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Aortic dissection masquerading as a code stroke: A single-centre cohort study

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Abstract

Introduction: Data on the incidence of acute aortic dissection in the code stroke population are scarce. We report estimated incidence, clinical manifestations, treatment and outcomes of patients with an acute aortic dissection in a code stroke cohort from a comprehensive stroke centre.

Patients and methods: We used data from a single-centre prospective registry of consecutive adult patients who presented to the emergency department between 2015 and 2018 with neurological deficits suggestive of an acute stroke ('code stroke'). All patients routinely underwent non-contrast computed tomography of the brain and computed tomography-angiography of the aortic arch, cervical and intracranial arteries.

Results: Of 2874 code stroke patients, 1563 (54.4%) had acute ischaemia (ischaemic stroke or transient ischaemic attack). Fifteen patients (0.5% of code stroke patients and 0.8% of patients with acute ischaemia) had an acute aortic dissection (all Stanford classification type A). Discerning clinical manifestations were decreased consciousness in 11/15 (73%), pain in 8/15 (53%) and low systolic blood pressure (mean 106 mmHg, SD30). Acute aortic dissection was an incidental finding during computed tomography-angiography in 4/15 (27%). Two out of 15 patients (13%) received intravenous thrombolysis, 9/15 (60%) underwent aortic surgery and 10/15 (67%) died. Of those who survived, 3/5 (60%) had a good functional outcome (modified Rankin Scale 0–2).

Discussion and Conclusion: In our comprehensive stroke centre, about 1/200 code stroke patients and 1/125 patients with acute ischaemia had an acute aortic dissection. Multicentre studies are necessary to acquire a more reliable estimate of the incidence of acute aortic dissection in the code stroke population. Given the ramifications of missing this diagnosis, imaging of the entire aortic arch is important in these patients.

Keywords

Aortic dissection, ischaemic stroke, transient ischaemic attack, stroke

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Introduction

Acute aortic dissection has an incidence of 2.5–3.5 per 100,000 person-years in the general population[1–3] and is associated with a high rate of mortality and morbidity. In its natural evolution, without surgical treatment, Stanford classification type A acute aortic dissection has a reported mortality rate of about 1% per hour initially, 50% after three days, and almost 80% after two weeks.[4] Approximately 17–40% of patients with acute aortic dissection present with neurological symptoms, half of which are attributable to acute ischaemic

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stroke (cerebral or spinal).[5] However, data on the incidence of acute aortic dissection in the code stroke population are scarce. Code stroke refers to patients who present to the emergency department (ED) with neurological deficits suggestive of an acute stroke.

New international guidelines allow for more ischaemic stroke patients to receive acute treatment, resulting in an increase of patients presenting with neurological symptoms suggestive of an acute stroke to EDs, particularly in comprehensive stroke centres.[6,7] Since management of patients with an underlying aortic dissection differs tremendously from that of other causes of ischaemic stroke, it is important to study the incidence of this condition in a code stroke population.

We estimated the incidence and report the clinical manifestations, radiological findings, and outcomes of patients with an acute aortic dissection in a single-centre code stroke cohort.

Patients and methods

We used data from a prospective registry of consecutive adult patients admitted to the ED of our comprehensive stroke centre between 1 January 2015 and 30 June 2018 with a code stroke. Code stroke was activated by ambulance nurses according to the following criteria: (1) patients with neurological deficits suggestive of an acute stroke, identified in a prehospital setting using the face arm speech time (FAST) test,[8] (2) within a time window of 6 h from symptom onset between January 2015 and February 2018 and 24 h from symptom onset between March 2018 and June 2018. The expansion of the time window in our acute stroke treatment protocol was in reaction to the publication of the DAWN and DEFUSE-3 trials.[9,10] When code stroke is activated, our stroke team is pre-notified of an incoming code stroke presentation. At our ED code stroke patients are primarily evaluated by a neurologist or experienced neurology resident and routinely undergo non-contrast computed tomography (CT) of the brain and CT-angiography of the aortic arch, cervical and intracranial arteries. Ischaemic stroke diagnosis was established based on clinical and radiological findings.

From this registry, we selected patients in whom aortic dissection was identified with CT-angiography, and if necessary, confirmed with magnetic resonance-angiography, echocardiography or autopsy. Imaging of patients with aortic dissection was re-evaluated by the authors. We defined aortic dissection as suspected on admission if aortic dissection was included as a specific question for the radiologist in the CT-angiography request. We also used the Dutch financial coding system for hospital care to identify potential missing cases of aortic dissection. We excluded patients who did not present to the ED or who were not primarily evaluated by neurology.

In our registry of consecutive code stroke patients, the following data are prospectively collected: demographics, National Institute of Health Stroke Scale (NIHSS) score, medical history, diagnosis at discharge from ED, details on treatment, time metrics, whether the patient was a direct presentation or referred from another hospital, and three-month modified Rankin Scale (mRS) score. Clinical data were extracted from the medical records. For outcome, we used in-hospital mortality and the mRS score at last follow-up. For the purpose of this study, baseline electrocardiograms (ECGs) were re-evaluated by a cardiologist (SMB) to detect common acute changes.[11] Acute changes were defined as ST elevation or depression, T inversion, atrioventricular block, new atrial fibrillation, premature atrial or ventricular contraction and sinus bradycardia.[11] The institutional review board of Amsterdam UMC, location AMC approved the study and waived the need for written informed consent (approval number W18_300 # 18.346). All statistical analyses were performed using IBM SPSS Statistics V.23 (IBM). Data were summarised using descriptive statistics (medians, means, SDs, frequencies).

We also conducted a literature search in PubMed to identify previous studies on the incidence of acute aortic dissection in code stroke populations using a search strategy including the terms aortic dissection and acute ischaemic stroke (full search strategy is provided in the online Supplemental Material). Title and abstract screening and full-length review of potentially relevant studies were conducted by VG.

Results

During the study period, 2874 code stroke patients (50% male; median age 68 years, IQR 54–78) presented to the ED. Of these, 1563 (54%) had acute ischaemia (1381 ischaemic stroke and 182 transient ischaemic attack (TIA), respectively). Fourteen patients had an acute aortic dissection. Three other patients had a suspected acute aortic dissection, but these were excluded because they died before the diagnosis could be confirmed and autopsy was not performed. Search of the financial coding system yielded one additional case. Therefore, 15 patients with an acute aortic dissection were included in the study. Overall, 0.5% (15/2874, 95% confidence interval (CI): 0.3–0.8%) of patients who presented with a code stroke had an acute aortic dissection. Among patients with a confirmed ischaemic stroke or TIA, the proportion was 0.8% (12/1563, 95% CI: 0.4–1.3%).

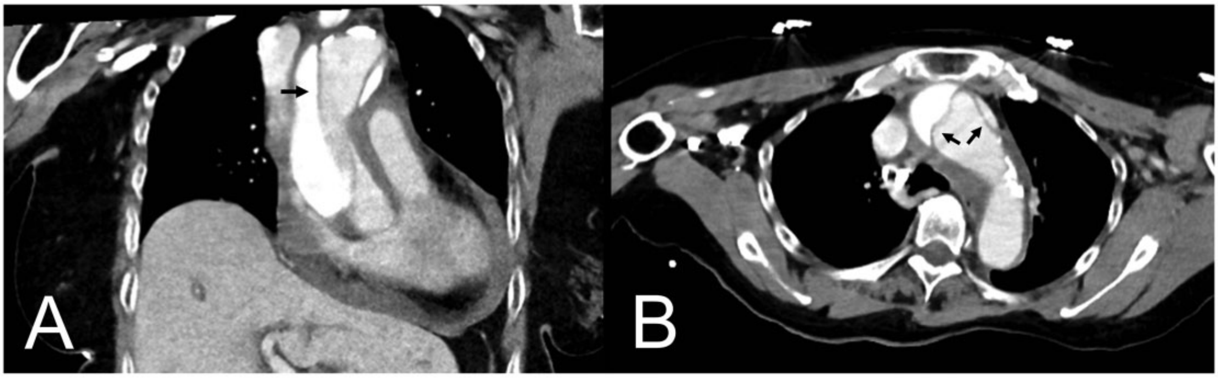

Figure 1. CT-angiography of patient No. 11, coronal (a) and transverse (b) planes. Arrows indicate Stanford classification type A acute aortic dissection.

All 15 cases had Stanford classification type A acute aortic dissection. The diagnosis was established with CT-angiography in all patients (Figure 1), six (40%) also underwent transthoracic echocardiography. In 11/15 (73%) patients, aortic dissection was suspected before imaging was performed, while in 4/15 (27%) patients, it was an incidental finding during CT-angiography. Baseline characteristics are provided in Table 1. Six patients (40%) were male and the median age was 73 years (range 53–88). Mean baseline systolic blood pressure was 106 mmHg (SD 30). In seven patients, blood pressure was measured bilaterally, and a discrepancy between left and right arm of 10 mmHg or more was found in 5/7 (71%). Acute ECG changes were present in 8/13 (62%) patients. Most common changes were ST elevation (n = 5), sinus bradycardia (n = 5), ST depression (n = 2) and T inversion (n = 2). Eight patients (53%) reported pain and 11 (73%) had decreased consciousness. Abnormal auscultation of the heart was observed in 2/4 (50%) of patients in whom this was examined. Cold extremities and asymmetric pulses were noted in 3/6 (50%) and 3/12 (25%) of patients, respectively. Perspiration was reported in 4/15 (27%) of patients. In patients presenting with unilateral motor deficits, the left side was affected in 4/5 (80%) of cases.

Information on radiological findings, treatment and outcome are provided in Table 2. Ten cases (67%) had cerebral ischaemia, two (13%) had spinal cord ischaemia (patients Nos. 7, 8), and in three cases (20%, patients Nos. 2, 5 and 10), the neurological symptoms were most likely attributable to hypoxic encephalopathy due to decreased cardiac output rather than acute ischaemia. None of the patients had an intracranial large vessel occlusion on CT-angiography. In two patients, intravenous thrombolysis was started, but was discontinued immediately after diagnosis of aortic dissection (Nos. 1, 6). The administered/total dosage of recombinant tissue plasminogen activator was 25/55 mg and 21/63 mg, respectively. Patient No. 1 became haemodynamically unstable in the operating room prior to incision as a result of a ruptured ascending aorta. The patient underwent a Bentall procedure (replacement of aortic root and ascending aorta), but died during the procedure due to left ventricular failure despite inotropic medication and cardiac massage. Patient No. 6 underwent a supracoronary ascending aorta replacement without any complications and recovered with an mRS score of 1 at last follow-up at nine months. In total, nine patients underwent aortic surgery, with a median interval of 201 min (range 75 min – 70 h) between presentation and incision. The other patients did not undergo surgery because of poor neurological condition (n = 5) or expected high risk of complications (n = 1).

At follow-up, 10/15 (67%) patients had died. Cause of death was directly related to the dissection in nine. In these patients, the median time between presentation and death was one day (range 0–2). Patient No. 4 died 20 months after presentation as a result of esophageal cancer. Of the patients who did not undergo surgery, none survived. Three out of five (60%) survivors had a good functional outcome (mRS 0–2; median time to last follow-up five months, range 0.5–9).

Of the 204 articles identified in the literature search, two were on the incidence of acute aortic dissection in code stroke or acute ischaemic stroke populations.[12,13] These studies are described in the 'Discussion' section.

Discussion

To our best knowledge, this is the largest report of an estimated incidence of acute aortic dissection among code stroke patients and the first in a European population. We found that during a 3.5-year period, approximately 1 in 200 patients who presented to the ED of

Table 1. Baseline clinical manifestations of code stroke patients with Stanford classification type A acute aortic dissection.

Pt	Sex	Age (yrs)	HT	BP (mmHg)	Pain	Cold limbs	Asymm. pulses	Acute ECG changes	GCS score	NIHSS	Focal neurological deficits
1	F	72	–	76/41	Chest	NA	NA	+	15	5	Dysarthria, paralysis and hypesthesia R leg
2	F	73	+	135/80	–	+	NA	–	9	14	–
3	M	76	–	100/52	–	NA	+	–	3	36	Pupil anisocoria (L>R)
4	F	67	+	128/59[a]	–	–	–	+	12	5	–
5	M	53	+	115/107	Chest	+	–	NA	12	5	–
6	M	67	+	135/81	Head	NA	–	+	15	17	Gaze deviation, L facial weakness, L hemiparalysis and hypesthesia
7	M	73	–	174/77[a]	Chest, back	–	–	+	15	2	Paraparesis
8	F	73	+	70/41	Chest	NA	+	–	15	10	Paraparesis, para-anesthesia
9	M	54	+	100/60	Chest	NA	–	–	3	36	Gaze deviation, pathological stretching
10	F	56	–	90/60[a]	–	NA	–	+	13	3	–
11	F	76	+	96/56[a]	–	+	+	+	12	15	L facial weakness, L hemiparalysis
12	M	74	–	114/61	Head, neck	NA	–	–	13	8	Aphasia
13	F	88	+	125/60	Jaw	–	–	+	14	5	Gaze deviation
14	F	54	+	76/35	–	NA	–	+	7	27	Gaze deviation, L hemiparesis
15	F	74	+	58/40[a]	–	NA	NA	NA	6	25	Pupil anisocoria (R>L), L hemiparalysis

+: yes; –: no; BP: blood pressure; ECG: electrocardiogram; GCS: Glasgow Coma Scale; HT: hypertension in medical history; NA: data not available; NIHSS: National Institutes of Health Stroke Scale; Pt: patient.
[a]Interarm blood pressure discrepancy, present in 5/7 (71%) patients.

our comprehensive stroke centre with a code stroke had a Stanford classification type A acute aortic dissection. An incidence of 0.5% may seem low, but because of the severe consequences of missing this diagnosis and increase of code stroke presentations as a result of recent therapeutic advancements, neurologists and radiologists should be vigilant about a possible aortic dissection in patients who present to the ED with a code stroke. Typical symptoms of acute aortic dissection are not always present. To increase the chance of diagnosing aortic dissection, imaging of the entire aortic arch is important in code stroke patients. As CT-angiography of cervical and intracranial arteries has become routine for selection of code stroke patient who are eligible for reperfusion therapy, extending routine CT-angiography to include the entire aortic arch is a minor adjustment.

Our estimated incidences of Stanford classification type A acute aortic dissection in code stroke patients and patients with acute ischaemia are in line with two smaller previous studies in Japanese populations.[12,13] One study reported 0.3% (95% CI: 0.0–0.6) acute aortic dissections in patients with suspected acute ischaemic stroke and 1.1% (95% CI: 0.1–3.4) in acute ischaemic stroke patients presenting <24 h from onset.[12] The other study reported 1.0% (95% CI: 0.1–3) acute aortic dissections in patients with acute ischaemic stroke presenting <3 h from onset.[13]

The typical aortic dissection patient is a male in his seventh decade with a history of hypertension who presents with abrupt onset of thoracic pain.[14] In our study, there were slightly more females than males and only about half of patients presented with pain. This is in agreement with a previous study comparing patients with acute ischaemia (ischaemic stroke or TIA within 4.5 h of symptom onset) and acute aortic dissection to those without acute aortic dissection.[15] Many presented with additional manifestations such as decreased consciousness, perspiration, low systolic blood pressure, asymmetrical blood pressure values, acute ECG changes, abnormal cardiac bruits and cold extremities. However, absence of typical medical history and additional manifestations do not rule out aortic dissection, as exemplified by our case series.

Table 2. Radiological findings, treatment and outcomes of code stroke patients with Stanford classification type A acute aortic dissection.

Patient	Affected arteries on CT-angiography[a]	Surgery	In-hospital mortality
1	Proximal: brachiocephalic trunk, bilateral common carotid arteries Distal: R common iliac artery	Bentall	+
2	Proximal: ascending aorta and aortic arch Distal: normal	David	–
3	Proximal: brachiocephalic trunk, bilateral common carotid arteries, R subclavian artery, origin R vertebral artery, L internal carotid artery, L subclavian artery Distal: origin celiac trunk, origin bilateral renal arteries, mesenteric superior artery, bilateral common iliac arteries	–	+
4	Proximal: brachiocephalic trunk, bilateral subclavian arteries, L common carotid artery, axillary artery Distal: L external iliac artery, origin L renal artery	Bentall	–
5	Proximal: brachiocephalic trunk, R common carotid artery, bilateral subclavian arteries Distal: CT-angiography of thorax and abdomen not performed due to poor condition of patient	–[b]	+
6	Proximal: R common carotid artery, R intracranial internal carotid artery Distal: normal	Supracoronary ascending arch replacement	–
7	Proximal: origin L subclavian artery Distal: celiac trunk, superior mesenteric artery, bilateral external iliac arteries	Supracoronary ascending arch replacement	–
8	Proximal: brachiocephalic trunk, bilateral common carotid arteries, L subclavian artery Distal: R common iliac artery	Bentall	–
9	Proximal: brachiocephalic trunk, R vertebral artery, bilateral common carotid arteries, bilateral intracranial internal carotid arteries Distal: celiac trunk, superior and inferior mesenteric arteries, L renal artery, bilateral external iliac arteries	–	+
10	Proximal: ascending aorta Distal: normal	Bentall	–
11	Proximal: brachiocephalic trunk, bilateral common carotid arteries, R internal carotid artery Distal: normal	Supracoronary ascending arch replacement	+
12	Proximal: brachiocephalic trunk, L common carotid artery, origin L subclavian artery Distal: L common iliac artery, L internal and L external iliac arteries	Sternotomy[b]	+
13	Proximal: brachiocephalic trunk, R common carotid artery, syphon R internal carotid artery, L subclavian artery Distal: celiac trunk, L renal artery	–	+
14	Proximal: brachiocephalic trunk, bilateral common carotid arteries, R vertebral artery, L subclavian artery Distal: celiac trunk, R external iliac artery	–	+
15	Proximal: brachiocephalic trunk, bilateral common carotid arteries, R internal carotid artery, L subclavian artery Distal: celiac trunk, superior mesenteric artery	–	+

–: no; +: yes; L: left; R: right.
[a]Affected segment(s) of aorta only described in patients in which dissection was limited to aorta.
[b]Patient died prior to/during surgery.

Notably, we were able to verify the predominance of left hemiparesis observed in a previous study.[13] It is plausible that the pathophysiological explanation lies in the anatomical order of the aortic arch branches. When Stanford classification type A aortic dissection spreads to other arteries, the brachiocephalic and right common carotid arteries are nearer to the origin of the dissection than the left common carotid artery, which could explain why these arteries are affected more often.

The risk of acute aortic dissection in a code stroke population confronts the physician with a dilemma. Timely start of intravenous thrombolysis in ischaemic stroke patients is of vital importance, and in many hospitals thrombolysis is started prior to or simultaneous with CT-angiography. However, if a patient turns out to have an acute aortic dissection, thrombolysis may be detrimental.[16,17] Thrombolysis for acute ischaemic stroke is contraindicated in concomitant aortic dissection, as it could lead to aortic rupture causing haemopericardium and cardiac tamponade, postpone a life-saving surgical procedure or result in the potential expansion of intramural or periaortic haematoma.[17] This dilemma is demonstrated by the fact that thrombolysis was started in two of our patients because aortic dissection had not been diagnosed yet, with an aortic rupture and fatal outcome for one patient. Performing CT-angiography prior to administering intravenous thrombolysis and fast evaluation of imaging after acquisition can help to reduce the risk of administering intravenous thrombolysis in patients with an aortic dissection.

Emergency aortic surgery provides the only chance for survival in patients with acute aortic dissection. Surgeons often weigh the presence of neurological manifestations in their decision whether to perform surgery.[18] Before and during surgery, a low blood pressure is maintained for several hours, which is necessary to prevent aortic rupture, but unfavourable in the presence of brain ischaemia. Prolonged hypotension may induce negative effects on the penumbra, increasing the risk for expansion of the infarct. In our series, the cardiothoracic surgeons decided to refrain from surgery in all patients who were comatose (GCS $\leq$ 8). In a recent cohort study of 53 Stanford classification type A acute aortic dissection patients with preoperative neurological deficits, half of the patients completely recovered from focal neurological deficits after aortic surgery.[18] Interestingly, preoperative GCS did not adequately predict neurological recovery; the proportion of patients with preoperative GCS $\leq$ 8 was 20% in those who fully recovered and 37% in those with persistent deficits. Surgery within 5 h after onset of symptoms increases the likelihood of favourable outcome in patients who present with coma.[19] Given the fact that CT-perfusion can differentiate between irreversibly damaged and salvageable brain tissue, this technique may potentially aid in decision making regarding surgery in these patients, although this hypothesis has not been studied yet.

Our study has several limitations. First, we may have missed patients with acute aortic dissection who presented to the ED with neurological deficits. At least three patients died before a suspected diagnosis could be confirmed. Also, we did not systematically re-evaluate imaging data of all code stroke patients. Therefore, it is possible that in some patients, the imaging did not capture the entire aortic arch or that the diagnosis was missed despite complete imaging of the aortic arch. Second, the observed incidence in our single-centre study may not be representative of all hospitals, since code stroke definition, the organisation of stroke care, and the proportion of stroke mimics[20] may differ per region. Multicentre studies are necessary for a more reliable estimate of the incidence of acute aortic dissection in the code stroke population.

Conclusion

In conclusion, in our comprehensive stroke centre, about 1/200 patients who presented with a code stroke and 1/125 patients with acute ischaemia suffered from an acute aortic dissection. Neurologists and radiologists should be vigilant about aortic dissection in this population, especially in patients with pain, decreased consciousness and low blood pressure, often with asymmetrical blood pressure values. Routine CT-angiography of the entire aortic arch can expedite early diagnosis of aortic dissection and help prevent administering intravenous thrombolysis to these patients.

Declaration of Conflicting Interests

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Ethical approval

The institutional review board of Amsterdam UMC, location AMC approved the study.

Informed consent

The institutional review board of Amsterdam UMC, location AMC waived the need for written informed consent (approval number W18_300 # 18.346).

Guarantor

JMC.

Contributorship

JMC, LP and VG conceived the study. VG and JMC were involved in protocol development and gaining ethical approval. N-SG, AEG, LP and VG were involved in data collection. VG performed data analysis and wrote the first draft of the manuscript. All authors reviewed and edited the manuscript and approved the final version of the manuscript. JMC oversaw all stages of the project.

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Supplemental Material

Supplemental material for this article is available online.

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