INTRODUCTION

More than a decade after tuberculosis (TB) was identified as a global health emergency; it still remains one of the world's leading infectious causes of death among adults[1]. Incidence of TB has been on the rise since the 1980s, with its spread concentrated in Southeast Asia, sub-Saharan Africa and the Middle East[1]. TB-related sudden death (TBRSD) is a rare, as it has been described in the literature by several authors. Surprisingly, even though TB is an endemic in the Middle East, there is only 1 report of TBRSD from this region[2]. This report presents a rare case of TBRSD in a 28-year-old Saudi Arabian woman who was admitted to our hospital for active pulmonary tuberculosis without any other comorbidities.

CASE REPORT

A 28-year-old woman was admitted through the outpatient clinic of our hospital complaining of cough and fever that had persisted for 3 months. Her cough was productive of a moderate amount of yellow sputum with no haemoptysis. The patient had a history of shortness of breath on exertion, loss of appetite, loss of weight by 10 kg, and a generalized fatigability with decreased activity. There was no history of chest pain or palpitation, and her past medical and surgical history were negative. She was on no medications and had no history of any allergies. Her father died of pulmonary-TB 2 years previously. She was single, a non-smoker and lived in Jeddah. A systemic review yielded unremarkable results. On her physical examination, she appeared ill, pale and cachectic. Her pain score was 0/10. She was febrile with temperature of 38.7°C and had tachycardia (heart rate: 113 bpm); her blood pressure was 102/63; respiratory rate, 22 breaths/min; and oxygen saturation at room air, > 92%. Chest examination showed an equal air entry and coarse crackles in both lungs, although, the latter were mainly noted in the left side. Cardiovascular, abdominal and neurological examinations revealed no significant abnormalities. The working diagnosis was pulmonary tuberculosis. Blood examination revealed a white blood count (WBC) of 24.4; haemoglobin (HGB) level, 8.6; platelet count (PLT), 769; erythrocyte sedimentation rate (ESR), 91 mm/h; and C-reactive protein (CRP) level, 192.2. The results of her liver function tests were normal, apart from an alkaline phosphatase (ALKP) level of 193 and a gamma-glutamyl transferase (GGT) level of 51. The test results for the levels of urea, creatinine and electrolytes; coagulation profile; and thyroid function were all within the normal limits. A chest X-ray scan showed a diffuse architectural distortion on both lungs plus multiple cystic and bronchiectatic changes; more obvious in the left lung, with compensatory hyperinflation of the right lung. No pleural effusion or pneumothorax was seen.

Anti-TB medication was started empirically, and the patient was admitted to the general ward isolation as a case of suspected pulmonary tuberculosis. Subsequently, a sputum smear tested positive for acid-fast bacilli, which were confirmed to be Mycobacterium tuberculosis (MTB) by polymerase chain reaction (PCR). Even though the patient was administered the complete anti-TB management protocol (600 mg rifampicin, 300 mg isoniazid, 2 g pyrazinamide and 800 mg ethambutol daily), her fever continued to spike. Since all septic screens remained negative, moxifloxacin was empirically added to treat any other bacterial infection, and the possibility of drug resistance was considered. A high-resolution computed tomography (HRCT) scan of the chest showed multiple cavities, and bronchiectatic changes mainly in the left lung associated with consolidation mainly in the left upper and left lower lobes; the right lung was relatively spared. There were no signs of aspergilloma. The patient's fever continued to spike, and in view of the extensive bronchiectasis observed in the HRCT scan, moxifloxacin was replaced with ciprofloxacin to treat any possible Pseudomonas infection. Seven days after admission, the patient collapsed suddenly and was unresponsive. A cardiac arrest call was announced, and an electrocardiogram (ECG) showed asystole. The patient was intubated, and 35 min of cardiopulmonary resuscitation (CPR), doctors failed to revive the patient. However, during CPR, the endotracheal tube and the patient's large airways were found to be filled with blood. The cause of death was therefore suspected to be massive haemoptysis. The subsequently obtained results of sputum culture were positive for Mycobacterium Tuberculosis (MTB); sensitive to all anti-TB medications. A post-mortem examination could not be conducted for cultural and social reasons.

DISCUSSION

TBRSD is a rare outcome of tuberculosis, and it has been reported in the literature mostly among alcoholics as they are more likely to seek medical attention[3]. Surprisingly, it has been found to occur especially after the initiation of anti-TB treatment[3]. In a 7-year review of the hospital case-notes and autopsy reports of 60 patients who were certified as having died of tuberculosis, a group of 16 (27%) patients who died suddenly and unexpectedly were identified. Furthermore, the cause of death in these patients was obscure. In 14 of these 16 patients, death occurred following the initiation of anti-TB chemotherapy[3], as in our patient. Although TBRSD has been described in the literature by several authors, there are no reports from the Middle East about this complication, apart from 1 paper from Baghdad published in 1984[2]. To our knowledge, this is the first report describing TBRSD from Saudi Arabia and the second in the Middle East.

The possible causes of TBRSD include tuberculosis bronchopneumonia, massive haemoptysis, tuberculosis myocarditis, and adrenal insufficiency secondary to tuberculosis adrenalitis[4]. In 2001, Alkhuja and Miller reviewed reports on TBRSD published in MEDLINE (1966 to October 2000)[4] in which forty-six cases of TBRSD were reported. The most common cause of TBRSD was tuberculous bronchopneumonia in 30 (64%) patients, followed by haemoptysis in 14 (30%) patients. Tuberculosis myocarditis and TB of the adrenal glands were found to be

rare causes of TBRSD; accounting for 4% and 2% of the cases, respectively[5-7].

Massive haemoptysis was believed to be the cause of death in our patient. It occurs in pulmonary tuberculosis due to the ruptured arterial aneurysms called Rasmussen's aneurysms. These are localized, dilated, small-to-medium sized arteries continuous with the tuberculous cavities that often result in a fatal massive haemoptysis. Haemoptysis can be detected by helical CT angiography and can be successfully treated by steel coil embolization[8].

More recently, Krishnan *et al.* reported a case of TBRSD due to dehiscence of the aortic wall caused by necrotising tubercular lymphadenitis[9]. Whereas, Bhagavath and co-workers reported a case of fatal hemoptysis due to tuberculous aspergilloma[10]. Other causes of TBRSD were considered in the differential diagnosis in our case, including tuberculous pneumonia. However, the presence of a large amount of blood in the endotracheal tube observed during CPR, suggested that the cause of death was asphyxia secondary to massive haemoptysis. Acute adrenal crisis may occur especially after anti-TB medication was initiated, as rifampicin is an enzyme inducer and may unmask sub-clinical adrenal insufficiency[11]. Though, adrenal insufficiency as a cause of TBRSD has not been reported to be directly related to rifampicin therapy. If the levels of urea and electrolytes are normal, and gastrointestinal symptoms are absent, adrenal crisis is unlikely to be the cause of death, as in our patient. Moxifloxacin has been shown to block potassium channels in the heart; a mechanism that has been proven to prolong the QT interval, which increases the incidence of torsades de pointes, especially in women and in heart failure patients[9]. However, this was unlikely to have been the cause of death in our case, as the patient had no cardiovascular risk factors and her potassium levels were normal.

Another possible cause of TBRSD is tuberculous myocarditis with granulomatous proliferation in the ventricular septum, leading to ventricular arrhythmia[5,6]. Tuberculous myocarditis is a very rare complication and is usually diagnosed only after death. Acid-fast bacilli are rarely present and detection depends on PCR. This condition affects young patients, usually men, who are generally asymptomatic and lead normal lives[5,6]. In contrast, the woman whose case was described in this report was symptomatic and ill, with extensive pulmonary involvement and sputum positive for acid-fast bacilli.

Our case highlights a rare but important complication of tuberculosis, and despite the development of specific chemotherapeutic agents for TB, TBRSD may still affect patients receiving treatments.

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CLINICAL IMAGES IN MEDICINE: A BREATHLESS AND HYPOTENSIVE PATIENT

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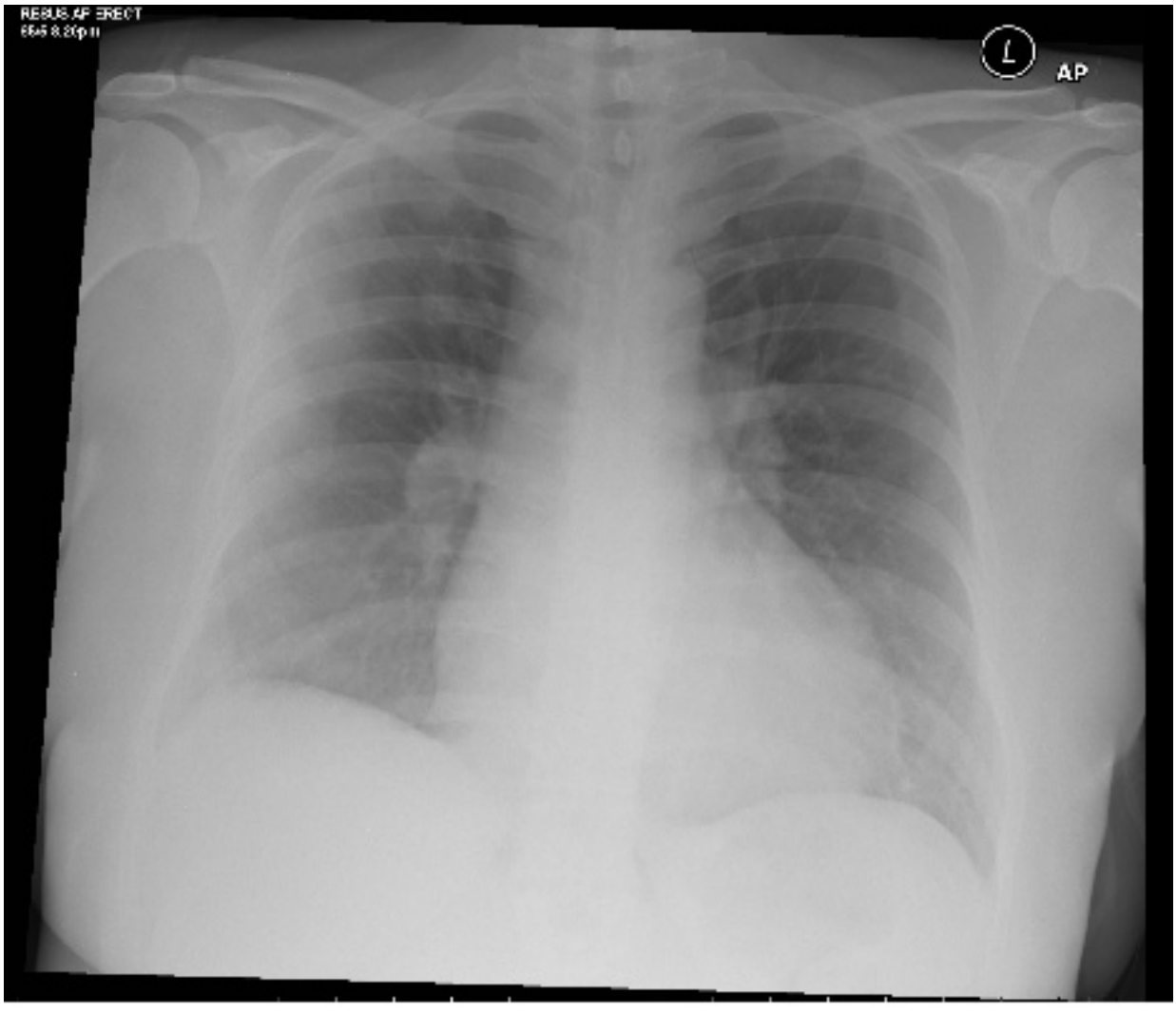

Figure 1. Plain chest X-ray of the patient on admission.

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CASE PRESENTATION

A forty-two-year-old lady was admitted through emergency department complaining of shortness of breath with central chest pain of sudden onset. The chest pain was mainly in the right side, sharp in nature, and aggravated by inspiration and coughing. The patient had a low grade fever and a mild dry cough. There was no significant past medical history, a part from thyrotoxicosis four years ago, treated medically. She is a non-smoker and worked in an office as a clerk. No recent history of long travel or surgery. Clinical examination demonstrated low blood pressure of 96/50 mmHg, pulse 110 per minute, respiratory rate of 24 per minute and oxygen saturation of 86% at room air. The rest of the clinical examination was unremarkable. Blood gases showed pH of 7.53, PCO_2 of 3.9 kPa, PO_2 of 6.8 kPa and bicarbonate of 24 mm mol/L. Her D-dimer was greater than 10,000 units. However, her echocardiogram showed a dilated right ventricle with a moderate tricuspid regurgitation and pulmonary systolic pressure of 60 mm Hg. Her electrocardiogram showed no abnormality apart from sinus tachycardia and non-specific T-wave changes. In addition, her chest X-ray (Fig. 1) and computed tomography pulmonary angiography (CTPA) are shown in Figure 2.

QUESTIONS

- Describe two abnormalities on the chest X-ray (Fig. 1)
- Describe seven abnormalities seen on the CTPA (Fig. 2)

ANSWERS AND DISCUSSION

This patient presented of shortness of breath, chest pain, hypoxemia and hypotension suggesting an acute massive pulmonary embolism. Her blood gases revealed non compensated respiratory alkalosis with hypoxemia and hypocapnia, signifying respiratory failure type 1. Although there was no significant history of a risk factor for PE, alternative diagnosis to explain the clinical presentation was lacking. Therefore, the clinical probability for PE was intermediate, according to the British Thoracic Society scoring system[1-3]. The chest X-ray showed bilateral large prominent pulmonary arteries suggestive of a pulmonary hypertension (Fig. 1A). In addition, the right pulmonary artery appeared amputated (Fleischner's sign) with a peripheral oligemia (Westermark's sign) in the right infra-hilar region (Fig. 1A) [4]. Computed tomography pulmonary angiography (CTPA)

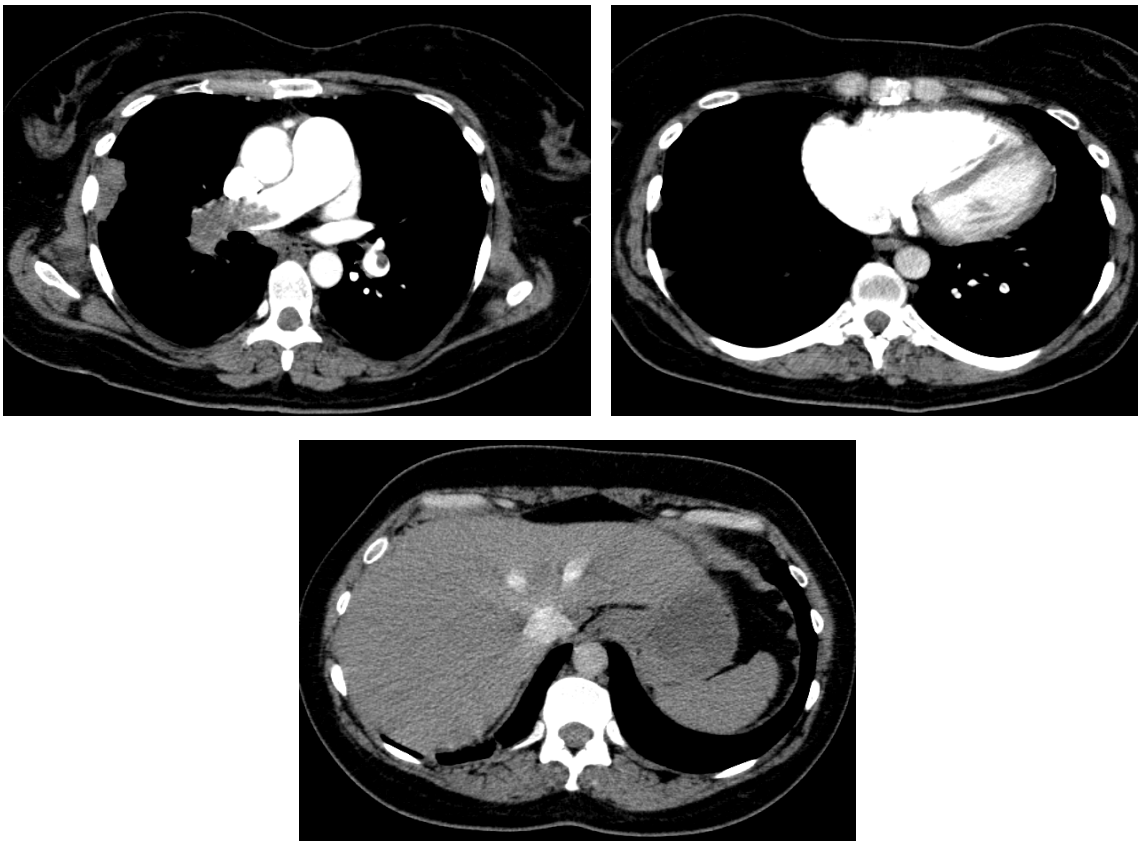

Figure 2. Computed tomography pulmonary angiography (CTPA) of the patient on admission

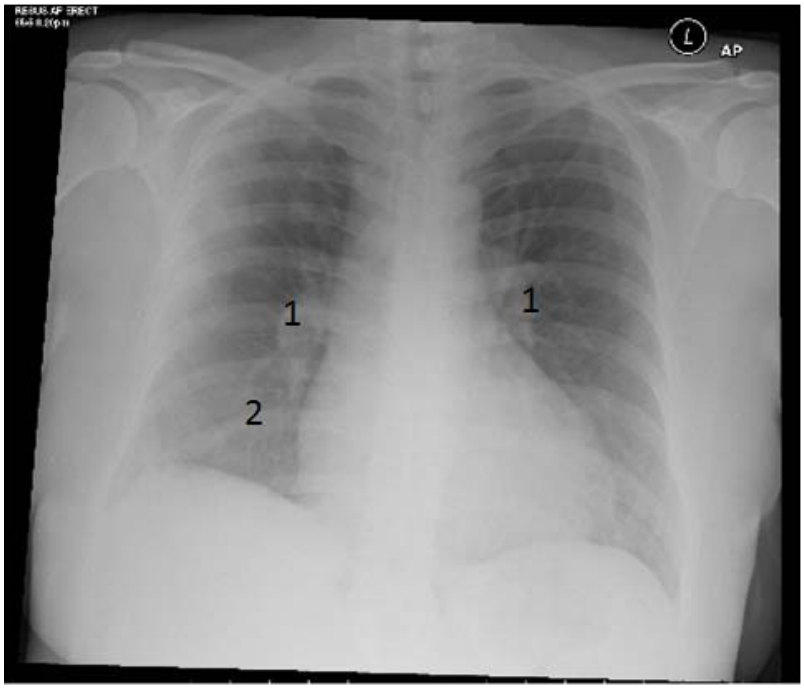

Figure 1A. The chest X-ray of the patient on admission showing bilateral large prominent pulmonary arteries (1), indicating pulmonary hypertension. In addition, the right pulmonary artery appeared prominent and amputated (Fleischner's sign) with small triangular area of peripheral oligemia (Westermark's sign) in the right side (2).

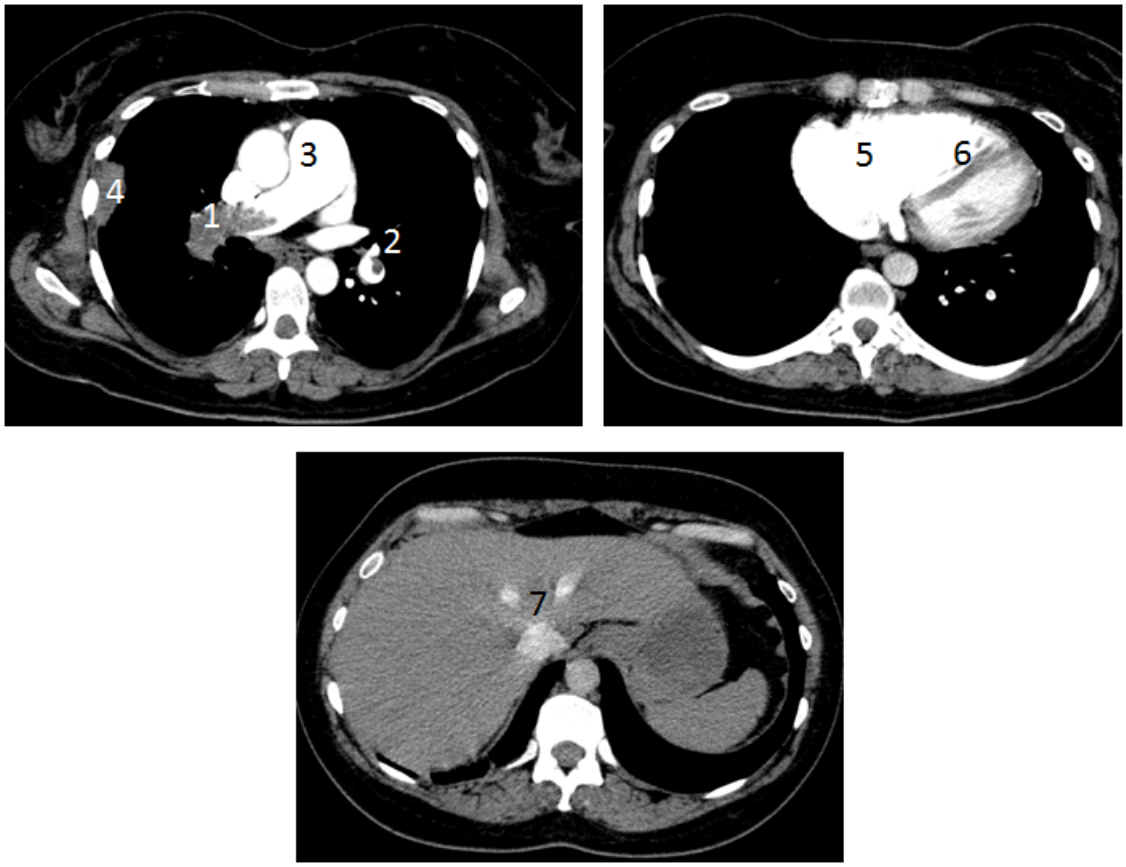

Figure 2A. Computed tomography pulmonary angiography (CTPA) scan of the patient on admission, showing a large pulmonary embolism in the right pulmonary artery (1); an embolus in a left segmental artery (2); a large pulmonary artery trunk diameter greater than the aorta (3); a pleural based pulmonary infarction (Hampton hump) in the right side (4); dilated right ventricle which is larger than the left ventricle size (5); bowing of the inter-ventricular septum to the left (6) and reflux of contrast medium in the inferior vena cava and hepatic veins (7).

on the other hand, showed features diagnostic of massive PE. These features included a large pulmonary embolus in the main right pulmonary artery, a large pulmonary artery trunk with a diameter greater than that of the aorta, a dilated right ventricle which was larger than the left ventricle size, and bowing of the inter-ventricular septum to the left, plus reflux of contrast medium in the inferior vena cava and hepatic veins (Fig. 2A). Furthermore, the CTPA demonstrated a small clot in the left segmental artery, and a pleural based pulmonary infarction (Hampton hump) in the right side (Fig. 2A). As the patient was relatively young and there was no obvious risk factor, screening for thrombophilia was warranted[5].

Although, massive pulmonary embolism account for only 4.5% of cases of pulmonary embolism, identification of this condition and early institution of thrombolytic therapy can be a life saving[6-7]. The use of CTPA scan as screening diagnostic test for PE has many advantages over ventilation/perfusion nuclear (VQ) scan, being quicker to carry out and easier to interpret. It is also less affected by the presence of cardio-pulmonary disease or abnormal chest X-ray[3,8-10]. Furthermore, CTPA can give an alternative diagnosis in many cases and provides a quantitative assessment of PE, which correlates well with the severity of the clinical picture[3,8-10]. Massive pulmonary embolism causes pulmonary hypertension leading to right ventricular overload and dysfunction. Right ventricular failure with consequent decreased right ventricular output, can cause decreased venous return to the left side with an end result of under filling of the left atrium and can impair the left ventricle preload[11,12]. This can also be impaired by the decreased left ventricular compliance as a consequence of a leftward shift of the inter-ventricular septum[11,12]. In this setting, CTPA is especially valuable as it may show the central location and the large size of the clot, as shown in this case. In addition, CTPA can show features indicative of right ventricular overload and dysfunction[11,12]. These features include a right ventricle size larger than the size of left ventricle, bulging of the inter-ventricular septum to the left and a large pulmonary artery diameter that may exceed the aortic diameter; all of which are seen in this patient and shown in Figure 2. Moreover, CTPA may exhibit signs suggestive of tricuspid valve incompetence, as manifested in our case by the reflux of contrast medium in the inferior vena cava and in the hepatic veins[13].

The accuracy of CTPA, compared to echocardiography in detecting right ventricular dysfunction was studied by Lim *et al.* in 14 patients with massive PE. These authors found that CTPA had a sensitivity of 91.6% and a specificity of 100% in detecting right ventricular dysfunction, when using echocardiography as a reference gold standard test[14]. Others studied the sensitivity and specificity of both CTPA and echocardiography in detecting right ventricular dysfunction using 30% pulmonary vascular obstruction as a reference standard test. In this study, however, the sensitivity and specificity were found to be 81% and 47% for CTPA, and 56% and 42% for echocardiography[15]. These studies suggest that CTPA is as good as an echocardiograph, if not better in detecting right ventricular dysfunction in the presence of massive PE.

This case highlighted the value of CTPA as a diagnostic test that can be also used in assessing the severity of PE. Particularly it can be an invaluable investigation in evaluating the pathophysiological effects of massive PE, and can be used to detect the presence of right ventricular dysfunction, as in this setting.

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