



A RARE PRESENTATION OF NON BACTERIAL THROMBOTIC ENDOCARDITIS IN HIV PATIENT

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Abstract : Nonbacterial thrombotic endocarditis (NBTE) or Marantic endocarditis is a rare type of endocarditis is seen in patients with HIV-wasting syndrome (1) . Its incidence is 3 to 5 percent of AIDS patients(2). Marantic endocarditis is known to be associated with malignant neoplasms, and hypercoagulable states. It can involve all four cardiac valves though left sided lesions are more common(3-5). The vegetations of NBTE were found on the aortic valve in 46.1percent, on the mitral valve in 40.6percent and on the both valves in 8.3percent and leading to valvular dysfunction (2).

Keyword : Non bacterial thrombotic endocarditis, marantic endocarditis, Human immunodeficiency virus.

Introduction: Marantic (nonbacterial thrombotic) endocarditis have been seen in HIV-infected patients. Marantic endocarditis is a non infectious type of endocarditis that can become secondarily infected. It is usually associated with hypercoagulation diseases such as disseminated intravascular coagulation, systemic lupus erythematosus, and malignancies. Surprisingly, it has been reported to be the most common endocardial lesion associated with HIV infection . It is caused by friable vegetations that consist of platelets within a fibrin mesh with few chronic inflammatory cells. It affects cardiac valves leading to valvular dysfunction with left-sided lesions being more common. Systemic and pulmonary embolization can occur leading to significant end-organ damage. Cerebral, pulmonary, splenic, renal and myocardial infarctions can result. systemic embolisation from marantic endocarditis is a rare cause of death in hiv patients.

Development of Nonbacterial Thrombotic Endocarditis:

Two major mechanisms appear pivotal in the formation of NBTE: **Endothelial injury and a Hypercoagulable state.** Marantic NBTE, thought to be a result of hypercoagulability, and is more common with increasing age and in patients with malignant disease, HIV-wasting syndrome, disseminated intravascular coagulation, uremia, burns, systemic lupus erythematosus, valvular heart disease, and intracardiac catheters. NBTE occurs at the valve closure contact line on the atrial surfaces of the mitral and tricuspid valves and on the ventricular surfaces of the aortic and pulmonic valves. Three hemodynamic circumstances may injure the endothelium, initiating NBTE.

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: (1) a high-velocity jet striking endothelium; (2) flow from a high-pressure to a low-pressure chamber; and (3) flow across a narrow orifice at high velocity.

When blood flows through a narrowed orifice, vegetations are deposited at low pressure sink immediately beyond an orifice as a consequence of **venturi effect** or at the site where a jet stream strikes a surface (**Fig1**). This deposition occurs as a result of endothelial injury or hypercoagulability.

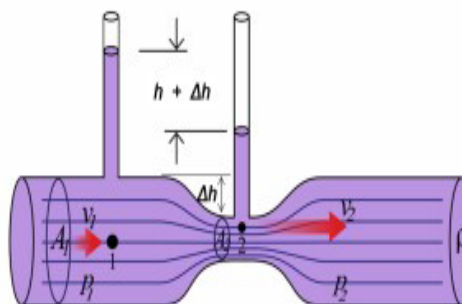


Figure 1

The pressure at "1" is higher than at "2", and the fluid speed at "1" is lower than at "2", because the cross-sectional area at "1" is greater than at "2".

NBTE is characterized by the deposition of small sterile thrombi on the leaflets of the cardiac valves (Fig 2A,B).

The lesions are 1 mm to 5 mm in size, and occur singly or multiply along the line of closure of the leaflets or cusps. Histologically they are composed of bland thrombi that are loosely attached to the underlying valve. The vegetations are not invasive and do not elicit any inflammatory reaction.



FIG 2A Nonbacterial thrombotic endocarditis (NBTE) Nearly complete row of thrombotic vegetations along the line of closure of the mitral valve leaflets (arrows).

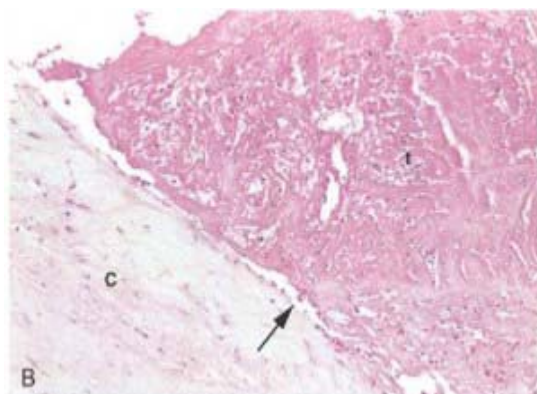


FIG2B, Photomicrograph of NBTE, showing bland thrombus, with virtually no inflammation in the valve cusp (c) or the thrombotic deposit (t). The thrombus is only loosely attached to the cusp (arrow).

CASE REPORT:

A 38 years old male admitted with complaints of breathlessness and left sided chest pain 2 months duration. Breathlessness occurs during minimal physical activity and relieved by rest (Grade III dyspnoea). He had central chest pain and squeezing in nature, non radiating type. He had generalised anasarca of the body. Edema initially arises in the legs and it progresses to generalised. He had orthopnoea and paroxysmal nocturnal dyspnoea. He had fever for past 2 months duration, Low grade, continuous in type. He had cough and no expectoration. He had h/o decreased urine output for past 1 month duration. No h/o palpitation. No h/o syncope. He had H/o extra marital contact 5 years back. He was diagnosed as HIV Positive 3 years back. He is not under ART. No past h/o tuberculosis. Not a known diabetic. Not a known Hypertensive. No past H/o coronary artery disease. No H/o of IV drug abuse. He was an known alcoholic for past 5 years, stopped alcohol 6 months back. He was an known smoker, 5 pack years, stopped Smoking 6 months back.

On physical examination Patient was moderately build. He was febrile, his temperature was 100.2 OF. Dyspnoeic, tachypnoeic, Respiratory rate was 22/min.

He was anemic and cyanosed. Grade II clubbing was present. Bilateral pitting pedal edema was present. no lymphadenopathy, no icterus, heart rate was 84/min . peripheral pulses - upper limb pulses were large volume and collapsing and all other peripheral pulses were felt and equal. Blood pressure in upper limb was 160/40 mm Hg, lower limb Bp was 200/20 mmHg in right lower limb. Other peripheral signs of AR was present. 1. Positive hill sign, 2. brisk brachial, 3. dancing carotid, 4. pistol shot femoral.

On inspection of cardiovascular system no precordial bulge, no visible pulsation, no scar over the chest, no engorged veins over the chest. JVP was increased 7cm above the angle of Louis when pt was on 450, with normal wave pattern. On palpation apical impulse was felt at 6th intercostal space, 1cm lateral to the midclavicular line, S3 was palpable, no thrill, no precordial bulge. On auscultation of cardiovascular system- S1, S2 +, A2 soft, early diastolic murmur heard with diaphragm of the steth along the left 3rd

intercostals space at and was well heard in expiration with the patient leaning forward, S3 was present, no fourth heart sound, no ejection click, no pericardial rub. On auscultation of respiratory system patient had normal vesicular breath sound and Bilateral basal crepts were present. Abdomen was distended and free fluid was present, moderate splenomegaly was present. CNS- no focal neurological deficit, Fundus was normal. Bone and joints normal.

ECG shows LVH with strain pattern.

X-ray shows cardiomegaly, bilateral minimal pleural effusion.

Ultrasound abdomen and chest : Bilateral minimal pleural effusion and moderate ascitis present. splenomegaly 8cm. liver architecture normal. both kidneys normal.

Blood culture and sensitivity: 3 sets of blood culture and sensitivity was taken at 1hr interval at 3 different sites on the same day. One set was for the aerobic organisms and the other set was for anaerobic organisms. All the cultures were negative for the organisms. Fungal culture was found to be negative.

Serological test: serological tests for Brucella, Bartonella, Legionella, C. burnetii, and HACEK organism were found to be negative

Examination of fundus : normal

Complete blood count	Total WBC count	10,200 cells/cmm
	Differential count	
	Polymorphs	60%
	Lymphocytes	35%
	Eosinophils	5%
	RBCs	3.5 million cells/cmm
	Platelets	1.5 lakhs/cmm
	HB	9 gms%
	MCV	78 ft
	MCH	27 pg
	MCHC	32%
	ESR ½ hr	50 mm
	1 Hour	110 mm

Blood	Sugar random	96mg/dl
Serum	Urea	29mg/dl
	Creatinine	0.9mg/dl
Serum Electrolytes	Sodium	138meq/l
	Potassium	3.8meq/l
Liver Function Test	Serum Bilirubin	1.1mgm%
	SGOT	35u/l
	SGPT	17u/l
	Alkaline Phosphatase	45u/l
	Serum Proteins	6.8gms%
	Serum Albumin	3.7gms%
	Serum Globulin	3.1gms%

Others	Hbs Ag	Negative
	Anti HCV	Negative
	HIV 1 & 2	Reactive
	CRP	Positive
	Rheumatoid Factor	Negative
	VDRL	Non reactive
	CD 4	418 cells/cmm

Urine routine	albumin	nil
	Sugar	nil
	Pus cells	2-3
	Epi cells	3-5

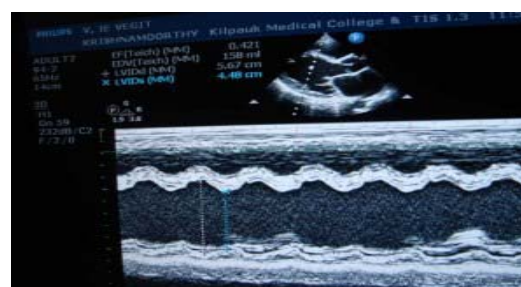
With the consent of cardiology department echo was picturised .
ECHOCARDIOGRAM done on 03/01/2012
ECHO done before antibiotic therapy showed in pic 3A,B,C
 LV - 5.6/4.4/42%
 Severe aortic regurgitation
 MR - Trivial
 Vegetation seen over non coronary cusp. It was 5mm in size.
 Severe LV systolic dysfunction



PICTURE 3A



PICTURE 3B



PICTURE 3C

After the initial echo, antibiotic and ART treatments were given. Repeat echo was taken 6 weeks later and it was same as the initial echo. Shown in **picture 4A,B,C**



PICTURE 4A



PICTURE 4B



PICTURE 4C

Patient was treated with inj.ceftriaxone (2 g IV qd), plus gentamicin (3 mg/kg qd IV q8h) after collection of blood sample for culture. Patient was also treated with T.Digoxin 0.125mg od 5days in a week, T.Enalapril 2.5 mg bd, T.Spironolactone 25mg bd, T.Frusemide 20mg bd and co-trimoxazole d.s. bd. Cardiac failure symptoms controlled after starting treatment. Simultaneously with patient consent ART counselling done and ART medications started from our ART clinic. Echo was done initially it shows 5mm of vegetation over non coronary cusp, severe aortic regurgitation and LV systolic dysfunction . Blood culture for Gram positive and Gram negative organism was negative. serological tests for Brucella, Bartonella, Legionella, C.burnetii, and HACEK organism were found to be negative. The patient was continue with antibiotics for 6 weeks. Repeat echo was taken after 6 weeks, the vegetation was persist 5mm in size, aortic regurgitation and LV systolic dysfunction also persists.

DISCUSSION

Non bacterial thrombotic endocarditis generally is a rare presentation. Its incidence is 3 to 5% in HIV patients. It affects ardiac valves mostly left sided valves. Aortic valve is the commonest valve to be affected. It leads to valve dysfunction. In our patient known HIV 3years not under treatment developed infective endocarditis with acute severe AR. The presentation showing Non bacterial thrombotic endocarditis and culture negative.

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