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Endovascular treatment of ruptured pica aneurysms and association with its extradural origin: A single-center experience

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Abstract

Background: Posterior inferior cerebellar artery (PICA) like other intracranial arteries is prone to aneurysm formation. Aneurysms usually arise from the vertebral artery (VA)—PICA junction and the proximal segment of the PICA. The surgical clipping of PICA aneurysms can be challenging and carries a potentially significant risk of morbidity and mortality. Experience with endovascular therapy has been limited to a few studies; however, the use of endovascular therapy as an alternative treatment to surgery has been increasing. We present our experience of last 5 years in treating the ruptured PICA aneurysms. **Materials and Methods:** A total of 11 patients with PICA aneurysms, out of them 7 were at proximal PICA, 2 at the vertebral-PICA junction, and 1 each at mid and distal PICA, underwent endovascular treatment at our institution between 2011 and 2016. **Results:** All the patients presented with an acute intracranial hemorrhage, confirmed on CT head. Most of the aneurysms were at proximal PICA (anterior and lateral medullary segments) with the partial incorporation of PICA origin in the sac. Low origin of PICA was seen in 7 (out of 11) cases, out of these cases, 5 had proximal PICA, aneurysm, and one (*n* = 1) had VA-PICA, junction aneurysm (1/7) and. one distal PICA aneurysm. There were seven proximal PICA aneurysms, and out of them, parent vessel occlusion was done in six and selective coiling in one (*n* = 1) case. From seven (*n* = 7) proximal PICA aneurysms, there were five cases of low origin and rests showed normal course and origin. Two (*n* = 2) junctional aneurysms were treated with simple coiling. Low origin was seen in right VA-PICA junction aneurysm. Endovascular treatment of all the 11 aneurysms was successful. The treatment consisted of selective aneurysm coiling in four (36.3%) patients and aneurysm with parent vessel trapping in seven patients (63.6%). Out of these seven patients, in one (*n* = 1) patient where aneurysm was distal PICA, glue embolization was done. There was no intra-procedural rupture/contrast extravasation or any thrombo-embolic complications. Follow-up studies ranged from 6 months to 5 years. **Conclusion:** Endovascular therapy of ruptured proximal PICA aneurysms is possible and safe with the use of adjuvant devices and should be considered as first-line treatment.

Key words: Aneurysm; coiling; embolization; endovascular treatment; PICA

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Introduction

Posterior inferior cerebellar artery (PICA) is the largest branch of the vertebral artery (VA), originating above the foramen magnum in more than 80% of the cases. It has the most complex and variable course of the cerebellar arteries and is differentiated into anterior, lateral, posterior medullary, supra-tonsillar segments, and perforating branches based on its relationship with the medulla oblongata and cerebellum.[1-3] PICA aneurysms account for 0.5–3% of all intracranial aneurysms.[4-6] The majority of the aneurysms arise from the VA-PICA junction and the proximal segment of the PICA. These aneurysms can be either saccular or fusiform in nature. The treatment of saccular aneurysms can be done with surgery or with an endovascular approach. Surgery at this location is difficult because of the intimate relationship of the proximal PICA with the medulla and lower cranial nerves.[7] Endovascular treatment as an alternative treatment has been increasing because it avoids the manipulation of important posterior fossa structures.[2,3,8-12] The management of PICA aneurysms is challenging, in particular for the VA-PICA junction and the proximal segment aneurysms. The low origin of PICA also plays an important role in the development of the aneurysms. This study presents our experience of 5 years of treating these aneurysms with emphasis on the importance of the origin of PICA-deciding treatment strategy.

Materials and Methods

A total of 11 patients with PICA aneurysms, out of them 7 were at proximal PICA (anterior and lateral medullary segments), 2 at the vertebral-PICA junction, and 1 each at mid and distal (cortical) PICA, underwent endovascular treatment at our institution between 2011 and 2016. Pre- and postoperative neuroimaging studies and endovascular operative reports were reviewed. Follow-up studies ranged from 6 months to 5 years.

All the 11 patients (8 females, 3 males; mean age: 40.1 years, range: 22-62 years) presented with acute onset severe headache and non-contrast-enhanced computed tomography (NCCT)-documented subarachnoid hemorrhage (SAH) and intraventricular hemorrhage (IVH). Other presenting symptoms included headache (n = 11), nausea/vomiting (n = 11), altered sensorium (n = 3), and seizures (n = 1). Clinical grading was documented using Glasgow coma scale (GCS), Hunt and Hess grade and World Federation of Neurological Surgeons Grading System (WFNS) for SAH. According to the institutional management protocol, all patients underwent NCCT head, computed tomography (CT)-cerebral angiography, and four-vessel cerebral digital subtraction angiography (DSA) and then were taken up for endovascular treatment. CT grading of SAH was done using modified Fisher scale.

The type of endovascular treatment of acutely ruptured VA-PICA/proximal PICA aneurysms mainly depended on the morphology and geometry of the aneurysm and width of the aneurysmal neck based on the DSA and 3D rotational angiography, the origin of PICA in relation to aneurysm, and the presence and size of collaterals. The anterior and lateral medullary segments constitute the proximal PICA and beyond that the distal portion.

All patients were treated during the acute phase of SAH. Baseline pre-procedure NCCT head was done for evidence of re-bleed, infarction, or hydrocephalus. Endovascular coiling was performed on a uniplane angiographic unit (Philips Allure Xper FD 20/10; Philips Medical Systems, Netherlands) with the patient under general anesthesia. On the day of the procedure, a pre-procedure diagnostic cerebral angiogram was done after administering a bolus of 2500 U of heparin. During the entire duration of the procedure, heparinized drip infusion through the guiding catheter was given and if there was angiographic visible vasospasm then nimodipine infusion was given through the guiding catheter. The access to the involved VA was taken with a 6F or 5F guiding catheter (Envoy; Codman; USA or Chaperon; Microvention; USA). Aneurysm coiling was performed using echelon-10 micro catheter (Ev3; USA) with axium (Ev3, USA) or microplex (Microvention; USA) coils without or with balloon assistance (4 mm × 7 mm Hyperform, Ev3, USA). In one patient where aneurysm was very distal and fusiform, glue embolization was done. The rest of them were packed with simple coiling.

The aim of coiling was to obtain an optimal packing density of aneurysm with or without parent vessel occlusion and was done until no residual opacification of aneurysmal sac was seen on an angiogram. Procedure related complications (aneurysm rupture or thromboembolism) of coiling and adverse events were recorded. Postprocedure imaging was done as and when required, and the patients were monitored in intensive care units.

The patients were scheduled for clinical follow-up at 3, 6, and 12 months. The final clinical outcome was assessed at the last clinical visit using modified Rankin score (mRS).

Results

Patients and aneurysms characteristics are shown in Table 1. All the patients presented with acute intracranial hemorrhage on NCCT head. Fisher's grade of SAH was: Grade 2 in three; grade 3 in three; and grade 4 in five patients. Hunt and Hess grading at admission was: grade II in seven patients; grade III in three patients; and grade IV in one patient. The aneurysm was located on the right side in five patients and on the left side in six patients. Seven aneurysms were located at proximal PICA with the partial or complete incorporation of its origin in the sac [Figures 1 and 2].

One aneurysm was seen at middle PICA [Figure 3], two aneurysms were at VA-PICA junction [Figure 4] and only single aneurysm noted involving distal PICA [Figure 5]. The size of the aneurysm ranged from 4 mm to 13.0 mm, mean size being 5.8 mm. The timing of treatment after SAH was within 4 days in eight patients and >4 days in three patients. No pre-procedural neurological deficit was noted. The angiographic evidence of local vasospasm was there which responded well to nimodipine infusion, and there was no clinical manifestation due to vasospasm in any patient.

Low origin of PICA was seen in 7 (out of 11) cases; out of these cases, 5 had proximal PICA aneurysm, one (n = 1) had VA-PICA junction (1/7), and the other had mid PICA aneurysm (1/7). There were seven proximal PICA aneurysms, and out of those, parent vessel occlusion was done in six and selective coiling in one (n = 1) case. From seven (n = 7) proximal PICA aneurysm, there were five cases of low origin and rests showed normal course and origin. Two (n = 2) junctional aneurysms were treated with simple coiling. The treatment consisted of selective aneurysm coiling in four (36.3%) patients and aneurysm with parent vessel trapping in seven patients (63.6%). Out of four cases of dissecting aneurysm, low origin of PICA was seen in 50% of cases. The endovascular treatment of all the 11 aneurysms was performed in a single sitting; no patient required a repeat procedure. Balloon-assisted coiling was done in one patient where vertebral PICA junction was involved; in this patient, we kept a super compliant balloon in the vertebral artery and overinflated it so as to cover the neck of the aneurysm, which was coiled with sparing of the PICA. Parent vessel embolization with aneurysm coiling was done in seven (n = 7) patients, and in one patient, glue [Figure 5] was used to do the same where aneurysm

Table 1: Patient characteristics, aneurysms location, management and follow up

Age/ sex	Hunt and Hess Grade	Fisher Grade	WFNS grade	CT Findings	Location	Etiology/ Shape	Parent vessel occlusion	Treatment	Low origin of PICA	Clinical outcome
M/45	2	3	2	SAH	Left VA-PICA junction	Saccular	No	Balloon Assisted coiling	No	Improved
F/58	2	4	2	SAH/IVH	Left- proximal PICA	Fuso-saccular	Yes	Simple coiling	no	Improved
M/35	2	3	1	SAH	RT Mid PICA	Saccular	No	Simple coiling	Yes	Improved
F/50	4	4	3	SAH/IVH	Lt distal PICA	Fuso-saccular	Yes	Glue embolization	No	Improved
F/55	2	4	1	SAH/IVH	Rt VA-PICA junction	Saccular	No	Simple coiling	Yes	Improved
M/59	2	3	1	SAH	Rt proximal PICA	Saccular	Yes	Simple coiling	Yes	Improved
F/22	2	2	1	SAH/IVH	Lt proximal PICA	Fuso-saccular	Yes	Simple coiling	Yes	Improved
F/62	3	4	2	SAH/IVH	Lt proximal PICA	Saccular	No	Simple coiling	Yes	Improved
F/52	2	2	1	SAH/IVH	Rt proximal PICA	Fuso-saccular	Yes	Simple coiling	Yes	Improved
F/38	3	2	2	SAH/IVH	Lt proximal PICA	Saccular	Yes	Simple coiling	No	Improved
F/45	3	4	2	SAH/IVH	Lt proximal PICA	Saccular	Yes	Simple coiling	Yes	Improved

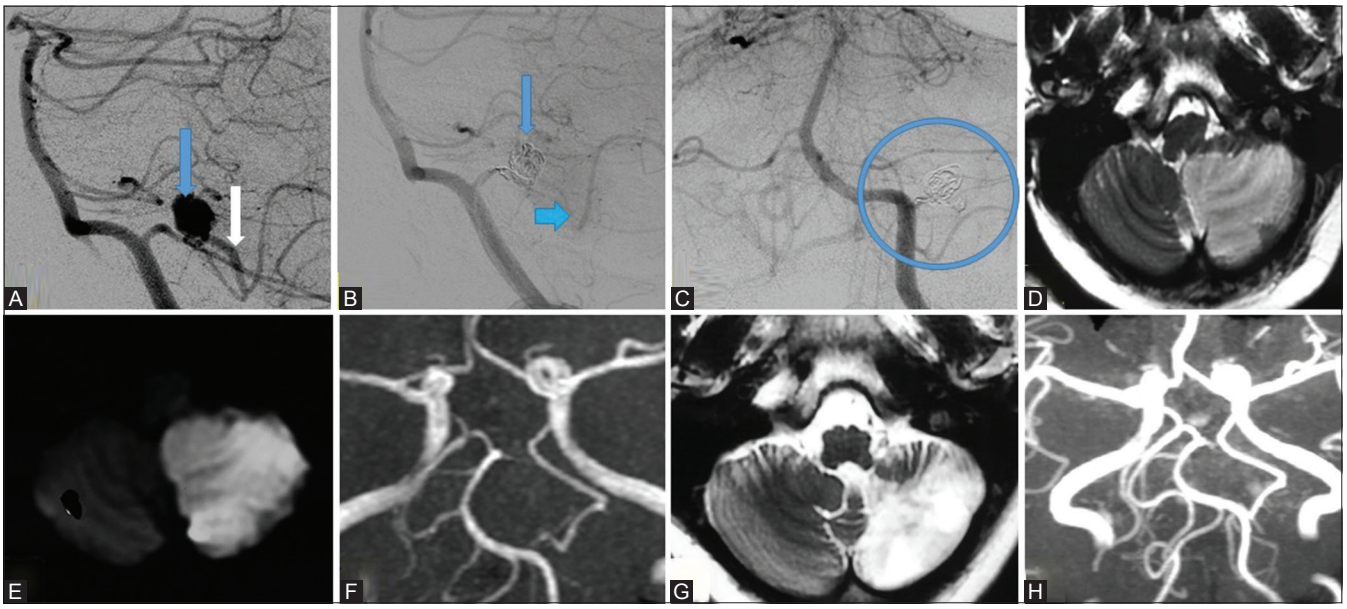

Figure 1 (A-H): 38Y/F; A large saccular aneurysm (blue arrow, A) in a lateral medullary segment of LT PICA (white arrow, A) with parent vessel occlusion. Faint enhancement of PICA (Blue arrow, B). Image C (circle) shows non-visualization of PICA. Follow-up MRI; T2 (D) and DWI images (E) at 6 months shows left cerebellar infarct with no recanalization of the sac on MRA (F). At 3-year follow-up MRI; T2WI (G) showed chronic infarct in left cerebellar hemisphere with no recanalization of aneurysm on MRA (H). The patient is clinically asymptomatic

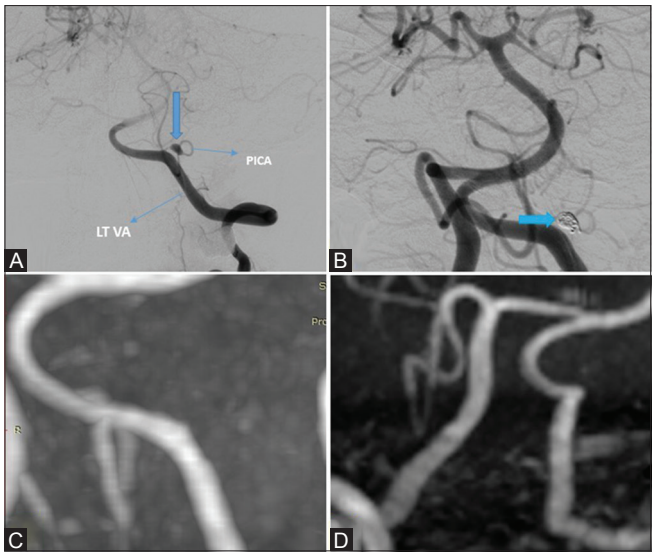

Figure 2 (A-D): 58y/F; A fuso-saccular aneurysm in a lateral medullary segment of LT PICA (arrow; A). Parent vessel occlusion with coiling (blue arrow; B). Follow-up MRA at 3 (C) and 6 (D) months; showed no e/o aneurysm or PICA

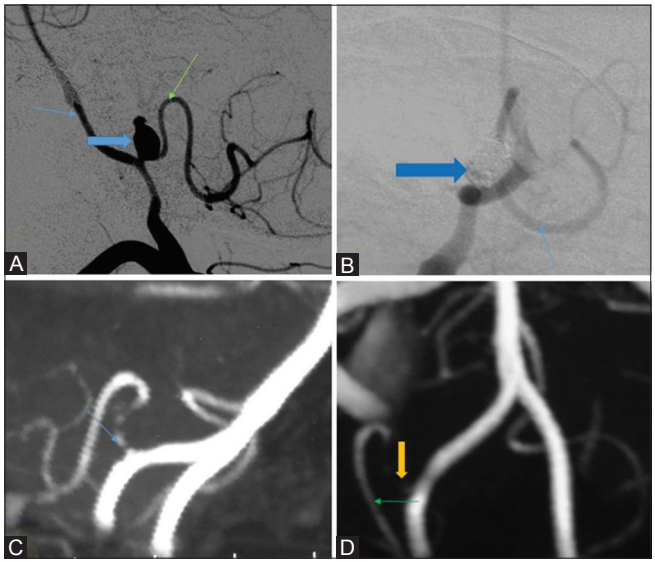

Figure 4 (A-D): 55y/F; A fuso-saccular aneurysm (arrow; A) at the junction of VA (small arrow)-PICA (green arrow) on RT side managed with coiling (blue arrow; B). PICA (small red arrow; B) was seen post coiling. Follow-up MRA at 3 (C) and 6 (D) months; shows narrowing of proximal PICA (arrow; C) and no e/o aneurysm. Narrowed proximal PICA (yellow arrow, D). Distal portion is normal (green arrow, D)

was in distal cortical branch and appear to be dissecting. No procedural complication, either aneurysm rupture or thromboembolic event, was noted.

Clinical outcome

Out of 11 patients, 10 patients (90.9%) had no neurological deficit post-procedure and 1 patient (9.1%) had a postprocedural complication as the patient was Hunt and Hess grade 4 and developed seizures following which her condition deteriorated and was put on ventilator support.

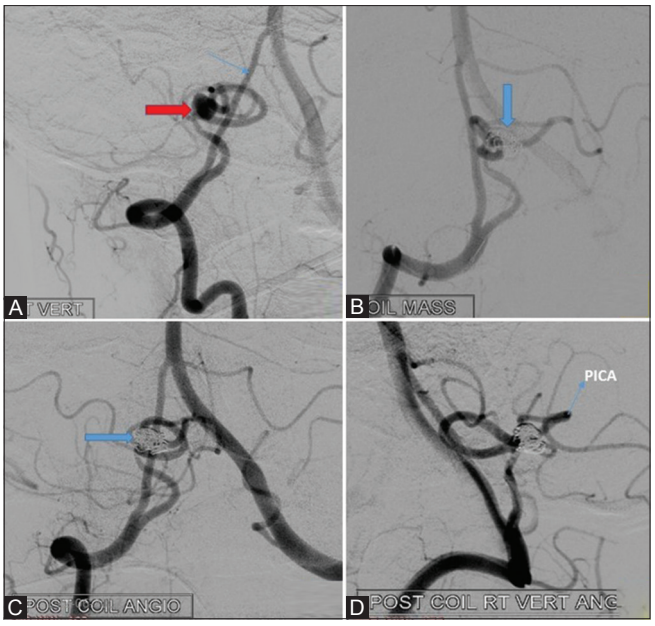

Figure 3 (A-D): 35Y/M; A saccular aneurysm (red arrow; A) in mid (Tonsillo-medullary segment) of RT PICA with simple coiling (arrow; B, C). RT V4 is hypo plastic (small arrow; A). Parent vessel is spared (C and D). The patient was clinically asymptomatic on follow up

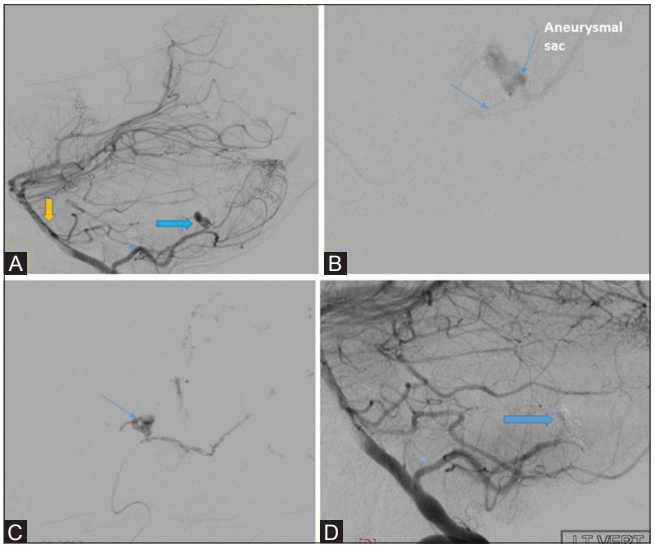

Figure 5 (A-D): 50Y/F; A fuso-saccular aneurysm (blue arrow; A) in a cortical segment of LT PICA (small red arrow; A). Lt Vertebral artery (yellow arrow) Glue Embolization (small arrow; C) with micro-catheter (small arrow, B). (D) Post-procedural angio showed no filling of aneurysmal sac (red arrow). PICA (small arrow) is patent

Imaging showed obstructive hydrocephalus, which was managed with a ventricular peritoneal (VP) shunt. Long-term follow-up (3 years) showed mRS grade 2.

Long-term (up to 5 years) clinical outcome was available for nine patients. Two patients lost to follow-up, but none had neurological deficient at 6 weeks. None had re-bleeding after the endovascular treatment or during the follow-up period. We followed the patients with sequential MRA every

6 months, and no recanalization of an aneurysm has been witnessed.

Discussion

PICA typically arises from the intracranial portion of the VA (80–95% of cases). The PICA can be divided into five segments and two loops, based on its relationship with the medulla oblongata and the cerebellum. The segments are the following: anterior medullary, lateral medullary, tonsillomedullary, telovelotonsillar, and cortical.[13] Dissecting aneurysm is diagnosed if an irregular fusiform dilatation, with or without associated narrowing of the PICA, has been demonstrated angiographically.[14] In the majority of cases, the etiology of dissecting aneurysms is unknown.[15]

PICA dissecting aneurysms account for approximately 0.5–0.7% of all intracranial aneurysms. SAH is the most common initial clinical presentation (74%) with ischemia accounting for the remaining cases.[16] Kanou *et al*.[17] reported that dissecting aneurysms of the proximal PICA tend to cause infarctions and that peripheral dissections tend to lead to SAH. In our series, all patients presented with SAH, with or without intraventricular hemorrhage.

The dissecting aneurysm of the PICA carries a 24% risk of re-bleeding in the acute phase,[18] associated with a high mortality rate; thus, early intervention is essential.

The management of PICA aneurysms that cause SAH is challenging and remains controversial. Most cases reported in the literature have been treated by the surgical occlusion of the PICA or trapping of the dissected segment. They suggested that the trapping of the involved segment with distal revascularization is the treatment of choice, which is done with open neurosurgical techniques but carries a high risk of lower cranial nerves palsies. More recently, successful endovascular treatment of dissecting PICA aneurysms has been reported.[19,20] The endovascular approach includes the occlusion of the aneurysm and the parent artery at the dissected segment.

The parent artery occlusion of the PICA is potentially associated with two types of ischemic complication. First, brain stem ischemia caused by the occlusion of the perforating arteries supplying the brain stem, which may originate from the most proximal segments of the PICA, and second, cerebellar ischemia distal to the occlusion site.

In all patients, the aneurysm and the dissected segment had been occluded without neurological consequences. In addition, in the three most recent publications,[19,20] which reported the results of endovascular treatment in dissecting PICA aneurysms, there were nine aneurysms located in the two more proximal segments. All aneurysms were treated endovascularly with parent artery occlusion, resulting in an excellent neurological outcome. The risk of brain stem ischemia in cases of permanent occlusion at the first two segments is limited because of the absence of normal perforating branches in aneurysmal dilatations of the dissected arterial segments.[21] Moreover, the risk of brainstem ischemia is also limited because of the numerous anastomoses of the perforating arteries forming a plexiform network on the medullary surface.[22] Furthermore, in both surgical and endovascular treatments, the diseased segment is occluded, thus perforating vessels in that area are already compromised.

On the other hand, the occlusion of PICA distal to the telo-velo tonsillar segment generally does not result in brain stem injury. Balloon test occlusion can be performed to accurately evaluate the patient's capability to tolerate permanent occlusion. In cases where collateral supply (from AICA and contralateral PICA) is insufficient, potentially there is the option of surgical bypass. We did not do balloon test occlusion or preprocedural collateral evaluation in any of our patients as even if permanent PICA occlusion is followed by cerebellar infarct, it is usually of limited size, and patients usually tolerate it well,[23] as we have seen in our cases of parent vessel occlusion where demonstrable cerebellar infarcts occurred without clinical manifestation. It is well known that MRI is more accurate in depicting ischemic lesions, especially in the posterior fossa, and therefore, the incidence of postprocedural cerebellar infarcts could be higher, but most of them go silent. The ideal treatment for a dissecting aneurysm is the trapping of the aneurysm with distal revascularization, which is done by surgical occipital artery to PICA bypass, but again as stated earlier this carries a high risk of lower cranial nerve palsies, along with other neurosurgical complications; however, if a patient can tolerate this then the long-term outcome, as stated by Wang *et al*., is good.[24]

When we compare our results to a larger series of PICA aneurysms, like one of Peluso *et al*.,[25] our results with parent vessel occlusion are better, may be because of limited numbers, and they have also stated that most cerebellar infarcts are asymptomatic, which we have also found. Overall procedural complications such as aneurysm rupture during the coiling of PICA aneurysm were not there in our series, probably owing to advancements in the technique and hardware.

Origin of PICA whether intradural or extradural (C1 or C2 origin) is important in deciding the treatment strategy for proximal PICA aneurysms. As illustrated by Lasjaunias *et al*.,[26] PICA can have both intradural and extradural origin but in all extradural origin, the medullary perforator of PICA arises from the location where the classical PICA would come from. So, extradural PICA occlusions would not lead to the classical lateral medullary syndrome, but the C2 origin

of PICA needs a special word of caution as lateral or posterior spinal artery supply might be there. In all our cases, low or extradural origin of PICA was there in seven cases, and out of these, proximal artery aneurysms were there in five cases, and we did parent vessel, along with aneurysm occlusion, in four of them with no patients presenting with lateral medullary syndrome, and in this way, we fully agree with Lasjaunias that the extradural origin of PICA can be occluded with no risk of lateral medullary syndrome. The tendency of the low origin of PICA associated with dissection and dissecting aneurysms is probably because of shearing trauma at the dural entry site, which is well known for vertebral arteries,[27] and a case report of extradural origin PICA dissection following chiropractic manipulation is also there.[28] The low origin of PICA and dissecting aneurysms have opened a new window for further exploration of the association between them, thereby deciding their proper management.

Conclusion

According to our results, the endovascular treatment of dissected PICA aneurysms with the occlusion of both the dissecting aneurysm and parent artery has an excellent clinical outcome. Despite the positive clinical outcome, according to the recent reports, the preprocedural angiographic evaluation of collateralization may be studied carefully prior to taking a decision on the treatment strategy to avoid extensive cerebellar damage and edema, especially in the setting of SAH.

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Conflicts of interest
There are no conflicts of interest.

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