Original Article

Spectrum of Posterior Cerebral Artery Dissection: A Retrospective Observational Study and a Critical Review

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Abstract

Background and Aims: Intracranial arterial dissections commonly involve the vertebrobasilar system leading to subarachnoid hemorrhage (SAH) or cerebral infarction attributable to a dissecting aneurysm of the vessel or occlusion of the lumen depending on the depth of dissection. However, isolated posterior cerebral artery dissections (PCADs) are rare and sparsely reported in the literature. **Methodology:** A retrospective multicentric observational study was carried out after collecting data from 14 patients admitted with PCAD in three hospitals of Kolkata, Jaipur, and Patna within the period of July 2021 to June 2022. **Results:** The median age of the population was 48.5 years, and 64.28% were females. SAH was the most common presentation with dissecting aneurysms in all patients barring one, who presented with a left occipital infarct consequent to ipsilateral PCAD. Among the 14 patients, three patients denied endovascular intervention and were lost to follow-up; one patient with an occipital infarct and another patient with a dissecting left P3 aneurysm, which underwent spontaneous thrombosis, were managed conservatively. Among the nine patients scheduled for endovascular coiling, one patient succumbed before intervention and one patient succumbed to sepsis in the postoperative period. A complete recovery was noted in six patients, whereas residual neurodeficits were present in three patients. Among the six patients who had an uneventful recovery at the end of 3 months, five patients had an endovascular intervention. **Conclusion:** PCAD may present with large-scale neurodeficits and is associated with high morbidity and mortality, hence necessitating prompt management. Conservative management is preferable for consequent infarcts, whereas endovascular management is desirable in cases of dissecting aneurysms, which usually tend to have a favorable outcome if intervened early.

Keywords: Dissecting aneurysm, endovascular intervention, ischemic stroke, posterior cerebral artery dissection, subarachnoid hemorrhage

Introduction

Extracranial and intracranial arterial dissections are considered as one of the most important causes of stroke in young population. Spontaneous intracranial artery dissections tend to cause cerebral infarction and subarachnoid hemorrhage (SAH) in the young and middle-aged population. The carotid and vertebral arteries in the neck are the most susceptible sites for extracranial dissection, while among intracranial arteries, dissections commonly involve the vertebrobasilar system.[1] Intracranial arterial dissections may lead to aneurysmal dilation of the vessel or may occlude the vessel lumen depending upon the depth of the dissection into the vessel wall. However, isolated dissections involving the posterior cerebral arteries (PCAs) are extremely rare and sparsely reported in the literature, but warrant accurate diagnosis to necessitate appropriate management at the earliest. The authors present a series of PCA dissections (PCADs), which depicts the opposite spectrum of presentation and management.

Methodology

A retrospective multicentric observational study was carried out among patients presenting with PCAD at Bangur Institute of Neurosciences, IPGMER and SSKM Hospital Annex-1, Kolkata, SMS Medical College, Jaipur, and GS Neuroscience Clinic and Research Centre, Patna, within the period of July 2021 to June 2022. The authors collected data from the existing hospital records, which revealed that a total of 14 patients were diagnosed with PCAD in the specified study period. The recorded data of these 14 patients were secondarily analyzed and tabulated, and a detailed assessment was carried out. The etiological workup for PCAD was extensively performed in these patients ranging from relevant clinical examination (to negate any stigmata of connective tissue and collagen vascular disease, systemic infections) to biochemical investigations (antinuclear antibody panel, antineutrophil

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cytoplasmic antibodies, prothrombotic panel) and radiological investigations (angiogram of renal vessels and arch of aorta, magnetic resonance imaging (MRI) of cranio-cervical junction, MR vessel wall imaging, echocardiography). The collected data were followed by a comparison with the existing literature. Since the spectrum of PCAD is diverse and intriguing, we looked up the literature available on MEDLINE, PubMed, and other databases using MeSH terms such as "Posterior cerebral artery dissection" for information and review.

Results

The median age of the population was 48.5 years (IQR 24), and the majority of them were females (64.28%). Three patients were hypertensives on irregular medications and one patient had poorly controlled diabetes, while another patient had both diabetes and hypertension and was on regular medications. Twelve patients had a headache as their initial presentation, among which nine patients reported a thunderclap headache. Three patients had left hemiparesis, and two of them had concomitant visual dysfunction. SAH was the most common finding on brain imaging seen in 11 patients (78.57%). Four patients had left PCAD, while the remaining 10 patients had right PCAD. Dissecting aneurysm of PCA was noted in all patients barring one who developed a right hemianopia consequent to a left occipital infarct attributable to ipsilateral PCAD.

Among the 14 patients, one patient with an occipital infarct and another patient with a dissecting left P3 aneurysm, which underwent spontaneous thrombosis, were managed conservatively. The remaining patients were offered endovascular intervention. Three patients denied any endovascular intervention, and one patient succumbed before his scheduled flow diversion procedure. Endovascular coiling was performed in the remaining eight patients, of which one patient succumbed to sepsis in the postoperative period. Among the six patients who had an uneventful recovery at the end of 3 months, five patients had an endovascular intervention. Three patients who denied intervention were lost to follow-up, and residual neurodeficits were noted in the remaining three patients at the end of 3 months. The demographic details, clinical profile, neuroimaging, management, and outcome of these patients are presented in Table 1. The authors hereby describe the first three cases to give a comprehensive view on the opposing spectrum of presentation of PCAD with highlights on management and outcome.

Case 1

A 42-year-old man without any comorbidity presented with a thunderclap headache followed by right hemianopia a day before admission. The headache was predominantly occipitonuchal and gradually progressed to holocranial distribution. He was nauseous and the headache was continuous, neither relieved at any position nor with analgesics. He did not have any motor or sensory deficit, and no history suggestive of any cranial nerve dysfunction or alteration of bladder and bowel habits was obtained. There was no preceding history of any trauma, diarrhea, or vomiting. History of past illness and family history were noncontributory. On examination, he had a right congruous homonymous hemianopia with macular sparing in the absence of any other focal neurodeficits. Brain imaging revealed a hyperdense blot in the left ambient cistern with a well-demarcated infarct in the left posterior cerebral arterial territory [Figure 1a]. An interval MRI revealed an iso-hypointense globular structure in the left crural cistern with associated hypodensity in the distribution of left PCA territory suggestive of dissected left PCA (P3) segment resulting in infarction [Figure 1b-d]. MR angiography time of flight (TOF) sequence suggested faint visualization and cutoff of the left PCA beyond the distal P2 segment [Figure 1e], which was further confirmed by a subsequent digital subtraction angiography (DSA) [Figure 1f]. He was managed conservatively and had persistent hemianopia on follow-up after three months.

Case 2

A 36-year-old healthy man without any preceding trauma presented with a left-sided hemiparesis for three days before admission preceded by a thunderclap headache. The headache was holocranial in distribution, increased with sitting up from a supine position, and associated with bouts of vomiting. An emergent brain imaging revealed diffuse SAH with right upper quadrant midbrain hematoma with an elliptical hyperdensity possibly representing the dissected segment of right PCA [Figure 2a-c]. A three-dimensional rotational CT angiography revealed a dissection of right PCA with a pseudoaneurysm at the junction of P1–P2 segments [Figure 2d-f]. The patient was scheduled for a flow diversion procedure, but unfortunately the patient succumbed after a day of hospitalization.

Case 3

A 47-year-old woman without any history of trauma was rushed to a tertiary care hospital with a thunderclap headache with repeated bouts of vomiting for 6 hours. The pain was predominantly occipitonuchal in distribution and radiated along the nape of her neck. She did not have any focal neurodeficit, and an emergent brain imaging revealed a crescent-shaped hyperdensity in the anterior aspect of pons and hyperdensity in peripontine cistern suggestive of suspected aneurysm with subarachnoid hemorrhage [Figure 3a]. A wide-necked bilobed aneurysm involving the right P1 and basilar top junction was detected in DSA, which was posted for a stent-assisted endovascular coiling using coils of decremental size [Figure 3b and c]. Late angiographic runs revealed tight filling of the aneurysm without any prolapse of the coil mass [Figure 3d]. The patient had an uneventful recovery and was discharged after 3 days.

Discussion

Intracranial arterial dissections are less commonly encountered than extracranial dissections. Intracranial dissections may

Table 1: Spectrum of patients presenting with posterior cerebral artery (PCA) dissection in this series

Case	Age (years)	Sex	Comorbidities	Presentation	Etiological workup (clinical/biochemical/imaging)	Imaging	DSA	Treatment	Outcome at 3 months
1	42	M	Nil	Thunderclap headache followed by right hemianopia	Marfanoid habitus	Occipital lobe infarction and subsequent gliosis	Left PCA (P3) dissection	Conservative	Persistent hemianopia
2	36	M	Nil	Thunderclap headache followed by left hemiparesis	Undetected	SAH with brainstem bleed	Right P1-P2 junction dissecting aneurysm	Scheduled for flow diversion	Succumbed before intervention
3	47	F	Nil	Thunderclap headache	Undetected	SAH with aneurysm in the anterior part of pons	Right P1 dissecting aneurysm	Coiling using a modified assisted approach using two microcatheters	Complete recovery
4	58	M	Hypertensive	Thunderclap headache	Hyperhomocysteinemia	SAH	Left P3 dissecting aneurysm with left anterior choroidal aneurysms	Conservative; spontaneous thrombosis of both aneurysms within 3 days	Complete recovery
5	33	F	Nil	Thunderclap headache	Undetected	SAH with midbrain compression	Right P2-P3 junction dissecting aneurysm	Coiling	Complete recovery
6	66	F	Diabetic, hypertensive	Holocephalic headache	Undetected	SAH	Right P2 and P3 large elongated dissecting aneurysms	Denied intervention	Lost to follow-up
7	59	F	Nil	Headache	Hyperhomocysteinemia	SAH	Right P3 dissecting aneurysm	Denied intervention	Lost to follow-up
8	66	F	Diabetic	Thunderclap headache	Undetected	SAH	Right P2 dissecting aneurysm	Coiling	Complete recovery
9	68	F	Hypertensive	Left hemiparesis and left hemianopia	Undetected	Right midbrain bleed	Right P1 dissecting aneurysm	P1 occlusion by coils	Residual left hemiparesis and hemianopia
10	55	F	Nil	Thunderclap headache	Undetected	SAH	Right P3 dissecting aneurysm	P3 occlusion by coils	Succumbed
11	30	M	Nil	Left hemiparesis and binocular horizontal diplopia	Marfanoid habitus	Right midbrain bleed	Right P1 dissecting aneurysm	Flow diverter-assisted coiling	Residual left hemiparesis
12	22	F	Nil	Postpartum thunderclap headache	Undetected	SAH, perimesencephalic	Left P2 dissecting aneurysm	Coiling	Complete recovery
13	50	F	Hypertensive	Headache	Undetected	SAH	Left P2 dissecting aneurysm	Denied intervention	Lost to follow-up
14	35	M	Nil	Thunderclap headache	Hyperhomocysteinemia	SAH	Right P2 dissecting aneurysm	Coiling with glue embolization	Complete recovery

DSA-digital subtraction angiography, SAH-subarachnoid hemorrhage

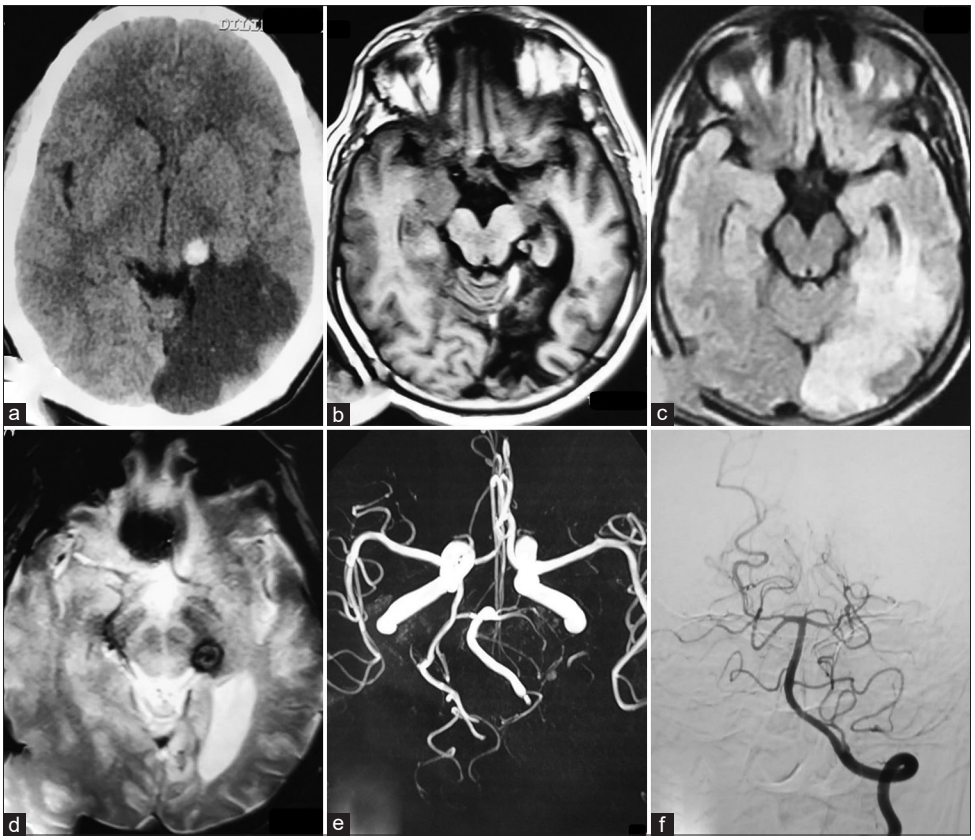

Figure 1: Nonenhanced brain CT (axial section) showing a hyperdense blot in the left ambient cistern with a well-demarcated hypodensity conforming to the left posterior cerebral artery (PCA) territory, suggestive of an infarct (a); interval nonenhanced magnetic resonance imaging (MRI) scan T1 axial sequence revealing an iso-hypointense globular structure in the left crural cistern with associated hypodensity in the distribution of left PCA territory (b); interval nonenhanced MRI FLAIR axial sequence showing hyperintensity in the same distribution (c); interval nonenhanced MRI GRE sequence revealing an ovoid hypointense signal change. The preliminary CT head and the interval T1 and GRE sequences most likely represent the dissected left PCA (P3) segment, which resulted in the PCA infarction (d); MR angiography time of flight (TOF) sequence revealed faint visualization and cutoff of the left PCA beyond the distal P2 segment (e); left vertebral digital subtraction angiography (DSA) confirming the MRI TOF imaging findings (f)

lead to occlusion of the arteries, which may not be recognized as dissections in the absence of any precipitating event and in children and young adults. The anterior circulation is predisposed to dissection in young adults compared to posterior circulation, which is a common site in the elderly.[2] Intracranial dissections may lead to cerebral ischemia as a consequence, whereas in extracranial carotid and vertebral artery dissections artery-to-artery embolism is considered the most probable mechanism of cerebral ischemia. The course of the large arteries within the subarachnoid space is usually invested with pain-sensitive nerve endings, and hence, any arterial dissection is associated with excruciating pain. Extracranial dissections of carotid and vertebral arteries are usually preceded by a sudden vigorous motion of the head and neck, while physical perturbations are less likely to cause intracranial dissections.[3]

Etiology

The cause of intracranial arterial dissections and dissecting aneurysms remains undetectable in majority of the cases. Various degrees of trauma with or without predisposition may lead to intracranial dissections. The trauma or triggering events may not always be remembered. In this study, none of the cases had antecedent trauma, and hence, all of them were considered spontaneous dissections. Connective tissue disorders, such as fibromuscular dysplasia, Marfan syndrome, Ehlers–Danlos syndrome, osteogenesis imperfecta, homocytinuria, cystic medial necrosis, and alpha-1 antitrypsin deficiency, have been associated with intracranial dissections.[4] A number of other conditions, such as systemic infections, hypertension, hyperhomocysteinemia, smoking, oral contraceptive use, and hypercoagulable states, have also been associated with intracranial dissections.[5] Hypertension is common in patients with extracranial carotid dissection in contrast to PCAD. In this study, only two patients had marfanoid habitus (cases 1 and 11) and three patients had serum hyperhomocysteinemia (cases 4, 7, and 14). Genetic analysis (clinical/whole-exome sequencing) could not be performed in these patients due to financial constraints.

Presentation

Vertebrobasilar dissections are commoner in males, while PCADs have a male–female ratio of 1.12:3.12.[6] PCAD presents commonly with moderate-to-severe headache and thunderclap at times predominantly in the occipitonuchal distribution.

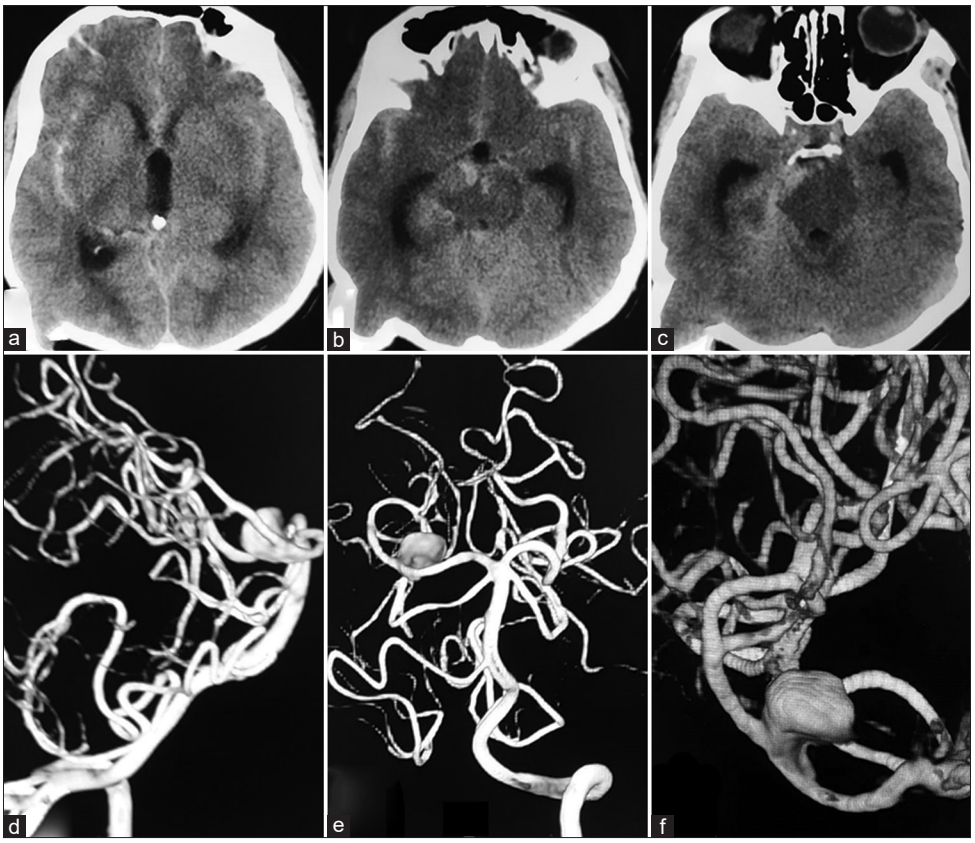

Figure 2: Non-contrast axial CT head revealing diffuse subarachnoid hemorrhage (a); non-contrast axial CT head revealing an elliptical hyperdense signal intensity occupying the right upper quadrant of midbrain, possibly representing the dissected segment (b); preferential distribution of subarachnoid hemorrhage in the right basal cistern having localization value (c). Both B and C have associated features of early hydrocephalus; 3D rotational angiography (3DRA) of left vertebral artery (LVA) profiling the dissected pseudoaneurysm in lateral view (d); 3DRA of LVA profiling the dissected pseudoaneurysm in AP view (e); 3DRA of LVA profiling the dissected pseudoaneurysm in the broad base and its location in the P1–P2 junction (f)

Dissecting intracranial aneurysms may lead to SAH or cerebral infarcts or both with variable neurodeficits.[7] Isolated PCADs commonly occur near P1–P2, lead to infarction, and have a relatively better prognosis.

Pathophysiology

Isolated PCADs most commonly occur near the P1–P2 junction, which is close to the free border of the tentorium cerebelli. Intracranial dissections occur between the intima and the media in comparison with extracranial dissections, which occur in the outer layers of the tunica media or between the adventitia and media. In contrast to extracranial arteries, intracranial arteries lack an external elastic membrane and have thin tunica adventitia and few elastic fibers in tunica media along with thick internal elastic lamina. These changes are more pronounced in the base of the skull for carotid arteries and 1 cm proximal to the dural perforation of the vertebral artery into the base of the skull. Focal stenosis in intracranial dissection and pseudoaneurysm in extracranial dissection may be attributable to these anatomical and ultrastructural variations. Moreover, high flow states, such as arteriovenous malformation, have been linked to the formation of aneurysms post-dissection.[8] After dissection, an intramural hematoma usually tracks between the internal elastic lamina and the tunica media with consequent mass effect on the lumen leading to focal stenosis with or without post-stenotic dilation.[9] However, in some instances intracranial arterial dissections may involve the subadventitia and consequently lead to subarachnoid hemorrhage. In extracranial dissection, the hematoma tracks between the media and adventitia, leading to compression of the vessel lumen and subsequent pseudoaneurysm formation. Head trauma may lead to shear injury of the vessels, especially along the edges of the tentorium and subsequent traumatic dissecting aneurysms. None of the patients in this series had an antecedent history of trauma.

Imaging

Digital subtraction angiography reveals narrowing of the vascular lumen, which may be irregular/asymmetric with or without dilation proximal or distal to the stenosis giving a string and pearl appearance. The pathognomonic double-lumen sign represents antegrade flow in both true and false lumens, separated by an intimal flap.[10] Stenotic lesions predominantly manifest ischemic symptoms compared with dilated lesions, which usually present with hemorrhage. A baseline non-contrast CT scan followed by a brain MRI is usually required to evaluate the stroke as per the requirement. The MR angiography, though useful, is limited by motion artifact and

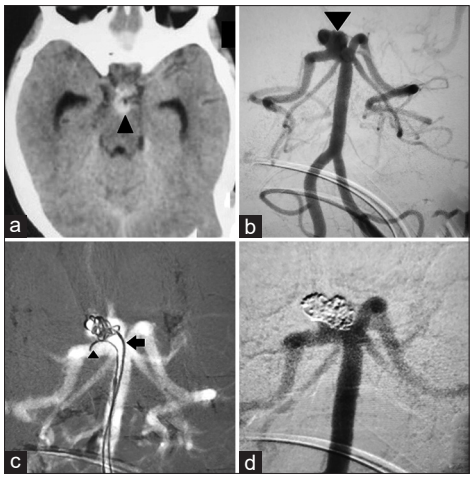

Figure 3: Brain CT shows crescent-shaped hyperintensity in the anterior aspect of pons and hyperintensity in peripontine cistern suggestive of basilar tip aneurysm with subarachnoid hemorrhage (a); digital subtraction angiography after vertebral artery injection showing broad-based dissected pseudoaneurysm in P1 segment (b); DSA roadmap showing two microcatheter, through one of them a microwire is hinged at right P1 segment (black arrowhead) and the other being used for coiling of the dissecting aneurysm (black arrow) (c); final check DSA showing near total obliteration of the aneurysm with patent right posterior cerebral artery (d)

small caliber of the intracranial vessels in which vessel wall irregularity or narrowing is often nonspecific. T1-weighted imaging shows hyperintensity within the dissected vessel wall and varies with the chronological age of the bleed.

Intracranial atherosclerotic disease vs intracranial dissection

Intracranial atherosclerotic disease (ICAD) may mimic intracranial dissections; however, the two entities may be differentiated by distinct imaging characteristics. ICAD refers to atherosclerosis of large intracranial arteries with focal stenotic segments revealing calcified or noncalcified atheromatous plaque with or without intraplaque hemorrhage. Although DSA serves as the gold standard investigation to determine the severity of stenosis in ICAD, it is unable to delineate the plaque morphology compared with MR angiogram (MRA) or CT angiogram (CTA).[11] ICAD tends to be multifocal and generalized, whereas multifocal dissection is relatively rare. The presence of intimal flap, double lumen, crescent sign, intravascular hematoma, or aneurysms in CTA or contrast-enhanced MRA serves as imaging indicators for intracranial dissections compared with plaques, eccentric thickening, and positive/negative remodeling in high-resolution MR vessel wall imaging in ICAD.[12]

Management

There is no universal consensus regarding the use of antiplatelets or anticoagulation in intracranial dissections; however, antiplatelets are preferred compared with anticoagulants due to the potential risk of intramural hematoma expansion and rupture.[13] Patients with resolving neurodeficits without any aneurysmal dilation may be managed conservatively. Adequate blood pressure optimization is essential to prevent the progression of the dissection and subsequent ischemia or hemorrhage. Dissecting aneurysms with SAH are prone to rebleed and associated with a high mortality rate necessitating early definitive management. Surgical or endovascular intervention is required in patients with dissecting aneurysms and consequent SAH.[14] Surgical intervention encompasses excision of the aneurysm, proximal ligation, reinforcement, and an intracranial–extracranial bypass as per the requirement.[15] The prognosis after neurosurgical intervention of intracranial aneurysms involving the posterior circulation is relatively dismal than that of anterior circulation, especially in ruptured aneurysms.[16] Expanding intramural hematoma or expanding dissecting aneurysm requires urgent surgical intervention. However, spontaneous recovery with fusiform dissecting aneurysms and subsequent recanalization of PCA have been reported in the literature.[17]

Endovascular treatment has been preferred as the management modality of choice in posterior fossa intracranial aneurysms.[18] Endovascular management includes flow diversion or therapeutic occlusion by using detachable microcoils with or without balloons.[19] A definitive endovascular approach to treat dissecting PCA aneurysms is to occlude the parent artery in aneurysms distal to P2 segment, as sacrificing the parent vessel in more proximal lesions may lead to large neurodeficits. A favorable outcome has been reported in the literature with proximal artery occlusion (PAO) with minimal chance of rebleeding or subsequent ischemic complications, but angiographic recurrence has been reported considerably.[20] However, if a large impending deficit is expected from occlusion of the parent artery, or migration of thrombus to the basilar tip is a concern, treatment modalities without occluding the parent artery may be considered.[21] Partial coiling of dissecting aneurysms may be an option, but prognostically dismal due to continuous growth and risk of rebleeding. Procedural complications encompass the inability to occlude the entire dissected lesion and consequent coil compaction and placement of microcatheter into the false lumen. Endovascular occlusion includes placement of the permanent occlusion device at the site of dissection in the true lumen followed by the collapse of the false lumen. Recently, the use of flow diversion has been rapidly adapted due to its ability to reconstruct the neck of large intracranial aneurysms, the outcomes being favorable. A prompt antiplatelet therapy needs to be ensured, the feasibility of which may be questionable in acute ruptured aneurysms.[22]

In this study, flow diversion was planned for cases where distal arteries were patent and supplying eloquent structures. In cases with distant vessel occlusion, unfavorable morphology, and good collateral supply, vessel sacrifice by endovascular coiling was planned.

Conclusion

PCAD is relatively less common, but may present with large-scale neurodeficits and is associated with high morbidity

and mortality, hence necessitating prompt management. PCAD may present with infarct or SAH with a dissecting aneurysm, and symptoms may evolve over days in a few cases. Conservative management is preferable for ischemic symptoms, whereas endovascular management is desirable in cases of dissecting aneurysms, which usually tend to have a favorable outcome if intervened early.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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