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Case Report
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Unexplained Cyanosis and Clubbing in a Patient of Disseminated Tuberculosis Leading to a Diagnosis of Pulmonay A-V Malformation

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Abstract

Pulmonary Arteriovenous Malformations (PAVMs) are caused by abnormal communications between pulmonary artery and pulmonary veins, which are mostly congenital in nature. Although these lesions are quite uncommon, but they are important part of the differential diagnosis of hypoxemia, central cyanosis, clubbing, haemoptysis and remains a diagnostic challenge.

We describe a case of 23-year-old, female who presented with hypoxemia, cyanosis, pandigital clubbing and exertional dyspnoea along with symptoms of tuberculosis and was diagnosed as complex PAVM on bubble ECHO and CTPA. Patient also fulfilled the criteria of possible HHT, according to Curacao Criteria. Later patient underwent percutaneous endovascular therapy with good clinical outcome.

PAVM should be suspected in patients with unexplained central cyanosis and clubbing without any cardiac cause as they carry high mortality risk. Treatment is strongly recommended to prevent mortality and complications related to PAVMs.

Keywords: Pulmonary; Hypoxemia; Cyanosis; Endovascular therapy

Introduction

Pulmonary Arteriovenous Malformations (PAVMs) are rare abnormalities of the pulmonary vascular system characterized by an abnormal communication between the pulmonary artery and vein, resulting in a low resistant right-to left shunt [1]. Churton was the first to report PAVM on an autopsy study in 1897 [2]. PAVMs are relatively uncommon, with a relative incidence of 1 in 2,600 individuals, with a female to male ratio of 1.67:1 [3,4]. The exact etiology and pathogenesis of PAVM remains a challenge so far [5-7]. Most of the patients are asymptomatic, many present as exertional dyspnoea, cyanosis and haemoptysis [8]. The natural course of PAVM is not well studied but most of the lesions remain either stable (75%) or demonstrate slow growth (25%). If untreated, the right to left shunt can result in symptoms of hypoxemia, paradoxical emboli to the left side circulation, stroke and intracranial abscess [9]. PAVM are usually congenital; however, they may also occur in many acquired conditions like tuberculosis and parasitic infections [8]. This review highlights about the diagnosis of PAVMs in patients with unexplained cyanosis and clubbing.

Case Presentation

A 23, old female presented with chief complains of fever on and off, along with headache and vomiting from past one month. Patient also complained of dry cough from past 2 month. On provocation patient gave history of intermittent dyspnea, associated with exercise intolerance, occurring every 2-3 months from past many years. There was a history of weight loss and decreased appetite for 1 month. There was no history of chest pain, orthopnea, PND, palpitation, sputum production, haemoptysis, epistaxis, decreased sensorium, cyanotic spells in childhood. No stigmata of infective endocarditis were present. There was no past history of congenital heart disease, pulmonary disease, malignancy or tuberculosis. No significant family history was present.

Patient was conscious and well oriented with BP of 100/64 mm of Hg, PR- 80/min, RR- 24/min, SpO_2-78% to 80% on RA. On examination patient had cyanosis (both central and peripheral) and grade 4 pandigital clubbing. We also noticed telangiectatic lesion present on nose of the patient.

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Cardiac examination was normal with normal apex and no added sounds was present. Rest of the systemic examination including respiratory examination was unremarkable.

On the basis of the above examination meningitis with some congenital heart disease was suspected. Basic investigation revealed Hb-14.8, Hct- 45.9, with normal WBC index, platelets, LFT, KFT. ABG revealed hypoxemia, $p0_2$ of 48% and $Sp0_2$ of 79%. CSF studies showed 120 cells, (80% lymphocytes) with CSF protein and sugar 192 and 45 respectively. Chest X ray showed left lower lobe opacity with miliary pattern. 2D echo was done twice which was normal. USG was suggestive of multiple mesenteric lymphadenopathy (largest SAD 10-12 mm) with mild ascites. On the basis of above examination disseminated tuberculosis (acute meningitis with pulmonary & abdominal kochs) was made and patient was started on ATT. But what was unexplained was cyanosis and pandigital clubbing since years in a young female. So PAVM was suspected and 2D bubble echo was done which showed microbubbles initially in RA followed by left atrium after 4 beats and the entry of microbubbles were from left side of the pulmonary vein likely to be PAVM of left lung. Later CTPA was done which confirmed the diagnosis of complex PAVM (serpiginous nidus in left lower lobe with multiple dilated feeding segmental arteries and early draining left inferior pulmonary veins with nidus showing multiple venous varices and intra-nidal aneurysms along with changes of active pulmonary kochs.). In our case cyanosis and grade 4 pandigital clubbing was unlikely to be due to tuberculosis. Later on, we also noticed that an AV nevus was present on the abdomen of the patient. So, we suspected HHT (hereditary hemorrhagic telangiectasia) and patient fitted into the criteria for possible HHT. Thus, a diagnosis of Complex PAVM with possible HHT was made along with disseminated tuberculosis. Later on, percutaneous endovascular therapy (plugging and coiling) was done in cardiology department after which the patient saturation improved to more than 94% on room air.

Discussion

PAVMs are defined as arteriovenous communications between the pulmonary and systemic circulations causing a right to left shunt through the PAVM bypassing the normal filter function of the lungs [10] Thus, PAVMs are structurally abnormal, direct vascular communications between pulmonary arteries and veins Figure 1, which bypass capillary beds to create low-resistance, high-flow continuous intrapulmonary right-to-left shunts [11]. PAVMs are relatively uncommon. Most PAVMs (over 90%) are typically unilateral and solitary as in our case [6]. Although PAVMs can be diagnosed at any age Figure 2 and 3, they are mostly discovered by the age of 30 years [12].

The exact etiology and pathogenesis of PAVM is unknown so far. However, researchers suggest that dilatation of thin-walled capillary sacs results from a defect in terminal arterial loops [6]. PAVM may be present from birth and continue developing in adulthood [6]. The etiology of PAVMs can be congenital or acquired. About 80% of cases are congenital of which 80% to 90% are associated with HHT or could be acquired later in life (<20%) [12,8]. Acquired conditions such as Fanconi syndrome, schistosomiasis and hepatic cirrhosis are associated with PAVMs [13]. Chest trauma, hepatopulmonary syndrome and congenital portosystemic shunts, metastatic carcinoma, mitral stenosis, parasitic infections, tuberculosis and systemic amyloidosis are other etiologies which are associated with PAVMs [8].

Figure 1: Shows cyanosis and pandigital clubbing.

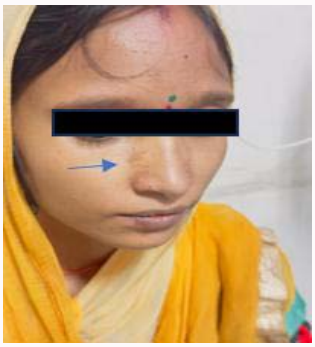

Figure 2: Shows nasal telangiectasia (blue arrow).

Figure 3: Shows cutaneous AV nevus (blue arrow).

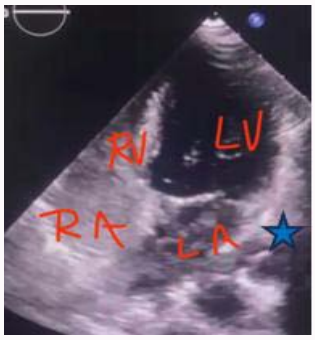

Figure 4: Contrast ECHO showing filling of the left atrium from the left pulmonary vein(star).

Hereditary Hemorrhagic Telangiectasia (HHT) is an autosomal dominant disorder, characterized by a wide variety of clinical manifestations due to the presence of multiple arteriovenous

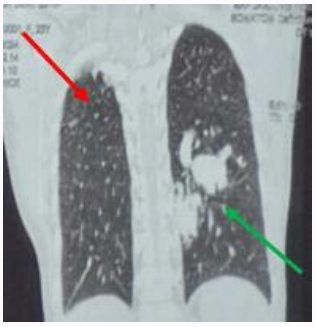

Figure 5: CTPA showing left lower lobe PAVM (green arrow), fibro-atelectatic changes in apical segment of RUL (red arrow).

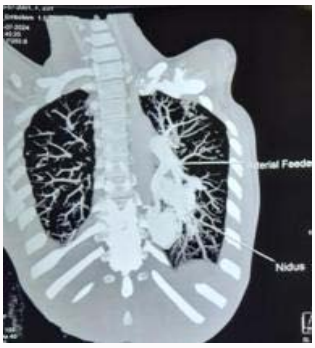

Figure 6: CT Pulmonary angiogram showing left Lower lobe PAVM.

malformations [10]. About 50% of patients with HHT have Pulmonary Arteriovenous Malformations (PAVMs) which are often multiple in nature [3]. In our case telangiectasia & visceral lesion in the form of PAVM was present, fulfilling 2 out of 4 criteria of HHT (possible HHT) Figure 4 and 5.

PAVM can be classified into 3 types- simple (most common) type, complex and diffuse type [3,12]. Most of the patients are asymptomatic (13% to 55%). The symptoms are related to degree of hypoxemia and the amount of shunt which includes exertional dyspnea and cyanosis [8]. Hypoxemia associated with orthodeoxia and platypnea are classical presentations as 65% to 83% of PAVMS are located in the lower lobes of the lungs [4]. Haemorrhages and complications from distant embolization including stroke and brain abscesses and are some of the other clinical presentations of PAVMs [14]. The gold standard for the diagnosis of PAVM is pulmonary angiography, and it is used both to diagnose PAVMs and to determine the vascular architecture of individual PAVMs Figure 6. It is especially valuable tool in planning further therapy [12]. The differential diagnosis of PAVMs is broad and includes pulmonary aneurysmal disease pulmonary varices, bronchocele and tumors [6,14].

Treatment decisions are based on a number of criteria such as patient's tolerance level, the size of the feeding artery, and symptoms of PAVMs [6]. There are two main PAVM treatment options: 1-Surgical resection, 2-Percutaneous endovascular therapy of the feeding vessel by coils or plugs/balloons (treatment of choice) [8] which was done in our patient. PAVMs do not spontaneously resolve. Mortality caused by PAVM is due to rupture, brain abscess, and stroke due to paradoxical embolization. High rates of morbidity and mortality are associated with PAVMs; mortality from an untreated PAVMs can reach up to 50%, while mortality from treated PAVMs can only reach 3% [6].

Conclusion

Pulmonary vascular malformations represent a multifaceted disease with different causes and manifestations. PAVMs should be carefully screened in patients of unexplained cyanosis and clubbing with normal 2D ECHO as early identification of PAVM cases is of paramount importance for the prognosis of patients. Current recommendations favour treatment of all symptomatic PAVMs. The treatment of PAVM should be aimed at improving symptoms like dyspnoea and hypoxemia and preventing complications such as cerebral abscesses and strokes.

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