

# Discordant Electrocardiogram Left Ventricular Wall Thickness and Strain Findings in Influenza Myocarditis

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A 42-year-old man presented with a viral prodrome and tested positive for influenza A. He rapidly deteriorated developing cardiogenic shock, rhabdomyolysis, and acute kidney injury. Patient improved 1 week later with supportive measures including vasopressors, inotropes, and an intraaortic balloon pump. We report this case as it highlights the discordance between echocardiographic ventricular wall thickening as a result of myocardial edema, and electrocardiographic findings at presentation, with a reversal in findings at time of resolution. Additionally, there was some suggestion of a regional pattern to the reduced longitudinal strain. (Echocardiography 2015;32:1880–1884)

**Key words:** influenza, myocardial edema, longitudinal strain

## Case Description:

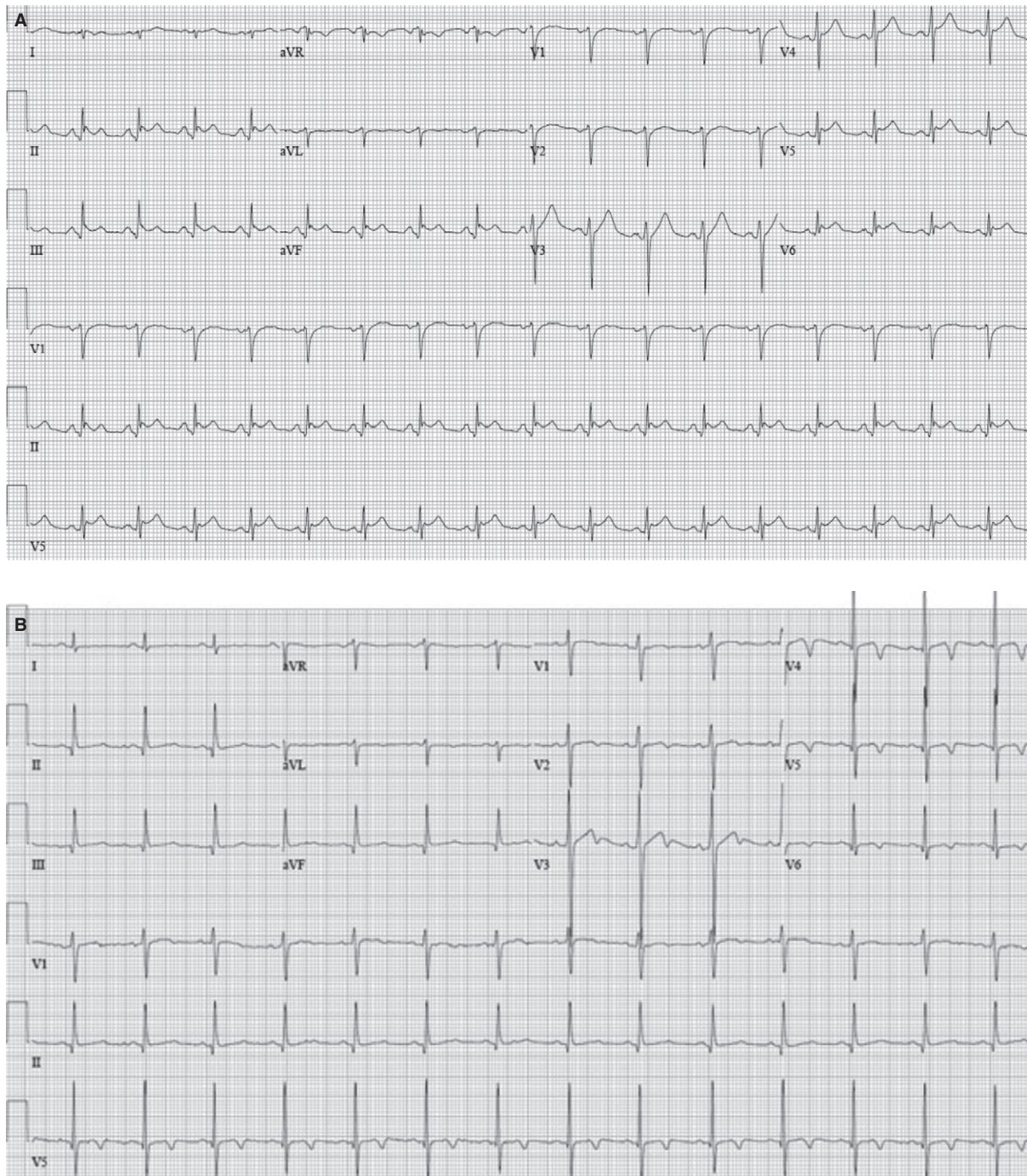
A 42-year-old previously healthy man presented to an outside facility with fatigue, myalgia, and fever of 1-week duration. Vital signs revealed a blood pressure of 97/66 mmHg, pulse of 116 beats/min, and temperature of 35.2°C. On physical examination, he appeared toxic and had a regular tachycardia and bibasilar crackles. Laboratory data demonstrated a creatinine of 2.2 mg/dL, lactate of 9.8 mmol/L, CK of 1035 U/L, MB of 22 ng/mL, and TnT of 0.05 ng/mL. Microbiological workup was positive for influenza A. Electrocardiogram (ECG) showed sinus tachycardia, PR elevation and ST depression in lead aVR, with PR depression and ST elevation in the remaining leads (most pronounced in the inferior/lateral leads; Fig. 1A). Over the following 24 hours, the patient required hemodynamic support and intubation for airway protection. An echocardiogram was obtained which showed significantly increased interventricular septal thickness at 1.7 cm and posterior wall thickness at 1.6 cm, with a left ventricular diastolic dimension (LVDd) of 3.6 cm, left ventricle mass indexed to body surface area (LVMI) of 136 g/m<sup>2</sup>, a globally reduced left ventricular ejection fraction (LVEF) of 30%, and a small circumferential pericardial effusion (Fig. 2A). Patient was subsequently transferred to our hospital for further management.

Patient had evidence of rhabdomyolysis with a significant elevation in CK, peaking at 38 000 U/L. The MB fraction was normal, and TnT was only minimally elevated (0.05–0.07 ng/mL). He remained oligoanuric despite aggressive volume resuscitation eventually requiring temporary renal replacement therapy. Patient required increasing doses of norepinephrine and dobutamine, and a Swan-Ganz catheter showed a low mixed venous oxygen saturation and cardiac index (53%, and 1.7 L/min per m<sup>2</sup>, respectively), with a pulmonary capillary wedge pressure of 18 mmHg. An intraaortic balloon pump (IABP) was placed, with successful weaning of vasopressors and inotropes. Over the following 7 days, hemodynamics improved, IABP was weaned, patient was extubated, and urine output and renal function normalized. Repeat ECG showed resolution of ST segment elevations and more prominent QRS voltages (Fig. 1B). Echocardiogram 10 days following the initial demonstrated an interventricular septal and posterior wall thickness at 0.9 cm, with an LVDd of 4.7 cm, LVMI of 73 g/m<sup>2</sup>, normal LVEF (60%), and the absence of pericardial effusion (Fig. 2B). Strain imaging was performed on the initial echocardiogram at time of presentation and upon resolution of myopericarditis (Fig. 3). Initial images showed a reduction in global longitudinal strain at –6.4%, with an increase to –13.7% on day 10.

## Discussion:

This case presents interesting observations linking ECG findings, echocardiographic findings, and

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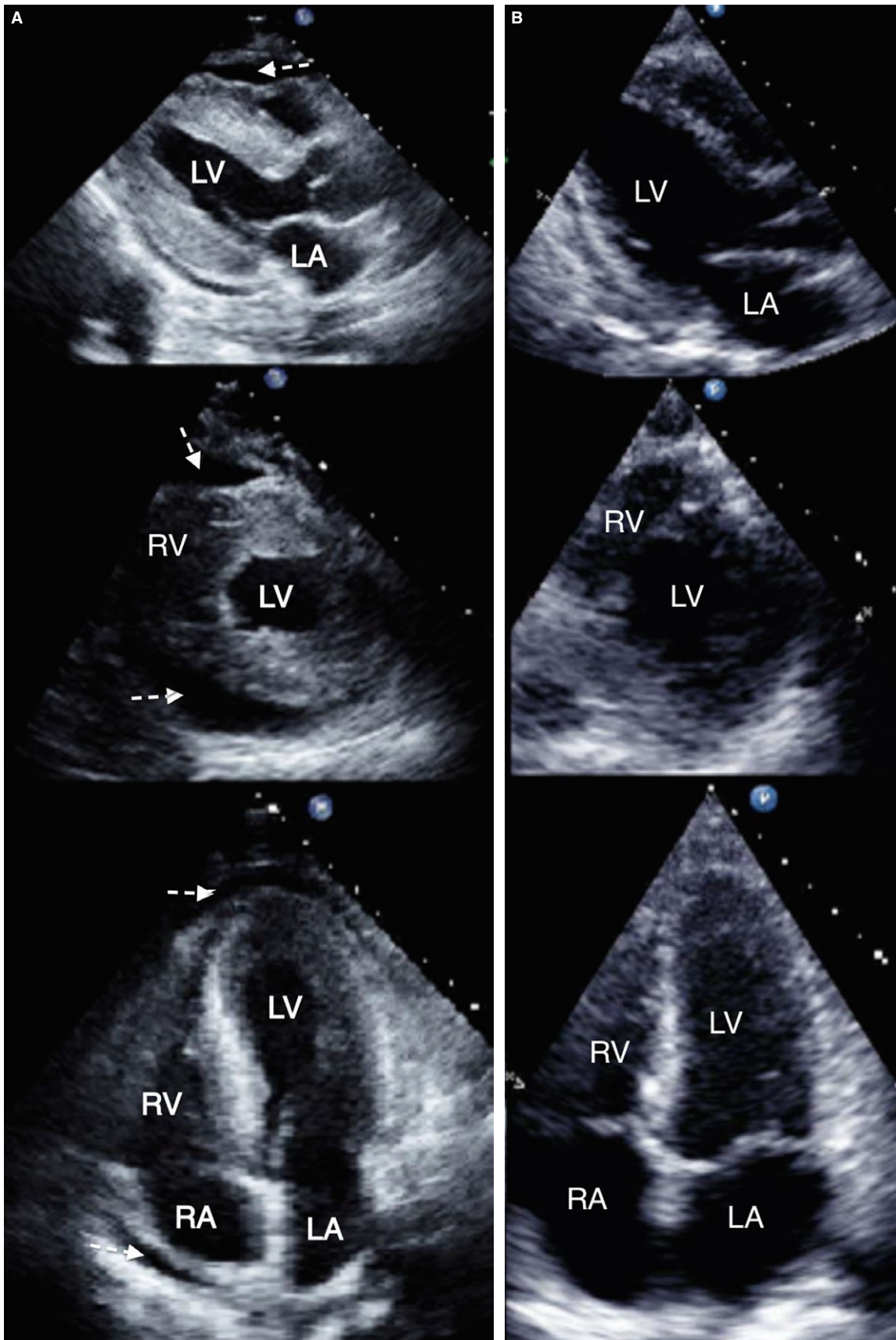


**Figure 1.** Initial electrocardiogram (ECG) **A.** on presentation and **B.** 10 days after presentation. ECG evidence of pericarditis is seen on presentation. Note the change in R-wave amplitude, most pronounced in leads I and V<sub>3</sub>–V<sub>6</sub>. The pericardial effusion was small in size and would likely not explain such changes.

strain imaging in a case of influenza-induced myopericarditis. The simultaneous involvement of both cardiac and skeletal muscle in the setting of influenza, as in our patient, is a rare entity with only a few reports in the literature.<sup>1</sup>

Cardiovascular complications of influenza were well described during the Asian influenza

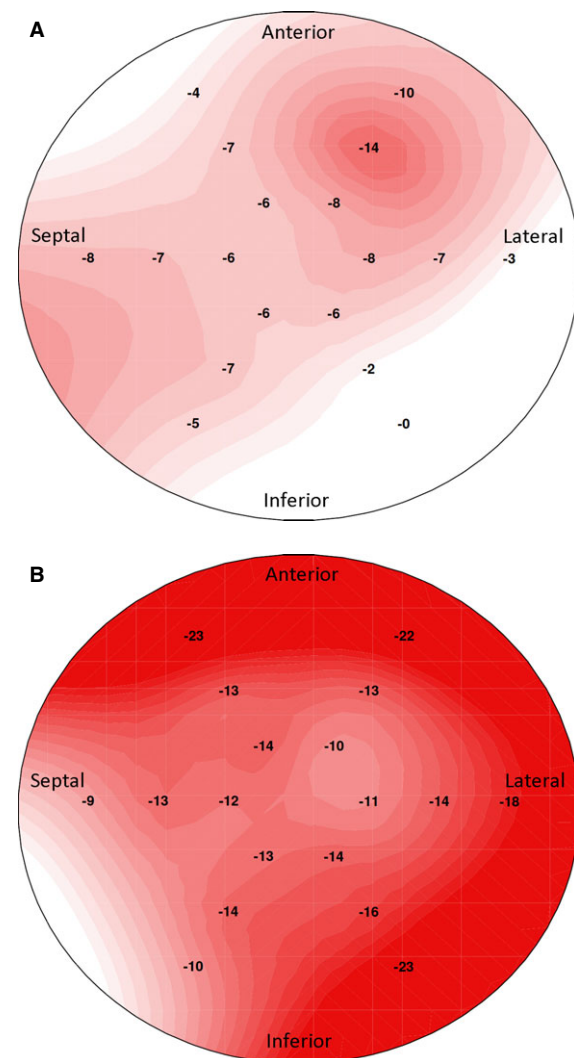
pandemic of 1957. In the Cleveland, Ohio area, multiple unexplained deaths were noted in young individuals, with autopsy revealing findings consistent with influenza-related complications. This prompted the Coroner's Office to study subsequent influenza-related deaths.<sup>2</sup> Of the 33 subsequent deaths, 13 had acute



**Figure 2.** Echocardiogram **A.** on presentation and **B.** 10 days after presentation. At resolution of myocarditis, the following changes were noted: interventricular thickness 0.9 cm (from 1.7 cm), posterior wall thickness 0.9 cm (from 1.6 cm), LV diastolic dimension 4.7 cm (from 3.6 cm), LV mass index 73 g/m<sup>2</sup> (from 136 g/m<sup>2</sup>), and LV ejection fraction 60% (from 30%). Pericardial effusion is indicated with a dashed arrow. LA = left atrium, LV = left ventricle, RA = right atrium, RV = right ventricle.

myocardial involvement at autopsy; 10 had acute myocarditis with varying degrees of interstitial cellular infiltration and edema; and four had fibrinoid changes in myocardial arterioles.

The R-wave amplitude in the ECG of our patient paralleled left ventricular function as assessed by speckle tracking echocardiography.



**Figure 3.** Strain imaging **A.** on presentation and **B.** 10 days after presentation. Note the basal inferior and lateral segments demonstrated a more pronounced involvement on presentation as well as the most recovery at resolution of myocarditis.

Conversely, the R-wave voltage did not correlate with LV wall thickness. The LVMI was nearly twice as high on presentation due to myocardial edema, in contrast to the R-wave voltage that was higher at resolution. Data suggest a correlation between anasarca in heart failure and lower ECG voltages.<sup>3</sup> Although our patient did have some evidence of anasarca and a small pericardial effusion, we believe that the predominant cause of reduced R-wave voltage was actually myocardial edema. This concept is similar to the discordance between voltage and wall thickness reported in amyloidosis.<sup>4</sup>

Interstitial inflammation and edema in acute myocarditis may translate into thickening of the ventricular wall as demonstrated in our patient. Hiramitsu et al. conducted a series of studies on this topic. They described 25 patients with acute myocarditis who underwent echocardiographic and histopathologic examination, demonstrating the association between ventricular wall thickening and interstitial edema.<sup>5</sup> A subsequent study established a direct correlation between stroke volume (SV) and LVDd and an inverse correlation between SV and ventricular wall thickness.<sup>6</sup> The sudden increase in LV wall thickness that may accompany acute myocarditis results in reduced LVDd and diminished stroke volume. In our patient, LVDd was 3.6 cm at the time of his transient wall thickening, increasing to 4.7 cm at time of resolution. The same authors demonstrated the poor prognostic implications of transient ventricular wall thickening in acute myocarditis.<sup>7</sup>

On presentation, there was a global reduction in strain with the basal inferior and lateral segments being predominantly affected. Interestingly, these segments had the highest relative rebound in strain at recovery. A case report illustrated a regional reduction in strain in the basal inferior and lateral segments in a patient with acute viral myocarditis who otherwise had preserved LVEF.<sup>8</sup> Moreover, a case series found reduced longitudinal strain to be associated with lymphocytic infiltrates in acute myocarditis even among those with normal LVEF.<sup>9</sup> At follow up, strain was similarly found to be lower in patients with ongoing inflammation. A detailed description of segmental analysis of strain was not provided in that series. Reports have indicated that delayed enhancement on cardiac MRI (cMRI) is most prominent in the lateral and inferior

segments.<sup>10</sup> Although cMRI was not performed in our patient, we speculate that an underlying delayed enhancement pattern may have been present. Future studies assessing segmental strain in acute myocarditis, as well as its association with delayed enhancement by cMRI, may prove useful.

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