Online April 20, 2023
Journal of Neuroendovascular Therapy 2023; 17: 107–113
DOI: 10.5797/jnet.cr.2023-0004

Case Report

A Case of Acute In-Stent Mobile Plaque after Carotid Artery Stenting Aspirated with a Distal Access Catheter

Takayuki Ishikawa, Takashi Yamanouchi, and Ryusuke Kabeya

Objective: Carotid artery stenting (CAS) is common procedure for carotid stenosis, but sometimes acute in-stent thrombosis or plaque protrusion after CAS leads to postoperative stroke. There are few reports of aspiration of in-stent plaque protrusion. This paper reports a case of acute in-stent mobile plaque aspirated with a distal access catheter.

Case Presentation: A 74-year-old male underwent CAS for symptomatic internal carotid artery stenosis and postoperative course was thought to be good, but in-stent mobile plaque was detected by carotid duplex at postoperative day 6. As mobile plaque is a high risk for stroke, we performed plaque aspiration with a distal access catheter, without neurological deficit or a new cerebral lesion in magnetic resonance imaging. We present a case report, including a literature review, of acute thrombosis or in-stent plaque protrusion.

Conclusion: Aspiration removal may be effective for in-stent mobile plaque, which is expected to be fragile, avoiding the disadvantages of increasing stents.

Keywords carotid artery stenting, acute in-stent thrombosis, in-stent plaque protrusion, aspiration of in-stent plaque, mobile plaque

Introduction

Vulnerable carotid plaque is a high risk for carotid artery stenting (CAS), and carotid endarterectomy is recommended in such a case. However, patients who are not able to tolerate general anesthesia sometimes receive CAS instead of vulnerable plaque. Acute in-stent protrusion may occur after CAS for vulnerable plaque, and acute in-stent thrombosis or plaque protrusion (ISTP) is major risk factor for postoperative stroke.[1] We report a case of acute in-stent mobile plaque aspirated with a distal access catheter.

Department of Neurosurgery, Ichinomiya Municipal Hospital, Ichinomiya, Aichi, Japan

Received: February 6, 2023; Accepted: April 10, 2023
Corresponding author: Takayuki Ishikawa. Department of Neurosurgery, Ichinomiya Municipal Hospital, 2-2-22, Bunkyo, Ichinomiya, Aichi, Japan
Email: takazxrstcr@gmail.com

Case Presentation

This case report was prepared after informed consent was obtained from the patient.

A 74-year-old male, with a previous left cerebral cortical infarction 1 month prior, was under treatment by neurologists. Although he took double antiplatelet agents, clopidogrel, acetylsalicylic acid, and atorvastatin, cerebral cortical infarction increased. Therefore, we were consulted about surgical treatments. The plaque had high intensity on time of flight of magnetic resonance imaging and low on carotid duplex (**Fig. 1A** and **1B**), indicating the plaque was vulnerable. The lesion was approachable by direct surgery, but the patient also had a history of myocardial infarction and chronic obstructive pulmonary disease. A cardiologist, respiratory physician, and anesthesiologist decided that the patient's cardiac and respiratory function was high risk for surgery under general anesthesia; therefore, we chose CAS.

Under local anesthesia, we started the procedure with a right femoral approach. On DSA, the diameter of the proximal common carotid artery (CCA) was 8.5 mm, the diameter of the distal internal carotid artery (ICA) was 4.0 mm, and the diameter of the narrowest portion of the ICA was

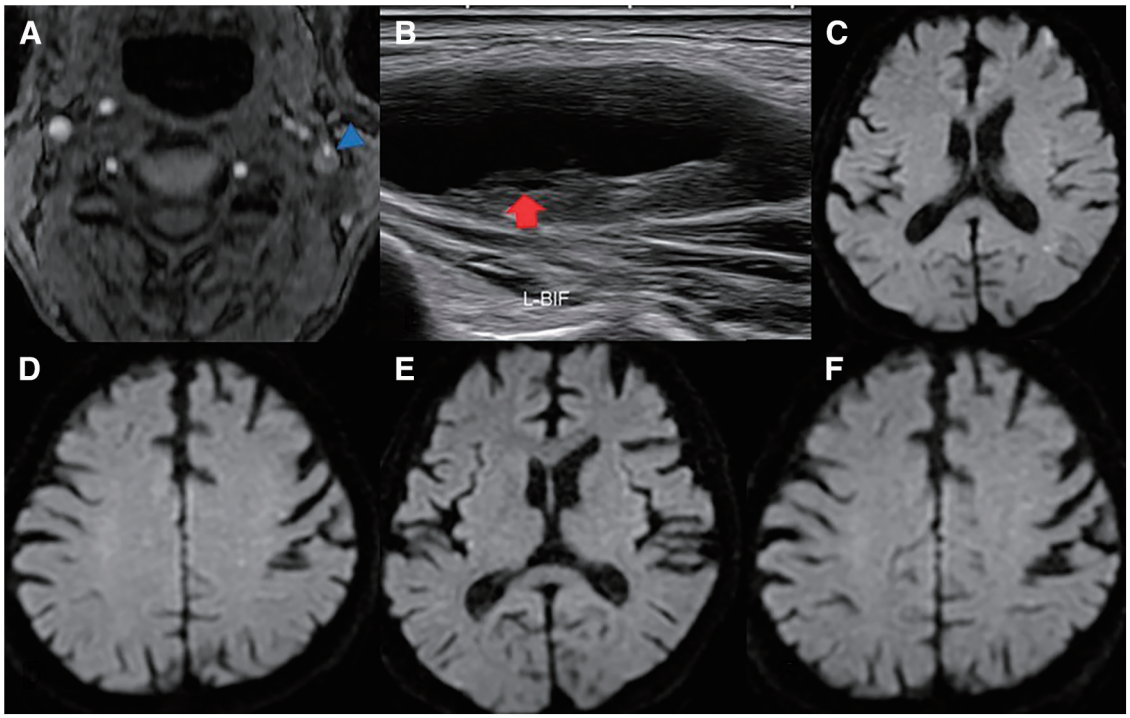

Fig. 1 Preoperative plaque images and postoperative MRI. (**A**) Preoperative plaque image by TOF. The plaque shows high intensity compared to the sternocleidomastoid muscle (blue arrowhead). (**B**) Preoperative plaque image by carotid duplex. The plaque appears low overall, and a linear echo-free space is seen in the plaque (red arrow). (**C** and **D**) The DWI images after the first surgery. A few tiny high-intensity spots can be seen (asymptomatic). (**E** and **F**) The DWI images after the second procedure. There are no additional lesions. TOF: time-of-flight

1.7 mm. We initiated occlusion of the CCA with an occlusion balloon (8Fr OPTIMO EPD; Tokai Medical Products, Aichi, Japan) and that of the external carotid artery (ECA) with another occlusion balloon (Pinnacle Blue 20; Tokai Medical Products). The lesion was crossed by a guide wire with a filter (FilterWire EZ; Boston Scientific, Natick, MA, USA) and a filter was developed at the distal ICA. After deflating the ECA occlusion balloon, the lesion was dilated using a 3 mm × 4 cm angioplasty balloon (Sterling; Boston Scientific) at 6 atm for 30 seconds and implanted with a 10 mm × 31 mm closed-cell stent (Carotid Wallstent; Boston Scientific). After that, post-dilation was performed with the same 3 mm × 4 cm angioplasty balloon at 6 atm for 10 seconds. Although slight contrast pooling outside the stent remained, thrombotic tendency was not detected, and we completed the procedure (**Fig. 2A** and **2B**).

Although MRI at postoperative day (POD) 1 showed a few spots of high-intensity lesion in the diffusion weighted image (DWI) (**Fig. 1C** and **1D**), the patient did not show any new neurological deficit. The postoperative course appeared to be good, but carotid duplex at POD 6 detected two in-stent mobile plaques (3.6 mm × 4.5 mm and 2.0 mm × 1.6 mm) (**Fig. 3A** and **3B**, respectively). We decided that the mobile plaque was highly vulnerable, and the possibility of embolic stroke was high; therefore, we decided to first attempt to remove it by aspiration using a thrombectomy catheter and, if necessary, perform overlapped stenting.

We again started the procedure with a right femoral approach. After placing the CCA occlusion balloon (8Fr OPTIMO EPD), the two plaques were detected by DSA and carotid duplex (**Figs. 2C**, **3C**, and **3D**). In the state of CCA occlusion with the use of an occlusion balloon, the lesion was crossed by a distal ICA occlusion balloon (SHOURYU HR 4-7; Kaneka Medical Products, Osaka, Japan). After deflating the CCA occlusion balloon, a distal access catheter (AXS CATALYST 7; Stryker, Kalamazoo, MI, USA) was inserted near to the plaques, applying suction coaxially with the ICA occlusion balloon. The larger plaque was aspirated well, but the smaller plaque was not aspirated. We believed that the ICA occlusion balloon limited the movement of the distal access catheter, and after inflating the CCA occlusion balloon as proximal protection and removing the ICA occlusion balloon, the residual plaque was aspirated by the distal access catheter. A tiny residual in-stent plaque (1.4 mm × 1.4 mm) was detected

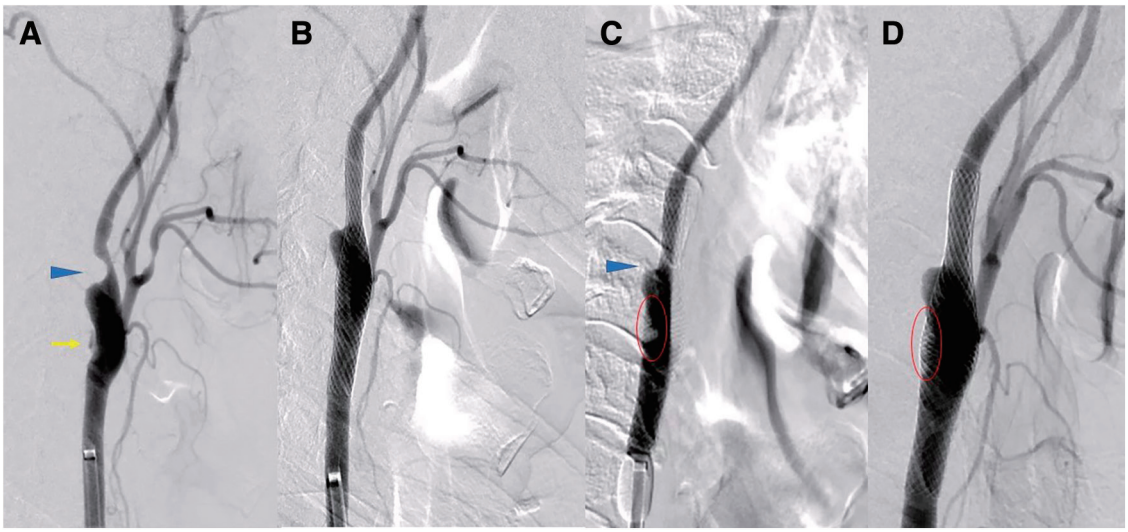

Fig. 2 DSA view. (**A**) DSA performed before the balloon dilation and stenting. Ulcer formation is detected (yellow arrow) and the narrowest region is placed at distal end of the plaque (blue arrowhead). (**B**) DSA after CAS. There is extra-stent contrast pooling, but ISTP is not detected. (**C**) DSA performed in the state of CCA occlusion at the second operation. ISTP is detected, not at the narrowest region (blue arrowhead) but at the ulcer site (red circle). (**D**) After aspiration, there is a slight residual ISTP, but the plaque is flattened and thrombosis does not occur (red circle). CAS: carotid artery stenting; ISTP: in-stent thrombosis or plaque protrusion

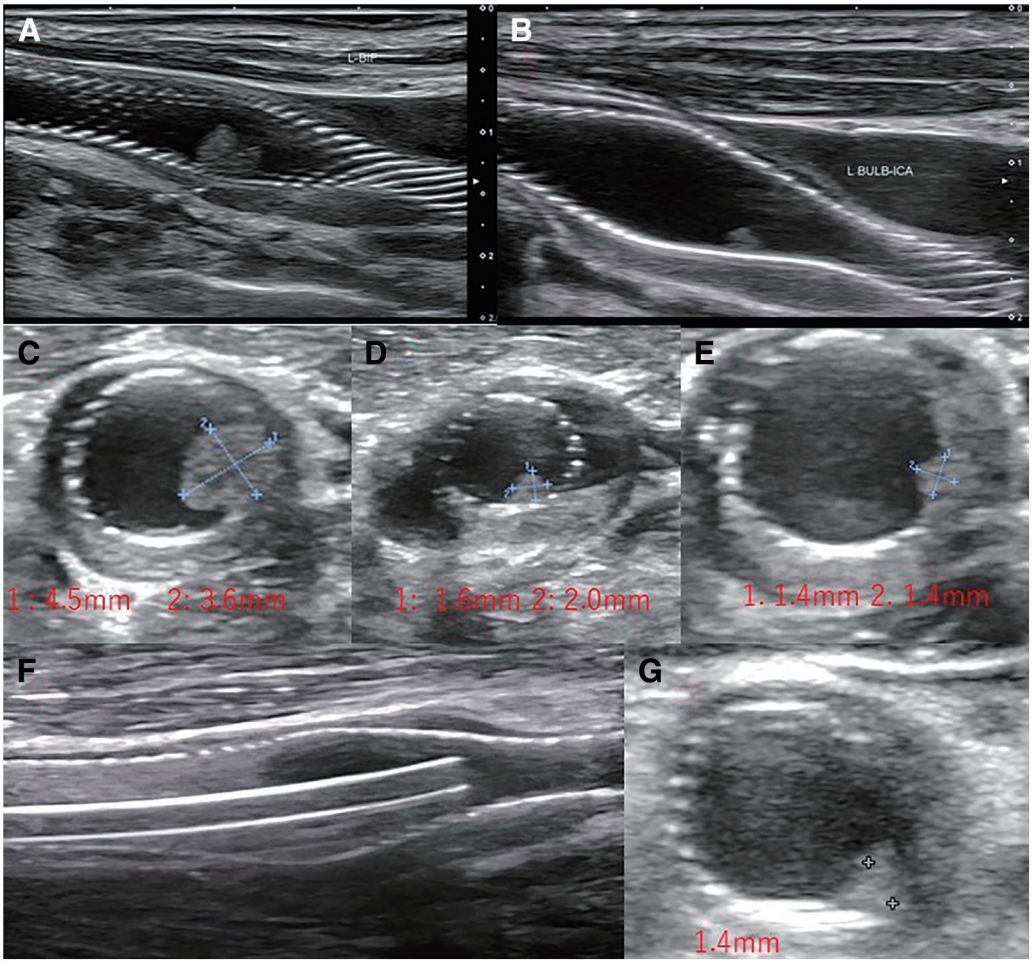

Fig. 3 Carotid duplex view. (**A** and **B**) The two mobile plaques are detected in the stent. (**C** and **D**) Two plaques before aspiration detected by intraoperative carotid duplex. (**E**) The residual plaque after aspiration. (**F**) The distal access catheter is clearly visualized by intraoperative carotid duplex. (**G**) The residual plaque at POD 1. POD: postoperative day

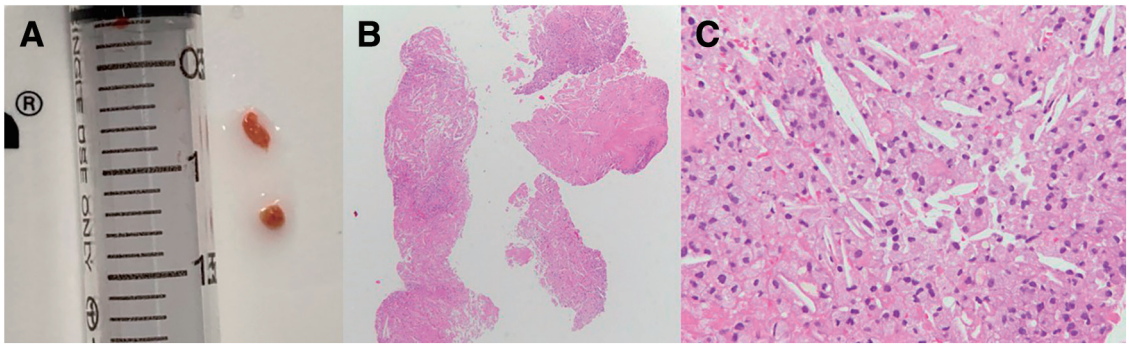

Fig. 4 Macro- and microscopic findings of the aspirated plaques. Appearance is yellowish (**A**) and microscopically, atherosclerotic plaque with fibrin; aggregation of inflammatory cells, such as macrophages; and abundant cholesterol cleft (HE staining; original magnification, ×20 [**B**], ×200 [**C**]). HE: hematoxylin and eosin

by DSA and carotid duplex, but the plaques were flattened, mobility of plaques were lost, and thrombus formation was not detected approximately 30 minutes after aspiration. We completed the procedure without stent-in-stent development (**Figs. 2D**, **3E**, and **3F**).

After the procedure, we administrated argatroban (60 mg/day) for 3 days, in combination with double antiplatelet therapy and statins. The postoperative course was good. After the operation, the patient had no additional neurological deficits and MRI showed no additional lesions on DWI or any other sequences. (**Fig. 1E** and **1F**). Carotid duplex was performed at 1 day, 1 week, 1 month, and 3 months postoperatively; and fortunately, the residual tiny plaques did not increase from 1 day to 1 month (**Fig. 3G**) and disappeared by 3 months.

The aspirated plaque was diagnosed pathologically as atherosclerotic plaque with fibrin; aggregation of inflammatory cells, such as macrophages; and abundant cholesterol cleft (**Fig. 4**).

Discussion

In this case, we aspirated in-stent mobile plaque with a distal access catheter 1 week after CAS.

There have been several reports about acute ISTP after CAS. Acute ISTPs are reported to occur in 20%–40% of cases after CAS.[2,3] However, it is reported that symptomatic stroke occurs at the rate of 0.5%–2% in ISTP cases.[4] Beppu et al. reported that ISTP detected with intraoperative intravascular ultrasound (IVUS) is an independent risk factor for postoperative ipsilateral stroke within 30 days, with an odds ratio of 27.03.[1] In addition, although the frequency of ISTP detected with intraoperative DSA is lower than with IVUS, 66.7% of ISTP detected with DSA cases led to ipsilateral stoke within 30 days after CAS.[5] According to the International Carotid Stenting Study (a randomized trial), post-CAS ipsilateral stroke is most common within 1 day after the procedure (74%), relatively seen up to 30 days (26%) postoperatively, but markedly less frequent after 30 days (0%) postoperatively.[6] In addition, it has been reported that the presence of ISTP at 1 month does not increase the risk of subsequent stroke from occurring 1 month to 1 year later.[2] From these studies, it is evident that patients, especially the high-risk group for CAS, should be monitored closely for up to 30 days after CAS.

The causes of acute or subacute ISTP, which can lead to postoperative stroke after CAS, are complex. The reasons for thrombosis are reported to be insufficient antithrombotic agents (including resistance to antiplatelet agents), hypercoagulation (patients with carcinoma, diabetes mellitus, or thrombocythemia), and intraprocedural carotid dissection.[7,8] On the other hand, the reasons for plaque protrusion are thought to be vulnerable plaque[1] or ulcer formation.[9] Among these factors, vulnerable plaque and ulceration were most likely to apply to this case, although drug resistance was not tested. In spite of a closed-cell stent, which we used in this case, at the time of reoperation, the stent was shortened by approximately 5 mm in length and expanded by a maximum 1.5 mm in diameter, compared to immediately after deployment in the first operation. The in-stent plaque protrusion site was consistent with the ulcer formation site, not the most stenotic site (**Fig. 1A** and **1C**). In addition, the plaque was pathologically diagnosed as lipid-rich atherosclerotic plaque. From these results, it can be inferred that the unstable plaque around the ulcer was extruded from the ulcer site by self-expansion of the stent after surgery.

There are reports that ulcer formation does not increase the risk of post-CAS stroke[1,10]; however, in this case, ulceration was still a risk of post-CAS stroke. From the viewpoint of stent design, controversy continues regarding differences in in-stent protrusion or postoperative stroke rates due to differences in stent design. As discussed above, the cause is multifactorial and not only due to plaques, so it is conceivable that there is no significant difference. In reports of direct observation of in-stent protrusion by angioscopy, it occurred in both open-[11] and closed-cell stents.[12] Angioscopy can detect intima- and ISTP, which cannot be detected by IVUS or optical coherence tomography, and is thought to be useful for elucidating the pathology of acute ISTP and improving CAS treatment outcomes. However, in Japan, it is off-label for carotid artery lesions and future development is awaited.

Controversy also continues regarding treatments for acute in-stent thrombus or plaque protrusion. Some reports recommend aggressive percutaneous transluminal angioplasty (PTA) and overlapped stent during the first operation,[13] and other reports recommend catheter thrombolysis or addition of antithrombotic drugs.[1] In addition, there are reports about open surgical treatments, including stent removal[14–16] and superficial temporal artery–middle cerebral artery anastomosis.[17] The treatment policy is influenced by many factors, such as the speed of disease progression, timing, expected pathology, degree of antithrombotic therapy being administered, patient's general condition, and devices and procedures available at the facility. It seems that it is difficult to set a certain guideline.

In this case, we chose aspiration of the mobile in-stent plaque. The plaque was shown to flow out from the strut of the closed-cell stent and protruded greatly into the lumen of the blood vessel. At that time, no stable endothelium was present on the plaque surface. Consequently, the possibility of thrombus formation and scattering of the plaque seemed high. Based on past reports, mobile plaque, even in stents, are highly likely to be resistant to medical treatment, and early surgical intervention is recommended.[18,19] In such cases, PTA and overlapped stent, or surgical stent removal is commonly used. There have been several reports of surgical stent removal in the early postoperative period.[14–16] This is very effective for removing the plaque but there are two limitations. First, in many cases, the stent is several millimeters to 1 cm distal to the distal end of the plaque, making it technically difficult to secure the distal ICA. Second, general anesthesia was high risk in those cases. In the present case, general anesthesia was also difficult, and CAS was selected for the first surgery; therefore, direct surgery was not selected for the second procedure. Although an overlapped stent was thought to be an effective option, risks include exudation of fragile plaque,[20] promotion of thrombus formation,[21] and prolongation of postoperative hypotension.[22] Recurrence of fragile plaque and acute thrombus formation are expected to require further surgical treatment. Prolonged hypotension increases the risk of myocardial infarction, heart failure, or stroke. These are major risks for a decline in the patient's activities of daily living. In addition, PTA alone is not risk free; balloon dilation for progressive in-stent protrusion was reported to result in complete occlusion, and aspiration removal was performed.[1] Plaque aspiration can avoid the disadvantages of these conventional strategies and was considered a technically feasible treatment. However, several risks are associated with plaque disruption. First is the promotion of delayed thrombosis. If the plaque outside the stent is aspirated, it is possible that thrombus formation progresses around the damaged plaque, which may become like an ulcer. Since this may cause serious intraoperative and postoperative thrombosis, the following precautions should be taken during aspiration. Aspiration should be performed while maintaining antegrade or retrograde blood flow within the stent to prevent the stent from shrinking. Additionally, aspiration should be performed while manipulating the catheter parallel to the stent so that the catheter tip does not aspirate the stent wall. Second is the risk of intraoperative embolism via ECA–ICA or ECA–vertebral arteries. Consequently, plaque removal was planned while preserving either anterograde or retrograde blood flow to the ECA for the same reason mentioned above. Therefore, there was a risk of cerebral embolism through intracranial and extracranial anastomoses. In this procedure, there seemed to be no method to completely avoid this risk of embolism, so we planned sufficient intraoperative heparinization as a preventative measure. Considering the risks and benefits of each treatment, we chose to proceed with plaque aspiration, reserving PTA and overlapped stenting as a secondary option if the plaque could not be removed by aspiration.

Finally, there are few reports on aspiration for in-stent thrombus or plaque protrusion. Although it is sporadically observed in patients with complete occlusion,[1,21,23,24] there have been no reports of its use for non-obstructed in-stent protrusions. From the experience of this case, aspiration

removal of an in-stent mobile plaque with a thrombectomy catheter may a viable treatment option preceding balloon dilatation and overlapped stents. However, it should only be performed after carefully evaluating the risk of embolism associated with aspiration and other treatments. We believe that the current indication for this method is limited in patients with mobile in-stent plaque who have poor cardiac function.

Conclusion

A distal access catheter was used to aspirate the in-stent plaque protrusion that occurred early after CAS and the plaque was removed successfully. Aspiration removal was thought to be effective for this case of mobile in-stent plaque, which was expected to be fragile, while avoiding the disadvantages of increasing stents.

Acknowledgments

We would like to thank Editage (www.editage.com) for English language editing.

Disclosure Statement

The authors declare that they have no conflicts of interests.

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