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Case Report

Role of drug-eluting stent on Takayasu arteritis with renal artery stenosis

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Takayasu arteritis (TA) is defined as granulomatous inflammation of large arteries involving the aorta and its primary branches. Takayasu arteritis with renal artery stenosis (TARAS) is a common cause of pediatric renovascular hypertension. The main purposes of TARAS management are to improve high blood pressure and recover renal function. When general medication fails to improve symptoms, renal revascularization may be attempted. Implantation of a drug-eluting stent (DES) has been used as an alternative strategy for pediatric renal revascularization. Here, we report on a 10-year-old, female, Javanese patient with bilateral TARAS who underwent DES implantation. Her clinical presentation was hypertensive crisis and worsened renal function. Bilateral renal artery DES implantation was performed successfully without complications. The child's blood pressure was controlled using two anti-hypertensive medications after DES implantation and her renal function recovered. Dual anti-platelet therapy was given to minimize the risk of stent thrombosis. **[Paediatr Indones. 2022;62:422-9; DOI: https://doi.org/10.14238/pi62.6.2022.422-9**].

Keywords: *Takayasu arteritis with renal artery*

The case

A Javanese girl aged 10 years presented with vomiting and oliguria. The patient had previously been hospitalized for 45 days with a clinical presentation of seizures, headache, vomiting, blurred vision, and a hypertensive emergency, with blood pressure reaching 190/110 mmHg. Previous treatment history consisted of intensive care with intravenous nicardipine at 0.25 μg/kg/min followed by intravenous furosemide at 1x25 mg and oral captopril at 3x6.25 mg. Sublingual nifedipine at 5 mg was given due to a recurrent hypertensive crisis, but then deterioration of her renal function and oliguria occurred. The patient was subsequently referred for hemodialysis.

Upon examination, the patient was awake, alert, and fully oriented. She had a weak general condition. Her right upper limb blood pressure (BP) was 120/80 mmHg, left upper limb BP 130/80 mmHg, lower extremity BP (both right and left) 120/80 mmHg, pulse 104 times/minute, respiratory rate 24 times/minute, axillary temperature 36.5°C, and 95% SpO_2 measured from the four extremities. Head and neck examination revealed no evidence of anemia, jaundice, cyanosis, or dyspnea. Cardiac examination revealed single S1 and S2 heart sounds, without murmurs or gallops. Pulmonary examination showed no signs of pulmonary edema. Examination of the extremities revealed warm extremities without leg edema or difference in pulses.

The laboratory examination results on the first day of the prior hospitalization were as follows: Hb 11.5 g/dL, white blood cell count (WBC) 20,500/μL,

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platelets 376,000/μL, erythrocyte sedimentation rate (ESR) 62 mm/hour, blood urea nitrogen (BUN) 14.2 mg/dL, serum creatinine 0.68 mg/dL, Na 140 mmol/L, K 3.3 mmol/L, and Cl 110 mmol/L. Laboratory examinations on the 19th day of post hypertensive crisis showed Hb 12.1 g/dL, WBC 10,400/μL, plateles 479,000/μL, ESR 98, BUN 47.6 mg/dL, and creatinine 2.11 mg/dL. Laboratory examinations at the time of referral (day 46) included Hb 10.8 g/dL, WBC 7,050/μL, PLT 331,000/μL, ESR 82 mm/hour, C-reactive protein (CRP) 31 mg/dL, BUN 83 mg/dL, serum creatinine 9.64 mg/dL, glomerular filtration rate (GFR) 7.9 mL/min/1.73m^2, Na 134 mmol/L, K 4.0 mmol/L, and Cl 92 mmol/L. Blood gas examination revealed the following: pH 7.444, PO_2 100 mmHg, PCO_2 23.4 mmHg, SO2 98.2%, HCO_3 16.2 mmol/L, $AaDO_2$ 19.6 mmHg, and base excess (BE) -8.1 mmol/L. Eye funduscopy examination revealed grade I hypertensive retinopathy in both the right and left eyes. Chest X-ray revealed no heart abnormalities.

The patient underwent hemodialysis. Post-hemodialysis, the child's BP fluctuated from 140/90 to 190/110 mmHg, and her laboratory results were Hb 10.4 g/dL, WBC 6,750/μL, platelets 232,000/μL, CRP 22, BUN 41 mg/dL, and serum creatinine 1.5 mg/dL. She received intravenous injection therapy of furosemide 1x25 mg, amlodipine 1x1 mg, and captopril 3x6.25 mg. Nifedipine 5 mg was given sublingually when her BP fluctuations exceeded 140/90 mmHg.

After hemodialysis, the patient undewent renal ultrasonography (USG), echocardiography, and angiography. Renal USG revealed no abnormality in either the right or left kidney. The echocardiography results were as follows: solitus situs; atrioventricular-ventriculoarterial concordance (AV-VA: concordance); normal pulmonary venous drainage; cardiac chamber dimensions include left ventricular dilatation; valves: trivial mitral regurgitation, mild tricuspid regurgitation, moderate pulmonary regurgitation; intact atrial and ventricular septae; aortic arch on the left; renal artery stenosis and multiple abdominal aortic stenoses.

Angiography revealed the following results (**Figure 1**): short segment and long segment abdominal aortic stenosis before renal artery branching and long segment after renal artery branching; a significant 90% stenosis in the ostial-proximal to both the left and right renal arteries.

We performed percutaneous transluminal renal angioplasty (PTRA) on the patient. Hemodynamic examination before PTRA showed a BP of 180/110 mmHg, pulse 84 beats/minute, and respiratory rate 18 beats/minute. Laboratory results before PTRA were Hb 8 g/dL, WBC 10,420/μL, PLT 328,000/μL, CRP 1.9, BUN 20 mg/dL, and serum creatinine 1.2 mg/dL. The patient had increased plasma prothrombin time (PPT) physiostasis of 15.5 seconds (control 9-12 seconds) and activated partial thromboplastin time (aPTT) of 64.5 seconds (control 21-23 seconds) without signs of bleeding. Percutaneous transluminal renal angioplasty was carried out with a transfemoral approach using a 6F sheath, guiding catheter right judgment (JR) 4.0 6F, and *Runthrough NS Hypercoat* guiding wire. Heparin at 2000 units was given intravenously before the guiding wire was inserted. The PTRA procedure was followed by angioplasty of the left and right renal arteries using a 3.0x15 mm sapphire balloon. However, there was recoil after balloon angioplasty, so stents were placed in the left renal artery [*DES Biomime* (Sirolimus) 4.0x24 mm] and the right renal artery [*DES Ultimaste*r (Sirolimus) 4.0x15 mm]. Post-DES dilation was performed on both the left and right renal arteries. There was no residual stenosis after stent placement (**Figure 2,3**).

Post-PTRA stenting, the child had BP 120/70 mmHg, HR 84 times/minute, and RR 20 times/minute. Blood test results were BUN 21 mg/dL, serum creatinine 0.8 mg/dL, GFR 96.25 mL/minute/1.73m^2, PPT 10.3 seconds (control 9-12 seconds), and APTT 30.10 seconds (control 23-33 seconds). Post-PTRA with DES, the patient was given 1x100 mg aspirin therapy and 1x40 mg clopidogrel to prevent stent thrombosis. Amlodipine 1x2.5 mg and furosemide 1x25 mg were given as anti-hypertensives. Three days after PTRA-stenting, the patient had no signs of complications at her clinic visit.

Discussion

Hypertensive crisis, either emergent or urgent, is severe hypertension (HT), which can be fatal. A hypertensive emergency is severe, with complications from target organ dysfunction, especially nervous, renal, or cardiac. Hypertensive urgency refers to a state of severe hypertension without damage to target

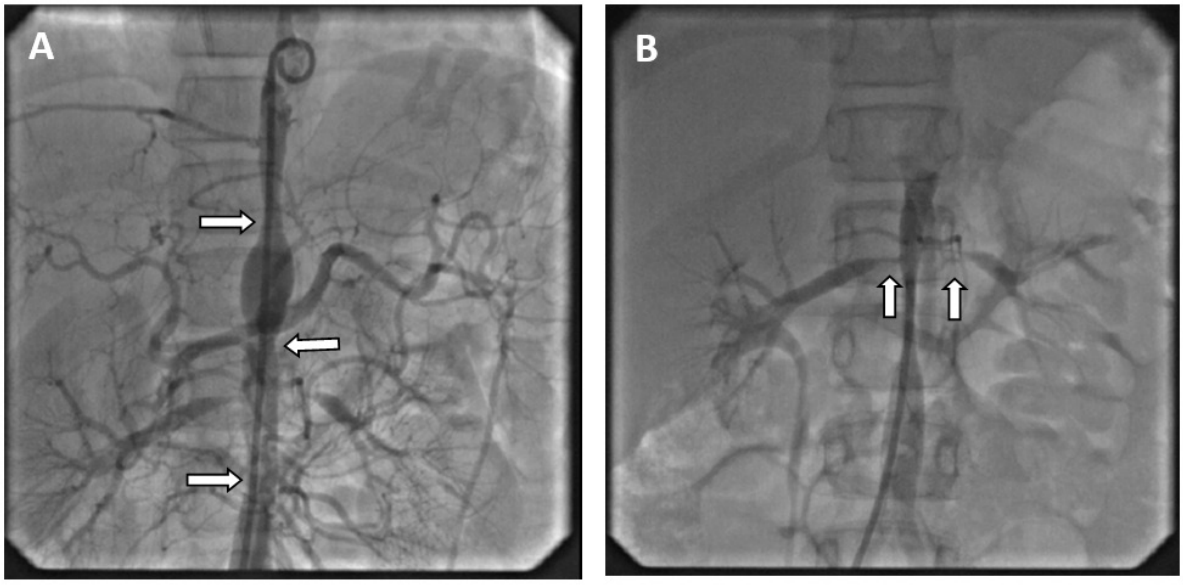

Figure 1. (A) The aortography shows segmental stenosis in the abdominal aorta (arrow) (B) Significant 90% stenosis in the ostial-proximal right and left renal arteries

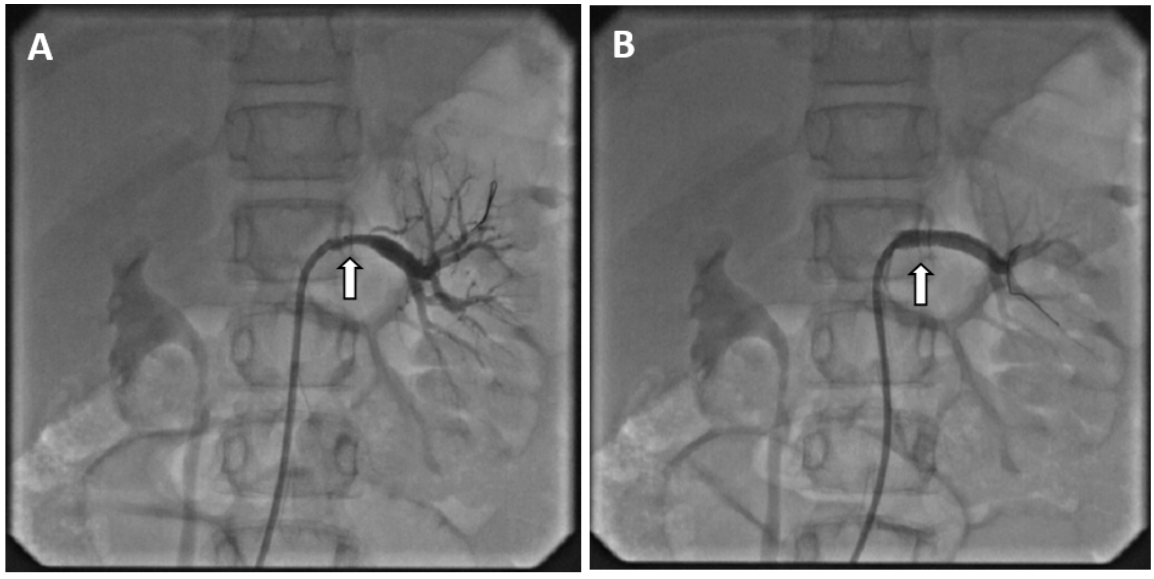

Figure 2. (A) Recoil of left renal artery after PTRA with balloon (arrow); (B) Good left renal artery patency after insertion of DES (arrow), without residual stenosis

organs, such that therapy can be given through oral agents. The target BP reduction based on clinical experience is no more than 25-30% in the first 6-8 hours, followed by a gradual decrease in BP over the next 24-48 hours.[1]

Resistant HT is a condition in which the target of lowering systolic and diastolic BP cannot be achieved, despite treatment for lifestyle changes and at least three kinds of anti-hypertensive agents, including diuretics. Hypertension that is generally persistent can be classified as secondary HT, if a specific etiology is obtained. The etiology of secondary HT generally varies based on the age of the patient. Hypertension in children <6 years is most often caused by renal

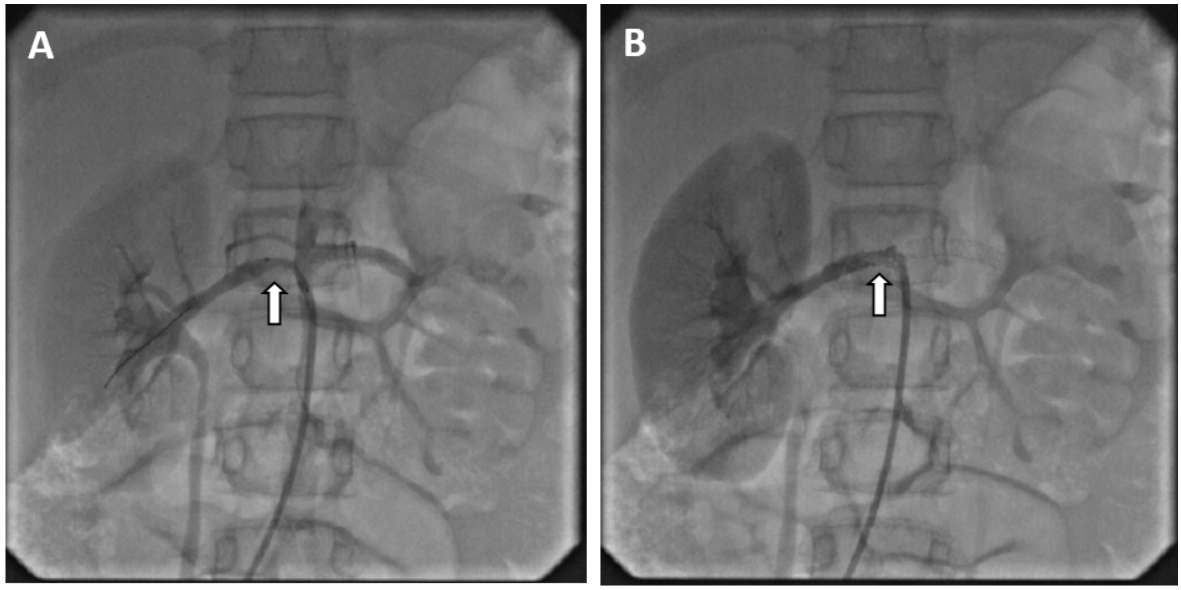

Figure 3. (A) Recoil of the right renal artery after PTRA with a balloon (arrow); (B) Good right renal artery patency after insertion of DES (arrow), without residual stenosis

parenchymal diseases, such as glomerulonephritis, polycystic renal disease, renal artery stenosis, and renal dysplasia.[1]

Children with uncontrolled HT who require more than two anti-hypertensive drugs are advised to undergo further renovascular HT screening procedures.[2] The examination consists of monitoring plasma electrolytes, serum creatinine, and glomerular filtration rate, as well as renal and renovascular imaging. Agents affecting the renin-angiotensin axis are generally contraindicated in pediatric patients suspected of renovascular HT. Angiotensin-converting-enzyme inhibitors (ACEI) and angiotensin-receptor blockers (ARBs), result in decreased glomerular filtration rate by dilating the glomerular efferent arterioles. Acute renal failure due to renal artery stenosis occurs in many cases, which is generally reversible once the etiology is resolved.[2]

Our patient had hypertensive emergency, characterized by severe HT (190/110 mmHg) accompanied by target organ dysfunction (nervous, renal, and eye) such that the patient was admitted to the ICU and treated with intravenous continuous pump nicardipine therapy. After the hypertensive emergency resolved, therapy was continued with 25 mg intravenous furosemide and oral captopril. In the course of progressive deterioration of renal function, bilateral renal ischemia occurred and it was exacerbated by ACEI therapy. Hemodialysis was performed to treat acute kidney failure. Echocardiography was suspicious of renal artery stenosis, which was confirmed by angiography. Angiography revealed significant stenosis of both left and right renal arteries. From the ultrasound, there was no suggestion of right-left renal abnormalities, so renal ischemia due to renal artery stenosis may have just occurred, with a possibility of reversible renal function, if the renal artery stenosis were to resolve.

The two main causes of renal artery stenosis in children are fibromuscular dysplasia (FMD) and Takayasu arteritis (TA).[3] Takayasu arteritis is a chronic inflammatory condition, with a predilection for the aorta and its branches. The pathogenesis of Takayasu's vasculitis is not known with certainty, but some evidence suggests autoimmune etiology and/or genetic predisposition. An association was noted between tuberculosis and Takayasu in Japan. Analysis of the vascular pathology of Takayasu's patients showed granulomatous inflammation that resulted in fibrosis and vascular damage. This results in changes in the blood vessel walls in the forms of stenosis and dilation that intersect with the normal blood vessel area.[4]

The diagnostic criteria for TA were initially established based on the recommendations of the *American College of Rheumatology* (ACR) in 1990,[5] which were later updated to the diagnostic criteria

for pediatric TA by the *European League Against Rheumatism* (EULAR)/*Paediatric Rheumatology International Trials Organization* (PRINTO)/*European Pediatric Rheumatology Society* (PRES) year 2008.[5] Angiographic criteria for diagnosis should be identified in any case of pediatric TA. The EULAR/PRINTO/PRES consensus emphasizes the differential diagnosis of TA with non-inflammatory etiologies such as fibromuscular dysplasia (FMD) or mid-aortic syndrome, where future imaging technologies are expected to add value to the differential diagnosis. The EULAR/PRINTO/PRES consensus then added the criteria for acute phase reactants associated with angiographic abnormalities as one of the important inflammatory criteria identified before complications arise, such as HT or claudication.[5]

The results of TA angiography were classified based on the *1994 International TA Conference* in Tokyo. There are 5 types of TA: type I includes branching of the aortic arch; type IIa includes the ascending aorta, aortic arch, and its branches; type IIb is a combination of type IIa and the descending aorta; type III includes the descending aorta, abdominal aorta, and/or renal artery; type IV includes the abdominal aorta and/or renal artery; and type V is a combination of type IIa and type IV.[6]

The most common clinical presentations of pediatric TA are HT (83%), headache (31%), fever (29%), dyspnea (23%), weight loss (22%), and vomiting (20%). Organ-specific manifestations of TA are due to decreased blood supply as a result of vascular stenosis and ischemia in areas supplied by TA-associated blood vessels. Claudication (13%) and pulse deficit (13%) occur in the area of the blood vessels involved. However, bruits are rarely found. Secondary heart disease was noted in 19% of patients with pediatric TA. Neurological manifestations include headache (31%) and stroke (17%). Skin manifestations of nodules and rash are rare. Likewise, ocular manifestations such as retinopathy are rarely seen in cases of pediatric TA.[7]

The diagnosis of TA in our patient was made based on the presence of type IV angiographic criteria (segmental stenosis of the abdominal aorta and renal arteries), hypertension, and increased ESR. The elevated ESR and CRP at the initial patient presentation, although not a specific marker of vascular inflammation, contributed to the child's inflammatory etiology. Her clinical presentation of hypertension, headache, and retinopathy supported the picture of a hypertensive crisis due to bilateral renal ischemia.

Renal revascularization can be performed through endovascular therapy or surgery. Takayasu arteritis patients should be not in acute phase when revascularization is performed.[8] The ESR and/or CRP values are expected to return to normal before revascularization.[3] Surgical or endovascular interventions when the patient is in an active inflammatory phase can trigger neointimal proliferation and result in restenosis. Renal revascularization in our patient was indicated by the presence of severe HT that was resistant to medical therapy and progressive deterioration of renal function within 45 days of treatment accompanied by oliguria, which then required dialysis which was possibly due to ACEI therapy. There are limited studies available on renal artery revascularization in children. Surgical revascularization has a high degree of difficulty and variable success rates, but is associated with good 5-year patency outcomes and good blood pressure control in adult patients. In pediatric patients, surgery is reported to have a good outcome characterized by a decrease in blood pressure in 96% of patients. Nonetheless, several studies reported varying failure rates of 4-57% in pediatric patients.[9] In that case, major complications such as graft thrombosis, the occurrence of aneurysms and anastomotic stenosis result in persistent hypertension, renal failure and early post-operative mortality.[9] The main difficulty in revascularization of the renal arteries through pediatric surgery is the selection of graft conduits. There is no ideal autogenous conduit that makes it possible to follow a child's growth. The use of saphenous veins as a graft in children is also not an ideal choice because there is a risk of aneurysmal degeneration of more than 20% each year. As such, endovascular therapy is generally carried out while waiting until definitive revascularization with a prosthetic graft can be performed as an adult.[2]

There is no definitive consensus on the use of endovascular therapy (PTRA), with or without stents in cases of pediatric vasculitis. The PTRA without stents is more common than with stents. The lesions in vasculitis cases are narrower and harder to dilate. It is generally difficult to penetrate the lesion

using conventional balloon angioplasty, so a high-pressure balloon or cutting of the balloon is required. The PTRA with balloon was reported to have a good blood pressure-lowering outcome in 72.7% of pediatric patients. In addition, this method has a lower restenosis rate than PTRA with stents in TA patients, which may be related to chronic stress and stretching resulting in inflammation, cellular proliferation, and renal artery restenosis when using stents.[10] In addition, children have not finished growing so their renal artery size may increase with age, hence, there is a risk of restenosis. Therefore, the indication for stent use in cases of renal artery stenosis is limited to conditions in which recoil occurs immediately after PTRA, impaired flow due to arterial dissection due to PTRA, and recurrent arterial stenosis after clinical and technical PTRA success.[11]

To date, reports on the use of stents in pediatric renal artery stenosis (RAS) cases generally use bare metal stents (BMS). However, a study reported a high rate of in-stent restenosis (37%) with BMS in cases of RAS. [11] Neointimal proliferation is the etiology of in-stent restenosis (ISR). The BMS strut stimulates a reaction against "foreign bodies," accompanied by the proliferation and migration of smooth muscle cells to the intima layer. In contrast to BMS, DES contains anti-proliferative and anti-inflammatory agents to inhibit ISR.[12] As such, the use of DES in pediatric RAS cases is being considered.

There have been few reports on the use of DES in renal artery stenosis, especially in adult and pediatric TA patients. A study reported that the ISR rate of BMS use in pediatric RAS cases was not much different from that in adult coronary artery stenosis patients. They noted that sizes of coronary arteries in adults and renal arteries in children are similar. In addition, another study reported that DES use in adult coronary artery stenosis patients was superior to BMS, in terms of preventing ISR.[11] Likewise, a case report showed good patency 6 months after DES insertion in a pediatric RAS case.[13] A previous study also reported good DES patency at 9 months post-insertion in a TARAS case of a 17-year-old girl.[14]

The endovascular revascularization strategy was decided considering the patient's age, as it was not optimal for the surgical alternative in terms of graft selection, high morbidity, and variable failure rates. The PTRA attempt was initially carried out with a balloon which led to post-dilatation recoil of the renal artery. Hence, stenting was performed to overcome the recoil. The DES was chosen in an effort to reduce the possibility of ISR that often occurs when using BMS. Post-stent dilation was performed to ensure optimal expansion of the stent. Good patency was obtained without residual stenosis after stent placement. There were no complications during and after the endovascular procedures.

Assessment of clinical success of renal artery endovascular intervention is based on the presence of clinical complications, blood pressure control, restoration of renal function, renal artery patency, and restenosis.[15] Better clinical outcomes of endovascular therapy are generally obtained when TA patients are in the inactive phase. Early complications such as infection, bleeding, thrombosis, aneurysm, dissection, vascular rupture, and stent dislocation are complications of early intervention that can occur in the stent insertion procedure.[3,15] While the major long-term complication is in-stent restenosis, little study has been done on it.[16]

To date, the use of DES in pediatric TARAS is rare, and there are no large studies on its long-term outcomes. A case report on the use of DES in children aged 8 years reported good blood pressure control within 8 years of post-DES evaluation using 2 low-dose anti-hypertensives.[13] In addition, two case reports of DES use in pediatric RAS mentioned good patency at 6 and 9 months post-stent insertion.[14,15] However, arterial endothelialization that is hampered by DES use results in a prothrombogenic condition that can trigger stent thrombosis. So dual antiplatelet therapy is required in adult patients.[12] The use of dual antiplatelet agents in children has not been well-studied, to date. Pre-operative heparin and aspirin after stent placement are therapies commonly used in children to prevent stent thrombosis. However, the stent type used in such cases is generally BMS.[15] Given the high risk of stent thrombosis in DES use, dual antiplatelet (DAPT) agents should be considered. Although there is no consensus on the duration of DAPT administration after DES placement in children, a study reported good DES patency with 6 months of post-stenting DAPT administration.[13]

Our 10-year-old patient had a good outcome after DES placement. Her blood pressure was controlled with two anti-hypertensives (amlodipine

1x 2.5 mg and furosemide 1x25 mg) and her renal function improved (BUN 21 mg/dL, creatinine 0.8 mg/dL, and GFR 96.25 mL/min/1.73 m2) after stenting. There were no complications during and after the endovascular procedures. Intravenous heparin therapy was given prior to stenting. Dual antiplatelet agents (aspirin 1x100 mg and clopidogrel 1x40 mg) were given after stent insertion to prevent stent thrombosis. There were no bleeding complications during the 3 days post-stent observation period.

Takayasu arteritis is a chronic inflammatory condition with a predilection for the aorta and its branches, including the renal artery. In summary, we present here a TA case of a 10-year-old girl with renal artery stenosis. The patient had a clinical presentation of a hypertensive crisis and progressive deterioration of renal function requiring hemodialysis. Angiographic examination revealed segmental stenosis of the abdominal aorta and both renal arteries. Percutaneous renal transluminal angioplasty with DES was performed to control blood pressure and improve renal function. After DES insertion, the child's renal function improved and her blood pressure control was good with two anti-hypertensives. Dual antiplatelet agents were given to prevent stent thrombosis. Long-term periodic monitoring is needed to assess outcomes and prevent complications related to the risk of ISR, stent thrombosis, and bleeding.

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