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A case report of breathlessness on exertion with an asymptomatic honeymoon period

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Background

Chronic thromboembolic pulmonary hypertension (CTEPH) results from fibrous organization of incomplete pulmonary emboli resolution which leads to increased pulmonary vascular resistance and promotes adverse pulmonary vascular remodeling. CTEPH develops after acute or recurrent pulmonary emboli, but a majority of CTEPH cases may originate from asymptomatic chronic thromboembolic disease (CTED). Here, we report the 6-year clinical journey of a patient from acute pulmonary thromboembolism to chronic thromboembolic pulmonary hypertension. Attentively, during the clinical course, there is an asymptomatic period from CTED to CTEPH. We draw a conclusion a thorough clinical work-up is required to increase clinicians awareness of finding the asymptomatic honeymoon period and guide patients through either surgical and/or medical treatment.

Case report

A woman aged 53 years was admitted to our hospital in April 2010 due to a shortness of breath on exertion during the past 10 days, as well as the mildly impaired exercise tolerance. Her physical examination revealed that her pulse rate was 96 beats per minute, the respiratory rate was 20 breaths per minute, and the blood pressure was 100/60 mmHg. On addition, her jugular veins were not distended. Her breath sounds were symmetry and clear. Her heart sound was regular at 96 beats per minute without accentuated P_2. Her bilateral legs were symmetry and no swelling. Her arterial blood gases analysis showed a pH 7.516, partial pressure of oxygen (P_aO_2) 52.7 mmHg, partial pressure of carbon dioxide (P_aCO_2) 26.50 mmHg, suggesting hypoxemia and hypocapnia. Blood tests evidenced elevated level of serum D-dimer at 993 μg/L (normal 0–300 μg/L), cardiac tronopin I (Tpn-I) at 0.30 μg/L (normal <0.14 μg/L) and the brain natriuretic peptide (BNP) at 1859 pg/ml (normal 0–125 pg/ml). An electrocardiogram (ECG) showed normal sinus rhythm, inversion of the T waves at leads V_2-V_3 and a bi-directional T wave at lead V_4 (Figure 1A). Initial echocardiography (Echo) showed the internal diameter of right ventricle was 20 mm, the diameter of outflow tract of right ventricle was 30 mm, the internal diameter of the main pulmonary artery was 18 mm, and the speed of tricuspid regurgitation was 2.7 m/s. Computed tomography pulmonary angiography (CTPA) and computed tomography venography (CTV) indicated bilateral filling defects in branches of pulmonary arteries, mosaic attenuation on both lungs and filling defects in local lumen of right popliteal vein (Figure 2A). Diagnosis of pulmonary thromboembolism (PTE) and right leg deep (popliteal) venous thrombosis (DVT) was established by above examinations. Risk stratification of this patient should belong to intermediate-high risk PTE, previously sub-massive PTE. Combined with her clinical manifestations, she qualified for anticoagulant indications without

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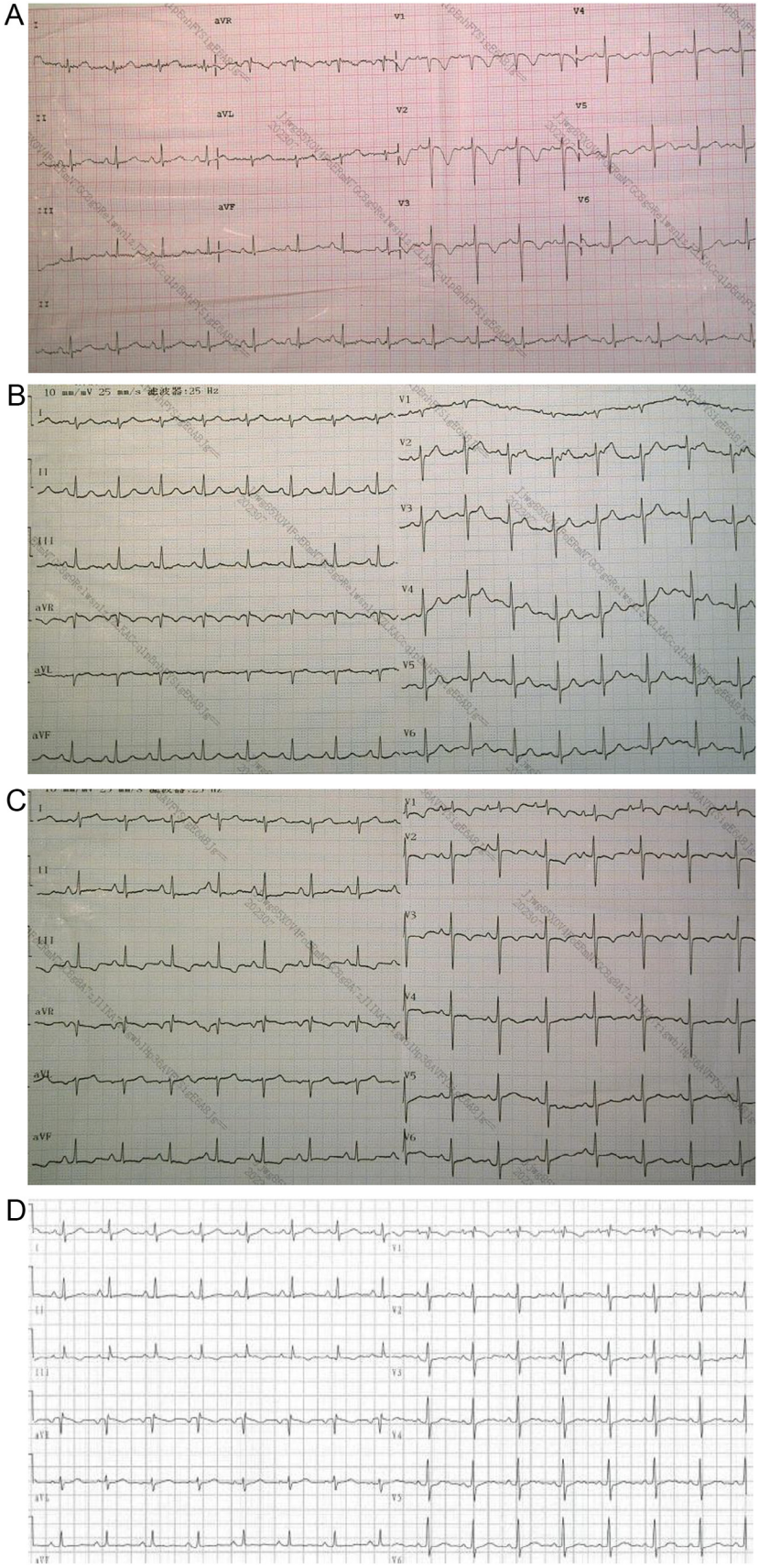

Fig. 1. (A) Electrocardiogram at first clinical presentation showed normal sinus rhythm, inversion of the T waves at leads V_2-V_3 and a bi-directional T wave at lead V_4. (B) Electrocardiogram in November 2011 showed normal. (C) Electrocardiogram when the patient felt breathless again after long-distance travel in March 2013 showed sinus rhythm, inversion of the T waves at leads III, V_2-V_3 and a bi-directional T wave at lead V_4. (D) Electrocardiogram reviewed in March 2015 showed sinus rhythm and low level of the T waves at leads V_2-V_6.