Case Study

Diphtheric Myocarditis: A Case Report on ECG and Cardiac Imaging Findings with The Levels of Cardiac Enzymes in A 17-Year-Old

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ABSTRACT

Background: Diphtheria, caused by Corynebacterium diphtheriae, remains a life-threatening infectious disease in regions with low vaccination uptake. Myocarditis, a severe cardiac complication of diphtheria, is a leading cause of mortality. Despite the success of vaccines in reducing diphtheria cases, sporadic cases continue to occur, leading to significant health complications.

Objective: This case report aims to present the clinical course, electrocardiogram (ECG) and cardiac imaging findings, and serial levels of serum troponin in a 17-year-old patient with diphtheritic myocarditis, and to discuss the management and outcomes.

Methods: This study was conducted at Lady Reading Hospital, Peshawar, Pakistan. Ethical approval was obtained from the hospital’s ethical review board, and informed consent was acquired from the patient and guardians. A comprehensive diagnostic workup was performed, including clinical examination, laboratory tests, ECG, transthoracic echocardiography (TTE), and cardiac magnetic resonance imaging (MRI). The patient’s vital signs, blood pressure, pulse rate, and jugular venous pressure were recorded. Laboratory tests included leukocyte count, erythrocyte sedimentation rate (ESR), and serial serum troponin measurements. ECG findings were documented, and TTE was used to assess left ventricular ejection fraction (LVEF). Cardiac MRI was performed to identify myocardial edema and delayed enhancement. Treatment included intravenous diuretics, ACE inhibitors, SGLT2 inhibitors, benzylpenicillin, and corticosteroids. Data were analyzed using SPSS version 25.0.

Results: The patient presented with a blood pressure of 110/70 mmHg and a pulse rate of 88 bpm. Initial laboratory tests showed a normal leukocyte count, ESR of 45 mm/hr, and troponin levels of 25 ng/ml, which decreased to 21.85 ng/ml and then to 8 ng/ml. ECG revealed ST segment depressions in the precordial leads. TTE showed an initial LVEF of 50%, declining to 35% and then to 28% within a week. Post-treatment, LVEF improved to 44% and subsequently to 55% at four weeks. Cardiac MRI indicated myocardial edema in the apical anterior and apical septal walls, with intramyocardial and subepicardial delayed enhancement.

Conclusion: Diphtheritic myocarditis can lead to rapid and severe deterioration of cardiac function. Early diagnosis and aggressive treatment, including the use of advanced imaging techniques and comprehensive medical therapy, can result in significant improvement in cardiac function and patient outcomes. Public health initiatives to improve vaccination coverage are essential to prevent such life-threatening complications.

Keywords: Diphtheria, myocarditis, diphtheritic myocarditis, cardiac imaging, serum troponin.
INTRODUCTION

Diphtheria, an infectious disease caused by Corynebacterium diphtheriae, remains a significant public health concern in regions with suboptimal vaccination coverage. Despite the widespread success of vaccination programs in reducing the incidence of diphtheria globally, outbreaks continue to occur in areas where vaccine uptake is insufficient, leading to severe and potentially fatal complications (1, 2). Among these complications, myocarditis stands out as the most fatal, contributing significantly to diphtheria-related morbidity and mortality. Myocarditis, characterized by inflammation of the heart muscle, can rapidly progress to heart failure and death if not promptly and adequately managed (3-5). The pathophysiology involves the diphtheria toxin directly damaging cardiac myocytes and the cardiac conduction system, resulting in both systolic and diastolic dysfunction (1, 3, 5).

The clinical presentation of diphtheritic myocarditis is often insidious, with symptoms such as chest pain, dyspnea, and orthopnea, progressing rapidly as the disease worsens. Electrocardiographic (ECG) abnormalities, including ST segment depression, T-wave inversion, and various conduction disturbances, are common and can aid in early diagnosis (6, 7). Echocardiographic evaluation typically reveals decreased left ventricular ejection fraction (LVEF) and other indicators of impaired cardiac function (7-9). Advanced imaging techniques such as cardiac magnetic resonance imaging (MRI) provide detailed insights into myocardial involvement, identifying areas of edema and delayed enhancement indicative of myocarditis (3). Serial measurements of cardiac biomarkers, particularly troponin, are crucial for assessing the extent of myocardial injury and guiding treatment strategies (8-11).

This case report discusses the clinical course, diagnostic findings, and management of a 17-year-old male with diphtheritic myocarditis. The patient presented with a constellation of symptoms including chest pain, nausea, vomiting, and dyspnea, which rapidly progressed to severe cardiac dysfunction. Initial assessments revealed significant elevations in troponin levels and characteristic ECG changes (10-12). Transthoracic echocardiography showed a marked decline in LVEF, while cardiac MRI confirmed the presence of myocardial edema and delayed enhancement. The treatment regimen, comprising intravenous diuretics, ACE inhibitors, SGLT2 inhibitors, benzylpenicillin, and corticosteroids, led to significant clinical improvement, as evidenced by the recovery of LVEF over subsequent weeks (13).

Despite the successful management of this case, the challenges in diagnosing and treating diphtheritic myocarditis underscore the need for heightened clinical vigilance and timely intervention. The variability in clinical presentation and rapid progression of the disease necessitate a comprehensive diagnostic approach, incorporating ECG, echocardiography, cardiac MRI, and serial biomarker assessments (10, 12). Furthermore, this case highlights the critical importance of public health initiatives to improve vaccination coverage, thereby preventing the occurrence of diphtheria and its associated complications. Future research should focus on optimizing treatment protocols and developing strategies to enhance early diagnosis and intervention in diphtheritic myocarditis (2, 5, 8, 11).

MATERIAL AND METHODS

This case report was conducted at Lady Reading Hospital, Peshawar, Pakistan, in collaboration with multiple institutions. Ethical approval was obtained from the hospital’s ethical review board, and the study adhered to the principles outlined in the Declaration of Helsinki. Informed consent was secured from the patient and his guardians for participation and publication of the case details.

The patient, a 17-year-old male, presented with a history of chest pain, nausea, vomiting, recurrent episodes of epistaxis, dyspnea on exertion, and orthopnea. He had a recent history of diphtheria, treated with anti-diphtheria serum at a tertiary care hospital. Comprehensive clinical and diagnostic evaluations were performed upon admission, including physical examination, laboratory tests, ECG, transthoracic echocardiography (TTE), and cardiac magnetic resonance imaging (MRI).

Vital signs were recorded, showing a blood pressure of 110/70 mmHg and a pulse rate of 88 bpm. Physical examination noted elevated jugular venous pressure and signs of cardiomegaly. Laboratory tests included leukocyte count, erythrocyte sedimentation rate (ESR), and serial serum troponin measurements. The initial troponin level was 25 ng/ml, which decreased to 21.85 ng/ml and subsequently to 8 ng/ml. The ECG demonstrated ST segment depressions in the precordial leads, suggesting myocardial ischemia. TTE was utilized to assess cardiac function, revealing global hypokinesia of the left ventricle (LV) with an initial left ventricular ejection fraction (LVEF) of 50%, which declined to 35% and further to 28% over one week. A cardiac MRI was performed to evaluate myocardial involvement, showing myocardial edema in the apical anterior and apical septal walls with intramyocardial and subepicardial delayed enhancement. T1 color mapping values of >1150 m/s were observed, consistent with myocarditis.

The patient was treated with intravenous diuretics, ACE inhibitors, SGLT2 inhibitors, and benzylpenicillin. Additionally, corticosteroids were administered due to the fulminant nature of the myocarditis. Treatment led to significant clinical improvement, with LVEF increasing to 44% after one week and 55% at four weeks, as shown by follow-up echocardiography.
Data collection was systematic, including detailed clinical history, physical examination findings, laboratory results, ECG, echocardiographic measurements, and cardiac MRI findings. All data were anonymized before analysis to maintain patient confidentiality. Data analysis was conducted using SPSS version 25.0, ensuring robust statistical evaluation of clinical and diagnostic data.

The multidisciplinary team involved in this case report followed stringent protocols to ensure the accuracy and reliability of the findings. This comprehensive approach provided valuable insights into the management of diphtheritic myocarditis in a clinical setting. The study highlighted the importance of early diagnosis and aggressive treatment in improving outcomes for patients with severe cardiac complications of diphtheria (1, 2, 3, 4).

**RESULTS**

The case of the 17-year-old male with diphtheritic myocarditis was thoroughly evaluated and monitored over time, with significant findings documented in both numerical and descriptive formats. The patient’s clinical presentation, laboratory findings, ECG changes, echocardiographic measurements, and cardiac MRI results are detailed below.

The patient presented with chest pain, nausea, vomiting, recurrent epistaxis, dyspnea on exertion, and orthopnea. Initial vital signs recorded a blood pressure of 110/70 mmHg and a pulse rate of 88 bpm. Elevated jugular venous pressure and cardiomegaly were noted on physical examination and radiographic assessment.

**Table 1: Initial ECG**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Initial Value</th>
<th>Follow-up Value 1</th>
<th>Follow-up Value 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leukocyte Count</td>
<td>Normal</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>ESR (mm/hr)</td>
<td>45</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Troponin (ng/ml)</td>
<td>25</td>
<td>21.85</td>
<td>8</td>
</tr>
</tbody>
</table>

The initial ECG showed ST segment depressions in the precordial leads.

**Table 2: Measurement Week1-4**

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Initial</th>
<th>After 1 Week</th>
<th>After 4 Weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEF (%)</td>
<td>50</td>
<td>35</td>
<td>28</td>
</tr>
<tr>
<td>LVEF Post-Treatment (%)</td>
<td>-</td>
<td>44</td>
<td>55</td>
</tr>
</tbody>
</table>

Transthoracic echocardiography revealed global hypokinesia of the left ventricle with an initial ejection fraction of 50%. This declined to 35% over one week and further to 28%. After treatment, the patient's LVEF improved to 44% and then to 55% at four weeks.

Cardiac MRI showed myocardial edema in the apical anterior and apical septal walls of the left ventricle, with intramyocardial and subepicardial delayed enhancement. The T1 color mapping values of >1150 m/s indicated findings suggestive of myocarditis. The patient received intravenous diuretics, ACE inhibitors, SGLT2 inhibitors, benzylpenicillin, and corticosteroids. Following this treatment regimen, the patient showed clinical improvement with a subsequent increase in LVEF to 44% after a 7-day course of treatment and to 55% at four weeks.

The patient presented with symptoms indicative of severe cardiac involvement, consistent with diphtheritic myocarditis. Laboratory findings showed elevated ESR and significantly raised troponin levels, indicating myocardial injury. The ECG revealed ST segment depressions, a common finding in myocarditis. Echocardiography demonstrated a progressive decline in left ventricular ejection fraction (LVEF), which improved significantly post-treatment. Cardiac MRI confirmed the diagnosis of myocarditis, showing characteristic myocardial edema and delayed enhancement. The comprehensive treatment regimen, including intravenous diuretics, ACE inhibitors, SGLT2 inhibitors, benzylpenicillin, and corticosteroids, led to a marked improvement in cardiac function and clinical symptoms.

**Table 3: Comprehensive Numerical Analysis**

<table>
<thead>
<tr>
<th>Time Point</th>
<th>BP (mmHg)</th>
<th>Pulse (bpm)</th>
<th>ESR (mm/hr)</th>
<th>Troponin (ng/ml)</th>
<th>LVEF (%)</th>
<th>EF (%)</th>
<th>MRI (%)</th>
<th>LV (ml)</th>
<th>EDV (ml)</th>
<th>LV (ml)</th>
<th>ESV (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial Presentation</td>
<td>110/70</td>
<td>88</td>
<td>45</td>
<td>25</td>
<td>50</td>
<td>-</td>
<td>206.06</td>
<td>145.24</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>After 1 Week (Pre-Treatment)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>21.85</td>
<td>35</td>
<td>31</td>
<td>206.06</td>
<td>145.24</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
DISCUSSION
The presented case of a 17-year-old male with diphtheritic myocarditis underscores the severe cardiac implications of diphtheria, especially in regions with inadequate vaccination coverage. Despite global efforts to control diphtheria through vaccination, areas with low immunization rates continue to experience sporadic outbreaks, leading to severe complications such as myocarditis. This case highlights the rapid and progressive nature of diphtheritic myocarditis, which, if not promptly diagnosed and treated, can result in significant morbidity and mortality (12-14).

The patient’s initial presentation with symptoms of chest pain, dyspnea, and orthopnea, alongside elevated troponin levels and characteristic ECG changes, is consistent with previous reports of diphtheritic myocarditis (3, 15-17). The rapid decline in left ventricular ejection fraction (LVEF) from 50% to 28% within a week exemplifies the acute and severe impact of the disease on cardiac function. The findings of myocardial edema and delayed enhancement on cardiac MRI provided further confirmation of myocarditis, aligning with established diagnostic criteria for myocardial involvement in diphtheria (3, 15-17).

The treatment regimen employed in this case, including intravenous diuretics, ACE inhibitors, SGLT2 inhibitors, benzylpenicillin, and corticosteroids, resulted in significant clinical improvement. This therapeutic approach, particularly the use of corticosteroids in managing fulminant myocarditis, has been supported by previous studies highlighting their role in reducing inflammation and improving outcomes (13, 18, 19). The patient’s LVEF improved to 55% over four weeks, demonstrating the effectiveness of early and aggressive intervention.

Several strengths of this case report include the comprehensive diagnostic workup and the multidisciplinary approach to treatment. The use of advanced imaging techniques such as cardiac MRI provided detailed insights into myocardial involvement, aiding in accurate diagnosis and management. Serial measurements of cardiac enzymes and echocardiographic evaluations allowed for close monitoring of the patient’s progress and response to treatment (20).

However, the study also had limitations. The findings are based on a single case, limiting the generalizability of the results. Further research involving larger cohorts is necessary to validate the observed treatment outcomes and refine therapeutic protocols for diphtheritic myocarditis. Additionally, the short follow-up period precludes a comprehensive assessment of long-term cardiac function and outcomes. Future studies should include extended follow-up to evaluate the sustained impact of treatment and potential late complications (20).

This case underscores the critical need for heightened clinical awareness and prompt management of diphtheritic myocarditis. Early diagnosis, facilitated by ECG, echocardiography, and cardiac MRI, is crucial for initiating appropriate treatment and improving patient outcomes. The significant improvement observed in this case highlights the potential for favorable outcomes with timely and aggressive intervention. Public health initiatives to improve vaccination coverage are essential to prevent diphtheria and its severe complications. Enhanced surveillance, early diagnosis, and comprehensive management strategies are vital for reducing the burden of diphtheritic myocarditis in affected regions (6, 9, 16, 19).

CONCLUSION
In conclusion, this case report provides valuable insights into the clinical course, diagnostic findings, and management of diphtheritic myocarditis. The successful treatment and significant improvement in cardiac function observed in this patient underscore the importance of early and aggressive intervention. Continued research and public health efforts are necessary to optimize management strategies and prevent the occurrence of diphtheria-related cardiac complications.

REFERENCES
Diphtheric Myocarditis in a 17-Year-Old: ECG, Imaging, and Enzyme Levels
Hayat FS, et al. (2024). JHRR:42(2): DOI: https://doi.org/10.61919/jhrr.42.1162