Two Cases of Sudden Cardiac Death. First Manifestation of Arrhythmogenic Cardiomyopathy of the Right Ventricle, Interventricular Septum and Left Ventricle

Sudden cardiac death is defined as unexpected death within an hour of the onset of symptoms or occurring in patients found dead within 24 h of having been observed in good conditions (unwitnessed event). Some patients die suddenly, but most of them have prodromal symptoms. (1) This arbitrary limit differentiates death due to progression of a known disease from unexpected death. (1)

We report two clinical cases of apparently healthy and asymptomatic young patients without a history of cardiovascular disease or previous check-ups, who died suddenly.

Case 1. A 32-year-old apparently healthy housewife and mother of 3 children was found dead in her bed by her husband at noon, after the night rest. The family reported that the patient had no personal or family history of cardiovascular diseases or of sudden death. At necropsy, serial cross sections showed areas of myocardial replacement by adipose tissue involving 50-80% of the right ventricular wall. Both ventricles had normal dimensions, and the right ventricular free wall was replaced by lard-like fatty tissue with only a few millimeters of normal myocardium in some areas. (Figure 1 A). The lesion extended into the interventricular septum and left ventricular free wall. The histological examination showed extensive replacement of myocardial free wall by adipose tissue, indicating the "wavefront phenomenon" of replacement by adipocytes. (Figure 1 B and C). Cardiac Doppler echocardiography was normal in all cases in the immediate family, parents, 2 siblings and their 2 children. The genetic test was positive for mutation in the KCNH2 gene.

Case 2. A 27-year-old male patient was found dead by his coworkers on the floor of the locker room at his workplace. The family reported that the patient had no personal or family history of cardiovascular diseases or of sudden death. On gross examination, an intracavitary thrombus was observed in the right ventricle; the right ventricular free wall measured a few millimeters in some thin areas and was replaced by lard-like fatty tissue (Figure 2 A). The lesion extended into the interventricular septum and left ventricular free wall. The microscopic examination with different magnifications showed extensive areas of myocardial replacement by fatty tissue, scarce atrophic fibers and residual myocardial fibers separated in small fascicles. Necrotic myocytes, myocytolysis and myocardial atrophy with nuclear and cytoplasmatic abnormalities were also observed (Figure 2 B and C). Cardiac Doppler echocardiography was normal in all cases in the immediate family who refused to undergo genetic testing.

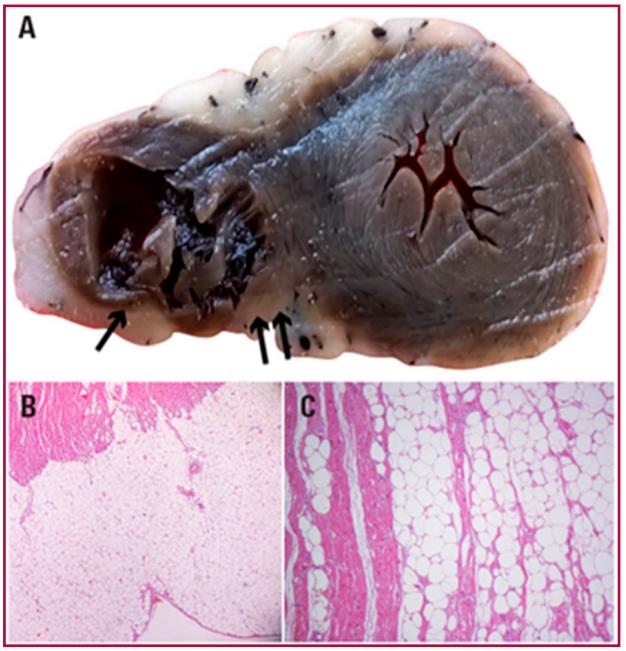

Fig. 1. A. Cross section showing the right ventricle on the left. The thin free wall measures 2-3 mm (single arrow). Abundant subepicardial adipose tissue is observed below. Fatty infiltration of the interventricular septum and left ventricle (arrows). **B.** Extensive subepicardial fatty infiltration with infiltration of the underlying myocardium. **C.** The fatty infiltration separates the atrophic myocardial fibers. **B** and **C** hematoxylin and eosin stain. 100 and 200×.

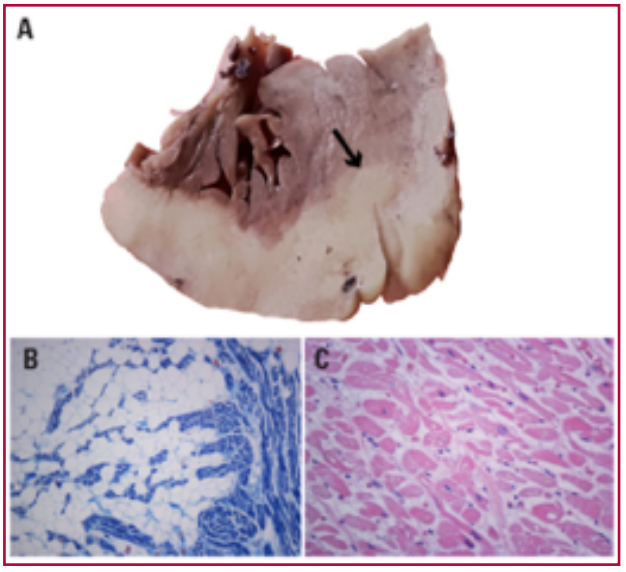

Fig. 2. A. Cross section showing the right ventricle on the left. Extensive subepicardial fatty infiltration of both ventricles and interventricular septum (arrow). **B.** Extensive subepicardial fatty infiltration with infiltration of the underlying myocardium. Masson's trichrome staining 100×. **C.** Section of the free wall distant to the fatty infiltration described in B with interstitial edema, atrophic myocardial fibers with myocytes with discrete fatty infiltration and nuclear abnormalities. Hematoxylin and eosin stain 200×.

The final diagnosis of both autopsies was arrhythmogenic cardiomyopathy (arrhythmogenic right ventricular dysplasia with partial involvement of the interventricular septum and left ventricle (biventricular variant).

The residual myocardial fibers described in both cases are the cause of malignant arrhythmias. Fibrofatty replacement hinders and delays interventricular conduction, thus favoring reentrant mechanisms. (1)

This condition, known as arrhythmogenic right ventricular cardiomyopathy (ARVC) or more recently as "arrhythmogenic cardiomyopathy" (ACM), is characterized by replacement of the myocardium by adipose and fibrous tissue predominantly in the right ventricle, and eventually thinning of the right ventricular wall with dilatation of cardiac chambers. Residual myocardial fibers are embedded in fibrous tissue, and this substrate favors reentrant arrhythmias. It is the most common cause of sudden cardiac death in young patients in Italy and accounts for 17% of sudden cardiac death cases in young patients in the United States. (2)

The diagnosis should consider genetic and non-genetic causes. Myocardial tissue is a complex system involving structural and functional factors; the identification of significant abnormalities in these factors allows the diagnosis of ACM by appropriate electrophysiologic and morphologic analysis. Family screening should include genetic evaluation to detect cases of premature cardiac events (sudden cardiac death, heart failure) and associated cardiac (arrhythmias, conduction disease) and non-cardiac (skeletal myopathy, renal failure, auditory/visual defects) phenotypes. (2) The abnormalities of the final common pathway may lead to the development of a complex phenotype, such as dilated cardiomyopathy with significant arrhythmic risk. (2)

In ACM, ultrastructural abnormalities exist at the level of the intercalated disc, which combine anomalies in the mechanical coupling that would be determined by mutation of desmosomal proteins, and in ionic conductance. The latter would be affected by abnormalities in the intrinsic structure of the ion channel and in the intercellular coupling structure. Although the pathogenesis of ACM in still not known, different theories have been suggested. (2) In the inflammatory theory, myocardial damage is explained by a continuous cycle of damage and repair that simulates chronic myocarditis. According to the genetic theory, mutations of genes that encode specific proteins would give rise to myocardial "dystrophy." The progressive myocardial replacement by fatty and fibrous tissue occurs after an exaggerated and inadequate process of apoptosis. Animal models lend support to the imbalance caused by cellular mechanical stress as a trigger of apoptosis. (3)

Cell-cell junctions are formed by proteins. These include plakoglobin (JUP) and desmoplakin (DSP) which play a key role in the transduction of mechanical stress and cell-cell communication. Plakoglobin is a cytoplasmic protein that forms part of desmosomes3-5 and adherens junctions. It helps link intermediate and cytoskeletal actin filaments with the transmembrane complexes that connect adjacent cells. The mutated form of JUP favors an unstable cell substrate, (3,5) and also regulates expression of the antiapoptotic protein Bcl-2.2. (4,5)

Desmoplakin is a component of the desmosome plaque where its functions include anchoring the intermediate filaments to the plasma membrane and is an essential platform for maintenance of cell integrity.

As for case 1, it is known that mutations in KCNH2 (which encodes potassium voltage-gated channel subfamily H member 2) are responsible for LQTS in many patients, and that mutations in JUP are associated with arrhythmogenic right ventricular cardiomyopathy. (5,6)

The clinical presentation is variable and includes palpitations, syncope, right heart failure or biventricular heart failure, or sudden cardiac death. It rarely manifests before puberty, generally between 12 and 45 years, and sudden cardiac death is often the first symptom. The hallmark presentation is symptomatic or documented arrhythmia. Arrhythmias are more common during exercise, and 3-4% of sudden deaths in athletes are due to this disorder. The diagnosis of ACM is particularly difficult; there are no pathognomonic signs and consensus criteria have been developed according to its structural and functional manifestations.

Recently, Cadrin-Tourigny et al. (6) developed a specific prediction model of potentially fatal ventricular arrhythmias as a closer surrogate for sudden cardiac death in a retrospective cohort of 864 definite cases of ACM over 5.75 years of follow-up. The variables considered were age, sex, prior sustained ventricular arrhythmia (≥30 s, hemodynamically unstable, or implantable cardioverter-defibrillator treated ventricular tachycardia; or aborted sudden cardiac death), syncope, 24-hour premature ventricular complexes count, number of anterior and inferior leads with T-wave inversion and left and right ventricular ejection fraction. Ninety-three (10.8%) patients experienced life-threatening ventricular arrhythmias, including 15 with sudden cardiac death/aborted sudden cardiac death (1.7%). Of the prespecified clinical predictors, only 4 (younger age, male sex, premature ventricular complex count, and number of leads with T-wave inversion) were associated with subsequent life-threatening ventricular arrhythmias, but no association was found with prior sustained ventricular arrhythmia or extent of functional heart disease. (6)

Unfortunately, in both cases reported, the absence of previous clinical-cardiological examinations hindered the correct characterization of the potentially lethal nature of arrhythmogenic cardiomyopathy

Conflicts of interest
None declared.

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Not applicable.

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Hybrid Treatment of Two Aneurysms in the Anterior Descending Artery

Coronary artery aneurysm (CAA) is a rare entity, with an incidence ranging from 0.2% to 10% according to different publications. It can be defined as the dilatation of the coronary vessel that exceeds the diameter of the rest of the anatomy by 1.5 times.

This condition was described by Morgagni in pathology studies in 1761, and by Munker in 1958 through a coronary angiography in a living patient.

Histopathological studies reveal hyalinization and fatty deposits affecting the intima and media layer, and the involvement of elastic muscle fibers causing vessel dilatation.

Its multiple etiologies include birth defects, vasculitis, collagen diseases, drugs, and trauma, but the most common is atherosclerosis, accounting for more than 50% of the cases.

CAAs are defined as saccular or fusiform according to morphology, and as small (< 5 mm), medium (5 - 8 mm) and large (> 8 mm) according to diameter.

CAAs are predominantly located in the right coronary artery, followed by the circumflex artery and the anterior descending artery. Clinical presentation ranges from asymptomatic patients to angina pectoris and sudden death due to rupture, tamponade, distal embolization or myocardial infarction.

In most cases, diagnosis is incidental during a coronary angiography, computed angiotomography or cardiac magnetic resonance.

CAA treatment may follow a conservative, surgical, or endovascular approach. (1-3) Since atherosclerosis is the main cause of CAA, conservative treatment consists of platelet antiaggregation, anticoagulants, statins and vasodilators. As for percutaneous revascularization, exclusion can be performed with covered stents; surgical treatment is applied in those cases in which the diameter is > 10 mm.

There is no consensus on CAA management, therefore, therapeutic decisions must be tailored to each patient, after assessing their risks and benefits. (4)

In the case discussed below, a hybrid treatment was decided due to the presence of two large aneurysms —the proximal CAA closely related to the pulmonary artery and left main coronary artery— making it very difficult to use the surgical approach alone.

We describe the case of a 59-year-old male patient coming in for a follow-up visit. The patient had a history of non-insulin diabetes, ex-smoker, obesity, dyslipidemia and hypertension, on treatment with enalapril 10 mg every 12 hours. He presented with sleep apnea-hypopnea syndrome under continuous positive airway pressure (CPAP), and a history of stent graft placement due to right iliac aneurysm, complicated with pseudoaneurysm and postoperative intermediate-risk pulmonary thromboembolism (PESI Score 88) with no right ventricular involvement, on treatment with rivaroxaban 20 mg daily.

While patient denied cardiac symptoms during questioning, control tests were requested given his medical history. Pulmonary scintigraphy revealed a defect of the anterior superior segment of the upper lobe and anterior basal segment of the lower lobe in the right lung, and a defect of the anterior segment of the upper lobe and upper lingula in the left lung. The final diagnosis was chronic pulmonary thromboembolism.

A cardiac magnetic resonance imaging (CMRI) was requested to assess pulmonary and pleural involvement, since computed tomography had already been performed and the aim was to reduce radiation expo-

sure. (5) CMRI showed an image in the interventricular groove, which received contrast medium in the angiography arterial time and appeared to be associated with the anterior descending artery. For this reason, a high-resolution CT angiography was performed for a possible aneurysm in the anterior descending artery (Figure 1). The study exhibited two tandem aneurysms at the level of this artery: one spherical aneurysm with a diameter of 27 mm, and another one with a diameter of 12 mm. The artery proximal to the aneurysm was severely calcified, with no lesions in the distal bed. The well-developed obtuse marginal artery originated in the circumflex artery, with multiple calcifications but insignificant stenosis. The right coronary artery was occluded at the proximal third level, and its distal bed was visualized through collaterals; the occluded path was 28 mm. On further questioning, the patient referred 1-year history of habitual angina in functional class II progressing to functional class III in recent months.

Given the presence of large aneurysms and in order to confirm the diagnosis, a coronary angiography was performed, revealing no significant lesions in the left main coronary artery, large aneurysms in the proximal and middle third of the anterior descending artery causing coronary steal, a significant lesion in the ramus intermedius, and collateral circulation from the anterior descending artery to the right coronary and circumflex arteries. A chronic occlusion was observed in the middle third of the circumflex artery, and in the right coronary artery.

Pre-surgical angiotomography of the neck vessels showed no evidence of lesions or aneurysmal images.

The case was discussed in the surgical grand round; a hybrid treatment for this disease was decided because the first aneurysm was very close to the pulmonary artery, making it very difficult to remove it surgically. (6)

Left mammary artery bypass graft to the anterior descending artery was performed, with pump and clamping times of 70 and 37 minutes respectively. Once the procedure was completed and still in the operating room, a right femoral puncture was performed using the Seldinger technique, a 0.035" wire was advanced, the needle was removed, and a 6 Fr introducer was placed in the right femoral artery. After flushing with heparinized solution, a JL 4 guiding catheter was advanced and the left coronary artery was selectively catheterized; control CT angiography showed the two aneurysms described above. Embolization using the Amplatzer Vascular Plug 4 - 7 mm x 12.5 mm was performed, which was fixed and stable within the anterior descending artery. Final control showed significant decrease of intraaneurysmal flow. (Figure 2)

Complications were Type-V subendocardial lateral infarction (CPK 2076 IU/L and high-sensitivity troponin > 40000 ng/L), and renal failure in the immediate postoperative period, which improved with hydration and IV diuretics.

Coronary aneurysms are a rare entity, and should be treated on an individual basis. Today, non-invasive imaging studies provide a great deal of information about their size and location, making it possible to decide on the best therapeutic strategy. In our case, due to their location and the fact that there were two aneurysms, we combined surgical and endovascular treatment to achieve positive outcomes.

Conflicts of interest

None declared.

(See authors' conflicts of interest forms on the website/ Supplementary material).

Ethical considerations

Not applicable

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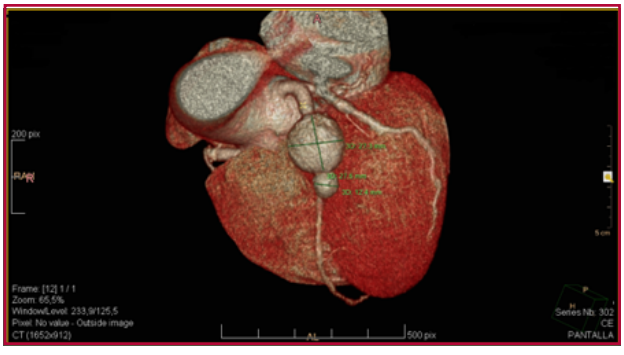

Fig. 1. Computed angiotomography showing two coronary aneurysms in the anterior descending artery.

Fig. 2. Follow-up angiography showing the exclusion of both aneurysms in the anterior descending artery territory.

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A Novel Continuous Airborne Disinfection and Isolation Unit for Stress Echocardiography

By the end of 2019, the world witnessed the emergence of the COVID-19 pandemic caused by the SARS-CoV-2 coronavirus and its subsequent multiple health impacts worldwide.

In view of the progressive and intense spread of the virus, protecting healthcare workers became a priority among the measures taken in the healthcare environment. Exposure to this disease results in unprecedented morbidity and mortality; therefore, timely implementation of protective measures for this group is imperative. (1-3)

As we speak and breathe, our airways usually release aerosols of different sizes, ranging from nanometers to hundreds of microns; people with respiratory tract infections exhale aerosols called bioaerosols, containing airborne pathogens. Bioaerosols are particles smaller than 100 microns that may remain in the air for some time and be inhaled at a distance greater than 2 meters from the emitter, or even in the absence of an emitter. Aerosols are the main channel for community transmission of COVID-19.

Since many cardiovascular diseases are diagnosed through functional stress tests, a modality that generates a large volume of aerosols, extreme caution must be taken and health recommendations should be followed, so that patients or health workers are not exposed. Initially, stress tests were limited to what was strictly necessary, or were performed under pharmacological stressor. However, with the passing of time and because of higher stress test sensitivity, they could not be postponed indefinitely, due to the risk of cardiac morbidity and mortality in non-diagnosed patients. (4)

Therefore, the following recommendations for safe practice were set: to perform tests in well-ventilated spaces; otherwise, to have a purification system with high particulate filtration (carbon or HEPA filters); to space the schedules as much as possible; and to ask the patient about symptoms and close suspected cases of COVID-19. In addition, a signed informed consent adapted to the situation and the use of masks during the entire test are required. Regarding healthcare workers, the recommendations were that the smallest possible number of people should stay in the room, as well as hand-washing, keeping a distance of 2 meters at all times, wearing personal protective items according to the type of study: face shield, N95 mask, and disposable gown and gloves, and cleaning all the accessories and work items used once the study was finished. (5, 6)

Apart from the standards and recommendations still in place, we intended to move forward on a safer method for both healthcare workers and patients. Based on a joint project of the Department's team of cardiologists and cardiology technicians, together with engineers from a company specialized in developing hospital isolation equipment (INGENIARG), an isolation unit with a continuous airborne extraction and disinfection system was designed. The aim was to create a structure that would provide safety for the healthcare workers involved in the test, by means of correct isolation, patient visualization, feasibility of performing the echocardiography and ECG, location of the airborne extraction system, etc.

The interesting part of this implemented method is that, with a device (which consumes only 80 Watts, equivalent to a light bulb), a continuous air transfer is achieved towards the inside of the cover where the patient is, generating not only a physical wall but also a second dynamic immunity wall.

As shown in Figure 1, the structure has an outlet on its upper part —at the level of the patient's airway— from where the unit's air is continuously extracted by a silent filtering handler, disinfected by two filters —one of them a high efficiency (99.99%) particulate air filter (HEPA)— and finally subjected to radiation exposure with an ultraviolet germicidal lamp and returned sterile to ambient air. The air filtration

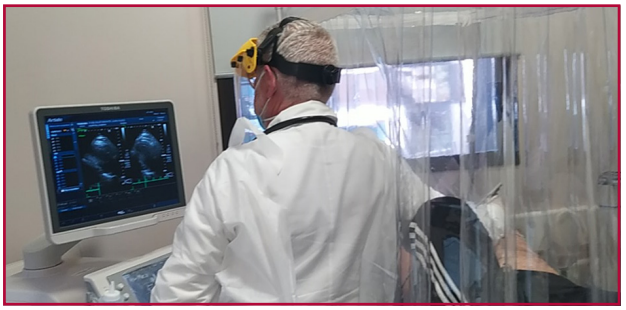

Fig. 1

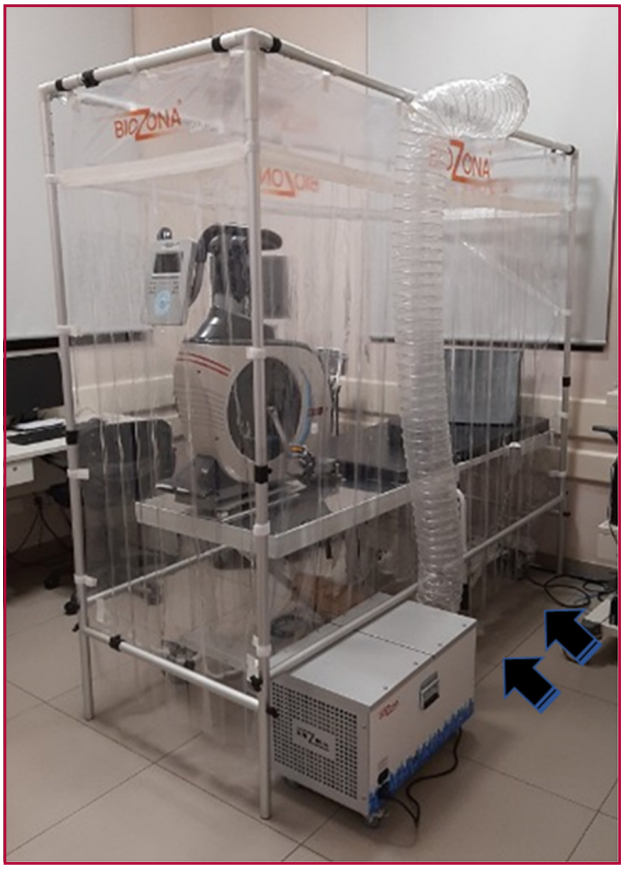

Fig. 2

unit is HEPA filter certified, and is regularly performed filter integrity tests according to ISO 14644-3 by qualified technicians.

The unit, based on an adjustable, periscope anodized aluminum structure, covers the entire stretcher sideways and the stationary bicycle with a high-density polyvinyl chloride crystal cover, allowing for normal visualization of the patient anatomy, management of the transducer from the lateral surface by the echocardiographer, and technicians' operability to place and remove the electrocardiograph electrodes from the other side and to adjust the resistance of the cycle ergometer from its anterior surface. It is important to highlight that the air flow direction with the extractor unit in operation is continuous and uninterrupted from the outside to the inside of the structure (Figure 2). At the end of each study, the interior, especially the inside of the cover, is cleaned with alcohol. Patients reported no discomfort after the study. On the contrary, they felt safer. It should be noted that this type of unit has never been published to date.

Thus, the test is performed even above the standards and recommendations of the World Health Organization, and provides both healthcare workers and patients with maximum safety conditions in times of spreading of airborne infectious diseases.

Conflicts of interest

None declared.

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Ethical considerations

Not applicable

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