Dear Editor,

A 33-year-old male who was undergoing treatment with phosphodiesterase 5 inhibitor and anticoagulant for idiopathic pulmonary arterial hypertension presented to the emergency department with acute onset of dyspnoea and chest pain. His cardiovascular examination had shown features of cardiomegaly and pulmonary arterial hypertension with no pathological murmur. His blood pressure was 138/100 mm Hg with heart rate 86 per minute. Electrocardiography showed changes suggestive of right ventricular hypertrophy with no evidence of ST segment changes. His troponin T markers for infarction were within normal limits. Chest X-ray (not shown) demonstrated cardiomegaly with biventricular enlargement. There was dilated pulmonary trunk with evidence of peripheral pruning. Patient was referred for urgent multi-detector computed tomography (MDCT) pulmonary angiography to rule out pulmonary thromboembolism. Computed tomography (CT) pulmonary angiography was performed using 64 slice machine (GE, VCT XTE); 120 mL of contrast was administered at the rate of 4.5 mL/sec followed by 40 mL of saline at the same rate. Scanning was performed with bolus track technique with region of interest (ROI) in the right ventricle. Sections of thickness 0.625 mm were acquired. Volume rendered and contrast images were then interpreted. CT scan showed that the main and left pulmonary arteries were grossly dilated. There was evidence of intimal flap which originated just cranial to the pulmonary valve (Fig. 1) and extended superiorly up to the bifurcation (Fig. 2). True and false lumens could be differentiated due to lesser enhancement and slow flow as compared to the true lumen (Fig. 3). Volume rendered (VR) images had shown grossly dilated main and left pulmonary arteries (Fig. 4). No evidence of any pulmonary thromboembolism was
seen. The patient was managed conservatively with oxygen inhalation, beta blocker, diuretics and vasodilators. He was advised surgical intervention for which he refused.

Discussion

Pulmonary artery dissection is an extremely rare and fatal disease which is usually diagnosed on post-mortem examination.¹ It usually occurs as a complication of chronic pulmonary arterial hypertension. Other rare causes include chronic inflammation of the pulmonary arteries, right heart endocarditis, amyloidosis, trauma and severe atherosclerosis.² Although rare but in any patient with pulmonary hypertension who presents with cardiogenic shock, collapse or sudden cardiac death, diagnosis of pulmonary artery dissection should be considered as a cause.³ Often, presenting signs include dyspnoea, chest pain and cyanosis. It usually occurs at the site of maximal dilatation of pulmonary artery and had been seen related to degeneration of media on pathological studies. Reports have shown that false lumen tends to rupture rather than developing a re-entry site.⁴ Multiple modalities can be used to diagnose this entity such as echocardiography,⁵ CT,¹ Magnetic resonance imaging (MRI)⁶ and angiography.⁷ Echocardiography is usually the first-line investigation because of its ready accessibility, ease of use, and low cost. However, CT or MRI may be used if echocardiography fails to detect an intimal flap or to provide additional information related to the dissection and associated abnormalities such as pericardial tamponade.⁸ MDCT has shown many advantages over other modalities including fast volume coverage as well as isotropic resolution that allows multiplanar reformation and 3D rendering. It can accurately display the intimal flap, extent of the dissection and intraluminal thrombi. It can also demonstrate haemopericardium secondary to pulmonary artery dissection into the pericardium resulting in acute cardiac tamponade. CT can accurately evaluate the diameter and extent of the aneurysm and can exclude the presence of pulmonary embolism.¹ Isolated reports of medical management of the condition in cases where the patient had refused or was unfit for surgery are present. Although it is usually a post-mortem diagnosis but recent reports have described successful surgical repair of the dissection.⁸

Conclusion

In conclusion, we recommend that contrast-enhanced MDCT examination should be the preferred initial investigation in any patient of pulmonary hypertension to rule out this rare but fatal complication. MDCT can act as a single best modality to rule out other pathologies such as embolism, aneurismal rupture and haemopericardium. New generation CT scanners can perform this task within seconds even in uncooperative patients.

REFERENCES


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