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Clinical Profile and Outcome of Non Traumatic Shock Patients Requiring Vasopressors for More Than Twelve Hours

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Abstract: ***Background: Shock is defined as a syndrome resulting in inadequate tissue perfusion and cellular oxygenation affecting multiple organ systems. Globally it is thought that shock of any etiology, septic shock especially is a terminal event between life and death, and is like handing over death warrant to a patient. This study was undertaken to find out various mortality predictors in shock, and to see, if their application can postulate remedy in future. Methods: This study was conducted on all admitted non-traumatic shock patients aged more than 18 years. Data was collected on daily basis till the day of discharge. Follow up of these patients was done at six months and at one year. Results: A total of 200 patients met the inclusion criteria. Of these patients 39% died in initial admission, 61% of patients were discharged from hospital. Out of survivors 10% died at six months and 4.5% died at one year, contributing total mortality of 53.5%. Significant mortality predictors identified include; Age > 60 years (p value= 0.001), Number of organ failures (p value < 0.001), treatment with vasopressors for more than 24 hours (p value < 0.001), high APPACHE –II score and Mean Spao2 values. Conclusion: Overall one-year mortality of all shock patients was around 53.5%. Age> 60 years, High APACHE score, Mean Spao2 values, Number of organ failures, and duration of shock were the main predictors and should be used for assessment of survival outcomes in shock patients.***

Keywords: Shock, Vasopressors, Mortality

1. Introduction

Shock is one of the most frequently diagnosed, yet poorly understood clinical conditions encountered in critically ill patients. The very definition of what constitutes "shock" remains controversial, largely due to its variable presentation and multifactorial etiologies. High-dose vasopressor dependent shock is often seen as a terminal event in the intensive care unit. On one hand it is commonly argued that it is futile to administer high-dose vasopressors in the critically ill patients with multiple organ failure [(2)], and on the other hand, survival rate is up to 50% in "severe" septic shock patients receiving early treatment with a specific algorithm [(3)]. Since mortality in refractory shock can be as high as 94% (4), efforts should be made to find the cause(s) for this refractory syndrome. In the extreme clinical scenario of refractory cardiovascular failure, the etiology and specific treatment of shock should be aggressively pursued in every cause of shock, including Sepsis [(5)], Myocardium ischemia complicated by cardiogenic shock [(6)], massive pulmonary embolism, [(7)] and in other shock etiologies.

1.1 Why we have chosen 12 hours in our study

This study was selected in the view that these patients are supposed to have worse outcome than those who do not require vasopressor support, or require it for less duration of time. Secondly, we wanted to know the outcome predictors for this sick group. Since globally, every institution is trying to find the causes or reasons of high mortality in stroke, AIDS, malignancy, but mortality related to shock lacks similar attention. We tried to find out various etiologies, clinical as well as lab parameters for discovering the reason of high mortality, so that; ways to lower it can be understood. Twelve hours of treatment is arbitrary time duration chosen by us, as there is no such study till date which has chosen any time duration for vasopressor administration. The main reason for mentioning twelve hours in our study was to choose severe shock patients and to know the reason of high death rates in this group of patients, to find out the predictors of mortality, whether the providence of treatment to them was beneficial and what the outcome in these chosen individuals was.

1.2 Aims and Objectives

To study and evaluate the clinical profile of non-traumatic shock patients requiring vasopressors for more than 12 hours in terms of:

1) Etiology of shock (Septic, Cardiogenic, Pulmonary, Neurogenic, Anaphylactic and Hypovolemic).
2) To study demographic, physical and Lab parameters.
3) To look for treatment received by patients of shock.
4) Follow up of patients at six months and at one year.
5) Correlate Etiology, Organ dysfunction, demographic profile, lab parameters and APACHE –II (Acute physiology and chronic health evaluation) scores at admission with early and late mortality and morbidity.

The main aim is to find predictors of short and long-term outcome and suggest ways to improve survival of shock patients.

2. Materials and Methods

The study entitled “Clinical Profile and outcome of non-traumatic shock Patients requiring vasopressor therapy for more than twelve hours” was conducted in Post Graduate department of Emergency medicine, Internal medicine and Intensive care sher-i-Kashmir institute of medical sciences, J&K India. The study was conducted on all admitted non-traumatic shock patients aged more than 18 years for duration of two years. It was a prospective observational, non-interventional study. The selection of patients was made on the basis of detailed history, thorough general physical and systemic examination. All the patients enrolled for the study underwent thorough investigations as per the clinical profile of patient. After securing airway, breathing and

circulation, every type of shock was initially resuscitated with fluids, and the preferred fluid for resuscitation was 0.9% NS. All patients included in this study did not respond to fluid challenge and were labeled as non-responders, and vasoactive agents were added as second line management.

Exclusion Criteria:
Traumatic shock patients. All shock patients who responded to initial fluid resuscitation and did not require vasopressor support. And all shock patients requiring vasopressors for less than 12 hours.

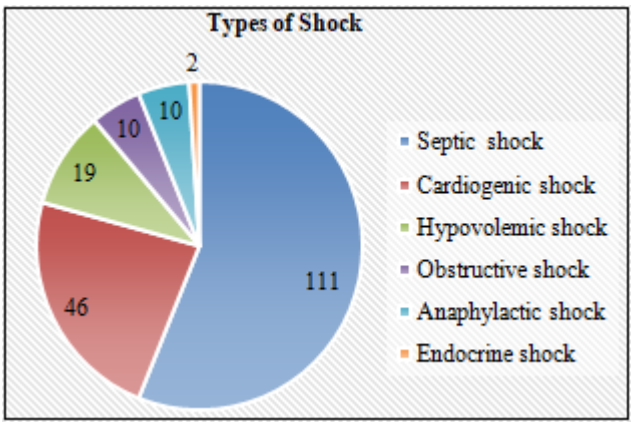
3. Results

We conducted a study in Department of Emergency Medicine, Internal Medicine and in Intensive care unit from June 2014 – July 2016 for a period of two years to classify shock into various etiologies, their clinical profile, and to determine various factors affecting and influencing outcome in these patients. We studied all non-traumatic shock patients aged > 18 years who were admitted in this institution within these two years in department of Emergency Medicine, and following results were concluded:

Following table showing patient profile, lab parameters, and treatment characteristics:

Gender	Male =109(54.5%)
	Female=91(45.5%)
Age group	18 – 39 yrs. =28(14%)
	40 - 59 yrs. =59(29.5%)
	>60 yrs. =113(56.5)
Comorbidities	Zero= 44(22%)
	Single=96(48%)
	Multiple=60(30%)
Mean APACHE II Score	38.37
Mean MAP	59.97mmHg
Mean Spao2 of patients on Admission	Ward=87.48
	ICU=67.13
Mean HB	9.8g/dl
Pressor used	Single=128(64%)
	Double=54(27%)
	Triple=18(9%)
Duration of Pressor therapy	12-18 hrs.=89(44.5%)
	18 – 24 hrs.=38(19%)
	>24 hrs.=73(37%)
No. of organ failures	No=77(38%)
	Single=62(31%)
	Multiple=61(30.5%)

Below given the pie chart shows types of shock studied:



Following table gives the detailed work out of classification of shock

Source of sepsis in septic shock;		
Chest	75	37%
Urine	19	7.50%
Abdomen	12	6%
Other	5	3%
Causes of cardiogenic shock:		
Myocardial Infarction	17	8.50%
DCM	26	13%
Arrythmias	3	1.50%
Causes of Hypovolemic Shock:		
Acute Gastroenteritis	11	5.50%
GI Bleed	5	2.50%
Pancreatitis	2	1%
Hemorrhage	1	0.50%
Causes of Anaphylactic Shock:		
Envenomation	7	3.50%
Drug	2	1%
contrast	1	0.50%
Causes of Obstructive shock:		
PTE	9	4.50%
Cardiac Tamponade	1	0.50%
Causes of Endocrine Shock:		
Adrenal crises	1	0.50%
Autonomic Failure	1	0.50%

On follow up, One year mortality was around 53.5% and mortality predictors studied are given in below table:

Mortality predictors	P value
Age	0.001
Comorbidity	0.001
MAP	0.001
Spao2	0.001
No. of organ failures	0.001
No. of pressor support used	0.001
Duration of pressor support	0.001

4. Discussion

We studied all non-traumatic shock patients aged > 18 years who were admitted in this institution for a period of two years. There were total of 200 patients, majorities were males (54.5%) and most of them were above 60 years of age. Majority of our patients (77%) had comorbid illness like T2DM (23.2%), HTN (24.2%), Malignancy (28.2%) and CKD (12.2%). Our study agrees with **Beck et al,** [8] who studied the epidemiology of shock patients; among them the major existing comorbidities were T2DM (26.6%), hypertension (19.1%), chronic renal failure (23.1%), and immune suppression (14.7%). This was consistent with our study.

We calculated APACHE -II and MAP in our studied population, on the day of admission and their mean was 38.7 ± 17.3, and 59.97 ± 7.7 respectively, comparable with **Samuel M et al** [9], where APACHE- II & MAP was 36 & 65 respectively, however were not comparable to those of **Daniel De Backer et al** [10] (APPACHE –II =20 MAP =58) **& Beck et al** [8] (APPACHE-II= 26.1 ± 8.2). This variability in these scores could be explained by the fact that our patients were sick than those who were enrolled by Daniel De Backer & Beck et al. We also measured oxygen

saturation ($Spa0_2$) of all patients enrolled in study, on the day of admission, day of admission to ICU and on the day of discharge and it was 87.48 ± 6.8, 67.13 ± 6.7 and 92.6 ± 2.5 respectively. It was comparable to **Daniel De Backer et al.** [10] were $Spa0_2$ mean was 95± 9. However, they have not assessed its utility in terms of outcome, and to our knowledge no other study has determined statistical significance of oxygen saturation to mortality. We found a direct correlation between Spao2 levels (estimated by ABG analysis) on admission and mortality. In the patients who died of shock, the mean spao2 level at admission was 84.36 while in the survivors it was 91.5 and this was statistically significant. (P-value <0.001). Hence it is safe to conclude that Spao2 level below 85 at admission is a strong predictor of mortality.

Patients enrolled in our study, presented to the hospital with varied symptoms e.g. fever, cough, breathlessness, oliguria, mental obtundation, upper gastrointestinal bleed, pain, vomiting and loose motions, and few patients had history of reptile bite and orthopod bite. Based on this presentation and other lab parameters we classified our studied patients into various etiologies of non-traumatic shock, among them the majority was that of septic shock (55.5%), with source of sepsis being chest (37%), urine (9.5%), abdomen (6.0%) and others (3.0%). Cardiogenic shock was the second leading cause of shock in our study (23%); etiologies were myocardial infarction (8.5%), cardiomyopathies (13%), and rarely shocks were secondary to arrhythmias (1.5%).Out of cardiomyopathies, dilated cardiomyopathy was most common. The third commonest etiology of shock was severe hypovolemia (9.5%) and etiologies were related to acute gastroenteritis (5.5%), UGI bleed (2.5%) and pancreatitis (1%). We also encountered obstructive type of shock (0.5%), e.g. PTE (4.5%) and cardiac tamponade (0.5%), however we did not receive any patient of shock secondary to tension pneumothorax. Shock related to anaphylaxis had an incidence of (5.0%), majority being related to snake bite (3%), one patient had shock secondary to insect bite (0.5%), two patients had anaphylaxis with drugs (Quinolones and Ondansetron) and one patient had anaphylactic shock related to contrast given for imaging. In our study we received two patients with endocrine shock, one with Pure Autonomic Failure and other with Adrenal Insufficiency secondary to disseminated tuberculosis. Our findings regarding etiology of shock (septic shock being most common) and also regarding the source of sepsis in septic shock are consistent with Daniel **De Backer, et al**[10] and **Samuel M Brown et al**[9].

Shock of any etiology leads to vital organ failure due to microcellular damage. In our study group single organ failure was found in 62 patients (31%) and multiorgan failure in 59 (29.5%) patients. Among organ failures Renal involvement was at the top (52.5%), followed by Respiratory (24.0%), Hepatic (15%), Hematological (11.5%), and Central nervous system (4%). Which was cognizant with **Vance Beck, et al.** [8] when organ failures were related to mortality, our results were comparable to Vance beck et al as adjusted P values are given as in table below:

Study	Organ failure with P values				
	Renal	Respiratory	CNS	Coagulation	Hematological
Vance beck	0.01	0.001	0.02	0.008	0.07
Present study	0.001	0.001	0.02	0.06	0.09

In our study total mortality was (53.5%), among them most of patients died in hospital. Mortality was different for different etiologies, in septic shock it was the highest 64.8%. Upon commenting on source of sepsis, maximum mortality was in patients who had respiratory sepsis followed by those with abdominal and urinary sepsis. In cardiogenic shock, mortality was (47%), deaths being highest in MI complicating cardiogenic shock, followed by cardiomyopathies (42.3) and arrhythmias (%). Mortality in obstructive shock was (70%), PTE being the major culprit. Other group of patients was victims of endocrine shock, in which no deaths were noticed. In anaphylactic shock, death rates were significant about (60%), most deaths occurred due to envenomation. Other category of shock was because of hypovolemia, among them the mortality rate was around (1.5%). No patient died because of AGE and pancreatitis. There was one death related to UGI bleed (0.5%) and one death related to massive hemorrhage (0.5%) secondary to heparin prophylaxis. '50 to 60 percent of patients die within one month of the onset of septic shock.' **Bone RC** [11]. 'In cardiogenic shock; mortality is estimated to be 60 to 90 percent' **Moscucci M et al** [12]**.** 'Mortality due to hypovolemic shock is variable', **Hochman JS et al** [13]. Which were consistent with mortality rates of our study.

These patients were followed after admission, till the day of discharge, at six months and at one year. It was found significant number of patients expired during hospital admission and the in -hospital death predictors were, type of shock (mortality was more in Septic & PTE shocks rather than shocks related to other etiologies), patient characteristics like Age (≥ 60 years when compared to younger individuals P value was 0.001), Comorbid illness (single comorbidity verses no comorbidity P value was 0.002), APPACHE –II scores (higher scores were associated with more deaths), duration of vasopressor infusion(p value was 0.001 when mortality of 12- 18 hour duration of pressor therapy was compared to mortality of > 18 hour duration of pressor treatment), and the number of organ failures (p value of 0.001 when organ failure mortality was associated with patients who did not have organ dysfunction.

Vast et al (2008), had follow up of 90 days, with corresponding mortality of 52%, Park et al (2005), had follow up of 28 days with mortality of 65%, Castro et al (2008), had follow up of 28 days, being mortality of 48%, Torgerson et al (2011), and mortality of 61%, and Dunsers et al (2009), had six-month mortality of 71%.

5. Follow-up

On follow up, One year mortality was around 53.5% and mortality predictors studied were: Age { p value = 0.001 (when mortality in age group of > 60 years was compared with mortality in age group less than 60.)}, APPACHE – II scores, Number of organs failures{ p value=0.001 (no organ failure vs single organ failures)}, Duration of pressor therapy{ p value= 0.001(≤ 12 -18 hours vs > 18 hours)}, and

Etiology of shock obstructive shock 70%, septic shock 64.0%, other shocks contributed less to mortality. Mortality was 47% in cardiogenic, 1.5% in Hypovolemic shocks, and 0% in adrenal and neurogenic shocks.

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