IJGC

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HIGHLIGHTS

Announcement

Geriatric Society of India West Bengal Branch

(Under the aegis of GSI WB Institute of Training, Education and Research (GWITER)- Established by a resolution of the registered body, Geriatric Society of India West Bengal Branch)

Launching Soon!

Geriatric Social Worker (Caregiver) Training Programme

(Pre-recorded online video training programme)

Course Duration – 06 (Six Month)

Eligibility - Pan India, 10+2 Passed and above

(Under exceptional cases educational qualification can be lowered)

Medium of Study: English

Course Fee: 1000/-

Inclusive of a copy of "A guide for a Geriatric Social Worker Caregiver

Invited Faculty: Pan India GSI Members and also non-Medicos.

Contact:
Dr. Kaushik Ranjan Das (krdas58@yahoo.co.in)

Announcement!!

Mid-Term Conference 2023

Geriatric Society of India®

26 – 27 August 2023

at

Scientific Convention Centre, Lucknow

Organizer

Dr. Kauser Usman

kauserusman87@gmail.com

Further details will be emailed soon

Editorial Board

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Editorial

Dr. A. K. Singh
President
Geriatric Society of India

Elderly and Comorbidities

INTRODUCTION

With the advancement in medical sciences number of deaths due to acute infectious diseases and pandemics has decreased, resulting in increased longevity and life expectancy. With increasing age functional and physiological decline occurs in nearly all organs and systems of body resulting in various derangements in them. So there is a hike in number of persons suffering from chronic diseases globally with global increase of life expectancy.[1] With passage of time, due to accumulation of these changes elderly become more prone to various metabolic and chronic diseases e.g. DM, HTN, IHD, dyslipidemia, atherosclerosis, osteoporosis, sarcopenia, dementia, Alzheimer's disease etc. and various geriatric syndromes like urinary incontinence, instability with falls, cataract, visual and hearing impairment, intellectual impairment, joint pain and other locomotor problems etc. Occurrence of chronic diseases increases with increasing age.[2] In between 75 to 79 years of age the chances of having 2 or more diseases is nearly 60% which increases to more than 75% in those between 85 to 89 years of age. Almost 2/3rd of older persons above the age of 80 years have 3 or more chronic conditions or comorbidities / multimorbidities.[3] Usually the subclinical pathology in multiple organ systems is present even in those apparently healthy older persons who present with single disease only.

DEFINITION

In 2008 WHO defined comorbidity as the simultaneous occurrence of 2 or more diseases in a single person which may be interrelated or of independent occurrence.[4]
Although comorbidity and multimorbidity are often used interchangeably to describe a state of multiple chronic conditions, actually they are different in meaning. In comorbidity there is an index condition which predominates management decisions. Contrary to it, multimorbidity is not dominated by any index condition rather here all co-existing conditions are given equal importance while taking management decisions.[5]

PREVALENCE

In USA incidence of 4 or more chronic conditions was 31.4% in medical beneficiaries above 85 years of age.[2] Al Modeer *et al* found that 89% elderly of southern Saudi Arabia had 2 or more comorbidities.[6] In Australia about 80% of elderly had 3 or more illnesses.[7] In China multimorbidity was found among 44% of elderly.[8] In Saudi Arabia, HTN was the commonest condition in elderly followed by DM and hyperlipidemia.[9] Similar disease pattern in elderly was also observed from other countries. In our study arthritis was the commonest condition in rural elderly patients of Varanasi followed by hypertension, visual problems, IHD, hearing impairment, DM, chronic bronchitis, asthma and emphysema etc.[10]

MANAGEMENT

In elderly with comorbidities management decisions are very difficult. First because most of the evidence based guidelines exist for single index condition only and very few of them address comorbid conditions. Secondly most of the randomized clinical trials exclude elderly population from their studies. Hence, proper decision or protocol making for elderly with comorbidities is really difficult. The Ariadne principles are a suitable approach in such situations,

especially in primary care settings.[11] It is a consensus framework for primary care consultations. Though it lacks a strong evidence base, it provides a broader approach to care which is consistent with general practice settings and needs of the patient. The principles are as follows –

1 – To assess the potential interactions – here the interactions among the patient's existing conditions, interactions among treatments of those conditions, possible drug interactions, constitution of the patient and the context should be properly assessed.

2 – To elicit preferences and priorities – here the prognosis, worst and best outcomes, possible alternatives (if they exist) and patient's priorities should be taken into consideration. Here the patient's choice is of paramount importance and all further actions and treatment plans should be performed accordingly.

3 – To individualise management to reach the negotiated treatment goals – here the individualized management plan according to preferences and priorities of the patient should be framed properly and performed to reach the desired goal.

IMPLICATIONS

Comorbidities and particularly multimorbidities are associated with poorer quality of life, greater burden on healthcare services, increased hospitalization rates and higher chances of polypharmacy and adverse drug reactions in such patients. They have great implications on the management and prognosis of a patient. They also impose various restrictions over the sufferer. Comorbidities are often associated with increased mortality, increased length of stay in hospital, readmission in hospitals, increased frequency of falls, poorer physical functioning, increased chances of persistent depression, lower level of social well being and a negative influence on quality of life.

CONCLUSION

Advancements in medicine have led to an increase in the average life expectancy which in turn has been associated with an increased prevalence of chronic diseases. The elderly age group is more prone to these conditions due to age-related derangements in metabolism and homeostasis. Hence, this age group in particular tends to have multiple co-morbidities. This is associated with a poor quality of life, various functional limitations and polypharmacy. Management of multiple co-morbidities is also a challenge in this age group. Comorbidities are often associated with increased mortality, increased length of stay in hospital, readmission in hospitals and increased frequency of falls and confer a poor prognosis whenever present. Co-morbidities in elderly significantly increase the burden on the healthcare system. It is imperative that these co-morbidities be tackled from an early age through population education and implementation of a healthy lifestyle and other preventive measures.

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Clinical Study of Septicemia In Geriatric Patients In ICU

Pradnya Diggikar[1], Nelabhotla Sai Satya Saranya[2]

ABSTRACT

Sepsis and septic shock can affect anyone of any age, but the elderly is the most likely to be affected by them. Currently, patients older than 60 years old account for most of sepsis cases that are observed.

When two or more of the following criteria for systemicinflammatory response syndro me (SIRS) are met, sepsis is considered to be present, regardless of whether an infection is present 1. Tachycardia, 2. Hypothermia or hyperthermia, 3. Tachypnoea or hypocapnia as a result ofhyperventilation), 4. White blood cell counts of less than 4,000 cells/mm or more than 12,000 cells/mm or more than 12 x 109 cells, or a band formation rate of more than 10 percent (immature white blood cells).

Septic Shock: A subset of sepsis in which underlying circulatory and cellular/metabolic abnormalities lead to substantially increased mortality risk.

INTRODUCTION

In January 1999, the Government of India adopted the "National policy on older persons", which defines an "elderly" person as one who is 60 years of age or older.

Sepsis and septic shock can affect anyone of any age, but the elderly is the most likely to be affected by them. Currently, patients older than 60 years old account for most of sepsis cases that are observed. In the progression of severe forms of sepsis, advanced age is a risk factor for obtaining a bloodstream infection that was caused by a nosocomial pathogen.

Because of its complexity from a pathophysiology, clinical, and therapeutic point of view, sepsis is one of the most important problems in the field of medicine. Although numerous definitions for this illness have been put out, it can generally be believed that they all refer to the clinical manifestation of the body's systemic response to an infection or an acute disease accompanied by inflammation.

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Although these definitions have been proposed, they are not universally accepted. In spite of advances in medical treatment, sepsis, severe sepsis, and septic shock are illnesses that greatly limit both the quality of life and the eventual survival of intensive care unit (ICU) patients. These conditions are linked with varying grades of organ dysfunction or failure.[1]

When two or more of the following criteria for systemic inflammatory response syndrome (SIRS) are met, sepsis is considered to be present, regardless of whether an infection has been strongly suspected or proven.[2]

1. A heart rate that is faster than 90 beats per minute, also known as tachycardia.
2. Body temperature below 36 degrees Celsius (96.8 degrees Fahrenheit) or above 38 degrees Celsius (100.4 degrees Fahrenheit) (hypothermia or hyperthermia).
3. A respiratory rate of more than 20 breaths per minute or, as measured by blood gases, a PaCO2 of less than 32 mm Hg (4.3 kPa) (tachypnoea or hypocapnia as a result of hyperventilation).
4. White blood cell counts of less than 4,000 cells per millimeter three or more than 12,000 cells per millimeter three or more than 12 x 109 cells, or a band formation rate of more than 10 percent (immature white blood cells).

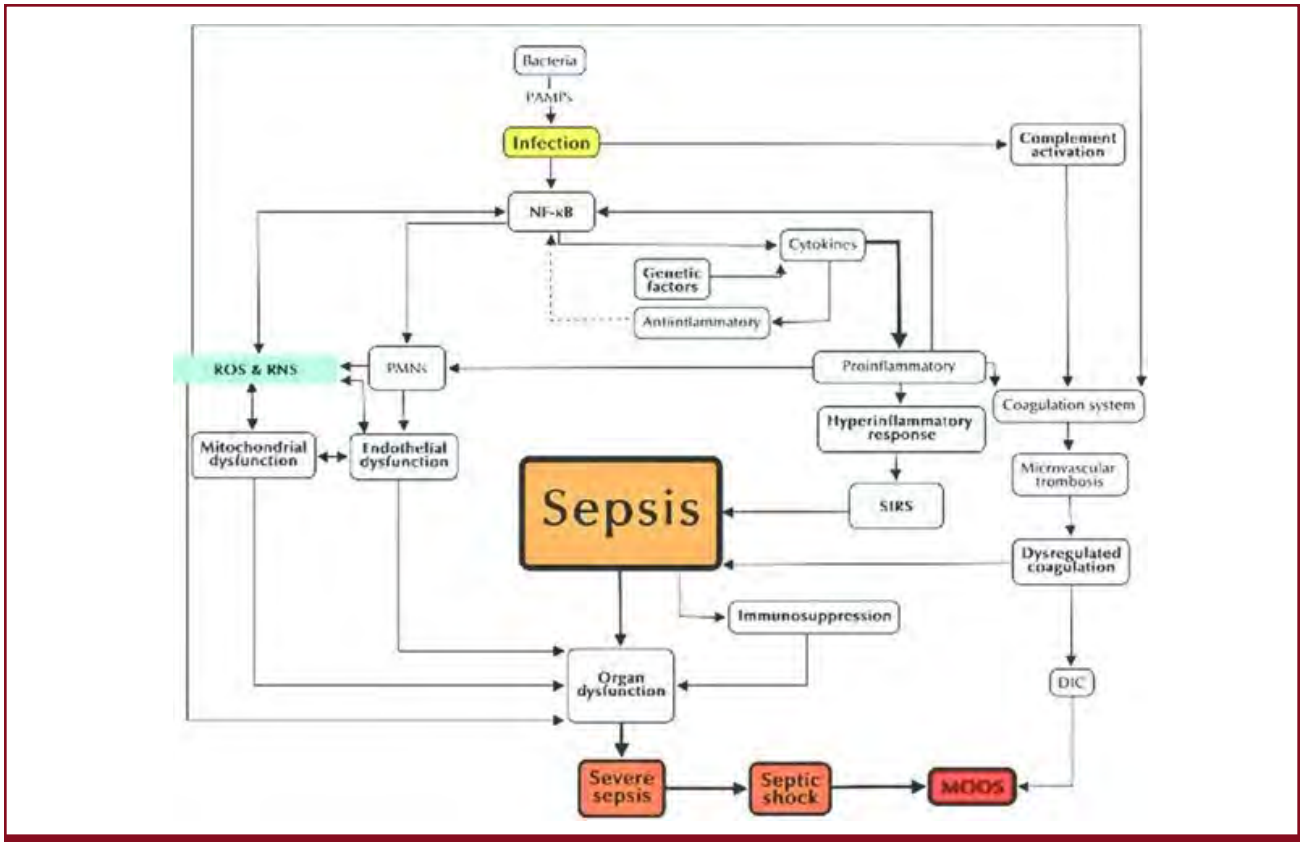

Figure 1. Pathophysiology of sepsis

SEPSIS: A life-threatening organ dysfunction caused by a dysregulated host response to infection.[2]

SEVERE SEPSIS: The presence of sepsis in addition to malfunction in one or more organs.[2]

SEPTIC SHOCK: A subset of sepsis in which underlying circulatory and cellular/metabolic abnormalities lead to substantially increased mortality risk.[2]

Septic shock can be described as severe sepsis along with the demand for vasoactive drugs. A subtype of sepsis in which the patient has underlying circulatory and cellular/metabolic problems that contribute to a significantly higher risk of death. Patients require vasopressor therapy in order to raise their mean arterial pressure to a level that is more than 65 mmHg, despite the fact that they have received an acceptable amount of fluid resuscitation.[2]

REFRACTORY SEPTIC SHOCK: Septic shock with refractory symptoms, an episode of septic shock that lasts for one hour and does not react to the administration of fluids or pressors.[2]

MULTI ORGAN FAILURE SYNDROME: The term "Multi organ Failure Syndrome" (MODS) refers to a condition that occurs when a patient has sepsis in addition to dysfunction in more than one organ and needs medical intervention to maintain homeostasis.[1]

REACTIONS NORMAL IN SEPSIS

When innate immune cells, particularly macrophages, recognize and bind to components of microorganisms, this triggers the beginning of the host's response to an infection. There are a few possible routes that lead to this result.[3]

On the surface of host immune cells is a protein complex called a pattern recognition receptor, or PRR, which has the ability to recognize and bind to a specific molecule called a pathogen-associated molecular pattern, or PAMP. Toll-like receptors, also known as TLRs, nucleotide-oligomerization domain leucine-rich repeat proteins, and retinoic acid-inducible gene I (RIG-I)-like helicases are the three families of PRRs. The TLRs play a particularly significant role among these.[3]

PRRs are able to recognize endogenous danger signals, also known as alarmins or danger-associated molecular patterns (DAMPs), such as the release of HMGB 1, S 100

proteins, and mitochondrial DNA during an inflammatory insult. When they are released into the extracellular environment, structures that are either nuclear, cytoplasmic, or mitochondrial in origin acquire new functions.[3]

On host immune cells, the triggering receptor expressed on myeloid cell (TREM-1) and the myeloid DAP12-associating lectin (MDL-1) receptors may recognize and bind to components of microorganisms.[3]

The interaction of TLRs with constituents of microbial cell walls triggers the activation of cytosolic nuclear factor-kb, which in turn triggers a signaling cascade (NF-kb). Activated NF- kb travels from the cytoplasm to the nucleus, where it then binds to transcription sites and induces the activation of a large set of genes involved in the inflammatory response of the host. These genes include proinflammatory cytokines (tumor necrosis factor alpha [TNFa], interleukin-1 [IL-1]), chemokines (intercellular adhesion molecule-1 [ICAM-1], and vascular cell adhesion.[3]

Activation of neutrophils results in the expression of adhesion molecules, which leads to the aggregation and margination of neutrophils to the endothelium of blood vessels. This is made easier by the fact that endothelium expresses adherence molecules, which leukocytes use to adhere to. Warmth and erythema due to local vasodilation and hyperemia, and protein-rich edema due to enhanced microvascular permeability are the symptoms of local inflammation. These indicators are caused by the release of mediators by neutrophils at the site of the infection.[4]

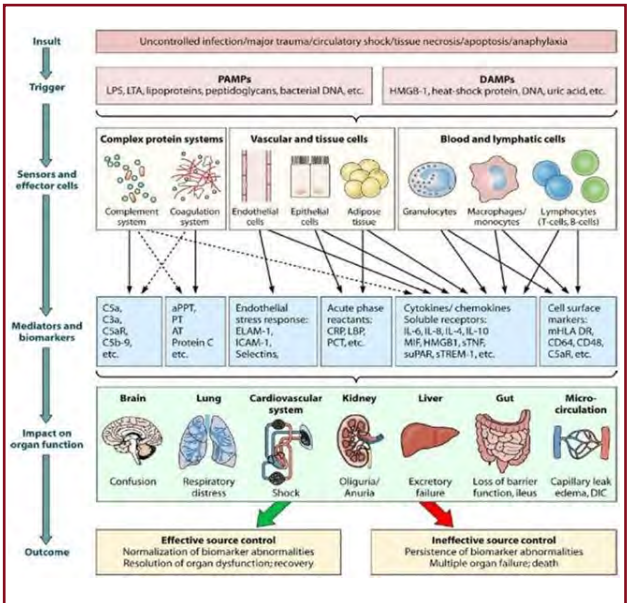

Figure 2. Sepsis induced organ dysfunction[7]

Macrophages become activated when bacteria invade tissue, and they regulate the process of local inflammation by producing an equilibrium between pro-inflammatory cytokines (such as TNF alpha and IL-1) and anti-inflammatory cytokines (such as IL-6 and IL-10), which are cytokines that inhibit the production of inflammatory cytokines. Macrophages become activated when bacteria invade tissue.[5]

The complement cascade is also activated by microbial components, both directly and indirectly through the proteolytic activity of plasmin. This activation results in the production of anaphylatoxins (C3a, C5a), chemotactic fragments (C5a), and opsonin's (C3b), all of which contribute to the proinflammatory state. Both directly through factor XII and indirectly through a changed state of endothelial function, the microbial components can activate the complement system.[5]

SYSTEMIC EFFECTS OF SEPSIS

Cellular damage, which is often the first step toward organ dysfunction, can occur if the immune response is allowed to become widespread. Several different mechanisms have been proposed to explain the cellular damage.

- Tissue ischemia (insufficient oxygen relative to oxygen need).
- Damage caused by cytopathy, which is the direct harm of cells caused by proinflammatory mediators and/or other inflammatory products.
- An abnormally high or low rate of apoptosis (programmed cell death).[6]

ORGAN-SPECIFIC EFFECTS OF SEPSIS

Circulation

Widespread vasodilation is the root cause of the circulatory dysfunction that occurs in sepsis. This occurs as a direct consequence of the production of vasoactive mediators, the function of which is to induce adequate vasodilation to enhance metabolic autoregulation, which is the process by which oxygen availability is matched to the changing tissue oxygen requirements. Prostacyclin, a vasodilator, and nitric oxide, often known as NO, are examples of vasoactive mediators. Endothelial cells are responsible for their production.[8]

Effects on the circulation that are localized

Cytokines can suppress myocardial activity in the central circulation, which includes the heart and major vessels. This results in a reduction in the systolic and

diastolic ventricular function of the heart. Cardiac myocytes may still be able to use the Frank Starling mechanism to raise cardiac output, which is necessary to maintain blood pressure in the context of systemic vasodilation. This is the case despite the fact that ventricular function has been diminished. Folks who already have a heart condition, such as older patients, are the ones who won't have this compensatory mechanism available to them.[9]

Lung

Injury to the endothelial lining of the pulmonary vasculature causes disruptions in the blood flow of the pulmonary capillaries and an increase in the permeability of the microvascular network, both of which contribute to edema in the interstitial and alveolar spaces. The damage to the alveolar capillary membrane is both initiated and exacerbated by the capture of neutrophils within the pulmonary microcirculation. Acute respiratory distress syndrome (ARDS) is a common symptom of these consequences.[10]

The digestive and intestinal tracts

Damage to the endothelium and other circulatory irregularities also reduce the normal barrier function of the gastrointestinal tract. This makes it possible for bacteria and endotoxin to enter the systemic circulation, which in turn makes the septic reaction more severe. This is supported by the findings of a prospective cohort study, which found that an increased intestinal permeability, as measured by the urinary excretion of orally administered lactulose and mannose, was predictive of the development of multiple organ dysfunction syndrome. This study was carried out on rats.[11]

Liver

When germs and products produced by bacteria pass through the portal system from the digestive tract and reach the liver, the reticuloendothelial cells of the liver serve as the body's first line of defense against them. Liver dysfunction can inhibit the removal of enteric-derived endotoxin and bacteria-derived toxins, allowing for the direct spillover of these potentially harmful compounds into the systemic circulation. This can be harmful in several ways.[12]

Kidney

Acute renal failure is a common complication that occurs alongside sepsis. There is a gap in our understanding of the mechanisms through which sepsis can lead to acute renal failure. One of the mechanisms is acute tubular necrosis, which can be caused by hypoperfusion and/or hypoxemia. It is also possible that direct renal vasoconstriction, systemic arterial hypotension, and the release of cytokines all contributed to the renal injury.[13]

Nervous system

Sepsis typically begins with symptoms in the nervous system, particularly in the central nervous system, before spreading to other organ systems. Although an altered sensorium (encephalopathy) is the most common complication of the central nervous system, the pathogenesis of how it develops is not well understood. In one study, a high incidence of brain abscesses was discovered; however, the role of hematogenous infection as the primary mechanism is still unknown due to the heterogeneity of the observed pathology.[14]

POOR PROGNOSTIC FACTORS IN SEPTIC SHOCK

- Hypotension that is resistant to treatment.
- Coma Leukopenia (WBC <5000 / > 50,000 cells) .
- Thrombocytopenia, which is defined as a platelet count of less than one million.
- Low fibrinogen (<150mg/dl) .
- There is evidence of MODS (acute respiratory distress syndrome, acute hepatic failure, central nervous system dysfunction, and myocardial depression).[18]

SOURCES OF SEPSIS

The majority of patients diagnosed with sepsis have an identifiable source of infection, with the exception of individuals who are immunocompromised and have neutropenia. In these patients, a clear source of infection is sometimes not detected in cases where sepsis has developed. The most common causes of sepsis are infections of the respiratory and urinary tracts, followed by infections of the abdominal cavity and soft tissues. One of the known risk factors for developing health care-associated sepsis is the use of intravascular devices. Sixteen to twenty-five percent of individuals may have an infection at more than one site.[19]

- Bacterial infections of the lower respiratory tract. Bacterial infections of the urinary tract.
- Infections of the central nervous system. Infections of the gastrointestinal tract.
- Intravascular implants and other types of foreign bodies that might cause infections.[19]

METHODS AND MATERIALS

Methodology

The study was conducted after getting permission from institutional ethics committee. Geriatric patients

were taken up for the study. A detailed clinical history was taken from all the patients regarding symptoms of sepsis. Patients were examined for signs and symptoms of sepsis.

TYPE OF STUDY: Prospective cross-sectional hospital based observational study.

SAMPLE SIZE: out of 100 patients with sepsis, 30 patients who were above the age of 60 years were taken as sample size from November 2022 to April 2023.

INVESTIGATIONS

1. Complete blood count/ESR
2. ABG
3. CRP
4. D- dimer
5. PT-INR
6. Renal function test/Serum electrolytes
7. Liver function test/ Serum Proteins
8. Urine R/M
9. Culture and sensitivity
10. Blood culture and sensitivity
11. S. Ferritin
12. ECG
13. Radiological imaging investigations
    - Chest X-ray PA view
    - USG Abdomen and Pelvis
14. Procalcitonin
15. Sputum/Endotracheal tube suction culture and sensitivity
16. SIRS

INCLUSION CRITERIA

All patients with sepsis aged 60 years and above.

Sepsis is considered present if infection is highly suspected or proven and two or more of the following Systemic inflammatory response syndrome (SIRS) criteria are met.

- Heart rate >90beats per minute (Tachycardia)
- Respiratory rate>20 breaths/minute or $PaCO_2$<32mm Hg (Tachypnoea or hypocapnia due to hypoventilation)
- White blood cell count-<4000cells/microliter or >12,000cells/microliter or greater than 10% band forms (immature white blood cells) (leukopenia/ leukocytosis)
- Body temperature<36 degree Celsius (96.8degreeF) or >38 degree Celsius (100.4-degreeF)

The new sepsis guidelines now identify organ dysregulation in sepsis as an increase in the Sequential organ failure assessment (SOFA) > or equal to 2.

TABLE 1: Descriptive analysis of gender in the study population (N=30)

GENDER	FREQUENCY	PERCENTAGES
MALE	21	70%
FEMALE	9	30%

Table 2: Summary of Present history of various parameters in the study population (N=30)

Present history of various parameters	Frequency	Percentage
Fever		
Present	27	90%
Absent	3	10%
Weakness		
Present	24	80%
Absent	6	20%
Abdominal Pain		
Present	18	60%
Absent	12	40%
Breathlessness		
Present	15	50%
Absent	15	50%
Burning Micturition		
Present	9	30%

EXCLUSION CRITERIA

All patients below the age of 60 years.

ANALYSIS

Among the study population, 70% of them were Male, 30% of them were Female (Table 1).

Among the study population with Present history, 90% of them had fever, 80% of them had Weakness, 60% of them had Abdominal Pain, 50% of them had Breathlessness, 30% of them had Burning Micturition, 30% of them had Altered Sensorium, 6% of them had Vomiting, 20% of them had Cough, 10% of them had Loose Stools, 10% of them had Chest Pain (Table 2).

Among the study population, 80% were febrile, 70% of them had pulse rate >90bpm, 90% of them had respiration rate >20 per minute, 80% of them had SBP<=120mmHg, 80% of them had DBP <=80mmHg, 10% of them had

TABLE 3: Descriptive analysis of vital parameter in the study population (N=30)

Temperature	Frequency	Percentages
Febrile	24	80%
Afebrile	6	20%
PR In Bpm		
<=90	9	30%
>90	21	70%
RR		
<=20	3	10%
>20	27	90%
SBP		
<=120	24	80%
>120	6	20%
DBP		
<=80	24	80%
>80	6	20%
Mean Arterial Pressure		
<=65	3	10%
>65	27	90%
Spo2 On RA		
<=90	9	30%
>90	21	70%

mean arterial pressure <=65, 30% of them had SPO2 on RA <=90% (Table 3).

Among the study population, 43% of them had Hb >12, 40% of them had between 7 to 10 and 6% had less than 7 and in population regarding WBC, 70% of them had >=12000, 14% of them had 4000 to 12000 and 16% had <4000, population with Platelet count, 90% of them had >100000, 10% of them had <100000, 60% of their HBA1C were >6.4, 30% of them were <5.7 (Table 4).

Among the study population, in 30% them ESR was increased, in 100% of them CRP was increased, in 90% of them Procalcitonin was increased, in 50% of them LDH was increased, in 80% of them Ferritin was increased, in 90% of them D- Dimer was Increased (Table 5).

Among the study population with urine Rm, 60% of them were WNL, 26% of them had pus cells (Table 6).

Among the study population with CXR-PA, 70% of them had WNL, 20% of them had B/L inhomogeneous opacities, 3% of them had Rt LZ inhomogeneous opacity

Table 4: Summary of laboratory parameters in the study population(N=30)

Hb	Frequency	Percentages
<7	2	6%
>12	13	43%
10 to 12	3	10%
7 to 10	12	40%
WBC		
<4000	5	16%
>=12000	21	70%
4000 to 12000	4	14%
Platelets		
<100000	3	10%
>100000	27	90%
Hba1C		
<5.7	9	30%
>6.4	18	60%
5.7 to 6.4	3	10%

(Table 7).

Among the study population in Urine C/S, 20% of them had E coli, 6% of them klebsiella pneumonia, 3% of them had Candida species (Table 8).

Among the study population in Blood C/S,67% of them had no growth, 10% of them had E coli, 10% of them Klebsiella pneumonia (Table 9).

Among the study population in Sputum C/S, 3% of them had Pseudomonas aeruginosa, 3% of them were AFB positive, 3% of them had Pseudomonas (Table 10).

Among the study population with USG A+P, 30.0% of them had WNL, 6.0% of them had Changes of cystitis, 9.0% of them had Pancreas bulky,10% of them had heterogenous echotexture, 10% of them had B/L Raised renal echogenicity (Table 11).

Among the study population, 20% of them undergone dialysis (Table 12).

Among the study population, 33% of them were under mechanical ventilation (Table 13).

Among the study population, 30% mortality was noted (Table 14).

TABLE 5: Descriptive analysis of parameter in the study population (N=30)

ESR	Frequency	Percentages
Normal	21	70%
Increased	9	30%
CRP		
Increased	100	100%
Procalcitonin		
Increased	27	90%
Normal	3	10%
LDH		
Increased	15	50%
Normal	15	50%
Ferritin		
Increased	24	80%
Normal	6	20%
D-Dimer		
Increased	27	90%
Normal	3	10%

TABLE 6: Descriptive analysis of urine rm in the study population (N=30)

Urine R/M	Frequency	Percentages
WNL	18	60%
Pus cells	8	26%
Pus cells, RBC	1	3%
Protein	3	10%

Among the study population with DIAGNOSIS, 30% of them had Urosepsis, 3% of them had Acute gastroenteritis, 10% of them had Acute pancreatitis, 16% had respiratory tract infections (Table 15).

Among the study population, 94% of them were febrile, 70% of them had pulse rate was >90bpm , 70% of them had WBC >=12000, 14% of them had 4000 to 12000 and 16% had < 4000 and 90% had RR >20cpm (Table 16).

Table 7: Descriptive analysis of CXR-PA in the study population (N=30)

CXR-PA	Frequency	Percentages
WNL	21	70%
B/L inhomogeneous opacities	6	20%
B/L blunting of CP angles	1	3%
Rt LZ inhomogeneous opacity	1	3%
Rt side pleural effusion	1	3%
Rt Upper lobe inhomogeneous opacity	0	0%

TABLE 8: Descriptive analysis of urine c/s in the study population (N=30)

Urine C/S	Frequency	Percentages
No growth	20	66%
E coli	6	20%
klebsiella pneumonia	2	6%
Candida species	1	3%
Pseudomonas	1	3%

TABLE 9: Descriptive analysis of blood c/s in the study population (N=30)

Blood C/S	Frequency	Percentages
No growth	20	67%
E coli	3	10%
Klebsiella pneumonia	3	10%
Acinetobacter	1	3%
Enterobacter species	1	3%
Enterococcus	1	3%
Pseudomonas aeruginosa	1	3%

Table 10: Descriptive analysis of sputum c/s in the study population (N=30)

Sputum C/S	Frequency	Percentages
No growth	**27**	90%
Pseudomonas aeruginosa	**1**	3%
AFB positive	**1**	3%
Pseudomonas	**1**	3%

Table 11: Descriptive analysis of USG A+P(N=30)

USG A+P	Frequency	Percent
B/L Kidneys small, CMD lost	2	6.0
B/L pyelonephritis changes	1	3.0
B/L Raised renal echogenicity	3	10.0
B/L raised renal echogenicity, Grade 1 hepatomegaly	1	3.0
Changes of cystitis	2	6.0
Changes of pyelonephritis	2	6.0
Heterogenous echotexture of pancreas	3	10.0
Mesenteric lymphadenopathy	1	3.0
Mild hepatomegaly	2	6.0
Multiple cysts in B/L kidneys	1	3.0
Pancreas bulky, heterogenous echotexture	3	9.0
WNL	9	30.0

TABLE 12: Descriptive analysis of dialysis in the study population (N=30)

Dialysis	Frequency	Percentages
Yes	6	20%
No	24	80%

TABLE 13: Descriptive analysis of ventilation in the study population (N=30)

Ventilation	Frequency	Percentages
Yes	10	33%
No	20	66%

TABLE 14: Descriptive analysis of outcome in the study population (N=30)

Outcome	Frequency	Percentages
Cured	21	70%
Death	9	30%

TABLE 15: DIAGNOSIS in the study population (N=30)

DIAGNOSIS	Frequency	Percent
Urosepsis	9	30%
Acute pancreatitis	3	10%
Acute gastroenteritis	1	3%
Respiratory tract infections	5	16%
Others	12	40%

TABLE 16: SIRS CRITERIA in the study population (N=30)

Temperature	Frequency Percentages	Percentages
Febrile	94	94%
Afebrile	6	6%
PR In Bpm		
<=90	9	30%
>90	21	70%
RR		
<=20	3	10%
>20	27	90%
WBC		
<4000	5	16%
>=12000	21	70%
4000 to 12000	4	14%

DISCUSSION

The present prospective cross-sectional hospital based observational study was conducted among 30 patients admitted in ICU Dr DY Patil Medical College Hospital and Research Centre, Pimpri, Pune during the period from NOVEMBER 2022- A P R I L 2023.

The salient findings of the present work in the form of summary are presented here.

1) In the study population majority were males constituting around 70% and females were around 34%.
2) In the study population, majority (90%) of patients presented with fever, (20%) had cough, (80%) had generalized weakness, (60%) had abdominal pain, (50%) had breathlessness, (30%) had burning micturition.
3) In the study population, 20% underwent Dialysis.
4) Majority chest X-ray finding was bilateral inhomogeneous opacities seen in 20% of patients due to acute respiratory distress syndrome as a complication of sepsis.
5) Majority of patients tested for Gram stain culture sensitivity in urine showed E. coli (20%) and Klebsiella Pneumoniae (6%).
6) The majority of patients tested for Gram stain culture sensitivity in blood showed E. coli (10%) , Klebsiella Pneumoniae (10%).
7) The majority of ultrasound abdomen and pelvis was bilateral raised renal echogenicity due to acute renal injury as a sequela of severe sepsis.
8) In the study population, 33% of patients needed mechanical ventilation because of sepsis leading to respiratory failure.
9) Among the study population, 30% of them had Urosepsis, 3% of them had Acute gastroenteritis, 10% of them had Acute pancreatitis, 16% had respiratory tract infections.
10) Inflammatory markers like ESR, CRP, S.LDH, Ferritin , D-Dimer and Procalcitonin are raised in the majority of the patients in study population.
11) 30% mortality is seen in the current study population who were in septic shock and multi organ failure.

CONCLUSION

In intensive care units, sepsis continues to be a leading cause of death. In the last 15 years, sepsis, severe sepsis, and septic shock have been managed with a more standardized approach, improving survivability. Early detection of sepsis includes symptoms and signs, such as leukocytosis or leucopenia, confusion, hypoxia, hypotension, pyrexia, and tachycardia. In addition to taking cultures and starting therapy early (Broad spectrum antibiotics, vasopressors, and fluids), an examination must include looking for the infection's source. In general, respiratory infections are responsible for around half of all sepsis cases. Genitourinary and abdominal sources of infections are also common causes. Even if sepsis fatality rates are on the decline, the overall number of sepsis deaths each year is still rising because of the rising incidence of illnesses.

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Estimating the Level of Cystatin C and its importance in managing Geriatric Patients

Prashant B, Edwin Gomes

ABSTRACT

There will be a growing population of geriatric population both in the wards of the hospital and / or at home and these will need cost effective care givers specially to improve their quality of life at the end of their life. They have already done enough and this is the least the state can do to them. It is time that each state has a pool of at least 30 to 40 trained care givers from which they can be appointed as a bed basis to look after the elderly and probably us when we grow old.

Keywords: *Elderly / Geriatric Population: Caregiver.*

INTRODUCTION

It would be important to estimate the correct glomerular filtration rate (eGFR) in a patient, because important clinical decisions like drug dosing, avoiding nephrotoxic drugs, and timely renal replacement therapy depends on the eGFR.

There are many ways to estimate the renal function, like the routine methods are the serum creatinine and creatinine clearance tests. The Cockcroft – Gault (CG) equation was then developed to estimate creatinine clearance, on the presumption that creatinine clearance was a direct measure of GFR.

The reason why this method of estimation of the renal function in the elderly may be not good is because there are many factors which can affect the formation of creatinine such as the amount of muscle mass and the rate of secretion of creatinine in the tubules. In the elderly, creatinine is affected by muscle mass.

The most widely used equation for eGFR is the four variable MDRD equation. However the MDRD equation does not consider the population over 70 years of age. It also excluded patients with normal kidney function, and the proportion of diabetics was small.

The limitations of creatinine mentioned above, have made physicians look for other biomarkers. One such biomarker is Cystatin C. Cystatin C is a protease inhibitor that is freely filtered through the glomeruli, reabsorbed and degraded by proximal tubules. Cystatin C levels are not affected by age or by muscle mass of an individual.[1,2] There was also a better correlation between serum Cystatin C and Inulin clearance, than serum creatinine and plasma inulin clearance. This suggests that Cystatin C is superior to creatinine for estimating GFR in the elderly.[3]

The KDIGO clinical practice guidelines suggest that, the Cystatin C based equations or plain Cystatin C estimation is limited to be used in individuals with eGFR between 45 to 59 ml/min/1.73m^2 with no other evidence of CKD to verify the presence of CKD.[4]

The use of Cystatin C has the potential to improve CKD diagnosis and epidemiology and its physiological behaviour is different to creatinine.

A study wherein estimating GFR using Serum Cystatin C alone and in combination with serum creatinine has been done and published, and the conclusion was that Serum Cystatin C alone provides GFR estimates that are nearly as accurate as serum creatinine adjusted for age, sex and race, thus providing an alternative GFR estimate that is not linked to muscle mass.[5]

Hence in our study we would want to do serum Cystatin C levels in the patients admitted with low body muscle mass, blood urea and serum creatinine on the lower side of normal range and aged above 70 years of age, where the calculated eGFR is more than 70 ml/min/ 1.73m^2.

If we find that the serum Cystatin C levels are high, indicating that there is decreased renal function, we could include it as a standard of care investigation in our patients more than 70 years of age whose serum creatinine is normal.

Dr. Prashant B. Senior Resident, Department of Geriatric Medicine, Dr. Edwin Gomes. Professor In Geriatric Medicine. Department of Geriatric Medicine.

AIMS AND OBJECTIVES

To establish the significance of estimating eGFR using Serum Cystatin C levels in elderly patients who have a Serum Creatinine value in the normal range, and near normal renal function when the eGFR was calculated using the Serum Creatinine levels.

MATERIALS AND METHODS

All patients admitted in the Geriatric Medicine Ward or seen in the Geriatric Medicine OPD at Goa Medical College and Hospital (A Tertiary Hospital In Goa) with a normal serum creatinine, and whose age is more than 70 would be included in the study.

Consent for the same would be taken in English from the patient or relative after explaining the need of the study.

The Blood Urea and Serum Creatinine values would be evaluated in Biochemistry Department on an auto analyser.

Serum Cystatin C would be estimated in a private standardised laboratory.

Serum Cystatin C would be compared to Serum Creatinine on the basis of the Calculated eGFR.

Calculated eGFR with serum creatinine will be done using the MDRD formula.

Calculated eGFR with serum Cystatin C will be done using the 2012 CKD-EPI Cystatin formula.

Patients with raised renal parameters would be excluded from the study.

SAMPLING

The sampling will be random sampling and the sample will be taken from the first 70 patients who are more than 70 years of age and have a normal creatinine.

We have taken a sample size of 70 as we would want to get Serum Cystatin C estimation as a standard investigation for at least once a year in those patients who are 70 years and above and have normal creatinine values.

ELIGIBILITY CRITERIA

Inclusion Criteria

- Age 70 years or more
- Normal serum creatinine
- Consent given

Exclusion Criteria

- Age less than 70 years
- Serum creatinine above normal
- No consent given

Data Collection and Statistical Analysis

The patients data will be collected by either the principal investigator or the co investigator and the proforma made will be filled so as the estimate

- Serum Creatinine
- Blood Urea
- Serum Cystatin C

As we routinely do the renal functions for all admitted patients or patients following up in the OPD, Serum Cystatin C will be done only in those with a normal creatinine.

For all the patients who meet the inclusion criteria the proforma will be filled.

Statistical Analysis

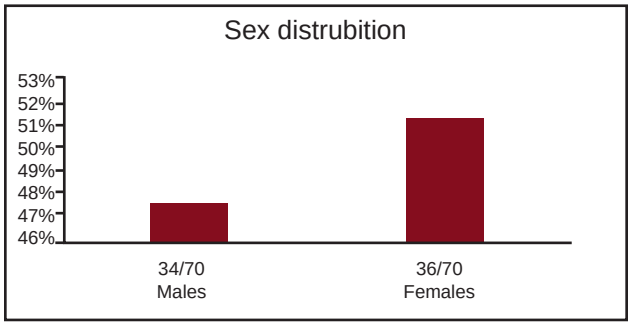
The Statistical analysis used will be calculating the percentage comparison, by comparing the standard eGFR calculation in these patients using the standard time tested test (MDRD) to the eGFR calculated using the serum Cystatin C Value.

Results

1. Demographics: Sex Distribution

Male versus Females

Males	34/70	48%
Females	36/70	52%

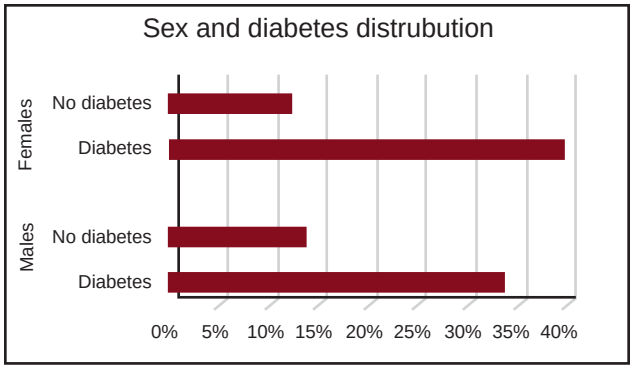


2. Diabetics versus Non Diabetics amongst Males and Females

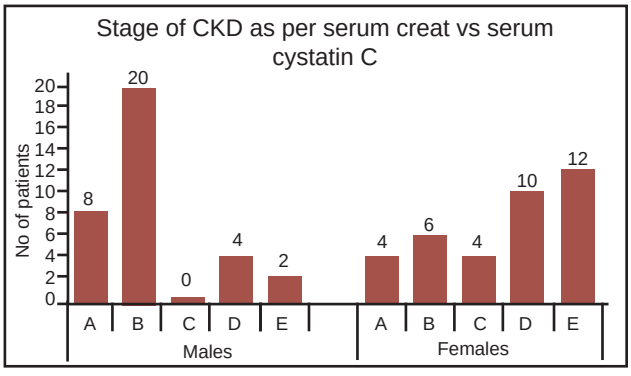
Males	Diabetes	34%	24
	No Diabetes	14%	10
Females	Diabetes	40%	28
	No Diabetes	12%	08

3. All Patients were between 70 to 82 years of age.
4. All the patients were hypertensive.
5. Stages Of CKD as per Serum Creatinine Vs Serum Cystatin C.

   Males. Creat Stage 1 / Cystatin C Stage 1. A 08



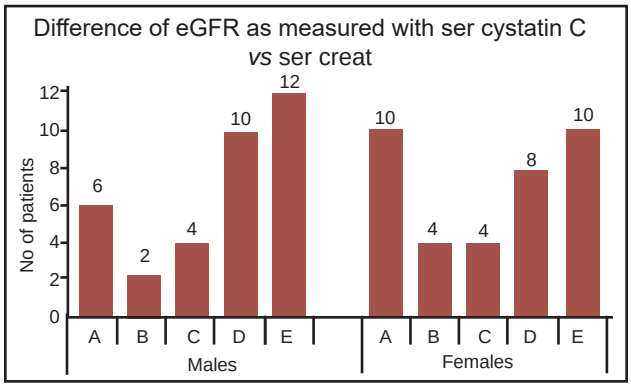
	Creat Stage 1 / Cystatin C Stage 2.	B	20
	Creat Stage 1 / Cystatin C Stage 3.	C	00
	Creat Stage 2 / Cystatin C Stage 2.	D	04
	Creat Stage 2 / Cystatin C Stage 3.	E	02
Females.	Creat Stage 1 / Cystatin C Stage 1.	A	04
	Creat Stage 1 / Cystatin C Stage 2.	B	06
	Creat Stage 1 / Cystatin C Stage 3.	C	04
	Creat Stage 2 / Cystatin C Stage 2.	D	10
	Creat Stage 2 / Cystatin C Stage 3.	E	12



The groups A and D where the Stage remained the same were mostly the 18 patients who were hypertensive only and not diabetic. There were 06 diabetic patients also who showed no change in the CKD stage.

Stages Of eGFR as measured based on Serum Creatinine versus Serum Cystatin C where in all the eGFR measured with Cystatin C was lower than that measured with serum creatinine.

Males	Difference 00 to 05 ml/min/1.73m²	A	06
	Difference 06 to 10 ml/min/1.73m²	B	02
	Difference 11 to 20 ml/min/1.73m²	C	04
	Difference 21 to 30 ml/min/1.73m²	D	10
	Diff more than 30 ml/min/1.73m²	E	12
Females	Difference 00 to 05 ml/min/1.73m²	A	10
	Difference 06 to 10 ml/min/1.73m²	B	04
	Difference 11 to 20 ml/min/1.73m²	C	04
	Difference 21 to 30 ml/min/1.73m²	D	08
	Diff more than 30 ml/min/1.73m²	E	10



The groups A, B, C from both groups which total 30 patients included the 26 patients who were hypertensive and not diabetic. That means only 4 patients who were diabetic had a eGFR difference of less than 20 ml.

In table 5,

22 of the 34 males (64.71%) showed worsening of the CKD stage when evaluated by Serum Cystatin C as compared to those evaluated by Serum Creatinine. This number became more significant as 22 0f the 24 (91.67 %) diabetic males had a worsening of the CKD stage when evaluated by Serum Cystatin C as compared to those evaluated by Serum Creatinine.

And similarly, 22 of the 36 females (61.11%) showed worsening of the CKD stage when evaluated by Serum Cystatin C as compared to those evaluated by Serum Creatinine. And this number became more significant as 22 of the 28 (78.57 %) diabetic females had a worsening of the CKD stage when evaluated by Serum Cystatin C as compared to those evaluated by Serum Creatinine.

Again in Table 6,

26 of the 34 males (76.47%) showed eGFR of more than 10 ml/min/1.73m², when evaluated by Serum Cystatin C as compared to those evaluated by Serum Creatinine. And this number became more significant as all the diabetic males (100%) had a eGFR of more than 10 ml/min/1.73m², when evaluated by Serum Cystatin C as compared to those evaluated by Serum Creatinine.

And Similarly, 22 of the 36 females (76.47%) showed eGFR of more than 10 ml/min/1.73m², when evaluated by

Serum Cystatin C as compared to those evaluated by Serum Creatinine. And this number became more significant as 22 of the 28 diabetic Females (78.57 %) had a eGFR of more than 10 ml/min/1.73m², when evaluated by Serum Cystatin C as compared to those evaluated by Serum Creatinine.

The results in Tables 5 and 6 clearly show that Cystatin C is a better indicator to measure the eGFR and grade the CKD severity specially in elderly diabetes patients with a normal creatinine.

Although not the aim of the study, the calculation of eGFR with both serum Cystatin C and serum Creatinine values were as close to the values got when they were measured using serum Cystatin C only.

DISCUSSION

In our study conducted at Goa Medical College and Hospital, Department Of Geriatric Medicine, which is the only Government Tertiary Hospital in Goa, among the the elderly patients above 70 years of age with a normal Serum Creatinine Level, Serum Cystatin C was evaluated to calculate the eGFR value.

There was worsening of the CKD stage when the Stage based on the eGFR was calculated with Serum Cystatin C level as compared to the Stage when evaluated with the Serum Creatinine value.

So also the eGFR when calculated with Serum Cystatin C level was more than 10 ml/min/1.73m², similar observations are there in literature as mentioned below.

Cystatin C was an alternate and more accurate serum marker than serum creatinine in discrimination type 2 diabetes mellitus patients with a reduced GFR from those who seen to have a near normal GFR based on serum creatinine.[6]

Studies have shown that Cystatin C is a better predictor of cardiovascular disease and mortality than serum creatinine based on estimation of eGFR.[7]

In the study of Kedam *et al*., Of 239 type 2 diabetic patients (normoalbuminurics: 110, microalbumin- urics: 81, macroalbuminurics: 48) were evaluated. The serum cystatin C levels were found negatively correlate with MDRD eGFR ($r = -0.364$, $p<0.0001$), and significantly higher in the macroalbuminurics than in the normoalbuminuric and microalbumin- uric groups.[8]

CKD- EPI-cys eGFR had better predictive value than the others for DN and it can be useful in detecting the early stage of DN.[9]

Serum cystatin C concentration was significantly higher in T2DM patients than in healthy control. Concentration of cystatin C increases with the progression of nephropathy in T2DM patients. In addition, the cystatin C level rises with the longer period of diabetes and UACR. Thus, serum cystatin C had higher accuracy in diagnosing nephropathy than serum creatinine in the Nepalese T2DM patients.[10]

CONCLUSION

eGFR is an important routine estimation that is needed in elderly patients specially when using medications which may be nephrotoxic, or using medications which cannot be used in an eGFR less than 25 ml/min/1.73m², (eg Dapglifozin, Finnerinone, Gliptins).

Hence all elderly patients with Type 2 Diabetes Mellitus who have a normal creatinine should have their serum Cystatin C estimation and their eGFR calculated based on serum Cystatin C or Serum Cystatin C with Serum Creatinine, to see their renal function and to monitor their renal function during their management.

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Sleep Disorders In Elderly-Pharmacotherapy And Beyond

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INTRODUCTION

Several physical and psychological changes are known to occur with normal ageing. Although sleep disturbance is a common complaint among all ages, studies suggests that older adults are especially at risk. A study of over 9000 people of more than 65 years age showed that 42 per cent among them have difficulty in initiating and maintaining sleep, follow up after 3 years revealed that 15 per cent of people who did not have sleep difficulty at baseline had sleep disturbances, suggesting an annual incidence rate of approximately 5 per cent.[1] Sleep disturbances in adults are related to various factors, like use of caffeine, tobacco, and alcohol, sleep habits, and comorbid diseases. Sleep apnea syndrome, rapid eye movement sleep behavior disorder, restless leg syndrome, and psychiatric disorders like depression and anxiety should always be ruled out in subjects who present with sleep disturbances. An epidemiological study in Japan reported an insomnia prevalence of 21.4 per cent, when insomnia was defined as at least one instance of difficulty in initiating sleeping, maintaining sleep, or early morning awakening.[2] More than half of the older adults suffer from insomnia, and these people are often untreated.[3] The annual incidence of insomnia in older people is reported to be 5 to 8 per cent.[4-6] So the sleep disorders which are more prevalent in older adults should receive clinical attention and treatment. Furthermore, the elderly, apart from the vagaries of ageing have comorbidities and are also subjected to Polypharmacy. These will compound felony of preexisting distorted sleep architecture further. Thus, it is imperative for a review of the sleep in elderly. This article is adumbrated down the path of Sleep physiology, Age related changes in the elderly, Comorbidities and Sleep, Polypharmacy and sleep, Preventive and therapeutic measures and conclusion.

1. SLEEP PHYSIOLOGY

Suprachiasmatic nuclei of the hypothalamus regulate the human sleep-wake cycle by regulation of the melatonin secretion which modifies circadian rhythm. This circadian rhythm is synchronized by external factors, such as light and food. Day after sleep loss, more sleep is needed to compensate, induced by homeostatic sleep pressure.

Normal human sleep was classically divided into rapid eye movement (REM) sleep and non-REM (NREM) sleep, consisting of three stages N1, N2, and N3 according to American association of sleep medicine.[7] Sleep starts with NREM while REM takes place after a short period of NREM sleep and this alteration occurs about four or five times during a normal night sleep.[8,9] The sleeper makes postural adjustments about every twenty minutes, heart rate and blood pressure decline in NREM sleep. REM sleep is characterized by a profound loss of muscle tone, and bursts of rapid eye movements.

2. AGE RELATED CHANGES IN SLEEP

The quantity and quality of sleep change profoundly across the lifespan.[10] New-born shows several sleep-wake cycles over 24 hours, with a total duration of sleep of 14 to

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ABBREVIATIONS: REM-Rapid eye movement; Nrem-Nonrapid Eye movement, SDB-Sleep dsordered Breathing, RBD-Remsleep Behaviour Disorder, Rls/Plms-Restless Leg Syndorme /Periodic Limb Movement In Sleep, AHI-Apnoea Hypopnoea Index, Osa-Obstructive Sleep Apnoea, AD-Amyloidal Degeneration, PND-Paroxysmal Nocturnal Dyspnoea, GERD-Gastroesophageal Reflux disease, BPH-Benign Prostatic Hypertrophy

15 hours per day. In young adults, average sleep duration is 7 to 8.5 hours, where as in old age overnight sleep is fragmented and lasts for less than 6 to 7.5 hours.

Sleep fragmentation happens in older people as they spend more time in lighter stages of sleep (N1 and N2),[11-14] resulting in their waking up several times during the night. Increased age in women is corelated significantly with the reduction in percentage of REM sleep, whereas in men it corelates with the reduction in percentage of slow wave sleep.[14] Older adults report early awakening in the morning, increased sleep onset latency, time spent in bed, night time awakenings, napping, and decreased total sleep compared to younger adults. Polysomnography support reports of such sleep disturbances.

3. SLEEP DISORDERS IN THE ELDERLY

The most common primary sleep disorders in the elderly population are: sleep-disordered breathing (SDB), REM sleep behaviour disorder (RBD) and restless legs syndrome/periodic limb movements in sleep (RLS/PLMS).

3.1 Sleep Disorderd Breathing (SDB)

In SDB,subjects experience complete cessation of breathing and/or partial or reduced respiration during sleep, which encompasses a spectrum of breathing disorders ranging from snoring to obstructive sleep apnoea. Each event lasts for more than 10 seconds and recurs throughout the night, resulting in arousals and desaturations in sleep, as diagnosed on polysomnography. Five or more events of apnoeas and hypopnoea per hour of sleep,otherwise called apnoea hypopnoea index (AHI), confirms the diagnosis of SDB.

AHI was found to be >5 in 81%, >10 in 62%, and >40% in 24% people in a large series of older adults (age 65-95).[15] Another sleep health study,[16] in 6,400 older adults (mean age= 63.5 years) showed SDB prevalence rates of 32% for AHI 5-14, and 19% for AHI >15 in 60-69 years old people, 33% for AHI 5-14 and, 21% for AHI >15 in 80-98 years old people. These numbers are significantly higher when compared to middle aged adults, whose SDB prevalence with AHI >5 were 4% in men and 2% in women.

Old age, male gender, obesity were the risk factors associated with SDB along with use of sedative medication, alcohol consumption, family history, race, smoking and, upper airway anatomy.

3.2 Periodic Limb Movement in Sleep (PLMS)/ Restless Leg Syndrome (RLS)

Subjects with restless leg syndrome experience dysesthesias in the legs, characterized by “pins and needles” or a “creepy and crawly” sensation in the legs which get relieved with movement. The diagnosis is made based on the history. Prevalence of RLS increases with age and is doubled among women compared to men.[17] Most of the patients with RLS also have comorbid PLMS, and vice versa.

PLMS is characterized by leg jerks causing brief arousals and/or awakening occurring approximately every 20-40 seconds over the night.

PLMS is diagnosed when patient is having at least 5 jerks per hour of sleep associated with arousal during an overnight sleep recording. PLMS prevalence is 45% among older adults compared to that of 5-6% in younger adults.[18] Dysfunction of the dopaminergic system and iron deficiency are thought to be involved in its pathogenesis.[19,20]

Patients report an inability to fall or stay a sleep,[21] associated with daytime sleepiness, fatigue and impairment in daytime functioning.[22]

3.3 Rapid Eye Movement Sleep (REM) Behaviour Disorder (RBD)

REM sleep behaviour disorder is a parasomnia, characterized by complete mototric behaviour that occurs during REM sleep. It is likely the result of intermittent lack of the skeletal muscle atonia typically present during REM phase of sleep. Unpleasant and vivid dreams are also a common symptom.[23,24] This disorder include walking, sleeping, eating and may harm the patient or bed partner and may be more violent. RBD is most prevalent and typically diagnosed among older adult males of ages of 55-58 years.[25,26]

Many patients with RBD are also subsequently diagnosed with neurodegenerative disorders, such as Parkinson’s disease, dementia with lewy bodies, and multiple system atrophy.27Definitive diagnosis of RBD requires PSG.28 Differential diagnoses includes obstructive sleep apnoea (OSA), sleep walking, sleep terrors, nocturnal frontal lobe epilepsy, periodic limb movement, arousals associated with confusion, dissociative states and malingering.[29]

3.4 Insomnia

Insomnia is one of the most prevalent sleep complaints reported by older adults, characterized by difficulty in initiating and maintaining sleep. American academy of sleep medicine defines insomnia, as a persistent difficulty with sleep initiation, duration, consolidation, or quality that occurs despite adequate opportunity and circumstances for

sleep, and results in some form of daytime impairment. Studies report 40-50 percent of adults to have disturbed sleep over the age of 60.30 There is a wide variation in reports from different parts of the world regarding the prevalence of insomnia in the elderly population.[31-47]

The 2004 national health interview survey in the USA showed that 20-25 percent of elderly men and women sleep less than 6 hours.[33] Subtypes of insomnia include, sleep onset insomnia (difficulty initiating insomnia), sleep maintenance insomnia, early morning insomnia (early morning awakenings with difficulty in returning to sleep), and psychophysiological insomnia (behaviourally conditioned sleep difficulty resulting from maladaptive cognitions and/or behaviours). Most common types in older adults are maintenance and early morning insomnia. Insomnia puts the older adults at greater risk for falls, cognitive impairment, poor physical functioning and mortality even after controlling for medication.[34, 35]

3.5 Circadian Rhythm Disturbances

Changes in the circadian rhythms, that control many physiological functions, can also contribute to sleep disturbances. Deterioration of suprachiasmatic nucleus occurs with the ageing, resulting in less synchronized sleep-wake circadian rhythms due to decreased response to external factors.[36] This results in less consistent periods of sleeping and waking across the 24 hours day.

Factors likechanges in core body temperature cycle, decreased light exposures and genetic predisposition may result in Circadian rhythm advancement causing patients to become sleepy early in the morning.

3.6 Sleep and Menopause

Sleep disturbance is one of the hallmark symptoms of menopause, with approximately 25 to 50 percent of women undergoing menopause reporting sleep complaints compared to approximately 15% of general population.[36] Sleep architecture disruption in menopausal women is associated with vasomotor symptoms, such as hot flashes. Decreased estrogen in menopause may also be associated with hot flashes, and thus increased arousals. Evidence suggests that estrogen is associated with increased sleep time and decreased sleep latency, night time awakenings, and arousals.[55] A study by Okatani and colleagues37reported that postmenopausal women with insomnia have lower levels of melatonin compared to their cohorts.

4. SLEEP AND COMORBIDITIES

Sleep architecture is distorted in the elderly both in males and females, as seen above, worsened by the impending comorbidities.

4.1 Dementia

Sleep and dementia have a bidirectional relationship, as sleep disruption represents both a risk factor for, and symptom of neurocognitive syndrome.38,39,40Problems including both short and long sleep duration, insomnia, OSA, impaired circadian rhythm and sleep quality, were associated with increased relative risk of preclinical Amyloidal degeneration (AD), cognitive impairment and AD.[40.41]

Patients with AD can have increased sleep onset latency, and reduced time spent in restorative slow wave sleep and REM sleep.[41]

4.2 Chronic Respiratory Disorders

Impaired sleep quality has been associated with chronic obstructive pulmonary disease and asthma and lower oxygen saturations has been observed.[42,43]

4.3 Depression

Older patients with depression and sleep also have bidirectional relationship, with sleep disturbance representing both a risk factor for, and symptom of depression.[44]

4.4 Heart Failure

Increased sleep onset latency, night time arousals, early morning wakening, non-restorative sleep, SDB etc. are common in patients with heart failure. Sleep disturbance is associated with several cardiovascular risk factors, and cardiovascular diseases in addition to higher sympathetic activity.45In contrast, nocturnal symptoms of heart failure, such as orthopnea and PND may exacerbate sleep disturbance.[45]

4.5 Gastro-Esophageal Reflux Disease (GERD)

.Nocturnal GERD symptoms affected sleep in 75%, inability to sleep throughout the night in 42%, and sleeping in a seated position in 34% in a study.[46]

4.6 Nocturia

Severity of nocturia has been associated with poor sleep quality and decreased sleep duration.[47,48] Nocturia may be caused by prostatomegaly (BPH), which is common in old age, primary or secondary detrusor overactivity and impaired bladder contractility.48In addition, excess fluid intake, diabetes and, hypercalcaemia are associated with nocturia.

These multiple comorbidities are associated with multiple drug intake or polypharmacy.Presence of two or more long-term health conditions is called as multimorbidity as per the definition of WHO.49Because of multimorbidity, polypharmacy is prevalent in geriatric population which is a major preventable contributor to sleep disorders, morbidity, and mortality.[50]

Polypharmacy in a managed care setting presents a unique set of challenges and opportunities 51as it is more prevalent and a less addressed issue.

. Though standard definition is lacking, routine use of five or more medications is often called polypharmacy according to WHO.

A systematic review of 44 studies conducted worldwidein long-term care facilities reported a 38.1–91.2% prevalence of polypharmacy, with use of ≥5 medications. When considering ≥ 9 medications, the prevalence ranged from 12.8–74.4% and 10.6–65.0% when ≥ 10 medications were considered.[52]

A prospective surveillance study from two teaching hospitals in India done over 814 hospitalized patients aged ≥60 years reported 45.0% and 45.5% of patients to experience polypharmacy and high-level polypharmacy respectively .[53]

The number of drugs increases in relation to the multiple chronic conditions; such as diabetes mellitus, hypertension, chronic obstructive pulmonarydisease, and heart failure.

Unfortunately, there are many negative outcomes; such as high expenses on healthcare, adverse drug events, drug- drug interactions,increased risks of inappropriate medications, non-adherence to medication and geriatric syndromes associated with polypharmacy.[54]

5. POLYPHARMACY AND SLEEP DISORDERS

The use of prescription medications, over-the-counter medications , dietary supplements, traditional and complimentary medicines are on the rise in geriatric population.[52]

A recent study of adults aged 62-85 years found that 88% used at least 1 prescription medication, 38% used over-the-counter medications, and 64% used dietary supplements.[55]

In older adults using activating medications insomnia symptoms are common. On the other hand, sedating medications such as antihistamins, anticholinergics, anticonvulsants cause daytime drowsiness in patients with chronic illnesses or sleep apnoea.[56,57,58]

Medications such as pseudoephedrine, beta agonists, corticosteroids, antidepressants, methylphenidate or selegiline can have activating or stimulating effects on older adults.[57, 58]

Antidepressants can worsen restless leg syndrome and periodic limb movement symptoms, while opiates or benzodiazepines are known to exacerbate sleep disordered breathing.

Certain beta blockers have been shown to suppress melatonin secretion and increase sleep fragmentation. Others can worsen parasomnias, induce REM sleep behaviour disorder, or change the amount of time spent in REM sleep.[57]

Selective serotonin uptake inhibitors and tricyclic antidepressants may impair sleep continuity, total sleep time, and cause nightmares or especially vivid dreams, 58

A final factor to consider is whether a medication might be interfering with sleep by worsening other conditions or causing sleep disruptive symptoms. Several examples of such effects include medications that worsen heart failure, have diuretic effects, create bothersome coughing, or cause nocturnal hypoglycaemia.

A study of 5213 participants in England found the rate of falls was 21% higher in people taking 4 or more medications compared with those taking fewer. Using a 10-drugs threshold, there was an increase in rate of falls by 50%.[58]

5.1 Substance use and Sleep

Alcohol consumptionmay decrease sleep latency, but increases arousals leading to poor quality and shorter sleep duration.58 it can also exacerbate sleep disordered breathing by decreasing pharyngeal muscle tone58; on the other hand, the stimulating effect of caffeine can increase sleep latency and number of arousals, leading to shorter sleep duration.[58]

Nicotine in Tobacco is a potential mediator which promotes wakefulness via an effect on acetylcholine transmission in CNS59 without a causal relationship be.

6. OTHER CONTRIBUTING FACTORS

Beyond treatment guidelines [which includes prescribing multiple drugs to treat chronic diseases such as diabetes mellitus and hypertension], there are health care contributors to the problem polypharmacy, including multiple prescribers; multiple pharmacies; accessibility to drugs online, in storesand in herbal shops.

7. HEALTH SYSTEM APPROACH TO POLYPHARMACY

A WHO report, The World Medicines Situation, estimated in 2004 that half of all medicines are inappropriately prescribed, dispensed, or sold.The necessary requirements to address this issue are good quality and economically sustainable prescriptions; public awareness about the problem; periodical review of prescriptions and deprescribing by using tools like STOP/ START criteria.

7.1 Common Medications

BENZODIAZEPINS are helpful in sleep initiation, muscle relaxation and stress relief. Decreasing limb movements, reduced oxygen desaturation witha usual dosage of 0.5 mgs taken one hour before the bedtime.60The sedated elderly may have tendency to fall in the nights, especially in the washrooms.

AMITRIPTYLINE has multifarious benefits by causing pharyngeal muscles relaxation, prolonging the sleep duration, ensuing the REM phase and effective antidepressant. This has atropine like action with side effects like drymouth etc.[61]

7.2 Curcumin

It is supposed to be the ancient wisdom of the east with modern medicine in vitro and vivo. Nano enhanced curcumin has antioxidant activity preventing metal induced aggregation of beta-amyloidalformation, destabilizing preformed beta amyloidal fibrils and accumulation of the same, which blocks the interneuron transmission.[62] It can cause Inactivation of the transcription factor NF-kB (nuclear factor-kappa B)-inhibiting the activation of the inflammatory compounds COX-2.[62] Over and above cur cumin can act as a powerful cytokine modulator down regulating the expression of several pro-inflammatory cytokines like TNF-a, IL-1,IL-2, AND IL-8. Thus, because of anti-inflammatory action, it can be an acceptable sleep medicine and even preventing the degenerative diseases.

7.3 Natural ways or non-morphological methods

The modern man spends 90% time indoors only, resulting in low energy, depression, physical health problems, altogether culminating in irregular sleep. Harvard Medical School found that exposure to sunlight for 90 minutes or 10000 Lux from light emitting box for 20 minutes can cause good sleep in elderly. This will help in prevention of melatonin production in the day time, reducing day time sleepiness and fortifying circadian rhythm.[63]

CONCLUSION

Polypharmacy is a significant problem faced by geriatric population due to multi-morbidities causing significant sleep disturbances related morbidity and mortality

It is preventable by multimodality approaches, aimed programmes, strengthening health care system, deprescribing, counselling, adopting natural methods and sleep study.

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Challenges in Anaesthesia Management and Management of common Complications during Total Hip Replacement in Geriatric Patient

Surekha S Chavan

ABSTRACT

In elder patient hip fractures due to falling is important cause of death, about 15-30% of patients with hip fracture die in a year. Orthopedic surgeries of hip and femur are most common surgeries due to falls and osteoporosis in elders. Physiological changes due to aging, multiple comorbidities, polypharmacy and occurrence of perioperative complications during hip surgery e.g. surgical blood loss, bone cement implantation syndrome, pulmonary embolism, and fat embolism; increases the risk of perioperative mortality and morbidity in elders. Thorough preoperative evaluation of elder patients, appropriate selection of anesthesia techniques, vigilant monitoring for perioperative complications and postop pain control is key to success during hip arthroplasty.

Keywords: *Anesthesia management, Complications, Geriatric, Hip arthroplasty,*

DEMOGRAPHY BACKGROUND

Elder population > 60 yrs of age is projected to rise from 8% (2015) to 19 % (2050) and by 2100 it will rise to 34 % of the total population in India. Geriatric population has surgical rate 2-3 times more than younger and 25-35% geriatric population will be occupying operation tables of total surgeries. Orthopedic surgeries of hip and femur are most common surgeries due to falls and osteoporosis in elders. Apart from physiological changes due to ageing, elders are associated with multiple comorbidities; which increases risk of perioperative mortality and morbidity. There is necessity of trained anesthesiologist and hence in future Most Anesthesiologists will be Geriatric Anesthesiologists!!!

INTRODUCTION

There is increased patient variability in geriatric patients due to mechanism of basic physiology of ageing. Onset and rate of progression of changes varies individual to individual resulting into heterogeneous geriatric patients for anesthesia management. Hence All Geriatric Patients are not Equal!

Increased life expectancy has increased the incidence of hip fracture in advanced age. This is due to, decreased bone density, osteoporosis, and decreased balance resulting in falls. Hip fractures due to bone metastasis are highly neglected. In elder patient hip fractures due to falling is important cause of death, 30% of these are above 85 years of age. About 15-30% of patients with hip fracture die in a year. Geriatric patients who undergo hip surgery are frail, associated with multiple co-morbid diseases, polypharmacy and risk of surgical blood loss.

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PHYSIOLOGICAL CHANGES IN ELDERS AND ITS ANESTHESIA IMPLICATIONS

1) Central Nervous System: there is decrease in brain mass & Grey matter shrinkage. Hippocampus and sub-cortical white matter decrease (15%), ventricular size increases. CBF, CMR, electrical activity is unchanged. Decreased number of receptors- Ach, DA, NA, 5HT3. Glutamate unchanged.

Anaesthesia Considerations: There is increased sensitivity to anaesthetic drugs. Decreased Nerve root fibres & Schwann cell distance, it increases conduction velocity & block sensitivity. The epidural space & CSF is decreased, leads to increased dural permeability for drugs.

2) Cardiovascular System: I. Vascular Ageing: There is loss of elasticity of vessels, and reflected pressure from stiffened arteries increases pressure in aortic root

during late systole, eventually result in ventricular hypertrophy and impaired diastolic filling.

II. Decreased Venous Compliance: In elder people veins stiffen with age like arteries and stiff veins are less able to "buffer" changes in blood volume. Geriatric patients are more sensitive to hypovolemia. Volume shifts cause exaggerated changes in cardiac filling pressure.

III. Myocyte Death: Cardiac muscle cells die over time, remaining cells hypertrophy to compensate which causes ventricular hypertrophy. SA nodal cell number is reduced with fibrotic infiltration of conduction system.

IV. Ventricular Contraction- Slows with Ageing, Ventricle may not be fully relaxed during beginning of diastolic filling phase due to myocardial fibrosis. This leads to early diastolic filling impairment and preload depends on atrial kick.

V. Dependence on High Filling Pressure

VI. Consequences of Delayed Relaxation- Late diastolic filling depends on high left atrial pressure and atrial kick, Tachycardia and atrial fibrillation are not well tolerated. There is narrow range between inadequate filling pressure and fluid overload (preload sensitive). Diastolic dysfunction is most common cause of heart failure in > 75 yrs of age!!!

VII. Decreased Beta-Receptor Responsiveness- In response to stress, the elderly demonstrate diminished increase in heart rate.

Hemodynamic Response to Anesthesia

Anesthetic agents can remove sympathetic tone, directly depress heart & vascular smooth muscles, diminished baroreceptor reflex. Changes in sympathetic tone can be due to surgical stimulus, variable depth of anesthesia & patient's volume status.

Volume Dependence of Elderly Heart

- Elderly heart depends on late filling that in turn depends on left atrial pressure
- Elderly heart is also stiff, so the left atrial pressure must be high in order to fill the LV
- Prone to diastolic dysfunction

3) Pulmonary Changes: Intrapleural pressure increases because of increased lung compliance & stiff thorax; there is decreased height & calcification of vertebrae, diaphragmatic flattening, higher work of breathing, Decreased CNS responsiveness causes blunted response to hypoxia & hypercapnia. Lung Volumes - Decreased VC, Increased RV, Increased FRC, Increased dead space. Increased CC > FRC, leads to V/Q mismatch & shunting, Flow Rates altered (due to loss of muscle with weakening).

Decreased Efficiency of Gas Exchange

It is due to breakdown of elastins between connective tissue and alveolar tissue, Poor tethering of lung tissue to airways permits atelectasis and increases risk of hypoxia and pneumonia. Smaller airways become collapsible. This increases shunting and dead space.

Pre-Oxygenation before Anesthesia is must for the elderly patient & it takes longer!!

Challenges in Airway in Geriatrics: Diminished Receptor sensitivity: Stimulus threshold for vocal cord closure is increased with blunted cough, laryngeal & pharyngeal reflexes. Also gag reflex is weak with loss of OG sphincter tone. These changes lead to increased risk of aspiration! Arthritic Changes: needs gentle and nontraumatic handling of upper airway due to decreased cervical spine and neck mobility and restricted mouth opening. Fragile teeth, Altered facies, TM joint dysfunction, loose teeth.

Considerations & Precautions during Intubation

Use smaller endotracheal tube due to smaller glottis opening, Cervical arthritis- Avoid neck overextension, prevent stress response due to laryngoscopy & intubation.

Always Remember Geriatric patients have risk of respiratory failure postoperatively as, Prone to airway collapse (risk of pneumonia), Lower blood oxygen levels (greater need for supplemental oxygen), In PACU, hypoxia more likely from residual drug/CNS effect.

Renal Changes: Glomerulosclerosis leads to decreased GFR (10% per decade). Serum Creatinine is poor indicator due to decreased muscle mass. Renal blood flow & drug clearance is reduced. Electrolyte changes – Reduced sodium conservation capacity, Reduced rennin and aldosterone + raised ADH= Hyperkalemia & Hyponatremia, Hypocalcaemia due to Osteoporosis, Subclinical hypothyroidism-increased TSH, Glucose intolerance

Liver And Billiary Tract: Decreased liver Wt. and Bl. Flow by 20 %, no change in liver function tests, catalytic enzymes activity decreased, drug metabolism slowed, synthesis Of Protein Binding & Coagulation Factors is decreased.

Haematological and Immune System: Reduction in bone marrow production & spleen size, Stem cells ageing, erythropoietin resistance and predisposed to anaemia & immunosenescence.

Musculoskeletal System: Decreased muscle mass and bone density. Osteoporosis causes fragile bone fractures. Increased incidence of Osteoarthritis, Osteomalacia, RA,

and Gout. Degenerative joint disease.

Loss of disc spaces leads to difficulty in regional anaesthesia techniques. Elders are prone to injuries due to various special surgical positioning.

Integumentary System: Fragile skin, decreased skin elasticity/subcutaneous fat- difficult IV access, Prone for pressure ulcer due to dehydration, poor circulation and nutrition. They need extra care during positioning and padding and proper venous access

Gastrointestinal System: Gastric acidity increased & GERD, malnutrition, loss of appetite, indigestion, Decreased colon motility leading to fecal impaction and constipation.

PHARMACOLOGICAL CHANGES

Pharmacodynamics -reduced receptors number & sensitivity and postreceptor transduction. Geriatrics have increased brain sensitivity to anaesthetic drugs.

Changes in pharmacokinetics

Intravenous Induction Agents: Thiopental: Peripheral vasodilatation +blunted baroreceptor reflex = profound BP drop. Elimination delayed, T1/2 =13-25hrs (6-12hrs in young), hence 25-75% dose reduction. Etomidate-hemodynamically stable, 50% dose reduction needed. Propofol: Greater blood pressure fall than Thiopental, hence inject slowly. Less post anaesthetic mental impairment, 30-40% dose reduction, induction dose 1.2-1.7mg/kg.

Inhalational Anaesthetics: V/Q mismatch –reduced rate of action. Reduced Cardiac Output leads to rapid onset of action. Lipid soluble agents-Increased Vd (fat)-prolonged recovery (isoflurane 7 min). Lower lipid soluble Sevoflurane, Desflurane -rapid depth control -faster emergence. Minimum Alveolar Conc. reduced 6%/decade after 40yr age.

Neuromuscular Blockers (Muscle Relaxants): Senile motor neuron degeneration with muscle atrophy. atypical extrajunctional Ach-receptors proliferation –thickening and spread of post junctional area, compensate for reduced motor end plates- dose unchanged. Scoline sensitivity is more due to reduced cholinesterase levels. Recovery from Suggamedix is delayed (Table 1).

GOAL OF ANAESTHESIA MANAGEMENT IN GERIATRICS

- Return to previous level of activity
- Preserve Independence
- Complete functional and cognitive recovery (ADL)
- Prevent disability

OBJECTIVES during preoperative evaluation of geriatric patient: Assess for following,

1. Personal history, mode of injury, other injuries, habits, past hospital/ICU admissions, physical examination, Age, BMI, vitals, review of medications, laboratory tests.
2 Information about Artificial dentures, hearing aid, pacemaker etc
3. Osteoarthritis/ rheumatoid arthritis. Cervical/ lumbar spine examination,
4. TMJ, Airway assessment, preparation for difficult airway, Regional/General Anesthesia
5. ASA physical status
6. Information of Adequacy of remaining organ reserve,

Table 1

Drug	Pharmacokinetic	Pharmacodynamic
Thiopentone, Etomidate,	Decreased initial Vd, Decreased clearance (T1/2-13-25hrs Vs 6-12 in young)	
Propofol, Midazolam	Decreased clearance	Increased brain sensitivity
Morphine (morphine 6 glucouronide)	Decreased clearance	Reduced renal clearance Increased brain sensitivity
Fentanyl, Sufentanyl, Alfentanyl		Increased brain sensitivity
Remifentanyl	Reduced central compartment volume	Increased brain sensitivity
NM blockers	Prolonged action of drug undergoing hepatic metabolism Dose unchanged	
Neostigmine	Dose unchanged	More cardiac adverse effects

neurological assessment, renal assessment, glycemic control, nutrition, pain education and treatment.

7. Functional status assessment- comprises:
- Activities of daily living (ADL) & Instrumental activities of daily living (IADL)
- Breath holding time, Pulmonary function test, Metabolic equivalents (METs),
- Cognitive assessment, Confusion assessment method (CAM score)

8. Age related diseases -Perioperative risk is proportional to medical comorbidities + reduced functional reserve. e.g. IHD, diabetes, stroke/TIA, focal deficit. Cognitive dysfunction, atypical disease presentation, Malnutrition, Immobility, Dehydration, Chronic pain,
9. Frailty- 6-9% incidence, Criteria- Weight loss >10lb/yr, Exhaustion, Decreased physical activity, walk time, hand grip. Symptoms-weakness, wasting, malnutrition, poor exercise tolerance, unstable gait

ANESTHESIA MANAGEMENT: Premedications -Lower doses, Opioid valuable, Avoid Anticholinergic. Use Benzodiazepine, H2 antagonist & metclopramide, DVT prophylaxis. Careful positioning, vigilant monitoring. Prefer shorter acting drugs.

Go low & Go slow! You can always give more if necessary! (Table 2)

Anesthesia techniques:

General Anesthesia- It is safest for restless/agitation due to shock and pain due to trauma to remain immobilized on operating table. Better haemodynamic control with higher O2 concentration than RA. Avoid premedication that causes respiratory depression, complete neuromuscular block reversal before extubation, postoperative analgesia.

Regional Anesthesia (SA/EA) - Both techniques are widely used. They have advantages of early mobilization postoperative pain relief, reduced risk of DVT/Hypoxia, preserved Consciousness.

Peripheral Nerve Block (PNB) - if SA, EA is contraindicated, fascia iliaca block, femoral nerve block, sciatic nerve block can be applied. They provide postoperative analgesia and do not have sympathetic blockade or sudden hypotension.

Advantages of PNB- Protection of consciousness, unaffected respiration. provides unilateral blockade, less sympathetic blockade than RA, USG used in PNB-reduces dose, blockage application time

Management of Perioperative Complications due to Hip Arthroplasty (THR), having high Morbidity/Mortality

I. HEMORRHAGE:

Average surgical blood loss during THR is about 1-1.5L. To Reduce Blood Loss, use controlled hypotension; SA/EA causes 30-50% less blood loss than GA. Using autologus/allogenic Blood Transfusion, Tranaxemic acid may prevent blood loss without causing thromboembolism.

ii. BONE CEMENT IMPLANTAION SYNDROME:

Clinical signs are: Hypoxia (increased pulmonary shunt), Hypotension, Arrhythmia, Decreased cardiac output, Pulmonary HTN (increased pulmonary vascular resistance), Embolism commonly associated with femoral prosthesis placement.

Methods to reduce bone cement related complications: By increasing FIO2 before cement procedure, vigilant monitoring of vitals and fluid balance, opening a hole in the distal end of femur(vent hole), cleaning femoral shaft with high pressure lavage to remove debris, and using non cemented prosthesis.

Advantages of Regional Vs General Anaesthesia

Regional Anaesthesia (RA)	General Anaesthesia (GA)
Less chance of DVT, PE	More chances of DVT, PE
Less blood loss	More blood loss, needs blood transfusion
Maintain airway & pulmonary function	Airway handling and related complications
Less risk of hypoxia	More risk of post op hypoxia
Opioid sparing	Opioid needed, respiratory depression risk
Post Operative Cognitive Dysfunction	Post Operative Cognitive Dysfunction
Altered landmarks & difficult positioning	Difficult airway, pressor response

iii. THROMBOEMBOLISM:

Risk factors: in patients with past h/o thromboembolism, varicose surgery, orthopedic surgery, Advanced age, Malignancy, Congestive heart failure, Prolonged immobilization, Obesity, use of OC pills and estrogen, non indicated blood transfusion.

How to Reduce Thromboembolism: Neuraxial anesthesia reduces incidence of thromboembolism (DVT). Use of intermitant compression leg devices and low dose anticoagulant prophyaxis also reduce the risk. DVT is responsible for 90% PE.

Diagnosis: CXR, ECG, ABG. Hypoxemia, hypocapnia, respiratory alkalosis, increased alveolar–arterial gradient. CPKMB, D dimer, FDP, SGOT are Normal. LDH, bilirubin high. Physiological dead space and tidal volume ratio increased.

Management- Ventilation with 100% O2, heparinisation, ionotropic agents, surgical embolectomy/ anticoagulation. Prevention methods- avoid venous stasis, leg elevation, early mobilization postoperatively.

iv. VENOUS AIR EMBOLISM (VAE)

Earliest sign is cardiovascular collapse. Blood pressure decreases disproportionately; sudden hypotension, tachycardia, arrhythmia, cardiac arrest occur in succession. CVP increases, a metallic sound heard on auscultation (mill wheel murmur). Increased respiratory rate, irregular breathing, apnea can be seen. Diagnosis – PAP, ETCO2, Doppler ultrasonic flow meter.

v. FAT EMBOLISM

Etiology: bone fractures, bone surgery. Diagnosis- Free fat in sputum /urine, petechiae, Hyperlipidemia, with maximum level of free fat in 3-4 days .Treatment- hypovolemia and shock should be treated.

POSTOPERATIVE CARE: aims for early detection and treatment of common complications.

Pain management: multimodal approach by using epidural analgesia, local wound site infiltration, NSAIDs preferred. Intraoperative analgesia with EA catheters, patient controlled analgesia (PCA) - via peripheral IV or epidural/PNB catheter. Periarticular injection intraop.– Bupivacaine 0.5% 100-200mg, morphine 4-10, methylprednisolone 40 mg.

Hydration: fluid supplementation based on strict input output chart. Sedation: low dose opioids or BZDs to reduce incidence of post op cognitive dysfunction and ICU psychosis. Aspiration prophylaxis: Antacids, 5HT3 blockers. Ulcer prophylaxis: Proton pump inhibitors. resume oral feeds early. Antibiotics, DVT prophylaxis: resume anticoagulants, early ambulation, compression stocking. Hypothermia: prevention/treatment.

Vigilance for- Desaturation (hypoxia), Sleep disordered breathing, Urinary retention, Sepsis, DVT, Hypotension, Arrhythmia, Postoperative CVA, Acute renal failure, Iatrogenic complications e.g ADR, dehydration, Postoperative Delirium, Mild POCD, Depression.

CONCLUSION

Thorough preoperative evaluation of elderly patients should include evaluation of coexisting morbidities & atypical symptoms, polypharmacy, organ based functional evaluation. Appropriate selection of anesthesia techniques, drug doses, vigilant monitoring, postop pain control, cognitive function along with vigilant monitoring and prompt management is important for complications due to hip arthroplasty as they are having high morbidity/ mortality.

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Frailty Scores in Clinical Assessment

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ABSTRACT

Frailty is defined as that condition when a person loses the ability to carry out important, practiced social activities of daily living when exposed to either psychological or stressful conditions. There are several types of frailty scoring, out of which the most commonly used scale in clinical practice is 'Clinical Frailty Scale'. The most widely used tool in the scales is the Gait Speed Test. Frailty assessment is important in prognosis, morbidity and mortality of diseased geriatric population and hence, if regularly used in clinical practice, improves the treatment process and the outcome of geriatric patient care.

Keywords: *Frailty, Frailty Scale, Comprehensive Geriatric Assessment*

Frailty is defined as that condition when a person loses the ability to carry out important, practiced social activities of daily living when exposed to either psychological or stressful conditions. Objectively defined by Linda Fried[1] and colleagues as inclusion criteria of weight loss, exhaustion, weakness, walking speed and low physical activity. Rockwood *et al* defined frailty as an increasing number of disabilities which heralds the beginning of a cascade that leads to functional deterioration, hospitalization, institutionalization and death.

TYPES

1. PHYSICAL FRAILTY PHENOTYPE OR FRIED'S FRAILTY SCALE

It has five core domains which are Slowness, Weakness, Low physical activity, Exhaustion and unintentional weight loss.[1] Patients meeting one or two criteria are considered as pre-frail, and those meeting three or more are considered frail.

2 SHORT PHYSICAL PERFORMANCE BATTERY

It measures a series of three timed physical performance tests such as Gait speed, Chair rise and Tandem balance.[2] Performance on each test is scored from 0 to 4, with a total score ≤5 (of a possible 12) indicating frailty. It is relatively simple, cheap and takes approximately 10 minutes to complete, however it is difficult to administer in acute situation.

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3. FRAILTY INDEX OR DEFICIT ACCUMULATION INDEX

It is also known as the Deficit Accumulation Index (DAI) and considers frailty across multiple domains and may include physical, psychological and social components in addition to laboratory values.[3] The number of deficits identified in an individual is correlated with the level of frailty. The proportion of deficits over the number of items evaluated is expressed as a fraction, and an FI >score 0.25 is usually considered as frail.

4. SURVEY OF HEALTHY AGEING AND RETIREMENT IN EUROPE

FRAILTY INDEX

It is based on the Fried criteria and evaluates exhaustion, appetite, ambulation, resistance, physical activity and handgrip strength measurement.[4] It is easier to measure than the original Fried scale, because the questionnaire can be easily completed at the bedside and does not require the measurement of gait speed.

5. TILBURG FRAILTY INDICATOR

It is a multidimensional structured questionnaire that evaluates the physical, psychological and social domains.[5]

It consists of two parts. Part A has 10 questions on frailty determinants (age, sex, marital status, education level, social circumstances and lifestyle). Part B has 15 frailty elements across three domains: Physical, consisting of eight items (physical health, unintentional weight loss, difficulty in walking and problems with balance, hearing, vision, hand strength and physical tiredness), Psychological, consisting of four items (cognition, depression, anxiety and coping), Social, consisting of three items (living alone, social relationships and social support). Each item in Part B scores 1 point, and patients are considered frail if they score at least 5 out of a possible 15.

6. CLINICAL FRAILTY SCALE

The Clinical Frailty Scale (CFS) was designed for the Canadian study of health and ageing (CSHA) and can be readily administered in most clinical settings.[6,7] It is based on fitness, active disease, activities of daily living (ADL) and cognition, and the expanded scale ranges from 1 (very fit) to 9 (terminally ill). Because assessment relies upon the subjective judgement of a clinician, the measure is prone to interobserver variability.

7. EDMONTON FRAIL SCALE

It is a multidimensional scale, comprising 10 domains with 17 potential deficits covering cognition, general health status, functional independence, social support, medication use, nutrition, mood, continence and functional performance.[8] It includes the clock test for assessment of cognitive impairment, and the Timed Get Up and Go (TUG) for balance and mobility. The cut-off point for frailty is 12 or more deficits. It is a rapid screening tool for the non-geriatric specialist.

8. REPORTED EDMONTON FRAIL SCALE

It includes nine frailty domains: cognition, general health status, functional independence, social support, medication use, nutrition, mood, continence and functional performance.[9] It is based on self-reported functioning, and is appropriate in patients able to complete a questionnaire. Frailty is identified by a score of at least 8.

9. HOSPITAL FRAILTY RISK SCORE

It uses ICD-10 diagnostic codes from electronic healthcare records to identify frailty and includes more than 100 variables derived from routinely collected data and has been validated against both the Fried scale and other FI measures.[10]

10. FATIGUE, RESISTANCE, AMBULATION, ILLNESSES AND LOSS OF WEIGHT SCALE

The Fatigue, Resistance, Ambulation, Illnesses and Loss of weight (FRAIL) scale is a brief, interview-based screening tool. It is commonly used in the acute setting because it does not include items that are difficult to measure (e.g., walk speed, handgrip strength, stand-up test).[11]

11. COMPREHENSIVE GERIATRIC ASSESSMENT

It is a Gold standard for frailty assessment. It Involves a holistic, multidimensional and interdisciplinary assessment of an individual, culminating in the formulation of an individualised management plan.[12] Time consuming and is not part of the routine care of older people. Potentially useful brief screening tests include measuring 5 m gait speed, which is highly predictive of cardiovascular mortality, or handgrip strength.

Frailty can also be assessed as physiologic process[12-16] comprising of following characteristics:

1. **Weight loss**- Loss of more than 10 pounds unintentionally last one year.
2. **Exhaustion:** Felt last week that "everything I did was an effort" or "I could not get going".
3. **Slowness:** Time required to walk 15 ft (cut off depends on sex and height)
4. **Low activity level:** Expends < 270 kcal/week (calculated from activity scale incorporating episodes of walking, household chores, yard work etc)
5. **Weakness:** Grip strength measured using hand dynamometer (Cut off depends on sex and BMI).

CONCLUSION

The knowledge of a person's frailty status provides valuable information on the prognosis which may be useful in guiding informed shared decision making regarding treatment strategy. As discussed above, all the frailty scales are useful, and personal preference and ease of implementation determines the usage. Even though the Fried criteria and Frailty Index (FI) are the most commonly used in research, maybe the use of an easy and quick

scale, such as the Clinical frailty scale (CFS) and Fatigue, Resistance, Ambulation, Illness and Loss of weight (FRAIL), or one which is based on routinely collected data, such as the FI or Hospital frailty risk score (HFRS), may be more practicable in clinical practice. Currently, however, there is no agreement on the optimal frailty assessment tool.

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De-escalation in Geriatric Practice

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ABSTRACT

De-escalation includes communication, self-regulation, continual assessment taking appropriate actions and safety consideration. The goal is to reduce the risk of harm to patients, families and caregivers in Senior Care. One clinical aspect pertaining to drug safety in common situations as exemplified in Diabetes Mellitus, Coronary Artery Disease, COPD, Infection Control, and Psychiatry is discussed and significance underlined.

Keywords: *Therapy Modification. Geriatric Care, De-escalation situations.*

INTRODUCTION

Deintensification or de-escalation is a process to simplify, reduce or withdraw medications to avoid overtreatment in order to reduce the risk of polypharmacy and associated adverse events. Polytherapy / combination therapy has been the hallmark of modern therapeutics, be it an acute infection like pneumonia treated in ICU, diseases where natural course is progressive chronic disease like Diabetes Mellitus, condition with frequent and recurrent exacerbations such as chronic obstructive pulmonary disease (COPD) and progressive degenerative ones such as Alzheimer's, Dementia or Multiple Sclerosis. In response and as a consequence, "Treatment Intensification" and "Stepwise Escalation" tend to be recommended and unequivocally emphasized.

Over the last few years, evidence accumulated that simplification of complex treatment regimens and step-down therapy is required to be considered in certain cases. The elderly, with a relatively fragile physiology, degenerating organ function and proneness to drug toxicity, immune -senescence drawing infections frequently, co-morbidities adding to complex interactions in therapeutics / diagnostics / multimodalities of treatment. The increased risk from interventions inevitably become a distinct subset where the principles of 'De-escalation' become essential to be considered and applied judiciously, with due care and caution. This is not purported to be an exhaustive review, but an attempt to capture the essence of de-escalation with illustrative examples in geriatric practice. Needless to add, lifestyle modifications and adherence play a significant role for treatment to be effective and control established.

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DISEASE CONDITIONS AND CLINICAL SITUATIONS

Diabetes Mellitus

By and large, diabetes is the commonest metabolic disorder managed by Indian physicians, the control often is intermittent and frequently erratic. De-escalation approach in anti-hyperglycemic treatment should be considered in patients of T2DM a) after bariatric (metabolic) surgery, b) with significant weight reduction irrespective of its origin, c) with complex insulin regimens where re -evaluation of this treatment was missed, d) with continuously decreasing renal function, e) Among elderly persons with comorbidities, f) in social deprivation.[1]

Both ageing and Diabetes are recognized as important risk factors for functional decline and disability.[2] Generic metabolic targets with regard to glycemia, lipid levels or even blood pressure, ignore the importance of holistic personalized care in the presence of multi morbidity or moderate to severe frailty.[2] This frailty is now seen as major factor in the increased risk of death and disability in older people with Diabetes.[3] Existence and assessment of Frailty is important and vital.[4]

Chronic Obstructive Lung Disease

There is solid evidence from randomized clinical trials (RCTs) supporting the rationale for withdrawal from

inhaled corticosteroids (ICSs) in most patients suffering from chronic obstructive lung disease. While only a minority of severe COPD patients that are symptomatic and /or are at high risk of exaggeration may really need triple therapy, most patients should be de-escalated /switched from ICS containing regimen toward dual bronchodilator therapy, or even a single bronchodilator regimen in patients affected by less severe form of COPD.[5]

Coronary Artery Disease

In patients with acute coronary syndromes (ACS) with or without invasive treatment, Dual Anti Platelet Therapy (DAPT) is the treatment of choice. On de-escalation, there were no significant differences in ischemic or bleeding outcomes between de-escalation to clopidogrel or low dose prasugrel.[6]

Infections in ICU

De-escalation generally refers to a reduction in the spectrum of administered antibiotics through the discontinuation of antibiotics or switching to an agent with narrower spectrum, a reassessment of treatment when culture results are available.[7]

Trials alert us about the possibility that this strategy may be linked to a higher rate of reinfections but without an impact on mortality.

Psychiatry Disorders

De-escalation involves transferring a sense of calm and genuine interest in the patient's needs and wishes by using respectful, clear, limit setting. The goal is to build rapport and a sense of connection with the agitated patient.

To sum up. the concept of de-escalation requires to be considered and practiced in diverse set ups of clinical practice. Geriatric Medicine has a specific case in view of profile - comorbidities, altered physiology and frailty.

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News from Headquarters

Welcome executive committee of Geriatric Society of India for the year 2023-2024.

Patrons: Dr. P. S. Shankar, Dr. B. C. Bansal & Dr. V. K. Arora

President: Dr. A. K. Singh

General Secretary: Dr. O. P. Sharma

Jt. Secretary: Dr. Manoj Kumar Srivastava

Past President: Dr. Kaushik Ranjan Das

President Elect: Dr. Sajesh Asokan

Vice Presidents: Dr. J. K. Sharma, Dr. Atul Kulshrestha & Dr. Anand P. Ambali

Treasurer: Dr. Garima Handa

Committee Members: Dr. B. B. Gupta, Dr. Krishnanjan Chakraborty, Dr. Manisha Arora, Dr. Dominic Benjamin, Dr. H. K. Raogupta & Dr. P. V. Rao

Co-Opted Members: Dr. Rajiv Garg, Dr. (Col.) Pramod Kumar, Dr. Chinmoy Maity, Dr. Sachin Desai, Dr. Pradnya Diggikar, Dr. Puneet Khanna, Dr. M. S. Gudi, Dr. Anil Kumar Manchanda, Dr. Arunansu Talukdar, Dr. Kausik Majumdar, Dr. I. S. Jain, Dr. Purna Chandra Das, Dr. Mohit Sharma & Dr. Samudra Gooptu

Zonal Co-Ordinator: Dr. Aniruddha De (East), Dr. Sandeep P. Tamane (Central), Dr. Nikhil Sarangdhar (West), Dr. Anita Nambiar (South) & Dr. Parvaiz A. Kaul (North)

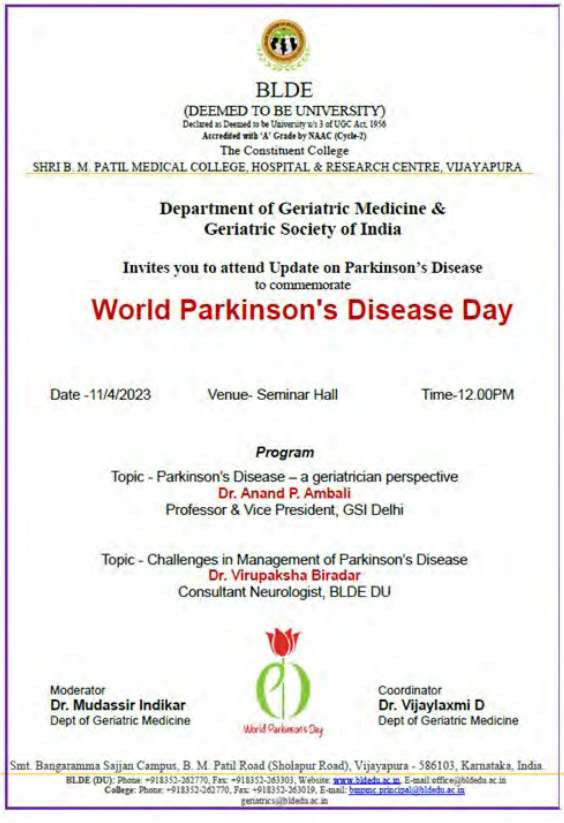
Advisors: Dr. Prabha Adhikari, Dr. H. L. Dhar, Dr. K. Satyanarayana, Dr. M. V. Jali, Dr. S. Ramanathan Iyer, Dr. Satish Gulati, Dr. N. S. Neki, Dr. S. N. Gaur, Dr. M. S. Sridhar, Dr. Vivek Handa & Dr. Agam Vora

Overseas Co-Ordinators - Dr. Renu Wadhwa (Japan), Dr. B. K. Mondal (United Kingdom), Dr. Lochana Shrestha (Nepal), Dr. Arvind Modawal (USA) & Dr. Subrato Ghosh (Bangladesh)

News from Vijayapura

WORLD PARKINSON'S DISEASE DAY

Department of Geriatric Medicine BLD Hospital Vijayapura observed World Parkinson's disease day on Tuesday 11th April 2023. On this occasion, Dr. Anand P. Ambali spoke about Parkinson's Disease - a geriatrician perspectives & Dr. Virupaksha Biradar, Neurologist spoke about the challenges in management of Parkinson's Disease. This was attended by staff members & students of BLDE Hospital.



BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

Department of Geriatric Medicine &
Geriatric Society of India

Invites you to attend Update on Parkinson's Disease
to commemorate

World Parkinson's Disease Day

Date -11/4/2023 Venue- Seminar Hall Time-12.00PM

Program

Topic - Parkinson's Disease – a geriatrician perspective
Dr. Anand P. Ambali
Professor & Vice President, GSI Delhi

Topic - Challenges in Management of Parkinson's Disease
Dr. Virupaksha Biradar
Consultant Neurologist, BLDE DU

Moderator
Dr. Mudassir Indikar
Dept of Geriatric Medicine

World Parkinson's Day

Coordinator
Dr. Vijaylaxmi D
Dept of Geriatric Medicine

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Solapur Road), Vijayapura - 586103, Karnataka, India.
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College: Phone: +918352-262770, Fax: +918352-263019, E-mail: bmpmc.principal@bldedu.ac.in
geriatrics@bldedu.ac.in

SENSITIZATION PROGRAM FOR INTERNS IN GERIATRIC MEDICINE

Sensitization program for Interns in geriatric medicine was organised virtually by the department of Geriatric Medicine for Interns from 25th March 2023 to 01st April 2023.

The program covered Geriatric medicine in 15 lectures by faculty of national repute from all over the country. Pre and post tests were also conducted and certificate of participation were awarded. The program was supported by Geriatric Society of India & Association of Physicians of India – Karnataka Chapter.

BLDE DU Shri B M Patil Medical College Hospital and Research Centre, Vijayapura 586103 Karnataka

Department of Geriatric Medicine, Geriatric Society of Inda, Delhi & Association of Physicians of India, KC

Online Sensitization Program in Geriatric Medicine for Medical Interns (27/3/23 to 01/4/23) 8PM to 9PM

	Topic	*Faculty*	*Moderator*	*Time*
Day 0	Inauguration, Pre-test		Dr Vigneshwaran S	30
Day 1	Clinical Approach to the Elderly	Dr A P Ambali	Dr Ravikeerthi M	40
	Communication with Elderly	Dr Mangala Borker	Dr Anita Basavraj	20
Day 2	Vaccines in Elderly	Dr O P Sharma	Dr A K Singh	20
	Caregiver role in Elder care	Dr Kaushik R Das	Dr Satish Gulati	20
	Diabetes in Elderly	Dr J K Sharma	Dr Purnachandra Das	20
Day 3	Elder Abuse	Dr A P Ambali	Dr Vijayalaxmi D	20
	Dementia in Elderly	Dr Prabha Adhikari	Dr N S Neki	20
	Various Government schemes for Elderly	Dr Sagar Borker	Dr Sachin Desai	20
Day 4	Role of Physiotherapy in Elderly	Dr Basavraj G Chandu	Dr Pradnya Diggikar	20
	Future Perspectives in Geriatric Medicine	Dr A P Ambali	Dr Vishwanath K	20
	Depression & Delirium in Elderly	Dr Alka Ganesh	Dr Sajesh Ashokan	20
Day 5	Approach to the elderly in Emergency Department	Dr Prem Narasimhan	Dr Md Abrar Ul Huq	30
	Care modalities in elderly care (Rehabilitation, Hospice)	Dr A P Ambali	Dr Tejaswini	15
	Palliative care in Elderly	Dr Pratibha Pereira	Dr Sangram Biradar	15
Day 6	Polypharmacy in Elderly	Dr Sandeep Tamane	Dr Mudassir Indikar	20
	Importance of Geriatric Medicine for Interns	Dr P S Shankar	Dr A K Arora	20
	Feed Back, Valedictory & Post test		Dr Kushal, Dr Harini	20

Dr.Rajeev Malipatil, HoD, Geriatric Medicine, geriatrics@bldedu.ac.in
Dr.Aravind Patil, Principal & DFoM, www.bldedu.ac.in
Dr.O. P. Sharma, General Secretary,GSI, www.geriatricsindia.com
Dr.Vishwanath Krishnamurthy, Hon.Secretary API KC, www.apikarnataka.org

News from Pune

A HEALTH CHECK-UP CAMP FOR ELDERLY

A Health Check-up Camp for Elderly was conducted by Department of Medicine at Dr. D. Y. Patil Medical College Hospital and Research Centre, Pimpri, Pune on the occasion of the birthday of our respected Chancellor, Dr P. D. Patil and Dr Smita Jadhav Madam on Friday 17th February 2023 in Medicine OPD.

Health check-up and following investigations were done free of cost: Haemoglobin Blood sugar random, ECG and bone densitometry by USG for 50 elderly citizens.

The camp was organized by Dr. Shubhangi Kanitkar, Professor & HOD, Dr. Shiddhapur, Professor & HOU, Dr. Nilesh Jagdale, Asst. Professor and residents from Department of Medicine.

INFLUENZA VACCINATION CAMP

Influenza vaccination camp was organized on 23rd March by Dr. Pradnya Diggikar at Dr. D. Y. Patil Medical College Hospital and Research Centre, Pimpri, Pune.

On this occasion a quiz was organized for Undergraduate students on the topic “Adult Vaccination”.

PG Students who participated in this program were Dr. Nelabhotla Saisatya Saranya, Dr. Nirali Chaudhary, Dr. Hanisini Raju Reddy, Dr. Mundada Mayank, Dr. Yammanuru Bhavyasri & Dr. Tushar Pancholi.

WORLD HEALTH ORGANIZATION DAY 2023 CELEBRATION

World Health Organization Day 2023 Celebration - Health for All was organized on 05th April 2023 by Dr. Pradnya Diggikar.

Diabetes awareness talk was delivered to The Patients, their relatives and undergraduate students. Demonstration of BSL testing and insulin administration were explained. Steps to take on improving lifestyle to prevent diabetes mellitus and its complications.

PG Students who participated in this program were Dr. Hanisini Reddy, Dr. Mayank Mundada, Dr. Arun Oommen, Dr. Yammanuru Bhavyasri, Dr. Tushar Pancholi & Dr. Sreevidya.

News from Eastern Zonal Branch

EXECUTIVE COMMITTEE OF GSI EZ BRANCH

General Body of GSI Eastern Zonal Branch on 11th April 2023 chaired by Chairman Dr Arunansu Talukdar have virtually approved the following Executive Committee of GSI eastern Zonal Branch for the year 2023-2025.

Patrons :
Dr. Amusana Singh ;L-755
Prof.Dr. Subhas Chandra Mahapatra ;L-996
Prof.Dr. Hari Shankar Pathak ;L-325
Advisors :
Dr.(col.) Promod Kumar : L- 1205
Dr. Aloke Dasgupta :L-799
Dr. Surendra Daga ; L-937
Dr.J.K.Mitra
Prof.Dr. Sanjeeb Kakoty ; L-470
Prof.Dr.Jayanta Panda
Chairman: Prof. Dr. Arunansu Talukdar
Vice Chairmen :
(1)Prof.(Retd.)Dr. Ashoke Das;L-1198
(2) Dr. Mrs. Taruni Ngangbam ;L-1055
(3) Dr.Purna Chandra Dash
(4) Dr.Ajay Kumar Bakhla
(5) Dr. Anuradha Deuri
General secretary: Dr. Kaushik Ranjan Das ; L-817
Joint Secretary: Dr. Pranjal Kumar Dutta ;L-684
Dr. Prasanna Kumar Rathor; L-969
Dr.Santosh Kumar Swain
Assistant secretary: Dr. Sudhir Kumar Gupta ;L-1182
Dr. Yogiraj Das
Dr. Shankha Shubhra Sen
Treasurer: Dr. Aniruddha De;
L-1160 ;Contact:+919830202538
Assistant Treasurer : Birja Prasad Biswal;L-1007

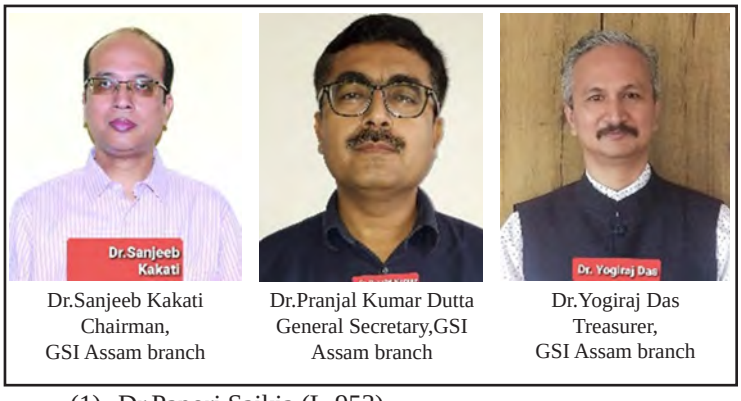
Committee Members :
(1) Dr.Thoidingiam Bijoy Singh;L-1054
(2) Dr.Manash Ghose
(3) Dr. Shyama
(4) Dr. Anirban Mohanta ; L-686
(5) Dr.Rakesh Kumar Jha;L-1222
(6) Dr. Ajit Narayan Deb ; L-895
(7) Dr. Amulya Kumar Das ;L-822
(8) Dr. Sudhir Ranjan Samal ;L-695
(9) Dr. Soumik Ghosh :L-1272
(10) Dr. Dhires Kumar Chowdhury,L-1263
(11) Dr. Soumi Chakraborty : L-854
(12) Dr. Tarun Kumar Das; L-1082
(!3) Dr.Rakesh Kumar Jha
AND Other Ex-Officio members.

News from Assam Branch

"GERIATRIC SOCIETY OF INDIA ASSAM BRANCH

"Geriatric Society of India Assam Branch" was installed on Tuesday 28th March 2023 with the following executive Committee

Patron: Dr. Ramaprasad Medhi (L-481), Dibrugarh.
Chairman: Prof.(Dr) Sanjeeb Kakati (L-471)
Vice Chairman:
(1) Dr.Alok Dasgupta (L-799)
(2) Dr.Anuradha Deuri (L-1427)
General secretary : Dr. Pranjal Kumar Dutta(L-684)
Assistant Secretary : Dr.Manash Ghose (L-1126)
Treasurer : Dr.Yogiraj Das (L-1407)
Committee members :



Dr.Sanjeeb Kakati
Chairman,
GSI Assam branch

Dr.Pranjal Kumar Dutta
General Secretary,GSI
Assam branch

Dr.Yogiraj Das
Treasurer,
GSI Assam branch

(1) Dr.Papori Saikia (L-953)
(2) Dr.Pradip Kumar Sarma (L-680)
(3) Dr. Anirban Mohanta (L-686)

News from Jharkhand Branch

GERIATRIC SOCIETY OF INDIA, JHARKHAND BRANCH

The GSI members from Jharkhand unanimously passed the resolution to create Jharkhand Branch of Geriatric Society of India, and formed the Executive Committee on 31.03.2023 for the same with following details :

(a) Name of the Branch - Geriatric Society of India - Jharkhand Branch

Address: H- 85 (SANSKRITI), Harmu Housing Colony, Ranchi-834002

(b) Chairman : Dr. J.K. Mitra, (L-1435)

(c) Vice chairman:
- (i) Dr. Sanjay Kumar Singh (L-1419)
- (ii) Dr. Sudhir Kumar (L-1182)

(d) Gen Secretary: Dr. (Col) Pramod Kumar (L-1205)

(e) Asst Secretary:
- (i) Dr Manju Mishra (L-1240)
- (ii) Dr. Gagan Gunjan (L-1421)

(f) Treasure : Dr. R.T. Guria (L-1417)

(g) Co- Treasurer: Dr. Sujeet Marandi (L1418)

(h) Executive Committee Members:
- (i) Dr. D.K. Sinha (L-1416)
- (ii) Dr. Arun Sarkar (L-1420)
- (iii) Dr. Ajay Kr Bakhla (L-1439)
- (iv) Dr. Ajit Dungdung (L-1434)
- (v) Dr Mrityunjay Mundu (L-1438)

(j) Life Members:
- (i) Dr. Kamal Kanti lal (L-941)
- (ii) Dr. Surya Kr Basu (L-1437)
- (iii) Dr. Lakshman Mandal (L-1436)
- (iv) Dr. Gajendra Kumar (L-1433)

Prof Dr.J K Mitra
Chairman,GSI Jharkhand

Dr.(Col) Pramod Kumar
Branch General Secreatry
,GSI Jharkhand

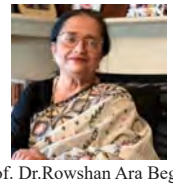
Dr. R T Guria
Treasurer,
GSI Jharkhand Branch

Members of GSI of Jharkhand State at the installation of the branch

News from Bangladesh

On 31.03.2023, doctors of different discipline met in a Zoom meeting and declared installation of "Geriatric Society of Bangladesh". Dr. Kaushik Ranjan Das, President GSI & Dr. Jyotirmoy Pal had joined virtually. GSI overseas members (Bangladesh) have worked hard under the continuous motivational effort of Dr. Kaushik Ranjan Das and made it in reality. Convener Geriatric Society of Bangadesh being Prof.Dr.Rowshan Ara Begum and Member Secretary being Dr.Sushanto Ghosh (Heroic Freedom Fighter);person instrumental being Dr.Subrato Ghosh ,who is also Bangladesh coordinator of GSI.GSI assures all possible help toward furtherance of Geriatrics in Bangladesh.

Chief Patron
Dr.Subhagata Choudhury
Geriatric Society of Bangladesh



Prof. Dr.Rowshan Ara Begum
Convenor
Geriatric Society of Bangladesh

Dr.Sushanto Ghosh
Member Secretary
Geriatric Society of Bangladesh

Dr. Subrato Ghosh
Coordinator
Geriatric Society of Bangladesh

CONGRATULATION DR. ANAND P. AMBALI

The National Institute of Social Defense (NISD), New Delhi organized two days National Conference on Elderly Issues on 24th March 2023 and 25 March 2023 in New Delhi. Dr. Anand P. Ambali was invited as Guest Faculty and his topic was Rural Geriatric Health Care Services which was scheduled on 25th March 2023. He discussed in detail regarding the need for rural health services exclusively for senior citizens, challenges faced and how we have overcome through our program "Reaching the Unreached".

- The heads of various NGO committees, students of geriatric care course and office bearers of NISD had attended the program. Dr. Indira Murthy, Joint secretary of Ministry of Social Justice and Empowerment, Government of India felicitated Dr. Anand P. Ambali.

Dr. Giriraj, Director Dr. H. C. Sridhara Channakeshava Ranga Reddy, Deputy Director of NISD were also present.

LETTER TO EDITOR

Letter to Editor

Dear Sir,

Ever since the Department Of Geriatric Medicine was started in Goa medical College on the 1st of October 2021, we have noticed that the lack of trained caregivers is the main cause of morbidity and readmissions in the Department.

In a scenario where both the family members are working and the grandchildren are schooling, there is deficiency of trained caregivers both in the hospital and at home (once the patient is discharged) to look after the elderly who have multiple co morbidity.

Although there are well defined modules to train caregivers under the National Health Care of the Elderly, there are no dedicated caregivers in the community.

Like paramedical staff, (Nurses, Occupational Therapists, and Physiotherapists) a pool of caregivers has to be built to take care of the elderly.

Thanking You,
Yours Sincerely

Dr. Edwin Gomes,
Professor and Head,
Department Of Geriatric Medicine,
Goa Medical College

With Best Compliments From

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Consultant Physician & Geriatrician
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Dr. (Mrs.) Rita Gulati
M.D., D.G.O., F.I.A.M.S.
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Dr. Dishant Gulati
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Consultant Physician
(Former Assistant Professor SGT Medical College, Budera (Gurgaon)
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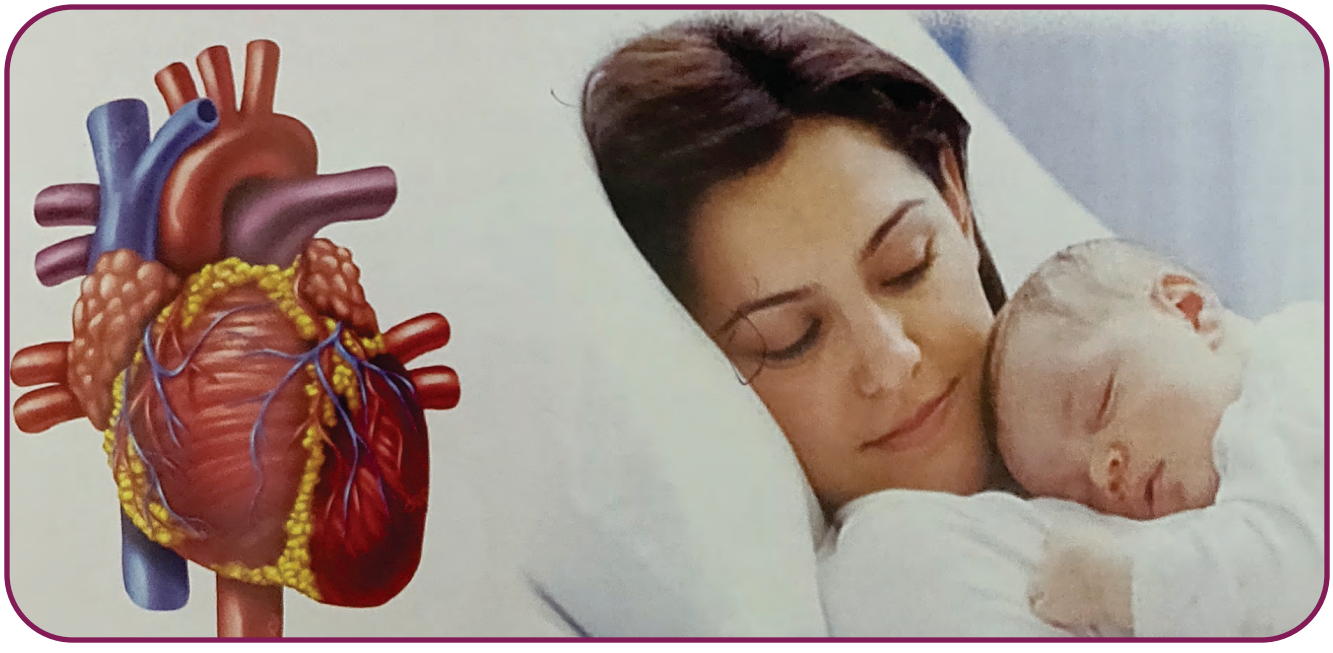
Cell: +91-9996233397



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