GMR
The Original

COMPLEX RIGHT ICA ANEURYSM WITH COLLATERAL REFORMATION: A CASE REPORT

Dr Jemi Daffodil S[1], Dr.Premraj. T[2*]

[1]PG Resident, Dept of Radiology, Sree Balaji Medical College & Hospital, Chennai, Tamil Nadu, India, Jemidaffodil0211@ Gmail.com
[2*]Assistant Professor, Dept of Radiology, Sree Balaji Medical College & Hospital, Chennai, Tamil Nadu, India, Premraj1611medico@gmail.com, Orcid id: 0009-0003-2268-2735

ABSTRACT
A 34-year-old female presented for evaluation of an incidentally detected intracranial aneurysm on CT cerebral angiography. Imaging revealed a complex aneurysm involving the supraclinoid segment of the right internal carotid artery (ICA), consisting of a fusiform dilatation with peripheral wall calcification and an associated saccular component measuring 5.5×7.0×6.1 mm. The M1 segment of the right middle cerebral artery (MCA) was markedly attenuated, yet the distal MCA branches (M2–M4) were opacified, likely via collateral circulation. The A1 segment of the right anterior cerebral artery (ACA) was reduced in calibre (possible hypoplasia). Venous anomalies including hypoplasia of the left transverse and sigmoid sinuses were also noted. There was no evidence of subarachnoid or intraparenchymal hemorrhage, thrombus, or arteriovenous malformation. This case highlights a complex, unruptured aneurysm in a young adult, with associated vascular anomalies and collateral-dependent distal perfusion. Given the absence of hemorrhage and the high surgical risk, conservative management with rigorous blood pressure control, lifestyle modification, and serial imaging surveillance was instituted. The case underscores the importance of individualized decision-making in complex cerebrovascular lesions.

KEYWORDS: Complex aneurysm, fusiform aneurysm, saccular aneurysm, internal carotid artery, collateral circulation, conservative management

INTRODUCTION
Intracranial aneurysms are pathological dilations of cerebral arteries that carry a risk of rupture, leading to subarachnoid hemorrhage (SAH) with high morbidity and mortality [1]. The prevalence of unruptured intracranial aneurysms in the general population is approximately 3-5%, with most being saccular (berry) aneurysms [2]. Fusiform aneurysms, characterised by circumferential dilatation of the arterial wall, are less common, accounting for fewer than 10% of all intracranial aneurysms, and often involve the vertebrobasilar system or the internal carotid artery [3].
Complex aneurysms – those with a combination of fusiform and saccular morphology, or those incorporating major branch vessels, present unique therapeutic challenges [4]. In young adults, such lesions may arise from congenital vessel wall weakness, haemodynamic stress, or underlying arteriopathies [5]. The supraclinoid segment of the ICA is a critical location because it gives rise to the posterior communicating artery (PCOM), anterior choroidal artery, and the terminal bifurcation into the MCA and ACA [6].
Management decisions for unruptured complex aneurysms require weighing the risk of rupture against the risks of intervention (microsurgical clipping, endovascular coiling, flow diversion) [7]. In select cases, particularly when the aneurysm morphology is unfavourable or when distal perfusion is collateral-dependent, conservative management with risk factor modification and serial imaging may be preferred [8]. This report describes a case of a complex right ICA aneurysm with associated hypoplasia of adjacent vessels and venous anomalies, managed conservatively after a multidisciplinary review.

Case Presentation
A 34-year-old female, Gayathri, with no prior known medical comorbidities, underwent a CT cerebral angiogram as part of a work-up for recurrent non-specific headaches. There was no history of hypertension, diabetes mellitus, smoking, or substance abuse. Family history was negative for intracranial aneurysms or subarachnoid hemorrhage. Neurological examination was unremarkable, with no focal deficits, normal cranial nerves, and intact higher mental functions.
The CT angiogram (axial study of brain and neck) revealed a normal aortic arch and normal left carotid system. On the right side, a fusiform dilatation with peripheral wall calcification was noted involving the supraclinoid segment of the right ICA, extending into the proximal right posterior communicating artery. Arising from the terminal part of this dilated segment, at the level of the ICA bifurcation, was a focal saccular outpouching measuring approximately 5.5 × 7.0 × 6.1 mm (AP × TV × CC). There was no filling defect or thrombus within the aneurysmal sac, and no evidence of subarachnoid or intraparenchymal hemorrhage.
The M1 segment of the right middle cerebral artery (MCA) was markedly attenuated in calibre. However, the distal M2, M3, and M4 segments of the right MCA were opacified, described as “likely due to collateral reformation”. The A1

segment of the right anterior cerebral artery (ACA) appeared reduced in calibre (possibly hypoplastic), while the A2–A4 segments of the right ACA and the anterior communicating artery were normal. The left ACA, left MCA, bilateral posterior cerebral arteries, basilar artery, and bilateral vertebral arteries were all normal.
Venous evaluation showed hypoplasia of the left transverse and sigmoid sinuses, with otherwise normal dural venous sinuses. No aneurysm or arteriovenous malformation was identified. The impression was a complex aneurysm involving the terminal/supraclinoid right ICA, consisting of both fusiform aneurysmal dilatation and a saccular component, resulting in marked attenuation of the right M1 segment with distal MCA filling via collaterals, and a reduced-calibre right A1 (hypoplastic).

Imaging Findings in Detail
A CT cerebral angiogram was performed. Figure 1 shows an axial view demonstrating fusiform dilatation with peripheral wall calcification involving the supraclinoid segment of the right internal carotid artery (ICA). The dilatation measures approximately 5.5 × 7.0 × 9.3 mm (AP × TV × CC) and arises from the terminal part of the dilated supraclinoid segment at the level of the ICA bifurcation. No filling defect or thrombus is seen within the aneurysmal sac, and there is no evidence of subarachnoid or intraparenchymal hemorrhage.
Figure 2 illustrates the extension of this fusiform dilatation to involve the proximal part of the right posterior communicating artery. Peripheral wall calcification remains evident along the dilated segment. Figure 3 shows the marked attenuated calibre of the M1 segment of the right middle cerebral artery (MCA), a consequence of the mass effect or flow diversion by the adjacent complex aneurysm. Figure 4 (starred) reveals that despite the attenuated M1, the distal M2, M3, and M4 segments of the right MCA are opacified likely due to collateral reformationfrom leptomeningeal anastomoses. No other vascular anomalies were noted apart from the described findings. The venous sinuses were unremarkable.

DISCUSSION
This case illustrates a complex, unruptured aneurysm in a young adult with several notable features. First, the combination of fusiform and saccular morphology is atypical; most intracranial aneurysms are purely saccular. Fusiform aneurysms are more common in the posterior circulation and are often associated with atherosclerosis or dissection [3]. In a 34-year-old without risk factors, congenital vessel wall weakness (e.g., from fibromuscular dysplasia or Ehlers-Danlos syndrome) should be considered, though no stigmata were clinically evident.
Second, the collateral-dependent MCA circulation is striking. The marked attenuation of M1 suggests that the aneurysm has effectively "stolen" flow, yet the patient remains neurologically intact because of robust leptomeningeal collaterals from the ACA and/or PCA. This finding has direct implications for treatment: any intervention that compromises the aneurysm or the parent artery risks causing distal ischemia unless collaterals are preserved.
Third, the absence of hemorrhage and the chronic appearance (calcification) favour a conservative approach. The annual rupture risk for unruptured intracranial aneurysms depends on size, location, morphology, and patient factors. For small (<7 mm) saccular aneurysms of the ICA, the rupture risk is very low (0.1-0.5% per year) [1]. However, fusiform morphology and the presence of a daughter sac may increase risk [7]. Nevertheless, the lack of symptoms and the high surgical risk of treating a complex aneurysm at the ICA bifurcation (which involves both MCA and ACA origins) lead most neurovascular teams to recommend conservative management with close follow-up [8].

Conservative Management
For this patient, a multidisciplinary team recommended conservative management based on the unruptured status, small saccular component (<7 mm), complex fusiform morphology at the ICA bifurcation, and the presence of collateral-dependent MCA perfusion – all of which make surgical or endovascular intervention high-risk. The primary goal is to reduce the risk of aneurysm rupture and prevent progression. Strict blood pressure control is the cornerstone, targeting <130/80 mmHg through lifestyle measures (low-sodium diet, regular aerobic exercise, stress reduction, adequate sleep) and, if necessary, low-dose antihypertensive therapy such as amlodipine or lisinopril. Lifestyle modifications also include avoidance of heavy lifting, straining (Valsalva manoeuvres), smoking (patient is already a non-smoker), and excessive alcohol intake. No antiplatelet agents (aspirin, clopidogrel) are prescribed because they may increase bleeding risk if rupture occurs, unless another compelling indication arises.
Serial imaging surveillance is essential: follow-up with MRA or CTA at 6 months, then annually for two years, and subsequently every 1-2 years if the aneurysm remains stable. Any growth of the saccular component (>1 mm) or new symptoms would prompt re-evaluation for intervention. Patient education includes recognition of sentinel headache (sudden severe "thunderclap" headache), new ptosis, diplopia, or focal neurological deficits, with an emergency action plan. Genetic counselling is offered to screen for underlying connective tissue disorders (e.g., Ehlers-Danlos type IV, Loeys-Dietz syndrome) given the patient's young age and complex vascular morphology. This conservative approach aligns with current AHA/ASA guidelines, which favour observation for small, unruptured, asymptomatic aneurysms in favourable locations. This conservative approach aligns with current guidelines from the American Heart Association/American Stroke Association, which recommend observation for small (<7 mm), unruptured, asymptomatic saccular aneurysms in favourable locations, especially in patients without prior SAH [7].

CONCLUSION
This case of a complex right ICA aneurysm (fusiform + saccular) with attenuated M1 and collateral-dependent MCA perfusion in a 34-year-old female highlights the importance of detailed vascular imaging and individualized management. Conservative management, including blood pressure control, lifestyle measures, and serial imaging, is a safe and

appropriate strategy in the absence of hemorrhage or high-risk features. The presence of venous anomalies (hypoplastic left transverse/sigmoid sinuses) may suggest an underlying vascular dysplasia, warranting further evaluation. Long-term follow-up is essential to monitor for aneurysm growth or morphological change.

REFERENCES

1. Brown RD Jr, Broderick JP. Unruptured intracranial aneurysms: epidemiology, natural history, management options, and family screening. Lancet Neurol. 2014;13(4):393-404.
2. Vlak MH, Algra A, Brandenburg R, Rinkel GJ. Prevalence of unruptured intracranial aneurysms, with emphasis on sex, age, comorbidity, country, and time period: a systematic review and meta-analysis. Lancet Neurol. 2011;10(7):626-36.
3. Sorteberg A, Sorteberg W. Fusiform and dolichoectatic aneurysms. In: Handbook of Clinical Neurology. Vol 143. Elsevier; 2017:227-240.
4. Raymond J, Roy D. Safety and efficacy of endovascular treatment of complex intracranial aneurysms. Neuroimaging Clin N Am. 2006;16(3):475-485.
5. Schievink WI. Genetics of intracranial aneurysms. Stroke. 2017;48(3):779-786.
6. Rhoton AL Jr. The supratentorial arteries. Neurosurgery. 2002;51(4 Suppl):S53-S120.
7. Thompson BG, Brown RD Jr, Amin-Hanjani S, et al. Guidelines for the management of patients with unruptured intracranial aneurysms. Stroke. 2015;46(8):2368-2400.
8. Wiebers DO, Whisnant JP, Huston J III, et al. Unruptured intracranial aneurysms: natural history, clinical outcome, and risks of surgical and endovascular treatment. Lancet. 2003;362(9378):103-110.
9. Brinjikji W, Murad MH, Lanzino G, Cloft HJ, Kallmes DF. Endovascular treatment of intracranial aneurysms with flow diverters: a meta-analysis. Stroke. 2013;44(2):442-447.
10. Tóth G, Cerejo R. Intracranial aneurysms: review of current management options. Cleve Clin J Med. 2019;86(5):331-340.

Figure Legends

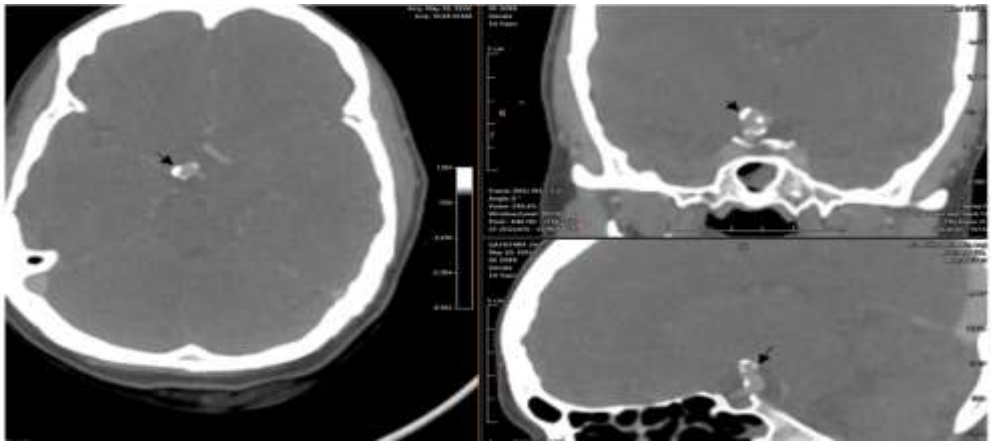

Figure 1: Axial CT angiogram image showing fusiform dilatation with peripheral wall calcification involving the supraclinoid segment of the right ICA (arrow). The dilatation measures approximately 5.5 × 7.0 × 9.3 mm. No thrombus or hemorrhage is present.

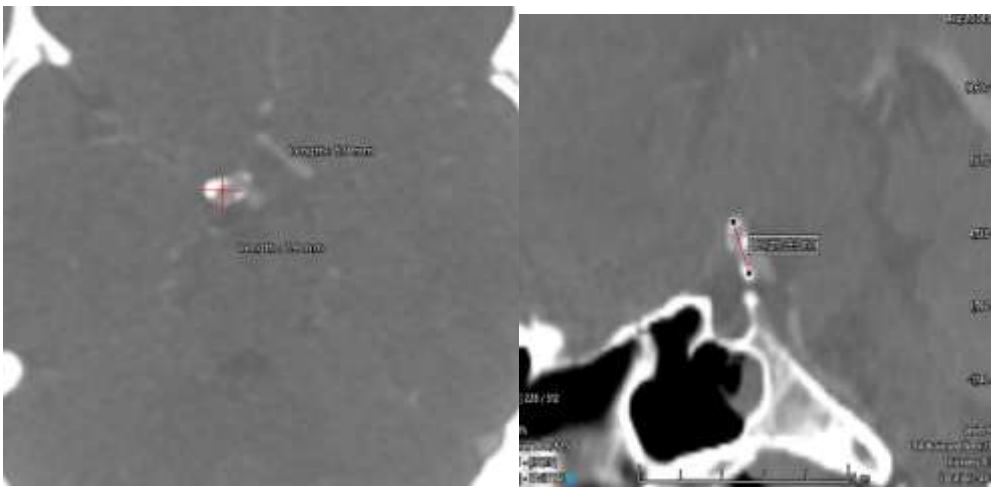

Figure 2: Coronal (or oblique) view demonstrating extension of the fusiform dilatation to involve the proximal part of the right posterior communicating artery (arrow). Peripheral wall calcification persists.

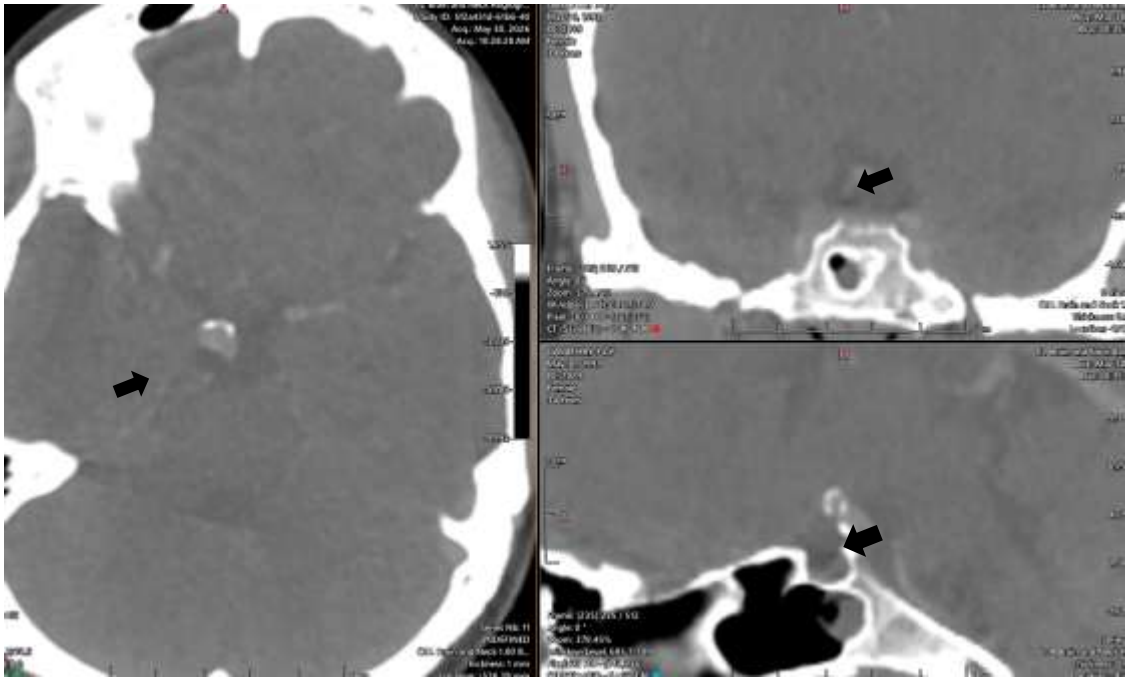

Figure 3: Axial image revealing marked attenuation of the M1 segment of the right MCA (arrowhead) secondary to the adjacent aneurysm.

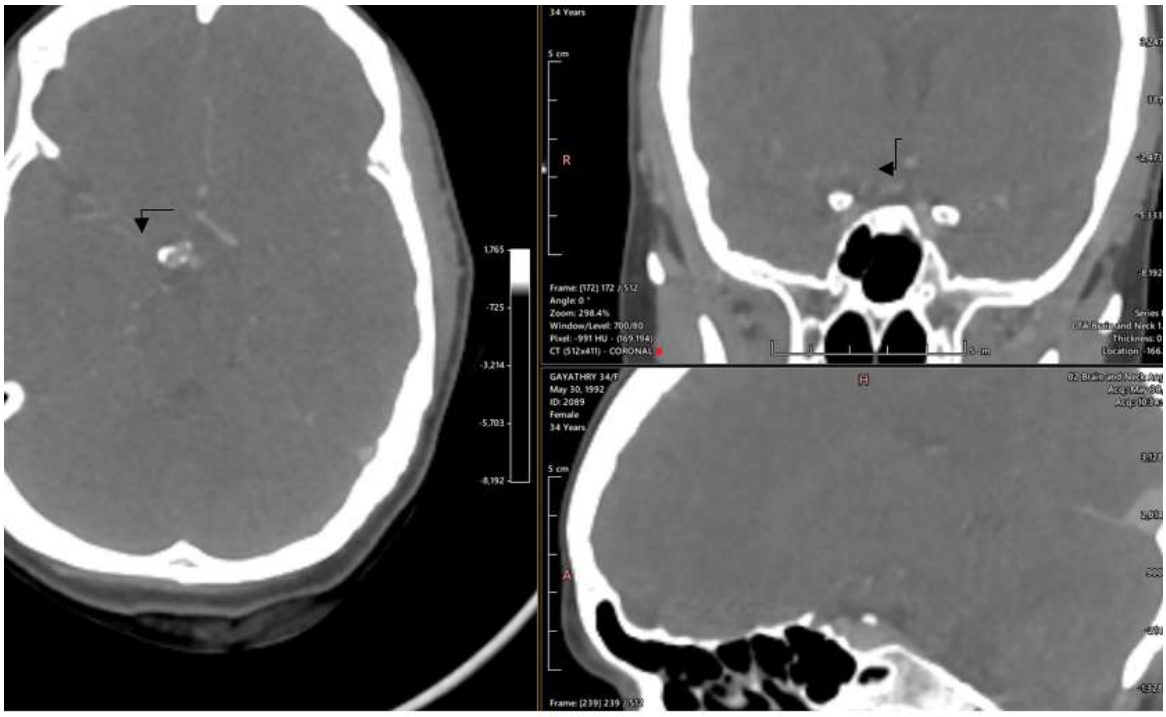

Figure 4: Axial image at a higher level showing opacification of the distal M2, M3, and M4 segments of the right MCA (stars), consistent with collateral reformation compensating for the attenuated M1 segment.