CASE REPORT

Delayed stenosis following stentriever use in acute stroke intervention

Jeremy James Macke, Michael P Bellew, Frank R Hellinger Jr

Department of Radiology,
Florida Hospital, Orlando,
Florida, USA

Correspondence to

Dr F R Hellinger Jr, Department
of Radiology, Florida Hospital,
601 East Rollins Dr, Orlando,
FL 32803, USA;
fhellingerjr@gmail.com

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SUMMARY

We report two cases of delayed post-embolectomy stenosis—that is, a new stenosis in the vascular bed of a previous endovascular embolectomy. The first case was discovered incidentally in a patient returning after embolectomy for evaluation of cerebral aneurysms. The second case was discovered on an MR angiogram obtained to screen for stenosis. This was prompted by our experience with the first case. To our knowledge, this phenomenon has not been reported previously.

BACKGROUND

The use of stentriever is a relatively recent development in the treatment of acute strokes. With these devices, higher recanalization rates and reduced time to flow restoration have been achieved. Complications described in the literature have been limited to hemorrhages, thromboembolic events, and vasospasm.^{1–3} Clinical reports of stenosis have been described in 1–12% of cases following permanent implantation of stents with mechanical properties similar to stentriever.^{4–8} Microscopic narrowing has been noted after stentriever embolectomy in animal models.^{9–10} Furthermore, histopathological examination of swine after Trevo stent thrombectomy demonstrated severe disruption of the intima.^{1–10} However, no reports of stentriever related stenosis have been reported to date in humans.

CASE PRESENTATION

Stenosis after acute stroke intervention (ASI) with stentriever was identified in two patients at our

institution. The first, a right handed patient in their fifties without significant past medical history, presented as a wake-up stroke. On examination, the patient was awake, had a spastic quadriplegia, was non-verbal, and followed no commands. Pupils were 2 mm and equal and reactive; gaze was conjugate and midline. CT was negative for intracranial hemorrhage or established infarction. The patient underwent an ASI and was found to have an upper basilar artery occlusion beginning at the level of the superior cerebellar artery origins and extending to the P3 segment of the left posterior cerebral artery (figure 1A). A 7 Fr balloon guide catheter access was used in the left vertebral artery (LVA), the clot was laced with 5 mg of tissue plasminogen activator, and then an embolectomy was performed using a 4×20 Solitaire. After two passes, there was complete recanalization with restoration of Thrombolysis in Cerebral Infarction 3 flow (figure 1B). There was no underlying stenosis or evidence of intimal injury, such as dissection. The patient was discharged home without deficits on day 7 on aspirin and Zocor.

Initial angiograms also demonstrated bilateral carotid–ophthalmic artery aneurysms. The patient was seen in follow-up and was doing well. Following this, an angiogram was performed 5 months after the ASI to carefully evaluate the aneurysms. The right carotid angiograms demonstrated sustained filling of the upper basilar artery through a small posterior communicating artery. This prompted a vertebral injection which demonstrated a severe concentric stenosis of the basilar artery just above the left anterior inferior cerebellar artery origin, somewhat proximal to the level of

Figure 1 (A) Anteroposterior (AP) left vertebral artery angiogram shows upper basilar artery occlusion (arrow). (B) AP left vertebral artery angiogram after embolectomy shows complete recanalization and no underlying stenosis or intimal injury. (C) AP left vertebral artery angiogram 5 months after embolectomy shows new severe basilar stenosis (arrow).

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Figure 2 (A) Anteroposterior (AP) right vertebral artery angiogram showing left vertebral artery occlusion (arrow). (B) AP left vertebral artery angiogram after left vertebral artery embolectomy shows recanalization with no underlying stenosis or intimal injury. *Note:* The location of the vertebrobasilar junction (arrow) is defined by reflux into the right vertebral artery. (C) MR angiogram 6 months after left vertebral artery embolectomy shows new severe stenosis at the left vertebral artery terminus (arrow).

the initial occlusion (figure 1C). This finding led us to review our previous ASI patients to see if any of them had such an occurrence and also to consider screening subsequent patients for the development of such a stenosis.

Our second patient was a left handed patient in their fifties who presented with a minor posterior circulation stroke. Angiographically, a distal LVA occlusion and a hypoplastic distal right vertebral artery were identified (figure 2A). The patient was in atrial fibrillation and was felt to have had a cardioembolic LVA occlusion. Their symptoms suggested postural posterior circulation ischemia. An LVA embolectomy was performed several days after the acute event. An 8 Fr balloon guide catheter was positioned at the C3 spinal nerve level in the LVA. A 4×20 Solitaire was used in two passes. The distal tip was in the basilar artery for the first pass and in the LVA for the second. Subsequently, a Penumbra 0.041 aspiration system was used in the LVA and a final pass was done with a Solitaire 6×30 with the distal end deployed in the LVA proximal to the pica origin. Following this procedure, the LVA was widely patent with no evidence of intimal injury, such as dissection (figure 2B). The patient underwent an MR angiogram 6 months later which showed a severe (70+%) stenosis of the LVA at the vertebrobasilar junction (figure 2C).

INVESTIGATIONS

All 340 ASI cases performed by a single operator (FRH) between January 1, 2000 and June 30, 2013 were reviewed. Of these, 244 underwent an embolectomy as part of their intervention. Within this subset, stentriever were used in 41 patients. Subsequent cerebrovascular imaging performed at least 3 months after the ASI was available in 27 of these patients. None of these studies demonstrated a new intracranial stenosis in the vascular bed of the intervention. Cases during which an intracranial angioplasty and/or stenting was performed were excluded.

OUTCOME AND FOLLOW-UP

Our patients have been followed for a total of 13 and 8 months, respectively, are asymptomatic, and being treated medically.

DISCUSSION

The current study is limited in that only two cases are reported. Given that many thousands of patients have been treated with stentriever, the incidence of the phenomenon reported here may be small. Asymptomatic stenosis could be common and go undetected but then its clinical relevance is reduced. However, we believe these findings are likely to be of intellectual interest to neuroendovascular practitioners and may be of clinical importance in young patients who experience good outcomes from their acute stroke. The mechanism by which these stenoses develop is unknown. While it is logical that microscopic damage and repair occurs, the embolectomy procedure may incite more trauma than has been thought. Both of the stenoses we observed were focal, severe, and involved only a small portion of the vessel exposed to the stentriever. It is interesting that both of these stenoses developed at a site in proximity to collateral inflow. In the first case, the basilar stenosis involved the basilar trunk above the anterior inferior cerebellar arteries but below collateral inflow from the posterior communicating–P1 connection. In the second case, the stenosis was just below the inflow from the contralateral vertebral artery. Perhaps changes in the arterial wall begin during the period of the vessel occlusion and have little or nothing to do with the embolectomy per se.

While the mechanism and incidence of delayed post-embolectomy stenosis reported here remains unclear, we believe this observation is potentially important to patients enjoying a good outcome after embolectomy.

Learning points

- ▶ The incidence of stentriever associated stenosis is unknown.
- ▶ Follow-up imaging to look for post-embolectomy stenosis may be warranted in selected patients.
- ▶ Further studies with longer clinical follow-up are needed to elucidate the incidence and significance of post-embolectomy stenosis.

Contributors The three authors are justifiably credited with authorship, according to the authorship criteria. FRH Jr conceived of the study. JJM and FRH Jr initiated

the study design and MPB helped with implementation. All authors contributed to the interpretation of the data. FRH Jr and JJM critically reviewed the manuscript. All authors approved of the final manuscript.

Competing interests None.

Patient consent Obtained.

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