Extensive Mobile Thrombus of the Internal Carotid Discovered After Intravenous Thrombolysis: What Do I Do Now?

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Abstract

This case report describes a rare presentation of ischemic stroke secondary to an extensive internal carotid artery thrombus, subsequent therapeutic dilemma, and clinical management. A 58-year-old man was administered intravenous (IV) thrombolysis for right middle cerebral artery territory ischemic stroke symptoms. A computed tomography angiogram of the head and neck following thrombolysis showed a longitudinally extensive internal carotid artery thrombus originating at the region of high-grade calcific stenosis. Mechanical embolectomy was deferred because of risk of clot dislodgement and mild neurological symptoms. Recumbency and hemodynamic augmentation were used acutely to support cerebral perfusion. Anticoagulation was started 24 hours after thrombolysis. Carotid endarterectomy was completed successfully within 1 week of presentation. Clinical outcome was satisfactory with discharge modified Rankin Scale score 0. A longitudinally extensive carotid artery thrombus poses a risk of dislodgement and hemispheric stroke. Optimal management in these cases is not known with certainty. In our case, IV thrombolysis, hemodynamic augmentation, delayed anticoagulation, and carotid endarterectomy resulted in a favorable clinical outcome.

Keywords

stroke, cerebrovascular disorders, carotid artery thrombosis

Introduction

Free-floating carotid thrombus is rarely a documented cause of stroke but when found poses a therapeutic dilemma. Originating from the extracranial internal carotid artery, this entity has been previously defined as an “elongated thrombus attached to an arterial wall.”¹ Thrombolysis of a large, mobile clot poses a theoretical risk of stroke worsening from incomplete fibrinolysis, detachment from its origin, and subsequent large-vessel occlusion. However, vessel imaging is not always performed prior to thrombolysis, so in many cases, these are only found after thrombolysis has already been given. The optimal management acutely of an extensive intraluminal carotid thrombus is not known with certainty, but existing case series suggest successful management with anticoagulation followed by carotid surgery.^{2,3} Hemodynamic augmentation in acute ischemic stroke can be used in settings of perfusion dependence⁴ and specifically carotid T occlusion.⁵

field disturbance while driving to work. He was brought to an emergency department where a head computed tomography (CT) was negative for signs of ischemia or hemorrhage. A telestroke consultation commenced, and the patient was found to have moderate stroke symptoms with the National Institute of Health Stroke Scale (NIHSS) score of 6 (+1 hemianopia, +2L face, +2L arm, +1 dysarthria). He was hypertensive with systolic blood pressure greater than 200 mm Hg. After the review of contraindications, discussion of risks and benefits, and reduction of systolic blood pressure to less than 185 mm Hg, he was treated with intravenous (IV) recombinant tissue plasminogen activator (rtPA), approximately 1 hour after symptom onset. He was transferred to a tertiary facility for continued management. Of note, on a review of systems, the patient had given a several-month history of episodic and transient right-sided painless monocular vision loss.

Case History

A 58-year-old man with no known medical history developed sudden-onset left-sided weakness, facial droop, and visual

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Upon arrival to our facility, his NIHSS score was 1 for a mild left facial palsy. He underwent CT angiography (CTA) because of the history of right-sided visual symptoms and new left-sided deficits. He developed a recrudesence of his deficits while upright in the emergency department awaiting the scan. He was laid supine, and after several minutes, his deficits improved back to his previous baseline. The CTA showed a high-grade calcific stenosis of the right internal carotid artery distal to the bifurcation with longitudinally extensive intraluminal thrombus terminating at the petrous junction (Figure 1A-D). This case was reviewed by the neurointerventional team who deferred mechanical thrombectomy given the risk of clot dislodgement. In consideration of anticoagulation, the patient underwent urgent magnetic resonance imaging with diffusion-weighted sequences, which revealed a mild burden of right middle cerebral artery territory infarction (Figure 2A). Anticoagulation was deferred for 24 hours because of recent IV thrombolysis and risk of hemorrhagic transformation. The patient demonstrated perfusion-dependent changes in his clinical examination, which responded to volume replacement and hemodynamic augmentation with phenylephrine to a goal systolic blood pressure of 160 to 185 mm Hg. After 24 hours from symptom onset, anticoagulation with IV unfractionated heparin was started. At 48 hours, repeat magnetic resonance imaging was remarkable for additional embolic infarctions (Figure 2B) and resolution of the extensive intraluminal internal carotid artery thrombus (Figure 2C). Phenylephrine was weaned, and he was transitioned to dual-antiplatelet therapy in advance of definitive carotid surgery. He underwent an uncomplicated carotid endarterectomy 8 days after symptom onset. Intraoperatively, he was noted to have a large ulcerated plaque at the sight of the stenosis. At 1 month, he had no neurological symptoms referable to his stroke. The patient provided verbal consent to the author Scharf for the publication of this report.

Discussion

This case describes the successful management of a unique presentation of ischemic stroke caused by a large artery embolus with residual longitudinally extensive thrombus. Presumably, given his history of right-sided visual loss, he had a long-standing carotid stenosis and subsequently developed an acute occlusion with embolic events at the time of his stroke symptoms. Administering IV thrombolysis within 1 hour of symptoms may have led to partial dissolution of the thrombus, leading to the residual intraluminal clot. Our knowledge of clinical outcomes associated with IV thrombolysis in the setting of free-floating carotid thrombus originates from case reports and 7 cases included in a retrospective of 409 patients receiving IV rtPA.⁶ In the 11 total cases described in the literature, 1 patient was associated with worsening after IV thrombolysis.

Figure 1. Computed tomography angiography. A high-grade calcific stenosis was present in the right internal carotid artery just distal to the bifurcation on axial sequencing (blue arrow, A). Emanating from the stenosis was an intraluminal thrombus (blue arrow, B). The intraluminal clot was longitudinally extensive as seen on sagittal views (blue arrows, C) and terminated at the petrous junction (blue arrow, D).

This case highlights the controversy over whether anticoagulation may still play a role in an acute stroke. Anticoagulation for acute stroke, a formerly common practice, is intended to halt progression of thrombus and reduce early recurrent embolism. However, large clinical studies did not show a benefit in reducing stroke recurrence and actually have repeatedly shown an increased risk of hemorrhagic transformation. As such, current guidelines recommend against acute anticoagulation for acute stroke.⁷ However, these aggregated patients with stroke show different mechanisms, and there may still be individual circumstances not well studied on their own, in which a clinician may feel the potential benefits outweigh the risks (eg, intracardiac thrombus, acute and extensive intra-arterial thrombi). The case series of free-floating carotid thrombus causing acute stroke^{1-3,8} report no adverse events from early anticoagulation even in severe strokes (eg NIHSS 14), but a publication bias almost certainly exists and the true risk is unknown.

Additionally, administration of any antithrombotic is contraindicated for 24 hours following thrombolysis because of the National Institute of Neurological Disorders and Stroke protocol,⁸ however, the true time range of increased susceptibility to bleeding following IV fibrinolysis is unknown and possibly less than traditionally considered, at

Figure 2. Magnetic resonance imaging. Three representative diffusion-weighted slices completed within hours of symptom onset show hyperintense acute infarctions with embolic appearance in the distribution of the right middle cerebral artery (A, arrows). Repeat diffusion-weighted imaging completed at 48 hours shows development of interval ischemic infarctions consistent with continued embolization (arrows, B). Magnetic resonance imaging with 2-dimensional time-of-flight sequencing completed at 48 hours shows focal carotid stenosis (arrow) and the intraluminal clot is not seen (C).

least in mild strokes. There are no reports of IV rtPA in the setting of acute stroke and free-floating thrombus so the risk of clot dislodgment is not known but still must be considered. Further research might reveal a narrower time window of increased hemorrhagic risk following thrombolysis, thus allowing for earlier antithrombotic administration in a subset of patients.

Other aspects of the case management relate to the role of endovascular treatment and hemodynamic augmentation. Mechanical thrombectomy was considered but deferred after review of the extensive but nonocclusive nature of the thrombus, the patient's mild neurological deficits, and the wish to avoid manipulation of the clot with respect to a perceived high risk of further embolization. If the patient had clinically deteriorated, he would have been taken urgently to attempt mechanical thrombectomy. Because the patient developed worsening of his symptoms with upright posture, cerebral perfusion was augmented for 48 hours with maintenance of adequate intravascular volume and IV vasopressors, with good response. The patient had a good clinical outcome with delayed heparinization and carotid endarterectomy. In summary, the case highlights the extremely complex nature of the

management of acute stroke with a free-floating carotid thrombus.

Declaration of Conflicting Interests

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