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Guidelines For Author
ABSTRACT

Aim: Aim of this study is to evaluate T2DM demographically including age and sex, awareness for self monitoring of blood glucose (SMBG) and as well as to find out the effects of prescribed anti-diabetic drugs on maintaining optimal blood glucose level in diabetic patients.

Methodology: This was a prospective, non interventional observational study, conducted at Department of Pharmacology in association with Department of Medicine, Dr. S. N. Medical College, Jodhpur. Information of patients collected included age, sex, diagnosis, duration of medicine, frequency of blood glucose monitoring, Fasting Blood Glucose level and Post-Prandial Blood glucose level were noted in case record form and analyzed.

Results: Total 250 patients, 193 (77.2%) male and 57 (22.8%) female were included in the study. Male to female ratio was 3.38:1. 30.8% of T2DM patients were recorded with age of ≤60 years and 69.2% of T2DM patients recorded with age more than 60 years, whereas 50% of patients had poor glycemic control.

Conclusion: Demographic features of patients with type 2 diabetes treated in primary care are associated with optimal glycemic control. This study strongly highlights the domination of OHA but documents shifting trend towards insulin in the treatment of Type 2 diabetes and the need for periodic blood-glucose monitoring in patients receiving anti-diabetic drug treatment to identify inadequately controlled glycemic levels, so that drug therapy can be intensified and multiple drug interventions can be planned in order to obtain an optimal glycemic level.

Keywords: Type 2 DM, FBS, PPBS, SMBG

INTRODUCTION

Diabetes mellitus (DM) is a metabolic disorder that adversely affects the normal physiological ability to produce or utilize insulin1. It is characterized by hyperglycemia. Elevated blood sugar level is found in diabetes that can cause severe short-term and long-term consequences ranging from brain injury to heart disease and amputations2. So, diabetes mellitus is one of the heterogeneous carbohydrate metabolism disorders where defects occur in insulin utilization and secretion.

DM has dreadful complications and can significantly compromise the quality of life. In 2017 according to International Diabetes Federation

1 Resident, 2 Professor, 3 H.O.D. and Senior Professor, Department of Pharmacology
4 Professor, 5 Resident, Department of Medicine,
Dr. S.N. Medical College Jodhpur (Rajasthan)
Corresponding Authors: Dr. Akhtar Ali e-mail: drakhtar06@gmail.com Mobile No.: 8058961071
Atlas 424.9 million people suffer from DM and the number is expected to rise further to 628.6 million by 2045.3 Healthcare expenditures for people with diabetes are assumed to be on average two-fold higher than people without diabetes3. Middle and low economic countries are having 79% of the global burden of DM as a result of population growth, aging and sedentary lifestyles3. Currently, India is having second position in respect of the most number of diabetes patients after China. By 2045 it is expected that India will have 134.3 million diabetes patients, the most in world3. Worldwide, DM is regarded as one of the most complex chronic disease. T2DM is the 7th leading cause of morbidity and mortality in the USA. Diabetic patients require life-long personal care to decrease the chance of developing long-term complications. The chronic nature of the disease, which is burdened with many complications, and the high costs of treatment contribute to rising demand for high-quality diabetes care. In diabetes care should be involved the construed to have the degree to which medical services, in relation to individual buyers and to the entire population, increase the likelihood of obtaining desired outcomes of treatment and are consistent with current knowledge4,5.

The course and level of metabolic control of type 2 diabetes are affected by many factors related to lifestyle, physical activity, diet control and as well as the quality of medical care. The place of residence of the patient also matters as it determines the availability of health care, education, therapy, specialist advice, and the degree of patient adherence to medical recommendations. Striving for the highest quality of patient care, as well as multidirectional action to improve the health and living conditions of people with diabetes, is of great importance, especially in the context of a number of reports based on a multi-centre clinical trials, which confirm the importance of good metabolic control in preventing complications and improving the quality of life of patients6,7,8,9.

In India, limited studies have focused on diabetes care and provide insight into the current profile of patients and their management. Therefore, this study was carried out to find the efficacy of prescribed anti-diabetic drugs in maintaining adequate glycemic control in diabetic patients attending a tertiary care teaching hospital in Western Rajasthan.

MATERIALS AND METHODS
This study was a prospective, noninterventional and observational study. It was conducted in association with the Department of Medicine in Mathura Das Mathur (M.D.M.) Hospital, Jodhpur (Tertiary Care Teaching Hospital) which is the largest hospital in Western Rajasthan. This study included 250 outpatients with Type 2 Diabetes Mellitus. Patients were not advised any new drug(s), by the investigator during the study period. All patients were included after receiving informed consent as well as clearly explained the purpose and nature of study in their language. All data of patients were kept confidential.
We visited the diabetic OPD every Wednesday and collected all informations pertaining to every patient, such as the name, age, gender, address, relevant medical history, past history, family history etc. Complete information was obtained either direct conversation with patients or from prescribed OPD slips and then mentioned in Case Record Form.

Known cases of Type 2 Diabetes Mellitus with and without complications, patients aged more than 20 years and only outpatients were included in this study. Pregnant women, Gestational diabetes patients, Type 1 Diabetic patients, patient not willing to participate and bedridden patients were excluded from this study. After recording the obtained information in the Case Record Form the data were subjected to further analysis. Data collection was analyzed further as a number of patients, gender-wise distribution of study patients, patients were divided into age group for the occurrence of type 2 diabetes mellitus, fasting blood glucose level, post-prandial blood glucose level and frequency of blood glucose monitoring.

**RESULTS**

The demographic profile of patients

A total of 250 patients who fulfilled the inclusion criteria were included in the study at Mathura Das Mathur Hospital, Attached group of Hospitals of Dr. S. N. Medical College, Jodhpur a largest hospital in Western Rajasthan.

**Gender wise distribution of study patients**

<table>
<thead>
<tr>
<th>Sex</th>
<th>Number of patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>193</td>
<td>77.2</td>
</tr>
<tr>
<td>Female</td>
<td>57</td>
<td>22.8</td>
</tr>
<tr>
<td>Total</td>
<td>250</td>
<td>100</td>
</tr>
</tbody>
</table>

![Figure 1: Gender wise distribution of study patients.](image)

Table 1: Gender wise distribution of study patients

In incidence of T2DM is more in male compare to the female Out of 250 patients, 193 (77.2%) were male and 57 (22.8%) were female. Male to female ratio was 3.38:1 which is quite high, shown in table 1 and figure 1.
The major group of patients was between the age of 61-70 years (56.8%), followed by 71 years or above (12.4%) and least number of patients were in the age group 60 years and below the age of 60 years (30.8%) as shown in table 2 and figure 2. This is also indicating that majority of T2DM patients were more than 60 years of old.

In the age group less than 60 years, fasting Blood Glucose was ≤130 mg/dl in 29 patients and was ≥131 mg/dl in 48 patients. While Fasting Blood Glucose was ≤130 mg/dl in 93 patients ≥61 years and was ≥130 mg/dl in 80 patients as mentioned in table 3 and figure 3. Overall 48.8% of patients had Fasting Blood Glucose ≤130 dg/dl and 51.2% had fasting blood glucose ≥130 mg/dl.

Significantly more controlled optimal FBS levels were found in age more than 60 years diabetic patients compared to less than 60 years old diabetic patients.
Distribution of study patients as per their Prandial Blood Sugar (PPBS):-

Table 4: Post-Prandial Blood Sugar (PPBS) wise distribution of study patients

<table>
<thead>
<tr>
<th>Age (Years)</th>
<th>=≤180 mg/dl</th>
<th>=≥181 mg/dl</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>Percentage</td>
<td>Number</td>
<td>Percentage</td>
</tr>
<tr>
<td>≤60 years</td>
<td>11</td>
<td>66</td>
<td>77 (30.8%)</td>
</tr>
<tr>
<td>61 years</td>
<td>30</td>
<td>143</td>
<td>173 (69.2%)</td>
</tr>
<tr>
<td>Total</td>
<td>41</td>
<td>209</td>
<td>250</td>
</tr>
</tbody>
</table>

Among the patients age less than 60 years, Post-Prandial Blood Sugar was ≤180 mg/dl in 11 patients and ≥180 mg/dl in 66 patients. Post-Prandial Blood Sugar was ≤180 mg/dl in 30 patients who were ≥61 years and 143 patients in the study group had fasting blood glucose levels ≥181 mg/dl (table 4 and figure 4). Overall 16.4% patients had Post-Prandial Blood Sugar ≤180 mg/dl and 83.6% had post-prandial blood sugar ≥181 mg/dl Post-Prandial indicating poor Post-Prandial Blood Sugar control, which is shown in table 4 and figure 4.

No significant difference was found in age less than 60 years and age more than 60 years for controlling optimal Post-Prandial Blood Sugar level.

Frequency of blood glucose monitoring (Days) distribution of study patients: -

Maximum number of patients were from the group 16-30 days (79.6%) frequency of blood glucose monitoring, followed by 8-15 days (14.8%), ≤7 days (4.4%) and minimum in the group >30 days (1.2%). Majority of patients belonged to frequency of blood glucose monitoring 16-30 day (79.6%) shown in table 5 and figure 5. There was no significant difference in frequency of blood glucose monitoring in ≥61 years and ≤60 years age groups, respectively. Frequency of blood glucose monitoring was quite low in patients shown in table 5 and figure 5. Patients who were doing glucose monitoring in 15 days or less than 15 days were also having a low self-monitoring of blood glucose.

Table 5: Frequency of blood glucose monitoring (Days) distribution of study patients

<table>
<thead>
<tr>
<th>Frequency of blood glucose monitoring (Days)</th>
<th>Age (Years)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>Percentage</td>
</tr>
<tr>
<td>≤7 days</td>
<td>2</td>
<td>2.6%</td>
</tr>
<tr>
<td>8-15 days</td>
<td>11</td>
<td>14.3%</td>
</tr>
<tr>
<td>16-30 days</td>
<td>63</td>
<td>81.8%</td>
</tr>
<tr>
<td>&gt;30 days</td>
<td>1</td>
<td>1.3%</td>
</tr>
<tr>
<td>Total</td>
<td>77</td>
<td>30.8%</td>
</tr>
</tbody>
</table>

Figure 5: Frequency of blood glucose monitoring (Days) distribution of study patients.

Maximum patients monitoring their blood glucose levels were in the group 16 to 30 days.
DISCUSSION

As Diabetes mellitus is reaching potentially epidemic proportions in India. The level of morbidity and mortality due to diabetes and its potential complications are enormous and pose significant healthcare burdens on both families and society.

Our aim in this study was to evaluate T2DM demographically like age and sex wise, awareness for self-blood glucose monitoring (SBGM) and as well as to find out the effect of prescribed anti-diabetic drugs on maintaining optimal blood glucose level in diabetic patients.

In the current study 77.2% of diabetic patients were male and 22.8% diabetic patients were female. Male to female ratio was 3.38:1 which was not accordance to Mathur et al10 and Nordstrum et al11 they found in their study that sex ratio in T2DM is 1:1 and 1.6:1, respectively.

In the present study incidence of T2DM patients with age ≤60 years was 30.8% while the incidence of patients with age more than 60 years was 69.2%, which was not similar to the study of Muhammad et al12 who reported that 42.2% diabetic patients had age 60 years or above 60 years. Observations in our study were quite similar to the study conducted by Mathur et al 10 who reported that 65.2% of patients were more than 60 years old. This incidence is justifiable because T2DM is an old age disease and routine diagnosis of T2DM remain quite late as patients remain asymptomatic for a long time.

In the current study, SMBG was less than 20% of and was performed in a range from 3 days to 15 days. This less frequency of blood glucose monitoring and may be because of high-cost of SMBG, lack of awareness and poor health education. This results in poor optimal glycemic control in T2DM patients.

Franciosi et al13 investigated the frequency of SMBG and its association with metabolic control and quality of life by use of a questionnaire. No association was found between a higher frequency of SMBG and better glycemic control in patients with type 2 diabetes who are not using insulin. However, SMBG frequency of at least one time a day was significantly related to higher levels of distress, worries, and depressive symptoms. Distress and worries were also significantly related to SMBG frequency of at least one time per week.

Karter et al14 used a cohort design (n = 17,601) to assess the association between SMBG and glycemic control. They found that monitoring at the recommended frequency (at least daily) was associated with a better HbA1c level of 0.4% (P < 0.0001) compared with less frequent monitoring. Because of the study design of current study, we could not find out the causal association between SMBG and glycemic control; it is possible that more motivated subjects choose to initiate SMBG.

Soumerai et al.15 evaluated a policy providing free blood glucose monitors, and they found that initiating SMBG was associated with a significant reduction in HbA1c levels.

In current study majority of the patients were receiving oral hypoglycemic agents (OHAs) which did not need strict SMBG.
of blood glucose and HbA1C are integral components of the standards of care in diabetes. They are designed to assess the effectiveness of a treatment plan and provide guidance in selecting appropriate medications and dosage/s16. SMBG allows patients to assess their own response to medication, minimize the risk of hypoglycemia, and determine whether they are achieving glycemic control. Optimal glycemic control is achieved when FPG is 70–130 mg/dl, 2 h postprandial <180 mg/dl, and bedtime glucose is 90–150 mg/dl. However, testing six to eight times daily may burden patients and may result in non-compliance. Therefore, it is recommended to ensure that patients are properly instructed and are given regular evaluation and follow-up17. Self-monitoring of blood glucose is essential in patients with diabetes who are on an intense insulin regimen (three to four injections of basal and prandial or insulin pump). It monitors and prevents hyperglycemia and a possible side effect of hypoglycemia. Blood glucose level is usually checked prior to meals, prior to exercise, prior to driving, and at bedtime. The evidence is insufficient to prescribe SMBG for patients not receiving an intensive insulin regimen18.

In the present study, 37.67% diabetic patients were ≤60 years old and had FBS ≤130 mg/dl. 53.76% diabetic patients were ≥61 years old and had FBS ≤130 mg/dl. Overall 51.2% patients had FBS ≥131 mg/dl. 69.2% of patients had Post-Prandial Blood Sugar ≥181 mg/dl and there was no significant difference in age more than 60 years or age less than 60 years. This indicates that more than 50% of patients had not achieved optimal glycemic control and this was similar to several other studies19,20, 21 et al where more than 50% of diabetic patients were having poor glycemic control. This poor glycemic control is not justifiable and can occur due to various reasons like increase insulin resistance with age, the lack of awareness to SMBG, poor health education, poor dietary control, lack of follow up, high cost of medicine and as well as poor socioeconomic status of patients.

Diabetes control has a chance to improve due to standardized guidelines implementation in many countries. Many of these recommendations scope on holistic patient care delivered by general practitioners, dietitians, nurses, lifestyle consultants, social workers, psychologists and also including patients' self-management22. Guidelines implementation is meeting many barriers, such as lack of follow-up, lack of awareness and lack of awareness of novel recommendations among physicians23.

CONCLUSION
Demographic features of patients with type 2 diabetes treated in primary care are associated with glycemic control. Physicians should take into consideration patient demographic characteristics, especially being a younger man, when planning treatment of type 2 diabetes. The study strongly highlights the domination of OHAs but documents shifting trend towards insulin in the treatment of
Type 2 diabetes and the need for periodic blood-glucose monitoring in patients receiving anti-diabetic drug treatment to identify inadequately controlled glycemic levels, so that drug therapy can be intensified and multiple drug interventions can be planned in order to obtain an optimal glycemic level. It also highlights the need for lifestyle modification measures along with anti-diabetic drug treatment for achieving better glycemic control in Type 2 diabetes.

LIMITATION OF STUDY
HbA1c level was not performed because of the high cost of investigation so, long-term control of blood glucose was not analyzed. In our study rural population was not included and data of the study was very small, so for final conclusion further studies are warranted, with a large amount of data.

ACKNOWLEDGMENTS
The authors express their thanks to the all Residents, Department of Medicine, Dr, S. N. Medical College, Jodhpur for their assistance in conducting the study.

CONFLICT OF INTEREST
The authors declare that no conflict of interest, financial or otherwise, exists.

REFERENCES


ABSTRACT

**Background**: The most serious, life-threatening infections caused by a group of drug-resistant bacteria (Enterococcus faecium, Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa and Enterobacter species) are named as "ESKAPE" pathogens by the Infectious Diseases Society of America (IDSA), because they effectively escape the effects of antibacterial drugs. The purpose of this study was to monitor the incidence of ESKAPE pathogens at tertiary care hospital.

**Material and Method**: Present study was a hospital-based observational study conducted for a period of 6 months (July 2019 to December 2019), all samples received in microbiology laboratory were processed for pyogenic aerobic culture sensitivity and phenotypic detection of superbugs.

**Result**: Out of 2176 samples, 437 (86.87%) ESKAPE pathogens were isolated. Maximum cases were isolated from Gynaec wards followed by NICU and PICU (37.52%, 27.23%, 14.18% respectively). Maximum isolates were from Blood culture followed by pus and urine (39.67%, 21.74% and 16.25% respectively). In ESKAPE isolates there was predominance of Gram positive isolate Staphylococcus aureus (36.61%) and Enterococcus faecium (14.64%) as compared to Gram negative Klebsiella pneumoniae (29.97%), Acinetobacter baumannii (10.75%), Pseudomonas aeruginosa (7.78%) and Enterobacter species (0.22%)(224:213). Amongst S. aureus predominated 65% were MRSA and 15.62% were Inducible Clindamycin Resistance (ICR), ESBL (1.14%) and none of them were VRE.

**Conclusion**: Judicious use of antibiotics is the need of the day to control the spread of MDR "ESKAPE" bugs. There is also an urgent need to develop Antimicrobial Stewardship.

**Key Words**: Extended spectrum β-lactamases, Methicillin Resistant Staphylococcus aureus, Vancomycin Resistant Enterococci, Multidrug resistance.

INTRODUCTION

Infectious Diseases Society of America (IDSA), has highlighted a group of antibiotic resistant bacteria as “ESKAPE pathogens”, because they effectively escape the effects of antibacterial drugs (Jack N. Pendleton et al., 2013) [1].

ESKAPE is an acronym for the group of bacteria, encompassing both Gram-positive and Gram-negative species, made up of Enterococcus faecium, Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa, and Enterobacter species.

ESKAPE pathogens causes severe...
diseases in human like blood stream infection, respiratory infection, urinary tract infection and soft tissue infection etc. Antimicrobial resistance has been on the rise in few decades over the globe and has become a challenge to health care system [2]. The presence of multidrug-resistant (MDR) pathogens has become a cause for serious concern with regard to nosocomial infections [3,4]. Antibiotic use is unnecessary or inappropriate in as many as 50% of cases and this creates unnecessary pressure for the selection of resistant species [2]. The various mechanisms of acquired resistance include mutation in drug target site, enzymatic degradation of antibiotics, active efflux through porins and other permeability barriers. Due to progressive selective pressure of separate and distinct groups of antibiotics, the transfer of gene responsible for drug resistance through plasmids, integrons and transposon has accelerated this spread [5,6]. Therefore, the treatment of MDR infections remains a serious problem all over the globe due to restricted availability of drugs. The aim of the present study was to detect MDR ESKAPE Pathogens and superbugs amongst ESKAPE pathogens

MATERIAL AND METHOD
Present study was the hospital based observational study which was conducted at Umaid hospital, associated group of Dr. SNMC, Jodhpur, Rajasthan for period of 6 months from July to December 2019. All samples which were received in microbiology lab during study time were included in study. Blood Samples were collected under all aseptic precaution, with care to avoid contamination from the commensal organism from surrounding skin and inoculation was done in Bactec automated blood culture [figure-1]. All collected specimen were inoculated on blood agar, Mac Conkey agar, Mannitol salt agar, Chromogenic media and Thioglycolate broth. Incubation were done at 370C, aerobically. First examination was made after 24 hours and if there was no growth subculture was done. After obtaining pure isolates further identification were done according to text book of medical microbiology [7,8]. Antimicrobial susceptibility testing was done on Muller Hilton agar as recommended by CLSI 2019 [9]. ESBL, MRSA & VRE detection were made by using Disc Diffusion method. For ESBL Ceftazidime & Ceftazidime with Clavulanic acid (>5MM zone of inhibition), For MRSA Cefoxitin / Methicillin / Oxacillin (at 35 degree C) and Vancomycin antibiotic discs for VRE detection were used. S. aureus ATCC 25923, E. coli ATCC 25922 and Pseudomonas aeruginosa ATCC 27853 were used as quality control. Statics: All data were collected and analyzed in EXCEL accordingly. Categorical data were presented as frequencies and percentages.

RESULTS
In present study time total 2176 samples were collected out of which 437(86.87%) were ESKAPE pathogens and 66 (13.12%) were non ESKAPE pathogens were isolated. Total 2176
sample were collected out of which blood culture (476), CSF culture (253), urine culture (821), vaginal culture (178), pus (303), throat swab (47), Endotracheal culture (45) and other culture (53). Maximum ESKAPE pathogens were isolated from Gynaecology followed by NICU (Table-1).

Enterococcus faecium were isolated in 64 cases (14.64%) maximum isolates were seen in urine samples 24 (33.80%) received from gynaecology ward followed by blood 18 (10.65%) and vaginal swabs 09 (16.66%) which showed maximum resistance with gentamycin 64 (14.64%), cefazolin 64 (14.64%), ciprofloxacin 49 (11.21%) and piperacillin and tazobactam 29 (6.63%). All Enterococcus faecium sensitive to vancomycin and none of them were Vancomycin Resistant Enterococcus (VRE). In linezolid 04 (0.91) were resistant.

Staphylococcus aureus were isolated in 160 cases. Maximum isolates seen in blood culture 70 (41.42%), followed by pus, urine and vaginal swabs i.e 39 (41.05%), 19 (26.76%) and 19 (35.18%) respectively. Out of 160 Staphylococcus aureus 104 were Methicillin Resistant Staphylococcus aureus (MRSA). They were having highest resistance with ciprofloxacin 109 (24.94%) followed by cefazolin 104 (24.94%). Maximum sensitive with vancomycin 100%. Out of 160 Staphylococcus aureus isolates 104 (65%) were MRSA positive and 21 (20.19%) isolates were having inducible clindamycin resistance (ICR) while amongst 56 non MRSA isolates 4 (7.14%) were having inducible clindamycin resistance (ICR).

Klebsiella pneumoniae were isolated in 131. Maximum Klebsiella pneumoniae were isolated from blood culture 59 (34.91%) followed by pus 25 (26.31%), vaginal 18 (33.33%) and urine 16 (22.53%). Maximum resistance showed with ciprofloxacin 100 (22.88%) and ceftazidine 99 (22.65%) and lowest resistance noted in nitrofurantoin 03 (0.68%) and meropenem 09 (2.05%). Out of 131 Klebsiella pneumoniae 04 (3.05%) isolates were ESBL producers and had shown multi drug resistance.

Total 47 isolates of Acinetobacter baumannii were isolated. Maximum isolates were isolated from blood 16 (9.4%) followed by Endotracheal tube 11 (34.37%) and pus 10 (10.95%). Maximum resistance noted in aztreonam 34 (7.37%) followed by ciprofloxacin 32 (7.32%) and ceftazidine 31 (7.09%) while minimum resistance showed with nitrofurantoin 03 (0.68%) followed by piperacillin and tazobactum 08 (1.83%). No ESBL was detected.

Total 34 Pseudomonas aeruginosa were isolated. Maximum isolates were isolated from pus 17 (17.89%) followed by blood culture and urine i.e 06 (3.55%) and 06 (8.45%) respectively. Maximum resistance showed with ciprofloxacin 27 (6.1%) followed by aztreonam 20 (4.5%) while minimum resistance showed with nitrofurantoin 100% in urine samples while meropenem in 01 (0.22%). Out of 34 Pseudomonas aeruginosa isolates 1 was ESBL producer.

01 (1.85%) Enterobacter species was isolated from 01 (1.85%) vaginal swab maximum resistance showed with
ciprofloxacin 01 (0.22%). No ESBL was detected.

**Discussion**

In 2011, the theme of World Health Day was “Antimicrobial resistance: no action today, no cure tomorrow”, and WHO published a six-point policy package to assist countries with tools to combat antimicrobial resistance [10]. The global emergence of antimicrobial resistance constitutes serious human and public health burdens, especially due to limited availability of treatment options.

In the hospital setting, different bacterial species may be the causative agents of infectious diseases. Due to their high level of pervasiveness and association with antimicrobial resistance, the ESKAPE group of pathogens deserve particular attention [11]. Enterococci are important nosocomial agents and serious infections caused by them are often treated with a combination of cell wall inhibitor and aminoglycoside. However, the presence of high level aminoglycoside resistance in these isolates makes this treatment combination ineffective [10]. However in present study though we had 14.66% Enterococcus faecium isolates which is similar to the studies done by Anuradha S. De et al. and Butch et al. [11,12]. In present study none of them were VRE perhaps may be due to community acquired isolates. In 36.61% Staphylococcus aureus isolates 104 (65%) were MRSA having and 15.62% Staphylococcus aureus were having ICR. Staphylococcus aureus exhibited 100% sensitivity with vancomycin which is similar to studies by conducted by Jagadevi et al, Ravindra S et al. and Abbas et al. [2,13,14].

In pediatric NICU, PICU and Pediatric ward maximum 57.25% Klebsiella pneumoniae were detected and maximum were from blood samples 59 (34.91%) followed by pus and urine. 04 ESBL isolates were detected.

Simple measures such as hand washing and barrier precautions can significantly reduce the spread of ESKAPE bugs. As these are multidrug resistant, they might pose a therapeutic challenge to the clinicians as well as microbiologists. Physicians should be aware of the local epidemiology of antimicrobial resistance to properly guide the initial therapy [15,16,17]. A strict antibiotic policy should be followed in every hospital which restricts the use of the broad spectrum agents (especially the third-generation cephalosporins). The cephalosporins should only be used as reserve drugs, in the fluoroquinolone resistant cases, with evidence based indications only. The reserve drugs such as vancomycin or those which are used against the resistance to the carbapenems, like polymyxin B and E (colistin), tigecycline and Fosfomycin should never be used indiscriminately [18-22] Most of these resistance problems are attributed to uncontrolled use of antimicrobial agents. Therefore, there is an urgent need to develop antimicrobial stewardship, to curb this threat [23-27].

**Conclusion:**

Simple measures such as hand washing and barrier precautions can significantly reduce the spread of
ESKAPE bugs. Most of these resistance problems are attributed to uncontrolled use of antimicrobial agents. Judicious use of antibiotics is the need of the day to control the spread of MDR “ESKAPE” bugs. Recommendations Hospital associated infection especially in hospitalised patients can be prevented by:

1. ICU protocols should be followed, all samples should be sent for Microbiological evaluation & detection of superbugs.
2. Isolation of patients harboring superbug is must.
3. Antibiotic policy should be followed meticulously, hand hygiene should be followed as a rule, critical evaluation of all HAI patient should be done in concordance with all staff.
4. Long-Line and IV line should be changed frequently & Silver Coated or Medicated Long Lines can be alternative option of choice.
5. ICU air surveillance and sterilization should be done according to protocol.
6. Protocols of Biomedical Waste Management (BMW) should be followed strictly. For that Posters should be displayed / BMW TRAINING / Inspection or monitoring should be done by Hospitals BMW team & pollution control board. Regular meeting of Infection Control Committee & BMW Committee should be mandatory in all hospitals.
7. Death monitoring and death analysis due to HAI should be done, critical analysis of each case is must to improve health standards, to decreases cost of treatment and stay in hospital & Improves Accreditation of any ICU & Hospital.

REFERENCES


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9) Clinical Laboratory Standards Institute. Performance standards for antimicrobial susceptibility testing. 20th Informational Supplement M100 S20. Wayne, Clinical and Laboratory Standards Institute, 2019
22) Jorge Martin Llaca-Diaz a Soraya
Mendoza-Olazaran b Adrian Camacho-Ortiz c Samantha Flores d Elvira Garza-Gonzalez a, b One-Year Surveillance of ESKAPE Pathogens in an Intensive Care Unit of Monterrey, Mexico Chemotherapy 2012;58:475–481


Table 01: SHOWING DISTRIBUTION OF ESKAPE PATHOGENS IN DIFFERENT ICU AND WARDS.

<table>
<thead>
<tr>
<th></th>
<th>NICU</th>
<th>PICU</th>
<th>GICU</th>
<th>GYNAE WARD</th>
<th>PAEDS WARDS</th>
<th>Others</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enterococcus faecium</td>
<td>06</td>
<td>08</td>
<td>09</td>
<td>27</td>
<td>08</td>
<td>06</td>
<td>64</td>
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<tr>
<td></td>
<td>(9.37%)</td>
<td>(12.5%)</td>
<td>(14.06%)</td>
<td>(12.5%)</td>
<td>(12.5%)</td>
<td>(9.37%)</td>
<td>(14.64%)</td>
</tr>
<tr>
<td>Staphylococcus aureus</td>
<td>47</td>
<td>26</td>
<td>73</td>
<td>62</td>
<td>12</td>
<td>00</td>
<td>160</td>
</tr>
<tr>
<td></td>
<td>(29.37%)</td>
<td>(16.25%)</td>
<td>(45.12%)</td>
<td>(38.75%)</td>
<td>(7.5%)</td>
<td>(0%)</td>
<td>(36.61%)</td>
</tr>
<tr>
<td>Klebsiella pneumoniae</td>
<td>51</td>
<td>18</td>
<td>08</td>
<td>42</td>
<td>12</td>
<td>00</td>
<td>131</td>
</tr>
<tr>
<td></td>
<td>(38.93%)</td>
<td>(13.74%)</td>
<td>(6.10%)</td>
<td>(32.06%)</td>
<td>(9.16%)</td>
<td>(0%)</td>
<td>(29.97%)</td>
</tr>
<tr>
<td>Acinetobacter baumannii</td>
<td>08</td>
<td>06</td>
<td>07</td>
<td>16</td>
<td>01</td>
<td>09</td>
<td>47</td>
</tr>
<tr>
<td></td>
<td>(17.02%)</td>
<td>(12.76%)</td>
<td>(14.89%)</td>
<td>(34.04%)</td>
<td>(2.12%)</td>
<td>(19.14%)</td>
<td>(10.75%)</td>
</tr>
<tr>
<td>Pseudomonas aeruginosa</td>
<td>07</td>
<td>04</td>
<td>02</td>
<td>17</td>
<td>04</td>
<td>00</td>
<td>34</td>
</tr>
<tr>
<td></td>
<td>(20.58%)</td>
<td>(11.76%)</td>
<td>(5.88%)</td>
<td>(50%)</td>
<td>(11.76%)</td>
<td>(0%)</td>
<td>(7.78%)</td>
</tr>
<tr>
<td>Enterobacter spp.</td>
<td>00</td>
<td>00</td>
<td>01</td>
<td>00</td>
<td>00</td>
<td>01</td>
<td>01</td>
</tr>
<tr>
<td></td>
<td>(100%)</td>
<td>(100%)</td>
<td>(100%)</td>
<td>(100%)</td>
<td>(100%)</td>
<td>(100%)</td>
<td>(100%)</td>
</tr>
<tr>
<td>Total</td>
<td>119</td>
<td>62</td>
<td>40</td>
<td>164</td>
<td>37</td>
<td>15</td>
<td>437</td>
</tr>
<tr>
<td></td>
<td>(27.23%)</td>
<td>(14.18%)</td>
<td>(9.15%)</td>
<td>(37.52%)</td>
<td>(8.46%)</td>
<td>(3.43%)</td>
<td>(100%)</td>
</tr>
</tbody>
</table>

**ESKAPE PATHOGENS IN DIFFERENT ICU AND WARDS**

- Enterococci
- Staph aureus
- Klebsiella
- Acinetobacter
- Pseudomonas
- Enterobacter

17
### Table 2: ESKAPE Isolates in various clinical samples

<table>
<thead>
<tr>
<th>Isolates</th>
<th>Blood culture (476)</th>
<th>CSF (283)</th>
<th>ET Tube (45)</th>
<th>Pus (308)</th>
<th>Throat Swab (47)</th>
<th>Urine (821)</th>
<th>Vaginal (178)</th>
<th>Other (53)</th>
<th>Total (2176)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enterococci</td>
<td>18 (10.65%)</td>
<td>00</td>
<td>02 (5.25%)</td>
<td>04 (4.21%)</td>
<td>07 (70%)</td>
<td>24 (33.60%)</td>
<td>09 (16.69%)</td>
<td>00</td>
<td>64 (14.64)</td>
</tr>
<tr>
<td>Staph aureus</td>
<td>70 (41.42%)</td>
<td>02</td>
<td>05 (15.62%)</td>
<td>29 (41.05%)</td>
<td>03 (30%)</td>
<td>19 (26.76%)</td>
<td>19 (35.18%)</td>
<td>03</td>
<td>160 (36.61)</td>
</tr>
<tr>
<td>Klebsiella</td>
<td>59 (34.91%)</td>
<td>00</td>
<td>13 (40.62%)</td>
<td>25 (26.31%)</td>
<td>00 (22.53%)</td>
<td>16 (33.33%)</td>
<td>18 (33.33%)</td>
<td>00</td>
<td>131 (29.98)</td>
</tr>
<tr>
<td>Acinetobacter</td>
<td>16 (9.46%)</td>
<td>01 (33.33%)</td>
<td>11 (34.37%)</td>
<td>10 (10.95%)</td>
<td>06 (8.45%)</td>
<td>03 (5.55%)</td>
<td>00 (0%)</td>
<td>00</td>
<td>47 (10.75)</td>
</tr>
<tr>
<td>Pseudomonas</td>
<td>06 (3.55%)</td>
<td>00</td>
<td>01 (3.12%)</td>
<td>17 (17.89%)</td>
<td>06 (8.45%)</td>
<td>04 (7.40%)</td>
<td>00 (0%)</td>
<td>00</td>
<td>34 (7.78)</td>
</tr>
<tr>
<td>Enterobacter</td>
<td>00</td>
<td>00</td>
<td>00</td>
<td>00</td>
<td>00</td>
<td>00</td>
<td>01 (1.85%)</td>
<td>00</td>
<td>01 (0.22)</td>
</tr>
<tr>
<td>Total</td>
<td>169 (39.67%)</td>
<td>03 (0.69%)</td>
<td>32 (7.32%)</td>
<td>95 (21.74%)</td>
<td>10 (2.29)</td>
<td>71 (16.25%)</td>
<td>54 (12.35)</td>
<td>03 (0.69)</td>
<td>437</td>
</tr>
</tbody>
</table>

### ESKAPE Isolates in various clinical samples

- **Enterococci**
- **Staph aureus**
- **Klebsiella**
- **Acinetobacter**
- **Pseudomonas**
- **Enterobacter**

### Antibiogram of ESKAPE Isolates

- **AK**
- **GEN**
- **CAZ**
- **CPM**
- **CX**
- **CZ**
- **CIP**
- **PIT**
- **NIT**
- **LZ**
- **VA**
- **MRP**
- **AT**
### Table 3 Antibiotic use of ESKAPE pathogens

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Total Cases</th>
<th>AK</th>
<th>GEN</th>
<th>CAZ</th>
<th>CPM</th>
<th>CK</th>
<th>CZ</th>
<th>CIP</th>
<th>PIT</th>
<th>NIT</th>
<th>LZ</th>
<th>VA</th>
<th>MRP</th>
<th>AT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enterococci</td>
<td>64</td>
<td>64</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>64</td>
<td>49</td>
<td>29</td>
<td>12</td>
<td>0</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>Staph aureus</td>
<td>160</td>
<td>44</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>104</td>
<td>164</td>
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<td>50</td>
<td>08</td>
<td>0</td>
<td>0</td>
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</tr>
<tr>
<td>Klebsiella</td>
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<td>58</td>
<td>-</td>
<td>99</td>
<td>88</td>
<td>-</td>
<td>-</td>
<td>100</td>
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<td>-</td>
<td>03</td>
<td>0</td>
<td>0</td>
<td>09</td>
</tr>
<tr>
<td>Acinetobacter</td>
<td>47</td>
<td>24</td>
<td>-</td>
<td>31</td>
<td>29</td>
<td>-</td>
<td>-</td>
<td>32</td>
<td>08</td>
<td>-</td>
<td>03</td>
<td>0</td>
<td>0</td>
<td>09</td>
</tr>
<tr>
<td>Pseudomonas</td>
<td>34</td>
<td>10</td>
<td>-</td>
<td>21</td>
<td>17</td>
<td>-</td>
<td>-</td>
<td>27</td>
<td>07</td>
<td>-</td>
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<td>-</td>
<td>01</td>
<td>20</td>
</tr>
<tr>
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<td>-</td>
<td>00</td>
<td>00</td>
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<td>-</td>
<td>00</td>
<td>-</td>
<td>00</td>
<td>00</td>
</tr>
<tr>
<td>TOTAL</td>
<td>437</td>
<td>52</td>
<td>108</td>
<td>154</td>
<td>134</td>
<td>104</td>
<td>168</td>
<td>318</td>
<td>142</td>
<td>21</td>
<td>06</td>
<td>0</td>
<td>19</td>
<td>120</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(54.28%)</td>
<td>(24.71%)</td>
<td>(30.66%)</td>
<td>(23.64%)</td>
<td>(18.44%)</td>
<td>(72.76%)</td>
<td>(32.72%)</td>
<td>(04.83%)</td>
<td>(1.83%)</td>
<td>(4.34%)</td>
<td>(24.45%)</td>
<td></td>
</tr>
</tbody>
</table>

### Table 4 ESKAPE pathogen and Superbugs (MRSA, VRE and ESBL)

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Total Isolate</th>
<th>MRSA</th>
<th>MSSA</th>
<th>D test Positive IN MRSA</th>
<th>D test Positive IN NON MRSA</th>
<th>VRE</th>
<th>ESBL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enterococci</td>
<td>64</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>00</td>
<td>-</td>
</tr>
<tr>
<td>Staph aureus</td>
<td>160</td>
<td>104</td>
<td>56</td>
<td>21</td>
<td>04</td>
<td>00</td>
<td>-</td>
</tr>
<tr>
<td>Klebsiella</td>
<td>131</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>00</td>
<td>04</td>
</tr>
<tr>
<td>Acinetobacter</td>
<td>47</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>00</td>
<td>00</td>
</tr>
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<td>Pseudomonas</td>
<td>34</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>00</td>
<td>01</td>
</tr>
<tr>
<td>Enterococci</td>
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<td>-</td>
<td>-</td>
<td>-</td>
<td>04</td>
<td>00</td>
<td>05</td>
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<tr>
<td>TOTAL</td>
<td>437</td>
<td>104</td>
<td>56</td>
<td>21</td>
<td>04</td>
<td>00</td>
<td>05</td>
</tr>
</tbody>
</table>
FIGURE 1:
AUTOMATED BLOOD CULTURE MACHINE (BD BACTEC)

Figure 2: Mannitol salt agar
( Yellow colonies for Staphylococcus aureus and Pink colonies for CONS)

Figure 3: Chromogenic agar
a. Blue mucoid colonies: Klesiella spp.
b. Golden yellow colonies: Staphylococcus aureus
c. White colonies: Acinetobacter spp.
d. Pink colonies: Escherichia coli
e. Blue pinpoint colonies: Enterococcus spp.
f. White colonies: CONS

Figure 4: Detection of MRSA and ICR (negative).

Figure 5: Antimicrobial sensitivity testing on Muller Hinton Agar for GPC with ICR positive (D-test).
AN EPIDEMIOLOGICAL STUDY OF PDM09 INFLUENZA CASES - A RETROSPECTIVE STUDY

Dr. Himani Tak¹, Dr. Suman Bhansali², Dr. Afzal Hakim³, Dr. Harendra Bhakar⁴

ABSTRACT

Background and Aim - Seasonal influenza is an acute respiratory infection caused by influenza viruses which circulate in all parts of the world and can affect people in any age group. In August 2010 World Health Organisation (WHO) declared the halting of pandemic and H1N1 influenza virus is now considered as seasonal influenza virus. The present study was conducted with aim of observing epidemiology of disease.

Material and Methods - The records of 271 patients who were tested positive for swine flu in the period of Jan-17 to Feb-18 (14 months) at Dr. S. N. Medical College, Jodhpur were evaluated retrospectively to study the epidemiology.

Results - 271 patients were tested positive out of 1873 samples. 48.7% patients were male and 51.3% were females. Case fatality rate (CFR) was 18.5% for all cases, and in females it was 23%. The ages of the patients varied from 15 days to 85 years, but most commonly affected were in age group 20 to 59 (65.3%). Most deaths were observed in patients’ ≥60 years of age (CFR- 22.8%). 34% of patients who died had some form of co-morbidity associated. The peaks with case were observed in post monsoon and winter months corresponding to decrease in temperature.

Conclusion - The mortality and morbidity due to seasonal influenza is still high. Preventive measures should be engaged in high risk population during increased activity of disease.

Keywords - epidemiology, swine flu, H1N1, pdm09 influenza.

INTRODUCTION

Influenza is an acute respiratory infection caused by influenza viruses which circulate in all parts of the world. There are 3 types of influenza viruses which cause infection in humans, types A, B, and C. Influenza A and B viruses cause seasonal epidemics of disease.¹ Influenza affect all age groups; globally incidence is higher in young children and those above 65 years.²,³ Persons with co-morbid conditions; such as lung disease, heart disease, liver disease, kidney disease, blood disorders, Diabetes; pregnancy and immuno-compromised persons are at higher risk. The transmission is air borne from person-to-person, through droplets generated by the act of coughing, sneezing, hand-shaking, talking and laughing. The hallmark of influenza is the sudden, rapid onset of symptoms. Influenza symptoms may include fever, chills, body aches, sore throat, non-productive cough, runny nose and headache. Gastrointestinal symptoms and muscle inflammation occur more often in young children, and infants can present with a sepsis-like syndrome. Confirmation of seasonal influenza (including HI1N1) infection is through Real time polymerase chain reaction (RT-PCR).⁴
The WHO declared H1N1 as a pandemic on 11th June, 2009 originating in Mexico. The first laboratory-confirmed case was reported from Hyderabad in India on 16th May 2009. The pandemic was started in the western part of Rajasthan in August 2009 and lasted until April 2010. In August 2010 WHO declared H1N1 influenza virus as seasonal influenza virus. Since 2010 a total of 14,826 positive cases are reported from the state along with 1314 deaths. From Jodhpur alone 1,623 case and 221 deaths were reported.

AIMS AND OBJECTIVE
This study was conducted at Dr. Sampurnanad Medical College and attached hospitals, Jodhpur to discern the trends and epidemiology of seasonal influenza in January 2017-February 2018.

MATERIALS AND METHODS
The study was conducted retrospectively, and confirmed H1N1 cases were included from 14 months (January 17 to February 18). The samples (nasopharyngeal/ nasal/throat swab) were taken from suspected patients and are tested at microbiology department of our centre i.e. Dr. Sampurnanad Medical College, Jodhpur with RT-PCR technique. Nasopharyngeal swabs are preferred for isolation of large amount of virus infected cells. Infected persons are assumed to be shedding the virus from the day prior to onset of symptoms until resolution of fever. For intubated patients endotracheal secretions are collected. Specimens are placed in sterile viral transport media and are transported to laboratories in ice or cold packs at temperature of 4 degrees. The positive cases were described according to their demographic characteristics. Case fatality rates were also calculated. The data are collected in Microsoft-Excel sheets and represented as percentages and proportions in the form of tables and graphs.

RESULTS AND OBSERVATIONS
A total of 271 (14.4%) out of 1873 patients' samples were tested positive for H1N1 virus from January 2017 to February 2018. Out of 271 patients 132 (48.7%) were male and rest (51.3%) were female. There were 50 deaths reported in the same period, of which 18 (36%) were male and 32 (64%) were female. Case fatality rate for each sex i.e. male and female was 13.6% and 23% respectively. Patients were in both the extremes of ages. Majority of patients i.e. 177 (65.3%) belonged to age group 20-59 years. Most deaths, 38 (76%) occurred in ≥60 years age group with 22.8% case fatality rate. 116 (42.8%) patients belonged to rural background with rest (57.2%) belonging to urban area. Maximum number of patients i.e. 232 (85.6%) were recorded in the months September-17, December-17, ...
January-18 and February-18 when temperature dip was observed. Same was true for the deaths, i.e. 45 (90%) deaths were in the same months. Out of 50 patients who died, 17 (34%) had various co-morbid factors associated such as chronic heart, lung and liver diseases, severe anaemia, other infections (Dengue, Malarial parasite, HIV), type 2 Diabetes Mellitus, and pregnancy or pregnancy related complications).

Table - 1. Distribution of patients according to various socio-demographic factors (N=271)

<table>
<thead>
<tr>
<th>INDICATOR</th>
<th>TOTAL NO. OF PATIENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>GENDER</td>
<td></td>
</tr>
<tr>
<td>MALE</td>
<td>132 (48.7%)</td>
</tr>
<tr>
<td>FEMALE</td>
<td>139 (51.3%)</td>
</tr>
<tr>
<td>AGE GROUP</td>
<td></td>
</tr>
<tr>
<td>0 TO 5</td>
<td>41 (15.1%)</td>
</tr>
<tr>
<td>6 TO 10</td>
<td>10 (3.7%)</td>
</tr>
<tr>
<td>11 TO 19</td>
<td>8 (2.9%)</td>
</tr>
<tr>
<td>20 TO 59</td>
<td>177 (65.3%)</td>
</tr>
<tr>
<td>=60</td>
<td>35 (13%)</td>
</tr>
<tr>
<td>RESIDENCE</td>
<td></td>
</tr>
<tr>
<td>RURAL</td>
<td>116 (42.8%)</td>
</tr>
<tr>
<td>URBAN</td>
<td>155 (57.2%)</td>
</tr>
</tbody>
</table>

Table-2. Distribution of patients who died due to H1N1 according to gender, age and residence (N=50)

<table>
<thead>
<tr>
<th>INDICATOR</th>
<th>TOTAL NO. OF PATIENTS</th>
<th>CASE FATALITY RATE</th>
</tr>
</thead>
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<tr>
<td>GENDER</td>
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<td></td>
</tr>
<tr>
<td>MALE</td>
<td>18 (36%)</td>
<td>13.6%</td>
</tr>
<tr>
<td>FEMALE</td>
<td>32 (64%)</td>
<td>23%</td>
</tr>
<tr>
<td>AGE GROUP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 TO 5</td>
<td>2(4%)</td>
<td>4.9%</td>
</tr>
<tr>
<td>11 TO 19</td>
<td>2 (4%)</td>
<td>20%</td>
</tr>
<tr>
<td>20 TO 59</td>
<td>38 (76%)</td>
<td>21.5%</td>
</tr>
<tr>
<td>=60</td>
<td>8 (16%)</td>
<td>22.8%</td>
</tr>
<tr>
<td>RESIDENCE</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RURAL</td>
<td>22 (44%)</td>
<td>18.9%</td>
</tr>
<tr>
<td>URBAN</td>
<td>28 (56%)</td>
<td>18.0%</td>
</tr>
</tbody>
</table>

Table 3. Distribution of cases and deaths due to swine flu according to months

<table>
<thead>
<tr>
<th>Month</th>
<th>No. of cases (N=271)</th>
<th>No. of deaths (N=50)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jan-17</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Feb-17</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Mar-17</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Apr-17</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>May-17</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Jun-17</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Jul-17</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Aug-17</td>
<td>12</td>
<td>3</td>
</tr>
<tr>
<td>Sep-17</td>
<td>72</td>
<td>8</td>
</tr>
<tr>
<td>Oct-17</td>
<td>10</td>
<td>1</td>
</tr>
<tr>
<td>Nov-17</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>Dec-17</td>
<td>40</td>
<td>13</td>
</tr>
<tr>
<td>Jan-18</td>
<td>72</td>
<td>19</td>
</tr>
<tr>
<td>Feb-18</td>
<td>48</td>
<td>6</td>
</tr>
<tr>
<td>Total</td>
<td>271</td>
<td>50</td>
</tr>
</tbody>
</table>
DISCUSSION
Seasonal influenza viruses circulate worldwide and can affect people in any age group. In temperate climates, seasonal epidemics occur mainly during winter. It is a serious public health problem that causes severe illness and death in high-risk populations.\(^1\) In our country two peaks are observed, one mild peak post-monsoon and one much severe in winter months as shown in the figure 1. Severity of disease was more in months of Jan-Feb-18 as compared to Jan-Feb-17. This change corresponds with the change in recommendation of vaccine by WHO, which recommended replacement of the A/California/7/2009 (H1N1)pdm09-like virus component with an A/Michigan/45/2015 (H1N1)pdm09-like virus.\(^9\) For the duration Jan-17 to Feb-18, Rajasthan reported a total positive cases of 4,753 and 376 deaths due to swine flu, out of these 271 (5.7%) cases and 50 deaths (13.3%) were reported from Dr. S.N. Medical College, Jodhpur respectively. In a study conducted by Gelotar P. S. et al. in hospital-based swine flu cases in Gujarat, a total 246 were recorded positive for H1N1. Most numbers of these cases were from young adults (34.55%) in age group between >15-30 years and paediatrics (25.61%) in age group of <15 years. Male (56.91%) were infected more than female (43.09%). Infection rate was higher in urban population (64.22%) than rural population (35.78%).\(^10\) Total two peaks of infection were noticed. One was in rainy season & second was in winter season. In our study females patients were more than males (51.3% v/s 48.7%), most common age group affected was 20-59, and urban patients were more than rural. In the present study a case fatality of 18.5% was observed. In a similar study conducted by Singh M. et al. case fatality rate was found to be 14.7%, with CFR among males (16.5%) higher than among females (14.5%) and CFR was highest in 50 to 60 years age group. In contrast, the present studied showed a higher case fatality in females than males (23% v/s 13.6%) and highest in the geriatric age group (22.8% in ≥60 years of age).\(^5\) Co-morbidities such as chronic heart disease, chronic lung disease, diabetes, etc. were present in 34% patients who died, 3 of whom were pregnant and 1 in immediate post-
partum period. Similar findings were reported by Sharma V. et al. in a study conducted in a tertiary care institute where deaths due to swine flu were associated with co-morbidities.11

Conclusion-
There was marked increase in number of cases in Jan-Feb-18 as compared to Jan-Feb 17. This can be attributed to change of strain.12 Also temperature changes are responsible in increase in cases in winter months. Persons with co-morbid conditions are at increased risk for mortality, therefore preventive measures in these groups should be made available by the government at the beginning of season.

Conflict of interest- none.

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1. Influenza (Seasonal), Fact sheet, Reviewed January 2018 (http://www.who.int/mediacentre/factsheets/fs211/en/).
5. Epidemiology of Pandemic Influenza A (H1N1) 2009 in Western Rajasthan, India: A Retrospective Study. Dr. Mahendra Singh, Dr. Afzal Hakim, Dr. G. L. Saini, Dr. Suman Bhansali. Sch. J. App. Med. Sci., 2014; 2(18):142-146.
6. Seasonal Influenza (H1N1)–State/UT-wise, Year-wise number of cases and death from 2010 to 2018. (http://idsp.nic.in/showfile.php?id=3933).
INTRODUCTION

Discovery of Landsteiner ABO group system provided a major breakthrough in transfusion system. The four main blood groups validated till date are A, B, AB and O. The Subgroups in the ABO system result from polymorphism in the genes coding for ABO group. Two main subgroups of A group based on the Anti A1 lectin reactivity are A1 and A2. A1 reacts with anti A1 whereas A2 does not show any agglutination with anti A1 lectin. Frequency of A1 subgroup varies greatly in Indian population. Around 80% of blood group A and AB population are A1 and A1B respectively while the remaining 20% of the population are either A2 or A2B. Rarely few individuals with subgroup A either show very weak reactivity or no reactivity with A antisera. These individuals are categorized as weaker subgroups. These weaker subgroups sometimes pose problems in ABO grouping.

1 - Senior Professor, 2 - Senior Demonstrator, Department of Immunohematology and Blood Transfusion, Dr. S. N. Medical College, Jodhpur (Rajasthan) India
Corresponding Author : Dr. Manju Bohra
E-mail : manjubohra59@gmail.com Mobile No. : +91 9460803698 / 9782788879
A1 antibody appear as an atypical cold agglutinin, however it becomes clinically significant when react at 37°C and leads to destruction of A1 cells.6 Around 0.4% of A2 and 25% of A2B individuals show presence of these antibodies.7,8 These antibodies cause difficulty in interpretation of blood groups which may sometimes cause transfusion reaction.9 The present study aimed to find the prevalence of A1, A1B, A2 and A2B subgroups in Western Rajasthan and their clinical implications in transfusion medicine.

METHODS
This was a prospective cross sectional observational study, which enrolled blood donors for blood grouping from Jan 2017 to Jan 2018 at Umaid Hospital blood bank. The donor red cells were washed three times with 0.9% saline. For forward grouping, monoclonal antisera Anti A Anti B and anti D were used. One drop of 5% suspension of donor red blood cells was mixed with two drops of antisera and centrifuged for 1 minute at 1000 rpm. Test results were examined microscopically. For further sub classification of A and AB blood groups into A1, A2, A1B and A2B, Anti A1 lectin was used. Reverse grouping was performed using slide method. Pooled A, B and O cells were made. Interpretation of blood groups was done on the basis of agglutination reaction seen with both forward and reverse grouping.

STATISTICS
Data were entered in Microsoft excel sheet version 10. The proportions of data were calculated among different groups. The proportions of A1 and A2 were calculated out of A blood group while proportions of A1B and A2B were calculated out of AB blood group. The data were also presented graphically.

RESULTS
A total of 11,624 healthy donors were enrolled for the study. Out of total study sample, A antigen was present in 3486 donors (29.98%). Of these, 2444 (21.02%) belonged to A group and 1042 (8.9%) belong to AB group. A1 antigen was present in 2402 (98.28%) and A2 was present in 42 (1.72%). A1B and A2B was found in 956 (91.75%) and 86 (8.25%) donors respectively (Table 1; Figure 1, 2). 207 (5.94%) donors with A antigen were Rh negative. Of these 207 donors, 122 (58.94%) were A negative and 85 (41.06%) AB negative (Table 2). No weaker sub groups was found in the study. Blood groups other than A and AB was found in 8138 donors.

DISCUSSION:
ABO blood grouping system was first introduced by Karl Landsteiner in early 20th century.10 Since then it has played a major role in transfusion medicine. Heterogeneity in the genes coding for A gene results in formation of subgroups. A1 and A2 are the major subgroups of A group. These subgroups can result in disparities in ABO grouping. Individuals with A2 subgroup have specific anti A1 antibody. These antibodies do not react with A2 red cells however they recognize A1 red cells as foreign antigen and cross react with them.11 According to our study results the prevalence of A2 and A2B in Western Rajasthan population was 1.22% and 8.25%. These findings were similar to those seen in Japanese and black population where frequency of A2B is...
higher than A2 subgroup. Higher frequency of A2B in black population has been attributed to presence of strong B genes which subdue the activity of A1 antigen. In a study done in Karnataka by Shastri et al, the prevalence of A2B and A2 was found to be 10.5% and 1.85% respectively. Shastri et al. (2012) from Andhra Pradesh found 4.1% A2 and 19.2% A2B subgroup prevalence in the study conducted by them. Mehra Ruhi et al. (2016) from Maharashtra found prevalence of A2 and A2B 1.6% and 9.68% respectively while the prevalence of A1 and A1B was 98.3% and 9.32% respectively. In Hiroshima A2 individuals comprised 0.17% of A group population while A2B individuals were 1.14% of AB type population. In Nagasaki prevalence of A2 and A2B individuals was found to be 0.08% and 2.44% respectively. These findings closely correlated with our study findings. In our study 98.28% donors of A blood group belong to A1B while 8.5% donors belong to A2B subgroup. In a study conducted by Sharma et al in 2013 in Greater Gwalior region of India and Hassan in Sudanese population the prevalence of A2B was similar to that in our study however the prevalence of A2 was higher in both the studies being 8% and 14.10% respectively. The prevalence of A2 negative in present study was 0.14% and A2B negative was 0.25%. A1 negative was prevalent in 0.14% and A2B negative in 0.25%. In a study done by Sujata S. Giriyan et al, A2 negative was prevalent in 0.004% and A2B negative in 0.014%. This study helps to link Rh status of the individuals with A and AB subgroups.

CONCLUSION:
This is the first prevalence study of A subgroup conducted in Western Rajasthan population. The two major subgroups of A blood group are A1 and A2. These subgroups cause difficulties in interpretation of ABO grouping which may sometimes lead to lethal transfusion reaction. Hence, all A and AB blood group donors should be tested for anti A1 sera. It will improve overall performance of blood transfusion and also prevent transfusion reaction.

REFERENCE:
6. Boorman KE, Dodd BE, Loutit JF, Mollison PL. Some results of transfusion of blood to
recipients with cold agglutinin.


Figure 1 legends: Distribution of A1 and A2 subgroup among A group (%)

Figure 2 legends: Distribution of A1B and A2B subgroup among AB group (%)
### Table 1: Distribution of A and AB Subgroup Among A and AB Groups

<table>
<thead>
<tr>
<th>Blood Group</th>
<th>Number</th>
<th>Subgroup</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>2444</td>
<td>A1</td>
<td>2402</td>
<td>98.28%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>A2</td>
<td>42</td>
<td>1.72%</td>
</tr>
<tr>
<td>AB</td>
<td>1042</td>
<td>A1B</td>
<td>956</td>
<td>91.75%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>A2B</td>
<td>86</td>
<td>8.25%</td>
</tr>
<tr>
<td>Total</td>
<td>3486</td>
<td></td>
<td>3486</td>
<td></td>
</tr>
</tbody>
</table>

Values expressed as number and percentage

### Table 2: Distribution of A and AB Negative Subgroups

<table>
<thead>
<tr>
<th>Group</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1 Negative</td>
<td>117</td>
</tr>
<tr>
<td>A2 Negative</td>
<td>5</td>
</tr>
<tr>
<td>A1B Negative</td>
<td>76</td>
</tr>
<tr>
<td>A2B Negative</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>207</td>
</tr>
</tbody>
</table>

Values expressed as numbers
ABSTRACT

**Background**: Pre-eclampsia is a multiorgan disorder unique to pregnancy typically characterized by blood pressure ≥140/90 mmHg after 20 weeks gestation associated with proteinuria ≥300 mg/24 h or ≥1+dipstick. Preeclampsia and the other hypertensive disorders of pregnancy remain leading causes of maternal and perinatal morbidity and mortality, complicating 5 to 10 percent of all pregnancies and together they form one member of the deadly triad, along with haemorrhage and infection. Objective: To study maternal outcomes in terms of severity, complications of pre-eclampsia; and maternal mortality and foetal morbidity and mortality.

**Material and Methods**: This was a retrospective observational study over July 2018 to June 2019 at Mathura Das Mathur hospital, Dr S N Medical College, Jodhpur Rajasthan. The diagnosis of all cases was made on the basis of history, clinical examination and ultrasonography.

**Results**: Total 100 patients were analysed in this study. Majority of the women studied (67%) were in age group of 20-30 years; 72% were term and 28% were preterm; and most common mode of delivery was caesarean section (61%). Various maternal complications arising due to preeclampsia were also studied, and it was found that abruption was seen in 6%, HELLP/DIC was seen in 4% patients, eclampsia in 3%, pulmonary oedema in 2%, NICU admission in 9% whereas maternal mortality occurred in 1% of patients. Foetal outcome also observed in the form of prematurity, IUGR, Birth asphyxia and neonatal intensive care unit admission.

**Conclusion**: Pre-eclampsia is one of the most common medical complications of pregnancy associated with significant maternal and neonatal morbidity.

**Keywords**: Pre-eclampsia, Maternal and foetal outcome.

Introduction:

“Safe pregnancy is a beautiful voyage ending with birth of two lives But, if gets affected by preeclampsia, becomes a journey through a ring of fire”

Hypertensive disorders are among the most common medical disorders during pregnancy and continue to be the major cause of maternal and perinatal morbidity and mortality. Pre-eclampsia is a multiorgan disorder unique to pregnancy typically characterized by blood pressure ≥140/90 mmHg after 20 weeks gestation associated with proteinuria ≥300 mg/24 h or ≥1+dipstick.1,2 Pre-eclampsia and the other hypertensive disorders of pregnancy remain leading causes of maternal and perinatal morbidity and mortality, complicating 5 to 10 percent of all pregnancies and together they form one member of the deadly triad, along with haemorrhage and infection. Of these
disorders, preeclampsia syndrome is the most dangerous, affecting 3.9 percent of all pregnancies globally. The WHO reviews of maternal mortality worldwide suggest 16 percent maternal deaths in developed countries due to hypertensive disorders. Importantly, more than half of these hypertension-related deaths are preventable. India accounted for 19% of maternal deaths worldwide. Five percent of maternal deaths in India are due to hypertensive disorders.

Pre-eclampsia can affect virtually every organ system leading to thrombocytopenia, renal insufficiency, liver dysfunction, pulmonary edema, visual disturbances, cerebrovascular and cardiovascular complications, placental abruption, acute renal failure, disseminated intravascular coagulation, multiple organ system failures, postpartum haemorrhage and maternal death. Prematurity, intrauterine growth restriction, fetal distress, neonatal asphyxia, low birth weight, stillbirth and perinatal death are fetal and neonatal complications to be anticipated and dealt with when the mother has preeclampsia. Preeclampsia is a principal cause of fetal morbidity and mortality, also the leading reason of maternal ICU admissions, and responsible for 15–20% of maternal deaths worldwide.

Delivery of the infant and placenta is the only effective treatment. Delivery at an earlier gestational age, however, is associated with an increased risk of adverse neonatal outcome. Women with preeclampsia have an increased rate of cesarean section consequent upon the high incidence of intrauterine growth restriction, fetal distress, and prematurity. Cesarean section on the other hand increases the risk of cardiopulmonary morbidity associated with pre-eclampsia. This is due to the altered hemodynamics in women with pre-eclampsia, particularly in an emergent situation.

AIMS
1. To study the distribution pattern of pre-eclampsia among different group of patients with special reference to age, parity, booked/unbooked status and gestational age at presentation.
2. To study maternal outcomes in terms of severity, complications of pre-eclampsia, and maternal mortality.
3. To study foetal morbidity and mortality.

MATERIALS AND METHODS
This was a retrospective study done during one year period from July 2018 to June 2019 at Mathura Das Mathur hospital, Dr S N Medical college, Jodhpur, Rajasthan. This study was done by analysis of hospital records of patients admitted with diagnosis of pre-eclampsia after approval by the ethical committee of the institution. Blood pressure was measured using standard parameters with a proper size cuff with patient in sitting position. Proteinuria was measured using dipstick method and ≥1+ on at least two occasions was taken as positive. Inclusion criteria were patients with diagnosis of preeclampsia. Exclusion criteria were patients with chronic renal disease, systemic lupus...
erythematous, connective tissue disorder, molar pregnancy, thyroid disorder, and diabetes. All patients were evaluated for fetal and maternal condition using various biochemical and radiological test. Biochemical test includes complete blood count, liver function test, kidney function test, urine for proteins with dipstick/24 h urine collection, and fundus examination. Fetal state was assessed using ultrasonography and nonstress test wherever required. Patients were managed as per clinical protocols, progression of disease, and fetomaternal condition.

RESULTS:
A total of 100 patients were analysed in this study. Majority of the studied women (67%) were in mean age group of 20-30 years, with 14% below 20 years and 19% above 30 years. In our study, 62% were primigravida and 38% were multigravida; 72% were term and 28% were preterm. In 61% of patients mode of delivery was caesarean section and 39% were vaginally delivered. Out of the 100 babies 57% babies weigh >2500gm, and 32% babies weight between 1500-2500gm and 11% were lesser than 1500gm.

Various maternal complications arising due to preeclampsia were also studied, and it was found that abruption was seen in 6%, HELLP/DIC was seen in 4% patients, eclampsia in 3%, pulmonary oedema in 2%, NICU admission in 9% whereas maternal mortality occurred in 1% of patients.

In all cases, fetal outcome was also observed in the form of prematurity, IUGR, Birth asphyxia, neonatal intensive care unit admission. It was seen that prematurity was present in 28% of patients, 4% of babies had IUD/still birth, 16% of babies had birth asphyxia, 7% of babies had IUGR whereas NICU admission was seen in 12.5% of patients.

Discussion
Preeclampsia, which occurs only in the presence of placenta, continues to be a major cause of maternal and perinatal morbidity and mortality world-wide. We observed that chances of preeclampsia were significantly higher in younger age group (20-30 years). Similar results have also been obtained by various authors Yadav et al12 and Singh A et al13.

Nulliparity has also been found to be associated with pre-eclampsia. We also observed that pre-eclampsia occurred more frequently in primigravida (62%) as compare to multigravida (38%).

<table>
<thead>
<tr>
<th>Foetal outcome</th>
<th>Number</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prematurity</td>
<td>28</td>
<td>28%</td>
</tr>
<tr>
<td>IUD/Still birth</td>
<td>4</td>
<td>4%</td>
</tr>
<tr>
<td>IUGR</td>
<td>7</td>
<td>7%</td>
</tr>
<tr>
<td>Birth asphyxia</td>
<td>16</td>
<td>16%</td>
</tr>
<tr>
<td>NICU admission</td>
<td>21</td>
<td>21%</td>
</tr>
</tbody>
</table>

MATERNAL COMPLICATIONS

<table>
<thead>
<tr>
<th>Maternal Outcome</th>
<th>Number</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abruptio</td>
<td>6</td>
<td>6%</td>
</tr>
<tr>
<td>DIC/HELLP</td>
<td>9</td>
<td>9%</td>
</tr>
<tr>
<td>Eclampsia</td>
<td>3</td>
<td>3%</td>
</tr>
<tr>
<td>PPH</td>
<td>7</td>
<td>7%</td>
</tr>
<tr>
<td>ARF</td>
<td>3</td>
<td>3%</td>
</tr>
<tr>
<td>Pulmonary Oedema/ARDS</td>
<td>2</td>
<td>2%</td>
</tr>
<tr>
<td>ICU admission</td>
<td>9</td>
<td>9%</td>
</tr>
<tr>
<td>Mortality</td>
<td>1</td>
<td>1%</td>
</tr>
</tbody>
</table>
Sibai and Cunningham reviewed a number of worldwide studies and concluded that the incidence of pre-eclampsia in nulliparous populations was more than that for multiparous. It could be because of the failure of normal invasion of trophoblastic cells that leads to maladaptation of the spiral arterioles.

We also studied distribution of pre-eclamptic cases according to gestational age of presentation and severity. It was observed that most of the cases (72%) occurred at advanced gestational age (>37 weeks) and preterm in 38% cases. Similar results have also been observed by Sajith et al.

We also observed that 61 out of 100 (61%) patients were delivered by lower segment cesarean section (LSCS), and 39 patients were delivered by vaginally. Similar results have also been observed by Yelmizaitun et al. (2010). This finding was in contrast with other authors Chaim et al (2008) Yadav et al (1997) in India (14.8%). This difference in LSCS rate may be due to difference in medical facilities and quality of antenatal care (ANC) in different parts of the world.

This present study showed that among all partial HELLP and HELLP syndrome and DIC were the most common maternal complications seen in 9% of patients; whereas PPH in 7% and pulmonary edema each was seen in 2%; eclampsia developed in 3% and maternal mortality rate was 1%. Similar results have also been obtained by Murphy and Stirrat (2000). In their study, 21% had developed HELLP/ELLP syndrome, 1.4% had eclampsia. In another study conducted by Minire et al. (2013), lower complications rate were reported. In their study, 3.2% patients had eclampsia, 4.2% had HELLP syndrome, and 5.58% had pulmonary edema, whereas DIC was seen in 0.46% of patients. Higher complication rates in our study could be because of late patient presentation for medical care.

Prematurity was the most common complication associated with pre-eclampsia, which was seen in 28% cases. These results lower than observed by Tuffnell et al (65.3%) and Singhal et al. (66%). Birth Asphyxia was observed in 16% patients. Similar results were also observed by Singhal et al. (2009) who found the risk of birth asphyxia (21.43%). It could be because these studies were conducted in different countries where patient's socioeconomic background and medical facilities are better.

**CONCLUSION:**

“Prevention is Better Than Cure”

But this is not completely true for pre-eclampsia. Since it cannot be completely prevented, timely diagnosis of high-risk patients and prompt treatment in mild stage is the key to prevent complications. This study highlights various risk factors and complications both to mother and fetus for pre-eclampsia. Therefore, the study advocates more research into the field of pre-eclampsia to develop effective strategy for prediction and prevention of adverse outcomes. Routine screening for hypertensive diseases of pregnancy (HDP) based on measurement of blood pressure...
among all pregnant women should be practised as recommended by WHO and where resources are available, it is desirable to do urinary protein analysis at every antenatal visit as a complement to routine blood pressure measurement in remote areas of India like our western Rajasthan.

REFERENCES:

14. Sibai BM, Cunningham FG. Prevention of pre-eclampsia and eclampsia. In : Lindheimer MD, Roberts JM, Cunningham FG, editors. Chesley’s Hypertensive


ABSTRACT

Background - To evaluate the influence of post natal growth on Glycemic status and insulin resistance up to 9 months of age in LBW & VLBW babies. We compared these parameters with normal birth weight babies and these babies served as controls.

Methods - 100 successive low birth weight, (birth weight between 1000-2499 gms) were successively enrolled in the study and 50 neonates with birth weight >2.5 kg included as controls. Of the 150 enrolled babies only 101 babies could be followed till 9 months of age (in NBW group-36, in LBW group -35, in VLBW group-30 were followed up to nine months of age). At the time of enrolment anthropometry and routine blood investigation and fasting blood sugar level of neonates (after 2-4 hours fasting) was performed. After discharge, enrolled babies were followed every 3 monthly (+10 days) up to 9 months of age on each follow up anthropometry assessment and fasting blood sugar of neonates (after 2-4 hours fasting) was done. At the age of 9 months venous blood was taken for serum insulin levels (after 2-4 hours fasting). Statistical analyses were performed using t-test.

Results - Mean plasma Glucose and pondral index were at enrolment in NBW babies was higher than LBW and VLBW babies and mean P.Glucose levels at 9 month was higher in VLBW followed by LBW than in NBW (P value<0.01). Maximum S. Insulin level at 9 month found in VLBW babies followed by followed by LBW and NBW but statistically significant in LBW and VLBW in comparison to NBW. Maximum IRI at 9 month occurred in VLBW babies followed by LBW and NBW. When we compared weight gain at 9 month of age in three group these had been maximum weight gain in VLBW babies followed by LBW &NBW.

Conclusion - Low birth weight is an independent risk factor for insulin resistance and this can lead to deleterious effect in their later life in the form of development of diabetes, hypertension, coronary heart disease.

Key Words - Insulin resistance, Plasma Glucose, LBW & VLBW babies

INTRODUCTION

Type-2 diabetes poses a major health problem globally because it has reached epidemic proportion and affects more than 170 million individuals world wide. The WHO predicts a 40% increase in diabetes over the next 25 years in the industrialised world and a 170% increase in the developing world (1). The relationship between low birth weight and insulin resistance in adult life grew out of several UK population-based studies of middle-aged and older individuals living in areas where birth records were available from 1905 onward.

It was noted that the percentage of men aged 59-70 years who had IGT or type-2 diabetes on an oral glucose tolerance test (OGTT) fell progressively with increasing birth weight, from 40% of those who weighed 5.5 lb or less at birth to 14% of those who weighed 9.5 lb or more. After data were corrected for adult

* Senior Professor, ** Senior Registrar, *** Senior Prof. & Unit Head (Retd.)
Department of Pediatrics, Umaid Hospital, Dr. S.N. Medical College, Jodhpur (Raj.)
Correspondence Author - Dr Rakesh Jora E-mail - Jorarakesh@gmail.com
total obesity (BMI), men who were small babies were six times more likely to have type-2 diabetes or IGT (impaired glucose tolerance) than men who were large babies (2,3). There are several studies that LBW is a risk factor for type-2 diabetes; these studies mainly done in older children and adults, very few studies have been done in LBW and VLBW babies below one year that LBW and VLBW is a risk factor for type-2 diabetes in late life, depending upon their glycemic control and insulin resistance. So, we have planned a study to see a effect of growth on glycemic status and insulin resistance below one year of age in LBW, VLBW babies and compare with the normal babies.

**MATERIAL & METHODS**

The study was approved by the medical college institutional research review board. The Present study was a prospective study, in which we have included 150 newborns. These were further divided in three groups according to their weight. Each group had 50 neonates.

1. First group included neonates with birth weight -2500-3999 gms. (NBW), designated as a control group.
2. Second group neonates with birth weight - 1500 – 2499 gms (LBW)
3. Third group neonates with birth weight -1000 – 1499 gms (VLBW)

A written informed consent was obtained from the parents or guardian. The nature, purpose and possible risks of the study were explained to the parents in detail before consent was obtained. Of the 150 enrolled babies, only 101 babies could be followed till 9 months of age (in NBW group-36 , in LBW group -35 , in VLBW group-30 were followed up to nine months of age). At the time of enrolment all were examined thoroughly basic information complete history including family history and antenatal, natal and postnatal history was taken in all subjects. General Physical Examinations and systemic examinations was done in all subjects. Accurate assessment of gestational age of the enrolled babies was done by using the expanded new Ballard score. Anthropometry and routine blood investigation and fasting blood sugar level of neonates (after 2-4 hours fasting) was performed.

After discharge, enrolled babies were followed every 3 monthly (+10 days) up to 9 months of age on each follow up anthropometry assessment and fasting blood sugar of neonates (after 2-4 hours fasting) was done.

At the age of 9 months venous blood was taken for serum insulin levels (after 2-4 hours fasting) were done by Radio immuno assay of all enrolled babies and all the findings as well as lab parameters was recorded in pre-design Performa.

The statistical analysis was performed by using student’s “t” test and Chi square test to find out the significance of difference in mean between two variables.

**RESULTS**

Various clinical and biochemical indices in the three groups at baseline...
are shown in Table 1. Mean plasma Glucose in at enrolment in NBW babies was higher than LBW and VLBW babies (NBW 70.2±10.4, LBW 66.6±8.71, VLBW 62.4±7.82) and same as true for ponderal index these value were statistically significant (p <0.01).

Physical and biochemical characteristics at nine months had shown that there is statistically significant difference in mean serum insulin levels between NBW and LBW and between LBW and VLBW babies. Plasma glucose levels also have similar results between study groups (Table 2). Mean IRI in Study Groups at 9 months have statistically significant difference (Table 3).

Maximum rise of weight gain, plasma glucose, S.insulin level & IRI was seen with VLBW babies followed by LBW & NBW respectively and all parameters had statistically significant correlation between NBW&VLBW babies (p value 0.001) (Table 4).

Discussion

Most observational studies describing the association between infancy weight gain and later insulin resistance did not include repeated measurements during infancy and therefore could not identify a narrower critical period. Therefore, to understand the association of infancy weight gain with later insulin resistance we have planned a study to test the hypothesis that rapid weight gain in infancy is associated with insulin resistance.

This study shows that rapid weight gain specially in IUGR & premature babies increases insulin resistance and make them more prone to diabetes mellitus. Mean weight gain, plasma glucose levels and serum insulin levels at nine months of age were highest in VLBW followed by LBW and NBW babies and correlation between these parameters were statistically significant. These results have shown that lower the weight at birth and more the weight gain after that increases the chances of insulin resistance in later in life. Several studies have shown that a rapid weight gain in infancy is associated with the development of childhood obesity in populations of European, African and Asian ancestry. It is unclear, however, whether this association is present later in adulthood and which specific period of infancy is critical for long-term risk of obesity.

Whincup PH et al (4) studied the relation between size at birth (birth weight, thinness at birth) and concentrations of plasma glucose and serum insulin in children, and compared the associations with those occurring with measures of current childhood size. They suggested that reduced birth weight might be related to the early development of insulin resistance, but in contemporary children, obesity is a stronger determinant of insulin concentration and insulin resistance than size at birth131.

In animals, acceleration of neonatal growth is thought to increase the later propensity to insulin resistance and non-insulin dependent diabetes, whereas slow growth as a
consequence of undernutrition is thought to have a beneficial effect. Developing organisms have a compensatory increase in growth after a period of nutritional deficit (5). Although such growth is beneficial in the short term, it could have a profound adverse effect on the subsequent long-term life history of an organism.

Atul Singhal, Tim J Cole, Alan Lucas et al (6) studied the effect of fast early postnatal growth on later insulin resistance and their results suggest that relative undernutrition early in life in children born preterm may have beneficial effects on insulin resistance. Rolland – Cachera MF et al (7) reviewed the studies which investigated the association between early growth pattern and future metabolic risks. They found that rapid growth in early life in terms of rapid weight and length gain is associated with various health risks in later life like – obesity, cancer, cardiovascular diseases & diabetes. Moreover, the pattern of growth rather than absolute level of fatness seems to be of most important.

Improvement in neonatal care has dramatically increased survival among premature infants. The costs of neonatal and early health care for these infants are considerable but may be even more substantial if one considers that they appear to be at increased risk for disease in adulthood, with its attendant costs. Prematurely born children represent a relevant and increasing proportion of society. Although both premature infants and term infants who are small for gestational age are confronted with an adverse environment at a similar stage of biologic maturity, premature infants primarily fall an adverse postnatal environment, whereas term infants who are small for gestational age have experienced this adverse environment during intrauterine life. If this adverse environment is responsible for the reduction in insulin sensitivity observed in term SGA infants, than premature infants would have a similar, early and permanent reduction in insulin sensitivity.

The intrauterine environment and early postnatal life are now generally accepted as important determinants of the risk of disease in adulthood. Low birth weight, a marker of intrauterine adversity has consistently been associated with a variety of adult-onset diseases, including type-2 diabetes mellitus, essential hypertension, dyslipidemia, coronary artery disease and cerebrovascular accidents.

To date, almost all studies linking children whose birth weights were low to the propensity toward disease in adulthood have focused on those who were small for gestational age and born at term. Low birth weight, however, is even more prevalent among children born prematurely, most of whom are smaller at birth than term infants.

Paul L. Hofman, Fiona Regan et al (8) conducted a study to determine insulin resistance in prematurely born children and found both the premature
appropriate for-gestational age group and the premature small for gestational age group had elevated acute insulin release, with values that were approximately 50 percent higher than those in the term control group (P<0.001).

Various studies (9,10&11) suggest that inverse associations of size at birth with raised glucose levels in children and adults are consistently seen in population of European descent. The present study had shown that rapid weight gain specially in IUGR & premature babies increases insulin resistance and make them more prone to diabetes mellitus. Moreover low birth weight is an independent risk factor for insulin resistance and this can lead to deleterious effect in their later life in the form of development of diabetes, hypertension, coronary heart disease. So we should make parents of these babies aware of the fact that the normal growth is good for these babies not the rapid catch up growth. The study also have important message for the infant nutrition policies and support the guidelines of the pediatric societies that recommend slow growth in undernourished children and recommend exclusive breast feeding in early months of life.

References
Table 1: BASELINE CLINICAL AND BIOCHEMICAL CHARACTERISTICS IN THE STUDY GROUPS

<table>
<thead>
<tr>
<th>Indices</th>
<th>NBW(1) 50</th>
<th>LBW(2) 50</th>
<th>VLBW(3) 50</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight(kg)</td>
<td>2.950±0.24</td>
<td>2.048±0.23</td>
<td>1.373±0.06</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Height(cm)</td>
<td>48.29±1.3</td>
<td>44.81±2.24</td>
<td>40.96±2.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>H.C.(cm)</td>
<td>34.38±0.68</td>
<td>32.12±1.77</td>
<td>28.94±1.43</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PI</td>
<td>2.623±0.23</td>
<td>2.290±0.32</td>
<td>2.057±</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>P.Glucose</td>
<td>70.2±10.4</td>
<td>66.6±8.71</td>
<td>62.4±7.82</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Table 2: PHYSICAL AND BIOCHEMICAL CHARACTERISTICS AT NINE MONTHS

<table>
<thead>
<tr>
<th>Indices</th>
<th>NBW(1) 36</th>
<th>LBW(2) 35</th>
<th>VLBW(3) 30</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight(kg)</td>
<td>8.038±0.51</td>
<td>7.990±0.58</td>
<td>7.140±0.51</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Height(cm)</td>
<td>68.53±1.16</td>
<td>67.58±1.95</td>
<td>63.71±2.02</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>H.C.(cm)</td>
<td>44.29±1.06</td>
<td>42.71±1.26</td>
<td>41.08±1.57</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>C.C.(cm)</td>
<td>42.49±1.06</td>
<td>40.32±1.53</td>
<td>38.25±1.91</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>P.Glucose</td>
<td>79±8.30</td>
<td>86.6±8.62</td>
<td>93.2±8.99</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Mean S. insulin level(mIU/ml)</td>
<td>3.1±2.96</td>
<td>17.32±17.19</td>
<td>21.22±11.35</td>
<td>1 vs 2 &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1 vs 3 &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2 vs 3 &gt; 0.3</td>
</tr>
</tbody>
</table>
### TABLE 3 - MEAN IRI IN STUDY GROUPS AT 9 MONTHS WITH SIGNIFICANCE OF DIFFERENCE

<table>
<thead>
<tr>
<th>IRI</th>
<th>NBW(1) 36</th>
<th>LBW(2) 35</th>
<th>VLBW(3) 30</th>
<th>P VALUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-1</td>
<td>32(88.8%)</td>
<td>7(20%)</td>
<td>1(3.3%)</td>
<td>-</td>
</tr>
<tr>
<td>1-3</td>
<td>4(11.2%)</td>
<td>17(45.7%)</td>
<td>9(30%)</td>
<td>-</td>
</tr>
<tr>
<td>3-5</td>
<td>-</td>
<td>7(20%)</td>
<td>5(16.7%)</td>
<td>-</td>
</tr>
<tr>
<td>&gt;5</td>
<td>-</td>
<td>5(14.3%)</td>
<td>15(50%)</td>
<td>-</td>
</tr>
<tr>
<td>Mean IRI</td>
<td>0.59±0.57</td>
<td>3.70±3.69</td>
<td>4.38±2.71</td>
<td>1vs2&lt;0.001 1vs3&lt;0.001 2vs3&gt;0.2</td>
</tr>
</tbody>
</table>

### TABLE 4 - CORRELATION OF PARAMETERS AT 9 MONTH OF AGE IN STUDY GROUPS

<table>
<thead>
<tr>
<th>Parameters</th>
<th>NBW(1) 36</th>
<th>LBW(2) 35</th>
<th>VLBW(3) 30</th>
<th>P- Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Weight Gain (kg)</strong></td>
<td>5.086±0.54</td>
<td>5.580±0.53</td>
<td>5.780±0.50</td>
<td>1vs2&lt;0.001 1vs3&lt;0.001 2vs3&gt;0.3</td>
</tr>
<tr>
<td><strong>P.Glucose (mg/dl)</strong></td>
<td>79±8.30</td>
<td>86.6±8.62</td>
<td>93.2±8.99</td>
<td>1vs2&lt;0.001 1vs3&lt;0.001 2vs3&lt;0.01</td>
</tr>
<tr>
<td><strong>S.Insulin level (mIU/ml)</strong></td>
<td>3.1±2.96</td>
<td>17.32±17.19</td>
<td>21.22±11.35</td>
<td>1v2&lt;0.001 1vs3&lt;0.001 2vs3&gt;0.3</td>
</tr>
</tbody>
</table>
SUBHEPATIC PERFORATED APPENDICITIS WITH SUBHEPATIC CAECUM : A CASE REPORT

Dr. Ram Dayal¹, Dr S.S. Rathore², Dr. Amit Sharma³, Dr Naresh⁴

ABSTRACT
Appendicitis is one of the common problem in surgical practice. Position of appendix varies, a rare variant is sub hepatic appendix with sub hepatic caecum. It happens due to gut malrotation and fixation of gut. Appendicitis is rare occurrence in abnormal subhepatic location. Presentation mimics other surgical emergency conditions so its diagnosis and management is difficult. Here we present an 8-year female patient with pain right iliac fossa and right hypochondrium quadrant. The final diagnosis was perforated appendicitis with localised abscess at sub hepatic location.

INTRODUCTION
Appendix is present at junction of teniae coli, 2.5cm inferior to ileocecal valve. Malrotation and nonrotation of gut places appendix in mid abdomen is 4/100000. [2] Average length OF APPENDIX is 6-12 cm and breadth about 0.3 -1 cm and is covered by peritoneum. [3] Ileum opens on left wall of cecum with its right and left caecal frenulum and upper horizontal and lower concave lip. The appendix open in cecum with her valve of Gerlach. Sub hepatic caecum is due to lack of growth factor and unable to attach with abdominal wall.

CASE PRESENTATION
An 8 years old female child was admitted in emergency department with complaint of pain abdomen in a right iliac region and right hypochondrium. She had pulse 90 /min and 100 /60mm Hg blood pressure right arm supine. On per abdominal examination she had tenderness in right hypochondriac region and right iliac region without guarding and rigidity. Her haemoglobin was 12.0 gm/dL, total leucocyte cell count was 22190/uL with 81% neutrophills and platelets was 2,43,000/Ui. Her LFT, RFT were normal. Erect abdomen X-RAY and x ray chest were normal. In USG finding non-peristaltic tubular blind ended tubular structure with Diameter of 5-6 mm in sub hepatic region with edematous fat plane few enlarge lymph node and distended bowelloop in RIF.

After confirming the diagnosis the patient was taken for surgery. Informed consent, including risk, benefit and alternatives given to the patient and family & documented. Open appendicectomy through Mc Buney’s incision was performed Right iliac fossa was empty on exploration. Hence incision was extended as Gridiron incision. The Ruptured appendix was found near tip with 50 cc pus. Omentum was found adherent the inferior border of liver . ceacum was adherent to posterior abdominal wall and Right iliac fossa was empty. The appendicectomy was done and abdominal drain was placed. The specimen was sent for histopathological examination and it
revealed acute inflammation of appendix. Patient recovered well and discharged with no post operative complication after surgery.

DISCUSSION
Early fetal fixation and rotation of gut take place in normal position. The caecal decent is due to decrease size of right lobe of liver. The appendix has variable positions most common is retrocecal others are pelvic post-ileal, post-ileal and they have their own clinical presentation. Sub hepatic caecum with appendicitis is rare and incidence is about 6%. Perforation is common in children and elderly patients and its presentation is late than normal appendicitis. In children perforation is due to delayed diagnosis. Extremes of age risk of mortality increase due to pressure necrosis perforation leads to pelvic abscess or peritonitis. [4]

Liver abscess or cholecystitis are common misdiagnosis during abdominal ultrasound for subhepatic caecum with perforated appendix.[5]
We gave Mc Burney incision which was laterally converted in to Rutherford Morrison incision for subhepatic appendix. Now laparoscopy is a good option for these kind of patients because it has diagnostic and therapeutic benefit. [6,7]

CONCLUSIONS
Subhepatic location of appendix is uncommon but subhepatic cecum with perforated appendix is very rare. This unusual site of appendicitis is confused with other very common conditions like cholecystitis liver abscess and hepatic flexor mass. For early diagnosis and surgical intervention ultrasonography and laparoscopy are helpful tools.

REFERENCES
INTRODUCTION

Expectant women with mechanical heart valves need to undergo an event-free period of gestation with a successful outcome for both mother and child. Unfortunately, pregnancy in these patients does not follow its usual benign course and is associated with high maternal and foetal risks.

The incidence of rheumatic heart disease has declined dramatically in the industrialised nations. However, in India it has not shown a similar decline, with hospital admissions ranging from 16.5–50.6% of all cardiac in-patients. As rheumatic heart disease primarily affects younger patients, most women undergoing a heart valve replacement belong to the child-bearing age group and this potentially magnifies this problem in the developing countries.

During pregnancy there is a 50% increase in blood volume, an increase in cardiac output and a decrease in systemic vascular resistance (from uterine circulation and hormonal changes). During labour and delivery, cardiac output increases abruptly followed by a sudden increase in preload (due to autotransfusion of uterine blood and caval decompression). This increased haemodynamic burden, which is well tolerated by most women, may lead to cardiac decompensation in patients with prosthetic valves, especially those with left ventricular (LV)
dysfunction or a relatively small-sized valve prosthesis.

For patients with mechanical heart valve, lifelong anticoagulation is mandatory. However in pregnant women, anticoagulation management is a complex issue. Pregnancy is a hypercoagulable state, due to increase in fibrinogen, factors VII, VIII and X, von Willebrand factor; and relative decrease in protein S activity; stasis and venous hypertension. This further increases the already existing risk of thrombo-embolic complications (TEC) in these patients. This state of hypercoagulability extends into the postpartum period too and requires a persistently higher maintenance dose of warfarin. Similarly, increase in total blood volume affects the distribution of heparin and low molecular weight heparin (LMWH). The presence of placental heparinase further contributes to unpredictable changes in the quantum of medication required. Thus, optimal anticoagulation therapy is considered essential, but the appropriate choice of agent among the options available (warfarin, heparin or LMWH) is highly debatable.

RISK ASSESSMENT

PATIENT FACTORS

There is an increased haemodynamic load during pregnancy, labour and delivery. The published experience indicates that most patients that were asymptomatic or only mildly symptomatic before conception, tolerate this haemodynamic burden well. However, cardiac decompensation may occur, especially in patients with impaired LV function and/or possible patient-prosthesis mismatch. In addition, an increased incidence of arrhythmia is reported during pregnancy and may add to patient discomfort. Thus it is not surprising that decreased functional capacity, pulmonary oedema and death are not uncommon in pregnant women with mechanical valves. Patients with prosthetic heart valves and markedly impaired LV function that are moderately or severely symptomatic (New York Heart Association, class III and IV) are best advised against pregnancy.

Residual tricuspid incompetence often co-exists in patients with prosthetic heart valves. The reported incidence of foetal loss in mothers suffering from tricuspid incompetence severe enough to require diuretics is around 73%. This risk is significantly higher when compared with foetal loss in pregnancies in which the mother did not exhibit tricuspid incompetence.

PROSTHESIS RELATED FACTORS

The commonest cause of maternal death in patients with mechanical heart valves is the device thrombosis. In addition, there is also a high incidence of thromboembolic events in these patients, ranging from 7% to 23%.

DRUG THERAPY

Foetal complications related to maternal anticoagulant therapy are teratogenicity and foetal loss. The incidence of abortion or foetal wastage (resulting from retroplacental haemorrhage, congenital malformations, etc) in these patients is
high, with reported rates ranging between 23% and 50%.
Maternal risk of haemorrhage while on anticoagulation is estimated at around 2.5%, with majority of such episodes (almost 80%) occurring in association with delivery.

Moreover, in addition to anticoagulants, the use of other cardiovascular drugs during pregnancy may also adversely affect the foetal outcome. Cardiac drugs that are relatively safe during pregnancy include heparin, propranolol (and other beta blockers), verapamil, digoxin and few antihypertensives such as labetolol, methyldopa, hydralazine, nifedipine and prazosin. Amiodarone is associated with foetal hypothyroidism and intrauterine growth retardation. It should be reserved only for cases with refractory arrhythmias.

In these patients, a planned pregnancy is preferred to an unplanned one. Evaluation of pregnant women with prosthetic heart valves should include information about her pre-pregnancy functional capacity, ongoing drug treatment, a full clinical assessment, details of valvular prosthesis, an ECG, as well as an echo-Doppler study to evaluate cardiac status. A fairly good estimate of maternal and foetal risk can then be made. Patient should also be advised on the potential complications that may occur during pregnancy: symptomatic worsening, higher incidence of thromboembolism, and potential harmful effects to the foetus.

ANTI-COAGULANTS AND PREGNANCY
Choice of anticoagulant is limited to warfarin, heparin or LMWH. The advantage of warfarin lies in its ease of administration, dependability and low cost. However, the associated risk of embryopathy has limited its use in pregnant women, particularly in the first trimester. Heparins need to be administered parenterally and produce less dependable anticoagulation, but are not teratogenic.

WARFARIN
Oral anticoagulants interfere with the cyclic inter-conversion of vitamin K and its epoxide, thus inhibiting the production of vitamin K dependant clotting factors. Dosage is adjusted to attain a desired international normalized ratio (INR) level (Table 2), which is calculated by the formula:

\[ \text{INR} = \frac{\text{patient PT}}{\text{mean normal PT}} \times \text{ISI} \]

(PT stands for prothrombin time and ISI denotes International Sensitivity Index of thromboplastin used at the laboratory).

UN-FRACTIONATED HEPARIN
Measuring activated partial thromboplastin time (aPTT) remains the most frequently used method for monitoring the anticoagulant response of unfractionated heparin (UFH) and should be measured about 6 hours after the bolus dose, the continuous intravenous dose is adjusted accordingly. Long-term heparin therapy may cause osteoporosis.

LOW MOLECULAR WEIGHT HEPARIN
LMWH has a better bioavailability than unfractionated heparin, and may also have a lower risk of bleeding, thrombocytopaenia and osteoporosis. These advantages, however, are partly offset by its longer half life (making it more difficult to handle during premature labour), and its unpredictable reversal with protamine. As already mentioned, because of an increased volume of distribution of LMWH in pregnancy and placental heparinase, dose adjustments based on plasma anti-Xa levels 4 hours after the morning dose are essential. The target is to achieve an anti-Xa level of 1.0 to 1.2 units per ml.

**ASPIRIN**

Small-doses of aspirin are safe during the 2nd and 3rd trimesters of pregnancy. Aspirin reduces the incidence of systemic embolization or death when added to oral anticoagulation in the non-pregnant population with mechanical heart valve. Thus, based on current data, 80-100 mg of aspirin during the second and third trimesters may be added to improve antithrombotic effects.

**EMBRYOPATHY**

Heparin (both UFH and LMWH) does not cross the placenta, and does not cause teratogenicity. On the contrary, warfarin readily crosses the placenta. Vitamin K acts as a co-factor for carboxylation of glutamic acid residues of osteocalcin and matrix Gla protein, which modulate calcium deposition. Oral anticoagulants when used during the first trimester, may thus cause a failure in the synthesis of osteocalcin and Gla matrix protein resulting in nasal hypoplasia and stippling seen on X-ray of proximal epiphyseal growth areas (Chondroplasia punctata). Exposure during the second and third trimesters may lead to central nervous system and eye abnormalities (optic atrophy, cataract, blindness, microphthalmia, intraventricular haemorrhage, microcephaly, hydrocephalus, seizures, and growth/mental retardation).

Warfarin when used in the post-partum period does not cause an anticoagulant effect on the breast fed infant. Likewise neither UFH nor LMWHs are secreted into breast milk.

**FOETAL LOSS**

Spontaneous abortion is by far the most frequent foetal complication, associated with pregnancy in women with mechanical heart valves. Both oral anticoagulants and heparin carry this risk. Inhibition of the immature liver enzyme system of the foetus by warfarin may result into an increased risk of haemorrhagic complications and stillbirth. Although heparin will not cross the placenta, bleeding at the utero-placental junction is still possible. Replacing warfarin with heparin during the first trimester prevents the occurrence of malformations, but this does not translate into an improved pregnancy outcome.

**MODE OF DELIVERY**

Several studies suggest that heparin therapy is safe for the foetus particularly at the time of delivery, when trans-placental transfer of oral anticoagulants may lead to bleeding in the neonate. Caesarean section is indicated, if labour starts while the
mother is still on oral anticoagulants; rapid reversal of the mother's anticoagulation is attempted with liberal use of fresh frozen plasma. Avoiding vaginal delivery decreases birth trauma to the anticoagulated baby.

Recommendations on the management of elective deliveries, however, are more controversial. There are suggestions that continuing warfarin till 38 weeks of pregnancy, followed by a 2-day interruption of anticoagulant therapy and cesarean section may improve outcome and decrease the time for which the mother remains unprotected. This represents a non-obstetrical indication for cesarean section, which is itself known to increase venous thromboembolic risk over that of natural childbirth. Also, anticoagulated preterm infants are at risk for intracranial haemorrhage during both vaginal and caesarean delivery. Therefore, stopping warfarin at the 36th week, replacing it with adequate heparin and planned induction of labour at 38th week is a more appealing alternative, and is recommended by most authorities on this subject.

AVAILABLE RECOMMENDATIONS

As in most areas of medicine, management of pregnant patients with mechanical heart valves is now covered by guidelines. Yet, this controversial issue remains unabated, partly from the need of well-designed prospective studies and partly due to the lack of consensus among various study groups. In this regard, the Task Force of the European Society of Cardiology, (ESC) On the Management of Cardiovascular Diseases during Pregnancy in its expert consensus statement2 avers: “In pregnant patients with mechanical prosthesis, the choice of anticoagulant therapy during the first trimester should take into account the greater thromboembolic risk with heparin and the risk of embryopathy with vitamin K antagonist. The use of vitamin K antagonist during the first trimester is the safest regimen for the mother.”

REFERENCES

1. American College of Cardiology; American Heart Association Task Force on Practice Guidelines (Writing Committee to revise the 1998 guidelines for the management of patients with valvular heart disease); Society of Cardiovascular Anesthesiologists;
As LMWH is not approved for use in prosthetic valve patients in pregnancy due to the high risk of thrombosis, and as subcutaneous unfractionated heparin throughout pregnancy carries a similar high risk, strategies which should be discussed with patients are:

(a) Heparin during the 1st trimester (to avoid warfarin embryopathy), followed by oral anticoagulation up to the 36th week with subsequent replacement by heparin until delivery.

(b) Oral anticoagulation throughout pregnancy, until the 36th week, followed by heparin until delivery.

Because of high rate of maternal complications with heparin therapy, particularly when given throughout the pregnancy, the committee strongly recommends strategy (b).

LMWH: Low Molecular Weight Heparin

Table 4. ACC/AHA 2006 Guidelines for the Management of Patients With Valvular Heart Disease. Selection of Anticoagulation Regimen in Pregnant Patients With Mechanical Prosthetic Valves

| Class I | All pregnant patients with mechanical prosthetic valves must receive continuous therapeutic anticoagulation with frequent monitoring. (Level of Evidence: B) |
| Class IIa | In patients with mechanical prosthetic valves, it is reasonable to avoid warfarin between weeks 6 and 12 of gestation showing to the high risk of foetal defects. (Level of Evidence: C) |
| Class III | LMWH should not be administered to pregnant patients with mechanical prosthetic valves unless anti-Xa levels are monitored 4 to 6 h after administration. (Level of Evidence: C) |

Class I: Conditions for which there is evidence for and/or general agreement that the procedure or treatment is beneficial, useful, and effective. Class II: Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment. Class IIa: Weight of evidence/opinion is in favor of usefulness/efficacy. Class III: Conditions for which there is evidence and/or general agreement that the procedure/treatment is not useful/efficacious and in some cases may be harmful. Weight of evidence in support of the recommendation is listed as follows: Level of Evidence A: Data derived from multiple randomized clinical trials. Level of Evidence B: Data derived from a single randomized trial or nonrandomized studies. Level of Evidence C: Only consensus opinion of experts, case studies, or standard-of-care. ACC: American College of Cardiology, AHA: American Heart Association, UFH: unfractionated heparin, LMWH: low molecular weight heparin, aPTT: activated partial thromboplastin time, INR: international normalized ratio.
One of the most significant demographic trends of the 20th century was what has been described as “Greying of Population”. Of all the people who have ever lived to age 65, more than two thirds are currently alive. Advances in medical and surgical therapy over the past three decades have increased the longevity of life. Fungal infections have increasingly become a problem among older adults as increasing longevity and affluence have enabled retirees to travel and participate in outdoor activities. Opportunistic fungal infections have increased because older patients are now receiving transplanted solid organs or bone marrow, undergoing aggressive treatment of malignancies, and taking immunosuppressive medications for dermatologic and rheumatologic diseases. Newer technology and therapies result in immune compromised individuals. Use of invasive monitoring devices, parenteral nutrition, broad spectrum antimicrobial agents and assisted ventilation have helped to treat patients suffering, provides life to non-viable but in turn results in proliferation of nosocomial invasive fungal infection in severely ill, immunocompromised, hospitalized patients, with fungi which were previously considered of low virulence. Older patients are also less able to handle invasive endemic or opportunistic infections, and outcomes of infection are frequently worse for patients who are elderly if not recognized and cared early.

Fungi are remarkable organisms. They are eukaryotic, rank in animal rather than in the plant kingdom. Aspergillus and Candida are found everywhere on earth. In past candida was known as Monilia (Monilia albicans and Odium albicans) the term first used by John Hill in 1751 while in 1890 Zopf termed it Monilia albicans to the fungus causing thrush in humans. Pathogenic fungi may be divided on the basis of their pathogenicity into true pathogens and opportunistic pathogens. While Candida albicans is the major pathogen most commonly isolated from ICU patients, other species of Candida include C. glabrata, C. parapsilosis, C. tropicalis, C. kruzi etc. Apart from Candida species and Aspergillus the other fungi which are frequently isolated from ICU patients include Fusarium, Mucor, Rhizopus, Penicillium, Trichosporon, Cryptococcus sps. etc. Data on the burden of opportunistic mycoses in India is not clear though the climate in this country is well suited for a wide
variety of fungal infections; yet a definite rising trend has been noted. The major factors which increase the risk of developing severe fungal infections in ICU patients include major abdominal surgery, perforation of G.I. tract, broad spectrum antibiotics, candida colonization, immunosuppression, burns, total parenteral nutrition, mechanical ventilation, central venous lines and other invasive procedures. To improve and optimize the level of predictive validity and specificity of tests analyzing colonization, two indices namely Candida Colonization Index [CCI] and Corrected Candida Colonization Index [CCCI] have been developed. In candidiasis, prevalence of disease depends on various factors. Candida reservoir is endogenous, as Candida albicans colonizes mucosa of G.I. tract and Vagina. Alteration in host-defence mechanism associated with altered intestinal barrier function and decreased immunity characterizes critically ill patients and results in life threatening fungal infections. Use of broad spectrum antibiotics eliminates bacteria of G.I. tract which confer colonal protection. After colonization, there is proliferation of organisms followed by translocation and invasion of mucosal barrier. This is followed by a period of inapparent candidaemia and finally invasive candidiasis and pathological manifestations of invasiveness. Thus, colonization is an independent risk factor and also a prerequisite for candida infection. This has been proved in neutropenic and nonneutropenic critically ill patients. Signs and symptoms of systemic candidiasis are variable and nonspecific. Clinical presentation varies from fever to septic shock. Other manifestations include skin nodules, myopathy, endophthalmitis, meningitis etc. Although the usual presentation is an unremitting fever that fails to respond to standard antibiotics within 96 hours of initiation of therapy.

It is essential to understand the host defence processes involved in initiating immune responses at mucosal surfaces and how it discriminates between the “commensal” and “pathogenic” states of this fungus. Nearly all studies investigating the epithelial response to C. albicans utilise cytokine and chemokine production as the sole read-out mechanism. We and others have shown that infected ECs produce cytokines/chemokines with a proinflammatory profile, including IL-1α/β, IL-6, G-CSF, GM-CSF and TNF α as well as the chemokine RANTES, IL-8 and CCL20. Infection of epithelial cells by C. albicans results in the production of cytokines and chemokines which recruit and activate various other immune cells. The best documented of these networks is initiated by IL-8. IL-8 recruits circulating neutrophils (PMNs) that are then activated by a variety of cytokines including GM-CSF, G-CSF and IL-1 family members. Activated PMNs then produce TNF α which then affect epithelial gene transcription. TGF β is produced constitutively by epithelial cells and will act with IL-1β and IL-6 to induce T
cell differentiation to the Th17 phenotype. Mucosal homing cells including Th17 T cells and activating dendritic cells will also be recruited by the increased expression of CCL20 and β-defensin 2, acting through the CCR6 receptor. This will lead to the presence of active Th17 T cells in the region to combat the fungal infection. Finally, infection of epithelial cells leads to the production of IL-20 family cytokines including IL-19, IL-20 and IL-24. These cytokines will function in an autocrine fashion, although their role in fungal immunity is not fully understood.

Amongst these host factors could be local and/or systemic. Wearing dentures, inadequate care of appliances, disturbed oral microbial flora due to long term use of antibiotics or corticosteroids, change in dietary factors and diseases like oral cancers or xerostomia are few important local factors. While extremes of age, chronic smoking, immunosuppression, nutritional deficiency, long term use of systemic antibiotics or diseases like diabetes mellitus, Cushing's syndrome, malignancies, severe blood dyscrasias, radiation to head and neck, oral epithelial dysplasia are systemic factors.

People are living longer, and older people are more likely to have compromised immune systems, a major risk factor for fungal infection. Similarly, the widespread use of broad spectrum antibiotics has contributed to the growing infection rate as fungal infections are known to occur after antibiotic therapy, which has the effect of killing the normal commensal bacteria. The global change in spectrum of candida species is also observed in India; cases with non-albicans Candida especially with C. tropicalis are commonly seen. Candida infections can spread to vulnerable people with depressed immune systems who are in the hospital, where the fungus is commonly found on the hands of caregivers and where indwelling catheters, central IV line, can allow an infection to take hold. Infection rates in intensive care units (ICUs) have been documented to be the highest of all hospital acquired infections in large multicentre studies. This is related to the use of large numbers of invasive monitoring devices, endotracheal and tracheostomy tubes; patient factors including extremes of age, immunocompromised state, malnutrition and severe underlying disease; and to a high incidence of cross infection. Bloodstream infections cause substantial morbidity and mortality. Increasing rates of antimicrobial resistance, changing patterns of antimicrobial usage, and the wide application of new medical technologies (e.g., indwelling catheters and other devices) may change the epidemiology and outcome of bloodstream infections. It is therefore important to continually review and update the epidemiology. These infections are costly to treat, prolong ICU stay and increase mortality rates. The three most common nosocomial infections are ventilator-associated pneumonias,
urinary tract infections and bloodstream infections.
Infection of skin, mucous membranes and nails by endogenous Candida can be caused by conditions that result in chronic maceration of these areas, physiologic changes in the host or a compromised immune status. Patients with chronic mucocutaneous candidiasis are generally immunocompromised adults undergoing treatment with steroids, cytotoxic drugs or antibacterial antibiotics. Oral candidiasis is an opportunistic infection of the oral cavity which is caused by an overgrowth or infection of the oral cavity by a yeast-like fungus, candida and often referred to as thrush. The lesions may be singular, patchy or confluent and a whitish pseudo membrane composed of yeasts and pseudohyphae may cover the tongue, soft palate and oral mucosa. There are various strains of oral candidiasis but the important ones are C. albicans, C. tropicalis, C. glabrata, C. pseudotropicalis, C. guillierimondii, C. krusei, C. lusitaniae, C. parapsilosis and C. stellatoidea. C. albicans, C. glabrata, and C. tropicalis represent more than 80% of isolates from clinical infection. Incidence of oral candidiasis is 54-93% in HIV positive patients with history of chronic smoking. CR-3 like protein enhances adherence of candida with epithelial cells in HIV positive patients. Vaginal thrush, patches of grey-white pseudo membrane develop on vaginal mucosa and a yellow-white discharge may accompany the infection especially in elderly diabetic females or those who are on hormone therapy.

Invasion of nail plate by fungus is known as onchomycosis, which is derived from the Greek word “onyx”, a nail and “mykes” a fungus. There is considerable difference in prevalence of onchomycosis in various geographical areas. In India, Pakistan, Korea, Canada and UK dermatophytes are major causative pathogens while Yeasts are frequently reported in Spain, Italy, Saudi Arabia and Iran. Saprophyte moulds are common cause of toenail infections. Onchomycosis are usually caused by dermatophytes and rarely by Candida or some saprophytic fungi. Patients who are diabetic, in extremes of age, having hyperhydrosis or frequent trauma suffers with onchomycosis frequently. Onchomycosis is being viewed as cosmetic problem, more prone to have secondary bacterial infections, cellulitis, idiopathic reaction and chronic urticaria. Infected toenail in elderly population may act as reservoir for fungus and in female patients social and emotional impairments are seen. Mycotic keratitis manifested as corneal ulcer or hypopyon or both, precipitated due to corneal trauma, tear deficiency, lid abnormality or chronic dacrocystitis, are commonly seen in elderly population. The mycosis has emerged into prominence since the advent of antibacterial antibiotics and the use of steroid ointment as they tend to enhance mycotic activity if spores of fungi are present on abraded cornea or in eye, surgical intervention or
antibacterial medication has altered normal host response. Fusarium solani, Aspergillus and Candida sp. are commonest causative agents. Most strains of F. solani are isolated from plants; they multiply rapidly at 37°C and survive at 40°C and causes mycotic keratitis in elderly due to trauma by vegetative plants.

Though Aspergillus is responsible for pulmonary aspergillosis which is typical invasive form with eventual haematogenous dissemination to other organs commonly caused by A. flavus, A. niger, A. clavus, A. terreus while A. niger and A. fumigates may colonise an ectatic bronchus, without invasion of lung parenchyma, forming a compact “fungus ball” known as Aspergilloma. In elderly Aspergillus can also lead to chronic fibrosing granulomatous reaction, allergic reactions as allergic asthema, allergic bronchopulmonary aspergillosis, sinusitis and otomycosis, CNS, sino-orbital infection or GIT-infarction (rarely). Mucormycosis are commonly seen in elderly with endocrinopathies (e.g. uncontrolled diabetes).

Candida biofilm is difficult to treat as they show resistance to antifungal treatment through slow penetration of drugs through the bio film, expression of resistance genes and presence of persister cells. Antifungal drug resistance especially with fluconazole, Amphotericin- B and newer triazoles are rapidly becoming major therapeutic challenge in immunocompromised patients. Newer antifungal agents, such as the echinocandins and liposomal formulations of Amphotericin B, have shown increased activity against candida bio films.

Numerous systemic manifestation of candidiasis may follow introduction of Candida into the blood stream. Candidaemia may result from contamination of indwelling catheters, surgical procedures, and trauma to the skin or gastrointestinal tract. The extent and severity of the infection is determined by the inoculum size and virulence of organism and most importantly the host defences. Clinical indications of acute systemic candidiasis include candiduria (in the absence of catheterization and an imbalanced flora), candida endophthalmitis and maculonodular skin lesions. Although Candida albicans is the most common agent of candidiasis, C. guilliermondii, C. parapsilosis and C. tropicalis are frequent causes of endocarditis. Prophylaxis is considered for a selected group of patients in whom the frequency of Candidaemia is high enough to make such treatment beneficial. On the other hand, presumptive antifungal therapy should be given to individuals with well known risk factors and a known degree of Candida colonization.

Currently, active and extensive investigation is focused on preventive strategies for invasive fungal infections. Fungal infections are common in elderly patients admitted to intensive care unit due to impaired immunity and presence of multiple risk factors. These infections have a profound effect on morbidity and mortality.
rates of ICU patients. Appropriate interventions must be undertaken to hasten the recovery of patients and to decrease the health care cost. More than 20% of Candidaemia cases are associated with increased morbidity and mortality rates. Persons at extremes of age are most susceptible to fungal colonization. Because of their dependency in personal hygiene, dentures responsible for mucocutaneous infection, vulnerability for getting injured while outdoors due to diminished vision because of age related ocular changes, along with co morbid diseases of old age. Physical dependency becomes more relevant for the institutionalized or admitted elders who may find it difficult to get assistance from the caretakers. Use of invasive monitoring devices, parenteral nutrition, broad spectrum antimicrobial agents and assisted ventilation have helped to treat patients' suffering, provides life to non-viable elderly but in turn results in proliferation of severely ill, immune-compromised, hospitalized patients which are at higher risk of acquiring nosocomial fungal infections which were previously considered of low virulence. Fungal infection in these patients is often severe, rapidly progressive and difficult to diagnose or manage. While Candida albicans species are the most common cause of severe fungal infections in ICU, the incidence of candida non-albicans and Aspergillus infection are rapidly rising due to the increased spectrum of patients at risk for developing mould infections. These fungal infections increase both morbidity and mortality to a significant extent in ICU patients and also prolong the duration of stay in ICU and increase health care cost. Candidaemia means isolation of Candida species in the blood and candidiasis means tissue invasion demonstrated by culture or histology at nonadjacent, normally sterile sites. Proven invasive Candida infections include Candidaemia and histological evidence of tissue invasion and positive Candida blood culture from sterile site. In the United States, Candida is reported to be the fourth leading organism responsible for nosocomial blood stream infections, accounting for 10-20% of infections in ICU patients. There are very few data available regarding the incidence of fungal infections in ICU patients in India, but the overall incidence is said to range from 10 to 16%.

For laboratory diagnosis of fungal infections, it is must that contamination of samples should be avoided, sterile instruments and containers should be used and appropriate sample should be ordered for a particular suspected disease. Commonly used samples are scrapings, fluids sp.CSF, BAL, pus, sputum and biopsy or blood culture to diagnose fungemia. As far as possible, swabs should be avoided as these are the poorest way to collect samples. The diagnosis of invasive candidiasis is still based on blood cultures in a substantial proportion of cases, a single positive blood culture being considered enough for a definitive diagnosis of candidaemia.
and invasive candidiasis. Unfortunately, blood cultures have quite a low sensitivity value ranging between 50 to 60%, other serological methods for the detection of candida infection include (1-3)-B-D-Glucan, Mannan, D-Arabinitol, Enolase, Galactomannan detection. Germ tube test (for C.albicans confirmation), antibody or antigen detection by Polymerase chain reaction or Latex agglutination, positron emission testing scan are the newer methods used in the diagnosis of fungal infections. For diagnosis of Cryptococcal infections, Indian Ink staining along with detection of antigen in CSF or blood are confirmatory diagnostic techniques along with culture on Niger Seed Agar medium and rapid urease test positive (within 4 hours). Nowadays automated identification system like Vitek-2 helps in early identification of yeasts, along with antimycotic sensitivity testing and their MIC can be known, which combats problem of drug resistance. While in Aspergillus infection diagnosis depends upon serological or molecular techniques or high resolution CAT scan. All dermatophytes are diagnosed by 10%KOH preparation of skin, hair or nail (40%KOH) and culture is done on Sabouraud's Dextrose Agar medium (selective media for fungus culture) or dermatophyte selective agar for identification.

Among the available antifungal drugs,azole group of drugs like fluconazole, voriconazole, posaconazole are commonly used. Amphotericin-B is reserved for severe systemic infections only, owing to its high nephrotoxicity potential and other systemic side effects. Echinocandins are a new class of drugs with improved spectrum of activity and less side effect profile. But they are available in parenteral form only for administration. Due to irrational use of antifungal drugs which is associated with emergence of azole-resistant strains of Candida and also the changes in candida species i.e. increase in candida non-albicans strains compared to candida albicans strains, they must be avoided.

Prophylaxis is considered for a selected group of patients in whom the frequency of candidaemia is high enough to make such treatment beneficial. On the other hand, presumptive antifungal therapy should be given to individuals with well known risk factors and a known degree of candida colonization. Currently, active and extensive investigation is focused on preventive strategies for invasive fungal infections. Prophylaxis with antifungal drugs has been demonstrated to reduce the incidence of invasive fungal infections in immunocompromised patients specially with CD-4count <200cu/mm, nonneutropenic patients, but their role in immunocompetent, nonneutropenic patients is less well defined. Clinically useful antibiotics include Amphotericin B, nystatin, griseofulvin and the azole antifungals. Invasive fungal infections are not only observed in immunocompromised hosts, but they are increasingly recognized as a growing problem in
critically ill nonimmunocompromised patients and in patients undergoing major surgical procedures. Cases of drug resistance are commonly seen, hence antifungal sensitivity test is recommended by E-Test method or disc diffusion method. Strictly all the following recommendations should be followed by ICU workers and doctors to minimise hospital acquired fungal infections in elders:

a) Hand hygiene continues to be corner stone of infection control practices
b) Judicious use of antibiotics and steroids
c) Central venous lines should be changed within 15 days
d) Minimize stay in ICU
e) Minimize civil work near OT and ICU
f) Antifungal prophylaxis should be given to high risk elderly patients
g) Avoid food that will feed the mutated Candida (A diet that is high in sugar and refined carbohydrates speeds up the process of the mutation in candida).

Fungal infection whether in ICU settings or environmentally acquired in geriatric patients is an important problem in Indian hospitals. Diagnostic delays could be shortened by more active screening for fungus especially in intensive care settings. Undiagnosed fungal infections may increase both the morbidity and mortality rates to a significant extent and increase the burden on health system in terms of cost and manpower. Correct diagnosis and prompt treatment with antifungal drugs remains a key factor in managing such patients.

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Types of manuscripts

Original Articles (Total number of words including References should be less than 3000)

These include original research work in all fields of medical sciences and bio-allied sciences. The article file should be accompanied by a structured abstract of no more than 250 words under the following headings: 1. Background and Aims 2. Methods (make a brief mention of statistical methods used) 3. Results and 4. Conclusion. The abstract should be followed by ‘MeSH’ compatible 3-7 keywords (https://www.nlm.nih.gov/mesh). The abstract shall not contain references.

The main article file should be written under only the following four headings: Introduction, Methods, Results, Discussion and Conclusion.

Introduction: State the purpose and summarize the rationale for the study or observation. The introduction should describe in brief the background related to the study and also the need for carrying out the present study. Limit the number of references cited in the introduction to 4-6 only. Please include aims and objectives in introduction itself.

Methods: It should include and describe the following aspects (do not use the headings in the manuscript /article):

Ethics:

When reporting studies on human beings, indicate whether the procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional or regional) and with the Helsinki Declaration of 1975, as revised in 2013 (available at http://www.wma.net/en/30publications/10policies/b3/index.html). For prospective studies involving human participants, authors are expected to mention about approval of (regional/ national/ institutional or independent Ethics Committee or Review Board, obtaining informed consent from adult research participants and obtaining assent for children aged over 7 years participating in the trial. The age beyond which assent would be required could vary as per regional and/ or national guidelines. Ensure confidentiality of subjects by desisting from mentioning participants’ names, initials or hospital numbers, especially in illustrative material. When reporting experiments on animals, indicate whether the institution’s or a national research council’s guide for, or any national law on the care and use of laboratory animals was followed.

Evidence for approval by a local Ethics Committee (for both human as well as animal studies) must be supplied by the authors on demand. Animal experimental procedures should be as humane as possible. The ethical standards of experiments must be in accordance with the guidelines provided by the CPCSEA and World Medical Association Declaration of Helsinki on Ethical Principles for Medical Research Involving Humans for studies involving experimental animals and human beings, respectively). The journal will not consider any paper which is ethically unacceptable. A statement on ethics committee permission and ethical practices must be included in all research articles under the ‘Methods’ section.

Study design:
The methods section should start out describing the nature of the study (randomized / blinded / prospective / retrospective, etc). Selection and Description of Participants: Describe your selection of the observational or experimental participants (patients or laboratory animals, including controls) clearly, including eligibility and exclusion criteria and a description of the source population. Technical information: Identify the methods, apparatus (give the manufacturer's name and address in parentheses), and procedures in sufficient detail to allow other workers to reproduce the results. Give references to established methods, including statistical methods; provide references and brief descriptions for methods that have been published but are not well known; describe new or substantially modified methods, give reasons for using them, and evaluate their limitations. Identify precisely all drugs and chemicals used, including generic name(s), dose(s), and route(s) of administration.

Reports of randomized clinical trials should present information on all major study elements, including the protocol, assignment of interventions (methods of randomization, concealment of allocation to treatment groups), and the method of masking (blinding), based on the CONSORT Statement (http://www.consort-statement.org).

Statistics:

Start this section in a separate paragraph (without placing the heading “statistics”). Whenever possible quantify findings and present them with appropriate indicators of measurement error or uncertainty (such as confidence intervals). Authors should report losses to observation (such as dropouts from a clinical trial). When data are summarized in the Results section, specify the statistical methods used to analyze them. Avoid non-technical uses of technical terms in statistics, such as ‘random’ (which implies a randomizing device), ‘normal’, ‘significant’, ‘correlations’, and ‘sample’. Define statistical terms, abbreviations, and most symbols. Specify the computer software used with the version/year. Use upper italics (P 0.048). For all P values include the exact value and not less than 0.05 or 0.001. P values are not needed for demographics routinely and are mentioned where study involves directly a correlation of study parameter with the demographics. Mean differences in continuous variables, proportions in categorical variables and relative risks including odds ratios and hazard ratios should be accompanied by their confidence intervals. Statistics related to sample size calculation and power estimation should be provided as the last paragraph in the methods section.

Results:

Present your results in a logical sequence in the text, tables, and illustrations, giving the main or most important findings first. Do not repeat in the text all the data in the tables or illustrations; emphasize or summarize only important observations. Extra- or supplementary materials and technical detail can be placed in an appendix where it will be accessible but will not interrupt the flow of the text; alternatively, it can be published only in the electronic version of the journal. Restrict tables and figures to a total of 6 only (preferable to have most relevant tables and figures), needed to explain the argument of the paper and to assess its support. Use graphs as an alternative to tables with many entries; do not duplicate data in graphs and tables. The legends must be brief and relevant and the units of measurement must be clearly mentioned in tables and graphs, with the group names also mentioned in the same fashion as in the Methods section. While reporting results related to VAS score, it is better to categorize the score as mild (0-3), moderate (4-7) and severe (8-10) and analyse accordingly rather than reporting mean VAS scores in decimals (e.g., 3.45) because there is no such value as 3.45 (the VAS can either be 3 or 4). Similarly, while analyzing time, better to analyze in seconds or minutes (as applicable to a study) rather than as minutes or hours and then reporting the value in decimals (e.g., 3.7 min or 10.6 hours does not convey the meaning correctly; 60 seconds= one minute and 60 minutes= one hour, NOT 100 seconds and 100 minutes respectively).

Discussion:

Include summary of key findings (primary outcome measures, secondary outcome measures, results as they relate to a prior hypothesis): Confounding variables, strengths and limitations of the study. Interpretation and implications in the context of the totality of evidence (is there a systematic review to refer to, if not, could one be reasonably done here and now?, what this study adds to the available evidence, any new possible mechanisms etc): Controversies raised by this study; and Future research directions (for this particular research collaboration, underlying mechanisms, clinical research etc). Do not repeat in detail data or other material given in the Introduction or the Results section. In particular, contributors should avoid making statements on economic benefits and costs unless their manuscript includes economic data and analyses. Avoid claiming priority and alluding to work that has not been completed. New hypotheses may be stated if needed, however they should be clearly labelled as such. These articles generally can have 6-8 authors, with correct details of their contribution entered in the first page / cover page file.

Review Articles: (Total number of words including References should be less than 3000)

It is expected that these articles would be written by individuals who have done substantial work on the subject or are considered experts in the field and their contribution is solicited by the editorial board. A short summary of the work done by the contributor(s) in the field of review should accompany the manuscript. The manuscript should
have an unstructured Abstract (250 words) representing an accurate summary of the article. The section titles would depend upon the topic reviewed. The journal expects the contributors to give post-publication updates on the subject of review. The update should be brief, covering the advances in the field after the publication of the article and should be sent as a letter to editor, as and when major development occurs in the field. The number of images / figures / tables / graphs are to be limited to 4-6 only. They may be merged side by side when a change is intended to be shown. The legends must be brief and relevant and the units of measurement must be clearly mentioned in tables and graphs.

**Case reports:** (Total number of words including References should be less than 1500)

New, interesting and rare cases can be reported. They should be unique, describing a great challenge and providing a learning point for the readers. Cases with clinical significance or implications will be given priority. These communications should have the following headings: Abstract (unstructured), Key-words, Introduction, Case report, Discussion, Conclusion, References, Tables and Legends in that order. Please note that case reports are low priority articles. The number of images / figures / tables / graphs are to be limited to 2 only. They may be merged side by side when a change is intended to be shown. The legends must be brief and relevant and the units of measurement must be clearly mentioned in tables and graphs.

**Brief communications** (Total number of words including References should be less than 1000)

The manuscript should have the following headings: Introduction, Case report (for Case reports) (Methods and Results for Clinical investigations), Discussion, Conclusion, References, Tables and Legends in that order. The number of images / figures / tables / graphs are to be limited to 2 only. They may be merged side by side when a change is intended to be shown. The legends must be brief and relevant and the units of measurement must be clearly mentioned in tables and graphs. Please note that brief communications do not need an abstract.

**Letter to the Editor:** (Total number of words including References should be less than 1500)

These should be short and decisive observations. They can also be related to articles previously published in the Journal or views expressed in the journal. They should not be preliminary observations that need a later paper for validation.

**Comments on Published Articles:**

The comments, addressed to the Editor, should include reference of the published article, should be concise with critical comments to the point, with references in support. Maximum number of word count allowed is 250 with not more than 4 references, the first reference being that of the article being commented upon.

**Response to Comments:** The author is allowed to present his case/response to the observations made by the reader, in concise, with upto 250 words, with not more than 4 references, the first reference being that of the comments and the second, of the article being commented upon.

**Other:**

Editorial, Guest Editorial and Commentary are solicited by the editorial board.

**References**

References should be numbered consecutively in the order in which they are first mentioned in the text (not in alphabetic order). Identify references in text, tables, and legends by Arabic numerals in square bracket after the punctuation marks. References cited only in tables or figure legends should be numbered in accordance with the sequence established by the first identification in the text of the particular table or figure. No references to be used in abstract and Conclusion/Summary. Use the style of the examples below, which are based on the formats used by the NLM in Index Medicus. The titles of journals should be abbreviated according to the style used in Index Medicus. Use complete name of the journal for non-indexed journals. Avoid using abstracts as references. Information from manuscripts submitted but not accepted should be cited in the text as "unpublished observations" with written permission from the source. Avoid citing a "personal communication" unless it provides essential
information not available from a public source, in which case the name of the person and date of communication should be cited in parentheses in the text.

The commonly cited types of references are shown here.

**Articles in Journals**


**Books and Other Monographs**


b. Editor(s), compiler(s) as author: Norman IJ, Redfern SJ, editors. Mental health care for elderly people. New York: Churchill Livingstone; 1996.


**Electronic Sources as reference**

**Journal article on the Internet**

**Monograph on the Internet**

**Homepage/Web site**

**Part of a homepage/Web site**

**Tables**

- Tables should be self-explanatory and should not duplicate textual material.
- Tables with more than 10 columns and 25 rows should be avoided.
- Number tables, in Arabic numerals, consecutively in the order of their first citation in the text and supply a brief title for each. Place explanatory matter in footnotes, not in the heading.
- Explain in footnotes all non-standard abbreviations that are used in each table.
- Obtain permission for all fully borrowed, adapted, and modified tables and provide a credit line in the footnote.
- For footnotes use the following symbols, in this sequence: *, †, ‡, §, ||,¶ , **, ††, ‡‡
- Tables with their legends should be provided at the end of the text after the references. The tables along with their number should be cited at the relevant place in the text

**Illustrations (Figures)**

- Upload the images in JPEG format. The file size should be within 2 MB in size while uploading.
• Figures should be numbered consecutively according to the order in which they have been first cited in the text.
• Labels, numbers, and symbols should be clear and of uniform size. The lettering for figures should be large enough to be legible after reduction to fit the width of a printed column.
• Symbols, arrows, or letters used in photomicrographs should contrast with the background and should be marked neatly with transfer type or by tissue overlay and not by pen.
• Titles and detailed explanations belong in the legends for illustrations not on the illustrations themselves.
• When graphs, scatter-grams or histograms are submitted the numerical data on which they are based should also be supplied.
• The photographs and figures should be trimmed to remove all the unwanted areas.
• If photographs of individuals are used, their pictures must be accompanied by written permission to use the photograph.
• If a figure has been published elsewhere, acknowledge the original source and submit written permission from the copyright holder to reproduce the material. A credit line should appear in the legend for such figures.
• Legends for illustrations: Type or print out legends (maximum 40 words, excluding the credit line) for illustrations using double spacing, with Arabic numerals corresponding to the illustrations. When symbols, arrows, numbers, or letters are used to identify parts of the illustrations, identify and explain each one in the legend. Explain the internal scale (magnification) and identify the method of staining in photomicrographs.
• Final figures for print production: If the uploaded images are not printable quality, the publisher office may request for higher resolution images which can be sent at the time of acceptance of the manuscript. Send sharp, glossy, un-mounted, color photographic prints, with height of 4 inches and width of 6 inches at the time of submitting the revised manuscript. Print outs of digital photographs are not acceptable. If digital images are the only source of images, ensure that the image has minimum resolution of 300 dpi or 1800 x 1600 pixels in TIFF format. Send the images on a CD. Each figure should have a label pasted (avoid use of liquid gum for pasting) on its back indicating the number of the figure, the running title, top of the figure and the legends of the figure. Do not write the contributor/s' name/s. Do not write on the back of figures, scratch, or mark them by using paper clips.
• The Journal reserves the right to crop, rotate, reduce, or enlarge the photographs to an acceptable size.

Authors

• Last name and given name provided along with Middle name initials (where applicable)
• Author for correspondence, with e-mail address and mobile number provided
• Number of contributors restricted as per the instructions. If authors are more then a proper justification have been provided.
• Identity not revealed in paper except title page (e.g. name of the institute in Methods, citing previous study as 'our study', names on figure labels, name of institute in photographs, etc.)

Presentation and format

• Double spacing, No.12 Times New Roman
• Margins 1.5 cm from all four sides
• Page numbers included at bottom
• Title page contains all the desired information
• Running title provided (not more than 50 characters)
• Abstract page contains the full title of the manuscript
• Abstract provided (structured/unstructured abstract of 250 words for original articles, unstructured abstracts of about 150 words for all other manuscripts excluding brief communications and letters to the Editor)
• Key words provided (three or more)
• Introduction of 75-100 words
• Headings in title case (Capitalize each word)
• The references cited in the text should be after punctuation marks, in square bracket.
• References according to the journal’s instructions, punctuation marks checked

Language and grammar

• Proper English with no grammatical mistakes
• Write the full term for each abbreviation at its first use in the title, abstract, keywords and text separately unless it is a standard unit of measure. Numerals from 1 to 10 spelt out
• Numerals at the beginning of the sentence spelt out
• Check the manuscript for spelling, grammar and punctuation errors: language and grammar check may be performed using option in MS word or with professional help
• If a brand name is cited, supply the manufacturer’s name and address (city and state/country).
• Species names should be in italics

Tables and figures

• No repetition of data in tables and graphs and in text
• Actual numbers from which graphs drawn, provided
• Figures necessary and of good quality (colour)
• Table and figure numbers in Arabic letters (not Roman)
• Labels pasted on back of the photographs (no names written)
• Figure legends provided (not more than 40 words)
• Patients’ privacy maintained (if not permission taken)
• Credit note for borrowed figures/tables provided

• Write the full term for each abbreviation used in the table as a footnote
### Summary of requirements:

<table>
<thead>
<tr>
<th>Type of submission</th>
<th>Maximum number of authors allowed</th>
<th>Abstract (Maximum word count)</th>
<th>Article (Maximum word count)</th>
<th>Supplementary material</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Original Article</td>
<td>Six (Upto 8 if clear and convincing role of all is produced and approved by Editor)</td>
<td>Structured: Background and Aims, Methods, Results and Conclusion (250)</td>
<td>Structured: Introduction(with 4-6 references), Methods, Results, Discussion and Conclusion. (3000)</td>
<td>Max. Six (Tables, graphs and Figures included)</td>
<td>Maximum 30</td>
</tr>
<tr>
<td>Review article</td>
<td>Three</td>
<td>Unstructured (250)</td>
<td>Introduction (with 4-6 references), And Summary mandatory (3500)</td>
<td>Upto 4-6 (Tables, graphs and Figures included)</td>
<td>Maximum 35</td>
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<tr>
<td>Special article</td>
<td>Four</td>
<td>Unstructured (150)</td>
<td>Introduction, (with 2-4 references) And Summary mandatory (2000)</td>
<td>Max. Four (Tables, graphs and Figures included)</td>
<td>25</td>
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<td>Case report / series</td>
<td>Four</td>
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<td>1500</td>
<td>Max. Two (Table, graph or Figure)</td>
<td>15</td>
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<tr>
<td>Brief communications</td>
<td>Four</td>
<td>NONE</td>
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<td>10</td>
</tr>
<tr>
<td>Letter to Editor</td>
<td>Four</td>
<td>NONE</td>
<td>600</td>
<td>Max. Two (Table, graph or Figure)</td>
<td>6</td>
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<tr>
<td>Comments on previously published article</td>
<td>Two</td>
<td>NONE</td>
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<td>Response to comments</td>
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<td>NONE</td>
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