glomerulonephritis, 7 with focal segmental glomerulosclerosis and 3 with IgA nephropathy. RNA for amplification of specific viral sequences was provised by terminal continuation methodology and compared with the expression profile of HCV core protein. For each case two glomeruli and two tubular structures were microdissected and processed. HCV RNA sequences were demonstrated in 26 (65%) od 40 glomeruli, but in only 4 (10%) of the tubules (P<0,05). HCV core protein was concomitant with viral sequences in the glomeruli and present in 31 of the 40 tubules. HCV RNA and/or HCV core protein was found in all four disease types. The immunohistochemical picture of HCV core protein was compared with the LCM-based immunoassays of the adjacent tissue sections. Immune deposits were detected in 7 (44%) of 16 biopsy samples shown to be positive by extraction methods. The present study indicates that LCM is a reliable method for measuring both HCV RNA genomic sequences and HCV core protein in kidney functional structures from chronically HCV-infected patients with different glomerulopathies and provides a useful baseline estimate to define the role of HCV in the production of renal injury. The different distribution of HCV RNA and HCV-related proteins may reflect a peculiar 'affinity' of kidney microenvironments for HCV and point to distinct pathways of HCV-related damage in glomeruli and tubules.

17. Relative interstitial volume is correlated with renal function even in non-representative biopsy

Okon K, Szumera A, Kuzniewski M, et al.

Pol J Pathol 2005; 56 (1): 9-13.

An useful renal biopsy should be representative, that is should conatin a sufficient number of glomeruli. However, a non-representative biopsy could possibly provide some information. The aim of the study was to evaluate the relationship between interstitial expansion, glomerular sclerosis and renal function in such material. The material consisted of 28 renal biopsies containing less than 5 non-sclerosed glomeruli. For each case the percentage of completely sclerosed glomeruli was recorded. The relative interstitial volume was evaluated by point counting method. Clinical data as sex, age, serum creatinine and urea levels were included into analyis. The mean percentage of completely sclerosed glomeruli was 39,6%; mean relative interstitial volume was 29,8%. Creatinine level was strongly correlated to relative interstitial volume (R=0,70), but the correlation of craetinine level to percentage of sclerosed glomeruli was much weker (R=0,38). The relationship between interstitial expansion and renal function is seen also in deficient biopsy material. The

correlation of renal function with interstitial expansion is stronger the correlation of renal function with glomerular sclerosis. These findings can indicate that the better representation is responsible for stronger prognostic impact of interstitial lesions

18. Study of the biopiesed nephrotic syndrome for 20 years in the Cadiz Bay Area: histological correspondes, renal prognosis and clinical prognostic factors

Quires PL, Ceballos M, Remon C, et al.

Nefrologia 2005; 25 (2): 147-54.

Aims: To analyse the histological correspondence, the renal survival and the clinical prognostic factors in the nephrotic syndrome for more than 20 years in the our environment as well as the influence of the nephrotic proteinuria in the renal survival in the different histological particular types of glomerulonephritis.Patients and Methods: Among the 542 primary and secondary glomerulonephritis

Diagnosed by kidney biopsy for two decades in the Cadiz Bay Area, we selected 242 patients whose clinical presentation and the biopsy indication was the nephrotic syndrome. Statistical methods: means +/- typical deviation, percentiles, percentages, Kaplan-Meier curves, long-rank test, student's t-test, chi-square analysis and Cox prportional hazards model test. Results: 242 patients with nephrotic syndrome (44,68% out of the total glomerulonephritis), average age of 39,15+/- 18 years old. Average proteinuria 6,75 +/- 4,53 g/day. Etiology: membranous nephropathy (33,85%), lupus nephritis (14,46%), minimal change disease (11,57%), focal segmental glomerulosclerosis (10,33%), renal amyloidosis (9,95%). 38%, 45%, 63% and 72% of the patients with nephrotic syndrome developed to the End-Stage Renal Disease and starting point of dialysis in 5, 10, 15 and 20years respectively. After the multivariate model, the age older than 60 years vears old, then high levels of proteinuria and the coexistence with hypertension or renal failure, in the moment of diagnosis, showed to be independent clinical prognostic factors. The nephrotic proteinuria had a negative influence in the prognosis in the different histological types, especially in the IgA nephropathy and the lupus nephritis. Conclusions: The nephrotic syndrome is the main indication of the renal biopsy in our environment. In general, as an independent gorup, its development is slowly progressive to the End-Stage Renal Disease, having the possibility of being also conditioned by certain clinical factors present in the moment of biopsy. The presence of nephrotic proteinuria is also a negative factor in the progression in many of the glomerulonephritis.

19. Immunotactoid glomerulopathy with microtubular deposits, with reference to the characteristics of Japanese cases

Fukuda M, Morozumi K, Oikawa T, et al.

Clin Nephrol 2005, 63 (5): 368-74.

We present the case of a 69-year-old man with nephrotic syndrome and renal insufficiency, who developed lobular glomerulonephritis. An electron microscopy examination of a renal biopsy showed microtubular structures of 24 nm in diameter in the subendothelial space and the paramesangial area. These deposits were PAS-positive and Congo red negative, and revealed predominantly positive staining for kappa light chain. There was no evidence of disease with highly organized glomerular deposits, such as amyloidosis, cryoglobulinem, systemic lupus erythematosus or paraproteinemia. Therefore, the patient was diagnosed to have immunotactoid glomerulopathy (ITG). During a seven-year course he has not developed any disease known to be associated with organized glomerular immune deposist. Hence, we believe IG occured as a primary glomerular disease in this case. We also highlight cases of ITG with microtubular deposits that have been reported in Japan, compare these cases to previous reports, and show that the characterictics of the Japanese cases are male predominance; a high incidence of membranoproliferative glomerulonephritis (MPGN); a low incidence of monoclonal gammopathy and hematological malignancies and higher incidence of hypocomplementemia.

20. Renal effects of non-steroidal anti-inflammatory drugs and selective cyclooxygenase-2 inhibitors

Cheng HF, Harris RC.

Cur Pharmaceut Design 2005; 11: 000-000.

Abstract: Nonsteroidal antiinflammatory drugs (NSAIDs) are one of the most commonly used medications worldwide to inhibiting COX activity for the treatment of pain and inflammation. Their nephrotixicity has been well documented. With the development and clinical implementation of new COX-2 inhibitors, the safety, including the effects on renal function and blood pressure, is attracting increasing attention. In the kidney, COX-2 is constitutively expressed and is highly regulated in response to alterations in intravascular volume. COX-2 metabolites have been implicated in mediation of renin release, regulation of sodium excretion and maintance of renal blood flow. Similar to conventional NSAIDs, inhibition of COX-2 may cause edema and modest elevations in blood pressure in a minority of subjects. COX-2 inhibitors may also ecacerbate preexisting hypertension or interfere with other antihypertensive drugs. Occasional acute renal failure has also been reported. Caution should be taken when COX-2 inhibitors are prescribed, especially in high-risk patients (including elderly and patients with volume depletion). Recently, agents with combined lypooxygenase/COX inhibition and agents that combine NSAIDs with a nitric oxide (NO) donor have been reported to reduce adverse renal effects.

21. Thrombotic thrombocytopenic purpura associated with rapidly progressive lupus nephritis: report of two cases

Kapoulas S, Liakos S, Karkavelas G, et al.

Clin Nephrol 2005; 63 (4): 297-301.

Abstract: There are a few reported cases in the literature of thrombotic thrombocytopenic purpura (TTP), associated with systemic lupus erythematosus (SLE). We describe two cases of TTP which have been represented during rapidly progressive lupus nephritis, with grand-mal seizures, thrombocytopenia and microangiopathic hemolytic anemia. Both cases were treated with hemodialysis, plasma exchange, corticosteroids, cyclophosphamide and intravenous gamma-globulin. In both cases the TTP was improved but not the renal function. Further experience is needed to determine whether intensive and prompt treatment with plasma exchange, corticosteroids and chemotherapy leads to a favorable outcome, in cases of TTP associated with SLE.

22. Association between polymorphisms of the renin-angiotensin system and more severe histological forms of lupus nephritis

Sprovieri SR, Sens YA, Filho M.

Clin Nephrol 2005, 64 (1): 20-27.

Abstract. Aims: Pathogenesis of the lupus nephritis (LN) has not been fully understood. The renin-angiotensin system (RAS) is implicated in various immunological and non-immunological phenomena, and the polymorphism of the RAS genes has been associated with cardiovascular and renal disease onset and outcome. Therefore, we evaluated the possible association between the polymorphism of the RAS genes and the development of different types of histological lesions of LN in Brazilian patients. Methods: 72 LN patients and 65 healthy subjects (sex-and ethnic-matched) were enrolled and compared in this study. Following the extraction of genomic DNA from the lukocytes of the peripheral blood, the genotypes of the angiotensin converting enzyme (ACE I/D), of the angiotensinogen (AGT M235T) and the angiotensin II type 1 receptor (AGTR1) A1166C) were determined by polymerase chain reaction. The renal lesions of the patients with LN were classified by the histological findings according to the WHO criteria. In addition, the activity and chronicity indicies were used to assess the severity of renal involvement. Results: Among the 72 patients with LN, there were17 class II, 8 class III, 40 class IV and 7 class V, according to the WHO criteria. Individuals with the III and IV clesses of LN showed a significantly increased DD genotype frequency of ACE I/D genes when compared to the control group (48% vs. 27,7%, x^2=4,885, df=1,, p=0,0442). No difference was found in the distribution of the AGT M235T and AGTR1 A1166C genotype frequencies among the LN of the different histological classes and healthy controls. There was no association between genetic polymorphism of ACE, AGT235T and AGTR1 A1166C and susceptibiliy to lupus nephritis, nor histological activity and chronicity indicies in renal biopsy among the patiens studied. Conclusions: This study suggest that the DD genotype of the ACE may be associated with the development of the more severe histological forms of lupus nephritis.

23. Inhibition of protein kinase CK2 prevents the progression of glomerulonephritis

Yamada M, Katsuma S, Adachi T, et al.

Proc Natl Acad Sci USA 2005, 102 (21): 7736-41.

Glomerulonephrits (GN) is a progressive inflammation that may be caused by a variety of underlying disorders. It is the primary cause of chronic renal failure and end-stage renal disease, which require dialysis and transplantation worldwide. Immunosuppressive therapy has been used to treat GN clinically, but this treatment has had insufficient therapeutic effects. Here, we show that protein kinase CK2 is key molecule in the progression of GN . cDNA microarray analysis identified CK2alpha, the catalytic subunit of CK2, as a GN-related, differentially expressed gene. Overexpression of CK2alpha was noted in the proliferative glomerular lesions in rat GN models and in renal biopsy specimens from lupus nephritis or IgA nephropathy patients. Administration of either antisense olygodeoxynucleotide against CK2alpha or low molecular weight CK2-specific inhibitors effectively prevented the progression of renal pathology in the rat GN models. The resolution of GN by CK2 inhibition may result from its suppression of extracellular signal-regulated kinase-mediated cell proliferation, and its suppression of inflammatory and fibrotic process that are enhanced in GN. Our results show that CK2 plays a critical role in the progression of immunogenic renal injury, and therefore, CK2 is a potential target for GN therapy.

24. Lupus erythematosus proliferative glomerulonephritis in fetus

Daikha-Dahmane F, Bault JP, Molina-Gomes D, et al.

Lupus 2005; 14 (4): 328-30.

We report the case of a fetus with proliferative glomerulonephritis in the context of maternal systemic lupus erythematosus (SLE). The pattern of the renal lesions correspond to the class III of revisited WHO classification of glomerulonephritis in SLE. Amniotic fluid analysis showed a high level of albumin and the presence of anti-Ro and anti-DNA antibodies that were possibly responsible for the renal injury.

25. HIV-associated immune complex glomerulonephritis with „lupus-like" features: a clonicopathological study of 14 cases

Haas M, Kaul S, Eustace JA.

Kidney Int 2005, 67 (4): 1381-90.

Summary: See P2/ET/2.

26. Apoptosis and proliferating cell nuclear antigen in lupus nephritis (class IV) and membranoproliferative glomerulonephritis

Kirim S, Tamer T, Saime P, et al.

Ren Fail 2005; 27 (1): 107-13.

Background: The role of apoptosis in the pathogenesis of renal diseases has not been clearly estatabilished. Proliferating cell nuclear antigen (PCNA) is also a proliferation marker. In this study, we investigated the relationship between clinical course and PCNA apoptosis on baseline renal biopsy in patients with lupus nephritis (LN) and membranoproliferative glomerulonephritis (MPGN). Methods: Thirty-nine patients with proliferative glomerulonephritis LN [21] and MPGN [18] were included in this study. PCNA and apoptosis on renal biopsies were detected by immunohistochemical and terminal deoxnucleotidyl transferase mediated dUTP nick end labeling (TUNEL) methods, respectively. We calculated the ratios of intraglomerular apoptotic cells and PCNA positive cells per glomeruli, and total number s of apoptotic tubular cells and PCNA positive tubular cells among the 100 tubular cells, and PCNA positive cell and apoptitic cell on two different tubulointerstitial areas (40x10). Results: in LN: Apoptotic indexes of glomerulus and tubulus were 1,08+/- 0,49 and 3,71+/-1,38, respectively. PCNA positivities were found at 16,76+/-11,34%, 46,57+/-22,54%, and 40,28+/-23,14% on glomerulus, tubulus, and interstitium, respectively. The activity index was 11,23+/-3,41, and the chronicity index was 3,81+/-1,99. In MPGN: Apoptotic indexes were found at 0,83+/-0,25 and 3,55+/-1,75 on glomerulus and tubulus, respectively. PCNA positivities were found at 21,33+/-18,42%, and 35,5+/-25,99%, and 34,66+/-26,84% on glomerulus, tubulus and interstitium, respectively. In controls, apoptosis was not found. In LN: PCNA positivity on tubulus and interstitium were correlated with the activity index ($r=0,768$, $p<0,001$, $r=0,721$, and $p<0,001$, respectively). Glomerular PCNA and apoptosis on interstitium and glomerulus were not correlated with the activity index. The activity index also was not correlated with creatinine clearence and daily proteinuria ($p=0,35$ for both). At the end of the first year, patients with recovered or stabilized renal function had higher interstitial and tubular PCNA than others in G1 and G2. Conclusin:It can be said that expression of PCNA on renal biopsy was correlated with activity indexes in LN. PCNA may be a prognostic indicator in MPGN and LN. However, apoptosis does not have a predictive value for MPGN and LN.

27. Quantitative morphometry of lupus nephritis: the significance of collagen, tubular space, and inflammatory infiltrate.

Hunter Mg, Hurwitz S, Bellamy CO, et al.

Kidney Int 2005; 67 (1): 94-102.

Background: Lupus nephritis encompasses a wide range of parenchymal injuries and severity. Better predictors of outcome are needed for patients newly diagnosed with lupus nephritis., so that an appropriated management strategy may be selected. Methods: A single-center cohort of first renal biopsies for lupus nephritis was chosen. Histologic sections of whole biopsy cores were stained with picro-Sirius red, and light microscopic image (x100) were digitally captured. Using a simple, freely available software package, the cortex of each biopsy was evaluated for four different parameters: area occupied by nuclei, intratubular space, fibrillary collagen, and colagenous matrix. Clinical and laboratory data were collected retrospectively from the time of biopsy and throghout

follow-up. Results: A hihg nuclear index at initial biopsy correlated with clinical parameters of disease activity, at the time of biopsy. High collagen matrix index predicted both relapse and progression to end-stage renal disease (ESRD). The fibrillary collagen index predicted progressive disease as assessed by doubling of serum creatinine, and relapse. Increased tubular space also predicted progressive disease as determined by doubling of creatinine and renal death. Conclusions: A simple automated system for objectively scoring biopsies of lupus nephritis predicts renal survival and may provide a useful adjunct to guide patient management.

28. Glomerular podocytopathy in patients with systemic lupus erythematosus

Kraft SW, Schwartz MM, Korbet SM, et al.

J Am Soc Nephrol 2005; 16 (1): 175-9.

A series of patients with systemic lupus erythematosus (SLE) and proteinuria were studied to determine whether nephrotic-range proteinuria was associated with diffuse epithelial cell foot process effacement in the absence of peripheral glomerular immune aggregate deposition. Biopsies from patients with kown or suspected SLE and a histologic diagnosis of (1) normal by light microscopy, (2) mesangial proliferative glomerulonephritis, or (3) focal segmental glomerulosclerosis were studied. Biopsies were excluded when they demonstrated endocapillary proliferation or necrosis by light microscopy or electron-dense glomerular basement membrane deposits by electron microscopy. Patients were required to fulfill four of 11 American Rheumatologic Association criteria for the diagnosis of SLE, and proteinuria could not be associated with nonsteroidal anti-inflammatory drug use. Eighten biopsies were studied, eight from patients with nephrotic-range proteinuria (>/=3g/d) and 10 from patients with non-nephrotic proteinuria. The time from diagnosis of SLE to biopsy was shorter for nephrotic patients that for non-nephrotic patients: Seven of eight biopsies from nephrotic patients demonstrated at lest 80% foot process effacement, whereas no biopsy from non-nephrotic patient exhibited >20% effacement. There were no other significant pathologic differences between the nephrotic and non-nephrotic patients. The single common morphologic feature associated with nephrotic proteinuria was diffuse visceral epithelial cell foot proces effacement. It is concluded that the development of nephrotic-range proteinuria in patients with SLE without peripheral immune aggregate deposition or endocapillary proliferation on renal biopsy is more likely a manifestation of SLE than the coexistence of idiopathic minimal-change glomerulopathy and SLE.

29. Kidney disease associated with primary antiphospholipid syndrome: clinical signs and histopathological features in an experience of five cases

Saracino A, Ramunni A, Pannarale G, et al.

Clin Nephrol 2005; 63 (6): 471-476.

Abstract: Background: The primary antiphospholipid syndrome (PAPS) is characterized by

presence of circulating antiphospholipid antibodies, clinically associated with blood hypercoagulability. Renal involvement in course of PAPS is very frequent, although the true prevalence of PAPS-correlated kidney disease is difficult to estimate. Material and Methods: We reviewed 270 consecutive renal biopsies examined in our Nephrology Division of Bari University Hospital between 1998- and 2004 to identify those performed in patients with PAPS. Results: We identified five biopsies performed in patients with PAPS. In three patients the diagnosis of PAPS was made in onset of the kidney disorder, while in the other two cases the initial diagnosis was primary focal segmental glomerulosclerosis (FSGS). In these caes the subsequent finding of positive antiphospholipid antibodies reoriented the diagnosis toward PAPS.correlated nephropathy. The clinical onset of kidney disease consisted of acute renal failure in three patients and urinary abnormalities in the other two. Histological examination of renal biopsies showed vascular lesions (intimal fibrous hyperplasia, arteriolar hyalinosis, double outline of the capillary walls) in four patients. Focal segmental glomerulosclerosis was present in four patients, two of whom showed double outline of the capillary walls. All patients had tubulo-interstitial lesions, while immunofluorescence was positive in only two patients. All patients preserved stable renal function throughout follow-up (mean value: 10.6 years, range 40 months – 24 years). The prevalence of PAPS-correlated nephropathy in our population was 1,85%. Conclusion: Our data confirm that PAPS-associated nephropathy has slow progression and rarely leads to end-stage renal failure. The prevalence of PAPS-correlated nephropathy is likely underestimated beacuse some patients with diagnosis of primary focal sclerosis may actually be affected by PAPS.