Volume 07, Issue 01 (January-February 2024), PP 52-58 www.ijmsdr.org
ISSN: 2581-902X

International Journal of Medical Science and Dental Research

Case Study on Young Male With Myocarditis with Cardiogenic Shock With IWMI

SHAZIYA TAHNIYATH,GUNISETTI TEJASWINI, TEJASWI CHILLARA*

ABSTRACT: Myocarditis is a condition characterized by the inflammation of the heart muscles leading to decreased cardiac output. The destruction of myocyte from acute injury results in triggering the innate and humoral immunity systems resulting in long-term cardiac damage, contributing to left-ventricular dilatation and cardiac failure. The clinical manifestations includes fever, myalgias, chest pain, heart palpitations, dyspnoea, fatigue etc. The etiological factors such as infectious and non-infectious agents like viruses, bacteria, protozoa, fungi, toxins, physical conditions may cause myocarditis. A 25 years male patient was presented with complaints of epigastric pain, dyspnoea on exertion since 3 days. H/O fever with chills since 3 months, vomitings (4 episodes), neck pain, generalized weakness, diarrhoea (4-5 episodes). The patient was hypotensive and the ejection fraction was dropped to 15%. Troponin-I levels were elevated. The ECG was abnormal. Serum electrolytes analysis was abnormal. The patient was treated with inotropic agent, vasopressor, antibiotics, antiplatelets, anticoagulants, statins and diuretics. The ejection fraction was improved to 55% and the patient was discharged in a stable condition.

KEYWORDS: Myocarditis, Cardiac Output, Ejection Fraction, Troponin

I. CASE PRESENTATION:

HISTORY OF PRESENT ILLNESS:

A 25-years-old male presents to the hospital with the chief complaints of epigastric pain and dyspnoea on exertion since 3 days. History of fever with chills (on & off) since 3 months and worsened since 3 days, 4 episodes of vomitings, neck pain, generalized weakness, diarrhoea(4-5 episodes). On Day 1 Patient was hypotensive(80/40 mmHg) and the BP was normalisedto 100/80 mmHg with Inj. NORAD @14ml/hr and Dopamine infusion – 4ml/hr, patient underwent CBP, LFT, RFT, 2D Echo, ECG, Viral markers and Serum Electrolytes. On Day 2 patient was drowsy and experienced chest pain (Troponin-I levels were elevated), sweating+, peripheries cold, Ejection fraction was 15% and treated with Antibiotics, Anti-platelets, Anti-coagulants, Statins and Diuretics. Based on the above investigations patient provisionally diagnosed as viral pyrexia with Thrombocytopenia with cardiogenic shock with severe LVD with DCMP. On Day 3 chest pain decreased and he underwent Urine examination, ECG and 2D Echo. On Day 4 Based on the evidences patient was finally diagnosed as Myocarditis with Cardiogenic shock with IWMI. On Day 5, the Ejection fraction was increased to 33%. On Day 7, patient was stable, vitals were normal, Ejection fraction was increased to 55% and doctor advised angiogram and the report was normal, patient got discharged.

PAST MEDICAL HISTORY:
H/o Electric shock injury (7 years back)
FAMILY HISTORY:

No significant family history.

SOCIAL HISTORY:

Patient is occasionally alcoholic since 3 years.

ALLERGIES:

No known medicine, food and environmental allergies.

GENERAL EXAMINATION:

Pt – c/c

Dehydrated

Temp – afebrile

BP – 80/40 mmHg

PR – 130 bpm

RR – 20 /min

CVS – S1+S2+

RESP – BAE +

CNS – NAD

SPO2 – 98%

P/A – tenderness+

GRBS – 99mg/dl

CBG – 130 mg/dl ↑

LABORATORY INVESTIGATIONS:

CBP

Hb – 14.5 gm%→15.3gm% (12-14gm%)

RBC – 4.8 million/cmm→5.1million/cmm (4-6M/cmm)

WBC – 17,500cells/cmm (4,000 – 11,000)

PLT – 1.7 lakhs/cells→2.29 lakhs/cells (1.4 lakhs – 4.4lakhs)

CRP: 41mg/L

RENAL FUNCTION TESTS:

Serum creatinine – 1.0 mg/dl (0.7 – 1.2 mg/dl)

Blood urea – 36 mg/dl→28mg/dl (7-30 mg/dl)

URIC ACID:5.0mg/dl

CUE:

Colour – pale yellow

Appearance – clear

pH – Acidic

Glucose – ++

Pus cells – 6-8 /hpf

Epithelial cells – 2-4 /hpf

ANALYSIS OF SERUM ELECTROLYTES:

Sodium -139mmol/l

Potassium -4.8mmol/l

Chloride - 103mmol/l

LIVER FUNCTION TEST:

Total bilirubin: 0.7 mg/dl

Direct bilirubin: 0.3 mg/dl

SGPT: 29U/L

SGOT: 75U/L

ALP: 56U/L

Total proteins: 5.8 gm/dl

Albumin: 3.0 gm/dl

Globulin: 2.8 gm/dl

A/G ratio: 1.0:1

VIRAL MARKERS:

Hepatiits-C virus: Negative

HBsAg: Negative

HIV-I/II: Negative

Malaria parasite: negative

Dengue IgM Antibody: Positive

CARDIAC BIOMARKER:

Troponin-I: 3.05 ng/dl (0-0.30 ng/dl) ↑…..

SCANS:-

2D ECHO

Dilated LVD

Global LV hypokinesia

Severe LVD (EF-15%) → improved to 35% → improved to 55%

Grade-3 diastolic dysfunction

Mild MR, IVC congested.

ECG

Sinus tachycardia

Right atrial enlargement

Nonspecific intraventricular conduction delay

Anterolateral infarct

Acute MI.

ASSESSMENT:

Based on the Physical, General examination, Laboratory findings and Scan evidences, the patient was diagnosed with **Myocarditis with Cardiogenic Shock with IWMI.**

TREATMENT:

S.no	DRUGS	DOSE	FRQY	R.O.A	D1	D2	D3	D4	D5	D6
1.	INJ.ZOSTUM	1.5GM	IV	BD	✓	✓	✓	✓	✓	✓
2.	TAB.DOXY	100MG	PO	BD	✓	✓	✓	✓	✓	✓
3.	TAB.ECOSPRIN	325MG ↓ 75MG	PO	STAT ↓ OD	✓	✓	✓	✓	✓	✓
4.	TAB.AXCER	180MG ↓ 90MG	PO	STAT ↓ BD	✓	✓	✓	✓	✓	✓
5.	TAB.ATORVA	80MG ↓ 40MG	PO	STAT ↓ HS	✓	✓	✓	✓	✓	✓
6.	INJ.HEPARIN	5000IU	IV	QID	✓	✓	✓	×	×	×
7.	INJ.NORAD	5ml/hr	IV	STAT	✓	✓	✓	✓	✓	✓
8.	INJ.ZOFER	4MG	IV	SOS	✓	✓	✓	✓	✓	✓
9.	INJ.PAN	40MG	IV	OD	✓	✓	✓	✓	✓	✓
10.	TAB.OXRA	10MG	PO	OD	✓	✓	×	×	×	×
11.	TAB.ALDACTONE	25MG	PO	OD	✓	✓	✓	✓	✓	×
12.	INF.DOPAMINE	4ml/hr	IV	STAT	✓	✓	✓	✓	✓	✓
13.	SYP.LUPITUS	5ML	PO	TID	✓	✓	✓	✓	✓	✓
14.	SYP. KCL	10ML	PO	TID	✓	✓	✓	✓	✓	✓
15.	SYP.LACTULOSE	20ML	PO	HS	✓	✓	✓	✓	✓	✓
16.	TAB.DYTOR PLUS	10/50MG	PO	OD	×	×	×	×	×	✓

II. DISCUSSION:

DEFINITION: Myocarditis is a medical condition characterized by inflammation in the heart muscles, resulting in severely reduced cardiac output. Depending on the age, this unusual and potentially fatal condition presents

with a wide range of symptoms. In affluent nations, viral infections are the most frequent cause of myocarditis; however, other causes include sarcoidosis, autoimmune illnesses, giant cell myocarditis, toxins, medication responses, and bacterial and protozoal infections. Myocyte destruction from acute injury triggers the innate and humoral immune systems, which in turn leads to severe inflammation.[1]

EPIDEMIOLOGY:

Myocarditis has a global prevalence ranging from 10.2 to 105.6 per 100,000 people, with an estimated 1.8 million cases each year. This wide range of reported instances implies ambiguity about the disease's true prevalence and likely misdiagnosis.[2]

ETIOLOGY:

Many infectious and non-infectious agents, including viruses, bacteria, protozoa, fungi, toxins, cardiac involvement in systemic disorders, and physical conditions, can cause myocarditis; however, the underlying cause is frequently unknown. [9]

Pathogens, such as adenoviruses, emerge following a respiratory infection. [2]

Bacterial pathogens that cause myocarditis include Corynebacterium diphtheria, Beta-haemolytic streptococci, Meningococci, Salmonella typhior paratyphi, Borrelia burgdorferi, Mycoplasma pneumonia, and Chlamydia psittaci.[2]

Corynebacterium diphtheria infection is now less frequent in western nations, whereas it is still a serious public health problem in many impoverished countries, and may be the most common cause of myocarditis globally.[2]

Many different agents can cause toxic myocarditis. Dobutamine, phenytoin, antibiotics (e.g., ampicillin, azithromycin, cephalosporins, and tetracyclines), psychiatric medications (tricyclic antidepressants, benzodiazepines, and clozapine), recreational/illicit drugs (e.g., methamphetamine or cocaine), heavy metals (copper, lead, and arsenicals), and antineoplastic agents (e.g., anthracyclines, cyclophosphamide, 5-fluorouracil, and tyrosine kinase inhibitor) may result in causing myocarditis.[2]

Immunologic syndromes like Takayasu arteritis, Diabetes mellitus, Thyrotoxicosis may cause myocarditis. [6]

CLINICAL FEATURES:

Myocarditis can present with a wide range of symptoms, from no symptoms at all to myocardial infarction-like presentations to life-threatening illness with cardiogenic shock. During the course of the illness, chest discomfort, cardiac arrhythmias, and acute or chronic heart failure (HF) might happen.[10]some of the clinical manifestations like congestive heart failure, cardiac arrest, fever, myalgias, fatigue, left and right ventricular dysfunction, pericardial effusion are seen in the patients.[3] The most common symptoms includes chest pain, dyspnea, palpitations and fatigue. [5]

PATHOPHYSIOLOGY:[4]

Myocarditis is commonly seen as a series of three pathologically different phases. During the first phase, virus-mediated lysis destroys cardiomyocytes directly, causing cell structural disintegration and allowing the virus to enter the cells, resulting in myocyte damage and cardiac dilatation. This initial phase generally goes unreported since the innate immune response prevents the initial damage.

The second phase occurs as a result of immunological dysregulation caused by the initial cardiomyocyte injury. The first cellular and humoral immune responses may improve the outcome during phase one; nevertheless, they are accountable for the negative effect during phase two. This is partially generated by molecular mimicry, which is caused by imitated epitopes shared by the viral and cardiac antigens.

Finally, during the third phase, significant myocardial damage causes a classic appearance of dilated cardiomyopathy (DCM). Cross-reacting antibodies with autoantigens have been detected in myocarditis patients, indicating a progression to DCM.

Among the phases of myocarditis are:[8]

1. Viral entrance and replication within cardiomyocytes, resulting in acute damage and necrosis.

2. The immune system becoming activated.

3. An immunological response involving the production of antibodies and cytokine activity.

Thereby results in causing initial myocardial injury and chronic low-grade inflammation, which leads to long-term cardiac damage and reparative fibrosis. Inflammatory cells also release matrix-degrading proteases, all of which contribute to left ventricular (LV) dilatation and cardiac failure in phase 3.

DIAGNOSIS: [5]

1. Cardiac biomarkers (Elevated serum troponin-I, T) and natriuretic peptide levels.
2. Creatinine-kinase
3. Electrocardiogram
4. Myocardial imaging (Echocardiography, Contrast-enhanced MRI)[6]
5. Cardiac Magnetic resonance imaging (identifies edema, hyperemia, necrosis)[8]
6. Elevations may be seen in non-specific markers of inflammation in the serum, such as leucocyte count, C-reactive protein, and erythrocyte sedimentation rate. [9]

TREATMENT:[4]

Immunosuppressive therapy-Glucocorticoids (both oral & IV)-Prednisone, Methylprednisolone

Intravenous immunoglobulins (Mycophenolate, Infliximab)

Diuretics

β-blockers

Angiotensin-converting enzyme-inhibitors

Angiotensin II receptor blockers

Alemtuzumab (anti-CD52 antibody), abatacept (a CTLA-4 agonist)

Clinical remission may result after anti-T-lymphocyte-based (antithymocyte globulin-anti CD3 antibody) and calcineurin inhibitor therapy.[7]

It has been reported that interferon-α and interferon-β both enhance hemodynamics and clinical outcomes in dilated cardiomyopathy and myocarditis.[6]

III. CONCLUSION:

According to my case report cardiogenic shock was caused due to infection and the patient treated conservatively and recovered within 3 weeks.

REFERENCES:

[1.] Lori A. Blauwet, Leslie T. Cooper; Myocarditis; Progress in Cardiovascular Diseases Volume 52, Issue 4, January–February 2010, Pages 274-288; https://doi.org/10.1016/j.pcad.2009.11.006

[2.] AinooshGolpour, Dimitri Patriki, Paul J. Hanson, Bruce McManus, and Bettina Heidecker. 2021. "Epidemiological Impact of Myocarditis" Journal of Clinical Medicine 10, no. 4: 603. https://doi.org/10.3390/jcm10040603

[3.] Elisa Carniel, Gianfranco Sinagra, RossanaBussani Andrea Di Lenarda, Bruno Pinamonti, GerardinaLardieri, FurioSilvestri* Fatal myocarditis: morphologic and clinical features, Department of Cardiology, *Department of Pathology, University of Trieste, Trieste, Italy

[4.] Robert Dennert, Harry J. Crijns, Stephane Heymans, Acute viral myocarditis, *European Heart Journal*, Volume 29, Issue 17, September 2008, Pages 2073–2082, https://doi.org/10.1093/eurheartj/ehn296

[5.] Nicolas Palaskas, Juan Lopez- Mattei, Jean Bernard Durand, Cezar Iliescu and Anita Deswal; Immune Checkpoint Inhibitor Myocarditis: Pathophysiological Characteristics, Diagnosis, and Treatment; Journal of the American Heart Association. 2020;9:e013757; Originally published21 Jan 2020https://doi.org/10.1161/JAHA.119.013757

[6.] Jared W.Mangani and G.WilliamDec;Current Trends in Diagnosis and Treatment Originally published14 Feb 2006https://doi.org/10.1161/CIRCULATIONAHA.105.584532Circulation. 2006;113:876–890

[7.] EnricoAmmirati,Maria Frigerio,Eric D. Adler,Cristina Basso,David H. Birnie, MichelaBrambatti,Matthias G. Friedrich,Karin Klingel,JukkaLehtonen,Javid J. Moslehi,PatriziaPedrotti,Ornella E. Rimoldi,Heinz-Peter Schultheiss,Carsten Tschöpe,Leslie T. Cooper Jr andPaolo G. CamiciOriginally published12 Nov 2020https://doi.org/10.1161/CIRCHEARTFAILURE.120.007405Circulation: Heart Failure. 2020;13:e007405Management of Acute Myocarditis and Chronic Inflammatory Cardiomyopathy An Expert Consensus Document.

[8.] Guglin, M., Nallamshetty, L. Myocarditis: Diagnosis and Treatment. *Curr Treat Options Cardio Med* **14**, 637–651 (2012). https://doi.org/10.1007/s11936-012-0204-7

[9.] Heinz-Peter Schultheiss, Uwe Kühl, Leslie T. Cooper, The management of myocarditis, *European Heart Journal*, Volume 32, Issue 21, November 2011, Pages 2616–2625, https://doi.org/10.1093/eurheartj/ehr165

[10.] Kindermann, I, Barth, C, Mahfoud, F. et al. Update on Myocarditis. J Am CollCardiol. 2012 Feb, 59 (9) 779–792. https://doi.org/10.1016/j.jacc.2011.09.074