The definition of acute renal failure (ARF) has not gotten common understanding yet in a long time, which leads to the difficulty in comparing the outcomes of some different studies, and has impacted the advance of diagnosis and treatment on the illness to certain extents. Most of the scholars hold that the attention paid to the early diagnosis and intervention of ARF was insufficient in recent years. Lots of clinical researchers indicated that even a slight impairment of renal function could result in the increasing of the morbidity and mortality of ARF. Therefore, the international circles of nephropathy and emergency medicine incline to rename the ARF as acute kidney injury (AKI), expecting that the illness could be noticed as early as possible, in the beginning of glomerular filtration rate (GFR) falling down and even in the initial stage of kidney injury, when there presented only the histological or biomarker change but GFR remains normal.

In the first conference of Acute Kidney Injury Network (AKIN) at Amsterdam in September 2005, the standard for the diagnosis and classification of AKI was set up based on the RIFLE. AKI is defined as the sudden (within 48 h) deprivation of renal function induced by the structural or functional injury of the kidney. Clinically it is manifested by the increase of serum creatinine content (SCr) for $\geq 0.3$ mg/dL ($\geq 26.4$ μmol/L) in absolute value or $\geq 50\%$ (up to 1.5-fold of baseline); or by amount of urine decrease to $< 0.5$ mL/kg per h, and this lasts for over 6 h. AKI was classified into three stages, in correspondence respectively with the stages of Risk, Injury, and Failure in the RIFLE.

Glomerular AKI commonly manifests the symptoms of rapidly progressing nephritis syndrome or acute nephritis syndrome, as hematuria, proteinuria, edema, hypertension, etc., with short-term renal function deterioration. The most important point for differentiation is oliguria ($< 400$ mL/day) or anuria ($< 100$ mL/day), which may be accompanied with hematuria. Pathological examination shows the changes of crescentic glomerulo-nephritis or endo-capillary proliferative glomerulo-nephritis, revealing diffusive proliferation of glomerular endothelial cells and mesenchymal cells, which are often seen in clinics in primary glomerular nephropathies, such as rapidly progressing nephritis and severe acute nephritis, also in some secondary glomerular nephropathies such as lupus nephritis type IV, antineutrophilic cytoplasm antibody associated small vasculitis renal injury, purpura nephritis and Goodpasture syndrome, etc.

Typical Case 1

CHEN, male, 26 years old, his first visiting took place on May 19, 2004.

He asked for emergent aid in the 6th People's Hospital at the end of March due to a gross hematuria presented, which was followed by a
fever (38 ℃) the next day. There he was treated with anti-infection therapy for three days and hospitalized on April 1, 2003, for the unattenuated fever. Examination on renal function showed SCr 190 μmol/L, urea nitrogen 5.3 mmol/L. After treating him three-day pulse therapy of methylprednisolone 500 mg, the re-examination showed no improvement of renal function: SCr 809 μmol/L, urea nitrogen 38.2 mmol/L. Hematodialysis was then started from April 11, three times a week. Meanwhile, CTX intravenous dripping was given intermittently, 50 mg on April 8 and 100 mg q.o.d. from April 9 onward for 22 days totally. The pathological examination of kidney biopsy found five crescentiform cells in eight glomeruli, matrix and mesenchymal cell proliferation, obstructed glomerular capillary, interstitial cell infiltration, and focal tubular atrophy and fibrosis, suggesting the diagnosis of rapidly progressing glomerular nephritis. Electron microscopic feature showed only few glomerular tissues, moderate amount of electronic dense matter deposition and lots of inflammatory cell infiltration in the mesenchymal area.

Laboratory indices indicate ANA (-), ANCA (-), ENA (-), and anti-GBM antibody (+). Pulse therapy with Methylprednisolone 500 mg was applied again for three days from April 24 and repeated with adding dosage to 1.0 g for three days from May 6, followed by 60 mg per day via intravenous dripping, and one day of pulse therapy with CTX 1.0 g for a meantime. Blood plasma replacement was conducted from May 7, with a total of 11 times. When discharged from the hospital, his renal function related indices showed SCr 213 μmol/L, urea nitrogen 16.6 mmol/L, uric acid 426 μmol/L, 24h urinary albumin 0.369 g, and anti-GBM antibody (+). Those were re-examined on May 12 and showed SCr 140 μmol/L, uric acid 472 mmol/L, and 24 h urinary albumin 0.147 g. The dosage of hormone was reduced to the regular level (12.5 mg/d) when the patient visited our hospital asking for Chinese medicine therapy on May 19.

At the first visit, his symptoms were self-perceived severe innerheat, dry mouth, much sweating, red tongue proper, yellow-greasy coating on tongue root, and taut pulse. His syndrome was differentiated as heat-dampness accumulation with flurry turbid-toxin, and the therapeutic principle of clearing heat, dispelling dampness, dredging Fu to excrete turbidity was adopted. The recipe was prescribed as the follows: *Herba Hedyotis diffusae* 30 g, *Scutellariae barbatae* 30 g, *Caulis Lonicerae* 30 g, *Herba Violae* 30 g, *Radix Paeoniae rubra* 15g, *Radix Rehmanniae* 15 g, *Rhizoma Polygonati* 20 g, *Radix Codonopsis* 30 g, *Radix Salviae miltiorrhizae* 30 g, *Semen Areca* 30 g, *Radix Puerariae* 20 g, *Prepared Radix et Rhizoma Rhei* 12 g, *Fructus Chaenomelis* 15 g, and *Herba Dendrobii* 15 g.

At the second visit on July 1, the patient complained liable to get common cold, dry mouth, but with normal appetite and sleep, one defecation a day, pink tongue body with yellow-greasy coating, and taut pulse. He was taking 10 mg of prednisone per day orally. Since his syndrome was differentiated as Shen (腎)-yin insufficiency with inner blocking of dampness-stasis, the treatment of nourishing yin, reinforcing Shen, activating blood, and removing dampness was applied. The recipe was prescribed as follows: *Herba Hedyotis diffusae* 30 g, *Folium Hibisci mutabilis* 30 g, *aulis Lonicerae* 30 g, *adix Rehmanniae* 15 g, *Fructus Corni* 12 g, *Radix Codonopsis* 30 g, *Radix Salviae miltiorrhizae* 30 g, *Rhizoma Ligusticum wallichii* 15 g, *Radix Puerariae* 15 g, *Rhizoma Polygonati* 15 g, *Fructus Lycii* 20 g, *Prepared Radix et Rhizoma Rhei* 10 g, *Radix Angelicae sinensis* 10 g, *Radix Paeoniae rubra* 15 g, *Radix Paeoniae alba* 15 g, and *Semen Juglandis* 15 g.

And coordinated with Huoxue Tongmai Capsule (活血通脉胶囊), three times a day, 3 capsules each time.

Re-examination of renal function showed SCr 140 μmol/L and urea nitrogen 5.7 mmol/L. B-ultrasonographic picture showed the normal size of bilateral kidney.

In the third visit on September 30, he has received dosage of CTX reaching 10 g in total, and continued to take 7.5 mg of prednisone a day. He said his dry mouth symptoms was alleviated significantly, only occurring in the morning. He had possessed good appetite, pink tongue with yellow-greasy coating, taut pulse. The treatment principle was supporting qi, nourishing Shen, coordinated with dissolving dampness, dispelling turbidity, activating blood and eliminating lump. The recipe was prescribed as follows:

*Radix Astragali* 30 g, *Radix Puerariae* 15 g, *Rhizoma Ligusticum wallichii* 15g, *Rhizoma Polygonati* 20 g, *Fructus Lycii* 20 g, *Cortex Eucommiae* 15 g,