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Case Report

Successful endovascular reconstruction of symptomatic vertebrobasilar dolichoectasia using multiple Leo stents and nondominant vertebral artery occlusion: a case report

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ABSTRACT

Vertebrobasilar dolichoectasia (VBD) is a rare vascular anomaly of increased diameter, length, and tortuosity of vertebral and/or basilar artery, but debilitating due to its risk of ischemia, hemorrhage, and nerve or brain compression. The management is also controversial due to various possible clinical manifestation and outcome. This study aimed to describe a combined approach of multiple scaffolding Leo stents and nondominant vertebral artery occlusion as a definitive approach to reconstruct vertebrobasilar arteries. A 40-year old male presented with severe headache and reduced consciousness, which was explained with brain CT findings of subarachnoid hemorrhage and hydrocephalus. Further etiologic approach until digital subtraction angiography revealed VBD. An endovascular reconstruction approach was considered one month following the event onset using multiple scaffolding Leo stents from left vertebral to basilar artery with right vertebral artery occlusion. This stent had the best radial strength, lowest bending stiffness, highest kink resistance, highest bending wall coverage, and lowest cell size, which provided strong vascular reconstruction properties. Combined nondominant vertebral artery occlusion was also performed to avoid the disturbance of flow-diverting pathway by the stents. Double antiplatelet was administered from three weeks following the event onset afterwards. The patient's condition improved at three-month follow-up. This case report presented that combined multiple Leo stents and nondominant vertebral artery occlusion may be considered as an approach to successful endovascular reconstruction for symptomatic VBD.

Keywords: VBD, Endovascular reconstruction, Vertebral artery occlusion, Scaffolding stent

INTRODUCTION

Vertebrobasilar dolichoectasia (VBD) is vascular anomaly of dilation, elongation, increased length, diameter, and/or tortuosity of basilar and vertebral artery.[1] It comprised up to 80% of intracranial arterial dolichoectasia.[2,3] It is discovered incidentally in 1.3-4.4% of general population and symptomatically in 7.6-18.8% of stroke population. Another report described that the incidence was 0.06-5.8%, but it contributes up to 12% cases of stroke.[2,3]

While it may be asymptomatic, VBD may manifest in 46.8% subjects as vascular or compressive symptoms, including ischemic stroke, and subarachnoid hemorrhage, cranial nerve compression, brainstem compression, hydrocephalus, cerebellar dysfunction, and central sleep apnea.[1,4] Wolters et al studied 375 VBD subjects and reported that the 5-year new event included ischemic stroke (17.6%), brainstem compression (10.3%), transient ischemic attack (10.1%), spontaneous intracerebral hemorrhage (4.7%), hydrocephalus (3.3%), and

subarachnoid hemorrhage (2.6%). Another study by Passero and Rossi (2008) reported that of 156 subjects with VBD and median follow-up of 11.7 years, 43% had progression of VBD and approximately 60% experienced at least one symptomatic event. Of all VBD subjects, the five-year mortality was reported to be 36.2%.[1]

The detrimental complication of VBD was still not supported by the advances its recommendation of management. Due to its critical anatomical location to vital structure asymptomatic VBD is suggested to be managed conservatively including observation and cardiovascular risk factor management. However, approach to symptomatic VBD, whether open cerebrovascular surgery, microsurgical decompression or endovascular reconstruction, were still controversial and confined to highly selective moderate or severe VBD, including those with low flow or more oscillatory flow or those with greater risk of bleeding or large aneurysm.[1] Open surgery are performed by occluding the parent artery with or without the construction of bypass, microsurgical decompression reduces the contact between VBD and associated cranial nerve symptoms, and endovascular technique aimed at vascular reconstruction.[5,6] Some techniques for endovascular reconstruction included multiple stent reconstruction, distal vertebral artery occlusion, aneurysm coiling, and thrombectomy (for VBD with acute ischemic stroke).[1-5] This study aimed to report symptomatic VBD of subarachnoid hemorrhage, brainstem compression, and hydrocephalus who was successfully treated with combined endovascular treatment of multiple scaffolding Leo stent and nondominant vertebral artery coiling.

CASE REPORT

A 40-year old male presented to the emergency ward with severe headache one day prior to admission followed with sudden loss of consciousness a day after. Physical examination showed increased blood pressure of 160/80 mmHg, Glasgow coma scale of 10, nuchal rigidity, and no lateralization.

The first brain CT scan at admission revealed a markedly ecstatic left vertebral artery, the second brain CT scan while presented with sudden loss of consciousness demonstrated diffuse subarachnoid hemorrhage (SAH), and the third brain CT scan at 10-day admission showed obstructive hydrocephalus as well as diffuse and trans-ependymal brain edema.

Digital subtraction angiography (DSA) revealed left vertebral dolichoectasia with ectasia of basilar and right vertebral artery.

He was diagnosed with VBD, treated with symptomatic subarachnoid management as per guideline, and admitted to the intensive care unit (ICU) for two weeks. Ventriculoperitoneal shunt was also performed due to hydrocephalus.

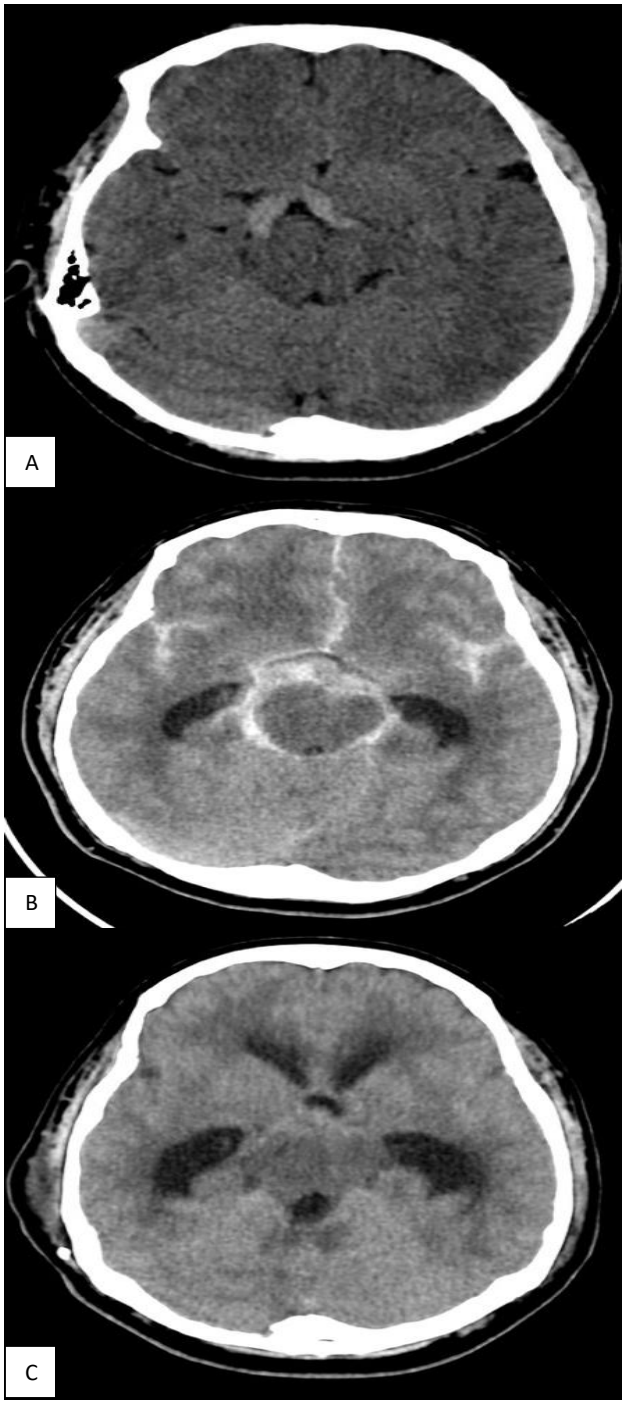

Figure 1 (A-C): Brain CT scan at admission showed bilateral vertebral artery ectasia; brain CT scan at the second day of admission showed spontaneous diffuse subarachnoid hemorrhage and brain CT scan at two weeks of admission revealed hydrocephalus.

Following a month after spontaneous SAH, an attempt for endovascular reconstruction was performed using two LEO stent. He received double antiplatelets of aspirin 80 mg and clopidogrel 75 mg pre-procedure, which was loaded to aspirin 320 mg and clopidogrel 300 mg at the day of procedure. Coiling was performed at the distal right vertebral artery. Multiple stents were deployed and scaffolded to the left vertebral to basilar artery to ensure

the coverage of the proximal and distal normal segment of artery.

The patient was observed post procedure for three days and discharged without remarkable symptoms. Brain CT angiography at three-month follow-up described no VBD. Double antiplatelet was continued for one year.

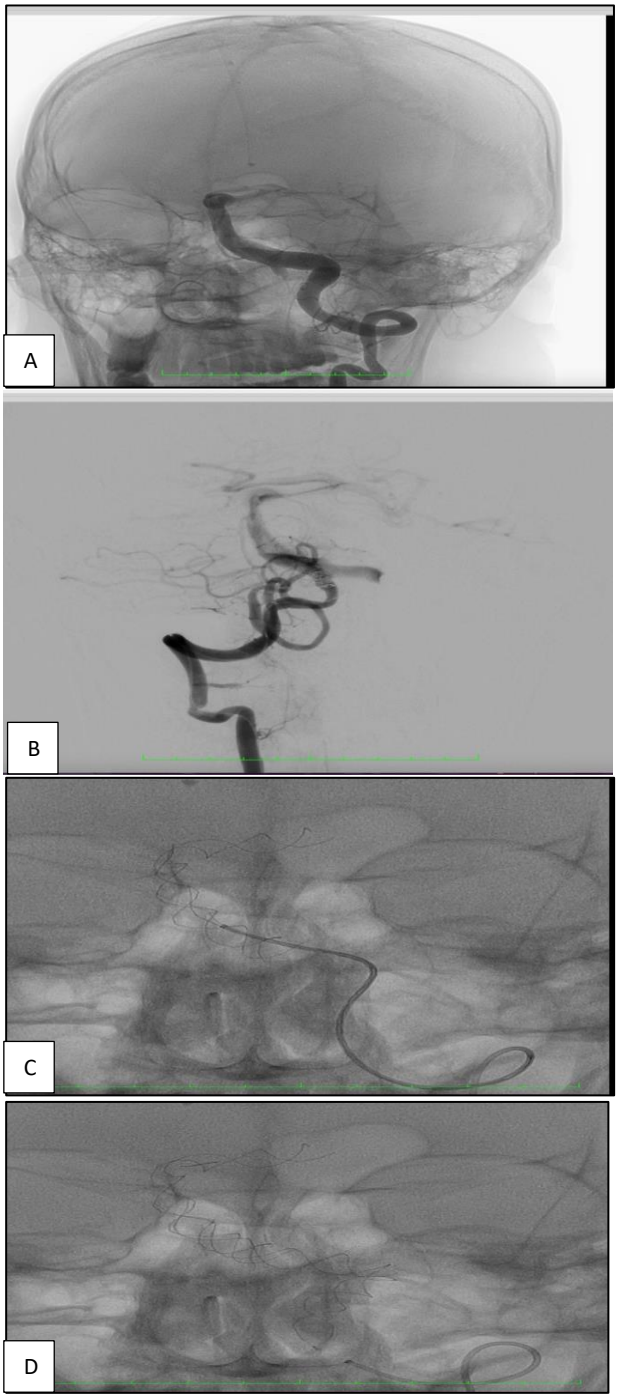

Figure 2 (A-D): Digital subtraction angiography on anterior-posterior left vertebral artery view showed left vertebral and basilar artery ectasia; distal right vertebral artery was coiled; 2 LEO stent-assisted coil was deployed and scaffolded from the distal left vertebral to basilar arteries.

DISCUSSION

This study presented middle-aged adult with symptomatic severe VBD who was successfully managed with combined endovascular approaches. In addition, this was one of first publicized VBD reconstruction in Indonesia using multiple scaffolding Leo stents and nondominant vertebral artery coiling. This approach may be a treatment choice to be considered in symptomatic VBD.

This patient had severe headache followed with reduced consciousness, which was consistent with spontaneous subarachnoid hemorrhage and its complications of hydrocephalus from CT scan findings. In addition, non-contrast brain CT scan prior to spontaneous subarachnoid hemorrhage demonstrate mild hyperdense vertebrobasilar artery elongation at midbrain suspected due to VBD, which was confirmed with DSA. Until recently, there was still no consistent consensus for definition of VBD, but it generally included ectasia, described as increased diameter of vessels, and lengthening, described as lateral and/or upward displacement, of vertebrobasilar arteries. It can be described with one of the following: (1) increased diameter of basilar artery of >4.5 mm at the level of mid pons; (2) lateral and/or upward displacement of vertebrobasilar artery beyond the lateral margin of clivus; (3) upward displacement of tip of basilar artery reaching the level of the floor of 3rd ventricle. Digital subtraction angiography was still regarded as gold standard for detecting anatomical structure of VBD whereas computed tomography/magnetic resonance MR angiography may provide information regarding intraluminal thrombus/ atherosclerosis and brain parenchyma changes.

The pathophysiology of VBD was still unclear, but it was thought due to abnormal connective tissue or abnormal vascular remodeling of arterial wall which contributed to imbalance between clotting and hemorrhagic system inside the arterial wall.[1,6] It may result in internal elastic lamina thinning due to hypertension, arterial wall degeneration due to senescence, or excessive atheromatous degeneration.[6] Cerebrovascular risk factors including hypertension and atherosclerosis as well as other diseases including Marfan syndrome, collagen vascular disease, Fabry disease, neurofibromatosis type I, and polycystic kidney were also thought to be associated to VBD. The increased vessel diameter in VBD may reduce blood flow velocity producing either ischemia or compressive symptoms, or provide flow turbulence inside vessels resulting in the formation of aneurysm which was prone to rupture. The course of VBD may be benign and stable or progressive after long time of stable condition.[1]

Due to its rare occurrence and unavailability of evidence-based management recommendation, tailored-made decision and individual approach were suggested. Mild VBD with smooth vertebrobasilar artery lumen may be managed with “watch and see” approach whereas VBD reconstruction was considered in moderate and severe symptomatic VBD, defined with unstable, stenosis,

fusiform dilation, or large aneurysm.[1] This patient had severe symptomatic clinical presentation of hemorrhagic and compressive VBD, which was therefore considered as a candidate for VBD reconstruction.

The endovascular approach to VBD reconstruction depended on the clinical manifestation of VBD, either ischemic, hemorrhagic, or compression.[1,4] This patient had hemorrhagic symptoms due to VBD rupture, which resulted in the decision to perform VBD reconstruction using multiple scaffolding Leo stents in combination with nondominant vertebral artery occlusion.[1] The stents were deployed along the VBD with anchors at both proximal and distal normal vascular sites. While there was no single stent superior to another, compared with Enterprise and solitaire, Leo stent was the only braided stent from a single self-expanding nitinol wire which had the best radial strength for 15% to 50% oversizing, low bending stiffness, highest kink resistance, lowest ovalization, highest bending wall coverage, lowest cell size, and can be repositioned after partial deployment.[8] This properties provided Leo with strong vascular reconstruction properties and vessel wall protection. Leo stent has been indicated for intracranial aneurysm, but it has also been reported to be used in a few case reports of VBD besides enterprise and Solitaire.[7-9]

Besides using multiple Leo stent, nondominant vertebral artery occlusion was also performed to reduce the blood flow into the space between the stents and the vascular wall, which can further improve the hemodynamics of the VBD. The occlusion should be performed as distal vertebral artery as possible to prevent occlusion of posterior inferior cerebellar artery and anterior spinal artery, but not to distal to obstruct This combination of technique has been reported as well in several studies.[7] Prior and following the endovascular reconstruction procedure, we administered double antiplatelet with additional dose at the day of procedure to prevent thrombogenic event by the stent including Leo.[8]

The outcome of VBD after endovascular reconstruction was heterogeneous. A case series of 22 cases by Wang et al reported that 7 cases of mortality and two (8%) cases of increased modified Rankin scale. The heterogeneity of clinical manifestation of VBD, the endovascular approach, and type of stent may contribute to the variation of outcome. This is one of the first studies using the combination of Leo stent and coiling with successful outcome, which may be further practiced and evaluated for the effectiveness and safety at a larger scale.

CONCLUSION

VBD is rare, but debilitating when it was symptomatic. Multiple approaches may be considered to decrease the complication and increase the success of endovascular reconstruction of VBD. Deploying multiple scaffolding Leo stents with occluding nondominant vertebral artery may be considered as an approach to successfully manage symptomatic VBD.

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