e-ISSN: 0975-1556, p-ISSN:2820-2643

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2024; 16(3); 268-273

Case Report

A Patient with Sudden Onset Massive Pericardial Effusion with Right Sided Infective Endocarditis with Multiple Pulmonary Septic Emboli - A Case Report

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Received: 25-12-2023 / Revised: 23-01-2024 / Accepted: 26-02-2024
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Conflict of interest: Nil

Abstract:

Septic embolism is an obstruction of a blood vessel, typically by an infected thrombus that travels through the bloodstream from a distant infectious source and blocks a blood vessel. Here a case is presented of pulmonary septic emboli in a middle aged patient who complains of fever, chest pain, vomiting and shortness of breath. He was diagnosed with massive pericardial effusion along with right atrial thrombus and multiple pulmonary emboli. On pericardiocentesis, reports were in favour of tuberculosis along with pyogenic features. Further investigations revealed presence of multiple septic / thrombotic embolus. Patient had septicemia which resolved slowly.

Keywords: Septic Embolism, Pericardiocentesis, Right Atrial Thrombus.

Introduction

Tricuspid valve infective endocarditis (TVIE) is an uncommon entity, especially when compared to left sided infective endocarditis. Right sided infected infective endocarditis (RSIE) accounts for approximately 5-10% of all cases of infective endocarditis.[1]

It is observed that the majority of Right sided Infective endocarditis (RSIE) involve the tricuspid valve, with some estimates as high as 90 %. Most of the Tricuspid valve Infective endocarditis (TVIE) cases are associated with intravenous drug use (IVDU).

Since 2006, there has been a considerable increase in the rates of TVIE, which can be attributed to the rise in IVDU in the US. [2] Tricuspid valve Infective endocarditis (TVIE) can be caused by various risk factors such as IVDU, hemodialysis catheters, pacemakers, and defibrillators leads. Staphylococcus aureus is the most common organism that leads to TVIE, but other bacteria such as different Staphylococcal and streptococcal species can also infect the tricuspid valve.

The diagnosis of TVIE can be established using Duke's criteria, but there are certain features of TVIE that can make its detection more challenging, such as absent murmur, concurrent pneumonia and less peripheral phenomena like splinter hemorrhage. [3] It is important to note that both TVIE and RSIE are treatable diseases eith favourable outcomes when detected and treated early. Although the prevalence of these diseases is increasing, antibiotics and surgical options are still the most effective treatments. In fact, one study showed a 12% increase in hospitalisations for IVDU related infective endocarditis. [4] IVDU is responsible for 86% of TVIE cases.

While more common among males, the ratio is evening out. The population with TVIE is becoming younger groups. [4] The diagnosis of IE is determined using the Duke's criteria which are widely accepted standard for diagnosing all types of IE. This criterion categorizes IE as definite, possible or rejected, based on specific clinical criteria.

Definite endocarditis is defined as meeting two primary criteria, or one major and three minor criteria, or five minor criteria. Possible endocarditis is diagnosed when one major and two minor criteria are met, or when three minor criteria are present. [5] In IE, routine laboratory values are usually not specific.

Therefore, a basic workup should be done. Some of abnormalities include leucocytosis, elevated CRP, ESR, normocytic anemia or urinalysis revealing

hematuria, proteinuria or pyuria. In addition ECG may reveal conduction anomalies such as heart block. [6]

To diagnose thrombotic vegetation's in infective endocarditis (TVIE), following investigations needs to be done.

- Chest X-rays to check for septic emboli or infiltrates.
- CT scans of the abdomen to look for metastaic emboli such as splenic or renal infarcts. If TVIE is suspected, blood cultures from at least three different sites.
- Echocardiography is essential in diagnosing TVIE. A transthoracic echocardiogram (TTE) is done, in case this is inconclusive or negative, Trans esophageal echocardiography (TEE) is necessary. [7]

Case Report

A Male patient 26 year old presented to the hospital with complain of fever with chills since 15 days , bilateral chest pain on and off which increases on inspiration and exertion , multiple episodes of vomiting , shortness of breath with MMRC grade 2 since 15 days. There was mild cough and decreased appetite. There was no h/o palpitation, pedal edema and PND. There was no h/o Diabetes, Hypertension, jaundice or tuberculosis. Patient was chronic alcoholic (250 ml /day) and tobacco chewer since 8-9 years. On examination BP was 150/120 mm Hg, pulse 90/min, regular, Spo2 98%, RBS was 108 mg/dl. On auscultation fine crepitations were present along with muffled heart sounds. Per abdomen showed tender hepatomegaly. Patient was conscious, oriented and responding to commands. Clinical diagnosis was also in favour of acute pancreatitis and investigations were supported by raised serum Amylase and Lipase. But CT scan of abdomen did not revealed acute pancreatitis and some of symptoms improved after few days.

Following investigations were done:

2D echo was done on urgent basis which revealed Right atrial pedunculated mass attached to septal tricuspid leaflet/ interatrial septum (size- 23×25). No IVC thrombosis noted. Large Pericardial effusion was present. Posterior to LV- 24 mm, anterior to RV- 11 mm, lateral to LV- 20 mm, lateral to RV – 24 mm, at apex – 17 mm. No RWMA, mild TR, no PAH, no cardiac tamponed. Urgent Paricardiocentesis was planned, but in view of deranged PT /INR of 4.5 it was postponed.

Antibiotics, inj vitamin K , nebulisation with duolin and budecort along with diuretics was given. After 1 week patient had complain of two episodes of hemoptysis. He was given injection Tranexamic acid and CT Pulmonary angiography was advised. After 4-8 FFP transfusion, PT/INR was normalised and pericardiocentesis was done. Later he was shifted to ICU in view of falling saturation and increased breathlessness. Respiratory opinion was taken and it was suspected to have pulmonary Koch's.

Bronchoscopy was advised but patient's condition was not allowing. Cardiothoracic vascular surgeon opinion was taken and in possible of high suspicion of right sided infective endocarditis, antibiotics were started but any surgical intervention was denied in view of poor condition of patient.

Serum calcium (ionic – 0.9 mmol/l) suggesting hypocalcemia. Correction done S. calcium (I) 1.12 mmol/l

Serum potassium: 6.14, 5.59, 5.62, 2.9 mmol/l, 3.42 mmol/l, 3.40 mmol/l, 2.89 mmol/l, 2.73 mmol/L, 2.99 mmol/l, serum Sodium – 125, 126, 131, 135, 133 mmol/l, 132 mmol/l, S. urea - 229.72 mg/dl, 227.90 mg/dl, 258.27 mg/dl, 174 mg/dl, creatinine- 2.74 mg/dl, 2.42 mg/dl, 2.40 mg/dl, 1.39 mg/dl, S. Bilirubin (total) 2.34, 1.79 mg/dl, SGOT 2444 U/L, 1654 U/L, SGPT 2586 U/L, 1951 U/L, S. Alk. Phosphatase 161.40 U/L, Anti-HCV – ve, HbsAg -ve, HIV –ve, S. Albumin 3.14 g/dl , S. Protein (T) 7.67 gm/dl, NT-PROBNP 880 pg/ml.

Autoimmune profile was sent.

Test name	Result	Dilutional factor
ANA	-ve	1:100
AMA	-ve	1:100
ASMA	-ve	1:100
Anti- LKM-1	+ve	1:100
Actin	-ve	1:100

Procalcitonin assay 1.55, 1.13 ng/ml, HsCRP 82.30 mg/dl, PT/INR 47.80/4.03, 36.40/3.01, 37.60/3.12, 23.00/1.44, ABG – Po2- 75.3 mmHg, PCo2-19.9 mmHg pH 7.466, s. K – 5.31 mmol/l, HCO3A 13.9 mmol/l.

HCO3S 21.1 mmol/l, O2 sat 96.2% (showing partially compensated respiratory alkalosis.), S. Amylase – 279.23 U/L, 225.24 U/L, S. Lipase 196.64 U/L, 165 U/L, S. uric acid 20.19 mg/dl, Hb 13.60 gm/dl, TLC - 25.46× 10^3 /µl, 16.17 × 10^3 /µl, platelet count – 185 × 10^3 /µl, MCV 97.40 fl, RDW-CV 18.3 %, HCT 44.1%.

Polymorphs 89%, lymphocyte 7%, Eosinophil 0, monocyte 4 %, T3 = 80.15 ng/dl, T4 = 7.70 µg/dl

TSH = 0.76 uIU/ml, Blood culture -ve, urine culture –ve.

USG whole abd

Liver –

- 14.5 cm with altered echotexture with altered hepatic echotexture with prominent hepatic veins (8.8mm).
- No evidence of any focal lesions noted in the present study.
- No intrahepatic biliary dilatation.
- CBD is normal in diameter (3.5mm), Portal vein is prominent (12mm), Pancreas normal.
- Spleen is normal in size (11mm).
- No focal lesion.
- Kidney normal.
- B/L raised echogenicity with maintained CMD suggestive of GRADE 1 MRD.
- Urinary bladder - normal
- Prostate normal in size with volume (~24 cc)

Gross pericardial effusion: Bilateral moderate pleural effusion.

Impression:

- Altered hepatic echotexture with prominent hepatic veins.
- Gross Pericardial effusion
- B/L moderate pleural effusion.
- Grade 1 MRD.
- ECG showing low QRS voltage, Tachycardia, some electrical alternans present.

More obvious in figure 2 in V2 lead.

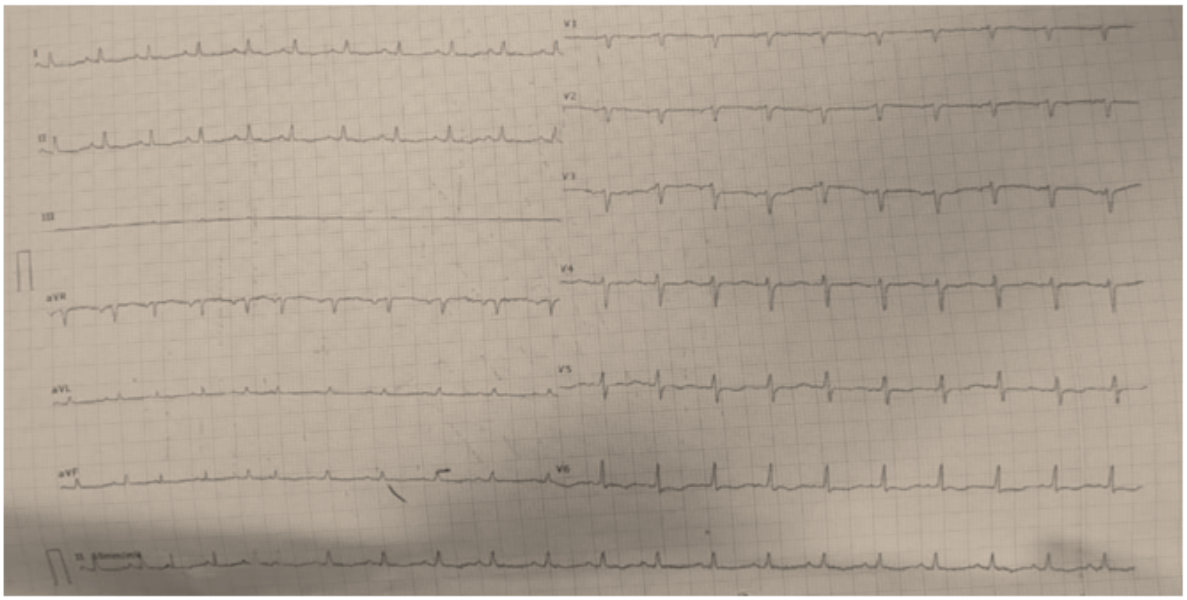

Figure 1:

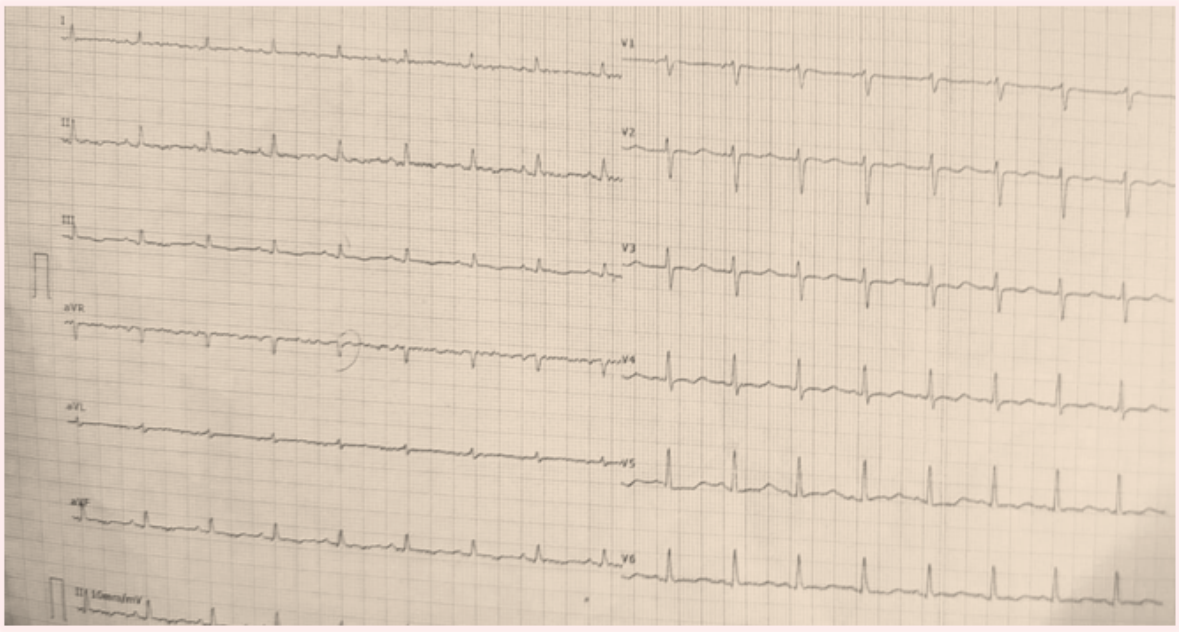

Figure 2:

2D ECHO and M-MODE Examination Values

- Aortic root – 24 mm
- Left Atrium – 28 mm
- Left Ventricle
- IVS ED – 08 mm IVS ES - 10 mm EDV – 124 ml
- LVPW ED – 09 mm LVPW ES- 10 mm ESV- 48 ml
- LVID (D)- 48mm LVID (S) – 30 mm
- Ejection Fraction 60 % (60-70%)
- Shortening Fraction 32% (24-42%)
- Doppler Study: Velocity M/S Pattern Gradient Regurgitation
- Mitral Flow: E = 0.55 0/4
- A = 0.89
- Aortic Flow: = 0.85 0/4
- Pulmonary Flow = 0.99 0/4
- Tricuspid Flow = 0/4
- Summary of Findings & ECHO Diagnosis
- Pedunculated Mass Attached to Septal Tricuspid LEAFLET/IAS (SIZE- 23×25 mm) Thrombus? Mass?
- NO IVC Thrombus Noted.
- Moderate Pericardial Effusion, POST TO LV = 24 mm, ANT TO RV= 11 mm, LAT TO LV = 20 mm, LAT TO LV = 24 mm, APEX = 17 mm.
- Normal Chamber Dimensions LVDD Grade II
- Normal LV Systolic Function, LVEF 60% NO RWMA.
- NO Significant Valvular Regurgitations.
- NO PE/CLOT/VEGETATIONS.
- Cardiac CT Advised with Pericardiocentesis.
- 3 Days After Repeat 2 D ECHO –
- Massive pericardial Effusion.
- Ant to RV = 13 mm.
- Apex 32 mm.
- Lat to LV = 32 mm.
- Post to LV = 32 mm.
- No Tamponade

HRCT Thorax

- Moderate right and mild left pleural effusion seen with underlying atelectasis
- Variable sized nodular lesions (largest measuring ~ 3.3× 2.7 cm in right lower lobe) seen in both lungs, some of them showing small cavitation within.
- Patchy consolidation with ground glass opacity as well as reverse halo seen in right lower lobe, predominantly in the basal segments with superimposed mild inter-lobular septal thickening.
- Marked pericardial effusion seen with compression of underlying heart chambers.
- Note is made of minimal per-hepatic free fluid in visualised sections of upper abdomen.
- No significant mediastinal or hilar lymphadenopathy is seen.
- Aorta and pulmonary arteries are normal in caliber.
- Trachea, proximal bronchi and esophagus are normal.
- Chest wall is normal.

Impression: Multiple Variable Sized Cavitating Nodules (Likely Septic Emboli) Along With Consolidation & Ground Glass Opacities As Described With Bilateral Pleural and Pericardial Effusion. Likely Infective.

After PT/INR correction, Pericardiocentesis was done. Following reports available

- Colour- yellow turbid
- Glucose- 33.80 mg/dl
- Protein- 4.61 gm%
- ADA- 75.10 u/l
- TLC- > 10,000cells/cmm
- Lymphocytes 10%, Polymorphs 90%

CT Pulmonary Angiography

- Moderate right and mild left pleural effusion seen with underlying atelectasis. (Increased in amount with increased underlying atelectasis as compared to prior scan)
- Variable sized nodular lesions (largest measuring 3.5 × 2.9 cm in right lower lobe) seen in both lungs, some of them showing small cavitation within.
- Patchy consolidation with ground glass opacity as well as reversible halo seen in right lower lobe , predominantly in the basal segments with superimposed with mild inter lobular septal thickening (there is increase in consolidation and ground glass opacities).
- Marked pericardial effusion seen with compression of underlying heart chambers.
- Irregular mildly enhancing hypodense lesion (~ 15 × 22 × 26 mm) is seen adherent to the region of tricuspid valve and inter-atrial septum seen in right atrium of heart? Thrombus/infective vegetation.
- Pulmonary trunk is normal bifurcating into right and left pulmonary arteries. There is evidence of non-enhancing hypodense content in right anterior segmental artery and left anterior basal segmental artery? Thrombotic occlusion.
- Few enlarged mediastinal lymph nodes are seen, largest measuring ~16.8 mm SAD in right para-tracheal region.
- Note is made of minimal peri-hepatic free fluid in visualised sections of upper abdomen.

Impression:

Multiple variable sized cavitating nodules (likely septic emboli) along with consolidation & ground glass opacities as described with b/l pleural and pericardial effusion.

Irregular mildly enhancing hypodense lesion adherent to the region of tricuspid valve and inter-atrial septum in right atrium of heart (? thrombus/ infective vegetation) with non-enhancing hypodense content in right anterior segmental artery and left anterior basal segmental artery (?thrombotic occlusion).

Discussion

This patient has been diagnosed to be a case of right sided infective endocarditis with multiple septic pulmonary emboli. Though patient has not given any h/o possible risk factors except that he was chronic alcoholic.

This patient underwent 2D echo and pericardiocentesis which revealed neutrophilic predominance with raised ADA. This lead to the start of Antituberculosis treatment.

When a patient is suspected to have TVIE, the treatment should begin with an empiric antibiotic regimen. Coverage for Staphylococcus and streptococcus is recommended and Vancomycin is a good choice of antibiotic. However, once blood cultures have been taken and analysed, the antibiotics should be tailored to the specific organism causing the infection .This should be continued for six weeks from the date of the first negative blood cultures. Short courses of antibiotics include in cases of isolated TVIE. Those without any complications secondary to IE, and those with IE from HACEK organisms. [9]. Treatment in this case also started with broad spectrum antibiotics. Blood cultures came negative. Surgical treatment of IE is reserved for individuals with the following: [10]

- TV vegetation > 2cm with septic pulmonary emboli.
- Persistent bacteremia for one week despite adequate treatment.
- Severe tricuspid regurgitation with right-sided heart failure.

The fatality rate is low in TVIE and prognosis is also good in case of uncomplicated TVIE. [11] In one study of native valve endocarditis, the overall mortality rate was 6% [12].

Vegetation size is an important prognostic factor. Those with vegetation greater than 1cm have significantly increased mortality. Individuals who have concurrent left-sided infective endocarditis have a worse prognosis. Complications of TVIE include [13]

- Peri-annular abscesses
- Septic pulmonary emboli
- Splenic abscess
- A mycotic aneurysm
- Heart failure

Here patient presented with complications of septicemia with MODS along with septic pulmonary emboli and heart failure.

Septic emboli results in two insults- the early embolic/ischemic insult due to vascular occlusion that may lead to infarction and the infectious insult that leads to inflammation and possible abscess formation [14].

Septic emboli originate depending on the anatomical location of the infection and the vasculature surrounding that area. For example, orbital cellulitis with Streptococcus constellatus was complicated with cavernous sinus thrombosis and pulmonary embolism. [15]

There are reports of septic pulmonary emboli from the right atrial thrombus related to tunnelled hemodialysis catheters. [16]

In a recent study of Infective endocarditis by Erdem et al., 44.4% of patients had major embolic events. The distribution of embolism was as follows; cerebral (26.3%), splenic (6.8%), pulmonary (6.1%), renal (2.9%), peripheral (2.2%), coronary (n=4), mesenteric (n=3). [17]

Treatment & Management

Patient was started with antibiotics and Anti tuberculosis drugs. Due to extensive patchy consolidation and atelectasis, Spo2 decreased. Patient was shifted to ICU. After broad spectrum antibiotics, ATT and suppostive treatment along with steroids, slowly patient's condition was improved. Paricardial drain was kept for daily drainage. After repeat 2D echo confirmation of decreased pericardial effusion, drain was removed and patient was discharged.

Conclusion

A patient with right sided infective endocarditis may present with complications of septic pulmonary emboli. This varies based on the severity and size of the damaged pulmonary parenchyma from asymptomatic lung nodules like lesions on pulmonary imaging to severe dyspnea and hypoxemic respiratory failure.

High degree of suspicion is needed if a patient presents with pneumonia like symptoms and hemoptysis. Early recognition and institution of antibiotics will lead to decreased mortality and early surgery performed within the first two days after diagnosis, plays a pivotal role in treating these patients.

Ethical Approval: The ethical approval was not required as patient's identity was not disclosed.

Author's Contribution

Jyoti Verma did the plan of manuscript preparation, investigations and management, Upendra have

contribution in interdepartmental references and timely as required inward management and Inderjeet have contribution in sampling and collection of reports.

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