



Original Research Article

Assessment of Myocardial Function by Echocardiogram in Neonatal Sepsis in Basrah

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Abstract: This case-control study was conducted to assess myocardial function in term neonates with sepsis over a ten-month period from June 2020 to March 2021 at Basrah Maternity and Children Hospital. A total of 105 neonates aged 1–28 days with gestational ages of 37–41 weeks were enrolled. Forty-six neonates with sepsis were recruited from the neonatal care unit, and 59 relatively healthy neonates served as controls. Both groups were matched for age, sex, and gestational age. Blood investigations including complete blood count, C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and blood culture were performed in all participants. Chest X-ray and echocardiography—including ejection fraction (EF), fractional shortening (FS), Tricuspid Annular Plane Systolic Excursion (TAPSE), Mitral Annular Plane Systolic Excursion (MAPSE), and Tei index—were performed within the first two days of admission or follow-up. The septic group had a mean age of 9.33 ± 5.61 days, mean weight 3100 ± 505 g, and a male-to-female ratio of 1:1. Illiterate mothers were more frequent in the septic group (15.22%) compared to controls (3.38%) with $p < 0.05$. Echocardiographic abnormalities were observed in 86.95% of septic neonates. Three parameters were significantly affected: TAPSE (0.68 ± 0.23 cm), MAPSE (0.57 ± 0.21 cm), and Tei index (0.5 ± 0.14), with Tei index being the most commonly affected (67.4%). MAPSE and TAPSE abnormalities were observed in 58.7% and 15.22%, respectively. Echocardiographic changes were not significantly associated with demographic or laboratory parameters, except for CRP in relation to TAPSE. In conclusion, a high proportion of neonates with sepsis exhibited myocardial dysfunction, predominantly affecting the left ventricle. Early cardiac evaluation in neonatal sepsis is essential to improve outcomes and reduce mortality.

Keywords: neonatal sepsis, myocardial dysfunction, echocardiography, Tei index, TAPSE, MAPSE.

INTRODUCTION

Sepsis is a systemic inflammatory process resulting from an inappropriate or excessive host response to infection. In neonates, an immature immune system limits the ability to control infection effectively, increasing the risk of systemic inflammatory response syndrome (SIRS) and progression to septic shock and multiorgan dysfunction [1]. The heart is among the organs most commonly affected in neonatal sepsis, and cardiac dysfunction is a major contributor to mortality. Early identification of myocardial involvement is essential, with echocardiography being the most reliable non-invasive method to assess cardiac function [2]. Neonatal sepsis is a systemic illness caused by bacteria, viruses, or fungi, resulting

in hemodynamic changes and clinical manifestations within the first month of life [3]. It is classified as early-onset (symptoms within 7 days, sometimes 72 hours) or late-onset (≥ 7 days), with differing risk factors. Early-onset sepsis is often associated with maternal and perinatal risk factors, including prolonged rupture of membranes, maternal fever, prematurity, bacteriuria, and prior delivery of infants affected by group B streptococcus (GBS) [5]. Late-onset sepsis is usually acquired horizontally from the hospital environment, family, or healthcare providers, with coagulase-negative staphylococci being the most common pathogens, followed by *Staphylococcus aureus* and GBS [6].

The incidence of neonatal sepsis varies globally. Developed countries report an incidence of approximately 0.7%, responsible for 15% of neonatal deaths, whereas developing countries report higher rates of 5–10%, with mortality two to three times higher [7]. Studies in low- and middle-income countries suggest mortality due to neonatal sepsis of 17.6% between 2009–2018 [8]. In Basrah, Iraq, infections accounted for 10.9% of early neonatal deaths and 45.6% of late neonatal deaths in 2020 [9]. Male infants are reported to have higher sepsis incidence than females [10].

Early-onset infections usually arise from exposure to pathogens in the birth canal, predominantly GBS and *Escherichia coli* [11], or from intrauterine infections such as chorioamnionitis [12], [13]. Late-onset infections typically result from hospital-acquired sources [14]. The infectious process triggers an inflammatory response mediated by white blood cells. If uncontrolled, this leads to diffuse tissue damage, organ dysfunction, and progression along the septic cascade, encompassing sepsis, septic shock, and multiple organ dysfunction syndrome (MODS) [15]–[18].

Clinical manifestations in neonates are often nonspecific due to immature immunity. Presentations range from localized symptoms to multiorgan involvement, including neurologic (convulsions, lethargy, bulging fontanel), respiratory (tachypnea, grunting, cyanosis), cardiovascular (poor perfusion, weak pulse), gastrointestinal (poor feeding, jaundice), dermatologic, musculoskeletal, and temperature abnormalities [19]–[21]. Laboratory tests support diagnosis but no single test is definitive [22]. Key investigations include complete blood count (CBC) with evaluation of WBC, neutrophil indices, and platelet counts [23], [24], microorganism isolation via blood, CSF, urine, or other cultures [25], [26], and inflammatory biomarkers including CRP, ESR, procalcitonin, cytokines, and others [27]–[29]. Additional assessments may include arterial blood gas, renal and hepatic function tests, and chest radiography if respiratory involvement is suspected [4], [30].

Sepsis-induced SIRS can lead to pulmonary complications such as ARDS and acute hypoxic respiratory failure, immune system suppression (immunoparalysis), central nervous system injury (periventricular leukomalacia), cardiovascular dysfunction, endocrine disturbances, and acute kidney injury, all of which contribute significantly to morbidity and mortality [30]–[32].

Neonatal sepsis may cause reversible intrinsic systolic and diastolic dysfunction in both ventricles. Prevalence of myocardial depression in pediatric sepsis ranges from 23–71%, reaching higher rates in

septic shock [34]. Neonatal cardiac response differs from adults due to structural, genetic, metabolic, and cellular differences, including altered calcium handling and excitation–contraction coupling [10], [35]. Mechanisms of dysfunction include elevated myocardial depressant factors (TNF- α , IL-1 β), endotoxin-mediated myocyte injury, calcium homeostasis disturbances, nitric oxide dysregulation, and oxidative stress leading to mitochondrial dysfunction [35], [36], [42]. Pediatric septic shock is often hypodynamic, contrasting with hyperdynamic adult shock, and may be complicated by persistent pulmonary hypertension, increasing right ventricular workload [37]. Anatomical factors, including reduced left ventricular mass and altered collagen ratios, limit neonatal myocardial compliance and contractile reserve [38], [39]. Inflammatory mediators (IL-1, IL-4, IL-6, IL-8, IL-10, IFN- γ , TNF- α , C5a) further impair cardiac function [40]. Circulatory changes such as venous pooling, microcirculatory imbalance, and altered preload/afterload also contribute to myocardial depression [36]. Nitric oxide can exert both protective and deleterious effects on contractility and vascular tone, depending on the context [35].

Sepsis-induced myocardial dysfunction is often reversible within 7–10 days of illness onset, with improvement following resolution of inflammation. Supportive measures including optimized preload and inotropes are essential to maintain tissue perfusion. Even in the absence of shock, neonates with sepsis may exhibit subclinical cardiac dysfunction, and blood pressure alone is often insufficient to assess systemic perfusion [34], [43].

Echocardiography is a first-line, non-invasive tool for evaluating cardiac anatomy, physiology, and hemodynamics in neonates with sepsis [44]. Functional echocardiography allows assessment of myocardial performance, preload, afterload, and cardiac output, aiding early intervention. Advanced echocardiographic parameters, including tissue Doppler imaging and speckle tracking, facilitate detection of subtle cardiac dysfunction. Myocardial dysfunction in neonates with sepsis may involve left ventricular (LV) systolic dysfunction (EF, FS, MAPSE), LV diastolic dysfunction (TDI), right ventricular (RV) systolic dysfunction (TAPSE), and/or global dysfunction (MPI/Tei index, TDI, myocardial strain) [45]. Diastolic and global dysfunction are more common than systolic dysfunction. LV diastolic dysfunction is prevalent due to limited myocardial elasticity, reduced ventricular mass, and high baseline contractile state, while systolic dysfunction is often less pronounced and measured via EF and FS, which may remain normal despite early longitudinal fiber impairment [46]–[50]. MAPSE detects longitudinal fiber shortening and may reveal early LV dysfunction even when EF is normal. MPI/Tei index provides a global assessment of

systolic and diastolic function, is highly sensitive, and carries prognostic value, though it is moderately load dependent [50]. RV function assessment is challenging due to complex geometry; TAPSE measures longitudinal shortening and identifies RV dysfunction, though it is load dependent and reflects lateral wall function only [51].

This case-control study aims to assess myocardial function in neonates with sepsis compared to healthy neonates using echocardiography and to evaluate the relationship between myocardial dysfunction and selected clinical, demographic, and laboratory variables.

METHODOLOGY

Study Design and Setting

This case-control study was conducted at the Neonatal Care Unit of Basrah Maternity and Children Hospital over a ten-month period from June 2020 to March 2021. The study included 105 term neonates with postnatal ages ranging from 1 to 28 days and gestational ages between 37 and 41 weeks. Participants were recruited from both inpatient units and outpatient clinics and were divided into two groups: neonates with sepsis and healthy controls. Ethical approval was obtained, and all procedures adhered to the Declaration of Helsinki.

Study Population and Inclusion/Exclusion Criteria

The sepsis group comprised 46 neonates diagnosed based on at least two clinical signs of infection and at least two laboratory indicators of inflammation. Eligible neonates exhibited mild to moderate sepsis, requiring only supportive care such as intravenous fluids, antibiotics, and oxygen; none required mechanical ventilation, inotropes, or treatment for septic shock. Neonates with severe sepsis who did not survive until echocardiographic assessment were excluded.

The control group included 59 age- and sex-matched term neonates without clinical or laboratory evidence of infection. This group consisted of neonates with simple jaundice requiring only phototherapy, neonates with transient tachypnea of the newborn who were clinically stable at discharge, and healthy outpatients attending routine follow-up.

Exclusion criteria for both groups included major congenital malformations, such as congenital heart disease, chromosomal abnormalities, diaphragmatic hernia, gastrointestinal atresia, suspected syndromes, birth asphyxia, and metabolic disorders, which could independently influence cardiovascular function.

Data Collection

Data were collected using a structured questionnaire. Neonatal data included age, sex, gestational age, birth weight, residence, duration of hospital stay, and clinical outcomes. Physical and neurological examinations were performed for all neonates, with attention to signs of systemic infection such as temperature instability, poor feeding, apnea, bradycardia, respiratory distress, jaundice, seizures, irritability, lethargy, and abnormal urine output. Maternal data collected included age, parity, educational level, occupation, medical conditions, pregnancy complications, and obstetric history such as maternal fever, urinary tract infections, chorioamnionitis, prolonged rupture of membranes, meconium-stained amniotic fluid, place of delivery, and mode of delivery.

Laboratory and Imaging Investigations

Laboratory evaluations included complete blood count, C-reactive protein, erythrocyte sedimentation rate, blood cultures, and blood glucose levels. Age-specific reference ranges were used for leukopenia, leukocytosis, neutropenia, thrombocytopenia, and thrombocytosis. CRP levels above 10 mg/L and ESR above 15 mm/hr were considered abnormal. Blood cultures were processed using standard microbiological techniques, with preliminary results at 72 hours and final results at 7 days.

Chest radiography was performed for neonates with respiratory symptoms to assess for pneumonia or respiratory distress syndrome.

Echocardiography was conducted within the first two days of assessment by a single pediatric cardiologist using a Philips CX50 machine. Parameters evaluated included left ventricular ejection fraction, fractional shortening, myocardial performance index (MPI), tricuspid annular plane systolic excursion (TAPSE), and mitral annular plane systolic excursion (MAPSE). Standard reference values were used to define left and right ventricular systolic dysfunction and global myocardial performance.

Statistical Analysis

Data were analyzed using SPSS version 26.0. Continuous variables were expressed as mean $\pm$ standard deviation, and categorical variables as frequencies and percentages. Comparisons between groups were performed using the Mann-Whitney U test for continuous variables and the chi-square or Fisher's exact test for categorical variables. A p-value of <0.05 was considered statistically significant.

RESULTS

Selected characteristics of patients and control neonates

Selected demographic features of patients and control were assessed and compared, the results were demonstrated in Table (3-1).

Table (1): Description of specific features of the cases and control groups

Characteristics		Type of sample		P value
		Sepsis (n = 46) (mean ± SD)	Control (n = 59) (mean ± SD)	
Age (Days)		9.33 ± 5.612	7.87 ± 6.526	0.0530 [#]
Weight (Grams)		3100 ± 505	3086 ± 407	0.9300 [#]
Character		n (%)	n (%)	P Value
Sex	Male	23 (50%)	20(33.9%)	0.152 [*]
	Female	23(50%)	39(66.1%)	
Residence	Rural	23 (50%)	33 (55.93%)	0.693 [*]
	Urban	23 (50%)	26 (44.07%)	
Gestational age of the neonate (weeks)	37- 38	3 (6.5%)	4 (6.78%)	0.292 ^{**}
	>38-40	43 (93.5%)	52 (88.14%)	
	>40-41	0 (0.0%)	3(5.08%)	
Mode of Delivery	Spontaneous VD	30 (65.22%)	39 (66.1%)	0.906 ^{**}
	Elective CS	14 (30.43%)	17 (28.81%)	
	Emergency CS	2 (4.35%)	3 (5.09%)	
Outcome of the patient	Improved	45 (97.83%)	59 (100%)	0.181 ^{**}
	Other complication	1 (2.17%)	0 (0%)	

Mann-Whitney U Test

* Chi-Square Tests

** Fisher's Exact Test

Table (3-1) demonstrate that both septic neonates and controls groups were matched for age, weight, sex, residence, mode of delivery and gestational age. With septic neonates having a female to male ratio of 1:1. The greatest percentage of neonates in both cases and controls completed the 39th week of gestational age forming the majority of the sample. All the cases recovered and discharged home apart from one case developed pneumothorax and was referred to another hospital, no mortality was found in the total study sample.

1.1. Selected maternal characteristics of cases and controls

Some of the maternal characteristics were assessed and compared in table (3-2) between the mothers of septic neonate and control groups.

Table (2): Description of the maternal features of the septic neonate and control groups

Characteristics	Type of sample		P value [*]
	Sepsis (mean±SD)	Control (mean±SD)	
Maternal age (years)	25.64 ± 5.94	25.68 ± 6.51	0.8330

Parity		2.91 ± 1.81	2.72 ± 1.61	0.7200
		n (%)	n (%)	P value**
Maternal Educational level	Illiterate	7 (15.22%)	2 (3.38%)	0.027
	Primary	31 (67.39%)	53(89.83%)	
	Secondary	5 (10.87%)	1(1.69%)	
	Higher education	3 (6.52%)	3(5.1%)	
Job	House wife	45 (97.8%)	56 (94.92%)	0.633
	Employee	1(2.2)	3(5.08%)	

*Mann-Whitney U Test

** Fisher's Exact Test

In table (3-2), mothers of both groups were similar for age and parity. Regarding education it was higher among controls. Most of the mothers of both groups were found to be house wives.

1.2. Laboratory characteristics of both cases and controls

In table (3-3) laboratory tests were assessed and studied in relation to both septic neonate and control groups.

Table (3): Description of laboratory tests of the cases and control groups

Laboratory characteristics		Type of sample		P value*
		Sepsis (mean ± SD)	Control (mean ± SD)	
Total white blood cell count (×10⁹/L)		16.38 ± 13.19	13.56 ± 5.45	0.6670
Neutrophil count (/mm³)		9.42 ± 9.90	6.43 ± 4.13	0.4580
Platelet count (×10⁹/L)		301.96 ± 129.40	284.23 ± 83.80	0.3280
ESR (mm/hr)		17.71 ± 19.17	0.80 ± 3.55	0.0001
CRP (mg/L)		40.36 ± 38.05	0.20 ± 1.54	0.0001
		n (%)	n (%)	P value**
Chest X-ray	Abnormal	12 (26.09%)	0 (0%)	0.000
	Normal	34 (73.91%)	59 (100%)	
Blood culture	Positive	5 (10.9%)	0 (0%)	0.064
	Negative	27 (58.7%)	23 (39%)	

*Mann-Whitney U Test

** Chi-square test

According to table (3-3) it was found that sepsis patients had a higher CRP and ESR mean (40.36 mg/L) and (17.71 mm/hr) respectively both showing a highly statistically significant value (p = 0.0001). CXR also with a significant P value with (26.09%) sepsis neonates with abnormal x-ray finding. Only 5 cases of sepsis neonates had a positive blood culture, results including one case with *Escherichia coli*, one case of *Klebsiella pneumonia*, and three cases of *Staphylococcus aureus*.

1.3. Echocardiographic parameters of both cases and controls

Specific echocardiographic parameters conducted in this study were studied in septic neonate and control groups.

Table (4): Cases and controls in relation to Echocardiographic parameters

Echocardiographic parameters	Type of sample		P value*
	Sepsis (mean $\pm$ SD)	Control (mean $\pm$ SD)	
Ejection fraction (%)	62.62 $\pm$ 5.66	61.75 $\pm$ 4.15	0.594
Fractional shortening (%)	31.76 $\pm$ 3.93	31.18 $\pm$ 2.84	0.606
TAPSE (cm)	0.68 $\pm$ 0.23	0.78 $\pm$ 0.12	0.023
MAPSE (cm)	0.57 $\pm$ 0.21	0.77 $\pm$ 0.08	0.0001
Tei index	0.50 $\pm$ 0.14	0.34 $\pm$ 0.08	0.0001

Mann-Whitney U Test

This table shows nearly equal values of EF and FS of both groups, while there is a lower mean of both TAPSE and MAPSE in the cases which was statistically significant. Higher levels of Tei index also were found in the patient's group, it was also statistically significant.

1.4. percentage of abnormal echocardiographic parameters among septic neonates

Description of the number and percentage of patients who had abnormal echocardiographic parameters out of all sepsis patients (total number of neonates with sepsis equals 46).

Table (35): Frequency of specific echocardiographic parameters with abnormal finding in cases

Echocardiographic parameter	N (%)
EF	1 (2.2%)
FS	4 (8.7%)
TAPSE	7 (15.22%)
MAPSE	27 (58.7%)
Tei index	31 (67.4%)

This table describes the number of patients who had an abnormal finding in echocardiogram. About 40 patients were found to have some abnormal echocardiographic measure of the sepsis group, about (33 (71.74%)) neonates with sepsis had left ventricular dysfunction, Tei index being the most common one to be found abnormal (67.4%) followed by MAPSE (58.7%). While (15.22%) had RV dysfunction in combination with LV involvement represented by TAPSE

1.5. Description of selected cardiac parameters according to different selected neonatal characteristics in septic group

Echocardiographic abnormalities were studied in relation to septic group neonatal characteristics in table (3-6).

Table (-6): Selected echocardiographic parameter in relation to selected neonatal features in septic neonates

Characteristics		TPASE		MAPSE		Tei index	
		Abnormal (n= 7)	Normal (n= 39)	Abnormal (n=28)	Normal (n= 18)	Abnormal (n=25)	Normal (n=21)
Age (days)		10.29 $\pm$ 9.827	9.16 $\pm$ 4.647	9.92 $\pm$ 6.31	8.53 $\pm$ 4.51	8.64 $\pm$ 4.67	10.20 $\pm$ 6.63
P value**		0.657		0.587		0.377	
Weight(gram)		3116.86 $\pm$ 586.39	3097.37 $\pm$ 497.82	3143 $\pm$ 473.16	3042.11 $\pm$ 554.09	3096 $\pm$ 510.29	3105.9 $\pm$ 512.19
P value**		0.890		0.413		0.775	
Duration of admission (days)		5 $\pm$ 1.63	6.71 $\pm$ 4.25	6.35 $\pm$ 4.74	6.58 $\pm$ 2.79	6.92 $\pm$ 5.09	5.85 $\pm$ 1.87
P value**		0.259		0.203		0.881	
Sex	Male	5 (71.43%)	18 (46.15%)	16(57.14 %)	7(38.89%)	9(36%)	14(66.67 %)

	Female	2 (28.57%)	21 (53.85%)	12(42.86 %)	11(61.11 %)	16(64%)	7(33.33%)
P value*		0.188		0.55		0.075	
Gestational age (weeks)	37-38	1 (14.29%)	2 (5.13%)	3(10.71%)	0(0%)	0(0%)	3(14.29%)
	>38-40	6 (85.71%)	37 (94.87%)	25(89.29 %)	18(100%)	25(100%)	18(85.71 %)
	>40-41	0(0%)	0 (0%)	0(0%)	0(0%)	0(0%)	0(0%)
P value*		0.277		0.637		0.289	
Residence	Rural	4 (57.14%)	19(48.72 %)	15(53.57 %)	8(44.44%)	15(60%)	8(38.09%)
	Urban	3 (42.86%)	20 (51.28%)	13(46.43 %)	10(55.56 %)	10(40%)	13(61.9%)
P value*		0.526		0.767		0.236	

* Fisher's Exact Test

** Mann-Whitney U test

Table (6) showing no significant relation between these three echocardiographic parameters and the weigh, age, duration of admission, sex, gestational age, and residence.

1.6. Description of selected cardiac parameters according to different laboratory characteristics in septic group

Assessing the relation between the different laboratory studies included in the study some of the echocardiographic parameters used in cases of neonatal sepsis were presented in table (3-7).

Table (7): Relation between selected echocardiographic parameter and selected laboratory parameters of the neonatal sepsis sample

Laboratory parameter		TAPSE		MAPSE		Tei index	
		Abnormal (n=7)	Normal (n=39)	Abnormal (n=28)	Normal (n=18)	Abnormal (n=25)	Normal (n=21)
Total WBC count (×10 ⁹ /L)		17.30 ± 11.32	16.11 ± 13.69	16.50±11.53	16.02±15.62	17.33±15.31	15±10.34
P value**		0.570		0.483		0.964	
Neutrophil count (/mm ³)		11.55 ± 10.06	8.95±10	9.30±7.95	9.43±12.40	9.66±11.46	8.96±7.91
P value**		0.509		0.543		0.802	
Platelet count (×10 ⁹ /L)		287.43±92.49	304.63±135.93	305.58±127.02	297±135.92	286.92±141.88	320.75±112.6
P value**		0.725		0.827		0.515	
ESR (mm/L)		22.14±30.25	16.89±16.85	16.35±19.72	19.58±18.75	18.32±18.1	16.95±20.88
P value**		0.818		0.326		0.634	
CRP (mg/L)		17.36±12.12	46.49±41.07	35.61±29.2	50.63±49.73	49.14±45.84	32.98±28.07
P value**		0.023		0.389		0.207	
Blood culture	Positive	2(28.57%)	3(7.7%)	4(14.29%)	1(5.56%)	2 (8%)	3 (15%)
	Negative	5(71.43%)	22(56.41%)	19 (67.86%)	8(44.44%)	14 (56%)	13 (65%)
	Missed or contaminated	0 (0%)	14 (35.89%)	5 (17.85%)	9 (50%)	9 (36%)	4 (20%)
P value*		0.059		0.061		0.466	
CXR	Abnormal	2(28.6%)	10(25.64%)	6(21.42%)	6(33.33%)	7 (28%)	5 (23.81%)

	Normal	5(71.4%)	29(74.36%)	22(78.57%)	12 (66.67%)	18 (72%)	16(76.19%)
P value*		0.112		0.306		0.721	

* Fisher's Exact Test

** Mann-Whitney U Test

It is shown in table (7) that there is no statistically significant difference between abnormal and normal groups of each echocardiography study (TAPSE, MAPSE, and Tei index) mentioned in this table and the following laboratory studies (total WBC count, neutrophil count, platelet count, ESR, CRP, Blood culture, CXR). Apart from one statistically significant relation between CRP and TAPSE, but it has a higher value of CRP (46.49 mg/L) as a mean in the group with normal echocardiographic findings.

DISCUSSION

Sepsis results from a defect in the body's immune response to an infectious agent and can progress to multiorgan failure, with myocardial dysfunction being a major contributor to sepsis-related mortality. Mechanisms involved include elevated cytokines, endotoxin-induced dysfunction, altered calcium homeostasis, and increased nitric oxide production, highlighting the importance of early detection and treatment of cardiac involvement in sepsis [53], [54].

This case-control study evaluated full-term neonates with sepsis, age-, sex-, and gestational age-matched with healthy controls. The study found that TAPSE values were lower in septic neonates, with approximately 15.22% showing right ventricular (RV) systolic dysfunction. This finding aligns with a study from Egypt [55] and other reports demonstrating RV dysfunction in septic neonates using Tei index and tissue Doppler imaging [56], [57]. Reduced systemic venous return due to vasodilatation and, in some cases, increased pulmonary vascular resistance contribute to RV preload reduction, decreasing RV contractility and longitudinal shortening reflected in TAPSE measurements [45].

MAPSE was also significantly reduced in septic neonates, consistent with studies by Alzahrani et al. and Yousef et al. [55], [58]. Mechanisms include decreased LV contractility due to vasodilatation and, in cold shock, compensatory mechanisms such as increased systemic vascular resistance, limited contractility, and reduced ventricular compliance due to neonatal myocardial characteristics, leading to decreased longitudinal fiber shortening even if EF remains normal [39], [45], [50]. No significant differences were observed in LV fractional shortening or EF between septic and healthy neonates, consistent with previous studies by Fahmey et al., Yousef et al., Al-Biltagi et al., and Tomerak et al. [6], [55], [49], [57], although Alzahrani et al. reported significant

differences in certain follow-up assessments [58]. EF and FS may remain normal despite early myocardial dysfunction due to compensatory changes in afterload and systemic vascular resistance [36].

Septic neonates demonstrated higher myocardial performance index (MPI), indicating global LV dysfunction, consistent with Al-Biltagi et al., Yousef et al., and Tomerak et al. [49], [57], [55]. MPI elevation occurs when LV relaxation is impaired, prolonging isovolumetric relaxation time. Some studies showed no baseline differences between septic and control groups, with significant changes observed only after recovery [58].

No significant correlation was observed between CRP, ESR, CBC, chest X-ray, or blood culture results and echocardiographic parameters, except a single association between CRP and TAPSE, suggesting that CRP reflects systemic inflammation rather than direct myocardial dysfunction [57]. Other biomarkers such as troponin and brain natriuretic peptide may better indicate myocardial involvement.

Maternal educational level showed a significant association, with illiterate mothers more frequent in the sepsis group, whereas literate mothers predominated in controls (96.7%). This aligns partially with studies from Nepal [59] and Ghana [60], suggesting that maternal education may influence neonatal sepsis risk through better prenatal care and neonatal management.

CONCLUSION

Most neonates with sepsis exhibited some degree of myocardial dysfunction, demonstrated by abnormalities in at least one echocardiographic parameter, primarily Tei index, MAPSE, and TAPSE. Left ventricular dysfunction was the most prevalent form, whereas right ventricular systolic dysfunction was less common and occurred only in combination with left ventricular involvement. A higher incidence of neonatal sepsis was observed among infants of illiterate mothers. Although CRP and ESR levels were elevated in septic neonates, these inflammatory markers were not directly associated with myocardial dysfunction.

REFERENCES

1. A. Bethou and B. V. Bhat, "Neonatal Sepsis—Newer Insights," *Indian J. Pediatr.*, vol. 89, no. 3, pp. 267–273, 2022.

2. G. Via, S. Price, and E. Storti, "Echocardiography in the sepsis syndromes," *Crit. Ultrasound J.*, vol. 3, no. 2, p. 71, 2011.
3. M. Singer et al., "The third international consensus definitions for sepsis and septic shock (sepsis 3)," *JAMA*, vol. 315, pp. 801–810, 2016.
4. D. B. Haslam, in *Nelson Textbook of Pediatrics*, R. M. Kliegman et al., Eds., 21st ed., Philadelphia: Elsevier, 2020, pp. 996–1005.
5. M. Alemu et al., "Determinants of neonatal sepsis among neonates in northwest Ethiopia: Case-control study," *Ital. J. Pediatr.*, vol. 45, no. 1, p. 150, 2019.
6. S. S. Fahmey, M. Hodeib, K. Refaat, and W. Mohammed, "Evaluation of myocardial function in neonatal sepsis using tissue Doppler imaging," *J. Matern. Fetal Neonatal Med.*, vol. 33, no. 22, pp. 3752–3756, 2020.
7. A. J. Cant, A. R. Gennery, A. B. Russell, and D. Isaacs, in *Rennie and Robertson's Textbook of Neonatology*, 5th ed., Churchill Livingstone, Elsevier, 2012, p. 1015.
8. C. Fleischmann et al., "Global incidence and mortality of neonatal sepsis: Systematic review and meta analysis," *Arch. Dis. Child.*, vol. 106, no. 8, pp. 745–752, 2021.
9. R. Lafta and H. Habeeb, "Assessment of neonatal mortality major factors," *Iraqi Natl. J. Med.*, pp. 124–130, 2019.
10. J. Wynn, T. T. Cornell, H. R. Wong, T. P. Shanley, and D. S. Wheeler, "The host response to sepsis and developmental impact," *Pediatrics*, vol. 125, no. 5, pp. 1031–1041, 2010.
11. B. J. Stoll et al., "Early onset neonatal sepsis: The burden of group B Streptococcal and E. coli disease continues," *Pediatrics*, vol. 127, no. 5, pp. 817–826, 2011.
12. R. Rampersaud, T. M. Randis, and A. J. Ratner, "Microbiota of the upper and lower genital tract," *Semin. Fetal Neonatal Med.*, vol. 17, no. 1, pp. 51–57, 2012.
13. J. M. Wortham et al., "Chorioamnionitis and culture confirmed early onset neonatal infections," *Pediatrics*, vol. 137, 2016.
14. S. El Manouni El Hassani et al., "Risk factors for late onset sepsis in preterm infants: A multicenter case-control study," *Neonatology*, vol. 116, no. 1, pp. 42–48, 2019.
15. B. Goldstein, B. Giroir, and A. Randolph, "International pediatric sepsis consensus: Definitions for sepsis and organ dysfunction in pediatrics," *Pediatr. Crit. Care Med.*, vol. 6, no. 1, 2005.
16. I. Adams Chapman and B. J. Stoll, "Systemic inflammatory response syndrome," *Semin. Pediatr. Infect. Dis.*, vol. 12, no. 1, pp. 5–16, 2001.
17. S. L. Weiss et al., "Surviving sepsis campaign international guidelines for the management of septic shock and sepsis associated organ dysfunction in children," *Intensive Care Med.*, vol. 46, pp. 10, 2020.
18. R. Aufieri, S. Picone, and P. Paolillo, "Multiple organ failure in the newborn," *J. Pediatr. Neonatal Individ. Med.*, vol. 3, 2014.
19. J. L. Wynn et al., "Defective innate immunity predisposes murine neonates to poor sepsis outcome," *Blood*, vol. 112, no. 5, pp. 1750–1758, 2008.
20. M. A. Glaser et al., "Neonatal sepsis: A review of pathophysiology and current management strategies," *Adv. Neonatal Care*, vol. 21, no. 1, pp. 49–60, 2021.
21. WHO, *Pocket Book of Hospital Care for Children*, 2nd ed., Geneva, 2012.
22. K. A. Simonsen et al., "Early onset neonatal sepsis," *Clin. Microbiol. Rev.*, vol. 27, no. 1, pp. 21–47, 2014.
23. M. Gilfillan and V. Bhandari, "Neonatal sepsis biomarkers: Where are we now?," *Res. Rep. Neonatol.*, vol. 9, pp. 9–20, 2019.
24. C. P. Hornik et al., "Use of the complete blood cell count in early onset neonatal sepsis," *Pediatr. Infect. Dis. J.*, vol. 31, no. 8, pp. 799–802, 2012.
25. E. Tappero and P. Johnson, "Laboratory evaluation of neonatal sepsis," *Newborn Infant Nurs. Rev.*, vol. 10, no. 4, pp. 209–217, 2010.
26. A. L. Shane, P. J. Sánchez, and B. J. Stoll, "Neonatal sepsis," *Lancet*, vol. 390, no. 10104, pp. 1770–1780, 2017.
27. J. R. Delanghe and M. M. Speeckaert, "Translational research and biomarkers in neonatal sepsis," *Clin. Chim. Acta*, vol. 451, pp. 46–64, 2015.
28. N. Chauhan, S. Tiwari, and U. Jain, "Potential biomarkers for effective screening of neonatal sepsis infections: An overview," *Microb. Pathog.*, vol. 108, pp. 234–242, 2017.
29. C. Chiesa et al., "Fetal and early neonatal interleukin 6 response," *Cytokine*, vol. 76, no. 1, pp. 1–12, 2015.
30. J. L. Wynn, in *Fetal and Neonatal Physiology*, 6th ed., R. A. Polin et al., Eds., Elsevier, 2022, pp. 1620–1621.
31. M. G. Conti et al., "Immunometabolic approaches to prevent, detect, and treat neonatal sepsis," *Pediatr. Res.*, vol. 87, no. 2, pp. 399–405, 2019.
32. E. Sewell, J. Roberts, and S. Mukhopadhyay, "Association of infection in neonates and long term neurodevelopmental outcome,"

- Clin. Perinatol., vol. 48, no. 2, pp. 251–261, 2021.
33. E. Antonucci et al., “Myocardial depression in sepsis: From pathogenesis to clinical manifestations and treatment,” *J. Crit. Care*, vol. 29, no. 4, pp. 500–511, 2014.
34. A. Jain et al., “Prevalence and outcome of sepsis induced myocardial dysfunction in children,” *J. Trop. Pediatr.*, vol. 64, no. 6, pp. 501–509, 2018.
35. R. Habimana et al., “Sepsis induced cardiac dysfunction: A review of pathophysiology,” *Acute Crit. Care*, vol. 35, no. 2, pp. 57–66, 2020.
36. L. Tiwari, J. Chaturvedi, and C. Anand, “Myocardial dysfunction in sepsis,” *J. Pediatr. Crit. Care*, vol. 5, no. 4, pp. 41–?, 2018.
37. R. K. Aneja, R. V. Aneja, M. Good, and J. A. Carcillo, in *Neonatology: A Practical Guide to Neonatal Disease*, 2nd ed., G. Buonocore et al., Eds., Springer, 2018, pp. 1774–1780.
38. M. M. H. Marijianowski et al., “The neonatal heart has a relatively high content of total collagen,” *J. Am. Coll. Cardiol.*, vol. 23, no. 5, pp. 1204–1208, 1994.
39. W. A. Luce, T. M. Hoffman, and J. A. Bauer, “Developmental influences on cardiovascular dysfunction in neonatal versus adult sepsis,” *Crit. Care*, vol. 11, no. 5, 2007.
40. J. E. DeJesus, J. J. Wen, and R. Radhakrishnan, “Cytokine pathways in cardiac dysfunction following burn injury,” *J. Pers. Med.*, vol. 12, no. 11, 2022.
41. R. Fallach et al., “Cardiomyocyte Toll like receptor 4 is involved in heart dysfunction following septic shock,” *J. Mol. Cell. Cardiol.*, vol. 48, no. 6, pp. 1236–1244, 2010.
42. M. Da S. Campista, W. De A. Martins, M. De A. Guedes, and A. J. L. Jorge, “From echocardiographic evaluation to biomarkers measurement,” *Int. J. Cardiovasc. Sci.*, vol. 31, no. 6, pp. 643–651, 2018.
43. S. Deshpande et al., “Cardiac output in late onset neonatal sepsis,” *J. Clin. Diagn. Res.*, vol. 11, no. 11, pp. SC25–SC28, 2017.
44. C. Tissot, V. Muehlethaler, and N. Sekarski, “Basics of functional echocardiography in children and neonates,” *Front. Pediatr.*, vol. 5, p. 235, 2017.
45. A. Kharrat and A. Jain, “Hemodynamic dysfunction in neonatal sepsis,” *Pediatr. Res.*, vol. 91, no. 2, pp. 413–424, 2021.
46. D. S. Wheeler, H. R. Wong, and B. Zingarelli, “Pediatric sepsis—Part I: ‘Children are not small adults!’,” *Open Inflamm. J.*, vol. 4, pp. 4–?, 2011.
47. J. Sankar et al., “Prevalence and outcome of diastolic dysfunction in children with fluid refractory septic shock,” *Pediatr. Crit. Care Med.*, vol. 15, no. 9, p. e370, 2014.
48. M. Yousef et al., “Assessment of cardiac functions in neonatal sepsis,” *Al Azhar Int. Med. J.*, vol. 2, no. 12, pp. 46–50, 2021.
49. R. H. Tomerak et al., “Echocardiogram done early in neonatal sepsis: What does it add?,” *J. Investig. Med.*, vol. 60, no. 4, pp. 680–684, 2012.
50. L. Mertens and M. K. Friedberg, in *Echocardiography in Pediatric and Congenital Heart Disease*, 2nd ed., Wiley Blackwell, 2016, pp. 96–131.
51. C. R. Breatnach et al., “Novel echocardiography methods in the functional assessment of the newborn heart,” *Neonatology*, vol. 110, no. 4, pp. 248–260, 2016.
52. J. L. Wynn, T. T. Cornell, H. R. Wong, T. P. Shanley, and D. S. Wheeler, “The host response to sepsis and developmental impact,” *Pediatrics*, vol. 125, no. 5, pp. 1031–1041, 2010.
53. O. T. El Razaky and M. Al Biltagi, “Cardiac functions by tissue Doppler and speckle tracking in neonatal sepsis,” *J. Pediatr. Neonatal Care*, vol. 5, no. 3, 2016.
54. H. E. Abdel Hady, M. K. Matter, and M. M. El Arman, “Myocardial dysfunction in neonatal sepsis: Tissue Doppler imaging study,” *Pediatr. Crit. Care Med.*, vol. 13, no. 3, 2012.
55. A. K. Alzahrani, “Cardiac function affection in infants with neonatal sepsis,” *J. Clin. Trials*, vol. 7, no. 5, 2017.
56. G. Shah, S. Budhathoki, B. K. Das, and R. Mandal, “Risk factors in early neonatal sepsis,” *Kathmandu Univ. Med. J.*, vol. 4, no. 2, pp. 187–191, 2006.
57. M. Siakwa, D. Kpikpitse, S. C. Mupepi, and M. Semuatu, “Neonatal sepsis in rural Ghana: A case control study,” *Int. J. Res. Med. Health Sci.*, pp. 77–88, 2014.