Case Report

Proximal basilar artery hemorrhage after submaximal angioplasty for intracranial atherosclerotic disease presenting as a large vessel occlusion treated with pipeline embolization device

Ryan M. Johnson[1], Michael Young[1], Gina N. Guglielmi[1], Hamad Farhat[2]

[1]*Department of Neurosurgery, Carle BroMenn Medical Center, Normal, IL, USA*
[2]*Department of Neurosurgery, Advocate Christ Medical Center, Oak Lawn, IL, USA*

Correspondence to
Ryan M. Johnson

Department of Neurosurgery, Carle BroMenn
Medical Center, 1300 Franklin Ave, Ste 110,
Normal, IL 61761, USA

Tel +1-740-632-3117
Fax +1-309-268-3713
E-mail ryan.johnson@carle.com
ORCID http://orcid.org/0000-0001-8700-2217

Iatrogenic vessel perforation from endovascular intervention is a devastating complication that commonly is treated with vessel sacrifice. We present a unique case of an iatrogenic proximal basilar artery perforation after submaximal angioplasty in a 67-year-old male presenting with an acute basilar artery occlusion with underlying intracranial atherosclerotic disease. Telescoping flow-diverting stents were then deployed to reconstruct the vessel wall with resulting active hemorrhage resolution. Our case documents a successful deployment of flow-diverting stents with resolution of active hemorrhage after an iatrogenic basilar artery perforation.

Keywords Intracranial atherosclerosis, Basilar artery, Iatrogenic disease, Angioplasty

INTRODUCTION

Intracranial atherosclerotic disease (ICAD) is becoming an increasingly well-recognized cause of ischemic stroke.[12] To date, treatment for symptomatic ICAD starts with maximal medical therapy (MMT) involving dual antiplatelet agents (aspirin and clopidogrel), statin therapy, and smoking cessation. Endovascular intervention for symptomatic ICAD refractory to MMT has been a topic of debate in recent years after the results of large randomized controlled trials showed higher periprocedural stroke and mortality rate for angioplasty and stenting.[5)13] Submaximal balloon angioplasty has arisen as a possible therapeutic intervention for symptomatic ICAD refractory to MMT.[12] Iatrogenic vessel perforation, either from an open surgical or

endovascular approach, is a well-recognized complication and has been treated with vessel sacrifice and covered stents in the literature.[8)9)] To our knowledge, there are no reports of an iatrogenic vascular perforation as a result of endovascular therapy treated with telescoping flow-diverting stents resulting in cessation of active hemorrhage. Our case documents such an experience in a patient with acute occlusion of the basilar artery with underlying severe ICAD treated with submaximal balloon angioplasty.

CASE DESCRIPTION

A 67-year-old male patient with a known history of symptomatic multifocal posterior circulation intracranial stenosis, treated only with aspirin 81 mg daily, was transferred to our institution with imaging findings concerning for an acute large vessel occlusion. His initial symptoms included headache and nausea occurring the night prior. The following morning the patient was unarousable and demonstrated acute symptoms of vomiting and seizure-like activity prompting emergency medical services. Initial head computed tomography (CT) was negative for acute intracranial hemorrhage. A subsequent computed tomography angiogram (CTA) of the head was obtained, demonstrating bilateral distal vertebral artery occlusion and complete basilar artery occlusion (Fig. 1A, B). Neurologically, the patient demonstrated bilateral upper extremity decorticate posturing, and flaccid bilateral lower extremities with a Glasgow Coma Score of five. The overall National Institutes of Health Stroke Scale was 24. The patient was intubated for airway protection. In the setting of acute neurological deterioration and concerning imaging findings, the patient was taken emergently to the neuro-interventional suite for treatment of basilar artery occlusion.

Initial angiogram was performed demonstrating complete occlusion of the basilar artery distal to the posterior inferior cerebellar artery (Fig. 2A, B), raising
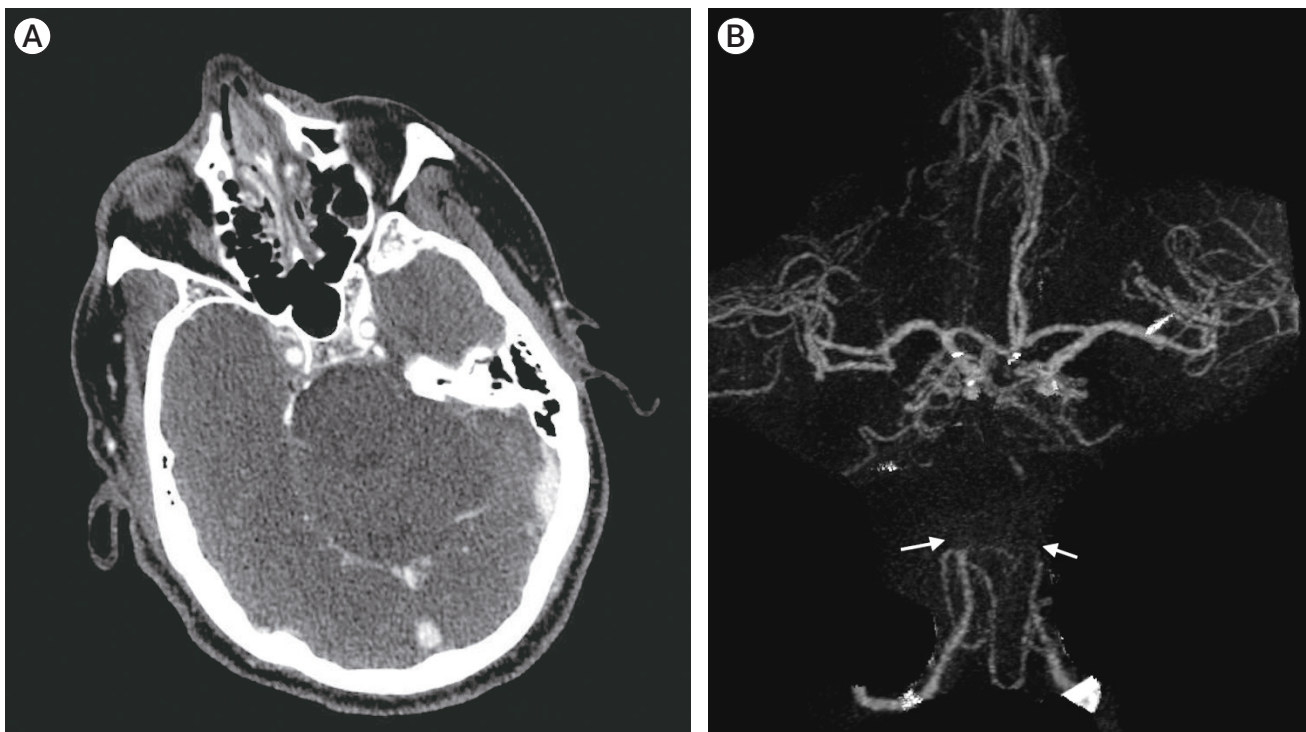
Fig. 1. (A) CT angiography of the head, axial view, demonstrating absence of intravascular contrast in the basilar artery at the level of the pons. (B) 3D-vessel reconstruction of the CT angiogram of the head showing bilateral intracranial vertebral artery occlusion distal to bilateral posterior inferior cerebellar arteries (arrows). CT, computed tomography.

Fig. 2. Left (A) and right (B) vertebral artery angiogram demonstrating complete occlusion of the proximal basilar artery just distal to the posterior inferior cerebellar artery origin.

the concern for acute occlusion of the basilar artery with superimposed atherosclerotic disease. A 5 F Sofia aspiration catheter (MicroVention, Aliso Viejo, CA, USA), which was then navigated into the right V4 segment. Aspiration thrombectomy was attempted with the aspiration catheter, which failed to recanalize the basilar artery. Focus then shifted to addressing the severe basilar artery atherosclerosis. A 2.5 mm×25 mm Sprinter balloon (Medtronic, Irvine, CA, USA) and a Synchro 2 microwire (Stryker, Kalamazoo, MI, USA) was then advanced into the distal basilar artery. Submaximal balloon angioplasty was then performed in the distal mid-basilar artery by inflating the balloon to a sub-nominal pressure of 6 atm (nominal pressure 8 atm). Post-angioplasty angiogram was then performed after the first attempt, which demonstrated persistent significant stenosis in the proximal basilar artery stenosis (Fig. 3A). The 2.5 mm×25 mm Sprinter balloon was re-inflated to a sub-nominal pressure of 6 atm. After the second attempt, post-angioplasty angiogram demonstrated active contrast extravasation in the proximal basilar artery (Fig. 3B). The balloon was immediately re-inflated for forty-five minutes to occlude the basilar artery and stop the active hemorrhage. The effects of

heparin were reversed by administration of protamine. During the forty-five minute balloon time, an external ventricular drain was inserted for intracranial pressure management. After forty-five minutes, the balloon was deflated and there was persistent contrast extravasation. The decision was made to place overlapping flow-diverting stents in attempt to re-direct flow distal to the luminal defect. A phenom-27 microcatheter (Medtronic) was advanced over a Synchro 2 microwire into the distal basilar artery. A 3.5 mm×16 mm Pipeline Embolization Device (PED, Medtronic) was deployed. The phenom-27 microcatheter was then advanced over a Synchro 2 microwire into the proximal mid-basilar artery where a second 3.5 mm×14 mm PED was deployed. Follow up angiogram after placement of both PEDs was unremarkable for active contrast extravasation (Fig. 4A, B). A post-procedure head CT confirmed diffuse subarachnoid hemorrhage with intraventricular hemorrhage (Fig. 5A, B). The patient's neurologic examination continued to deteriorate with gradual loss of brainstem reflexes. The patient died on post-procedure day two after family decision to transition to comfort care measures.
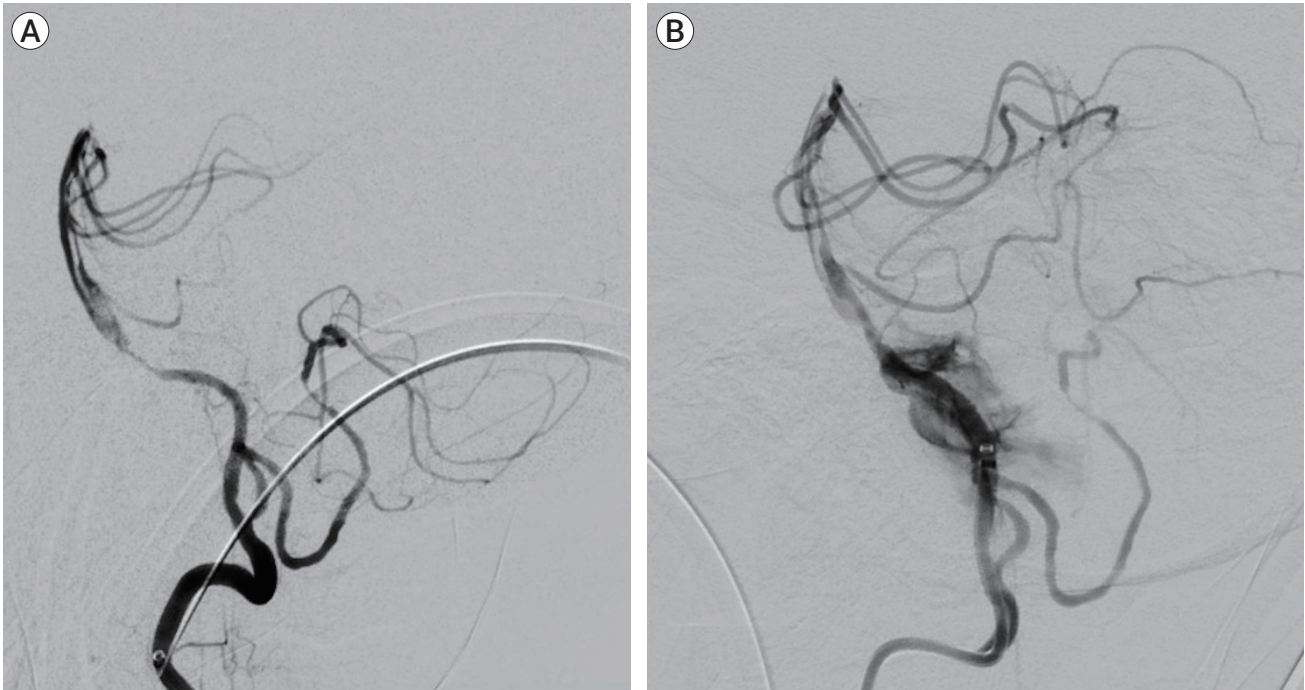
Fig. 3. (A) RVA angiogram after first attempt submaximal balloon angioplasty demonstrating residual stenosis of the proximal basilar artery. (B) RVA angiogram after second attempt submaximal balloon angioplasty demonstrating active contrast extravasation from the basilar artery. RVA, right vertebral artery.
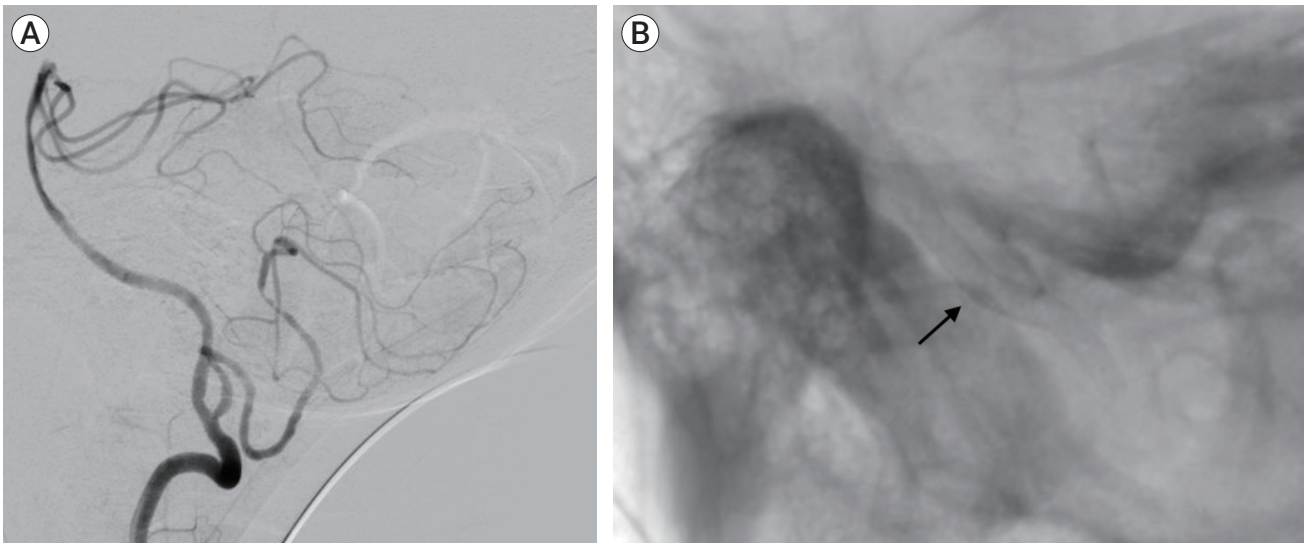

Fig. 4. (A) Right vertebral artery angiogram demonstrating cessation of contrast extravasation and vessel wall reconstruction after overlapping pipeline embolization device insertion into the basilar artery. (B) Lateral fluoroscopic x-ray demonstrating pipeline embolization devices (arrow).

DISCUSSION

ICAD is a well-documented risk factor for ischemic stroke and is reported to be the underlying cause of ischemic stroke in 5-10% of cases.[12] Vertebrobasilar ICAD is believed to be associated with a higher morbidity
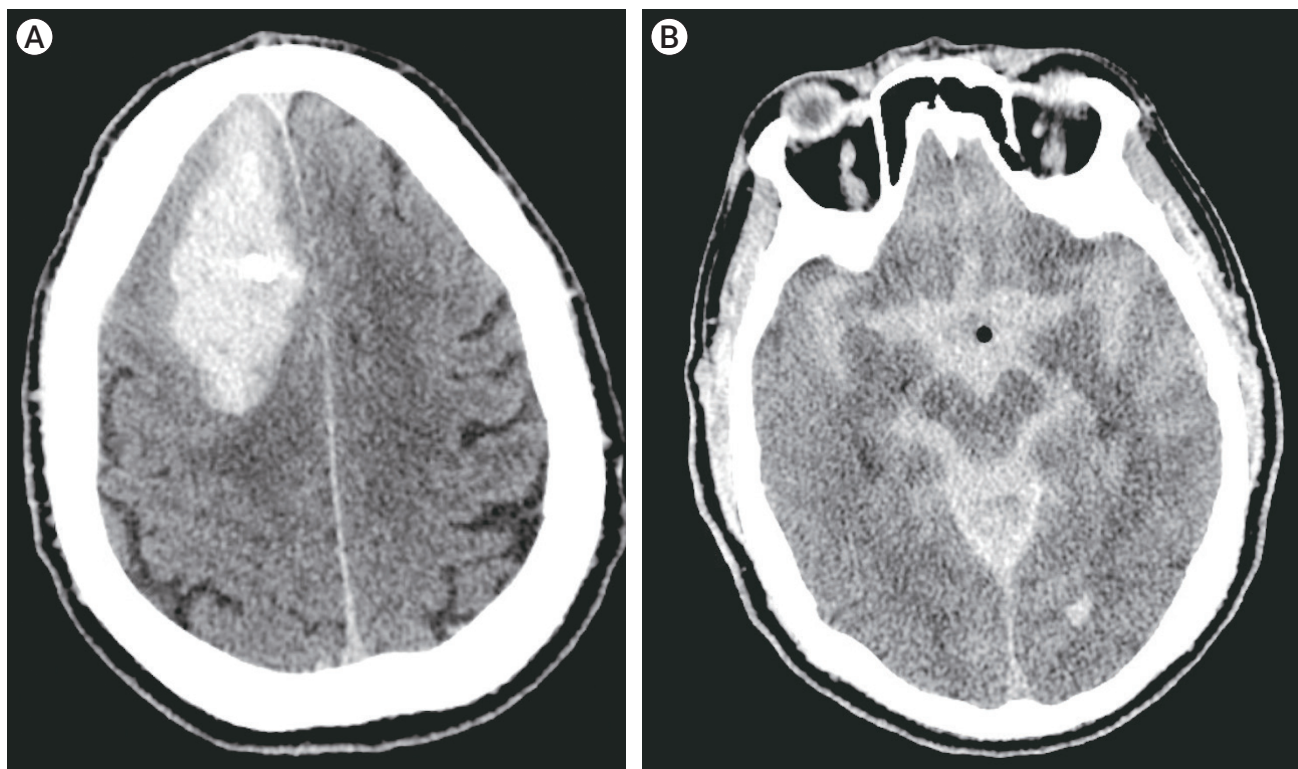
Fig. 5. (A) Post-procedure CT of the head demonstrating a right frontal intraparenchymal hematoma along the entry site of the external ventricular drain, and (B) diffuse subarachnoid hyperdensity in the perimesencephalic and sylvian cisterns likely representative of residual contrast and subarachnoid hemorrhage. CT, computed tomography.

and mortality due to the critical anatomical structures supplied by branching vessels and perforators arising from the vertebral and basilar arteries.[1)4)] Additionally, the posterior circulation has been shown to be more likely to have ICAD superimposed with an acute vertebrobasilar occlusion compared to anterior circulation large vessel occlusion.[10)11)] Advances in endovascular techniques have been challenged with the task of providing therapeutic intervention for symptomatic ICAD. Unfortunately, the higher periprocedural stroke and death rate associated with intracranial angioplasty and stenting in the SAMMPRIS Trial and the VISSIT Trial compared to aggressive medical therapy alone has resulted in a decreased utilization of these endovascular techniques for symptomatic ICAD.[5)13)] More recently, however, 19 studies incorporated into a meta-analysis with 777 patients documented stroke and death rate reduction at 30 days and 1-year associated with submaximal angioplasty (SA) for ICAD, providing support for SA in combination to aggressive medical therapy involving dual antiplatelet medications and statin therapy as a viable treatment option for symptomatic ICAD.[12)] An advantage of SA is related to the decreased radial force applied to the diseased vessel wall reducing the risk of iatrogenic injury to that vessel.[7)] There are case reports that document the utility of endovascular intervention for iatrogenic injuries to the intracranial vessels. Utilization of flow-diverting stents for treatment of unruptured iatrogenic extracranial and intracranial pseudoaneurysms has been documented in the existing literature as an effective treatment with high obliteration rate.[2)3)6)] The role of intravascular flow-diversion in the setting of frank vessel laceration or perforation has not been fully elucidated, and has only been documented in small volume case series' or isolated case reports. A brief review of the literature identifies two case series' totaling 7 patients with iatrogenic internal carotid artery (ICA) injury treated primarily with intravascular stent recon-

struction, six being treated with covered stents and one treated with a Pipeline Embolization Device (PED).[8)9)] Only one patient from these two papers suffered a frank laceration of the ICA, which was unsuccessfully treated with PED, and required coil embolization to sacrifice the vessel.[9)] The authors hypothesized that the laceration in the vessel created a pressure gradient from the intravascular space to the extravascular space that was greater than the flow-diverting properties of the PED. This questions the utility the flow-diverting stents in the setting of active hemorrhage due to vessel laceration, especially when there will be a need for dual antiplatelet therapy post-stent deployment.

It should be noted that the indications for Medtronic's PED does not include situations of active hemorrhage from vessel injury or ruptured aneurysms. The interventionalist needs to balance the risks and benefits of inserting a flow-diverting device that requires dual-antiplatelet therapy in the setting of active hemorrhage. Anti-platelet therapy would hasten an ongoing hemorrhagic event if the event was not controlled prior to placement of the device. In contrast, withholding anti-platelet therapy after placement of a flow-diverting device would increase the risk of developing stent thrombosis and a subsequent ischemic event.

We present a unique case of iatrogenic injury to the proximal basilar artery as a consequence of SA that was successfully treated with PED in the setting of acute vertebrobasilar occlusion with superimposed ICAD. The reason why intravascular flow-diversion resulted in cessation of hemorrhage in our patient, and not in the case series report by Griauzde et al.,[8)] cannot be definitively stated. Our patient experienced a vessel perforation as a result of internal to external pressure from balloon angioplasty, which, theoretically, created a small defect in the tunica intima of the vessel due to a stretching motion with extravasation of blood through the tunica media and tunica adventitia. In contrast, the vessel laceration created in the case series by Griauzde et al.[9)] demonstrates an external to internal disruption caused by a tearing motion, which likely created a larger defect in the tunica adventitia and tunica media of the vessel. We

hypothesize the pressure gradient between the intravascular and extravascular compartments in our patient's vessel perforation is smaller than a surgically created laceration, allowing the flow-directing properties of the PED to adequately reconstruct the vessel with resulting resolution of active hemorrhage. Despite our patient's negative outcome, our case documents the successful deployment of flow-diverting stents with resolution of active hemorrhage from an iatrogenic vessel perforation as a complication of endovascular therapy.

CONCLUSIONS

Vertebrobasilar ICAD is a risk factor for ischemic stroke with higher morbidity and mortality due to the eloquent anatomic structures supplied by those vessels. Iatrogenic injury to the basilar artery during endovascular intervention is a potentially devastating complication with a high morbidity and mortality. Our case documents, to our knowledge, the first successful deployment of flow-diverting stents with resolution of active hemorrhage after an iatrogenic vessel perforation.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

REFERENCES

1. Alexander MD, Rebhun JM, Hetts SW, Amans MR, Settecase F, Darflinger RJ, et al. Technical factors affecting outcomes following endovascular treatment of posterior circulation atherosclerotic lesions. Surg Neurol Int. 2017 Nov;8:284.

2. Ambekar S, Sharma M, Smith D, Cuellar H. Successful treatment of iatrogenic vertebral pseudoaneurysm using pipeline embolization device. Case Rep Vasc Med. 2014;2014:341748.

3. Amenta PS, Starke RM, Jabbour PM, Tjoumakaris SI, Gonzalez LF, Rosenwasser RH, et al. Successful treatment of a traumatic carotid pseudoaneurysm with the Pipeline stent: Case report and review of the literature. Surg Neurol Int.

2012;3:160.

4. Baek JH, Kim BM, Heo JH, Kim DJ, Nam HS, Kim YD. Endovascular and clinical outcomes of vertebrobasilar intracranial atherosclerosis-related large vessel occlusion. Front Neurol. 2019 Mar;10:215.

5. Chimowitz MI, Lynn MJ, Derdeyn CP, Turan TN, Fiorella D, Lane BF, et al. Stenting versus aggressive medical therapy for intracranial arterial stenosis. N Engl J Med. 2011 Sep;365(11):993-1003.

6. Dolati P, Eichberg DG, Thomas A, Ogilvy CS. Application of pipeline embolization device for iatrogenic pseudoaneurysms of the extracranial vertebral artery: A case report and systematic review of the literature. Cureus. 2015 Oct;7(10):e356.

7. Dumont TM, Sonig A, Mokin M, Eller JL, Sorkin GC, Snyder KV, et al. Submaximal angioplasty for symptomatic intracranial atherosclerosis: a prospective Phase I study. J Neurosurg. 2016 Oct;125(4):964-71.

8. Griauzde J, Gemmete JJ, Pandey AS, McKean EL, Sullivan SE, Chaudhary N. Emergency reconstructive endovascular management of intraoperative complications involving the internal carotid artery from trans-sphenoidal surgery. J Neurointerv Surg. 2015 Jan;7(1):67-71.

9. Griauzde J, Ravindra VM, Chaudhary N, Gemmete JJ, Ma- zur MD, Roark CD, et al. Use of the Pipeline embolization device in the treatment of iatrogenic intracranial vascular injuries: a bi-institutional experience. Neurosurg Focus. 2017 Jun;42(6):E9.

10. Lee JS, Hong JM, Lee KS, Suh HI, Demchuk AM, Hwang YH, et al. Endovascular therapy of cerebral arterial occlusions: Intracranial atherosclerosis versus embolism. J Stroke Cerebrovasc Dis. 2015 Sep;24(9):2074-80.

11. Lee YY, Yoon W, Kim SK, Baek BH, Kim GS, Kim JT, et al. Acute basilar artery occlusion: Differences in characteristics and outcomes after endovascular therapy between patients with and without underlying severe atherosclerotic stenosis. AJNR Am J Neuroradiol. 2017 Aug;38(8):1600-4.

12. Seyedsaadat SM, Yolcu YU, Neuhaus A, Rizvi A, Alzuabi M, Murad MH, et al. Submaximal angioplasty in the treatment of patients with symptomatic ICAD: a systematic review and meta-analysis. J Neurointerv Surg. 2020 Apr;12(4):380-5.

13. Zaidat OO, Fitzsimmons BF, Woodward BK, Wang Z, Killer-Oberpfalzer M, Wakhloo A, et al. Effect of a balloon-expandable intracranial stent vs medical therapy on risk of stroke in patients with symptomatic intracranial stenosis: the VISSIT randomized clinical trial. JAMA. 2015 Mar;313(12):1240-8.