Original Research Paper

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General medicine

HIV- RELATED PULMONARY ARTERIAL HYPERTENSION: A CASE REPORT

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ABSTRACT The use of highly active antiretroviral therapy (HAART) has increased the life expectancy of HIV-infected patients. With prolonged survival and improved control of infectious susceptibility, vascular complications have emerged as a significant source of morbidity and mortality in HIV-infected patients [1]. HIV-associated pulmonary arterial hypertension (HIV-PAH) is an important lung disease in HIV-infected persons who live longer with antiretrovirals. HIV-PAH may be detected via chest radiographs, CT scans, or electrocardiograms, but Doppler echocardiography is the most useful screening test to identify candidates for right heart catheterization. Because the survival for HIV-infected individuals with PAH with advanced symptoms (New York Heart Association, NYHA class III-IV) is worse compared with less symptomatic individuals (NYHA class I-II) (2), identification of asymptomatic individuals is of critical importance. Histologically, THE lesions in HIV-infected patients with PAH include concentric laminar intimal fibrosis, medial hypertrophy, recanalized thrombi, and plexiform lesions (3). According to a more recent study by Sitbon et al [4] in 2008, the prevalence has remained at 0.5% even in the modern era of HIV therapy, suggesting that HAART has not made a dramatic impact on the prevention of HIV-PAH. We present a case of HIV-PAH to highlight the importance of the non-infectious pulmonary complications associated with HIV disease.

KEYWORDS : HIV, PAH

CASE REPORT

A 30-year-old male, a known case of AIDS on HAART (Highly active antiretroviral therapy) for last 1 year, presented to the emergency department with chief complaints of chest tightness, progressively increasing shortness of breath and bilateral lower limb swelling for 1 year. Shortness of breath occurred even during his day to day activity since last 1 month. There was no history of wheeze, dry cough, haemoptysis, acute episode of shortness of breath, joint pain, exaggerated response to cold and any drug abuse. There was no history of febrile illness, night sweats or weight loss.

Examination showed an ill looking patient with pulse-110/min, regular, low in volume, BP-110/70 mm Hg, respiratory rate-19 breaths/min, raised JVP with large 'v' wave and pitting pedal edema. There was no clubbing, sclerodactyly or telangiectasia. Cardiovascular examination showed normally placed apex beat and a normal cardiac impulse. He had a left lower parasternal lift and a palpable P2.On auscultation, a loud P2 was present in pulmonary area and a pansystolic murmur in tricuspid area with inspiratory accentuation and basal crepitations. Tender hepatomegaly and splenomegaly were present.

Blood investigations showed Hb-12.23 g/dl, WBC count-13900/cumm, serum creatinine-2.02 mg/dl, total bilirubin-1.65 mg/dl, direct bilirubin-0.93mg/dl, ALT-69 IU/l, AST-81 IU/L, ALP-205 IU/L, and serum sodium-125.51 mmol/l. HBsAg and anti HCV-antibody were non-reactive. ANA and autoimmune panel for rheumatic disorders were negative. CD4 count was 112 cells/cumm. Electrocardiogram showed sinus tachycardia, p pulmonale and right ventricle hypertrophy.

Pulmonary function tests were normal. Diffusion capacity of lungs for carbon monoxide was reduced. Chest x-ray showed cardiomegaly with enlarged right ventricle and enlarged pulmonary arteries with oligemic lung fields. 2D echocardiography showed dilated RA and RV along with severe tricuspid regurgitation with PAH (with RVSP-59 mm Hg). Venous doppler of bilateral lower limb was normal. CT angiography ruled out any thromboembolic disease.

A clinical diagnosis of AIDS with pulmonary hypertension was made. He was started on diuretics, antifungals, intravenous antibiotics and later on sildenafil. His symptoms gradually improved after starting sildenafil. He started experiencing ease in performing his day to day activity. He was discharged on sildenafil, lamivudine, nevirapine, zidovudine, furosemide and fluconazole.

On out-patient follow up two months later, he was able to perform 6 –minute walk test with ease. He is planned for repeat echocardiogram after 12 months for estimation of his pulmonary arterial pressures and response to treatment.

DISCUSSION

Although HIV is a rare cause of PAH, this form of PAH is indistinguishable from idiopathic PAH and is an important cause of mortality in the HIV-infected population.

A diagnosis of HIV-PAH was made after excluding common differentials like chronic thromboembolic disease, connective tissue disease, left heart failure and PAH associated with lung disease.

History was not suggestive of thromboembolic disease as the dyspnea was not acute in onset and there was no sign of DVT. Moreover, CT angiography excluded this possibility. Connective tissue disease was ruled out as ANA and autoimmune panel for rheumatic disorders were negative.

Left heart disease was kept as a possibility due to presence of dyspnea, fatiguability and basal crepitations but was excluded due to absence of orthopnea and paroxysmal nocturnal dyspnea, normally placed apex beat and absence of S3. Elevated JVP, palpable P2, large 'v' wave and chest x-ray findings of oligemia in lung field with enlarged RV supported the diagnosis of pulmonary arterial hypertension with right sided heart failure which was confirmed by echocardiography.

As other major causes of PAH were excluded, a clinical diagnosis of HIV-PAH was made.

CONCLUSION

The management of PAH should not only include providing symptomatic relief to the patient but an attempt should be made to find out the etiology of PAH in the patient. A detailed history and examination along with relevant investigations must be done to reach to the final diagnosis. HIV related PAH must be kept in mind as an important cause of dyspnoea in HIV patients.

Figure 1: 2D ECHO showing dilated right atrium and ventricle with PAH (RVSP= 59 mm Hg)

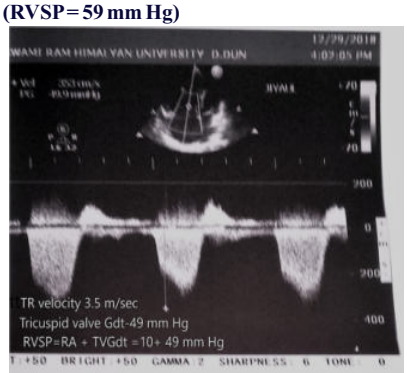
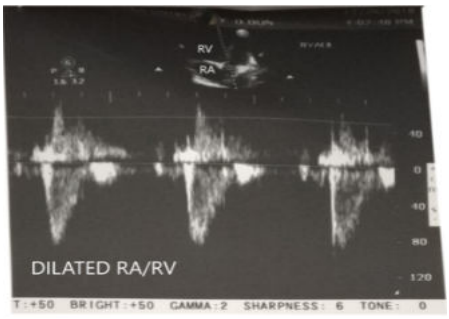


Figure 2: 2D ECHO showing severe tricuspid regurgitation



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