Management and outcomes of carotid artery extension of aortic dissections

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ABSTRACT

Background: Aortic dissection (AD) is the most common aortic catastrophe. Carotid artery dissection due to extension of AD (CAEAD) is one severe complication of this condition. Despite years of refinement in the techniques for repair of AD, the optimal management strategy for CAEAD remains yet to be described. We hypothesized that CAEAD eventually resolves on antiplatelet therapy with a low but not insignificant risk of cerebrovascular accident (CVA).

Methods: This was a single-institution retrospective review of patients admitted with nontraumatic coincident aortic and carotid dissection between 2001 and 2013.

Results: CAEAD was present in 38 patients (24 men [53%]). The median age was 59.5 years (range, 25-85 years). A Stanford type A AD was diagnosed in 36 patients (95%). CVA or transient ischemic attack was identified in 11 patients (29%). Eight were potentially attributable to the carotid lesion. Two of these eight strokes resulted in death. Of the 11 CVAs and transient ischemic attacks, 8 were evident at presentation, 2 were diagnosed postoperatively during hospitalization, and 1 was diagnosed during early follow-up. Only one of these three postadmission strokes was attributable to the carotid lesion. Nonoperative management of aortic and carotid dissections was pursued in 9 patients (24%), 26 (68%) underwent open repair, and 4 (11%) had endovascular management of AD (2 thoracic endovascular aortic repair, 2 endovascular fenestrations), including 1 patient with a staged hybrid procedure (frozen elephant trunk). There were eight inpatient deaths (21%) and nine deaths in the follow-up period. Of the 30 patients who survived to discharge, 24 (80%) were managed with antiplatelet therapy. At a median follow-up of 14.5 months in 22 patients with follow-up computed tomography scans available, a minority of lesions had resolved, and only one CVA was reported.

Conclusions: This study found that CAEAD was associated almost exclusively with type A AD, was typically unilateral, most often on the left, and usually persisted at follow-up. Many CAEAD patients presented with CVA and experienced significant early mortality. Notably, not all CVA events were attributable to the CAEAD. CVAs were not common after admission, and there appeared to be a low risk of new or subsequent stroke during follow-up with routine antiplatelet and antihypertensive therapy. (J Vasc Surg 2017;■:1-9.)

Aortic dissections (AD) are the most common aortic catastrophe. Carotid artery extension of aortic dissection (CAEAD) is a potentially severe although relatively rare complication of AD. In fact, one of the most common causes of carotid artery dissection cited is AD.[1] Stroke, defined as a transient ischemic attack (TIA) or cerebrovascular accident (CVA), is seen in 6% to 16% of patients presenting with type A AD.[2-4] Isolated cervical carotid and vertebral artery dissections carry a <5% risk of stroke on medical management alone.[5] Despite a robust body of literature detailing the technical aspects of AD repair, the ideal management of CAEAD, whether surgical or medical therapy, is not well delineated.

Historically, data on CAEAD has been scarce, and most guidelines are derived from expert opinion based on case reports and single-institution series. This is likely due to the often asymptomatic nature of this condition and previously limited sophistication of imaging techniques. Prior studies have also lacked long-term follow-up specifically addressing late sequelae. Modern imaging, including high-definition computed tomography (CT) angiography and the more prevalent use of duplex ultrasound imaging, has improved the recognition and diagnosis of CAEAD.

Classically, early revascularization of CAEAD was advocated by multiple international case reports.[6-9] This was because of concern for recurrent CVA after the initial presentation, likely due to a belief that mural thrombus would result in carotid stenosis or distal thromboembolism. However, a recently published study advocated a strategy of aortic repair, followed by delayed carotid artery revascularization. The authors advocated that carotid repair was indicated only for recurrent symptoms attributable to CAEAD.[9] We hypothesize that CAEAD

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can be safely managed with antiplatelet therapy with a low risk of late CVA.

METHODS

This was a single institution analysis of a retrospectively collected database at an urban tertiary referral center. All data accrual was conducted under the University of Maryland Institutional Review Board protocol #HP-00049876, which waived consent. The database was developed by medical record abstraction for patients identified in administrative and billing data. The database included all patients diagnosed with any aortic pathology from 2001 to 2013. Patients were identified based on International Classification of Diseases, Ninth Revision, codes for thoracic aortic (441.01), thoracoabdominal aortic (441.03), and carotid artery (443.21) dissections. Cases of coincident aortic and carotid dissection were identified for review of electronic medical records, including all imaging reports, all CT scans, operative reports, and hospital dictations, to collect demographic, perioperative, and follow-up data. Patients were included if they had an initial contrasted CT study or clear documentation of the lesion at the time of operation by the attending surgeon to ensure accurate diagnosis of CAEAD. We excluded traumatic aortic and carotid injuries. Data concerning cerebrovascular ischemic events were obtained from clinic notes and imaging reports.

For the purpose of comparison, we identified four patient groups of interest: patients who did and did not survive the index admission, patients who survived and had interval CT follow-up (CTF) of their carotid lesions, and patients lost to CTF (LCTF) of their carotid lesions. Postdischarge mortality data were collected from commercially available databases that provided Social Security Death Index data through 2014 and allowed searching for published obituaries for patients not listed in the Social Security Death Index.

The Fisher exact test and Student *t*-test were used to compare groups where appropriate, with a P value of $<.05$ accepted as significant. Kaplan-Meier survival curves were generated for overall survival and survival in patients with and without CVA. The log-rank test was used to evaluate the significance of differences between survival curves.

RESULTS

We queried records for 3160 patients and found relevant diagnoses present in 453 patients, including 358 patients with thoracic AD and 95 with thoracoabdominal AD. CAED was identified by CT in 38 of these patients (8.4%).

The CAEAD group was 63% male (n = 24), 66% Caucasian (n = 25), 32% African American (n = 12), and was a median age of 59.5 years (range, 25-85 years). Comorbidities included hypertension (30 [79%]), tobacco use (18 [47%]), coronary artery disease (8 [21%]), and hyperlipidemia (8 [21%]). A family history of Marfan syndrome was rare (2 [5%]), and no patients bore a connective tissue diagnosis at the time of their index admission. Differences between the demographics and comorbidity profiles of survivors and nonsurvivors of the index admission were not significant. Complete demographics are reported in Table I.

Patient and lesion characteristics are outlined in Table II. In short, 36 (95%) of the included patients were diagnosed with a Stanford type A dissection as the primary pathology. Two patients had Stanford type B lesions with retrograde extension of the dissection that involved their left common carotid arteries. The respective attending surgeons at the time determined that the patients' primary disease involved the descending aorta and that the proximal extension into the base of the left common carotid artery did not constitute involvement of the ascending arch and so the patients were managed as type B ADs. There were 17 patients (44.7%) with left common carotid artery dissections, 9 (23.7%) with right, and 12 patients (31.6%) with bilateral involvement. The differences between subgroups were not significant. Most patients, 32 of 38 (84%), arrived as transfers from other medical facilities. The most common presenting symptoms were chest, back, or neck pain, seen in 27 patients (71%), lower extremity malperfusion (10 [26%]), and stroke (7 [18%]). The analyzed subgroups were not significantly different (Table II).

Management strategies are reported in Table III, and relative distribution is seen in Fig 1. Of the 38 patients with CAEAD, 26 (68%) underwent open aortic repair, and four (11%) had endovascular management. Nonoperative management of AD in the setting of a diagnosis of CAEAD was pursued in nine patients (24%). In only eight patients (21%) undergoing open repair was great vessel replacement or bypass performed. One patient, with bilateral CAEAD, had a later right-sided carotid stenting procedure for >95% stenosis on interval imaging. The endovascular procedures included two endovascular fenestrations (5%) with branch vessel stenting, and thoracic endovascular aortic repairs in two patients (5%). One of these aortic stenting procedures was performed as part of a staged frozen elephant trunk hybrid procedure.

We found no significant difference in treatment patterns between survivors and nonsurvivors. There were, however, some notable differences in the distributions of interventions, with relatively more nonoperative management (63% vs 47%) and less open repair (38% vs 86%) in the LCTF group compared with the CTF group (Table III and Fig 1).

Nonoperative management was pursued for a variety of reasons. This was most often (5 [55%]) because of prior aortic intervention, including three patients with chronic type A dissections (2 after repair >5 years earlier at another center). These three patients had newly diagnosed extension into the carotids that was found incidentally during evaluation for other conditions. The other two patients had recent repair of ascending AD