DIAGNOSTIC PROBLEMS IN A PATIENT WITH NEPHRITIC SYNDROME

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ABSTRACT Acute nephritic syndrome is a collection of clinical symptoms due to a sudden decrease in glomerular filtration rate (GFR) accompanied by water and salt retention, positive erythrocytes, erythrocytes cast and albumin in urinalysis. Information about nephritic syndrome is essential to know for accurate diagnoses and treatment. A 13-year-old adolescent with nephritic syndrome has been reported with rapid progressive glomerulonephritis (RPGN). He has been treated with corticosteroid, antibiotic, immunosuppressant, antihypertensives, and diuretic followed by improvement of clinical condition and laboratory of the patient. The biopsy was done for definitive diagnosis. The biopsy results showed a picture of IgA nephropathy (IgAN) and resolving stage of acute poststreptococcal glomerulonephritis (APSGN). Acute poststreptococcal glomerulonephritis (APSGN) was suspected due to finding of apparent hematuria, swollen and acute renal failure after streptococcal infection. To support the diagnosis, evidence of laboratory streptococcal infections and low levels of C3 complement are needed. Whereas IgAN was known by examining the IgA serum. Management of patients was including supportive and symptomatic therapy. Giving corticosteroids or cytotoxic agents is needed for RPGN therapy. The prognosis is generally good, with improvement of more than 90% of cases. Long-term observation is needed to observe the possibility of the disease being chronic. In this case, the symptoms and results of the laboratory examination were following the results of the biopsy, namely IgAN and resolving APSGN. Immediate and proper management provides an improvement in general and laboratory conditions.

KEYWORDS acute poststreptococcal glomerulonephritis, IgA Nephropathy, Nephritic Syndrome

Introduction

Acute nephritic syndrome is a collection of clinical symptoms due to a sudden decrease in glomerular filtration rate (GFR) accompanied by water and salt retention, positive erythrocytes, erythrocytes cast and albumin in urinalysis. Although the common cause of the acute nephritic syndrome is APSGN [1,2] however, it is necessary to consider another differential diagnosis. Nephritic syndrome and nephrotic syndrome has similar symp-
on urinating. Four days afterwards the patient complained of fever, cough, runny nose and pain when swallowing.

On physical examination, the patient was alert and in good orientation, blood pressure 160/110 mmHg, pulse 104 times/minute, respiratory rate 24 times/minute, temperature 37.3°C, body weight 60 kg, and height 155 cm, and with normal body mass index (BMI). No eyelid edema, no signs of anemia or jaundice. Tonsil enlargement was found, no pharyngeal hyperemia, normal jugular venous pressure (JVP). Normal heart sounds without murmurs, vesicular pulmonary sounds, no rhonchi nor wheezing. In the abdomen not found ascites, liver and spleen were not palpable, and all four limbs appear edema and warmth. The color of the urine looked like tea-colour.

Laboratory tests showed leukocyte or white blood cells (WBC) 5.9 x 10³ / ul; hemoglobin 11.85 g / dl; platelets 246,5 x 10³ / ul; blood urea nitrogen (BUN) 30 mg / dl; serum creatinine (SC) 3.3 mg / dl; aspartate aminotransferase (AST) 17.8 IU / l; alanine aminotransferase (ALT) 8.4 IU / l; sodium 135 mmol / l; potassium 3.7 mmol / l; albumin 1.94 g / dl; total cholesterol 466.7 mg / dl; high density lipoprotein (HDL) 47.31 mg / dl; low density lipoprotein (LDL) 351.4 mg / dl; triglycerides (TG) 230 mg / dl, from urinalysis showed turbidity (+); pH 5; specific gravity 1,020; leukocytes (++) 500; protein (+4); nitrite (-); blood (+5); bacteria (++); bilirubin (-); many leukocyte cells / field of view; many erythrocytes / morphic / visual field, virally marker for hepatitis was negative, anti-streptolysin O (ASTO) negative results. On examination of protein Esbach showed protinuria of 3.8 grams / liter / day. Abdominal ultrasound (USG) was done and found no abnormalities on examination. Throat swab confirmed Streptococcus viridans as the infectious agent. Complement C3 and C4 examination were normal.

Treatment given were furosemide 40 mg OD, pulse dose methylprednisolone 500 mg for three days, tapering down untill dose of 4 mg BID, Irbesaran 150 mg OD, spironolactone 25 mg OD, Cefoperazon 1 gr OD intravenous. Patients continued treatment at the polyclinic using azathioprine 25 mg TID, cyclosporine A (Sandimmun Neural®) 25 mg BID, irbesaran 150 mg OD, furosemide 40 mg OD, spironolactone 25 mg OD, methylprednisolone 4 mg BID, and amloidipine 5 mg OD.

After continued the treatment, improvement as no swelling, no tea-colored urine and no fever. On physical examination not found abnormalities of heart and lungs. No eyelid and extremities edema. Urine production in 24 hours is 1000 cc (0.92 cc / kgBW / hour).

Laboratory results were improved, WBC 7.5.10³ / ul; hemoglobin 14.9 g / dl; platelet 376.6.10³ / ul; BUN 9 mg / dl; SC 0.67 mg / dl; AST 12.2 IU / l; ALT 10.9 IU / l; Na 138 mmol / l; K 4.24 mmol / l; albumin 4.24 g / dl; total cholesterol 269.4 mg / dl; HDL 67 mg / dl; LDL 198 mg / dl; TG 123 mg / dl, from urinalysis showed turbidity (-); pH 6; BJI 1,019; leukocytes (-); protein (+1); nitrite (-); blood (-); bacteria (-); bilirubin (-); leukocyte cells 1.4 / field of view; erythrocytes 2.3 / morphic / field of view. Esbach protein showed proteinuria of 2 grams / liter / day. Renal biopsy was performed an confirmed a picture of IgA Nephropathy, and resolving GNAPS.

Discussion

Rapid progressive glomerulonephritis is characterized by rapid deterioration of kidney function, usually a 50% reduction in GFR in less than 3 months.[2-4] Although various diseases can cause RPGN, all types of RPGN have the characteristics of glomerular injury and crescent form on renal biopsy. Severe damage
Renal biopsy showed that >50% glomeruli with mild-moderate hyperscellularity in the form of increased global mesangial cellularity, as well as endocapillary cell proliferati-
tion. Some glomeruli show focal epithelial proliferation. One glomerulus shows a neutrophil infiltrate, endocapillary impres-
sion. Some glomerules also show the attachment of the tuft glomerulus to Bowman’s capsule. At some tubular lumen ery-
throcytes appear. In another focus, mononuclear cells infiltrates in interstitial tissue appear. The conclusion of the biopsy re-
results was the picture can be found in IgAN and found APSGN resolving.

Conclusion
A 13-year-old male adolescent with nephritic syndrome has been reported with RPGN. Treated with corticosteroid, antibi-
otic, immunosuppressant, antihypertensives and diuretic, fol-
lowed by improvement of clinical condition and laboratory of the patient. Kidney biopsy was performed for diagnostic and therapeu-
tic considerations. The biopsy results showed a picture of IgA nephropathy and resolving stage of GNAPS. The prognosis is generally good, with improvement more than 90% of cases. Long-term observation is needed to observe the possibility of the disease being chronic.

Conflict of Interest
There are no conflicts of interest to declare by any of the authors of this report.

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