# Row 3812

Visit Number: e56ffe72137b034694ca0c8c577ad2fb5a376dc7399a09d170827a6be984d8c4

Masked\_PatientID: 3810

Order ID: c48c8313c12ebdc3a455322b7704598ade71da05760b08d628619ecc5fb45247

Order Name: CT Pulmonary Angiogram

Result Item Code: CTCHEPE

Performed Date Time: 19/1/2018 16:00

Line Num: 1

Text: HISTORY ASD Eisenmenger. SOB + URTI. TRO PE TECHNIQUE Scans obtained during the pulmonary angiogram phase. Contrast: Omnipaque 350 - Volume (ml): 50 FINDINGS The radiograph of 17 Jan 2018 and CT pulmonary angiogram of 29 Apr 2013 (CGH) were reviewed. There is suboptimal opacification of the pulmonary arteries. Accounting for this, filling-defects are seen in the middle lobe pulmonary artery (se 3-45) and extending to the medial segmental artery, in keeping with acute pulmonary embolism. There is a focal wedge-shaped ground-glass opacity with a rim of consolidation in the medial middle lobe, suspicious for pulmonary infarct. There is a linear filing-defects in the left lower lobe pulmonary artery, possibly extending into the lateral basal segmental branch, suspicious for a web (3-38 and 6-21). An eccentric filling defect is also noted in the right lower lobe pulmonary artery (6-30). These changes are likely due to chronic pulmonary embolism. Known atrial septal defect (ASD) (se 3-50). There is stable dilatation of the pulmonary trunk to 4.3 cm, in keeping with pulmonary arterial hypertension. Cardiomegaly is present, with enlargement of the right sided cardiac chambers. There is also reflux of contrast into the hepatic veins, compatible with right heart strain. Bilateral centrilobular ground-glass opacities are present, less prominent compared to the prior CT of 29/04/2013, and are presumably related to underlying chronic pulmonary arterial hypertension. No suspicious pulmonary mass. There is no pleural effusion. The central airways remain patent. Prominent right hilar nodes measuring 8 mm (se 3-46) are probably reactive. No significant abnormality is seen in the included upper abdomen. There is no aggressive bony lesion. CONCLUSION 1. Acute-on-chronic pulmonary embolism (PE). Middle lobe acute PE with infarct of the middle lobe medial segment. Chronic PE in both lower lobes. 2. Known ASD with pulmonary arterial hypertension and right heart strain. Bilateral centrilobular ground-glass opacities may be attributed to the chronic pulmonary arterial hypertension, with superimposed infection/pneumonitis as a less likely differential consideration. The above pertinent finding (1) were conveyed to Dr Sharon Harvinder by Dr Felicia Teo via telephone on 19/01/2018 at 4.35pm. Further action or early intervention required Reported by: <DOCTOR>

Accession Number: 960ed02a3bf2c19e9245bff843643580be642ae5f1607f8f54821cba6a56fced

Updated Date Time: 19/1/2018 17:48

## Layman Explanation

This radiology report discusses HISTORY ASD Eisenmenger. SOB + URTI. TRO PE TECHNIQUE Scans obtained during the pulmonary angiogram phase. Contrast: Omnipaque 350 - Volume (ml): 50 FINDINGS The radiograph of 17 Jan 2018 and CT pulmonary angiogram of 29 Apr 2013 (CGH) were reviewed. There is suboptimal opacification of the pulmonary arteries. Accounting for this, filling-defects are seen in the middle lobe pulmonary artery (se 3-45) and extending to the medial segmental artery, in keeping with acute pulmonary embolism. There is a focal wedge-shaped ground-glass opacity with a rim of consolidation in the medial middle lobe, suspicious for pulmonary infarct. There is a linear filing-defects in the left lower lobe pulmonary artery, possibly extending into the lateral basal segmental branch, suspicious for a web (3-38 and 6-21). An eccentric filling defect is also noted in the right lower lobe pulmonary artery (6-30). These changes are likely due to chronic pulmonary embolism. Known atrial septal defect (ASD) (se 3-50). There is stable dilatation of the pulmonary trunk to 4.3 cm, in keeping with pulmonary arterial hypertension. Cardiomegaly is present, with enlargement of the right sided cardiac chambers. There is also reflux of contrast into the hepatic veins, compatible with right heart strain. Bilateral centrilobular ground-glass opacities are present, less prominent compared to the prior CT of 29/04/2013, and are presumably related to underlying chronic pulmonary arterial hypertension. No suspicious pulmonary mass. There is no pleural effusion. The central airways remain patent. Prominent right hilar nodes measuring 8 mm (se 3-46) are probably reactive. No significant abnormality is seen in the included upper abdomen. There is no aggressive bony lesion. CONCLUSION 1. Acute-on-chronic pulmonary embolism (PE). Middle lobe acute PE with infarct of the middle lobe medial segment. Chronic PE in both lower lobes. 2. Known ASD with pulmonary arterial hypertension and right heart strain. Bilateral centrilobular ground-glass opacities may be attributed to the chronic pulmonary arterial hypertension, with superimposed infection/pneumonitis as a less likely differential consideration. The above pertinent finding (1) were conveyed to Dr Sharon Harvinder by Dr Felicia Teo via telephone on 19/01/2018 at 4.35pm. Further action or early intervention required Reported by: <DOCTOR>. In simpler terms, this means...

## Summary

No diseases detected.  
No specific organs mentioned.  
No symptoms mentioned.