Evaluation of Cardiac Involvement by Echocardiography in Children with Acute Meningococcemia

by

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Summary

Mycarditis in acute meningococcemia is rare in children. Impaired cardiac contractile function may contribute to cardiovascular collapse in endotoxemia. We evaluated prospectively the clinical and echocardiographic findings in 25 consecutive children with acute meningococcemia. All of the patients had good prognostic indicators. Febrile (44%) of the 25 children had echocardiographic evidence of myocardial dysfunction. The mean LVFS of these eleven children was 0.23 ± 0.00, as compared with the mean LVFS of 0.34 ± 0.87 in the remaining children. LVFS returned to normal within 7 days in all patients. We observed minimal pericardial effusion in 4 patients (16%) that resolved within 3 days. We demonstrated that acute meningococcemia is frequently associated with myocardial dysfunction. We could not though, find the relation between myocardial dysfunction and mortality.

[Additional indexing words: Myocardial dysfunction, acute meningococcemia, echocardiography]
Introduction

Meningococcal infections usually occur between 3 months and 4 years of age [1]. Acute meningococcemia is frequently associated with cardiovascular collapse, and the mortality ranges from 20% to 70%, even with adequate therapy [2,3]. The cause of death is unknown and the pathogenesis of meningococcal shock is controversial. Shock itself may cause myocardial ischemia and dysfunction.

Pericarditis and myocarditis in acute meningococcemia occur fairly frequent in adults, but is rare in children [1]. Histologic evidence of myocardial inflammation and cellular damage has been reported with meningococcemia [4,5]. Cardiac involvement may be related to the severity of endotoxemia may produce a fatal outcome in meningococcal septicemia [6].

The purpose of this study was to investigate the incidence and prognosis of myocardial dysfunction and pericarditis in acute meningococcemia by echocardiography. Furthermore, we evaluated the association between myocardial dysfunction and cardiovascular collapse.

Materials and Methods

We evaluated prospectively the clinical and echocardiographic findings in 25 consecutive patients with meningococcemia admitted to Dr. Sami Ulus Children's Hospital over 6 months.

The clinical diagnosis of meningococcemia was based on the acute onset of fever, lethargy, petechiae or purpura, and signs of meningeal irritation. The diagnosis was confirmed in 11 of the 25 patients by positive culture of Neisseria meningitidis from the blood or cerebrospinal fluid. Neisseria meningitidis was documented by Gram stain of cerebrospinal fluid in 7 patients. A presumptive diagnosis of meningococcemia was made in 7 patients with sterile cultures who had received antibiotic before cultures were obtained. We could not make serologic typing of the Neisseria meningitidis.

The clinical criteria of myocarditis were tachycardia, gallop rhythm, cardiomegaly, non-specific evaluations or depression of ST segment and low QRS voltage. The criteria of cardiovascular collapse with meningococcemia were elevated central venous pressure (≥12 mm Hg) associated with tachycardia, dyspnea, hypotension, decreased urinary output, and a gallop rhythm.

Telecardiograms (TELE) and electrocardiograms (ECG) were evaluated. Echocardiogram was carried out by Toshiba Sonolayer TSA-250A using 3.7 and 5 MHz transducers. Echocardiography was performed in 48 occasions in 25 patients. There were 11 patients with myocardial dysfunction (group I) and 14 patients (group II) without myocardial dysfunction. Interventricular septum and posterior or wall of the left ventricle were measured both in systole and diastole and the results were as follows; left ventricular internal diastolic dimension (LVIDd), left ventricular internal systolic dimension (LVIDs), left ventricular ejection fraction (LVEF) and left ventricular fractional shortening (LVFS). All values were averaged for at least three cardiac cycles. The LVFS is a measurement of left ventricular function and relatively independent of age and heart rate. The normal mean (±SD) of LVFS is 0.56 ± 0.04. A LVFS of 0.28 or less (2 SD below the mean) was defined as depressed myocardial dysfunction. Data were compared between groups using the student t test. The criterion for significance was chosen as P < 0.05.

Results

The mean age of the patients with myocardial dysfunction (group I) was 4.02 ± 0.73 years (Table I) and 54% of this group were males. The mean age of the children with normal myocardial function (group II) was 4.55 ± 1.25 years (p > 0.05) and 46% of this group were males. The onset of symptoms prior to admission was <24 hour in 20 children, and >24 hours in five. Physical examination revealed fever (93%), petechiae or purpura (80%), and meningeal irritation (84%). All patients with meningococcemia had meningitis. Meningococcal shock was diagnosed in three patients, two of whom had associated depressed myocardial function. All patients were given intravenous penicillin and chloramphenicol. Three children with meningococcal shock were heparinized during the first 24 to 48 hours of hospitalization and were given corticosteroid and dopamine.

Echocardiographic results of myocardial function:

Eleven (44%) of the 25 children had myocardial dysfunction by echocardiographic criteria (group I). The mean ± SD LVFS in group I was 0.23 ± 6.00. Contrastingly, normal myocardial function was found in 14 (56%) of the 25 children in group II (Table I). The LVFS in group II was 0.34 ± 8.87 and three was a significant difference between the two groups (p < 0.05). The LVIDd in group I (32.60 ± 1.19) was not significantly different from that in group II (34.37 ± 1.85), and both were within the normal range. Echocardiographic examination was performed as early as possible. LVFS returned to normal within 2 days in one child. LVFS was normal within 3 days in eight children. However, the LVFS remained depressed in two children as long as 7 days (Table I). We observed minimal pericardial effusion in 4 patients (16%) by echocardiography. Pericardial effusion resolved within 3 days.

The telecardiogram revealed the cardiac size to be slightly enlarged in 4 children and moderately enlarged in 3 children. The ECG demonstrated voltage supression and non-specific elevation or depression of ST segment and sinusal tachycardia in 10 patients. One child with myocardial dysfunction developed sinusoidal bradycardia.

Peripheral WBC count was ≥ 10,000/μl in 23 patients. Two children with myocardial dysfunction had lower peripheral WBC count (< 10,000/μl). Cerebrospinal fluid WBC count was 5 cells/mm³ in all patients. Abnormal prothrombin time (PT) and partial thromboplastin time (PTT) values were present in three patients with cardiovascular collapse. All patients were discharged from hospital within a week.

Discussion

The pathophysiologic basis of myocardial dysfunction in children with acute meningococcemia is unknown. Experimental studies in animals have demonstrated decreased contractility, decreased velocity of contraction, and altered ventricular compliance [7]. Release of myocardial depressant factor has been demonstrated to have a strong correlation with the depressed myocardial function seen in endotoxicemia [8]. Myocardial dysfunction may be a significant cause of mortality in children. But the incidence of myocarditis in children remains controversial. Because of the variety of manifestations of infectious myocarditis, the diagnosis is
Table I. Echocardiographic measurements in children with meningococcemia

<table>
<thead>
<tr>
<th>Age</th>
<th>Heart rate (yrs)</th>
<th>LVDD (mm)</th>
<th>LVDS (mm)</th>
<th>LVEF (mm)</th>
<th>LVFS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>6.0 ± 0.73</td>
<td>16.35 ± 4.02</td>
<td>32.60 ± 1.19</td>
<td>24.78 ± 0.90</td>
<td>0.49 ± 0.92</td>
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<td>(n = 11)</td>
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<tr>
<td>Group II</td>
<td>4.55 ± 1.25</td>
<td>116.5 ± 4.09</td>
<td>34.37 ± 1.05</td>
<td>22.80 ± 1.40</td>
<td>0.64 ± 1.25</td>
</tr>
<tr>
<td>(n = 14)</td>
<td></td>
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</tbody>
</table>

LVDD = Left ventricular end diastolic dimension
LVDS = Left ventricular end systolic dimension
LVEF = Left ventricular ejection fraction
LVFS = Left ventricular fractional shortening

Table II. Left ventricular shortening fraction in each child serial determined by echocardiography

<table>
<thead>
<tr>
<th>Patients</th>
<th>LFVS</th>
<th>1st day</th>
<th>2nd day</th>
<th>3rd day</th>
<th>7th day</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td>0.24</td>
<td>0.26</td>
<td>0.27</td>
<td>0.36</td>
</tr>
<tr>
<td>2</td>
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<td>0.25</td>
<td>0.27</td>
<td>0.28</td>
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<td>3</td>
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<td>0.37</td>
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<tr>
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<td>0.21</td>
<td>0.28</td>
<td>0.41</td>
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<td>5</td>
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<td>0.33</td>
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</tr>
<tr>
<td>6</td>
<td></td>
<td>0.22</td>
<td>0.27</td>
<td>0.36</td>
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<tr>
<td>7</td>
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<td>0.23</td>
<td>0.28</td>
<td>0.38</td>
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<tr>
<td>8</td>
<td></td>
<td>0.26</td>
<td>0.28</td>
<td>0.37</td>
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<tr>
<td>9</td>
<td></td>
<td>0.24</td>
<td>0.26</td>
<td>0.36</td>
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<tr>
<td>10</td>
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<td>0.23</td>
<td>0.28</td>
<td>0.38</td>
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<tr>
<td>11</td>
<td></td>
<td>0.25</td>
<td>0.27</td>
<td>0.36</td>
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</tr>
</tbody>
</table>

LVFS = Left ventricular fractional shortening

Echocardiography in myocarditis has proven to be a sensitive technique for detecting the degree of myocardial depression. The motion of the interventricular septum and of the ventricular walls may be poor and abnormal and the ejection fraction may be decreased [15,16]. There is nearly always dilatation of the heart chambers, usually the left ventricle [15]. None of our patients demonstrated increased LVDD and LVSD. Boucek et al. also reported that LVDD was normal with myocardial dysfunction. Transient increases in left ventricular wall thickness have been found in myocarditis, perhaps representing inflammatory myocardial edema [17,18]. In our study, increased left ventricular wall thickness was not observed at all. With these findings, we concluded that our patients had mild myocarditis. Finally, we demonstrated that acute meningococcemia was frequently associated with myocardial dysfunction. But we couldn't determine the relation between myocardial dysfunction and the cardiovascular collapse.

Meningococcal pericarditis occurs fairly frequent in adults, but rarely in children (1). Pericarditis is usually benign although pericardiocentesis is occasionally required [1,11,12]. Our results showed that the pericardial effusion was minimal and resolved within three days. But sterile pericardial effusion develops late in the course of the disease, suggesting a hypersensitivity phenomenon [12,13]. Nichols et al. reported an unusual case of constrictive pericarditis that developed 13 years after an episode of meningococcal pericarditis [14].

REFERENCES


ORIGINAL ARTICLE

**Serum IgG, and IgM Levels in Children with Febrile Convulsions**

by

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Abstract

At the Dr. Sami Ulus Children's Hospital Ankara, Turkey, 20 patients, twelve being between 6 months-2 year old and eight being between 2-4 years, with their first febrile convulsions (FC) were examined for serum IgG, IgA, IgM levels during the period of March 1989-July 1989. Twenty healthy children were used as controls, seven being between 6 months-2 years old and thirteen being between 2-6 years. The serum IgG, IgA and IgM levels of the patients between 6 months-2 years were 805.000 + 307.8984 mg/dl, 49.7167 + 27.9807 mg/dl and 155.1833 + 62.9696 mg/dl respectively. The serum IgG, IgA and IgM levels of the patients between 2-4 years were 989.1250 + 314.5359 mg/dl, 92.6125 + 34.9663 mg/dl and 159.8750 + 45.6647 mg/dl respectively. The mean IgA levels of the 12 FC patients between 6 months-2 years were 49.7167 + 27.9807 mg/dl and the mean level of IgA in the age matched control group was 81.0427 + 31.3551 mg/dl and the difference between them was statistically significant (p < 0.005).

We conclude that FC under 2 years of age is associated with low serum IgA levels.

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