Case Report

Case report of secondary hypertension due to renal artery stenosis in young patient

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Abstrak

Hipertensi sekunder jarang terjadi, namun harus menjadi kecurigaan pada pasien usia muda. Hipertensi sekunder harus didiagnosis dan ditatalaksana secara tepat. Stenosis arteri renalis merupakan salah satu penyebab hipertensi sekunder. Tujuan dari laporan kasus ini adalah menjelaskan diagnosis, patofisiologi dan tata laksana hipertensi sekunder akibat stenosis arteri renalis pada usia muda. Seorang pria berusia 17 tahun dengan gejala sesak nafas didiagnosis menderita hipertensi tahap 3 pada pemeriksaan rutin di Rumah Sakit Pasar Rebo, Jakarta. CT scan abdomen menunjukkan stenosis arteri renalis bilateral. Prosedur invasif PTA (Percutaneous Transluminal Angiography) *arteri renalis kiri dilakukan di Pusat Jantung Nasional Harapan Kita dan satu stent dipasang dengan hasil baik. Tekanan darah setelah pemasangan stent normal.*

Abstract

Secondary hypertension is rare to occur, but should become suspicion in young age. Secondary hypertension must be appropriately diagnosed and treated. Renal artery stenosis is one of many causes of secondary hypertension. The aim of this case report is to describe diagnosis, pathophysiology and management of secondary hypertension due to renal artery stenosis in young patient. A 17 year old man with symptom of shortness of breath was diagnosed with hypertension stage 3 on his medical examination at Pasar Rebo Hospital, Jakarta. Abdominal CT scan examination revealed bilateral renal artery stenosis. Percutaneous transluminal angiography (PTA) of left renal artery was performed at National Cardiovascular Centre Harapan Kita. Stent was placed succesfully and the blood pressure was normalized.

Keywords: renal artery stenosis, secondary hypertension, young age

pISSN: 0853-1773 • eISSN: 2252-8083 • http://dx.doi.org/10.13181/mji.v23i2.666 • Med J Indones. 2014;23:117-21
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Secondary hypertension is an elevated blood pressure that results from an underlying, identifiable, often correctable cause. It is rare to occur, only about 5 to 10 percent of hypertension cases are thought to result from secondary causes.[1] However, because of the overall high prevalence of hypertension, secondary forms of hypertension can affect millions of patients worldwide. Secondary hypertension must be appropriately diagnosed and treated, so patients with a secondary form of hypertension might be cured, or at least show an improvement in blood pressure control and a reduction of cardiovascular risk.[2]

The etiology of secondary hypertension could be one of the diseases that include: renal problem (renal parenchymal disease, renal vascular disease, etc.), endocrine (hypothyroidism, hyperthyroidism, Cushing syndrome, etc.), drugs and exogenous hormones, neurological causes, obstructive sleep apnea, acute stress related secondary hypertension, diseases of the aorta, pregnancy-induced hypertension, and isolated systolic hypertension due to an increased cardiac output. Renovascular disease was the second most often to cause secondary hypertension.[3,4]

The prevalence of renal artery stenosis (RAS) in the general population is small. In general, about 1 to 6% of hypertensive patients have some element of RAS. In patients who undergo diagnostic coronary arteriography, the prevalence rises to greater than 20%. In a prospective study of 1302 patients undergoing coronary arteriography, concurrent abdominal aortography demonstrated significant RAS in 15% of patients.[5]

The process to identify secondary hypertension in young adults and to diagnose the cause is always challenging. The rarity of the case and the complexity make it become important to present this case.

Case ilustration

A 17 year old man was admitted to adult ward of National Cardiovascular Center Harapan Kita (NCCHK) after undergoing renal artery angiography. About 2 years ago, he experienced breathlessness and coughing and was told that he had heart problem. His blood pressure was about 140/80 mmHg. One month ago, on his regular medical control in Pasar Rebo Hospital, the doctor found his blood pressure was about 200/90 mmHg. The physician prescribed him captopril and amlodipine for one month but with no improvement. He has no history of weight gain, fatigue, weakness, hirsutism, weight loss, hair loss, heat intolerance, tremor, fever, prolonged cough, light headedness, arthralgia, skin rash, weight loss, claudication or colour changes on cold exposure. The abdominal CT-scan showed aneurysm of abdominal aorta, stricture of bilateral proximal renal artery, and diverticles of proximal descending aorta. He was then referred to NCCHK for further investigation.

At the adult ward, patient seemed mildly ill, compos mentis, blood pressure was checked at the four extremities. The results for right arm, left arm, right leg, left leg were 171/80, 170/82, SBP 165, SBP 164. Both radial pulses were 75 beats/min, regular, high volume and symmetrical on both sides. Both dorsal pedis pulse were weak, symmetrical on both sides. Heart sounds were found normal and regular, with diastolic murmur grade 2/4 along the left sternal border, no gallop sound. Lung auscultation revealed no rales or wheezing. No bilateral ankle edema was noted. No abdominal bruit, supraclavicular bruit could be heard by auscultation nor abdominal mass could be palpable.

ECG show sinus rhythm, rate 78 bpm, axis was about -60^{o}, P wave was normal, PR interval was 0,12 s, QRS duration was 0,06 s, no ST-T changes. The conclusion was a sinus rhythm electrocardiogram with sign of left ventricular hypertrophy (axis criteria).

Bloods test results (Pasar Rebo Hospital) showed: hemoglobin 15.2 g/dL, hematocrite 47%, leucocyte count 8270/uL, platelet count 270.000/uL, SGOT 10 U/L, SGPT 11 U/L, ureum 19 mg/dL, ceatinine 1.04 mg/dL, total cholesterol 182 mg/dL, LDL cholesterol 117 mg/dL, HDL cholesterol 39 mg/dL, triglyceride 129 mg/dL, uric acid 6.4 mg/dL, ASTO negative. Electrolyte levels were in normal ranges. The urinalysis showed protein +1, with no eritrocyte.

CT scan of upper and lower abdomen (November, 26th 2013) showed aneurysm of abdominal aorta, stricture of proximal bilateral renal artery, and diverticle of proximal descending aorta. Right kidney was smaller in size. Excretion function of both kidney was normal (Figure 1).

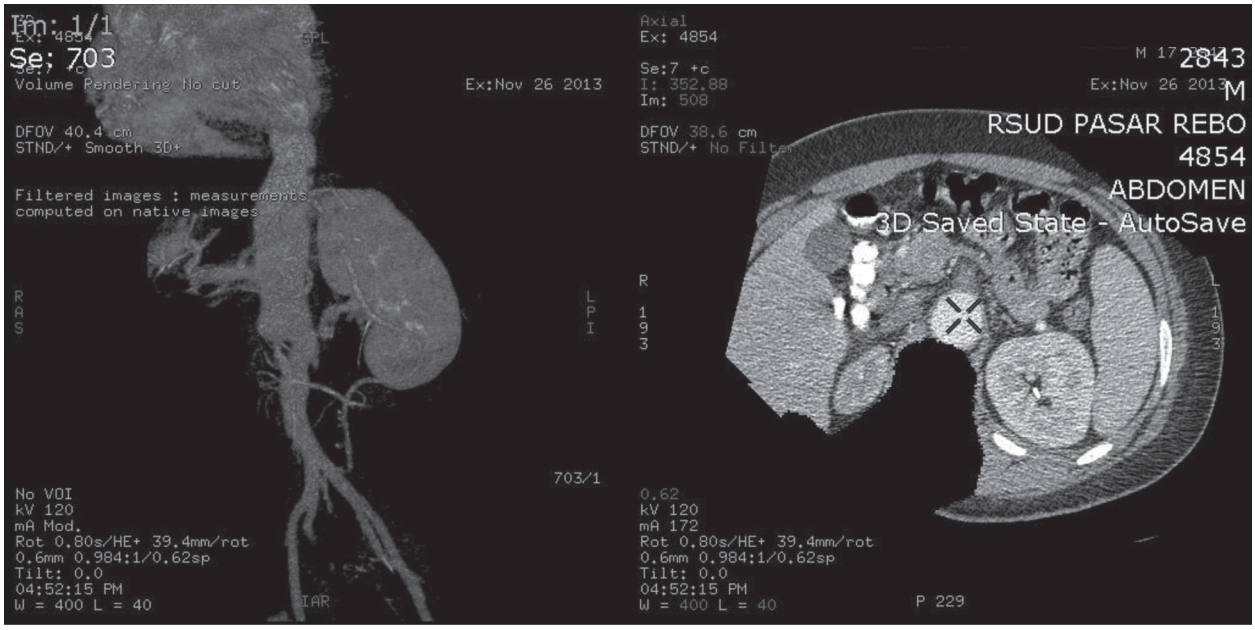

Figure 1. The abdomial CT scan revealed aneurysm of abdominal aorta, stricture of proximal bilateral renal artery. Right kidney was smaller in size

Transthoracic echocardiography was performed on December 19th 2013, showed that LV end diastolic diameter was 68 mm, end systolic diameter was 49 mm, tricuspid annular plane systolic excursion (TAPSE) 2.8 cm. There was aorta aneurysm starting from ascending aorta to abdominal aorta with no signs of aortic dissection. Global systolic LV function was normal with ejection fraction (EF) 40%, global hypokinetics, LV diastolic dysfunction (relaxation disorders), mild-moderate aortic regurgitation (AR) with etiology of suspected NCC prolapse.

Percutaneous Transluminal Angiography (PTA) of renal artery was performed on January 17th 2014 in NCCHK, showing stenosis of left renal artery. Pressure measured: AoD 186/82/119 mmHg; Left renal artery: 98/52/63 mmHg. Peak systolic pressure gradient: 88 mmHg. Mean systolic pressure gradient: 56 mmHg. Right renal artery and right renal contour difficult to visualized (Figure 2). A stent was placed in the left renal artery (Figure 3). After the procedure his blood pressure was decreased to 112/50.

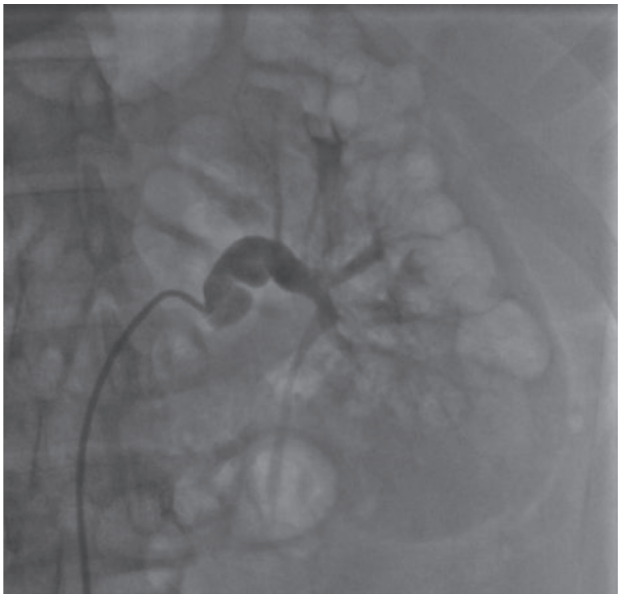

Figure 2. PTA of renal artery revealedostial stenosis of left renal artery with dilatation in the post stenosis area

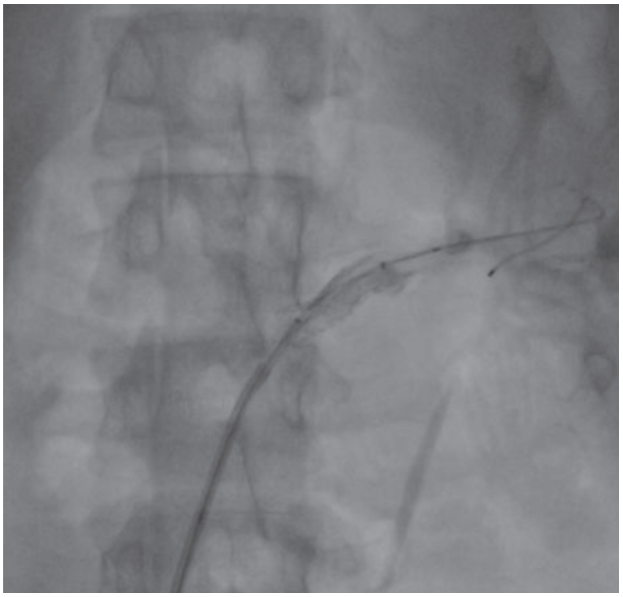

Figure 3. A stent was placed in the left renal artery successfully

DISCUSSION

Diagnosis of secondary hypertension

After confirmed hypertension, screening of the etiology of hypertension must be done according to history, physical examination, and laboratory test. Further investigation into a possible secondary etiology in the absence of suggestive signs and symptoms is indicated in resistant hypertension (defined as elevated blood pressure although the patients have been treated with three antihypertensive agents, including diuretics), onset of hypertension in persons younger than age 20 or older than age 50, a severe or accelerated course of hypertension, worsening of control in previously stable hypertensive patient, stage 3 hypertension, significant hypertensive target organ damage, lack of family history of hypertension, or specific drug intolerances.[1,6]

The most common etiologies in children, in whom 70 to 85 percent of cases of hypertension have a secondary cause, are different from those in older persons, therefore, an age-based approach to the differential diagnosis is recommended.[6]

In this case, patient's blood pressure was measured about 140/80 before. When he came again for medical check-up, his blood pressure was about 200/90, a grade 3 hypertension. At that time he was 17 year old. He was then treated with two antihypertensive agents with no satisfying improvement. These findings bring us to suspect that the patient had secondary hypertension. Further investigation was done to determine the etiology of hypertension.

The most common cause of secondary hypertension at the age of adolescent is renal disease.[6] From his anamnesis and physical examination we still can not

decide the etiology. His laboratory examination towards renal function shows normal creatinine level but protein +1 from urinalysis. This finding leads us to do more investigation about his renal problem.

Some imaging modalities could be done to visualize renal and renal artery. Both spiral computed tomography (CT) and magnetic resonance imaging (MRI) are being increasingly used to visualize the renal arteries. General agreement exists across the literature that renal angiography is the "gold-standard" for the diagnosis of renal artery stenosis. Similarly, nearly all authorities agree that individuals who have a very high absolute risk of renal artery stenosis or renovascular hypertension should proceed directly to renal angiography.[4,7]

In this case, imaging modalities help us to make a more definite etiology of the secondary hypertension. The noninvasive modality was chosen first by the internist in Pasar Rebo Hospital. His CT-scan showed stricture of proximal bilateral renal artery. Renal angiography was performed later in NCCHK as the gold standard diagnostic tool to confirm the diagnosis.

Renovascular hypertension and renal artery stenosis

Renovascular hypertension refers to hypertension induced by renal ischemia or by renal artery stenosis.[7,8] The diagnosis of renovascular hypertension can be made only retrospectively, unlike diagnosis of most other cardiovascular and nephrologic conditions. Blood pressure in renovascular hypertension will response to an intervention. Classically, we can diagnose renovascular hypertension correctly and properly 6 to 12 weeks afteran intervention, only if the BP is lower than it was before the intervention, and the patient taking the same or fewer antihypertensive medications.[7]

In our case, we can conclude that patient's hypertension was a renovascular hypertension because the blood pressure was lower than it was before the intervention (from 154/65 to 112/50 mmHg). Our suspicion toward a renovascular hypertension came because of the onset of hypertension in young age. No other comorbidities such as diabetes, dyslipidemia, nor tobacco use, were found in our patient that could lead to hypertension and then renal artery stenosis.

Renal artery stenosis is a diagnosis based on anatomic criteria. Classically, renal artery stenosis was diagnosed when the patient had a greater than 75% narrowing of the diameter of a main renal artery or a more than 50% luminal narrowing with a poststenotic dilatation.[7]

Pathophysiology of hypertension in renal artery stenosis begins with an increase in plasma renin activity. This increase results in an immediate increase in blood pressure. Subsequent elevation in plasma aldosterone results in an increase in salt and water retention, thereby potentiating the impact on hypertension. If unilateral RAS occurs, the contralateral normal kidney may compensate for the salt and water effects; however, this compensatory mechanism will not occur in the case of bilateral RAS.[5]

There are some subtypes of renovascular disease with renal artery stenosis: fibromuscular dysplasia (FMD), atherosclerotic disease, and other (less common) causes of renovascular disease. FMD is a vascular disease of unknown cause that can occur in multiple vascular beds. In the kidney, it usually occurs in the mid or distal portion of the renal artery, and it can also be found in smaller accessory renal arteries. FMD is more common in younger patients and in women. Fibromuscular dysplasia also affects other arteries, including the carotid and vertebral arteries, and less commonly, the iliac and mesenteric arteries. Atherosclerotic disease is usually seen in older patients with traditional risk factors for atherosclerosis.[8]

Many additional causes (either extrinsic or intrinsic to the vessel) of renovascular hypertension have been described. On a population basis, Takayasu's arteritis may be the most important, especially in India or Japan.[7] In 1990, the American College of Rheumatology (ACR) defined specific diagnostic criteria for Takayasu's arteritis: age at disease onset $\leq$ 40 years, claudication of extremities, decreased brachial artery pulse, blood pressure difference > 10 mmHg in systolic blood pressure between arms, bruit over subclavian arteries or aorta, arteriogram abnormality. A diagnosis of Takayasu arteritis requires that at least 3 of the 6 criteria are met.[9]

In this case, the patient is a young adolescent, with no traditional risk factors noticed. From this information, we can exclude atherosclerotic disease

as the etiology of renal artery stenosis. According to the ACR criteria for Takayasu's arteritis the patient only meets 2 of the 6 criteria which is: age at disease onset ≤ 40 years, and the arteriogram arteritis. Another possible cause of renal artery stenosis in this patient is fibromuscular dysplasia (FMD). FMD is more common in younger patient (15-30 years of age).

Management of hypertension with renal artery stenosis

The goals of management of secondary hypertension due to renal artery stenosis in this patient are: control of blood pressure, preservation of renal function, and avoidance of complications and adverse effects of treatment. The managements could include: medical therapy, percutaneous intervention, and surgical intervention.

Combination of more than one antihypertensive agent is usually suggested to control blood pressure. A combination of ACE-inhibitor or angiotensin receptor blocker with diuretic and calcium channel blocker is common to use. The major concern about intensified antihypertensive drug therapy is the risk of acute deterioration in renal function that sometimes occurs when an angiotensin-converting enzyme inhibitor or an angiotensin receptor blocker is added to the regimen. These drugs are effective in reducing BP in 86% to 92% of patients with renovascular hypertension, most commonly when used in combination with other agents. Acute renal failure with ACE inhibitors was rare and generally reversible with discontinuation of the agent.[8] The increase in serum creatinine concentration typically reverts to baseline after stopping the angiotensin converting enzyme inhibitor or angiotensin receptor blocker; this can be an indication for renal revascularization.[7]

Percutaneous revascularization need to be performed to this patient. The American Heart Association (AHA) recommends percutaneous revascularization for patients with resistant hypertension, malignant hypertension, hypertension with an unexplained unilateral small kidney, and hypertension with intolerance to medication.[10]

Acknowledgments

We thank to dr. Rarsari Soerarso, Sp.JP as the members of Division of Clinical Cardiology and Vascular, Faculty of Medicine Universitas Indonesia.

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