Abstract of the case

Case 1

A case of Henoch-Schönlein purpura nephritis with marked hump-like subepithelial deposition.

Akiko Fujii,
Department of Pathology, Mitsui Memorial Hospital

A 72-year old woman with was referred to our hospital due to acute kidney injury. The patient had abdominal pain, melena and purpura in extremities for a month and was first admitted to the regional hospital. She was transferred to our hospital because of deep duodenal ulcers detected by gastrointestinal endoscopy, suggestive of Henoch-Schönlein purpura with acute kidney injury and cutaneous lesions.

On admission, she was almost anuric and demonstrated systemic edema. Urinalysis showed a 3+ test for protein with many red and white blood cells. Blood test showed creatinine concentration of 2.3mg/dl and Hb 12.0g/dl. Total protein and albumin were decreased to 4.7g/dl and 2.1g/dl, respectively. Serum C3, C4 and CH50 were within normal limits. ANA was positive with a titer of 1:40. Anti-glomerular basement membrane antibody, anti-neutrophil cytoplasmic antibody, rheumatoid factor were negative. Anti streptolysin O antibody titer was elevated to 664IU/ml. Renal biopsy was performed on the following day. Microscopically, renal tissue revealed marked neutrophil infiltration in glomeruli. Hyalinous deposits distributed mainly on subepithelial and subendothelial region. There was also severe tubulointerstitial neutrophil infiltration. Immunofluorescent study showed mesangial IgA deposits. IgG and other complement components were also positive in the same distribution. Electron microscopy showed hump-like subepithelial electron dense deposits. After first course of steroid pulse therapy, duodenal ulcer and renal function improved and purpura disappeared immediately. However, nephrotic-range proteinuria and refractory edema persisted after administrating a second steroid pulse therapy. The second biopsy was performed 67 days after the first biopsy. Microscopically, glomerular neutrophils and hump-like deposits decreased markedly, although tubulointerstitial damage still remained. After second

biopsy, urinary protein excretion slowly decreased to 1.0g/day. Here we report an unusual case of Henoch-Schönlein purpura nephritis with marked hump-like deposits mimicking acute glomerulonephritis.

Case 2

Varicella-zoster virus infection and acute kidney injury

Gu-ming Zou,

The Center of Nephrology, Beijing China-Japan Friendship Hospital, Beijing China

A 15-years-old boy who had good health until ten weeks prior to hospital admission, when he began to experience a fever, up to 39.5□, with herpes on face, tunk and extremities. He was diagnosed as chickenpox and was treated with Ganciclovir and Ribavirin in local hospital. Nine weeks prior admission, he noticed gross hematuria and edema and his urinary volume decreased to 500ml per day. Meanwhile he suffered from hypertension of 145/100mmHg. Urinary analysis revealed proteinuria and hematuria. Serum creatinine level was 212μmol/L and blood albumin was 16.8g/L. The diagnosis of nephrotic syndrome and acute renal failure was made. Eight weeks prior admission, his serum creatinine increased to 451.9μmol/L and blood albumin decreased to 16g/L. Intravenous methylprednisolone was administered at a dose of 200mg/d for six days and followed by oral prednisone at 1mg/d/kg. He also received hemodialysis in local hospital. Three weeks before admission, leflunomide was added. After above treatment, his renal function recovered gradually, but no improvement in edema and hematuria. During hemodialysis, he experienced epilepsy

twice without obvious reasons.

The patient was admitted to the China-Japan Friendship Hospital on September 21, 2008. His blood pressure was 148/114mmHg and temperature of 36.7□. The numerous and small scabs in face and other sites were observed. Edema was found at both lower extremities and scrotum. Laboratory investigation found hemoglobin 141g/L, blood albumin 17g/L, cholesterol 9.3mmol/L. serum creatinine 65μmol/L, creatinine clearance rate 47.6ml/min; his serum complements C3 was decreased at 45.1mg/dl (normal 70~128mg/dl); urinary protein 3.85~4.22g/d, urinary osmotic pressure 725mOsm/kg; Antistreptolysin O titer and C reactive protein were normal. Autoantibodies (ANA, ANCA, anti-GBM) were negative. Urinary sediments showed deformed RBC and RBC casts. Ultrasonography displayed normal size of both kidneys.

The patient experienced an episode of epilepsy on the third day after admission. Electroencephalogram showed widespread continuous slow waves at occiput and spikes at frontal part occasionally. Neurologist's consultation was that intracalvarium lesion and possible secondary epilepsy caused by the Varicella-zoster virus infection.

The clinical diagnosis: Nephrotic syndrome, Acute Kidney Injury

Positive varicella-zoster virus (VZV) specific IgM antibodies were detected.

The percutaneous renal biopsy was performed on the fourth day after admission.

Immunofluorescence: IgG(2+-3+), IgA(2+-3+), IgM(2+-3+), C3(3+),

C1q(1+-2+), Fibrin(1+-2+), along glomerular mesangium and capillary wall. Light microscopy: glomerular mesangial cells and endothelial cells diffuse proliferation, podocyts proliferation and vacuolar degeneration, fuchsinophil material deposit in mesangium and some at subepithelial site, tubular epithelial cells show severe vacuolar and granular degeneration and focal broken, some smudgy inclusions occur. Electron microscopy: electron dense deposit was noticed on mesangium, subepithelial, subendothelial sites. Furthermore, virus-like particles and viral inclusions could also be found.

Special staining for viral inclusions (Methylene Blue and Eosin) was further investigated. VZV antigen and RNA transcript were identified in glomerular and tubular cells by immunohistochemical staining and in situ hybridization of renal tissues, respectively.

The pathological diagnosis: VZV-related endocapillary proliferative glomerulonephritis with podocyte proliferation and severe renal tubular injury.

Case 3

Histological alteration of recurrent dense deposit disease in renal allograft in a series of allograft biopsy.

Kumi AITA[1], Kazunari TANABE[2], Keiko SAI[2], Shigeru HORITA[3] and Yutaka YAMAGUCHI[4]

[1] Division of Pathology, Sayama hospital, [2]Department of Urology, Tokyo Womens Medical Univertisy, [3]Kidney center, Tokyo Womens Medical Univertisy, [4]Department of Pathology, Kashiwa Hospital, Jikei University

A 37-year-old man had living-related renal transplantation from his mother after 2 years of hemodialysis. He was originally diagnosed as MPGN type II (dense deposit disease, DDD) at 18 years old, and had been presented marked hypocomplementemina thereafter. He received three sessions of

DFPP prior to transplantation and started initial immunosuppressive therapy consisted of MP, MMF, FK506 and basilituximab. Sixteen days after transplantation, he showed proteinuria and increased serum creatinine level.

The allograft biopsy showed marked endocapillary proliferative GN with numerous subepithelial humps. The immunofluorescence study revealed single intense granular deposits of C3 along the glomerular basement membrane (GBM). Electron microscopy revealed a large number of electron dense deposit in both the subendothelial area of the GBM and the mesangium. The recurrence of DDD was suspected and he received MP pulse therapy. Despite the treatment, his proteinuria increased gradually. The second allograft biopsy two months post-transplant showed similar glomerular lesions as the previous biopsy, except for slight amelioration of endocapillary hypercellularity. The electron microscopy revealed dense, ribbon-like material along the basement membrane. Although his proteinuria ameliorated temporary after plasmapheresis, it returned to the previous level once plasmapheresis was stopped.

Eight months posttransplant, he was treated with rituximab at 100mg, and CD19+ B cells depleted from 10% to 1% of lymphocytes after treatment. Since he was recovered from leukopenia, MMF was restarted with 500mg/day 19 months after transplantation. Twenty-three months after transplantation, the complement levels suddenly increased and after 23 months posttransplant, proteinuria disappeared completely. The 3rd allograft biopsy showed marked mesangial hypercellularity with thickening of GBM. Subepitheial deposits were also remained focally. Immunofluorescence study showed less C3 staining area than the previous biopsies.

<Summary> This is the precious case of recurrent DDD in renal allograft. We could purchase the histological alterations in a series of biopsy and it's of great help to understand the natural course of DDD in native kidney. Despite the clinical remission, glomerular lesions were remained with substantial deposition. It's also intriguing to assess the discrepancy between the clinical course and histological features.

Case 4

Exudative glomerulitis with mesangial deposits

Muyi Guo

Dept. of Pathology, Shanghai medical college , Fudan university

Patient: A 51-year-old female native Taichang city, Jiangsu province of P.R.China, complained of thirsty, weight loss, polyuria with proteinuria for more than 2 months, and then admitted to local hospital for renal biopsy. Only slight diabetic changes (I ^{0}) of the retina were seen on physical examination. Clinical laboratory examinations disclosed that serum glucose of the patient was elevated to 10.0g/L, urine protein excretion was 1.5g/ 24 hours, urine glucose was 1+～2+, and serum immunoglobulins (IgG, IgA, IgE, and IgD) , complements (CH50 , C3) , routine tumor markers and Scr, BUN were all normal.

Pathological changes of renal biopsy

LM: 23 glomeruli , 3 sclerotic, were seen in the glass slides of renal biopsy. Glomerular changes include irregular thickening of capillary wall, GBM, with slight proliferation of the mesangial cells , increase of the mesangial matrix and a few neutrophil infiltration..Dissipated neutrophil and lymphocyte infiltration in renal interstitium, and mild hyalinosis or intimal fibrosis of the arterioles were found.

IF : Only positive C3 deposition was mainly seen in glomerular mesangial areas.

EM: Electron dense deposits with increased mesangial matrix were predominantly located in glomerular mesangial areas, associated with a little deposition in the lamina densa of GBM

Goal of discussion: To determine the pathological diagnosis.

Case 5

Enlargement of the lymph nodes and renal failure

Gan Liu

The Department of Nephrology, Peking University First Hospital, Beijing China

A 33-year-old Chinese man was presented with cervical lymph node swelling for 7 months and macrohematuria for 1 month. Seven months before admission, he was found to have cervical lymph node swelling; a presumed diagnosis of lymphadenitis was made and he was treated with cephalosporin, without any effect. One month before admission, macrohematuria and generalized edema developed. His medical history was notable for a 6-month history of fever in 2003 without clear etiology. On admission to the hospital, his temperature was 37.0°C, blood pressure was 140/90 mm Hg, and heart rate was 80 beats per minute. Bilateral, small, rubbery lymph nodes were palpable in the cervical, axillary, and inguinal regions. Examination of the lungs showed mild diminished breath sounds in the left side. The rest of the examination was unremarkable.

Blood testing revealed the white-cell count was 9400/mm^3, hemoglobin level 6.9 g/dL (69 g/L), and platelet count 84,000/mm^3. Erythrocyte sedimentation rate was 107 mm/hr. Urinalysis showed protein in 4＋ and urinary sediment 100 to 150 red cells per 10×40 field but no granular casts. Urine protein excretion was 7.25 g/24 hrs. Serum total protein was 7.0 g/dL (70 g/L), albumin, 2.9 g/dL (29 g/L), aspartate aminotransferase, 38 IU/L, alanine aminotransferase, 20 IU/L; serum bilirubin was 0.7 mg/dL (12 μmol/L), serum creatinine, 1.3 mg/dL (118 μmol/L), urea nitrogen, 19.6 mg/dL (7 mmol/L). Serum protein electrophoresis showed a polyclonal gammopathy with increased IgG (21.1 g/L). Serum interleukin-6 (IL-6) was 401.8 pg/mL (normal <100), vascular endothelial cell-derived growth factor (VEGF) was 209.5 pg/mL (normal <29). Indirect Coombs test (anti-IgG, anti-C3d) was positive. His autoantibody profile including antinuclear and

antineutrophil cytoplasmic antibodies, antinuclear antibodies were all negative. However, anti-GBM antibodies were detected in 80% (normal <13%) by enzyme-linked immunosorbent assay. Hepatitis B, hepatitis C, syphilis, and HIV screening were negative. Human herpes virus 8 was not detected in lymph node tissue by polymerase chain reaction amplifications. Chest radiograph showed a left-sided pleural effusion but was otherwise unremarkable. Ultrasound scanning of the abdomen showed splenomegaly and an enlarged lymph node (diameter 2 cm); both kidneys were symmetrical and normal in size.

The clinical diagnosis: lymph node swelling and acute renal failure

Kidney and lymph node biopsies were performed. The biopsy of the left cervical lymph node demonstrated hyperplastic lymph follicle and sheets of plasma cells in the interfollicular space. Therefore, the patient was diagnosed as multicentric plasma cell type of Castleman disease. A renal biopsy revealed 22 glomeruli: cellular crescents were present in 13 of the 22 glomeruli, with a focal segmental necrotizing glomerulonephritis in 2 of the 22 glomeruli. Some glomeruli showed rupture of Bowman's capsule, with a giant cell response. The interstitium was infiltrated with a dense of focal mononuclear cells, and tubules had focal dedifferentiation with many red cell casts. Immunofluorescence studies showed strong deposition of IgG in a linear pattern along the GBM. The renal biopsy diagnosis was crescentic glomerulonephritis , type□.

Clinical Follow-up

Plasma exchange was performed daily for 12 days using 2 L human albumin with 1 L fresh frozen plasma, accompanied by methylprednisolone pulse therapy (500 mg/day) for 3 days. The patient showed a good response to the treatment (Figure 3). Anti-GBM antibodies returned to the normal range by 2 weeks and indirect Coombs test became negative. In addition, other clinical manifestations including, lymphoadenopathy, splenomegaly, generalized edema, anemia, thrombocytopenia, and anemia resolved. The serum creatinine, CRP, IL-6, and VEGF levels completely normalized within 1 month of admission. A follow-up renal biopsy was performed 6 weeks later following intensive treatment to evaluate the effect of the therapy. The biopsy showed extensive evidence of glomerular and tubulointerstitial scarring (not shown). Of note, 11 of the 18 glomeruli had fibrocellular crescents and 5 of the 18 glomeruli now demonstrated cellular crescents.

During the follow-up period, he received COP chemotherapy (cyclophosphamide 1200 mg, vincristine 4 mg, and prednisone 100 mg) every month. Complete remission was achieved with negative of anti-GBM antibodies by 6 months after admission. However, approximately at this time he was diagnosed with herpes zoster and his chemotherapy needed to be discontinued. Consequently, his anti-GBM antibody level increased (21%–26%), his serum IL-6 increased to 198 U/mL and his renal function as measured by serum creatinine declined to the 1.8 to 1.9 mg/dL range. One month after discontinuation of chemotherapy, he was restarted on another course of chemotherapy: his IL-6 and anti-GBM antibodies returned to normal again. After a follow-up of 20 months, the patient was normal, urinalysis showed 20 to 30 red cells per 10×40 field and urine protein excretion was 2 to 3 g/day, and his serum creatinine was in the range of 1.6 to1.8 mg/dL.

Case 6

A case of rapidly progressive glomerulonephritis with rare crescent formation

Emiko Fujita, Du Xuanyi, Keiko Hara, Naoaki Hayama, Akiko Mii, Kaoru Aki, Yukinari Masuda, Yasuhiko Iino, Akira Shimizu
Department of Pathology, and Department of Internal Medicine, Chiba-Hokusoh Hospital, Nippon Medical School, Tokyo Japan

Patient: 60 years old, Male
Past history: 50 y/o chronic sinusitis, Hypertension, Diabetes Mellitus (no treatment, HbA1c is 9.4 on admission)
Present illness: A 60 years old male, who had cough, bloody sputum, and 38□ fever from 1.5 months ago. He went to outpatient clinic and received antibiotics and NASIDs. However, his symptoms continued, he came our hospital (BUN 19.8 CRE 0.90) at 2 weeks ago. Chest X-P and CT showed multiple pulmonary nodules with cavity. He found macrohematuria 3 days before admission, and his renal functions deteriorated (BUN 33.3, CRE 2.45). Then he admitted to our hospital.
Physical and laboratory findings on admission were; Temp: 36.7°C, BP: 162/100mmHg, no evidence of sinusitis in nasal cavity, pitting edema on

lower limbs, no skin rash, WBC 18700/□l (Neu St1.0 Seg88.0 L4.0 E3.0), RBC 509x10⁴/□l, HGB 15.5g/dl, HCT 44.5%, PLT 23.6x10⁴/□l, BUN 33.3mg/dl, CRE 2.45mg/dl, Alb 3.6g/dl, CRP 17.67mg/dl, Glu 217mg/dl, HbA1c9.4, Ferritin 1144 ng/ml, HBs-Ag(-), HCV(-), ASK/ASO(-) (ELISA): PR3-ANCA <10, MPO-ANCA <10, IF: ANCA (-), IC-C1q <4, anti-GBMab (-), CH50 50U/ml, C3 156mg/dl, C4 33mg/dl, ANA <40, anti-ds-DNA(-), Urinalysis: Prot (2+), Occult blood (+/-), Sugar (3+), urinary segiment: RBC 1-4/HPF, WBC 5-9/HPF, cast (-) U-NAG9.3U/l, U-□2MG 3515μg/l, Culture: blood (-), urine (-), sputum (Klebsiella pneumonia), Tbe(-), Abdominal CT: kidney size 110mm,

On admission, he had RPGN and pulmonary multiple nodules, and his clinical diagnosis is Wegener granulomatosis, although c-ANCA is negative. He also had Diabetes Mellitus. He received antibiotics (ABPC 4g/day and CLDM 1.8 g/day) for 5 days. After 1st renal biopsy, he received methylpredonisolone pulse therapy (1g/day for 3 days) and cyclophosphamide pulse therapy (700mg). Our pathological diagnosis of 1st renal biopsy was glomerular and interstitial hemorrhage, may associated with vasculitis, no necrotizing and crescent formation in glomeruli. His symptoms (cough and bloody sputum), pulmonary multiple nodules in CT findings, and severe inflammatory reactions in peripheral blood resolved after treatment, however, renal dysfunction progressed to end-stage renal disease at 1 month after 1st renal biopsy. Hemodialysis was started and steroid therapy also was continued (60mg/day for 4 weeks then reduced 5-10mg/week). During he received hemodialysis, 2nd renal biopsy was performed (Our diagnosis was focal segmental crescentic glomerulonephritis, 4-5 crescents/ 19 glomeruli). His renal function was gradually recovered and disengaged from hemodialysis. Now his renal function is BUN40.5 and CRE2.61 without hemodialysis.

Discussion points:

1) Clinically, patient had rapidly progressive renal failure and pulmonary multiple nodules, like as Wegener glanulomatosis. PR3-ANCA(-). What is diagnosis in this case?.

2) Why rapidly progressive renal failure developed in 1 st renal biopsy findings.

3) Why rapidly progressive renal failure did not inhibit after steroid and cyclophosphamide pulse therapy.

Mini-lecture-1

Proposal of podocytic infolding glomerulopathy: A review of 25 cases from nationwide research in Japan

Kensuke Joh,
A research working group of the Japanese Society of Nephrology
Division of Renal Pathology, Clinical Research Center, Chiba-East National Hospital, Chiba, Japan

Background A rare and peculiar glomerulopathy has begun to be recognized in Japan. The Japanese Society of Nephrology has established a research working group and has collected cases from all over Japan in an attempt to understand the complete spectrum of this glomerulopathy.
Method The diagnostic criterion, which was needed to collect the cases, was proposed as a glomerulopathy showing microspheres or microtubular structures or both associated with podocytic infolding into the glomerular basement membrane (GBM) on electron microscopy. The lesion shows a non-argentaffine hole in the GBM with periodic acid methenamine silver staining and is similar to membranous glomerulonephritis.
Results Twenty-five cases were collected from 17 institutions. Patients were 20 to 69 years old (5 women, 20 men). Seventeen patients also had collagen diseases such as lupus nephritis and Sjoegren's syndrome. All patients had proteinuria. Proteinuria showed a remission in 15 of 23 patients within 12 months, but proteinuria remained higher than 1.0 g/day in 5 patients despite different types of therapy. Podocytic infolding including microspheres showed either positive or negative staining for immunoglobulins. Cluster formation of microspheres was found in 4 of 17 patients with collagen disease, and in 5 out 8 patients without collagen disease. Electron-dense deposits in the GBM were also found in 6 of 17 patients with collagen disease but were not found in 8 patients without collagen disease.
Conclusion Some patients might have a subtype of lupus nephritis, class V, or membranous glomerulonephritis. However, we propose a new disease entity, podocytic infolding glomerulopathy, as a common basis of all 25 patients, because we suspect that microspheres or microtubular structures or both can be derived from podocytic infolding.

Mini-lecture-2

Virus Infection and Nephropathy

Zou Wan-zhong

Department of Pathology, School of Basic Medical Science, Peking University

Institute of Nephrology, Peking University.

The pathogenic links between virus infection and renal diseases are often difficult to establish [1] . Viruses are a group of pathogenic microorganisms, with only 20nm to 300nm in diameter. They are simple in structure, without any cytopharyngeal apparatus, such as mitochondria, endoplasmic reticulum, Golgi complex, etc. They contain only one kind of nucleic acid, ether RNA or DNA. Thus, viruses are not independent reproductive organisms, but may parasite in host living cells. Their reproduction occurs within the host cells, since the viruses depend on the host cells for replication of their own nucleic acid and synthesis of their own protein coat. The viruses entry into host cells by phagocytosis or pinocytosis, usually the host cells have specific receptors on their cell surface for the viruses [2] .

The renal cells, including glomerular, tubular, interstitial and vascular endothelial cells, could be infected by viruses and thus result in virus-related nephropathy. Different mechanisms are operative in different viral nephropathies : (1) Virus infection provides continuous antigenic stimulation, resulting in antibody production and formation of immune complexes, which can be derived from circulation or formed in situ. Acute glomerulonephritis and glomerular capillary endotheliosis are formed. In chronic forms of

glomerulopathy, persistent viral infections cause inflammatory renal diseases, various pro-inflammatory mediators can result in sclerosis and exacerbate glomerulopathy. (2) Viruses could enter host cells and then induce reproduction or multiplication leading to cellular dysfunction and cell death, such as acute necrotic glomerulopathy and tubulointerstitial nephritis. (3) Release of various pro-inflammatory mediators could directly induce inflammation, tissue injury and functional deterioration [3] .

The diagnostic criterion of viral nephropathy are as follows: (1) Viruses infection is conformed by serological assay, such as PCR and Elisa etc; (2) Presence of acute or chronic renal injuries; (3) Renal biopsy shows glomerular or tublointerstitial diseasees by immunopathologic techniques (immunofluorescent, immunohistochemistry etc.) and light microscopy examination; (4) Immunopathologic techniques to confirm the presence of viral antigens in kidney; (5) The viral gene in renal cells is identified by in situ hybridization method; (6) The special stain (Mann special stain method for viral inclusion: Methylene Blue and Eosin method) or electron microscopy showed viral inclusion body in renal cells; (7) The electron microscopy examination found virus particles in renal cells. The positive serological viral markers, viral antigens and viral gene in renal cells are necessary [4] .

The various nephropathies may be induced by virus infections with different mechanisms. (Table 1) The most frequent and well recognized virus related glomerulonephritis are those associated with hepatitis B virus (HBV), in which formation of immune complexes is important. Hepatitis C virus (HCV) induces cryoglobulinemia related membranoproliferative

glomerulonephritis in most cases. Infection with human immunodefiency virus (HIV) is associated with a broad spectrum of glomerular lesions and with multiple pathogenic mechanism. Parvovirus B19 (PVB19) is associated with focal segmental glomerulosclerosis (FSGS) and immune complex mediated glomerulonephritis. Polyoma BK virus and Hantavirus most frequently cause tubulointerstitial damage. Some rare nephropathies are currently attributed to other viruses, such as those causing yellow fever, mumps, measles, varicella and adenovirus, etc [5]

Table 1. Viral infections and associated nephropathies

Viral infections cause nephropathies by immune complex:	
Hepatitis B virus	atypical MN, MPGN
Hepatitis C virus	MPGN with cryoglobulinemia
Hepatitis A virus	GN
Varcella-zoster virus	GN and TIN
Parvovirus B19	GN
Yellow fever virus	GN
Mumps virus	GN
Measles virus	GN
Virus directly (or unknown) damage to renal cells:	
Hantavirus	TIN
Severe acute respiratory syndrome (SARS) virus	TIN

Adenovirus	TIN
Epstein-Barr virus(EBV)	TIN
Cytomegalovirus	TIN
Human immunodeficiency virus(HIV)	FSGS
Polyoma BK virus	TIN
Dengue fever virus	TIN
Influenze A virus	TIN
Coxsackie B virus	TIN

Note: GN: glomerulonephritis; MN: membranous nephropathy; MPGN:membrano-proliferative glomerulonephritis; TIN: tubulointerstitial nephritis; FSGS: focal segmental glomerulosclerosis.

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