Introduction

Vertebral artery-posterior inferior cerebellar artery (VA-PICA) aneurysms constitute 0.5-3% of all intracranial aneurysms.[1-3] Surgery for these aneurysms is extremely challenging. PICA has the most complex and variable course among all arteries of the posterior circulation. The complexities involved in the surgical approach to the PICA aneurysms relate to the narrow corridor limited by the brain-stem, petrous-occipital bones, and multiple neurovascular structures occupying the cerebellomedullary and cerebellopontine cisterns. Moreover, an anomalous PICA is reported in 4-16% patients.[2,5]
PICA originates from the intracranial portion of VA in 80-95% patients, approximately 8.6 mm above the foramen magnum and 1 cm proximal to the verteobasilar junction. PICA aneurysms may take origin from one of its six segments and two loops (based on its relationship to the medulla oblongata and the cerebellum including: a) the BA-VA-PICA junction; b) the anterior medullary segment, from VA-PICA origin to the inferior olivary prominence; c) the lateral medullary segment, extending until the origin of IX-X-XIth cranial nerves; d) the tonsillomedullary segment, until the caudal portion of tonsils (including the caudal loop); e) the telovelotonsillar segment, from the midportion of its ascent along the medial surface of tonsil to the cortical cerebellar surface (including the cranial loop); and, f) the cortical segment, extending until the cerebellar vermis and hemisphere. 

We focus on the surgical management of saccular VA-PICA aneurysms with emphasis on the modifications in the approach based upon the location of aneurysms and variation in PICA, and also study the perioperative dilemmas encountered.

Materials and Methods

Patient spectrum

Twenty consecutive patients (mean age: 41.6 years, male: female ratio 3:17) admitted to Department of Neurosurgery and harboring ruptured PICA aneurysms underwent surgical management over an eight-year period. Their mean ictus-surgery interval was 23.5 days (range 3-150 days). Sudden onset severe holocranial headache was the presenting feature in 19 patients; 8 experienced transient loss of consciousness and 4 had associated seizures [generalized tonic clonic (n=2), focal with secondary generalization (n=1) and, tonic seizure without clonic movements (n=1)]. Neck stiffness was present in 15 patients. Their Hunt and Hess score (HandH) grade at admission was I in 15; II in 2; and III in 3 patients [Table 1].

Radiographic features

All patients underwent a CT scan at the time of ictus [Table 2]. The site of hemorrhage was localized to posterior fossa cisterns in 5 (25%) patients; supra- and infratentorial cisterns in 6 (30%) patients; and in the cerebellar parenchyma, and in the interhemispheric/tentorial areas in one patient each, respectively. Third ventricular blood was seen in 2 (10%) patients; intra-IVth ventricular blood in 12 (60%) patients; and lateral ventricular blood in 3 (15%) patients. One patient (5%) had a normal CT scan. Among the 12 patients with intra-IVth ventricular hemorrhage, 6 each had purely ventricular or ventricular associated with cisternal hemorrhage, respectively. When IVth ventricular hemorrhage was not associated with cisternal hemorrhage, PICA aneurysms were usually located peripherally on the vessel (on lateral medullary (n=1), tonsillomedullary (n=1) and telovelotonsillar (n=4) segments, respectively) with blood entering the IVth ventricle either through the foramen of Luschka or Magendie. The PICA aneurysm was diagnosed on the basis of digital subtraction angiogram (DSA) in 16 patients (two of these also underwent an additional computed tomodraphic angiogram (CTA)); and only on CTA/MRA images in 4 patients. The location of PICA aneurysms was as follows: VA-BA-PICA junction (n=5); anterior medullary segment (n=4); lateral medullary segment (n=3); tonsillomedullary segment (n=4); and telovelotonsillar segment (n=4). There was no aneurysm located on the cortical segment of PICA. Notable findings on angiograms were large variations in the course of VA-PICA and the existence of numerous vascular anomalies [Table 3]. These included fenestrated or tortuous PICA; brain-stem compression by a giant PICA aneurysm [Figures 1 and 2]; two PICA aneurysms on a common trunk proximal to an arteriovenous malformation (AVM); bilobed or multiple aneurysms; low PICA situated at or below foramen magnum with or without a hypoplastic VA [Figures 3-5]; and partially or completely thrombosed aneurysms. In two patients, the preoperative CTA was unable to pick up the PICA aneurysm that was subsequently detected on DSA.

Surgical treatment

In patients who were admitted within 2 weeks of ictus, oral nimodipine and steroids (intravenous dexamethsone 4 mg hourly) were instituted. All patients also received phenytoin (100 mg 6 hourly). Following angiographic confirmation of the aneurysm, surgery was performed usually as the first case the next day morning. The surgical approaches included a retromastoid suboccipital cranietomy (n=9; usually for aneurysms located lateral to brain stem); midline suboccipital

Figure 1: Patient 1– (a) Axial CT scan shows a giant, thrombosed PICA aneurysm causing brain-stem compression; (b) T2-axial and, (c) Sagittal MRI showing aneurysm surrounded by hemosiderin ring and having a laminated core; (d) Contrast coronal MR showing intimate relationship of aneurysm to brainstem; (e) MR angiogram showing the aneurysm lateral to midline
Table 1: Spectrum of patients with PICA aneurysms

| Age, sex | Symptom, signs | Grade,* Ictus- surgery interval (days) | Pre-operative CT findings | Aneurysm location,† surgical approach | Important imaging‡ and operative findings | Post operative additional neurological deficits, outcome§ |
|----------|----------------|--------------------------------------|---------------------------|--------------------------------------|-----------------------------------------|--------------------------------------------------|
| 54, F    | Headache, vomiting, neck stiffness | I, 4 | Posterior fossa cisternal bleed | 2, RMSO | Saccular aneurysm, pica extremely tortuous | None,0 |
| 20, F    | Headache, vomiting, transient unconsciousness, neck stiffness | I, 8 | Intracerebellar and intra IVth ventricular bleed | 4, Midline suboccipital craniectomy | Saccular aneurysm, large thrombus in the sac | Right IVth nerve paresis, mild right cerebellar signs, 1 DSA: aneurysm clipped but non-visualisation of main PICA trunk |
| 32, F    | Headache, vomiting, transient unconsciousness, | I, 88 | IIIrd and IVth intraventricular bleed with hydrocephalus | 3, Midline suboccipital craniectomy | Bilobed, saccular aneurysm | None,0 |
| 40, M    | Headache, vomiting, altered sensorium, follows simple command, neck stiffness | III, 19 | Cistern magna, IVth and lateral ventricular bleed with hydrocephalus | 3, Midline suboccipital craniectomy | AVM filling from left pica and draining in transverse sinus, two aneurysm present proximal to AVM, microcatheterization and embolization attempt failed | Mild left cerebellar signs, IX-Xth nerve paresis, 1 DSA: Aneurysms clipped and AVM excised but non-visualisation of main PICA trunk |
| 32, M    | Headache, vomiting, transient unconsciousness, Left VIth nerve palsy and left cerebellar signs | III, 13 | IVth and lateral ventricular bleed with hydrocephalus | 4, RMSO | Partially thrombosed saccular aneurysm, temporary clip applied for 15 minutes, no vasospasm | Left VIth nerve palsy and cerebellar signs persisted,1 |
| 17, F    | Mild holocranial headache, neck stiffness | I, 38 | IVth ventricular bleed, no hydrocephalus | 4, Midline suboccipital craniectomy | Saccular aneurysm, with pica originating below foramen magnum, intraoperative rupture with temporary clip application in total for 11 minutes, small residual neck wrapped with surgicel | None,0 |
| 31, F    | Occipital headache, one episode of GTCS, neck stiffness | I, 4 | Basal cistern bleed | 4, Midline suboccipital craniectomy | Right pica had anomalous origin below foramen magnum being in continuation of vertebral artery as distal vertebral artery was hypoplastic. Aneurysm was saccular, bilobed | CSF leak from surgical incision wound, increased cerebellar signs, bilateral VIth nerve paresis, 2 |
| 70, F    | Headache, vomiting, transient unconsciousness, neck stiffness | I, 8 | Sellar, suprasellar, preoptine, perimesencephalic and intra IVth ventricular bleed with hydrocephalus | 2, RMSO | Pica had fenestrated origin. Saccular aneurysm with neck in close proximity to lower cranial nerves | None,0 |
| 40, F    | Headache, vomiting, neck stiffness | I, 7 | Right sylvian, preoptine and ambient cistern bleed | VA-pica junction, RMSO | Saccular aneurysm | Left MCA Infarction, septicemia, lower cranial nerve involvement, 6 |
| 40, F    | Headache, vomiting, transient unconsciousness, one episode of right focal seizure with secondary generalization | II, 17 | Normal | VA-pica junction, Far lateral approach | Saccular aneurysm | Left MCA Infarction, septicemia, lower cranial nerve involvement, 6 |
| 58, F    | Headache, vomiting, neck stiffness | I, 3 | Bleed in suprasellar, ambient cistern and IIIrd and IVth ventricular bleed with hydrocephalus | 3, RMSO | Saccular aneurysm | Developed right hemiplegia during digital subtraction angiography due to vertebral dissection which persisted postoperatively, 4 |

(Continued)
cavernous ICA aneurysm close to midline; additional left cavernous ICA aneurysm

47, F Headache, vomiting, neck stiffness I, 5 Suprasellar, bilateral sylvian, right ambient cistern blood 1, Far lateral approach Dominant right VA looping close to midline; additional left cavernous ICA aneurysm None, o

The tortuosity of VA-BA complex also influenced the decision to use either a retromastoid suboccipital craniectomy or a far lateral approach while addressing PICA aneurysms of BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysms (that is, BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysms (that is, BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysms (that is, BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysms (that is, BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysms (that is, BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysms (that is, BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysms (that is, BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysms (that is, BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysms (that is, BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysms (that is, BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysms (that is, BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysms (that is, BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysms (that is, BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysms (that is, BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysms (that is, BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysms (that is, BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysms (that is, BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysms (that is, BA-VA-PICA junction (n=5) and anterior medullary segment (n=4). In these anteriorly situated aneurysm
Table 2: The radiological features seen on plain computed tomography at admission

| Features on plain computed tomography at admission | No of patients (n=20) | Percentage |
|---------------------------------------------------|-----------------------|------------|
| Subarachnoid hemorrhage                           |                       |            |
| Limited to infratentorial cisterns                | 5                     | 25         |
| Supra- and infra-tentorial cistern                 | 6                     | 30         |
| Intraventricular hemorrhage                        |                       |            |
| Fourth ventricle hemorrhage                        | 12                    | 60         |
| Third ventricular hemorrhage                       | 3                     | 15         |
| Lateral ventricle hemorrhage                       | 3                     | 15         |
| Isolated tentorial and posterior interhemispheric bleed | 1                     | 5          |
| Hyperdense prepontine mass with subarachnoid hemorrhage | 1                     | 5          |
| Hydrocephalus                                      | 8                     | 40         |
| Intracerebellar bleed                              | 1                     | 5          |
| Normal study                                       | 1                     | 5          |

Table 3: Radiological variations in PICA aneurysm and related anatomy

| Variations                                             | Number of patients |
|--------------------------------------------------------|--------------------|
| Brain stem compression with a thrombosed, giant aneurysm | 1                  |
| Two PICA aneurysms on the common trunk proximal to an AVM | 1                  |
| Bilobed aneurysm                                       | 2                  |
| Low PICA below foramen magnum with hypoplastic VA       | 1                  |
| Low PICA originating below foramen magnum               | 2                  |
| PICA origin at foramen magnum                          | 1                  |
| Multiple aneurysms, one at VA-PICA junction and other at basilar and vertebral artery junction on the contralateral side | 1 |
| Multiple aneurysms, one in anterior medullary segment of PICA and other at cavernous carotid location | 1 |
| Thrombosed aneurysm                                    | 1                  |
| Partially thrombosed aneurysm                          | 3                  |
| Fenestrated PICA origin                                | 1                  |
| Extremely tortuous PICA                                 | 1                  |

PICA – Posterior inferior cerebellar artery; AVM – Arteriovenous malformation; VA – Vertebral artery

well as anterior medullary), a relatively straight segment of BA-VA in 5 patients favored utilization of the far lateral approach to access the aneurysm located close to the midline anterior to the brain stem; while the looping of the vessels toward the cerebellomedullary cistern made the aneurysm easily accessible using the conventional retromastoid approach in 4 other patients. Temporary clipping was utilized in two patients (patient 5 and 6). None of these patients had any additional neurological deficits in the postoperative period. Following surgery, nimodipine and hypertensive, hypervolemic, hemodilutional (triple H) therapy were continued for two weeks in 4 patients who were operated within a week of their ictus, had a transient neurological deterioration in the form of transient aphasia and right hemiparesis. None of these patients had any additional neurological deficits in the postoperative period. 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Figure 5: Patient 2 – Operative images showing (a) Retracted cerebellum exposing lower cranial nerves; (b) Clot in cerebellomedullary cistern; (c and d) PICA aneurysm from anterior medullary segment with draped lower cranial nerves; (e and f) Neck clipped sparing the origin of PICA

Figure 6: Patient 3 – (a and b) Axial CT scans showing SAH within suprasellar, sylvian, preopticine and ambient cisterns; (c) Anteroposterior; and (d) Lateral left vertebral angiogram showing PICA aneurysm near anatomical midline; (e and f) CTA shows dominant left VA harbouring aneurysm; (g) Oblique right carotid angiogram showing an additional cavernous ICA aneurysm

Figure 7: Patient 3 – Far lateral approach; (a) Dural exposure; (b) Exposed cerebellum and cistern magna; (c) Opening cistern magna reveals PICA below tonsil; (d) Cerebellar retraction exposes anterior medullary segment aneurysm anterior to lower cranial nerves and brain-stem with fundus attached to jugular tubercle; (e) Aneurysm clipped sparing PICA origin

Figure 8: Patient 4 – (a) Three-dimensional reconstructed axial; (b) coronal; (c) sagittal; and (d) three dimensionnal reconstructed oblique CTA showing BA-VA-PICA aneurysm in a high, anterior location. Left VA-BA junction is incorporated in aneurysm wall. Right VA is draped over aneurysmal fundus

Figure 9: Patient 4 – Far lateral approach; (a) Cerebellar retraction exposes lower cranial nerves; (b) Aneurysm is anterior to lower cranial nerves; (c) Left VA trunk is incorporated in aneurysm neck; (d-f) Angulated fenestrated clip clips aneurysm and encloses proximal PICA within fenestration

Table 4: Location of aneurysm on the PICA and the surgical approach adopted

| Location of aneurysm (n=20) | Surgical approach                        | Patient number |
|-----------------------------|------------------------------------------|----------------|
| VA/BA-PICA junction (5)     | Far lateral approach                      | 3              |
|                             | RMSO                                     | 2              |
| Anterior medullary (4)      | RMSO                                     | 2              |
|                             | Far lateral approach                      | 2              |
| Lateral medullary (3)       | RMSO                                     | 3              |
| Tonsillo medullary (4)      | RMSO                                     | 1              |
|                             | Midline suboccipital craniectomy         | 3              |
| Telovelotonsillar (4)       | RMSO                                     | 1              |
|                             | Midline suboccipital craniectomy         | 3              |
| Cortical (0)                | Midline suboccipital craniectomy         | 0              |

VA – Vertebral artery; BA – Basilar artery; PICA – Posterior inferior cerebellar artery; RMSO – Retromastoid suboccipital craniectomy
of mild alteration in the level of consciousness immediately following surgery and whose preoperative angiogram had shown vasospasm. Prior to institution of the triple H therapy, they underwent a CT scan/MRI to rule out an infarct, hematoma, increased postoperative edema or hydrocephalus.

Results

The patient outcome was assessed at discharge and at follow-up (range: 6 months-2.5 years) utilizing modified Rankin’s score (mRS) [Table 5].[10,11] Of the 15 patients in HandH grade I, 12 remained in mRS 0 (no symptoms); and one was in mRS 2 (mild disability, due to increased cerebellar signs in the form of appendicular ataxia and VIth nerve paresis). One patient in this group developed vertebral artery dissection with hemiplegia during the DSA. He had hemiparesis grade III at follow-up (mRS 4; moderately severe disability). Of the two patients in HandH grade II, one remained asymptomatic at follow-up (mRS 0). Among the three patients with HandH grade III, two improved to mRS 1 (no significant disability despite mild symptoms of cerebellar ataxia and occasional difficulty in swallowing at follow-up). Three patients (one each with preoperative modified HandH grade I, II, and III, respectively) expired in the perioperative period (mRS 6). A patient in HandH grade I had two aneurysms, a saccular aneurysm at the PICA origin and another at the BA-VA junction contralateral to the side of the PICA aneurysm. Following the uneventful clipping of the ruptured PICA aneurysm, this patient died suddenly in the intensive care unit probably because of rupture of the unclipped BA-VA aneurysm. The second patient with a VA-PICA junctional aneurysm and in preoperative HandH grade II developed patchy infarction in the MCA territory and increased lower cranial nerve paresis. He succumbed to aspiration pneumonitis and septicemia. Finally, a patient in HandH grade III had a VA-PICA junctional aneurysm and developed vasospasm leading to left cerebellar and right ACA territory infarction. He also developed cerebrospinal fluid leak through the operative wound and succumbed to meningitis. The postoperative DSA revealed non-visualization of the distal trunk of PICA in two patients, but fortunately without significant morbidity. In the first patient, a partially thrombosed aneurysm of the telovelotonsillar segment was successfully clipped and its luminal thrombus excised. In the second patient, an AVM was filling from the left PICA and draining into the transverse sinus. He also had two aneurysms present proximal to the AVM in the tonsillomedullary segment. Both the aneurysms were clipped and the AVM excised in the same setting.

Discussion

Pathogenesis

The size of saccular aneurysms of PICA that produced SAH ranged from 4mm to greater than 2.5cm. Some of them represented extremes of sizes of aneurysms that are usually not prone to bleeding at other locations. The complex and tortuous course of PICA with multiple acute angulations promoted increased luminal shear stress. This resulted in the development of aneurysms even where there were no visible bifurcation sites or branching vessels. Thus, these saccular aneurysms were on thinner and weaker vessel walls (commonly associated with fusiform and dissecting aneurysms) irrespective of whether the aneurysms were extremely small or giant.[5,12,13] They could, however, be adequately clipped (usually with parent vessel preservation) due to their saccular shape and a well defined neck. One of our patients had the unique presentation of two aneurysms on the parent vessel proximal to an arteriovenous malformation (AVM). This association has rarely been reported.[8,14-17] The risk of hemorrhage under these circumstances is high both from the AVM and the associated aneurysm.[8,14,15,17] Thus, they required simultaneous addressal during surgery in our patient with the aneurysms being treated before proceeding to the more distal AVM.[16,17]

Radiological features

Seventeen patients (85%) presented with intraventricular hemorrhage (IVH) that was associated nearly 50% of the time with cisternal blood. In the reported literature, the incidence of IVH due to PICA aneurysm rupture has ranged from 83-100%.[18-20] The high incidence of hydrocephalus in our patients (8 patients, 40%) was in all probability, consequent to the

| Preoperative score | Patient | Postoperative outcome and neurological deficits number (n=20) |
|--------------------|---------|----------------------------------------------------------|
| H and H I          | 15      | 1: Rankin 0: 1. Rankin 2: increased cerebellar S/S and VIth N paresis |
|                    |         | 1: Rankin 4: VA dissection during DSA caused persistent hemiplegia |
|                    |         | 1: Rankin 6: expired |
| H and H II         | 2       | 1: Rankin 0 |
|                    |         | 1: Rankin 6: expired |
| H and H III        | 3       | 1: Rankin 6: expired |
|                    |         | 2: Rankin 1: persisting cerebellar S/S and IX-X N paresis |
|                    |         | 1: Rankin 6: expired |

Table 5: Surgical outcome at discharge and follow up range of 6 months-2.5 years
frequent presence of intra-ventricular bleed. The cisternal hemorrhage, when present, was not entirely confined to the posterior fossa, but often extended to the supratentorial cisterns. It resulted in severe cerebral ischemia due to anterior circulation vasospasm in two patients. These cases exemplify the fact that a ruptured PICA aneurysm cannot be discounted while encountering a patient with an extensive supratentorial subarachnoid hemorrhage (SAH).

A unique subset of patients were those who had only IVth ventricular hemorrhage without any cisternal, cortical, or sulcal blood. This manifestation was due to seepage of blood through the foramen of Luschka or Magendie following aneurysmal rupture. These patients nearly always had distally located PICA aneurysms. One must be aware of this frequent association between a primary IVth ventricular hemorrhage and a distal PICA aneurysm in order not to miss these aneurysms that may rebleed later on if left uninvestigated and untreated. In two of our patients, the CTAs missed a PICA aneurysm that was later detected on a DSA. Aneurysms of the posterior fossa may be missed on a CTA due to the beam hardening effect in the region. An additional DSA is, therefore, mandatory before a PICA aneurysm may be unequivocally ruled out after a normal CTA. This is especially true when a high index of suspicion regarding the presence of a PICA aneurysm exists based upon the clinicoradiological manifestations. Numerous variations of PICA and its associated aneurysms were also evident in this series. Evaluation of the VA and the origin of PICA on both sides is useful in picking up additional aneurysms or anomalies in the vasculature on the side contralateral to the side of evident bleeding. This is especially important when perimedullary cisternal hemorrhage, IVH, hydrocephalus or unilateral or bilateral 6th nerve paresis are encountered in the clinical setting of SAH. This is also important when perimedullary cisternal hemorrhage, IVH, hydrocephalus or unilateral or bilateral 6th nerve paresis are encountered in the clinical setting of SAH. These aneurysms may occasionally assume giant proportions mimicking a tumor or a large cavernous angioma. They may also thrombose spontaneously causing significant brain stem compression. This was seen in one of our patients. The laminated appearance of the thrombus in various stages (the target sign) within the lumen of the lesion pointed towards the existence of an aneurysm rather than a tumor. An MRA and DSA further ruled out partial filling of this giant aneurysm. Removal of the clot, clipping of its neck and excision of its fundus relieved the brain stem distortion.

Surgical nuances

While planning the surgical trajectory, the location of the aneurysm based upon the segmental anatomy of PICA; its relationship to the anatomical midline (10 mm or less being considered significant), brainstem and cerebellum; and, the variability in the vessel’s origin should be carefully assessed. The brain-stem and cranial nerves may be compromising the surgical view to these aneurysms. Moreover, one or more parent vessels at the VA-BA junctional region may be ending into or taking origin from the neck of this aneurysm. Application of clip may cause narrowing or twisting of these vessels leading to luminal compromise.

In VA-BA-PICA junctional and anterior medullary aneurysms situated close to midline anterior to the brain-stem, the far lateral or extreme lateral transcondylar approach may be utilized. Removal of the posterior condyle; C1 lateral mass; and/or the jugular tubercle help in widening the surgical corridor. In those patients in whom far lateral approach was used, control of the proximal ipsilateral VA was obtained in its extradural portion in the suboccipital triangle after its emergence from C1 foramen transversarium. The VA has a fairly constant and well-localized position while traversing over the C1 posterior arch prior to its entry into the dura. A tortuous VA with a high shoulder may be exposed from the contralateral “aneurysmal dome” side or by using the translabyrinthine approaches.

PICA usually arises dorsal or lateral to the aneurysm; occasionally, however, it may be posterior or medial to its neck. The applied clip blades are usually pointing forwards, distal to VA-PICA junction parallel to the long axis of VA. Fenestrated clips may protect lower cranial nerves or the origin of PICA. Most of these anterior aneurysms are located ventral to lower cranial nerves. While traversing the plane of these nerves, an occasional patient may develop dysphagia, rhinolalia, hoarseness, nasal regurgitation or aspiration pneumonia related to their functional impairment even when the arachnoidal plane has been meticulously preserved. The incidence of lower cranial nerve paresis following clipping of proximal PICA aneurysms has been reported to be varying between 10-45%; in our series, it was seen in 3 (15%) patients. The related morbidity was fortunately self-limiting and usually improved in approximately three to six months. However, occasionally, lower cranial nerve paresis may lead to disastrous consequences particularly in moribund patients with poor preoperative HandH scores. The definite risk factors for the development of nosocomial pneumonia (that carries a mortality rate of 20-50%) are low nutritional status, lower Glasgow Scale Score, vertebrobasilar stroke, and the need for mechanical ventilation.

We concede that an endovascular intervention may have provided similar good results. These patients, when given an option between surgical clipping and endovascular approach chose the former due to the lesser expense of the surgical procedure. A complication rate of as high as 13% has been reported with embolization of PICA aneurysms. As is well illustrated in this study, PICA often has extremely variable and tortuous course with multiple anomalies and variability in position of the aneurysm relative to the brain-stem. Superselective catheterization of the PICA is often not feasible or may be associated with complications during the endovascular procedure as exemplified by our patient in whom vertebrobasilar dissection occurred during catheterization.
Identification of the neck and selective catheterization may be difficult or impossible especially in peripheral aneurysms. When aneurysms are small, the risk of abrupt “jumping” of the catheter or guide wire into the sac increases the chances of its rupture. Moreover, if the microcatheter tip does not conform favorably to the anatomy, the system may be very unstable and further coils may migrate or displace the catheter into the parent vessel. It is apparent from this study that the mortality was related more to vasospasm as a result of subarachnoid hemorrhage rather than due to technical aspects of surgery. Thus, surgeons have a responsibility to be well-versed with the surgical approaches and nuances for clipping these aneurysms in case the endovascular treatment fails or is not possible; or, if salvage surgery is required for SAH occurring during the interim period between the endovascular coiling and the development of luminal thrombosis within the aneurysm.

Limitations
The medical personnel assessing outcome were not blinded to the initial clinicoradiological diagnosis and were participating in the care of the patients. The subjects included patients with saccular aneurysms amenable to surgical clipping; fusiform or dissecting PICA aneurysms do not form a part of the study. Trapping of PICA with occipital artery-PICA anastomosis and excision of aneurysm and end-to-end anastomosis were not performed in any of the patients including the two in whom surgical clipping of the PICA aneurysm was associated with non-visualization of the parent blood vessel on postoperative DSA.

Conclusions
This series presents the surgical experience in dealing with both proximal and distal, ruptured, saccular PICA aneurysms based on the anatomical segments of PICA. The patients often presented late with the majority being in a good preoperative HandH grade; this may be a major factor in the usually gratifying postoperative outcome seen. The presence of SAH in the supratentorial cisterns does not rule out the existence of a PICA aneurysm. Unilateral or bilateral VIth nerve palsy, intraventricular hemorrhage and hydrocephalus in the clinical setting of SAH mandates a four-vessel (rather than the conventional three-vessel) angiogram to assess the anatomy of VA-PICA complex on both sides especially since PICA had significant variations and is associated with numerous anomalies. VA-PICA junctional aneurysms require special surgical considerations based upon tortuosity of the main vessels and distance from the midline.

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