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(CASE REPORT)

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Acute post-streptococcal glomeru bilateral pleural effusion in a 12-year-old child in rural area: A case

Ana Silvi Ni'ma [1, *] and Mochamad Chabibi [2]

[1] *General Practitioner in Emergency Department of Ngudi Waluyo Regional General Hospital, Blitar, East Java, Indonesia.*
[2] *Pediatrician in Pediatric Department of Ngudi Waluyo Regional General Hospital, Blitar, East Java, Indonesia.*

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Abstract

Background: Nephritic syndrome is a group of symptoms such as hematuria, hypertension, oliguria and edema. The most common cause of acute nephritic syndrome in children is acute glomerulonephritis after streptococcal infection.

Case Presentation: A 12-year-old child came to the ER with shortness of breath for 3 days and swelling of the face. There was no history of fever or diarrhea. The patient had a history of itching of the feet 2 weeks earlier. Vital signs found blood pressure of 160/120 mmHg and edema was found on the face especially in the eyelids. Urine examination found a high number of leukocytes and erythrocytes and there was albumin +2 in the urine. Examination of kidney function showed an increase in urea and creatinine. Chest X-ray examination showed pulmonary edema and bilateral effusion. ASTO titer examination obtained a level of 200 IU/ml. The patient was given therapy in the form of antihypertensives, antibiotics and corticosteroids. After 1 week of hospitalization, the blood pressure target and clinical improved, the patient was able to leave the hospital, checked into the polyclinic to continue treatment and evaluated urine examination of kidney function every 2 weeks.

Summary: Acute nephritic syndrome is a disease that is rarely found in pediatric patients, most of the causes are acute glomerulonephritis after streptococcal infection with clinical manifestations in the form of throat infection or skin infection in the form of impetigo. With proper diagnosis and management, this disease can be cured with perfect healing, but some cases can occur acute kidney failure so that monitoring is needed.

Keywords: Nephritic syndrome; Acute glomerulonephritis After *streptococcus*; Almonry edema

1. Introduction

Acute post-streptococcal glomerulonephritis is a condition in which there is a decrease in kidney function due to an inflammatory response in the form of a type III hypersensitivity reaction due to streptococcal infection. This condition is specifically caused by group A beta hemolytic streptococci or nephrogenic streptococci. This disease occurs in the kidneys, especially in the glomerulus and surrounding blood vessels. GNAPS often attacks children, especially 1-2 weeks after they have a sore throat or 6 weeks after a skin infection in the form of impetigo. This nephritic syndrome mostly consists of various symptoms in the form of hematuria, oliguria, hypertension and edema. A small part of the symptoms are similar to nephrotic syndrome with symptoms in the form of proteinuria.[1]

This study is a case report on a 12-year-old child diagnosed with GNAPS who had bilateral pleural effusion in both lungs. This case report was chosen with the aim of studying the condition in more depth. Data were obtained from medical records and the results of direct examinations of patients supported by several literature reviews. Patient medical

* Corresponding author: Ana Silvi Ni'ma

records in the form of anamnesis, physical examination results, laboratory results and x-ray examinations related to cases of GNAPS and bilateral pleural effusion in children.

In this case, the patient experienced swelling in the face especially after waking up in the morning. The patient also experienced shortness of breath. The patient had a history of skin infection about 2 weeks before the complaints of shortness of breath and swelling appeared. On examination, there was high blood pressure and swelling was found in both eyes and face.

Pharmacological therapy for GNAPS in the form of diuretics, antihypertensives in the form of calcium channel blockers, ace inhibitors or arbs and symptomatic therapy. Antibiotics can be given in the acute phase and there is evidence of streptococcal infection. Antibiotics are not given as therapy for glomerulonephritis, but are useful for reducing the spread of existing streptococcal infection. Immunosuppression therapy can be given to reduce the progression of kidney failure. Dialysis is also needed for cases of kidney failure and electrolyte imbalance. [2]

In conclusion, GNAPS is a condition that must be taken seriously considering that this disease can cause kidney failure and several other conditions that can cause death. However, this disease has a good prognosis with complete recovery in children if treated appropriately.[3]

2. Case Report

2.1. Identity: An F/12 years/35 kg/male/Blitar

2.1.1. Anamnesis

A 12 years old male was admitted to the emergency room with complaint of shortness of breath 3 days before going to the hospital. Shortness of breath was felt both during activity and rest, shortness of breath did not get worse and did not improve with changes in position. Complaints of fever, cough, runny nose and sore throat were denied by the patient. The patient also complained of swelling in the face, especially around the eyes. Complaints were felt mainly in the morning and the swelling decreased during the day. The swelling was felt 3 days before coming to the ER. Facial swelling was not accompanied by pain. The patient also complained of abdominal pain and felt hard since 4 days before came to ER. Normal bowel movements, no complaints of diarrhea or constipation.

About 2 weeks before shortness of breath, the patient had a history of itchy feet. The itching was initially reddish, initially filled with clear fluid then burst. The itching was initially only slight, but then spread to both feet, especially the right foot. The patient was given antibiotic ointment. When in the emergency room, the itching had healed and the lesions were scabs.

The patient had been treated at the health center and given medication for shortness of breath in the form of salbutamol, but the shortness of breath did not improve.

The patient's delivery history was a spontaneous normal birth, full term and assisted by a midwife. The patient's birth weight was 3500 grams, cried loudly, was not blue. During pregnancy, the patient's mother routinely checked her pregnancy with a midwife and never had any specific complaints. The patient's growth and development history is according to age. The patient can lift her head at the age of 3 months, lie on her stomach at the age of 6 months, sit at the age of 8 months, stand at the age of 12 months and walk at the age of 15 months. The patient was able to talk at the age of 1.5 years and until now there have been no communication problems between the patient and those around her. As a baby, the patient received sufficient breast milk until the age of 15 months, then continued with formula milk. The patient started MPASI at the age of 6 months with sufficient nutritional composition. At the age of 1 year the patient was able to eat family food. There is no history of food allergies such as chicken, eggs and fish. The patient has no history of food, dust, and drug allergies. A history of congenital heart disease is also denied. There is no history of surgery. Complete basic immunization. There is no history of the same illness as the patient's family. The patient is currently in 6th grade of elementary school and currently lives at home with the patient's parents and grandmother with adequate ventilation and clean water sources.

2.2. Physical Examination

General condition is good, consciousness compos mentis, Anthropometric examination BB 35 kg, TB 135cm good nutritional status.Vital signs found BP 160/120 mmHg, pulse 90x/minute lifting strength, RR 24x/minute, T 37.5 C

On examination of the head and neck, periorbital edema was found. No signs of anemia and jaundice were found. No enlarged tonsils T2/T2, no pharyngeal hyperemia. The first and second heart sounds are normal without murmurs. On lung examination, rhonchi and dull percussion were found in the inferior 1/3. Abdomen was found swollen, extremities no edema, lower extremities found hypopigmented macular lesions, no crusts, acral felt warm.

2.2.1. Supporting examination

Laboratory examination showed hemoglobin 11.6 g.dl; wbc 10.7. 103 / ul; platelet 450.103 / ul; BUN 52 mg / dl; SK 0.86 mg / dl; AST 19 IU / l; ALT 14 IU / l; Na 139.5 mmol / l; K 4.86 mmol / l; albumin 3 g / dl; cholesterol 140 mg / dl, ASTO 200. Urinalysis showed turbidity; PH 5.0; BJ 1,015; leukocytes 10-12 / LPB; uncountable erythrocytes / LPB, urine albumin +2; bilirubin (-). On ECG examination, normal sinus rhythm was found 89x/minute; chest x-ray showed cardiomegaly, pulmonary edema and bilateral pleural effusion.

2.2.2. Analysis

Based on the patient's anamnesis, periorbital and abdominal swelling was found. The patient also felt short of breath, from the patient's medical history, an infectious rash appeared on the legs 3 weeks earlier. On physical examination, the patient's blood pressure was high, periorbital edema and breath sounds contained rhonchi and dull percussion in the inferior 1/3 of the lung field. Supporting examination found on chest x-ray bilateral effusion, cardiomegaly and pulmonary edema. Blood examination found increased BUN (52) Albumin (3) ASTO (200). Urinalysis examination showed high uncountable erythrocytes and urine albumin (+2). So, the working diagnosis in the patient is acute glomerulonephritis with bilateral pleural effusion.

2.2.3. Management

On the first day of treatment, the patient was given diuretics and CCB antihypertensives. The patient was given injection of furosemide 40 mg every 8 hours and amlodipine 5 mg every 24 hours. Given a low-salt diet of a maximum of 2 grams per day and 1700 kcal. Monitoring of fluid balance, electrolytes and vital signs was carried out. On the third day of treatment, a steroid therapy program was started, with oral methylprednisolone 4mg-3mg-2mg. On the fifth day of treatment, complaints of swelling and shortness of breath decreased and blood pressure had decreased so that furosemide 40 mg was given orally once a day and PO erythromycin 4x400 mg was given after the ASTO results came out. On the seventh day, complaints of swelling and shortness of breath had greatly decreased, the blood pressure target had been achieved, the UL results had shown improvement, so outpatient treatment was planned with per oral treatment. Patient gave per oral amlodipine 1x5 mg, erythromycin 4x400 mg and methylprednisolone 4-3-2. Urinalisis and renal function test evaluation would be carried out every 2 weeks.

3. Discussion

Acute post-streptococcal glomerulonephritis is a disease involving decreased kidney function due to an inflammatory response process, namely a type 3 hypersensitivity reaction due to a streptococcal infection. The type of streptococcus that causes this reaction is group A beta hemolytic streptococcus which is also known as nephrogenic streptococcus. This disease attacks the glomerulus and blood vessels of the kidneys. GNAPS usually occurs in children who previously had symptoms of sore throat 1-2 weeks earlier or 3-6 weeks earlier had a skin infection in the form of impetigo. The prevalence of streptococcal infection is higher in developing countries because it is related to sanitation and access to health workers. [4]

The prevalence of streptococcal infection is higher in developing countries because it is related to sanitation and access to health workers. The incidence of GNAPS is rare in children under 2 years of age because they still have immature antibody responses. The population of children who often get this infection is aged 3-12 years. This incident often occurs in boys than girls with a ratio of 2:1. Frequent symptoms are the appearance of edema or swelling and hematuria both macroscopic and microscopic. Some cases can develop into acute renal failure and require further treatment. [5]

Nephrogenic streptococci that cause APSGN begin with infections of the skin or oropharynx. Nephritis associated plasmin receptor (NAPIr) and streptococcal pyrogenic exotoxin B (SpeB) are the two antigens most frequently associated with the pathogenesis of APSGN. Both antigens activate the complement pathway and have affinity for plasmin and glomerular proteins. [6]

The mechanism of this disease involves a type III hypersensitivity reaction. The body responds to nephrogenic streptococcal infection by forming an immune complex consisting of streptococcal antigens and human antibodies. Some theories suggest that this immune complex process is deposited in the glomerulus and spreads into the body's

circulation. Another theory suggests that this immune complex mechanism is a condition of "in situ" formation in the glomerulus. The presence of an immune complex activates the complement pathway which can cause an increase and infiltration of leukocytes and proliferation of mesangial cells in the glomerulus which affects capillary perfusion and affects the glomerular filtration rate. Decreased GFR can lead to renal failure, acid-base and electrolyte imbalances, edema and hypertension. [7]

Histopathologically, in GNAPS disease, microscopic examination shows that the glomerular cells are hypercellular, there are deposits of electrons on the basement membrane in the glomerulus and there is evidence of deposits of IgG and C3 in the glomerulus in the first 2-3 weeks. There are several other causes of APSGN other than Group A B hemolytic streptococcus, including endocarditis, enterocolitis, pneumonia, viral infections such as hepatitis B and C, HIV, CMV, Epstein Barr, parvovirus, fungal infections and parasitic infections such as malaria, leishmania, toxoplasma and schistosomiasis. [7]

Poor sanitation and hygiene and low socioeconomic status are the main factors causing streptococcal infections. Genetic factors can also influence about 40% of the incidence of this disease, but the specific gene that causes APSGN has not been found. [8]

About 50% of patients with asymptomatic APSGN are accidentally found in urine tests. The triad of APSGN is hypertension, edema and hematuria. Usually patients previously had infections such as pharyngitis, tonsillitis or impetigo. However, several cases have been found in patients with APSGN who previously had no bacterial infections elsewhere. [9]

Kidney function is greatly affected by this infection. Depending on the severity, symptoms that can appear range from anuria to kidney failure that affects acid-base and electrolyte imbalances that can be life-threatening. Fluid overload can be caused by this mechanism. About 60-80% of patients have high blood pressure and usually recover on the 10th day. [7] The incidence of edema or swelling is around 60-90% of cases. Swelling of the eyelids (periorbital edema) is a typical symptom of nephritic syndrome. This symptom appears clearly in the morning and decreases during the day. swelling throughout the body can also occur. Some cases can also cause patients to experience shortness of breath and respiratory distress originating from pulmonary edema. Some also complain of decreased appetite, nausea and vomiting. [8]

GNAPS should be suspected in patients with hypertension and heart failure (cardiomegaly), even though the patient does not have hematuria or a history of sore throat and pyoderma. Several supporting examinations can be used to diagnose and evaluate GNAPS, Among them are:

- Evidence of previous streptococcal infection with ASO titer examination. ASO titer is a test that is often used, but can be low due to previous antibiotic administration. Serum complement C3 can also be examined to measure inflammatory reactions. Complement C3 levels can decrease before ASO titer increases. Complement C3 levels can be normal in the 6-8th week.
- Urine analysis shows macroscopic or microscopic hematuria. The presence of erythrocytes and mild proteinuria, only 5% of patients with GNAPS have high proteinuria. Leukocytosis usually also appears on urinalysis.
- Kidney function tests such as BUN (Blood Urea Nitrogen) and SK (serum creatinine) can also increase during the acute phase.
- In patients with heart failure, NT pro BNP examination also increases along with pulmonary congestion on X-ray examination.
- Renal biopsy is not recommended for diagnosing patients with PSGN, and is only performed if there is suspicion of renal pathology.[9][10]

3.1. Some other supporting examinations are

Ultrasound examination shows kidney enlargement. X-ray examination shows pulmonary congestion.

GNAPS can be categorized as a self-limiting disease or can heal itself in most cases, but supportive treatment is still needed to prevent complications from volume overload such as hypertension and edema that occur in the acute phase. [7]

3.2. Pharmacological therapy includes

- Antibiotics: patients with evidence of streptococcal infection need to receive antibiotics to kill any remaining bacteria, but do not prevent the development of GNAPS itself
- Diuretics: Loop diuretics such as furosemide are preferred over thiazides because the efficacy of thiazides is less than furosemide in GFR conditions <30 ml/minute.
- Antihypertensives: Blood pressure can be lowered by salt and fluid restriction and diuretics. Some cases require other antihypertensive therapy such as calcium channel blockers, ACE I and ARB. However, CCB is more recommended.
- Immunosuppressive therapy: there is no scientific evidence to show the effectiveness of this therapy in patients with GNAPS. However, therapy with corticosteroids is needed in patients suspected of having kidney failure. [14]
- Dialysis: dialysis is needed if there is already an acid-base imbalance and electrolyte abnormalities such as hyperkalemia [12][14]

3.3. The Differential diagnosis of GNAPS

- IgA nephropathy: usually occurs after upper respiratory infection or infection in the digestive tract. To differentiate with AGNPS is the time of appearance of nephritis symptoms and previous infection. Usually the time is almost the same.
- Membranoproliferative glomerulonephritis: nephritis symptoms preceded by upper respiratory infection with a mechanism of hypocomplementeria. The difference is that in this disease, the measured complement level takes longer to return to normal than AGNPS
- Lupus nephritis: the symptoms are similar to AGNPS symptoms, the difference is the presence of ANA test, ds DNA, positive cytopenia in Lupus nephritis and the involvement of other organ damage
- Nephrotic syndrome: the presence of massive proteinuria is characterized by the examination of protein in urinalysis obtained more than 3.5 grams / day. other symptoms that appear are hypoalbumin, edema, and increased risk of thrombosis
- Henoch Schonlein purpura (HSP): clinical symptoms that are classed as purpura on the palpebra, kidney failure and manifestations in the digestive system and musculoskeletal system. Complement levels are normal. [13]

The prognosis of APSGN is complete recovery in 6-8 weeks. In adults, about 50% of patients can fall into a condition of decreased kidney function, hypertension, and persistent proteinuria. Death in adults can occur due to complications of heart failure and kidney dysfunction, studies show that in long-term examinations, adult patients still have abnormalities in urine production, proteinuria and persistent hypertension. Mortality during the acute period of APSGN is 2-12 percent. Most cases result from untreated kidney failure [11][15]

The most dangerous complications during the acute phase of APSGN are congestive heart failure and azotemia. Chronic complications include chronic renal failure and nephrotic syndrome [13]

4. Conclusion

From the case described, the patient can be concluded to have acute post-streptococcal glomerulonephritis with complications in the form of bilateral pleural effusion. GNAPS is a condition in patients that appears after infection with group A beta hemolytic streptococcus. This is based on the patient's history of illness that has an infection in the upper respiratory tract or skin infection.

Physical examination found swelling on the face especially on the eyelids, high blood pressure and bilateral pleural effusion. Laboratory examination found hematuria, proteinuria and high ASTO levels can support this diagnosis. Based on the analysis and examination found, appropriate and comprehensive management is needed for the patient.

Management of patients with GNAPS is to relieve symptoms, prevent complications and worsening of the kidneys and provide supportive therapy. Patients are given antibiotic therapy to kill the bacteria that cause GNAPS. In addition, patients are given diuretics to treat swelling, antihypertensives to lower blood pressure. A low-salt diet is recommended to reduce urinary retention. The important thing is to regularly monitor kidney function in patients.

Therapy should be given based on the patient's condition and it is necessary to monitor the patient's clinical condition and treatment response and detect any complications. With comprehensive therapy, it is expected that patients can achieve recovery from this disease and its complications.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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