

Original Research Article

Study of prevalence, etiology, response to treatment and outcome of paediatric shock in a tertiary care hospital

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ABSTRACT

Background: Shock accounts for 2% of children admitted to Paediatric casualty worldwide as per most western literature and in Nelson text book of Paediatrics. About 10 million children die of shock every year in the world. Highest mortality is observed in under 5 children in developing countries. Clinical manifestations are due to decreased perfusion to tissues, the compensatory mechanisms that are triggered by the decreased perfusion and the inadequate removal of metabolic wastes. This study was carried out to assess the prevalence of paediatric shock in children admitted to Paediatric ICU, to identify possible aetiology and the response to treatment and outcome in patients admitted with shock in Paediatrics Department of Government Mohan Kumaramangalam Medical College, Hospital, and Salem.

Methods: All sick children admitted to Paediatric intensive care unit of Government Mohankumaramangalam Medical College Hospital, Salem with the suspicion of shock are assessed by using the rapid cardiopulmonary assessment and diagnosed suffering from shock. Possible etiology, type and severity of shock would be arrived at using a targeted history, clinical examination and relevant laboratory investigations.

Results: All children who had unstable airway or bradypnea, were having decompensated shock and except one among them all expired despite prompt airway management. Respiratory distress noticed in 23 (40.4%) of children and all of them had either cardiogenic, septic shock or a combination of both. Capillary refill time was prolonged in 52 (91.2%) of children and the remainder 5 (8.8%) had flash refill and managed as warm septic shock. Decompensated shock as evidenced by low blood pressure was seen in 57.9% children. All of them had altered mental status. Urinary output was monitored in 38 children of which 31 (81.6%) had oliguria.

Conclusions: Septic shock accounts for majority of decompensated shock and poor outcome to management. Infancy decompensated shock, septic shock and those requiring ventilator support were the factors influencing the outcome of management.

Keywords: Airway obstruction, Metabolic waste, oliguria, Septic shock

INTRODUCTION

Shock is a clinically diagnosed altered physiological status defined as a complex state of circulatory dysfunction that results in inadequate delivery of oxygen and metabolic substrates to the tissues.¹ Clinical

manifestations are due to decreased perfusion to tissues, the compensatory mechanisms that are triggered by the decreased perfusion and the inadequate removal of metabolic wastes.² Shock accounts for 2% of children admitted to Paediatric casualty worldwide as per most western literature and in Nelson text book of Paediatrics.

About 10 million children die of shock every year in the world.³ Highest mortality is observed in under 5 children in developing countries. In order to prevent cellular death, once lactic acidosis sets in, various compensatory mechanisms come into play. The neural and humoral receptors are activated by decreased perfusion and decreased oxygen concentration in the blood and result in an increase in heart rate and stroke volume and help preserve the blood flow to brain, heart and kidneys. Respiratory rate also increases to compensate for metabolic acidosis. Oxygen extraction is increased. All these mechanisms defend the blood pressure and circulation to vital organs. This state of shock is called compensated shock. Decompensated shock occurs when cardiovascular system fails to maintain the blood pressure.⁴ This rapid cardiopulmonary assessment provides the best tool for decision making in emergency management.⁵ Most effective and sensitive physiologic status monitoring repeatedly by a competent and experienced physician cannot be replaced by the best monitors. Once diagnosed, shock has to be managed aggressively. First hour is considered the golden hour. Evaluation and treatment of underlying cause should proceed simultaneously. Airway must be managed as necessary.⁶ All children with shock must be administered high flow oxygen as there is tissue hypoxia. Intubation may be required in the following situations. Vascular access must be achieved rapidly. If not after 90 seconds, intraosseous route could be used to administer isotonic fluids which are the first-choice fluids for correction of shock. Rapid boluses of RL or NS at 20 ml/kg in 5-10 mm is given. Reassessment is done, and further fluids administered depending on the clinical situation. Significant reduction in mortality is achieved when >40 ml/kg of isotonic fluids are administered in the first hour. No difference in occurrence of ARDS due to rapid fluid bolusing has been noticed in between groups of patients who were given large boluses and groups given lower volumes.⁷

METHODS

The cross-sectional study was conducted period from Government Mohan Kumar Mangalam Medical College and Hospital Salem for 1 year from November 2016 to September 2017. Sample size was around 50 children.

Inclusion criteria

All patients between ages of 1-month and 12 years admitted to Paediatrics ward, Government Mohankumaramangalam Medical College Hospital, Salem.

Exclusion criteria

Neonates are excluded from the study.

All sick children admitted to Paediatric Intensive Care Unit of Government Mohankumaramangalam Medical

College Hospital, Salem with the suspicion of shock are assessed by using the rapid cardiopulmonary assessment and diagnosed suffering from shock. Possible aetiology, type and severity of shock would be arrived at using a targeted history, clinical examination and relevant laboratory investigations. These children are managed as per the paediatric advanced life support guidelines for shock with modifications for individual cases as necessary. The outcome of treatment is studied. Children are classified based on severity as compensated or decompensated shock and based upon their aetiology as hypovolemic, cardiogenic, septic, distributive, anaphylactic or obstructive.

RESULTS

This study aimed at assessing the prevalence of paediatric shock, the etiological profile and the management outcome. Children diagnosed to have shock by clinical cardiopulmonary assessment were classified according to aetiology and severity and managed appropriately as per PALS guidelines and the outcome of management studied. The data obtained were classified, analysed and interpreted with the help of statistical package S.P.S. S (13.0) at the 5% level of significance.

Table 1: Age and sex wise classification of trials.

Age Group	Male		Female		Total	
	No.	%	No.	%	No.	%
<12 month	15	46.9	12	48.0	27	47.3
1- 5 years	5	15.6	7	28.0	12	21.1
5-10 years	8	25.0	4	16.0	12	21.1
>10 years	4	12.5	2	8.0	6	10.5
Total	32	100.00	25	100.00	57	100.00
Range	1 month to 12 years		1 month to 10 years		1 month to 12 years	
Median	13.5 months		12 months		12 months	
Mean	44.9 month		32.8 month		39.6 month	
SD	46.9		37.6		43.1	

Table 2: Sex wise distribution of paediatric shock cases.

Sex	Total children admitted in ward / PICU	Total children admitted with shock	%	Prevalence per 1000/p
Male	1189	32	2.7	26.9/1000
Female	846	25	3.0	29.8/1000
Total	2035	57	2.8	28/1000

The subjects were studied and described according to their demographic characteristics namely sex and age. The total no of paediatric shock cases was 57. Among them 32 (56%) were male and 25 (43.9%) were females.

Nearly half 47.3% were infants. Children between 1-5 years and 5-10 years were 21.1% in each category >10 years children accounted for 10.5% of shock cases. The mean age of study population was 12 months. The median ages of male and female were 13.5 months and 12 months respectively.

Table 2 explains the prevalence as 28/1000 patients. In males it was 26.9/1000 and in females it was 29.8/1000. The difference between the two groups was not statistically significant.

Table 3: Clinical findings.

Clinical finding	No.	%
Unstable airway/Bradypnea	19	33.3
Effortless tachypnea	24	42.1
Respiratory distress	23	40.4
Tachycardia	42	73.7
Relative/absolute bradycardia	15	26.3
CRT prolonged	52	91.2
Flash refill	5	8.8
Blood pressure low	33	57.9
Liver span increased	24	42.1
Altered mental status (AJV/P/U)	57	100
Urinary output (>1ml/kg/hr)	31 (out of 38)	81.6

All children who had unstable airway or bradypnea, were having decompensated shock and except one among them all expired despite prompt airway management. Respiratory distress noticed in 23 (40.4%) of children and all of them had either cardiogenic, septic shock or a combination of both. Capillary refill time was prolonged in 52 (91.2%) of children and the remainder 5 (8.8%) had flash refill and managed as warm septic shock. Decompensated shock as evidenced by low blood pressure was seen in 57.9% children. All of them had altered mental status. Urinary output was monitored in 38 children of which 31 (81.6%) had oliguria.

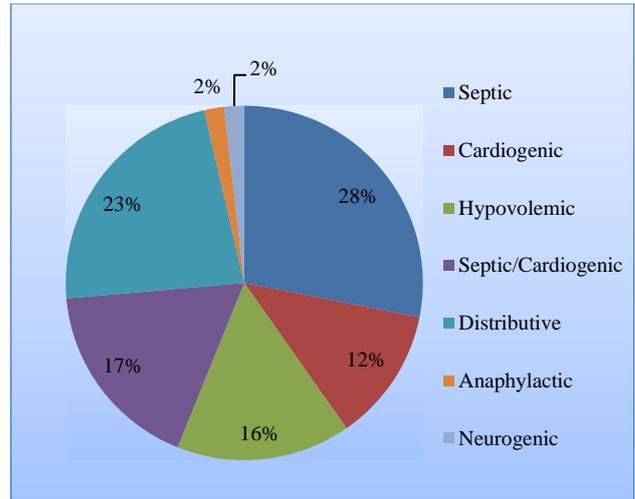


Figure 1: Percentage of distribution of etiology of shock.

Among the 57 cases studied septic (19.3%) was the major type among infants and 28.1% among the total group. This did not include the septic/cardiogenic type which accounted for 17.5% of cases. Hypovolemic was seen in 15.8% of cases and distributive in 22.8% of case. Cardiogenic shock was seen in 12.2%. One child (1.8%) had anaphylactic shock and another one (1.8%) had neurogenic shock due to omam water poisoning.

Table: 4 Renal function and liver function tests in children with shock.

RFT And LFT Elevated	Compensated		Decompensate		Total intubation required	
	No	%	No	%	No	%
RFT ↑	6	24	17	51.5	23	40.4
LFT↑	5	20.8	11	33.3	16	28.1

Table 5: Outcome based on etiological classification.

Etiology	Improved		Died		E	Duff	Significant
	No	%	No	%			
Septic	8	25	8	320	0.5	55	p>0.05
Cardiogenic	6	18.8	1	4.0	1.864	55	p>0.05
Hypovolemic	8	25	1	4.0	2.442	55	p>0.05
Septic / cardiogenic	1	3.1	9	36.0	3.264	55	p>0.01
Distributive	7	21.9	6	24.0	1.463	55	p>0.05
Anaphylactic	1	3.1	0	0.0	2.171	55	p>0.05
Neurogenic	1	3.1	0	0.0	1.011	55	p>0.05
Total	32	100	25	100			

Of the 45 children whose liver function test was available, 16 have elevated values, 11 from the

decompensated category and 5 from the compensated category. The difference was not statistically significant

($t = 1.072$ d.t 55 and $p > 0.05$). Renal function tests were done only in 50 children and liver function tests were done only in 45 children during the study due to difficulty in obtaining blood sample due to severity of shock while presentation and shorter duration of stay in the hospital. Of the 50 children whose renal function test was available, 23 have elevated values, 17 from the decompensated category and 6 from the compensated. The difference was statistically significant ($t = 2.23$ duff 55 and $p < 0.05$).

Death and improvement following management of shock were the two variables measured in study. Among the septic shock category 8 improved and 8 died. Among cardiogenic shock 6 improved and 1 died. Both there were not statistically significant. Where as in hypovolemic shock 8 improved and 1 died and the difference was statistically significant in children who had both septic + cardiogenic shock only 1 survived and 9 died which was also significant statistically.

DISCUSSION

Studies analysing the demographic profile and prevalence of shock in paediatric patients who present to a tertiary care hospital are very few in both western and Indian literature. Sex wise distribution of shock patients did not show any significance though of those children admitted, 846 were females and 1189 were males and 3% and 2.7% of them respectively were diagnosed to have shock.⁸ Neither did the severity of shock - compensated nor decompensated have any difference among the two sexes. All 57 cases were assessed by rapid cardiopulmonary assessment at presentation and the data of clinical findings obtained is discussed below. The most consistent finding noticed in the cases was altered level of sensorium at presentation.⁹

This was done using the A/V/P/U scale. All children (100%) had impaired consciousness of varying degrees. Next common finding was that of decreased urinary output noticed in 81.6% of children. Only 38 children with shock were catheterized for monitoring urine output out of which 31 had oliguria. Capillary refill time was prolonged in 91.2 % ($n = 52$) and flash refill noted in 8.8% ($n = 5$). All these 5 children were among the warm septic shock category at presentation. Tachycardia surprisingly was seen in only 73.7% ($n = 42$) children. The rest had relative/absolute bradycardia.¹⁰ Respiratory problems ranged from bradypnea, respiratory arrest, effortless tachypnea to respiratory distress. Respiratory distress was seen in 40.4% ($n = 23$) children and all of them had septic/cardiogenic shock.¹¹ Unstable airway/bradypnea was noticed in 33.3% ($n = 19$) and these children were having decompensated shock I imminent arrest. 57.9% ($n = 33$) cases of shock were decompensated while presentation to this hospital in while only 40% ($n = 39$) cases were decompensated in the study conducted by Singh D et al. In the present study children presented to the hospital in a more severe degree

of shock. 63.6% ($n=21$) of the 33 compensated shock cases died and 16.7% ($n=4$) of the 24 compensated shock cases died in our study while the percentage of death among the two groups was 67% and 2% respectively.¹²

The next common form of shock noticed was distributive shock which accounted for 22.8% ($n=13$) of 57 cases of shock. All these cases were suspected and later proved to be children with dengue shock syndrome or dengue haemorrhagic shock. Hypovolemic shock came in next with 15.8% ($n = 9$) of cases. All of them were due to diarrheal dehydration. One case of anaphylactic shock due to multiple bee sting was admitted in decompensated shock and responded well to isotonic fluid replacement, IM adrenaline and IV Hydrocortisone. One case of neurogenic shock was a result of Oman water poisoning and the child succumbed to decompensated shock. End points of management were achieved with isotonic fluids alone in 9 (15.8%) of cases with compensated shock and 2 (3.5%) of cases with decompensated shock.¹³ These two children who had received more than 80 ml/kg of isotonic fluids were hospitalized with severe diarrheal dehydration. Dopamine in addition to isotonic fluids was administered to achieve end points in 14 (24.6%) with compensated shock and 13 (22.8%) of patients with decompensated shock. Adrenaline infusion was used in 16 children (28%) of which 8 were administered Adrenaline following post arrest stabilization and 8 were administered Adrenaline infusion because they were catecholamine resistant. All the 16 children were in decompensated group.¹⁴

Intravenous Hydrocortisone was used in 5 children with septic decompensated shock who were resistant to inotropic support. Indicators were not used in the present study. In the present study 40 children (70.2%) out of the 57 cases had received >40 ml/ kg of fluid resuscitation in the first hour of management of these 40 children 20 of them died of which 85% ($n=17$) and 15% ($n=3$) of them suffered from decompensated and compensated shock respectively.¹⁵ Remaining 20 of those children survived. 19 (33.3%) of 57 children required endotracheal intubation and one more child required bag and mask ventilation. All of these children 94.7% ($n=18$) were among the decompensated group except for one child 5.3% ($n=1$) who was compensated at time of presentation. Only one child of the 19 requiring intubation survived.¹⁶ Liver function tests were elevated in 28.1% (16 out of 45) of children with shock and no significant difference was found between the compensated and decompensated groups. Renal function tests were elevated in 40.4% (23 out of 50) of children with shock and a significant difference was noticed with more children from the decompensated category having increased values.^{17,18}

CONCLUSION

Shock constitutes a significant percentage of diagnosis in critically ill children. Infants are affected by shock and

have severe degree of shock at diagnosis than more than any other age group in the study. No difference in prevalence or severity of shock at presentation between the two sexes was noticed. Septic shock accounts for majority of decompensated shock and poor outcome to management. Infancy decompensated shock, septic shock and those requiring ventilator support were the factors influencing the outcome of management.

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REFERENCES

1. American Academy of Pediatrics. Pediatric Education for Prehospital Professionals. Elk Grove village IL Jones and Barletta; 2000:133-138.
2. American Heart Association. Recognition of shock and respiratory failure. In: Camelids L. Hazinski MF. Pediatric Advanced Life Support; 2006:347-352.
3. Brierley J, Carrillo JA, Choong K, Cornell T, Decaen A, Deymann A et al. Clinical practice parameters for hemodynamic support of pediatric and neonatal septic shock: 2007 update from the American College of Critical Care Medicine. Crit Care Med. 2009;37(2):666-88.
4. Bollaert FE, Bauer P, Bert A. Effects of epinephrine on hemodynamics and oxygen metabolism in dopamine resistant shock. Chest. 1990;98(4):949-53.
5. Carrillo JA, Fields AT. American college of critical care medicine task force members. Clinical practice parameters for hemodynamic support of pediatric and neonatal patients in septic shock. Crit Care Med. 2002;30(16):1365-78.
6. Harris PA, Taylor R, Thielke R, Payne J, Gonzalez N, Conde JG. Research electronic data capture (REDCap): A metadata-driven methodology and workflow process for providing translational research informatics support. J Biomed Inform. 2009;42(2):377-81.
7. Cochrane Injuries Group Albumin Reviews. Human albumin administration in critically ill patients: systematic review of randomised controlled trials BMJ. 1998;317:235-40.
8. Conran RS, Kumar V, Robbins SL. Shock in fluid and hemodynamic derangements. In: Robbins Pathologic Basis of Disease. WB Saunders; 1989:114-119.
9. Fleisher G, Ludwig S. Textbook of Pediatric Emergency Medicine. 4th ed. Philadelphia Lippincott; 2000.
10. Carrillo JA, Tasker RC. Fluid resuscitation in Hypovolemic shock: acute medicine's great triumph for children. Intensive Care Med. 2006;32(7):958-61.
11. Frankel LR, Mathers LH. Shock, In: Nelson Textbook of Pediatrics. 17th Edition. Philadelphia: Saunders; 2003.
12. Irwin RS, Rippe JM. Irwin and Rippe's intensive care medicine, Philadelphia, Lippincott Williams and Wilkins; 2011.
13. Duke LT, Molineux EM. IV fluids for seriously ill children. Lancet. 2003;362:1320-3.
14. McConnell MS Perkin RM. Shock states. In: Pediatric Critical Care. 2nd ed. Fuhrman BP, Zimmerman JJ. St. Louis, Mosby; 1998;293-305.
15. Murphy K. Pediatric Triage Guidelines. Mosby St. Louis; 1997.
16. Finfer S, Bellomo R, Boyce N, French J, Myburgh J, Norton R. SAFE study investigators. a comparison of albumin and saline for fluid resuscitation in the intensive care unit. N Engl J Med. 2004;350(22):2247-56.
17. Singh S. Shock. Principles of Pediatric and Neonatal emergencies 2nd ed. New Delhi: Jaypee Medical Publishers; 2006.
18. Tobin JR, Wetzel RC. Shock and multiorgan system failure. Textbook of Pediatric Intensive Care. 3rd Edition. Lippincott Williams and Wilkins; 2008;123-8.

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