

International Journal Of Medical Science And Clinical Inventions

Volume 3 issue 4 2016 page no. 1751-1757 e-ISSN: 2348-991X p-ISSN: 2454-9576

Available Online At: <http://valleyinternational.net/index.php/our-jou/ijmsci>

Postoperative Delirium In The Elderly Patients.

Haxhire Gani¹, Vjollca Beqiri², Majlinda Naco³, Aurel Janko⁴, Bilbil Hoxha⁵, Rudin Domi⁶.
UHC. "Mother Teresa", Dept. of Surgery, Tirana, Albania.

Summary.

Age-related changes in physiology and pharmacology can affect every aspect of perioperative care. The purpose of this study was to describe the natural history, identify risk factors, and determine outcomes for the development of postoperative delirium in the elderly.

Pathogenesis :The underlying mechanisms contributing to delirium are poorly understood. Risk factors for delirium: The following are risk factors which are associated with an increased risk of delirium: Age ≥ 65 years. Male sex. Pre-existing cognitive deficit, e.g. dementia, stroke. Severity of dementia. Co-existing medical problems etc. The recognition and identification of appropriate interventions for patients with acute postoperative delirium continues to challenge perianesthesia nurses, anesthesia care providers, and other health care providers.

Introduction:

Age-related diseases such as cerebral arteriosclerosis, Alzheimer's and Parkinson's disease are all more common with advancing age. Most strokes affect those older than 70 years and the risk doubles every 10 years after age 55. The prevalence rates for dementia and Alzheimer's disease double approximately every five years from rates of 2 to 3 percent in the age category of 65 to 75 years to more than 30 percent in persons age 85 and older. Onset of symptoms in Parkinson's disease usually occurs between ages 60 and 69, although in 5 percent of patients the first signs are seen prior to age 40. About 1 percent of persons age 65 and older and 2.5 percent of those older than age 80 have Parkinson's disease.^[1,2,3] Increase in magnitude with advancing age, represent aging. Effects of aging on the nervous system include: selective attrition of cerebral and cerebellar cortical neurons neuron loss within certain areas of the thalamus, locus ceruleus, and basal ganglia, general reduction in neuron density, with loss of 30 percent of brain mass by age 80 decreased numbers of serotonin receptors in the cortex, reduced levels of acetylcholine and acetylcholine receptors in several regions of the brain decreased levels of dopamine in the neostriatum and substantia nigra and reduced numbers of dopamine receptors in the neostriatum. The association of serotonergic, cholinergic and dopaminergic systems, respectively with mood, memory, and motor function, may partially account for depression, loss of memory and motor

dysfunction in the elderly. According^[15] to the American Psychiatric Association, Delirium is defined as "a disturbance of consciousness with the reduction of the ability to focus, sustain, or change in focus, a change in the recognition (memory deficit, disorientation, spoken of untidiness) or the development of perception mess. Postoperative complications specific to elderly surgical patients such as delirium will be increasingly relevant in the coming decades. The prevalence of post-operative delirium in elderly patients ranges from 0% to 73%, depending on the study and type of surgery^[29]. Post-operative delirium is a medical emergency, which can occur within hours of surgery and has the potential to last up to 7 days^[30]. At least a quarter of elderly patients who develop delirium post-operatively may continue to have symptoms for up to 6 months after hospital discharge^[31]. Delirium postoperative has been associated with increased morbidity and mortality and long hospital stay. With increase of the average age the elderly surgery has increases as well. **The purpose of this selective review is to recognize risk factors, and strategies for prevention and treatment of acute postoperative delirium in elderly patients.** The reason of Delirium postoperatively to elderly patients is multifactorial.

Pathogenesis : Many theories emphasize aberrant neurotransmission. One of the most widely accepted mechanisms is cholinergic deficiency; increased serum

anticholinergic activity is associated with delirium^[4] Other hypotheses invoke abnormalities in melatonin and serotonin,^[5,6] with abnormal tryptophan metabolism unifying these ideas because tryptophan is Neuronal damage is an alternative explanation, secondary either to oxidative stress.^[7] or inflammation. Proinflammatory cytokines increase in postoperative delirium,^[9] especially interleukin-6 and interleukin-8.^[8] In addition, elevations in C-reactive protein occur in delirious patients. A link between inflammation and neurotransmission has been proposed, with inflammation-induced perivascular edema leading to hypoxia and subsequent reduced synthesis of acetylcholine.^[10] It is generally thought that delirium represents global brain dysfunction. Electroencephalographic findings reveal a decrease in the fast alpha frequencies and an increase in the slower theta rhythm.^[11] In hypoactive delirium, hypoperfusion occurs globally in the frontal, temporal, and occipital lobes, and focally in the caudate head, thalamus, and lenticular nuclei. Delirium improves once blood flow returns to normal, suggesting that cerebral hypoperfusion may play a role.^[12]

Mini-Mental State Examination.^[13]

This test takes about 10-15 minutes, and requires some written material for the last test.

Score Results:

30 – 29 Normal

28 – 26 Borderline cognitive dysfunction

25 – 18 Marked cognitive dysfunction – may be diagnosed as demented

<17 Severe dysfunction – severe dementia

CAM^[14]

Score Section Task

Orientation

5. What is – the year, season, date, day, month

5. Where are we – country, county, town, hospital, floor

Registration

3. Name 3 objects – 1 second to say each, then ask patient to recall all three. Repeat until the patient has learnt all three.

Count and record trials

5. Serial 7s – one point for each correct. Stop after 5 correct. Alternatively – spell ‘world’ backwards

3. Ask for the 3 objects repeated above – Give an example of each.

Language

2. Name a pencil and a watch

1. Repeat the following ‘ no ifs, ands, or buts’

3. Follow a 3-stage command: ‘take a paper in your right hand, fold it in half, and put it on the floor.’

3. Read and obey the following: ‘close your eyes’, ‘write a sentence’, ‘copy a design’

30 Total Score

Co-existing medical problems .In 1995, Dyer et al^[16] More recently, Edlund et al^[17] also stressed the importance of identifying riskfactors for delirium and the mechanisms involved in the development of delirium. Inouye^[18] describes delirium as typically resulting from a combined action of predisposing and precipitating factors.

An inter-relationship exists between patient vulnerability to delirium at the time of admission into the hospital (predisposing factors) and the occurrence of noxious insults during hospitalization (precipitating factors). Predisposing factors include vision impairment, severe illness, cognitive impairment, and dehydration.^[18] Increasing numbers of predisposing factors lead to increasing risk of delirium development. Precipitating factors include the use of physical restraints, malnutrition, the addition of more than 3 new medications on the previous day, use of an indwelling bladder catheter, and any iatrogenic event (including complications of diagnostic or therapeutic procedures, transfusion reactions or adverse drug reactions).^[18] Again, multiple precipitating factors lead to increasing risk. Targeting preventive strategies toward these riskfactors can reduce the development of delirium in hospitalized older patients by 40%.³ Various investigators cite differing conditions associated with the onset of delirium. The most prevalent conditions associated with the pathogenesis of delirium include pharmacologic agents, dehydration, hypoxia, infection (especially upper respiratory and urinary tract), metabolic disturbances, and nutritional deficiencies.^[16] Dyer et al found that age, preoperative cognitive

impairment, and the use of anticholinergic drugs were significantly associated with delirium, whereas gender, type and route of anesthesia, and sleep deprivation were not. Older patients are at higher risk than younger patients. Preexisting cognitive impairment is a risk factor. Higher rates of postoperative delirium are seen in patients with preexisting central nervous system disorders, such as dementia and Parkinson's disease.^[17] Depression is significantly associated with postoperative delirium. Disorientation can occur quickly in the unfamiliar hospital surroundings and with the various auditory and visual stimuli leading to sensory overload in busy PACUs and ICUs.

Clinical Mercantonio et al identified clinical predictors for the development of postoperative delirium. They are age 70 years or older; alcohol abuse; preoperative Telephone Interview for Cognitive Status (TICS) score of 30 or less (TICS, a modification of the Mini-Mental Status Exam [MMSE], does not require written responses and is comparable to the MMSE in validity and reliability. Scores below 30 indicate impaired cognition); Specific Activity Scale (SAS) class IV (class IV represents severe physical impairment); markedly abnormal preoperative serum sodium, potassium, or glucose level; aortic aneurysm surgery; and noncardiac thoracic surgery. Patients with no risk factors (0 points) had a rate of less than 1% postoperative delirium; those with one point, a rate of 8%; patients with 2 points, a rate of 19%; and patients with 3 or more points, a rate of 45%. Postoperative delirium also is associated with other adverse outcomes.^[19] These outcomes may include cardiac arrest, ventricular tachycardia or fibrillation, myocardial infarction, pulmonary edema, pulmonary embolus, bacterial pneumonia, respiratory failure requiring intubation, renal failure requiring dialysis, and stroke. DENISE O'BRIEN. Mercantonio et al^[19] found that in patients in whom delirium developed, the rate of major complications was 15% compared with a 2% rate among patients who did not have delirium. Mortality rate in patients with postoperative delirium was 4% compared with 0.3% mortality rate in patients who did not have delirium. Mean length of stay for patients with postoperative delirium was

15 days compared with 7 in patients without delirium. Rate of discharge to new long-term care or rehabilitation facilities was 36% for patients with postoperative delirium, whereas patients without delirium went to new facilities 11% of the time.

Biochemical Derangements: Metabolic alterations can also precipitate the onset of delirium. These alterations include dehydration, hyponatremia, hyperglycemia, hypoglycemia, acid-base disorders, hypercalcemia, hyperphosphatemia, and hepatic, renal, and endocrine disease.

Drugs, Anesthetics, and Intraoperative Risks:

Cholinergic pathways appear to play a significant role in the pathogenesis of delirium.^[20] Serotonin, norepinephrine, and other neurotransmitters have been implicated in the development of delirium. Medications associated with delirium include anticholinergic drugs, anesthetics, opioids, antihistamines, antiasthmatic agents, benzodiazepines, antiparkinsonian agents, histamine₂-receptor antagonists, antihypertensive and cardiovascular agents, antimicrobials, corticosteroids, immunosuppressive agents, anticonvulsants, anti-inflammatory agents, muscle relaxants, and oral hypoglycemics.^[20] Mercantonio et al^[21] found that meperidine and benzodiazepines (long-acting and higher doses more significant) were significantly associated with delirium that occurred in postoperative patients and recommended the use of alternative therapies whenever possible. Ketamine, in use for many years, is well known for its potential to precipitate emergence delirium in patients. The product insert states "emergence reactions have occurred in approximately 12% of patients." The incidence of these reactions is least among the young (under 15 years old) and the elderly (over 65 years of age).^[21] Mercantonio et al^[22] identified occurrence of postoperative delirium associated with greater intraoperative blood loss, more postoperative blood transfusions, and postoperative hematocrit level less than 30%. Route of anesthesia and intraoperative hemodynamic complications such as hypotension were not associated with postoperative delirium. **Postoperative Factors/Hypoxemia** In acute emergence delirium in the postanesthesia care unit, hypoxemia is always considered the primary cause until

proven otherwise.^[23] Pulse oximetry is essential in the monitoring of the postoperative patient.

Risk Factor Points

Age 70 yr 1

Alcohol abuse 1

TICS score 30*1

SAS class IV† 1

Markedly abnormal preoperative sodium,potassium, or glucose level‡ 1

Aortic aneurysm surgery 2

Noncardiac thoracic surgery 1

Total Points Risk of Delirium (%)

0- 2% / 1 or 2- 11% / 3- 50%

*TICS indicates Telephone Interview for Cognitive Status (scores less than 30 suggest cognitive impairment). †SAS indicates Specific Activity Scale (class IV represents severe physical impairment). ‡Markedly abnormal levels were defined as follows: sodium, less than 130 or greater than 150 mmol/L; potassium, less than 3.0 or greater than 6.0 mmol/L; or glucose, less than 3.3 or greater than 16.7 mmol/L (60 or 300 mg/dL). Estimates of risk were based on the true incidence of delirium in the validation population.

Etiologic Factors/Preoperative. 1. Brain affectation caused by. Physiologic causes—aging. Pathologic causes—congenital, traumatic, neoplastic, vascular, idiopathic

2. Drugs. Drug polypharmacy. Drug intoxication or withdrawal

3. Endocrine and metabolic. Hyper/hypothyroidism. Hyponatremia. Hypoglycemia

4. Mental status. Depression. Dementia. Anxiety. Gender

Intraoperatively. Type of surgery. Orthopedic. Ophthalmic. Cardiac

2. Duration of surgery

3. Anesthetic drugs used

4. Type of anesthesia used—general versus regional

5. Complications during surgery. Hypotension. Hyperventilation.

Embolism. Hypoxemia

Postoperative. Hypoxia. Respiratory causes. Perioperative hypoxia. Residual anesthetics

. Hypocarbia,. Pain,. Sepsis,. Sensory deprivation or overload,. Electrolyte or metabolic problem

Prevention of Postoperative Delirium

Preoperative assessment. Detailed history of drugs. Medical problem evaluation

Detection of sensory or perceptual deficits. Mental preparation before surgery. Neuropsychiatric testing. Use of geriatric-anesthesiologic program

Intraoperative precautions. Adequate oxygenation and perfusion. Correct the electrolyte imbalance. Adjust drug dose. Minimize the variety of drugs. Avoid atropine, flurazepam, scopolamine.

Postoperative care. Environmental support. Well-lit cheerful room. Quiet surroundings. Keep patient oriented. Visit by friend or family. Treat pain. Identify risk-associated drugs. Anticholinergics. Depressants, H₂ antagonists. Digoxin, lidocaine. Meperidine Benzodiazepines. Reassure patient and family

Medical management: Using drugs to treat delirium can lead to adverse effects and worsening of delirium; therefore, careful consideration is required.

Antipsychotics have beneficial effects in selected patients who are agitated and their onset is relatively fast. Haloperidol is preferred, although there is a risk of extrapyramidal side-effects (EPSEs). Small doses should be used, e.g. 0.5-2.0 mg PO/IM/IV. It may be appropriate to consider a lower dose in elderly patients, e.g. 0.5 mg. This can be repeated after 30 minutes if there is no response. Lorazepam is generally the first choice of benzodiazepines, as it has a rapid onset of action and a short duration. Lorazepam can be used alone or with low doses of haloperidol if there are concerns regarding EPSEs, e.g. Parkinson's disease or Lewy body dementia. A starting dose of 0.5-1 mg PO can be given 2-hourly or 0.5-1 mg IV/IM (both to a maximum of 3 mg in a 24-hour period). Atypical antipsychotics have also been used; however, olanzapine and risperidone should be avoided in the elderly, as they have been associated with an increased risk of cerebrovascular disease.^[24] Awareness of high-risk patients and subsequent close observation for delirium with prompt

assessment and management can potentially reduce morbidity and mortality. One study has looked at methods of reducing postoperative delirium in patients with multiple risk factors.^[25] Their results are interesting and suggest that simple factors like hyperglycaemia, poor nutritional and poor functional states (e.g. delayed mobility) - all of which are easily prevented - account for adverse clinical outcomes in a subset of patients. The National Institute for Health and Clinical Excellence (NICE) has thus recommended a "tailored team approach to the prevention of delirium. Patients should be assessed within 24 hours of admission, making note of factors that may precipitate and worsen delirium. There are various interventions listed in the NICE guidance, based on the identified clinical factors - for example: Cognitive impairment or disorientation - provide appropriate lighting and regularly orientate the person. Promote cognitively stimulating activities and regular visits from people well known to the patient. Hypoxia - identify and correct with the appropriate amount of oxygen.

Pain - assess verbally and non-verbally and treat. Medications - should be reviewed on a daily basis and non-essential medication stopped. Other factors include dehydration, constipation, reduced mobility, infection, poor nutrition, sensory impairment and sleep disturbance. Further education of medical staff and awareness are required. Guidelines may be effective in the management of delirium. This has been studied by one group and they reported that guidelines reinforced by teaching sessions are effective - although statistical significance was not reached.

Future Considerations. Optimizing patients preoperatively to minimize clinical predictors and known risk factors is the goal. Research is needed in the area of acute postoperative delirium to better define the phenomenon, its incidence, risk factors and causes; enhance recognition; and develop additional interventions. Unfortunately, much of the literature on postoperative delirium is over 10 years old. Some basic work defining acute delirium needs to be accomplished before new studies are undertaken on specific acute postoperative delirium. Schuurmans et al^[27] recommend that to change clinical practice and improve

early recognition the following should occur: 1. The word *confusion* should be dropped; it causes misunderstanding in the process of observation and diagnosis. Replacing *confusion* with exact behavioral descriptions would help nurses describe delirium more accurately.

2. Emphasis should be given to nurses' views and knowledge related to health in older patients. Basic nursing curricula should include knowledge of delirium, its causes, and risk factors. 3. Instruments for systematic screening of symptoms need further study related to reliability, validity, and ease of use in the clinical practice setting. Foreman^[26] suggests consensus in nomenclature to facilitate communication. Study designs must incorporate fundamental characteristics of acute delirium and phenomenologic approaches be undertaken to gain insight into the human experience of delirium. Also, Foreman^[26] suggests that instrumentation be improved. Instruments are needed that are practical, sensitive and specific, and discriminating. Franco et al^[28] believe that substantially reduced health care costs may be one of the outcomes of future interventions designed to improve clinical outcomes and lower complication rates.

Bibliography:

1. Muravchick S. The physiologic and pharmacologic implications of aging. 37th Annual Refresher Course Lectures and Clinical Update Program. American Society of Anesthesiologists. 1986; No. 275
2. The Aging Brain. *Geriatrics*. 1998; 53.
3. Hendrie HC. Epidemiology of Alzheimer's disease. *Geriatrics*. 1997; 52:S4-S8.
4. Uitti RJ. Tremor: How to determine if the patient has Parkinson's disease. *Geriatrics*. 1998; 53:30-36.
4. Mussi C, Ferrari R, Ascari S, et al. Importance of serum anticholinergic activity in the assessment of elderly patients with delirium. *J Geriatr Psychiatry Neurol* 1999; 12(2):82-6.
5. Balan S, Leibovitz A, Zila SO, et al. The relation between the clinical subtypes of

- delirium and the urinary level of 6-SMT. *J Neuropsychiatry Clin Neurosci* 2003; 15(3):363–6.
6. Lewis MC, Barnett SR. Postoperative delirium: the tryptophan dysregulation model. *Med Hypotheses* 2004;63(3):402–6.
7. Karlidag R, Unal S, Sezer OH, et al. The role of oxidative stress in postoperative delirium. *Gen Hosp Psychiatry* 2006;28(5):418–23.
8. Rudolph JL, Ramlawi B, Kuchel GA, et al. Chemokines are associated with delirium after cardiac surgery. *J Gerontol A Biol Sci Med Sci* 2008;63A(2):184–9.
9. de Rooij SE, van Munster BC, Korevaar JC, et al. Cytokines and acute phase response in delirium. *J Psychosom Res* 2007;62(5):521–5.
10. Hala M. Pathophysiology of postoperative delirium: systemic inflammation as a response to surgical trauma causes diffuse microcirculatory impairment. *Med Hypotheses* 2007;68(1):194–6.
11. Plaschke K, Hill H, Engelhardt R, et al. EEG changes and serum anticholinergic activity measured in patients with delirium in the intensive care unit. *Anaesthesia* 2007;62(12):1217–23.
12. Yokota H, Ogawa S, Kurokawa A, et al. Regional cerebral blood flow in delirium patients. *Psychiatry Clin Neurosci* 2003;57(3):337–9.
13. Folstein MF, Folstein SE, McHugh PR. A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatry Research*. 1975; 12: 189-98
14. Mercantonio ER, Goldman L, Mangione CM, et al: A clinical prediction rule for delirium after elective noncardiac surgery [Electronic version]. *JAMA* 271:134-139, 1994
15. American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders (ed 4)—Text revision (DSMIV-TR). Washington, DC, American Psychiatric Association, 2000
16. Dyer CB, Ashton CM, Teasdale TA: Postoperative delirium: A review of 80 primary data-collection studies [Electronic version]. *Arch Intern Med* 155:461-465, 1995
17. Edlund A, Lundstrom M, Brannstrom B, et al: Delirium before and after operation for femoral neckfracture [Electronic version]. *J Am Geriatr Soc* 49:1335-1340, 2001
18. Inouye SK: Prevention of delirium in hospitalized older patients: Riskfactors and targeted intervention strategies [Electronic version]. *Ann Med* 32:257-263, 2000
19. Mercantonio ER, Goldman L, Mangione CM, et al: A clinical prediction rule for delirium after elective noncardiac surgery [Electronic version]. *JAMA* 271:134-139, 1994
20. Drugs in the Peri-operative Period. *Drugs and Therapeutics Bulletin* 1999; 37: 62-64
21. Mercantonio ER, Juarez G, Goldman L, et al: The relationship of postoperative delirium with psychoactive medications [Electronic version]. *JAMA* 272:1518-1522, 1994
22. King Pharmaceuticals, Inc: Ketalar CIII Product information, 2000
23. Litwack K: Post Anesthesia Care Nursing. St Louis, MO, Mosby, 1995
24. Young J, Inouye SK; Delirium in older people. *BMJ* 2007;334:842-846
25. Rockwood K; Need we do so badly in managing delirium in elderly patients? *Age Ageing*. 2003 Sep;32(5):473-4.
26. Foreman MD: Acute confusion in the elderly. *Annu Rev Nurs Res* 11:3-30, 1993
27. Schuurmans MJ, Duursma SA, Shortridge-Baggett LM: Early recognition of delirium: Review of the literature [Electronic version]. *J Clin Nurs* 10:721-729, 2001
28. Franco K, Litaker D, Locala J, et al: The cost of delirium in the surgical patient [Electronic version]. *Psychosomatics* 42:68-73, 2001
29. Dyer CB, Ashton CM, Teasdale TA: Postoperative delirium. A review of 80 primary data-collection studies.

Arch Intern Med 1995, **155**:461-465. [PubMed Abstract](#) |
[Publisher Full Text](#)

30. Dodds C, Kumar CM, Servin F: *Oxford Anaesthesia Library: Anaesthesia for the elderly patient*. 1st edition. Oxford: Oxford University Press; 2007.

31. Levkoff SE, Marcantonio ER: Delirium: a major diagnostic and therapeutic challenge for clinicians caring for the elderly. *Compr Ther* 1994, **20**:550-557. [PubMed Abstract](#)