Valvular heart disease is one of the most common causes of cardiovascular disease worldwide, with a prevalence of 2.5%/1,2. To treat this disease, valvular heart replacement is the most important form of treatment for rheumatic valvular disease. From 1963, until the late 90s, Starr–Edwards prosthetic valves were placed in aortic and mitral position but due to the high occurrence of thrombotic events, they were replaced with other prosthetic options. In this case, we report a patient with a long-standing Starr–Edwards mechanical prosthetic valve in mitral position who had a myocardial infarction.

A 64-year-old woman treated in 1968 with a Starr–Edwards prosthetic for rheumatic mitral stenosis when she was 13 years old, had typical chest pain so she was admitted to the emergency room in December 2018. At her arrival, physical findings included a normal neurologic examination, intense second heart sound, and protosystolic mitral murmur with normal opening and closing clicks. The electrocardiogram showed sinus rhythm with Q wave in inferior leads, while troponin I essay was positive with no elevation in brain natriuretic peptide or alteration in renal function. Furthermore, echocardiogram reported a normofunctioning prosthetic caged-ball valve with the following parameters: maximum velocity of 1.5 m/s, maximum gradient of 10 mmHg, mean gradient of 32 mmHg, velocity time integral 36 cm, and in vitro diagnostics 1.4. Due to the findings, she was admitted to catheterization laboratory and no angiographically significant lesions were found (Figs. 1 and 2). Due to the typical chest pain, the electrocardiogram, and laboratory findings, she was diagnosed with myocardial infarction with non-obstructive coronary arteries.

Starr–Edwards prosthetic valves have considerable risk of thrombotic events, with a rate of 4-6%/year1,3. In this valve, the ball travels completely outside the orifice, reducing the possibility of thrombus or pannus because the contact points are constantly changing therefore reducing surface contact. Nevertheless, they are more dangerous when placed in mitral position due to greater risk of the left ventricle outflow tract obstruction by compression of the ventricular wall3,5.

Worldwide, there are case reports of patients that have lived up to 45 years after surgery without any serious complications or loss of the valve's structural integrity in mitral position6. This case is relevant because, to the best of our knowledge, this is the first report of a patient with a Starr–Edwards prosthetic valve with a follow-up of 51 years without loss of integrity but with a well-known complication such as myocardial infarction with non-obstructive coronary arteries7-9.

Myocardial infarction with non-obstructive coronary arteries has a prevalence of 5-10%, which means that...
at least 1-2 of every 20 acute myocardial infarctions do not have significant coronary stenosis. The mechanisms causing this kind of myocardial infarction are plaque disruption, spasm, thromboembolism, dissection, microvascular dysfunction, supply/demand mismatch, myocarditis, or Takotsubo cardiomyopathy\textsuperscript{10}. In our case, a possible etiology is thromboembolism that may have spontaneously resolved because the patient had typical chest pain and troponin I elevation, and both cannot be explained by other causes such as acute heart failure or acute kidney injury.

Fortunately, nowadays, safer prosthetic valves are available. However, we must not forget that on our daily practice, we can still find patients with Starr–Edwards prosthetic valves. This fact expands our boundaries to have medical evidence-based facts on this particular subject. We must recognize that Starr–Edwards valve carriers who have survived to our days – more than 25 years after these valves were replaced for newer prosthetic valves – can carry a good prognosis if we make adequate surveillance to prevent potential complications. We must recognize that with longer survival, there is greater risk for embolism but also an increase in the risk of bleeding because of longer anticoagulation and complications such as myocardial infarction can occur in this patient.

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**Conflicts of interest**

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**Ethical disclosures**

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