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Aorta Code: a pilot study of a health care network for patients with acute aortic syndrome

Código Aorta: proyecto piloto de una red asistencial para la atención al paciente con síndrome aórtico agudo

To the Editor,

Acute aortic syndrome (AAS) is a rare condition associated with high mortality.[1]

The Aorta Code project was designed to create a dedicated health care network for patients with AAS. The project had 3 main goals: to increase vigilance and improve the diagnosis of AAS, to expedite the transfer of patients to the network's aortic referral center, and to provide optimal treatment through a highly specialized team.

The Aorta Code network is formed by 4 hospitals from the autonomous community of Madrid and the area's medical emergency response service (SUMMA-112). It provides coverage to a population of 1.04 million inhabitants.

The project set-up included specialized training at the hospitals' emergency departments and SUMMA-112 and the development of a diagnostic and a treatment algorithm based on clinical practice guidelines.[2] A highly specialized AAS care team formed by 2 cardiologists, 2 cardiac surgeons, and 2 vascular surgeons (the Aorta Team) was created to take responsibility for decisions on incoming patients and their treatment. The project was approved by the relevant health authorities in Madrid and the local ethics committee at *Hospital Clínico San Carlos*. Informed consent was obtained from all patients involved.

The full Aorta Code protocol has been published elsewhere.[2] In brief, as soon as a patient is diagnosed with AAS, the emergency responders activate the Aorta Code via a dedicated telephone line. The coordination committee at SUMMA-112 dispatches a unit for the urgent transfer of the patient to the referral hospital, where their case is assessed by the members of the Aorta Team, who decide on the definitive treatment.

To evaluate the Aorta Code project, we compared outcomes from the 18 months before the project was implemented (care-as-usual period) with outcomes from the first 2 years of its implementation (March 15, 2019 to March 15, 2021).

Categorical variables are expressed as number and percentage and were compared using the chi-square or Fisher exact test. Quantitative variables are expressed as mean ± standard deviation or median [interquartile range] and were compared using the *t* test. Statistical significance was set at $P < .05$ and the statistical analyses were performed in STATA (version 12.0).

The AAS code was activated 59 times during the 2 years analyzed (this included a 3-month period in which the service was interrupted due to the COVID-19 pandemic). AAS was confirmed in 42 patients (table 1); 5 patients did not have aortic disease, 8 had nonacute aortic disease, and 4 had a ruptured abdominal aortic aneurysm.

Following the implementation of the Aorta Code project, the number of patients diagnosed with AAS doubled (from a mean of 1 patient a month to 2 patients a month). No significant differences were observed between the groups for the presence of risk factors, imaging findings, or clinical presentations (table 1). Transfer times to the hospital were also significantly reduced (table 2). There were no significant differences between the groups for time from first symptom to diagnosis or time from diagnosis to surgery. Preoperative AAS complication rates also decreased (table 2).

The number of surgeons treating patients diagnosed with AAS fell from 13 (7 cardiac and 6 vascular surgeons) during care-as-usual to 4 (2 cardiac and 2 vascular surgeons) during the project. Sixty percent of patients with type A AAS were treated with the Bentall-De Bono procedure during the care-as-usual period. This percentage fell to 42% after implementation of the project, as 58% were treated with aortic root repair surgery. There was also a relative increase of 80% in the number of complete aortic arch procedures performed ($P = .09$) (table 2). The project achieved a relative reduction of 28% in surgery mortality rates compared with care-as-usual, although the difference was nonsignificant (risk difference, 8.5%; odds ratio = 0.64; 95% confidence interval, 0.15-2.6; $P = .559$). There were no differences between the groups in overall mortality.

The clinical presentation of AAS is variable and often leads to diagnostic errors.[3] Implementation of the Aorta Code project improved the knowledge and skills of emergency service physicians and increased the number of AAS diagnoses. Although management of acute aortic disease by a small team of dedicated surgeons at a limited number of specialized centers has been found

Table 1
Baseline and presenting characteristics of patients with acute aortic syndrome during care-as-usual and after implementation of the Aorta Code project

Variable	Aorta Code (n = 42)	Care-as-usual (n = 18)	*P*
Age, y	67.1 ± 2.2	63.4 ± 4.3	.403
Male sex	57.1% (24)	77.8% (14)	.129
Diagnosis			
Aortic dissection	80.9% (34)	77.8% (14)	.720
Hypertension	17.1% (7)	22.2% (4)	
DTTA rupture	2.4% (1)	0	
Type			
A	73.8% (31)	77.8% (14)	.745
B	26.8% (10)	22.2% (4)	
Risk factors			
Hypertension	76.2% (32)	55.6% (10)	.169
Diabetes mellitus	7.2% (3)	5.6% (1)	.821
Hypercholesterolemia	45.2% (19)	22.2% (4)	.172
Smoking	28.6% (12)	27.8% (5)	.950
COPD	11.9% (5)	5.6% (1)	.453
Chronic kidney failure	2.4% (1)	5.6% (1)	.530
Aortic aneurysm	5.4% (2)	11.1% (2)	.445
Signs, symptoms, and complications on admission			
SBP, mmHg	134.6 ± 6.5	132.3 ± 8.8	.840
Chest pain	80.5% (33)	88.9% (16)	.428
Syncope	19.1% (8)	11.1% (2)	.450
Neurological deficit	16.7% (7)	27.8% (5)	.324
Pulse deficit	19.1% (8)	22.2% (4)	.778
Peripheral ischemia	12.8% (5)	27.8% (5)	.168
Acute kidney failure	15.8% (6)	22.2% (4)	.557
Myocardial infarction	12.8% (5)	11.1% (2)	.855
Shock	23.1% (9)	16.7% (3)	.581
Tamponade	19.5% (8)	16.7% (3)	.796
Need for intubation	7.5% (3)	5.6% (1)	.787
Other studies			
Normal electrocardiogram	52.4% (22)	61.1% (11)	.533
Normal chest radiograph	7.3% (3)	5.88% (1)	.844
Hemopericardium	28.6% (12)	33.3% (6)	.712
Pleural effusion	23.8% (10)	23.5% (4)	.982
Hemomediastinum	14.3% (6)	18.8% (3)	.675
Hemothorax	7.2% (3)	0% (0)	.550
Periaortic hematoma	31% (13)	29.4% (5)	.907
Supraortic branch involvement	48.8% (20)	58.8% (10)	.486
True lumen compression	60% (21)	43.8% (7)	.279
Renal artery involvement	48.8% (20)	35.3% (6)	.347
D-dimer level, ng/mL	7187 (4230-54 411)	6817 (2390-49 739)	.871
Maximal aortic diameter, mm	50.3 ± 2	51 ± 3.3	.836
Maximal AIH thickness, mm	12 ± 1.4	17.4 ± 4.9	.197

AIH, aortic intramural hematoma; COPD, chronic obstructive pulmonary disease; DTAA, descending thoracic aortic aneurysm; SBP, systolic blood pressure.

to improve survival outcomes,[4] our project is the first of its kind to be implemented in Spain. The Aorta Code project has had 2 main effects: *a*) it has increased the complexity of surgical procedures (by assigning all interventions to just 2 surgeons) and *b*) it has reduced surgical mortality rates, although the difference with care-as-usual was not significant due to the small sample. Extending surgical repair to include the aortic arch and root increases complexity, but performance of this surgery by an expert team of surgeons in carefully selected patients does not increase in-hospital morbidity or mortality and reduces the need for reoperation in the mid- to long-term, thereby improving prognosis.[5]

In conclusion, *a*) the creation of a health care network for patients with AAS is feasible and requires the extensive involvement of emergency departments and response services and the creation of a team of experts in aortic conditions, *b*) the Aorta Code project has improved the diagnosis of AAS and shortened transfer times to the referral hospital, and *c*) the performance of AAS surgery by a small team of highly specialized surgeons has improved outcomes.

Table 2
Treatment and prognosis of patients with acute aortic syndrome during care-as-usual and after implementation of the Aorta Code project

Variable	Aorta Code (n = 42)	Care-as-usual (n = 18)	*P*
Time from symptoms to diagnosis, h	4.2 (1.01-8.9)	5.8 (2.5-9.6)	.508
Transfer time, min	150 (114-196)	259 (180-273)	.046
Treatment			
Medical	26.2% (11)	22.2% (4)	.745
Surgical	64.3% (27)	77.8% (14)	.303
Endovascular	14.3% (6)	0% (0)	.091
Complicated AAS before surgery	40.7% (11)	76.9% (10)	.046
Type A AAS surgery	87.1% (27)	92.8% (13)	.569
Surgery (segments)			
Valve	40.7% (11)	30.8% (4)	.542
Root	70.4% (19)	38.5% (5)	.054
Ascending aorta	92.6% (24)	92.3% (12)	.974
Hemiarch	9.5% (2)	30.8% (4)	.114
Complete arch in type 1 AAS	72.2% (13)	40% (4)	.094
Time in cardiocirculatory arrest, min	27.8 ± 2.9	30.7 ± 4.9	.593
Preoperative complications			
Kidney failure	55.6% (15)	53.9% (7)	.919
Mesenteric ischemia	11.1% (3)	15.4% (2)	.702
Peripheral ischemia	7.4% (2)	0	> .999
Tamponade	11.1% (3)	7.7% (1)	.736
Myocardial infarction	7.4% (2)	0	.314
Neurological complications	37.1% (10)	30.8% (4)	.697
Reoperation	29.6% (8)	23.1% (3)	.664
Overall mortality	23.8% (10)	22.2% (4)	.894
Surgical mortality	22.2% (6)	30.8% (4)	.559

AAS, acute aortic syndrome.
Complicated AAS: occurrence of any of the following complications before surgery for AAS: poor perfusion, kidney failure, myocardial infarction, tamponade, shock, and neurological complications.

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AUTHORS' CONTRIBUTIONS

C. Ferrera, I. Vilacosta, P. Busca, A. Martín Martínez, F.J. Serrano, and L. Maroto Castellanos contributed significantly to the collection and interpretation of data. C. Ferrera analyzed the data. C. Ferrera and I. Vilacosta wrote the first version of the manuscript. All the authors revised the full manuscript and approved its final version. All the authors agree on the content of the article and guarantee the veracity and accuracy of all parts of this study. The authors declare that they are responsible for the content of this study.

CONFLICTS OF INTEREST

None.

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◇See the Appendix for the full list of researchers from the Multidisciplinary Aorta Code CardioRed1 Group.

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Epicardial adipose tissue attenuation in admitted patients with COVID-19

Atenuación de grasa epicárdica en pacientes ingresados por COVID-19

To the Editor,

Since the outbreak of the acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the coronavirus disease 2019 (COVID-19) pandemic has spread worldwide and has caused almost 4 million deaths while more than 171 million people have contracted the virus 1 year later.[1] It has been previously reported that obesity increases the risk of COVID-19 complications.[2] Obesity not only indicates increased subcutaneous adipose tissue, but is also associated with increased visceral ectopic fat, including epicardial adipose tissue (EAT). It is known that increased visceral fat distribution promotes chronic proinflammatory, prothrombotic, and vasoconstrictive states[2] and it has been linked to a worse prognosis in COVID-19 patients.[3] Due to its local and systemic effects, EAT has been proposed as a leading actor in myocardial inflammation in COVID-19.[4,5]

Chest computed tomography (CT) examination allows quantification of adipose tissue and assessment of its metabolic activity by measuring its radiodensity or attenuation with a quantitative scale: Hounsfield units (HU). It has been proven that CT attenuation measured in HU can distinguish the metabolic activity of adipose tissue; highly active adipose tissue is characterized by more positive HUs, and hence greater tissue densities.[6]

Our aim was to investigate the association of EAT attenuation with the clinical outcomes of COVID-19 infection. We retrospectively analyzed EAT attenuation in chest CT scans performed in 75 patients admitted to our hospital due to COVID-19 infection between March and May 2020. The decision to perform the CT scans was made clinically by the treating physician. We compared EAT attenuation values of COVID-19 patients with those of controls individually matched for age and sex. All controls were identified from a prospectively collected database of 3792 patients who underwent a cardiac CT scan in our hospital. The eligibility criteria for controls included 256 patients undergoing the test due to chest pain that were reported as normal (Agatston score = 0, normal epicardial coronary arteries and no other pathology seen on the CT scan). After randomly matching for age and sex, we obtained 46 pairs to compare. The study was performed in accordance with the institutional review board of our center, and there was no requirement for informed consent due to the retrospective and observational study design. CT images were analyzed using a state-of-the-art workstation tool (AW Server, General Electric Healthcare, USA). EAT was considered as all tissue with a HU threshold between − 190 to − 30 contained within the parietal pericardium. EAT attenuation was measured as the mean of 3 values obtained by manually drawing 3 regions of interest: 1 anterior to the great vessels, 1 in the pericoronary fat, and 1 anterior to the right ventricle.

Baseline characteristics of the study cohort were mean age 71 ± 11 years, 56% male patients, and median in-hospital stay of 15 days. Comorbidities were frequent: 55% of patients had hypertension, 27% had a history of cancer, and 25% had diabetes mellitus. A total of 42.7% of patients was overweight and 18.7% obese. Ten patients required intensive care, all of them with mechanical ventilation and 18 patients died (24%).

COVID-19 patients had significantly higher EAT attenuation than controls (−91 HU vs − 105 HU, $P < .001$, figure 1A). In our population of cases, we found no differences in EAT attenuation in patients with different cardiovascular risk factors or comorbidities. Regarding the laboratory results, we found no correlation between EAT and any markers (including inflammatory, troponin and D-dimers). EAT attenuation was higher among patients with bacterial coinfection and in those requiring intensive care, mechanical ventilation, and vasopressor support. We found no differences between survivors and nonsurvivors (figure 1B).

The main factors associated with higher mortality included age, hypertension, dyslipidaemia, prior cancer, baseline hemoglobin, total white cell count, D-dimers, troponin, C-reactive protein, and lactate dehydrogenase levels. Table 1 summarizes their relationship with EAT attenuation values.

Our COVID-19 patients had higher EAT attenuation values than controls. Among COVID-19 patients, EAT attenuation was higher in cases with a complicated clinical course: in hemodynamically unstable patients and among those requiring intensive care and mechanical ventilation. These findings are compatible with the hypothesis that EAT could act as a contributor to SARS-CoV-2 entry into the heart and promote an augmented inflammatory response causing myocardial complications, suggested by Kim et al.[4] Due to the small population, size we did not perform a multivariable analysis to explore whether this finding is simply a marker of poor prognosis or whether it plays an independent role in the clinical outcome of COVID-19 infection but we found no association between EAT attenuation and the rest of the variables of poor prognosis identified in the series. In addition to the small sample,