LETTERS TO THE EDITOR

The de Winter electrocardiographic pattern as an ST-elevation myocardial infarction equivalent in a young patient with COVID-19

Patrón de "de Winter" como equivalente de infarto de miocardio con ST elevado en un paciente joven con COVID-19

To the editor:

The COVID-19 pandemic has had a major impact on emergency medical services (EMS)[1]. The barriers created to combat infection and minimize exposure hinder adequate patient assessment and expose patients to diagnostic and therapeutic delays. Thus, while clinical pulmonary COVID-19 focuses much of the effort on EMS, other manifestations such as acute myocardial infarction (AMI) may go unnoticed. An example would be electrocardiographic equivalents (ECG) of ST-elevation (STE), which are associated with acute coronary thrombotic occlusion in the absence of a truly elevated ST segment[2-4].

A 33-year-old male smoker with no family history of heart disease, a positive SARS-CoV-2 PCR (polymerase chain reaction), and a mild viral infection; as a sequela he had reactive panic disorder. During the second week of isolation, he came to the emergency room showing extreme aggressiveness and agitation with difficult anamnesis. He was afebrile and had no specific findings on examination. The narcotic test and chest radiology were normal. After controlling the initial state of agitation, a picture of oppressive chest discomfort and other pericardial characteristics, started the day before admission, was identified as a trigger. The ECG without pain did not suggest ischemic etiology (Figure 1). It was classified as acute pericarditis, and myocarditis and acute coronary syndrome were included in the differential diagnosis. After detecting ultrasensitive troponin T elevation, the bedside echocardiogram revealed apical and anterolateral hypokinesia with an image suggestive of apical thrombus (Figure 2a) which was confirmed with echocardiography (Figure 2b). The ECG was reinterpreted as a "de Winter" pattern secondary to evolving AMI, complicated with pericarditis (Dressler's syndrome) and intraventricular thrombus in the context of COVID-19. Coronary angiography confirmed proximal thrombotic occlusion of the proximal anterior descending (ADA) (Figure 2c), with a high thrombotic burden that required manual thrombectomy with aspiration catheter (Figure 2d) and subsequent stent implantation in the proximal ADA. Given the high thrombotic bur-

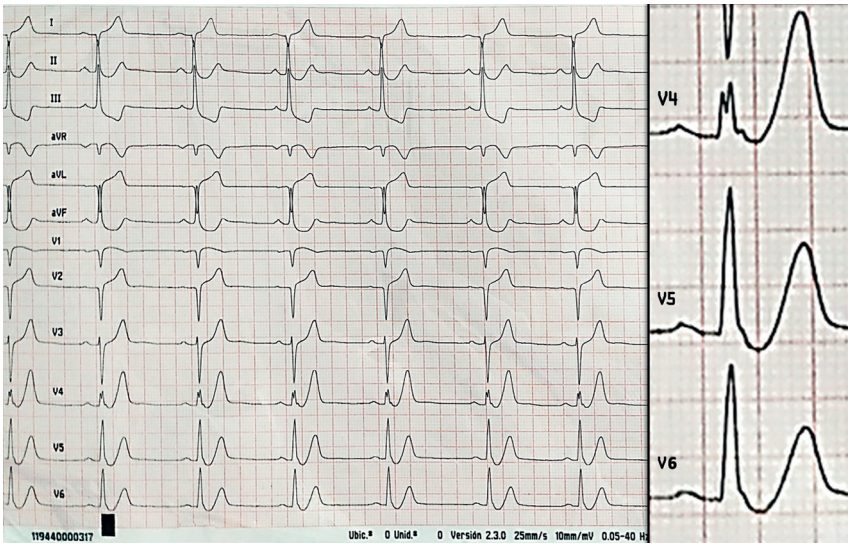

Figure 1. ECG on admission. Ascending ST-segment depression at the J-point level, followed by symmetrical high T waves from V4 to V6, compatible with a "Winter's" pattern.

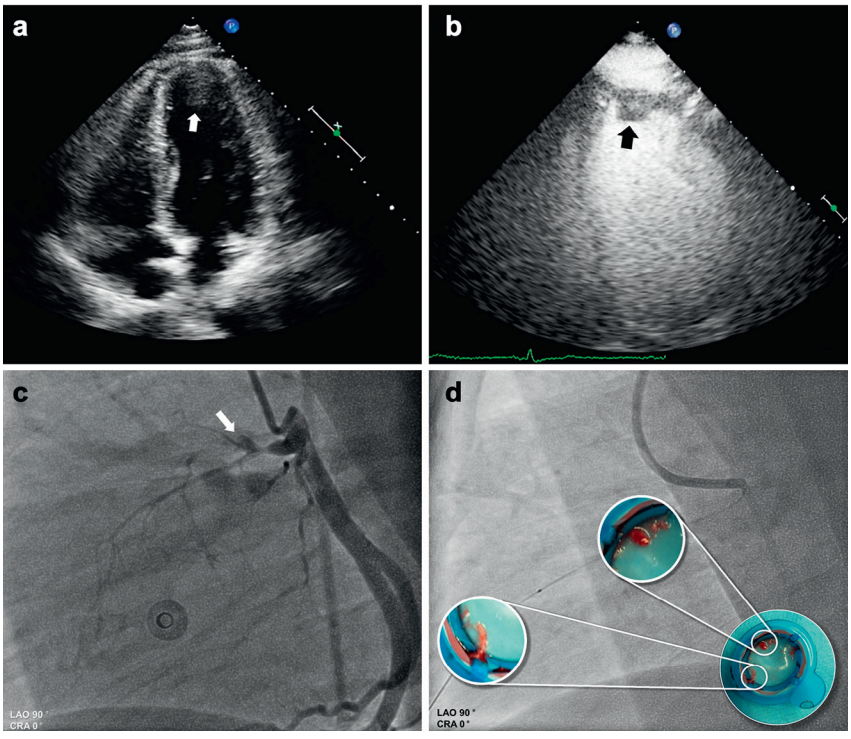

Figure 2. Echocardiography and coronary angiography. a. Apical 4-chamber echocardiographic window: mass compatible with AT (white arrow). b. Echocardiogram with echocardiographic echocardiographic echocardiography: AT (black arrow). Echocardiogram with echopotentiators: AT (black arrow) after opacification of the ventricular cavity. c. Thrombotic occlusion of the proximal LAD (white arrow). d. Coronary thrombectomy. Lower left circle: recent platelet-rich "white" thrombus obtained from the distal LAD. Upper-right circle: older "red" thrombotic material rich in red blood cells extracted from the proximal occlusion.
ADA: anterior descending artery; AT: apical thrombus.

den and low bleeding risk, dual antithrombotic therapy with prasugrel 10 mg/day and dabigatran 150 mg/12 hours (off-label indication, due to intraventricular thrombus) was decided. Two months after discharge, a cardiac MRI showed complete resolution of the thrombus, with no hemorrhagic complications.

The "de Winter" pattern is considered an equivalent of STE reflecting proximal ADA occlusion, with a positive predictive value of 95%[2]. This pattern is not specific for COVID-19: it appears in 2-3% of anterior AMI, especially in young men in whom it could be misinterpreted as early repolarization or pericarditis, causing reperfusion delays[4]. In contrast to the "hyperacute" T waves that precede ST-elevation in AMI, this pattern was described as a static marker, although recent data question it. Regardless of age, myocardial damage is a frequent finding in patients hospitalized for COVID-19[5] and, although they often have asymptomatic nonobstructive coronary artery disease, they may have a high associated thrombotic burden6. Echocardiography is essential for the differential diagnosis of chest pain in EMS. The thrombotic material extracted suggested a thrombotic phenomenon of different chronology, with older proximal occlusive thrombus rich in red cells ("red thrombus"), and a superimposed thrombosis in distal segments of the of more recent chronology and with higher platelet content ("white thrombus"), which would explain the subacute clinical course of the patient. Although the pathophysiology of COVID-19-associated coagulopathy remains to be resolved, the role of an individual excessive inflammatory response secondary to the "cytokine storm" triggered by SARS-CoV-2 is accepted; distinct from forms of disseminated intravascular coagulation in the context of sepsis. In the current epidemiological scenario, it is necessary to maintain a high index of suspicion to identify acute cardiovascular manifestations by COVID-19 in EMS, even in apparently healthy young patients, with early identification of STE equivalents, such as the "de Winter" pattern.

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