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CASE REPORT

Biventricular pulsus alternans: An echocardiographic finding in patient with pulmonary embolism

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Abstract

Pulsus alternans is characterized by regular rhythm with beat-to-beat alternation of systolic pressures. Left ventricular alternans is usually found in severe left ventricular dysfunction due to cardiomyopathy, coronary artery disease, systemic hypertension, and aortic stenosis. Right ventricular alternans is usually associated with left ventricular alternans, right ventricular dysfunction, pulmonary embolism, and pulmonary hypertension. Biventricular alternans is rare and associated with severe left ventricular dysfunction and left anterior descending coronary artery disease. The exact mechanism of pulsus alternans has not been clearly delineated, and it has been remained a subject of investigation and conjecture since the nineteenth century. Two fundamental mechanisms have been proposed to explain ventricular alteration. The first, based on the Frank-Starling mechanism, proposes beat-to-beat alteration in end-diastolic volume accounted for the alternating contractile force. The second proposed mechanism which explains the physiology of pulsus alternans involves the abnormal calcium handling by cardiac myocytes. To the best of our knowledge, biventricular alternans in pulmonary embolism has not been previously reported in the medical literature. We present and discuss the mechanisms of pulsus alternans and its clinical implications.

Key words: Pulsus alternans; Biventricular alternans; Pulmonary embolism

Core tip: Biventricular pulsus alternans is a rare phenomenon and has only been described in few cases of severe left ventricle systolic dysfunctions and left anterior descending coronary artery disease. Pulsus alternans is an ominous sign that suggests severe heart failure; early recognition can aid in appropriate management and intervention, which may change patient outcome.

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INTRODUCTION

Pulsus alternans was first described by Traube in 1872, characterized by regular rhythm with beat-to-beat alternation of systolic pressure[1,2]. Left ventricular alternans occurs commonly in setting of severe left ventricular dysfunctions. Right ventricular alternans are rare, and biventricular alternans are even less common, with only a few case reported in the literature[3]. To the best of our knowledge, biventricular pulsus alternans in pulmonary embolism has not been previously reported in the medical literature. We describe a case report of biventricular pulsus alternans in pulmonary embolism and discuss its mechanisms and clinical implications.

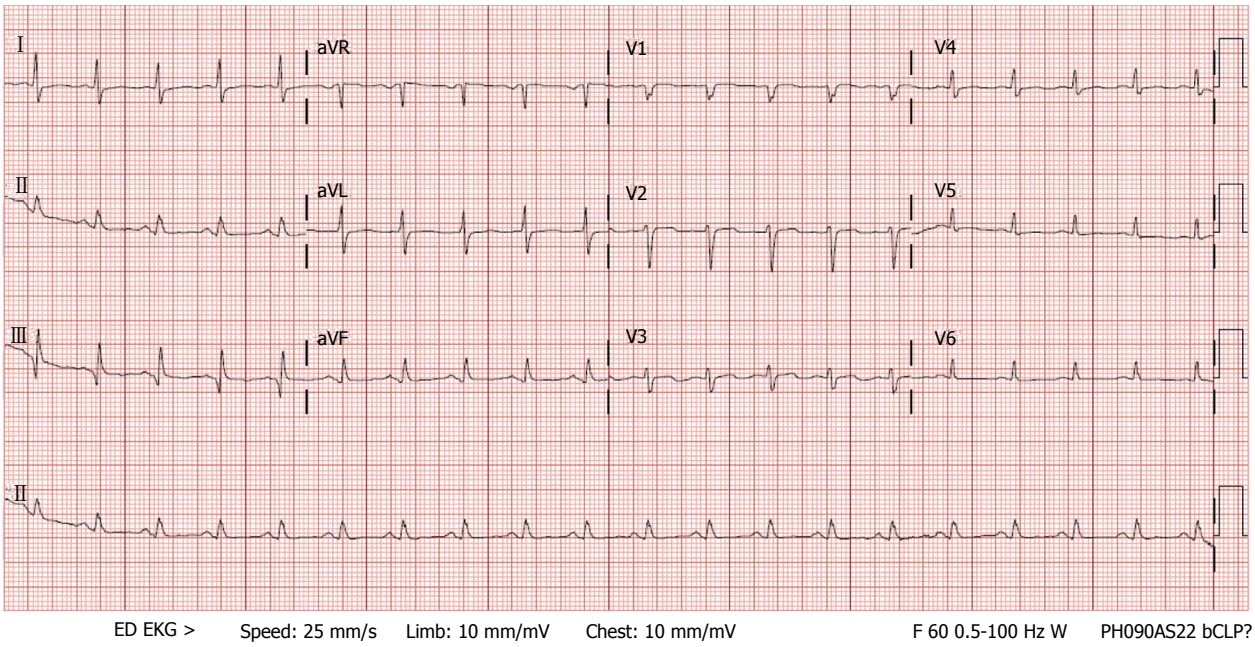

Figure 1 Electrocardiogram shows sinus tachycardia with S1Q3T3, a sign of acute cor pulmonale.

CASE REPORT

A 47-year-old African American male with obstructive sleep apnea, noncompliant with continuous positive airway pressure and stage Ⅱ chronic kidney disease developed what he thought was the "flu" associated with shortness of breath and chest pressure for the past 6 d. On the morning prior to his hospital admission, the symptoms persisted and were accompanied by rapid palpitations and syncope. Physical examination revealed a morbidly obese man in respiratory distress with a heart rate of 122 beats/min, blood pressure of 120/87 mmHg, respiratory rate of 33 breaths/min, and room air oxygen saturation of 88%. Patient had no jugular venous distention or lower extremity edema, lungs clear to auscultation, no gallops, murmurs or rubs. The electrocardiogram showed sinus tachycardia with S1Q3T3 consistent with acute cor pulmonale (Figure 1). Ventilation perfusion scan showed multiple perfusion defects highly suspicious for multiple pulmonary emboli. Echocardiogram revealed marked biventricular alternans with elevated pulmonary systolic pressure of 50 mmHg. The right ventricle was visually larger than the left ventricle in the four chamber view. In diastole, the right ventricle basal diameter measured approximately 4.8 cm. TAPSE was 1.2 cm. Left ventricular ejection fraction (LVEF) was 60% visually. By Modified Simpson's method, an LVEF of 70% was calculated. In the parasternal long axis view, the interventricular septal diameter was 1.4 cm, the left ventricular posterior wall diameter was 1.5 cm, the left ventricular end diastolic diameter was 4.6 cm, and the left ventricular mass index was increased at 103 g/m^2 (Figure 2). There was concentric remodeling by relative wall thickness calculation. Pulmonary embolism was diagnosed, and the patient was treated with intravenous heparinization, followed by 6 mo of coumarin therapy.

DISCUSSION

Pulsus alternans is usually found in severe left ventricular systolic dysfunctions due to cardiomyopathy[3], coronary artery disease[2], systemic hypertension[2], and aortic stenosis[4]. It has also been reported in acute transient ischemia and in patient with normal heart during or after supraventricular tachycardia[5] (Table 1). Right ventricular alternans is usually associated pulsus alternans on the left side[3]. Isolated right ventricular alternans seems to be related to right ventricular dysfunctions and increased pulmonary resistance due to reactive air way disease[2], pulmonary embolism[6], and pulmonary hypertension[7]. Biventricular alternans is rare, and only few cases have been described in patient with severe left ventricular dysfunction and left anterior descending coronary artery disease[1,3]. Cournand *et al*[8] reported biventricular alternans in 9 patients, most of whom had dilated cardiomyopathy, systemic hypertension, congestive heart failure and myocardial fibrosis. In comparison, the patient we described has marked biventricular alternans secondary to pulmonary embolism without left ventricular dysfunction. Massive pulmonary embolisms can cause acute cor pulmonale and can lead to right ventricular alternans; however, it has not known to cause biventricular alternans.

There has been continuing interest in understanding the mechanisms and clinical manifestations of pulsus alternans since its first description by Traube in 1872, although its cause and exact mechanism are poorly understood. Two main mechanisms have been proposed to account for pulsus alternans. The first, based on the Frank-

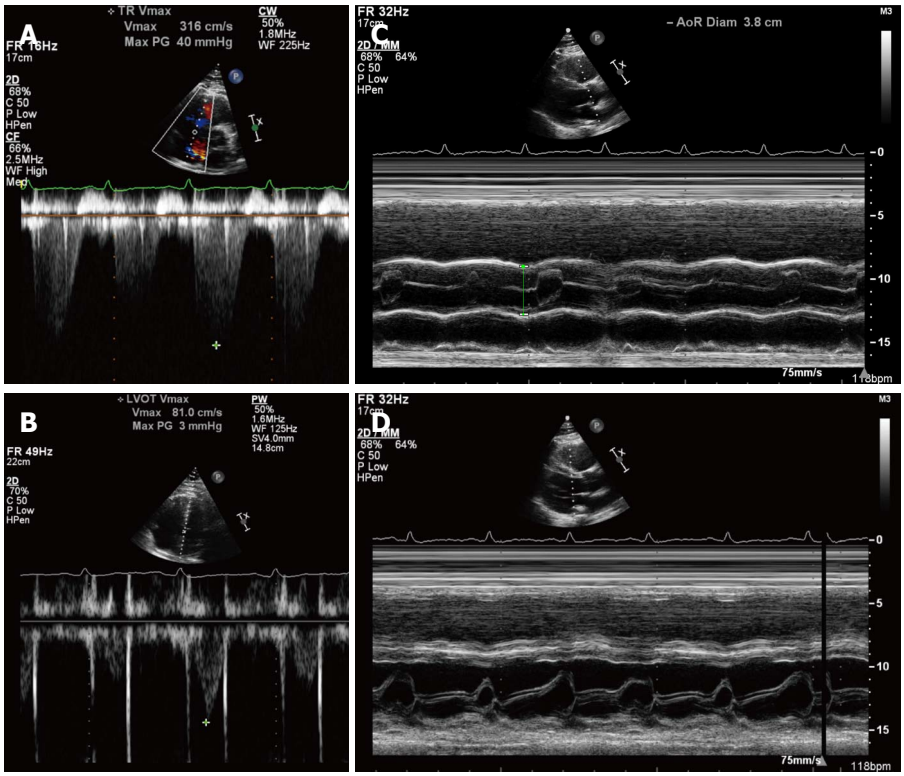

Figure 2 M-mode tracing. A: Beat to beat marked variation of continuous wave Doppler blood flow velocities of tricuspid regurgitation indicative of right ventricular pulsus alternans; B: Beat to beat marked variations of continuous wave Doppler blood flow velocities of left ventricular outflow tract indicative of left ventricular pulsus alternans; C: M-mode tracing showing beat to beat marked variation of aortic valve opening indicative of left ventricular pulsus alternans; D: M-mode tracing showing beat to beat marked variation of mitral valve opening indicative of left ventricular pulsus alternans.

Table 1 Causes of left ventricular alternans

Left ventricular dysfunctions
Systolic and diastolic dysfunctions[6,13]
Coronary artery disease[2]
Hypertrophic cardiomyopathy[3]
Outflow track resistance
Systemic hypertension[2]
Aortic stenosis[4]
Mitral stenosis[5]
Prosthetic valve dysfunction[14]
Normal heart
Transient ischemia[5]
Marked acceleration of rate[9]-dobutamine infusion[15]
Hypocalcemia, hypothermia, acidosis[11,12]

Starling mechanism, proposes beat-to-beat alteration in end-diastolic volume accounted for the change in force of contraction[1,9]. Impaired systolic contraction of a failing ventricle reduces stroke volume, resulting in an elevated end diastolic volume for the next contraction. Elevated end diastolic volume results in increased myofibril length and therefore, increased contraction on the next beat[1,9]. An increase heart rate accentuates this process as diastolic filling is further impaired. Although this mechanism may be contributory, the popularity of this mechanism has waned over the recent years as there is experimental evidence which suggests the absence of alternation in end diastolic volume with the stronger beats of pulsus alternans[10]. Also in animal studies, pulsus alternans has been produced and maintained with constant preload and lack of alternation in mitral inflow[5]. The alternation in the contractile state of the myocardium, the "myocardial theory," underlies some instances of pulsus alternans but in no way negates the possibility that Frank-Starling principle accounting for some instances of pulsus alternans[10].

The second proposed mechanism which explains the cause of pulsus alternans is the alteration in cellular handling of calcium during the cardiac contractility[11,12]. The physiological action-involves the abnormality of intracellular calcium cycling, involving the sarcoplasmic reticulum [11]. Calsequestrin is a protein in the inner membrane surface of the sarcoplasmic reticulum that binds and stores calcium. Schmidt *et al*[11] found that mice that overexpressed calsequestrin had pulsus alternans during high heart rates. The study concluded that the mice had a delay between the uptake and release of calcium from the sarcoplasmic reticulum and this alteration in calcium cycling and use led to development of pulsus alternans *in vivo*.

In conclusion, isolated left and right ventricular alternans usually occur in the failing hearts. Biventricular pulsus alternans is a rare phenomenon and has only been described in few cases of severe left ventricle systolic dysfunction and left anterior descending coronary artery disease. To our knowledge, this is the first reported case of pulmonary embolism with echocardiographic finding of biventricular pulsus alternans. In most cases, pulsus alternans is an ominous sign that suggests severe heart failure; early recognition can aid in appropriate management and intervention, which may change patient outcome.

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