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Editorial Board

General Information

The Journal of Diabetic Association Medical College, Faridpur (JDAMC) is the official journal of Diabetic Association Medical College, Faridpur. The journal is published twice in a year i.e. January and July. It accepts original articles, review articles, and case reports. Complimentary copies of the journal are sent to libraries of all medical and other relevant academic institutions in the country and selected institutions abroad.

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Editorial

A Suicidal Teen: Help Knowing the Warning Signs and Risk Factors

Akhiruzzaman

Introduction

Suicide is such an unexplained, meaningless, tragic, contradictory and mystified complex public health problem having dimensions of physical, biological, somatic, mental, psychological, psychiatric, cultural, social and spiritual phenomenon. In spite of the several identified background factors, the real reasons behind suicide is not clear, because suicide is multi-causal, and can never be traced back to one single cause. However, the strongest suicide risk factor is an unrecognized and untreated mental disorder. Suicide among young people is one of the most serious public health problems.[1] According to the Centers for Disease Control (CDC) suicide is the second leading cause of death for teens and young adults.[2] The suicide rate among teenage girls reached an all-time high in 2015. The analysis found an increase in teen suicides across the board between 2007 and 2015. The suicide rate increased 31% for teen boys and doubled for teen girls during this time period[3]

The numbers are a sobering reminder that suicide is a growing public health concern and that teens are a particularly vulnerable group. Research shows that teen depression is on the rise [4] and suicide is always a risk during the course of a major depressive episode.[5] World Health Organization Global Health Observatory estimates reported suicide rate in Bangladesh is 7.8 per 100,000 population in 2012.[6] However, in a paper published by Feroz et al., reported that suicide rate was 30.4% below 20 years of age in Bangladesh.[7] The Bangladesh Police are the only authorities to keep track of the suicides in the country. According to their 2017 statistics, on average around 30 people commit suicide every day. Statistics show that 9,665 people committed suicide in 2010 and the number rose to 11,095 in 2017 among them the largest portion was below 20 years of age. But the police, activists and experts concur strongly that the actual numbers would be much higher as many incidents go unreported.[8]

So it is time to take action against teen suicide. At first we have to know about the warning signs of suicide and also know about, how to help the teens to prevent the catastrophic event.

Warning signs for teen suicide

Four out of five teens who attempt suicide give warning signs. But often, those signs are missed or ignored.[9] All teens are different and many are adept at masking their feelings. To the end, it isn't always possible to predict signs of suicidal ideation. Many do, however, exhibit some symptoms. The following are some (but not the only) potential warning signs of suicidal ideation as follows.[10]

- Talking about death, suicide, and/or self-harm
- Changes in personality or behavior that is out of character
- Talking about feeling worthless, helpless, and/or hopeless
- Changes in sleep patterns and eating habits
- Risky or self-destructive behavior
- Lack of concentration and changes in school performance
- Isolating from peers and/or family
- Giving away prized possessions
- Expressing feelings of overwhelming shame and guilt, and making statements that others don't care or others will be better off without me
- Lack of hope for the future, feeling like things can't possibly improve
- Visiting or calling on loved ones
- Getting affairs in order.

Factors trigger suicidal behavior in teens:[2,10,11]

- Depression, anxiety and other mental health disorders
- Anger
- Sadness
- Rejection by peers
- Loneliness
- Irritability
- Social issues
- Family discord
- Social media use (Negative ideation)

There are also risk factors that put youth at an increased risk level for suicidal ideation:

- Perfectionism
- Substance abuse
- History of sexual or physical abuse
- Low self-esteem
- Academic struggles
- Teens lacking social and family support
- Family history of suicide.
- Frequent conflicts with friends or family

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- Impulsive behavior
- A tendency to take unhealthy risks (behaviors that could result in physical harm)

Help a suicidal teen

Suicide remains the second leading cause of death among teens. To prevent this consequence everybody should be aware and ready to help a suicidal teen.

Help your Friend:[12, 13]

1. Know the warning signs. Read over the list above and keep it in a safe place.
2. Do not be afraid to talk to your friends. Listen to their feelings. Make sure they know how important they are to you, but don't believe you can keep them from hurting themselves on your own. Preventing suicide will require help from friends.
3. Make no deals. Never keep secret a friend's suicidal plans or thoughts. You cannot promise that you will not tell or disclose to others. You have to tell to save your friend.
4. Tell an adult: Talk to your parent, your friend's parent, your school's psychologist or counselor or any trusted adult. Don't be afraid that the adults will not believe you or will not take you seriously. Keep talking until they listen, Even if you are not sure your friend is suicidal, talk to someone. This is definitely the time to be safe, not sorry!
5. Ask if your school has a crisis team: Many schools have organized crisis teams, which include teachers, counselors, social workers, school psychologists and principals. These teams help train all staff to recognize warning signs of suicide as well as how to help in a crisis situation. These teams can also help students understand warning signs of violence and suicide. Whether or not you think someone at your school might be suicidal, find out if your school has a crisis team in place. If your school does not have a crisis team, ask your student council or faculty advisor to look into starting a team
6. Pay attention to their language and behavior: People often talk about suicide in vague or unclear ways.Your friend could say things that reflect a sense of shame, hopelessness, or failure. They may not say, “I want to die,” or “I want to kill myself.” Instead, they might say:“I just want the pain to stop.” or “I don't know if I can go on.” or “I'm a burden to everyone.” or “I'll never feel better.”
7. Offer compassion: To validate their feelings do not argue, respect their opinion and offer hope e.g: “That sounds so painful, and I appreciate you sharing that with me. How can I help?” “I know things seem bleak now, but it can be hard to see possible solutions when you feel so overwhelmed.” “I'm concerned about you because I care, and I want to offer support however I can. You can talk to me.”

Help your family members:

As a family member of a suicidal teen you can do the following things:[10]

Make time to connect with your teen 1 on 1, If your teen keeps a busy, high-pressure schedule, find ways to slow down and decrease commitments, Encourage your teen to connect with positive, supportive friends, Practice relaxing activities together (going for a walk, journaling, drawing, using a mindfulness app), Exercise together, Help your teen create a list of people to call or text when feeling overwhelmed,

Acknowledge your teen's pain and validate your teen's emotions, Be patient, it will take time to break the cycle, Remove means of suicide from suicidal teens, and reach out to the school counselor to assist with accommodations in school

Help your students:

Every student spent a huge amount of time at school and with their teachers. So a teacher can do the following things:[1]

1. Strengthen students' self-esteem.
2. Increase the level of mental education/nurturing.
3. Help the students to express emotion in controlled and positive way.
4. Prevent bullying and violence at school.
5. Provide information about mental care services.
6. Recognize and treat every depressed student.
7. Built trustful communication.
8. Improve school staff's communication skills.
9. Every school needs to have an emergency plan on how to inform school staff, especially teachers, fellow pupils and parents when suicide has been attempted or committed in school.

Conclusion

Thoughts of suicide, even if it seems vague, should always be taken seriously. If someone is at risk for suicide, it's better to take action and offer help. Nobody can control someone's thoughts, but some words and actions have more power than people think. Feeling hopeless, helpless, loneliness or can result in extreme emotional pain and depression. Sometimes these feelings result in thoughts of suicide, but it is important to let the person (with suicidal thoughts) know that *‘you are not alone, there is help and hope’*.

References

1. Kalmar S. The possibilities of suicide prevention in adolescents.A holistic approach to protective and risk factors.Neuro PsychoPharmacol Hung. 2013 Mar 1;15(1):27-39.
2. Risk and Protective Factors [Internet]. Cdc.gov. 2021 [cited 26 December 2021]. Available from:

https://www.cdc.gov/suicide/factors/index.html

3. Centers for Disease Control and Prevention, "*Quick Stats:* Suicide Rates for Teens Aged 15-19 Years, by Sex- United States, 1975–2015," *Morbidity and Mortality Weekly Report*, August 4, 2017 / 66(30); 816.
4. Mojtabai R.,Olfson M., Han B. "National Trends in the Prevalence and Treatment of Depression in Adolescents and Young Adults," *Pediatriacs*, November, 2016.
5. American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition*, American Psychiatric Publishing, Washington, D.C., 2013: Pages 160-168
6. GHO by Category Suicide Rates-Data by Country. Available online: http://apps.who.int/gho/data/view.main.MHSUICIDEv
7. Feroz, A., Islam, S.N., Reza, S., Rahman, A.M., Sen, J., Mowla, M., &Rahman, M.R. (2012). A Community Survey on the Prevalence of Suicidal Attempts and Deaths in a Selected Rural Area of Bangladesh. *Journal of Medicine, 13(1).* doi:10.3329/jom.v13i1.10042
8. Examining the alarming suicide trends in Bangladesh [Internet]. Dhaka Tribune. 2021 [cited 26 December 2021]. Available from: https://archive.dhakatribune.com/opinion/special/2018/05/08/examining-alarming-suicide-trends-bangladesh
9. Warning Signs. The Jason Foundation, Inc. [Internet]. The Jason Foundation, Inc. 2021 [cited 26 December 2021]. Available from: https://jasonfoundation.com/youth- suicide/warning-signs
10. Teen Depression and Suicide Risk Factors [Internet]. Psycom.net- Mental Health Treatment Resource Since 1996. 2021 [cited 26 December 2021]. Available from: https://www.psycom.net/teen-suicide-risk-factors
11. Teens and Depression: Diagnosis, Treatment, and Risks [Internet]. Psycom.net - Mental Health Treatment Resource Since 1996. 2021 [cited 26 December 2021]. Available from: https://www.psycom.net/depression.central.teens.html
12. Save a Friend: Tips for Teens to Prevent Suicide [Internet]. National Association of School Psychologists (NASP). 2021 [cited 26 December 2021]. Available from: https://www.nasponline.org/resources-and-publications/resources-and-podcasts/school-safety-and-crisis/mental-health-resources/preventing-youth-suicide/save-a-friend-tips-for-teens-to-prevent-suicide
13. How to Help a Suicidal Friend: 11 Tips [Internet]. Healthline. 2021 [cited 26 December 2021]. Available from: https://www.healthline.com/health/mental-health/how-to-help-a-suicidal-friend#helpful-resources

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Original Article

Antioxidant Status in Type-2 Diabetes Mellitus Compared to Diabetic Foot Patients- A Hospital Based Study in Rajshahi

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Abstract

Background: Chronic hyperglycemia in diabetes mellitus leads to decrease total antioxidant level in the body which might play important role in the development of chronic complications of diabetes due to oxidative stress.

Objective: It is aimed to evaluate the antioxidant status in type-2 diabetes mellitus patients & diabetic foot patients.

Methods: This was a descriptive cross sectional study carried out in the department of Pharmacology & Therapeutics in collaboration with Rajshahi Diabetic Association General Hospital, Rajshahi from July 2017 to June 2018 to evaluate total antioxidant status in type-2 diabetes mellitus. In this study, 20 patients with type-2 diabetes mellitus and 20 diabetic foot patients were evaluated. Antioxidant status was determined by using spectrophotometer. Most of the patients were age ranging 40-60 years of both sexes.

Results: The mean of fasting blood glucose and total antioxidant capacity of type-2 diabetes mellitus with diabetic foot patients was 11.23±2.76 mmol/l and 18.78±6.70 µmol/l and that of type-2 DM patients was 9.13±2.57 mmol/l and 26.07±7.67 µmol/l. Antioxidant level were less in type 2 diabetic foot patients.

Conclusion: There is low level of antioxidant in plasma which may be regarded as an important causative factor for development of type-2 diabetes mellitus and its chronic complication. The findings can be a basis of generating a hypothesis for further testing towards a meaningful conclusion.

Key words: Antioxidant status, type-2 diabetes mellitus, Diabetic foot.

Introduction

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by chronic hyperglycemia. The prevalence of type-2 DM in Bangladesh is alarmingly increasing over the past two decades and continues to rise as a disease of immeasurable class. The World Health organization has predicted that the worldwide number of patients with diabetes will double by the year 2025.[1] Human body is continuously exposed to different types of agents that results in the production of reactive species (RS) called free radicals. Free radicals transfer their unpaired electron and cause the oxidation of cellular machinery. In order to encounter the deleterious effects of such species, body has got endogenous and exogenous antioxidant that neutralizes such species and keeps the homeostasis of the body. Any imbalance between the reactive species and antioxidant leads to produce a condition known as oxidative stress that results in the development of different pathological conditions of which diabetes and its later complications are very important.[2] Persistent uncontrolled hyperglycemia secondary to insulin resistance and diminished insulin secretion in type-2 DM leads to many complications such as diabetic ketoacidosis, hyperosmolar coma, ischemic heart disease, chronic kidney failure, retinopathy, neuropathy, non-ketotic hyperosmolar coma and foot ulcer.[3] Hyperglycemia causes release of tissue damaging reactive oxygen species (ROS) and diminishes antioxidant agents. Diabetes mellitus not only stimulates the generation of reactive oxygen species, but also impairs the ability of a cell or tissue to cope up with the increased oxidative burden. Oxidative stress is an important mediator of diabetic complication.[4] The most notorious complications of

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diabetes in the lower extremity are the diabetic foot which is commonly observed in our country. For this reason, it is important to determine the total antioxidant status in patients with type-2 diabetes mellitus compared to its complication.

Materials & Methods

This was a descriptive cross-sectional study conducted in the Department of Pharmacology and therapeutics, Rajshahi Medical College, in collaboration with Rajshahi Diabetic Association General Hospital, Rajshahi. The study was conducted among 20 type-2 diabetes mellitus & 20 type-2 DM with diabetic foot.
The inclusion criteria were, Clinically diagnosed type-2 diabetes mellitus & type-2 DM with diabetic foot patients in the age group of 40-60 years, both genders.

The exclusion criteria were, Patients with serious comorbid diseases (stroke, myocardial infarction, major surgery etc, patients with liver and kidney dysfunction, history of using drugs significantly affect glucose metabolism (glucocorticoids, oral contraceptives, thiazide diuretics etc.) or taking vitamin supplements. The study variables were age, total antioxidant status (µmol/l) and glucose (mmol/l).

A formal permission was obtained from the Ethical Review Committee of Rajshahi Medical College, Rajshahi to select this study. After getting permission from the concerned authority, every patient was informed about the study and they were also informed that there was no chance of any significant harm by inclusion in this study. The data were collected from inpatients as well as outpatients fulfilling the inclusion criteria attending Rajshahi Diabetic Association General Hospital, Rajshahi. An elaborate history was taken for each individual regarding present & previous history of illness suggesting type2 diabetes mellitus and any diabetic complication. After taking informed consent, complete history taking, physical examination was done and recorded in a preformed data sheet. Then 4 ml blood was taken from each group of patients in a test tube containing anticoagulant tri-potassium EDTA (Ethylene di-amine tetra acetic acid). Plasma was collected after centrifuging for 15 minutes at 3000 rpm. Then plasma total antioxidant status was measured in Pharmacology laboratory of Rajshahi Medical College, Rajshahi. The generated laboratory data were recorded using a prepared checklist. Then the data were then analyzed using SPSS version 16 for Windows by applying descriptive statistics and cross tabulation. Frequency and percentages were calculated. The unpaired t-test was used for comparing means. Significance was kept at p-value less than 0.05.

Measurement of total antioxidant status:

Total antioxidant activity was measured by ferric reducing antioxidant power assay (FRAP) of Benzie and Strain 1999.

Principle:

The FRAP assay uses antioxidants as reluctant in a rodox-linked colorimetric method employing an easily reduced oxidant, Fe(III). Reduction of a ferric tripyridyltriazine complex to ferrous-$(2,4,6\text{-tripyridyl-s-triazine})_2$ ie: Ferric (III) [colorless] to Ferrous (II) [blue] can be monitored by measuring absorbance at 593nm. The absorption readings are related to the reducing power of the electron-donating antioxidants present in the test compound. Hence the FRAP assay can rank the reducing power and the antioxidant potential of a wide range of test compounds.

Sample:
Plasma

Reagents:

300 mM Acetate buffer

40 mM HCl

10 mM TPTZ

20 mM $FeCl_3.6H_2O$

0.001 M $FeSO_4.7H_2O$ Standard

FRAP working reagent

Sample: 10:1:1 (acetate buffer: TPTZ:$FeCl_3.6H_2O$)

Standard: 10:1:1 (acetate buffer: TPTZ:H_2O)

Procedure:

100 µl plasma was mixed with 900 µl distilled water and 2 ml of FRAP working reagent and absorbance 593 nm was measured after 30 min against FRAP reagent blank. Standard were preceded in same manner. The result was expressed as µM/L of ferrous equivalent.

Calculation:

The FRAP equation is:
FRAP value of sample (µM)= {Abs(sample) x FRAP value of Std (µM)} / Abs(Std)

Parameters of study:

Demographic parameter: Age & duration of the disease
Study parameter: Total antioxidant status

Results

Table 1: Demographic parameters of type-2 DM and type-2 DM with Diabetic Foot patients

Parameters	Group	
	DM (Mean±SD)	DM with diabetic foot (Mean±SD)
Age	51.75±5.12	53.95±5.48
Duration (year)	5.63±3.58	7.5±2.60

Table-1 Shows that demographic parameter of each group. The mean age and duration of disease in DM with diabetic foot were more than that of DM patients.

Table 2: Study parameter of type-2 DM and type-2 DM with Diabetic foot patients.

Variables	Group	
	DM patients (20)	**DM with diabetic foot patients (20)**
FBS (mean±SD)	9.13±2.57 **mmol/l**	11.23±2.76 **mmol/l**
TAC (mean ±SD)	26.07±7.67 **µmol/l**	18.78±6.70 **µmol/l**

Table 2 Shows that 20 patients belonged to each group. The mean of FBS and TAC in type-2 DM with diabetic foot was 11.23±2.76 mmol/l and, 18.78±6.70 µmol/l. The mean of FBS & TAC in type-2 DM was 9.13±2.57 mmol/l & 26.07±7.67 µmol/l. The mean of FBS were more in DM with diabetic foot patients. The mean of TAC was lower in DM with diabetic foot patients.

Table 3: Comparison of biochemical parameter between type-2 DM and Diabetic foot patients

Variables (biochemical characteristics)	Group		Test of Significance
	DM (Mean±SD)	**DDM with diabetic foot (Mean±SD)**	
FBS	9.1350±2.56767	11.2300±2.75702	**t**=-2.487**df**=38 **P**=.017
TAC	26.0750±7.67133	18.7800±6.70299	**t**=3.202 **df**=38 **P**=.003

Table 3 Shows that biochemical parameters between DM patients and DM with diabetic foot patients. It was observed that biochemical parameters were statistically significant ($P<0.05$) when compared between these two groups.

Discussion

In this study, 20 were type-2 DM patients and 20 were type-2 DM with diabetic foot patients. The mean age and duration of disease of type-2 DM and type-2 DM with diabetic foot was 51.75±5.11, 5.62±3.58 and 53.95±5.48, 7.50±2.60 years respectively. It was found that the mean of FBS and TAC of type-2 DM with diabetic foot was 11.23±2.75 mmol/l and 18.78±6.7 µmol/l. The mean of FBS and TAC of type-2 DM was 9.13±2.56 mmol/l and 26.07±7.67 µmol/l. These results revealed that FBS were significantly increased in type-2 DM with diabetic foot and TAC was decreased in type-2 DM with diabetic foot patients compared with type-2 DM patients. The similar findings observed by Bolajoko *et al.* (2008)[5] and Pinaki Saha *et al.* (2015)[6]. They suggested that decreased level of antioxidant and elevated oxidative stress associated with increased risk of type-2 DM and its complications. A study performed by Kedziora-Komatowska *et al.* (1998)[7]; Bandeira *et al.* (2013)[8]; Li *et al.* (2013)[9] and Ganjifrockwale *et al.* (2017)[10] showed the low level of TAS in patient with type-2 DM & type-2 DM with complication compared to healthy individual. This is in agreement to the present study. In our study it was observed that decreased TAC in type-2 DM with diabetic foot patient compared to type-2 DM (18.78±6.7 µm/l; 21.07±7.67 µm/l). Bikkad *et al.* (2014)[11]; Chopra *et al.* (2012)[12] and Djordjevic *et al.* (2014)[13] performed a study and found that decreased level of enzymatic antioxidant in type-2 DM with complication compared to type-2 DM. Oliveira *et al.* (2014)[14] performed a study and found decreased level total antioxidant capacity (0.61±0.09 mg/ml) and reduced protein thiol (369.22±46.13 mg/ml) in type-2 DM with diabetic foot patient compared to type-2 DM without diabetic foot (0.69±0.06 mg/ml; 413.41±28.71 mg/ml) which was similar to the present study.

Conclusion

Finding in this study are compatible with the hypothesis that persistent hyperglycemia leads to decreased TAC in diabetic patients. This is more pronounced in patients with diabetic foot. A high level of lipid peroxidation accompanied by insufficient antioxidant capacity in plasma could attribute to the chronicity of diabetes mellitus disease. Thus delivery of antioxidants and employing the mechanism based approach; clinical pathology and concentration based dosage schedule in antioxidants trial might help us in preventing development of complication of type-2 diabetes. However, the findings are having the scope for generating a clear hypothesis for further testing towards a significant conclusion of the facts.

Conflict of interest: No

References

1. Siddique MAH, Begum A, Begum S, Khan MH, Saiedullah M, Haque, A, Rahman MN and Ali L. Comparison of Antioxidative Effects of Biguanides and Sulfonylureas Monotherapy on Total Antioxidant Status in Newly-Diagnosed Patients with Type 2 Diabetes Mellitus. Diabetes Case Re 2016;1(1):1-5.
2. Ullah A, Khan A and Khan I. Diabetes mellitus and oxidative stress-A concise review. Saudi Pharmaceutical Journal. 2016;24:547-553.
3. Dries SS, Soares BDS, Verza SG, Ziulkoski AL, Linden R, Andrade FMD and Perassolo MS. Oxidative stress in patients with type-2 diabetes mellitus treated with metformin. Sci Med. 2017;27(2):1-8
4. Vairamon SJ, Babu M and Viswanathan V. Oxidative Stress Markers Regulating the Healing of Foot Ulcers

in patients with Type 2 Diabetes. WOUND. 2009;21(10):273-279.

5. Bolajoko EB, Mossanda KS, Adeniyi F, Akinosun O, Fasanmade A and Moropane M. Antioxidant and Oxidative stress status in type-2 diabetes and diabetic foot ulcer. SAMJ. 2008;98(8):614-617.
6. Saha P, Banerjee P, Auddya L, Pal P, Das M, dutta M, Sen S, Mondal M C., Kumar, A. and Biswas UK. Simple Modified Colorimetric Methods for Assay of Total Oxidative Stress and Antioxidant Defense in Plasma: Study in Diabetic Patients. iMedPub Journals. 2015;5(1):1-7.
7. Kedziora-Kornatowska KJ, Luciak M, Blaszczyk J and Pawlak W. Lipid peroxidation and activities of antioxidant enzymes in erythrocytes of patients with non-insulin dependent diabetes with or without diabetic nephropathy. Nephrol Dial Transplant. 1998;13(1):2829-2832.
8. Bandeira SDM, Fonseca LGSD, Guedes GDS, Rabelo LA, Goulart MOF and Vasconcelos SML. Oxidative Stress as an Underlying Contributor in the Development of Chronic Complication in Diabetes Mellitus. International Journal of Molecular Sciences. 2013;3265-3284.
9. Li J, O W, Li W, Jiang ZG and Ghanbari HA. Oxidative Stress and Neurodegenerative Disorders. Int. J. Mol. Sci. 2013;14:24438-24475.
10. Ganjifrockwala FA, Joseph JT and George G. Decreased total antioxidant levels and increased oxidative stress in South African type 2 diabetes mellitus patients. Journal of Endocrinology, Metabolism and Diabetes of South Africa. 2017;22(2):21-25.
11. Bikkad MD, Somwanshi SD, Ghuge SH and Nagene NS. Oxidative Stress in Type II Diabetes Mellitus. Biomed Res-India. 2014;25(1):84-87.
12. Chopra M, Kaul K, Tarr J, Choudhury R, Kohner EM and Chibber R. Oxidative stress in diabetic neuropathy: source of reactive oxygen species. Endocrinology Studies. 2012;2(6):22-28.
13. Dejordjevic GM, Djuric SS, Dejordjevic VB, Apostolski S and Zivkovic M. The Role of Oxidative Stress in Pathogenesis of Diabetic Neuropathy: Erythrocyte Superoxide Dismutase, Catalase and Glutathione Peroxidase Level in Relation to Peripheral Nerve Conduction in Diabetic Neuropathic Patients. Research Gate. 2014;152-177.
14. Oliveira ACP, Teixeira CJ, Stefanello TF, Carrara MA, Bazotte RB, Sa-Nakanishi AB, Comer JF and Batista MR. Oxidative Stress Parameters as Biomarkers of Risk Factor for Diabetic Foot among the Patients with Type 2 Diabetes. Braz. Arch. Biol. Technol. 2014;57(2):223-227.

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Original Article

Role of Dexamethasone in Post-tonsillectomy Morbidities

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Abstract

Objective: The aim of this study was to find out the effect of Dexamethasone on pain following tonsillectomy. In addition to post-tonsillectomy pain, this study also explore the comparative outcome of use of additional analgesic, fever, nausea & vomiting as well as oral intake of semisolid food between the groups.

Methods: A double blind, randomized control trial using a single pre-operative dose of Dexamethasone (0.15mg/kg body weight) or normal saline in the randomly selected 100 patients of age more than 3 years, undergoing tonsillectomy fulfilling inclusion criteria of this study, in the Department of Otolaryngology and head Neck surgery, Dhaka Medical College Hospital, Dhaka during July 2009 to June 2010. Surgical techniques, anesthetic drugs, use of analgesic and other medication were standardized. Both groups were assessed clinically at 6, 12 and 24 hours post-operatively for pain, requirement of analgesic, fever, nausea & vomiting, oral intake, condition of the tonsillar fossa and reactionary haemorrhage. Visual Analogue Scale (VAS) used for the assessment of pain.

Results: Post-operative pain was assessed by Visual Analogue Scale (VAS) in both Dexamethasone and Control groups. Mean±SD of the VAS in Dexamethasone and Control groups at 6 hours was 5.04 ± 1.09 vs 5.96 ± 1.03 ($p = < 0.0001$), at 12 hours was 3.94 ± 0.93 vs 5.14 ± 0.61 ($p = < 0.0001$) and at 24 hours was 2.64 ± 0.69 vs 3.84 ± 0.87 ($p = < 0.0001$). Requirement of additional analgesic ($p<0.05$), fever ($P<0.001$), Post-Operative Nausea and Vomiting (PONV) ($P < 0.005$), oral intake of semisolid food ($P<0.005$) between Dexamethasone and control group were significantly different. Regarding condition of the tonsillar fossa and reactionary haemorrhage, no significant differences were observed.

Conclusion: A single preoperative IV dose of Dexamethasone (0.15 mg/kg), given just before starting surgery, provided good and prolonged analgesia. It also reduces fever, nausea & vomiting and odynophagia. Those enhance earlier and better postoperative quality of oral intake and decrease morbidities.

Keywords: Tonsillectomy, Dexamethasone, VAS.

Introduction

The history of tonsillectomy dates back over 2000 years when Celsus used the first primitive scalpel in approximately 50 AD to remove tonsil tissue. The earliest description of the procedure was by Paul of Aegina in 625 AD. Approximately, 600,000 tonsillectomies are performed in each year on children and adults. According to the American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS), it is the second most common paediatric surgery. A recent survey conducted by Harris Interactive on behalf of the AAO-HNS identified that pain associated with tonsillectomy was a top concern for more than 90% of the parents surveyed.[1] Despite advances in anaesthetic and surgical techniques, post-tonsillectomy morbidity (emesis, poor oral intake, pain and bleeding) remains a significant clinical problem for the patient, family and physician.[2]

As the postoperative pain is a significant concern after tonsillectomy. This pain inhibits chewing and swallowing, which leads to dehydration and contributes to lassitude and delayed recovery of strength and well-being. Many studies have attempted to use various interventions to reduce postoperative pain. Dexamethasone is one of the drugs used to reduce postoperative pain in various operations.[3] The patients also return to a normal diet more quickly and there appear to be no serious adverse effects.[4,5] Dexamethasone has anti-inflammatory actions. These are mediated by inhibition of production of inflammatory cell factors, such as cytokines in macrophages, monocytes, and lymphocytes, which results in decreased extravasation of leukocytes, lysosomal enzyme release, and vascular permeability in areas of injury. This reduces oedema and decreases fibrosis during healing. By inhibiting phospholipase A_2 enzyme, corticosteroids block both the cyclooxygenase and lipoxygenase pathway and thus prostaglandin production, thereby leading to pain relief.[6]

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In an attempt to formulate an objective recommendation regarding the efficacy of routine use of a single, intra-operative, intravenous dose of corticosteroid on post-tonsillectomy pain, a systematic review of the published literature was performed using established meta-analysis techniques with a predetermined protocol.[7,8] As Dexamethasone reduces pain, so patient returned to normal diet and activity with in early postoperative period. The Dexamethasone is sometimes given in a single intravenous dose during surgery to try to prevent vomiting after the operation. The review of trials found that single dose of corticosteroid during tonsillectomy or adenotonsillectomy can prevent vomiting, as Dexamethasone has antiemetic properties and used in chemotherapy as an antiemetic.[3] Dexamethasone for tonsillectomy has become standard care in many institutions. The dose response of Dexamethasone for prevention of Post-Operative Nausea and Vomiting (PONV) symptoms in paediatric tonsillectomy remains unclear, although doses up to 1mg/kg have been tested.[9-12] If Dexamethasone were indeed analgesic in this setting, fewer postoperative analgesics such as NSAIDs would be needed postoperatively and theoretically, patients would be less at risk of postoperative bleeding.[13]

A Visual Analogue Scale (VAS) is an instrument that is used for measuring various parameter in medical science. VAS tries to measure a characteristic or attitude that is believed to range across a continuum of values and cannot easily be directly measured. Operationally a VAS is usually a horizontal line, 100 mm in length, anchored by word descriptors at each end. The patient marks on the line the point that they feel represents their perception of their current state. The VAS score is determined by measuring in millimetres from the left hand end of the line to the point that the patient marks.[14]

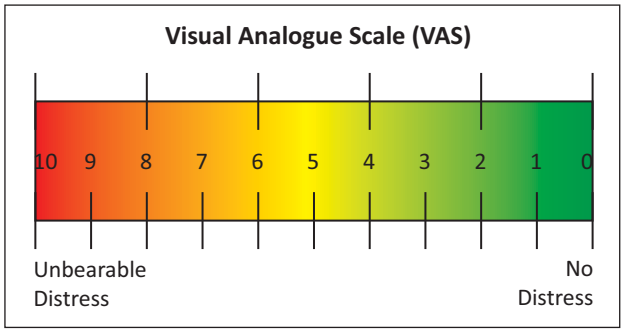
The results of this study with wide number of variables will reflect the overall effect of the Dexamethasone in post-operative morbidities. The aim of the present study is to find out the effect of single pre-operative dose of Dexamethasone on pain and other morbidities following tonsillectomy.

Materials and Methods

This is a double blind (Blind-1 is Surgeon and Blind-2 is Data Collector), randomized control trial and was conducted in Department of ENT, Dhaka Medical College Hospital, Dhaka, during the period of July 2009 to June 2010 after getting permission from Ethical Committee of this institute. According to inclusion and exclusion criteria, total 100 patients were included in this study. The patients were randomly divided into two groups, based on odd and even serial number of the subjects during their inclusion in this study. Odd serial numbers were included in Control group and even serial numbers were included in Dexamethasone group. The researcher gave a single pre-operative I.V. dose of Dexamethasone (0.15mg/kg body weight) or normal saline to these patients undergoing tonsillectomy fulfilling the inclusion criteria of this study.

Data generation procedures:

Anesthetic drugs used, surgeons, surgical technique, use of analgesics and other medication were standardized in both groups. Tonsillectomy was done by cold dissection method under general anaesthesia with endotracheal intubation. Surgeons of same level of experience were carried out the surgery. Haemostasis was done by ligation and electro-coagulation. Postoperative assessment and data collection were done by an assistant surgeon of the Department of ENT, Dhaka Medical College Hospital, Dhaka. Both groups were clinically assessed for pain, additional analgesics, fever, nausea, vomiting, oral intake, condition of the fossa and haemorrhage. All the generated data were recorded on a questionnaire form at 6, 12 and 24 hours after operation. A Zero to Ten point Visual Analog Scale (VAS) was used for pain assessment. Generally, higher numbers in VAS indicate more pain (0 = No pain, 10 = Most imaginable pain). The pain level was graded in to following types: VAS score 2 - 4 = Mild pain, VAS score 4 - 6= Moderate pain and VAS score 6 and above = Severe pain.



Paracetamol was used as a first line analgesic at 10 mg/kg body weight per dose, if pain is more than 4 in Visual Analogue Scale (VAS) in both groups. If the pain was not controlled with the paracetamol, then additional analgesics (Diclofenac injection or suppository) was added. Regarding fever, nausea & vomiting data was collected within the 24 hours post-operatively from both groups. Numbers of episodes of vomiting were also noted. Data about the pattern of oral intake of the semisolid food was collected at 24 hours. The pattern of the oral intake was judged as follows: Excellent = Patient requests it, Good = Patient accepts it when offered and Poor = Patient refuses. Data for the reactionary haemorrhage and condition of the fossa were collected at 24 hours after surgery. All the collected data was checked and compiled first. Then they were processed and analyzed with the help of software Statistical Package for Social Science (SPSS -17). Parametric data were analyzed using unpaired 't' test and non-parametric data were analyzed using Chi square (χ^2) test. $P < 0.05$ was considered as significant. The generated data was presented as a tabulated form.

Results

This study comprising of 100 patients between 3-40 years of age. Patients were randomly divided into two groups with 50 patients in each group. The mean age of Dexamethasone

group was 15.90 ± 6.71 years (5-33), while in Control group, the mean age was 16.12 ± 6.72 years (7-28). Most of the patients participated in the study (60%) was from 10 to 20 years. Fifty one percent of the study population was male and 49% was female. In Control group, there was 23 (46%) male and 27 (54%) female. In Dexamethasone group, there was 28 (56%) male and 22 (44%) female. There were no significant differences in age and sex distribution of the patients. The visual analogue scale (VAS) was used to assess the pain level of these patients. The pain level was graded in to following types: VAS score 2-4= Mild pain, VAS score 4-6= Moderate pain and VAS score 6 and above = Severe pain.

Table 1: Pain status of the patients in visual analogue scale (VAS)

	Post tonsillectomy pain					
	At 6 hours		At 24 hours		At 12 hours	
Pain	**Dexa**	**Control**	**Dexa**	**Control**	**Dexa**	**Control**
Severe	22	36	12	28	5	16
Moderate	19	10	14	12	7	14
Mild	9	4	24	10	38	20
Total	**50**	**50**	**50**	**50**	**50**	**50**

Dexa= Dexamethasone.

Result of Chi-Square between Dexamethasone and Control group at 6 hours was 8.095 (p= 0.017), at 12 hours was 12.319 (p=0.002) and at 24 hours was 13.681(p= 0.001). Postoperative pain in between Dexamethasone and Control groups was significantly different. The intensity of pain in VAS was lower in Dexamethasone group.

Table 2: Mean±SD of pain score in VAS and its comparison between the groups.

	Post tonsillectomy pain (Mean ± SD) in VAS		
Group	**At 6 hours**	**At 12 hours**	**At 24 hours**
Dexamethasone	5.04 ± 1.09	3.94 ± 0.93	2.64 ± 0.69
Control	5.96 ± 1.03	5.14 ± 0.61	3.84 ± 0.87
***p* value**	<0.0001	<0.0001	<0.0001

The 't' test results at 6, 12, 24 hours were considered to be statistically significant. These two groups were significantly different.

Table 3: Use of additional analgesic, comparison between two groups.

Additional analgesic	**At 6 hours**		**At 24 hours**		**At 12 hours**	
	Dexa	**Control**	**Dexa**	**Control**	**Dexa**	**Control**
Required	11	23	6	17	2	10
Not required	39	27	44	33	48	40
Total	**50**	**50**	**50**	**50**	**50**	**50**

Dexa= Dexamethasone

- At 6 Hour Chi-Square test = 5.392 with 1 degrees of freedom. (p = 0.020)
- At 12 Hour Chi-Square test = 5.647 with 1 degrees of freedom. (p = 0.017)
- At 24 Hour Fisher Exact Test= The proportion of observations in the different categories which define the contingency table is significantly different than is expected from random occurrence (p = 0.028).

Post-operative use of additional analgesic in patients, between Dexamethasone and Control groups was significantly different.

Table 4: Comparison of fever between two groups

Fever	**Dexamethasone group**	**Control group**	**Total**
Fever present	06 (12%)	23 (46%)	29
Fever absent	44 (88%)	27 (54%)	71
Total	**50**	**50**	**100**

Chi-square= 12.433 with 1 degrees of freedom (p = <0.001). The fever in Dexamethasone and Control group was significantly different.

Table 5: Comparison of Post-Operative Nausea and Vomiting (PONV) in between two groups.

PONV	**Dexamethasone group**	**Control group**	**Total**
Present	08 (16%)	22 (44%)	27
Absent	42 (84%)	28 (56%)	73
Total	**50**	**50**	**100**

Chi-square = 8.04 with 1 degrees of freedom (p = 0.005). Post-Operative Nausea and Vomiting (PONV) in both Dexamethasone and Control group was significantly different.

Table 6: Comparison of oral intake of semisolid food in between two groups

Oral intake	**Dexamethasone group**	**Control group**	**Total**
Excellent	15	04	19
Good	28	38	66
Poor	07	08	15
Total	**50**	**50**	**100**

Chi-square= 7.950 with 2 degrees of freedom (p=0.019). Oral intake of semisolid food in Dexamethasone and Control group was significantly different.

After 24 hours, post-operative condition of the tonsillar fossa and reactionary haemorrhage were compared in between the groups. However, they were not significantly different.

Discussion

Tonsillectomy is the most common surgery that performed by an otolaryngologist. Despite advances in surgical and anaesthetic techniques, postoperative pain remains a significant problem after tonsillectomy. The pain inhibits chewing and swallowing, which leads to dehydration and contributes to lassitude and delayed recovery of strength and well-being. Oropharyngeal pain and irritation of gastric mucosa by swallowed blood are two main contributors towards high incidence of PONV (Post-Operative Nausea and Vomiting) following tonsillectomy.[5] Corticosteroids act as an antiemetic, but the exact mechanism of which is not known. Two studies have proved relationship between low levels of cortisol (endogenous or exogenously administered) and nausea, vomiting.[15,16] In a meta-analysis of 17 trials involving use of Dexamethasone for prevention of PONV in surgical patients. They concluded that when there is a high risk of PONV, a single prophylactic dose of Dexamethasone is antiemetic compared with Control, without evidence of any clinically relevant toxicity in otherwise healthy patients.[5]

The main aim of the study was to find out the effect of Dexamethasone on pain following tonsillectomy. Dexamethasone is highly potent and has long half-life (36-72 hours) for glucocorticoid activity, so that the effect would remain even after the discharge of the patient. Single IV dose was used, as it is devoid of side effects like gastritis, adrenal suppression etc.[17] In a study conducted by Splinter and Roberts have used 0.15 mg/kg Dexamethasone with good results.[18] So, in this study the selected dose of Dexamethasone was 0.15 mg/kg. Intravenous Dexamethasone was given just before the surgery to achieve peak effect in the early postoperative period. Both anesthetic drugs and surgical techniques were standardized in both groups. Usually Paracetamol was used as a first line analgesic, when the VAS score become more than 4 then the patients get this medicine. Before administering additional analgesic, a time period of 15 minutes was allowed to see if patient responded to tender loving care or pain subsided. Some patients need additional analgesic like Diclofenac sodium.

For the post-operative pain, Visual Analogue Scale (VAS) scores were assessed in both Dexamethasone and Control group at 6 hours, 12 hours and 24 hours. In both groups, post-operative pain in VAS (Mean±SD) at 6 hours was 5.04 ± 1.09 vs 5.96±1.03 (p= <0.0001), at 12 hours was 3.94± 0.93 vs 5.14 ± 0.61 (p=< 0.0001) and at 24 hours was 2.64± 0.69 vs 3.84±0.87 (p=<0.0001). Throughout the postoperative period VAS score was significantly lower in Dexamethasone group. With increasing time after surgery, the VAS scores difference between the two groups increased. Majority of Dexamethasone treated patients were pain free in 6-24 hours. This indicates prolonged analgesic effect of Dexamethasone. Number of patients requiring additional analgesic and the doses needed was less with Dexamethasone group than that of Control group.

Post-operative fever was remarkably less in Dexamethasone group (p= <0.001). In Control group, it is about 46% and Dexamethasone group 12%. A study conducted by Dhiwakar M. et al, showed that the fever was found in 54% of the post-tonsillectomy patient.[19]But, no data was available about post-tonsillectomy fever and its relation with Dexamethasone, though it was theoretically possible of less fever in Dexamethasone group.

PONV (Post-Operative Nausea and Vomiting) was significantly lower in Dexamethasone group (p= 0.005). Overall incidence of PONV (Post-Operative Nausea and Vomiting) in this Control group (44%) was comparable with previous studies (40-70%).[5] The incidence of PONV (Post-Operative Nausea and Vomiting) in Dexamethasone group (16%) was almost same in comparison to previous studies (17%).[20]

Oral intake of semisolid food was significantly better in Dexamethasone group (p=0.019). Perhaps by decreasing pain and inflammation, Dexamethasone improved the oral intake. In a meta-analysis Steward et al, showed that the children receiving Dexamethasone were more likely to advance to a soft or solid diet on post-tonsillectomy day one.[4] Condition of the tonsillar fossa and reactionary haemorrhage were compared in Dexamethasone and Control groups. However, there were no significance difference in between the groups. Complications from use of corticosteroids for less than 24 hours, however, are virtually non-existent, even in the presence of viral or bacterial infection.[6] Short courses of corticosteroids can be stopped abruptly and do not require a tapering regimen. Including the present study, no complications related to steroid use were reported in any of the studies examining the effect of preoperative steroids in tonsillectomy.[18,20,21]

Conclusion

The result comes out in this study is statistically significant in most of its variables. Pre-operative single dose of Dexamethasone is simple and easy procedure that can be practicable by all ENT surgeons. As the Dexamethasone appears to have positive influence on postoperative pain, provided good and prolonged analgesia. Side by side, reduce the requirement of additional analgesic and reduce the chance of analgesic induced side effect. Dexamethasone also reduces the incidence of post-operative fever, nausea and vomiting. As Dexamethasone reduces pain, fever, nausea and vomiting in post-tonsillectomy patient, it will help in earlier and better quality of oral intake. In conclusion, a single IV dose of 0.15 mg/kg Dexamethasone, given just before the starting surgery, provided good and prolonged analgesia, reduced fever, nausea and vomiting. Then enhance the earlier and better quality of oral intake without side effects.

Conflict of interest: No

Reference

1. Shapiro N. Tonsillectomy myths. AAO-HNS Bulletin. 2005; 24:12-13.
2. Randall D, Hoffer M. Complications of tonsillectomy and adenoidectomy. Otolaryngol Head Neck Surg. 1998; 118:61-68.
3. Panthila R, Viraporn A, Thida U, et al. Effect of Dexamethasone on Postoperative Pain after Adult. Thai J Anesthesiology. 2008; 34:1-8.
4. Steward D, Welge J, Myer C. Do steroids reduce morbidity of tonsillectomy? Meta-analysis of randomized trials. Laryngoscope. 2001; 111: 1712-1718.
5. Henzi I, Walder B, Tramèr MR. Dexamethasone for the prevention of postoperative nausea and vomiting: a quantitative systematic review. Anesth Analg. 2000; 90:186-194.
6. Schimmer BP and Parker KL. Adrenocorticotrophic hormone; adrenocortical steriods and their synthetic analogs: inhibitors of the synthesis and actions of adrenocortical hormones. In: Hardman JG, Limbird LE, Molinoff PB, Ruddon RW and Gilman AG, editors. Goodman and Gilman's The Pharmacological basis of therapeutics. 9th ed. Toronto: McGraw-Hill. 1996; 1459-1485.
7. Boissel J, Blanchard J, Panak E, et al. Considerations for the metaanalysis of randomized clinical trials: summary of a panel discussion. Control Clin Trials. 1989; 10:254-281.
8. Sacks H, Berrier J, Reitman D, et al. Meta-analyses of randomized controlled trials. N Engl J Med. 1987; 316:450-455.
9. April MM, Callan ND, Nowak DM, Hausdorff MA. The effect of intravenous dexamethasone in pediatric adenotonsillectomy. Arch Otolaryngol Head Neck Surg. 1996;122(2):117-120.
10. Tom LW, Templeton JJ, Thompson ME, Marsh RR. Dexamethasone in adenotonsillectomy. Int J Pediatr Otorhinolaryngol. 1996;37(2):115-120.
11. Pappas AL, Sukhani R, Hotaling AJ, et al. The effect of preoperative dexamethasone on the immediate and delayed postoperative morbidity in children undergoing adenotonsillectomy. Anesth Analg. 1998;87(1):57-61.
12. Hanasono MM, Lalakea ML, Mikulec AA, Shepard KG, Wellis V, Messner AH. Perioperative steroids in tonsillectomy using electrocautery and sharp dissection techniques. Arch Otolaryngol Head Neck Surg. 2004;130(8): 917-921.
13. Christoph C, Nadia E, Christopher L, et al. Dexamethasone and risk of nausea and vomiting and postoperative bleeding after tonsillectomy in children. JAMA. 2008; 300:2621-2630.
14. D. Gould et al. Visual Analogue Scale (VAS). Journal of Clinical Nursing. 2001; 10: 697-706.
15. Fredrikson M, Hursti T, Furst CJ, Steineck G, Borjeson S, Wikblom M et al. Nausea in cancer chemotherapy is inversely related to urinary cortisol excretion. Br J Cancer. 1992; 65:779-780.
16. Hursti TJ, Fredrikson M, Steineck G, Borjeson S, Furst CJ, Peterson C. Endogenous cortisol exerts antiemetic effect similar to that of exogenous corticosteroids. Br J Cancer. 1993; 68:112-114.
17. Melby JC. Drug Spotlight Program: Systemic corticosteroid therapy- pharmacology and endocrinologic considerations. Ann Intern Med. 1974; 81: 505-512.
18. Splinter WM. Roberts DJ. Dexamethasone decreases vomiting by children after tonsillectomy. Anaes Analg. 1996;83: 913-916.
19. Dhiwakar M, Eng CY, Selvaraj S, McKerrow WS. Antibiotics to Improve Recovery Following Tonsillectomy: A Systemic Review. Otolaryngology-Head and Neck Surgery (2006) 134, 357-364.
20. Malde AD, Sonawane VS, Jagtap SR. Effect of dexamethasone on post-tonsillectomy morbidities. Indian J. Anaesth. 2005; 49 (3): 202-207.
21. Khani A, Jaafarpour M, Khajavikhan J, Dyrekvandmogadam A. The effect of dexamethasone on morbidity related to vomiting, pain and oral intake in children after tonsillectomy. Journal of Clinical and Diagnostic Research. 2009 Aug ;(3)1641-1646.

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Original Article

Injury Pattern of Victims in Fatal Motor Bike Accident

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Abstract

Background: Road traffic accidents (RTA) are a major health problem worldwide, responsible for significant morbidity and mortality. The increase in the number of motorcycles circulating over the years is a consequence of the low cost, ease of locomotion and fuel efficiency of this vehicle.

Objective: To find out the injury pattern of victims in fatal motor bike accident in addition to socio-demographic characteristics of victims.

Methods and Materials: The cross-sectional study was conducted Sylhet M. A. G. Osmani Medical College Hopital, in Sylhet. Data were collected from the emergency unit and hospital ward registers. Information from the different unit & ward registers was entered into a standardized questionnaire. Data were collected by the study principal investigator, during study period from January 2021 to June 2021.

Results: More than two third (68.0%) victims belonged to age group 21-40 years. Male was found 49(98.0%) and 40(80.0%) were employer. Half (50.0%) of the victims were driver followed by 16(32.0%) were passenger and 9(18.0%) were pedestrian. Every victim has been injured in different parts of the body at the same time. Out of 50 patients, head injuries accounted for 49 (98 percent), thorax (chest) injuries for 44 (88%), abdominal injuries for 100%, and limb injuries for 27 (54%). Majority 47(94.0%) victims were alive and 3(6.0%) were death. In died patients, 1(33.3%) had severe haemorrhage and 2(66.7%) had craniocerebral injury.

Conclusion: Male was predominating, half of the victims were driver, and more common type of injury was Head, Thorax (chest), Abdomen and Limbs. Mortality rate was found 6% and most of them were craniocerebral injury.

Keywords: Road Traffic Accidents, hemorrhage, cranio-cerebral injury.

Introduction

Road traffic accidents (RTA) are a major health problem worldwide, responsible for significant morbidity and mortality.[1,2] According to the recent Global Status Report on Road Safety (2018), RTA are the current leading cause of death for children and young adults (5-29 years) and the eighth for all age groups.[1]

The World Health Organization estimates that RTA will become the third leading cause of disability in the world by 2030.[3] Therefore, the Sustainable Development Goal (SDG) target 3.6 has called for initiatives to halve the number of global deaths and injuries from RTA by 2020.[4]

Two-wheeled vehicles are increasing in number across the world especially in developing countries because compared to other vehicles, motorcycles are relatively cheap to own and operate.[5]

The increase in the number of motorcycles circulating over the years is a consequence of the low cost, ease of locomotion and fuel efficiency of this vehicle. However, this increase has grown proportionally to the number of traffic accidents, making them a major problem for public health worldwide, since they have been considered one of the main causes of morbidity and mortality in the world.[6]

The poor state of the roads in the country and the inefficiency of the public transportation system, as well as worsening vehicular congestion and increasing unemployment, are major reasons for the thriving motorcycle transport industry.[7]

Factors such as helmet wearing, use of alcohol and other

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drugs, inexperience of riders and poor driver training, conspicuity of the motorcycle and rider, issues of licensure and ownership, riding speed, and risk taking behaviour of riders have been identified as contributory factors to the increased risk of fatal motorcycle crashes.[8]

Motorcycle accidents are a major cause of RTIs and deaths. Almost half of individuals killed in road traffic accidents (RTAs) are motorcycle users.[9] The problem is more pronounced in developing countries owing to many factors such as rapid motorization, using motorcycles for commercial transport, and failure of motorcyclists to wear protective helmets; the burden of motorcycle accidents is aggravated by the habit of reckless driving with tendency to over speed by some motorcycle riders, as well as a significant number of drivers lacking proper certification and valid licensing. Poor traffic regulations and law enforcement and the abuse of recreational drugs and alcohol are also major contributing factors to motorcycle accidents.[10,11] The present study aimed to measure the magnitude of motor bike accident in the Bangladesh community and to determine the common patterns of major injuries after these accidents in patients attending the Sylhet M.A.G Osmani Medical College Hospital. The study also suggested possible counter measures and solutions to reduce the incidence and mortality of these accidents.

Materials & Methods

The cross-sectional study was conducted Sylhet M. A. G. Osmani Medical College Hospital, Sylhet. Data were collected from the emergency unit and hospital ward registers. Information from the different registers was entered into a standardized questionnaire. Data were collected by the study principal investigator, during study period from January 2021 to June 2021. Finally the size of the sample was 50 in number for investigation and selected purposively. Socio-demographic characteristics captured included age group, sex, occupation, and type of road user (pedestrian, car passenger, car driver, or motorcyclist (drivers) and motorcycle passengers). Clinical characteristics captured from medical record included type of injury (not injured, traumatic brain injury, wound, fracture, and two or more injuries), Glasgow Coma Score (classified as normal 15, mild coma 14–10, heavy coma, 9–7, or deep coma (6–3), and death (yes or no). Type of RTA was designated as involving a motorcycle or others (vehicle or bicycle). All deaths due to motorcycle accidents, being coroner's cases are usually accompanied by a duly signed order from a Coroner, requesting for an autopsy to be performed on the body. The coroner's papers contain information such as the name of the deceased (if known), sex, age, and residential address, place where the body was found and a report of the accident as documented in the extract from the police diary. The records of all deaths resulting from motorcycle accidents were extracted from the autopsy registers; further information was recovered from autopsy reports, hospital case notes, where applicable, extract from police diary. The data retrieved were analysed using the IBM Statistical Package for Social Sciences (SPSS) version 23, and the results were presented in percentages, tables, pie charts and bar chart.

Results

Table 1: Socio-demographic characteristics of the victims (n=50)

Variable	Frequency	Percentage
Age (years)		
≤20	5	10.0
21-40	34	68.0
>40	11	22.0
Gender		
Male	49	98.0
Female	1	2.0
Occupational status		
Student	4	8.0
Employer	40	80.0
Unemployed	6	12.0

Table 1 shows majority victims (68.0%) were in age group 21-40 years, male (98.0%) and employer (80.0%).

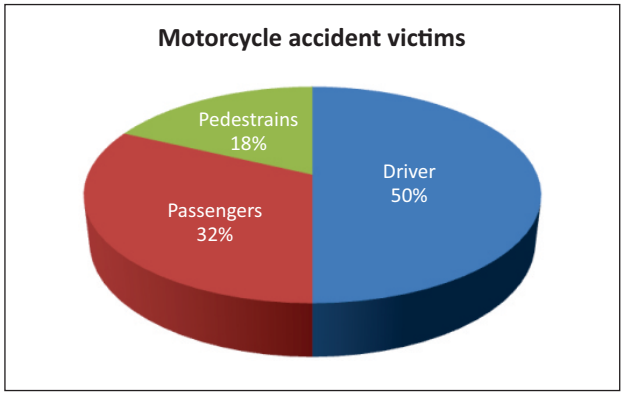

Figure 1: Pie diagram showing distribution of victims by types (n=50)

Table 2: Type of injury of the victims

Variable	Frequency	Percentage
Head		
Abrasion/laceration	15	30.0
Skull fracture (vault)	9	18.0
Skull fracture (base)	7	14.0
Intracranial haemorrhage	10	20.0
Brain injuries (contusion and laceration)	8	16.0
Thorax (chest)		
Abrasion/laceration	15	30.0
Lung laceration	10	20.0
Heart laceration	3	6.0
Vascular (aorta and jugular)	1	2.0
Rib fracture	15	30.0
Abdomen		
Abrasion/laceration	19	38.0
Liver laceration	13	26.0
Splenic laceration	10	20.0
Kidney laceration	3	6.0
Bowel perforation	5	10.0
Limbs		
Facture pelvic	10	20.0
Facture femur	7	14.0
Facture radius	5	10.0
Facture tibia	4	8.0
Facture ankle joint	1	2.0

Every victim has been injured in different parts of the body at the same time. Out of 50 patients, head injuries accounted for 49 (98 percent), thorax (chest) injuries for 44 (88 percent), abdominal injuries for 100 percent, and limb injuries for 27 (54 percent). The most prevalent type of injury encountered in the head was a skull fracture (32.0 percent), which included vault (18.0 percent) and base of the skull fractures (14.0 percent). Abrasions and lacerations were discovered in 15 (30.0%) of the thorax and 19 (38.0%) of the abdomen. In limbs, ten (20.0 percent) were discovered to have pelvic fractures.

Table 3: Outcome the victims (n=50)

Variable	Frequency	Percentage
Death	3	6.0
Alive	47	94.0

Table 3 shows that majority 47(94.0%) victims were alive and 3(6.0%) were death.

Table 4: Cause of death the victims (n=3)

Variable	Frequency	Percentage
Severe haemorrhage	1	33.3
Craniocerebral injury	2	66.7

In died patients, 1(33.3%) had severe haemorrhage and 2 (66.7%) had craniocerebral injury.

Discussion

In this study showed that more than two third (68.0%) victims belonged to age group 21-40 years. Male was found 49(98.0%) and 40(80.0%) were employer. Faduyile et al.[12] reported the peak age of victims was 31-40 years (29.6%).There were 122 (86%) males and 20 (14%) females with Male: Female ratio of 6:1. An overwhelming male preponderance of between 87.9% and 90.8% were reported in studies in other parts of the world.[13,14] Delamou et al.[15] also compared to victims of other RTA, victims of motorcycle RTA were more likely to be male (73.5% vs. 63.6%) and youth (53.4% vs. 41.3% children or youth; median age 23 years (IQR 17-33). Occupational distribution differed significantly by the RTA type: the commonest occupational groups involved in motorcycle RTA were students (29.7%), employees (23.6%), and farmers/housewives (23.3%), while employees (24.9%) and farmers/housewives (22.2%) represented the most affected group for other types of RTA. Fouda et al.[16] reported there were 181 (90.5%) males with a mean age of 30.7±10.5 years (range, 7-65years). Sharma et al. [17]observed young adults of the age-group 21-25 years constituted the majority of the victims, 48 (36%) and the 16-30 year age group accounted for 98 (73%) motorized two wheeler deaths. The overall male: female ratio was 1.6:1.

In this study showed that half (50.0%) of the victims were driver followed by 16(32.0%) were passenger and 9(18.0%) were pedestrian. Faduyile et al.[12] reported majority of the victims 67 (47.2%) were Motorcycle riders, followed by pillion passengers who accounted for 48 (33.8%), while the remaining 27 (19.0%) of the victims were pedestrians. Delamou et al.[15] regarding the type of road users, motorcyclists were predominant (50.7%) among motorcycle RTA victims, whereas car passengers represented the majority (89.3%) of victims of other types of RTA.

In current study showed that every victim has been injured in different parts of the body at the same time. Out of 50 patients, head injuries accounted for 49 (98 percent), thorax (chest) injuries for 44 (88 percent), abdominal injuries for 100 percent, and limb injuries for 27 (54 percent). The most prevalent type of injury encountered in the head was a skull fracture (32.0 percent), which included vault (18.0 percent) and base of the skull fractures (14.0 percent). Abrasions and lacerations were discovered in 15 (30.0%) of the thorax and 19 (38.0%) of the abdomen. In limbs, ten (20.0 percent) were discovered to have pelvic fractures. Faduyile et al.[12] reported analysis of head injuries shows that skull fracture was the commonest form of injury seen (32.7%), which comprises fracture of the vault (19.5%) and fracture of the base of the skull (13.2%). This was followed by abrasions and lacerations to the scalp and face, which accounted for 30.5%. This finding is similar to that of Nwadiaro et al.[18] who reported that head injury constituted 40.1% of the injuries in a clinical-based study in Jos.[18] Studies in Ghana by Kudebong et al.[19] and Uganda by Kigera and Naddumba also showed that head injury was

the commonest type of injury, accounting for 32.2% and 20.0% respectively.[19,20] The smaller figure reported in Ghana and Uganda when compared to this study may be due to a higher level of helmet use in those countries. Heydari et al. in a study in the Fars province in Iran observed that the head was the most frequently injured site (87.8%).[21] They opined that helmet use among motorcycle occupants was very low in that area and that may readily explain this high level of head injury seen in that study. Delamou et al.[15] the number of victims of motorcycle RTA who sustained two or more injuries was about four times higher than that of other RTA (5,091 vs. 1,461). Fouda et al.[16] The most common pattern of injury in polytraumatized patients was combined head and orthopedic injuries in 30.4% of patients. Isolated head injuries were observed in 34 (56%), patients and combined head injuries (skull fracture with one or more types of hemorrhage) were seen in 27 (44.3%) patients.

Current study showed abrasions and lacerations was found 15(30.0%) in thorax. Faduyile et al. reported the most common thoracic injury in this study was rib fracture (46.7%). This is close to 40.3% reported by Sharma in Northern India [13], and 45.9% reported by Kraus et al. in the US.[22]Fouda et al.[16] also observed 18(9%) patients had variable chest injuries, combined chest injuries were the most common pattern occurring in a third; 12 (67%) patients with chest injuries were managed conservatively and three of them died, whereas six (33%) patients required insertion of intercostal tube and only one of them died. There was no significant relationship between final outcome and the different types of chest injuries.

In this study abrasions and lacerations was found 19(38.0%) in abdomen. Faduyile et al. [12]reported the pattern of abdominal injuries in this study showed that lacerations to the liver were the most frequent visceral injury (16.7%), followed by injuries to the spleen and kidney which accounted for 13.6% and 7.6% respectively. Bowel perforation constituted only 10.6%. This general pattern is similar to the observation by Sharma in India, who reported the following pattern of abdominal injuries; liver laceration (27.6%), splenic laceration (20.1%), kidney rupture (10.4%) and intestinal perforation (4.5%).[13] Similarly, Kraus et al. in the U.S reported that liver laceration was the commonest type of intra-abdominal injury in a fatal motorcycle accident, representing 31.8%.[22] Fouda et al.[16] reported Internal hemorrhage was diagnosed in 17 (9.5%) patients by FAST; 11 (65%) of these patients were admitted to the ward, and four patients to ICU.

In present study showed 10 (20.0%) was found facture pelvic in limbs. This is very similar to the finding by Heydari et al. which was 9.8%.[21]This study showed that fracture of the humerus accounted for 8.3%, a figure very close to 9.7% reported by Sharma et al. in India. [13] Lower extremity injuries accounted for 18.2% of the injuries seen in this study, comparable with 14.8% reported by Heydariet al.[21] On the other hand, Solagberu et al. reported that lower extremity was the most frequently injured part of the body in motorcycle accidents, representing 70.5%.[23]

In this study showed that majority 47(94.0%) victims were alive and 3(6.0%) were death. Sharman et al. in India opined that motorcycle accident deaths among females were mainly due to the act that nearly all female pillion riders sit sideways with both legs to the left of the vehicle because the common mode of dress, the sari, prevents them from sitting astride and they do not wear helmets.[13]Delamou et al.[15] reported overall, 4.4% of RTA victims died; mortality was 1.1%amongst motorcycle RTA victims compared with 3.3%amongst other RTA victims ($P < 0.001$).Fouda et al.[16] reported 82.5% of the studied patients were admitted to the ward and 13% required ICU admission, with an overall mortality rate of 4.5%.

In died patients, 1(33.3%) had severe haemorrhage and 2(66.7%) had craniocerebral injury. Faduyileet al.[12] reported majority of the victims, 72 (50.7%) died of Craniocerebral injuries. This is consistent with the findings in some studies from within Nigeria and from other parts of the world with frequency ranging from 33.3% to 87.8%.[21,24] Nzegwu et al.[25] in Benin City observed that none of the dead victims in their study wore a crash helmet at the time of the accident.

Conclusion

Male was predominating, half of the victims were driver, and more common type of injury was Head, Thorax (chest), Abdomen and Limbs. The fact that majority of victims die of head injuries also signals the need for more research efforts geared towards head protection for motorcycle riders and passengers. Mortality rate was found 6% and most of them were craniocerebral injury.

Reference

1. WHO World Health Organization, *Global Status Report on Road Safety Summary 2018*, WHO World Health Organization, Geneva, Switzerland,, 2018, http://apps.who.int/bookorders.
2. WHO World Health Organization, Road Traffic Injuries: Key Facts, https://www.who.int/ violence_ injury_prevention/road_traffic/en/? fbclid=I wAR26ikgm8o OCO2mwdAkqigLGfSxPlF6376_ Qv4wr-WBNSVCdu2PG-lcNJ3I, p. 6, WHO World Health Organization, Geneva, Switzerland, 2015
3. C. D. Mathers and D. Loncar, "Projections of global mortality and burden of disease from 2002 to 2030," PLoS Medicine, vol. 3, no. 11, p. e442, 2006.
4. WHO World Health Organization, Fact sheets on sustainable development goals: health targets, Road Safety. In: Urban Transportation and Logistics: Health, Safety, and Security Concerns, WHO World Health Organization, Geneva, Switzerland, 2013, http://www.euro.who.int/__data/assets/pdf_file/ 0003/351444/3.6-Fact-sheet-SDG-Road-safety-FINAL-10- 10-2017.pdf?ua=1.

5. Aderamo AJ, Olatujoye S. Trends in motorcycle accidents in Lokoja, Nigeria. EurInt J Sci Technol. 2013; 2: 251-261.
6. Bittar CK, Cliquet A Jr, Costa VSDA, Pacheco ACF, Ricci RL. Socioeconomic impact of motorcycle accident victims in the emergency room of a hospital (Part 2). ActaOrtop Bras. [online]. 2020;28(3):149-51.
7. Olubomehin OO. The development and impact of motorcycles as means of transportation in Nigeria. Res Humanities and Soc Sci. 2012; 2: 231-239.
8. Lin LR, Kraus JF. A review of risk factors and pattern of motorcycle injuries. Accid Anal Prev. 2009; 41: 710-722.
9. Vafaee-Najar A, Esmaeili H, Ibrahimipour H, et al. Motorcycle fatal accidents in Khorasan Razavi Province, Iran. Iran J Public Health 2010; 39: 95–101.
10. Museru LM, Mcharo CN and Leshabari MT. Road traffic accidents in Tanzania: a 10-year epidemiological appraisal. East Central Afr J Surg 2002; 7: 23-26.
11. Galukande M, Jombwe J, Fualal J, et al. Boda–boda injures a health problem and the burden of disease in Uganda: a tertiary hospital survey. East Central Afr J Surg 2009; 14: 33-37.
12. Faduyile F, Emiogun F, Soyemi S, Oyewole O, Okeke U, Williams O. Pattern of injuries in fatal motorcycle accidents seen in Lagos State University teaching hospital: an autopsy-based study. Open access Macedonian journal of medical sciences. 2017;5(2): 112.
13. Sharma BR, Gupta N, Sharma AK, Sharma S. Pattern of fatal motorised two-wheeler crash injuries in Northern India: is safety helmet adequate prevention? Trends in Med Res. 2007; 2: 27-36.
14. Carasco CE, Godinho M, Barros MB, Rizoli S, Fraga GP. Fatal motorcycle crashes: a serious public health problem in Brazil. World J Emerg Surg. 2012; 7:S5
15. Delamou A, Kourouma K, Camara BS, Kolie D, Grovogui FM, El Ayadi AM, Ade S, Harries AD. Motorcycle accidents and their outcomes amongst victims admitted to health facilities in Guinea: a cross-sectional study. Advances in preventive medicine. 2020;2020:1-7.
16. Fouda EY, Youssef M, Emile SH, Elfeki H, Thabet W, Abdallah E, Elshobaky A, Toma MS, Khafagy W. Pattern of major injuries after motorcycle accidents in Egypt: The Mansoura Emergency Hospital experience. Trauma. 2017;19(1):39-45.
17. Sharma BR, Gupta N, Sharma AK, Sharma S. Pattern of fatal motorized two-wheeler crash injuries in Northern India: Is safety helmet adequate prevention. Trends in medical research. 2007;2(1):27-36.
18. Nwadiaro HC, Ekwe K.K, Akpayak IC, Shitta H. Motorcycle injuries in north central Nigeria. Niger J ClinPract.2011; 14: 186-189.
19. Kudebong M, Wurapa F, Aikins M. Economic burden of motorcycle accidents in northern Ghana. Ghana Med J. 2011; 45: 135-142.
20. Kigera JNM and Naddumba EK. Patterns of injuries after road traffic crashes involving Bodabodas.AnnAfr Surg. 2010; 5:12-15.
21. Heydari ST, Maharlouei N, Foroutan A, Sarikhani Y, Ghaffarpasand F, Hedjazi S et al. Fatal motorcycle accidents in Fars province in Iran: a community based survey. Chin J Traumatol. 2012; 5: 222-227
22. Kraus JF, Peek-Asa C, Cryer HG. Incidence, severity and patterns of intra-thoracic and intra-abdominal injuries in motorcycle crashes. J Trauma. 2002; 52: 548-553 .
23. Solagberu BA, Ofoegbu CKP, Nasir AA, Ogundipe OK, Adekanye AO, Abdurahman LO. Motorcycle injuries in a developing country and the vulnerability of riders, passengers and pedestrians.Inj Prev. 2006; 12: 266-268.
24. Department for Transport. Reported Road Casualties Great Britain: 2010 Annual Report. Department for Transport, London, UK, 2011
25. Nzegwu MA, Aligbe JU, Banjo AAF, Akhiwu W, Nzegwu CO. Patterns of morbidity and mortality amongst motorcycle riders and their passengers in Benin City, Nigeria: one year review. Ann Afr Med. 2009; 7: 82-85

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Original Article

Evaluation of a Newly Developed Non-Culture Test for Bacteriological Diagnosis of Ventilator-Associated Pneumonia in RMCH

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Abstract

Background: The diagnosis and treatment of pneumonia in patients who are receiving mechanical ventilation remain a difficult challenge**.**

Objective: To investigate the utility of soluble triggering receptor expressed on myeloid cells1(s TREM-1) levels in Endotracheal aspirate (ETA) samples as early biomarkers for the diagnosis of VAP compared with quantitative culture result of ETA.

Methods: A total of eighty patients with clinically suspected VAP cases were included in this study and were selected purposely. ETA was collected and level Wes TREM-1are measured. On the other hand, VAP was diagnosed by the quantitative culture of ETA.

Results: The concentration of s TREM-1 in ETA did not discriminate VAP positive from VAP negative patients when compared to quantitative cultures of ETA as the gold standard. A cut-off value of 68.72pg/ml for human s TREM1 in ETA resulted in a sensitivity of 60.81% and specificity of 62.5%.

Conclusion: It may be concluded that the proposed ELISA kit cannot be used as a rapid diagnostic test for the immediate diagnosis of VAP.

Keywords: Ventilator-associated pneumonia (VAP), infection, Non-culture Test

Introduction

Ventilator-associated pneumonia (VAP) is the most common nosocomial infection in the ICU and contributes disproportionately to both poor outcomes and the high cost of care in critically ill patients.[1]

Ventilator-associated pneumonia (VAP) is defined as pneumonia that arises more than forty-eight hours after initiation of mechanical ventilation by tracheostomy or endotracheal intubation. It has emerged as an important challenge in ICU as it contributes to approximately half of all cases of hospital-acquired pneumonia.[2]

VAP is estimated to occur in 9-27% of all mechanically ventilated patients with the highest risk being early in the course of hospitalization and mortality rates in patients with VAP range from 20-50% and may reach more than 70% when the infection is caused by multidrug-resistant and invasive pathogens [3,4]

VAP is clinically suspected usually on the basis of the presence of fever or hypothermia, leukocytosis or leucopenia, purulent tracheal secretion and the presence of a new or persistent radiographic infiltrate. But these clinical parameters individually have limited diagnostic value. In several studies, the clinical pulmonary infection score (CPIS) was used as a diagnostic tool for pneumonia and calculated on the basis of points assigned for 6 clinical criteria including body temperatures, leukocyte count, volume and appearance of endotracheal aspirate, oxygenation chest X-ray, and culture and Gram staining of endotracheal aspirate.[5-7] Pugin *et al.,* found that a CPIS of >6 was associated with a high likelihood of pneumonia with 93% sensitivity and 100% specificity.[5] To establish an infection, clinical parameters should coexist with the culture of lower respiratory tract secretion. [8]

Culture of the lower respiratory tract secretions obtained by bronchoscopically such as bronchoalveolar lavage (BAL) or protected specimen brush(PSB) are essential for deciding the antibiotic susceptibility of the etiological agent.[7] But bronchoscopy is an invasive procedure which

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cannot be performed in all patients suspected to have VAP. Bronchoscopy maylead to cardiac arrhythmias, hypoxemia or bronchospasm. So it is usually performed only in the later stages of VAP.[9] But any delay in the administration of appropriate antibiotic therapy is associated with higher morbidity and mortality. So there is a need for a non-invasive technique which can be performed in patients suspected to have VAP.[10]

Endotracheal suction is performed in ventilated patients as part of routine care and for tracheal toileting. Endotracheal aspiration does not require any special expert or instrument for its collection. It is easy to collect and less time-consuming and less.[11] Examination of endotracheal aspirate by gram staining allows rapid insight into the number and types of bacteria as well as the number of polymorphonuclear neutrophils that are suggestive of inflammation and infection. The presence of bacteria on Gram-stained smear correlates with the culture of approximate 10^5 bacteria per ml of aspirate.[12]

Diagnosis of VAP is both contentious and frustrating for the intensivist. Early detection of VAP and its causative micro-organisms is a key challenge for clinicians whose aim is to establish a rapid and adapted antibiotic therapy. Identification of the causative microorganism relies on quantitative or semi-quantitative cultures of BAL or ETA with microbiological data provided 24-48hoursafter the sampling. This study will evaluate the effectiveness of using soluble triggering receptor expressed on myeloid cells (s TREM)-1, expressed in response to bacterial infection, as a tool for the rapid diagnosis of VAP. The triggering receptor expressed on myeloid cells is a member of the immunoglobulin superfamily. Its expression on phagocytes is upregulated by exposure to bacteria. A soluble form of TREM-1(s TREM-1) is proposed as a new biomarker that had been tested for acute infections with different diagnostic and prognostic value. S TREM-1 can be found in different body fluids, such as serum, broncho-alveolar lavage fluid (BALF), endotracheal aspirate (ETA), and exhaled breath condensate (EBC) where it can be assayed by ELISA using commercial immunoassay kits. Some clinical studies have proved that s TREM-1did has the ability to identify patients with sepsis while others come to an opposite conclusion.

The real effect of s TREM-1 on the diagnosis of VAP is still unknown and has not been well evaluated yet.

With the above view this study will measure s TREM-1 levels in different body fluid samples from patients who are clinically suspected for VAP and at the same time microbiological test of the sample for diagnosis of VAP and correlation between them will be done

Materials and Methods

This is a cross-sectional type of analytical study. The study was conducted from January to December; 2018. Clinically suspected VAP patients admitted in the Intensive Care Unit Department of Rajshahi Medical College Hospital were selected purposively as cases in this study. Moreover, endotracheal aspirate was collected from patients under mechanical ventilation by endotracheal tube or tracheostomy tube for more than 48 hours and was also purposive in nature. Exclusion criteria were patient's attendant who refused to give consent and patient without infection. After all aseptic precaution, a narrow soft sterile disposable suction catheter was introduced through the endotracheal tube or tracheostomy tube. A 10 cc disposable syringe was used for aspiration and 3 to 5ml aspirate was collected and then injected into a sterile test tube. The tube was then brought to the laboratory for further processing. Rapid diagnosis of clinically suspected VAP cases was done by measuring the s TREM-1 level in body fluid (tracheal aspirate) which it can be assayed by ELISA using commercial immunoassay kits.

Results

A total of 80 patients' endotracheal aspirates were collected from clinically suspected VAP (Ventilator-associated pneumonia) cases according to CPIS (Clinical Pulmonary Infection Score) system and were tested for isolation. Endotracheal aspirates were also tested for ELISA.

Table 1: Culture positivity of collected samples. n=80

Culture positivity	Single isolated cases (%)	Multiple isolated cases (%)	Total (%)
Culture positive	72 (90%)	3 (3.75%)	75 (93.75%)
Culture negative	-	-	5 (6.25%)
Total			80 (100%)

Table 1 shows culture positivity of collected samples. Among 80 cases 75 (93.75%) cases were culture positive and 5 (6.25%) cases were culture negative.Single and multiple isolated cases were 72 (90%) and 3 (3.75%) respectively among the culture positive cases.

Clinically suspected 80 VAP patient's endotracheal aspirates were tested for ELISA. For negative control another 5 patient's (non-VAP case) endotracheal aspirates were also tested for ELISA.

Figure1 shows that, the concentration of human s TREM-1 in VAP cases and non-VAP cases did not show any significant difference. The average concentration of human s TREM-1in non-VAP cases were 68.72 pg/ml (OD-0.105).

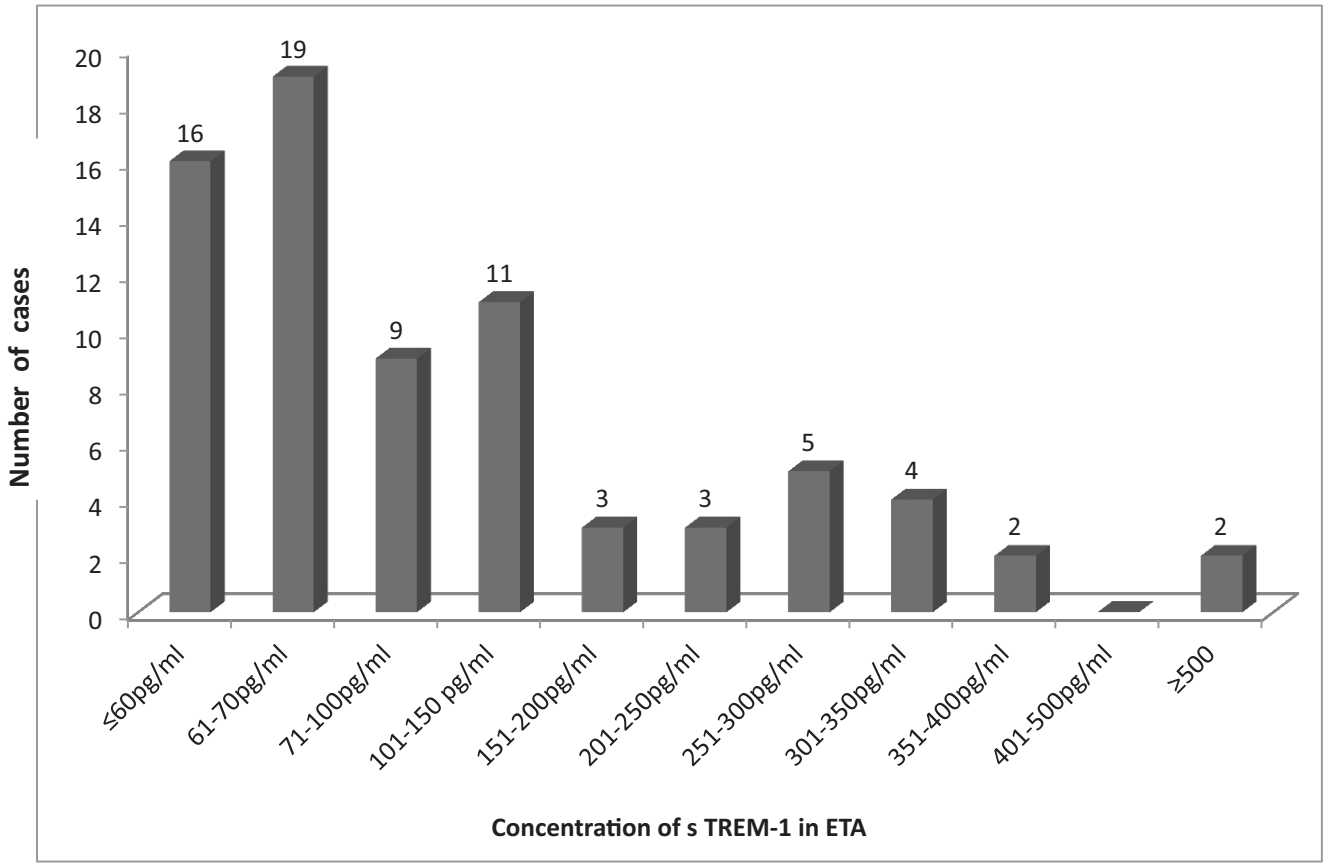

Figure- 1: Distribution of human s TREM-1 level in VAP cases. n=75

Table 2: Distribution of concentration of human s TREM-1 in VAP cases comparing with non-VAP cases.

Attributes	Above 68.72pg/ml	Average 68.72pg/m1	Below 68.72pg/ml	Total
VAP case	45 (60%)	(1.33%)	29 (38.67%)	75 (100%)
Non-VAP case	-	5	-	5 (100%)

A cut off value of 68.72 pg/ml for human s TREM-1 in ETA resulted in sensitivity of 60.81% and specificity of 62.5%.

Discussion

Ventilator associated pneumonia remains a major contributor to hospital-acquired infection in Asia. Early accurate diagnosis is fundamental in the management of patients with VAP.[1] Delayed diagnosis and subsequent delay in initiating appropriate therapy may be associated with worse outcomes in patients with VAP, on the other hand an incorrect diagnosis may lead to unnecessary treatment and subsequent complication related to therapy.[10]

In the present study soluble triggering receptor was measured which expressed on myeloid cell-1 in endotracheal aspirate by ELISA kit which is commercially available to see the diagnostic performance of s TREM-1.

It is evaluated that the concentration of s TREM-1 in Endotracheal aspirate (ETA) could not effectively categorize patients as VAP positive or VAP negative when using ETA culture samples as the comparison. This study found lower sensitivity 60.81% and specificity 62.5% and findings are consistent with some recent studies; a study Wang *et al.*, 2017 in china showed moderate sensitivity 85.5% and lower specificity 28.8%,[13] Palazzo *et al.*, 2012 in Washington also showed low sensitivity and specificity of s TREM-1 in ETA for VAP patient.[14] Dissimilarity was observed by Gibot*et al.*, 2004 in New England where they found high sensitivity 98% and specificity 90% but in that study they measures TREM-1 level in bronchoalveolar-lavage not ETA. Moreover that it could not differentiate between communitie acquired pneumonia from VAP.[15]

One of the goal of this study was to evaluate a rapid non-culture test that will be able to diagnose VAP effectively and will help the clinicians to start the treatment as early as possible so that ultimate reduction of the mortality rate of VAP patients can be obtained. It is decided that the proposed kit that measured the s TREM-1 in ETA needed a large scale evaluation to practice it as a rapid diagnostic procedure. Additionally, it would not be able to differentiate the micro-organism. It's expression on phagocytes is up regulated by exposure to any bacteria. As

a result, the microbiological diagnosis (culture) to know the exact causative organism and their drug sensitivity pattern to start accurate treatment of the VAP patient.

Conclusion

Ventilator associated pneumonia (VAP) is a significant cause of morbidity and mortality in critically unwell patients. In ELISA test, among five culture negative cases average concentration of human s TREM-1 was 68.72 pg/ml. But among 75 culture positive cases 45 cases showed higher concentration of human s TREM-1 in ETA (>68.72 pg/ml) and 29 cases showed lower concentration of human s TREM-1 (<68.72 pg/ml). The optimum cut-off value for s TREM-1 in ETA was 68.72 pg/ml, yielding sensitivity and specificity of 60.81% and 62.5%. Finally it may be concluded that the proposed ELISA kit need further evaluation to use it as a rapid diagnostic test for VAP.

Conflict of interest: No

References

1. Rea-Neto, A., Yousf, N.C.M., Tuche, F., Brunkhorst, F., Ranieri, V.M., Reinhart, K. and Sakr, Y. (April, 2008). Diagnosis of Ventilator associated Pneumonia : A systematic review off the literature. Available online: http:// ccforum.com/content/12/2/R56

02. Kalanuria, A.A., Zai, W. and Mirski, M. (2014). Ventilator associated Pneumonia in the ICU, BioMed Central, Critical Care; 18: 208. http:// Ceforum.com/ Content/18/2/208.

03. Selina, F.,Talha, K.A., Islam, A., Hasan, Z., Hyder, M. and S. Selvapandian. (2009). Organisms associated with ventilator associated pneumonia (VAP) in intensive care unit (ICU), Journal of the Bangladesh

04. Amanullah, S. and Mosenifar, Z. (Dec, 2015). Ventilator Associated Pneumonia Overview of Nosocomial Pneumonias. Online available https:// emedicine.medscape.com

05. Pugin, .J, Auckenthaler, R., Mili, N., Janssens, J.P., Lew, P.D. and Suter, P.M. (1991). Diagnosis of ventilator associated pneumonia by bacterilogical analysis of bronchoscopic lavage fluid. Am Rev RespirDis ; 143: 1121-1129.

06. Chastre J, Fagon JY. (2002).Ventilator associated pneumonia. AM J RespirCritCare Med; 165: 867-903.

07. Niederman, M.S. and Craven, D.E. (2005). Guidelines for the management of adults with hospital acquired ventilator associated pneumonia and health care associated pneumonia. AM J RespirCrit Care Med;171: 388-416.

08. Fartoukh, M., Maitre, B., Honone, S., Cerf, C., Zahan, J. and Brun-Buisson, C. (2003).Diagnosing Pneumonia during mechanical Ventilation: The clinical pulmonary infection score revisited.Am J RespirCrit Care Med; 168(2): 173-179

09. Joseph, N.M., Sistla, S., Dutta, T.K. and Badhe, A.S.(2010). Ventilator associated pneumonia in a tertiary care hospital in India: role of multidrug resistant pathogens. J infectdevCtries; 4(4): 218-225

10. Luna, C.M., Vujacich, P., Niederman, M.S., Vay, C., Gherardi, C., Matera, J. and Jolly, E.C. (1997). Impact of BAL data on the therapy and outcome of Ventilator associated Pneumonia. Chest; 676-685

11. EI-Ebiary, M., Torres, A., Gonzalez, J., Puig de la Bellacasa, J., Garcva, C. and Jiminez de Anta M.T. (1993). Quantitative cultures of endotracheal aspirates for the diagnosis of ventilator associated pneumonia. Am Revrespire dis.; 148: 1552-1557.

12. Nagendra et al.(2001). Sampling variability in the microbiological evaluation of expectorated sputum and endotracheal aspirates.J ClinMicrobiol; 39(6): 2344-2342.

13. Wang, K., Chen, Y., Salido, M.M., Kohli, G.S., Kong, J.L. and Liang, H.J. (2017). The rapid in vivo evolution of Pseudomonas aeruginosa in ventilator associated pneumonia patients leads to attenuated virulence. Open Biol.7: 170029.Online available at https://dx.doi.org/10.6084/m9.figshare.c.385829

14. Palazzo, S.J., Simpson, T.A., Simmons, J.M. and Schnapp, L.M. (Dec, 2012). Soluble Triggering Receptor Expressed on Myeloid Cells-1 (s TREM-1) as a Diagnostic Marker of Ventilator associated Pneumonia. Respir Care; 57(12): 2052-2058. doi: 104187/respcare.01703.

15. Gibot, S., Cravoisy, A., Levy, B., Bene, M., Faure, G. and Bollaert P. (2004) Soluble Triggering Receptor Expressed on Myeloid Cells and the diagnosis of Pneumonia.N. Engl J Med; 350(5): 451-458

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Original Article

Comparison between Two Methods of Preoperative Hair Removal with Surgical Site Infection

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Abstract

Background: Surgical site infections (SSI) are one of the major complications that develop in surgical patients and are the most common nosocomial infection in patients undergoing surgery, carrying significant morbidity and mortality rates. Several measures are taken to reduce surgical site infection. Removal of hair is also important for a surgery that helps the surgeon in operation and also after postoperative period during bandaging. Several methods of hair removal are present as razor shaving, depilatory method, clipping etc. Razor shaving is the most popular method of hair removal from operative site of preoperative patients in developing country like Bangladesh that can cause preoperative skin abrasion that may be source of skin infection and can induce surgical site infection.

Objective: To determine whether preoperative razor shaving or depilatory method of hair removal is preferred to reduce postoperative wound infection. In addition the study also considered variables under comparisons were age, sex, BMI & Hb%, condition of wound, type of operation, length of incision, duration of operation, pre-operative hospital stay as well as status of wound healing.

Method: This crosssectional comparative study was conducted on 100 patients of which in case of 50 patients' preoperative hair removal were done from operation site by razor shaving and in 50 patients' preoperative hair removal were done from operation site by depilatory method. Outcome variable was wound infection.

Results: Wound infection was compared of both groups of patients. Total 19% wound infections were detected. Among the group-I (who had razor shaving), 41 (82%) patients had satisfactory healing and among the Group-II (who had hair removal by depilatory method), 40 (80%) patients had satisfactory healing. There is no statistically significant difference of wound infection between the two groups of patients.

Conclusion: There is the same outcome of surgical site infection in case of preoperative hair removal by razor shaving and preoperative hair removal by depilatory method.

Keywords: Razor shaving, depilatory method, wound infection.

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Introduction

Postoperative wound infection may lead to significant morbidity, patient discomfort and increased cost of surgical care[1]. In the United Kingdom, it is estimated that postoperative wound infections cost the National Health Scheme about one billion pounds annually[2]. As part of the antiseptic steps taken to reduce postoperative wound infection, different methods of hair removal are employed when preparing patients for operations and many of these have been previously evaluated[3-5]. The most popular methods are the use of razor blade, clippers, and depilatory creams[6].

In many developing countries such as Bangladesh, the age-long practice of preoperative razor shaving is still popular. However, studies reviewing hair shaving, the commonest and most economical method of hair removal, have noted its association with a greater risk of wound infection.[3,7,8]

Furthermore, the psychological effect of hair removal on patients undergoing cranial surgeries has led to doubts about the necessity of hair removal.[4,9] These among other reasons make the practice of hair removal controversial today with both proponents and opponents.[3,7] Those who

advocate the practice of preoperative hair removal do so in the belief that presence of hairs can interfere with skin incisions and the subsequent closure as well as the application of adhesive drapes and wound dressings[10]. In Bangladesh, in many tertiary institutions, routine preoperative shaving to remove hair from the operative site and its surroundings, particularly when access would be through a hair-bearing area of the body, has been the practice. Patients for elective operations are usually shaved with a razor blade by nursing staff in the hospital on the morning of surgery. As razor shaving cause skin abrasion and greater risk of wound infection, this study is conducted to evaluate the relationship of preoperative razor shaving or depilatory method hair removal with postoperative wound infection. Outcome variable is wound infection.

Materials and Methods

This comparative cross-sectional study was conducted among 100 conveniently selected patients who have undergone elective operation in the Department of Surgery of Prime Medical College Hospital from 1st January 2017 to 31st December 2017. A sample of 50 in each study group (a total of 100 patients) was collected by considering 5% significance level, 9% precision level and considering the incidence of 10% wound infection in clean-contaminated opeeration[11].

Among the total respondents, preoperative hair removal were done from operation site by razor shaving in 50 patients who were renamed as Group-I and in Group-II, other 50 patients' preoperative hair removal were done from operation site by depilatory method.

Patient selection and preparation:

Patients of elective operations who fulfilled inclusion and exclusion criteria were given an arbitrary number. Each odd number of patient was included as group-I and even number of patient was included as group-II. All the patients were assessed before operation by history taking, physical examination and necessary investigations. Hb%, RBS, serum urea and creatinine were estimated of each patient to exclude anaemia, diabetes mellitus, uraemia respectively. Patients BMI were measured by measuring height and weight of the patient and calculating BMI.

Patients were searched for any focal source of sepsis. They have been informed about the purpose of data collection and written consent has been taken. They were asked to take a preoperative showering before the day of operation. In case of Group-I patients, shaving of the patient was done on the morning in the day of operation. In case of Group-II patients, hair removal from the operative site of the patient is done by depilatory method.Before operation hand scrubbing of the surgeon is done with aqua based povidone iodine. In all cases surgeon scrubbed his hand for 3 minutes. Skin preparation, draping and other aseptic procedures during operations were performed in both groups as standard method.

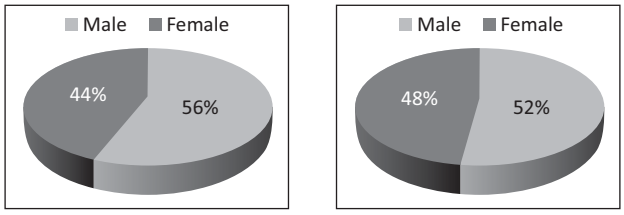
In all cases diathermy were used for haemostasis and drainage tube were inserted (if necessary) through a separate stab wound. If any discharge from the wound was present, it was collected and was sent for bacteriological examination and antibiotics were changed according to culture and sensitivity report. Adequate postoperative analgesia was ensured and patients were encouraged for early mobilization. Patients were followed up on 3rd to 7th postoperative day and regularly examined for surgical site infection on the basis of ASEPSIS score[12]. The potential for infection depends on a number of patient variables such as the state of hydration, nutrition and existing medical conditions as well as extrinsic factors, for example related to pre-, intra-, and postoperative care if the patient has undergone surgery. This often makes it difficult to predict which wounds will become infected. Consequently the prevention of wound infection should be a primary management objective for all healthcare practitioners.[13]

Results

Table 1: Distribution of both groups of patients by age

Age in years	Group- I (n= 50)		Group- II (n= 50)	
	Frequency	Mean (±SD)	Frequency	Mean (±SD)
17-30	18	21.3 (±10.1)	8	22.1 (±9.42)
31-40	16	36.4 (±6.22)	16	37.21 (±7.11)
41-50	14	43.5 (±8.41)	15	45.71 (±3.47)
51-50	2	52.1(±3.21)	11	54.21 (±4.25)
Total	50	35.56 (±10.88)	50	40.44(±10.66

The mean(±SD) age of group-I and group-II was 35.56(±10.88) and 40.44(±10.66) respectively.



Group-II Group-II

Figure 1: Pie diagram showing distribution of both group of patients by sex

Figure 1 shows that in the group-I, there were 28 (56%) male patients and 22 (44%) female patients. In the group-II, there were 26 (52%) male patients and 24 (50%) female.

Table 2: Distribution of both groups of patients by BMI and hemoglobin level

Parameters	**Group- I (n=50)** Mean (±SD)	**Group- II (n=50)** Mean (±SD)	**p value**
BMI (kg/m²)	19.72 (±1.73)	19.67 (±1.06)	> 0.05 [t = 0.2043,
Hb% (g/dl)	11.9 (±1.8)	12.1 (±1.5)	> 0.05 [t = 0.0785

Table-2 shows that the mean BMI of group-I patients was 19.72 (±1.73) and group-II was 19.67(±1.06). The difference of mean BMI of group-I and group-II patients was not statistically significant. The mean haemoglobin level of group-I patients was 11.9(±1.8) and group-II patients was 12.1(±1.5). The difference of mean haemoglobin level of group-I and group-II was not statistically significant

Table 3: Distribution of both groups of patients by condition of wound

Condition of wound	Group- I	Group- II	Total
Satisfactory healing	41 (82%)	40 (80%)	81 (81%)
Disturbance of healing	4 (8%)	5 (10%)	19 (19%)
Minor wound infection	3 (6%)	2 (4%)	
Moderate wound infection	2 (4%)	3 (6%)	
Severe wound infection	0 (0%)	0 (0%)	

Table 3 shows that after categorization of wound infection; out of 100 patients, 81 patients had found satisfactory healing and 19 patients had found from disturbance of healing to severe wound infection.

Table 4: Distribution of both groups of patients according to the operation

Operation	Group- I	Group- II	p
Cholecystectomy	23	21	>.05
Gastro jejunostomy	16	18	
Choledecho lithotomy	4	4	
Resection & anastomosis	2	3	
Interval appendisectomy	5	4	

Table 4 shows that in the group- I, 23 patients had cholecystectomy, 16 had gastrojejunostomy, 4 had choledecholithotomy, 2 had resection and anastomosis of small gut and 5 had interval appendisectomy. In the group-II; 21 patients had cholecystectomy, 18 had gastrojejunostomy, 4 had choledecholithotomy, 3 had resection and anastomosis of small gut and 4 had interval appendecectomy. There is no statistically significant difference in between 2 groups of patients.

Table 5: Distribution of both groups of patients by length of incision of operation

Length of incision (cm)	Group- I	Group- II	p
7-8	15	16	>.05
9-10	14	16	
11-12	12	12	
13-14	6	4	
15-16	3	2	

Table 5 shows that among the group- I; 15 patients had 7-8cm incision, 14 had 9-10cm, 12 had 11-12cm, 6 had 13-14cm and 3 had 15-16cm. Among the group- II; 16 patients had 7-8cm incision, 16 had 9-10cm, 12 had 11-12cm, 4 had 13-14cm and 2 had 15-16cm. No significant difference is found between the lengths of incision of both groups of patients.

Table 6: Distribution of both groups by duration of operation

Duration of operations (in minutes)	Group- I	Group- II	p
41-50	21	18	>.05
51-60	6	9	
61-70	14	14	
71-80	3	4	
81-90	6	5	

Table 6 shows that among the group-I; 21 patients had duration of operation 41-50 minutes, 6 had 51-60 minutes, 14 had 61-70 minutes, 3 had 71-80 minutes and 6 had 81-90 minutes. Among the group-II; 18 patients had duration of operation 41-50 minutes, 9 had 51-60 minutes, 14 had 61-70 minutes, 4 had 71-80 minutes and 5 had 81-90 minutes. No statistical difference is found between the duration of operation of both groups of patients.

Table 7: Distribution of both groups of patients by preoperative hospital stay

Preoperative hospital stay (days)	Group- I	Group- II	p
6-10	4 (8%)	4 (8%)	>.05
11-15	16 (32%)	12 (24%)	
16-20	12 (24%)	14 (28%)	
21-25	12 (24%)	14 (28%)	
26-30	4 (8%)	4 (8%)	
31-35	2 (4%)	2 (8%)	

Table 7 shows that Among the group-I; 4 patients had preoperative hospital stay 6-10 days, 16 had 11-15 days, 12 had 16-20 days, 12 had 21-25 days, 4 had 26-30 days and 2 had 31-35 days. Among the group-II; 4 patients had preoperative hospital stay 6-10 days, 12 had 11-15 days, 14 had 21-25 days, 4 had 26-30 days and 2 had 31-35 days. There is no statistically significant difference between the hospital stay of both groups of patients.

Table 8: Distribution of both group patients by status of wound healing

Status of wound healing	Group- I	Group- II	p
Satisfactory healing	41	40	>.05
Disturbance of healing	4	5	
Minor wound infection	3	2	
Moderate wound infection	2	3	

Table-8 shows that in the group-I, 41 patients had satisfactory wound healing, 4 had disturbance of healing, 3 had minor wound infection and 2 had moderate wound infection. In the group-II, 40 patients had satisfactory wound healing, 5 had disturbance of healing, 2 had minor wound healing and 3 had moderate wound infection. There is no significant difference of wound infection between the two groups of patients.

Discussion

Preparation of skin prior to operation is also a causative factor of wound infection. In this study, both study and control group of patients were distributed according to age and there was no significant difference of age variation between the groups. Again, regarding BMI and Hb% no statistical difference was found between the groups of patients.

In this study, total infection rate was 19%. It is higher than the international standard. This may be due to overcrowding of the hospital. In a study, surgical site infection rate was 3.03% in clean surgeries and 22.41% in clean-contaminated surgeries[14]. In a study it has been observed that the small skin incision, if associated with prolong operation time, may increase the overall insult in pediatric cardiac surgery[15]. So in this study some of the confounding variables like length of incision, duration of operation, preoperative hospital stay were compared between two groups of patients, which showed no significant difference between the groups. In a study, it was concluded that duration of operations at least partially determined by hospital factors and consequently, should be used as a quality indicator to compare SSI infections between hospitals, rather than being used as a patient factor to adjust comparisons between hospitals[16].

The compared the infection rate of patients who had preoperative razor shaving and who had hair removal by depilatory method. In a study of Adewale *et al*[17] showed that postoperative wound infection is strongly associated with the presence and degree of skin injuries inflicted during preoperative hair removal commonly after shaving. It also shows that depilatory cream is superior to razor shaving for preoperative hair removal.In our study no statistical significant difference was found between the groups who had razor shaving and who had hair removal by depilatory method.In a study of Dingmei *et al*[18], no significant differences between shaving, clipping, no hair removal and depilatory cream were observed in the frequency of surgical site infections which is similar to our result. However, Judith *et al*[19] showed that there are probably fewer surgical site infections when hair is not removed compared with shaving with a razor (moderate-certainty evidence)

Conclusion

From this study, it may be concluded that there is the same outcome of surgical site infection of postoperative patients in case of preoperative razor shaving and preoperative hair removal by depilatory method from operation site. Again this study is done in a limited scale. Further study with large scale sample size may give more conclusive findings.

Conflict of Interest: No

References

1. Tanner J and Khan D (2008) Surgical site infection, preoperative body washing and hair removal. J Perioper Pract 18:237-243.
2. National Audit Office 2004 Improving patient care by reducing the risk of hospital acquired infection: A progress report. Report by the Comptroller and Auditor General. HC 876, Session 2003–2004 London, The Stationery Office.
3. Alexander JW, Fischer JE, Boyajian M, Palmquist J, Morris MJ (1983) The influence of hair-removal methods on wound infections. Arch Surg 118: 347-352.
4. Scherpereel B, Rousseaux P, Bernard MH, Guyot JF (1979) Preoperative non-shaving for neurosurgical operations. Neurochirurgie 25:244-245.
5. Ratanalert S, Saehaeng S, Sripairojkul B, Liewchanpattana K, Phuenpathom N (1999) Nonshaved cranial neurosurgery. SurgNeurol 51:458-463.
6. Tanner J, Woodings D, Moncaster K (2006) Preoperative hair removal to reduce surgical site infection. Cochrane Database Syst Rev 19: CD004122.
7. Long AE (1967) The unshaved perineum at parturition: A bacteriological Study. Am J ObstetGynaecol 99: 333-336.
8. Court-Brown CM (1981) Preoperative skin depilation and its effect on postoperative wound infections. J Royal Col SurgEdinb 26: 238-241.
9. Gil Z, Cohen JT, Spektor S, Fliss DM (2003) The role of hair shaving in skull base surgery. Otolaryngol Head Neck Surg 128: 43-47.
10. Horgan MA, Piatt JH (1997) Shaving the scalp may increase the rate of infection in CSF shunt surgery. PedNeurosurg 26: 180-184.
11. David JL. 2004. Wound infection. Bailey & Love's Short Practice of Surgery; 24th ed, London; Arnold, 129.
12. Wilson APR, Treasure T, Sturridge MF, Grungnerg RN. Scoring method (ASEPSIS) for postoperative wound infection for use in clinical trials of antibiotic prophylaxis: Lancet, 1986;8: 311-312.
13. Richard J Howard 1999. 'Surgical infection' Schwartz Textbook of Principles of Surgery, McGraw Hill company, 7th international edition, p132.
14. Lilani SP, Jangale N, Chowdhary A, Dave GB.

Surgical site infection in clean and clean-contaminated cases. Indian Journal of Medical Microbilogy 2005;23(4): 249-252.

15. Ando M, Takahashi Y. Short operation time: An important element to reduce operative invasiveness in Pediatric Cardiac operative invasiveness in Pediatric Cardiac Surgery: The annals of thoracic surgery 2005;80 :631-635.
16. Gasmeier P, Sohr D, Breier A, Behnke M, Geffers C. Prolonged duration of operation: an indicator of complicated surgery or of surgical (mis) management? Infection 2012;39(3) 211-215.
17. Adewale OA, Olukayode OL, Olusanya A. Evaluation of two methods of preoperative hair removal and their relationship to postoperative wound infection. *J Infect DevCtries*2011; 5(10):717-722.
18. Dingmei S, Yao Y, Weifei Y. Comparison of preoperative hair removal methods for the reduction of surgical site infections: a meta-analysis.J Clin Nurs2017 Oct;26(19-20):2907-2914.doi: 10.1111/jocn. 13661. Epub 2017 Mar 12
19. Judith T, Kate M. Preoperative hair removal to reduce surgical site infection.Cochrane Database Syst Rev2021 Aug 26;8(8):CD004122.doi:10.1002/ 14651858.CD004122.pub5.

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Review Article

Role of Aspirin Anti-platelet Drug in Recurrent Pregnancy Related to Anti- phospholipid Anti-body Syndrome

Khanam S[1], Pramanik D[2], Akter T[3], Khan IA[4]

Abstract

In our day to day practice absence of viable issue due to recurrent history of pregnancy loss is not so uncommon. There are so many causes that are established as causative factors for recurrent pregnancy loss. We are first ruled out common abnormalities creating abortion by our available investigation procedure. Among recurrent pregnancy loss great significant cause is Antiphospholipid syndrome. Antiphospholipid syndrome causes recurrent pregnancy loss at any trimester of pregnancy which is pathetic for couple. Early diagnosis and preconceptional precaution for prevention of recurrent pregnancy loss is essential to deliver a healthy baby in a diagnosed case of Antiphospholipid syndrome. Antiplatelet drugs like Aspirin had a great play of role in reducing recurrent pregnancy loss due to Antiphospholipid syndrome.

Introduction

Recurrent pregnancy loss is a common problem now a day in our daily practice. Repeated pregnancy loss is a great burden for a couple as well as their surrounding circumstances. In our country this situation may breaks down the relationship between husband and wife and also from social circumstances. A tragedy arise when a couple has gone strong desire for a child but it is terminated as loss. Antiphospholipid Antibody plays like a great culprit in recurrent pregnancy loss. There are so many causes that are established as causative factor for recurrent pregnancy loss.[10] Among them systemic causes includes uncontrolled Diabetes Mellitus, uncontrolled chronic hypertension, hypothyroidism and local causes includes uterine polyp, uterine fibroid cervical incompetency etc.[1] When there are no other common investigations failed to identify then cause of recurrent pregnancy loss is suspicious for Antiphospholipid Antibody syndrome. Sometimes where confirm diagnosis of Antiphospholipid syndrome is not possible due to lack of available investigations procedure we used empirically Aspirin gives a positive result. However, recurrent pregnancy loss needs an indication to screen for Antiphospholipid syndrome.

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Incidence

Apparently among normal pregnancy approximately 10% to 30% terminated to spontaneous abortion. Among them recurrent pregnancy loss is 25 to 47 ACOG, 2001; (Dawood-Farqunarson & Queen by 2004). Antiphospholipid syndrome has a prevalence of 15% in women with 1st trimester recurrent miscarriage and this, as well as a single 2nd trimester miscarriage is one of the clinical component for diagnosis of the syndrome (Dewhurst's text book, 8th Edition, Page no-63).[2] 15% of future untreated pregnant women with Antiphospholipid syndrome miscarriage rate 90%.[3]

Pathophysiology of Antiphospholipid syndrome

Antiphospholipid syndrome is one example of an Autoimmune mediated pregnancy loss. In APS twenty auto antibodies detected against negatively charged phospholipids binding protein.[4] The spectrum of antibodies found in women with pregnancy loss encompasses nonspecific antinuclear antibodies as well as antibodies against individual cellular components like phospholipids, histones and double or single stranded DNA.[5]

Antiphospholipid syndrome (APS) or Antiphospholipid Antibody Syndrome or Hughes Syndrome is an autoimmune; hypercoagulable state caused by Antiphospholipid Antibodies APS provokes blood clot (Thrombosis) in arteries and veins as well as pregnancy related complications such as miscarriage, preterm delivery, severe preeclampsia or still birth.[1] Its serological marker is the presence of Antiphospholipid Antibodies in the blood of these patients. The relation between the presence of Antibodies against anionic phospholipid and thromboembolic complications well established over the last 25 years but the pathophysiology of the syndrome is largely unclear.

The primary antigenic determinant is β_2 glycoprotein which has an affinity for negatively charged phospholipids.[6] The Antiphospholipid syndrome encompasses

1) Lupus Anticoagulant Antibody (LAG)
2) Anti Cardiolipin Antibody (ACL) or
3) Anti β_2 Glycoprotein.[6]

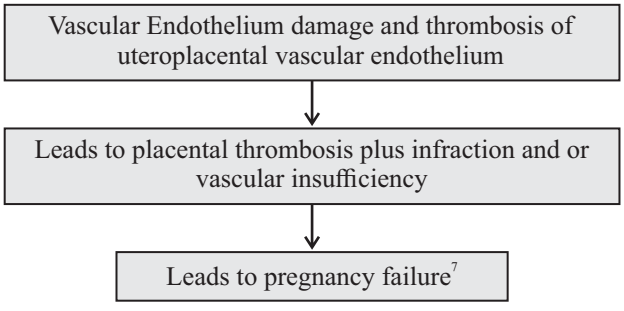
All antibodies play as culprit role in reproductive function. Maximum miscarriage occur in 1st trimester after establishment of fetal cardiac activity. Autoantibody act differently, possible and probable mechanism consist as follows.

1st Mechanism: The Antiphospholipid Antibody that are characteristic of APS are formed against normal plasma protein that are involved in the coagulation cascades and disrupt the phospholipid dependent anticoagulation process leading to a procoagulant state.[7]

2nd Mechanism: Antiphospholipid Antibodies (present in the serum of patients with APS) are known to induce platelet activation and the production of procoagulants such as Von Willebrand factor, thereby encouraging thrombosis. Antibodies have a direct toxic effect on trophoblasts tissue impending uteroplacental blood flow.[7]

3rd Mechanism: Interfere prostacyclin plus thromboxane balance by interfering with prostacyclin production.[7]

Ultimate mechanism involved pregnancy loss: Clinical presentation



Antiphospholipid Syndrome is an autoimmune multi system disorder characterized by arterial, venous or small vesseles thromboembolic events and/or pregnancy morbidity in the presence of persistent Antiphospholipid Antibodies (APS). Primary Antiphospholipid syndrome is used when APS occurs in the absence of any other related disease and secondary APS is used when APS occurs in the context of other autoimmune diseases such as, SLE. In rare case APS leads to rapid organ failure due to generalized thrombosis this is termed catastrophic APS (CAPS) and is associated with a high risk of death in pregnant women affected by APS.[1]

APS significantly increase pregnancy loss. It is estimated that 80% of women with APS experience at least one fetal loss (ACOG, 2005).[7] The high loss rate of pregnancy through recurrent spontaneous abortion. The presence of Antiphospholipid Antibodies does not produce symptom until a clot is precipitated. Primary symptom is 1st trimester pregnancy loss after establishment of fetal cardiac activity.[4] APS may be suspected when a patient gives a strong family history has an unproven suspicious personal history or has repeated spontaneous abortion when other underlying conditions have been ruled out.

Others common secondary signs/symptoms are

- Superficial thrombophlebitis
- Deep vein thrombosis
- Thrombosis (Arterial or Venous)
- Pulmonary embolism
- Septic pelvic thrombophlebitis.[7]

Diagnostic criteria for APS is dependable on Clinical and Laboratory criteria. According to the American College of Obstetrics & Gynaecologist (ACOG, 2005). APS is defined by certain laboratory & clinical criteria. On clinical plus on laboratory criterion must be present to confirm diagnosis.[7]

Clinical criteria

Vascular thrombosis includes venous and arterial.

Fetal loss variable certainity like:

I. One or more unexplained fetal death beyond 10 weeks gestation.
ii. One or more premature birth before 34 weeks of gestation.
iii. Three or more unexplained consecutive spontaneous abortion without hormonal or chromosomal abnormalities.[8]

Laboratory criteria:

Includes-

I. Anticardiolipin Antibody (ACL)
ii. Lupus Anticoagulant (LA)[9]

Role of Aspirin (Antiplatelet drug) in Antiphospholipid syndrome

Variants treatment options for APS including low dose Aspirin (LDA), Heparin, Prednisolone and Intravenous Immunoglobulin (IVIG) have been investigated.[10] A systemic review showed that Prednisolone and Intravenous Immunoglobulin (IVIG) do not improve pregnancy outcomes and are associated with increased risk of diabettis and premature birth.[11] The same review concluded that LDA along was not of significant benefit but a combination of LDA and unfractionated heparin reduced subsequent pregnancy loss by 54%. Thus LDA and heparin are the recommended treatment for women with recurrent miscarriage and APS. In clinical practice low molecular weight heparins are preferred as they have reduced risk of thrombocytopenia.

Only need once daily administration and levels do not need to be monitored. However, low molecular weight may not have the same effect in reducing risk of miscarriage in APS.[12] Aspirin is an Antiplatelet drug which designated as Anticoagulation process mediated by Antibody detected in Antiphospholipid syndrome.

It acts against main culprit mechanism of abortion in APS like prevention of thrombosis in placental bed and consequence placental insufficiency and fetal loss. And we continue the concurrent pregnancy wellbeing by using Aspirin.

Aspirin (Antiplatelet drug) play role in following pathways:

1st- Prevention of thrombosis with low dose of Aspirin (81 mg).[13] In our country circumstances dose is (75mg) giving throughout pregnancy and for (6-8) weeks postpartum.

2nd- Improvement of placental blood flow by decreasing thromboxane to prostacyclin ratio with low dose Aspirin.[13]

Conclusions

Although invention of Antiplatelet drug like Aspirin in Obstetrics practice have limited use but patients are beneficial suffering from Recurrent pregnancy loss. We can prevent the disaster from hazards of antibody mediated Recurrent pregnancy loss by using low dose Aspirin. Anticoagulation appears to prevent miscarriage in pregnant women. In pregnancy low molecular weight heparin and low dose aspirin are used instead of warfarin because of warfarin teratogenicity. Women with recurrent miscarriage are often advised to take aspirin and to start low molecular weight heparin treatment after missing a menstrual cycle. Thus aspirin play a great role the patients having Antiphospholipid Syndrome and as well as history of Recurrent pregnancy loss. APS is treated by giving Aspirin to inhibit platelet activation and/or Warfarin as an Anticoagulant. So, use of Aspirin greatly reduced Recurrent Pregnancy Loss. ith ill people or their environment, wearing mask etc.

References

1. Bangladesh Journal of Obstetrics and & Gynaecology, Volume-29(2) page 106-110, 2014
2. Original Article Levy Y et al 2005, Increased Platelet deposition on extra cellular matrix under flow condition in patients with antiphospholipid syndrome who experience thrombotic events. Arthritis Rheumatism(52) page 4011-4017
3. WarrenJ, Silver R: Autoimmune disease in pregnancy: Systemic lupus erythromatosis and antiphospholipid syndrome (Obstet Gynecol Clin North America 31(2): 345-372, 2004.
4. Obstetrics & Gynaecology, Dewhurst's Textbook of, 7th edition page 101-102, 8TH edition page 63
5. Pathologic Basis of Disease, Robbins & Cortan, 8th edition page 115-123
6. Landon MB, Galan HL, Jauniaux ER, Driscoll DA, Berghella V, Grobman WA, Kilpatrick SJ, Cahill AG. Obstetrics: normal and problem pregnancies. Elsevier Health Sciences; 2020 Feb 17.
7. Vincenzo B. Obsretric Evidence Based Guidelines. London: Informa. 2007:3-16.
8. Current Diagnosis and Treatment, Obstetrics and Gynaecology, 11th edition page 241
9. Dutta DC. Textbook of obstetrics. Hypertensive disorders in pregnancy. 2004;6:221-42.
10. CA Laskin, KA Spitzer, CA Clark, MR Crowther, The Journal of 2009, jrheum.org
11. American College of Obstetricians & Gynaecology (ACOG), Antiphospholipid Syndrome, ACOG practice Bulletin No. 68 Obstet Gynecol 106(5), 2005; 1113-1121.
12. Manual of High Risk Pregnancy and Delivery, Elizabeth Strepp Gilbert, 5th edition page 271-281, 312-313
13. Basic & Clinical Pharmacology, Bertram G. Katzung, 11th edition page 598-600

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Case Report

Management of Disseminated Intravascular Coagulation Following PPH: A Case Report

Ahmed S[1], Islam R[2]

Abstract

Disseminated intravascular coagulation is a life threatening complications of severe post-partum hemorrhage. That is the end point of a pathologically activated cascade leading to excessive consumption of platelets culminating bleeding. It results from washing out of all important procoagulants. A case of post-partum hemorrhage following caesarean section is reported. She was treated by emergency peripartum hysterectomy and aggressive blood and blood component therapy.

Keywords: Disseminated Intravascular Coagulations (DIC), Post-Partum Hemorrhage (PPH), Blood component therapy.

Introduction

Postpartum hemorrhage is a life threating obstretic emergency that is nightmare for an obstetrician during her professional life. It complications 3-6% of all deliveries and accounts for 15-20% of maternal deathin Bangladesh.[1] Disseminated intravascular coagulation (DIC) is a clinicopathological syndrome characterized by the formation of fibrin clots with concomitant consumption of platelets and coagulation factors that leads to organ failure and contributes to a high mortality if left tuntreated.[2] Anemia, malnutrition poorly supervised deliveries, delay in transfer of patient to tertiary care center and lack of adequate blood and blood components therapy contribute to the gross outcome.[3-5]

Although post-partum hemorrhage is anticipated in some high risk cases, it occurs unexpectedly in many others cases.[6] Unpredictability of primary PPH contributes the main hazard of home deliveries. Shock following severe hemorrhage and DIC are common life threating complications of post-partum hemorrhage.[7] Timely replacement of blood and blood products in an aggressive manner can save many young women dying from these serious but preventable complications of pregnancy and delivery.[8]

Case Presentation

30 years old woman with amenorrhea for 37 weeks presented with occasional lower abdominal pain. She hadD her first cesarean section 9 years back. She was under complete antenatal checkup. Now despite optimal medical therapy her pain did not reduce. Then her lower segment caesarean section (LUCS) was done on the next day of her admission at morning. After the operation she was reasonably well. Her uterus was contacted; urine output was good and no excessive per vaginal bleeding.

But after 2:30 hours of operation she developed excessive per vaginal bleeding. Then she was treated as per as treatment protocol of primary post-partum hemorrhage. Ballooncatheter was done. Bleeding was reduced in amount then after 1 hour again bleeding started, her peripartum hysterectomy was done at evening for saving her life. Patient was severely anemic so, combat her loss 3 unit blood was given on that day. She had no history of any bleeding and thrombosis or anticoagulant history. Before peripartum hysterectomy patient was receiving, oxytocin, methylergometrine, maleate, ceftriaxone, misoprostol. An emergent complete blood count was drawn and a significant platelet count drop (85000u/L) was noted then an emergent coagulations studies were taken .There were significant for an elevated international normalized ration (INR) of more than 2.70, clotting time 12 min 5 sec , D dimer 5.677 microgram, APTT 52.1sec . These findings indicated blood loss, fibrinogen and platelet depletion in the widespread thrombotic process and breakdown products of fibrinogen and hemoglobin due to extensive clotting and red blood cell sludging in thrombotic capillaries, respectively. To see end organ damage we performed renal and liver function test here. Creatinine was 1.20 mg/dl and S. ALT/ SGPT was 99.98 U/L.

Supportive therapy with blood products (fresh human blood) to replace lost blood components included 6 units fresh human blood was given. Over the next 24 hour with the above interventions. She improved clinically with stabilization of thehemoglobin and platelet counts. She was discharged from hospital after one week. During follow up she remained clinically stable with no evidence of recurrent haematogical abnormalities, residual end organ damage.

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Discussion

Disseminated intravascular coagulation (DIC) is a syndrome characterized by increased turnover of coagulation factors, platelet destruction, activation of fibrinolytic system, formation of thrombi in the micro circulation and uncontrolled thrombin activity. It is a life threatening complication seen during pregnancy or after delivery.[9] The conditions where DIC may occur are abruptio placenta, thrombocytopenic purpura, jaundice in pregnancy, HELLP (hemolysis elevated liver enzymes low platelet count) syndrome. Intrauterine death (IUD) of fetus, pre eclamsia, eclamsia, septicemia, hypovolemic shock, amniotic fluid embolism, vesicular mole etc.

These conditions trigger delicate hemostatic mehanismeither by endothelial injury or by release of thromboplastin and phospholipids. Because of a hypercoagulable state in pregnancy, prevalence of any provocative factor can easily unset the normal balance, culminating in disseminated intravascular coagulopathy. Following severe post-partum hemorrhage, DIC mayoccourue to diminished pro coagulants or increased fibrinolytic activity. DIC can be diagnosed by clinical signs and by laboratory investigations. Bleeding from venepuncture site, abdominal surgical wound site, gastric hemorrhage, appearance of petechial hemorrhage; raise suspicion of onset of DIC.

This involves maintenance of circulatory blood volume with appropriate fluid replacement. Rapid infusion of fresh frozen plasmas is recommended at 15ml/kg, with massive blood transfusion. One liter of fresh frozen plasma is recommended for 6 units of blood transfused. Platelet transfusion is recommended to maintain platelet count above 50,000/cumm and cryoprecipitate to be administered, if the fibrinogen level falls to less than 1 gm/dl. Recombinant factor VII has shown definitive role in the treatment of severe postpartum hemorrhage with disseminated intravascular coagulation.[10]

It is difficult to assess the particular disorders of coagulations, due to rapid changes in the disorder from one phase to another. Volume replacement by massive whole blood transfusion is the sheet anchor of treatment to replenish fibrinogen and other pro coagulants. A volume of 500 ml of fresh blood raises the fibrinogen level by 12.5mg/100ml. It also adds 10000-15000 platelets to the circulation. Fresh frozen plasma contains fibrinogen and other coagulation factors including V and VIII. It also contains anti thrombin III, which prevents intravascular clotting. Fresh frozen plasma must be ABO compatible but need not be Rh compatible.

Platelet concentrates may be given to the patient if platelet count is below 50,000/cumm. Platelets are administered rapidly over a period of 10 minutes. It should be ABO abd Rh compatible. One unit of platelet transfusionraises the platelet count by 5000 to 10,000/cumm; cryoprecipitates are rich in fibrinogen and factor VIII, XIII. One bag of cryoprecipitate will raise blood fibrinogen level by 5 mg/dl. Obstetric hysterectomy is a lifesaving procedure in intractable atonic postpartum hemorrhage.[11] At time the surgeon is in a dilemma; whether to sacrifice a women's reproductive capacity, especially if she is nulliparous or having less than 2 children. A timely decision to perform hysterectomy can make the difference between life and death of the patient.[12] A quick subtotal hysterectomy usually saves life in conditions of acute blood loss and shock. Training of resident doctors to perform obstetric hysterectomy in an emergency situation is important. Networking of regional blood blanks can help in timely procurement of requisite blood and its components in dire emergencies.

Conclusion

Disseminated intravascular coagulation is frequently found in primary postpartum hemorrhage and regarding this patient her life was saved in cost of hysterectomy. So early diagnosis and prompt action is the key to save life from the complications of postpartum hemorrhage.

Conflict of interest: No

References

1. International Relations and SecurityNetwork, Primary Resources in International Affairs. Constitution of the People's Republic of Bangladesh; 1972. [Last accessed on 2017 Jan 01]
2. H. Wada, T. Matsumoto, Y. Yamashita, "Diagnosis and treatment of disseminated intravascular coagulation (DIC) according to four DIC guidelines", Journal of Intensive Care, vol. 2, no. 1, p. 15, 2014.
3. J. S. Kim, J. S. Ryu, S. H. Jeon, et al., "Dramatic response of acute disseminated intravascular coagulation to erlotinib in a patient with lung adenocarcinoma with activating EGFR mutation", The Journal of International Medical Research, vol.46, no. 1, pp. 533-537, 2018.
4. R. A. Costello, S. M. Nehring, Disseminated Intravascular Coagulation (DIC). Stat Pearls. Stat Pearls Publishing Stat Pearls Publishing LLC, Treasure Island, FL, USA, 2018.
5. Text book of obstetrics DC Dutta , 6th edition 2004 new central book agency pvt ltd, calcutta complications of thrid stage of labour , page 411-422.
6. Postpartum hemorrhage ushakrishna, D.K tank, shirishdeftary 4th edition jaypee brothers, new delhi.
7. The management of labour S Anil Kumaran K BhaskarRaoorientslongmanPvt Ltd, Hyderabad The management of PPh. Page 209-229.
8. Practical guide to high risk pregnancies Fernando Arias 2nd edition Mosby year Book, London Hematological problems during pregnancy, pages 245-262.

9. Progress in obstretics and gynecology john stud, 17 th edition Churchill Livingstone, New Delhi Postpartum uterine artery, pages 264.
10. Abonen J, Jokela Recombinant factor VIIa for life threatening postpartum haemorrhage BV- JrAnesthe 2005, 94: 542-545.
11. SaheyPritiBala, KumariArchana, clinical review of emergency obstetrichysterectomy, J obstetGynacol India, vol 59, no 5: sept- oct 2009, pages: 427-431.
12. Preneshwari Devi RK, sing NN, singh TD, emergency obstrtric Hysterectomy, J ObstetGynaecology India, 2004: 54:127-129.

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