ORIGINAL ARTICLE

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. Evelen (44%) of the 25 children had echocardiographic evidenceof myocardial dysfunction. The mean LVFS in these eleven children was 0.23 ± 6.00, as compared with the mean LVFS of 0.34 ±8.87 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]

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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, petechia or purpura, and signs of meningeal irritation. The diagnosis was confirmed (in 11 of the 25 patients by positive culture of Neisseria meningitis from the blood or cerebrospinal fluid. Neisseria meningitidis was documented by Gram stain of te 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 rythm, cardiomegaly, non-specific evaluations or depression of ST sagment 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 wall of the left ventricle were measured both in systole and diastole and the results were as follows; left ventricle internal diastolic dimension (LVIDD), left ventricular internal systolic dimension (LVISD), left ventricular ejection fraction (LVEF) and left ventricle 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 0.36 ± 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 (92%), petechia 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 fisrt 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 II). 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 sinusal bradycardia.

Peripheral WBC count was ≥ 10,000/ml in 23 patients. Two children with myocardial dysfunction had lower peripheral WBC count (< 10,000/ml). Cerebrospinal in fluid WBC count was = 5 cells/mm^3 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 endotoxemia [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*

Age	Heart rate (yrs)	LVIDD (bpm)	LIVDS (mm)	LVEF (mm)	LVFS	
Group I (n = 11)	4.02 ± 0.73	146.36 ± 4.02	32.60 ± 1.19	24.78 ± 0.90	0.49 ± 9.24	0.23 ± 6.00
Group II (n = 14)	4.55 ± 1.25	116.5 ± 4.09	34.37 ± 1.85	22.80 ± 1.40	0.64 ± 1.25	0.34 ± 8.87
		p < 0.05			p < 0.05	p < 0.05

LVIDD = Left ventricular end diastolic dimension

LVIDS = Left ventricular end systolic dimensiion

LVEF = Left ventricular efectional fraction

LVFS = Left ventricular fractional shortening

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

Patients	LFVS			
	1st day	2nd day	3rd day	7th day
1	0.24	0.26	0.27	0.36
2	0.23	0.27	0.28	0.41
3	0.25	0.29	0.37	
4	0.21	0.28	0.41	
5	0.28	0.33	-	
6	0.22	0.27	0.36	
7	0.23	0.28	0.38	
8	0.26	0.28	0.37	
9	0.24	0.26	0.36	
10	0.23	0.28	0.38	
11	0.23	0.27	0.36	

LVFS = Left ventricular fractional shortening

often made on autopsy [1]. Boucek et al. [9] had evaluated the clinical, echocardiographic and hemodynamic findings in 12 children with acute meningococcemia. They reported that myocardial dysfunction was found in 7 children (58%) and three (43%) of these cases died [9]. Therefore, they emphasized that myocardial dysfunction might be a significant cause of mortality in children with meningococcemia. In our study, the incidence of myocardial dysfunction in acute meningococcemia was 44% . The course of myocarditis was benign and LVFS returned to normal within 7 days and none of the children died. Investigators reported that the myocardial dysfunction was demonstrated to peak at 36 to 48 hours and return to normal slowly over 7 - 10 days [8.10]. All of our patients with meningococcemia had meningitis and high WBC counts (≥ 10,000/mm^3) except in 3 cases. Two children with low WBC counts had myocardial dysfunction. Benign course of myocarditis was probably due to good prognostic factors. Two of the 11 children with myocardial dysfunction had meningococcal shock. Therefore, we could not find 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].

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 LVIDD and LVISD. Boucek et al. also reported that LVIDD 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 with acute meningococcemia and cardiovascular collapse. If the children with acute meningococcemia have good prognostic indicators, we can say that the course of myocarditis may be benign. The depression of myocardial contractility and pericarditis appear early in endotoxemia [8.10.19]. Therefore, we emphasized the importance of echocardiogarphy to be performed as early as possible to investigate the degree of myocardial dysfunction in acute meningococcemia.

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