

# Pseudo-Myocardial Infarction During an Episode of Herpes Zoster

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*The patient arrived at the emergency unit with a history of acute myocardial infarction, for which she was treated. Without improvement in the pain, the patient developed heart failure and underwent a hemodynamic study, which showed normal coronary arteries and extensive ventricular impairment. During evolution, the clinical findings improved and herpes zoster appeared on the right shoulder. In a few months the clinical findings subsided, and the findings of the electrocardiogram, chest X-ray, and ventricular function were normal. The patient is currently asymptomatic.*

Clinical manifestation of myocarditis may vary from an asymptomatic state secondary to focal infection to severe heart failure. In some cases, clinical manifestations may simulate myocardial infarction<sup>1</sup>, as electrocardiographic and laboratory findings do. Approximately 25 viruses may be associated with myocarditis, including the varicella-zoster virus. Myocardial involvement during infection by herpes zoster virus is rare, and when it occurs, it is usually asymptomatic or manifests as heart failure<sup>2</sup>.

We report the case of a patient with clinical findings of myocardial infarction and infection by herpes zoster virus.

## Case Report

The patient is a white 68-year-old female, who arrived at the emergency unit complaining of precordial pain that irradiated to the left upper limb and was accompanied by sweating and weakness for 3 hours. The patient reported malaise in the previous 2 days, and a continuous discomfort in her right shoulder with no alleviating or worsening factors. The patient was hypertensive and had been using 20mg of enalapril for the last 5 years. She also reported having a family history of longevity.

On physical examination the patient was restless, pale, sweating, and dyspneic. Her blood pressure was 134/80mmHg in both upper limbs, her heart rate was 116bpm, the lungs showed no rales, and the peripheral pulses were palpable.

The clinical hypothesis of myocardial infarction led the patient to immediately undergo electrocardiography (fig. 1), which confirmed the diagnosis. A thrombolytic (streptokinase) was started, and hypotension occurred during infusion; therefore, the dripping was decreased, dopamine was started followed by dobutamine. After infusion, the patient remained restless, more dyspneic, and still complained of pain. On pulmonary auscultation, crepitant rales could be heard in the inferior 2/3 of the lungs. The patient was given sodium nitroprusside and a diuretic. The chest X-ray (fig. 2) showed the classical pattern of pulmonary edema. As the pain persisted after 4 hours of thrombolytic infusion, the patient underwent coronary angiography, which revealed normal coronary arteries and an increase in left ventricular end-systolic volume due to an extensive anterior and inferior akinetic area, and also apical dyskinesia (fig. 3). Table I shows the levels of the cardiac enzymes.

The echocardiogram of 3/12/96 revealed an ejection fraction of 45%, mild mitral insufficiency, a deficit in left ventricular relaxation, middle apical dyskinesia, and anteroinferior and septal akinesia. The remaining walls were hyperkinetic.

The patient gradually improved. Twenty-four hours after admission, the patient reported aggravation of the pain in her right shoulder and upper limb, and vesicles with a clear content and surrounded by a pink halo appeared on her right shoulder. The clinical diagnosis of herpes zoster was then established. Evolution of the electrocardiogram is shown in figures 4 and 5 and the chest X-ray in figure 6. The echocardiogram of 3/19/96 revealed akinesia in the apical portion of the septum and anterior wall, with no mitral insufficiency. On 4/9/96, the echocardiogram was normal with no changes in the segmentary contractility and normal relaxation.

Currently, the patient is using a calcium channel blocker (diltiazem) and is clinically well, with normal cardiac function and no deficit.

## Discussion

At admission, the patient met the diagnostic criteria for acute myocardial infarction and was managed accordingly. On evolution, the small increase in enzymatic levels for the

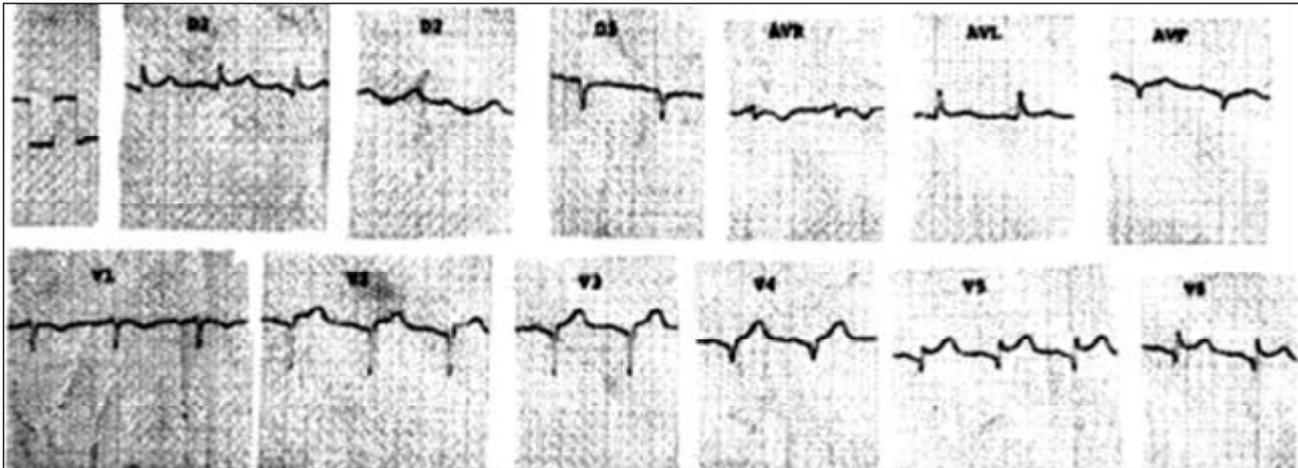


Fig. 1 – Electrocardiogram on hospital admission: elevation of the ST segment in D<sub>1</sub>, AV<sub>1</sub>, V<sub>2</sub>, V<sub>3</sub>, V<sub>4</sub>, V<sub>5</sub>, and V<sub>6</sub>; QS pattern in D<sub>2</sub>, D<sub>3</sub>, AVf, V<sub>2</sub>, V<sub>3</sub>, V<sub>4</sub>; and QR pattern in V<sub>5</sub> and V<sub>6</sub>. Conclusion: electrically inactive zone in inferior and anterolateral wall, anterolateral lesion.

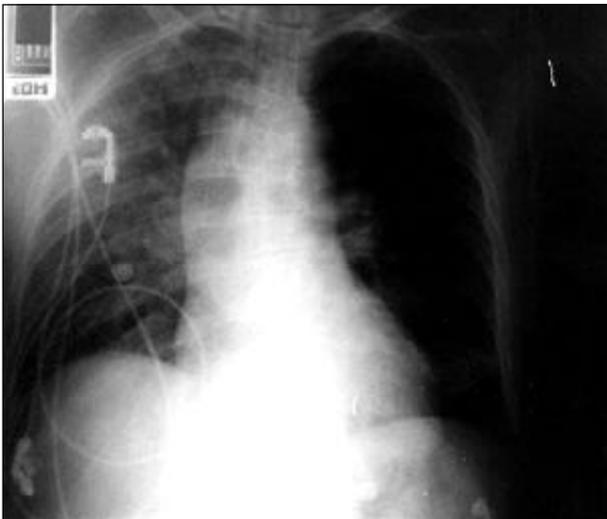


Fig. 2 – Chest X-ray (3/10/96). Pattern of pulmonary edema.

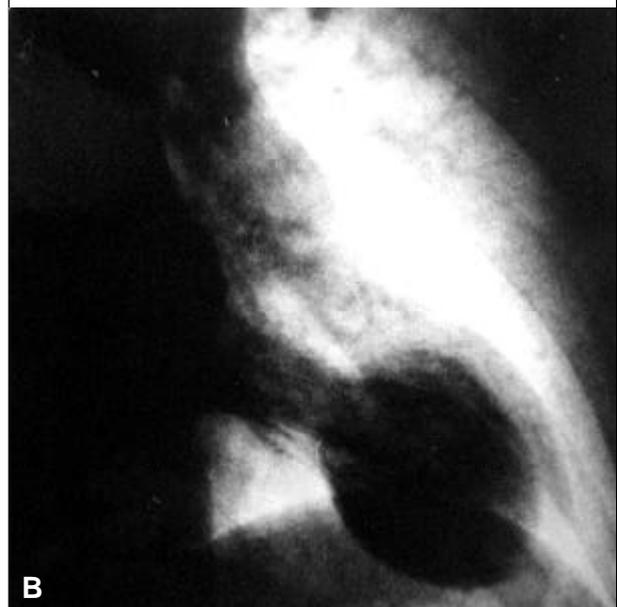
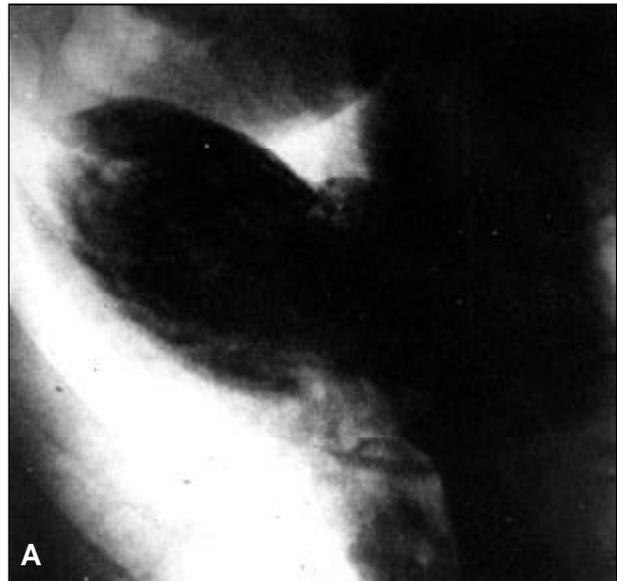


Fig. 3 – Angiographic study (3/10/96): normal coronary arteries, ventriculography shows an antero-inferior akinetic zone and an apical dyskinetic zone: a) end-diastole, b) end-systole.

Table I – Evolution of the cardiac enzymes		
	CPK	CKMB
Admission	140	18
6 hours	173	32
12 hours	261	64
Reference values: total CPK up to 60, CKMb up to 10.		

extension of the myocardial impairment was notable, the latter being clinically observed and angiographically and echocardiographically confirmed. During evolution, some vesicles appeared allowing the diagnosis of herpes zoster, which had already been manifesting as a discomfort in the right shoulder a few days prior to the episode of chest pain.

According to the report by Franken et al<sup>1</sup>, the new Q wave of the electrocardiogram should be interpreted and the findings referred to as an electrically inactive zone and not as a necrotic area.

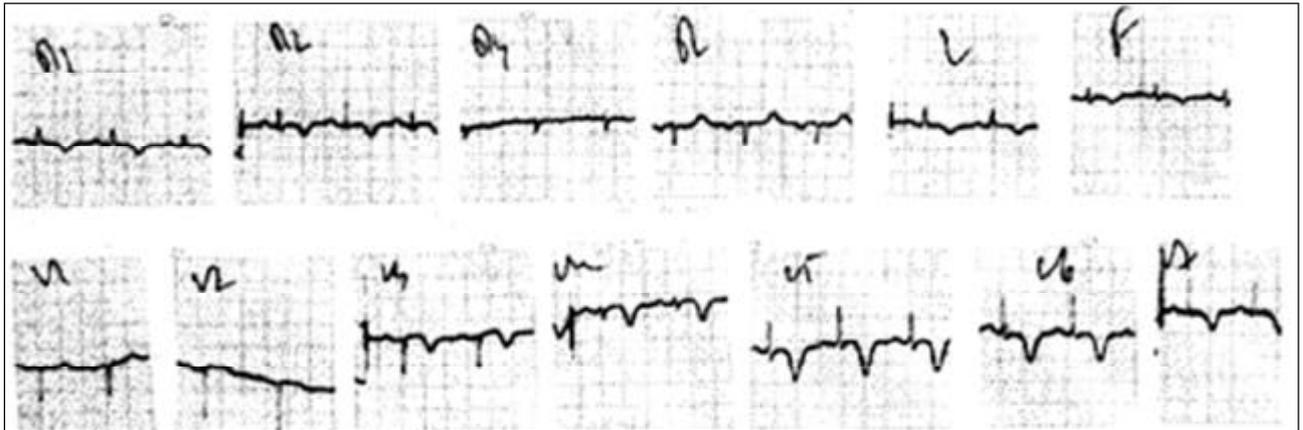


Fig. 4 - Electrocardiogram (3/30/96). Reappearance of R in D<sub>2</sub>, D<sub>3</sub>, AVf, V<sub>4</sub>, V<sub>5</sub>, and V<sub>6</sub>; diffusely negative T wave. Conclusion: anterior electrically inactive zone, diffuse alterations of ventricular repolarization.

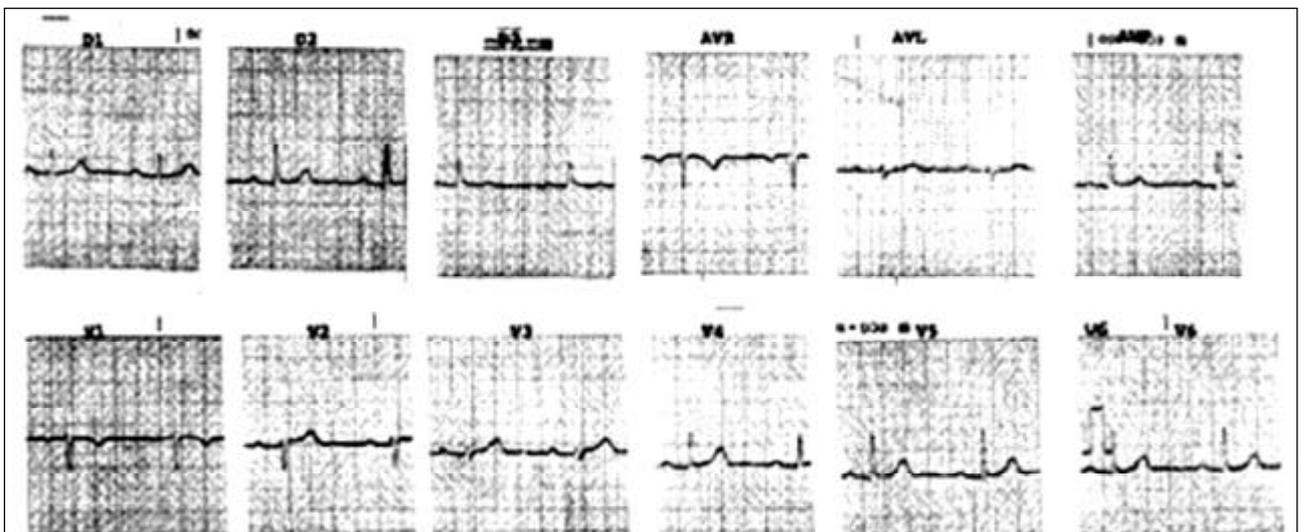


Fig. 5 - Electrocardiogram (6/10/96). Normal electrocardiogram.



Fig. 6 - Chest X-ray (6/10/96). Normal.

terized by transient myocardial impairment after an acute ischemic event, and results from cytoplasmic accumulation of calcium and free radicals. From the electrophysiological point of view, the stunned area becomes unresponsive to the electric stimulus and, consequently, noncontractile, perfectly mimicking an area of necrosis. The region stunned by electrolytic changes becomes incompletely repolarized (rest potential above  $-20$  mv) and, therefore, unresponsive.

We found reports in the literature of myocarditis associated with herpes zoster virus, in which the clinical manifestation is heart failure<sup>5</sup>. However, we found no report in the literature on stunned myocardium related to acute myocarditis, which would be perfectly acceptable from the pathophysiological point of view. Du Bois et al<sup>6</sup> reported a similar case induced by the use of interleukin-2 in the treatment of neoplasia.

We cannot, however, discard viral impairment of the coronary arteries, as has already been observed in epidemiological evidence relating acute coronary artery episodes to infectious disease<sup>7</sup>.

Heyndrick et al<sup>3</sup> reported the entity related to myocardial ischemia known as stunned myocardium. According to Braunwald and Kloner<sup>4</sup>, the stunned myocardium is charac-

## References

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