MANAGEMENT OF ACUTE GLOMERULONEPHRITIS IN A CHILD WITH ISCHEMIC STROKE AND HYPERTENSIVE CRISIS

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ABSTRACT

Acute glomerulonephritis refers to a specific set of renal diseases in which an immunologic mechanism triggers inflammation and proliferation of glomerular tissue. Some patients present with hypertensive emergency and cerebral involvement. It was uncommon case of ischemic stroke accompanied by acute glomerulonephritis. The patient was 8 year-old boy admitted to the hospital with the main complaint of unconsciousness. He had seizure and dark urine. Physical examination revealed blood pressure 160/120 mmHg, anisocor pupils and paresis. Urine examination showed proteinuria and urine sediment full of erythrocyte and white blood cells. ASTO titer 800 IU/ml and low level of C3. Head CT scan showed a wide embolic ischemic cerebral infarction in right fronto-temporo-parieto-occipital region that appropriate with right media cerebral artery and perifocal edema. He was treated with furosemide, nifedipin, manitol and dexamethasone. The condition improved with the treatment without decompressive trepanation. The patient was discharged in good condition.

Keywords: acute glomerulonephritis, ischemic stroke, hypertensive crisis, clinical signs, management, improved

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INTRODUCTION

Acute glomerulonephritis refers to a specific set of renal diseases in which an immunologic mechanism triggers inflammation and proliferation of glomerular tissue. It includes a variety of diseases with diverse etiologies and variable morphologic appearances clinically presenting with a nephrotic syndrome (Cole 2000; Sulyok 2004). It has been reported to range from 1 to 33% of the patients with the overall risk of approximately 15%. The disease is more frequent among children between the ages of 2 and 12 years. Boys are more often affected (Cole 2000; Hoyer 2000). The most common presenting symptoms are hematuria, edema, proteinuria and hypertension. Hypertension is observed in more than 80% patients. Some patients present with hypertensive emergency. Central nervous system involvement during the course of acute poststreptococcal glomerulonephritis may be secondary to hypertensive encephalopathy, but it can also be attributed to other causes (Hoyer 2000; Ughi 1999). The objective of this case report is to report a case of acute glomerulonephritis in a child with ischemic stroke focusing on successful management.

CASE REPORT

A, with body weight of 21 kg was referred by pediatrician to Emergency Unit, Dr Soetomo Hospital, with the main complaint of unconsciousness. He was unconscious since 4 days before admission with general seizure, low grade fever and swelling from foot and then spread all over the body since 8 days prior to admission. There was also a history of dark urine. Physical examination revealed a weak and unconscious boy with the blood pressure: 160/120 mmHg. Edema was present on both of lower extremities. The boy was sopor (GCS 2-1-4), anisocor pupils, 6 mm/3 mm (Figure 1) with decreased of light reflexes, paresis of left extremity and increase of physiological reflexes.

The laboratory examination showed creatin 1.3 mg/dL, BUN 30, 2 mg/dL. Urine examination: proteinuria, urine sediment: full of erythrocyte, full of leucocytes. ASTO titer 800 IU/ml and low level of C3. Electrocardiography was normal. Head CT scan revealed a wide embolic ischemic cerebral infarction in right fronto-temporo-parieto-occipital region that appropriate with right media cerebral artery and perifocal edema (Figure 2).
Based on the history, physical examination, laboratory and radiology evaluation the diagnosis of acute post streptococcus glomerulonephritis with hypertensive crisis, cerebral infarction and cerebral edema were established. The patient was given oxygen, fluid restriction, fasting, ampicillin 750 mg every 8 hours, furosemide 20 mg intravenously every 12 hours, nifedipine 2 mg every 4 hours sublingually, mannitol 10 g intravenously, and dexamethasone 10 mg followed by 2 mg every 8 hours intravenously and the patient was observed in pediatric intensive care.

On the follow up, he was still sopor. Blood pressure was 140/110 mmHg. The neurosurgeon decided to perform decompressive trepanation to decrease intracranial pressure. Fortunately, on the next day the consciousness improved. Blood pressure was 140/100 mmHg. The decompressive trepanation was cancelled. To overcome the hypertension, captopril 6.25 mg every 8 hours was administered orally and furosemide was discontinued. One week after admission, the child alert and the blood pressure became gradually normal (120/80 mmHg). Based on this result, mannitol infusion was ceased. To prevent neurological sequelae, physical rehabilitation was initiated. Two weeks after admission, the condition was well. Blood pressure was normal and the GFR 90 ml/minute/1.73mm². Repeated CT scan evaluation showed sub acute thrombo-embolic infarction in temporo-parietal region. No appearance of midline structure deviation (Figure 3).

The patient was discharged one month after admission in a good condition. He could open his lid normally and able to sit and walk by himself.

**DISCUSSION**

Childhood stroke is defined as a cerebrovascular event that occurs between 30 days and 18 years of age. Multiple risk factors may coexist in childhood stroke (Delsing 2001). The mechanism by which ischemic stroke occurs in children include the following: thromboembolism from an intracranial vessel, or the heart; acute, transient, or progressive arteriopathy and other rare causes, but in a large percentage of cases the cause is undetermined (Younkin 2003). It was uncommon case of ischemic stroke accompanied with acute glomerulonephritis. We did not find abnormality of the heart. Cerebral vasculitis was considered the etiology of ischemic stroke. Central nervous system vasculitis can lead to arterial thrombosis, central venous thrombosis, or intracranial hemorrhage (Delsing 2001; Younkin 2003).
The patient suffered from hypertensive crisis. Blood pressure should be decreased to prevent damage of the target organ. Herce said that nifedipine sublingually could be used for hypertensive crisis. Nifedipin acts as calcium-channel blockers interfere with the inward displacement of calcium ions through the slow channels of active cell membranes (Herce 1998). In our case, nifedipine sublingually was combined with furosemide. Furosemide acting as loop diuretic can induce a much greater peak diuresis than other agents. Furosemide has other advantages. It can decrease the cerebral edema. In greater peak diuresis than other agents. Furosemide has other advantages. It can decrease the cerebral edema. In hypertensive crisis, blood pressure reduction is suggested not more than 25% in 6 hours to prevent brain hypertensive crisis, blood pressure reduction is done in 24-48 hours. In this case, the blood pressure reduction was about 12.5% from 160/120 mmHg to 140/110 mmHg on the first day, and then the blood pressure decreased gradually. The blood pressure was normal after 3 days therapy.

ACE inhibitors are important in the management of hypertension. They inhibit the conversion of angiotensin I to angiotensin II, which lowers peripheral arteriolar resistance. In our case, captopril was given orally beside nifedipine until the patient discharged from hospital. A stepped-down therapy for hypertension management in children is considerable. Blood pressure is maintained for 6 until 8 weeks to change in monotherapy (Ingerfinger 1990). In this case, furosemide was ceased in few days after the blood pressure was normal. After 12-day therapy with nifedipine and captopril, nifedipine was stopped. The ACE inhibitors may be better drugs for first-line therapy of mild hypertension (Sinaiko 2000).

There have been no randomized, clinical trials for the acute treatment of ischemic stroke in children (Lynch 2002). The management of this kid was described to decrease the intracranial pressure caused by the cerebral edema. The treatment included decreasing of intracranial components volume; maintaining the brain metabolic function and avoiding conditions inducing elevated intracranial pressure (Morries 2002). Restriction fluid about 60% was given in this case to decrease brain volume (Morris 2002). In addition, mannnitol infusion was administered as osmotherapy to improve cerebral perfusion by decreasing viscosity and protect brain damage (Bereckzi 2000). Corticosteroid was used for this patient because it may decrease cerebral edema and fixes the membrane integrity by maintaining permeability of endothelial cells (Lanthier 2000).

In our case, we used piracetam to treat the ischemic stroke. Piracetam is an agent with neuroprotective properties. It has been reported in pilot studies to increase compromised regional cerebral blood flow in patient with acute stroke and given soon after onset, to improve clinical outcome (Kessler 2000). If the elevated intracranial pressure still persists with those types of therapies, the surgical correction is needed (Nicholas 2003). One rationale argues that edema compresses adjacent tissue, causing secondary damage and extension of the infarction into the ischemic penumbra, and it should be treated as early as possible (Berdnt 2000; Nicholas 2003). Decompressive hemicraniectomy as a lifesaving procedure in severe acute ischemic stroke is associated with lower mortality (Attia 2003). Fortunately, the patient was conscious after treatment and the surgical intervention was cancelled. Repeated CT scan of this patient revealed sub acute embolic-stroke and no deviation of midline structure. Compared with previous CT, it suggested an acute process of stroke had already passed and the reduction of cerebral edema made a good clinical appearance, although the probability of residual sequelae process remains high in this child.

Rehabilitation was needed for this patient to improve motoric function (Delsing 2001; Lynch 2002). Complete recovery occurs in more than 95% of children with acute post streptococcal glomerulonephritis. Mortality in the acute stage can be avoided by appropriate management of acute renal failure, cardiac failure, and hypertension (Cole 2000; Sulyok 2004). The outcome of childhood stroke can be very difficult to predict. Children with stroke are at risk for future cognitive impairment, motor impairment and epilepsy (Lynch 2002). In our case the patient was on critical illness when he was admitted, and he was discharged in a good condition after medical treatment.

REFERENCES


