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Case Report

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Pulsus Paradoxus from Anterior Mediastinal Mass

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Abstract

Spontaneous breathing comprises a variation of intrathoracic pressure transmitting to heart and great vessels. Under normal circumstance, arterial blood pressure falls with inspiration and rises with expiration, fluctuates within 10 mmHg range. Exaggeration of normal systolic blood pressure decrease during inspiration is defined as pulsus paradoxus [1]. Classical prototype scenarios are cardiac tamponade and acute asthma. Other causes of pulsus paradoxus include pulmonary embolism, constrictive pericarditis and pulmonary disease with large variations in intrathoracic pressure [2]. Few other conditions have been reported such as superior vena cava obstruction, thoracic outlet syndrome, etc. [3]. This report demonstrates a case of a young female patient who was sent for echocardiography due to dyspnea. There is minimal pericardial effusion seen; however, we found an exaggerated inflow variation without evidence of pericardial disease on echocardiography. On examination, pulsus paradoxus was positive by sphygmomanometer as well as with palpation. There is no evidence of pulmonary embolism or lung disease from computer tomography. We presented a case of pulsus paradoxus caused by an anterior mediastinal mass, and also proposed mechanisms behind the findings.

Keywords: *pulsus paradoxus, doppler inflow variation, anterior mediastinal mass, lymphoma.*

Case Presentation

A 25-year-old Thai female without underlying disease presented with one day of sudden dyspnea. Four weeks earlier she developed pleuritis chest pain with progressive dyspnea. She noticed a low graded fever in the evening once or twice a week. During the last month, her appetite lost along with her weights (four kilograms in one months). She denied other chest symptoms.

On examination, she had tachycardia of regular rhythm at rate of 120 b.p.m. and tachypnea at rate of 24 breaths/min and normal blood pressure (131/74 mmHg). Her SpO2 was 97% at room air. Her neck veins were engorged. Positive palpable pulsus paradoxus was suspected and confirmed by sphygmomanometer measurement. There was neither facial edema nor flushing. Cardiac auscultation showed decreased heart sound at parasternal border when compared with the apical region. Decreased breath sound of the left lung with dullness on percussion and decreased vocal resonance. Abdominal examination was normal. No pitting edema was found at both lower extremities. No lymphadenopathy was noted.

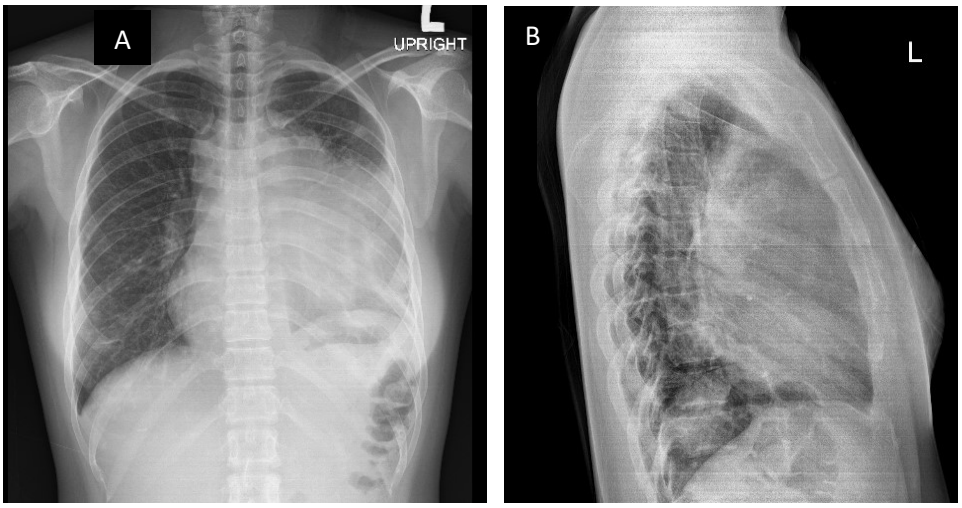

Figure1: Chest X-ray upright view(A) and lateral view (B) showing haziness in the left lung field obliterating anterior mediastinal cavity.

Chest X-ray upright view revealed haziness at left lung zone, silhouette with left heart border. In lateral view, there is haziness at anterior mediastinal cavity (figure 1A, 1B). Anterior mediastinal mass was suspected. Her electrocardiogram (EKG) was sinus tachycardia (Figure 2). Her hematocrit was 41.6%, platelet was 487,000, white blood cell was 8330 (Lymphocyte 7.5%). Her lactate dehydrogenase (LDH) was 3836 U/L (100–250U/L). The remaining laboratory results were within normal range with AFP <0.908, Beta-hCG <0.1.

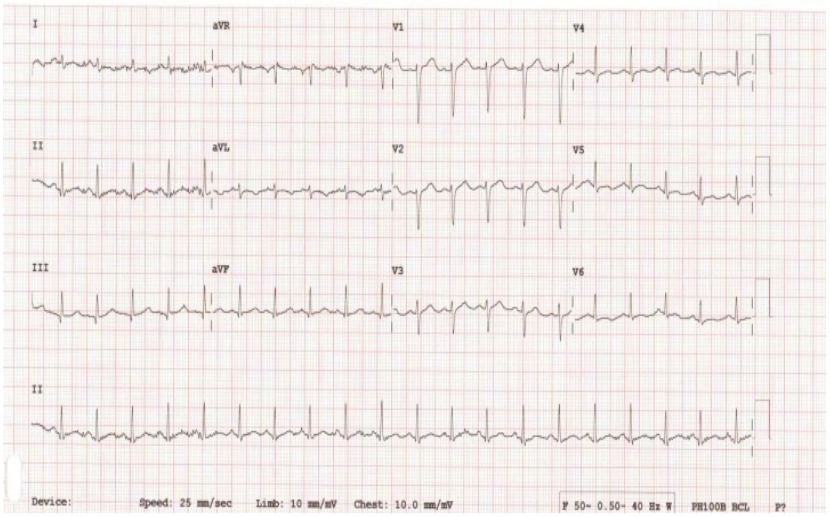

Figure 2: ECG showed sinus tachycardia without low voltage nor electrical alternans.

Echocardiogram showed a huge inhomogeneous mass compressing proximal to distal right ventricular outflow tract (RVOT) and extending to main pulmonary artery. In four chamber views, the mass compresses LV cavity form lateral causing small LV chamber. Short axis view demonstrates interventricular septum paradoxically shifts to left ventricle (Figure 3). Mitral and tricuspid doppler inflow velocity demonstrate exaggerated inflow variation (37%, 83% respectively) (Figure 4A, 4B). CW doppler of proximal descending aorta suggests respirophasic variation of peak velocity (Figure 5) which consistent with palpable pulsus paradoxus from physical examination in this patient. Computer tomography found 12x10x15.5cms inhomogeneous anterior mediastinal soft tissue mass encase the great vessels causing impending SVC obstruction with multiple mediastinal lymphadenopathies. No pulmonary embolism. Several irregular shape nodules scattering in both lungs. Various hypodensity lesions scattering in liver, spleen and left kidney. Minimal pericardial effusion with normal cardiac size. (Figure 5, Video5). Ultrasound guided core-needle biopsy was done. The immunohistochemical staining yielded diffuse strongly positive CD-45. Malignant lymphoma was diagnosed. Chemotherapy was planned. Unfortunately, the patient did not make it through the chemotherapy appointment in the following week as planned. She passes away shortly after emergency visit at nearby hospital due to sepsis and kidney failure.

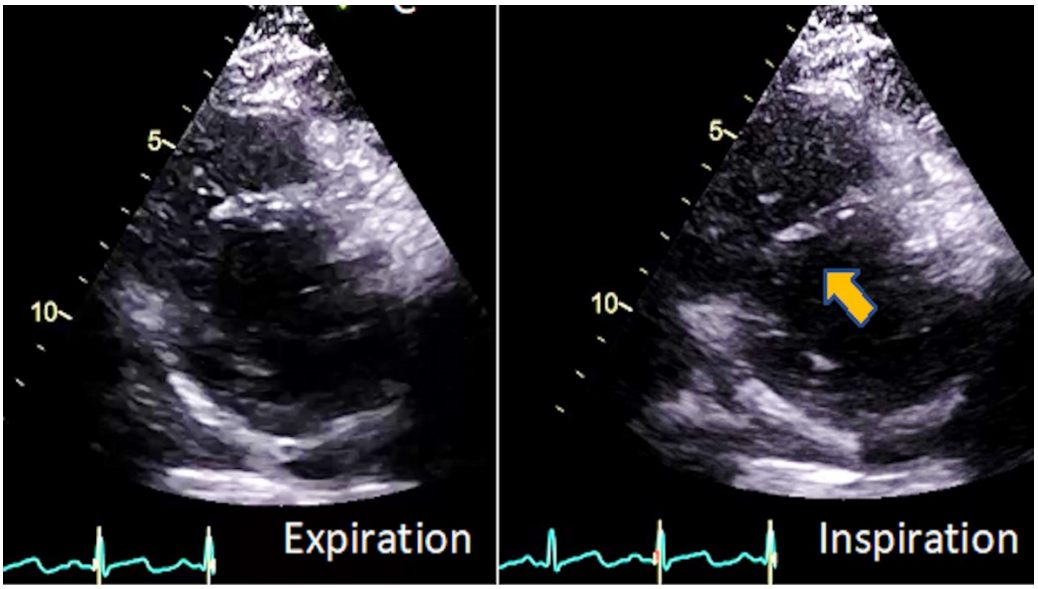

Figure 3: Short axis view demonstrates interventricular septum paradoxically shifts to left ventricle during inspiration (yellow arrow).

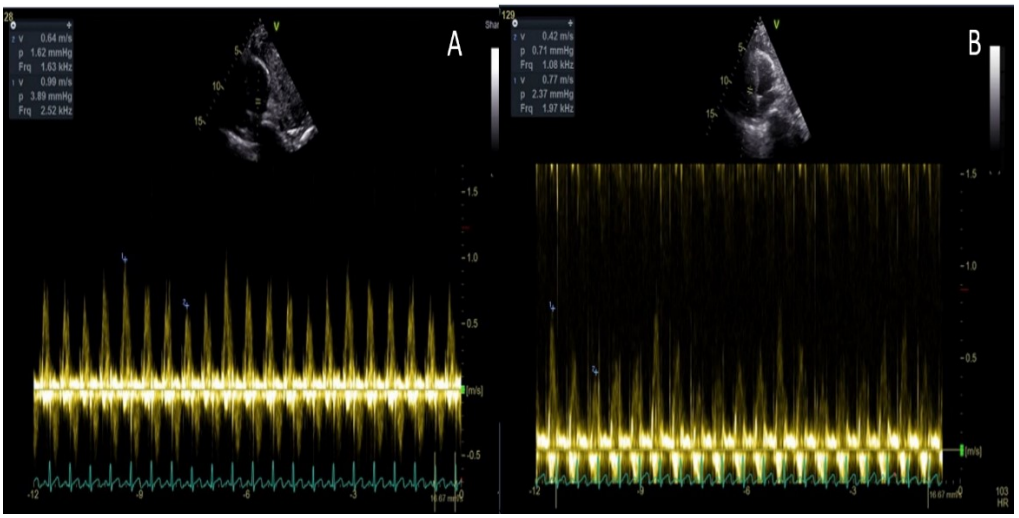

Figure 4: Doppler inflow velocity of mitral (A) and tricuspid (B) demonstrate exaggerated inflow variation of 37%, 83% respectively.

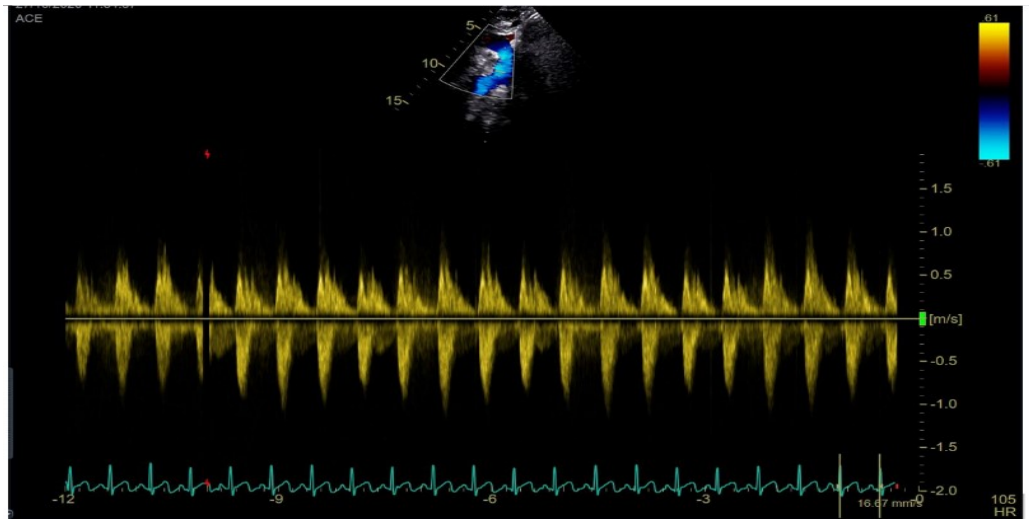

Figure 5: CW doppler of proximal descending aorta suggest respirophasic variation of peak velocity.

Discussion

In 1873, Adolf Kussmaul first described a phenomenon which a pulse simultaneously slight and irregular, disappearing during inspiration and returning upon expiration as pulsus paradoxus [1]. Pulsus paradoxus is defined as a fall in systolic pressure more than 10 mmHg with inspiration and may be palpable if the difference exceeds 15-20 mmHg [4]. Proposed mechanism of pulsus paradoxus comprised of [5].

1. Impaired pressure transmission: negative intrathoracic pressure of the intrapericardial structures is lesser compared with the extra-pericardial veins
2. Pulmonary venous pooling: pulmonary venous compliance increases during inspiration causing blood pooling in the pulmonary vasculature, leading to decreased left ventricular filling
3. Ventricular diastolic interdependence: Impaired filling of the left ventricle due to inspiratory filling of the right heart in a constricted pericardial space
4. Ventricular systolic interdependence: ventricular septal flattening causes impaired left ventricular systolic function
5. Afterload theory: increased impedance to left ventricular ejection from negative pleural pressure
6. Pericardial tug: constraint of cardiac filling due to inspiratory deformation of the pericardium
7. Systemic venous return variation: increased respiratory variability in systemic venous return in cardiac tamponade.
8. Pulmonary afterload: hyperinflation of the lungs in obstructive airway disease impede right ventricular ejection causing decreased filling of the left ventricle.

In our patient the pathophysiology of pulsus paradoxus cannot be only explained by mild pericardial effusion and there is no evidence of pulmonary embolism from computerized tomography image. Mediastinal mass is uncommon to cause hemodynamic of pulsus paradoxus. Although, there is a report of a hepatic mass causing tamponade physiology [6]. We propose mechanism behind the findings in our case as (Figure 2).

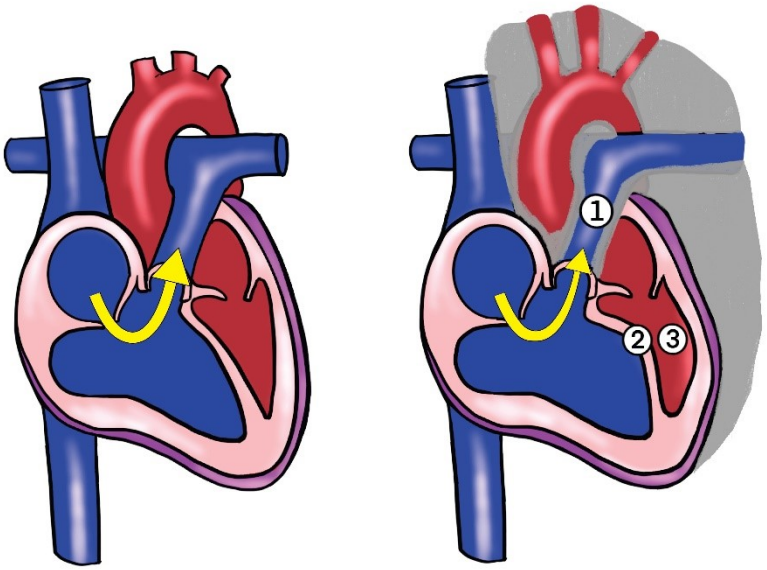

Figure 2: Diagram shows proposed mechanism of pulsus paradoxus in our case.

1. Increase of afterload of the right ventricle due to mass compress
2. Impaired in left ventricular filling due to ventricular diastolic interdependence and decrease intrathoracic pressure transmission to pericardial space leading to limit LV diastolic expansion
3. Impaired left ventricular systolic function and stroke volume due to ventricular systolic interdependence

Apart from being using for diagnosis of pericardial tamponade, higher pulsus paradoxus magnitude has also shown to correlate with decreased stroke volume and cardiac output. Thus, pulsus paradoxus may be used as a clinical prognosis predictor when follow patient with pulsus paradoxus. Pulsus paradoxus could be interpreted as impending hemodynamic compromise suggesting urgency management. In our case, we regret not to admit her immediately, instead we asked her to come back for chemotherapy in a week.

Conclusion

This report demonstrates a pulsus paradoxus sign in case without massive pericardial effusion. We proposed the possible mechanisms how anterior mediastinal mass can cause pulsus paradoxus. Recognizing and using pulsus paradoxus as an indicator for predicting the urgency of treatment could be useful.

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