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Case Report

# Intraprocedural Plaque Protrusion during Carotid Artery Stenting with a CASPER Stent: A Case Report

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**Objective:** Plaque protrusion (PP) during carotid artery stenting (CAS) is considered to be associated with periprocedural ischemic stroke. A new double-layer micromesh stent, the CASPER stent (CS), was approved for use in Japan in 2020. The expectation is that this micromesh stent system will reduce the risk of PP, but we report a case of PP during CAS despite the use of a CS.

**Case Presentation:** An 87-year-old man presented with left hemiparesis. MRI showed right brain infarction and angiography showed right internal carotid artery stenosis with thrombus. Follow-up angiography after medical treatment showed that thrombus disappeared. We therefore performed CAS for right internal carotid artery stenosis with unstable plaque. CAS was performed under local anesthesia with Mo.Ma Ultra and FilterWire EZ protection using a CS placed to sufficiently cover the stenotic region. Conservative post-dilatation was then performed. Intravascular ultrasonography (IVUS) after post-dilatation showed the presence of PP. A second CS was then added using the stent-in-stent technique. No postoperative neurological abnormalities were found and the patient was discharged without postoperative complications. No stroke or restenosis has been observed as of 16 months after CAS.

**Conclusion:** PP can occur even when CAS is performed using the CS for carotid artery stenosis with unstable plaque. The importance of checking for PP using IVUS is suggested.

**Keywords** carotid stent, plaque, CASPER stent, intravascular ultrasound

## Introduction

Although carotid artery stenting (CAS) is noninferior to endarterectomy for high- and standard-risk patients,[1,2] ischemic stroke as a perioperative complication related to CAS remains an important issue. Previous studies have reported that risk factors for cerebral infarction include the use of protection devices, insufficient operator skill, higher patient age, plaque properties, stent design, and statin use.[3] Kotsugi et al.[4] reported plaque protrusion (PP) as a new risk factor strongly associated with cerebral infarction. Preventing perioperative cerebral infarction may require selection of a procedure and devices that do not induce PP. Furthermore, they mentioned unstable plaque and open-cell stent use as predictors of PP. As a method to prevent PP in the case of unstable plaque, new stents such as the CASPER stent (CS; Terumo, Tokyo, Japan), a double-layer micromesh stent, have been clinically applied, and favorable results have been reported from several clinical studies.[5,6] Such results suggest that low ischemic stroke rate is associated with prevention of PP. To the best of our knowledge, no reports have described PP detected by intravascular ultrasonography (IVUS) during CAS with a CS. We therefore report this case of PP during CAS even while using a CS. All procedures of this case report were approved by the Ethics Institutional Review Board of our hospital and written informed consent was obtained from the patient.

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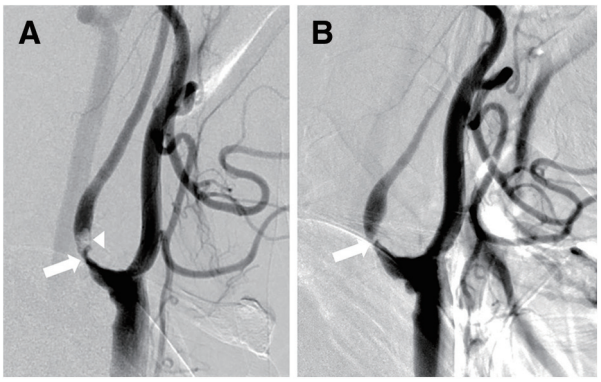


**Fig. 1** Lateral-view right common carotid angiography. (**A**) Angiography on admission shows severe stenosis (arrow) with thrombus (arrowhead) at the origin of the right ICA. (**B**) Angiography on hospital day 7 shows complete disappearance of thrombus (arrow). ICA: internal carotid artery

## Case Presentation

An 87-year-old man presented to our hospital with an 8-h history of left-sided weakness. MRI showed right internal carotid artery (ICA) occlusion and he was referred to our department. He presented with somnolence, left-sided paralysis of the arm and leg, and left-sided facial weakness. The National Institutes of Health Stroke Scale score was 6.

Diffusion-weighted imaging showed bright lesions in the right cerebral hemisphere, and MRA showed defect or weakness of the right ICA signal (not shown). Cerebral angiography showed the North American Symptomatic Carotid Endarterectomy Trial 90% stenosis at the origin of the right ICA, with thrombus observed on admission (**Fig. 1A**). The patient was immediately administered 300 mg of aspirin and 600 mg of clopidogrel as loading doses, and heparinization was started on admission. From the next day, aspirin and clopidogrel doses were reduced to 100 mg/day and 75 mg/day, respectively. On hospital day 3, we confirmed using the VerifyNow system (Accumetrics, San Diego, CA, USA) that aspirin reaction units and P2Y12 reaction units were 456 and 349, respectively, so clopidogrel was changed to prasugrel. On hospital day 7, angiography showed complete disappearance of thrombus (**Fig. 1B**). We therefore planned CAS for hospital day 14 to avoid ischemic complication. P2Y12 reaction units were checked on the operation day and found to be 207, almost within the effective ranges.

Plaque at the origin of the right ICA showed marked signal hyperintensity on axial-view T1-weighted imaging using the black blood method. The ratio of signal intensities for the sternocleidomastoid muscle and plaque was 1:2, suggesting unstable plaque (**Fig. 2**).

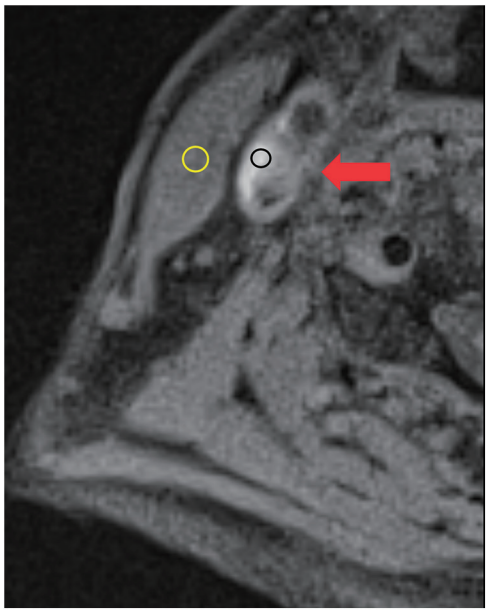

**Fig. 2** Preoperative axial-view MRA of the plaque with T1-weighted black blood method. Signal hyperintensity is evident at the origin of the right ICA (arrow). The black circle shows the ROI at the carotid plaque (mean signal intensity: 382.7) and the yellow circle shows the ROI in the muscle (mean signal intensity: 189.5). Signal intensity ratio is 2:1, suggesting the presence of unstable plaque. ICA: internal carotid artery; ROI: region of interest

Under local anesthesia, an 8-Fr. long sheath was inserted through the right femoral artery. After intraoperative systemic heparinization (activated clotting time ≥275 sec), the following two protection devices were inserted: an 8-Fr. Mo.Ma Ultra (Medtronic Vascular, Santa Rosa, CA, USA) and a FilterWire EZ (Boston Scientific, Natick, MA, USA). The lesion was crossed with a microcatheter (Aguru; Boston Scientific) and pre-pre dilatation at the site of stenosis was performed using a 2.0 mm × 20 mm balloon catheter (SHIDEN; Kaneka Medix, Osaka, Japan). The lesion was crossed with the FilterWire EZ, and predilatation at the site of stenosis was performed using a 3.0 mm × 40 mm balloon catheter (Oceanus; iVascular, Sant Vicenç dels Horts, Spain). Subsequently, an 8.0 mm × 30 mm CS was inserted in accordance with the stenotic lesion. Post-dilatation was conservatively conducted using a 4.0 mm × 40 mm balloon catheter (Rx-Genity; Kaneka Medix).

IVUS (Volcano Visions PV 0.014P catheter with Chroma Flo; Volcano, Rancho Cordova, CA, USA) immediately post-dilatation showed PP, representing the most stenotic lesion. A second CS (8.0 mm × 20 mm) was additionally inserted to cover the PP area considering distal embolism and acute occlusion. After inserting the second CS, IVUS immediately confirmed the disappearance of PP. The patient showed no neurological symptoms and the procedure was completed (**Fig. 3**).

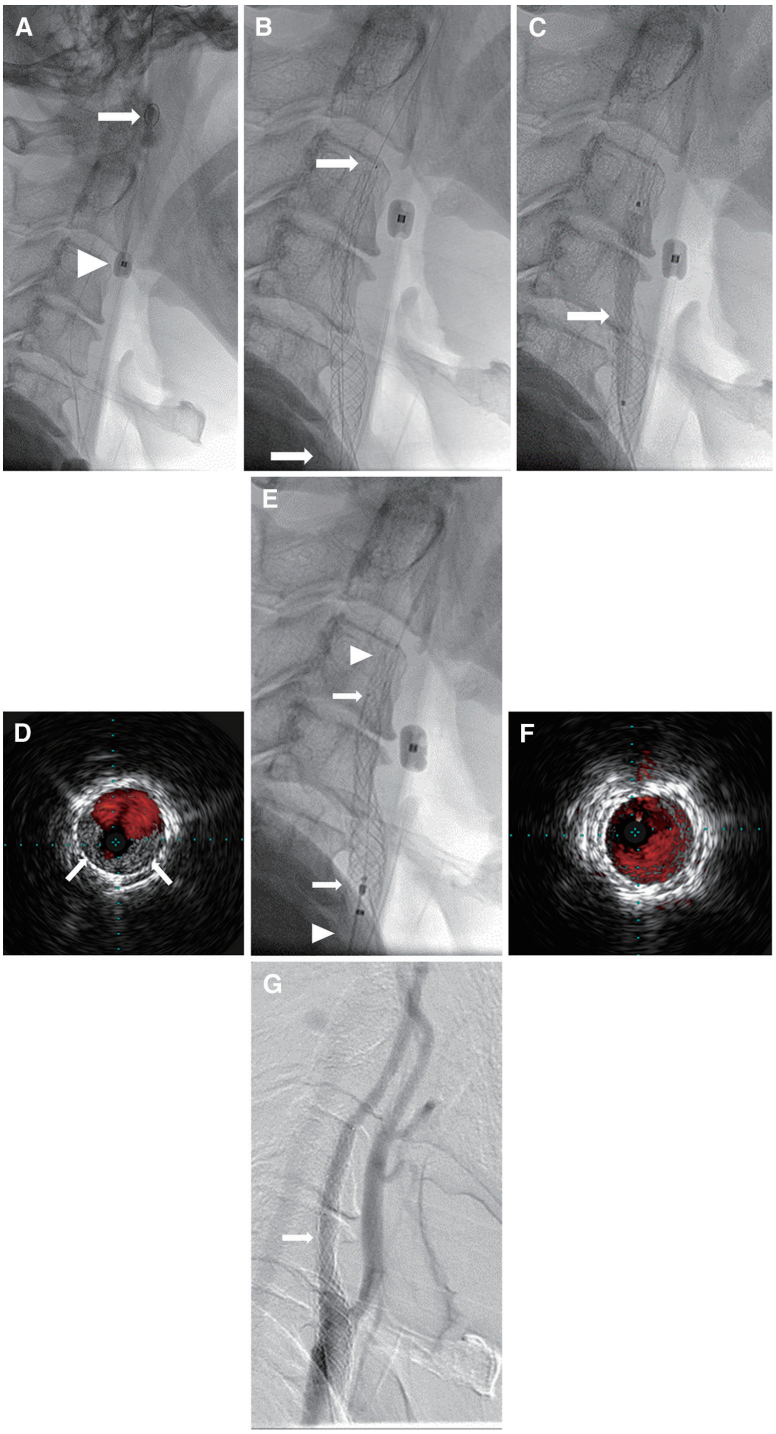


**Fig. 3** CAS using the CS to treat severe stenosis with unstable plaque at the origin of the right ICA. (**A**) Fluoroscopic image of protection device placement, with proximal protection provided by a Mo.Ma Ultra device (arrowhead) and distal protection by a FilterWire EZ (arrow). (**B**) An 8 mm × 30 mm CS is placed to cover the stenotic site (arrows). (**C**) Post-dilatation is performed using a 4 mm × 40 mm balloon catheter (arrow). (**D**) IVUS after stent placement. The isoechoic area in the vascular lumen indicates intraluminal PP (arrows). (**E**) A second CS (8 mm × 20 mm; arrows) is guided inside the first stent (arrowheads) and placed in a stent-in-stent manner. (**F**) After placement of the second stent, IVUS performed at the level of minimum lumen diameter confirms the absence of PP. (**G**) Lateral-view angiography of the right common carotid following additional second CS shows successful dilatation of the lesion without in-stent defect (arrow). CAS: carotid artery stenting; CS: CASPER stent; ICA: internal carotid artery; IVUS: intravascular ultrasonography; PP: plaque protrusion

No new neurological abnormalities have been seen since CAS. Diffusion-weighted imaging the day after CAS showed no signal-hyperintense areas. Evaluation of PP using ultrasonography on postoperative day 5 proved difficult and the patient was discharged on postoperative day 25. Follow-up angiography 6 months later confirmed the disappearance of PP. As of 16 months after CAS, no stroke or restenosis has been observed.

## Discussion

The main purpose of CAS is to prevent ischemic stroke, so ischemic stroke as a perioperative complication should be avoided as much as possible. Nevertheless, an incidence of 2.5%–6.0% has been reported, and the incidence of perioperative ischemic stroke is slightly higher than that after carotid endarterectomy (CEA).[1,2] Previous studies have reported many risk factors for perioperative ischemic stroke associated with CAS.[3] Kotsugi et al.[4] reported PP as a new risk factor in 2017. They identified PP in 2.6% of 328 patients who underwent CAS and found a strong association with perioperative ischemic stroke and no association with protection devices. Furthermore, they highlighted vulnerable plaque and the use of open-cell stents as predictors of PP. Considering the etiology of PP, they hypothesized that the use of a stent with strong radial force and post-dilation would lead to disruption of the fibrous capsule, resulting in soft PP through the stent. Unstable plaque was present in our case, corresponding to a high risk of PP.

Given the etiology of PP, fine-mesh stents may be suitable for preventing PP. Micromesh stents, which have a finer mesh than standard stents, are used in clinical settings. A meta-analysis of four micromesh stent study showed that all 556 patients achieved successful stent placement, with a perioperative stroke rate of 1.08% (n = 6).[5] A recent multicenter prospective study in Japan using the CASPER micromesh stent showed a perioperative ischemic stroke rate of 1.4%.[6] The incidence of perioperative stroke in both reports is lower than that in patients treated using conventional stents, which may have decreased through the inhibitory effects on PP.

Yamada et al.[7] reported on 46 consecutive patients with unstable plaque identified on magnetic resonance imaging who underwent CAS with optical frequency domain imaging (OFDI). OFDI analysis showed that the presence of PP was significantly lower in the CS group (44%) than in the conventional stent group (88%; p = 0.022). Moreover, mean PP area was significantly smaller in the CS group ($0.013 \pm 0.034$ mm$^2$) than in the conventional stent group ($0.057 \pm 0.09$ mm$^2$; p = 0.006). This study shows that the CS suppresses PP compared to conventional stents, and the reduction of ischemic complications of CS may be due to PP suppression. The optimal treatment for PP after CAS has yet to be established. Kotsugi et al.[4] considered treatment methods in the presence of PP and suggested that stents should be added until the PP disappears, followed by follow-up for 5–10 min, and recommended that patients with non-convex-type PP undergo careful clinical follow-up within 30 days after CAS. In the present case, PP geometry was assessed as convex, and an additional CS was inserted.

In-stent restenosis with CS is of concern because of the larger volume of metal stent material, which might be associated with hyper platelet aggregation compared with the standard single-layer stent. In fact, two recent studies showed the restenosis rate of CS compared with that of a single-layer stent. Sycora et al.[8] reported in a comparison of in-stent restenosis risk between dual-layer and single-layer stents that the rate of severe restenosis (≥70%) was significantly higher in the dual-layer stent group than in the single-layer stent group (13.3% [11/83] vs 3.4% [4/116], p = 0.01). Mazurek et al.[9] also performed a systematic review and meta-analysis of clinical studies on the topic (Preferred Reporting Items for Systematic Reviews and Meta-Analyses [PRISMA] methodology, 3302 records). They concluded that the dual-layer stent reduced the risk of ischemic stroke at 12 months (–3.25%, p <0.05) but was associated with an increased risk of in-stent restenosis (3.19%, p = 0.04).

In our case, the shape of the PP was estimated and an additional CS was inserted. After confirming the disappearance of PP, the procedure was completed and no postoperative ischemic complications were encountered. As PP may recur despite disappearance during the procedure, careful follow-up may be warranted after CAS in cases at high risk of PP with unstable plaque or a large volume of plaque, similar to the present case. Several investigators have reported the utility of IVUS for PP diagnosis. The incidence of PP in IVUS during CAS is 7.8%–10%.[10–12] Kotsugi et al.[4] reported that PP was detectable using IVUS in 27 (7.6%) of 352 patients, with DSA revealing PP in nine patients (2.6%). Furthermore, they reported no cases that could not be detected by IVUS were detected by DSA, suggesting the importance of IVUS in the diagnosis of PP. In the present case, PP diagnosis by IVUS was performed just before deprotection, and we again recognized the importance of PP diagnosis by IVUS post dilatation.

## Conclusion

PP may occur even when CAS is performed using the CS for carotid artery stenosis with vulnerable plaque. The importance of PP diagnosis by IVUS has been suggested.

## Disclosure Statement

The authors declare that they have no conflicts of interest.

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