Indian Journal of Emergency Medicine

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Indian Journal of Scientific Computing and Engineering	2	4000	280
Indian Journal of Surgical Nursing	3	3000	70
Indian Journal of Trauma & Emergency Pediatrics	4	8500	302
International Journal of Agricultural & Forest Meteorology	2	8000	800
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International Journal of History	2	6000	500
International Journal of Neurology and Neurosurgery	2	9000	276
International Journal of Political Science	2	5000	400
International Journal of Practical Nursing	3	3000	70
International Physiology	2	6500	240
Journal of Animal Feed Science and Technology	2	4000	280
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Journal of Orthopaedic Education	2	4500	190
Journal of Pharmaceutical and Medicinal Chemistry	2	15000	350
Journal of Psychiatric Nursing	3	3000	70
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Indian Journal of Emergency Medicine
Volume 1 Number 2 July - December 2015

A Study of Levels of Stress Among Physicians in A Tertiary Care Hospital In Kolkata India

Sanskar Pandey*, Indraneel Dasgupta, Indranil Mitra*****

Abstract

Background: It is essential to know to about work related stress, as prolonged stress at workplace reduces the performance of an individual and has an indirect/direct effect on health &on his/her professional social and personal life. In a health care system it is very important to know about the stressors ,as increased stress levels invariably affects interpersonal relationships ,doctor to patient communication , inter-colleague relationships and professional performance.*Aim:* To determine a) Levels of stress among physicians b) Factors associated with high stress levels c) Attempt to develop tips for reducing stress on a long term basis. *Materials and Method:* A group administered questionnaire based survey was done in which a pre validated questionnaire was used both in clinical settings and on epidemiological settings on mental health status of the participants. All full time consultants, associate consultants, attending consultants, Post graduate trainees from Masters in Emergency Medicine, DNB Medicine, family medicine, orthopedics, pediatrics.,full time residential medical officers attached to Peerless Hospital during the study period were included. All non –physicians, non medical and ancillary staff attached to Peerless Hospital were excluded. The sample size required for this survey was calculated as 72, rounded to 70. *Statistical Analysis:* In the present study 20 out of 43 physicians of age group 25-35 found to have moderate to severe stress. The another factor seen is physicians who are taking care of clinical work and working in-hospital are experiencing more stress the p values for these two factors came to be (p=0.01) which is statistically significant *Result:* Though majority of responders initially felt that they perfectly well, 25% realised the need of a good tonic/ refreshment. Later 36% of physicians felt that they are not feeling well in daily lives. Approximately 64% felt that they were feeling run down and out of sorts. Many (41%) experience headaches due to work stress. 41% of physicians complained both hot and cold spells recently. A large number of physicians had lost their sleep. 21% of physicians feel that they are nervous all the time. Though majority are able to keep themselves busy some are not. Some are taking longer time to complete their routine tasks. Though 89% feel they are able to do things better than usual a large number of physicians contradict them. A large number of physicians are taking longer time to complete their routine tasks. 10% feel less satisfied in the way they have carried their task. A large chunk (9%) feels themselves to be worthless though another small group (3%) believe that their life is entirely hopeless. A small population (3%) of physicians confesses that the idea of making away with themselves actually crossed their minds. Even some say (4%) that at some point of time they thought of taking their own life. 6% of physicians feel that their nerves are so bad that they are unable to do anything.. 4% physicians says that the idea of taking their life has crossed their mind. *Conclusion:* There is trend of higher GHQ scores that correlates with the higher stress levels amongst the emergency physicians and the physicians working in-hospital and taking care of clinical departments.

Keywords: Stress; Physician Work Load.

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Introduction

Stress can be either eustress or distress. i.e. in simple terms it can be good or bad. Whether physical or mental, it has been attributed to affect physical/mental health in some or the other way. So the need for evaluation of stress levels among health care provider so as to know the factors associated with high levels of stress and to modify the factors in a way that reduces the stress levels among physicians.

Various persons have defined stress in various ways few of those definitions are coded here:-

- Stress was defined as the *nonspecific response* of the body to any demands made upon it (Selye 1976).
- Stress was defined as environmental conditions that require *behavioral adjustment* (Benson, H. *The Relaxation Response*, 2000, pg. 41).

Various studies have shown that people adopt unhealthy life style to deal with stress. The term*"burnout"*which is a result of prolonged stress is characterized by progressive loss of idealism energy and purpose experienced by people working in the human services (Agius et al 1996). The burnout phenomenon as defined by Pines and Maslach was as a syndrome of emotional exhaustion involving the development of a negative self –concept, negative job attitudes and loss of concern for clients (Schweitzer, 1993). Maslach also devised an inventory to measure burnout in physicians known as Maslach burnout inventory.

Chronic stress reactions and depression are often characterized by long term activation of the hypothalamic-pituitary-adrenal axis and the sympathetic nervous system which were found to be associated with the development of abdominal obesity, and this may explain why depression or chronic stress increases the risk of diabetes (Björntorp, 2001; Vogelzangs et al., 2008). The vicious cycle of stress, excessive secretion of cortisol and other stress hormones which triggers the immune system as it has a direct line to the hypothalamus. When the immune system is activated to fight illness or infection, it sends a signal to the hypothalamus to produce its stress hormones, including cortisol. The flow of hormones, in turn, shuts off the immune response. This negative-feedback loop allows a short burst of immune activity, but prevents the immune system from over activity. In this way, some stress can be beneficial for the individual. But chronic stress produces such a constant flow of cortisol that the immune system is dampened too much. This helps explain how stress makes us ill (Sternberg, 2000).

Various studies indicate that physicians are not very good at taking care of themselves if stressed/ burnt out.

Since stress has effect on an individual's professional, social, personal life as well on physical/ mental health its evaluation should be done to understand the common stressors and levels of stress in an organization and ways and means to reduce it.

It is essential to know to about it as prolonged stress at workplace reduces the performance of an individual and has an indirect/direct effect on health &on his/ her professional social and personal life.

In a health care system it is very important to know about the stressors, the existing stress levels as increased stress levels invariably affects interpersonal relationships, doctor to patient communication, inter-colleague relationships and professional performance.

Aims of the Study

The study was carried out with the aim of determining the following.

a. Levels of stress among physicians
b. Factors associated with high stress levels
c. Attempt to develop tips for reducing stress on a long term basis.

The primary research question in study was to evaluate levels of stress , and to know about the common stressors associated with it.. The definition chosen for the present study is the definition stated by Seyle1976 and Benson, H as these definitions are easy to understand and easy to correlate with day today situations. . Evaluation of the factors will further help to reduce the stress and enhance performance levels amongst doctors.

Study Methodology

It dealt with the research methods that was used in this study to evaluate the levels of stress among physicians of Peerless Hospital, Kolkata, India. The study was conducted in the Peerless Hospital and B.K. Roy Research Centre, Kolkata. The duration of the study was 1 year between September 2013 to August 2014. It was an observational cross sectional nonrandomized, questionnaire based study, designed to look at the factors strongly associated with subjective stress levels among the all Physicians and Surgeons in Peerless Hospital. A group administered

questionnaire based survey was done in which a pre validated questionnaire was used both in clinical settings and on epidemiological settings on mental health status of the participants. All full time consultants, associate consultants, attending consultants, Post graduate trainees from Masters in Emergency Medicine, DNB Medicine, family medicine, orthopedics, pediatrics.,full time residential medical officers attached to Peerless Hospital during the study period were included. All non –physicians, non medical and ancillary staff attached to Peerless Hospital were excluded. The sample size required for this survey was calculated as 72, rounded to 70. Upon completion of data collection, data was coded, captured on Excel and then the statistical analysis was done.

Results

As this is questionnaire based study, several questions were formed & data interpretation was done

Fig. 1: Feeling perfectly well and in good health

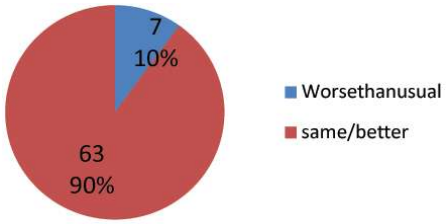


Fig. 2: Have you recently been feeling in need of a good tonic?

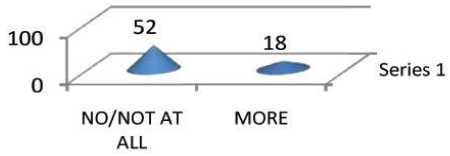


Fig. 3: Feeling run down and out of sorts

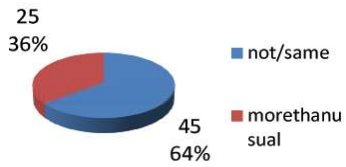
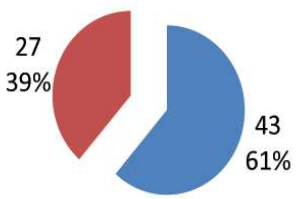


Fig. 4: Feeling ill



Though majority of responders initially felt that they perfectly well, 25% realised the need of a good tonic/ refreshment. Later 36% of physicians felt that they are not feeling well in daily lives. Approximately 64% felt that they were feeling run down and out of sorts.

Fig. 5: Loss of sleep or over worry

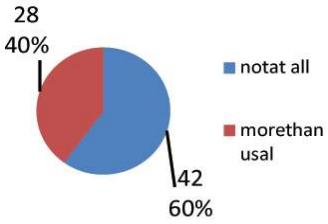


Fig. 6: Getting edgy and bad-tempered?

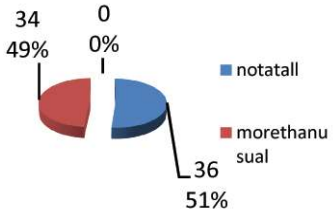


Fig. 7: Getting scared or panicky for no good reason

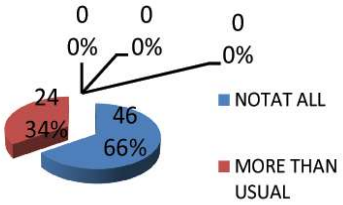


Fig. 8: Have you recently found everything getting on top of you?

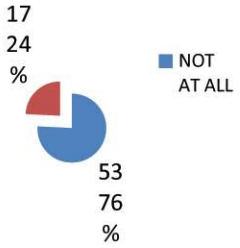


Fig. 9: Feeling nervous and strung-up all the time?

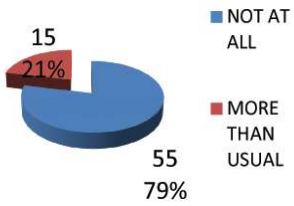
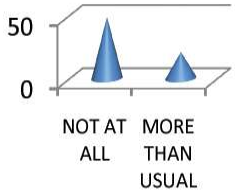


Fig. 10: Have you recently been managing to keep yourself busy and occupied?



Many (41%) experience headaches due to work stress. 41% of physicians complained both hot and cold spells recently. A large number of physicians had lost their sleep. 47% of physicians shared that they are experiencing difficulty in sleeping once they are off and feel that they are in constant strain. Many (49%) are getting edgy and bad tempered easily.

21% of physicians feel that they are nervous all the time. Though majority are able to keep themselves busy some are not. Some are taking longer time to complete their routine tasks. Though 89% feel they are able to do things better than usual a large number of physicians contradict them.

Fig. 11: Have you recently felt on the whole you were doing things well?

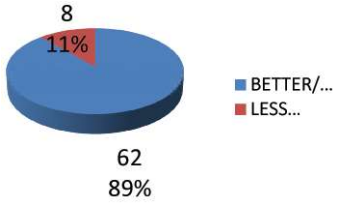


Fig. 12: Taking longer to do things

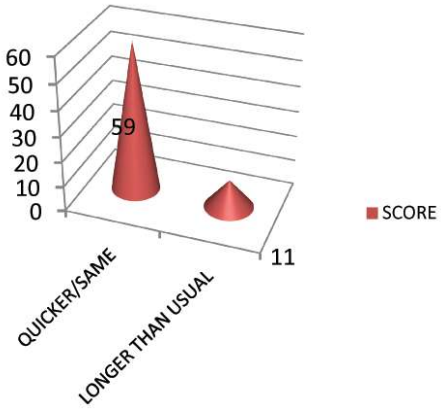


Fig. 13: Have you recently been satisfied with the way you've carried out your task?

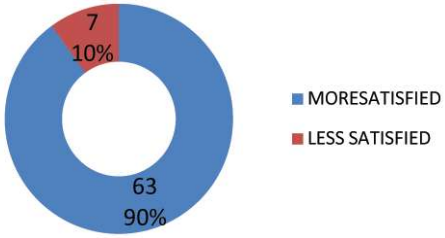
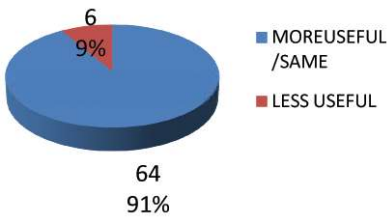


Fig. 14: Have you recently felt that you are playing a useful part in things?



A large number of physicians are taking longer time to complete their routine tasks. 20% feel they are unable to do things better than usual. 10% feel less satisfied in the way they have carried their task. Many physicians believe that they are not playing less useful part in their day today life.

Fig. 15: Have you recently felt capable of making decisions about things?

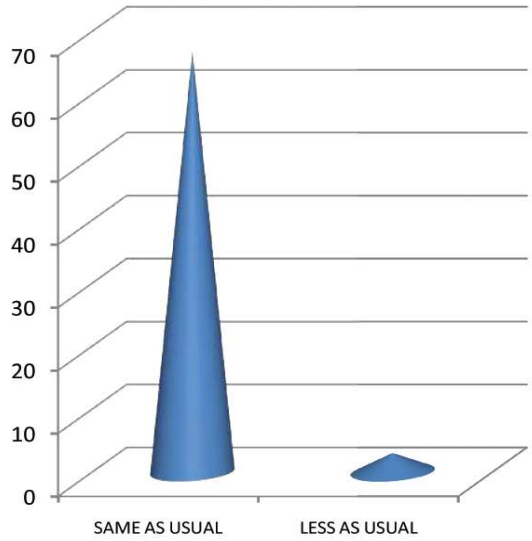


Fig. 16: Have you recently been able to enjoy your normal day-to-day activities?

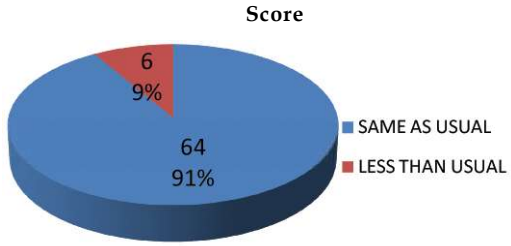


Fig. 17: Thinking oneself as a worthless person?

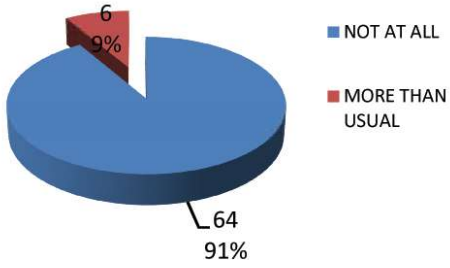
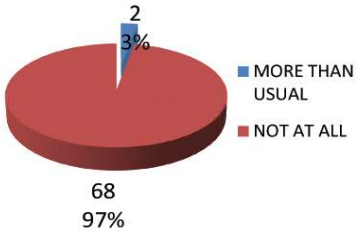


Fig. 18: Life is entirely hopeless



A small number (4%) of physicians had problems in decision making. Some (9%) say that they are not able to enjoy their day today activities same as usual. A large chunk (9%) feels themselves to be worthless though another small group (3%) believe that their life is entirely hopeless.

Fig. 19: Life isn't worth living

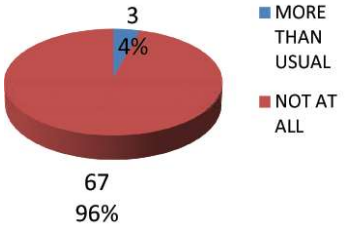
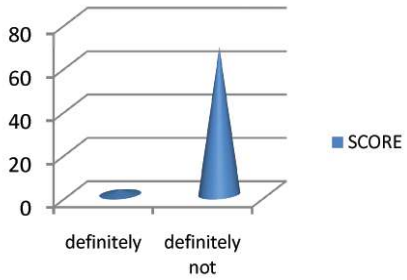


Fig. 20: Making away



A small population (3%) of physicians confesses that the idea of making away with themselves actually crossed their minds. Even some say (4%) that at some point of time they thought of taking their own life. 6%

Fig. 22: Wishing you were dead and away.

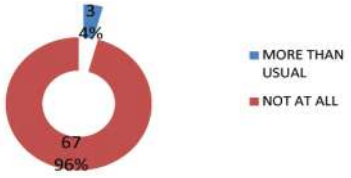


Fig. 23: Taking Life

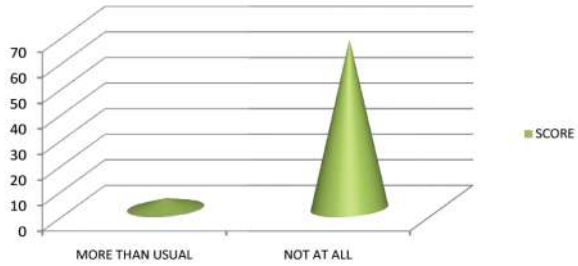
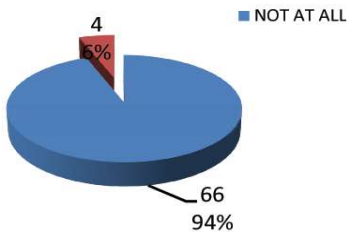


Fig. 21: Nerves were too bad



of physicians feel that their nerves are so bad that they are unable to do anything.. 4% physicians says that the idea of taking their life has crossed their mind..

Statistical Analysis

			score_3grp			
			< 8	8-14	15-28	Total
Age	25-35 yrs.	Count	23	16	4	43
		% within Age	.5	.4	.1	1.0
	36-45 yrs.	Count	14	5	0	19
		% within Age	.7	.3	.0	1.0
	46-65 yrs.	Count	3	5	0	8
		% within Age	.4	.6	.0	1.0

p=0.19

The physicians in the age group of 25-35 years, 16 out of 43 are in moderate stress and 4 out of 43 are in severe stress. i.e. 37% of physicians found to be in moderate stress and 9% of physicians found to be in severe stress. However it is not statistically significant.

Marital status

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Married	34	48.6	48.6	48.6
	Unmarried	36	51.4	51.4	100.0
	Total	70	100.0	100.0	

Speciality

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Admin	2	2.9	2.9	2.9
	Internal Medicine	13	18.6	18.6	21.4
	Endocrinology	2	2.9	2.9	24.3
	Accident & emergency	35	50.0	50.0	74.3

Family Medicine	2	2.9	2.9	77.1
Cardiology	2	2.9	2.9	80.0
Gastroenterology	3	4.3	4.3	84.3
Orthopedics	2	2.9	2.9	87.1
Anesthesia	2	2.9	2.9	90.0
Radiology	2	2.9	2.9	92.9
Critical care	5	7.1	7.1	100.0
Total	70	100.0	100.0	

			score_3grp			Total
			<8	8-14	15-28	
Speciality	Admin	Count	1	1	0	2
		% within Speciality	.5	.5	.0	1.0
	Internal Medicine	Count	9	4	0	13
		% within Speciality	.7	.3	.0	1.0
	Endocrinology	Count	2	0	0	2
		% within Speciality	1.0	.0	.0	1.0
	Accident & emergency	Count	16	16	3	35
		% within Speciality	.5	.5	.1	1.0
	Family Medicine	Count	1	0	1	2
		% within Speciality	.5	.0	.5	1.0
	Cardiology	Count	2	0	0	2
		% within Speciality	1.0	.0	.0	1.0
	Gastroenterology	Count	2	1	0	3
		% within Speciality	.7	.3	.0	1.0
	Orthopedics	Count	2	0	0	2
		% within Speciality	1.0	.0	.0	1.0
	Anesthesia	Count	1	1	0	2
		% within Speciality	.5	.5	.0	1.0
	Radiology	Count	2	0	0	2
		% within Speciality	1.0	.0	.0	1.0
	Critical care	Count	2	3	0	5
		% within Speciality	.4	.6	.0	1.0
	Total	Count	40	26	4	70
		% within Speciality	.6	.4	.1	1.0

p=0.54

Comments: The emergency physicians are having higher GHQ score as compared to other specialties.

Work Pattern

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Clinical	46	65.7	65.7	65.7
	Others	13	18.6	18.6	84.3
	Admin+clinical	2	2.9	2.9	87.1
	Clinical+Others	9	12.9	12.9	100.0
	Total	70	100.0	100.0	

Comments: The physicians working in the clinical departments are experiencing moderate to severe stress it is statistically significant (p value0.01)

			score_3grp			
			< 8	8-14	15-28	Total
Work_ place	Hospital	Count	33	20	3	56
		% within Work_place	.6	.4	.1	1.0
	Clinical	Count	0	0	1	1
		% within Work_place	.0	.0	1.0	1.0
	Others	Count	4	5	0	9
		% within Work_place	.4	.6	.0	1.0
	Hosp+clinic	Count	3	1	0	4
		% within Work_place	.8	.3	.0	1.0
Total		Count	40	26	4	70
		% within Work_place	.6	.4	.1	1.0

P=0.01

			score_3grp			
			< 8	8-14	15-28	Total
Nt_duty	Present	Count	29	23	4	56
		% within Nt_duty	.5	.4	.1	1.0
	Absent	Count	11	3	0	14
		% within Nt_duty	.8	.2	.0	1.0
Total		Count	40	26	4	70
		% within Nt_duty	.6	.4	.1	1.0

p=0.17

Comments: 41% of physicians who do nightshift seen to have moderately stressed however this is not statistically significant.

score

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	.00	20	28.6	28.6	28.6
	1.00	3	4.3	4.3	32.9
	2.00	3	4.3	4.3	37.1
	3.00	4	5.7	5.7	42.9
	4.00	3	4.3	4.3	47.1
	5.00	6	8.6	8.6	55.7
	6.00	1	1.4	1.4	57.1
	8.00	4	5.7	5.7	62.9
	9.00	1	1.4	1.4	64.3
	10.00	8	11.4	11.4	75.7
	11.00	4	5.7	5.7	81.4
	12.00	3	4.3	4.3	85.7

13.00	6	8.6	8.6	94.3
16.00	1	1.4	1.4	95.7
19.00	1	1.4	1.4	97.1
22.00	1	1.4	1.4	98.6
24.00	1	1.4	1.4	100.0
Total	70	100.0	100.0	

Score GRP

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	<=8	40	57.1	57.1	57.1
	8-14	26	37.1	37.1	94.3
	15-21	2	2.9	2.9	97.1
	22-28	2	2.9	2.9	100.0
	Total	70	100.0	100.0	

Comments: 37% of physicians found to have moderate stress

Result

Though majority of responders initially felt that they perfectly well, 25% realised the need of a good tonic/ refreshment. Later 36% of physicians felt that they are not feeling well in daily lives. Approximately 64% felt that they were feeling run down and out of sorts. Many (41%) experience headaches due to work stress. 41% of physicians complained both hot and cold spells recently. A large number of physicians had lost their sleep. 47% of physicians shared that they are experiencing difficulty in sleeping once they are off and feel that they are in constant strain. Many (49%) are getting edgy and bad tempered easily. 21% of physicians feel that they are nervous all the time. Though majority are able to keep themselves busy some are not. Some are taking longer time to complete their routine tasks. Though 89% feel they are able to do things better than usual a large number of physicians contradict them. A large number of physicians are taking longer time to complete their routine tasks. 20% feel they are un able to do things better than usual. 10% feel less satisfied in the way they have carried their task. Many physicians believe that they are not playing less useful part in their day today life. A small number (4%) of physicians had problems in decision making. Some (9%) say that they are not able to enjoy their day today activities same as usual. A large chunk (9%) feels themselves to be worthless though another small group (3%) believe that their life is entirely hopeless. A small population (3%) of physicians confesses that the idea of making away with themselves actually crossed their minds. Even some say (4%) that at some point of time they thought of taking their own life. 6% of physicians feel that their nerves are so bad that they are unable to do anything.. 4% physicians says that the idea of taking their life has crossed their mind.

Discussion

Stress levels or psychological stress is one of the important factor which is often overlooked in day to day life. It has been seen in various studies increased psychological stress causes various physiological changes in human body and directly or indirectly affect one's health.

The higher stress levels also leads a person to adopt unhealthy life styles and push towards addictions.

This study is done keeping in mind about the physician's stress levels as it is found in literature that physicians are exposed to number of stressors and it is also being seen that if physicians are having more stress the patient care is decreased above all the physician him/herself becomes ill.

As a saying says "HEALERS TO BE HEALED FIRST". Knowing about the stress levels and attempts to reduce the stress levels is of paramount importance.

For few demographic factors i.e. the working place(in- hospital) and type of work(clinical) the physicians having these in common found to have moderate to severe stress and this was statistically significant . The emergency physicians are having higher GHQ score as compared to other specialties but this was not statistically significant because of small sample size and number of physicians from other specialties are also less to do good comparison. In 37% of physicians the GHQ score is high(from

moderate to severe stress) this was not statistically significant .

There are few limitations to this study and hence the results cannot be generalised because it is a single centre study and a small sample size.

Conclusion

There is trend of higher GHQ scores that correlates with the higher stress levels amongst the emergency physicians and the physicians working in-hospital and taking care of clinical departments.

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Increasing Electrocardiograph Speed does not Improve the Accuracy of Diagnosis of Narrow Complex Tachycardias

Subhajit Sen*, Indraneel Dasgupta**

Abstract

Background: The diagnostic dilemma of ECG rhythm diagnosis is very common for patients presenting in ED and Critical Care Units. A widely practiced method for diagnosing ECG rhythms of narrow-complex tachycardia is the use of double-speed (50mm/sec) ECGs in addition to the 25mm/sec speed ECG. Though widely practiced, accuracy of diagnosis by this method has not been evaluated adequately. *Method:* This single-center, single-blinded, comparative, questionnaire-based study was done amongst Emergency and Critical Care Physicians to assess whether adding a double speed ECG help in making a correct diagnosis of narrow-complex tachycardia. *Results:* The study did not show any significant improvement in diagnostic accuracy with use of 50mm/sec speed ECG. 26 ECGs were interpreted by 35 observers, each ECG set was diagnosed twice, once with 25mm speed only and once 25mm and 50mm speed together. Correct diagnosis was made in 534/910 (58.68%) in the standard group and 537/910 (59.01%) with the addition of the 50mm/s ECG. None of the narrow complex tachycardia revealed any improvement in diagnostic accuracy with the aid of double-speed ECG. Proportion of correct diagnosis by physicians of different years of experience with the help of double-speed ECG is not greater than that with standard speed ECG. The proportion of correct diagnosis by physicians with opinion that double-speed ECGs are helpful is also not better than physicians who do not find double-speed ECGs helpful. *Conclusion:* Though widely practiced in diagnosing narrow complex tachycardia, double speed ECG is not a very accurate tool. Perhaps physicians need more training and practice in interpreting double speed ECGs for more accurate information of rhythm analysis.

Keywords: Tachycardia; ECG Rhythm; Atrial Fibrillation.

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Introduction

The clinical manifestations resulting from tachycardia are a common reason for presentation to Emergency Department. The narrow complex tachycardia comprises of Atrial fibrillation (AF), Atrial-flutter (AFl), Paroxysmal Supraventricular tachycardia (PSVT), AV nodal re-entrant Tachycardia (AVNRT), AV reciprocating tachycardia (AVRT), Multifocal atrial tachycardia (MAT) and Sinus tachycardia [1, 2, 6]. ECG remains the primary tool in arrhythmia analysis. However, the diagnosis of tachycardia is often difficult on standard ECG. In rapid narrow complex tachycardia, the intervals may be too narrow to appreciate qualities such as irregularity and flutter waves. The arrhythmias are often more complicated due to presence of bundle branch blocks. The patients with narrow complex tachycardia are difficult to diagnose using the 12-lead ECG. Hence, technique for improving diagnosis by a simple, quick, noninvasive test such as the 50 mm/s ECG is therefore attractive and very commonly used. The only study to investigate the clinical utility of this strategy suggests that the addition of a 50 mm/s ECG to a standard 25 mm/s ECG improves diagnostic accuracy in narrow complex tachycardia [3]. The study also suggests that inappropriate use of

adenosine may be reduced by implementing this strategy, as interpreters are more likely to correctly diagnose difficult tracings [4, 5, 7, 8, 9]. However, more research is needed in this topic. This study was also aimed to determine if addition of a 50mm/s ECG aids in the correct diagnosis of narrow complex tachycardia when compared to standard speed (25 mm/s) ECG. We hypothesized that this addition improves the diagnostic accuracy of such tachycardia.

Methodology

The study was conducted as a single-blinded comparative trial at an academic Emergency Department at a corporate hospital amongst Emergency Physicians and Critical Care Physicians.

ECGs were selected from the patients who have attended the Hospital Emergency and had tachycardia on presentation. The ECGs were selected based on the difficulty of diagnosis. These ECGs were considered difficult based on the initial difficulty in interpretation or perceived difficulty by the authors. All ECGs were printed at 25 mm/s and 50 mm/s speeds simultaneously. The gold standard for each patient's diagnosis was based on the final diagnosis from the patient's medical record and a second opinion by a Senior Consultant Cardiologist.

Initially, thirty sets of ECGs (both 25 mm speed and 50 mm speed) were selected for the final questionnaire. Four ECGs were excluded due to a disagreement between the official diagnosis and the cardiologist's diagnosis. The finalized ECGs were marked by a code to prevent mixing-up between the two sets of same patient's ECG and also to "blind" the interpreters.

Thirty-five doctors, with different years of experience were asked to diagnose the ECGs. All the doctors who were asked to diagnose ECGs worked in Emergency Department or Critical-care, areas which require for a rapid interpretation of ECGs, sometimes even without any available past medical history. The observers were instructed that all hypothetical patients were hemodynamically stable but were masked to all other clinical information and asked to give the diagnosis based on the ECG interpretation only. The observers were initially given ECGs at 25 mm/s. Each observer was asked to diagnose the ECG rhythm (Sinus tachycardia, Atrial fibrillation/ flutter, Supraventricular tachycardia, additional blocks etc). The questionnaire also asked about the participant's years of experience after graduation. After completion of the ECG observation with 25mm/sec the observers were asked not to discuss the ECGs with other readers until completion of the study.

After a gap of 2-weeks, the same physicians were given the same ECGs at both the standard speed (25mm/s) and at rapid speed (50 mm/s) together. For the hypothetical patient population, the physicians were asked to interpret the rhythm based upon the ECGs. Also, opinion of the participants about usefulness of 50mm speed ECG in diagnosing narrow complex tachycardia was taken. The answers to this question are based on a Likert scale as following: *Not helpful, Helpful, Very Helpful, Essential*. Any correlation between diagnostic accuracy of doctors with years of experience was also searched by comparing diagnostic accuracy of doctors with different years of experience.

Diagnostic accuracy between the two groups was compared by using McNemar's Chi-squared test. All tests except exact binomial test are large sample tests. Comparisons between categorical variables are performed using McNemar's Chi-squared test and k-sample test for equality of proportions. In these tests, a *P*-value <0.05 are considered significant.

Results

Twenty-six ECGs were interpreted by thirty-five observers, yielding 910 observations in total (each ECG set was diagnosed twice, once with 25mm speed only and once 25mm and 50mm speed together).

The distribution of cardiac rhythms of the study was as follows:

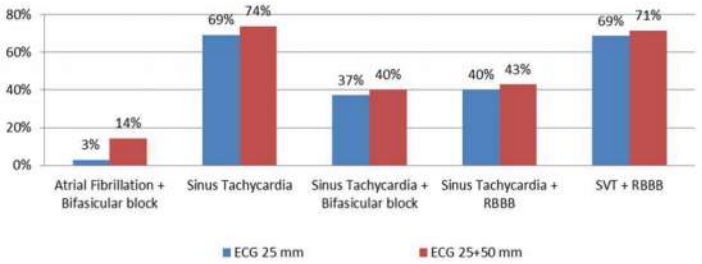
Rhythm	Quantity
Sinus tachycardia:	10
Sinus tachycardia with RBBB	1
Sinus tachycardia with bifascicular-block	1
Atrial flutter	2
Atrial fibrillation	5
Atrial fibrillation with bifascicular-block	1
Junctional rhythm	1
Supraventricular tachycardia	4
Supraventricular tachycardia with RBBB	1

Though widely believed to be useful in more accurate diagnosis of narrow-complex tachycardia, use of 50mm speed ECGs in addition to the standard ECG failed to show any significant improvement in interpretation of the various rhythms. Correct diagnosis was made in 534/910 in the standard group. With the addition of the 50mm/s ECG, correct diagnosis improved only to 537/910. Proportion of correct diagnosis with ECG 25mm and with ECG 25 mm + 50 mm speed is same with P-value 0.8538

ECG 25 mm (Mean ± Se)	ECG 25 + 50 mm (Mean ± Se)	McNemar's Chi-squared test P-value (two sided)
58.68% ± 1.63%	59.01% ± 1.63%	0.8538

Sl. No.	Disease category	Sample size	ECG 25 mm (Mean ± Se)	ECG 25+50 mm (Mean ± Se)	McNemar's test (one sided) P-value
1.	Atrial Fibrillation + Bifasccicular block	35	2.86% ± 2.90%	14.29% ± 6.09%	0.0668
2.	Sinus Tachycardia	350	69.14% ± 2.48%	73.71% ± 2.36%	0.0549
3.	Sinus Tachycardia + Bifascicular block	35	37.14% ± 8.41%	40.00% ± 8.52%	0.5000
4.	Sinus Tachycardia + RBBB	35	40.00% ± 8.52%	42.86% ± 8.61%	0.5000
5.	SVT + RBBB	35	68.57% ± 8.08%	71.43% ± 7.86%	0.5000

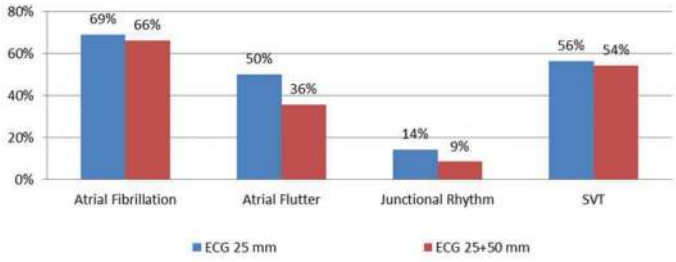
Fig. 1: Proportion of correct diagnosis of different rhythms.



As evident in the graph, there is a marginal improvement in the diagnostic accuracy of the above rhythms; however the statistical analysis using McNemar's Chi-square test reveals that the diagnostic accuracy is not significantly better using the 50mm speed ECG. The p-values of all the comparisons are non-significant with values 0.05 or more.

Sl. No.	Disease category	Sample size	ECG 25 mm (Mean ± Se)	ECG 25+50 mm (Mean ± Se)	McNemar's test (both sided) P-value	Exact Binomial test (one sided) P value
6.	Atrial Fibrillation	175	69.14% ± 3.51%	66.29% ± 3.59%	0.6025	0.7825
7.	Atrial Flutter	70	50.00% ±6.06%	35.71% ± 5.81%	0.0550	0.9915
8.	Junctional Rhythm	35	14.29% ± 6.09%	8.57% ± 4.87%	0.6831	0.8906
9.	SVT	140	56.43% ± 4.22%	54.29% ± 4.24%	0.7656	0.7243

Fig. 2: Proportion of correct diagnosis with ECG 25+50 mm and ECG 25 mm.



It is clear from this table and graph that use of 50mm/sec speed ECG has not been helpful in these rhythm analyses. The use of double speed ECG has produced a less accurate diagnosis compared with those made by normal speed ECG only. Since there was a deterioration in performance, p-value using Exact Binomial test (one sided), was used for interpretation of results, and it clearly shows no advantage in diagnostic accuracy using the 50mm speed ECG.

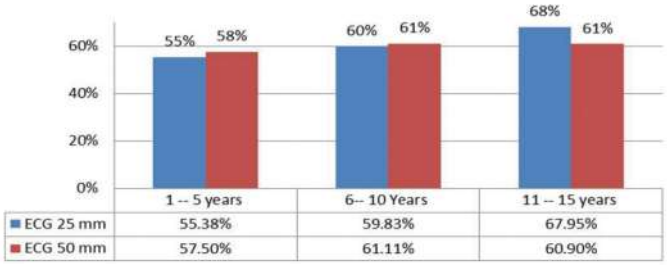
Proportions of correct diagnosis of respective diseases with ECG 25mm differ significantly amongst physicians (P value < 2.2e-16 < 0.0001, value of test statistic 112.7488) and the proportions of correct diagnosis of respective diseases with ECG 25+50 mm also differ significantly (P value < 2.2e-16 < 0.0001,

value of test statistic 129.1058). Test values correspond to 9-sample test for equality of proportions.

Proportion of correct diagnosis by Physicians of different years of experience

The ECG diagnostic accuracy of physicians with different years of experience was also observed in the study to determine whether years of experience improves the interpretation skills of double speed ECGs. The observer physicians were divided into 3 groups according to years of experience: 1- 5 years; 6 -10 years and 11- 15 years.

Fig. 3: Correct Diagnosis by Physicians of Different Year of Experience with Ecg 25+50 Mm and Ecg 25 Mm



Proportion of correct diagnosis by physicians of groups 1- 5 & 6 -10 years of experience with ECG 25+50 mm is not significantly greater than that with ECG 25 mm. Test values correspond to McNemar's Chi-square test (one sided).

Sl. No.	Years of experience of Physicians	Sample size	ECG 25 mm (Mean ± Se)	ECG 25 + 50 mm (Mean ± Se)	McNemar's test (one sided) P value
1	1 -- 5 Years	520	55.38% ± 2.18%	57.50% ± 2.17%	0.21815
2	6 -- 10 Years	234	59.83% ± 3.22%	61.11% ± 3.20%	0.3937

Though a slight improvement is noted in more experienced physicians; i.e., 11-15 years than the other groups in interpreting with the 25mm speed ECGs, there is no improvement, rather, deterioration in their interpretation with the aid of 25+50mm speed ECGs (P-value 0.9638). Test value corresponds to Exact Binomial test (one sided).

Sl. No.	Years of experience of Physicians	Sample size	ECG 25 mm (Mean ± Se)	ECG 25 + 50 mm (Mean ± Se)	McNemar's test (both sided) P value)	Exact Binomial test (one sided) P value
3	11 -- 15 Years	156	67.95% ± 3.76%	60.90% ± 3.93%	0.1360	0.9638

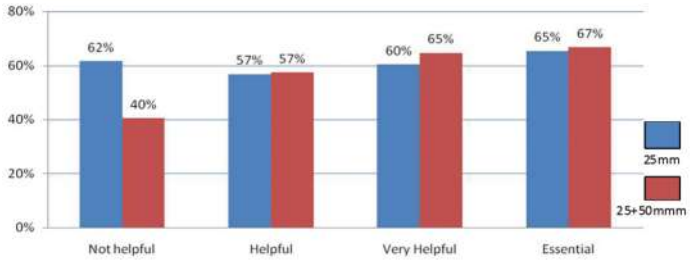
Proportions of correct diagnosis with ECG 25mm by physicians of different years of experience differ significantly (P value 0.01846, value of test statistic 7.9838) but the proportions with ECG 25+50 mm by physicians of different years of experience do not differ significantly (P value 0.5635, value of test statistic 1.147).Test values correspond to three sample test for equality of proportions (both sided). Thus, the study finds years of experience has no significant contribution over correct diagnosis of diseases with double-speed ECG though it's contribution is significant in correct diagnosis with ECG 25 mm only.

Proportion of correct diagnosis by Physicians of different opinions on utility of double-speed ECG

Proportion of correct diagnosis by physicians of the opinion 50mm "Not Helpful" with ECG 25+50 mm is less that with ECG 25 mm. Using Exact binomial test (one sided), the said proportion with ECG 25+50 mm is significantly less than that with ECG 25 mm (P value 0.01089). Though, proportion of correct diagnosis with ECG 25+50 mm by physicians of the other 3 groups is marginally better than diagnosis with 25mm speed ECG, McNemar's Chi-squared test P-values are >0.05 in all thus showing a non-significant improvement in all the groups.

Proportions of correct diagnosis with ECG 25+50 mm by physicians of different opinions on utility of ECG 50 mm differ significantly (P value 0.004777, value of test statistic 12.9362), though the proportions with ECG 25mm do not differ significantly (P value 0.4441, value of test statistic 2.677). Test values correspond to four sample test for equality of proportions (one sided).

Fig. 4: Proportions of correct diagnosis with ECG 25+50 mm and with ECG 25mm by physicians of different opinions



Discussion

Diagnoses of rhythm in high speed tachycardia are difficult and challenging. Some tachycardia occur secondary to fever, anxiety, pain etc. and responds to appropriate treatment of the pathologic insult. Some other tachycardia like Atrial fibrillation, SVT may indeed cause complaints like palpitations, light-headedness from poor perfusion and needs identification to be adequately managed. For diagnosis of rhythms, physicians rely chiefly on ECGs, at times a double speed ECG is used to aid in diagnosing very fast narrow-complex tachycardia. The study was aimed to evaluate the diagnostic accuracy using the double speed ECG.

Though commonly practiced as a diagnostic aid, accuracy of diagnosing the rhythm in narrow-complex tachycardia, using double speed ECGs is unsatisfactory and not above questionable merit. The physician are prone to give inaccurate diagnosis of rhythms when asked to make diagnosis with the aid of a static picture of rhythms by an ECG rhythm strip, specially without any knowledge about patients age, hemodynamic status, presenting complains etc. The accuracy of diagnosis is unaffected by the physicians experience or comfort level in interpreting ECGs. No specific narrow-complex rhythm can be said to be more accurately identifiable with the help of double-speed ECGs. At times, double speed ECGs may be more misleading than helpful in making diagnosis. Not all physicians are comfortable with the interpretation of double-speed ECG as they are used sparingly or by more qualified colleagues. The static picture as seen in ECG strip may give inadequate information of rhythm in fast paced tachycardia. Physicians also tend to consider the hemodynamic status and illness of patient when making a diagnosis, absence of such information too can mislead in making the correct diagnosis.

A weakness of the present study is that it was done on a single center with a limited sample size. A further weakness of the qualitative result was that we were unable to expand upon the results as we did not collect any information on aids and difficulties faced by physicians on making their diagnosis of various rhythms by the ECG strips or what they might think to be more helpful in diagnosing narrow-complex tachycardia.

Therefore, more research needs to be directed to identify information and methods which may help in diagnosing difficult narrow-complex rhythms and increase diagnostic accuracy.

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Ethical approval

The study was approved by the Ethical Committee of Peerless Hospital & B K Roy Research Centre, Kolkata

Conflicts of interest

The authors declare that there is no conflict of interest.

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Original Research Article

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A Prospective Study in the Indian Emergency Setting to Sample the Significance of Undetected Hypertension Presenting as Raised Blood Pressure

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Abstract

Study objective: To determine if the raised blood pressure at an ED visit was due to pain and anxiety or undetected hypertension. *Methods:* Patients visiting the Emergency department of Max Balaji Super Specialty Hospital, Delhi in a period of 6 months of 2013 were recruited for the study and a sample of 72 patients generated. Patients who passed the inclusion and exclusion criteria had an initial and a repeat ED blood pressure that were increased (SBP e"140 or DBP e"90 mm Hg). To gauge the causality of pain and anxiety in the rise in BP we used an ED Pain score, the Verbal descriptor scale and ED Anxiety score, the Beck's Anxiety Scale. With 95%CI and 10% precision we enrolled a total of 72 subjects in the study. *Results:* Out of 72 patients who fulfilled the criteria for study 40.28% were male and 59.72 were female. Average age of the participants was 48 years. Patients with raised ED blood pressure were older and more of females than males. The mean pain score (SD) for the sample was 4.1 and mean (SD) anxiety score was 37.8. Anxiety score showed a positive correlation change in systolic ED blood pressure than diastolic ED blood pressure. Mean (SBP) systolic ED blood pressure at the First reading was Mean 154.6 (SD 6.7) and Second reading was a Mean 128 (SD 9.3).Mean (DBP) diastolic ED blood pressure at the First reading was Mean 94.8 (SD 5.2) and second reading was a Mean 84.15 (SD 6.88). *Conclusion:* Even in Indian scenario non-hypertensive patients with raised blood pressure in ED, with pain and anxiety relief still remained in pre-hypertensive stage suggestive for routine reassessment and regular blood pressure follow up with the primary physician and lifestyle modification.

Keyword: Not Provided

Introduction

Hypertension has been identified as one of the leading risk factors for mortality, and is ranked third as a cause of disability-adjusted life-years [1]. Existing data suggests that the prevalence of hypertension has remained stable or has decreased in economically developed countries during the past decade, while it has increased in developing countries [2]. Given the rising prevalence of hypertension in developing countries undergoing epidemiological transition like India, increased awareness, treatment, and control of high blood pressure are critical to the reduction of cardiovascular disease risk and prevention of the associated burden of illness. This study was undertaken with the objective to gather both epidemiological data and data on awareness and control of hypertension in Delhi which represents urban north India. In 2003, the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure published their seventh report, which redefined hypertension categories and created a new category, prehypertension (systolic blood pressure 120 to 139 mm Hg or diastolic blood pressure 80 to 89 mm Hg) [5, 6]. The emergency department (ED) may be able to play an important public health role in early detection and prevention of hypertension by identifying patients who have not yet been diagnosed.

Recognizing the potential role of the ED and the importance of the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, the American College of Emergency Physicians published a clinical policy in 2006, recommending that patients with persistently increased blood pressure readings (i.e., 2 or more systolic blood pressure readings greater than 140 mm Hg) referred for follow-up of possible hypertension. This policy also recognizes barriers to routine ED screening and referral, including (1) the general belief by both the physician and the patient that blood pressure screening is beyond the scope of the ED and (2) the emergency experience may result in false increased blood pressures because of pain and anxiety [7]. In an effort to address these barriers, evidence is needed to ED setting and to understand the relationship between ED blood pressures and pain and anxiety.

Methodology

Study Design and Oversight

We conducted a prospective cohort study. The Institutional scientific committee and Institutional Ethics Committee of the Max Super Specialty Hospital, Patparganj, Delhi; approved the protocol. The Study was registered to *www.ctri.nic.in* prior to recruitment of the first patient into the study. Those patients who provided voluntary written informed consent were recruited into the study. The time frame for conduction of the study was a period of 5 months study from January 2013 to May 2013.

Sites and Patients

We recruited a total of 72 participants of the 96 eligible participants from the ED of Max Super Specialty Hospital, Patparganj, Delhi that has approximately 72,000 adult ED patient visits yearly. Patients eligible for inclusion were if they were at least 18 years of age.

All the patients were included in the study that had an initial ED SBP (systolic blood pressure) e"140 or DBP (diastolic blood pressure) e"90 mm Hg. When a repeat set of BP was measured SBP e"140 or DBP e"90 mm Hg was recorded were included. Another inclusion criterion was patients without any history of HTN (hypertension).

The patients excluded from the study were who were admitted to the hospital, , homeless, who were unable to do BP measurements at home, pregnant with previous or recently diagnosed history of medical instability or psychiatric illness. In the final data patients with inadequate contact information were excluded. Although there are no specific blood pressure upper limits for exclusion, we excluded patients if the emergency physician prescribed an antihypertensive agent at discharge.

Study Interventions

After a brief patient interview, ED physicians instructed subjects on use of home BP monitor. The pain and anxiety of each participant was evaluated by using individual scores including the *ED Pain score* (0-10 verbal descriptor scale) and an ED Anxiety score, the *Beck's Anxiety Scale*. The Beck's scale Scores patients' report 20questions grading low to high anxiety from *Not At All* as score 1 to *Very Much So* as score 4.

On an Initial ED SBP e"140 or DBP e"90 mm Hg recording in a patient and no history of HTN, patient recruitment was evaluated. A repeat ED SBP e"140 or DBP e"90 mm Hg was recorded too. The highest and lowest SBP and DBP recordings were deleted. Mean monitor SBP and DBP were calculated for each individual.

Statistical Analysis

Statistical calculations at the start and at the time of final analysis of the data were done by an Institutional Statistician who was blinded from the primary data. The prevalence of hypertension with a cut off mark of 140/90 mm of Hg is 24.9%. Sample size was calculated by n-Master (2.0) software. Sample size required for 95% confidence interval at 10 % precision minimum inclusion number of subjects in the study was calculated to be 75. Statistical Analysis was done using Chi-square and Fisher's exact test (categorical variables), t test (continuous variables), Pearson correlation coefficients to determine the correlations between the change from ED to home SBP and DBP with the ED mean pain score and anxiety score.

Results

Total no of patient approached were 96 who visited the Emergency Department fulfilling the criteria for our study, out of which only 72 patient agreed and gave consent for study, as the rest did not gave consent for different reasons. Out of 72 patients who

fulfilled the criteria for study 40.28% were male and 59.72% were female. Average age of the participants were 48 years, out of them 33% were non vegetarians and 66% were vegetarian. Family history of hypertension was present in 27.78% patients. Patients with raised ED blood pressure were older and more likely to be obese and female gender. The mean pain score (SD) for the sample was 4.1 and mean (SD) anxiety score was 37.8. Anxiety score showed a positive correlation change in systolic ED blood pressure than diastolic ED blood pressure. Mean (SBP) systolic ED blood pressure at the First reading was Mean 154.6 (SD 6.7) and Second reading was a Mean 128 (SD 9.3).Mean (DBP) diastolic ED blood pressure at the First reading was Mean 94.8 (SD 5.2) and second reading was a Mean 84.15 (SD 6.88). Anxiety score showed a positive correlation change in systolic ED blood pressure than diastolic ED blood pressure.

Table 1

96	Patients met inclusion criteria
24	Patients did not consent for the study
29	Patients were male
43	Patients were female
48	Patients were vegetarians
20	Patients had family history of hypertension

The table below lists the average blood pressures (systolic and diastolic) before and after survey in ED.

Table 2

Systolic	Min	Max	Mean	SD	Mean+SD	Mean - SD
Before Survey	145	180	154.64	6.71	161.35	147.927
After Survey	110	140	128.60	9.34	137.93	119.26
Diastolic	**Min**	**Max**	**Mean**	**SD**	**Mean+SD**	**Mean - SD**
Before Survey	84	110	94.89	5.30	100.19	89.59
After Survey	70	90	84.15	6.88	91.03	77.27

Limitations

Our analysis was limited to small time duration of only five months. Because many of the patients had to suffice multiple exclusion criteria, the presentation may be variable during a particular period of time. Nevertheless, we are of the belief that the obtained sample size is adequate to formulate a preliminary judgment of the original goal planned for the study. Secondly, this study was conducted at a single hospital setup which reduces the precision of predicting the application to the country as a whole. A similar limitation to the study was that it was conducted in a private institute wherein only a set group of economically well to do populace visits and the scenario may well end up being completely varied if conducted including patients from all possible setups including only government run institutes and charitable trusts. Finally only one invigilator was involved in collection of the data and hence introduces the possibilities of observer bias which could be eliminated by induction of more number of investigation members.

Discussion

Undiagnosed hypertension is common amongst the general population of every country, especially developing ones like India and presents a challenge to the emergency physicians. The minor practice of attention to raised blood pressure during a single ED visit could allow for earlier detection and prevention of long term morbidity and mortality associated with undetected hypertension. However no previous published studies were found that adequately describe the significance of single raised blood pressure measurement in Emergency Department in the Indian scenario. We conducted a prospective cohort study using increased blood pressure in the first ED visit along with Pain and Anxiety score at the ED Visit to validate these findings. In this cohort we had blood pressure readings that met the 7th report of JNC in prevention, detection, evaluation and treatment of raised blood pressure criteria for hypertension. Most patients had blood pressure in the pre-hypertensive range. As blood pressure in the pre-hypertensive range is associated with increased cardiovascular risk, referral for overall cardiovascular risk assessment and lifestyle modifications is warranted. ED clinicians may believe that patients with raised blood pressure who are anxious or in pain may be normotensive after their acute problems has passed over therefore do not require referral for evaluation and management of hypertension. We found a similar correlation with blood pressure changes in ED. However, simultaneously it was also observed that considerable percentages of patient were still in pre-hypertensive stage at the time of discharge from ED.

In conclusion, a single emergency department visit could bring about a major change in the outlook and outcome of a serious health problem like hypertension. However as per the date till date, further more studies are a definitive need to assess and quantify the right methodology and processes to validate the same.

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Review Article

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BNP and its Status as a Biomarker in Acute Ischemic Stroke

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Abstract

B-Type natriuretic peptide (BNP) has established itself as an important cardiovascular and cardio renal biomarker. Stroke is an emergency having high mortality, morbidity, social and economic implications. Acute ischemic stroke (AIS) accounts for approximately 70% of all strokes and is caused by embolic or atherosclerotic occlusion in the cerebral vessels. Identification of a biomarker for risk, severity and prognosis of stroke would be of great benefit. The mechanism by which the plasma levels of BNP are increased in patients with AIS independently of heart diseases is not clearly defined but the levels of BNP has shown a strong correlation with cardio-embolic stroke and has established its role as a surrogate marker for the same. Biomarkers like BNP should be used to supplement clinically guided therapy and not to substitute it. Proper interpretation of BNP would surely make the diagnosis, management and risk stratification better for stroke subjects.

Keywords: BNP; Biomarker; Acute Ischemic Stroke.

Introduction

Basic science discoveries and technological progress in the last decade have introduced a variety of circulating molecules in clinical research referred to as biomarkers. B-type natriuretic peptide (BNP) has established itself as an important cardiovascular and cardio renal biomarker. Stroke is defined as sudden onset of focal and global neurological symptoms due to cerebral blood vessels leading to haemorrhage and ischemia in brain [1]. Stroke is an emergency having high mortality, morbidity, social and economic implications. Acute ischemic stroke (AIS) accounts for approximately 70% of all strokes and is caused by embolic or atherosclerotic occlusion in the cerebral vessels. Identification of a biomarker for risk, severity and prognosis of stroke would be of great benefit. BNP produced as a result of cardiovascular changes following ischemic stroke has an important role in the hemodynamic of these patients. The mechanism by which the plasma levels of BNP are increased in patients with AIS independently of heart diseases is not clearly defined but the levels of BNP has shown a strong correlation with cardio-embolic stroke and has established its role as a surrogate marker for the same. Robust, widely-available, rapidly processed, inexpensive biomarkers such as BNP could potentially be used in the future to guide management of complex cerebrovascular patients in order to maximize their potential for recovery.

History of BNP and natriuretic peptides

The history of B-type natriuretic peptide (BNP) dates back to 1988 when it was first isolated from porcine brain tissue. It was subsequently also detected in rat brain where its expression is upregulated by middle cerebral artery occlusion[2,3] BNP belongs to the family of natriuretic peptides which comprises of three structurally related molecules, atrial natriuretic peptide (ANP), BNP and C-type natriuretic peptide (CNP) encoded by a gene NPPC. In addition to the mammalian natriuretic peptides (ANP, BNP and CNP), other natriuretic peptides with similar structure and properties have been isolated elsewhere in the animal kingdom. Trevonen (1998) described a salmon natriuretic peptide known as salmon cardiac peptide [4] and dendroaspis natriuretic peptide (DNP) is found in venom of the green mamba [5].

Physiology, Synthesis & functions of BNP

BNP is a 32-amino acid polypeptide released mainly from the ventricular myocardial cells and to some extent from cardiac fibroblasts in response to stretching secondary to pressure or volume overload. The release is modulated by calcium ions. BNP is synthesized as a 134-amino acid preprohormone (preproBNP), encoded by the human gene NPPB. Removal of the 25-residue N-terminal signal peptide generates the prohormone, proBNP, which is stored intracellularly as an O-linked glycoprotein, proBNP is subsequently cleaved between arginine-102 and serine-103 by a specific convertase, corin into NT-proBNP and the biologically active 32-amino acid polypeptide BNP-32, which are secreted into the blood in equimolar amounts. BNP is cleared from plasma through binding to the natriuretic peptide clearance receptor type C, but it seems relatively resistant to proteolysis by neutral endopeptidase NEP 24.11. The biological effects include diuresis, vasodilatation, and inhibition of renin and aldosterone production thus leading to natriuresis and inhibition of cardiac and vascular myocyte growth. BNP binds to and activates the atrial natriuretic factor receptors (NPRA). The biological half-life of BNP is twice as long as that of ANP.

Factors affecting levels of BNP

BNP is measured by immunoassay. There is no single cut off value to differentiate a normal level from an abnormal level. The value of less than 50pg per ml has a sensitivity of 97% and specificity of 62% in ruling out acute decompensated heart failure. There is a diagnostic 'grey area' between 100pg/ml & 500pg/ml. As with any biomarker several factors should be considered when interpreting BNP levels. BNP increases with age and is higher in women subjects without cardiovascular disease or cardiac dysfunction. An inverse relation exists between BNP and body mass index. Renal dysfunction increases the BNP levels. Cardiovascular drugs such as diuretics, spironolactone, angiotensin converting enzyme inhibitor and angiotensin receptor blockers may decrease BNP levels while with beta blockers the levels may increase for weeks and then decrease after a few months. There also exists intra-individual biologic variation. Several cardiac diseases, ventricular assist devices, sepsis etc., also have effect on the levels.

Brain Ischemia & BNP

There is limited data on the physiological and pathological role of BNP in human brain. Hypoxia increases cardiac BNP gene expression in pigs and circulating BNP levels in humans, and occlusion of the middle cerebral artery stimulates BNP mRNA expression in rat brain tissues [6]. Moreover, the human BNP gene promoter region contains a hypoxia-inducible factor (HIF)-1 binding site and BNP gene expression is activated by HIF-1 In this context, considerable attention should be paid to the positive correlation between brain infarct volume and the plasma BNP level in AIS and the possibility that the infarct or ischemic area in the brain could be a potential source of circulating BNP. Studies have shown that elevated plasma NTproBNP levels are involved in the pathogenesis of brain edema in ischemic and hemorrhagic stroke [7]. These findings suggest that the ischemic brain itself may also release NT-proBNP into the circulation. It has been reported that S-100protein, a calcium-binding protein abundant in glial and Schwann cells, is increased in blood and cerebrospinal fluid (CSF) after ischemic stroke and its plasma concentration correlates positively with the size of infarct volume [8]. Thus, it may be important to investigate whether the concentration of BNP in CSF would be increased after brain infarction or ischemia and BNP released from the ischemic brain tissues would exert a neuro-protective effect around the ischemic area. Handke et al reported that left atrial appendage (LAA) flow was closely related to elevated thromboembolic risks in the cerebral ischemia patients irrespective of the basic rhythm. To detect LAA flow transesophageal echocardiography (TEE) has to be done during the acute stroke. TEE being invasive, requiring expertise and having risk of pneumonia, a non-invasive marker to predict cardio-embolic stroke would be beneficial.

BNP and Stroke

Increase in the life expectancy of humans has led to increased number of stroke patients. Early diagnosis is required for applying efficient treatment like thrombolysis in ischemic stroke. Subsequent to differentiation of ischemic and haemorrhagic stroke, it is important to differentiate cardioembolic stroke from non-cardio-embolic stroke, since cardio-embolic stroke generally results in more severe disability and acute treatment and secondary prevention differ in cardio-embolic stroke from non-cardio-embolic stroke. However, it is difficult to diagnose the subtypes of ischemic stroke accurately at admission. In determining subtypes of ischemic strokes, combination of biomarkers such as BNP, D-dimer, Matrix metalloproteinase 9 (MMP-9) and C-reactive protein may be more predictive rather than using a single biomarker.

BNP & Acute Ischemic Stroke

The levels of BNP are higher in patients with Acute Ischemic stroke (AIS) as compared to haemorrhagic stroke and are higher in cardio-embolic stroke as compared to non-cardio embolic ischemic stroke. [9] BNP is increased in AIS presumably due to myocardium damage or elevated blood pressure. Tomita et al however clearly demonstrated after exclusion of heart disease, the plasma BNP level at admission was significantly higher in large artery occlusion (LAA) than in small artery occlusion (SAO) and control. (70.6±53.9 vs 38.2±28.4 and 28.5±19.9 pg/ml respectively, both $p < 0.05$)). In LAA group there was no difference between supratentorial and subtentorial lesions [10]. Yukiri et al observed significantly higher BNP levels at admission in cardio-embolic infarctions as compared to atherothrombotic infarctions. ($P<0.001$) and concluded that BNP can be a surrogate marker for CES with strong predictive power independent from atrial fibrillation. BNP levels positively correlate with infarct volume.

BNP & Stroke Severity

Plasma BNP level can be a clinically useful marker indicative of the severity of acute ischemic stroke. Significant correlations ($P=0.003$) are found between BNP level and the NIH stroke score (NIHSS) in AIS at admission. Since higher plasma BNP levels reflect greater infarct area, the correlation is compatible with the clinical manifestations [10]. Cakir et al found no statistically significant correlation between NIHSS score and BNP levels [11].

BNP & Functional Outcome of Stroke

Plasma levels of BNP in the acute phase of ischemic stroke predict post stroke mortality [12] and patients with high plasma BNP levels have four fold higher mortality. Long term functional outcome after stroke is one of the most important and difficult variables to predict and is subjected to complex interactions with multiple factors. The potential role of BNP in predicting long term functional outcome is controversial.

BNP & Intra-cerebral Haemorrhage

Tomita el al found no difference in BNP levels in ICH (47.3±26.6 pg/ml) and controls.[10] Nakagawa et al found that although patients with intracranial haemorrhage (ICH) had higher MAP levels than patients with ischemic stroke, the serum BNP levels were higher in patients with ICH [13].

Conclusion

Biomarkers like BNP should be used to supplement clinically guided therapy and not to substitute it. Proper interpretation of BNP would surely make the diagnosis, management and risk stratification better for stroke subjects. Using combination of biomarkers increases its predictive value. In stroke patients, elevated serum BNP on admission may not only further confirm a cardio-embolic etiology of stroke event, but also may signal increased risk for poor long-term outcome, including death. BNP testing has a role in risk stratification, identifying those likely to require intensive rehabilitative intervention. In addition, particularly in cases of cryptogenic stroke, the BNP level could help in forming the choice of antithrombotic agent for secondary stroke prevention.

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Review Article

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Pre-analytical Variables in Coagulation Testing: Avoiding Diagnostic Errors in Hemostasis

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Abstract

Emergency physician tends to assume that the sample analysed by the lab and the results authenticated and released reflects what is happening in the patient. It is important to note that the clinical decision making process based on correlation with lab reports will be flawed and may have disastrous consequences if a system exists where the pre-analytical variables of Lab testing, especially coagulation tests are not considered and monitored. This review essentially highlights most important pre-analytical variables with the underlying mechanisms that if ignored can lead to correct analysis but wrong results if correlated to patients condition.

Keywords: Coagulation; Hemostasis; Phlebotomy; Fibrinolytic System.

Introduction

In Accident & Emergency Medicine "Doing the right thing" alone will not suffice, "Doing the thing right also holds equal importance". Large multi-speciality tertiary care hospitals usually have busy Emergency Medicine department with a constant inflow of patients suffering from varying acute disorders together with a good number of road traffic accident cases. Emergency Physicians are always under pressure to deliver best possible care within a short time frame and save as many lives as possible. Although clinical decision making relies heavily on symptomatology and physical sign elicitation, backup results authenticated by Lab of basic parameters play a vital role in confirmation of primary diagnosis or narrowing down the differential diagnosis, choice of medications and short term prognostication.

Pre-analytical, Analytical & Post-analytical phase of Diagnostic Services

Test for Hemostasis is an important component of all primary tests ordered by the Emergency Physician in most of the patients streaming to the Emergency Department. These tests are used to assess whether a particular patient is at risk to bleed or clot. Choice of tests ordered, time to draw blood, positive patient identification, phlebotomy process, choice of anticoagulant, sample transport to Lab, sample accessioning by the Lab and sample storage before analysis form the pre-analytical aspect of Lab testing. Actual testing procedure constitutes the analytical phase of Lab testing. Laboratory Information System (LIS) with its integration to the Hospital Management System ensures that the test reports authenticated by the Lab Physician are accessed by the Emergency Physician in a regulated and timely fashion. This forms the post-analytical phase of Lab testing.

Brief overview of Hemostasis

Most of the time, we equate hemostasis with coagulation cascade and assessment of its surrogate markers. However hemostasis is far more complicated. It is a complex dynamic interaction between several key players in the body namely the vascular endothelium, platelets, coagulation factors, the fibrinolytic system and inhibitors of the fibrinolytic system [1, 2]. Checks and balances are present in every step. Bleeding or thrombosis occurs when the interactions are disrupted due to deficiency or dysfunction of the important components [3].

Hemostasis unit of the Hematopathology department usually performs the routine coagulation tests like the Prothrombin time (PT), Activated plasma thromboplastin time (APTT), Thrombin time (TT), Fibrinogen assay and D-Dimer /FDP tests. A major issue in coagulation testing is overlooked and which has in the recent times begun to be the topic of interest and research: Pre-Analytical Variables.

Concept of Pre-Analytical Variables

Are essentially the problems and deviations that may arises prior to sample testing including but not limited to sample collection, handling, transport, processing in accession and storage prior to testing. When a sample is inadequate, it is rejected by the lab. Accredited Labs have well defined sample rejection guidelines, its standard operating procedure (SOP) and records of its implementation. However the system is flawed because a major portion of the pre-analytical events occur outside the purview of the lab. Lab is unaware most of the time that an adverse event has occurred with a particular sample. Hence it is not always clear when an unsuitable sample has arrived in the lab, tested in good faith and results released [4, 5].

Pre-Analytical Variables

Patient Identification

Patient misidentification is associated wrong reports resulting in worst clinical outcome due to misdiagnosis and inappropriate treatment. In both outdoor and indoor settings double identifiers is preferred. Identification from the current prescription and talking with the patient/relatives in OPD and from the bar coded wrist band together with verbal communication with the patient/attendant/nursing staff will help reduce the incidents of blood being drawn from the wrong patient [6].

Sample Identification

Post phlebotomy collection vials usually are identified by bar coded tube labels pasted on it[7].These bar coded tube labels should be generated bedside after collection is completed for that particular patient. The practice of scribbling by pen few patient data on the filled vials and moving to the next patient for phlebotomy keeping the generation of bar coded tube labels and pasting on the vials based on the scribbled data for a later time at the nursing station has to be discouraged .

Phlebotomy Process

Tourniquet time of more than one minute results in hemoconcentration. Further it stimulates the endothelium and activates the coagulation cascade at multilevel in the vessel. In both the cases results are altered as ratio of plasma to anticoagulant is changed in the former event and consumption of factors occurs at the later event. Use of tourniquet of < 1 minute and release of the same as soon as blood flow into the vial, is recommended.

Slow venepuncture and difficult venepuncture irritates the vessel wall and causes in vivo activation of cascade resulting from local release of tissue factor. If the vein is located by multiple passes by the needle / manipulation of the needle or blood comes out in a slow stream another venipuncture site have to be selected.

Drawing blood from IV infusion line or phlebotomy from a site downstream to the infusion site is discouraged. It leads to dilution of coagulation factors leading to erroneous results, when drawing blood from peripheral or central venous lines predraw flushing and discard of the initial sample is necessary. This avoids sample dilution and sample contamination. Too large needle (<16G) and too small needle (>25G) needs to be avoided. Former causes more tissue damage in the wall of the vein causing premature start of the clotting process resulting in false low results. Later cases cause hemolysis of the sample. Heparinised needles used in blood gas analysis needs to be avoided at all costs [11].

According to CLSI guidelines on order of blood draw [8], coagulation test sample to be preferably drawn first (second to blood culture set). These avoid sample contamination from subsequent anticoagulants and clot activators. Also effect of local release of tissue factor is minimal. If winged collection set is used, it is necessary to discard the first sample to minimise the effect of contaminants and air in the tube [9].

Sample vial should be filled up to the mentioned mark or 90% of the total prescribed volume. Underfilling leads to low sample volume and excess calcium binding citrate causing falsely prolonged coagulation results [10]. Blood drawn into the citrate vial if not adequate in volume should never be topped up from another vial having same or different anticoagulant. In the former case it results in doubling up of the anticoagulant and dilution of plasma sample. In the later case introduction of calcium chelating EDTA or clot activators results early and spurious start of the coagulation process and results in false low test results.

Thorough mixing of sample after its collection in the vial with the anticoagulant present in the vial is to be done by end over end inversion 3-6 times gently. This prevents clot formation. Clot if formed however small it may be leads to erroneous results. The practice of manually removing a clot if formed in the vial and the sending the vial to the lab for testing instead of rejecting the sample is to be strongly discouraged. Conversely vigorous shaking of the filled vial by the phlebotomist/ others can lead to hemolysis of the sample or spurious factor activation resulting in wrong results with disastrous consequences to medication and safety.

Choice of the Anticoagulant

CLSI guidelines recommend use of 3.2% citrate instead of 3.8% citrate except for few specific applications. Samples drawn into 3.8% citrate overestimate PT & APTT results and lower fibrinogen values. Biological reference ranges derived from literature review is mostly based on coagulation study on sample drawn in 3.2% citrate vials [12].

Sample transport to the Lab

Current recommendation states, sample to be transported to the Lab as soon as possible in non refrigerated state at ambient temperature (15-22®C) [13]. Emergency Physician needs to understand that correct results for all coagulation tests are possible if analysis is done within 4 hrs. Hence sample should be send to lab stat and not collectively at predetermined time. Putting the sample in the refrigerator before despatch to Lab is to be avoided at all costs. It is important to note that APTT test of patients getting unfractionated heparin needs to be done within 1hr [14]. Delay causes heparin neutralization by platelets resulting in wrong results.

Conclusion

Preanalytical process variation remains an important cause of diagnostic errors. A large number of wrong results can be intercepted before release of reports if the concepts and knowledge of Pre-Analytical Variables is available to the Emergency Physician.

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A Case of Pulmonary Artery Stenosis

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Abstract

Stenosis of the pulmonary artery is a condition where the pulmonary artery is subject to an abnormal constriction .Peripheral pulmonary artery stenosis may occur as an isolated event or in association with Alagille syndrome, Berardinelli-Seip congenital lipodystrophy type1, Costello syndrome, Keutel syndrome, nasodigitoacoustic syndrome (Keipert syndrome), Noonan syndrome or Williams syndrome. It should not be confused with a pulmonary valve stenosis, which is in the heart, but can have similar hemodynamic effects. Both stenosis of the pulmonary artery and pulmonary valve stenosis are causes of pulmonic stenosis. The presentation of such a case may be very misleading. The case that we present here is about a 37 year old Carribean female who presented with progressive increase in SOB.

Keywords: Pulmonary Stenosis; Pulmonary Hypertension; Cardiovascular; Hypertension; Angiography; Congenital Heart Disease; Chest Pain.

Introduction

Pulmonary artery stenosis is a narrowing (stenosis) that occurs in the pulmonary artery, a large artery that sends oxygen-poor blood into the lungs to be enriched with oxygen. The narrowing may occur in the main pulmonary artery and/or in the left or right pulmonary artery branches. This narrowing makes it difficult for blood to reach the lungs to pick up oxygen. Without enough oxygen, the heart and body cannot function as they should. In an effort to overcome the narrowing, the pressure in the right ventricle (the chamber that pumps blood into the pulmonary arteries) rises to levels that can be damaging to the heart muscle. If the narrowing in the artery is less than 50 percent, patient may not experience any symptoms. However, if the narrowing of the artery is more than 50 percent, patient may experience any of the following symptoms:- Shortness of breath , Fatigue, heavy or rapid breathing , rapid heart rate ,swelling in the feet, ankles, face, eyelids, and/or abdomen.

Pulmonary artery stenosis is a congenital heart defect, meaning it is a defect that is inborn or exists at birth. Stated another way, the defect is an abnormality, not a disease. Pulmonary artery stenosis is often present in combination with other congenital heart defects. The pulmonary valve is found between the right ventricle and the pulmonary artery. It normally has 3 leaflets that function like a one-way door, allowing blood to flow forward into the pulmonary artery, but not backward into the right ventricle.

With pulmonary stenosis, problems with the pulmonary valve make it harder for the leaflets to open and permit normal blood flow from the right ventricle to the lungs in a normal fashion. In children, these problems can include:

- A valve that has leaflets that are partially fused together.
- A valve that has thick leaflets that do not open all the way.
- Narrowing of the area above or below the pulmonary valve.

There are four different types of pulmonary stenosis:

- *Valvar pulmonary stenosis:* The valve leaflets are thickened and/or narrowed.

- *Supravalvar pulmonary stenosis:* The portion of the pulmonary artery just above the pulmonary valve is narrowed.
- *Subvalvar (infundibular) pulmonary stenosis:* The muscle under the valve area is thickened, narrowing the outflow tract from the right ventricle.
- *Branch peripheral pulmonic stenosis*: The right or left pulmonary artery is narrowed, or both may be narrowed.

Pulmonary stenosis may be present in varying degrees, classified according to how much obstruction to blood flow is present.

Case History

A 37 year old female of afro carribean origin, married with two kids, had been referred to India for treatment / evaluation of gradually progressive dyspnoea, since past one year, with ejection systolic murmur.

No past history of any cardiac / systemic illness. The patient presented to the hospital with progressive dyspnoea. On presentation to the emergency the patient was dyspnoiec.

Vitals

BP: 130 / 80 mm hg

SPO2:

Pulse: 82 / Min

Respiratory Rate: 22 / Min

CVS : S1 N S2 Wide Split with ejection systolic murmur

ECG: Equivocal / Insignificant

Labs: Not Significant.

Pulmonary artery angiogram showed bilateral significant peripheral pulmonary artery stenosis.

Patient was taken for intervention in cath lab

Pulmonary stenting was done:

Procedure details: Access Rt femoral vein and Rt femoral artery catheter used Swan Ganz 6 F and pigtail 6F.

Pressures: Systemic arterial pressure: 117/70 (88) mm hg

RV Systolic pressure: 100 mm hg

Main pul artery: 100/ 21 (54) mm hg

Left lower distal PA: 21 /1 7 mm hg

RT Distal PA: 13/ 7 (10) mm hg

Pulmonary artery angoigram: Left lower PA 70% Stenosis, (Pullback Gradient 79 mm hg)

RT Pulmonary artery: 80 % Stenosis, Pullback Gradient 87 mm hg

Pulmonart Artery Stenting: Access RFA and RFV,

Systemic Pressure: 140 /80 mm hg

Pre procedure PA pressure: 98/16 mm hg

Guide – JR 6 F

In RT pulmonary artery 7/16mm Herculink Plus Stent was deployed @ 13 ATM Post Dilated

with Stent baloon itself.

In lpa 6/16 mm herculink plus stent was deployed @15 atm post dilated with stent baloon itself .

Post procedure

Systemic pressure: 142/ 80 mm hg

Pul Artery: 77/15 mmhg

Good end result, no procedural complications.

Post procedure RV pressure almost half of systemic.

Post procedure hospital stay was uneventful, the patient was symptomatically better and was discharged in a stable condition.

Discussion

This case tells us that as emergency physician our differences have to be very broad for patients presenting with sob and dyspnea on exertion. The patient was well managed and the different modalities that can aid us reaching a diagnosis in such atypical/ typical presentation are as under.

During examination in ED, doctor may hear abnormal heart sounds (a murmur) when listening to the heart. If abnormal sounds are identified, we can order for the below mentioned diagnostics from the ED itself.

- An electrocardiogram (ECG or EKG): A test that records the electrical changes that occur during a heartbeat; reveals abnormal heart rhythms (arrhythmias) and detects heart muscle stress.
- Chest X-ray: A test to show the size and shape of the heart and lungs and pulmonary arteries
- Echocardiogram: A test that uses sound waves to create a moving picture of the heart's internal structures.

- Doppler ultrasound: A test that uses sound waves to measure blood flow; usually combined with echocardiogram to evaluate both the internal structure of the heart and blood flow across the heart's valves and vessels.
- Cardiac magnetic resonance imaging (MRI): A test that uses three-dimensional imaging to reveal how blood flows through the heart and vessels and how the heart is working.
- CT scan: An X-ray procedure that combines many x-ray images with the aid of a computer to generate cross-sectional views of the heart. Cardiac CT uses the advanced CT technology with intravenous (IV) contrast (dye) to visualize cardiac anatomy, coronary circulation, and great vessels.
- Cardiac catheterization: A procedure that involves inserting a thin tube (a catheter) into a vein or artery and passing it into the heart to sample the level of oxygen, measure pressure changes, and make X-ray movies of the heart and its internal structures.
- Pulmonary angiography: A dye-enhanced X-ray of the pulmonary arteries and veins of the heart
- Perfusion scan: A test in which the patient is injected with a small amount of a radioactive material. A special machine shows how well blood is flowing through each of the two lungs.
- Additional tests may be ordered as necessary.

ED Management

Stabilizing A-B-C and then need to think of definitive management as under:

Balloon dilation

This treatment method consists of moving a balloon dilation catheter into the narrowed area of the artery. The balloon is carefully inflated – first under low pressure and then under higher pressure – until the narrowed area is widened. The balloon is then deflated and removed. Although the narrowing is improved in a majority of patients following balloon dilation, overtime the artery can again become narrow in as many as 15% to 20% of cases, requiring further ballooning. Different types of balloons are currently being developed that will likely lead to better and longer-lasting results.

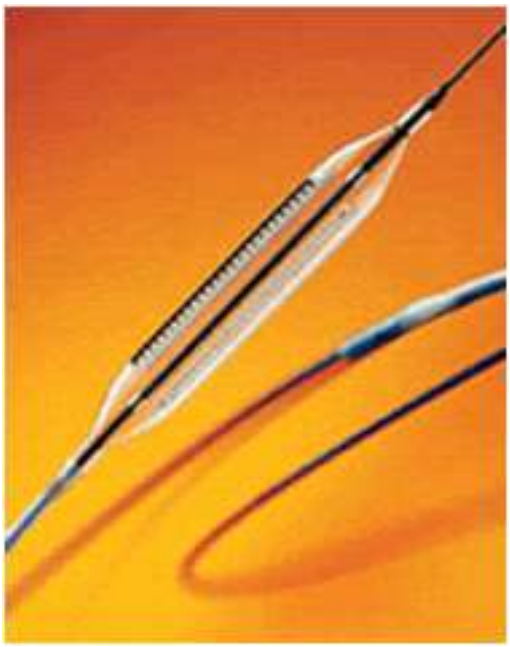
Balloon dilation and stent placement

In an effort to improve on the results of balloon dilation, a search for a more effective treatment was begun and led to the development of the stainless steel balloon-expandable stent. Stent placement is accomplished by positioning the stent across the narrowed segment of the artery. The stent is mounted on aballoon angioplasty catheter and covered with a sheath as it is moved into position. The sheath then is withdrawn off the stent-balloon angioplasty assembly and the balloon is inflated to its recommended pressure, expanding the stent and anchoring it in place.

The Cutting Balloon

This procedure is similar to standard balloon dilation but the balloon has been specially designed with small blades running up and down its length. When the balloon is inflated, the blades are activated and they cut through the narrowed area, making the vessel easier to dilate and resulting in a larger opening. Cutting balloons are available in different sizes.

Fig. 1: The Cutting Balloon Image with permission, from Boston Scientific Corporation



Surgery

Various methods of surgical repair of pulmonary artery stenosis are used, the choice of which depends on the characteristics of the stenosis and the surrounding vessels and other structures.

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Imperforate Hymen: A Cause of Acute Urinary Retention in Young Females that is often Overlooked

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Abstract

Children, especially females, very rarely present with acute urinary retention to the Emergency Department. In this case report, we present a case of urinary retention secondary to imperforate hymen leading to haematocolpos and mechanical obstruction of the urinary tract in a 12 year old adolescent girl. Imperforate hymen can be missed in adolescent girls presenting to Emergency Departments with urinary difficulty if genital examination or Emergency Ultrasound scan is not performed. There should be increased awareness among emergency physicians that imperforate hymen can be a possible cause urinary retention and lower abdominal pain in adolescent girls. We discuss the need of careful assessment along with a brief review of literature.

Keywords: Imperforate Hymen; Young Females; Cyclical Abdominal Pain.

Introduction

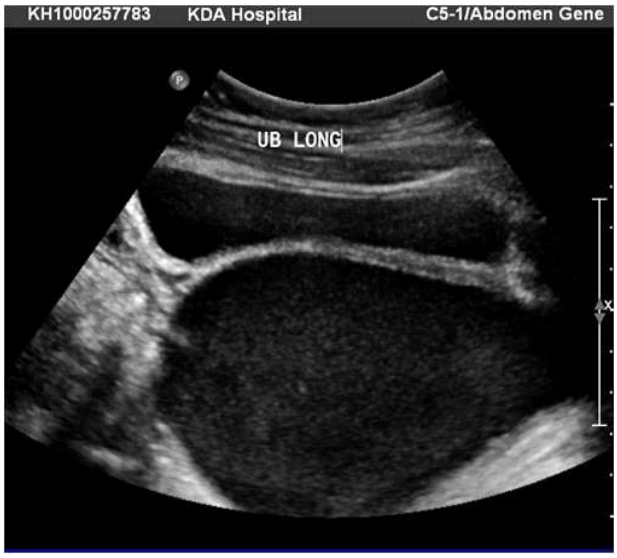
Though imperforate hymen is one of the commonest congenital anomaly of lower female genital tract, occurs in approximately 1/1000 newborn girls i.e. 0.1% of all new born female babies [1, 2, 3].

Acute urinary retention is a rare occurrence in females because of their short urethra and anatomic relationships [1]. However, imperforate hymen can present as urinary retention due to obstruction from a pelvic or perineal mass caused from haematocolpos, which is the accumulation of menstrual blood above an imperforate hymen leading subsequently to the distension of the vagina. This distension of the vagina leads to stretching of the urethra and eventually urinary retention. A case of imperforate hymen that presented with acute urinary retention is described to increase awareness of this condition amongst EM clinicians.

Case History

We present the case of a 13 year old girl who attended the Emergency Department with acute urinary retention and lower abdominal pain of about 6 hours duration. She did not have any nausea, vomiting, fever or bowel disturbance. She had no previous episodes of any urinary problems like retention or difficulty in passing urine. She had not attained menarche and denied any vaginal discharge or sexual activity. Her past medical history was unremarkable. Her mother had attained menarche at the age of 14.

Fig. 1



On physical examination her ABC's and vitals were stable. Abdominal examination revealed suprapubic tenderness and with no guarding and rigidity. Bowel sounds were present and hernial orifices were normal.

Vaginal examination revealed bulging bluish membrane beneath the urethral orifice.

Rest of the systemic examination was normal.

Bedside USG (Figure I) showed a distended bladder with grossly distended fluid filled uterus measuring 25cm*15*16cm. Our final diagnosis was acute urinary retention secondary to urethral obstruction due to imperforate hymen and haematocolpos and hematometra.

Foley's catheterization (12F) was done immediately under aseptic and antiseptic precautions to relieve the symptoms of acute urinary retention. The patient then underwent a vertical hymenotomy in the operation theatre and 2550 ml of blood mixed, chocolate coloured fluid was drained. Patient was discharged on day two without any complications.

Discussion

This case report helps us to think about imperforate hymen in the differential diagnosis and the use of bedside ultrasonography by the ED physician as an adjunct to the diagnosis.

The hymen develops from the embryonic vagina buds and the urogenital sinus and normally perforates in the later stages of embryonic development and forms a central canal that communicates between the upper vaginal tract and the vestibule of the vagina. Imperforate hymen occurs due to incomplete canalization of the mullerian system and the urogenital system [2]**.** It is a developmental abnormality and the most frequent cause of vaginal outflow obstruction which is reported in approximately of 0.1% of newborns.

Imperforate hymen is an isolated abnormality, where the diagnosis should ideally be done at birth by careful examination of the external genitalia of all newborn females [1]. Patients who are not diagnosed in their infancy can present in the early part of second decade with symptoms of cyclical abdominal pain, urinary retention or constipation due to hematometra or haematocolpos. Sometimes in severe cases hematosalpinx can occur due to retrograde menses with resultant development of intra-abdominal endometriosis.

The most common symptoms of an imperforate hymen are cyclical abdominal pain and urinary retention, usually presenting between the ages of 13 and 15 years (when menarche occurs) [3, 4]. In a previous report on twenty cases it was found that 55% of the patients presented with urinary retention as a result of mass effect [5].

Though, the etiology of this condition is still unknown, imperforate hymen results in vaginal outflow obstruction and menstrual blood accumulates in the vagina (haematocolpos) and the uterus (hematometra). This may lead to pressure and stretching effects on the urethra, bladder, intestines or pelvic blood vessels which result in urinary retention, intestinal obstruction or pedal oedema. [3, 5-9]. Low back pain may also result from pressure and irritation of the sacral plexus [4].

Imperforate hymen can mimic other lower abdominal conditions like appendicitis, urinary tract infection and cystitis, renal calculi or abdominal tumour (ovarian tumour), where patients have even undergone appendectomies [10].

Though imperforate hymen is not a very uncommon cause of acute retention, the lack of awareness amongst clinicians frequently leads to incomplete history and physical examinations leading to misdiagnosis and unnecessary tests and treatment [2] One should always consider an imperforate hymen if there is a discrepancy between the Tanner stage and menarche status [2].

Gynecological examination should be carried out in all adolescent females and will reveal a bluish bulging hymen and generally an abdominal mass. Abdominal ultrasound showing a pelvic cystic mass, which bulged when Valsalva maneuver is used confirms the diagnosis of imperforate hymen by differentiating it from transverse vaginal septum which should not bulge [2].

If imperforate hymen is not diagnosed early it can cause serious complications such as infections (pyocolpos), hydronephrosis, renal failure, endometriosis and subfertility [11, 12]. It has been shown in a previous study that eight of nine patients with imperforate hymen and outflow obstruction had developed endometriosis at the time of operation.

Imperforate hymen is surgically treated by a cruciate incision in the hymen from 4 o'clock position to 10 o'clock position and 2 o'clock position to 8 o'clock position which allows the accumulated blood to drain away. This should be done aseptically as a closed vagina has an alkaline or weakly acidic pH and lacks in protective Doederlein's bacilli. This

causes poor natural resistance to bacteria entering from the lower genital tract and the blood and debris provide a good culture medium after the drainage leading to intrauterine infection [4]. The complications of a hymenotomy are infection, bleeding, scarring and stenosis of the vaginal opening [13].

A previous study on the long term results of hymenotomy has shown that nine out of fifteen patients had irregular periods and six had dysmenorrhoea after hymenotomy. However their Pre-operative complaints like cryptomenorrhea (n=15), abdominal pain (n=11), palpable mass in the lower abdomen (n=9), urinary retention (n=6), dysuria (n=3) and problems defecating (n=4) disappeared after surgery. Most patients had no sexual dysfunction and [14] two of them who were attempting pregnancy and were successful. Another study showed that 86% of patients who attempted pregnancy succeeded after surgical correction of imperforate hymen [12]. Less invasive treatments for an imperforate hymen include the use of CO_2 lasers or a Foley catheter [12].

Conclusion

Imperforate hymen is one of the most common female genital tract malformations though thought to be uncommon cause of abdominal pain in pediatric population. A large number of patients with this condition (55%) presents with acute urinary retention. It is one of the conditions that can be overlooked in a busy emergency department leading to misdiagnosis and delayed or unnecessary investigations, treatment and serious complications. So, it is very important to take a complete gynecological history and perform a gynecological examination in adolescent girls presenting to the Emergency department with cyclical pain, lower abdominal mass or acute urinary retention especially if there is a discrepancy between the Tanner stage and menarche status.

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Vocal Cord Paralysis Induced Aspiration Pneumonia with Ards: An Unusual Presentation Seen in a Case of Intracranial Space Occupying Lesion

Deepika Mittal*, Shahid Mustafa Khan*, Kishalay Datta**

Abstract

Intra cranial space occupying lesion have been known to present with varied symptoms and signs, mostly related to CNS since the involvement of brain parenchyma directly or the pressure effects of the intracranial space occupying lesion lead to varied manifestations ranging in severity from a mild headache to coma and varied neurological deficits.

We take the opportunity to present a case report of this 63 year old male patient who was admitted with a primary diagnosis of pneumonia with ARDS, further investigation revealed a unilateral vocal cord paralysis secondary to anintra cranial space occupying lesion, although the patient was asymptomatic for any neurological symptoms previously and the prime presentation was with cough and fever of five days duration.

The unusual thing about this case was the masking effects of ARDS and shock which lead to ambiguity of diagnosis in view of no underlying medical ailment.

Keyword: Vocal Cord Paralysis; Intracranial Space Occupying Lesion; Aspiration Pneumonia.

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Case History

A 63 year old male patient previously normotensive, non diabetic no h/o cardiac disease, not on any medication, presented to the emergency medicine department with fever, cough, breathing difficulty since five days.

Vitals

BP: Not recordable

Pulse: Feeble, 126/min

Spo2: 85% on room air

Temp.: 102 F

RBS: 105 mg/dl

ECG: Sinus tachycardia

The systemic examination/Secondary survey was unequivocal except for bilateral crepitations scattered over the chest.

The patient was started on IV vasopressors in view of non response to initial fluid resuscitation with IV fluid bolus.

The patient's initial chest x-ray revealed B/L opacities in lower and mid zones suggestive of ARDS/? Aspiration pneumonia.

Investigations: ABG- pH: 7.3, pCO2: 41.3, pO2: 54, HCO3: 21.9, Hb: 11.7, Na: 133,

K: 3.4, LAC: 3.8

UREA: 18.0, S.CREATININE: 0.66

CBC- Hb: 12.1, WBC: 13000, PLATELETS: 150

In spite of aggressive resuscitative measures with iv fluids, vasopresors and appropriate antibiotic therapy, along with other supportive measures the patient's clinical status showed no improvement and the patient continued to be hypotensive with severe respiratory distress and tachypnea.

After consultation with the pulmonologist, the patient was planned for endotracheal intubation in view of severe respiratory distress and inability to maintain oxygen saturation beyond 89% .

While undergoing endotracheal intubation, on direct laryngoscopy the patient's laryngeal inlet was found compromised, which led to the suspicion of

laryngeal paralysis as a cause of aspiration pneumonia, since no other possible aetiology could be attributed to the pathogenesis of aspiration pneumonia in this patient with no co-morbid conditions.

Further inquest into the cause of suspected laryngeal paralysis, leads to the diagnosis of low density lesion at base of skull on left side with epicentre at petrous apex, left cord palsy with enlargement of ipsilateral laryngeal ventricle, on CT scan.

MRI Brain: Extra axial lobulated intra cranial space occupying lesion in the region of left side of skull, base of left middle crania fossa with left half of cavernous sinus, left apex, left meckel's cave.

Immediate neurosurgical review was done and patient was planned for decompression surgery in view of ICSOL, but in spite of best resuscitative efforts the patient's clinical status kept on deteriorating with ARDS and resistant shock.

The patient went into cardiopulmonary arrest and was declared dead after failure of resuscitation as per ACLS protocols.

Discussion

Respiratory insufficiency is a common presentation in the ED, and possible search into the causes of respiratory insufficiency in a patient with apparent pulmonary infection or X-ray suggestive of pneumonia with ARDS like picture would rarely ring the bells to look for a cause in CNS.

But again considering the scenario in this patient with no co-morbid conditions and clinical picture of a respiratory tract infection a logical way to proceed would be H1N1 screen rather than a CT brain.

The key to patient management in the emergency department is early aggressive resuscitation, but the clinician needs to be aware of subtle clinical signs, like in our case the suspicion of vocal cord paralysis on direct laryngoscopy, which can lead to formulating a conclusive diagnosis and improved patient outcomes.

Conclusion

What we intend to convey by means of this case is that no doubt most of the times, disease presents in the prime place with symptoms suggestive of the primary organ system involvement, but as we all have learnt from previous experience as clinicians we should not miss any minor details in the patients presentation since as the dictum goes, *biology is a science of exceptions*, any disease can present with atypical or exceptional manifestations.

Early recognition of important clinical signs can be vital in directing our diagnostics and management in critically ill patients, helping improve survival and prognosis.

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A Case Report of Acute Cerebrovascular Accident in Children

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Indranil Das*, Kishalay Datta*, Tamorish Kole*

Abstract

Stroke is defined as the sudden occlusion or rupture of cerebral arteries or veins resulting in focal cerebral damage and neurological deficits. Types of stroke resulting from vascular occlusion are arterial ischemic stroke and those resulting from vascular rupture are called hemorrhagic stroke. Stroke in children is relatively rare and frequently results in a lack of recognition and delay in diagnosis. The etiologies of stroke in children are multifactorial. In our present case the patient who is 13 yrs old and presented with symptoms suggestive of acute CVA. The child recovered within 4-5 days and was discharged in improved neurological status.

Keywords: Stroke; Arterial Ischemic Stroke; Sinovenous Thrombosis; Hemorrhagic Stroke; Antithrombotic Therapies.

Introduction

Stroke or CVA is rare in children. Heart disease whether it is congenital or acquired, metabolic and hematological disorders and vasospastic conditions like migraine are more often associated with childhood strokes. The treatment of stroke in children has been primarily directed toward treating underlying causes. Anticoagulant therapy appears to be increasing in pediatric ischemic Stroke. Mortality after stroke in children ranges from 20% to 30% depending on the location and the underlying cause. Residual neurological dysfunction is present in more than 50% of survivors. Stroke is a major cause of disability and death in children. 10% of children suffering stroke die, and at least 50% of survivors are left with neurological disabilities, learning difficulties or seizures. Arteriopathies and cardiac disease are the commonest risk factors for childhood Arterial Ischemic Stroke (AIS). The cause of perinatal AIS is poorly understood, despite affecting 1 in 4000 newborns. Sinus & venous thrombosis due to head and neck infections are one of the causes in AIS and AV malformation has been found to be the cause of hemorrhagic stroke. Infants, children and young adults account for less than 5% of all strokes. The incidence of stroke in the 0 to 14 yrs age group was found to be 2.5 cases per 100,000 per year. Out of these 25.2 % were of ischemic stroke and 75.6% is of hemorrhagic stroke. In India the incidence is quite high of around 13 to 33 cases per 100,000 per year. It has also been found that around 20-30 % of all infants born prematurely below 35 weeks of gestation have some forms of intraventricular or cerebral matrix hemorrhage.

Case History

A 13 yr old female was brought to the emergency department by her parents with complain of weakness of the Right Upper and Lower limbs gradually increasing since 4 hours. The patient is unable to walk or stand by herself and tends to fall while trying to do so. There is also mild slurring of the speech and deviation of the tongue to the left. There is no h/o any fever, cough, cold, loose motions, rash or joint pain. She was absolutely fine in the morning and then suddenly started complaining of weakness in the Right Upper and Lower Limb. No history of any trauma, fall, seizure in the past. The patient is not on any regular medication and has no h/o any hypertension, vision problems, sinusitis or upper respiratory tract infection. There was no travel history

and no h/o any recent vaccination. Birth history- Insignificant and normal vaginal delivery and no birth trauma. Developmental history – Normal, Diet history– on balanced Vegetarian diet; patient was fully vaccinated, and did not give any history of major illness in the past. Anthropometry- looking well built and well nourished. The vital parameters were Pulse – 88 /m Regular; BP- 100/60 mm Hg; RR- 24/m; Spo2 -100% in RA; RBS- 102 mg/dl; Temp- Normal; Cardiac monitor – Normal Sinus rhythm; Examination of the HEENT, PUPILS; CHEST, abdomen and CVS were normal. CNS examination revealed that patient had dysarthria; signs of right 7th nerve palsy and right sided hemiparesis and hypotonia of the right upper and lower limbs. Patient was otherwise fully conscious and oriented, GCS 15/15 and right Plantar response was indeterminate.

After initial stabilization, the patient was sent for MRI brain which showed an acute infarct in the Left MCA territory.

Baseline laboratory investigations sent from Emergency Department were normal – TLC 11.4, Hb 12.3, PCV 37.4, platelet 271, Neutrophil 82.3, ESR- 11mm/hr, urea 12.8, creatinine 0.4, Na+139, K+4.2, Chloride 106, Calcium9.8, Cholesterol 118, Triglyceride 132, HDL 30.9, LDL 73.3, VLDL 26.4.T4 0.63, T3 3.42, TSH 2.21. PT 12.9, INR 1.19, PTT 27.1. Special tests sent after admission revealed a very low Protein C level though ANA, Anti Phospholipids Ab (IgG, IgM), Homocystine Level, Factor V, Protein S, Antithrombin III and Lupus Anticoagulant were all normal.

Patient was treated conservatively with supportive medications physiotherapy & and Neuroscience Rehabilitation. By the 5th day, the patient regained near normal muscle tone in all limbs and fair voluntary control in Rt. Upper and lower limb. Berg balance score 44/56. Patient discharged on Aspirin with near normal Recovery.

Discussion

The primary pathophysiology of CVA is either Ischemic or Hemorrhagic. Also infection and substrate failure leads to damage to the fragile brain parenchyma in children. The main extent of damage to the brain is due to the impairment of the vascularity and metabolic demands of the brain tissue. The brain receives its blood supply from the carotid and the vertebro-basilar circulation. There are certain regions in the brain like the diencephalon which are supplied by the end arteries and the anastomosis are not efficient and hence have a dreadful consequence when these end arteries are occluded. There are certain areas of the brain which lies between the any two major arteries and are called the watershed zones. These zones are affected by the decrease in cerebral perfusion pressure.

Two types of brain injury are there which include the Primary Injury and the Secondary Injury. The Primary injury is due to cellular damage caused by direct insult and the Secondary Injury is the cascade of events which are ignited by the primary insult. There can also be focal hemorrhage following ischemia. There is a central area or core where there is severe ischemia and there is a surrounding area called the penumbra which can recover if the perfusion is restored. This will result in better recovery. There is also a gross difference between the adult and the infant/children brain. The lactic acid produced as a result of ischemia causes more damage to the adult brain than the neonatal brain. This is thought to be due to the greater permeability of the immature brain to the lactate as a result it cannot accumulate locally and causes less damage.

Common Causes of CVA in Children

1. *Congenital* e.g. congenital heart disease and Coarctation of aorta.
2. *Acquired* e.g. RHD, IE, cardiomyopathies, arrhythmias, myocardial infarction.
3. *Vasculitis/Vasculopathies* e.g. Infections, migraine, fibro muscular dysplasia.
4. *Hematological & hypercoagulable states* e.g. Sickle cell disease, polycythemia, Infections, Leukemia, Protein C&S deficiency, Antithrombin III deficiency, FactorV Leiden deficiency, nephritic syndrome.
5. *Metabolic causes* e.g. Homocystineuria, Ehler Danlos syndrome, Marfan's syndrome.
6. *Trauma* e.g. Blunt trauma.

This case report shows that Stroke in Children is due to Protein C deficiency which is also not a very common entity and the timely diagnosis and treatment led to complete recovery.

Conclusion

Although Acute Strokes in children are rare but still it is an emergency condition. So as Emergency physician any such presentation should be

considered with evidence based approach. The patient with such presentation needs extensive work up and should be evaluated as an inpatient.

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Case Report of Splenic Infarct with Proximal Splenic Artery and Coeliac Trunk Thrombosis

Gulati D.*, Ramsundar S.*, Datta K.*, Das I.*, Nagarani S.K.S.*

Abstract

A thrombus formation in the splenic artery occludes the vascular supply of the spleen, leading to ischemia of parenchyma of spleen and subsequently necrosis – Splenic Infarct. It is often clinically silent, the most common symptom being Pain Abdomen / LUQ (Left Upper Quadrant) pain and the sign being Left Hypochondrium Tenderness. There are a multitude of causes for splenic artery thrombosis and infarction, ranging from hematological disorders and malignancies to embolic disorders, vasculitis, autoimmune and collagen vascular diseases, trauma, systemic inflammatory disorders etc. As the presentation tends to mimic other diseases, a high degree of clinical suspicion is warranted for diagnosis. A contrast enhanced CT scan is the current diagnostic modality of choice. Splenic infarction alone is not an indication for surgery. Non-operative medical management requires close follow up and surgery is indicated for persistence of symptoms and/or complications.

We are reporting the case of a young male who presented to the emergency with 4 days of low grade fever and nausea with sudden onset severe pain in epigastrium and left hypochondrium. Normal lab investigations and USG abdomen were followed up with a CECT-abdomen that revealed splenic infarction and 90% stenosis of celiac trunk and hepatic artery with proximal splenic artery thrombosis.

Keywords: Splenic Infarct; Splenic Artery Thrombosis; Celiac Trunk Thrombosis.

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Introduction

A thrombus formation in the splenic artery occludes the vascular supply of spleen leading to ischemia of parenchyma of spleen and subsequently necrosis - Splenic Infarct. The infarction may involve the entire organ (global) or be localized to a segment. One of the earliest descriptions of splenic infarct was in 1896 in Germany where microscopic splenic infarcts were detected post-spleenectomy in a patient of endocarditis with septic emboli [1].

Splenic infarcts are most often clinically silent. In 1998, Nores and colleagues [2] reported 59 cases treated over a 30-year period at the University of California, Los Angeles (UCLA), and at the Cedars-Sinai Medical Center. In 1986, Jaroch and coauthors [3] identified 75 patients through clinical or autopsy reports at the Cleveland Clinic and found only an additional 77 cases in the literature. Most of the current literature consists of case reports only. However, there is a rising trend in the frequency of number of splenic infarcts identified due to increase in radiological imaging of patients, subsequently leading to an increase in incidental detection of splenic infarcts.

There are a multitude of causes for splenic infarction, majority (88%) comprising of infiltrative haematological diseases that result in congestion of splenic circulation by abnormal cells or obstruction of large vessels by thromboembolic events [2]. The causes may vary ranging from haematological disorders and malignancies to embolic disorders, vasculitis, autoimmune and collagen vascular diseases, trauma, systemic inflammatory disorders etc.

The spectrum of clinical presentation varies from asymptomatic infarction (discovered incidentally) to hemorrhagic shock. The most common symptom is Abdomen Pain in LUQ (Left Upper Quadrant) and the sign is tenderness in Left Hypochondrium. No lab investigations are specific for splenic infarct. Contrast enhanced CT scan is the diagnostic modality of choice [4]. A Gd-MRI clearly identifies area of infracted splenic parenchyma. Presence of luminal bowel gas and morbid obesity render this modality less useful. In a retrospective study of 49 episodes of acute splenic infarction, Antopolsky et al found ultrasonographic scanning to be diagnostically useful in only 18% of patients [5].

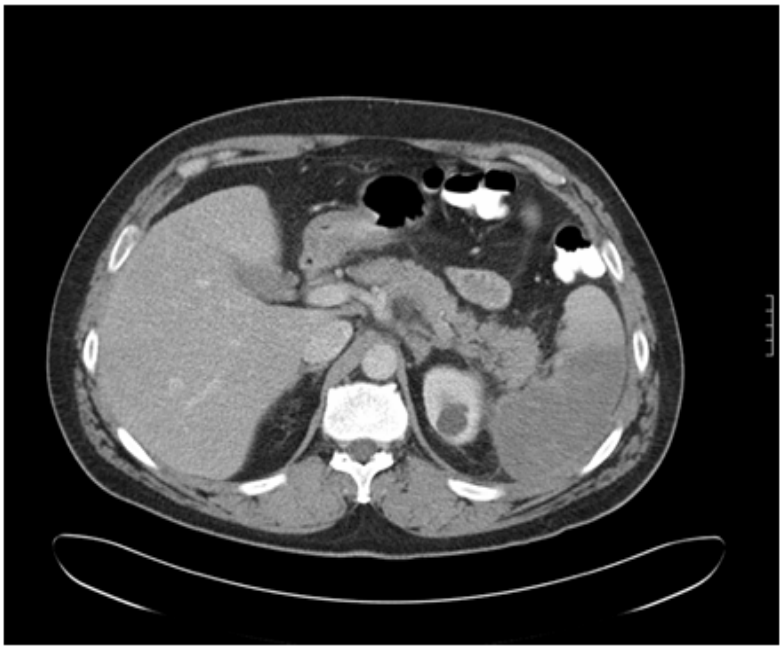
Splenic infarction alone is not an indication for surgery. Non-operative medical management requires close follow up. The mainstay of medical management comprises of adequate analgesia and close follow up. There is no scientifically supported information for the role of antiplatlet drugs and antibiotics. Surgery is indicated for persistence of symptoms and/or complications such as abscess, rupture, haemorrhage or pseudocyst. Because of the small but fatal risk for OPSI (overwhelming post-splenectomy infection), splenic salvage is preferred.

Case Report

A 45 yr old male presented to the ER with complaints of sudden onset very severe pain abdomen since morning (4-5hrs), mainly in the epigastrium and left hypochondrium, non-radiating, not associated with any aggravating or relieving factors, associated with fever, low grade, not associated with chills past 3-4 days and nausea and is not associated vomiting, loose motions, chest pain, syncope, sweating, SOB and cough. On arrival to the ER, patient is talking with no apparent respiratory distress and is haemodynamically stable, with P=86/min, BP=130/80, T= 98.4°F (36.9°C), RR=18/min, Spo2=99% on R, ARBS = 176 mg/dl. On systemic examination of the patient, there was tenderness present over the epigastrium and left hypochondrium with no palpable mass/ organomegaly and Bowel sounds were present, rest of the systemic examination was absolutely normal

Management in the Emergency department included intravenous cannulation, symptomactic pain medications and Investigations which were ECG,CBC, LFT, KFT, Blood cultures, S. Amylase, S. Lipase, PS for MP, Typhidot and USG whole abdomen was done which was absolutely normal. In view of persistence of pain abdomen in spite of adequate analgesia, associated nausea and a normal USG study without any GB or Hepatobiliary pathology, Acute Pancreatitis was suspected. Pt kept NPO, iv fluid, iv antibiotics, iv analgesics, iv antiemetics, iv antacids, supportive managementSurgical consultation was taken. The surgical specialist evaluated the patient and a CECT Whole Abdomen was planned.

Image 1: CECT abdomen showing hypodense area within the spleen—splenic infact



CECT Whole Abdomen (Fig. 1) Enlarged spleen with a Hypodense lesion within the splenic parenchyma– suggestive of Splenic Infarct. The splenic artery also does not enhance, suggestive of thrombosis. A cardiology consultation was taken for abdominal thrombus/Ischaemia. Patient was planned for a detailed 2D ECHO, CT Angiography, USG Abdominal Doppler (portocaval) and ANA. 2D- ECHO revealed LVEF 55% with no RWMA, Doppler – lower limb which was normal arterial and venous Doppler studies *Additional labs* included **C**-ANCA <6 u/ml (negative) P-ANCA <6 u/ml (negative) Phospholipids 207 mg/dL (151-264 mg/dL) Thrombin time 20 sec (16-23 sec) with no evidence of lupus anticoagulant and Cardiolipin antibodies normal. CT Angiography revealed 90% stenosis of Celiac trunk and Hepatic artery with proximal Splenic artery thrombosis. Patient was administered Inj. Clexane 0.6ml s/c stat and planned for DSA. The patient was Discharged on Request in stable condition on conservative management (anti platlets).

Discussion

Acute occlusion of the splenic artery results in infarction of the splenic parenchyma. As the spleen receives its blood supply from both splenic arteries (from celiac plexus) and short gastric arteries (from left gastroepiploic artery), occlusion of the main splenic artery may be compensated by collaterals that often preserve some or all of the splenic parenchyma. Within the spleen, the arterial supply is segmental. Occlusion of these secondary branches results in the classic wedge-shaped infarct.

Splenic infarction is often clinically silent and the presentation tends to mimic other diseases. Hence, a high degree of clinical suspicion is warranted for diagnosis. In patients presenting with left upper quadrant pain, fever, chills, nausea, vomiting, pleuritic chest pain and left shoulder pain, infarction of the spleen should be considered. Rapid diagnosis is the key to salvage the spleen. Multitude of causes need to be considered, especially, hematological diseases, thromboembolic states, vasculitis (SLE with lupus anticoagulant or antiphospholipid antibodies), cocaine abuse, trauma etc.

Not all cases require surgical intervention and the call for splenectomy should be taken judiciously, taking into consideration the lifelong risk of OPSI. Majority of cases are managed medically.

At one end of the spectrum of prognosis lies clinically occult splenic infarcts without any sequel while at the other end is high mortality associated with splenectomy. Asplenic individuals are at high risk of developing OPSI and require regular clinical follow up.

Although the incidence of splenic infarcts are rare but still quite prevalent and has been seen in adult populations. This case tells us that a simple case of pain abdomen which was initially thought to be a pancreatitis can turn out to be a case of splenic infarct and it carries along with it a whole set of complications. A high index of clinical suspicion has to be maintained for considering this among the differential diagnosis of patients with non specific abdominal pain.

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Early Recognition, Timely Intervention and Immediate CPR and its Outcome in a CKD Patient with Cardiac Arrest

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Abstract

Cardio-respiratory arrest is a real medical emergency. It can present as Pulseless VT (ventricular tachycardia), VF (ventricular fibrillation), Asystole, and PEA (Pulseless Electrical Activity). PEA is defined as any organized rhythm without a detectable pulse.

As per ACLS protocol 2010 guidelines, PEA should be treated with CPR and Epinephrine and/or Vasopressin as charted below and the most important step is to identify any reversible cause and to correct it.

Here in our present case, the 61 yrs old female patient presented as Cardio-respiratory arrest with PEA with severe metabolic acidosis and hyperkalemia.

She was treated as per ACLS guidelines and was revived successfully and she was discharged in a stable condition after 48 hrs.

Keywords : Pulseless Electrical Activity (PEA); Chronic Kidney Disease (CKD); End Stage Renal Disease; Cardiac Arrest; Compressions; Hyperkalemia; Hypercarbia; Metabolic Acidosis; Hemodialysis; Sudden Cardiac Death; Agonal Gasp.

Introduction

Cardio-respiratory arrest in patients of ESRD/CKD is not uncommon and can be due to various reasons like metabolic acidosis, hyperkalemia, hypoxia, coronary thrombosis, hypercarbia. PEA presents as cardio-respiratory arrest which is a real medical emergency. It includes rhythms like Sinus rhythm, Idioventricular rhythms, Post-defibrillation idioventricular rhythms, Ventricular escape rhythms.

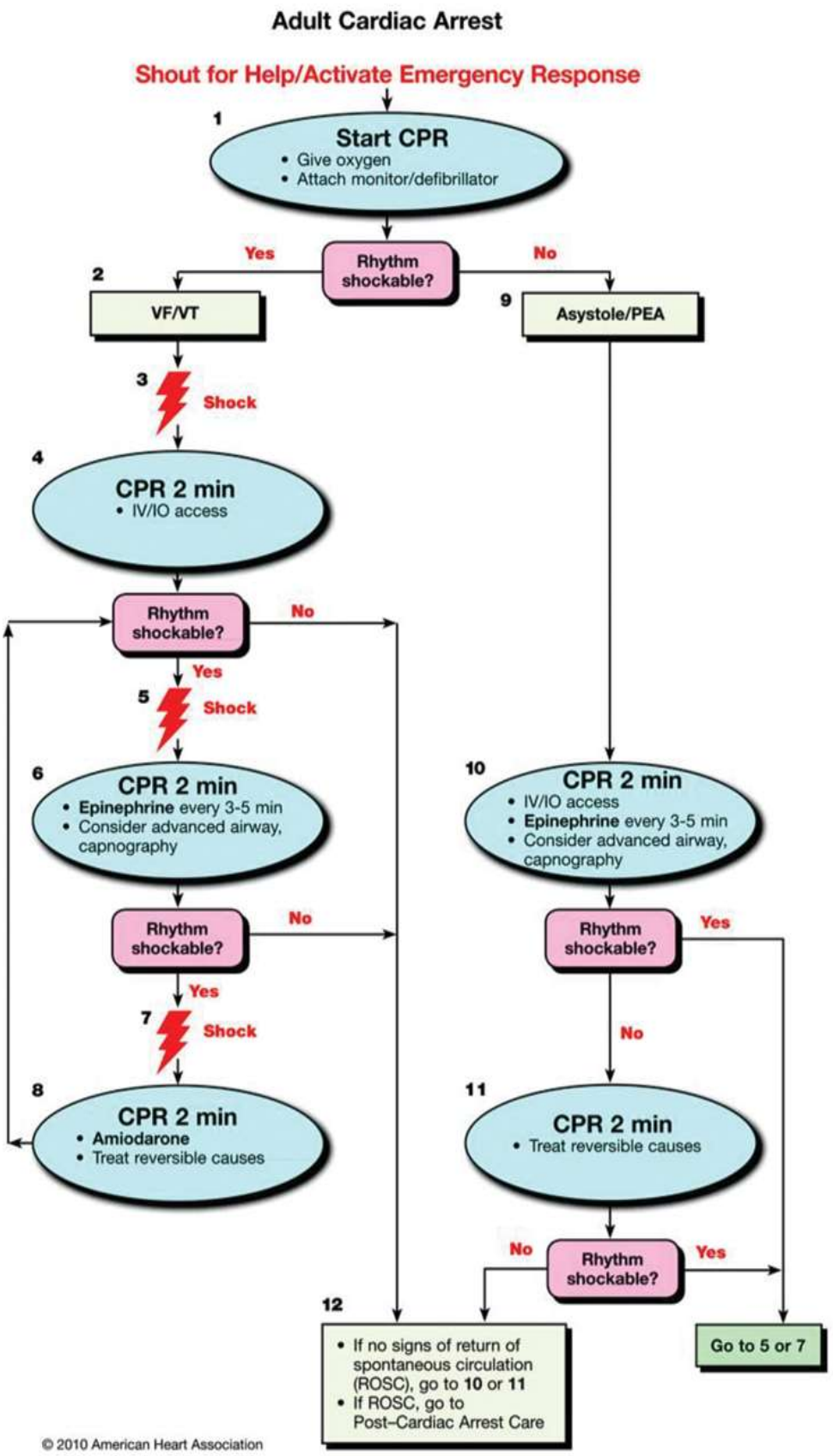
Previously PEA was termed as Electromechanical Dissociation (EMD) to describe patients who displayed electrical activity on cardiac monitor but lacked apparent contractile function because of an undetectable pulse. This means a weak contractile function is present – detectable only by invasive monitoring or echocardiography – but the cardiac function is too weak to produce a pulse or effective CO. This is also the most common initial condition present following successful defibrillation.

There are several reversible causes of PEA, popularly called 5 H's and 5 T's, including Hypovolemia, Hypoxia, Hydrogenion (acidosis), Hypo-/Hyperkalemia, Hypothermia and Tension pneumothorax, Cardiac Tamponade, Toxins, Coronary Thrombosis, Pulmonary Thrombosis.

In our case, cardiac arrest could have been due to hyperkalemia, and/or acidosis. The treatment of PEA is primarily directed towards treating the underlying cause as per the ACLS protocol.

On initial examination, if the patient is in cardiac arrest (unresponsiveness, no pulse/BP, no spontaneous respiration or only gasping), immediate chest compressions needs to be started within 10 seconds of identifying the diseased condition, i.e., PEA here. Drugs like Epinephrine 1mg every 3-5minutes or Vasopressin 40 U to replace the 1st or 2nd dose epinephrine can be given, along with other drugs as per the underlying cause of PEA.

The ACLS protocol for the treatment of PEA is charted as below.



CPR Quality
- Push hard (≥2 inches [5 cm]) and fast (≥100/min) and allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Rotate compressor every 2 minutes
- If no advanced airway, 30:2 compression-ventilation ratio
- Quantitative waveform capnography
  - If P_{ETCO_2} <10 mm Hg, attempt to improve CPR quality
- Intra-arterial pressure
  - If relaxation phase (diastolic) pressure <20 mm Hg, attempt to improve CPR quality

Return of Spontaneous Circulation (ROSC)
- Pulse and blood pressure
- Abrupt sustained increase in P_{ETCO_2} (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Shock Energy
- **Biphasic:** Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic:** 360 J

Drug Therapy
- **Epinephrine IV/IO Dose:** 1 mg every 3-5 minutes
- **Vasopressin IV/IO Dose:** 40 units can replace first or second dose of epinephrine
- **Amiodarone IV/IO Dose:** First dose: 300 mg bolus. Second dose: 150 mg.

Advanced Airway
- Supraglottic advanced airway or endotracheal intubation
- Waveform capnography to confirm and monitor ET tube placement
- 8-10 breaths per minute with continuous chest compressions

Reversible Causes
- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

Extract from ACLS (AHA) 2010 guidelines

Case History

A 61 years old female patient was brought by her attendants in a wheelchair in an unresponsive, unconscious and gasping state on 8th July 2015 at around 08:30 AM.

She was said to be in this state for about 20 minutes before her presentation in the ED. She was a known case of Diabetes mellitus, Hypertension, CAD Post-PTCA+Stenting and ESRD on maintenance hemodialysis (twice/week; last HD was 4 days back).

On examinations, patient was unconscious, unresponsive, and was gasping. Pulse was not palpable, BP was not recordable, SPO2 was 60% at room air. Cardiac monitor showed PEA.

Patient was immediately put on Bag-Mask ventilation and effective CPR was started as per ACLS protocol.

Two large bore IV cannulas were inserted and ABG was sent to the lab. During CPR inj epinephrine 2mg (1+1) was given. After 5 cycles (2 minutes) of CPR, ROSC was achieved in the ED.

Patient was intubated with ETT size 7.5 after giving Inj Etomidate 18mg and Inj Rocuronium 75mg and she was put on the ventilator with the following ventilator settings: ACV mode, FiO2 1.0, f 15, PEEP 5, TV 500.

Her ABG analysis showed: pH 6.88/ PO2 72mmHg / PCO2 84mmHg/ HCO3 15mmol/L/ Na+ 138mmol/L/ K+ 6.101mmol/L / Lactate 3.9

Inj Calcium Gluconate 10% 10ml was given over 10 minutes. Inj Fentanyl infusion was started @50mcg/hr. Inj Sodium bicarbonate 200ml was given stat.

Post-ROSC and Intubation, Pulse was 98/min regular, BP 170/110 mmHg, RR 18/min regular, SPO2 100% with FiO2 1.0 on ventilator. Patient was afebrile and RBS was 260 mg%

On Systemic Examination

RS: AE B/L equal but B/L basal crepts present. CVS: S1 S2 Normal, No murmur/bruit

PA: Soft, non-distended, BS+; No organomegaly

Neuro: Sedated and Paralysed; Pupils B/L 2mm and sluggishly reacting to light. Extremities: Warm, B/L Pedal edema++

AMPLE History

A - No known allergies

M - Regular Medication details not available

P - Known DM/HTN/CAD Post-PTCA+S/ ESRD on maintenance hemodialysis (2/week)

L - She had light breakfast that morning.

E - Events as per given history above.

She was provisionally diagnosed as PEA (Pulseless Electrical Activity) with Severe Metabolic Acidosis.

More investigations were sent as follows: CBC, LFT, KFT, ECG, CXR PA.

Foley's catheter no 14 was inserted and urine flow observed. Ryle's tube no 16 was inserted and its position was confirmed.

The following medications were given in the ED:

- Inj Emeset 4mg IV stat.
- Inj Pantoprazole 40mg IV stat.
- Inj Calcium Gluconate 10% 10ml was given over 10 minutes.
- Inj Dextrose 25% + Insulin 10 units IV stat.
- Inj Noradrenaline 5mcg/hr infusion started.
- Inj Fentanyl 50mcg IV stat and @ 50mcg/hr infusion started.

The case was discussed with the Nephrologist and the patient was admitted in ICU.

Cardiolology reference was given.

Her Vitals after 45 minutes of presentation

P 95/min regular BP 120/80 mmHg (on Noradrenaline @ 2.5mcg/hr) SPO2 100% on ventillator Cardiologist saw the patient at 09:25 AM and advised Troponin I. Guarded prognosis was explained to the attendants.

Patient was shifted to ICU AT 09:45 AM.

Patient was seen by Nephrologist and was started on SLED around 3 PM. Patient started gaining her consciousness by afternoon and was extubated in the evening.

Noradrenaline was tapered off and was stopped. Her vital stats were maintained throughout the day.

Reports of initial blood sampling

Urea 143mg/dl, Creatinine 9.83mg/dl, Na 135mmol/l, K 6 mmol/l, Calcium 8.2 mg/dl, Magnessium 2.9mg/l, Phosphorus 4mg/l, Hb 9.5gm/

dl, TLC 19900,Troponin-I Negative, Liver function test WNL. CXR showed B/L Infiltrates

Treatment given during the hospital stay

Tab. Azithromycin , Cap. Ecosprin AV, Inj. Elores (Ceftriaxone+Sulbactam), Inj. Novorapid , Inj. Ranitidine, Tab. Shelcal, Sub-Whey Protein Powder and other supportive medications. She remained stable in the ward.

Foley's catheter was removed the following morning (09/07/2015)

She underwent Hemodialysis on 9th July with ultrafiltrate of 2 L and was shifted to ward.

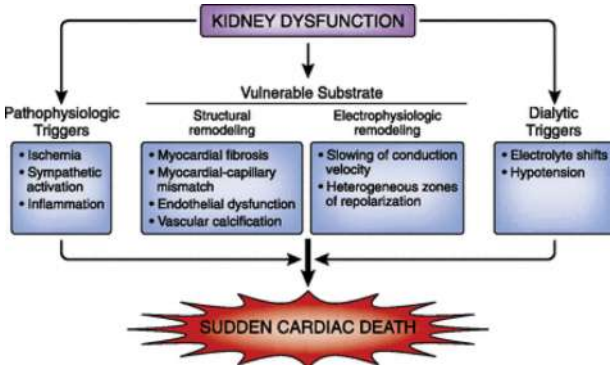
Reports

Na 130mmol/l, K 5.9mmol/l

She was discharged after 48 hrs in stable condition on 10/07/2015.

Discharge Medications

- Tab. Azithromycin 500mg once daily.
- Cap. Ecosprin AV 1 Capsule at bedtime.
- Inj. Elores 1.5gm IV twice daily.
- Inj. Novorapid SQ thrice daily started with low dose sliding scale.
- Tab. Ranitidine 150mg thrice daily.
- Tab. Shelcal 500mg thrice daily.
- Sub Whey Protein powder 2tsf twice daily.



After Discharge

She was followed up and hemodialysed on OPD basis thrice/week, last being on 25/07/2015, without any complications (followed up until 25/07/15).

Discussion

Chronic Kidney Disease (CKD) affects 13% of adults in the USA.

The majority of cardiovascular-related deaths in ESRD are attributable to SCD (Sudden Cardiac Death) events.

The incidence of SCD in the USA ranges approximately between 180,000 and 450,000 cases annually. Despite major advances in CPR and post-ROSC care, survival to hospital discharge after cardiac arrest remains very poor, estimated to be only 7.9% among out-of-hospital cardiac arrests that were eventually treated by emergency medical personnel.

The prognosis from cardiac arrests is even worse in patients with kidney dysfunction in which survival chances decreases with a declining GFR. The likelihood of survival following cardiac arrest is further low in dialysis patients.

Structural and electro-physiologic remodeling of the heart, vascular calcification and fibrosis, autonomic dysregulation, and volume and electrolyte shifts are some of the underlying processes thought to explain the increased predisposition for SCD in people with CKD.

This patho-physiology is depicted as in the flow chart below:

In patients with CKD, cardiomyopathy commonly occurs because of LV pressure and volume overload. Both atherosclerotic and arteriosclerotic vascular diseases also occur frequently.

This adverse cardiomyopathic and vasculopathic milieu predisposes individuals with CKD to arrhythmias, conduction abnormalities, and sudden cardiac death, which is likely to be exacerbated by electrolyte shifts, divalent ion abnormalities, diabetes, and sympathetic over-activity, in addition to inflammation and possibly iron deposition.

Impaired baroreflex effectiveness and sensitivity, as well as obstructive sleep apnea might also contribute to the risk of sudden death.

Cardiac arrest, due to whatsoever reason, does not give much time for interventions.

Therefore early recognition of cardiac arrest, immediate interventions like CPR and ACLS drugs administration and treating the underlying cause, are most important in not only reviving the patient but also to reduce mortality after revival. Agonal gasps can mislead the medical staff in detecting cardiac arrest. Agonal gasps are not adequate breathing; it

looks like the patient is drawing air in very quickly but if the patient is not responding to commands, it is actually a sign of cardiac arrest and must be intervened quickly, as done in our case.

The more the delay in interventions, the less the chances of survival and if revived, more the chances of end-organ damage like brain and kidneys.

This case report shows that early recognition of cardiac arrest and timely interventions and correcting the possible underlying cause of PEA, lead to revival of the patient without any end-organdamage.

Conclusion

Any cardiac arrest patient should be intervened early.

As Emergency physicians any such presentation should be considered with evidence based approach (as the ACLS 2010 guidelines in our case).

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