A 52-year-old woman presented with symptoms of increasing dyspnea on exertion (classified as NYHA class II-III), which did not improve with transfusion, and a pulsatile retrosternal sensation. She had neither cough nor sputum production. She had been diagnosed with hereditary hemorrhagic telangiectasia several years ago and had known multiple arteriovenous malformations in the liver. She also had a moderate insufficiency of the mitral valve. Chest radiography and contrast-enhanced multidetector CT of the thorax were performed. Chest X-ray showed a nodular opacity, paravertebral in the left lower lobe (Fig. 1). Contrast enhanced chest MDCT showed a well-defined nodule (diameter 2.5 cm), posteriorly in the left lower lobe with a hypertrophied feeding artery and a draining vein (Fig. 2, 3); and a second well-defined nodule (diameter 0.6 cm) was found posterior in the left lower lobe with also a feeding artery and a draining vein (Fig. 4). Maximum intensity projection (MIP) images (Fig 5) and volume rendered technique (VRT) images (Fig 6) showed the pulmonary arteriovenous malformation demonstrating the simple angio-architecture of an AVM with a single feeding artery and a single draining vein, both connected to the aneurysmal sac (nidus).

### Discussion:

In our patient, with hereditary haemorrhagic telangiectasia (Osler-Weber-Rendu syndrome), imaging findings led to the diagnosis of two pulmonary arteriovenous malformations in the left lower lobe.

Pulmonary arteriovenous malformations (PAVMs) are mostly congenital (80%) and classified as simple (80%; single feeding artery leading to a single draining vein) or complex (20%; two or more feeding arteries or draining veins) types. They commonly present in the lower lobes (53–70%) and can lead to chronic hypoxemia and systemic emboli. Approximately 70% of the PAVMs are associated with hereditary haemorrhagic telangiectasia (HHT), and about 15-30% of individuals with HHT have a PAVM. HHT is an autosomal dominant disorder, which presents with arteriovenous malformations in skin, mucous membranes and visceral organs. PAVMs are usually asymptomatic, but symptoms can occur in the 4th to 6th decade of life with the development of congestive heart failure, respiratory failure with cyanosis, life threatening cerebrovascular complications including stroke and cerebral abscesses.

The diagnosis of PAVMs can be suspected on chest radiography (98% abnormalities) as a well-defined oval mass of uniform density, ranging in size from 1-5 cm, with connecting vessels from the hilus (Fig. 1). However, chest CT
scanning is invariably required to confirm the diagnosis. The characteristic appearance of a PAVM on contrast-enhanced CT scans is the presence of a homogeneous, circumscribed, non-calcified nodule with connecting blood vessels (Fig. 2,3). Three-dimensional (3D) spiral CT images are useful in the pre-therapeutic evaluation of the angioarchitecture, namely, the number and orientation of the feeding arteries (Fig 5,6). In our case, maximum intensity projection (MIP) and volume rendered technique (VRT) were used as 3D techniques (Fig. 5, 6). Pulmonary angiography is therefore not longer the golden standard and is only required when further intervention is planned. Magnetic resonance imaging (MRI) is less useful than contrast-enhanced CT scanning and not used in the diagnosis of PAVMs. Contrast enhanced 2D echocardiography (sensitivity 92%) and a 100% oxygen study can only confirm a right-to-left intrapulmonary shunt, but they both need further investigation because they cannot determine the number of PAVMs, their location in the lung and their size. The treatment of choice in symptomatic patients and PAVMs with feeding arteries >3mm includes embolotherapy. Surgery is only indicated for larger malformations or when there is a significant risk of embolotherapy.

**Differential Diagnosis List:** Pulmonary arterio-venous malformation

**Final Diagnosis:** Pulmonary arterio-venous malformation

**References:**

Description: Nodular opacity, paravertebral in the left lower lung. Origin:
**Figure 2**

**Description:** A well-defined contrast enhancing nodule (diameter 2.5cm) posterior in the left lower lobe with an hypertrophied feeding artery and a draining vein. Note simultaneous enhancement of the right ventricle and left lower lobe nodule. **Origin:**
Description: A well-defined nodule (diameter 2.5cm) posterior in the left lower lobe with an hypertrophied feeding artery and a draining vein. Origin:
Description: A second well-defined nodule (diameter 0.6 cm) posterior in the left lower lobe with also a feeding artery and a draining vein. Origin:
Figure 5

Description: Left lower lobe pulmonary arteriovenous malformation demonstrating the simple angioarchitecture of an AVM with a single feeding artery and a single draining vein, both connected to the aneurysmal sac (nidus). Origin:
Figure 6

Description: Left lower lobe pulmonary arteriovenous malformation demonstrating the simple angioarchitecture of an AVM with a single feeding artery and a single draining vein, both connected to the aneurysmal sac (nidus). Origin: