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Hypertension due to renal artery stenosis

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ABSTRACT. A case of a 12-year-old boy with renal artery stenosis as a cause of hypertension is presented. The diagnosis of renal artery stenosis was established based on the bruit heard over costovertebral angle and the increased plasma renin secretion, and further confirmed by angiogram finding. The detection of bruit, either on the abdomen or costovertebral angle, in association with hypertension should initially lead to the consideration of the presence of renal artery stenosis. **[Paediatr Indones 2001;41:60-63]**

Keywords: *renal artery stenosis, hypertension*

HYPERTENSION IS DEFINED AS AN AVERAGE SYSTOLIC AND diastolic blood pressure greater than 95th percentile for age and sex measured on at least three separate occassions.[1] Hypertension associated with renal diseases is commonly found in children. Previous reports stated that most secondary hypertension in childhood and adolescence is duc to renal and renovascular disorder whereas other causes, including coarctation of the aorta, pheochromocytoma, Cushing's syndrome, and primary aldosteronism, accounts for only a minority of cases.[2,3] The prevalence of renal parenchymal diseases as the underlying causes of secondary hypertension in children adolescence is higher (60 to 80%)[3] than those of renovascular lesions (10% to 24%).[4,5]

Since hypertension may occasionally be an acute emergency in children because of the impact of severely elevated blood pressure on vital organ systems resulting in hypertensive encephalopathy, symptomatic pulmonary edema, cardiovascular failure and hypertensive retinopathy,[1,8] it should be treated appropriately without delay for the stabilization of the blood pressure. The purpose of this report is to describe a case of renal artery stenosis as the underlying cause of hypertension in a 12-year-old boy.

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Report of the case

A 12-year-old boy was referred to the hospital on July 28, 1997 with a diagnosis of acute glomerulonephritis. The patient complained of having a 6-month history of bifrontal headache, nausea, and vomiting. Periorbital and pretibial edema appeared 1 month before hospitalization. There was no history of urinary tract infection and other symptoms were unremarkable.

Physical examination revealed a well-nourished child with body weight of 25 kg and height of 126 cm. Rectal temperature, pulse rate, respiratory rate, and blood pressure were 37.4°C, 120 per minute, 28 per minute, and 170/120 mmHg (upper extremities) and 180/120 mmHg (lower extremities), respectively. Periorbital and pretibial edema were absent. Pertinent physical findings included normal heart and lungs, bruit on the costovertebral angle and absence of visual disturbances. Others were unremarkable.

Laboratory examination documented normal routine blood data and urinalysis, and urine culture yielded no growth of pathogenic bacteria. Blood ureum and creatinine were 11.5 mg/dl and 0.96 mg/dl, respectively. Creatinine clearance was 86 ml per minute per 1.73 m^2. Albumin, globulin, and cholesterol levels were normal. Urinary excretion per 24 hours was 1500 ml.

Chest X-ray and electrocardiogram were normal. The initial clinical impression was hypertension due to acute nephritic syndrome and suspicion of renal artery stenosis. The patient was advised to have a total bed rest and low salt diet. Ser-ap-es and furosemide were given orally with a good clinical response.

Clinical course

On the 4th day of hospitalization, carpopedal spasm developed. His blood pressure was 140/100 mmHg (upper extremities). Serum electrolytes showed sodium 137 mEq/l, potassium 2.1 mEq/l, chloride 88 mEq/l, and calcium 6.7 mg/dl. The patient was given Elkana (a preparation containing calcium phosphate, calcium lactate), potassium chloride, and Ser-ap-es (reserpin, hydralazine, hydrochlorothiazide) orally t.i.d. Furosemide was discontinued. Other hematologic data showed C3 complement of 145 mg/dl, ASTO < 200 IU. Urinalysis was normal.

On the 5th day from admission, carpopedal spasm disappeared. Cardiologic evaluation showed normal heart size. There was no evidence of retinopathy. On the 9th day of follow-up, the patient complained of headache, nausea, and vomiting. He became unconscious and general convulsion developed. His blood pressure rose up to 170/120 mmHg (upper extremities). He, therefore, was diagnosed as having hypertensive encephalopathy and treated with parenteral clonidine and furosemide. Serum electrolytes showed calcium 9.9 mg/dl, potassium 3.5 mEq/l, sodium 137 mEq/l, and chloride 98 mEq/l. Peripheral plasma renin activity at supine position was 26.64 ng per 100 ml per hour. Renal ultrasonogram showed a normal left kidney and a small right kidney measuring 8.5x4 cm and 5x2 cm, respectively (Figure 1). An intravenous pyelogram showed bilateral equal visualization with right kidney shadow smaller than the left. A renal computed tomogram using contrast medium revealed a normal left kidney and a small right kidney (see Figure 2).

On the 10th day of hospitalization, all signs and symptoms of hypertensive encephalopathy disappeared. His blood pressure decreased to 160/100 mmHg. Parenteral clonidine and furosemide was discontinued and oral captopril 12.5 mg t.i.d. was started. On the subsequent follow-up, the blood pressure decreased to 120/80 mmHg. The patient did not complain of headache, nausea, and vomiting. The bruit remained heard on the costovertebral angle. An abdominal and renal arteriogram demonstrated a normal right kidney and a small left kidney with renal artery stenosis associated with a mild narrowing of the abdominal aorta. (Figure 3).

By the 20th day of hospitalization, the patient remained in a good general condition. His blood pressure was stable at 120/80 mmHg. He was then referred to the Department of Radiology, Medical School, University of Indonesia, Jakarta for percutaneous transarterial angiography (PTA). (Figure 4).

Discussion

Acute renal parenchymal disease is the most common cause of secondary hypertension in children.[2] In acute poststreptococcal glomerulonephritis, 40% to 50% or more hospitalized patients are hypertensive, a quarter of these severely so.[1] Despite the evidence of bruit on his costovertebral angle, we suspected our patient of suffering from acute poststreptococcal glomerulonephritis, because of signs and symptoms of hypertension, and history of edema. Moreover, acute poststreptococcal glomerulonephritis is the most frequent cause of hospitalized patient with hypertension in our Division of Pediatric Nephrology, Dr. Wahidin Sudirohusodo Hospital, Ujung Pandang.

It is evident that bedside Ser-ap-es and furosemide, as the regimen frequently used for hypertension for our patients with acute poststreptococcal glomerulonephritis, failed to decrease the elevated blood pressure in this case, and other findings did not confirm the diagnosis of acute poststreptococcal glomerulonephritis. We therefore performed the modified three-stage investigations,[6] to find out other causes of renal hypertension in our patient. The first test carried out comprised of urinalysis, urine culture, renal function test, intravenous pyelography, and peripheral plasma renin activity. Since the peripheral plasma renin level was remarkably high, we there-

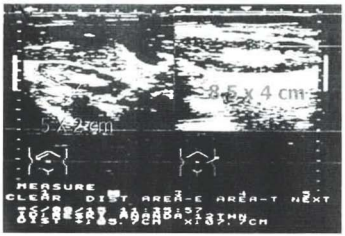

Figure 1. Renal ultrasonogram showed a small right kidney measuring 5x2 cm

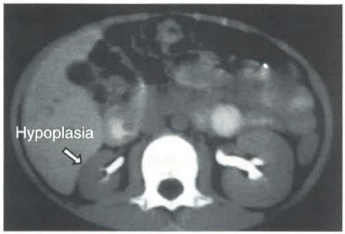

Figure 2. Renal CT using contras medium revealed the right kidney was smaller the left (arrow)

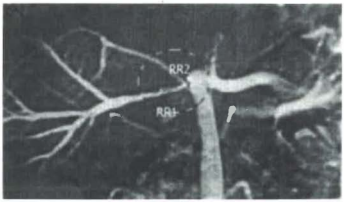

Figure 3. Renal arteriogram demonstrated the stenosis of the main branch of right renal artery (RR1) circle.

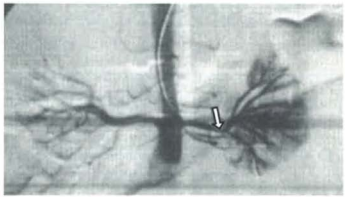

Figure 4. After PTA, the main branch of right renal artery appeared to dilate RR1, arrow.

fore, performed the subsequent tests including renal ultrasonography, renal arteriography, and renal computed tomography to look for renovascular lesions.

The renal ultrasonogram (Figure 1) and computed tomogram (Figure 2) revealed a normal left kidney and a small right kidney. The abdominal and renal arteriogram (Figure 3) demonstrated a normal right kidney and a small left kidney with renal artery stenosis associated with mild narrowing of the abdominal aorta. These findings confirmed the diagnosis of renal artery stenosis as the cause of hypertension.

It is stated that abdominal bruit heard in children with hypertension suggests renal artery stenosis as a cause. However, of 45 children reported with normal findings on renal arteriogram, 13% has abdominal bruit and 19 (30%) out of 63 children with documented renal artery stenosis have abdominal bruit.[4] A bruit may also be occasionally heard over the costovertebral angle.[7] The bruit in our case was only heard over the costovertebral angle.

Hypertensive encephalopathy develops preferentially with an acute rise in blood pressure. Seizures either focal or general seem to occur more frequently in children than adults and are regularly accompanied by loss of consciousness.[8,9] Our patient had a general seizure accompanied by disorder of consciousness. These symptoms disappeared within a few minutes after treatment but complete recovery was achieved within 24 hours and his blood pressure was back to normal within 48 hours.

Hypertension in our patient seemed to result from an overproduction of renin (known to be the main cause of renovascular hypertension)9 as the blood pressure decreased to normal within a few hours after captopril therapy. After the PTA was performed, the right renal artery stenosis appeared to dilate (RR1). It seems that if bruit is heard on the abdomen or costovertebral angle in children with hypertension, then renal artery stenosis as a cause of hypertension should be initially considered.

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