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Differential Diagnosis and Treatment of Glomerulonephritis

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Abstract: Based on clinical presentation, it is necessary to distinguish between nephrotic and nephritic spectrums. This distinction is crucial as it helps narrow down the differentiation of underlying glomerular pathology. Additionally, differential diagnosis includes primary and secondary causes depending on the age group and clinical presentation. Primary glomerulonephritis, presenting as nephrotic syndrome in young patients, is likely minimal change disease, while in adults, the membranous variant is more common.

Keywords: IgA nephropathy, Rapidly progressive glomerulonephritis (RPGN), Pulmonary edema, Hypertension, Chronic renal failure.

Introduction: It is critical to take a detailed history aimed at identifying the main cause of glomerulonephritis, such as systemic disease or recent infection. Acute glomerulonephritis often affects children aged 2 to 14, presenting with periorbital and facial swelling developing rapidly post-streptococcal infection. Urine is usually dark, foamy, or scanty, and blood pressure may be elevated. Nonspecific symptoms include general weakness, fever, abdominal discomfort, and malaise. In acute glomerulonephritis associated with staphylococcal infection, the patient is often a middle-aged or elderly person diagnosed with diabetes mellitus. Symptoms may coincide with infections like pneumonia, endocarditis, osteomyelitis, or methicillin-resistant Staphylococcus aureus (MRSA) skin infection, frequently with hematuria.

Clinical History and Symptoms: It is essential to gather history about the onset and duration of the disease. Symptoms typically appear suddenly. In acute post-infectious glomerulonephritis (GN), there is usually a latent period of up to three weeks before clinical manifestations appear. However, this period varies, usually being one to two weeks for cases following throat infections, and two to four weeks for cases following skin infections like pyoderma. Nephritis occurring within one to four days post-streptococcal infection usually indicates pre-existing kidney disease. Identifying the potential etiological agent is important, with recent fever, sore throat, arthralgia, hepatitis, valve replacement, travel, or intravenous drug use possibly being related.

Evaluation and Diagnosis: Symptoms include hypertension, edema (initially in low-tension areas like periorbital), abnormal urine sedimentation with microscopic or macroscopic hematuria, oliguria, azotemia, shortness of breath during exertion, secondary headache due to hypertension, and possible secondary confusion from malignant hypertension. There could also be side pain or symptoms related to underlying systemic diseases.

Physical Examination: Physicians should look for signs of excess fluid in the body, such as periorbital and peripheral edema, high blood pressure, small crackles in the lungs due to pulmonary edema, increased pressure in the neck veins, ascites, and pleural effusion. Other signs to note include vasculitic rashes, kidney angle fullness or tenderness, abnormal neurological examination, or altered sensory function, and arthritis.

Diagnostic Tests:

- ✓ Blood tests: A complete blood count can show anemia or pleocytosis in an infectious cause.
- ✓ Serum electrolytes: Increased potassium levels in severe renal failure.
- ✓ Kidney function tests: Elevated BUN and creatinine levels, possibly decreased GFR.
- ✓ Liver function tests: Can indicate the underlying etiology.

- ✓ Immunoglobulins: Important for identifying specific causes.
- ✓ C-reactive protein (CRP): Indicative of inflammation.
- ✓ Complement levels (C3, C4): Help narrow down the differential diagnosis.
- ✓ Autoantibodies (ANA, ANCA, anti-ds-DNA, anti-GBM): Rule out collagenopathies as primary causes of GN.
- ✓ Antistreptolysin O (ASOT) titer: Elevated in 60-80% of cases, indicative of streptococcal infection.
- ✓ Hepatitis serology: Various types of hepatitis can lead to different forms of GN.

Urine tests: Usually dark with red blood cells and specific gravity above 1.020, proteinuria, and decreased creatinine clearance.

Imaging and Biopsy:

- ✓ Chest X-ray: To identify signs of lung bleeding if present.
- ✓ Kidney ultrasound: To assess size and anatomy for biopsy.
- ✓ Kidney biopsy: Essential for evaluating glomerular lesions and confirming GN diagnosis.

Treatment:

1. Specific treatment: Based on immunosuppression determined by histological diagnosis, disease severity, progression, and comorbid conditions.

- ✓ Corticosteroids
- ✓ Rituximab
- ✓ Cytotoxic agents (e.g., cyclophosphamide)
- ✓ Plasmapheresis: Temporarily used in severe cases until chemotherapy takes effect.

2. Chronic treatment: Includes monitoring kidney function, managing blood pressure with loop diuretics and ACE inhibitors or ARBs, treating anemia, bone mineral disorders, acidosis, cardiovascular diseases, and preparing for renal replacement therapy if needed.

Prognosis and Complications: The outcome of nephritic spectrum diseases like PSGN is generally good in children but can result in reduced kidney function and hypertension in adults. IgA nephropathy and Henoch-Schönlein purpura generally have a benign course but can progress to ESRD in some cases. Prompt and aggressive treatment of immunocompromised GN usually leads to remission, but untreated cases have a poor prognosis. Membranoproliferative GN and nephrotic spectrum diseases like minimal change disease have variable outcomes, with some responding well to treatment and others progressing to ESRD. Regular monitoring and appropriate treatment are critical in managing glomerulonephritis and preventing progression to chronic kidney disease.

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