

Case 2

In-Stent Stenosis (ISS)

Keywords: Cerebral aneurysm, Bare stent, In-stent stenosis

Etiology of ISS

Intracranial stents and flow diverters induce neo-endothelial reaction (intimal hyperplasia). This enables the complete exclusion of the aneurysm from circulation (healing reaction), but it is also responsible for ISS [1].

ISS in Intracranial Stenting

A literature search for ISS does not mention or differentiate any specific data related to GIAs. Hence the underlying data applies to aneurysms in general. It is well known in intracranial atherosclerotic disease treated with angioplasty and/or stenting. In the wingspan® (Boston Scientific, Fremont, CA) study, the rate of ISS was as high as 38%. It was also more commonly seen in supraclinoid ICA particularly in young patients. Also supraclinoid ICA ISS was symptomatic in the majority of patients (up to 60%) [2]. However, in patients with aneurysms treated using stent-assisted coiling, the rate of ISS was significantly less at 2.5%. Both Enterprise® (Cordis, Miami Lakes, FL) and Neuroform® (Boston Scientific/Target, Fremont, CA) stents were used in the study. It was found that ISS was mild in most cases and did not require any intervention [1].

ISS in FD Therapy

Both self-expanding stents and FDs have a low radial force and cause minimal vessel injury and ISS. In a retrospective study from a single institution of 149 patients treated with PED® FD, ISS was relatively common occurring in around 16% of patients. Previous studies have reported the incidence of PED® ISS to vary from 3.5 to 10%. This is higher than in Neuroform® (Boston Scientific/Target, Fremont, CA) and Enterprise® (Cordis, Miami Lakes, FL) stents (2.5–5.8%). Furthermore, ISS is an early manifestation in both, and most cases are found within 6 months of treatment. The PED® ISS was mild in nearly 50% of cases and asymptomatic in all patients. The authors recommend continuation or re-institution of dual anti-platelet therapy with follow-up imaging in cases with ISS. The study also found ISS to be associated with anterior circulation FDs; all the affected patients were women (did not reach statistical significance) with a tenfold risk in patients not pretreated with aspirin [1].

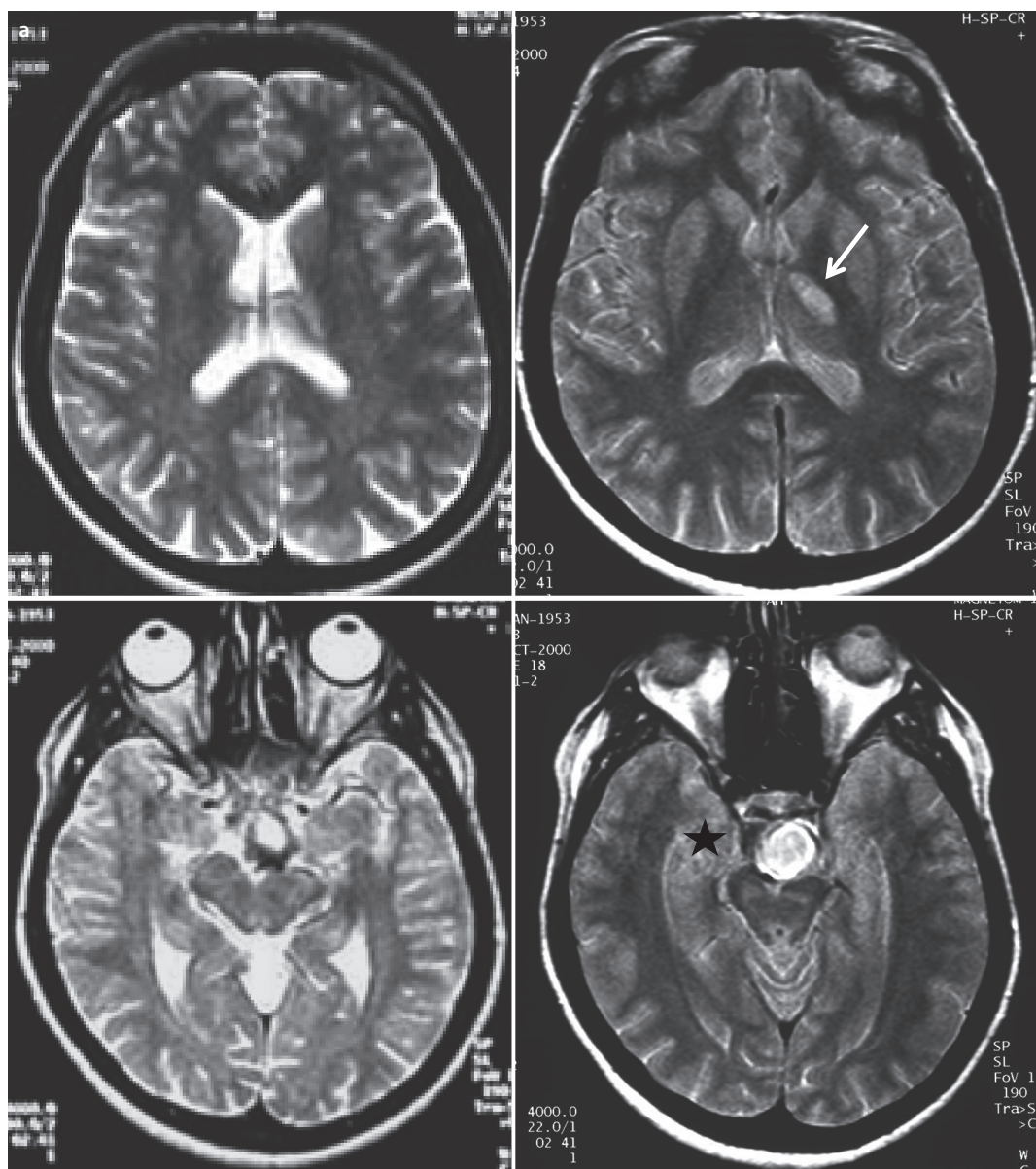
A retrospective review of 47 patients treated at two institutions, with both SILK® and the newer SILK+® FD, detected a midterm (mean follow-up 22 months) ISS of 57%. Severe ISS (>50% stenosis) was seen in 18.5% of patients. Among these patients with ISS, 60% improved or disappeared, 28% were stable, and 12% led to parent vessel occlusion. All ISS leading to PVO were seen in first-generation SILK® FD. However, the rate of ISS in SILK® FD was high in this series, as compared to previous series that were 7.8% in Berge et al., 6% in Byrne et al., and 5% in Murthy et al. [3]. The lead author also published a retrospective study examining 20 patients with 27 aneurysms treated at the same two institutions with PED® FDs. He found a very low rate of ISS (10%) at 3–6 month follow-up angiograms. All the ISS were mild (<50% stenosis) and were asymptomatic [4].

A prospective study of 63 patients treated with PED® FD detected a total of 18.4% ISS on a 3-month control angiogram. Of these, 8% were mild (<50%), 5% were moderate (50–70%), and 5% were severe (>70%). All stenoses were asymptomatic, and three cases showed regression by the 6-month control angiogram [5]. Another retrospective study examined intracranial ICA aneurysms less than 25 mm treated with both SILK® and PED® FDs and detected ISS in 38 and 39% of patients treated with SILK® and PED®, respectively. Out of the total of six patients with ISS, a single patient with distal tapered severe ISS was symptomatic and treated with balloon angioplasty. All cases of ISS were detected early on 2-month control angiograms with no case of delayed ISS [6].

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■ **Fig. 2.1** A 47-year-old lady presented with intermittent headaches, memory loss, and left anisocoria. (a) MRI reveals left anterior thalamic infarct (arrow) with a large well-delineated heterogeneous signal intensity mass lesion with pulsation artifact (star) in the interpeduncular cistern compressing the left mesencephalon suggestive of an aneurysm.

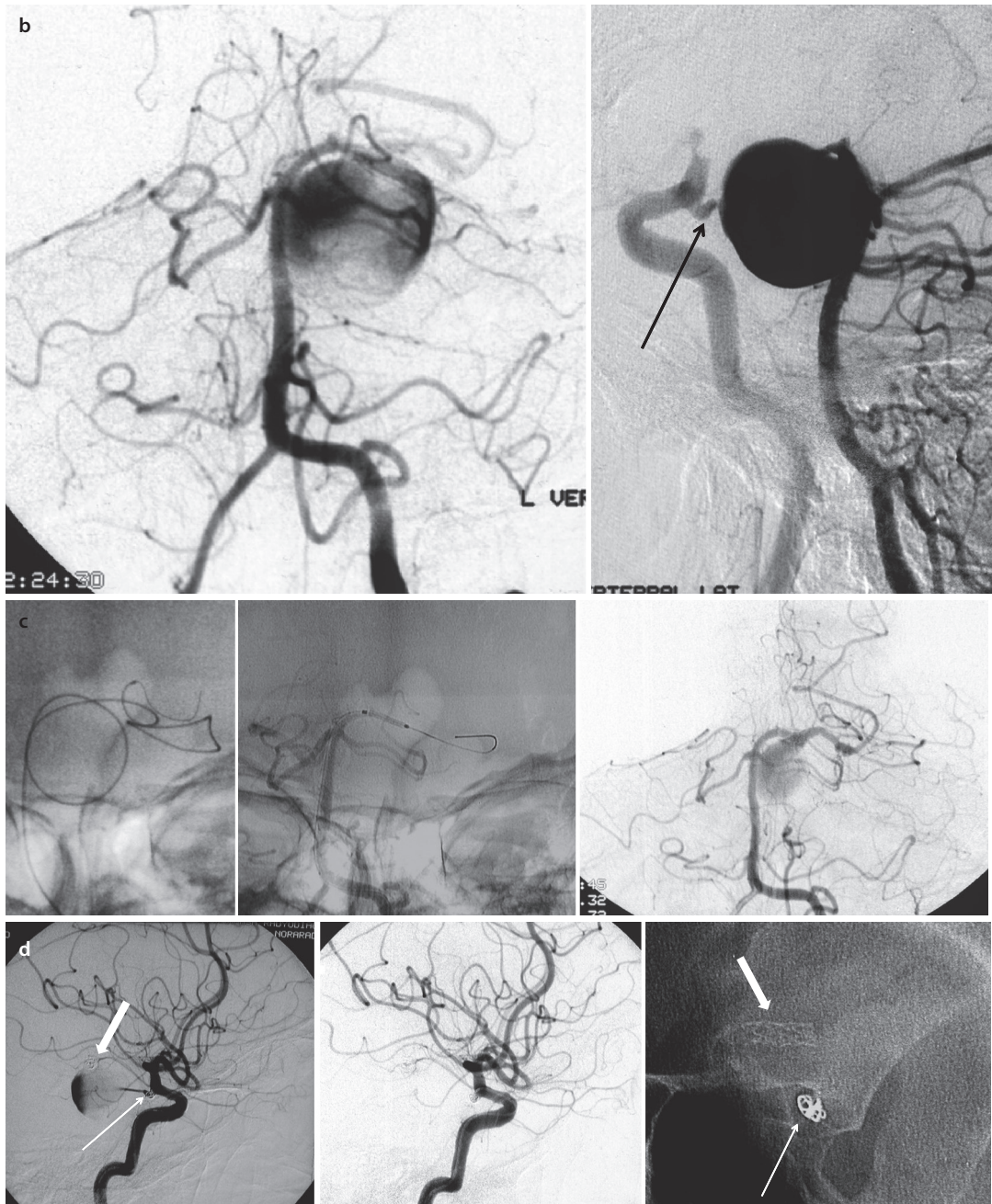
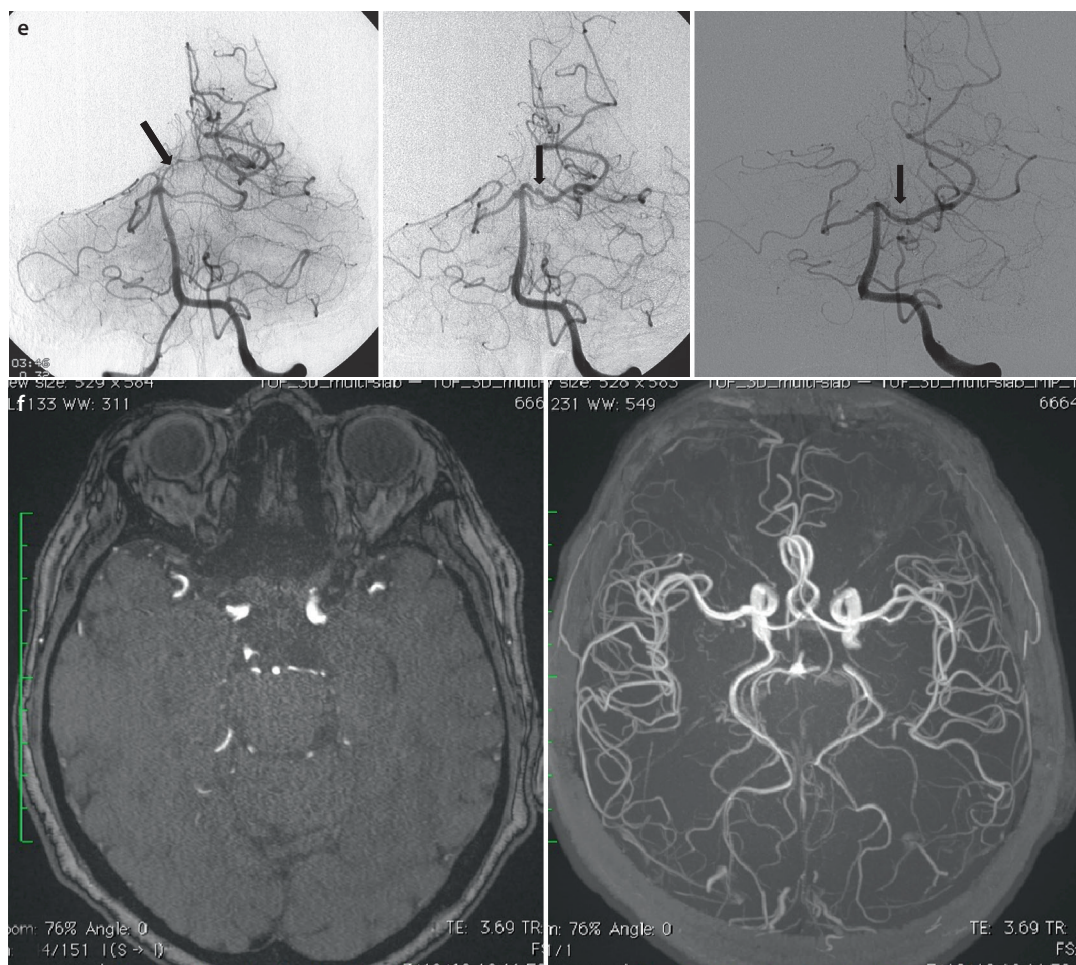


Fig. 2.1 (continued) **(b)** Cerebral DSA reveals a 25 × 22 mm-sized giant fusiform aneurysm in the middle and distal segments of the left PCoM. The aneurysm has a wide neck at its origin from PCA. The aneurysm had dominant filling from PCA with some flow from PCoM (*arrow*). The aneurysm had a slow luminal swirling flow with gradual opacification of the distal PCA branches. **(c, d)** On October 2000, the aneurysm neck was crossed using catheter-looping technique. A 2.5 × 9 mm-sized balloon-mounted coronary stent was deployed across the aneurysmal neck to create flow diversion. There was significant decrease in aneurysmal opacification with a small residual filling from PCoM. Hence, two coils were used to occlude the inflow zone from the PCoM infundibulum. Fluoroscopic AP image shows coils within the PCoM infundibulum (*arrow*) and coronary BMS (*block arrow*) within the PCA.



■ **Fig. 2.1** (continued) **(e)** Control DSA at 6 months, 1 year, and 5 years, respectively, reveals complete exclusion of the aneurysm from circulation. There is moderate to severe in-stent stenosis in left PCA (*arrow*) at 6 months. Note the elevation of P1 and P2 segments due to mass effect of thrombosed aneurysm. Follow-up DSA at 1 year shows regression of the in-stent stenosis (*arrow*) with residual contour irregularity. The PCA has reverted to its original configuration following shrinkage of aneurysm. Subsequent DSA at 5 years shows complete resolution of left PCA in-stent stenosis (*arrow*) with a normal PCA configuration. **(f)** On a 13-year follow-up MRA, there is aneurysmal occlusion with normal appearance of intracranial arteries

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