A 30-year-old woman presented 1-month postpartum with new-onset biventricular heart failure for which no other contributing cause could be found clinically or on laboratory investigations. Her 2 previous pregnancies were uncomplicated. She was HIV-positive for the preceding 3 years, but had never been on antiretroviral therapy.

Initial echocardiography revealed a left ventricular (LV) end-diastolic diameter of 59 mm and LV ejection fraction of 32% (online-only Data Supplement Movies 1 and 2). There was evidence of LV noncompaction involving the apex and midinferior and midlateral walls of the LV that satisfied the Jenni criteria (Figure 1). There was no echocardiographic evidence of other congenital or organic valvular disease. Speckle tracking revealed rigid body rotation (ie, rotation at both the basal and apical levels of the LV occurred in a counterclockwise direction during systole; Figure 2). The patient was treated with furosemide, carvedilol, an angiotensin-converting enzyme inhibitor, and spironolactone. On reevaluation 6 months later, her functional status was New York Heart Association Class 1. Echocardiography revealed that LV ejection fraction was now 48% (online-only Data Supplement Movies 3 and 4). Further speckle-tracking analysis revealed that the LV’s base and apex were now rotating in the opposite directions as would be normally expected (Figure 3).

This report is the first to our knowledge that describes a patient with evidence of reversible rigid body rotation who satisfies the clinical criteria for the diagnosis of both peripartum cardiomyopathy and LV noncompaction.

Discussion

Peripartum cardiomyopathy is defined as the development of heart failure in the last month of pregnancy or within 5 months of delivery with no other identifiable cause for its onset. An association of LV noncompaction and peripartum cardiomyopathy was recently described. Rigid body rotation often occurs in patients with LV noncompaction and has been suggested as a functional diagnostic criteria. The normal rotational patterns of the apex and base occur because of the opposite movements of the subepicardial and subendocardial fibers during systole. The subepicardial fibers have a longer rotatory arm and, thus, exert the dominant force that causes the base to move clockwise and the apex counterclockwise during systole.

We postulate that, in this case, a differential pathophysiological process (such as focal myocarditis/myocardial edema) caused dysfunction of the basal and midwall subepicardial fibers such that the predominant effect was exerted by the subendocardial fibers in this region, leading to counterclockwise rotation. Although some degree of apical dysfunction occurred, the degree of abnormality was less extensive and, thus, did not negate the overall dominance of the subepicardial fibers, thereby causing counterclockwise rotation at the apex. The resolution of this pathophysiological process, characterized by improved LV ejection fraction, was accompanied by restoration of a normal twist pattern.

There are several limitations to this report. No cardiac MRI was performed to exclude myocarditis, although there were no clinical or biochemical abnormalities to suggest it. The impacts of varying loading conditions in the postpartum period as well as neurohumoral abnormality may vary with a later compensated state, and may account for the remodeling changes and reversal of twist patterns.

Reversal of rigid body rotation is an important finding that may account for improvement of LV ejection fraction and clinical status in peripartum cardiomyopathy, and requires further investigation.

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Disclosures
None.

References

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