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Review Article

Systematic Review of Common Carotid Artery Dissection of Aortic Origin

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Abstract

Objectives: The management of common carotid artery dissection (CCAD) extending from acute aortic dissection is controversial. This systematic review examines the literature on CCAD secondary to aortic dissection and the association with stroke.

Methods: MEDLINE and EMBASE databases were searched using multiple interfaces, including Ovid: Medline, ProSearch, PubMed/PMC, Cochrane Library, Scopus, Google Scholar, and EBSCOhost. All documented cases with an abstract written in English were extracted. Additionally, manual reference search was performed and quality of evidence was assessed.

Results: A total of 165 articles, presenting 374 individual patients, were included. The main endpoints of interest included mortality and postoperative neurologic deficits. Thirty-two articles reported carotid artery interventions performed before or after aortic repair. Overall reported stroke incidence after aortic repair was 19.13% and five-year neurologic event-free survival was 54.5%. Overall five-year survival was 71.5%.

Conclusions: We did not find evidence to suggest a difference in outcomes for those who underwent intervention for CCAD prior to aortic repair compared to those patients who did not. There is a need for a high index of suspicion for recognition, timely diagnosis, and early repair of proximal aortic dissection to improve the prognosis of these high-risk patients.

Keywords: Common carotid artery; Dissection; Aortic dissection; Stroke in young adults; Systematic rev

Introduction

Carotid artery dissections represent the second leading cause of stroke in adults under the age of 45 [1,2]. The most frequent causes involve trauma or spontaneous dissection of internal and external carotid arteries, and sometimes involvement of the common carotid arteries. However, common carotid artery dissection (CCAD) due to an extension from proximal aortic dissection is a significant cause of stroke in adults that is rarely recognized as a separate entity.

Acute aortic dissection is a cardiovascular emergency and remains the most common cause of mortality due to aortic reasons [3]. Current estimates show an incidence of 5-30 cases of acute aortic dissection per million people per year in the United States [3-5]. Twenty percent die before reaching the hospital, with a risk of death in the surviving population of 1-4% per hour in the first 48 hours if left untreated [5]. Nearly 50% die within 24 hours and approximately 70% are dead within 48 hours of presentation [5]. Malperfusion is an independent perioperative risk factor for mortality and morbidity. Acute aortic dissection is complicated by cerebral malperfusion and stroke in 6%-20% of cases [3,6]. Data shows an 18.6% rate of stroke at presentation in those with CCAD vs. 8.1% in those without extension of aortic dissection into their carotid arteries [7]. However, it is unclear if the risk of postoperative stroke following aortic repair in those with CCAD is equally tangible as their risk of stroke at initial presentation.

The management of CCAD after or concomitant to acute aortic dissection is controversial. Although several reports describe concomitant endovascular repair of CCAD after open ascending aortic repair, others advocate a trial of medical management along the lines of management of traumatic or spontaneous CCA dissections. Some propose early carotid revascularization for CCAD of aortic origin prior to instituting a trial of medical therapy [8-10].

Some suggest a carotid artery intervention in the form of angioplasty and stenting at the time of aortic repair or in the immediate postoperative period [8,11-13]. Contrary to this approach, other authors recommend urgent repair of acute type A aortic dissection without preoperative carotid revascularization [7,14].

Medical management forms the mainstay of therapy for traumatic and spontaneous dissections and entails administration of antiplatelet and anticoagulant. The effectiveness of medical management in CCAD of aortic origin is, however, unknown. A systematic review of published studies on traumatic and spontaneous cerebrovascular dissections showed that the risk of recurrent stroke or transient ischemic attack in patients managed with medical therapy is less than 5% [15], but the natural history and risk of stroke in CCAD of aortic origin patients is still unclear. To the best of our knowledge, this is the first systematic review in the literature that examines CCAD of aortic origin as an independent entity.

Methods

Search Strategy

Because there are no randomized controlled trials or systematic reviews of literature on the topic, this review includes only observational studies. All descriptive and observational studies and reports in the online/print journals or forums worldwide that are indexed in the MEDLINE and/or EmBase databases were searched using five portals: OvidMedline, PubMed Central (PMC), Cochrane library, Scopus, Medline and EmBase, and EBSCOhost. Studies of patients with CCAD and acute aortic dissection were selected. The key terms used under all text fields for the search included "common carotid artery", "dissection", "aortic dissection", "carotid dissection", "carotid involvement" and "common carotid artery dissection". No restrictions on language or publication period were applied. Abstracts were included if all relevant information on presentation, diagnosis and management were available. Additionally, a manual bibliographic search of all included studies was performed. Detailed inclusion and exclusion criteria are available online (appendix I). (Figure 1) presents the pictorial view of the study methodology per Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.

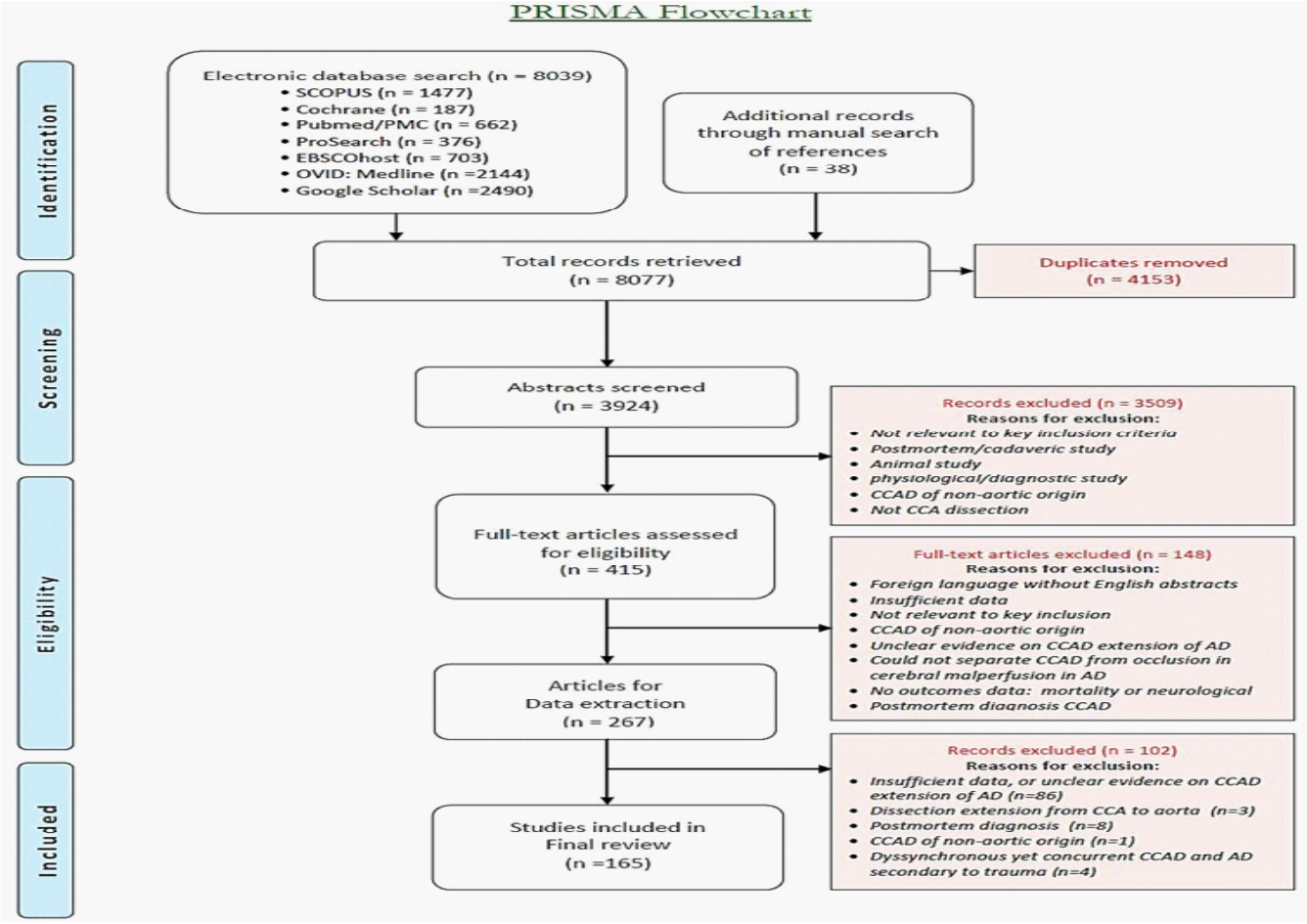


Figure 1: PRISMA flow diagram summarizing the selection, inclusion and exclusion of the reviewed studies.

Data Extraction

Two reviewers independently assessed the studies for eligibility, study quality and evidence synthesis. A third reviewer resolved any disagreement over the eligibility of a study. Extracted data points included: study design; case definition and patient demographics, presentation characteristics, proximal aortic repair, operative interventions for CCAD, anticoagulant and/or antiplatelet therapy, postoperative complications, postoperative neurological deficits; outcomes and times of measurement; follow-up time, if available; and information for assessment of the risk of bias (Table II)

Study	# Patients	Preop Stroke	Prox Aortic Repair	Mortality	New Postop ND/Stroke	ASA or anti-coag	CCAD Intervention
Case Reports*	140	62	75	25†	22‡	23§	30†
Guthaner 1979	3	.	3	0	3	0	.
Symbas 1980	2	1	2	0	0	0	0
Zurbrugg 1988	16	.	16	0	9 (4AF, 4TIA,1St)	7+2||	0
Bluth 1989	2	.	2	.	.	.	.
Fann 1989	6	6	6	1	0	.	0
Dany 1990	12	.	.	.	.	.	0
Jeng 1994	3	0	1	0	1	.	.
Hughes 1995	2	1	2	0	0	0	0
Zielinski 1999	15	1	15	0	0	0	0
Chen 2002	8	.	.	0	.	.	.
Htay 2003	2	.	2	0	0	.	0
Gaul 2004	2	0	2	0	0	.	0
Neri 2004	49 (42 cases + 7)	33 (26 cases + 7)	49	Mar-42	13/42	13	12
Panos 2005	2	0	2	0	0	0	0
Tanaka 2005	2	0	2	0	0	0	0
Sojer2007	3	1	2	1	1	0	0
Iguchi 2010	2	2	1	2	0	0	0
Tsukube 2011	21	.	17	.	.	.	0
Seliger 2011	3	1	3	0	0	0	0
Nakamura 2011	7	7	7	1	0	0	0
Folgoas 2012	2	2	1	2	0	1	0
Igarashi 2013	2	0	2	1	0	0	2
Orihashi 2013	3	2	3	0	0	0	0
Lee 2013	22	6	.	.	8	0	0
Charlton-Ouw 2013	43	8	43	13	6	27	0
Totals	**374**	**133**	**236**	**49**	**63**	**73**	**44**

Table II: Summary of Literature Review Data.

*All papers reporting one relevant patient with CCAD of aortic origin. Reports on 2 or more patients were considered a series.
†Data missing in 4 (of 140) case reports
‡Data missing in 5 (of 140) case reports
§Data missing in 11 (of 140) case reports
||7 Post-aortic repair anticoagulants and 2 post-ND development

AF – Amaurosisfugax; TIA – transient ischemic attack;– unknown due to unavailable full-text; St – Stroke; ND – neurological deficit; Pts – patients; Preop – preoperative; Prox – proximal; Postop – postoperative; and ASA – aspirin/acetylsalicylic acid

Quality Assessment

The data was cross-checked and validated using the PRISMA [16,17] guidelines and the quality of evidence was assessed based on Grading of Recommendations Assessment, Development and Evaluation (GRADE) [18] criteria before pooling all collected data into a single review database. The (appendix II) presents a summary of quality assessment on each included study.

Data Analysis

Individual level data was included for analyses from each of the included studies, structured around patient characteristics, intervention content and outcomes, whenever possible. Given the fact that the information presented in these articles was often heterogeneous and incomplete with varying follow-up time, there was a limited scope for meta-analysis. We synthesized and reported data as pooled analysis based on Meta-Analysis of Observational Studies in Epidemiology (MOOSE) reporting guidelines [19]. We provided aggregate descriptive summaries for each outcome (late neurological deficit, risk of stroke, stroke recurrence, and overall mortality) and intervention (CCAD and/or medical therapy) across each study as mean or percent of events reported. A pooled data analysis to analyze the overall recurrent neurologic deficit rate, mortality rate, survival and neurologic deficit-free survival was performed. No attempt was made to impute missing data. Statistical computations were performed using SAS 9.3 (SAS Institute Inc, Cary, NC).

Results

The initial search yielded 415 papers spanning from 1938-2013. Of these, 267 articles were retrieved for data extraction after exclusion criteria were applied during full-text review of the articles as shown in the PRISMA flowchart (Figure1). During data extraction, an additional 102 articles were excluded due to insufficient data, unclear evidence on CCAD traceable to aortic dissection, and other reasons. Four excluded articles presented patients with CCAD along with a concurrent aortic dissection due to iatrogenic or traumatic etiology [20-23].

The final review included 165 papers (please see appendix) presenting 374 individual patients with CCAD of aortic origin. Of these, 140 were case reports on a single patient with 25 other observational studies documenting 2 or more patients. Stroke on admission was reported in 43.2% (n=315). Ascending aortic repair was performed in 90.2% (n=327). There was a 67.9% mortality reported in those who did not undergo aortic repair (19/28 without aortic repair) and 12.2% (33/271) reported mortality among those who had aortic repair, although long-term follow-up was not always reported. A new neurological deficit after aortic repair was reported in 19.1% (n=324); 14.0% (19/136) of patients had some postoperative residual neurological deficit from the stroke at initial presentation even after aortic repair; and 22.1% of patients were given medical therapy consisting of aspirin or anticoagulants. (Tables I and II) provide a summary of available individual data characteristics collected from the included studies.

Characteristic	CCAD Int	No CCAD Int	Overall
	(n=44)	(n=281)	(n=374)
Demographics Age	56.79	56.72	57.02
	(n=42)	(n=259)	(n=350)
Males	21* (70%)	131 (66.8%)	186 (66.4%)
	(n=30)	(n= 196)	(n=280)
Females	9* (30)	65 (33.2%)	94 (33.6%)
	(n=30)	(n=196)	(n=280)
Presentation		51 (39.8%)	60 (28.8%)
Chest pain	8 (19.5%) (n=41)	(n=128)	(n=208)
Abdominal pain	0 (0%)	8 (6.3%)	8 (3.8%)
	(n=41)	(n=128)	(n=208)
Back pain	10 (24.4%)	20 (15.6%)	30 (14.4%)
	Back pain	(n=41)	(n=128)
Pulse/pressure	6 (15.8%)	67 (37.2%)	73 (28.4%)
deficit	(n=38)	(n=180)	(n=257)
AMS/LOC	11 (26.8%)	59 (46.5%)	72 (34.6%)
	(n=41)	(n=127)	(n=208)
Stroke on admission	10* (32.3%)	88 (39.5%)	136 (43.2%)
	(n=31)	(n=223)	(n=315)
Dissection	(n=32*)	(n=257)	(n=341)
RCCAD	16 (50%)	91 (35.4%)	131 (38.4%)
LCCAD	7 (21.9%)	81 (30.7%)	106 (31.1%)
Bilateral	9 (28.1%)	85 (33.1%)	104 (30.5%)
Treatment		32 (11.4%)	32 (9.8%)
	0 (0%)	(n=281)	(n=327)
	(n=44)		
Prox. aortic repair	44 (100%)	243 (86.5%)	295 (90.2%)
	(n=44)	(n=281)	(n=327)
rt-PA	0 (0%)	17 (8.2%)	17 (7.02%)
	(n=44)	(n=207)	(n=242)

Table I: Patient Characteristics.

ASA/Anticoag	22 (51.2%)	51 (20.6%)	73 (22.1%)
	(n=43)	(n=248)	(n=330)
Outcomes		38 (15.6%)	62 (19.1%)
Post aortic repair neuro-deficit	24 (55.8%)	(n=243)	(n=324)
	(n=43)		
In-hospital mortality	1 (2.3%)	21 (10.5%)	22 (8.1%)
	(n=44)	(n=200)	(n=272)
Overall mortality	6 (2.3%)	46 (16.8%)	52 (16.8%)
	(n=43)	(n=274)	(n=310)

CCAD – common carotid artery dissection; Int – intervention; AMS – altered mental status; LOC – loss of consciousness; RCCAD – right-sided common carotid artery dissection; LCCAD – left-sided common carotid artery dissection; rt-PA – recombinant tissue plasminogen activator; ASA – aspirin/acetylsalicylic acid; Anticoag – anticoagulation; prox. – proximal.

On aggregate, 47% of the patients were reported to have chest pain, abdominal pain, and/or back pain. However, the rest of the patients presented as painless aortic dissections. Pulse or pressure deficit with or without hypotensive shock was present in 28.4% of the patients (n=257). In addition, altered mental status and loss of consciousness was frequently reported as well. Of the 238 patients with reported presenting complaints, some had unusual symptoms. Eleven (4.6%) patients complained of headache on admission [24-34]. The second most frequently reported unusual symptom was visual disturbances that included visual field defects, blurring of vision, amaurosis, postoperative ischemic optic neuropathy, and visual features of stroke (homonymous hemianopsia, conjugate deviation of eyes) presenting without paralytic or paretic symptoms [24,25,27,29,33,35-40]. Another interesting manifestation was that of transient global amnesia reported in 4 patients [41-43]. Seventeen patients who presented with stroke received thrombolytic therapy on admission due to lack of suspicion of aortic origin at the time of presentation. Of these, 12 underwent emergent aortic repair after diagnosis of CCAD of aortic origin was established and thrombolysis infusion was stopped. There was no reported mortality in these patients. However, 4 out of 5 patients who did not undergo surgical repair, either due to refusal of surgery or because of delay in diagnosis, died soon after or during thrombolysis infusion.

Thirty-two articles (44 patients from 30 case reports and 2 case series) reported a common carotid artery intervention performed before, after or concomitant to primary aortic repair. (Table III) summarizes the key data points on outcomes and details of these interventions. Aspirin or anticoagulant therapy was initiated in 19/44 (43.2%) prior to CCAD intervention. Four (9.1%) patients had persisting/residual neurological deficits after CCAD and aortic interventions [38,44-46]. Twenty-three of these 44 patients (52.3%) underwent carotid artery intervention after the aortic repair. All of these patients were symptomatic and presented with a postoperative neurological deficit. Only 1 patient died in this group [38]. Six reports describe CCAD intervention prior to proximal aortic repair and included 1 femoral-carotid bypass, 1 CCA percutaneous transluminal angioplasty, 2 subclavian-carotid bypassesand 2 femoral-carotid shunts. Of these, 1 patient died [47]. Fifteen patients had CCAD interventions performed at the time of aortic repair and included procedures such as carotid fenestrations, bilateral CCA reconstruction with arch replacement, CCA ligation, carotid-carotid bypass, subclavian-carotid bypasses, femoral-carotid shunts and bypasses, and endovascular stenting. Eleven endovascular stenting procedures performed after or at the time of aortic repair were described (Table III). One mortality was reported in these patients. However, data was not always available on neurological status improvement owing to lack of long-term follow-up.

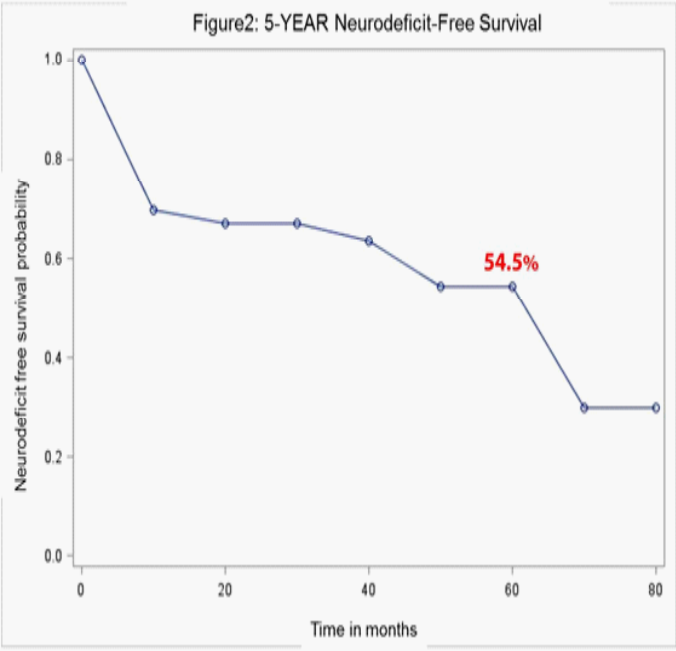
Article (32 papers)	# Patients	Preop Stroke	Mortality	New Postop ND/Stroke	ASA or anti-coag	Intervention for CCAD
Walterbusch 1984	1	N	Y	Y	Y	Fem-R CCA bypass, preop
Schievink 1994	1	N	1	Y	Y	R SCA-ICA bypass, L ICA interposition bypass postop
Nomoto 1997	1	N	N	N	N	b/l CCA reconstruction concomitant to aortic repair
Seelig 2000	1	.	N	Y	Y	R CCA ligation and car-car bypass postop
Kubota 2000	1	Y	N	N	N	R CCA PTA preop
Toda 2000	1	Y	N	N	N	R SCA-car bypass preop
Hama 2000	1	N	N	Y	Y	L CCA, L SCA, and IA stents postop
Shimazaki 2003	1	N	N	N	N	b/l CCA reconstruction concomitant to aortic repair
Shimazaki 2004	1	Y	N	N	N	ligation R CCA (no revasc) concomitant to aortic repair

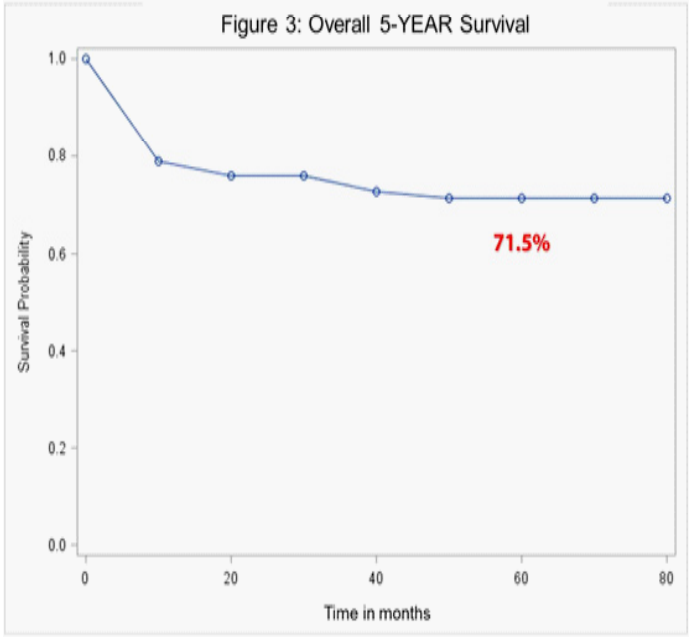
Pavkov 2004	1	.	N	Y	N	IA, R CCA/ICA stent postop
Imanaka 2004	1	Y	N	N	N	fem- R CCA shunt and aorto-R CCA by-pass concomitant to aortic repair
Neri 2004	12	.	N	12	12	4 pt with arch replace postop
						1 pt with fem-SCA bypass postop
						2 pt with carotid fenestration postop
						2 pt with carotid fenestration postop
						3 pt with stent (1 with R CCA/ICA and 1 with bilat CCA stent) postop
Eren 2005	1	Y	N	N	N	b/l CCA fenestration concomitant to aortic repair
Belov 2006	1	.	N	.	N	L SCA-car bypass preop
Sartipy 2006	1	N	N	Y	N	Ao-car bypass postop
Roseborough 2006	1	N	N	Y	N	R CCA/ICA stents postop
Cardaioli 2007	1	N	N	Y	N	Bilat CCA/ICA stent postop
Uyema 2007	1	Y	N	N	N	R CCA fenestration and R VA-ICA bypass concomitant to aortic repair
Schnoholz 2008	1	Y	1	N	N	fem-car shunt preop
Munakata 20091	1	Y	N	N	N	fem-car shunt preop
Lentini 2009	1	N	N	N	N	B CCA stents concomitant to aortic repair
Yoshida 2009	1	Y	N	N	N	R CCA/IA thrombectomy +/- fenestration concomitant to aortic repair
Umeda 2010	1	N	N	N	N	L CCA reconstruction via neck incision concomitant to aortic repair
Casana 2011	1	N	N	Y	N	R CCA/ICA stent postop
Chen 2011	1	N	N	N	N	Ao–R CCA bypass concomitant to aortic repair
Abe 2012	1	Y	N	N	N	Ao–L CCA bypass concomitant to aortic repair
Droeser 2012	1	N	N	Y	Y	R CCA fenestration and patch angioplasty postop
Elshikh 2013	1	N	N	Y	Y	R CCA/ICA stent and coil occlusion of FL postop
Matsuoka 2013	1	N	.	N	.	Ao–R CCA bypass concomitant to aortic repair
Rylski 2013	1	N	N	N	N	Ao–B CCA bypass concomitant to aortic repair
Alurkar 2013	1	N	N	Y	Y	R CCA/ICA stent postop
Igarashi 2013	2	N	1	N	N	CCA thrombectomy concomitant to aortic repair
Total	**44**	**10**	**3**	**23**	**19**	

Table III. Summary of CCAD interventions.

. – unknown due to unavailable full-text; ND – neurological deficit; Pts – patients; Preop – preoperative; ASA – aspirin/acetylsalicylic acid; Anticoag – anticoagulation; CCAD – common carotid artery dissection; Prox – proximal; Postop – postoperative; Fem-R CCA – femoral artery to right common carotid artery bypass; L CCA – left-sided common carotid artery dissection; R – right; L – left; SCA-ICA – right subclavian artery to internal carotid artery bypass; L ICA – left internal carotid artery ; rt-PA – recombinant tissue plasminogen activator; prox. – proximal; B – bilateral; car-car – carotid to carotid artery bypass; fem-car – femoral artery to common carotid artery; PTA – percutaneous transluminal angioplasty; IA – innominate artery; Ao-car – aorto-carotid; VA-ICA – vertebral artery to internal carotid artery bypass; +/- – with or without; Ao – aortic; FL – false lumen.

The overall reported incidence of neurological deficit after aortic repair was 19.1% and five-year neurologic event-free survival was 54.5% +/- 4.0% (Figure 2). The estimated median residual lifetime was 5.2 years initially, then gradually decreased to 2.4 years by the end of third year. The overall five-year survival was 71.5%(Figure 3). Although no postoperative neurological events were reported in patients who underwent an intervention for CCAD prior to proximal aortic repair (Table III), the heterogeneity in reported follow-up time limits the derivation of a statistical inference. We did not find statistically significant survival difference among those who underwent intervention for CCAD in addition to proximal aortic repair compared to those patients who were medically managed after aortic repair.





Discussion

CCAD presents a risk for stroke on initial presentation of acute aortic dissection. Nearly half of patients with aortic origin CCAD presented with stroke on initial presentation. We recently reported our experience on patients with aortic origin CCAD and compared them to patients with aortic dissection without CCAD [7]. Our results demonstrated a higher incidence of stroke on presentation in patients with aortic origin CCAD compared to those without CCAD (18.6% vs. 8.1%, p=0.05). Another series by Neri et al. on patients with residual brachiocephalic dissection of aortic origin following aortic repair also reported a high preoperative stroke rate (86%) [48].

Medical management with anticoagulation and antiplatelet therapy is the mainstay of treatment in traumatic and spontaneous carotid dissections. Kennedy et al. recently reviewed the literature and performed a meta-analysis to compare anticoagulation and antiplatelet for prevention of recurrent stroke after cerebrovascular dissections [15]. Their results show that the risk of recurrence of stroke or transient ischemic attacks following medical therapy was 2.3% and there was no difference (p = 0.29) between antiplatelet (7.5%) and anticoagulants (3.8%) in prevention of recurrent stroke. The natural history of aortic origin CCAD after aortic repair is less clear. Surgical management mainly involves repair and revascularization of the ascending aorta, often with hemi- or total arch replacement. However, some authors advocate early carotid revascularization before a trial of medical therapy and before or

concomitant to aortic repair. The most common documented reason for these procedures is as preemptive measure based on the assessment of the patient as high risk for postoperative neurological deficits. Elaborate shunt and cannulation methods performed as bailout procedure prior to or at the time of aortic repair have also been described [47,49,50]. Endovascular repair involving carotid angioplasty and stenting have also been reported [8,11-13,48]. Some advocate the use of carotid revascularization prior to or concomitant with central aortic repair (Table III) as an effective approach in patients with extended dissection to reduce postoperative risk for neurological complications and redo aortic repairs.

Based on our present results, the overall reported incidence of neurological deficit after aortic repair was 19.1% and the five-year neurologic event-free survival was 54.5%. Four major series in the literature that report long-term follow-up on 15 or more patients document an overall post-aortic repair stroke risk in patients with CCAD of 12.5%-31% [7,48,51,52]. Zurbrugg and colleagues reported two cases of (2/16 CCAD of aortic origin, 12.5%) neurological events in the postoperative period following aortic repair attributable to CCAD [51]. They concluded that the persistent dissection was not a risk factor for subsequent stroke. Moreover the authors speculated that anticoagulation therapy was perhaps not protective against postoperative neurological events. Similar conclusions were also provided by Zielinski and colleagues [52], as they reported absence of major neurological events in the 21-month follow-up period following aortic repair. In the study performed by our group [7], we observed 14.6% incidence of postoperative stroke in patients with CCAD of aortic origin following aortic repair compared to 10.3% in those without CCAD (P=0.442). Anticoagulation and/or antiplatelet therapy was given to 81.8% of these patients.

Contrary to these observations, Neri et al. report residual brachiocephalic arterial dissection as an independent risk factor for postoperative neurological events with a 4-fold increased risk of TIA or stroke in patients with aortic dissection [48]. They observed recurrent neurological symptoms in nearly 31% of cases with residual brachiocephalic arterial dissection despite medical management. Of these, 12 patients underwent 16 revascularization procedures for correction of their symptoms. No mortality or permanent neurologic deficit was reported in these repaired patients. Other similar reports on failure of medical therapy with postoperative neurovascular events requiring CCAD interventions are summarized in Table III .The pooled analysis demonstrates an overall occurrence of postoperative neurological deficits in reported cases of 19.1%. Results from our group reported a lower incidence and no recurrence of neurologic deficits during follow-up in patients with CCAD of aortic origin [7]. The overall rate of postoperative neurologic complications in patients following open type A aortic dissection repair ranges from 11.3-24.7% [7,53,54]. Therefore, at this time, there is no evidence available in support of performing carotid revascularization beyond the primary aortic repair over medical management with anticoagulation and or antiplatelet therapy.

The current literature is deficient in systematic and analytical studies on the current issue. However, based on the given results, the presence of CCAD poses a significant risk for stroke on initial presentation but does not necessarily increase the risk for postoperative stroke. The overall five-year survival and the five-year neurologic event-free survival was 71.5% and 54.5%, respectively. Currently, there is not enough evidence showing statistically relevant improved survival outcomes in those who underwent carotid artery intervention for CCAD when compared to those patients who had a primary aortic repair only. Moreover, reported data on postoperative events is insufficient to make a meaningful comparison on neurologic-event-free survival between those who had a CCAD intervention and those who did not.

Those with known or suspected preoperative CCAD should have a postoperative carotid duplex scan or CT aortic angiography for surveillance or follow-up imaging [55]. Unlike traumatic or spontaneous carotid dissections, there are no studies comparing the effectiveness of medical therapy in preventing stroke in aortic origin CCAD. However, it is reasonable to extrapolate that medical therapy may prevent a neurologic event. Until the results from randomized trials are known, medical therapy consists of either aspirin alone (81-325 mg daily), clopidogrel alone (75 mg daily), or full anticoagulation for 6 months after aortic repair and indefinite therapy in patients presenting with new symptoms after cessation of initial medical antithrombotic therapy. The patients who present with recurrent ischemic symptoms referable to CCAD on adequate medical therapy should be offered carotid revascularization. Both open and endovascular repairs options are feasible and depending on the patient's anatomy and extent of CCAD.

Limitations

One limitation of this study was the heterogeneity of information presented in these articles. Often the presented data was incomplete with varying follow-up times and inconsistency across studies, which made detection of interactions, correlations, risk stratification, and trends difficult. Although the existing literature does not show a clear-cut increased risk in postoperative stroke in patients with CCAD of aortic origin, stronger study designs and reports are needed to make firm conclusions on the subject. Also, there is a high likelihood of publication bias, since it is likely that patients with unfavorable outcomes were not reported. It is also impossible to comment on the exact incidence of CCAD of aortic origin, as the denominator is difficult to ascertain. Despite the potential biases and a limited scope for analysis of included observational studies, our study presents the first comprehensive review that can be tapped to reference available information from the world literature.

Conclusion

CCAD presents a significant risk for stroke on initial presentation, although it is not necessarily a risk for neurological events after aortic repair. The mere presence of CCAD or a residual dissection does not mandate operative repair of common carotid arteries. Extrapolating from the current guidelines on medical management for traumatic and spontaneous carotid dissections, treatment with antiplatelet agents or anticoagulation may be considered.

References

1. Stevic I, Chan HHW, Chan AKC (2011) Carotid artery dissections: thrombosis of the false lumen. Thromb Res 128: 317-324.
2. Lyrer P, Engelter S(2010) Antithrombotic drugs for carotid artery dissection. Cochrane Database Syst RevCD000255.
3. Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, et al. (2000) The International Registry of Acute Aortic Dissection (IRAD): New Insights Into an Old Disease. JAMA 283: 897-903.
4. Bickerstaff LK, Pairolero PC, Hollier LH, Melton LJ, Van Peenen HJ, et al. (1982) Thoracic aortic aneurysms: a population-based study. Surgery 92: 1103-1108.
5. Mészáros I, Mórocz J, Szlávi J, Schmidt J, Tornóci L, et al. (2000) Epidemiology and clinicopathology of aortic dissection. Chest 117: 1271-1278.
6. Cambria RP, Brewster DC, Gertler J, Moncure AC, Gusberg R, et al.(1988) Vascular complications associated with spontaneous aortic dissection. J. Vasc. Surg7: 199-209.
7. Charlton-Ouw KM, Azizzadeh A, Sandhu HK, Sawal A, Leake SS, et al. (2013) Management of common carotid artery dissection due to extension from acute type A (DeBakey I) aortic dissection. J. Vasc. Surg 58: 910-916.
8. Casana R, Tolva V, Majnardi a R, Bianchi PG, Addobati L, et al. (2011) Endovascular management of symptomatic cerebral malperfusion due to carotid dissection after type A aortic dissection repair. Vasc Endovascular Surg 45: 641-645.
9. Geirsson A, Szeto WY, Pochettino A, McGarvey ML, Keane MG, et al. (2007) Significance of malperfusion syndromes prior to contemporary surgical repair for acute type A dissection: outcomes and need for additional revascularizations. Eur. J. cardio-thoracic Surg 32: 255-262.
10. Seelig MH, SeeligG SK, Oldenburg WA (2000) Carotid-Carotid Bypass: An Excellent Procedure for the Treatment of Complex Extracranial Carotid Artery Disease.Vasc. Endovascular Surg34:607-612.
11. Lentini S, Tancredi F, Benedetto F, Gaeta R (2009) Type A aortic dissection involving the carotid arteries: carotid stenting during open aortic arch surgery.Interact. Cardiovasc. Thorac. Surg 8:157-159.
12. Roseborough GS, Murphy KP, Barker PB, Sussman M (2006) Correction of symptomatic cerebral malperfusion due to acute type I aortic dissection by transcarotid stenting of the innominate and carotid arteries.J. Vasc. Surg 44:1091-1096.
13. Cardaioli P, Rigatelli G, Giordan M, Faggian G, Chinaglia M, et al. (2007) Multiple carotid stenting for extended thoracic aorta dissection after initial aortic surgical repair. Cardiovasc. Revascularization Med 8: 213-215.
14. Estrera AL, Garami Z, Miller CC, Porat EE, Achouh PE, et al. (2006) Acute type A aortic dissection complicated by stroke: can immediate repair be performed safely? J. Thorac. Cardiovasc. Surg 132: 1404-1408.
15. Kennedy F, Lanfranconi S, Hicks C, Reid J, Gompertz P, et al. (2012) Antiplatelets vs anticoagulation for dissection: CADISS nonrandomized arm and meta-analysis.Neurology 79: 686-689.
16. Knobloch K, Yoon U, Vogt PM (2011) Preferred reporting items for systematic reviews and meta-analyses (PRISMA) statement and publication bias.J. cranio-maxillo-facial Surg 39: 91-92.
17. Moher D, Liberati A, Tetzlaff J, Altman DG (2009) Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement. J. Clin. Epidemiol 62: 1006-1012.
18. Atkins D, Best D, Briss PA, Eccles M, Falck-Ytter Y, et al.(2004) Grading quality of evidence and strength of recommendations. BMJ 328: 1490-1494.
19. Stroup DF, Berlin JA, Morton SC, Olkin I, Williamson GD, et al. (2000) Meta-analysis of observational studies in epidemiology: a proposal for reporting. Meta-analysis Of Observational Studies in Epidemiology (MOOSE) group. JAMA 283:2008-2012.
20. Zoffoli G, Saccani S, Larini P, Colli A, Gherli T (2006). Endovascular treatment of traumatic aortic dissection and innominate artery pseudoaneurysm. J. Trauma 61:447-450.
21. Methodius-Ngwodo WC, Burkett AB, Kochupura P V, Wellons ED, Fuhrman G, et al. (2008) The role of CT angiography in the diagnosis of blunt traumatic thoracic aortic disruption and unsuspected carotid artery injury. Am. Surg74:580–585; discussion 585-586.
22. Mitsos AP, Chantler J, Konstantinou E, Fotis T, Lambrinou E, et al. (2009) Right procedure, wrong organ, an unusual case report of aortic trauma in a multiple injured patient. Cases J2: 6795.
23. Doguet F, Canville A, Lebreton G, Clavier E, Benichou R, et al. (2011) Repair of intraoperative aortic dissection associated with malperfusion syndrome using a combination of open and endovascular techniques. J. Card. Surg 26: 212-214.
24. Estreicher M, Portale J, Lopez B (2013) Aortic dissection presenting as gait disturbance: a case report. Am. J. Emerg. Med 31: 262-269.
25. Fanelli F, Salvatori FM, Marcelli G, Bezzi M, Totaro M, et al.(2003) Type A aortic dissection developing during endovascular repair of an acute type B dissection. J. Endovasc. Ther10: 254-259.
26. Gleeson H, Hughes T, Northridge D, Prendergast BD (2000) Bulbar palsies and chest pain. Lancet 356: 826.
27. Jeng J-S, Yip P, Hwang B (1994) Ultrasonography of Common Carotid Artery Dissection Secondary to Aortic Dissection : Three Case Reports. J. Med. Ultrasound 2: 41-46.
28. Ko J-I, Park T (2014) Headache: a rare manifestation of Debakey type I aortic dissection. Am. J. Emerg. Med 32: 291.e5-e6.
29. Mathys J, Lachat M, Herren T (2004) Headache as a Manifestation of a Life-Threatening Vascular Disorder. Headache 44: 706-709.

30. Nohe B, Ernemann U, Tepe G, Ritz R, Bail D (2005) Aortic dissection mimicking subarachnoidal hemorrhage. Anesth Analg 101: 233-234.
31. Runyan D, Alexander P, Saba S (2010) Ascending aortic dissection with atypical presentation of headache. Texas Hear. Inst.J37: 614-615.
32. Stöllberger C, Finsterer J, Fousek C, Waldenberger FR, Haumer H, et al. (1998) Headache as the initial manifestation of acute aortic dissection type A. Cephalalgia 18: 583-584.
33. Chen CY, Lee FY, Kuo YL, Liu CF, Kung CT (2011) Mydriatic pupil as the presenting sign of aortic dissection. Am. J. Emerg. Med 29: 240e5-240e7.
34. Ramalingam VS, Sinnakirouchenan R, Sudhakar S, Brasch A V (2010) Acute ischemic stroke in aortic dissection: Case report and review of literature. Indian J. Med. Sci 64: 385-389.
35. Chen Y-W, Jeng J-S, Yip P-K (2002) Stroke in Patients with Common Carotid Artery Dissection Secondary to Dissecting Aortic Aneurysm: an Observational Vascular Imaging Study. J. Med. Ultrasound 10: 20-25.
36. Kanabuchi K, Yamaguchi M, Ueda T, Furuya H, Uchimua N (2011) Case of ischemic optic neuropathy developed eleven days after an aortic arch replacement. Tokai J. Exp. Cinical Med 36: 112-115.
37. Iguchi Y, Kimura K, Sakai K, Matsumoto N, Aoki J, et al. (2010) Hyper-acute Stroke Patients Associated with Aortic Dissection. Intern. Med 49: 543-547.
38. Schievink WI, Bjornsson J, Piepgras DG (1994) Coexistence of fibromuscular dysplasia and cystic medial necrosis in a patient with Marfan's syndrome and bilateral carotid artery dissections. Stroke 25: 2492-2496.
39. Seliger C, Turmanidze N, Schmid E, Wiethölter H, Boy S, et al. (2011) Three cases of stroke in patients with atypical presentation of type a aortic dissection--potential of neurosonography in the early diagnosis of atypical stroke. Ultraschall Med 32: 619-621.
40. Sojer M, Stockner H, Biedermann B, Spiegel M, Schmidauer C (2007) Common carotid dissection: a sign of emergency. Circulation 115: e181-e185.
41. Gaul C, Dietrich W, Tomandl B, Neundorfer B, Erbguth FJ (2004) Aortic dissection presenting with transient global amnesia-like symptoms. Neurology 2: 2442-2443.
42. Masoero M, Leotta G, Vischia F, Sechi A (2013) Transient global amnesia: an atypical presentation of acute aortic dissection. Emerg. Care J9: 11-13.
43. Colotto M, Maranghi M, Epifania A, Totaro M, Giura R, et al.(2011) Unmasking aortic dissection in patients of transient global amnesia: case report and diagnostic algorithm for the emergency department. BMJ Case RepEPub: p6.
44. Shimazaki Y, Minowa T, Watanabe T, Koshika M, Toyama H, et al.(2004) Acute aortic dissection with new massive cerebral infarction - a successful repair with ligature of the right common carotid artery. Ann Thorac Cardiovasc Surg 10: 64-66.
45. Munakata H, Okada K, Tanaka H, Yamashita T, Nakagiri K, et al.(2008) Acute dissection of the innominate artery: a case of report. Gen. Thorac. Cardiovasc. Surg 56: 131-133.
46. Imanaka K, Nishimura M, Masuoka A, Ogiwara M, Kato M, et al.(2004) Lethal thrombus in the carotid artery during operation for acute aortic dissection with cerebral malperfusion. Ann Thorac Surg 77: 1448-1449.
47. Schönholz C, Ikonomidis JS, Hannegan C, Mendaro E (2008) Bailout percutaneous external shunt to restore carotid flow in a patient with acute type A aortic dissection and carotid occlusion. J. Endovasc. Ther 15: 639-642.
48. Neri E, Sani G, Massetti M, Frati G, Buklas D, et al. (2004) Residual dissection of the brachiocephalic arteries: significance, management, and long-term outcome. J. Thorac. Cardiovasc. Surg 128: 303-312.
49. Reeps C, Gee M, Pelisek J, Maier A, Gurdan M, et al. (2010) Interaction of biomechanics and metabolic activity in abdominal aortic aneurysm wall.World Congress on Medical Physics and Biomedical Engineering, September 7 - 12, 2009, Munich, Germany. In: Dössel O, Schlegel WC, editors. Springer Berlin Heidelberg. p. 659-661.
50. Göbölös L, Philipp A, Foltan M, Wiebe K (2008) Surgical management for Stanford type A aortic dissection: direct cannulation of real lumen at the level of the Botallo's ligament by Seldinger technique. Interact. Cardiovasc. Thorac. Surg 7: 1107-1109.
51. Zurbrugg HR, Leupi F, Schupbach P, Althaus U (1988) Duplex scanner study of carotid artery dissection following surgical treatment of aortic dissection type A. Stroke 19: 970-976.
52. Zieliński T, Wołkanin-Bartnik J, Janaszek-Sitkowska H, Biederman A, Rynkun D, et al. (1999) Persistent dissection of carotid artery in patients operated on for type A acute aortic dissection--carotid ultrasound follow-up. Int. J. Cardiol 70: 133-139.
53. Chiappini B, Schepens M, Tan E, Dell' Amore A, Morshuis W, et al. (2005) Early and late outcomes of acute type A aortic dissection: analysis of risk factors in 487 consecutive patients. Eur. HeartJ 26: 180-186.
54. Sinatra R, Melina G, Pulitani I, Fiorani B, Ruvolo G, et al. (2001) Emergency operation for acute type A aortic dissection: neurologic complications and early mortality. Ann. Thorac. Surg 71: 33-38.
55. Reginelli A, Capasso R, Ciccone V, Croce MR, Di Grezia G, et al. (2016) Usefulness of triphasic CT aortic angiography in acute and surveillance: Our experience in the assessment of acute aortic dissection and endoleak. International Journal of Surgery 1: S76-S84.