CASE REPORTS

C3 glomerulonephritis

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Abstract

A case of rapidly progressive glomerulonephritis with pathological changes to the crescent glomerulonephritis "C3 glomerulonephritis" in the Third Affiliated Hospital of Inner Mongolia Medical University was collected and its diagnosis, clinical manifestation, and pathological characteristics were explored. The experts analyzed the disease from the perspective of its causes, diagnosis, complications and treatment. Timely renal biopsy is required to clarify pathological diagnosis as the primary glomerular disease was newly recognized with a lower incidence. So misdiagnosis and delayed healing is more frequent. The paper aims to enhance the clinician's understanding of "C3 glomerulonephritis".

Key Words: Acute kidney injury, Kidney failure, Crescent glomerulonephritis, C3 glomerulonephritis

1 Medical record

1.1 General information

A 5-year-old girl was admitted to our hospital due to fever, hematuria and oliguria for 5 days, abdominal pain for an hour. Her body temperature was up to 39.5°C five days ago, accompanied by tea colored urine in decreased urine output, daily approximately around 200 ml, with dysuria, without urinary frequency or urgency. She had antipyretics three times, and cold granules and azithromycin for three days, which posed no effect. The patient was, then, admitted to our department for routine urine tests: WBC +2, protein +2, occult blood +3, erythrocytes 976/HP, WBC 12.66 $\times 10^9$/L, neutrophils 0.79, hemoglobin 115 g/L, platelets 264×10^9/L, so "urinary tract infection" was suspected. The patient was treated with intravenous ceftriaxone twice and andrographolide once, consequently the fever was gone. She had systemic urticarial, but rash finally disappeared when desensitization was given for a day. Suddenly, she felt abdominal pain, unbearable and sustained periumbilial pain during intravenous ceftriaxone one hour prior to her admission. The symptoms of diarrhea, convulsions, poor appetite were presented without vomiting or headaches.

1.2 Physical examination

Data on the physical examination revealed her temperature 37°C, pulse 85 beats per minute, breathing 20 per minute, blood pressure 110/80 mmHg, and Wt 17.5 kg. She showed poor spirit, steady breathing, and had no rash in skin or body. Slightly swollen was caught in eyelids, double lung breath sounded rough and no signs hinted of the abnormality of heart. No enlargement of liver, spleen and kidneys beneath the rib was found, nor edema of lower limbs. Her abdomen was soft but tenderness could be felt in periomphalic area and right lower side. Neurological examination result was normal.

1.3 Auxiliary examination

Blood routine examination: WBC 13.3×10^9/L, N% 86%, L% 11.3%, Hb 120 g/L, PLT 298×10^9/L, eosinophils 0-

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0.15×10^9/L, reticulocyte count 0.009-0.014. Cellular morphology was normal, alanine aminotransferase and aspartate aminotransferase were normal, lactate dehydrogenase 428.0 U/L, HBDH 383.0 U/L, serum bilirubin was normal, albumin 30 g/L; cholesterol and lipoproteins were normal. Routine urine test: occult blood +++, erythrocytes 400/μl-13,500/μl with full vision; a large number of white blood cells, urine protein +-++ quantitative 0.53 g/24h, Ccr 7 ml/(min.1.73 m^2). Sodium 129-138 mmol/L, serum chloride 97-105 mmol/L, potassium 3.6-5.4 mmol/L, calcium 1.63-2.12 mmol/L, phosphorus 2.83-2.42 mmol/L, blood gas analysis: PH 7.3-7.43, HCO_3^- 9.3-16.2 mmol/L. Lactate dehydrogenase 428.0 U/L, HBDH 383.0 U/L, EB virus, mycoplasma antibody, herpes simplex virus antibodies, rubella virus, toxoplasma antibodies were negative; Four infections, hepatitis B five were normal. Hepatitis A, hepatitis C and hepatitis E antibody were negative. ANA was negative, anti-neutrophil cytoplasmic antibody was negative, CRP 16.74 mg/L, ESR 40 mm/h, ASO 98.1-33.7 IU/ml. Blood coagulation was normal; Changes of kidney function and complement were seen in the Table 1.

Table 1: Dynamic changes of kidney function and complement value

After admission (day)	BuN (mmol/L)	Cr (μmol/L)	UA (μmol/L)	Cys-c (mg/L)	C3 (g/L)	C4 (g/L)
1	20.7	395.0				
2	27.8	510.6	812.5		0.5	0.66
3	20.67	365.3	456.2	2.85		
4	27.15	438.6		4.3	0.3	0.62
5	24.58	343.2	461.4			
9	23.86	260.2	530.4	3.53	0.47	0.55
12	12.18	70.3	414.4	2.15	0.67	0.28
16	6.31	32.0	290.4	1.43	0.93	0.45
Reference value	1.7-8.3	40-60	150-430	0.4-1.35	0.8-1.85	0.18-0.4

Note BuN: Blood urea nitrogen; Cr: creatinine; UA: Uric acid; Cys-c: Cystatin C

Abdominal ultrasound examination results for three times: Parenchymal echo diffuse increased in the kidney and the volume increased as well. The left kidney was up to 10.6 cm ×5.4 cm, and its counterpart was 10.3 cm ×4.6 cm, with undefined boundaries of cortex and medulla. Sinus structural disorder was presented, but hydronephrosis and ureteral dilatation could not be seen or separation between renal pelvis. Chest X-ray, echocardiography and electrocardiogram clarified normal condition of the kidney.

Renal biopsy examination result: (1) Light microscopy: 21 complete glomeruli, no global glomerular sclerosis, capsular synechia or segmental sclerosis was detected, 12 cellular crescents (57.1%). Glomerular mesangium cell proliferation or glomerular mesangial matrixes could not be detected. While segment endothelial cell proliferation occurred, some capillary loop was pressed and bloacked with bad open, thickened or stratified glomerular capsule also appeared, accompanied by segmental proliferation of epithelial cell, glomerular neutrophil infiltration and segmental fibrinoid necrosis. Renal tubular atrophy was invisible, brush border depigmentation and vacuolar degeneration of epithelial cells was checked. There were protein casts, red blood cell casts, exfoliated epithelial cells and inflammatory cells in lumen, no significant inflammatory cell infiltration and fibrosis in mesenchymal, and no obvious changes in renal artery. (2) PAM, MASSON staining: No red substance addicted complex deposition, no thicken basement membrane, or track spikes. (3) Immunofluorescence: C3 (+) mass-like deposits along the mesangial area, IgG (-), IgA (-), IgM (-), C_4 (-), C1q (-), Fib (-). Pathological diagnosis: Crescent glomerulonephritis (CGN) (see Figures 1,2).

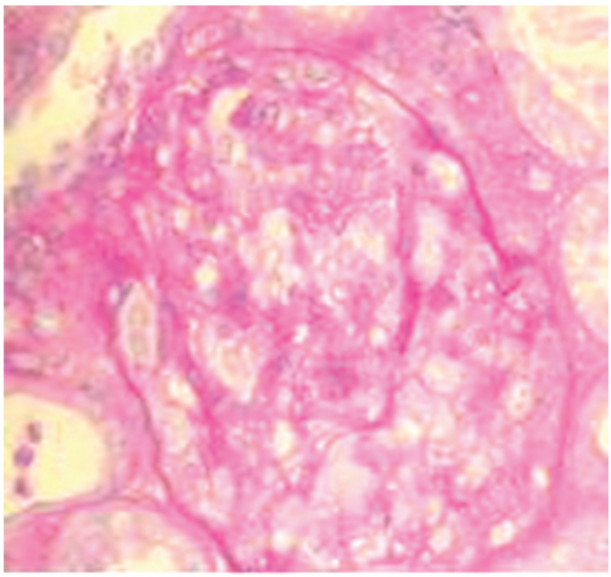

Figure 1: Cellular crescents

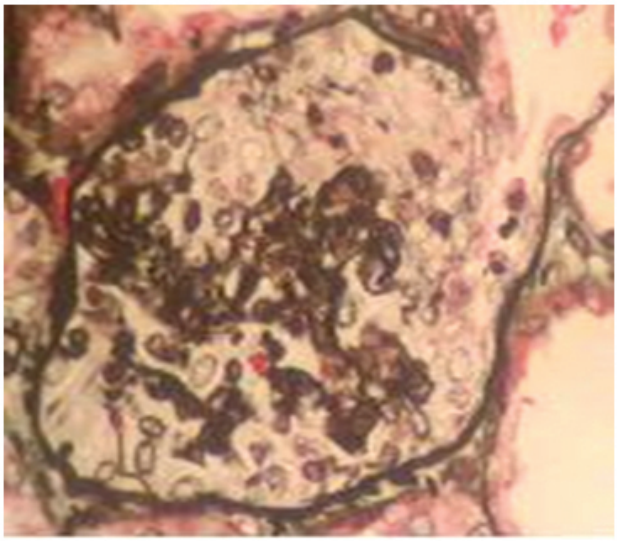

Figure 2: Glomerular filled with a large number of neutrophils

1.4 Primary diagnosis

(1) Acute glomerulonephritis
(2) Acute renal failure

1.5 Treatment

Blood electrolyte indicated hyponatremia and blood gas analysis showed metabolic acidosis after the examination. BUN was up to 27.9 mmol/L, creatinine progressed to 510.6 μmol/L, and potassium peaked 5.40 mmol/L within 24 hours. Right kidney was probed and a diameter of about 0.4 cm hyperechoic was reflected which supported the diagnosis of kidney stones on the day of admisson. While the possibility of pre-renal azotemia, could not be ignored due to light edema, poor appetite for 3 day's duration, continuous oliguria of tea and smoke gray color. Rehydration remedy acid treatment, then, was given, consequently kidney stones was tested to disappear by renal ultrasound the following day after admission. The patient was diagnosed as ARF, rapidly progressive glomerulonephritis could not be ruled out. Femoral vein catheter was performed under anesthesia three times in the afternoon beside the bed for blood filtration and hemodialysis on the next day. Methylprednisolone 0.3 $\times$3, qd was taken and administration of prednisone 20 mg, qd was conducted on the third day after admission. The patient started to eat and felt much better on the fifth day. Compared with previous examination results, urea, creatinine decreased, C3 dropped to 0.3 g/L, routine urine test: RBC full vision, a large number of white blood cells, protein +2, blood pressure has been normal, edema was not obvious. Puncture biopsy was conducted under general anesthesia on the sixth day and pathological diagnosis demonstrated crescent glomerulonephritis. The second course of treatment of methylprednisolone 0.3 $\times$3, qod was given and deep venous catheter was removed on the tenth day. Renal function, complement, erythrocyte sedimentation were normal on the sixteenth day. There was slightly swollen in the right leg on the eighteenth day, and ultrasound showed hypoechoic plaque adhesion in right femoral vein mural, stenosis rate of 75%, the color of blood stream thinning. IVUS hinted of thrombosis in right external iliac vein and general iliac vein. Meanwhile the diagnosis of deep venous thrombosis was made due to D-dimer 3.6 g/L, FDP 6.5 g/L. Additional use of 80,000 units of urokinase for 5 days, low molecular weight heparin 1,700 units $\times$6, bid, pulse-activating injection 10 ml for 5 days so that the symptom of right lower limb swelling subsided completely. Coagulation and vascular ultrasound were back to normal after reexamination. The patient had Xarelto orally for 20 days, finally femoral vein thrombosis disappeared. And extra use of rehabilitation in the treatment of nephritis was employed for a month, the patient only had microscopic hematuria, urinary red blood cells of 10 to 20/HP, microscopic hematuria disappeared due to 3 months' duration of therapy. He went to Peking Medical University for complement regulatory protein factor H examination, which revealed 416.5 μg/ml (reference value 380-762 μg/ml), anti-H factor antibody was negative.

1.6 Confirmation of the diagnosis

(1) Rapidly progressive glomerulonephritis
(2) Crescent glomerulonephritis
(3) C3 glomerulonephritis
(4) Femoral vein thrombosis

2 Discussion

2.1 Dr. Zhixia Wang

Dr. Zhixia Wang is the attending doctor of Pediatric Department at the Third Affiliated Hospital of Inner Mongolia Medical University, specializing in pediatric respiratory and intensive Care.

The patient experienced hematuria, oliguria and proteinuria during the course of the acute infection. Plus examination results demonstrate a continuing deterioration of renal function and a decrease in C3 levels so that an initial diagnosis of acute nephritic syndrome was established when kidney volume was testified to be enlarged. The disease refers to clinical manifestations of acute onset, such as edema, hematuria, hypertension, a decrease in estimated glomerular filtration rate, water and sodium retention of varying origins. It is also known as glomerulonephritis among children patients, and its degree of the clinical manifestations differs greatly. Patients with minor glomerulonephritis had no ob-

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vious clinical symptoms but microscopic hematuria could be seen. While, rapidly progressive course is presented in severe cases, heart and kidney failure are likely to occur in the short term. They are categorized into several different pathological patterns, which are broadly grouped into primary or secondary types. Symptoms of unknown origin usually belong to the former type. The vast majority of secondary infection glomerulonephritis includes bacteria, viruses, mycoplasma, fungus and parasites and other infections, among which acute post-streptococcal glomerulonephritis (APSGN) is the most common one, much associated with streptococcal infection, and occurs after respiratory or skin infections 1-3 weeks. It is a glomerular inflammatory lesions caused by serum through antigen-antibody immune complexes. The streptococcal and products antibodies could be detected in serum. The patient in the case had damage to the kidney during its infection course, and Antistreptolysin O (ASO) titer did not increase. But the syndrome was greatly relieved, complement rose soon and kidney function recovered after the patient received high-dose methylprednisolone pulse therapy so that the diagnosis of atypical APSGN was confirmed.

2.2 Dr. Chunyan Ma

Dr. Chunyan Ma is associate director of Pediatric Department at the Third Affiliated Hospital of Inner Mongolia Medical University, specializing in pediatric respiratory and intensive care.

The child was diagnosed as acute glomerulonephritis with acute renal failure (ARF). Causes of acute kidney failure fall into one of the following categories: prerenal failure, postrenal failure, and renal failure. (1) Prerenal ARF occurs when a sudden reduction in blood flow to the kidney (renal hypoperfusion) causes inadequate blood circulation (perfusion) to the kidneys and a decrease in glomerular filtration rate. Prerenal failure can be caused by dehydration and disruption of blood flow to the kidneys from vomiting, diarrhea, decreased urine output. Patients usually have dehydration and low blood pressure during physical examination, in which hemoglobin elevated, routine urine examination was normal, urine specific gravity, urine sodium as well as urine osmolality rose. The diagnosis of prerenal failure in the case was, of course, denied as the routine urine examination was severely subnormal and no history of vomiting and diarrhea was included. While the symptoms of poor appetite, obvious oliguria, edema, faint hypertension hint of hyponatremia hence rehydration therapy was treated. The outcome of the therapy showed that there was no obvious increase in urine output, blood pressure was normal, no aggravating edema so that hypovolemia was confirmed. (2) Postrenal ARF, caused by obstruction of the urinary tract, may be within the urinary tract or extrinsic. Obstruction is often accompanied by acute abdominal pain, tumor, stones, malformation and other changes could be found by renal ultrasonography, and renal pelvis, ureter or bladder expansion which is above the level of obstruction could be seen as well. The patient was checked to have a diameter of about 0.4 cm hyperechoic reflection on the right kidney after admission, and kidney stones were highly suspected. The stones disappeared after 1 day treatment of rehydration remedy acid treatment. However, the symptom of renal dysfunction aggravated so postrenal causes are ruled out. (3) Intrinsic ARF, can be due to untreated prerenal kidney failure, severe hypoxia, drugs, toxins, infections, renal parenchyma and renal vascular disease. Common symptoms are acute tubular necrosis, Acute Glomerulonephritis (AGN), acute interstitial nephritis and other diseases. The presence of progressive AGN, an increase of blood urea nitrogen and creatinine within 24 hours in the case were highly suggestive of intrinsic ARF.

2.3 Dr. Yajing Zhang

Dr. Yajing Zhang is leading expert of Pediatric Department at the Third Affiliated Hospital of Inner Mongolia Medical University, specializing in newborn's disease and children's growth.

Acute kidney injury (AKI) is much preferred than acute renal failure in international nephrology and emergency medical community in recent years.[1] ARF is a critical and complex renal dysfunction syndrome that occurs in a variety of clinical situations (child or adult, clinic or ward, ICU and non-ICU patients), characterized with sudden and sustained decrease of glomerular filtration rate, urea and other metabolites accumulate in the blood and any significant water and electrolyte imbalance, metabolic acidosis and azotemia. AKI is to advance the clinical diagnosis of ARF syndrome. AKI is fully recognized, and its prognosis may cause secondary to chronic kidney disease, and eventually progress to end-stage renal disease. Early diagnosis and treatment of the underlying AKI among children patients remains imperative for possible recovery. ARF is a common clinical manifestation of acute onset with rapid progress and high mortality, whose etiology and clinical presentation vary significantly, while most renal damage is reversible by timely and correct treatment. The patient was diagnosed as AKI stage 3 in accordance with the staging of AKI,[2] SCr was up to $\geq$ 26.5 μmol/L, peak 510.6 (> 353.6 μmol/L), oliguria continued > 24 hours within 48 hours after admission. Blood purification therapy is feasible to improve renal function.

2.4 Dr. Weiguo Jia

Dr. Weiguo Jia is the director of Nephrology Department at the Third Affiliated Hospital of Inner Mongolia Medical University.

The best time to perform renal replacement therapy in patients with AKI remains to be determined. Literature review in our country demonstrated that continuous veno-venous hemofiltration (CVVH) can significantly improved the prognosis in AKI stage 1 or 2, while it posed no effect on AKI stage 3.[3] Renal replacement therapy for AKI patients depends on various factors, such as intravascular volume, electrolyte and acid-base balance, uremia, nutritional requirements, urine output and assessment of the hemodynamic status. We should not only consider the positive aspects of renal replacement therapy but also weigh the factors of risk as the anticoagulant can cause central venous access and related mechanical damage and infectious complications of bleeding.[4] The child was at the condition of AKI stage 3 for hemodialysis indication so that blood filtration and dialysis can alleviate the syndrome. Central venous catheter should be performed under general anesthesia as the patient as young with great risk. Indwelling catheter may induce complications (such as infection, bleeding or hematoma, catheter dysfunction, thrombosis, *etc*.), active prevention, therefore, is required. We should also pay attention to the occurrence of acute interstitial nephritis as the patient had administration of traditional Chinese medicine, antipyretics, anti-inflammatory drugs, *etc*. and had systemic urticarial. The disease can be manifested as hematuria, proteinuria and kidney damage, mimicking acute nephritis. Fever, rash, eosinophilia, urinary eosinophils, *etc*. may occur as well. However, there was no further fever or body rash after admission, eosinophils ($0, 0.15 \times 10^9$/L) was checked to be low so that diagnosis of acute nephritis was ruled out. Biopsy is requisite for the establishment of the syndrome. The methylprednisolone pulse therapy is useful treatment to induce rapid remission of disease.

2.5 Dr. Jinli Hao

Dr. Jinli Hao is the director of Pediatric Department at at the Third Affiliated Hospital of Inner Mongolia Medical University, specializing in children's growth, pediatric Hemato-Oncology and newborn.

The pathological diagnosis of chronic glomerulonephritis (CGN) was initially made in the case, while clinical progression of the disease, progressive deterioration of renal function, and lower level of complement C3 indicates of Rapidly progressive glomerulonephritis (RPGN). The disease is extremely severe kidney disease with a high mortality and a variety of primary or secondary glomerular diseases can be manifested as RPGN clinically. Most of the formation of renal biopsy is with extensive crescents, also known as the CGN.[5] The severe glomerular disease could be commonly categorized into three types according to immunofluorescence features. Type I: Anti-glomerular basement membrane glomerulonephritis, linear deposition of IgG along the glioblastoma multiforme (GBM), serum anti-GBM antibodies, clinical manifestations of Goodpasture syndrome. Type II: Immune complex-mediated CGN, characterized by a lot of particulate immune complexes and complement deposition seen in glomerular capillary and mesangial area, accompanied by proliferative glomerular lesions and was mostly found in a variety of primary and secondary glomerular diseases, such as IgA nephropathy, lupus nephritis, allergic purpura. Type III: Oligonucleotide immune complex nephritis, no immunofluorescence or only a small amount of immune complex deposition in the glomeruli could be seen. CGN is generally considered as the type of systemic vasculitis manifestation of renal involvement, and some patients are with anti-neutrophil cytoplasmic antibody (ANCA positive). But ANCA were negative in few children. Now that the[6] type III RPGN refers to primary vasculitis, segmental glomerular necrosis caused by ANCA, resulting in plasma and mononuclear cells into the renal capsule, and forming a crescent. Its incidence is closely related to a variety of adhesion molecules and cytokines. Specific antigens of anti-ANCA include original particles in neutrophils, myeloperoxidase in monocytes lysosome, and serine proteases related components. The former antibody is perinuclear, and the latter is cytoplasm type. ANCA was tested to be negative in the child patient. No damage to blood system and skin was presented, so kidney damages were not caused by systemic vasculitis. It is more likely to be type II immune complex glomerulonephritis.

2.6 Dr. Hua Chen

Dr. Hua Chen is the director of Pediatric Department at the Third Affiliated Hospital of Inner Mongolia Medical University, specializing in kidney immune diseases.

Capillary segmental fibrinoid necrosis was found by light microscopy examination of renal biopsy in the case, immunofluorescence was with C3 deposition, so the diagnosis of C3 glomerulonephritis could not be excluded in accordance with clinical manifestation. The difference between APSGN and the disease is highly depended on electron microscopy. The case was of acute onset, without medical history of streptococcal infection, no presence of swelling, and blood pressure was normal, dynamic monitoring of ASO was not always high, but complement C3 levels rose quickly, renal pathology consistent glomerular capillary hyperplasia, whose symptoms were different from the typical APSGN. Now that the manifestations of acute glomerulonephritis tissue infection mainly includes four types:[7] (1) Glomerular hyperplasia; (2) Crescent glomerulonephritis; (3) Glomerular mesangial proliferation disease; (4) Mesangial proliferative lesions. Biopsy pathology demonstrated the glomerular capillary proliferative lesions with a large number of infiltrating cells in the case on the eleventh day after admission, which consistent with acute glomerulonephritis exudative lesions. Whether it is atypical APSGN

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deserved more attention. It is with much regrets that electron microscopy failed. Only complement C3 along the mesangial area mass-like deposits was found by immunofluorescence microscopy, which was in line with C3 glomerulonephritis.

C3 glomerulonephritis is a newly recognized independent disease,[8] and its pathogenesis is much associated with genetic or acquired factors that cause the complement system dysregulation. Histological changes in mesangial proliferative lesions is much common, immunofluorescence staining glomerular was simply presented with complement C3 deposition along the glomerular capillary loops, without other complement or immunoglobulin deposition. Electron microscopy confirms subendothelial glomerular capillary loops and (or) see mesangial electron dense deposits.[9] It is lack of characteristic clinical manifestations, with nephrotic syndrome, hematuria, hypertension and renal dysfunction as the main performance, and lower presence of complement is frequent. Recently, Servais, *et al.*[8] detected mutation of factor H, factor I or membrane cofactor in some C3 glomerulonephritis patients, C3 nephritis factor in serum, suggesting the relationship between complement bypass route dysregulation and the disease.[10] 17 cases of C3 glomerulonephritis was reported in our country, among which three cases occurred under 14 years. They all share a tedious medical history of the disease and delayed healing, and immunosuppressive agents carry poor efficacy.[11] The patient had two courses of methylprednisolone, then the indicators were normal and only microscopic hematuria was untreated. Therefore, the possibility of C3 glomerulonephritis could not be excluded. If the electron microscope showed no electron dense and foot process fusion of epithelial cell, the diagnosis of the disease was then confirmed. Further investigation of factor H of complement regulatory protein, C3 nephritic factor is required.

3 Conclusion

In summary, the patient was initially manifested as continuous deterioration of renal function and acute glomerulonephritis with rapid progression. However, systematic internal environment was stabilized after CVVH treatment, and the disease was under control after the impact of hormone therapy. Long-term follow-up in September demonstrated that there were no obvious abnormalities in renal function, and urine was normal up for six months. Our clinical diagnosis of children from the AGN to RPGN, pathological diagnosis from CGN to C3 glomerulonephritis recognition had been enhanced after retrospective analysis study so that treatment programs were also adjusted. Though the merger of the femoral vein thrombosis occurred, the outcomes of the child ultimately improved. With the improvement of diagnosis and treatment, treatment shows an increasingly important role in the prognostic factors. Treatment principle of AKI hinges on quick identification and correction of reversible factors, prevention of further damage to the kidneys and maintenance of water and electrolyte balance. CVVH treatment for AKI stage 3 obtained good results in the case as renal biopsy diagnosis of CGN provides a reliable basis. The light microscopy findings are cellular crescents, indicating the early crescent formation, with a certain degree of reversibility.[12] In addition, the disease rapidly improved after methylprednisolone pulse therapy, suggesting that no progresses to fibrous crescent. It has been reported[13] that eight cases of cellular crescents disappeared in 9 cases of IgA nephropathy with crescent. Unfortunately, we did not have electron microscope result and H factor was tested to be negative. Ruijuan He from Peking Medical University reported that three cases of C3 glomerular disease were associated with a decrease of factor H,[14] and the diagnosis of underlying C3 glomerulonephritis could not be made. It is noteworthy that the child had onset of the course again after 10 months, very similar clinical manifestations and AGN and soon progressed to ARF. Renal function after a course of methylprednisolone pulse therapy was rapidly recovered, hematuria disappeared in two months. It will be of great help with our understanding of the disease if we had repeated biopsy information. Long-term prognosis of C3 glomerulonephritis is not clear in children patients. Yagi, *et al.*[15] from abroad reported four cases of the disease with good prognosis in 10 years, and noted that the risk factor of renal tubular damage is end-stage renal disease (ESRD). Ying Wang, from Children's hospital of suzhou university, *et al.*[16] seven cases of C3 glomerulonephritis, with crescent formed in 1 case, and the prognosis was good during followed up for 2 months to 5 years. As the patients were followed up shortly, longer follow was required for further diagnosis of the disease.

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