

## Post-coital Hemoptysis Due to Silent Coronary Artery Disease

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### Abstract

Hemoptysis after physical activity is a quite common presenting symptom. However, hemoptysis during/after sexual intercourse is a rare condition, and cardiac decompensation has been mostly implicated in the etiology. This is because, exertion associated with sexual stimulation and coitus can cause transient hemoptysis secondary to a transient elevation of pulmonary capillary pressure with subsequent rupture of pulmonary capillaries and hemoptysis.

We present a 56-year-old Caucasian male, with a past medical history of hypertension, hyperlipidemia, obesity and sleep apnea who presented with hemoptysis of four-months duration. Echocardiography revealed normal left ventricular systolic function, with Ejection Fraction EF of 55%. Grade I diastolic dysfunction, mild mitral regurgitation and aortic regurgitation.

Ischemic mitral regurgitation is a complication of coronary artery disease. It is a dynamic lesion and its severity may increase with different forms of exercise leading to flash pulmonary edema. This case highlights that silent coronary artery disease can present as post-coital hemoptysis in middle aged patients, the importance to rule out underlying cardiac pathologies through an extensive cardiac workup in patients presenting with post-coital hemoptysis.

**Keywords:** Coronary artery disease; Pulmonary edema; Mitral regurgitation; Post-coital hemoptysis

### Introduction

Hemoptysis is a very common presenting symptom in the clinical settings, and has been described after strenuous exercise. Sexual stimulation causes transient elevations of pulmonary capillary pressure with subsequent rupture of these capillaries resulting in hemoptysis. Sexual intercourse, a form of isometric exercise, can cause severe increase in ischemic mitral regurgitation compared to other forms of exercise due to increase sympathetic activity, leading to flash pulmonary edema and hemoptysis. We present a 56-year-old Caucasian male, with past medical history of hypertension, hyperlipidemia, obesity and sleep apnea who presented with post-coital hemoptysis of four-month duration [1-11].

### Case Report

A 56-year-old Caucasian male, with a past medical history significant for hypertension, hyperlipidemia, obesity, sleep apnea, chronic hepatitis C infection and insomnia, who presented with complaints of shortness of breath and hemoptysis that occurs only after sexual intercourse. This has been consistently observed over the past four months. He is a truck driver, never smoked, denies alcohol, and illicit drug use. He is also a weight lifter, works out daily, and never had history of hemoptysis after a strenuous exercise or other physical activities. There is negative history of dyspnea on exertion, paroxysmal nocturnal dyspnea, chest pain, leg swelling, dizziness, palpitations, syncope, fever, night sweats, weight loss, recent travel, or sick contact. He has never been hospitalized for cardiac or respiratory diseases in the past (Table 1).

In the emergency room, his blood pressure was 153/85 mmHg, pulse 93 beats/min regular, temperature 97.6 F (36.6°C) Tympanic, respiratory rate 20, SpO<sub>2</sub> 91% on room air, height 1.88 m, weight 275 lb (124.739 kg), BMI 35.291 kg/m. Physical exam was essentially normal. EKG showed normal sinus rhythm, no ST, T wave changes and left ventricular hypertrophy.

Chest x ray revealed bilateral upper lobe infiltrates. CT angiogram of the chest revealed diffuse scattered ground glass infiltrates, non-specific etiology, secondary to volume overload or infection, with upper lobe

predominance, no pleural effusion, no evident pulmonary embolism. The patient was initiated on sublingual Nitroglycerine 0.4 mg prn, Aspirin 81 mg po daily, Metoprolol 25 mg po twice a day, Atorvastatin 40 mg po daily, Furosemide 40 mg po daily. 2D Echocardiography/Doppler color flow study: normal left ventricular systolic function, left ventricular end-diastolic pressure was measured at 27. The estimated ejection fraction was 50% with inferior wall akinesis and mild anterolateral wall hypokinesis. Grade I diastolic dysfunction, mild mitral regurgitation and aortic regurgitation. Estimated normal pulmonary systolic pressure, left atrial enlargement, no mass, no thrombus, no pericardial effusion.

Stress Echocardiogram: Patient reached his target heart rate, there was no chest pain, hypokinesis of the septal and lateral wall after the stress was noted. There is infero-posterior fixed wall motion abnormality/hypokinesis LVEF post stress 40% to 50%. (Evidence of reversible ischemia in LAD territory), and worsening mitral regurgitation. Patient then had Cardiac Catheterization which revealed: left main had mid to distal 30% to 40% plaquing, LAD had 70% to 80% stenosis, left circumflex had distal 90% stenosis. The first obtuse marginal branch was moderate to larger caliber and was occluded proximally. It filled from left to left collaterals. The ramus had a mid-80% to 90% stenosis. The right coronary artery was totally occluded proximally. Eventually, the patient underwent surgical revascularization with a 3-vessel CABG with complete symptoms relief thereafter (Table 2).

### Discussion

Sexual activity is associated with moderate hemodynamic stress and increases the risk of MI [10,11]. During sexual activity, there is increase in heart rate, right ventricular systolic and diastolic pressures,

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Labs	Results	Normal range
WBC	8.000 /microL	4.000-10,000 /microL
Hemoglobin Hgb	17.1 g/dl	14-17 g/dl
HCT	51.5 %	41-51 %
MCV	98 fL	80-100 fL
Platelets	347,000 /microL	150,000-350,000 /microL
INR	0.9	09-1.1
PTT	33 s	25-35 s
Glucose	99 mg/dl	70-100 mg/dl
Urea	13 mg/dl	8-20 mg/dl
S. Creatinine	1.14 mg/dl	0.7-1.13 mg/dl
Sodium	140 meq/L	135-145 meq/L
Potassium	4.5 meq/L	3.5-5.0 meq/L
S. Troponin I	0.22 ng/ml	0-0.5 ng/ml
BNP	88 pg/ml	< 100 pg/ml
D. Dimer	656 ng/ml	< 500 ng/ml

Table 1: Baseline investigations.

Labs	Results	Normal range
S. Troponin I	0.22- 0.22- 0.17 ng/ml	0-0.5 ng/ml
Total Cholesterol	262 mg/dl	150-199 mg/dl
HDL	24 mg/dl	>40 mg/dl
LDL	233 mg/dl	<130 mg/dl
Triglycerides	129 mg/dl	< 150 mg/dl
A1C	5.4%	5.7-6.4 %
Sputum culture	Negative	--
Coccidioides Immitis Ab	Negative	--
ESR	16 mm/hr	0-22 mm/hr
ANA	1:20	1:40 – 1:60
Rheumatoid factor RF	6 units/ml	<40 units/ml
ANACA	<1:20	<1:20

Table 2: The patient was admitted to the hospital and placed on telemetry, further work up showed.

and pulmonary artery pressures was found to be significantly enhanced during coitus when compared to usual forms of exercise in a patient with congestive cardiac failure and pulmonary hypertension [11,12]. Coitus may lead to a greater elevation in pulmonary venous pressure, increase in pulmonary capillary pressure, rupture of capillaries, and subsequent hemoptysis [9,10].

The standard clinical measure of exertion is the MET (metabolic equivalent of oxygen consumption); 1 MET is defined as 3.5 mL O<sub>2</sub> uptake/kg per min, which is the resting oxygen uptake in a sitting position. Sexual activity is often equated with 2 to 3 METS during the pre-orgasmic phase and 3 to 4 METS during orgasm; this is equivalent to walking at two to four miles per hour on a level surface [12]. Exercise testing is often used to assess both exercise tolerance and tolerance for sex.

In patients with ischemic heart disease, coitus may result in transient left ventricular compromise leading to pulmonary congestion and hemoptysis [1-3]. The rapid rise in systemic arterial pressure following sexual activity can also augment the severity of mitral regurgitation, thereby contributing to the rise in pulmonary capillary wedge pressure in patients with congestive heart failure [8-10].

In patients whose symptoms or valve disease severity are indeterminate and in those with asymptomatic severe valvular disease, exercise stress testing may provide an assessment of symptomatic and hemodynamic response to physical activity. Exercise echocardiography can provide additional information on the physiological response to exercise, including ventricular function, inducible increases in valve gradients, and inducible pulmonary hypertension [5,7,10].

In the index case, we concluded that hemoptysis resulted mainly from ischemic heart disease that caused transient left ventricular compromise, and increase in the severity of already existed ischemic mitral valve regurgitation with subsequent elevation in pulmonary venous pressure following coitus leading to flash pulmonary edema and hemoptysis.

## Conclusion

Post-coital hemoptysis is rare and mainly results from cardiovascular compromise. There is a need for proper evaluation to determine the cause of hemoptysis, which will determine the proper line of management. Owing to the fact that cardiac pathologies are more likely to cause post-coital hemoptysis than pulmonary pathologies, it may be wise to initially investigate ruling out the cardiac cause [1-3,5,7]. Sildenafil use has been reported to be associated with alveolar hemorrhage and should be considered in patients with recurrent hemoptysis with no obvious cause. We recommend physicians to ask patients about the use of sildenafil before sexual intercourse, and to rule out silent cardiovascular artery disease in cases of idiopathic hemoptysis [1,4].

Beta blockers reduce myocardial oxygen demand and can minimize or eliminate angina during sexual intercourse. The Determinants of Myocardial Infarction Onset Study found that beta blockers reduced the risk of MI following anger [11] but not sexual intercourse [10].

Our case highlights the importance to ask every patient independent of his or her age about the possibility of the hemoptysis being related somehow to sexual intercourse, and should be included in the differential diagnosis of every patient with unexplained hemoptysis.

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