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Nursing Care of a Child with Membranous Nephropathy Combined with Cerebral Venous Thrombosis

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Abstract: *Objective*: To explore the key points of holistic nursing for children with membranous nephropathy (MN) combined with cerebral venous thrombosis, providing a reference for similar cases. *Methods*: The course of illness data of an 8-year-old boy with MN in the recurrent phase complicated with cerebral venous sinus thrombosis (CVST) was retroactively analyzed. Systematic nursing was implemented based on evidence-based practices, including strict neurological monitoring, anticoagulation and thrombolysis medication nursing, fluid and electrolyte management, hypoalbuminemia and edema nursing, complication prevention, and mental health education. *Results*: After 16 days of continuous infusion of low molecular weight heparin and supportive treatment, the child's symptoms, such as headache and vomiting, disappeared. The reexamination of MRV showed significant absorption of thrombosis, and there was no residual neurological deficit. During the 3-month follow-up, the anticoagulation compliance was good, and there was no recurrence. *Conclusion*: Early identification of hypercoagulability risk, strengthening dynamic evaluation of neurological signs and coagulation indicators, standardizing anticoagulation and thrombolysis nursing, and cooperating with continuous health management can significantly improve the prognosis of children with MN combined with CVST.

Keywords: Membranous nephropathy; Cerebral venous sinus thrombosis; Children; Systematic nursing; Case report

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1. Introduction

Membranous nephropathy (MN) is an immune complex-mediated glomerular disease characterized by the deposition of immune complexes beneath the glomerular basement membrane, leading to damage to podocyte function and resulting in massive proteinuria. The immune complexes mainly consist of immunoglobulins (IgG), related antigens, and complements, predominantly IgG4 subtype and complement C3 [1]. The pathological features include diffuse thickening of the glomerular basement membrane with or without spike formation under light microscopy, granular IgG and complement deposition along the glomerular capillary loops by immunofluorescence, and subepithelial electron-dense deposits under electron microscopy. The incidence of MN

is about 5–10 per million population.

Recent studies have shown that MN has surpassed IgA nephropathy as the most common glomerular disease among adults. About 70% of adult MN patients have specific autoantibodies in their serum, known as primary membranous nephropathy (PMN). PMN is a kidney-specific autoimmune disease where circulating antibodies target glomerular podocyte antigens, leading to the deposition of immune complexes beneath the glomerular basement membrane. This activates the complement system, resulting in damage to the glomerular filtration barrier and proteinuria. The remaining 30% of MN cases are secondary to autoimmune diseases, malignancies, infections, or medications, known as secondary membranous nephropathy (SMN) [1].

MN (Membranous Nephropathy) is relatively rare in children, accounting for only 2% to 9% of renal biopsies during the same period. It is mainly secondary membranous nephropathy (SMN) and can be secondary to systemic lupus erythematosus, hepatitis B or C virus infection, secondary and congenital syphilis, malaria, or Epstein-Barr virus infection. Clinically, it usually manifests as nephrotic syndrome or asymptomatic proteinuria, accompanied by microscopic hematuria, azotemia, or mild hypertension.

During the development of nephrotic syndrome, changes in the balance between coagulation and anticoagulation in the body and the composition of the fibrinolytic system, such as activation of coagulation factors, insufficiency of antithrombin, elevation of coagulation factors and fibrinogen, as well as the use of drugs such as hormones and diuretics, can lead to a hypercoagulable state or thrombosis. Common thromboses include deep vein thrombosis of the extremities and renal vein thrombosis, while cerebral venous sinus thrombosis (CVST) is relatively rare. CVST can lead to severe cerebral infarction or cerebral hemorrhage.

2. Case report

An 8-year-old male child was admitted to the hospital due to a confirmed diagnosis of “membranous nephropathy” for more than 5 years and 4 months, and headache and dizziness for more than half a month. The diagnoses were: (1) Nephrotic syndrome; (2) Cerebral venous sinus thrombosis; (3) Left internal jugular vein thrombosis. In June 2019, the child patient developed edema in abdomen and both lower extremities without obvious inducement. The local hospital diagnosed it as “nephrotic syndrome” and treated it with cyclophosphamide pulse therapy and oral prednisone acetate. However, the urine protein did not turn negative. In August 2019, a renal biopsy at another hospital suggested membranous nephropathy. The child was treated with prednisone acetate 40mg and tacrolimus 1mg q12h, and the urine protein turned negative after 1 week. However, after discharge, the urine protein frequently relapsed and could only be turned negative after adding stress hormones for about 1 week. During relapses, the parents adjusted the drug dosage themselves, with the lowest reduction of prednisone acetate to 5 mg qod and tacrolimus 1mg qd. In August 2023, the urine protein relapsed again, with a 24-hour urine protein of 3700mg. Prednisone acetate was adjusted to 40mg qd, and the urine protein decreased to normal after 1 week. The prednisone acetate was then reduced sequentially. In January 2024, the urine protein relapsed again, and the parents increased the prednisone acetate to 40mg qd and tacrolimus to 1mg in the morning and 0.5mg in the evening. After the urine protein turned negative, they reduced the dosage themselves. The urine protein relapsed about 3–4 times in 2024 (with parents adjusting the drug dosage themselves).

On December 1, 2024, when the parents adjusted the hormone dosage to 25mg qod, the child developed cough, vomiting, and headache. He was treated at a local hospital with a 24-hour urine protein of 4380mg. He was given cefoperazone for infection control and intravenous infusion of methylprednisolone 32mg qd. The urine

protein turned negative after about 10 days. However, on December 11, he developed vomiting and headache, mainly in the bilateral temporal regions. A cranial MRI showed: 1. A few abnormal signals in the left temporal region, suggesting thickened blood vessels or subdural hematoma. On December 13, a cranial MRI enhancement scan showed: 1. Thrombosis of the left internal jugular vein, sigmoid sinus, transverse sinus, and superior sagittal sinus. Upper extremity vascular ultrasonography showed no abnormalities. On December 14, low molecular weight heparin sodium 75U/Kg and mannitol 2.5mg/Kg were added. From December 15 to December 16, his mental state improved compared to before. However, on December 17, he developed headache and dizziness again. A repeat cranial MRI enhancement showed thrombosis of the left internal jugular vein, bilateral sigmoid sinuses, transverse sinuses, and superior sagittal sinus, with an increased range compared to before. He was then transferred to this hospital. After admission, the child underwent complete blood routine, stool routine, urine + urine red blood cell phase, cortisol coagulation profile, comprehensive biochemical profile, cranial MRV, and other examinations. Neurology and radiology consultations were requested. Treatment included continuous infusion of low molecular weight heparin, intravenous infusion of mannitol, and supportive care.

3. Physical examination

Body temperature: 36.5°C; Pulse: 80 beats/min; Respiration: 22 breaths/min; Blood pressure: 102/69mmHg; Weight: 36kg. The child appeared to be in a general state of mind, complaining of headache and back pain, morning dizziness, occasional coughing and expectoration, steady breathing, independent posture, and cooperative during the examination. No obvious rash or edema was observed on the body, the thoracic cage was symmetrical without deformity, three concave signs were negative (-), both lungs had a clear percussion sound, both lungs had a rough respiratory sound, no wet rales were heard, the precordial area was not raised, the heart sounds were strong, the heart rate was 80 beats/min, the rhythm was regular, no murmur was heard in the auscultation areas of each valve, the abdomen was soft, the liver and spleen were not palpable under the ribs, no abnormal mass was felt, there was no tenderness or rebound tenderness, and bowel sounds were normal.

After admission, cranial MRI+MRV was performed, which showed uneven signals in the left internal jugular vein, sigmoid sinus, transverse sinus, torcular, and superior sagittal sinus on MRV. T1WI, T2W, and FLAIR sequences showed high signals, DWI showed high signals, ADC signal was reduced, and filling defects were seen on enhanced scanning. The remaining transverse sinuses, sigmoid sinuses, straight sinuses, superior sagittal sinuses, and inferior sagittal sinuses were normal in course, with no obvious stenosis or truncation of the lumen and no filling defects in the lumen. These findings suggested thrombosis in the left internal jugular vein, sigmoid sinus, transverse sinus, torcular, and sagittal sinus.

After the child's admission, the doctor ordered intravenous infusion of 40mg of methylprednisolone combined with tacrolimus to treat the primary disease, intravenous infusion of albumin to increase intravascular colloidal osmotic pressure, reduce tissue edema, and decrease blood viscosity; intravenous infusion of mannitol to lower intracranial pressure and relieve headache symptoms; considering the long duration of thrombosis when the child arrived at the hospital, recombinant streptokinase thrombolytic therapy was not given, and low molecular weight heparin calcium was chosen for intravenous injection. Vital signs, urine output, blood pressure, coagulation function, and other clinical indicators were closely monitored. After consultation with imaging experts, the child's cerebral thromboembolism condition was further clarified, and a cranial magnetic resonance review was performed to observe the thrombosis. After consultation with neurologists, surgical thrombectomy was excluded

as a treatment measure, and continuous infusion of low molecular weight heparin was recommended. Based on expert advice, the department director ordered the infusion of 10,000 units of low molecular weight heparin calcium, with daily coagulation function reviews. Later, based on coagulation function, the dosage was reduced to 5,000 units per day for continuous intravenous infusion.

After 16 days of anticoagulation therapy, the child's symptoms, such as headache, nausea, vomiting, disturbance of consciousness, and visual abnormalities gradually disappeared. Multiple magnetic resonance reviews showed a gradual reduction in infarction area compared to before, and collateral circulation was established. The child was then discharged with oral rivaroxaban 10mg qd and weekly coagulation function reviews. Two weeks later, magnetic resonance was reviewed, and the child continued medication for membranous nephropathy with regular follow-up.

4. Nursing care

4.1. Condition observation

(1) Vital signs: Closely monitor changes in the child's body temperature, pulse, respiration, blood pressure, and blood oxygen saturation. Report any abnormalities to the doctor immediately and take prompt action.

(2) Neurological symptoms: Closely observe the child's consciousness, state of mind, and pupil light reflex; observe for symptoms such as headache, vomiting, disturbance of consciousness, convulsions, and limb movement disorders. Report any abnormalities to the doctor promptly.

(3) Edema: Observe the location and degree of edema, associated symptoms and signs, presence of pleural or peritoneal effusion, closely monitor the child's weight, abdominal circumference, and 24-hour intake and output.

(4) Auxiliary examination: Closely monitor the child's urinary protein quantitation, urinalysis, serum creatinine, electrolytes, albumin, white blood cell count, coagulation function, etc. [2–4].

4.2. Rest and activity

Create a good environment in the patient room, ensure adequate sleep for the child, absolute bed rest during the acute phase, avoid strenuous activity and head vibration to prevent blood clots from falling off or aggravating bleeding. After the condition stabilizes, gradually increase activity levels under the guidance of a doctor.

4.3. Dietary care

Follow doctor's orders for a low-salt, low-fat, high-quality protein diet, eat more fresh fruits and vegetables, maintain regular bowel movements, avoid straining during defecation to increase abdominal pressure, which may lead to increased intracranial pressure. Protein intake should be 1.5–2g/(kg*d), preferably with high biological value animal protein (milk, fish, eggs, poultry, etc.). Food should be diversified, light, and avoid cold, spicy, seafood, and fried foods.

4.4. Medication care

4.4.1. Anticoagulants

Accurately administer medication as prescribed, closely observe for any bleeding tendencies such as skin petechiae, gum bleeding, etc., and regularly monitor coagulation function [5].

4.4.2. Dehydrating agents

Strictly follow doctor's orders for medication, closely observe the infusion drip rate and the skin condition at the puncture site; observe whether the child has symptoms such as headache, dizziness, and palpitations; observe for signs of water and electrolyte imbalance, such as fatigue and abdominal distension; closely monitor changes in urine output [6].

4.4.3. Glucocorticoids and immunosuppressants

When administering glucocorticoids and immunosuppressants, observe for drug side effects such as infection, elevated blood sugar, etc., and regularly check blood routine, blood sugar, liver, and kidney function.

4.5. Infection prevention

(1) Strictly implement sterile operation principles; keep the patient room clean and tidy, ventilate regularly; isolate and protect infected children in separate rooms.

(2) Perform good oral hygiene to prevent respiratory infections; provide good skin care, regularly turn over during bed rest to avoid long-term pressure on local skin and prevent skin infections.

(3) Strengthen genital care for children to prevent urinary tract infections.

4.6. Psychological care

Utilize the Rosenthal Effect nursing model, employing various methods such as attitude, expression, and behavior to provide positive psychological suggestions to the child, thereby fostering optimistic emotions and changing individual behavior [7]. After the child is admitted to the hospital, the responsible nurse actively communicates with the family members, carefully evaluates their knowledge of the disease, and provides random education targeting the family members' cognitive weaknesses. After the education is completed, invite family members to participate in the child's care, improving their caregiving abilities. Encourage family members to give more care and love to the child, enhancing the child's sense of security. The responsible nurse should fully understand the child's psychological development, incorporating psychological comfort throughout the entire nursing process. When the child's emotions stabilize and they show good treatment compliance, timely praise and affirmation should be given, conveying expectations and helping the child establish a strong and unyielding belief.

4.7. Continuing care

Establish a multidisciplinary continuing care team within the department, consisting of 2 dedicated nurses, 1 specialist doctor, 1 nutritionist, and 1 psychologist. All team members undergo professional training and assessment. Before discharge, nurses carefully evaluate the child's physical, psychological, and healthy behaviors, establish a continuing care file, communicate with family members to understand the child's daily preferences and home care needs, and develop a targeted continuing care plan. Arrange for team members to conduct regular follow-ups and systematically evaluate the child's condition. Establish a continuing care WeChat group where team members share knowledge about nephrotic syndrome recovery and self-care [8, 9].

5. Discussion

Proteinuria in children with nephrotic syndrome (NS) leads to the loss of anticoagulants such as antithrombin

III. Additionally, the use of hormones and calcineurin inhibitors can exacerbate hypercoagulability, which is a significant risk factor for cerebral venous sinus thrombosis (CVST). Domestic reports indicate that active thrombolysis, anticoagulation combined with albumin supplementation can significantly improve prognosis. However, during this period, rigorous nursing care is required to balance the risks of bleeding and re-embolization. In this case, a three-stage nursing model of "Complete, Continuous, Family" was adopted: acute phase in the hospital focused on neurological and coagulation monitoring; transition phase emphasized lifestyle guidance and psychological intervention; and post-discharge continuous follow-up ensured long-term compliance, achieving good results.

6. Conclusion

Currently, there is no consensus on the standardization of anticoagulant types and dosages used to treat deep venous thrombosis in children. Randomized controlled clinical trials on thrombolysis in children are relatively few, with limited reference data and a lack of evidence that thrombolytic therapy is superior to anticoagulation therapy. Thrombolysis carries a risk of bleeding, and there is no clear consensus on the optimal type, dosage, and administration method. Children are young and have incompletely developed blood vessels. Therefore, in the diagnosis, treatment, and care of such patients, doctors need to have rich clinical experience, and nursing staff need to have strong emergency response capabilities, proficient knowledge of the disease, and emergency skills to provide timely and effective treatment and care, improve patient prognosis, and enhance the quality of life. In this case, comprehensive nursing care was provided, including condition observation, diet management, psychological and cognitive intervention, and other perspectives, which effectively improved clinical symptoms and conditions, reduced the risk of complications, increased family members' awareness, enhanced the enthusiasm of children and their families for treatment, and ensured the smooth progress of clinical treatment to achieve early expected treatment goals.

Disclosure statement

The authors declare no conflict of interest.

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