Ventricular Septal Rupture Due to Evolved Acute Myocardial Infarction. Side Effects of Delayed Diagnosis During the COVID-19 Pandemic

Mechanical complications are among the worst phenomena that can occur with acute myocardial infarction (AMI).

After the introduction of early reperfusion therapy, the incidence of post-infarction ventricular septal defect (VSD) has decreased to 0.2% to 0.34%. (1, 2) It occurs within the first 24 hours and 3-5 days after symptom onset. Risk factors include anterior location of AMI, arterial hypertension, female sex, advanced age, and being the first ischemic event. Transthoracic Doppler echocardiography (TTE) is the main diagnostic tool. Surgical repair is the mainstay of treatment, with a high mortality rate between 20-50% depending on the series. (3)

The following two cases are reported in order to highlight the complications arising from the delay in consulting for fear of visiting hospitals during the pandemic.

The first case is a 66-year-old woman with a relevant history of hypertension, type 2 diabetes mellitus under 3 oral antidiabetic drugs, and dyslipidemia, who presented with 1-week history of non-irradiated oppressive central chest pain progressively increasing in intensity, with no vasovagal reactions, associated with general ill feeling and blood pressure tending to hypotension, with home systolic blood pressure (SBP) of 70 mmHg.

On the initial evaluation, the patient was in acceptable general condition, with BP 100/60mmHg, eupneic at rest; saturation 98% without supplemental oxygen. The ECG detected sinus rhythm at 110 bpm with QS in V1-V4 and ST segment elevation in V2-V5. The echocardiography showed a restrictive VSD at the septal level with clear identification of the shunt, an apical, anteroseptal, aneurysmal area, and akinesia extending to the middle septum. Overall, left ventricular ejection fraction (LVEF) was estimated in 40% (Figure 1 A & B).

The patient presented in the Emergency Room with blood pressure of 80/40mmHg, and was transferred to the Intensive Care Unit (ICU) under cardiogenic shock due to evolved anterior infarction with restrictive VSD as a mechanical complication. Hemodynamic stability was achieved through inotropic support and aortic balloon counterpulsation. Lab tests showed creatinine 1 mg/dL, natremia 132 mEq/L, potassium level 4.2 mEq/L and CRP 206.3 mg/dL. Peak high-sensitivity troponin T (hs-TnT) was 23 072 ng./L. Hemoglobin was 14.7g/ dL, white blood cells 18 820/mm^3, and platelets 298 000 /mm^3.

Coronary angiography showed no lesions in the left main trunk. The middle segment of the anterior descending artery was occluded, with a lack of visualization of the distal vessel. The circumflex artery showed non-significant wall irregularities. Occlusion of the proximal segment of the right coronary artery was observed, and the distal vessel was visualized by means of hetero-coronary collateral circulation. Left ventriculography showed septal, anterior, and apical akinesia with apical aneurysm and passage of contrast agent during systole from the left to the right ventricle through a septal defect with two proximal leaks.

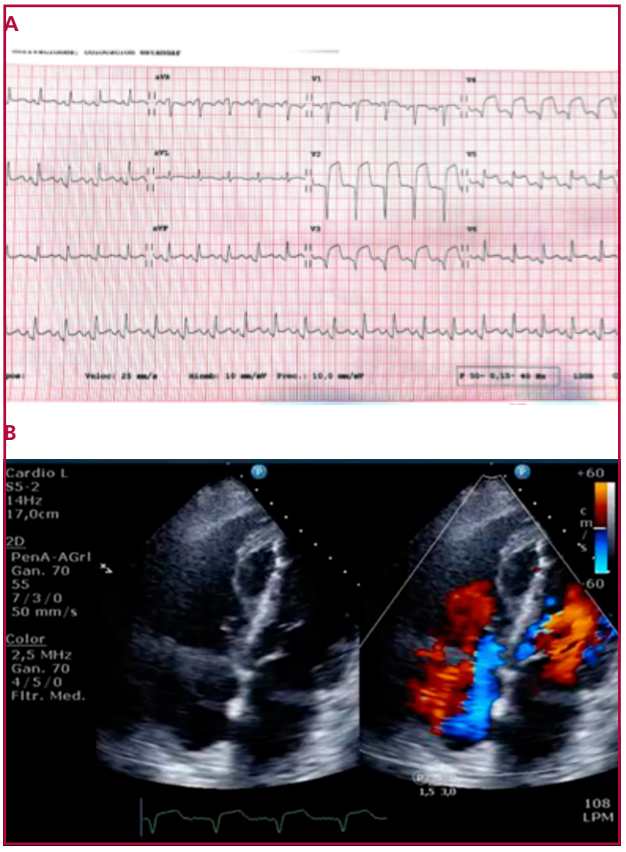

Fig. 1. A. ECG on admission: Sinus rhythm at 110 bpm with QS in V1-V4 and ST segment elevation in V2-V5. **B.** Color Doppler echocardiography: Pseudoaneurysm at the septal level with restrictive ventricular septal defect (VSD).

Once assessed by the Department of Cardiac Surgery, the patient was performed surgical repair of VSD and ventricular aneurysm with a bovine pericardial patch. Postoperative course was torpid; standard echocardiography revealed persistent defect with moderate left ventricular dysfunction and severe right ventricular dysfunction, severe pulmonary hypertension and severe tricuspid regurgitation.

Heart transplantation was ruled out due to poor metabolic control with HbA1c 11.6% and pulmonary hypertension, and conservative management was decided.

Six months later, the patient is closely followed up under home care, and is clinically stable.

The second case is a 64-year-old male patient with a history of hypertension, type 2 diabetes mellitus and dyslipidemia, under follow-up due to vascular surgery for intermittent claudication, who presented with a 10-day history of non-irradiated oppressive left chest pain, with no vasovagal reactions, which did not change with deep breathing or postural movements, and of variable duration (between minutes and hours). For 3 days, the patient was experiencing progressive dyspnea; upon consultation, ST-segment elevation in V2-V5 with pathological Q waves in V1-V6 was detected. Sublingual nitrites were administered to relieve the pain, and the patient was referred to the Emergency Department, where he was in an acceptable general condition and referred no chest pain but persistent mild dyspnea. Blood pressure was 108/70 mmHg, oxygen saturation 95% with supplemental oxygen at 3 liters, and heart rate was 115 bpm. Physical examination revealed distant heart sounds with continuous diastolic murmur and preserved global vesicular murmur.

Lab tests showed hemoglobin 15.5 g/dL, white blood cells 21 700/mm^3, neutrophils 18 700/mm^3, platelets 482 000 /mm^3, creatinine 0.96 mg/dL, CPK 72 IU/dL, natremia 137 mEq/L, potassium level 3.8 mEq/L. No alterations in coagulation. Troponin T: 593 ng/L.

Echocardiography showed dilated, aneurysmal left ventricle in the territory of the anterior descending artery, with a severely depressed LVEF (15-20%); in view of suspected apical VSD as a mechanical complication, the Department of Cardiac Surgery was contacted and the patient was transferred to the ICU.

During his stay in the ICU, the patient remained hypotensive, and inotropic support was started; synchronized electrical cardioversion was performed due to sustained ventricular tachycardia. Given the high risk of conventional surgery, percutaneous VSD closure was proposed. However, the patient's general condition worsened, with sudden dyspnea and cardiopulmonary arrest, and he finally died.

The global COVID-19 pandemic caused by SARS-CoV-2 puts stress on healthcare systems in many ways. In the paper by Rodriguez-Leor et al., the longer time from symptom onset to first medical contact is a cause for concern. (4) The most plausible initial interpretation is that patients took longer to call for out-of-hospital emergency services for fear of COVID-19. (5)

While the incidence of post-infarction VSD has declined due to early reperfusion therapy, delayed healthcare-seeking due to COVID-19 has the potential to reverse this trend. Since VSD is a life-threatening mechanical complication commonly leading to cardiogenic shock and subsequent death, urgent care is necessary, the mainstay of treatment being surgical VSD repair.

Despite its low incidence, this complication should be considered in the Emergency Department, in patients developing hemodynamic instability in the context of chest pain.

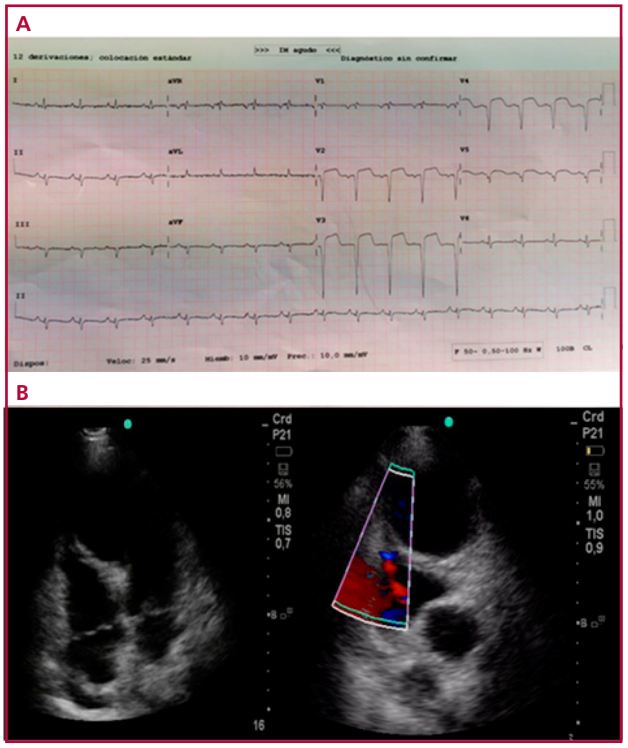

Fig. 2. A. ECG on admission: Sinus rhythm 120 bpm with ST segment elevation in V2-V5 and pathological Q waves in V1-V6. **B.** Color Doppler echocardiography: Dilated, aneurysmal LV in the apical VSD territory.

Conflicts of interest

None declared.

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Ethical considerations

Not applicable.

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Right Ventricular Hamartoma of Mature Cardiac Myocytes

Cardiac hamartoma is a rare tumor with a heterogeneous morphology, consisting of disorganized proliferation of mature and immature myocytes, fibrous tissue and vascular elements, as a result of abnormal development of embryonic cells. Three histological variants have been described depending basically on the dominant cellular proliferation: cardiac rhabdomyoma, which is predominantly composed of immature myocytes and spider cells and is associated with tuberous sclerosis; fibroma, mainly composed of fibrous tissue with some cardiomyocytes on the periphery and poorly vascularized; and hamartoma of Purkinje cells, the least common, consisting of myocytes with eosinophilic granular cytoplasm. (1) The first descriptions of mature cardiomyocyte hamartoma date back to 1998. (2) In this tumor, hypertrophied myocytes are arranged in a disordered fashion and mixed with fatty cells, vascular and fibrous tissue in different proportions. These tumors preferentially develop on the ventricle wall but have been reported on valve tissue. (3) Few cases in the right atrium have been reported, (4-6) and no cases in the right ventricle (RV) have been found.

We report the case of a 32-year-old male patient, with no known history or risk factors, long-standing dyspnea, weight loss of 5 kg in the month prior to hospitalization, and edema (+++) in the right lower limb. In another center, the clinical picture was diagnosed as secondary to chronic thromboembolic pulmonary hypertension, based on a pulmonary arteriography reporting "bilateral pulmonary embolism with manometric measurement of 50/30 mmHg, occlusive thrombosis within the right lower lobe artery, and segmental thrombosis within the left lower lobe".

Transesophageal echocardiography (TEE) prior to referral reported a heterogeneous image in the RV apex consistent with organized thrombus. Chest CT scan showed pleural and pericardial effusion, mediastinal nodes, dense fibrosis tracts of pleural-interstitial sequelae, bibasilar ground-glass pattern, and ascites.

Tests on collagen diseases antibodies (anti-myeloperoxidase, anti-proteinase 3, anti-cytoplasm, anti-citrulline, anti-RO, anti-La/SS-B, and anti-nuclear

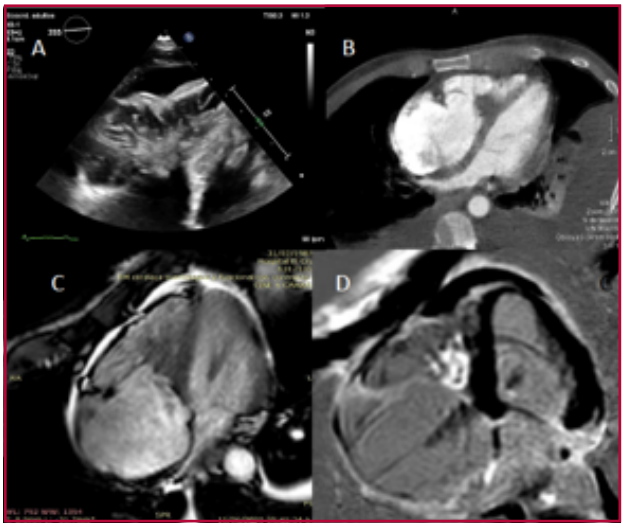

Fig. 1. Left to right and top to bottom. **A**. Two-dimensional transthoracic echocardiography with a focus on the right ventricle (RV), showing a heterogeneous right midventricular mass involving the tricuspid subvalvular apparatus, septal tricuspid leaflet retraction, focal retraction of the anterior RV wall, and anterior pericardial effusion. **B.** Cardiac CT scan with contrast, showing cold spot of the mass occupying the RV, implanted in the interventricular septum. **C.** Cardiac resonance imaging showing intraventricular mass in the RV, involving the tricuspid subvalvular apparatus, moderator band (causing retraction of the RV free wall), and septal tricuspid leaflet. **D.** Same image with positive late enhancement in the right intraventricular mass, and negative late enhancement in the left ventricle.

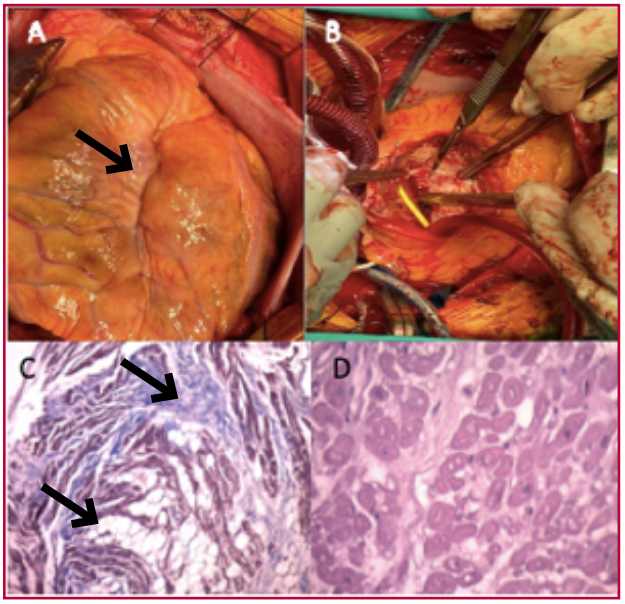

Fig. 2. Top: Surgical images, from right to left. A. View of the RV wall retraction. B. Anterior incision showing involvement of the tricuspid subvalvular apparatus. Bottom: Pathology, from left to right. C. Trichrome technique showing interstitial fibrosis in purple (arrow) and abundant fatty cells in white (arrow). Hematoxylin and eosin staining showing hypertrophy and mature myocyte vacuolization.

factor antibodies), as well as tumor markers (Ca 19-9, cyfra Ca 21, Ca 72.4) were negative.

In this scenario, the patient was referred to our hospital for thromboendarterectomy.

On admission, the patient was stable, with dyspnea FCII and without oxygen requirement.

Transthoracic echocardiography (TTE) showed a right midventricular mass with irregular borders and heterogeneous echogenicity, involving the tricuspid subvalvular apparatus and causing focal myocardial retraction of the RV free wall and the septal tricuspid leaflet, with secondary, moderate, eccentric valvular regurgitation. There were no indirect signs of pulmonary hypertension (Figure 1).

Cardiac CT scan ruled out thrombi associated with this structure, and cardiac magnetic resonance confirmed the findings described above, ruled out vascularization, and showed late enhancement.

Lower limb venous ultrasound showed an image consistent with proximal iliac vein thrombotic obstruction.

An endomyocardial biopsy was performed. The pathology diagnosis was hamartoma of mature myocytes. A surgical approach was decided. Macroscopic surgical findings included RV anterior wall retraction, intraventricular fibrous mass with retracted chordae tendineae and septal tricuspid leaflet involvement (Fig. 2). Immediate postoperative course was torpid, with refractory ventricular failure, requiring mechanical ventilation with extracorporeal membrane oxygenation (ECMO). The patient died within 48 hours of surgery.

Heart neoplasms are uncommon lesions, most of which are incidental findings on autopsy or imaging studies of the heart. Transthoracic echocardiography (TTE) is the diagnostic approach of choice in initial studies of cardiac masses. The safety, low cost and innocuous nature of echocardiography make it the technique of choice in initial studies of heart masses. The technique can localize such masses, define their shape, size, mobility, and point of anchorage, and determine whether they are solid or cystic in nature. However, abnormal tissue characterization, invasion or vascularization cannot be defined by this technique. CT scan is a second-line diagnostic tool to determine location, size and anatomical relationships of cardiac masses, followed by positron emission tomography (PET), which allows early detection of primary tumors of uncertain origin, and their initial staging. Magnetic resonance (MR) supplies structural and hemodynamic information since it offers both static and dynamic sequences, and images can be acquired from an unlimited number of planes and projections. Tissue characterization via images potentiated in T1 and T2 complete this information. Late enhancement sequences define tumor clearly and are essential for thrombus detection and characterization. (1) In our patient, these methods ruled out intracavitary thrombi, and cardiac tumor was suspected as we could not confirm it was a fibroma or a sarcoma (angiosarcoma, rhabdomyosarcoma) due to its characteristics and rare RV location. However, these methods determined the need for a biopsy, given that the mass was not vascularized.

We believe that the publication of our extremely uncommon case contributes to considering mature myocyte hamartoma within the differential diagnoses in cases of tumors located in the right chambers. While this is a benign tumor, it causes significant deterioration of the general condition with poor prognosis if early surgical treatment is not followed.

Conflicts of interest

None declared.

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

Ethical considerations

Not applicable

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Two Cases of Myocardial Infarction with Normal Coronary Arteries in the Context of Acute Coronavirus Infection

We report two cases of myocardial infarction with non-obstructive coronary arteries (MINOCA) in the context of acute (SARS-COV-2) coronavirus disease.

Case 1

A 63-year-old man with type II diabetes, who had suffered from COVID-19 (mild symptoms) two weeks before, consulted for prolonged precordial pain lasting 8 hours with ST-segment elevation on ECG (Figure 1A), elevated high-sensitivity troponin I (TnI US), and creatine phosphokinase (CPK).

On admission, the patient was hemodynamically stable and asymptomatic. The echocardiography showed basal inferior and basal inferolateral hypokinesis, with no thinning or thickening of ultrasound refraction. Left ventricular systolic function was preserved.

High-sensitivity troponin I was 1000 ng /mL (reference value: negative < 0.023 ng/mL).

The symptoms were defined as acute coronary syndrome with ST-segment elevation. The patient was admitted to the Coronary Care Unit, and was administered aspirin, clopidogrel, statins, and carvedilol. Coronary angiography showed angiographically normal coronary arteries. The patient made good progress, with typical CPK curve and a maximum value of 856 IU/L (Figure 1B). In view of suspected MINOCA, a cardiac magnetic resonance imaging (CMR) was performed, revealing a sequela of basal, inferolateral transmural necrosis (Figure 1 C, D & E).

Case 2

It is a 55-year-old man with untreated hypercholesterolemia (LDL cholesterol 190 mg/dL), and a history of COVID-19 with bilateral pneumonia, requiring hospitalization in the general ward for 7 days (under oxygen therapy and dexamethasone).

Fifteen days after discharge, the patient presented for an outpatient cardiology consultation due to persistent dyspnea on exertion. ECG showed sinus rhythm, with negative T-waves in V4 to V6, findings that were not present in the ECG on the day of admission due to COVID-19 (Figure 2 A & B). It should be noted that at no time during hospitalization or on the following days had the patient experienced precordial pain.

Echocardiography was normal. High-sensitivity troponin I value on the day of consultation was normal. In the absence of diagnostic findings in the routine testing, a CMR was performed, which revealed a small, apical transmural infarction with sessile apical thrombus of 0.8 cm (Figure 2 C&D). Coronary angiography showed no epicardial coronary artery obstruction. Treatment with aspirin and statins was indicated as secondary prevention.

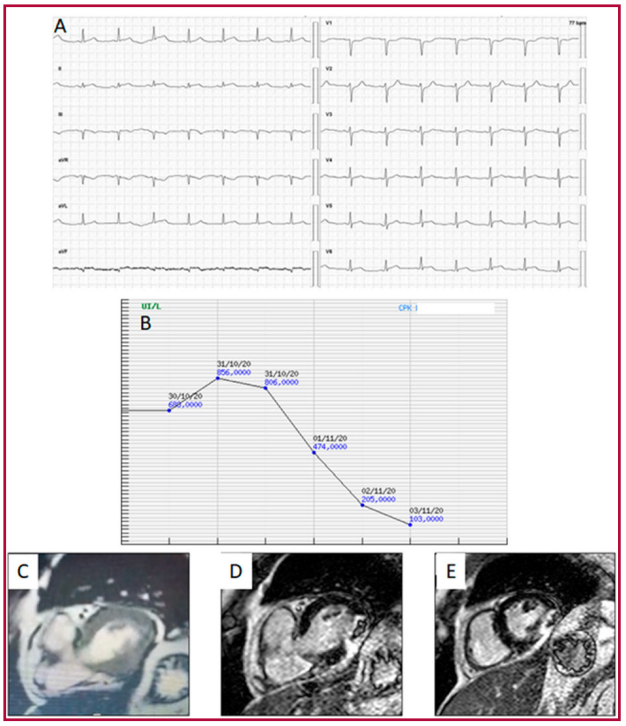

Fig. 1. A. ECG on admission. **B.** CPK curve. Cardiac magnetic resonance imaging: **C.** Short-axis cine sequence showing akinesia and thinning of the basal inferolateral segment. **D & E.** Late gadolinium enhancement sequence showing transmural sequela with no-reflow phenomenon.

Although COVID-19 is seen as a disease that primarily affects the lungs, cardiovascular involvement due to thromboembolic effect has been described. Moreover, the risk for an acute myocardial infarction is significantly increased in the context of acute/subacute COVID-19. (1) Regarding the incidence of MINOCA in the COVID-19 population, in a cohort of COVID-19 patients with acute coronary syndrome (ACS), 54% had coronary arteries without significant obstructions (6/11 patients). (2) In another study comparing 2019 (pre-pandemic) and 2020 cohorts of ACS patients, significantly fewer patients were found to have a culprit lesion in the 2020 cohort compared to the 2019 cohort (58.5% versus 74.2%, $p = 0.004$) and even fewer COVID-19 patients compared to patients without the infection (30.8% versus 61.9%, $p = 0.032$). (3) In view of these findings, it is particularly important to be aware of the coronary anatomy in coronavirus-associated ACS, since the presence or absence of epicardial coronary obstructions determines different nosological entities. (4) The causal pathway of myocardial damage in patients with COVID-19 has not been elucidated, but multiple pathophysiological mechanisms have been considered, such as direct viral infection of the myocardium, immune-mediated cardiac injury, cytokine storm, hypercoagulability and thrombosis of the microvasculature. (5) Furthermore, clinical presentation of the cases reported here (anginal pain, ECG compat-

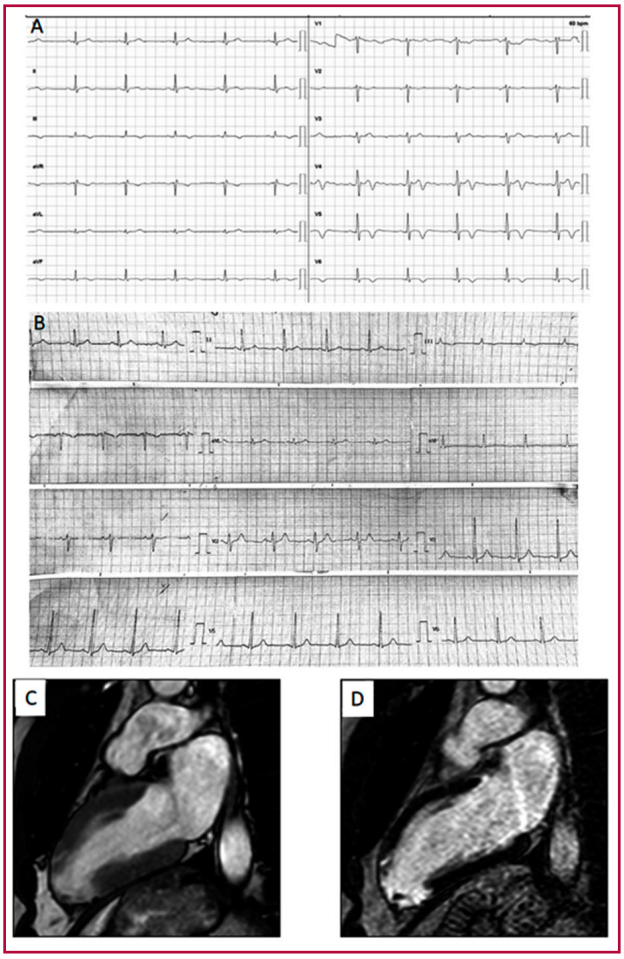

Fig. 2. A. ECG 3 weeks after hospitalization, showing negative T-waves in V3 to V6. **B.** Previous ECG (on the day of admission due to COVID-19) was normal. Cardiac magnetic resonance imaging: **C.** Two-chamber cine sequence showing akinesia, apical and inferoapical thinning with mass inside, consistent with thrombus. **D.** Late gadolinium enhancement sequence, 2-chamber view, showing transmural sequela with thrombus in the left ventricular apex.

ible with echocardiography, typical enzymatic curve in case 1; abnormal ECG in case 2 initially suggested ischemic etiology. However, once coronary disease was ruled out, it was CMR the tool that led to the definitive diagnosis of infarction, evidencing transmural fibrosis in the coronary territory. CMR is very useful for a wide spectrum of heart diseases, providing information on morphology, motility and tissue characterization. It has become an essential step for the diagnosis of MINOCA. (6) These two cases —presented as MINOCA occurring during the course of acute COVID-19— suggest a particular pathophysiology in this epidemiological setting. It is still unknown whether the management of this condition is equivalent to that of conventional ACS regarding the benefit of dual antiplatelet therapy and statins, or in terms of prognosis and likelihood of recurrence once the infection has been overcome. Finally, it is important to consider MINOCA among the differential diagnoses of ACS in general, but particularly in the context of COVID-19, where the absence of coronary obstructions seems to have a higher relative prevalence. While there may be pathophysiological differences in MINOCA between patients with or without COVID-19, the increased number of cases in pandemic provides an invaluable opportunity to obtain robust information on the progression and prognosis of this heart disease.

Conflicts of interest

None declared.

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Ethical considerations

Not applicable

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Electrical Cardioversion: An Almost Unknown Cause of Diffuse Alveolar Hemorrhage

Diffuse alveolar hemorrhage (DAH) is a potentially life-threatening event occurring due to disruption of the alveolar-capillary basement membrane. Systemic vasculitis is among the most frequent etiologies, (1) although theoretically any source of injury to the alveolar microcirculation may cause alveolar hemorrhage. Pulmonary capillaritis is the most usual histological

feature. ()

DAH is diagnosed with increased frequency, occuring at any age and usually with an established associated disease. Hemoptysis is the clinical hallmark, but may be absent in 33% of cases. Other symptoms are nonspecific, including cough, dyspnea, chest pain and fever. (3) DAH after cardioversion is a very rare event. To the best of our knowledge, only one case report has been published regarding this issue.

We report the case of a 47-year-old male with history of dyslipidemia, paroxysmal atrial fibrillation and a past episode of hemoptysis after electrical cardioversion. He had no occupational or toxic exposures and no current medication or allergies were known. The patient presented to the emergency department in 2019 with complaints of palpitations and fatigue. Electrocardiogram showed atrial fibrillation with rapid ventricular response. Electrical cardioversion was attempted four times, without success. After clinical stabilization, the patient was discharged with oral anticoagulation (apixaban) and amiodarone. A few hours later, before starting any medication prescribed, he was readmitted due to orthopnea and moderate volume hemoptysis. Physical examination revealed fine crackles on pulmonary auscultation and initial blood testing was normal. Chest x-ray showed de novo bilateral heterogeneous opacities. Chest computed tomography (CT) revealed diffuse bilateral peribronchial ground glass opacities, compatible with both pulmonary edema and hemorrhage, and also revealed small bilateral pleural effusion (Figure 1). Hemoptysis stopped after aminocaproic acid perfusion. Transthoracic echocardiography was performed due to uncontrolled heart rate and dyspnea, revealing severe left ventricular dysfunction with diffuse hypokinesis and ejection fraction of 25%. The patient was transferred to the cardiology intensive care unit for cardiovascular stabilization. Bronchoscopy was not performed at this time due to severe cardiac dysfunction/clinical instability and due to good response to treatment. He was clinically stabilized and discharged asymptomatic. Control chest CT scan was performed two weeks after the event, showing significant radiological improvement (Figure 2).

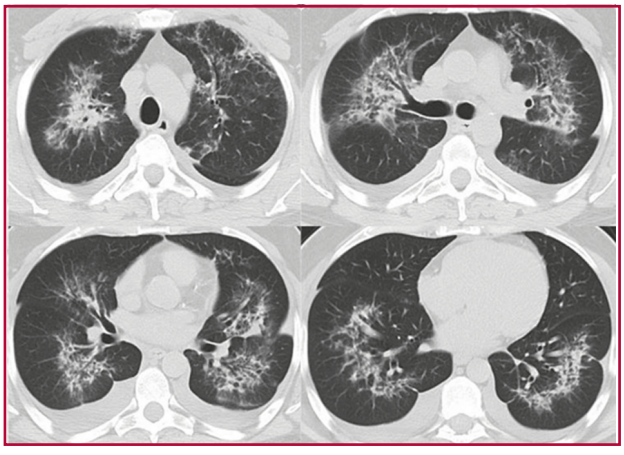

Fig. 1. Chest computed tomography with bilateral peribronchial ground glass opacities and small bilateral pleural effusion

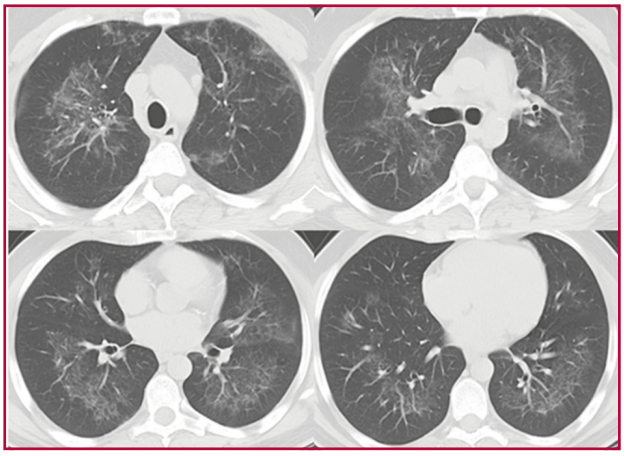

Fig. 2. Re-evaluation chest computed tomography performed 2 weeks after the event, showing significant improvement.

Secondary causes of hemoptysis, such as infection, bronchiectasis or vasculitis, were excluded. Hemogram, C-reactive protein and serum immunoglobulins were normal; autoimmune study (rheumatoid factor, ANA, ANCA, Anti-GBM, C3 and C4, dsDNA, SSA/SS-B, anti-ribonucleoprotein and anti-JO-1) was negative. The time relation between electrical cardioversion and hemoptysis occurrence, the radiological pattern, the exclusion of other potential causes and the past history of hemoptysis after a previous electrical cardioversion raised the suspicion of alveolar hemorrhage secondary to electrical trauma. The co-occurrence of pulmonary edema due to severe cardiac dysfunction made this diagnosis a greater challenge. Unfortunately early bronchoscopy was not possible to perform as indicated in most cases to confirm diffuse alveolar hemorrhage. Hemoptysis was crucial for the clinical suspicion of DAH. Moreover, a decreased red blood cell count would have agreed with a blood loss (hemorrhage).

There are a few reported cases of alveolar damage caused by amiodarone, yet most of them are described after long-term and high dose intake. (4) Electrical cardioversion is a widely performed procedure in the emergency department to treat arrhythmias with a low complication rate.3 It has been described as a cause of DAH once, in 2006, in a South Korean hospital. A 33-year-old female patient presented to the emergency department with dyspnea and hemoptysis, twelve hours after being submitted to electrical cardioversion. CT scan abnormalities were similar to the ones described in the present case and disappeared spontaneously on follow-up CT. (5)

It may be possible that the association between electrical cardioversion and alveolar hemorrhage is

underreported in the absence of overt blood losses. To conclude, though rare, alveolar hemorrhage due to electrical trauma should be considered as a differential diagnosis in a patient with hemoptysis after an electrical cardioversion procedure.

Conflicts of interest

None declared.

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

Ethical considerations

Not applicable

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Same-day-discharge after transcatheter aortic valve replacement

Aortic valve disease is the most common primary valvular affection worldwide. Degenerative etiology is the most frequent cause and its prevalence is increasing due to the aging population. Surgery or percutaneous interventions are the standards of care in symptomatic patients.

Transcatheter aortic valve replacement (TAVR) is a less invasive procedure that has been rapidly evolving for the past 20 years. The minimalist TAVR approach is currently applied in experienced centers allowing not only to obtain better outcomes but shorter hospital length of stay and faster social reinsertion, particularly important among elderly patients. Early discharge has shown to be beneficial for patients and hospitals in reducing complications and health care costs. Same day discharge (SDD) after TAVR has been reported during Covid-19 pandemic to avoid hospitalization. Due to the increment in the safety of the procedure, it is becoming an alternative strategy for a selected group of patients. We present 2 cases of SDD.

Patient 1: An 84-year-old male patient with symptomatic severe aortic valve stenosis with a transthoracic echocardiography (TTE) showing a mean gradient of 50mmHg and valvular area of 0.3 cm2, with STS risk score of 3.4% and EuroSCORE of 1.1% was presented in the Heart Team of our institution.

He had a history of permanent pacemaker implantation 4 years ago. After a multidisciplinary discussion, TAVR was selected for this patient due to his age and frailty. An elective procedure was performed with a transfemoral implantation of a Medtronic Evolut Pro 29 mm valve through a minimalistic approach with conscious sedation. During the procedure a transesophageal echocardiogram (TEE) revealed a correct valve implantation and a mild-moderate aortic regurgitation due to severe nodular calcification in the aortic annulus. The patient was continuously monitored and electrocardiograms were performed immediately, 3 and 6 hours post-intervention. Four hours post-procedure, the patient was allowed to walk around without any femoral complication. Before discharge, control TTE showed a mild aortic regurgitation. The clinical course of the patient was favorable without any complications during the 6hour observation period and was discharged with guidelines. Virtual follow up the day after the procedure and a return visit to the clinic was done on day 7 with a control TTE. The patient showed no adverse events and returned to daily activity after 48 hours of the procedure.

Patient 2: A 77-year-old male patient with symptomatic severe aortic valve stenosis with a TTE showing a mean gradient of 78 mmHg and area of 0.5 cm2. The patient had a history of complicated coronary artery bypass grafting (CABG) when he was 61 years old, so he rejected a surgical valve replacement. An elective procedure was performed with a transfemoral implantation of a Myval 27.5 mm valve using a minimalistic approach. The TEE during the procedure

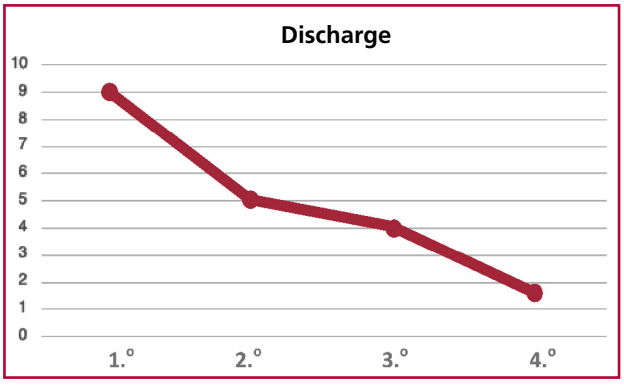


Fig. 1.Hospital length of stay (in days) after TAVR from 2009 to 2021.

showed mild paravalvular regurgitation without any other complications. The patient was continuously monitored for 6 hours and he did not present any conduction disturbances nor vascular complications. He was able to walk and was discharged on the same day without any complications with outpatient controls on day 1 and 7 after the procedure.

SARS-CoV-2 pandemic has led to reduce the number of procedures to preserve resources and hospital capacity in many institutions. Procedures with an early discharge protocol have popularized suddenly worldwide.

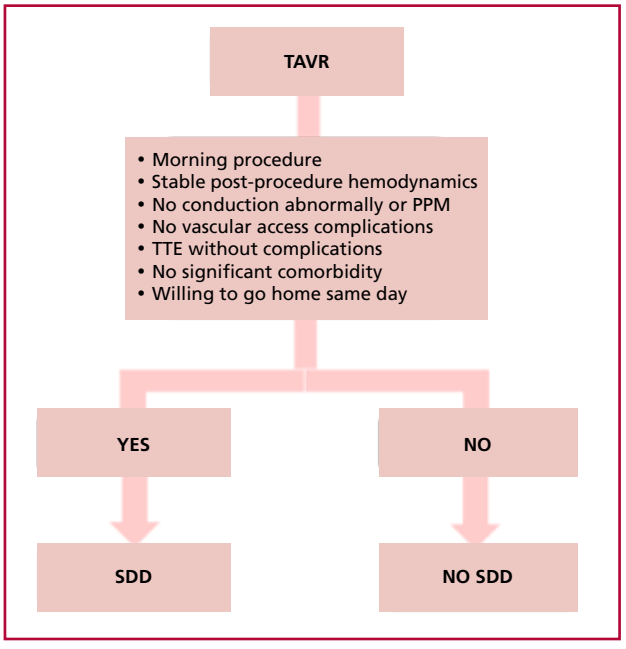
The evidence with SDD TAVR so far is limited to isolated case reports, small case series and a retrospective analysis. However, a selected group of patients could benefit with this approach. The adoption of a minimalistic approach for TAVR has allowed for a reduction in hospital length of stay, complications and cost.

The most common concerns in early discharge in TAVR have focused in late conduction disturbances and the need for permanent pacemaker implantation. Improvement in the devices design and in the technical aspects of the intervention have dramatically diminished these complications. According to our experience, hospital length of stay has been reduced during the past decade (Figure 1). Therefore, patients with previously implanted permanent pacemakers and/or those without prior conduction disturbances without any procedural complication undergoing TAVR represent a suitable group of patients to initiate SDD protocols safely.

TAVR registries suggest that it is unlikely that patients who do not develop conduction disorder during the index TAVR procedure or the following six hours would do so later on. Therefore, extended hospital monitoring might not be always mandatory.

In order to identify patients who could be potentially discharged home on the same day after TAVR, it is necessary to evaluate demographic and procedural variables associated with early morbidity, such as high-risk comorbidities, procedural complications (vascular complications, paravalvular leak, excessive contrast use) and discharge planning issues (lack of social support and difficulties in outpatient follow up). Medical follow up and real-time remote cardiac monitoring with many different devices might be crucial to react rapidly to potential complications among certain group of patients (Figure 2).

There are several limitations to SDD evidence so far. Highly selected, moderate to low-risk patients have been chosen to go home early worldwide. Event rates in these patients are low and therefore, the evidence is scarce. The need for standardized protocols and wider evidence is crucial before SDD TAVR becomes a daily practice.



SDD:same day discharge

Fig. 2. SDD algorithm.

Conflicts of interest

None declared.

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

Ethical considerations

Not applicable

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