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A rare case of tricuspid valve endocarditis with splenic infarct and Atrial Fibrillation-case report and review of literature.

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Abstract

Clinical and imaging findings from a rare case of a patient on intravenous drug abuse (IVDA) with a splenic infarct in the setting of Tricuspid valve endocarditis (TVE) and Patent Foramen Ovale (PFO) are presented through this research article. Literature does not portray significant information on medical vs. surgical approach in the management of TVE with a splenic infarct and PFO. In our attempt to highlight this unique clinical scenario, we are surmounted by the dilemma in absence of any available clinical management guidelines-either treat the patient with the closure of the septal defect inviting more complications or manage the patient medically in a setting where the patient is continuously under the effect of Intravenous drug Abuse.

Keywords- PFO, Endocarditis, IVDA, TVE.

Case History:

38 year old HIV-negative female presented with a history of fever, chills, productive cough with blood tinged sputum, dyspnea, chest pain and past history of hypertension, hepatitis C, acute renal failure and associated TVE (tricuspid valve endocarditis) secondary to intravenous drug abuse (IVDA) since the past two weeks. The patient presented to the hospital twice in the two week period, initially with complaints of fever and cough, which was treated with IV antibiotics and the patient, was discharged. During the second visit to the hospital, patient presented with severe shortness of breath, cough, malaise, chest pain and disorientation and patient was admitted to ICU and started on IV antibiotics. Patient developed new onset atrial fibrillation (A-Fib) which was treated with amiodarone and no associated history of nausea, vomiting, palpitations, diarrhea and change in bowel habits was reported. On physical examination, the patient was noted to be tachypneic, tachycardic, with elevated JVP (Jugular venous pressure) and

3/6 pansystolic murmur over lower left sternal border. Blood culture identified MRSA (methicillin resistant staphylococcus aureus) which is known to be associated with aggressive presentation of IE (infective endocarditis).

Past medical history is significant for asthma, hypertension, seizures, IV drug abuse (last reported use in 2007), polyarthritis, acute renal failure and chronic hepatitis C.

Labs revealed elevated WBC (white blood cell) count at $21000/\text{mm}^3$, an ESR (erythrocyte sedimentation rate) of 45, sodium at 117 mEq/L, creatinine at 2.18 mg/dL with a GFR (glomerular filtration rate) of 25, CRP (C-reactive protein) at 216 and lactic acid of 2.5U/ml. History eluted that patient is abusing IVD and her urine drug screen was positive for benzodiazepines and opiates. Chest exam reported bilateral scattered wheezes and bilateral multiple infiltrates.

IE was established as the Clinical diagnosis and was started on normal saline and IV antibiotics (cefipime and vancomycin per the standard protocol). Blood culture and sensitivity, sputum culture, urinalysis and urine culture/sensitivity were ordered. Blood culture was returned positive for staphylococcus epidermidis and enterococcus faecalis. Chest CT (computed tomography) revealed multifocal lesions in the lungs bilaterally suggestive of pulmonary infarction (Figure 1). Within the same CT, a splenic infarct was identified (Figure 2). The 2-D ECHO (echocardiogram) identified small tricuspid valve vegetation on a posterior tricuspid leaflet with severe tricuspid regurgitation (TR). Transesophageal echocardiogram (TEE) with bubble study revealed PFO, severely dilated right atrium (RA), mild mitral regurgitation with dilated left atrium (LA), prominent budd-chiari network and TV vegetation. During hospital stay, patient developed new onset atrial fibrillation which was managed by amiodarone and heparin induced thrombocytopenia (HIT), successfully treated with fondaparinux.

Before discharge, cardiothoracic surgery recommended valve replacement and possible PFO closure after completion of antibiotic therapy but the patient never returned to the hospital for future evaluations and to undergo surgery as recommended. Figure 3 TEE shows PFO during Bubble study on TTE.

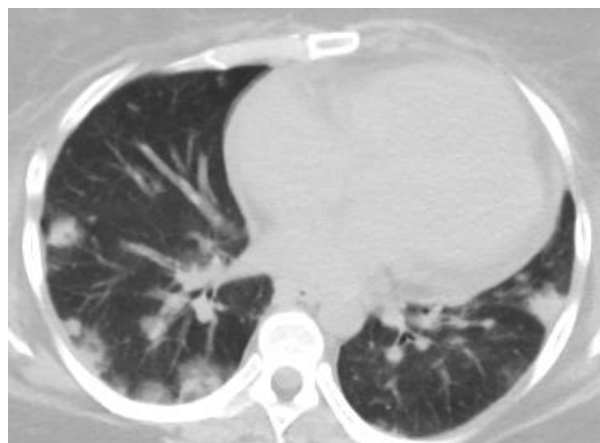


Figure 1. CT image of the Pulmonary Infarcts



Figure 2. CT Chest showing Splenic Infarct

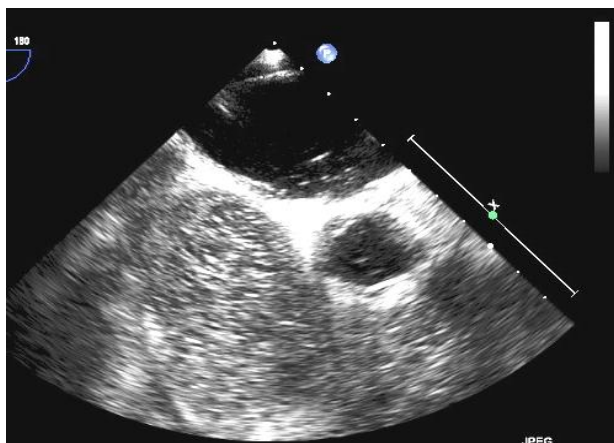


Figure 3:TEE showing PFO during Bubble study.

Discussion:

Occurrence of TVE is known within the patient population on IVDA.^{1, 4, 5} Literature also points to the diagnostic significance of the Transthoracic echocardiography (TTE) in diagnosing these rare presentations.² However, this case as per our knowledge, is one of the rare presentations in patients with TVE associated with IVDA. In this patient, the tricuspid valve vegetations could have possibly resulted in a series of paradoxical systemic emboli (splenic infarct, pulmonary infarcts) through a patent foramen ovale under a continued risk of re-infection due to IVDA.

Very few studies have reported on the existence of intracardiac shunts and possible paradoxical systemic emboli.^{2, 3} Negi et al⁶ have reported on the occurrence of septic pulmonary emboli and tricuspid valve endocarditis. Johri et al² published on the extension of tricuspid vegetation to the left atrium through a patent foramen ovale but the actual presentation of a patient with a splenic infarct and septic pulmonary emboli with a TVE and PFO (patent foramen ovale) is yet to be described in literature. It is important to understand that PFO alone is not an independent risk factor for cryptogenic stroke (CS) and incidental finding of PFO is not an indication for its closure.⁴ In our case, patient came back twice with the tricuspid valve endocarditis and had systemic manifestations, the closure of PFO was justified in our setting.

To our knowledge, the approximate % of patients suffering from a splenic infarct in conjunction with tricuspid valve endocarditis has not been published whereas up to 35% of the patients with patients with left sided endocarditis present with left upper quadrant abdominal pain.⁷ Occurrence of Atrial fibrillation (A-Fib) in a setting of TVE with a PFO with a prominent budd-chiari network is considered another unique finding for this clinical presentation. Chiari networks have a well-documented association with a PFO.⁸ Though Chiari networks are present in 1.5% to 4% of the population⁹, it has been demonstrated by Schneider et al⁹ that a PFO was

associated with 83% of patients with existent Chiari networks compared with 28% of controls. Johri et al² reported a patient presenting with TVE and a PFO with a mass in the LA and septic emboli to lungs confirmed through Pulmonary CT. Turek et al³ reported a case of tricuspid valve endocarditis in conjunction with a PFO in an IVDA with no atrial mass and severe TR (tricuspid regurgitation).

Contemplating Medical vs. Surgical treatment in absence of any published literature, was a challenge for our patient. Usually patients in similar situations do well on medical therapy and surgery is only indicated in case medical treatment fails or patient is going for another cardiac surgery like Mitral/Aortic valve surgery.⁴ Repair is usually preferred over valve replacement considering operative higher mortality in replacement surgery.⁴ However, with introduction of more novel approaches like percutaneous TVR, it may be possible to replace TV alone (in cases of RHF {right heart failure} with severe TR or vegetations>20mm).

Absence of any published randomized clinical trials data on safety and efficacy outcomes comparing the surgical or percutaneous PFO closure vs. medical management in patients with concomitant TR and TVE is lacking, though there is supporting data from observational studies. It is possible that percutaneous approach is likely to be associated with minimal complications, being minimally invasive. To close the PFO, various devices have been studied (Amplatzer, Gore helex, Premere, INTRASEPT) and are used off label but none of the devices have received FDA approval. Verma and Tobls⁶ published that 8000 PFO closures are performed each year without any major complications whereas occurrence of complications (0.28%) have required surgical explantation of these devices either due to a thrombus, chest pain, perforation, residual shunt or recurrent stroke. Yared et al¹⁰ have reported on 1355 patients undergoing PFO closure between 1998 and 2004 that 1.5% of the patients had major complications and 7.9% of the patients had minor complications. Findings from the randomized clinical trials (RESPECT, CLOSURE 1)^{11, 12} conducted for studying the occurrence of cryptogenic stroke in patients with PFO closure vs. medical therapy have reported no superiority of closure of a patent foramen ovale over medical therapy alone in preventing cryptogenic stroke.

However, our patient did not have other valve involvement; indication for Tricuspid valve repair and/or replacement could be justified by the following criteria in our case:

1. PFO and CS
2. Septic emboli
3. Severe Tricuspid Regurgitation

We would like to highlight some unusual occurrences which seem pertinent in the interest of Medical literature and have not been previously reported perplexed with the possible medical/surgical approach: 1) TVE and splenic infarct 2) Chiari network and PFO patency 3) PFO with A-Fib.

Conclusion

Our report describes a rare case of a patient with occurrence of splenic infarction in presence of a TVE and PFO which to our knowledge has not been often reported. In absence of any published randomized clinical trial data on Surgical vs. Medical therapy, existing dilemma remains unparalleled on how to mitigate the risk for future paradoxical emboli with significant risk of systemic complications and therapy based on medical or surgically management where there is a high likelihood of devices being infected under continuous IVDA abuse. Only a randomized clinical trial of PFO closure in patients with TVE may possibly answer this question, but it would be difficult due to the rare nature of this clinical scenario.

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